



APASL 2026

Istanbul

**35TH ANNUAL MEETING OF THE ASIAN PACIFIC ASSOCIATION
FOR THE STUDY OF THE LIVER**

22-25 April 2026
Istanbul / Türkiye

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- 1353 - 30% of Inactive HBsAg Carriers Achieved Clinical Cure at 48 Weeks with Pegylated Interferon Alpha-2b Therapy: A Multicenter Real-World Study (STARHB Project in China)- 3 years Data Update, Shan Ren
23 April 2026, Thursday | 17:10 - 18:30 | ORAL PRESENTATION 11
- 1356 - Severe Hepatic Steatosis Impairs PEG-IFN Response in HBsAg Clearance-Favorable Chronic Hepatitis B: A Retrospective Cohort Study, Pengxuan Wu
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 64
- 1361 - L-Taurine for Fatigue in Decompensated Cirrhosis: A Randomized Controlled Trial, Swarup Sasidharan
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- 1362 - Cell-Microcapsule-Loaded Potent Nanoenzymes Alleviate Drug-Induced Liver Injury via Dual Strategies, Xiaoyu Huang
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 07
- 1375 - Multidimensional analysis of diagnosis and treatment status of chronic hepatitis B, Yujuan Guan
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- 1377 - Computed tomography-based sarcopenic obesity can predict cardiovascular and cerebrovascular mortality in patients with cirrhosis, Yanhua Chen
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- 1378 - Leucine-rich alpha-2-glycoprotein 1 turnover imbalance impairs regenerative signaling in Alcohol-associated Hepatitis, Nupur Sharma
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 44
- 1380 - The SAFE-DILI Model for the Dynamic Prediction of Liver Transplantation or Death in Idiosyncratic Drug-Induced Liver Injury, Mengmeng Zhang
24 April 2026, Friday | 09:00 - 10:30 | ORAL PRESIDENTIAL SESSION
- 1384 - Hepatitis A and E Guidelines created by the Japan Agency for Medical Research and Development (AMED) HAV and HEV Study Group, Tatsuo Kanda
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 01
- 1385 - Direct chemical reprogramming of urine-derived stem cells from acute-on-chronic liver failure patients into hepatocyte-like organoids to attenuate liver injury, Pengfei Yu
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 01
- 1387 - The transcription factor PRDM5 is a novel pro-fibrotic factor in liver fibrosis, Chaiyaboot Ariyachet
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 05
- 1389 - Variability of Presentation of Cholelithiasis in Relation to Etiology and Stages of Cirrhosis of Liver in a Tertiary Care Hospital in Dhaka City, M Golam Azam Azam MG
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 28
- 1393 - Optimal timing of hepatectomy after conversion therapy and stratification by risk of recurrence in patients with hepatocellular carcinoma: a multicenter retrospective investigation (GUIDANCE005), Da-Long Yang
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 31
- 1396 - Exploring the efficacy of Systemic Inflammatory Markers in predicting outcomes post Transarterial Chemoembolization (c TACE & deb TACE), RAJAT PRABHAKAR PANDE
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 31
- 1399 - Trajectory-Based Multidimensional Risk Scoring in a Single-Center Retrospective Cohort: Exploratory Development for Predicting Poor HBsAg Seroclearance Response in Chronic Hepatitis B Functional Cure, Lin Tuo
23 April 2026, Thursday | 17:10 - 18:30 | ORAL PRESENTATION 11
- 1401 - Effects of the secondary metabolites of Terminalia arjuna on non-alcoholic fatty liver disease in experimental mice model, Mst. Rejina Afrin
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37
- 1408 - Predicting Recompensation and Exploring the Prognostic Benefit of its Durability in patients with HBV-related decompensated cirrhosis, Yuwei Wang
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 28
- 1410 - Characterization of Serum Cytokine Profile and Its Early-Warning Value in Elderly Patients with Hepatitis E, Zhao Qian
23 April 2026, Thursday | 17:10 - 18:30 | ORAL PRESENTATION 11
- 1414 - Multi-omics integration analysis identifies Molecular Signatures of Liver Stress in preclinical model of Acute Hypoxia, Sanju Yadav
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 1416 - PPAR γ Activation Restores GSDME Pyroptosis to Reverse Anti-PD-1 Resistance in HCC, Peng Chen
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 31
- 1417 - The Clarity Trial: Prospective evaluation of pemafibrate therapy in MASLD, Takefumi Kimura
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37

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- 1418 - Efficacy of Sulfasalazine in Patients with Primary Biliary Cholangitis and Incomplete Response to Ursodeoxycholic Acid, Li Wei
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 45
- 1419 - Evodiamine alleviates liver fibrosis by targeting the ANXA2/PI3K/AKT/CCND1 axis in hepatic stellate cells, Zongyi Liu
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 28
- 1422 - Unveiling the pathogenesis and early warning biomarkers of acute liver failure associated with disulfidptosis through bioinformatics analysis and experimental validation, Li Bai
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 20
- 1423 - Characterisation of people living with chronic hepatitis B virus infection in China and stratification by HBsAg levels, Myriam Drysdale
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 26
- 1426 - Double plasma molecular adsorption system improves 90-day survival in patients with acute-on-chronic liver failure, Xiao Che
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 20
- 1431 - Persistent sarcopenia confers a cumulative risk of fibrosis progression and liver-related events in biopsy-proven metabolic dysfunction-associated fatty liver disease, Siru Wu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37
- 1436 - HBV Inhibits Stress Granule Formation by Hijacking SG Core Proteins into the Nucleocapsids, Yi Wang
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 26
- 1447 - N acetyl cysteine plus corticosteroid versus corticosteroid alone for treatment of acute severe autoimmune hepatitis: a randomized clinical trial, Ahad Eshraghian
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 04
- 1456 - Determination of Ankyrin Repeats Domain (ANK) of RNASE L Gene in Hepatitis C Patients and its effects on Viral Load, anum LIAQUAT ALI
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 08
- 1459 - Revisiting HepaScore: New Evidence for Non-Invasive Fibrosis Assessment in Indonesian Patients with Chronic Hepatitis B, Meti Metiani
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 64
- 1461 - A Retrospective Study on PD-1/CTLA-4 Bispecific Antibody Therapy Promoting Partial Functional Cure of Hepatitis B in Patients with HBV-Related Hepatocellular Carcinoma, Chuyue Xiong
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 65
- 1463 - Real-World Outcomes of Durvalumab Plus Tremelimumab (STRIDE) for Unresectable Hepatocellular Carcinoma in Japanese Patients, Yuta Maruki
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 31
- 1466 - A new prediction model based on plasma ammonia and the Chinese Group on the Study of Severe Hepatitis B-ACLF-8545; score for patients with Hepatitis B virus-related acute-on-chronic liver failure., Yijing Cai
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 65
- 1468 - Multicenter evaluation of the efficacy and safety of rechallenge with immune checkpoint inhibitors in unresectable hepatocellular carcinoma, Jian-Hong Zhong
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 31
- 1469 - Targeting NEK9 in hepatocellular carcinoma to remodel immune microenvironment and enhance immunotherapy, Yulong Shang
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1471 - Biomolecular Mapping of Circulating Extracellular Vesicles to Develop AI Driven Prognostic Panels for Stratifying Acute Liver Failure Patients at Risk of Early Mortality, Neha Sharma
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 20
- 1472 - Divergent immune resolution and protracted immuno-metabolic memory in hepatitis E revealed by cross-species multi-omics, Xiaoman Liu
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 65
- 1473 - Dominant Neoantigens in Liver Cancer Malignant Ascites, Dongbo Chen
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35

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- 1476 - Prediction of microvascular invasion in HCC using spatiotemporal radiomics of contrast-enhanced ultrasound: a deep learning model with tumor microenvironment correlation, Chuan Pang
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- 1481 - Development and validation of brain age clock from psychometric hepatic encephalopathy score for early detection of hepatic encephalopathy, Xin Wen
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 15
- 1482 - From Gene Expression Profiles to Functional Networks: A Multi-Omics Integrative Analysis of Methyl Eugenol Promoting Cholangiocarcinoma Development, Jing Liang
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 45
- 1484 - Exploring the Anticancer Effects of Spirulina Extract in Human Hepatocellular Carcinoma Cells, Ceylan KÜPELİ
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 1486 - Serum bile acids profile analysis predicts severity and progression of liver disease, Yanpei Mai
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 21
- 1493 - Clinical determinants of treatment response in Asian hepatocellular carcinoma undergoing lenvatinib therapy: the impact of non-viral liver disease etiologies, Lu Li
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1494 - Distinct Clinical Profile of Wilson Disease-Related Cirrhosis: Insights from a Propensity Score-Matched Multicenter Study, Zhenhua Zhang
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 08
- 1497 - A Phase I Clinical Trial Evaluating the Safety and Efficacy of the QH-1A Combination Bio-artificial Liver Support System Incorporating Ex-vivo Mesenchymal Stem Cells for the Treatment of ACLF patients, Liang Peng
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 20
- 1507 - Hyperammonemia in pre-hepatic and intrahepatic portal hypertension: comparable levels, different clinical outcomes, Maria Nadinskaia
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 28
- 1516 - Serum HBP as a Mechanistic Predictor of Primary Resistance to Atezolizumab plus Bevacizumab in HCC., Satoshi Narahara
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1517 - Hepatitis B serology and vaccine response in uveitis patients treated with adalimumab: effects of the 1998 national hepatitis B vaccination program in Turkey, Fatma Merve Kocak
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 65
- 1525 - A Multi-Sectoral Co-Payment Model to Reduce Hepatitis C Treatment Costs and Advance Elimination Among Low-Income Populations: Experience from Yangjiang, China, Yuelong Lin
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 65
- 1526 - Transcriptional Activity of cccDNA in HBsAg-Negative Individuals with a History of Hepatitis B Virus Infection, Yanfei Feng
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 65
- 1527 - Development and Validation of an Early Diagnostic Model to Distinguish Bacterial Infection in Patients with Acute-on-Chronic Liver Disease (AoCLD) and Systemic Inflammatory Response Syndrome (SIRS), Ruochan Chen
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1528 - Palliative care reduces disease time burden but is under-utilized in terminal-stage hepatocellular carcinoma, Matthew Shing-Hin Chung
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1529 - CD39 Promotes Angiogenesis and Poor Prognosis in Hepatocellular Carcinoma via Activation of the PI3K/AKT Signaling Pathway, Zhengnan Cao
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1531 - Dynamic changes in gut microbiota associated with HBsAg decline in chronic hepatitis B patients undergoing IFN treatment, Demin Yu
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29

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- 1533 - Sex differences in cirrhosis among untreated chronic hepatitis B: a 10-year longitudinal cohort study from China, Qiao Tang
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1542 - Mechanistic Insights into the Protective Effects of Faecalibacterium prausnitzii and Its Metabolite Short Chain Fatty Acids on Biliary Injury in Primary Biliary Cholangitis, Qi Wang
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 27
- 1551 - Non Invasive Detection of Esophageal Varices in Compensated Cirrhotic Patients, Mohamed Abdel-Samiee
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 28
- 1552 - Multi-Omics Integration Identifies a Prognostic Signature Guiding Precision Survival in Advanced HCC, Fengna Yan
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1555 - Concordance Between Ultrasound Steatosis Grading and Elastography-CAP Classification: A Case Series of Chronic HBV Patients on TAF Therapy, Elif Nur Özbay Haliloğlu
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1559 - SIRPA as a Novel Regulator of T Cell Apoptosis in Hepatocellular Carcinoma: Implications for Immunotherapy, Xue Zhang
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 05
- 1560 - Correlation between transient elastography and liver biopsy in CHB patients with elevated HBV DNA with normal ALT, Dr Arifa Tasnim
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1564 - Second-line Therapy after Immunotherapy for Unresectable Hepatocellular Carcinoma: a Multicenter Data Registry Analysis, Takeshi Okamoto
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1565 - The Efficacy and Safety of Pegylated Interferon Alpha Treatment in Patients with HBV Related Cirrhosis and HCC after Radical Surgery (1 Year Data from the TERMINATOR Project), Qiuju Sheng
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1567 - Multi-omics Analysis of Bile Acid Dysregulation in Hepatitis E virus Infection, Conglin Zhao
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1568 - Role of CD146-High Endothelial Cells in Vascular Remodeling and Fibrosis Progression in Cirrhotic Liver: A Single-Cell Transcriptomic Study, Bingqing Yang
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 28
- 1573 - Comparison of ALBI, PALBI, MELD, and Child-Pugh Scores in Chronic Liver Disease: Evidence from Bangladesh, A B M Shakil Gani
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 21
- 1576 - Qilingxiaoji formula and the effective ingredients inhibit hepatocellular carcinoma progression by inhibiting miR32/GPR180 signaling pathway, Yilan Yang
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 35
- 1580 - Prognostic Evaluation of Hepatocellular Carcinoma: Impact of Etiological Factors on Survival Outcomes, ALİ ÇAĞRI ORAL
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1581 - Continuing or Switching Systemic Therapy Combined with Locoregional Therapy Defines a Personalized Strategy for Oligoprogression in HCC: A Multicenter Retrospective Study, Hongli Yu Yu
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1587 - Remodeling of the bile acid connectome across the hepatitis-cirrhosis-hcc axis: network analysis reveals stage-specific hubs and potential therapeutic targets, Qinshuo Wang
24 April 2026, Friday | 09:00 - 10:30 | ORAL PRESIDENTIAL SESSION
- 1588 - Role and Mechanism of TGF β /pSAP Signaling in Regulating Hepatocyte-Macrophage Crosstalk in Metabolic Dysfunction-Associated Steatohepatitis, Zheng Liu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37
- 1589 - Intermittent therapy restores anti-HBV efficacy of T_H1 by reducing elevated dysfunctional HLA-DR+CD38hiCD8+ T lymphocytes, Yanjie Lin
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29

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- 1592 - Fibroscan role in determining the patients who are at high risk for developing HCC in 1000 cirrhotic Egyptian patients., Abdel Ghani Atef Abdel Ghani Badran
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1596 - ABT score based clinical model predicts prognosis in unresectable hepatocellular carcinoma treated with TACE combined with Bevacizumab and Sintilimab therapy, Minjun liao
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1598 - Pneumonia incidence and pathogen spectrum in liver transplant recipients: a decade-long retrospective analysis comparing pre-coronavirus disease 2019 and pandemic-era temporal patterns and risk factors, Zehra İkbāl Karabal
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 18
- 1602 - The Association Analysis of Metabolic Comorbidities and the 10-Year Prognosis of Hepatic Events in Chronic Hepatitis B Patients, Xuejie Zhang
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 29
- 1603 - FcγR2b mediates CD8+ T cells dysfunction in Hepatocellular Carcinoma, Qianting Guan
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1607 - From Dysbiosis to Prediction: A Novel Gut Microbiota-Derived Index for Spontaneous Bacterial Peritonitis in HBV-Related Cirrhosis, Zhewen Zhou
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48
- 1608 - Two-Year Follow-up of a Dose-Escalation Trial of Mesenchymal Stromal Cell Therapy for Decompensated Liver Cirrhosis, Lei Shi
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48
- 1609 - Prediction of Liver-Related Events Using Non-invasive Fibrosis Scores in Patients with Metabolic Dysfunction-Associated Fatty Liver Disease, Yankun Gao
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37
- 1615 - Mechanistic Study of Celiac Sympathetic Denervation in the Treatment of Metabolic Dysfunction-Associated Steatohepatitis (MASH), Yi Xu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37
- 1619 - Safety and Effectiveness of Peginterferon Alfa-2b Combined with Tenofovir Amibufenamide in Patients with Chronic Hepatitis B, Qing-Lei Zeng
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1624 - A Noninvasive Model Based on Liver and Spleen Microcirculatory Perfusion Parameters for Predicting High-Risk Varices in CHB-Related Cirrhosis, Chenglin Sun
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48
- 1625 - Alcohol Consumption Accelerates Liver Fibrosis in Chronic Hepatitis B: Evidence from Mendelian Randomization and a Prospective Cohort Study, Yu Zhang
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 44
- 1629 - Analysis of Risk Factors and Establishment of a Predictive Model for Liver Disease Severity in Non-Alcoholic Fatty Liver Disease, Ziyu Zhang
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 37
- 1634 - Integrated Plasma and Fecal Metabolic Profiling in Alcoholic Cirrhosis: Association with Gut Microbiota Dysbiosis, Yifan Xu
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 44
- 1640 - The 4-hydroxybutyrylation modification of ACSL4 inhibits the growth of hepatocellular carcinoma, Xiangchang Zeng
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1642 - Heme oxygenase-1 inhibiting mitochondrial apoptosis in metabolic associated fatty liver disease, Wen Zhao
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 63
- 1646 - Mapping the Global Network of Viral Hepatitis A, B, C and E: Unveiling Disease-Specific Epidemic Architectures through Complex Network Analysis, Yuxi Shi
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46



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- 1647 - Antiviral Therapy in Patients with HBV Infection in the HBeAg-Negative Indeterminate Phase, Mingyang Feng
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- 1649 - Platelet-Albumin-Bilirubin (PALBI) Score: A non-invasive Predictor of Esophageal Varices in Patients with Liver Cirrhosis Compared to Child-Turcotte-Pugh (CTP) Score, ZANNATUL HABIBA
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 21
- 1652 - Investigating the Role of Gut Microbiota-Derived Valine in Treating MAFLD through PPAR Signaling, minshan huang
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 64
- 1653 - Efficacy of sofosbuvir/velpatasvir in patients with hepatitis C virus genotype 3 infection, Menghan Jin
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1656 - Analysis on the differences of clinicopathological characteristics in drug-induced liver injury patients with positive or negative autoantibodies and different clinical types, Suwen Jiang
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 07
- 1661 - Clinical Characteristics of Autoimmune Hepatitis in Patients with Hepatitis B-D Coinfection: A Case Series, Zain Majid
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1662 - Socioeconomic Disparities and Potential Impact of Delayed Presentation on the Burden of Portal Hypertension in a Bangladeshi Tertiary Care Setting, Md. Sabbir Hossain
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48
- 1664 - Matrix Stiffness Suppresses Hepatitis B Virus Replication via YAP-Mediated Intrinsic Antiviral Signaling, Hua-xiang Yang
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1672 - Hepatic steatosis in Wilson disease: phenotype or metabolic comorbidity?, Veronica Cumpata
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 08
- 1673 - Towards hepatitis B elimination in Ghana: vaccination coverage and its predictors among informal sector workers in Kejetia, Kumasi, Ghana, Pius Takyi
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1676 - Predictive Factors of Tyrosine Kinase Inhibitor Response in Patients with Advanced Hepatocellular Carcinoma, Hayriye Dilan Çağlar
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1677 - Establishment of the TBRI Biobank for Liver Diseases: Advancing Research Translation and Precision Medicine in Hepatology, Eman El-Ahwany
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 1679 - PASSIVE TOBACCO SMOKE INDUCED CHANGES IN THE EXPRESSION OF IRON METABOLISM GENES IN MOUSE PUPS, Naseer Kawish
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 05
- 1690 - Off-Therapy Outcomes After Long-Term Nucleos(t)ide Analogue Treatment in HBeAg-Negative Chronic Hepatitis B, Petek Şarлак Konya
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1694 - Non-Invasive Evaluation of Fibrosis Regression in HBV and HCV: A Real-World Comparative Analysis, Hamza Benazzouz
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 46
- 1698 - Etiological Predictors of Portal Vein Thrombosis in Patients with Porto-Sinusoidal Vascular Disease, rajaa boukhraiss
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 1708 - Males are emerging as the primary high-risk population for gallbladder and biliary tract cancers, Xiaoxiao Wang
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 06
- 1713 - Hepatocyte myb-like swrm and mpn domains 1 exacerbates liver fibrosis by stabilizing gelsolin via k29-linked deubiquitination, Han Ding
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48

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- 1715 - THE OUTCOME PREDICTION ROLE OF RED CELL DISTRIBUTION WIDTH IN 1000 HOSPITALIZED CIRRHOTIC EGYPTIAN PATIENTS, Eman Abdelsameea
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- 1717 - A new switch for lactate accumulation in metabolic dysfunction-associated steatotic liver disease, Miaomiao Tian
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 63
- 1722 - Paraneoplastic Hyperbilirubinemia in Hodgkin's Lymphoma: An Uncommon Presentation with Significant Diagnostic and Therapeutic Challenges, Jasmin Gondayao
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 07
- 1725 - Suboptimal sensitivity of quantitative HBcrAg assay for managing HBeAg-negative chronic hepatitis B, Ruifeng Yang
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 50
- 1726 - Glucagon-like peptide-1 receptor agonists and reduced liver and non-liver complications in patients with type 2 diabetes and viral hepatitis, Xinrong Zhang
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- 1727 - Diagnosis of the differentials of ascites in low-resource settings., Riad Mohammad Moshaed Hossain
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48
- 1729 - The expression of Fmn1 in monocyte-derived macrophages promotes hepatic inflammation in MAFLD, Zilong Wang
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 63
- 1732 - Associations of sweetened beverages consumption with the risk of MASLD and its related outcomes across genetic risk profiles: a prospective population-based cohort study, Jian Zhao
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 63
- 1735 - Policy Blind Spots and Clinical Realities: Determinants of Continued Alcohol Use in Alcohol-Associated Liver Disease at India's First Integrated AUD-ALD Clinic, Mohit Varshney
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 44
- 1738 - Combination Therapy with Toll-like receptor 7 Agonist and Programmed Death Ligand 1 Antibody Restores HBV-Specific CD8+T Cells in Virally suppressed Chronic Hepatitis B, Yuying Chen
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 50
- 1747 - Predictive Biomarkers for Hepatitis E Virus-Related Acute Liver Failure Based on Lipidomics and Transcriptomics, Jian Wu
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 50
- 1753 - Allogeneic Umbilical Cord Mesenchymal Stem Cell Transplantation in Patients with Cirrhosis Caused by Hepatitis B: A Single-Center Case Series, Jason Phowira
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 50
- 1754 - The mechanism of TMBIM5 in ameliorating alcohol-related liver disease by regulating mitochondrial dynamics, Qi Shen Shen
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 44
- 1757 - Impact of etiology on outcomes with atezolizumab plus bevacizumab in advanced hepatocellular carcinoma: A multicenter propensity-matched analysis, Yeo Wool Kang
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1758 - Predictive Role of Spleen Stiffness in Gastrointestinal Bleeding for Patients with Unresectable Hepatocellular Carcinoma Undergoing Systemic Therapy, Yue Huang
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 53
- 1768 - ALT Trajectories Differentiated by Early Treatment Response Determine Long-Term Outcomes in Autoimmune Hepatitis, Chao Cai
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 04
- 1769 - Admission NLR and APRI as Practical, Low-Cost Predictors of Severity in Cirrhosis: Data from an Indian Tertiary Center, UDIT VAISH
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 48
- 1771 - Our Journey Towards a Dedicated Transjugular Intrahepatic Portosystemic Shunt (TIPSS) Service: Teamwork Makes the Dream Work, Hak Keith Leung
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- 1775 - Longitudinal Analysis Reveals Comparable ALT Trajectories and Achievement of Biochemical Remission Regardless of Baseline Fibrosis in Autoimmune Hepatitis, Wenxuan Shang
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- 1776 - Immune characteristics of peripheral blood cells in inactive hepatitis B surface antigen carriers treated with interferon, Sujun Zheng
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 50
- 1781 - An Endothelial-Related Gene Signature Derived from Integrative Transcriptomics Predicts HCC in Cirrhosis, Jiahao Yuan
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 1782 - An Evaluation of the Effectiveness of Entecavir in Patients with Acute Chronic Hepatitis B liver Failure, Sharker Mohammad Shahadat Hossain
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- 1783 - LLGL1 Loss Attenuates Hippo Signaling and Sustains Pro-oncogenic YAP Activity in Hepatocellular Carcinoma Cells, Soner KARABULUT
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 05
- 1785 - Automatic Detection of Hepatocellular Carcinoma on MRI Using a Lesion-Aware Fine-Tuning Framework, Cong Wang
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- 1798 - East Asian subgroup analysis of the global Phase 3 GLISTEN study of linerixibat for the treatment of cholestatic pruritus in patients with primary biliary cholangitis (PBC), Atsushi Tanaka
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 27
- 1802 - Hesperidin inhibits HBV via promoting macrophage M1 polarization through activation of the NF- κ B/CXCL/TNF α pathway, Man Li
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 50
- 1804 - LOXL1 G153D Mutation Impairs Liver Fibrosis Regression by Enhancing Extracellular Matrix Stabilization, Wen Zhang
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- 1805 - Clinical Validation and Diagnostic Value Assessment of Plasma GSTM3 as a Novel Biomarker for Liver Fibrosis, Chenxue Hou
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- 1812 - Clinical characteristics and influencing factors of porto-sinusoidal vascular disease patients with metabolic dysfunction-associated steatotic liver disease, Weihua Cao
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- 1813 - Unmasking the unknown: the unexpected discovery of hepatic epithelioid hemangioendothelioma, Johanna Lynne La Rosa-Rimorin
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- 1815 - Efficacy of Silybin Phospholipid Complex in Metabolic-Associated Fatty Liver Disease, Qianli Zhao
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- 1816 - The roles of THBS2-SPP1 axis in hepatic stellate cells activation and liver fibrogenesis, Hong Li
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- 1818 - Selective Inhibition of Hepatic Stellate Cell-Derived Microfibril-associated glycoprotein 4 Attenuates Liver Fibrosis, Wenye Wu
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 14
- 1819 - Impact of the expanded program on immunization on Hepatitis B elimination efforts: Insights from a Turkish reference center, Ahmet Sertçelik
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- 1820 - Downstaging by Atezolizumab plus Bevacizumab in Advanced Hepatocellular Carcinoma: Identifying Candidates for Conversion Therapy Based on Tumor Biology and Vascular Invasion Patterns, Chihiro Miwa
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- 1821 - Adolescent MASLD Incidence Trends, 1990–2023: Global and National Perspectives, Tianyuan Yang
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- 1822 - Blood biomarkers for non-invasive diagnosis of advanced fibrosis or compensated cirrhosis in chronic hepatitis B, Xin Wang
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- 1824 - Utility of EUS-guided Liver Biopsy in Liver Disease., NIVEDITA NIVEDITA
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 25
- 1832 - An Unexpected Turmoil: Hepatitis B Reactivation with Atypical Serology Following Neoadjuvant Capecitabine-Oxaliplatin in an Unsuspected Patient with Gastric Adenocarcinoma: A Multidisciplinary Management Complexity, ADRIAN BUGAYONG
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 12
- 1834 - The national burden of acute liver failure from 2019 to 2022: A Thailand nationwide population-based cross-sectional time series study, Rungfah Saehan
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 20
- 1836 - Trends in hepatitis B-related diseases in Asia, 1990-2023: an analysis of the Global Burden of Disease 2023 update, Jingran Jiao
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- 1837 - Early Ablation for Sub-centimeter Hepatocellular Carcinoma: Divergent Outcomes in Primary vs Recurrent Tumors over 10 Years, Yan Zhou
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- 1843 - Gut-Liver Axis Mechanisms Underlying Spontaneous Reversal of Liver Fibrosis: A Gut Microbiota-Metabolomics Analysis, Yuanying Zhao
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- 1846 - Patient-Derived Liver Organoids as a Translational Platform to Validate Targeted Nanoparticles for Image-Guided Liver Cancer Surgery, Zulihumaer Aizimuaji
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 18
- 1848 - Metastatic Liver Involvement from Primary Adamantinoma-like Ewing Sarcoma of the Lung in an Elderly Male: A Rare Case Presentation, Daniele Alek Calda
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- 1849 - Yangyin Fuzheng Jiedu Prescription Reduces Recurrence Risk in Patients with Small Hepatocellular Carcinoma after Minimally Invasive Treatment: a Pragmatic Randomized Controlled Trial, Huiwen Yan
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- 1851 - Inhibiting PPM1D facilitates the phosphorylation and oligomerization of VDAC2 to elicit immunogenic tumor destruction by activating cGAS-STING-IFN response, Liang Zhang Zhang
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- 1857 - Therapeutic Role of N-Acetylcysteine in Acute Liver Failure Secondary to Anti-Tuberculosis Drug-Induced Liver Injury: Insights From Indonesia, Alfiyyah Rizqy
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 20
- 1861 - Test, Treat!: A Small Step For The World, A Big Step For Us; Hepatitis C Microelimination Efforts in a Small City in Türkiye, HATUN ÖZTÜRK ÇERİK
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 12
- 1867 - Identification of benefit subgroups from heterogeneous effects of adjuvant transarterial chemoembolization in hepatocellular carcinoma using causal model, Tianyi Xia
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- 1883 - Early-Onset PSC With Recurrent Cholangitis Requiring Early Transplant Evaluation: A Five-Year Case Trajectory and Prelude to the Transplant Course, Christine Velasquez
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- 1886 - The role of Alix and VPS4A in autophagy and endosomal pathways and their relation to HBV replication, Mengji Lu
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- 1889 - Liver Fibrosis: Explainable Deep learning Network for Automated Staging by Using Equilibrium Enhanced Phase MR Images, Junhao Zha
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- 1890 - Deep Learning CT Signature for Predicting Occult Liver Metastases and Neoadjuvant Therapy Benefit in Pancreatic Ductal Adenocarcinoma: A Multicenter Retrospective Study, Yaoyao Yu
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- 1895 - Add on traditional Chinese medicine for hospitalized acute on chronic liver disease, Yufei Bi
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- 1897 - Genotype-Phenotype Correlation of ATP7B Mutations in Wilson Disease, Sujun Zheng
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- 1899 - Exploring the value of imaging indicators in predicting TACE resistance in hepatocellular carcinoma based on contrast-enhanced CT, Ying Lei
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- 1903 - Performance of FIB-4 and MAFLD Fibrosis Score vs. Transient Elastography in a Filipino Cohort, Rafael Emmanuel Mendoza
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- 1906 - A Fe-Curcumin-based Strategy to Reinvigorate CAR-T Cells by Reversing Exhaustion and Senescence in Liver Cancer, Yuchan Xue
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- 1908 - Performance of Baveno VII Criteria in Predicting Clinically Significant Portal Hypertension in Burned-Out MAFLD, Adinda Amalia
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- 1909 - A Diagnostic conundrum in a Case of Obstructive Jaundice in Biliary Hamartoma, Cleo Christille Lynn Lom-oc
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 06
- 1912 - Association Between Fatty Liver and Risk of Liver Failure in Patients with Acute Hepatitis B: A Retrospective Cohort Study, Xiao-hao Wang
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 12
- 1916 - Restoration of ATP7B Trafficking and Biliary Function in Wilson Disease Hepatocytes through CRISPR/Cas9-Mediated Gene Correction, Jiaqi Li
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 08
- 1918 - Solving a Recurrent Hepatic Abscesses Through Discovery of a Cholecystoduodenal Fistula resulting from an Over-the-Scope Clip closure of Duodenal Perforation as a complication of Microwave Ablation for HCC, James Manuel Medalla
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- 1920 - A New Noninvasive Marker Predicting Liver Fibrosis in Patients with Chronic Hepatitis B, ESMA ASLIHAN AYDEMİR
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- 1923 - Women's Perspective on Non-Antibiotic Interventions for rUTI Prevention: Meta-Analysis of Efficacy, Safety, and Clinical Implications, Wenjing Wang
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- 1926 - Hepatitis and HIV Seroprevalence in Individuals with Substance Use Disorders: A 25-Year Retrospective Analysis, Sümeyra İstek
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 12
- 1934 - Comprehensive Evaluation of Demographic, Endoscopic, and Ultrasonographic Characteristics in First Episode Variceal Bleeding, Hakan Demiröz
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 14
- 1935 - Neuropilin-1 alleviates metabolic dysfunction-associated fatty liver disease by promoting lysosomal degradation of toll-like receptor 4 in macrophages, Yinuo Yang
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- 1938 - Prediction of Esophageal Varices in Primary Biliary Cholangitis: What Is the Role of Non-Invasive Tests?, ikram aluahabi
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- 1940 - Dual SGLT2-DPP-4 Inhibition Synergistically Restores Hepatopancreatic Integrity in Experimental Diabetes, MOHD. IBRAHIM KHAN
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- 1941 - Predictors and Immune Signatures of Baveno-VII–Aligned Recompensation in Pediatric Wilson Disease with Decompensated Cirrhosis, Anmol Anmol
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- 1943 - Investigation of the Therapeutic Effects of α -Adrenergic Stimulation on Paracetamol-Induced Acute Liver Damage, Seydanur Avci
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- 1945 - Impact of Microbiome in Obese vs Lean Steatotic Liver Disease in Children, Betül Tunç Demirci
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 63
- 1949 - Development and Evaluation of Polyherbal Chitosan Nanoparticles: Antioxidant and Cytotoxic Effects on HepG2 Cells, Mohammad Shariq
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- 1950 - Decellularized Liver Matrix from Healthy versus Acute Liver Failure Livers Possesses Distinct Structural and Functional Properties, Chen Zhou
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- 1954 - Effects of Obinutuzumab on Cecal Ligation and Puncture Induced Septic Liver Injury in Rats, Fatih Albayrak
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- 1965 - Modeling the burden of steatotic liver disease and its subcategories in people living with HIV, Maurice Michel
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- 1967 - Hepatitis C Virus MicroRNA elimination in the Hospital Setting: What Results Have We Achieved?, Lu Zhang
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- 1968 - Investigation of the Effects of Vasopressin V2 Receptor Inhibition on Aquaporin Expression and Liver Functions in Sepsis-Induced Hepatic Injury, Elif Cadirci
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 24
- 1973 - Prognostic Utility of baseline and SVR12/24 M2BPGi for Liver-Related Events in hepatitis C patients, Haiying Zhang
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- 1984 - Comparative Efficacy of Entecavir and Tenofovir Disoproxil Fumarate in Preventing HBV Reactivation in Patients Receiving Biologic Disease-Modifying Antirheumatic Drugs, Gule Cinar
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 12
- 1985 - Effects of Linalool–Regorafenib Combination on Anticancer Activity in HUH-7 Cells, Aysegül Öztürk
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- 1989 - Post-Polypectomy Bleeding in Patients with Cirrhosis: A Systematic Review of Risk, Predictors, and Procedure-Related Factors, TALAL BHATTI
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 56
- 2000 - Efficacy of Yangyin Fuzheng Jiedu Prescription for Hepatocellular Carcinoma with Portal Vein Tumor Thrombosis: An Evaluation Using Five Propensity Score Methods, Wanxin Shi
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 54
- 2002 - Acute Hepatitis B Infection Following Long-Acting Injectable Antiretroviral Therapy Drug Switch in HIV, Jacky Man Chun Chan
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 12
- 2006 - Phenotypic Pattern of Acute Liver Injury in Crimean-Congo Hemorrhagic Fever Among Patients with Chronic Hepatitis B in an HBV-Endemic Region, Faruk Karakeçili
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- 2012 - Eliminating Viral Hepatitis by 2030: Goals and Gaps from a Large-Scale Community-Based Study in Shenzhen, China, Deliang Huang
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- 2019 - Cardiometabolic Risks in Elderly Individuals With Fatty Liver Living in Mid-Mountain Regions of Central Asia, Nurgul Toktogulova
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- 2023 - Impact of Liver Fat Contents on Brain Structure and Cognitive Functions in Patients with MASLD, Zhuoru Jiang
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- 2028 - sepsis, Peng Qu
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- 2039 - Modelling liver fibrosis and MASH with humanized immune system mice, Clothilde Philouze
25 April 2026, Saturday | 13:40 - 15:10 | ORAL PRESENTATION 60
- 2041 - A Rare Presentation of Salmonella enteritidis Bacteremia With Coronary Mycotic Aneurysm and Non-Cirrhotic Portal Hypertension, Ferdinand Anzo
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 15
- 2047 - Controlled expansion stent-grafts in TIPS: a real-world analysis of in vivo diameter change and early hepatic encephalopathy, Zizhen Ye
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- 2065 - Comprehensive multiomics study of ballooned hepatocytes in MASH: Insights into cellular adaptation to metabolic overload, Yan Wang
25 April 2026, Saturday | 13:40 - 15:10 | ORAL PRESENTATION 60
- 2066 - Phenotypic Age is a Hepatocentric Marker of Biological Aging: Stronger Associations of Liver-Related and Metabolic Outcomes over Cardiovascular Events in Steatotic Liver Disease, SHUN LI
25 April 2026, Saturday | 13:40 - 15:10 | ORAL PRESENTATION 60
- 2067 - Hepatic Arterial Infusion Chemotherapy Combined with Donafenib and PD-1 Inhibitor for Advanced Hepatocellular Carcinoma with Lung Metastases: A Multicenter Retrospective Study, Jinpeng Li
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- 2068 - The impact of implementing the expanded antiviral therapy eligibility for HBsAg-positive pregnant women in China: a modelling study, Ruixuan Hong
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- 2081 - Late Presentation and Poor Outcomes of Hepatocellular Carcinoma in a Resource-Limited Setting: An Observational Study in a Tertiary Care Hospital, Bangladesh, Mohammad Forhad Abedin Abedin
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- 2083 - Targeting STK40 unleashes anti-tumor immunity by triggering tumor vulnerability and T cell reinvigoration, Cun Wang
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- 2087 - CD146 Regulates Hepatocyte Lipotoxicity through Modulation of Mitochondrial Energy Metabolism and Inflammatory Response, Yixue Shu
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- 2092 - Paris-II Outperforms Other UDCA Response Criteria to predict Transplant-Free Survival in Asian PBC Patients, Yu Jun Wong
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- 2094 - Inhibition of Aurora B Induces Senescence and Potentiates Immunotherapy in Hepatocellular Carcinoma, Xuhui Ma
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- 2100 - From Organ to Surface: Perforating Disorders in Chronic Liver Disease, Maisam Akroush
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- 2102 - Temporal Trends and Outcomes of AMA-Negative Primary Biliary Cholangitis in a Multi-Centre South-East Asian Cohort, Abhimati Ravikulan
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- 2104 - A Longer Way to Go: Chronic Hepatitis B Burden and Trends Across the Asian-Pacific Region, Zhengzhao Lu
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- 2105 - Breaking the habit: Evaluating the routine use of SBP prophylaxis, Shauna MADIGAN
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- 2107 - COVID-19 pandemic had minimal impact on the diagnostic and treatment uptakes for HCV – a territory-wide study, Terry Cheuk+Fung Yip
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- 2110 - THE RISING THREAT OF MASLD IN YOUNG ADULTS: A COMPREHENSIVE METABOLIC AND HEPATIC ASSESSMENT FROM SOUTH ASIA, Abdul Samad
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- 2113 - Unveiling occult squamous commitment as a clinical fate determinant in cholangiocarcinoma, Chen Yang
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- 2117 - Clinical Characteristics and Short-Term Outcomes of Patients with Cirrhosis and Subclinical Decompensation, Tong Bu
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- 2121 - Eliminating Viral Hepatitis in Pakistan: A Comprehensive Strategic Framework, Hassan Mahmood
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- 2124 - GENDER DISPARITIES IN HEPATOCELLULAR CARCINOMA: A COMPARATIVE ANALYSIS, Abdul Samad Bhatti
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- 2125 - Efficacy and Safety of Magnesium Isoglycyrrhizinate in Checkpoint Inhibitor-Induced Liver Injury in Patients with Advanced Solid Tumors: An Exploratory Study, Yong Liu
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- 2130 - Predictors and impact of complete pathological response to pre-transplant immune checkpoint inhibitors in hepatocellular carcinoma: Findings from the Global LIFT-HCC consortium, Ju Dong Yang
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- 2132 - Leveraging Decentralization, Integration, and Task-Shifting to achieve Micro-elimination of Hepatitis C among prisoners in Northern India, Ajeet Singh Bhadoria
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- 2134 - Iron Metabolism Defects in Wilson Disease Suspects - A Real-World Two-Phase Molecular Epidemiology Study, Yue Han
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- 2137 - Protective and Therapeutic Roles of Vitamin D in Reducing Chlorpyrifos Induced Liver Fibrosis Through Modulation of Fibrotic Pathways, Dr. Aftab Abbasi
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- 2143 - A model, screen test and treat hepatitis C elimination project among under-served communities in Islamabad the federal capital of Pakistan, Huma Qureshi
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- 2145 - Gestational cholestasis: single center experience, Ahmet Uyanikoglu
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- 2148 - B-Cell Depletion Therapy Shows Efficacy in Steroid-Refractory Autoimmune Hepatitis, YunLong Yao
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- 2150 - Early Skeletal Muscle Loss Predicts Poor Outcomes in Advanced Hepatocellular Carcinoma Treated with Hepatic Arterial Infusion Chemotherapy, Asahiro Morishita
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- 2152 - Circulating cell-free mitochondrial DNA predicts prognosis and triggers macrophage STING-dependent inflammation in acute-on-chronic liver failure, Qiankun Hu
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- 2161 - Circulating Tumor DNA Analysis Enables Early Detection of Hepatocellular Carcinoma in High-Risk Patients, Xi Zhang
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- 2168 - Colorectal Carcinoma in Young patients, single Centre study from Karachi, Pakistan., Ghulam Mohiuddin
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- 2172 - The coexistence of MAFLD exacerbates liver fibrosis in patients with chronic hepatitis B., Alaa M. Mostafa
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- 2185 - Nonthermal Histotripsy Enables Complete Ablation of Perivascular Liver Tumors While Preserving Adjacent Vessel Patency, KAI-WEN HUANG
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- 2188 - Personalised Risk-Based HCC Surveillance in HCV-Related Advanced Fibrosis/Cirrhosis After SVR: Real-World Evidence of Cost Effectiveness., Gamal Shiha
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- 2189 - HBsAg Dynamics and Seroclearance Maintenance After Interferon Cessation in Chronic Hepatitis B Patients, Shanshan Zhu
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- 2192 - Neutrophil recruited CD38hi cytotoxic CD8 T cells characterize the T cell infiltrate in checkpoint inhibitor-mediated cholangitis, Xinghuan Fu
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- 2195 - 3D Radiomics Analysis Based on Liver CT Imaging for Predicting Hepatic Encephalopathy in HBV-ACLF Patients, Xueyun Zhang
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- 2201 - Prospective Evaluation of Factors Affecting Measurement Quality in Liver Elastography, Murat Taner GÜLŞEN
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- 2215 - Innovative Insights: Hepatic Stellate Cells activation and ROS-Gene Signatures Predicting Prognosis and Immunotherapy Response in Hepatocellular Carcinoma, Ning Wang
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- 2218 - Nucleos(t)ide Analogue Treatment History Stratifies Prognosis in Hepatitis B Virus-Related Acute-on-Chronic Liver Failure, Shan Zhong
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- 2225 - Infected Choledochal Cysts Masking as Liver Abscesses in an Adult Male: A Case Report, Emerald T. Gersava
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- 2226 - Impact of Diabetes on Fibrosis Progression in NAFLD: A Systematic Review and Meta-analysis, Javaria Younas Muhammad Younas
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- 2229 - Discrepancies in the indeterminate phase in patients with chronic hepatitis B between two updated international guidelines, Shaoqiu Zhang
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- 2240 - Hepatitis B Management in Primary Care: Nationwide Survey Highlights Knowledge and Practice Gaps Among Family Physicians in Türkiye, Imran Hasanoglu
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- 2241 - Diagnostic Value of Liver Biopsy in Unexplained Liver Enzyme Elevations: A Tertiary Center Experience, Serap Türk
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- 2244 - Early Experience with Endoscopic Ultrasound-Guided Gastric Variceal Obliteration at an Australian Quaternary Referral Centre, Rajit Gilhotra
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- 2245 - Unravelling the placebo response in pharmacological trials for metabolic dysfunction-associated steatotic liver disease: a systematic review and meta-analysis, Kiavash Semnani
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- 2254 - Prognostic Impact of Sarcopenia Assessed by Handgrip Strength and Skeletal Muscle Index in Hepatocellular Carcinoma Patients Receiving Atezolizumab plus Bevacizumab Therapy, Hiroki Tai
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- 2255 - Role of Metabolic Risk Factors in the Early Detection of Metabolic-Associated Steatotic Liver Disease (MASLD) in Young Adults: A Cross-Sectional Study, Muhammad Siddique Chohan
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- 2256 - Efficacy of Combined Nutritional and Physical Activity Intervention in Reversing Early MAFLD, Hafsa Abdul Waheed Jadgal
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- 2274 - Blood-Stasis Constitution Drives Hepatic MDSC Accumulation in MASLD;;
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- 2276 - Long-Term Outcomes of Treatment-Induced Versus Spontaneously Achieved HBsAg <100 IU/mL in Chronic Hepatitis B, Jian Wang
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- 2282 - SIRT2 restrains HDV-associated hepatic macrophage pyroptosis by deacetylating NLRP3 and suppressing NF- κ B signaling, Leer Shen
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- 2290 - Coffee Consumption and Hepatocellular Carcinoma: An Umbrella Review of Meta-Analyses involving Over 12 Million Population, Nitesh Kumar Karna
24 April 2026, Friday | 17:10 - 18:30 | ORAL PRESENTATION 32
- 2291 - The Antagonism between Demographic Shifts and Epidemiological Progress: A Decomposition Analysis of the Liver Cancer Burden in the Asia-Pacific, 1990–2023, Wenxin Yan
24 April 2026, Friday | 17:10 - 18:30 | ORAL PRESENTATION 32
- 2292 - Analysis of Clinical Characteristics and Treatment Outcomes in HBV - Related Liver Failure Patients with or without Diabetes Mellitus, lufeng Li
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 42
- 2294 - Comparison of the Effects of Tenofovir Alafenamide and Entecavir on Lipid Profiles in Anti-HBc Positive Kidney Transplant Recipients, Elif Gucyetmez
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- 2296 - From Campaign to Validation: Operationalizing Egypt's National Sustainability Plan for Viral Hepatitis Elimination (2025–2030), Mohamed Abd Allah
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- 2297 - Ischemia-Modified Albumin in MASLD: Correlation With Ultrasonographic Steatosis Grade and Elastography-Based Liver Stiffness, Kubra Koken
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- 2300 - Old Drugs, New Targets: Repurposing Proton Pump Inhibitors for Autoimmune Liver Diseases: Molecular Docking Insights into the TLR4/MD-2, NLRP3, and C5aR1 Axes, Mehmet Yildiz
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 03
- 2302 - Stiffness Regression Fundamentally Alters Mortality Patterns and Improves Survival in Advanced Fibrosis After SVR: A Cause-Specific Outcome Analysis, Shuntaro Obi
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 30
- 2306 - Study on Key Inflammatory Proteins of Antiviral Treatment in Children with Chronic Hepatitis B, Yanwei Zhong
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- 2309 - Zonal Restoration and Inter-Cellular Signaling in Fibrosis Regression: Single-Cell Transcriptomics Decodes the Pericentral Hepatocyte-HSC Axis, Thi Thanh Thuy Le
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 30
- 2310 - NAFLD promotes early hepatocellular carcinoma recurrence via MDSC metabolic reprogramming: Insights from a two-phase recurrence model in mice, Li Mingming
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 38
- 2311 - LOXL1 Regulates Lipid Metabolism to Promote Hepatic Stellate Cell Activation via Ubiquitin-Mediated FASN Degradation, Xuzhen Yan
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 30
- 2314 - Gilbert syndrome increases the risk of gallstone disease: a cross-sectional study based on health examination data from 338,372 adults, Sujun Zheng
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 08
- 2318 - Gut Microbiota Transfer and Integrated Omics Identify Streptococcal Collagen-Like (Scl1) Protein as a key fibrogenic driver in Alcohol associated liver disease., Manisha Yadav
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- 2320 - Liver meta-proteome and kinome reveal *Pseudomonas aeruginosa* –pPKC; axis as a driver of steroid resistance in severe alcohol related hepatitis, Jaswinder Maras
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- 2326 - Asia-Pacific expert consensus on GAAD for hepatocellular carcinoma surveillance, Teerha Piratvisuth
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 56
- 2328 - Fatty Liver as a Key Risk Modifier for Adenoma Detection in FIT-Positive Young Adults: A Systematic Review of the Available Evidence, DESIREE JANE LACUIN
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 38
- 2329 - Multi-omics analyses identify a novel hepatocellular carcinoma subtype with better response to immune checkpoint inhibitor therapies, Junjie Xu
24 April 2026, Friday | 17:10 - 18:30 | ORAL PRESENTATION 32
- 2336 - Lean MAFLD in Chronic Hepatitis B: Clinical Characteristics, Fibrosis Risk and antiviral response in a Pakistani Cohort, Farhana Kayani
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 64
- 2340 - Metabolic Fatty Liver Masks Hepatitis B Activity: Implications for ALT-Based Treatment Decisions, a study from PAKistan, Jalal Khan
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- 2347 - Semaglutide demonstrates efficacy in improving liver fibrosis across diverse subpopulations in part 1 of the ESSENCE trial – outcomes in Asian and non-Asian populations, Jacob George
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- 2349 - Study on the Anti-Liver Cirrhosis of Luteolin: Targeting the PPAR; Axis to Regulate Macrophage Fatty Acid Metabolic Reprogramming, Xuemei WANG
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- 2350 - Generation of Macro-Scale Engineered Biliary Trees with P-gp Efflux Function and Enhanced Injury Resistance, Shiwen Ma
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 24
- 2351 - A-HOC Study on Patients Under Real-time Surveillance for Liver Cancer in Mongolia, Amarsanaa Jazag
24 April 2026, Friday | 17:10 - 18:30 | ORAL PRESENTATION 32
- 2353 - Analysis of Aggressive Phenotypes in Hepatocellular Carcinoma Using Transcriptomic Data: A Machine Learning Framework for Biological Signature Discovery, Erdi Şeker
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 22
- 2359 - Stage-Specific Survival in Hepatocellular Carcinoma: A Multiregional Analysis Across Japan vs Non-Japan Cohorts in the Asia-Pacific Region, Kyoko Oura
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- 2360 - Amlodipine activates an adaptive endoplasmic reticulum stress response in Huh-7 hepatocellular carcinoma cells, Neslihan Başgöz Karagüç
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 24
- 2361 - L-canavanine selectively inhibits hepatocellular carcinoma cell proliferation and induces apoptosis in vitro, Ahmet Ozan Kaleci
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 24
- 2364 - Hepatic Involvement and Drug-Induced Liver Injury in Brucellosis, Oğuzhan Acet
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 15
- 2368 - Dynamic Metabolic Reprogramming Drives Adipose Fibrosis in a High-Fat Diet-Induced Obesity in Mice Model, Abhishak GUPTA
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 45
- 2369 - AGIUS Score Predicts Re-bleeding Risk after Endoscopic Variceal Ligation in Cirrhotic Patients Introduction, Fei Wang
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 30
- 2374 - Non-Invasive Assessment of Steatosis and Stiffness in MASLD: Links to Body Composition and Dyslipidemia in High-Risk Metabolic Groups, Abdulvahap Hohluoğlu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 38



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- 2375 - Achieving Recompensation with Low-dose Diuretics in HBV-related Cirrhosis: An Expanded Baveno VII Strategy Yields Similar Long-Term Prognosis in A Multicenter Cohort Study, Zhiying He
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- 2377 - Efficacy Differences of Interferon Combined with Nucleos(t)ide Analogs in Treating HBeAg-Positive Chronic Hepatitis B Stratified by Baseline Liver Fibrosis Indicators, Yaqin Zhang
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- 2378 - Metabolic Dysfunction-Associated Liver Disease and Lung Injury: A Decade of Progress Toward Mechanistic Integration and Translational Insight, Tong Chen
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- 2385 - HECTD3 Mediates PRDX4 Stabilization via K63-Linked Ubiquitination to Ameliorate Hepatic Steatosis, Inflammation, and Fibrosis in MASH, Rui Jin
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- 2386 - Immunosuppressive Medication Non-adherence in Liver Transplantation Adult Recipients in Vietnam, Anh Nguyen Thi Van
24 April 2026, Friday | 10:50 - 12:10 | ORAL PRESENTATION 33
- 2389 - Towards Individualized Interferon Therapy in Hepatitis B: HBsAg Kinetics and Seroclearance upon Baseline HBsAg Levels and Treatment Duration, Qiran Zhang
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- 2390 - Ultrasonographic evaluation of gallbladder motor function in the diagnosis and prognosis of intrahepatic cholestasis of pregnancy, Zhen Tian
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 57
- 2394 - Demystifying primary hepatic Langerhans Cell Histiocytosis (LCH)- From pattern recognition to molecular diagnosis, Deepa Janakiraman
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- 2395 - Multicohort fecal metagenomes analysis contributes to non-invasive diagnosis for cholangiocarcinoma, Zhigang Ren
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- 2398 - Etiology-Specific Patterns of Liver Stiffness and Steatosis Assessed by Transient Elastography in a Tertiary Referral Center in Southern Philippines, Mark Siñagan
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- 2404 - Development and validation of a dynamic nomogram for predicting significant liver injury in chronic hepatitis B patients with indeterminate phase, Chao Jiang
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 52
- 2406 - Benefit of treatment expansion: A Comparative Analysis of EASL 2025, AASLD 2025, and APASL 2026 Clinical Practice Guidelines for Management of Chronic Hepatitis B, Jing Chen
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- 2410 - Hepatitis B Virus Core Antibody Seroconversion and Risk of Hepatitis B Reactivation after Allogeneic Hematopoietic Stem Cell Transplantation, Ruirui You
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- 2418 - AFP trajectories in HCC after ablation treatment: a 15 year retrospective study, Maria Fleurdeliz Goco-Siatan
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 17
- 2422 - Diagnostic Accuracy of Portal System Duplex Sonography Findings Among Filipinos with and Without Liver Cirrhosis; The DupLiCs Study, Yrah Damiene Fernandez
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 21
- 2426 - Association of Immune Nutritional Status with Histological Stage and Mortality in Patients with Metabolic Dysfunction-Associated Steatotic Liver Disease: A Population-Based Cohort Study, Yixuan Zhu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 38
- 2427 - Endoscopic Ultrasound-Guided Portosystemic Pressure Gradient Measurement To Determine Candidacy For Simultaneous Liver-Kidney Transplantation Vs Renal Transplant Alone In End-Stage Renal Disease Patients With Compensated Advanced Chronic Liver Disease or, Arka De
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 25

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- 2432 - Endoscopic Ultrasound Guided Ablative Therapies For Hepatocellular Carcinoma, Arpit Shastri
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- 2433 - Diagnostic performance of non-invasive fibrosis scores in treatment-naïve patients with autoimmune hepatitis, Mubin Ozercan
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- 2438 - Long-term Serial Liver Stiffness Monitoring, Histological Fibrosis, and HVPG as Determinants of Disease Progression and Decompensation in MASLD, ANKUR JINDAL
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- 2439 - A silent threat: A case of infiltrative cholangiocellular carcinoma developing without detectable mass, Abdullah Tanrikulu
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 06
- 2440 - Clinical outcomes and treatment response in primary biliary cholangitis with and without autoimmune hepatitis features: a Thai multicenter 10-year cohort, Kedsiree Sanit
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 47
- 2441 - MACHINE-LEARNING MODELS IMPROVES MORTALITY AND ANTIMICROBIAL RESISTANCE PREDICTION IN CULTURE-PROVEN SPONTANEOUS BACTERIAL PERITONITIS IN CIRRHOSIS: A MULTICENTER COHORT STUDY, Nipun Verma
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 21
- 2443 - Histological severity is not associated with clinical presentation or outcome in children with autoimmune hepatitis, Aditya Soni
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- 2450 - A rare case: Sporadic Burkitt lymphoma presenting with high serum-ascite albumin gradient and low ascite glucose, Abdullah Tanrikulu
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 56
- 2454 - Anticancer Effects of Bromelain on HUH7 Hepatocellular Carcinoma Cells through Modulation of the EGFR/PI3K Pathway, ONUR DURNA
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 17
- 2457 - Impact of the national Hepatitis B vaccination program on HBsAg seroprevalence in pregnant women and comparison with migrant pregnant women, Yelda Ögütmen
25 April 2026, Saturday | 10:50 - 12:10 | ORAL PRESENTATION 52
- 2460 - Comparative Efficacy of Lenvatinib Versus Sorafenib in Advanced Hepatocellular Carcinoma: A Meta-Analysis of Meta-Analyses, Akshyata Osti
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- 2461 - Early GI/Hepatology Subspecialty Access in Cirrhosis and Survival: A Retrospective Cohort Analysis, Mohamed Elsaid
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- 2463 - Association of Long-Term Glycemic Control with the Risk of Developing Cirrhosis in Type 2 Diabetes Mellitus: A 5-Year Multi-Center Retrospective Cohort Study of Liver Outcomes and Predictive Factors, Paul Miguel Perez
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- 2483 - Endoscopic treatment for esophagogastric variceal bleeding in acute-on-chronic liver failure patients, Yanan Sun
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- 2484 - CT-based Vasomics model for noninvasive diagnosis of Porto-sinusoidal vascular disease in patients with portal hypertension, Yanna Liu
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 21
- 2488 - SAFETY OF MIDAZOLAM AND NALBUPHINE IN CIRRHOTIC PATIENTS WHO UNDERGO UPPER GASTROINTESTINAL ENDOSCOPY, Lajpat Rai
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- 2491 - Hepatocellular Carcinoma in Armenia: Liquid Biopsies and Primary Assessment of Molecular Alterations Prevalence, Hasmik Ghazinyan
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- 2496 - Development and validation of an artificial intelligence driven convolutional neural network for automated stool colour classification to facilitate early diagnosis of biliary atresia, Arjun Maria
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- 2499 - Wilson disease mutation pattern with genotype-phenotype correlations in paediatric cohort in south India, Sindhuja Thatti Audikesavalu
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- 2504 - Long-term outcomes of patients with Hepatitis D recurrence after liver transplantation, Devrim Muge Ozari Gulnar
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- 2505 - Lonafernib as finite treatment for chronic hepatitis D: Long-term follow-up data, Cihan Yurdaydin
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- 2506 - Evaluation of anti-HCV positivity, HCV RNA testing, and treatment status among individuals presenting to the hospital, Selma Tosun
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- 2507 - High Treatment Adherence Reduces HCC in Cirrhosis, with Additional Benefit from TAF Running title : Adherence Lowers HCC Risk in Cirrhosis, Jeong-Ju Yoo
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- 2508 - Midodrine with albumin improves ascites control in Acute-on-chronic liver failure: A Double Bind Randomized trial (MIDAS trial), Anand Kulkarni
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- 2510 - Efficacy of PegIFN α 24; Add-on Therapy in Treatment-Experienced CHB Patients with Low-Level Viremia: A Large Prospective Cohort Study, Bei Jiang
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- 2511 - Diagnostic accuracy of blood-based non-invasive tests for screening of varices in children with portal-hypertension: A systematic review and meta-analysis, Kalpana Panda
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- 2514 - Long-term Clinical Outcomes of Delta Hepatitis in A Single Center Experience, Furkan Kirsoy
25 April 2026, Saturday | 13:40 - 15:10 | ORAL PRESENTATION 58
- 2518 - Antinuclear Antibodies Positivity Indicates a High-inflammatory Phenotype in Primary Biliary Cholangitis: A Multicenter Retrospective Analysis, shasha li
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 06
- 2519 - The long term results of EUS guided coil application for fundal varices, Sercan Kiremitci
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- 2525 - Adherence to an Admission Care Bundle and Its Impact on Outcomes in Decompensated Cirrhosis: A Multicenter Analytical Cross-Sectional Study, Federico Peralta
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- 2527 - Prognostic Stratification of Non-Ischemic Acute Liver Injury in the Emergency Department: A Retrospective Cohort Study, Pelin Telli
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- 2531 - Evaluation of Leptospirosis Cases in the Şanlıurfa Region, Velat AYTEKİN
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- 2533 - Cross-study analysis of HBsAg rebound following treatments of elebsiran/BRIL-179 in combination with peginterferon alfa, Jidong Jia
25 April 2026, Saturday | 13:40 - 15:10 | ORAL PRESENTATION 58
- 2534 - ASSESSMENT OF ACCURACY OF PLATELET COUNT/SPLEEN DIAMETER RATIO IN PREDICTING THE PRESENCE OF ESOPHAGEAL VARICES IN FILIPINO PATIENTS WITH CIRRHOSIS AT SLMC-QC, ALYSSA CHAN
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- 2541 - Alcoholic Liver Transplants Performed Due to Liver Disease: A Single-Center Experience, Hasan Buran
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- 2543 - Prevalence and risk factors of esophageal and gastroesophageal varices in myeloproliferative neoplasm patients with portal hypertension in a tertiary government hospital: a 5-year cross-sectional study, Sam Fandood
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- 2553 - EUS guided liver biopsy for diffuse liver diseases is an emerging alternative to percutaneous liver biopsy, Hakan Şentürk
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- 2554 - A Preclinical study on Protective Effects of Atorvastatin Against Methotrexate-Induced Hepato-Renal Toxicity: Role of Oxidative Stress and Inflammation, Jayadevan K
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 41
- 2559 - Evaluation of a FIB-4-Based Clinical Pathway for Fibrosis Assessment in Patients with Coronary Heart Disease, Chenlu Yang
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- 2560 - Predictive potential of hypermethylation of RASSF1A and CCND2 promoters in hepatitis B virus-induced hepatocellular carcinoma, Raad Rahmat
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- 2562 - Successful Endoscopic Ultrasound-Guided Ethanol Ablation of a 7-mm Non-Functional Pancreatic Neuroendocrine Tumor in a High-Risk Surgical Candidate, Irshad Batool
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 41
- 2564 - The relationship between the systemic immune-inflammation index and hepatitis C virus infection: NHANES 2005 to 2012, Yuyu Zeng
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- 2572 - Short-Course Adjuvant Tislelizumab plus Tyrosine Kinase Inhibitor Following Curative Ablation for Early-Stage Hepatocellular Carcinoma: A Prospective Cohort Study, Fanping Meng
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 17
- 2574 - Plasma microRNA-based non-invasive stratification of clinically significant and advanced fibrosis in metabolic dysfunction-associated steatotic liver disease among Arab adults, Ammal Metwally
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2576 - Point-of-care HBcrAg testing to identify high viral replication in chronic hepatitis B: a tool for supporting the WHO simplified treatment strategy in resource-limited settings, Takako Inoue
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- 2577 - Using Rose to Improve the Accuracy of Diagnosis of Biliary Tract Diseases through ERCP guided Biopsy, Yunzhi Shen
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- 2579 - YTHDF1high cancer-associated fibroblasts-derived kynurenine promotes tumor progression by supplying an immunosuppressive niche, Liren Zhang
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- 2580 - PYGL: A novel non-invasive biomarker for diagnostic and prognostic of HEV-ALF patients, Luyu Wang
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- 2583 - Head-to-Head Comparison Between MRI and A Novel Portable VCTE Device (Liverscan): A Pilot Study, Zhuoru Jiang
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- 2590 - The Predictive Value of Platelet-Derived Growth Factor for Evaluating Antiviral Therapy Response and Liver Stiffness Trajectory in Chronic Hepatitis B, Fadhyl Zuhry Lubis
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- 2593 - Spermidine suppresses liver fibrosis by remodeling the communication signal between liver sinusoidal endothelial cells and hepatic stellate cells Running title: Spermidine suppresses liver fibrosis by downregulation of LSEC-derived BGN, Cheng Zeng
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- 2594 - Risk factors for low-level viremia in Tenofovir Disoproxil Fumarate (TDF)-treated patients with chronic hepatitis B complicated by advanced hepatic fibrosis/compensated liver cirrhosis, Runling Zhang
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- 2596 - Baseline Determinants of Inadequate Ursodeoxycholic Acid Response in Primary Biliary Cholangitis: A POISE-Based Real-World Cohort Study, Phool Chand
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- 2600 - Hepatitis B virus recurrence in liver transplant recipients receiving subcutaneous hepatitis B immunoglobulin, Muhsin Murat Muhip HARPUTLUOĞLU
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- 2601 - Diagnostic Accuracy and Long-Term Prognostic Value of FIB-4 Index and De Ritis Ratio in Chronic Hepatitis C Patients: A Comparative Kaplan-Meier Analysis with MELD 3.0, İLYAS ETHEM ŞENOCAK
23 April 2026, Thursday | 09:00 - 10:30 | ORAL PRESENTATION 10
- 2602 - Effect of Midodrine on Short Term Mortality and Liver Function in Patients with Cirrhosis and Ascites: A Systematic Review and Meta-analysis, GİZEM DAĞCI
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- 2604 - Research on artificial liver support system for non-hepatocellular carcinoma patients with severe immune checkpoints related liver injury, Hua Liu
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 07
- 2605 - Mobile Ablation for Secondary Hypersplenism in Hepatocellular Carcinoma Patients with Liver Cirrhosis: A Safety and Efficacy Study, Yang Liu
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 49
- 2606 - A Simple Five-Variable Model Integrating GWAS Variants Predicts Chronic Outcomes in Drug Induced Liver Injury, Rongtao Lai
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 07
- 2607 - Inhibition of FKBP10 attenuates hepatic stellate cell activation and liver fibrosis via RAS signaling, Jingyu Zhang
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- 2608 - Optimizing Pegylated Interferon Consolidation Therapy and Predicting Recurrence After Functional Cure in Chronic Hepatitis B: A Study Based on Updated Definitions, Xiaoping Wu
23 April 2026, Thursday | 09:00 - 10:30 | ORAL PRESENTATION 10
- 2609 - Acetylation modification of PKM2 promotes macrophage reprogramming and accelerates the progression of MAFLD, Huiling Cao
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2612 - Accuracy of D-dimer Levels in Predicting Clinical Outcomes of Patients with Decompensated Liver Cirrhosis in a Philippine Tertiary Hospital, SAUL SUAYBAGUIO
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 49
- 2614 - A Cross-Section Study on the Co-prevalence of MAFLD and HBV Infection Among Adult Residents in Maojian District, Shiyan City., Zhongji Meng
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2617 - Spirulina Prevents Hepatic Steatosis by Promoting Macrophage M2 Polarization via Gut Microbiota-Derived 2-Hydroxy-3-Methylbutyrate, Feihu Bai Bai Feihu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2620 - The Gut Fungus Rhizopus microsporus var. rhizopodiformis Exacerbates Metabolic-associated Fatty Liver Disease by Impairing Intestinal Barrier Integrity, Daya Zhang Zhang Daya
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2623 - Transcriptomic Profiling Identifies Anti-Inflammatory and Anti-Steatotic Programs Mediated by ShenGe Formula in MAFLD, ZHU XIAONING
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2625 - Procedural-Related Bleeding Among Patients with Liver Cirrhosis in a Tertiary Hospital in the Philippines: A Five-Year Review, Jeremie Manarang
25 April 2026, Saturday | 07:30 - 09:00 | ORAL PRESENTATION 49
- 2626 - Clinical characteristics of colorectal polyps in patients with non-alcoholic fatty liver disease in high altitude areas, Chunyan Wang
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2627 - ZIP1⁺ CAFs Shape the Immunosuppressive Microenvironment to Drive Hepatocellular Carcinoma Progression, Wang Hao
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- 2630 - Single-cell transcriptomes reveal the immune characteristics of patients with chronic hepatitis B at different stages of disease progression, Liu Yang
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- 2634 - A Novel Murine Model of MetALD Recapitulates the Synergistic and Aggressive Phenotype of Combined Metabolic Dysfunction and Alcohol Consumption, Yingying Nie
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- 2635 - Endothelial RGS16 Attenuates Hepatic Fibrosis by Restricting Pathological, Yuzhu Song
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- 2639 - Machine learning(ML) based models predict beta-blocker response using non-invasive tests(NITs) in patients with acute variceal bleed -BE-RESPONSE STUDY, Harsh Vardhan Tevethia
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- 2640 - Necrolytic migratory erythema without glucagonoma: An unusual association with chronic pancreatitis and hepatic dysfunction, Mehmet Demir
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 41
- 2641 - Prevalence of Metabolic-Associated Fatty Liver Disease in Patients with Rheumatoid Arthritis: A Cross-Sectional Study, Eman Fares
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2643 - IL-24 attenuates hepatocyte apoptosis and ameliorates metabolic-associated steatohepatitis by activating the autophagy-lysosome pathway, Jiawei Cui
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 39
- 2644 - FKBP10⁺ CAFs Shape the Immunosuppressive Microenvironment to Drive Hepatocellular Carcinoma Progression, Liu Jiangxi
23 April 2026, Thursday | 18:30 - 20:00 | ORAL PRESENTATION 17
- 2647 - Quercetin ameliorates MASLD progression through the inhibition of the hexosamine biosynthesis pathway-mediated TAB2 protein O-GlcNAcylation, Yang Yang
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 62
- 2649 - Microsomal Glutathione S-Transferase 2 Links Copper Toxicity to Hepatic Inflammation via Leukotriene C4 in Wilson's Disease, Jie Bai
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- 2654 - Comparison of the diagnostic accuracy of magnetic resonance elastography, FibroScan, and serum fibrosis models for detecting liver fibrosis and cirrhosis in chronic liver diseases: a multicohort study across all aetiologies, Qiang Li
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 51
- 2660 - The Silent Summer Threat: Significant Seasonal Worsening of Electrolyte Imbalance in Cirrhosis Patients in a Tropical Climate Calls for Dynamic Diuretic Dosing, Md. Shakhawat Hossain
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- 2663 - High Prevalence of MASLD and MASH in Chronic Hepatitis B Patients: A Cross-Sectional Study, Müge Toygar Deniz
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- 2664 - Integrating genetic omics and functional omics to identify clinically actionable gut microbiota–host interface targets in MASLD, Jiyuan Zhou
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- 2666 - Outcomes of cholangiocarcinoma (ICC) or combined hepatocellular-cholangiocarcinoma after liver transplantation, Dali Zhang
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- 2670 - Endoscopic Ultrasound-Guided Partial Splenic Embolization in Hypersplenism: A Novel Approach Validated in a pilot study, Changqing Guo
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- 2671 - Mechanistic Study of Resmetirom in Treating MAFLD Mouse Model: Gut Microbiota Remodeling and Metabolic Regulation, hang chen
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- 2672 - Predictors of Post-TIPS Persistent Ascites, JAYASHREE BISWAS
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- 2673 - Parallel Comparison of Two HBV pg RNA Detection Assays, Yuyuan Jia
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- 2678 - HBV recurrence in patients with hepatocellular carcinoma and cholangiocarcinoma following liver transplantation, Xi He
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- 2679 - Outcomes of cholangiocarcinoma (ICC) or combined hepatocellular-cholangiocarcinoma after liver transplantation, Dali Zhang
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- 2681 - Characteristics of Autoimmune Hepatitis in elderly patients, Lili zhao
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- 2690 - TACE plus Lenvatinib and Envafolelimab for Conversion Therapy in Unresectable HCC: A Prospective, Phase II Study, Yuhao Su
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- 2691 - Splenic Stiffness Measurement to PREDict Decompensation in Patients with Compensated Advanced Chronic Liver Disease in India (SPRED-c-ACLD study), Sayan Malakar
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- 2693 - Muscle Matters: Impact of Sarcopenia on Mortality and Clinical Outcomes in Pediatric Liver Transplantation, Jyoti Bhutani
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- 2695 - Candidemia in Liver Transplant Recipients: A Ten-Year Retrospective Study, Adem Kose
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- 2699 - RAPID Procedure Enables Safe Use of Small Left Lobe Grafts: A Two-Center Experience, emre aray
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- 2700 - Does Primary Biliary Cholangitis and Autoimmune Hepatitis Increase the Risk of Precancerous Colon Polyps?, Ayse Sena Oguz
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- 2706 - A Novel Non-invasive Model to Identify High Risk Varices in Patients with HBV-related Cirrhosis, Haiyu Wang
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- 2711 - Related Factors for Low-Level Viremia and the Comparative Efficacy of Management Strategies in Treatment-Naïve Chronic Hepatitis B Patients Receiving Entecavir or Tenofovir Amibufenamide: A Real-World Study, Xiucheng Pan
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- 2714 - Exposure to Legacy and Replacement PFAS Is Associated with Metabolic Dysfunction-Associated Steatotic Liver Disease in Women, Guanjing Lang
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- 2729 - Lactate triggers HBV-Specific CD8+ T cell exhaustion through mitophagy suppression mediated mitochondrial dysfunction, Yuhao Wang
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- 2731 - Tbx1 alleviated hepatic encephalopathy by promoting meningeal lymphangiogenesis and drainage of senescent astrocytes via a CCL21-dependent manner, Chihao Zhang
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- 2733 - Joint Association of C-Reactive Protein and the Triglyceride Glucose-Chinese Visceral Adiposity Index with Incident Chronic Liver Disease: A National Cohort Study, Runci Jiang
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- 2736 - Thalidomide reduces transfusion requirements due to gastrointestinal vascular malformations in patients with liver cirrhosis, Manu Rajeev
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- 2739 - D-TACE Combined with HAIC Versus D-TACE in BCLC Stage C HCC: A Randomized Controlled Trial, Xuhua Duan
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- 2742 - PREDICTION OF NOSOCOMIAL INFECTION DEVELOPMENT REMAINS SUBOPTIMAL DESPITE USING MACHINE LEARNING TECHNIQUES IN A PROSPECTIVE GLOBAL COHORT OF HOSPITALIZED CIRRHOSIS PATIENTS, Jasmohan Bajaj
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- 2748 - A Retrospective Cohort Study on the Adherence to American Association for the study of Liver Diseases (AASLD) Quality Measures in Cirrhosis for Variceal Bleeding and its Association with Clinical Outcomes, Mario Rafael Estella
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- 2753 - Clinical and biochemical predictors of Hepatocellular Carcinoma: A Five-Year Cohort Study from a Tertiary Care Center in Pakistan, Gulzar khan
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- 2756 - Locoregional Therapy Combined with Continuing or Switching Systemic Therapy for Intrahepatic Oligoprogression in Hepatocellular Carcinoma: A Multicenter Retrospective Study, Hongli Yu
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 36
- 2759 - miR-945-KB downregulates miRNA-363-3p and targets SERCA2b to alleviate hepatic endoplasmic reticulum stress and steatosis in MAFLD, da wang
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 62
- 2760 - Real-world Biochemical Responses to First-line Azathioprine versus Mycophenolate and Upon Switching from Azathioprine to Mycophenolate in Autoimmune Hepatitis Patients, Astrid Amador
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- 2762 - The Effects of Nomenclature Change on the Epidemiology of Fatty/Steatotic Liver Disease, Danielle Lucero
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- 2765 - Disability Adapted Digital Therapeutics as a Precision Strategy for MASLD Risk Modulation: Longitudinal Neuromuscular in Lower Limb Amputees, Muh syaiful akbar
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- 2773 - Mechanistic Role of MFN2-FSP27 Mediated Lipid Droplet-Mitochondria Interactions in Metabolic Dysfunction-associated Steatohepatitis, QIQANQIAN ZHENG
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- 2774 - Adipose-derived FABP5 drives myeloid bias in hematopoietic stem cells and promotes tumor progression., Wanyue Cao
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- 2779 - Re-evaluation of Liver Biopsy Specimens Performed for Presumed Chronic Hepatitis B and Investigation of the Prevalence of MASLD, Ammar ALRAMADAN
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- 2781 - Dual noninvasive prediction of esophageal varices in cirrhosis using transient elastography and echocardiographic parameters: an interim machine learning analysis, EINA MARIE LI
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- 2784 - The Effect of HALP Score on Predicting Post-Transplant Outcomes in Patients with Hepatocellular Carcinoma, Fuat Aksoy
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- 2785 - Metabolic Dysfunction Increases the Risk of Liver-related Events in Virologically Suppressed HBV Related Compensated Cirrhosis: Refining Prognostication with the Baveno VI-SSM Model, Huan Cheng Cheng
24 April 2026, Friday | 13:40 - 15:10 | APASL - IASL JOINT MEETING
- 2789 - Comparison of the Safety and Efficacy of Tailored versus Fixed-Dose Albumin in Cirrhosis with Sepsis-Associated Acute Kidney Injury: A Randomised Controlled Trial, SHREYAS SARVESH
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 02
- 2793 - 4-1BB Costimulation Restore Tfh1 Function to Augment HBV-Specific Humoral Immunity in Chronic Infection, Yanchen Ma
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 27
- 2796 - Interferon- γ programs HBsAg-specific humoral immunity through the monocyte-IL6-Tfh cell axis to facilitate functional cure of hepatitis B, Shihong Zhong
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- 2799 - Cathepsin K Attenuates Liver Fibrosis by Suppressing TGF- β /Smad Signaling via TLR-1/2 Degradation, Zhandong Lin
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- 2809 - Long-Term Survival in De Novo Liver-Metastatic Colorectal Cancer: A Descriptive Cohort Study, Sati Coşkun Yazgan
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 36
- 2810 - Sodium Glucose Co-Transporter 2 Inhibitors Use in Diabetic Patients with Cirrhosis Improves Liver-related Outcomes and Survival, Sajid Jalil
25 April 2026, Saturday | 09:00 - 10:30 | ORAL PRESENTATION 51
- 2814 - Etiological distribution, clinical phenotypes and six-month outcomes of drug-induced liver injury: a single-center cohort study, Duygu Birinci
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 07
- 2815 - Associations Between Dietary recommendations/ diet quality and Steatotic Liver Disease Prevalence and Severity in U.S. Adults 2017–2023, Nicholas Dunn
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 2818 - Updated results of the observational study to assess virological effectiveness, safety, and tolerability of NVR/RTV+SOF combination in HCV infected people living with HIV, Emilia Krasavina
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 27
- 2832 - Immune-Excluded CD4⁺ and CD8⁺; T-Cell Depletion Predicts Early Recurrence in Hepatocellular Carcinoma with Microvascular Invasion, Rino Alvani Gani
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- 2833 - Hyperinsulinemia Establishes a Pathogenic Feedback Loop between Pyroptotic Hepatocytes and MDSCs in MAFLD, Huilian Shi
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- 2834 - Epidemiologic and Clinical Trends in Hepatocellular Carcinoma: A Glimpse Through the Lens of Systemic Therapy Availability, Ma. Angelica Goldamier Cannu
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 61
- 2839 - CAN EARLY CHANGES IN ALPHA-FETOPROTEIN PREDICT RADIOLOGIC RESPONSE AND OVERALL SURVIVAL IN IMMUNE CHECKPOINT INHIBITOR-BASED THERAPY OF HEPATOCELLULAR CARCINOMA?, Jann Christian Solis
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- 2841 - PLPP2 drives liver repair and regeneration via remodeling phosphatidylethanolamine metabolism after acute liver injury, Fan Li
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- 2844 - AARS1 promotes tumor progression and immune evasion via ATF6 lactylation-mediated tryptophan metabolism in hepatocellular carcinoma, Yiming Wang
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- 2848 - Physician–Patient Dual-Track Follow-Up Care Bundle in Hepatology: A Multidisciplinary Practice Model Integrating Clinical Care and Research Coordination, Hong-Li Liu
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- 2851 - From Synthetic to Clinical: The Effect of Domain Shift on Decision Support Systems, Sümeyye AĞIR
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- 2859 - Profile of Hepatocellular Carcinoma in India: A Five-Year Prospective Study, Dr. ANAND V KULKARNI
25 April 2026, Saturday | 15:30 - 17:00 | ORAL PRESENTATION 61
- 2860 - Advances in Artificial Intelligence for Liver Disease Research: Algorithmic Applications and Technical Challenges, YAO YUNLONG
24 April 2026, Friday | 07:30 - 09:00 | ORAL PRESENTATION 22
- 2871 - Genetic Variants in Bile Transporter Genes in Prolonged Cholestatic Jaundice Associated with HAV and Drug-Induced Liver Injury, KRISHNAKANTH SARDA
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- 2874 - Dynamic evolution of the gut microbiome predicts the therapeutic response in liver cancer: A large-scale longitudinal study with a cross-cohort meta-analysis, Jie Han
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- 2877 - The Effect of Portal Vein Tumor Trombosis on Survival in Patients with Hepatocellular Carcinoma Treated with Different Therapies, Hikmet Akkız
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- 2878 - From Stool to Personalized Screening Approach: A Comprehensive Meta-analysis of Metagenomic Gut Microbiota Assessment in Patient with MAFLD and Control Cohort, Krisanto Tanjaya
24 April 2026, Friday | 18:30 - 20:00 | ORAL PRESENTATION 40
- 2879 - AARC score predicts survival in patients with Acute on Chronic Liver Failure due to Severe Alcoholic Hepatitis undergoing Plasma Exchange, Jaifrin Daniel
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 02
- 2881 - Impact of an AI-Supported Triage System on the Diagnostic Workflow of Focal Pancreatic Lesions: A Real-World Clinical Utility Analysis, Bo WANG
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- 2886 - Comparative Analysis of Epidemiological Research Output: Metabolic Dysfunction-Associated Steatotic Liver Disease vs. Alcohol-Associated Liver Disease, Pavithra Senadheera
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- 2888 - Evaluation of selected Plasma Long Non-Coding RNAs as predictive Biomarkers in HBV-Related Hepatocellular Carcinoma, Munira Jahan Jahan
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- 2894 - Splenic probe (100 KPa) is superior to liver probe (75 KPa) in assessment of liver stiffness and predicting outcomes in Acute on chronic liver failure patients, Sudhir Verma
23 April 2026, Thursday | 07:30 - 09:00 | ORAL PRESENTATION 02
- 2897 - Integrative Metabolomic and Cytokine Profiling Reveals a "Metabolic-Inflammatory" Signature in Chronic Hepatitis B Combined with Metabolic Dysfunction-Associated Fatty Liver Disease, Yichen Peng
25 April 2026, Saturday | 17:10 - 18:30 | ORAL PRESENTATION 64
- 2904 - Clinical Analysis of Liver Transplantation in Patients With Severe Liver Disease, Jinzhen Cai
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- 2905 - A clinical study on the reduction of donor lymphocyte chimerism rate in recipient tissues after liver transplantation by extracorporeal perfusion of anti-thymocyte globulin via portal vein, Jinzhen Cai
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- 1002 - Assessing the Utility of Serum Kallistatin in the Diagnosis of Liver Cirrhosis: A Meta-analysis, Dr Rukesh Yadav
- 1003 - Is drug- induced autoimmune hepatitis being underdiagnosed?, Thanh Quynh NGAN Le
- 1004 - An acute flare of chronic hepatitis B in a patient with eosinophilic ascites and diabetic ketoacidosis represents a clinical challenge, Ngan Le Thanh Quynh
- 1005 - Drug- induced autoimmune hepatitis, three case reports., Ngan Le Thanh Quynh
- 1008 - Efficacy and Safety of Terlipressin versus Octreotide for esophageal variceal bleeding: A retrospective study at a tertiary hospital in the UAE, Thaer Swaid
- 1012 - Thromboangiitis obliterans in a Non-Smoker Complicated by Budd-Chiari Syndrome: A Rare Case of Visceral Thrombosis, Ziad W. Elmezayen
- 1014 - Validation of the CLIF-SIG score in patients with HBV-related acutely decompensated cirrhosis, Jiyang Chen
- 1015 - Gut Microbial Succession and Escalating Antibiotic Resistance from Chronic Hepatitis B to Acute-on-Chronic Liver Failure: A Metagenomic Study, Jiyang Chen
- 1016 - Single-cell analysis identifies RETN+ monocyte-derived resistin as a therapeutic target in hepatitis B virus-related acute-on-chronic liver failure, Xianbin Xu
- 1024 - Comparison of effectiveness of mycophenolate mofetil associated with standard dosage or reduced-dose tacrolimus for liver transplantation immunosuppression, Ulil Albab Habibah
- 1026 - EXPLORING LABORATORY VARIATIONS IN CIRRHOSIS ETIOLOGIES: A COMPARATIVE ANALYSIS OF CBC AND LIVER SYNTHETIC FUNCTION BETWEEN PATIENTS WITH ALCOHOLIC LIVER DISEASE AND OTHER ETIOLOGIES, Lilit Harutyunyan
- 1027 - Long-term follow-up of HCV patients who treated with DAA [Direct-Acting Antivirals], Eliza Vashakidze
- 1028 - Chronic viral hepatitis [HCV, HBV] course and outcome in patients with severe COVID 19], Eliza Vashakidze
- 1030 - A case of drug-induced liver injury caused by salt-fleshed psoralea corylifolia, Chaojian He
- 1031 - Idiopathic Inflammatory Myopathy Presenting as Hepatic Injury: A Case Report, Chaojian He
- 1032 - Drug-Induced Liver Injury from Shenbao Tablets in an Elderly Patient: A Case Report, Chaojian He
- 1033 - A Rare Case of Peptic Ulcer Perforation Associated with Lenvatinib in Hepatocellular Carcinoma, Zyrhah Lou Samar
- 1036 - Two cases of drug-induced liver damage in the elderly caused by Tusanqi (Gynura japonica), Chaojian He
- 1038 - Comparative Analysis of Epidemiological Characteristics of Hepatitis A and Hepatitis E in Beijing from 2015 to 2024, Chao WANG
- 1040 - Small Intestinal Bacterial Overgrowth: A Missing Link in Gut–Liver Axis Dysregulation of MASLD, Ziteng Wang
- 1041 - Controlled Attenuation Parameter Factors and Steatosis Grading in Metabolic Dysfunction-associated Steatotic Liver Disease, Ningrui Su
- 1043 - Non-invasive detection of occult yet significant liver pathology in ALT-normal chronic hepatitis B: a multicenter study, Xinjie Li
- 1044 - Multifactorial Anemia Unveiling Gastric Antral Vascular Ectasia in a Stage IV Breast Cancer Survivor: Endoscopic Resolution with Argon Plasma Coagulation, John Derek Clutario
- 1047 - Comparative Analysis of Epidemiological Characteristics and Co-infection of Hepatitis B and Hepatitis C in Beijing from 2010 to 2024, Gang Li
- 1048 - Analysis of the Epidemiological Characteristics of Hepatitis B in Beijing from 2008 to 2024, Quanyi Wang
- 1049 - Rifaximin to reduce the risk of overt hepatic encephalopathy in patients undergoing TIPS - real world experience in a diverse population, Charles Gallaher
- 1052 - Pan-cancer analysis identifies hemopexin as a tumor suppressor in HCC via TNF-alpha-mediated mitochondrial apoptosis, Liying Ren
- 1053 - Development and validation of the FAAP model for prognostic stratification in HCC patients treated with TACE, sintilimab plus bevacizumab: a multicenter study, Liying Ren
- 1057 - Serum N-glycomics for non-invasive detection of significant liver pathology across clinical phases of treatment-naïve chronic hepatitis B, Xinjie Li

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- 1059 - The predictive value of different frailty assessment tools for the prognosis of hospitalized patients with liver cirrhosis, **huiling zhu**
- 1060 - Transitional Hepatocytes and Mortality-Associated Gene Programs Drive NASH-Associated Liver Cancer Revealed by Single-Cell Transcriptomics, **Firdian Makrufardi**
- 1061 - Integrative AI-Based Multi-Omics Approach to Uncover Endothelial Diversity and Predict Cholestatic Liver Disease Across Vulnerable Populations, **Firdian Makrufardi**
- 1062 - GALNT7-Mediated Glycosylation of TAZ at Serine 307 Promotes Gallbladder Cancer Progression via Stabilization and Feed-Forward Activation, **Peng Qiu**
- 1063 - Paraspinal Vein Shunt: A Potential Key Factor Leading to Hepatic Myelopathy, **Yuhong Suo**
- 1064 - Analysis of clinical characteristics and risk factors of liver injury in treatment-naïve patients with lung cancer, **Yaqian Mao**
- 1066 - Efficacy of laparoscopic-assisted Microwave Ablation for treating giant hepatic hemangioma: a retrospective controlled study, **Changle Wu**
- 1068 - Gut Microbiota-Derived 3-Methylxanthine Regulating T-Cell Immune Response to Inhibit Viral Replication and Liver Injury in Chronic Hepatitis B comorbid with Metabolic Dysfunction-associated Fatty Liver Disease, **Baoju Wang**
- 1070 - Analysis of Epidemiological Characteristics and Risk Factors of Liver Function Impairment Among Middle-Aged and Older Community Residents in Beijing, **Quanyi Wang**
- 1074 - Mediating role of BMI in alcohol-linked liver enzyme elevation among adults at a tertiary care hospital in South India, **Arivarasan Barathi**
- 1077 - Performance of BAVENO VI and BAVENO VII Criteria for Excluding High-Risk Esophageal Varices in Compensated Cirrhosis with Hepatocellular Carcinoma, **Benjaphol Panyapornsakul**
- 1078 - "When Treatment Triggers Disease: Hepatic Abscess as a Paradoxical Reaction to Antitubercular Therapy", **REBEKA SULTANA MOUSUMI**
- 1081 - Risk factors and outcomes of carbapenem-resistant enterobacteriaceae infection in liver transplant patients: a systematic review and meta-analysis, **Kapil Khanal**
- 1082 - Prediction of Hepatic Functional Reserve Using a Novel "Severity Index" on Hepatobiliary Phase Gadoteric Acid-Enhanced MRI, **Fatih Işık**
- 1083 - Functional Liver Imaging Score on Gadoteric Acid-Enhanced MRI: Correlation with MELD-Na, ALBI, FIB-4 and Clinical Status in Chronic Liver Disease, **Fatih Işık**
- 1084 - Long-Term Virological Response Dynamics and Clinical Outcomes in HBV-Related Cirrhosis: A Real-World Cohort Study, **Yuwei Wang**
- 1086 - COVID-19 Pandemic's Impact on the Global Disease Burden of HBV-related Hepatocellular Carcinoma in the Elderly, **Yuwei Wang**
- 1087 - Bibliometric Analysis and Research Trends of Specialty Theses on Cirrhosis in Turkey, **AHMET UYANIKOGLU**
- 1088 - The cGAS-STING-IFITM3 Axis Drives Monocyte Phenotypic Shift and Immune Dysregulation in Acute-on-Chronic Liver Failure, **JUNYI WANG**
- 1089 - CEP290: a primary cilium-related gene as the positive feedback regulator of Nrf2 that modulates liver cancer cells ferroptosis, **Ruilian Ma**
- 1090 - Hermansky-Pudlak syndrome (HPS), **Vedat Goral**
- 1092 - Triple A (Allgrove) Syndrome, **Vedat Goral**
- 1093 - Towards HCV Elimination: Time for Clinical and Laboratory Collaboration, **Burcu Çalışkan Demirkıran**
- 1094 - Oxidative Stress-Immunotherapy Gene Signatures Define Prognosis and Immune Subtypes in Hepatocellular Carcinoma, **Yuxin Liang**
- 1095 - Efficacy and safety of postoperative adjuvant HAIC combining lenvatinib with or without PD-1 inhibitors in solitary large HCC: A multicenter retrospective study, **Yuxin Liang**
- 1097 - The Roadtrip : PEERS AND MEDICAL STAFF IS THE BEST MATCH TO TEST AND TREAT DRUG USERS FOR HEPATITIS C, **juliette PONT**
- 1100 - MRI differential diagnosis of hepatic metastasis of choroidal melanoma in the patient with hepatitis B cirrhosis, **Chenxi Liu**

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- 1106 - BEDSIDE NUTRITIONAL ASSESSMENT IN PATIENTS WITH LIVER CIRRHOSIS USING MINI NUTRITIONAL ASSESSEMENT PROFORMA, **Asif Khan**
- 1107 - Serum fibroblast growth factor 21 as a novel biomarker of cachexia in chronic liver disease, **Takatsugu Tanaka**
- 1108 - Comparison of Liver Biopsy with Shear-Wave Elastography and Other Non-Invasive Fibrosis Markers in Chronic Hepatitis B, **Fatih Albayrak**
- 1109 - Stereotactic body radiation therapy as an add-on to targeted agents in patients with locally advanced hepatocellular carcinoma, **Mohamed Hamouda Elkasaby**
- 1111 - Effectiveness and safety of Fibrate Therapy in the treatment of adults with Primary Sclerosing Cholangitis: A Systematic Review and Meta-Analysis, **Mark Angelo Pado**
- 1114 - Detection of HBV and HCV seroprevalence using rapid point-of-care tests among earthquake survivors in Southeastern Türkiye, **Muhammed Bekçi**
- 1115 - Effectiveness and Follow-up Challenges of Sofosbuvir/Velpatasvir in HCV/HIV Co-infected Patients: A Real-world Experience from a Chinese Cohort, **Wei Lv**
- 1119 - Metabolic Associated Fatty Liver Disease in Obese and At-Risk Children and Adolescents in Türkiye: A Meta-Analysis of Prevalence, **Eda Kaya**
- 1120 - Research Trends of Targeted Drugs for Nonalcoholic Fatty Liver Disease: A Bibliometric Analysis (2004–2024), **Xingru Wang**
- 1124 - Single-arm meta-analysis and systematic review on clinical cure of chronic hepatitis B after hematopoietic stem cell transplantation: immunoregulatory mechanisms and clinical translational insights, **He Ma**
- 1128 - Zinc-alpha 2-glycoprotein (ZAG) emerges as a diagnostic biomarker for Hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF), **Daxian Wu**
- 1129 - Efficacy of albumin infusion for hypoalbuminemia in post-operative period in living donor liver transplantation – An open label randomized controlled trial, **Vipul Gautam**
- 1131 - Dietary Total Antioxidant Capacity and Metabolic Dysfunction-Associated Steatotic Liver Disease: a cross-sectional study, **Yani Zhou**
- 1135 - Impact of Low-Dose Rifaximin on Gut Antibiotic Resistance Genes in Patients with Decompensated Cirrhosis, **Zhiyuan Hao**
- 1136 - Catastrophic unmasking: Fatal tuberculosis-associated Immune Reconstitution Inflammatory Syndrome presenting as acute liver failure in pregnancy, **Carmela Bianca Villacete**
- 1138 - Peripheral Single-Cell Immune Profiling of HBsAg Loss in HBV-Related Hepatocellular Carcinoma Receiving Anti-PD-1 Therapy, **Pengyuan Kang**
- 1142 - From Naïve to Non-Responders under DAA Pressure: Unveiling NS5A and NS5B Resistance Associated Substitutions in HCV Genotype 3a from Bangladesh, **Mahmuda jahan Jahan**
- 1143 - Association between AST/ALT ratio and Hemodynamic Parameters of Carotid Arteries in Patients with Primary Aldosteronism: A Cross-Sectional Study, **Mengbo Wu**
- 1145 - Targeting Cancer-Associated Fibroblast Subpopulations and Metabolic Pathways to Enhance Immunotherapy in Hepatocellular Carcinoma, **Xin Song**
- 1146 - Treatment with direct-acting antivirals in HCV-infected patients after liver transplantation, **Dorota Zarębska-Michaluk**
- 1148 - Deciphering the Mechanism of Aristolochic Acid I-Driven Hepatocellular Carcinoma through Integrated Network Toxicology and Bioinformatics, **Yuling Liang**
- 1150 - Evaluation of high-sensitivity HBcrAg and HBsAg assays as biomarkers for occult HBV infection and hepatocellular carcinoma risk, **Yi-Chung Hsieh**
- 1151 - HDV Screening Practices in Chronic Hepatitis B: Real world Data from a Decision Support App in the Context of Global Guidance, **Grace Wong**
- 1152 - Clinical Efficacy of Robot-Assisted Biliary-Enteric Reconstruction in Radical Resection of Hilar Cholangiocarcinoma: Single-Center Practical Experience, **Yang Wang**
- 1154 - Clinical and epidemiological profile of Hepatitis A outbreak among medical students at Shaheed Ziaur Rahman Medical College hostel: cross-sectional study, **Abu Saleh Md Sadequul ISLAM**

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- 1155 - MASLD AND GLYCEMIC IMPROVEMENT AFTER SUSTAINED VIROLOGIC RESPONSE IN TYPE 2 DIABETES PATIENTS WITH CHRONIC HEPATITIS C: A THREE-YEAR FOLLOW-UP STUDY, Ching-Chu Lo
- 1160 - Study on the Mechanism of Samhd1 Mediated NEMO Polyubiquitination in Liver Ischemia-Reperfusion Injury, Xianzhang Luo Luo
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ORAL ABSTRACTS

1006 - Ultrasound derived Fat Fraction is a Reliable Method to Grade Hepatic Steatosis in Patients with Chronic Liver Diseases A Multicenter Study in a Population with Mixed Etiologies

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Background / Hypothesis: To investigate the performance of ultrasound derived fat fraction (UDFF) on grading hepatic steatosis in CLD patients applying liver biopsy as the gold standard. Also, the potential factors that affect the diagnostic performance of UDFF will also be analyzed.

Materials and Methods: This prospective study enrolled patients suspicious for or confirmed CLD, and scheduled for liver biopsy from October 2024 to June 2025 at four tertiary care centers. UDFF was measured on the same day of liver biopsy and the region of interest (ROI) contained the biopsy site. The performance of UDFF on detecting hepatic steatosis $\geq S1$, $\geq S2$, and $=S3$ was analyzed by ROC curve.

Results: A total of 255 patients with CLD were included in the final analysis, with a mean age of 52.00 (14.00) years old. The counts of patients with hepatic steatosis of S0, S1, S2, and S3 were 146(57.25%), 40(15.69%), 30(11.76%), and 39(15.30%). The AUCs of UDFF on detecting hepatic steatosis $\geq S1$, $\geq S2$, and of S3 were 0.914 (0.873 ~ 0.946), 0.950 (0.916 ~ 0.974), 0.916 (0.875 ~ 0.947). The optimal cutpoints of UDFF to grade hepatic steatosis $\geq S1$, $\geq S2$, and of S3 in this cohort were $\geq 7\%$, ≥ 11 , and $\geq 18\%$. The AUC of UDFF on detecting hepatic steatosis $\geq S1$ in patients with inflammation of G2~3 is significantly higher than those with G0~1 [0.969 (0.929 ~ 0.990) vs. 0.888 (0.805~0.945), $p = 0.043$].

Conclusions: UDFF is a reliable method to noninvasively grade hepatic steatosis in CLD patients regardless of the etiology, stage of inflammation as well as fibrosis.

Keyword(s) : chronic liver disease; ultrasound; hepatic steatosis; liver biopsy

HCV Related Cirrhosis A Multicenter Retrospective Study in Egypt

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Background / Hypothesis:

HCV infection remains a major public health issue in Egypt, with a high prevalence of genotype 4. Direct-acting antivirals (DAAs) achieve >95% sustained virologic response (SVR), but their impact on variceal rebleeding in genotype 4 cirrhotic patients is underexplored. This study evaluated the association between DAA therapy and variceal rebleeding in Egyptian patients with HCV-related cirrhosis.

Materials and Methods: A multicenter retrospective cohort study included HCV genotype 4 cirrhotic patients from five Egyptian centers with a first variceal bleeding episode. Patients were divided into DAA-treated (Group A) and non-treated (Group B) groups and followed for 5 years. Propensity score matching, Cox regression, and competing risk analysis were adjusted for confounders.

Results:

DAA treatment significantly reduced variceal rebleeding (HR 2.57; 95% CI 1.39–4.72; $p = 0.002$), ascites development over 5 years (6.8% vs. 27.1%, $p = 0.006$), and hepatic dysfunction progression. During treatment, it improved liver function (lower MELD, stable Child-Pugh class) and reduced complications. All Group A patients achieved SVR by PCR, while Group B remained HCV positive, likely contributing to the observed reductions in rebleeding and hepatic decompensation. These benefits persisted over 5 years, with longer survival without rebleeding (4.5 vs. 3.2 years), lower MELD (7 vs. 12, $p < 0.001$), and reduced hepatic

1007 - Direct Acting Antiviral Therapy Reduces Variceal Rebleeding and Improves Liver Function in

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decompensation (Child-Pugh progression: 5.1% vs. 35.6%, $p < 0.001$). At 5 years, the DAA group had better liver function (higher albumin, lower INR, improved platelets), while the non-DAA group worsened. PSM confirmed these findings (HR: 0.45, 95% CI: 0.27–0.75, $p = 0.002$), and competing risk analysis showed sustained protection (sub distribution HR: 0.44, 95% CI: 0.26–0.74, $p = 0.002$). Endoscopy revealed variceal regression with DAA but progression in the non-DAA group. DAA therapy significantly reduced variceal rebleeding, hepatic decompensation, and mortality (8.5% vs. 20.3%, $p = 0.045$), with survival benefits linked to sustained virologic response (SVR). Additionally, it was associated with improved survival, with a lower 5-year mortality rate in the DAA group (8.5% vs. 20.3%, $p = 0.045$).

Conclusions:

DAA treatment in HCV-related cirrhosis significantly reduces variceal rebleeding, ascites development, and hepatic dysfunction progression. The 5-year follow-up data demonstrate sustained improvements in liver function and hematologic parameters, underscoring the long-term benefits of DAA therapy.

Keyword(s) : Direct-acting antivirals (DAAs), variceal rebleeding, portal hypertension, sustained virologic response (SVR), genotype 4, Egypt.

1009 - Liver Resection versus Trans arterial Chemoembolization for Huge Hepatocellular carcinoma (>10cm) A nationwide Propensity score adjusted analysis

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Background / **Hypothesis:**
The treatment of hepatocellular carcinoma (HCC) > 10 cm remains controversial. The ongoing debate regarding the treatment of patients with HCC > 10 cm is partly due to the necessity of tailoring treatment to individual patient conditions. This study aims to address the important controversy regarding the management of large HCC, focusing on providing insights into the comparative effectiveness of liver resection (LR) and Transarterial Chemoembolization (TACE).

Materials

and

Methods:

Patients registered in the Korean Primary Liver Cancer Registry (KPLCR) were selected. The KPLCR is a random sample consisting of approximately 15% of patients newly diagnosed with primary liver cancer registered in the Korean Central Cancer Registry (KCCR), which is a nationwide cancer registry and includes more than 95% of all cancer cases in Korea. The study design utilizes data from the KPLCR covering the years 2008 to 2017 to investigate the treatment outcomes for HCC larger than 10 cm. Starting with 15,307 cases, the study identified 9,310 HCC patients with follow-up data, excluding those with missing initial treatment data ($n=5,863$) and follow-up loss ($n=132$). From these, 7,662 patients with single HCC were selected, further excluding cases with multiple HCC ($n=1,394$) and distant metastasis ($n=254$). This refined the cohort to 504 patients with HCC tumors larger than 10 cm, who were then divided into two treatment groups: 122 patients underwent liver resection (LR), and 382 patients received TACE.

Results:

After PSM, the baseline characteristics showed no significant differences between the LR group ($n=99$) and the TACE group ($n=99$). Before propensity score matching (PSM), the hazard ratio (HR) for LR was 3.41 (95% CI: 2.62–4.43, $p<0.001$), indicating a significantly better survival probability for the LR group compared to the TACE group. After PSM, the HR for LR was slightly reduced to 3.06 (95% CI: 2.18–4.29, $p<0.001$), but the survival advantage of LR over TACE remained highly significant. Before PSM, the HR for LR was 3.29 (95% CI: 2.59–4.17, $p<0.001$), indicating a markedly better recurrence-free survival (RFS) for the LR group compared to the TACE group. After PSM, the HR for LR increased slightly to 3.37 (95% CI: 2.43–4.68, $p<0.001$), maintaining the significant survival advantage of LR over TACE.

Conclusions:

This study provides a comprehensive analysis of treatment outcomes for HCC > 10 cm, comparing LR and TACE using data from the KPLCR. The results indicate that LR is associated with significantly better OS and RFS compared to TACE, both before and after PSM.

Keyword(s) : HCC, Hepatectomy, Prognosis, Survival

1010 - A Five Year Retrospective Analysis of Hepatic Fibrosis and Steatosis Assessed by Transient Elastography in Patients Across the Glycemic Continuum

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Background / Hypothesis: Liver fibrosis and steatosis are key prognostic markers in metabolic dysfunction-associated fatty liver disease (MAFLD), which is now the leading cause of chronic liver disease worldwide. Since the liver is central to glucose and lipid metabolism, a two-way link exists between MAFLD and diabetes mellitus, with each condition raising the risk of the other. This study aimed to explore the relationship between glucose tolerance, liver stiffness, and steatosis measured by transient elastography (FibroScan), while also evaluating related metabolic parameters.

Materials and Methods: We conducted a five-year retrospective analysis of 564 adults who underwent FibroScan between 2020 and 2024. Participants were categorized by glucose status into normoglycemic, prediabetic, and diabetic groups. One-way ANOVA, chi-square tests, multinomial logistic regression, and multivariate analysis of covariance were used to analyze differences and associations among groups.

Results: The mean age was 50 ± 14 years, and the mean BMI was 27 ± 4.6 kg/m². Among participants, 54.8% were female. Significant differences were observed among glycemic groups in liver stiffness, steatosis, BMI, and lipid profile (all $p < 0.05$). Compared to diabetics, normoglycemic individuals had significantly lower odds of intermediate fibrosis (87.4% reduction) and probable cirrhosis (87.7% reduction), while prediabetics also showed reduced odds (71.7% and 86.2%, respectively). Diabetic patients had notably higher total cholesterol, triglycerides, and LDL cholesterol compared to other groups.

Conclusions: These findings show that impaired glucose tolerance is linked to more severe liver fibrosis and steatosis, with diabetic individuals experiencing the most significant hepatic and metabolic changes. The results emphasize the importance of early metabolic screening and blood sugar management to lower the risk of liver-related complications in people with prediabetes and diabetes.

Keyword(s) : fibrosis, steatosis, liver stiffness, transient elastography, FibroScan, CAP score, diabetes mellitus, prediabetes, metabolic factors, BMI, ALT, lipid profile, HDL cholesterol, LDL cholesterol, triglycerides

1011 - Predictors of non response to antiviral therapy for hepatitis C in Almaty the largest city of

Kazakhstan

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Background / Hypothesis: Despite the proven efficacy of direct-acting antiviral (DAA) therapies for chronic hepatitis C virus (HCV), treatment failures still occur in some patients. This study aimed to evaluate the effectiveness of these therapies in routine clinical settings and to identify factors that may influence the likelihood of achieving a sustained virological response (SVR).

Materials and Methods: A retrospective observational study was conducted at the Hepatological Center in Almaty involving 136 adult patients diagnosed with chronic hepatitis C and treated with Sofosbuvir + Daclatasvir ± Ribavirin within the state-guaranteed program (2018–2022). Cases (n=34) were defined as patients without SVR; controls (n=102) achieved SVR. Groups were matched 1:3 by age, sex, and ethnicity. Data on demographics, HCV genotype, fibrosis stage, comorbidities, laboratory parameters, treatment regimen, and adherence were collected. Univariable and multivariable logistic regression analyses were performed to identify independent predictors of treatment failure.

Results: The study population consisted predominantly of males (78.7%), with a mean age of 46 years and a mean BMI of 26.3 kg/m²; 52.2% were of Asian ethnicity. Previous interferon-based therapy was reported in 3.7% of patients and was significantly more frequent among non-responders (11.8% vs. 1.0%; $p = 0.0147$). In univariable analysis, genotype 3, advanced fibrosis, elevated ALT levels, obesity, peptic ulcer disease, HIV coinfection, and arterial hypertension were associated with an increased likelihood of treatment failure. Multivariable regression confirmed seven independent predictors of non-response: genotype 3 was associated with a more than two-fold increase in the odds of non-response compared to genotype 1 (AOR 2.33, 95% CI 1.26–3.35), and advanced fibrosis or cirrhosis (F4) also increased the risk almost two-fold (AOR 1.99, 95% CI 1.21–4.07), while arterial hypertension nearly doubled the risk of failure (AOR 1.94, 95% CI 1.03–3.36). Longer disease duration prior to treatment and elevated baseline ALT were modest but significant predictors (AOR 1.006 each, 95% CI just above 1.0). In contrast, extending therapy to 24 weeks proved protective, reducing the likelihood of failure by two-thirds

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compared with 12-week regimens (AOR 0.33, 95% CI 0.10–0.75). The most striking finding was that poor adherence to therapy dramatically increased the risk of non-response (AOR 87.1, 95% CI 23.7–313.1). Conclusions: DAA therapy remains highly effective for CHC in Kazakhstan; however, a subgroup of patients demonstrates suboptimal outcomes. Independent predictors of non-response include genotype 3, advanced fibrosis, elevated ALT, arterial hypertension, prolonged disease duration, and poor adherence, while extended treatment duration showed a protective benefit.

Keyword(s) : Hepatitis C, DAA, SVR, Non-response

1013 - Non invasive prediction of hepatic inflammation grade in chronic hepatitis B patients based on multiple parameters non enhancement MRI combined with clinical characteristics

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Background / **Hypothesis:**
Liver biopsy remains the gold standard for assessing hepatic inflammation in CHB (chronic hepatitis B) patients, yet its clinical utility is limited by drawbacks such as delayed reporting and potential complications. Building upon our prior research, multiple parameters MRI combined with clinical characteristics may serve as a potential non-invasive approach for evaluating hepatic inflammation.

Materials and **Methods:**
Between May 2020 and April 2024, we prospectively enrolled 100 consecutive CHB patients who underwent DWI ($b = 800 \text{ s/mm}^2$) and T2WI within 6 hours prior to liver biopsy on a GE 3.0T Signa HDxt MRI scanner. The imaging protocols were routine non-enhancement MRI protocols included RT-DWI (respiratory-triggered DWI), T2 FS (T2-weighted fat-suppressed imaging), and T1 DUAL (dual-echo T1-weighted imaging). ADC (Apparent diffusion coefficient) values derived from DWI and SII (signal intensity index) values derived from T2WI which were defined as the ratio of hepatic signal intensity adjacent to the liver biopsy site in the right liver lobe to that of the right erector spinal muscle at the same level were measured using ROI (region-of-interest) delineation by two observers. Meanwhile, clinical

characteristics of patients within 3 days before biopsy including age, age range, gender, ALT, HBV DNA exponents, HBsAg, TBIL, DBIL, ALB and PLT were documented. Using pathological hepatic inflammation grade as the gold standard, patients were classified into a case group (N=50, inflammation grade $\geq G2$) and a control group (N=50, inflammation grade $\leq G1$). Univariate and multivariable logistic regression were used to identify the significant independent risk factors and establish a predictive model. ROC (Receiver operating characteristic) analysis was performed to evaluate the diagnostic performance of radiological model, clinical model and combined model for predicting hepatic inflammation. Calibration plots and DCA (decision curve analysis) curve were generated using bootstrapping.

Results:

The radiological model was constructed by combination of ADC and SII. For the clinical model, age, ALT, and HBV DNA were identified as independent predictors of inflammation grade $\geq G2$ and thus included in its development. The combined model was established by merging the radiological and clinical predictors. AUC of radiological model in predicting liver inflammation grade $\geq G2$ was 0.904, the AUC of clinical model was 0.862, and the AUC of combined model was 0.947. Calibration plot and DCA curve of the combined model demonstrated good predictive performance. Conclusions: Multiple parameters MRI combined with clinical characteristics achieved superior accuracy in predicting hepatic inflammation grade $\geq G2$. This approach provides a non-invasive method, serving as a potential biomarker for early clinical assessment of pathological

Keyword(s) : Magnetic Resonance ; Chronic Hepatitis B ; Diffusion Weighted Imaging ; Apparent Diffusion Coefficient ; Liver

1022 - SLC1A5 dependent glutamine transport regulates macrophage senescence and efferocytosis during liver regeneration

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Background / **Hypothesis:**
Liver regeneration is an extraordinary process vital for the maintenance of homeostasis, governed by complex metabolic and immune interactions, particularly concerning the function of macrophages. Prior research underscores the significant role of these immune cells in the repair of tissue; nevertheless, the precise

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mechanisms that govern macrophage phenotypes and their functions throughout liver regeneration are not well understood. This study aims to investigate the role of SLC1A5, a neutral amino acid transporter responsible for glutamine transport, in macrophage senescence and efferocytosis during liver regeneration.

Materials and Methods: We employed myeloid-specific SLC1A5 knockout mice (SLC1A5^{fl/fl}/fLy2cre) in conjunction with a partial hepatectomy model to examine the effects of macrophage SLC1A5 deficiency on liver regeneration. Bone marrow-derived macrophages (BMDMs) were extracted and subjected to RNA sequencing and a range of functional assays to assess macrophage senescence, efferocytosis, and cytokine production levels.

Results:

The results indicate that SLC1A5 is essential for thwarting macrophage senescence and maintaining efferocytosis during liver regeneration. Macrophages lacking SLC1A5 showed elevated senescence markers and transitioned towards a pro-inflammatory phenotype, marked by increased levels of TNF- α and IL-6. Significantly, efferocytosis was compromised due to the reduced expression of Gas6, a critical ligand for macrophage efferocytosis. Treatment with L-glutamine and recombinant Gas6 effectively restored macrophage functions and enhanced liver regeneration in vivo.

Conclusions:

This investigation demonstrates that SLC1A5 is integral in modulating macrophage functions via glutamine transport during liver regeneration. The enhancement of glutamine availability may offer a new therapeutic strategy to alleviate macrophage senescence and promote liver recovery following injury.

Keyword(s) : liver regeneration, macrophage, SLC1A5, cell senescence, efferocytosis

1023 - Real world Clinical and Procedural Insights into Liver Abscesses A Multidimensional Retrospective Analysis from a Tertiary Center in North India

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Background / Hypothesis: Liver abscess is a major cause of morbidity in tropical countries, with varied etiologies and outcomes. Data on large cohorts from real-world settings remain limited. Our study aimed to evaluate demographic, clinical, etiological, imaging, management, and outcome patterns in liver abscess and identify predictors of

prolonged hospitalization and mortality.

Materials and Methods: We retrospectively analyzed 1,408 confirmed liver abscess cases admitted between January 2018 and December 2023. Data on demographics, comorbidities, imaging, abscess characteristics, complications, management, and outcomes were extracted from discharge records. Hospital stay was dichotomized as ≤ 7 vs. > 7 days. Statistical analyses included univariate tests and multivariate logistic regression.

Results:

The mean age was 39.1 ± 17.5 years; 84.0% were male. Overall mortality was 5.5% (n = 77). Common etiologies were amebic (7.5%), pyogenic (7.0%), and unidentified (82.0%). Amebic abscesses had larger volumes but no mortality; pyogenic abscesses were linked to prolonged stays and higher mortality. Bilobar involvement and complications such as rupture, peritonitis, sepsis, shock, and anemia significantly worsened outcomes. Culture positivity was low (3.3%), with E. coli, Pseudomonas, and A. baumannii predominating. Ultrasound was the primary diagnostic tool (77.4%). Percutaneous catheter drainage (PCD) was used in 61.9%; discharge with PCD in situ was associated with shorter stay (p = 0.001) and low mortality (0.5%). Multivariate analysis showed younger age (OR 0.82; p = 0.001) and female gender (OR 1.19; p = 0.0025) predicted prolonged stay, while older age independently predicted mortality (OR 1.64; p < 0.0001).

Conclusions:

Liver abscess remains a heterogeneous disease with significant morbidity and mortality. Early identification of high-risk patients based on age, gender, etiology, and complications, along with safe early discharge protocols using PCD in situ, may reduce hospital stay and improve outcomes. Enhancing microbiological yield and optimizing imaging strategies are key future priorities.

Keyword(s) : Liver abscess, Percutaneous catheter drainage, Hospital stay, Mortality predictors, Complications

1025 - Real time multidimensional system for predicting survival in patients with advanced biliary tract cancer treated with immuno chemotherapy

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Background / Hypothesis: First-line treatment for advanced biliary tract cancer (BTC) is now immunotherapy-chemotherapy. However, response varies significantly among patients, requiring ongoing monitoring and treatment adaptation. We aimed to develop an AI system that dynamically updates survival predictions for advanced BTC patients using evolving clinical data, enabling real-time prognostic stratification.

Materials and Methods: This study included patients with advanced BTC receiving immuno-chemotherapy across eight centres. Patients were divided into development, internal validation, and external validation cohorts (two retrospective, one prospective). We developed a Bayesian joint model, the Individualized Dynamic Risk Estimation Model for BTC (iDREM-BTC), that integrated baseline clinical and imaging data with serial measurements of C-reactive protein (CRP), carbohydrate antigen 19-9 (CA19-9), and total bilirubin (TBil). An enhanced model (iDREM(Pro)-BTC) also incorporated baseline immunohistochemistry and genetic data. Performance was evaluated via dynamic area under the curve (AUC) and calibration analyses.

Results: Data from 2314 patients were analysed (development cohort, n=841; internal validation cohort, n=360; external validation cohorts, n=1113). Machine learning methods identified age, tumour burden, biliary obstruction severity, ascites, CRP, CA19-9, and TBil as predictors of mortality. iDREM-BTC demonstrated robust performance in the development cohort (overall dynamic AUC 0.730 [95% CI 0.689–0.794]), internal validation cohort (0.718 [95% CI 0.668–0.778]), external validation cohort (0.755 [0.707–0.808]), external validation cohort 2 (0.705 [0.639–0.773]), and prospective external validation cohort (0.745 [0.691–0.802]). iDREM(Pro)-BTC achieved better discrimination in the development cohort (AUC 0.807 [95% CI 0.781–0.839]), and external validation confirmed these findings (0.718 [0.669–0.787]).

Conclusions: iDREM/(Pro)-BTC offer dynamic, real-time prognostic prediction for patients with advanced BTC undergoing immuno-chemotherapy. The two models suit different clinical scenarios, providing individualized prognostic stratification.

Keyword(s) :

1029 - Global burden of hepatitis A virus 1990–2023 results from the Global Burden of Disease Study 2023

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Background / Hypothesis: This study drew on data from the Global Burden of Disease (GBD) 2023 to examine global epidemiological patterns and long-term trends in hepatitis A virus (HAV) infection between 1990 and 2023.
Materials and Methods: Within the GBD 2023 framework, data from 204 countries and territories were synthesized from population-based surveys, clinical records, and published literature. HAV cases were defined according to ICD-10 codes B15–B15.9. Incidence and mortality were estimated using the Bayesian meta-regression tool DisMod-MR 2.1, and age-standardized rates (ASRs) were calculated.
Results:

In 2023, there were approximately 156,185,264 new cases of HAV worldwide, with an age-standardized incidence rate (ASIR) of 2,248.8 per 100,000 population. The global age-standardized mortality rate (ASMR) was 0.47 per 100,000, with 36,484 deaths. Incidence and mortality were highest in low-income regions, especially South Asia, followed by Oceania, and lowest in developed East Asia and Eastern Europe. Disability-adjusted life years (DALYs) from HAV fell from 8,747,097 in 1990 to 2,401,925 in 2023, with the lightest burden in Western Europe, Australasia, and high-income Asia-Pacific regions. Australasia saw the largest relative increase in the age-standardized DALY rate (4.9%). Across regions, there was a negative correlation between the Socio-Demographic Index (SDI) and the age-standardized DALY rate ($R = -0.58$). The age-standardized DALY rate declined sharply as SDI increased up to about 0.54, then the decline slowed but continued overall. Some exceptions were noted: San Marino, Qatar, Andorra, Estonia, and Latvia had higher-than-expected DALY rates despite high SDI, whereas Burkina Faso, Senegal, The Gambia, Timor-Leste, and Zimbabwe had lower-than-expected HAV burdens despite low SDI.

Conclusions: Between 1990 and 2023, the global burden of HAV fell sharply, though the impact varied widely across regions due to socioeconomic factors. These findings

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underscore the importance of targeted public health interventions and policies in areas where HAV remains a significant burden. Further research is needed to better understand the complex epidemiology of HAV and to inform the development of effective prevention strategies.

Keyword(s) : hepatitis A virus, disability-adjusted life years, incidence, mortality, public health

1034 - Charting immune and cancer metabolism co evolution during metastasis

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Metastasis, the leading cause of cancer death, exhibits a highly heterogeneous and suppressive cancer microenvironment. Here, we sequenced 97 matched samples by using single-cell RNA sequencing and spatial transcriptomics of colorectal cancer liver metastasis. Strikingly, the metastatic microenvironment underwent remarkable spatial reprogramming of immunosuppressive cells such as MRC1+ CCL18+ M2-like macrophages. We further developed scMetabolism, a computational pipeline for quantifying single-cell metabolism, and observed that those macrophages harbored enhanced amino acid metabolic activity. Interestingly, neoadjuvant chemotherapy could block this status and restore the antitumor immune balance in responsive patients, whereas the nonresponsive patients deteriorated into a more suppressive one. In parallel, metastatic cancer cells displayed the metabolic switch from OXPHOS to glycolysis, which is dependent on the metabolic master regulator LY6E. Our work described the metabolism co-evolution of both immune and cancer cells during metastasis and uncovered the black box of how tumors respond to neoadjuvant chemotherapy.

Keyword(s) : Metastasis, Metabolism, Immune microenvironment

1035 - Targeting PAK1 on tumour associated macrophages enhances the efficacy of immunotherapy in hepatocellular carcinoma

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Background

Hypothesis:

The tumour microenvironment (TME) is an important mediator of tumour immunotherapy resistance. Unfortunately, hepatocellular carcinoma (HCC) does not respond to immunotherapy due to a lack of enhancing inflammation in tumor microenvironment (TME), a contributing factor is P21 (RAC1) Activated Kinase 1 (PAK1). PAK1 is selectively highly expressed in tumour-associated macrophages (TAMs). How PAK1 impacts the TME remains under study.

Materials and Methods:

We investigated the role of the PAK1/mTORC1/TFEB pathway in HCC development by conducting metabolomic analysis, gene expression profiling and immunohistochemistry analyses in RAW264.7 cells, oncogene-induced HCC mouse models and human HCC samples, and further explored the molecular mechanism by which PAK1 promoted macrophages M2 polarization.

Results:

PAK1 is selectively expressed in different types of tumours, and was associated with adverse clinical outcome in patients with cancer. We show that targeting PAK1 is a novel and tolerable approach to significantly reverse M2 transition to inhibit tumor development in vitro experiment. This approach reshaped the tumour immune microenvironment, reduced IL-10 and TGF- β expression of macrophages, together with increasing expression and nuclear localization of TFEB. Mechanistically, PAK1 promoted M2 polarisation of macrophages by activating mTORC1/TFEB pathway.

Conclusions:

The PAK1/mTORC1/TFEB pathway plays an important role in HCC, targeting PAK1 represents a potential strategy to enhance the efficacy of immunotherapy in HCC patients.

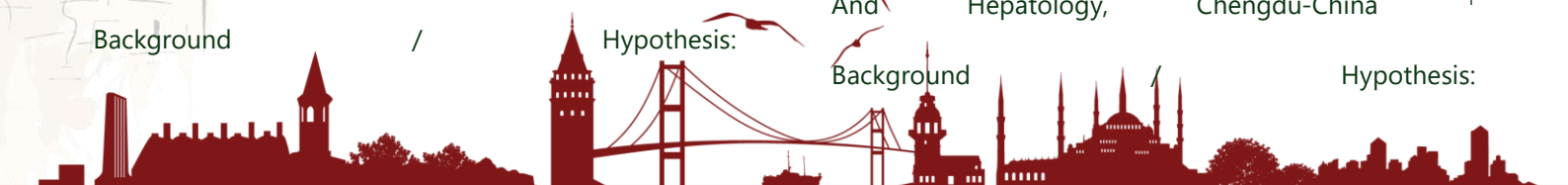
Keyword(s) : PAK1, TFEB, macrophage polarization, tumor microenvironment, hepatocellular carcinoma

1037 - Engineered Vesicle Mediated Metabolic Regulation and Immune Targeting for the Treatment of Autoimmune Hepatitis

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Background

Hypothesis:



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Metabolic disorder promotes CD4+ T-cell activation and associated inflammatory liver injury in autoimmune hepatitis (AIH). Strategies that target CD4+ T-cell metabolism hold great prospects for treatment. Here, we investigated the energy metabolic changes of CD4+ T cells in AIH patients and mice. Furthermore, we constructed a novel targeted delivery nanosystem, that is, a hybrid vesicle (Hy-EV) of metformin engineered mitochondria-rich extracellular vesicle (Met-EV) and dendritic cell membrane vesicle (DCMV), for the treatment of AIH.

Materials and Methods: The metabolic profiles of CD4+ T cells were characterized in human AIH and mouse hepatitis models. The liver protective effect of Hy-EV was evaluated by transaminase levels, histopathology, and inflammation. The targeting ability of Hy-EV was quantitatively detected using an IVIS imaging system and a confocal microscopy.

Results: Significant differences were found in the glycolysis and OXPHOS-related gene expression in CD4+ T cells between normal control and AIH group. Hy-EV treatment effectively rescued liver injury in AIH mice. In-depth exploration found that Hy-EV inherited Met-EV's ability to reduce CD4+ T-cell activation and cytokine release by reducing glycolysis while enhancing mitochondrial oxidative phosphorylation in such cells. In addition, DCMV component of Hy-EV displayed enhanced targeting potency for hepatic CD4+ T cells due to the overexpression of the key molecules which mediated DC-T cell contact. Lastly, multiple long-term administration of Hy-EV showed no systemic toxicity.

Conclusions: Hy-EV exploits a synthetic effect in enhancing efficacy and targeting in the treatment of AIH, which is of high value to serve as a viable strategy in clinical practice.

Keyword(s) : Autoimmune Hepatitis, Engineered Vesicle, Metabolic Regulation, Immune Targeting

1039 - Preoperative TACE with PD 1 Inhibitors and TKIs in Beyond "Up to Seven" Hepatocellular Carcinoma A Propensity Score Matched Analysis

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China ¹

Background / Hypothesis: Hepatocellular carcinoma (HCC) is the sixth most common malignancy globally, and patients with high tumor burden, particularly those exceeding the "up-to-seven" criteria, are at increased risk for recurrence. Despite advances in treatment strategies, effective perioperative management for these high-risk patients remains limited. This study aimed to evaluate the postoperative efficacy and safety of preoperative systemic therapy, including transarterial chemoembolization (TACE) combined with PD-1 inhibitors and tyrosine kinase inhibitors (TKIs), in HCC patients exceeding the "up-to-seven" criteria.

Materials and Methods: We retrospectively analyzed 126 HCC patients who underwent curative liver resection between January 2021 and June 2024. Of these, 73 patients received preoperative TACE with PD-1 inhibitors and TKIs (PST group), and 53 underwent direct surgery without preoperative therapy (Surgery group). Propensity score matching (PSM) was applied to control for confounding factors, resulting in 39 matched pairs. The primary endpoints were recurrence-free survival (RFS) and overall survival (OS), with secondary endpoints including intraoperative blood loss, surgical time, and complications.

Results: After matching, the PST group demonstrated significantly superior RFS and OS compared to the Surgery group ($P < 0.05$). The median RFS was 26 months in the PST group versus 13 months in the Surgery group ($P = 0.029$). There were no significant differences in postoperative complications between the two groups ($P = 0.365$). The PST group exhibited longer surgical times ($P < 0.001$) and slightly higher intraoperative blood loss, suggesting increased surgical complexity due to preoperative therapy. Treatment-related adverse events were predominantly grade 1-2, with few grade 3-4 events.

Conclusions: Preoperative TACE combined with PD-1 inhibitors and TKIs provides significant survival benefits without increasing the risk of severe postoperative complications in patients with beyond-up-to-seven HCC. This regimen may be an effective strategy for perioperative management in high-risk HCC patients, offering a promising option for improving long-term outcomes.

Keyword(s) : Neoadjuvant Therapy; Hepatocellular Carcinoma (HCC); Up-to-seven; Propensity Score Matching (PSM)

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1042 - Non invasive detection of occult yet significant liver pathology in ALT normal chronic hepatitis B a multicenter study

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Background / **Hypothesis:**
Individuals with chronic hepatitis B (CHB) may harbor occult yet significant liver pathology despite normal alanine aminotransferase (ALT) levels, representing a major diagnostic challenge. We aimed to identify and validate non-invasive biomarkers for detecting significant liver pathology among these population.

Materials and **Methods:**
This multicenter study screened 3,258 CHB cases from 2013-2023, enrolling 137 treatment-naïve HBeAg+ and 253 HBeAg- infection patients with normal ALT (≤ 40 U/L). All participants underwent liver biopsy (gold standard), liver function and novel biomarker measurement (including CK18-M30, CK18-M65, GP73, IL-10, and IL-2R). Significant liver pathology was defined as inflammation grade (G) and/or fibrosis stage (S) ≥ 2 . Sixteen CK18-M65-centered biomarker combinations were evaluated using eight machine learning algorithms with multicenter stratified validation.

Results:
Significant liver pathology was observed in 45.2% of HBeAg+ infection cases. CK18-M65, CK18-M30, and GP73 strongly correlated with pathology severity (G stage: 0.757, 0.688, and 0.453, respectively; $p < 0.0001$), independent of ALT levels. CK18-M65 demonstrated

optimal diagnostic performance (AUC=0.934, 95% CI: 0.896-0.973) with 85.5% sensitivity and 88% specificity, especially in those with low-normal ALT. High CK18-M65 levels conferred >40 -fold increased risk of severe liver injury (adjusted OR=40.64). The optimal biomarker combination (CK18-M65+CK18-M30+GP73) achieved AUROC of 0.942, significantly outperforming LSM (0.824), APRI (0.783), and FIB-4 (0.745; all $p < 0.01$), with enhanced clinical utility. HBeAg- infection cases showed 46% significant pathology but weaker diagnostic performance.

Conclusions:

CK18-M65-centered biomarker models provide promising non-invasive tools for detecting occult liver pathology and risk stratification of ALT-normal HBeAg+ CHB infection, potentially reducing unnecessary biopsies while identifying candidates for early intervention.

Keyword(s) : chronic hepatitis B (CHB); CK18-M65; non-invasive diagnosis; ALT-normal; liver pathology

1045 - The Denizli Model A Leading Strategy in the War on Hepatitis C

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Background

In Türkiye, an estimated 750,000 people are infected with Hepatitis C (HCV), but diagnosis and treatment rates remain low. This is a significant issue, especially among high-risk groups like people who inject drugs (PWID) and the prison population.

Material and **Methods**
A comprehensive HCV elimination project was initiated in Denizli in 2018 to address this. The project included a retrospective study at Pamukkale University and a screening study in a Denizli prison.

Results
The retrospective study at AMATEM analyzed data from 265 patients with opioid use disorder. The results showed a remarkably high Anti-HCV positivity rate of 50.2% in this group, which is approximately 50 times higher than the general population. Longer substance use duration was found to be associated with higher HCV positivity. However, treatment initiation and follow-up rates were low. The prison screening study, conducted between 2021 and 2022, screened 187 inmates. The Anti-HCV

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prevalence among the prison population was 8.56%, which is eight times higher than the Türkiye average. The HCV SPOT rapid diagnostic test proved to be a reliable screening tool with 93.75% sensitivity and 100% specificity. The main risk factors identified were injecting drug use and a history of tattooing or piercing. Conclusions

The findings from these studies underscore the need to prioritize PWID and inmates in HCV elimination strategies. The micro-elimination approach, which focuses on these specific high-risk groups, is proposed as an effective way to achieve the WHO's 2030 goals. To reach this objective, key recommendations include enhancing systematic screening programs for high-risk populations, improving the linkage of diagnosed patients to treatment, implementing multidisciplinary approaches that coordinate care between addiction treatment and infectious diseases specialists, and leveraging rapid diagnostic tests to increase the accessibility of screening.

Keyword(s) : Hepatitis C, people who inject drugs, prison population

1046 - EVALUATING THE PROGNOSTIC VALUE OF THE MELD 3.0 SCORE IN PREDICTING MORTALITY IN CIRRHOSIS PATIENTS WITH ACUTE VARICEAL BLEEDING

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INTRODUCTION

Variceal hemorrhage is a severe complication in cirrhotic patients, occurring in up to 70% of cases of upper gastrointestinal bleeding, with a 6-week mortality rate of 15–20%. Accurate early mortality prediction is essential for optimizing treatment. The MELD 3.0 score, an improved version of the MELD score, enhances mortality prediction by incorporating gender, sodium, albumin, and a maximum creatinine threshold of 3.0 mg/dL. This study evaluates the effectiveness of MELD 3.0 in predicting 6-week mortality in cirrhotic patients with acute variceal bleeding, comparing it with MELD, Glasgow-Blatchford (GBS), and AIMS65 scores.

METHODS

A prospective study was conducted at Cho Ray Hospital from November 2023 to May 2024, enrolling cirrhotic patients with variceal bleeding. The primary outcome was 6-week mortality, and in-hospital mortality was a secondary endpoint. The predictive accuracy of MELD 3.0, MELD, GBS, and AIMS65 scores was assessed using AUCROC analysis.

RESULTS

Among 212 patients, in-hospital and 6-week mortality rates were 4.7% and 19.8%, respectively. MELD 3.0 outperformed other scores, with AUCROC values of 0.88 (in-hospital) and 0.81 (6-week mortality). A cut-off score of 20 corresponded to a >25% mortality risk, with 69.1% sensitivity and 83.5% specificity.

CONCLUSION

MELD 3.0 is a reliable tool for predicting mortality in cirrhotic patients with variceal bleeding. A cut-off score of 20 at admission can help identify high-risk patients for early intervention.

Keyword(s) : MELD 3.0 score; variceal bleeding; liver cirrhosis; 6-week mortality

1050 - Multicenter Study of Ultrasound Guided Microwave Ablation for Hepatic Cystic Echinococcosis

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Background / Hypothesis: Hepatic cystic echinococcosis (HCE) is a endemic disease associated with significant morbidity. Microwave ablation (MWA) has recently emerged as a promising minimally invasive treatment alternative.

Materials and Methods: his retrospective multicenter study included 73 patients with 99 HCE lesions treated with ultrasound-guided MWA between 2016 and 2024. Primary endpoints were local lesion progression (LLP) and recurrence-free survival (RFS). Secondary outcomes included technical success and complication rates. Risk factors for recurrence were analyzed using Firth-penalized Cox regression. Four hierarchical risk models were developed to predict RFS.

Results:

The cohort comprised 73 patients (56.2% female, mean age 51.4±14.0 years) with mean lesion size of 7.5±2.5

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cm. Complete ablation was achieved in all cases without major complications. Over a median follow-up of 34.0 months, the LLP rate was 9.1%. The 1-, 2-, and 3-year RFS rates were 92.3%, 78.6%, and 64.2%, respectively. Three independent predictors of recurrence were identified: age >55 years, cyst diameter ≥ 11 cm, and elevated bilirubin. Four risk models were developed (Full, Extended, Basic, Mini) with C-index values ranging from 0.788 to 0.928. All models significantly stratified 3-year RFS (100% for low-risk vs 51-59% for high-risk groups, $p < 0.001$).

Conclusions:

Ultrasound-guided MWA is a safe and effective minimally invasive treatment for HCE. The proposed risk models facilitate individualized treatment planning and improved clinical decision-making.

Keyword(s) : hepatic cystic echinococcosis, microwave ablation, minimally invasive treatment, recurrence risk

1051 - Physical performance is associated with severe liver fibrosis in patients with chronic liver disease

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Background / Hypothesis: Physical activity is a key lifestyle factor that influences the development of chronic liver disease (CLD). However, the impact of physical performance on the severity of liver fibrosis in CLD patients remains unclear. This study aimed to investigate the association between physical performance and liver fibrosis by analyzing the relationship between noninvasive fibrosis markers and various assessments of physical performance in individuals with CLD.

Materials and Methods: This retrospective study included 200 patients with various chronic liver conditions who underwent physical performance evaluations at Dalin and Taipei Tzu Chi Hospital between July 2020 and June 2023. The evaluations included handgrip strength, the Short Physical Performance Battery, the Five Times Sit-to-Stand Test, the 4-meter usual walking speed test, and the 6-minute walking test. Liver fibrosis was assessed using the Fibrosis-4 Index (FIB-4), with scores ≥ 2.67 indicating advanced fibrosis and < 1.3 indicating its absence. Clinical data, FIB-4 scores and physical performance measures were analyzed for associations.

Results:

The cohort included 100 patients with chronic hepatitis B, 16 with hepatitis C, 56 with liver cirrhosis, 20 with alcoholic liver disease, and 123 with steatotic liver disease. All participants were nutritionally stable. Among them, 23 were identified with advanced liver fibrosis. Multivariate analysis showed that older age and lower handgrip strength were significantly associated with FIB-4 scores ≥ 2.67 . On the contrary, steatotic liver disease was significantly associated with FIB-4 scores < 1.3 .

Conclusions:

Reduced handgrip strength and the presence of steatotic liver disease may serve as potential indicators for screening advanced liver fibrosis in patients with CLD.

Keyword(s) :

1056 - Development of a Predictive Formula for Hepatic Steatosis in Apparently Healthy Individuals A Biopsy Proven Study in Turkish Liver Transplant Donors

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Background / Hypothesis: The prevalence of metabolic-associated fatty liver disease (MAFLD) is rising globally, with an estimated prevalence of around 30%. In Türkiye, reported prevalences are approximately 50%, depending on the non-invasive diagnostic methods used. However, not all centers have access to advanced liver care. In this study, we aimed to develop a simple formula based on routine laboratory and demographic parameters to detect hepatic steatosis, using biopsy-proven data obtained from liver donors within the national liver donation program.

Materials and Methods: We analyzed prospectively

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collected data from 367 individuals who underwent liver biopsy as part of the liver donation program and were considered healthy. We divided dose patients 50/50 age- and sex-matched. Parameters associated with the presence of hepatic steatosis were first evaluated using univariate analyses, followed by binary logistic regression. Based on the results of the multivariate analysis, a formula for detecting hepatic steatosis was derived. The Youden index was applied to determine the optimal cut-off value for the detection of hepatic steatosis.

Results: The median age was 34 [19-61] and 59.7% were male. In the univariate analysis, significant differences were observed in body mass index (BMI), hemoglobin, alanine aminotransferase, bilirubin, creatinine, natrium, insulin, HOMA, T3, T4, plateletcrit (PCT), and uric acid. In binary logistic regression analysis, BMI, bilirubin, PCT, and uric acid remained statistically significant. Using the binary logistic regression analysis the following formula was created:

$$p = 1 / \{ 1 + \exp[- (-10.750 + 0.179 \cdot \text{BMI} + 12.346 \cdot \text{PCT} + 0.698 \cdot \text{Uric\_acid} + 1.791 \cdot \text{Bil})] \}$$

This model achieved an area under the curve (AUC) of 0.837 [Confidence interval: 0.775-0.898]. The optimal cut-off for predicting steatosis was $p > 0.59$, yielding a sensitivity of 83% and a specificity of 76%. In the validation cohort the AUC was 0.659 [0.578-0.740] with sensitivity of 59% and specificity of 41%.

Conclusions: In our study, we developed for the first time a formula based on simple laboratory markers, using liver biopsy as the reference standard, to predict the presence of hepatic steatosis in apparently healthy Turkish population. This formula should be validated in future cohorts. However, this formula may offer a simple approach for use in primary care as a screening tool.

Keyword(s) : MAFLD, steatosis, healthy, general population, prediction formula

1065 - Epigenetic regulation and functional mechanisms of the ZSCAN23 gene in liver cancer

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Background / Hypothesis: Liver cancer poses a major global health threat. Epigenetic alterations, alongside genetic mutations, play critical roles in its pathogenesis. The ZSCAN23 gene, encoding a zinc finger protein, is often silenced by promoter hypermethylation in cancers like breast and pancreatic cancer, but its role in liver cancer remains

unclear.

Materials and Methods: ZSCAN23 expression and methylation in liver cancer and normal tissues were analyzed using TCGA. Six liver cancer cell lines were treated with the demethylating agent 5-aza-2'-deoxycytidine (5-aza), and ZSCAN23 expression was detected by RT-PCR. Methylation-specific PCR (MSP) was performed on DNA from cell lines, 403 liver tumor tissues, and 5 normal liver tissues. Clinical data were correlated with methylation status. ZSCAN23-overexpressing cell models were constructed. Co-immunoprecipitation (Co-IP) and mass spectrometry (MS) identified ZSCAN23-interacting proteins, validated by Western blot. Drug sensitivity and colony formation assays were conducted. Pathway protein levels were measured to assess ZSCAN23 methylation as a synthetic lethal biomarker.

Results:

TCGA analysis showed promoter hypermethylation of ZSCAN23 correlated with low expression and poor patient survival ($P < 0.001$). ZSCAN23 was silenced in the SNU-449, HepG2, Huh7, HCCLM3, SNU-182, and Bel-7405 cell lines, and promoter methylation was detected in all. 5-aza treatment restored ZSCAN23 expression in these cells, confirming methylation-dependent regulation. In primary tissues, ZSCAN23 methylation frequency was 54.5% (224/403) and significantly associated with advanced tumor stage ($P < 0.0001$), smoking, and alcohol consumption ($P < 0.05$). ZSCAN23 was found to interact with Ku80, a key DNA damage repair protein. Methylation-mediated ZSCAN23 silencing suppressed non-homologous end joining (NHEJ) and promoted ATR/CHK1 pathway activation. Consequently, ZSCAN23-silenced cells showed increased sensitivity to ATR inhibitors combined with cisplatin. Western blot confirmed significantly increased DNA damage under this combination treatment.

Conclusion:

In liver cancer, the ZSCAN23 gene frequently undergoes methylation, and its expression is regulated by methylation in the promoter region. The methylation status of ZSCAN23 is closely associated with tumor stage, smoking history, and alcohol consumption in liver cancer patients. ZSCAN23 is involved in DNA damage repair, activating the non-homologous end joining pathway and inhibiting the ATR/CHK1 pathway. Methylation of ZSCAN23 serves as a potential marker for liver carcinogenesis and is also a potential marker for ATR inhibitor "synthetic lethality" therapy.

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Keyword(s) : Liver cancer; Treatment; ZSCAN23; DNA methylation; Synthetic lethality

1067 - Clinical Profile and Short Term Outcomes of Patients with Acute on Chronic Liver Failure A Prospective Observational Study from a Tertiary Center in Southern India
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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) is associated with high short-term mortality, but data from South Asia remain limited, particularly regarding precipitating events and prognostic scores.

Materials and Methods:

This prospective observational study included 30 consecutive adult patients fulfilling APASL ACLF criteria, admitted between Jan–June 2025. Baseline demographic, clinical, and laboratory data were collected. Prognostic scores (MELD, CLIF-C ACLF, AARC) were calculated. Patients were followed for 28-day survival

Results:

The mean age was 47 ± 9 years; 24 (80%) were male. The underlying chronic liver disease was alcohol-related in 18 (60%), HBV in 6 (20%), and cryptogenic/NAFLD in 6 (20%). The commonest acute precipitating events were:

• Alcohol re-use	–	12	(40%)
• Sepsis	–	10	(33%)
• GI bleed	–	5	(17%)
• Drug-induced	–	3	(10%)

At baseline, median MELD was 26 (IQR 22–32), CLIF-C ACLF score 46 (IQR 40–54), and AARC: grade I – 6 (20%), grade II – 14 (47%), grade III – 10 (33%).

28-day mortality was 14/30 (47%). Independent predictors of mortality included bilirubin >15 mg/dL (p=0.01), INR >2.0 (p=0.02), grade III/IV encephalopathy (p=0.01), and sepsis (p=0.03). Prognostic discrimination was highest with CLIF-C ACLF (AUROC 0.83), followed by MELD (0.76) and AARC (0.71).

Conclusions: In this prospective cohort of ACLF patients,

alcohol and sepsis were the major precipitants. Short-term mortality was nearly 50%. CLIF-C ACLF score outperformed MELD and AARC for predicting 28-day outcome, supporting its role in early risk stratification and transplant prioritization

Keyword(s) : ACLF , APASL CRITERIA,SHORT-TERM MORTALITY , LIVER TRANSPLANTATION

1069 - Gut Microbiota Derived Butyrate Alleviates the Progression of Metabolic Dysfunction Associated Steatohepatitis by Regulating Intrahepatic CD4⁺T Cell Immune Response
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Background:
Metabolic dysfunction-associated steatohepatitis (MASH) is a critical stage in liver disease progression, where inflammation and fibrosis are core drivers of poor prognosis. Gut microbiota dysbiosis and abnormalities in its metabolite short-chain fatty acids (SCFAs) may affect disease progression by regulating T-cell immunity. This study focuses on the gut microbiota-SCFAs-T-cell immune axis, investigates its role and mechanisms in MASH, and provides a basis for disease prevention and treatment.

Methods:
Mendelian randomization (MR) was used to analyze the genetic association between gut microbiota and MASH, with inverse variance weighting as the main method for causal association analysis. In animal experiments, a MASH mouse model was established using a choline-deficient L-amino acid-defined high-fat diet (CDAHFD), and butyrate was supplemented via gavage combined with free drinking water. Fecal microbiota, SCFAs, serum liver function, blood lipids, inflammatory factors, liver pathological staining, and intrahepatic T-cell immune response (detected by flow cytometry [FACS]) were measured.

Results:
MR analysis showed genetically predicted higher abundance of butyrate-producing bacteria correlated with lower MASH risk, with no significant

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heterogeneity/horizontal pleiotropy and stable leave-one-out results.

Animal experiments confirmed MASH mice had reduced gut microbiota alpha diversity, lower GMHI, higher dysbiosis index, distinct beta diversity (vs control, PCoA), decreased butyrate-producing bacteria/fecal SCFAs (butyrate dropped most), plus higher visceral-to-body ratio, serum ALT/AST, intrahepatic TC/TG, and aggravated hepatic inflammation/fibrosis. Exogenous butyrate raised fecal butyrate, reduced visceral-to-body ratio, NAS score, fibrosis/steatosis, serum ALT/AST, intrahepatic TC/TG; downregulated serum IL-6/TNF- α , intrahepatic TNF- α / α -SMA/Collagen1/TIMP-1 mRNA, upregulated MMP mRNA; restored microbiota alpha diversity, improved GMHI/dysbiosis, normalized composition, and increased butyrate-producing bacteria. FACS showed that intrahepatic CD4⁺T cell proportion in MASH mice was significantly lower than in controls, restored after butyrate intervention. The MASH group had increased intrahepatic Th2 cells and decreased Treg cells; post-intervention, Th2 trended down while Treg trended up. Additionally, intrahepatic CD4⁺Perforin⁺ and CD4⁺TNF- α ⁺T cell proportions were elevated in the MASH group, which dropped significantly after butyrate treatment. No significant difference in intrahepatic T cell inhibitory phenotypes was noted among the three groups.

Conclusion:

Genetically, increased abundance of butyrate-producing bacteria is causally associated with lower MASH risk. In MASH mice, reduced butyrate-producing bacteria and butyrate correlate with hepatic injury; exogenous butyrate improves hepatic pathology and restores gut microbiota. Its protection may involve regulating intrahepatic CD4⁺T cell immune response, suggesting CD4⁺T cells mediate butyrate's role in delaying MASH progression.

Keyword(s) : Metabolic Dysfunction-Associated Steatohepatitis; Gut Microbiota; Butyrate; T Cells; Hepatic Fibrosis; Mendelian Randomization

1071 - Prevalence of significant fibrosis and metabolic correlates in patients with Metabolic dysfunction Associated Fatty Liver Disease A cross sectional study from a tertiary centre
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Background

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Hypothesis:

MAFLD is the most common chronic liver disease with an estimated prevalence of 25% worldwide. Patients with fibrosis $\geq$ F2 identified by transient elastography (TE) are considered the target population for pharmacological treatment and are most likely to benefit from antifibrotic drugs. However, FibroScan devices are expensive and not always available in resource-poor settings. Identification of metabolic correlates of advanced liver fibrosis helps in risk stratification and prioritisation of patients for further evaluation.

Materials

and

Methods:

This cross-sectional cohort study included consecutive adult patients visiting the gastroenterology department with fatty liver disease diagnosed by ultrasound scan / CAP measurement from January to June 2025(n=50). Clinical evaluation and routine lab works were performed and quantitative values of steatosis and fibrosis were measured using FibroScan. Patients with significant fibrosis defined as LSM $\geq$ 7.0 kPa ($\geq$ F2) were identified. Cross-sectional analysis assessed the prevalence and association of significant fibrosis with BMI, diabetes, hypertension and ALT.

Results:

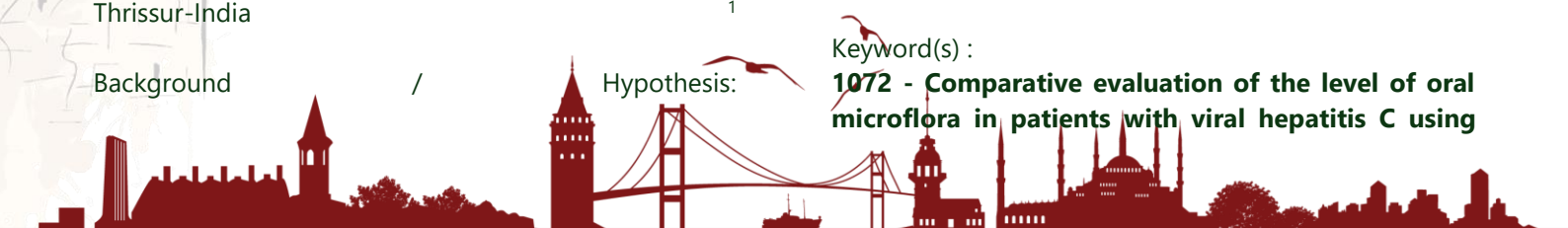
Mean age was 45 ± 10 years, of which 30 (60%) were males and 20 (40%) were females. Mean BMI was 31 ± 4 kg/m². Metabolic features: obesity 28 (56%), diabetes 20 (40%), hypertension 18 (36%), dyslipidemia 24 (48%) and low HDL 26 (52%). Steatosis (CAP): S0 5 (10%), S1 5 (10%), S2 15 (30%), S3 25 (50%). Fibrosis by TE: F0-F1 20 (40%), F2 15 (30%), F3 8 (16%), F4 7 (14%). Overall prevalence of significant fibrosis ($\geq$ F2) was 30(60%). On statistical analysis, significant fibrosis was associated with diabetes (p=0.01), BMI $\geq$ 30 kg/m² (p=0.02) and ALT >40 U/L (p=0.03).

Conclusions:

In this cross-sectional MAFLD cohort, 60% of study population had significant fibrosis. As diabetes, obesity and raised ALT were found to be associated with advanced disease, these parameters help in risk stratification in clinics. Hence these patients can be prioritised for fibrosis assessment using TE and early administration of antifibrotic therapy.

Keyword(s) :

1072 - Comparative evaluation of the level of oral microflora in patients with viral hepatitis C using



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heated tobacco product and cigarette smoking in Armenia a pilot study

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Background: Since the last decades of the 20th century up to the present time, there has been an increase in the incidence of viral hepatitis. The high frequency and diversity of the clinical picture of oral lesions in HCV are indicated. HCV contributes to the development of lesions of the oral mucosa (OM) and periodontium, but OM and periodontal diseases often precede HCV relapse. Microorganisms of periodontal pockets can cause a state of sensitization of the body and disturbances in the immunological status. Cigarette smoking (CS) also causes disturbances in the balance of oral microorganisms. In recent years a new type of products has emerged, such as Heated Tobacco Products (HTP), which are positioned as a harm reduction measure. The aim of the study was a comparative assessment of the level of oral microflora in patients with HCV using HTP and CS.

Materials and Methods: 50 patients with HCV who were treated in an infectious diseases hospital in 2025 were examined. The average age of the patients was 50.05 ± 13.29 years. Among them, there were 33 men (66%) and 17 women (34%). Bacteriological testing was performed on 50 patients in the HTP and CS groups.

Results: We have studied the features of the formation of pathological biotopes in the oral cavity in patients with HCV using HTP and CS. The comparative analysis of the frequency and intensity of inoculation of individual microorganisms between the groups is presented in Table 1. As can be seen from the table, most patients in the HTP group showed scanty growth of microorganisms. Moderate and abundant growth of pathogenic microorganisms *S. aureus* and *Str. pyogenes* are practically identical. *Veillonella* had a moderate growth in the HTP vs CS ($p < 0.001$; OR=0.01; 95% CI, 0.0–0.16). When analyzing the data of *Actinomyces* and *Candida* spp, moderate growth was revealed in HCV CS group, with a high degree of reliability compared with the HTP group ($p < 0.001$; *Actinomyces* OR=9.14; 95% CI, 3.05 to 27.43; *Candida* spp OR=8.13; 95% CI, 2.9 to 23.02).

Conclusion: Understanding patterns of interaction between different types of microorganisms will help identify new targets for the treatment of complex

polymicrobial

infections.

Keyword(s) : oral mucosae, microbiology, HCV, CS, HTP

1073 - Cognitive Impairment in Metabolic Dysfunction Associated Fatty Liver Disease (MAFLD) A Systematic Review and Meta analysis
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Background / **Hypothesis:**
This systematic review and meta-analysis aimed to comprehensively evaluate cognitive performance in individuals with metabolic dysfunction-associated fatty Liver Disease (MAFLD) compared to healthy controls. It further sought to investigate how differences in cognitive assessment tools, including the Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA), and key metabolic risk factors may influence the strength and direction of the association between MAFLD and cognitive impairment.

Materials and **Methods:**
We conducted a systematic search of PubMed, Scopus, Cochrane Library, and Google Scholar through May 2025 to identify studies comparing cognitive performance between individuals with MAFLD and healthy controls, assessed using either MMSE or MoCA. We performed a random-effects meta-analysis to calculate pooled standardized mean differences (SMDs) and assessed heterogeneity using I^2 statistics. Subgroup and meta-regression analyses were conducted to identify potential moderators influencing cognitive outcomes.

Results:
We included 14 studies involving 6,011 participants in the meta-analysis. The pooled SMD in cognitive scores between individuals with MAFLD and controls was -1.15 (95%CI: -2.48 to 0.19), indicating a trend toward poorer cognitive performance, though not statistically significant ($p=0.092$; $I^2=93\%$) (Figure 1). Subgroup analysis (Table 1) revealed significant effects in cross-sectional studies (SMD = -0.52 ; 95%CI: -0.75 to -0.29 ; $p < 0.001$) and cohort studies (SMD = -0.19 ; 95%CI: -0.25 to -0.13 ; $p < 0.001$). Studies using MoCA showed a significant association (SMD = -0.60 ; 95%CI: -0.89 to -0.31 ; $p < 0.001$), while MMSE did not (SMD = -1.76 ; 95%CI: -4.68 to 1.15 ; $p=0.235$). Quantitative steatosis assessment tools yielded smaller but significant effects,

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whereas ultrasound showed a larger but non-significant effect. Meta-regression (Table 2) identified higher BMI ($\beta = -0.071$; $R^2 = 81\%$), a greater prevalence of diabetes ($\beta = -0.130$; $R^2 = 92\%$), and increased liver enzymes—ALT, AST, GGT—as significant moderators. No significant associations were observed for age, hypertension, or lipid parameters. Across three studies, increasing steatosis severity was associated with progressively lower cognitive scores, suggesting a dose-response relationship. Egger's test detected publication bias; Trim and Fill analysis adjusted the effect to -0.28 ($p = 0.739$), indicating potential overestimation. Leave-one-out analysis identified one influential outlier study, which, when excluded, reduced heterogeneity and revealed a significant pooled SMD (-0.43 ; $p < 0.001$).

Conclusions:

This meta-analysis demonstrates a trend toward poorer cognitive performance in individuals with MAFLD, particularly in those with higher BMI, type 2 diabetes, and elevated liver enzymes. The findings highlight the greater sensitivity of MoCA over MMSE and underscore the importance of routine cognitive screening in at-risk populations. Well-designed longitudinal studies using standardized diagnostic and cognitive assessment methods are needed to clarify causal relationships.

Keyword(s) :

1075 - Effectiveness of Semaglutide in Non Alcoholic Steatohepatitis resolution A meta analysis and systematic review

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Background / Hypothesis: NAFLD can lead to complications such as fibrosis, cirrhosis, and an elevated risk of hepatocellular carcinoma, cardiovascular disease, chronic kidney disease, and mortality. The use of glucagon-like peptide-1 (GLP-1) analogues is currently being studied as a possible treatment option in NAFLD. GLP-1 receptor agonists have pleiotropic effects on several aspects of metabolic syndrome relevant to treatment of NASH, including body weight, inflammation, and glucose and lipid metabolism.

Materials and Methods: A comprehensive literature search showed 4 randomized controlled trials ($N = 604$) comparing Semaglutide (0.4mg/day or 2.4mg/week) with placebo.

Two reviewers independently selected studies, assessed quality, and extracted and pooled outcomes including liver histology, aspartate transaminase (AST), alanine transaminase (ALT), Triglyceride, Hba1c levels and body weight. All selected studies were found to be of low risk of bias based on Cochrane risk of bias assessment tool for randomized trials

Results:

4 RCTs (604 patients) were analyzed, improvement of NASH as compared to those receiving placebo was noted (WMD = 0.23 95%CI: 0.12; 0.46) with a p-value of 0.0001; I² = 66%, fibrosis stage (WMD = 2.21 95%CI: 1.04; 4.52) with a p-value of 0.03; I² = 0%, serum ALT activity (WMD = -0.19; 95% CI: -1.73; 1.34) with a p-value of 0.36; I² = 0% (low heterogeneity) and AST activity (WMD = -0.23 95% CI: -1.90; 1.43) with a p-value of 0.33; I² = 0%, body weight (WMD = -1.05 95%CI: -1.81; -0.29) with a p-value of 0.02; I² = 83%, Hba1c (WMD = 0.39 95% CI: -0.84; 0.06) with a p-value of 0.07; I² = 59%, Triglycerides (WMD = -0.28 95%CI: -0.17; 0.17) with a p-value of 0.12;; I² = 30%.

Conclusions:

Our meta-analysis shows that while semaglutide improves liver enzyme levels and cardiovascular health in patients with NAFLD, it has a limited impact on liver fibrosis and fat content. Semaglutide is effective for weight loss and blood sugar control in patients with NAFLD and metabolic syndrome. However, larger studies are needed to fully understand its long-term effects on liver disease.

Keyword(s) :

1076 - Optimizing Risk Stratification of Esophageal Varices for Endoscopic Surveillance Diagnostic Value of APRI FIB 4 King and Bonacini Scores in Hepatitis Related Liver Disease

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Background / Hypothesis: Chronic hepatitis B and C can lead to portal hypertension and esophageal varices, increasing the risk of upper gastrointestinal bleeding. In low-resource settings, non-invasive fibrosis scores offer a practical alternative to endoscopic screening.

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Materials and Methods: A retrospective cohort study was conducted on patients with upper gastrointestinal bleeding and chronic hepatitis B or C admitted to Moewardi Hospital, Indonesia (January–December 2024). AST, ALT, INR, and platelet levels were used to calculate APRI, FIB-4, King's, and Bonacini scores. All patients underwent EGD for EV grading (I–III). Ordinal logistic regression and ROC analysis assessed the association and diagnostic performance of each score.

Results: Among 44 UGIB patients meeting the criteria, the mean age was 56.91 ± 10.48 years, and 79.5% were male. Hepatitis B was present in 86.4%, the rest had hepatitis C. EV severity was distributed as 43.2% grade III, 40.9% grade II, and 15.9% grade I. Mean APRI, FIB-4, King, and Bonacini scores were 1.91 ± 2.63 , 5.81 ± 4.13 , 56.46 ± 81.12 , and 6.59 ± 1.66 , respectively. Ordinal regression identified FIB-4 and Bonacini as significant predictors of EV severity. FIB-4 was associated with a 29% increase in odds per unit ($p=0.01$, $OR=1.29$, $R^2=0.208$), and Bonacini with a 137% increase ($p<0.001$, $OR=2.37$, $R^2=0.379$). APRI and King scores were not significant. ROC analysis supported this, with Bonacini showing the best discrimination ($AUC=0.888$, $p=0.001$), followed by King ($AUC=0.761$, $p=0.03$), FIB-4 ($AUC=0.753$, $p=0.036$), while APRI had non-significant AUCs.

Conclusions: Bonacini index emerged as the most accurate and consistent non-invasive marker for predicting EV severity, suggesting its utility for risk stratification in hepatitis-related upper GI bleeding.

Keyword(s) : APRI, FIB-4, King Score, Bonacini Index, Esophageal Varices

1079 - Prognostic utility of the MELD score and AE model in predicting acute on chronic liver failure among patients with severe acute exacerbation of chronic hepatitis B.

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) remains one of the deadliest complications of chronic hepatitis B (CHB), particularly in Asia where HBV prevalence is high. Early identification of patients at risk is critical, yet existing prognostic models yield inconsistent results. Currently, predictive models for acute-on-chronic liver failure in hepatitis B virus infection remain limited in Vietnam.

Materials and Methods: We conducted a cohort study of 120 patients with severe acute exacerbation of CHB at University Medical Center, Ho Chi Minh City (2019–2024). Prognostic performance of MELD, MELD-Na, and AE models was compared using AUROC.

Results: Within 28 days, 35% of patients progressed to ACLF. MELD-Na (AUROC 0.82) and AE model (AUROC 0.84) demonstrated significantly higher predictive accuracy than MELD (AUROC 0.74). In multivariate logistic regression analysis, the factors independently associated with acute-on-chronic liver failure exacerbation included: ascites ($OR=24.65$, 95% CI = 3.77–161.29, $p<0.001$), hepatic encephalopathy ($OR=293.78$, 95% CI = 19.83–4353.16, $p<0.001$), palmar erythema ($OR=42.11$, 95% CI = 1.60–1105.74, $p=0.025$), and INR ($OR=677.89$, 95% CI = 6.91–66,468.85, $p=0.005$).

Conclusions: MELD-Na and AE models substantially outperform MELD in predicting ACLF, offering clinicians reliable tools for early risk stratification and timely intervention. This may reshape management strategies for HBV-related ACLF in high-burden regions such as Vietnam.

Keyword(s) : ACLF; CHB, MELD- Na, MELD, AE

1080 - Comparative Features of Viral and Metabolically Associated Liver Cirrhosis

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Background / Hypothesis: Metabolically associated steatotic liver disease (MASLD) is becoming one of the most common liver disorders worldwide. The prevalence of MASLD is increasing in parallel with the global rise in obesity and type 2 diabetes mellitus. The most advanced stage of this disease is metabolically associated liver cirrhosis (MALC), which is associated with high morbidity, disability, and mortality rates. **Objective:** To study the clinical features of MALC in comparison with viral liver cirrhosis (VLC).

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Materials and Methods:
A total of 37 patients (14 men and 23 women) with MALC and 83 patients (49 men and 34 women) with VLC (including 54 with HBV and 29 with HCV infection) were examined. Clinical decompensation was defined by the presence of ascites, variceal bleeding, or hepatic encephalopathy (HE).

Results:

Patients with MALC were significantly older (58.43 ± 1.91 years) than those with VLC (38.70 ± 1.34 years). Elderly individuals (≥ 65 years) accounted for 35.1% of the MALC group and only 3.6% of the VLC group. Compared with VLC patients, those with MALC had a higher prevalence of obesity (32.4% vs. 4.8%), diabetes mellitus (56.7% vs. 9.6%), hypercholesterolemia (27.0% vs. 9.6%), and arterial hypertension (62.2% vs. 20.5%). Elevated ALT activity was observed in 51.4% and 75.9% of patients with MALC and VLC, respectively. Clinically significant portal hypertension (CSPH) was diagnosed in 43.2% of MALC and 59% of VLC patients. The proportion of patients with decompensated cirrhosis was higher in the MALC group (62.2% vs. 49.4% in VLC). The most frequent manifestation of decompensation was ascites, while the incidence of variceal bleeding and hepatic encephalopathy was similar between groups. Decompensation occurred in 67% of patients with obesity and in 60% of those without. Among patients with diabetes, decompensation developed in 76%, compared with 44% in non-diabetic individuals.

Conclusions:

MALC develops predominantly in older individuals with metabolic risk factors and is often accompanied by decompensation. Decompensation of MALC may occur even in the absence of CSPH. A high degree of necroinflammatory activity in the liver and the presence of diabetes mellitus—but not obesity—contribute to the progression of cirrhosis decompensation.

Keyword(s) : metabolically associated liver cirrhosis, clinically significant portal hypertension, decompensation

1085 - Radiomics on Gadoteric Acid HBP MRI to Distinguish Subclinical Hepatic Parenchymal Disease from Normal Liver

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Background / Hypothesis:
Hepatobiliary-specific contrast agent gadoteric acid (Gd-EOB-DTPA) provides functional information reflecting hepatocellular uptake and biliary excretion. However, early parenchymal impairment may remain undetected on conventional biochemical tests or visual MRI assessment. We hypothesized that radiomics extracted from hepatobiliary-phase (HBP) MRI could identify subtle parenchymal alterations and distinguish subclinical hepatic disease from normal liver, enabling earlier noninvasive detection of hepatic dysfunction.

Materials and Methods:

This single-center retrospective study was approved by the Erzurum Atatürk University Ethics Committee (Approval No:B.30.2.ATA.0.01.00/692) and conducted in accordance with the Declaration of Helsinki. A total of 157 subjects who underwent Gd-EOB-DTPA-enhanced liver MRI were analyzed: 100 patients clinically diagnosed with subclinical chronic liver disease (CLD) based on laboratory scores and 57 normal controls without CLD. Exclusion criteria included large focal hepatic lesions and severe motion artifacts. Images were acquired on a 1.5T system using a 20-minute hepatobiliary phase protocol. All images were resampled to $2 \times 2 \times 2$ mm voxels and normalized using per-patient Z-scores. Automatic liver segmentation was performed using TotalSegmentator, manually corrected in 3D Slicer (Figure 1), and focal lesions with a 1.5 cm rim of surrounding tissue were excluded. PyRadiomics extracted 94 features across first-order statistics, GLCM, GLRLM, GLSZM, GLDM, and NGTDM matrices (Figure 2). The Boruta algorithm selected 10 most predictive features, primarily first-order energy-based and texture heterogeneity metrics. A logistic regression pipeline (standardization → PCA → classifier) was trained on 133 cases and tested on 24 independent cases. Model performance was evaluated by ROC-AUC, precision-recall (PR) analysis, accuracy, sensitivity, specificity, and F1-scores.

Results:

In the independent test cohort, the logistic regression model achieved ROC-AUC = 0.896 (Figure 3) and

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accuracy = 87.5%. Average precision (AP) on the PR curve was 0.93 (Figure 4). Class-based performance showed precision = 1.00, recall = 0.67, F1 = 0.80 for the normal group, and precision = 0.83, recall = 1.00, F1 = 0.91 for the subclinical group. The macro F1-score was 0.855, and weighted average F1 = 0.868. Confusion matrix analysis revealed correct classification of all 15 subclinical cases, with only 3 normal cases misclassified.

Conclusions:

Radiomics features derived from hepatobiliary-phase Gd-EOB-DTPA MRI distinguished subclinical hepatic parenchymal disease from normal liver with high accuracy and sensitivity. This machine-learning model demonstrates potential as a noninvasive imaging biomarker for detecting early hepatic dysfunction before clinical decompensation. Future multicenter validation will further define its prognostic and translational utility.

Keyword(s) : Radiomics; gadoxetic acid; hepatobiliary phase MRI; subclinical liver disease; chronic liver disease; logistic regression; Boruta; machine learning; ROC-AUC

1096 - Triplet Therapy with Envafolelimab Lenvatinib and TACE Enables Curative Conversion and Pathological Response in Unresectable HCC A Single Arm Phase II study

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Abstract

Background: Hepatocellular carcinoma (HCC) frequently presents at intermediate or advanced stages where curative resection is not feasible. Transcatheter arterial chemoembolization (TACE) is first-line for unresectable HCC but can induce angiogenesis via VEGF upregulation and often fails to cure, leading to recurrence. Combining systemic therapies with TACE may improve downstaging and conversion to surgery. Envafolelimab is a novel subcutaneous PD-L1 antibody offering convenient administration and potentially fewer infusion reactions. We conducted a single-arm, phase II trial of TACE plus Lenvatinib and envafolelimab in unresectable HCC, focusing on conversion to resectability. Methods: Fifteen patients with unresectable HCC (Barcelona Clinic Liver Cancer stage B or C) were enrolled from April 1–September 30, 2024. All received

conventional TACE (every 6 weeks as needed) combined with lenvatinib (12 mg or 8 mg daily by weight) and envafolelimab (300 mg subcutaneously every 3 weeks) until radiographic downstaging enabled surgery, or until progression, unacceptable toxicity, or death. The primary endpoint was conversion rate to curative resection. Secondary endpoints included objective response rate (ORR) and disease control rate (DCR) by mRECIST, R0 resection rate, recurrence-free survival (RFS), overall survival (OS), and safety. Results: The median age was 54 years, 86.7% were male, 80% had HBV infection, and 66.7% had multifocal tumors (median diameter 5.5 cm). ORR was 53.3% and DCR 86.7% per mRECIST. Nine patients (60%) achieved downstaging to resectable disease (conversion rate 60%), all of whom underwent R0 resections. Pathological examination showed viable tumor in most resected specimens, but 3 patients (33.3% of those resected) achieved pathological complete response. After a median follow-up of 16 months, the estimated median RFS was 15.0 months and 1-year RFS was 53%. Only one patient (6.7%) died during follow-up, yielding a 1-year OS of 100% and 18-month OS of 93.3%, median OS was not reached. Conclusion: TACE combined with envafolelimab and lenvatinib demonstrated a high conversion-to-surgery rate (60%) with favorable tumor responses and survival in unresectable HCC. All conversions led to R0 resections, and a subset achieved pathologic CR, highlighting the potential of this regimen to enable curative surgery. The safety profile was manageable and similar to known effects of TACE, VEGF inhibition, and PD-1/PD-L1 blockade. The subcutaneous delivery of envafolelimab was convenient and avoided infusion reactions. These findings support further investigation of envafolelimab plus lenvatinib and TACE as a conversion therapy strategy to improve resection rates and long-term outcomes in advanced HCC.

Keyword(s) : Envafolelimab; Lenvatinib; Transcatheter Arterial Chemoembolization; Hepatocellular Carcinoma; Conversion Therapy

1098 - Disease burden of chronic hepatitis D virus (HDV) infection among people living with chronic hepatitis B virus (HBV) infection in a low endemic city

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Background / **Hypothesis:** Despite European guidelines recommending anti-hepatitis D virus (anti-HDV) screening for all hepatitis B surface antigen (HBsAg)-positive cases, screening rates in the Asia-Pacific region remain low. We aimed to evaluate the prevalence of chronic HDV infection among individuals living with chronic hepatitis B virus (HBV) infection with an additional risk factor for HDV, namely human immunodeficiency virus (HIV) co-infection or HBV-related hepatocellular carcinoma (HCC).

Materials and **Methods:** People living with chronic HBV infection, with either HIV co-infection or HBV-HCC under the care of a tertiary, academic hospital, were invited to have HDV screening by anti-HDV antibody followed by reflex HDV RNA quantitative real-time PCR (Quest Diagnostics, New Jersey, USA, limit of detection 5 IU/mL) in anti-HDV-positive individuals. Subjects with positive anti-HDV also underwent a vibration-controlled transient elastography (VCTE) examination as the non-invasive assessment for liver fibrosis.

Results: A total of 125 individuals (102 HBV-HCC, 23 HBV-HIV, mean age 65 years, 70.4% men, all HBV DNA undetectable (< 10 IU/mL) with oral nucleos(t)ide analogues) were recruited. Three (2.4%, two HBV-HCC, one HBV-HIV) subjects had positive anti-HDV, and two (1.6%, both HBV-HCC) subjects had equivocal anti-HDV; four of these five subjects also had hepatitis C virus (HCV) co-infection. All of these five subjects had undetectable HDV RNA; their median (range) liver stiffness measurement was 7.5 (6.8 – 14.8) kPa.

Conclusions: Our data showed that the implementation of anti-HDV / HDV RNA reflex testing is feasible in a low-endemic city. Even in the tertiary setting, the disease burden of HDV among individuals living with HBV and other risk factors (HCC, HCV, HIV) was low.

Funding: This work was supported by the Investigator Sponsored Research of Gilead Sciences (Reference: IN-US-980-7495).

Keyword(s) : HBV, HCC HDV, HIV

1101 - Association of Serum ALT Levels with Histological Activity and Fibrosis in Chronic Hepatitis

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Background / Hypothesis: This study aimed to evaluate the relationship between serum alanine aminotransferase (ALT) levels and both the histological activity index (HAI) and fibrosis stages in patients with chronic hepatitis B (CHB).

Materials and Methods: A total of 268 patients who underwent liver biopsy with a diagnosis of CHB at Yozgat City Hospital between 2017 and 2025 were retrospectively analyzed. Demographic characteristics, biochemical parameters, HBeAg status, hepatitis B virus DNA (HBV DNA) levels, and histopathological findings of the patients were evaluated. The threshold value for ALT was accepted as 50 U/L, which is the cutoff used in our hospital laboratory.

Results: Among the patients, 27.6% (n=74) had ALT levels above 50 U/L. ALT levels were significantly higher in the group with an HAI score ≥ 6 compared to those with HAI < 6 [41 U/L (10–1434) vs. 22 U/L (8–260), $p < 0.001$]. Similarly, ALT levels were higher in patients with fibrosis stage ≥ 2 [32 U/L (7–1007) vs. 26 U/L (9–1434), $p = 0.016$]. However, there was no significant difference in ALT levels between patients with advanced fibrosis (stage 5–6) and those with mild-to-moderate fibrosis (stage 0–4) [25 U/L (11–80) vs. 27.5 U/L (8–1434), $p = 0.531$].

The higher ALT levels observed in patients with HAI ≥ 6 and fibrosis stage ≥ 2 support the clinical value of ALT as an indicator of hepatic injury. On the other hand, the absence of a significant difference in ALT levels between advanced and lower fibrosis stages suggests that ALT may not consistently reflect disease severity in advanced fibrosis. This finding may indicate that, as fibrosis progresses, hepatocyte loss and increased fibrotic tissue can limit serum ALT activity. Consistent with previous literature, ALT levels may remain within the normal range in patients with advanced fibrosis or cirrhosis, indicating that ALT alone may not accurately reflect histological damage. Although elevated ALT levels were observed in some patients with HBeAg positivity and high HBV DNA levels, no absolute correlation was found between ALT and viral load. This suggests that hepatocyte injury and viral replication may not always occur in parallel during the

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natural course of CHB.

Conclusions: In patients with chronic hepatitis B, serum ALT levels were found to be associated with necroinflammatory activity and fibrosis degree. However, in advanced fibrosis stages, ALT levels do not reflect histological severity. These findings indicate that ALT should not be used as the sole marker in clinical evaluation and should be complemented with biopsy or noninvasive fibrosis assessment methods.

Keyword(s) :

1102 - Natural History and Risk Factors of Metabolic Dysfunction–Associated Fatty Liver Disease in Chinese Cohorts A Scoping Review
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Background:

Metabolic dysfunction-associated fatty liver disease (MAFLD) has rapidly become the predominant chronic liver disorder in China. However, evidence on its natural history and determinants of progression remains fragmented. We conducted a scoping review to synthesize evidence from Chinese cohorts on disease trajectories from steatosis through fibrosis, cirrhosis, and hepatocellular carcinoma (HCC), and to identify key risk factors driving progression.

Materials and Methods: Following PRISMA-ScR guidelines, we searched PubMed, EMBASE, Cochrane Library, CNKI, Wanfang, and VIP databases from January 2012 to January 2024. Eligible studies included Chinese MAFLD cohorts reporting natural history outcomes or multivariate-adjusted risk factors for progression. We mapped outcomes: Metabolic dysfunction-associated steatohepatitis (MASH), fibrosis progression, cirrhosis, HCC, liver-related events, cardiovascular events, mortality, and tabulated risk factors evaluated across studies.

Results:

41 articles (36 natural history; 21 risk factor) met the inclusion criteria, involving 247,502 participants for

disease-course analyses and 66,143 for risk modelling. For MAFLD patients, in 5 years or less, fibrosis advanced in 27.5-38.4%, and cirrhosis developed in up to 1% (0.9%). Over longer follow-up (>5 years), 0-2.1% transitioned to cirrhosis and 0-2.7% to HCC (annual incidence 0.3-2%). Hepatic decompensation (ascites, encephalopathy, variceal bleeding) occurred in 0.1-1.1% by 11 years. Within 5 years, cardiovascular mortality (2.1-7.0%) and all-cause mortality (2.2-6.8%) outpaced liver-related mortality (0.3-0.6%). Key independent predictors of progression included older age, male sex, hypertension, and overweight/obesity, especially type 2 diabetes, each reported in ≥ 3 studies across endpoints. Additional risk factors variably linked to adverse outcomes were dyslipidemia, sarcopenia, elevated transaminases, viral hepatitis coinfection, and low albumin. Heterogeneity in study populations, endpoint definitions, and reporting of demographic covariates limited comparability.

Conclusions:

MAFLD in China trends similarly to the global pattern but may exhibit notably rapid progression in specific populations or stages. Multiple risk factors contribute to disease advancement. Further large-scale prospective cohort studies are warranted to elucidate the associations between risk factors and MAFLD, and to better inform early intervention strategies for this condition.

Keyword(s) : MAFLD, MASH, fibrosis, cirrhosis, mortality, type 2 diabetes, overweight, obesity, natural history, risk factor, Scoping Review

1103 - Unusual Partners in Anemia Zieve's Syndrome and Beta Thalassemia Trait in a Single Patient A case report and literature review
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Introduction: Zieve syndrome (ZS) — a triad of hemolytic anemia, jaundice, and hyperlipidemia due to chronic alcohol abuse — is often underdiagnosed. Beta-thalassemia trait also causes chronic hemolysis. This report presents what we believe is the first documented case of ZS with concurrent beta-thalassemia trait, establishes the diagnosis of concurrent hemolysis, and adds to the limited literature on the complexities of treating ZS.

Case Presentation: A 36-year-old Turkish man with known beta-thalassemia trait and 3-year history of

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heavy alcohol consumption presented with a 3-4-month history of jaundice, weakness, abdominal pain, nausea, vomiting, and significant weight loss. Clinical findings included severe non-immune hemolytic anemia (Hb 6 g/dL, low haptoglobin, high reticulocytes), marked jaundice (total bilirubin 28 mg/dL, direct predominant), hypertriglyceridemia (530 mg/dL), severe hepatic steatosis, splenomegaly, and signs of pancreatitis. Diagnosis, Interventions, and Outcomes: The main diagnoses were ZS, with beta-thalassemia trait likely exacerbating the anemia, severe alcoholic steatohepatitis, and pancreatitis. Interventions included blood transfusions, alcohol withdrawal therapy, supportive care, and prednisolone administered due to a high Maddrey score (62.5). Alcohol abstinence led to significant improvement, although relapse occurred after resumption of use. The side effects of the steroids disappeared after discontinuation of the medication. Conclusion: This case underscores the importance of considering ZS in patients with chronic alcoholism who present with the characteristic triad, even in the presence of pre-existing hematologic conditions like beta-thalassemia trait, which can exacerbate anemia. Total abstinence from alcohol remains the critical component of treatment to achieve clinical improvement.

Keyword(s) : Zieve syndrome, alcoholic hepatitis, hemolytic anemia, thalassemia, case report

1110 - Effectiveness and safety of Fibrate Therapy in the treatment of adults with Primary Sclerosing Cholangitis A Systematic Review and Meta Analysis

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Background / Hypothesis: Primary sclerosing cholangitis is characterized by the stricturing of the bile ducts, either intrahepatic or extrahepatic bile ducts, and co-existence of inflammatory bowel disease, of which it is usually found in around 70% of patients. The global prevalence of primary sclerosing cholangitis is estimated to be 16 per 100,000 individuals, and in the Philippines, the prevalence of primary sclerosing cholangitis was around 1%. The risk for complications in the presence of primary sclerosing cholangitis could lead to liver failure, development of cholangiocarcinoma, and possible colon malignancy. This study compared the fibrate therapy and placebo in terms of serum liver biomarkers and safety of patients with primary sclerosing cholangitis

Materials and Methods:

Meta-analysis was the research design used. Specifically, the main author obtained the research design, the methodology of these investigations, interventions used, results and outcomes of these respective studies, among many others. Two research studies of de Vries and colleagues and Hatami and colleagues were included in the meta-analytical assessment. Results:

The investigation yielded statistical difference with 95% CI between placebo and fibrate therapy in terms of serum biomarkers specifically alkaline phosphatase (399.24 ± 173.57 vs. 261.85 ± 191.92 respectively, $p .050 < .05$). On the other hand, there was no statistical difference between placebo and fibrate therapy in terms of aspartate aminotransferase (66.02 ± 43.95 vs. 59.29 ± 39.86 respectively, $p .390 > .05$), alanine aminotransferase (72.09 ± 62.32 vs. 65.14 ± 42.37 respectively, $p .630 > .05$), total bilirubin (11.07 ± 10.17 vs. 10.52 ± 10.64 respectively, $p .390 > .05$), and serum albumin (22.27 ± 1.80 vs. 21.89 ± 3.23 respectively, $p .460 > .05$).

Conclusions:

Fibrate therapy could potentially improve the outcomes of primary cholangitis of patients. In this regard, the initiation of fibrate therapy could be beneficial in improving the biochemical markers of the aforementioned disease, better prognosis, and may also alleviate pruritus in patients.

Keyword(s) : Fibrate Therapy, Liver Disease, Primary Sclerosing Cholangitis

1116 - Racial Disparities in Hepatocellular Carcinoma A TCGA Based Gene Expression Study of Caucasian and Asian Populations

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Background / Hypothesis: Hepatocellular carcinoma (HCC) continues to be one of the leading causes of cancer death globally and has notable racial differences in incidence. As a result of hepatitis B or C virus (HBV/HCV) infection, Asian populations have a higher incidence of HCC, while

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Caucasian populations are more likely to be affected by non-viral etiologies such as metabolic dysfunction-associated steatotic liver disease (MASLD) and alcohol-related liver injury. These differences may produce different tumor biology and treatment outcomes.

Materials and Methods: Transcriptomic and clinical data from Asian (n = 157) and Caucasian (n = 184) patients were obtained from The Cancer Genome Atlas Liver Hepatocellular Carcinoma (TCGA-LIHC) cohort. Gene expression analysis was carried out in PyDESeq2 with size factor normalization and dispersion estimation. Different expressed genes (DEG) were defined by $FDR < 0.05$ and $|\log_2FC| \geq 1.0$. Gene annotation was done using Sanbomics, data visualization was done using seaborn and functional enrichment was done with GSEA using GSEApv. Enrichment was focused on Gene Ontology (GO) and KEGG pathways. Prognostic significance was assessed with GEPIA and the human protein atlas.

Results:

In both cohorts, 387 genes were upregulated and 250 downregulated compared to non-tumour tissues. Altered expression of GPC3, PLVAP, FCN3, and OIT3 suggests their potential as universal HCC markers. A total of 16 genes were upregulated in Asians but downregulated in Caucasians, while 25 showed the opposite pattern. The upregulated genes in Asian population AKR1B10, UBE2C, S100P were linked to viral infection and immune modulation, while MDK, LCN2, NQO1, CYP2C19 up-regulated in Caucasian were linked to proliferation and metabolism and xenobiotic exposure. GO and KEGG analyses revealed distinct biological programs: Asians showed enhanced ubiquitin ligase activity and reduced inflammatory signalling consistent with immune evasion in HBV/HCV-related HCC, whereas Caucasians showed enrichment of cytokine signalling, complement activation, and xenobiotic metabolism linked to alcohol and environmental carcinogen exposure. Elevated AKR1B10 in Asians and MDK in Caucasians were both associated with poorer five-year survival.

Conclusions:

This study identifies population-specific molecular pathways underlying HCC in Asian and Caucasian groups. While shared genes such as GPC3 and PLVAP may serve as common therapeutic targets, distinct immune and metabolic signatures reflect differing etiologies. Integrating these molecular and demographic differences will be essential for advancing precision oncology and developing equitable,

population-tailored prevention and treatment strategies for HCC.

Keyword(s): Hepatocellular carcinoma, gene expression, populations, TCGA

1117 - ADH1B rs6850217 as a Novel Genetic Determinant of Hepatocellular Carcinoma Risk in Chronic Hepatitis B

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Background / Hypothesis:

While viral and host immune determinants of hepatocellular carcinoma (HCC) in chronic hepatitis B (CHB) are well established, the influence of alcohol-metabolizing gene polymorphisms remains unclear. We investigated whether variants in alcohol dehydrogenase 1B (ADH1B) and aldehyde dehydrogenase 2 (ALDH2) affect HCC risk and interact with alcohol consumption among hepatitis B virus (HBV) carriers.

Materials and Methods:

A total of 3,631 participants from the Risk Evaluation of Viral Load Elevation and Associated Liver Disease/Cancer-HBV (REVEAL-HBV) cohort were analyzed. Incident HCC was the primary endpoint. Associations between genotypes, alcohol intake, and HCC risk were assessed using multivariable Cox proportional hazards models and adjusted hazard ratios (HRs) were estimated. Gene-environment interactions were examined on an additive scale by calculating the relative excess risk due to interaction (RERI), attributable proportion (AP), and synergy index (SI).

Results:

During a median follow-up of 26.7 years, 416 participants developed HCC. The ADH1B rs6850217 variant was independently associated with an increased risk of HCC (adjusted HR = 1.60; 95% CI: 1.08–2.37; $p = 0.020$). The ALDH2 rs146752252 variant showed a crude association with HCC (HR = 2.06; 95% CI: 1.25–3.40; $p = 0.005$), but this association was attenuated after multivariable adjustment (adjusted HR = 1.38; 95% CI:

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0.81–2.36; $p = 0.242$). Alcohol consumption was also significantly associated with an increased risk of HCC (crude HR = 1.96; 95% CI: 1.54–2.49; $p < 0.001$). A significant synergistic interaction was observed between ADH1B rs6850217 and alcohol consumption (RERI = 1.635, AP = 0.408, SI = 2.19), whereas the ALDH2 rs146752252–alcohol interaction was comparatively weaker (RERI = 0.913, AP = 0.263, SI = 1.585).

Conclusions:

ADH1B rs6850217 is a novel, independent genetic determinant of HCC risk in chronic HBV carriers. Its synergistic interaction with alcohol highlights the importance of integrating genetic and lifestyle factors in HCC risk assessment and personalized prevention strategies.

Keyword(s) : Chronic hepatitis B; Hepatocellular carcinoma; Alcohol metabolism; ADH1B polymorphism; Gene–environment interaction

1118 - Targeting Premalignant Liver Lesions with Dietary Bioactive Compounds From Multi Targeted Mechanisms to Precision Prevention

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Background / **Hypothesis:** Hepatocellular Carcinoma (HCC) is a leading cause of cancer-related mortality globally, with over 800,000 new cases and 700,000 deaths annually (WHO data). It develops through preneoplastic stages including liver cirrhosis and dysplastic nodules, and its malignant transformation is driven by risk factors such as HBV/HCV infection, aflatoxins, alcohol intake, and MAFLD, via mechanisms like mutations, epigenetic changes, and pathway dysregulation. Advanced HCC has a poor prognosis, creating an urgent need for chemopreventive strategies. Dietary bioactive compounds, with low toxicity and multi-targeted properties, are hypothesized to address this need—they may regulate hepatocyte homeostasis, exert chemopreventive effects, and intercept HCC progression from preneoplastic lesions, though their clinical translation is constrained by bioavailability limitations and interindividual variability. **Materials** and **Methods:**

This review synthesized peer-reviewed studies retrieved from databases including PubMed, Web of Science, and Scopus. The search used keywords such as “liver cancer precancerous lesions” and “dietary active ingredients”. Included studies met three criteria: focused on dietary bioactive compounds, reported specific mechanisms of action and efficacy, and provided full-text access. **Results:**

Dietary bioactive compounds regulate hepatocyte homeostasis through multiple interconnected pathways, primarily by activating the Nrf2 pathway to enhance antioxidant defenses and suppressing NF- κ B-mediated inflammatory responses. These compounds further exert their chemopreventive effects by inducing apoptosis via caspase pathway activation and modulating key oncogenic signaling networks such as Wnt/ β -catenin and PI3K/AKT. Synergistic combinatorial strategies significantly enhance their efficacy; for instance, curcumin-resveratrol co-treatment inhibits Hepa1-6 cell proliferation and induces apoptosis in a dose- and time-dependent manner, mechanistically involving mitochondrial pathway activation, downregulation of XIAP/survivin, and ROS accumulation. Similarly, the combination of Grifola frondosa polysaccharide (DFP) and vitamin C triggers G2/M phase arrest and 65% apoptosis in SMMC-7721 cells through mitochondrial membrane destabilization, excessive ROS generation (3.2-fold increase), and inhibition of the PI3K/AKT/NF- κ B axis, demonstrating multifaceted anticancer activity.

Conclusions:

Dietary bioactive compounds show significant promise in intercepting the progression from preneoplastic lesions to HCC, with multi-targeted chemopreventive effects. Synergistic combinatorial strategies and advanced delivery systems (e.g., nanodelivery) address their bioavailability limitations, while genomics/metabolomics-guided interventions enable precision prevention. Current research gaps include limited large-scale clinical trials and unclear compound–host interactions. Future research should focus on exploring crosstalk with the tumor microenvironment, conducting phase II/III clinical trials, and promoting the clinical translation of delivery technologies and personalized strategies to reduce the global burden of HCC.

Keyword(s) : precancerous lesions of liver cancer, dietary active ingredients, molecular mechanisms, bioavailability, targeted delivery

1121 - The first application and feasibility assessment of 5G enabled remote robot assisted hepatobiliary and pancreatic surgery in patients

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with malignant tumors

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Background / Hypothesis: Remote surgery is gradually demonstrating its potential due to the application of the new generation of network and satellite communication technologies. Previously reported remote robot-assisted laparoscopic surgery has confirmed the technical feasibility of remote surgery at a physical distance of 4,670.2 km (5,031.2 km network communication distance), with a median network delay of 73 ms during the operation, no interruption or packet loss, and no abnormalities in the patient's 30-day follow-up. However, the feasibility assessment of remote robot-assisted complex hepatobiliary surgery for resectable malignant tumors is still lacking, and relevant research is urgently needed to fill this gap.

Materials and Methods: This study was the first to apply remote robotic surgery to four patients with liver, gallbladder and pancreatic tumors. The surgery was conducted using the MP2000 robotic platform independently developed in China (Shenzhen Edge Medical Company). The main surgeon operated from a location 900 kilometers away from the surgical site (1035 kilometers in terms of network communication distance), with 5G network as the primary communication method and dedicated fiber optic network as a backup. Network latency and fluctuations were monitored throughout the procedure, and experienced local surgeons were on standby at the surgical site to handle any emergencies.

Results: All the remote surgeries in this study were successfully completed. The average communication delay during the operation was only 21ms, and the end-to-end total delay was less than 100ms. There was no network interruption or packet loss throughout the process. The average operation time in the surgical cavity was 240 minutes, and the average intraoperative blood loss was 100mL. All patients achieved R0 resection. During the postoperative hospital follow-up

period, no complications such as pancreatic fistula, bile leakage, delayed gastric emptying, or postoperative hemorrhage occurred in the patients. Their physiological conditions remained stable, and intestinal function recovered within 24 hours after the operation, achieving rapid recovery. As of the time of this report, the short-term recovery effect of the patients was good, and no obvious abnormalities were observed.

Conclusions: This study confirms for the first time the technical feasibility of remote robotic surgery in treating resectable malignant tumors. Short-term follow-up shows favorable outcomes and safety, filling a gap in research on complex remote robotic procedures for cancer.

Keyword(s): 5G, remote robot-assisted, hepatobiliary and pancreatic surgery

1123 - From Cirrhosis to Cancer End to End Deep Learning Surveillance on Non Contrast CT

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Background / Hypothesis: Patients with liver cirrhosis are at high risk of developing hepatocellular carcinoma (HCC). However, the sensitivity of current surveillance strategies for early detection remains limited, often leading to delayed diagnosis and poor outcomes. To develop and validate an end-to-end deep learning model based on noncontrast-enhanced abdominal CT scans to predict the occurrence of HCC 3–12 months in advance among patients with liver cirrhosis.

Materials and Methods: This prospective study included 703 patients with liver cirrhosis from a single tertiary center between June 2018 and January 2020 (median age, 48.5 years; 86.2% male). Patients were divided into a training cohort (n = 568) and an external validation cohort (n = 135). For patients who later developed HCC, noncontrast CT scans obtained 3–12 months before diagnosis were analyzed; for those without HCC, CT images from the final follow-up were used. An end-to-end deep learning model (LCPC) based on a fine-tuned ResNet-50 architecture was trained with five-fold cross-validation and evaluated in an independent external validation cohort.

Results: The LCPC model achieved an area under the receiver operating characteristic curve (AUC) of 0.818 (95% CI, 0.795–0.892) in five-fold cross-validation, 0.829 (95% CI, 0.735–0.914) in the internal validation cohort, and 0.758 (95% CI, 0.646–0.861) in the external

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validation cohort. The model demonstrated good generalizability and discrimination for early HCC prediction using only noncontrast-enhanced CT images. Conclusions: The LCPC model enables prospective and individualized risk assessment of HCC in patients with liver cirrhosis using noncontrast abdominal CT scans. This approach may facilitate earlier clinical decision-making and expedite preparation for liver transplantation or donor registration in high-risk patients.

Keyword(s) : Liver Cirrhosis; Hepatocellular Carcinoma; Deep Learning; AI

1125 - Development of an Explainable Hybrid Artificial Intelligence System for Early Detection of Chronic Liver Diseases

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Background / Hypothesis: Chronic liver diseases such as metabolic-associated fatty liver disease, cirrhosis and hepatocellular carcinoma are major causes of global morbidity and mortality. Traditional scoring systems, including the fibrosis-4 (FIB-4) index and the model for end-stage liver disease (MELD), guide management decisions but primarily quantify disease severity rather than enabling early detection. MELD is calculated to determine how soon a patient needs a liver transplant; higher scores indicate more advanced disease. FIB-4 relies on age and specific enzyme tests, and its performance varies across populations, leading to false positives and missed cases. This situation highlights the need for tools capable of analysing complex clinical data to identify high-risk individuals at an early stage. We hypothesised that combining conventional scoring algorithms with modern machine-learning models and explainability techniques could enhance early diagnosis and personalised risk stratification.

Materials and Methods: A web-based clinical decision-support system was developed with separate modules for metabolic-associated fatty liver disease, cirrhosis and hepatocellular carcinoma. The system integrated established clinical scores and trained supervised algorithms (CatBoost, extreme gradient boosting and support vector machines) on publicly

available datasets. Data preprocessing included missing-value imputation via k-nearest neighbours, removal of outliers using z-scores and standardisation. Datasets were partitioned into 80 % training and 20 % test sets. Model transparency was achieved through Shapley additive explanation analysis, feature-importance tables, receiver-operating-characteristic curves and confusion matrices. To mitigate class imbalance in the hepatocellular carcinoma module, a balanced training set was used.

Results: The CatBoost model achieved the highest performance, attaining 92 % accuracy and a 0.95 area-under-the-curve value in predicting hepatocellular carcinoma. The extreme gradient boosting model used for cirrhosis classification reached 93 % accuracy on the test data. Explainability methods revealed cases in which FIB-4 suggested low risk yet the model predicted a high probability of cirrhosis because of factors such as elevated international normalised ratio and low albumin. In the metabolic-associated fatty liver disease module, the support-vector machine achieved comparable performance, and the system generated individual risk profiles and feature contributions.

Conclusions: Integrating traditional clinical scores with modern machine-learning algorithms and explanation methods improved early detection and risk stratification of chronic liver diseases. The system provides transparent predictions that highlight contributing factors, thereby alerting clinicians to high-risk patients who might otherwise be overlooked. These findings suggest that explainable artificial intelligence can augment existing screening strategies and support digital health solutions adaptable to resource-limited settings.

Keyword(s) : Chronic Liver Disease, Explainable Artificial Intelligence, Clinical Decision Support System, Machine Learning, Early Detection

1126 - Development of a Dual Module Artificial Intelligence System for Non Invasive Staging of Liver Fibrosis Using Blood Tests and Ultrasound Images

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Background / Hypothesis: Liver fibrosis staging guides therapy and predicts progression to complications such as cirrhosis. The current standard, percutaneous liver biopsy, is invasive and can cause pain, bleeding, infection and accidental injury. Moreover, sampling error and patient reluctance limit its utility. Existing non-invasive serum-based scores often lack accuracy in intermediate fibrosis stages. Evidence that combining different non-invasive modalities improves diagnostic accuracy suggests that integrating biochemical data with imaging could provide complementary information. We hypothesised that a dual-module artificial intelligence system that simultaneously analyses blood test data and ultrasound images can non-invasively predict fibrosis stage with high accuracy, offering a practical alternative to biopsy.

Materials and Methods: A web-based platform with two separate machine-learning modules was developed. The biochemical module used a random forest algorithm trained on 30691 records of liver biochemistry (aspartate aminotransferase, alanine aminotransferase, albumin and related markers) to estimate fibrosis stage. The imaging module employed a convolutional neural network based on ResNet-50 and transfer learning to classify 6323 B-mode ultrasound images. Each dataset was partitioned into 80 % training and 20 % test sets. Outputs from the two modules were fused and passed to a large language model that generated an interpretable report for clinicians.

Results: The imaging module achieved a test accuracy of 96.26 % in classifying ultrasound images across fibrosis stages. The random forest module analysing blood tests also demonstrated strong predictive performance. When predictions from both modules were combined, the system provided consistent stage estimates from F0 through F4 and produced detailed reports explaining the contributions of key laboratory and imaging features. The multi-modal integration improved robustness compared with using biochemical or imaging data alone.

Conclusions: Developing a dual-module artificial intelligence system that combines serum laboratory values and ultrasound images enables promising reliable non-invasive staging of liver fibrosis. By reducing reliance on biopsy and integrating complementary data sources, this approach may enhance diagnostic confidence, improve patient comfort and streamline clinical workflows. Such multi-modal decision-support systems illustrate the potential of explainable artificial intelligence in hepatology and could be adapted for other chronic liver diseases where early, non-invasive assessment is essential.

Keyword(s) : Liver Fibrosis, Non-Invasive Staging, Artificial Intelligence, Machine Learning, Multimodal Imaging

1127 - Development of a Multi Modal Artificial Intelligence Assistant for Early Diagnosis of Hepatocellular Carcinoma Using Imaging and Laboratory Data

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a leading cause of cancer-related death and is often detected at advanced stages. Early detection substantially improves eligibility for curative therapies and five-year survival rates. Standard surveillance with ultrasonography and alpha-fetoprotein (AFP) testing has limited sensitivity; in cirrhosis patients these methods detect early-stage HCC in only 45 % of cases, although adding AFP modestly increases sensitivity. Recent research highlights that integrating imaging, clinical and laboratory data can enhance HCC risk prediction and early detection. We hypothesised that a multi-modal artificial intelligence system combining magnetic resonance imaging (MRI) with laboratory results and computing personalised risk scores could improve early diagnosis and clinical decision-making.

Materials and Methods: A web-based multi-module assistant was developed. A U-Net convolutional neural network segmented the liver and tumours from three-dimensional MRI volumes. Liver fibrosis stages were predicted using a VGG16 network trained via transfer

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learning on 2843 medical images. Laboratory data—including serum AFP, the ratio of aspartate to alanine aminotransferase and bilirubin levels—were preprocessed and merged with imaging features. All data were integrated into a virtual liver model that computed personalised risk scores based on tumour-to-liver volume ratios and nodule counts. Outputs were summarised by a large language model to generate interpretable clinical reports. Model performance was evaluated on separate test sets.

Results: The segmentation module achieved a 92 % dice similarity score in separating liver and tumour regions. The fibrosis-staging module reached 93.13 % accuracy. Combining imaging and laboratory features within the virtual liver model yielded consistent risk estimates across tumour stages and fibrosis grades. The system produced detailed reports explaining the contribution of each feature, facilitating clinician interpretation. By integrating MRI with laboratory biomarkers, the assistant addresses the low sensitivity of ultrasound and AFP surveillance offers a data-driven alternative for early detection.

Conclusions: A multi-modal artificial intelligence assistant that combines MRI segmentation, fibrosis staging and laboratory data can provide accurate non-invasive assessment and personalised risk stratification for early detection of hepatocellular carcinoma. This approach overcomes limitations of current surveillance methods, has potential to increase early diagnosis rates and could be incorporated into clinical workflows to improve patient outcomes and reduce mortality.

Keyword(s) : Hepatocellular Carcinoma, Early Diagnosis, Artificial Intelligence, Multimodal Data, Magnetic Resonance Imaging

1130 - Effects of COVID 19 Vaccines on HBV Replication in Patients with Chronic Hepatitis B A Comparative Analysis of mRNA and Inactivated Vaccines

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Background and Aim: Chronic hepatitis B virus (HBV) infection remains a global health burden, affecting more than 250 million individuals and predisposing to cirrhosis and hepatocellular carcinoma. HBV DNA levels are the most reliable biomarker of viral replication and disease activity. With the global implementation of COVID-19 vaccination programs, questions have arisen regarding their impact on HBV replication, particularly with mRNA-based vaccines (BNT162b2) compared to inactivated vaccines (CoronaVac). This study aimed to evaluate the effects of COVID-19 vaccines on HBV DNA dynamics and to assess the modifying role of antiviral therapy (tenofovir, entecavir). **Methods:** In this retrospective cohort, medical records of 2,183 HBsAg-positive patients followed between January 2021 and December 2023 were screened. A total of 167 vaccinated patients met inclusion criteria, comprising 118 treated with antivirals and 49 untreated. An additional 80 unvaccinated HBsAg-positive patients served as controls. Patients were stratified according to vaccine type (mRNA vs. inactivated) and antiviral therapy status (tenofovir or entecavir). HBV DNA levels were recorded 3–6 months before vaccination and subsequently at 3-month intervals post-vaccination. Viral breakthrough was defined as $>1 \log_{10}$ IU/mL increase in HBV DNA, while smaller but significant increases were categorized as "HBV DNA rise. **Results:** Among antiviral-treated patients, HBV DNA rise was observed in 58.2% of BNT162b2 recipients and 12.8% of CoronaVac recipients, with no increase in the unvaccinated controls. In the untreated cohort, HBV DNA rise occurred in 84.6% of BNT162b2 recipients, 20% of CoronaVac recipients, and 17.5% of unvaccinated controls. Viral breakthrough was significantly more frequent in BNT162b2 recipients compared with both CoronaVac and control groups ($p<0.001$). In antiviral-treated subgroups, breakthrough rates were 22.8% for BNT162b2 and 2.6% for CoronaVac. Despite these increases, no hepatic flare or liver failure occurred, and HBV DNA levels in most patients returned to baseline within 12 months. **Conclusion:** COVID-19 vaccination, particularly with mRNA-based vaccines, appears to transiently increase HBV replication in patients with chronic infection, while inactivated vaccines show minimal effect. Antiviral therapy with tenofovir or entecavir reduces but does not completely eliminate this risk. These findings suggest the importance of close HBV DNA monitoring after vaccination, especially in untreated patients, and

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emphasize the need for prospective studies to clarify immunological mechanisms and long-term clinical significance.

Keyword(s) : Chronic Hepatitis B, HBV replication, COVID-19 vaccines, mRNA vaccine, Inactivated vaccine, Antiviral therapy

1132 - POTENTIAL HERB DRUG INTERACTIONS IN CHRONIC HEPATITIS B TREATMENT A CASE SERIES

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Background / **Hypothesis:**
The use of herbal supplements is increasingly widespread among patients with chronic diseases, including chronic hepatitis B (CHB). Although antiviral therapies such as tenofovir disoproxil fumarate (TDF), tenofovir alafenamide (TAF), and entecavir have high efficacy and safety, data on their pharmacokinetic and pharmacodynamic interactions with herbal products remain limited. Herb-drug interactions can alter antiviral drug metabolism, potentially leading to therapeutic failure or hepatotoxicity. This case series hypothesizes that concurrent use of herbal preparations with standard CHB therapy may influence liver enzyme levels or viral suppression by interfering with antiviral drug bioavailability.

Materials and **Methods:**
Four male patients (aged 35–60 years) with CHB receiving long-term antiviral therapy were evaluated between 2015 and 2024. All had previously achieved virological suppression and normal liver function tests (LFTs). Each patient presented with unexpected HBV DNA positivity or elevated transaminase levels during routine follow-up. Detailed histories were obtained to identify concurrent use of herbal products. After recognition of supplement use, patients were instructed to discontinue the herbal product while continuing antiviral treatment. Laboratory parameters, including HBV DNA and LFTs, were monitored at one- to six-month intervals.

Results:
Case 1: A 48-year-old on TDF developed detectable HBV DNA (1276 IU/mL) while using okra seed extract for osteoarthritis. HBV DNA became undetectable three months after discontinuation.
Case 2: A 35-year-old on TDF showed persistent low-level viremia (665 IU/mL) despite adherence; he had used cornflower extract tablets for four months. HBV DNA normalized within four months of cessation.
Case 3: A 42-year-old on TDF presented with HBV DNA

525 IU/mL after one month of artichoke capsule use; viral suppression was restored within three months.
Case 4: A 60-year-old on entecavir showed elevated LFTs (ALT 245 U/L) and detectable HBV DNA while taking garlic powder. After stopping garlic, both LFTs and HBV DNA normalized within six months. In all cases, normalization occurred without changes in antiviral therapy, suggesting reversible herb-drug interactions rather than resistance.
Conclusions:

This case series indicates that herbal supplement use may transiently impair virological control or elevate liver enzymes in CHB patients receiving standard antiviral therapy. The observed reversibility following supplement discontinuation supports a pharmacokinetic or pharmacodynamic interaction mechanism. Clinicians should routinely inquire about herbal product consumption in CHB follow-up, especially when unexplained viral breakthrough or hepatotoxicity occurs. Further controlled studies are needed to elucidate specific metabolic pathways and to guide safe co-administration practices.

Keyword(s) :

1133 - A Novel HFE Mutation C.1006+1G>A Disrupts Iron Homeostasis and Drives Hereditary Hemochromatosis Pathogenesis

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Background / **Hypothesis:**

Hereditary hemochromatosis (HH) is predominantly linked to pathogenic mutations in the homeostatic iron regulator (HFE) gene (NM_000410.3), yet non-classical variants remain understudied. We investigated a rare splice-site mutation (c.1006+1 G>A) in HFE, which retains intron 5 and introduces a premature stop codon, truncating the HFE protein's intracellular domain to elucidate its role in HH pathogenesis.

Materials and **Methods:**
Clinical data and liver MRI from a patient with iron overload were analyzed. Iron deposition was confirmed via histochemical staining of liver tissue. Sanger sequencing validated the c.1006+1 G>A mutation. Structural modeling (PyMOL) visualized the truncated HFE protein. Immunofluorescence and confocal microscopy quantified HFE expression in hepatocytes

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and gastric mucosal cells versus controls, supported by semi-quantitative analysis (Image Pro Plus 6.0).

Results:

Magnetic resonance imaging (MRI) of the liver and histopathological sections from the patient revealed typical iron deposition. Genetic testing confirmed the c.1006+1 G > A mutation in the HFE gene. The mutation retained intron 5 and introduced a stop codon, resulting in the truncation of the intracellular domain (R336-E348) of the HFE protein. Immunofluorescence and confocal microscopy revealed significantly reduced expression of HFE protein in hepatocytes and gastric mucosal epithelial cells compared to the control group ($p < 0.001$).

Conclusions:

The c.1006+1 G>A mutation disrupts HFE splicing, leading to a truncated protein with impaired iron regulatory function, directly contributing to HH. This study identifies a novel molecular mechanism underlying HH and underscores the diagnostic and therapeutic potential of targeting this mutation.

Keyword(s) : Hereditary hemochromatosis; homeostatic iron regulator; IVS5 mutation; liver; iron overload.

1139 - Sequential Application of the Fibrosis 4 Index and Serum Gamma Glutamyl Transferase for Enhanced Detection of Advanced Fibrosis in Type 2 Diabetes Mellitus Patients with Metabolic Dysfunction Associated Steatotic Liver Disease

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Background / Hypothesis:

The Fibrosis-4 index (FIB-4) reliably stratifies advanced fibrosis in patients with metabolic dysfunction-associated steatotic liver disease (MASLD). However, the limitation of FIB-4 is its considerable proportion of indeterminate results, notably in patients with type 2 diabetes mellitus (T2DM). Additionally, tools like vibration-controlled transient elastography (VCTE) are

often unavailable in resource-limited settings. This study aimed to compare the performance of FIB-4 in MASLD patients with and without T2DM and to identify potential biomarkers associated with advanced fibrosis.

Materials and Methods:

A cross-sectional study was conducted on patients who underwent VCTE for steatotic liver disease at Universiti Malaya Medical Centre. Clinical data, including T2DM status, FIB-4 index, and liver stiffness measurements by VCTE, were collected.

Results:

A total of 253 MASLD patients were analyzed, with 55% having T2DM. Among T2DM patients, 46.8% had indeterminate or high-risk FIB-4 ≥ 1.3 , compared to 25.4% of patients without T2DM. A greater proportion of T2DM patients had indeterminate FIB-4 (1.3–3.25) results (38.8% vs. 21.9% in non-T2DM). In T2DM patients with an indeterminate FIB-4, gamma-glutamyl transferase (GGT) levels above the upper limit of normal indicated a 10.5-fold increased risk of advanced fibrosis (odds ratio 10.5, 95% CI 2.94–37.55, $P < 0.001$). The proposed sequential use of FIB-4 followed by the GGT test in FIB-4 indeterminate cases improved stratification performance (sensitivity from 58.2% to 71.4%, specificity from 63.9% to 80.8%), while reducing referrals from 46.8% to 25.9%. FIB-4 ≥ 3.25 also appeared to predict advanced fibrosis effectively.

Conclusions:

A sequential FIB-4 followed by GGT approach improves fibrosis stratification and may reduce unnecessary tertiary-care referrals.

Keyword(s) : fatty liver, fibrosis, FIB-4, MAFLD, MASLD, NAFLD

1140 - EEF1E1 sustains EGFR signaling to drive immune evasion and tumor progression in hepatocellular carcinoma

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Background / Hypothesis: Despite the advent of immune checkpoint inhibitors, clinical benefit in hepatocellular carcinoma (HCC) remains limited. Robust, clinically actionable biomarkers are urgently needed to identify patients most likely to benefit from

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immunotherapy and to guide rational combination strategies.

Materials and Methods: We evaluated EEF1E1 expression and prognosis in HCC using an immunohistochemistry tissue microarray (n = 141). Pro-tumor effects were tested by gain/loss-of-function assays in vitro and in vivo, including an orthotopic tumor model. The impact on the tumor immune microenvironment was profiled by single-cell RNA sequencing and multiparameter flow cytometry. EEF1E1-EGFR interaction was examined by co-immunoprecipitation and related signaling assays.

Results: EEF1E1 was highly expressed in HCC tissues and served as an independent adverse prognostic factor. Functionally, EEF1E1 enhanced invasion and migration and promoted epithelial-mesenchymal transition in vitro and in vivo. EEF1E1 reshaped the tumor immune microenvironment toward immunosuppression, characterized by increased infiltration of PD-1+ CD8 T cells. Mechanistically, EEF1E1 interacted with EGFR, activated EGFR-STAT signaling, and facilitated EGFR accumulation by impeding endosomal-lysosomal degradation. Consistent with this, tumors with high EEF1E1 expression showed superior efficacy from combined PD-1 and EGFR inhibition compared with either monotherapy.

Conclusions: EEF1E1 drives aggressive biology and immune dysfunction in HCC by sustaining EGFR signaling and may serve as a prognostic and predictive biomarker to guide combined PD-1 and EGFR blockade.

Keyword(s) : hepatocellular carcinoma; immunotherapy; EEF1E1; EGFR

1141 - Sequential met inhibition from crizotinib to capmatinib in a patient with advanced met amplified intrahepatic cholangiocarcinoma a case report
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Background: Intrahepatic cholangiocarcinoma (iCCA) is the second most common primary liver malignancy. It carries a poor prognosis, with limited options after first-line therapy failure. MET amplification is a rare (2%-4%) but targetable driver in iCCA cases.
Case Presentation: We present the case of a 49-year-old male with metastatic iCCA complicated by cirrhosis and thrombocytopenia. After progressing on an adjusted first-line regimen (nanoparticle albumin-bound paclitaxel, S-1, radiotherapy, and simvastatin), next-

generation sequencing (NGS) of tissue confirmed a high-level MET amplification (copy number 13). The patient was initiated on crizotinib, achieving a rapid partial remission (PR) that lasted 4.1 months. Upon progression, liquid biopsy NGS confirmed persistent MET amplification (copy number 4), which guided a switch to capmatinib, a more selective MET inhibitor. This sequential strategy brought the patient a sustained progression-free survival (PFS) of 10 months on capmatinib and an overall survival (OS) of 24 months from the advanced diagnosis.
Conclusion: This case demonstrates that dynamic molecular monitoring via NGS can effectively guide sequential MET-TKI therapy. Switching to a more potent MET inhibitor, rather than abandoning the target, may be a promising strategy for MET-amplified iCCA that progresses on a first-generation TKI while maintaining MET dependency.

Keyword(s) : cholangiocarcinoma, MET amplification, sequential targeted therapy, crizotinib, capmatinib.

1144 - The GREM1-BMP7 axis orchestrates apoptosis driven fibrotic remodeling in biliary atresia

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Background / Hypothesis: Biliary Atresia (BA) is a life-threatening fibro-inflammatory cholangiopathy of infancy that progresses to cirrhosis despite timely Kasai portoenterostomy. The mechanistic interplay between apoptosis, necroptosis, and fibrogenesis remains poorly defined, impeding biomarker discovery and therapeutic targeting.

Materials and Methods: Peripheral blood and extrahepatic biliary tissue from BA patients, and controls were analyzed using targeted RT² Profiler PCR arrays. Data underwent PCA, hierarchical clustering, and Weighted Gene Co-Expression Network Analysis. Feature selection employed multi-model machine learning (LASSO, Elastic Net, Random Forest, XGBoost, SVM) with cross-validation. Gene Ontology, Reactome, and Ingenuity Pathway Analysis, were used for functional annotation. Correlations with clinical parameters and histopathology were evaluated. Relative mRNA expression of key molecules were quantified by

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qRT-PCR among healthy controls, biliary atresia, and non-BA neonatal cholestasis groups.

Results:

A molecular signature of FASLG, GREM1, SNAI1, MMP8, IL10, and BCL2A1 showed strong and reproducible discriminatory power between BA and controls, with consistently high AUC = 1.0 values across different ML models. WGCNA revealed a dominant turquoise module integrating apoptosis, necroptosis, and fibrotic regulators ($r = 0.82$, $p < 0.001$). Network analysis identified TNF, CASP8, TGFB1, and GREM1 as topological hubs bridging apoptotic and fibrotic pathways. IPA demonstrated co-activation of TNF/FAS signaling, TGF- β /SMAD2/3, and RIPK1-RIPK3-MLKL necroptosis, delineating a feed-forward inflammatory-fibrogenic loop. Upregulation of GREM1 and SNAI1 ($p < 0.001$) was functionally linked to suppression of BMP7-mediated antifibrotic signaling and epithelial-mesenchymal transition activation. Expression of GREM1 correlated strongly with serum γ -GT ($r = 0.73$) and histological fibrosis grade, establishing direct clinical association. GREM1 expression was significantly upregulated in BA compared to HC and NC ($p < 0.001$), with a diagnostic cut-off value of 5.373.

Conclusions:

The study establishes a unified apoptosis-necroptosis-fibrosis axis as the molecular driver of progressive liver injury in BA. The identified multi-gene panel holds promise as a minimally invasive biomarker set for disease diagnosis and progression monitoring where GREM1 emerges as a promising diagnostic and therapeutic target.

Keyword(s) :

1147 - Global regional and national burdens of cirrhosis in adolescents and young adults from 1990 to 2021

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Background

Cirrhosis is a leading cause of morbidity and mortality

Hypothesis:

worldwide, yet epidemiological data specific to adolescents and young adults (AYAs, aged 15–39 years) remain limited. This study aimed to evaluate global, regional, and national trends in the burden of cirrhosis among AYAs from 1990 to 2021 and to characterize etiologic contributions.

Materials

and

Methods:

Data on incidence, prevalence, mortality, and disability-adjusted life years (DALYs) of cirrhosis among individuals aged 15–39 years were obtained from the Global Burden of Disease (GBD) 2021 database. Temporal trends were assessed using estimated annual percentage change (EAPC) at global, regional, and national levels. Subgroup analyses were conducted by sex, age group, etiology, and Socio-demographic Index (SDI)

Results:

Globally, incident cases increased from 20,240,719 in 1990 to 32,087,179 in 2021 (EAPC = 0.55 [0.51 - 0.59]). The burden attributable to hepatitis B virus (EAPC = -1.51 [-1.65 to -1.37]) and hepatitis C virus (EAPC = -0.07 [-0.14 to 0.00]) decreased, whereas alcohol-related cirrhosis (EAPC = 0.13 [0.03 - 0.23]), non-alcoholic fatty liver disease (EAPC = 0.79 [0.74 - 0.84]), and other causes (EAPC = 0.18 [0.06 - 0.31]) showed rising trends. Males exhibited a higher burden than females, and incidence peaked at ages 20–24.

Conclusions:

Cirrhosis poses an increasing health burden. Given the changing etiology, early adulthood represents a period of peak vulnerability, and substantial geographic and sex disparities exist. Public health strategies should prioritize viral hepatitis prevention, alcohol control, and metabolic risk management, with targeted interventions for high-risk populations, particularly in low-SDI regions.

Keyword(s) : Cirrhosis, Adolescents, Young adults, Burdens.

1149 - Dead box helicase1 promote cancer stemness of hepatocellular carcinoma by enhancing FSCN1 mRNA translation

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Background

/ Hypothesis: Hepatocellular carcinoma (HCC) is a highly heterogeneous tumor with a high recurrence rate and mortality. Liver cancer stem cells are a major source of tumor heterogeneity and a critical factor in tumor

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recurrence, metastasis, and drug resistance. An in-depth exploration of the stem cell-like characteristics of HCC is essential for unraveling HCC heterogeneity, elucidating its pathogenesis, reducing recurrence, and overcoming therapeutic resistance. We aim to explore the intratumor heterogeneity through single-cell transcriptomic profiling and identify the key driver of cancer stemness in HCC.

Materials and Methods: Single-cell RNA sequencing (scRNA-seq) and bulk RNA sequencing of HCC were integrated to reveal key cell subpopulations and therapeutic targets that determine the stemness and tumorigenesis of HCC. Furthermore, in vitro and in vivo studies were performed to elucidate the underlying mechanism.

Results:

scRNA-seq was conducted on 10,548 cells from twelve primary HCC samples. After cluster analysis, CytoTRACE analysis and Scissor analysis of heterogeneous malignant cells revealed that cells with high stemness were enriched in the Scissor+ cell subpopulation, which was associated with poor prognosis. Next, integrated analysis with bulk RNA-sequencing profiles indicated that DEAD-box helicase 1 (DDX1) was a key driver of cancer stemness and tumorigenesis in HCC. Furthermore, DDX1 was found to be highly expressed in HCC tissues and liver cancer stem-cell-like cells. In vitro and in vivo assays showed, for the first time, that the stem cell-like properties of HCC cells were inhibited by DDX1 depletion, as evidenced by the decreased size and number of formed spheres, the downregulated expression of C-MYC, OCT4, and NANOG proteins, as well as the reduced proportion of CD133+ cells. In addition, depletion of DDX1 expression also restrained proliferation, migration, and invasion, yet facilitated apoptosis of HCC cells. Overexpression of DDX1 exhibited the opposite effects on the stemness and tumorigenesis of HCC. Mechanistically, high-throughput RNA sequencing, IP-MS, and RIP analyses revealed that DDX1 directly binds to FSCN1 mRNA and facilitates its translation without altering mRNA levels, thereby enhancing FSCN1 protein expression. Furthermore, DDX1 interacts with eIF4E and eIF4G1 to promote FSCN1 translation, which in turn activates the MAPK/ERK1/2 pathway, ultimately enhancing liver stemness and tumorigenesis.

Conclusions:

Our study unraveled the intratumor heterogeneity of HCC malignant cells and highlighted the functional significance of DDX1 in regulating HCC stemness and

tumorigenesis. These findings could facilitate the development of novel prognostic strategies and therapies for HCC.

Keyword(s) : Hepatocellular carcinoma, Tumor heterogeneity, Cancer stemness, DDX1, FSCN1

1153 - A Pilot Program of Community Volunteers to Improve Adherence to Hepatitis B Medications in Kiribati

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Background / Hypothesis: Chronic hepatitis B (CHB) is a significant global health concern affecting approximately 254 million people worldwide with sequelae of cirrhosis, liver failure, and hepatocellular carcinoma. While effective antiviral therapies exist, adherence remains a significant challenge due to barriers such as long-term treatment, medication fatigue, stigma, cost, transportation, and health literacy. Antiviral adherence is crucial for achieving and maintaining viral suppression and improving long-term outcomes and quality of life. Studies demonstrate poor adherence can lead to treatment failure, viral resistance, and increased risk of liver-related complications including hepatocellular carcinoma. Suboptimal adherence to antiviral therapy for CHB has been problematic in Kiribati, particularly amongst high-risk groups of pregnant women and people with cirrhosis.

Materials and Methods: A community volunteer intervention was initiated in Kiritimati, a remote island in Kiribati, to address adherence to antiviral therapy for CHB. Community volunteers were assigned to HBsAg-positive patients newly commencing antiviral therapy who were pregnant or had cirrhosis. During home visits (weekly for pregnant women and monthly for cirrhotic patients), volunteers provided education and encouraged medication adherence and clinic follow-up. Adherence in the intervention group was compared to HBsAg-positive patients on treatment on other remote islands in Kiribati without the intervention.

Results: Adherence was measured by medication bottle

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counts. Outcomes were assessed by two measures: overall group adherence (total bottles taken by the group divided by total months since treatment start) and the proportion of individuals in the group with adherence >80%. When compared to patients on treatment in other outer islands, group adherence was 77.4% with volunteer intervention (n=51) versus 52.5% without (n=205, $p<0.001$). Overall adherence amongst pregnant women in the intervention group was 89.7%. Individual adherence >80% was met in 58.8% of the intervention group and 34.1% in those without the intervention ($p=0.0012$).

Conclusions: Although these findings are preliminary and the control and intervention groups were not matched, this pilot program suggests community volunteers are a potential strategy to improve adherence to CHB in a high-prevalence setting in the Pacific region. Improved adherence can reduce vertical transmission, decrease hospitalizations and complications including HCC and deaths. CHB patients commonly have co-morbidities such as diabetes, hypertension, and heart disease requiring long-term medication adherence that could also be addressed during volunteer visits. Fostering a supportive healthcare environment with volunteers in the local community reduces stigma, promotes education, and encourages open communication that can empower patients to adhere to treatment.

Keyword(s) :

1156 - Contrast enhanced Ultrasound Features of Combined hepatocellular carcinoma and cholangiocarcinoma (cHCC CC) A Case Series Report

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Background / Hypothesis: Combined hepatocellular carcinoma and cholangiocarcinoma (cHCC-CC) a rare histologic variant of primary liver malignancy, accounting for approximately 4–14.2% of cases. Due to its concurrent composition of both hepatocellular carcinoma and cholangiocarcinoma elements, definitive preoperative diagnosis remains challenging. Non-specific clinical and imaging findings lead to a high misdiagnosis rate. The final diagnosis mainly depends on pathological examination. This article aims to explore the ultrasound and contrast-enhance ultrasound (CEUS) features of cHCC-CC to improve its early diagnosis.

Materials and Methods: A retrospective analysis of ultrasound imaging data and clinical manifestations of 10 patients pathologically confirmed with cHCC-CC from 2017 to 2024 was conducted.

Results:

The patients ranged from 38 and 71 years old, 9 male and 1 female. The lesions were solitary, with a maximum diameters of 2.3-8.6cm. Serum Alpha-fetoprotein (AFP) was elevated in the majority of patients (60%), whereas carbohydrate antigen 19-9 (CA19-9) was elevated in 20%, concurrent elevation of both markers was observed in only one case (10%). The conventional ultrasound showed irregular hypoechoic mass with blurred boundary (80%), frequently exhibited irregular margins (60%), and intratumoral hypervascularity was observed in half of the cases. The arterial phase of the CEUS was peripheral Homogeneous hyperenhancement/heterogeneous hyperenhancement, and the portal phase and delayed phase were both hypoenhancement. Three of the lesions had unenhanced zones. The most common enhancement patterns (70%) were high peripheral Homogeneous enhancement in arterial phase, hypoenhancement in portal phase and delayed phase. The arterial phase may be Homogeneous hyperenhancement (70%) or Inhomogeneous hypoenhancement (30%). Most of the lesions showed hypoenhancement in portal phase (90%) and delayed phase (100%). There were irregularly unenhanced zones in some lesions (30%).

Conclusions: The present ultrasonographic study revealed that the majority of combined hepatocellular – cholangiocarcinoma (cHCC-CC) lesions present as irregular, hypoechoic intrahepatic masses with conspicuous intratumoral vascularity. The arterial phase of the CEUS was peripheral Homogeneous hyperenhancement/heterogeneous hyperenhancement, and the portal phase and delayed phase were both hypoenhancement. There were irregularly unenhanced zones in some lesions. Combining these ultrasound characteristics with their laboratory test results (increased AFP and CA19-9) can assist in the correct diagnosis of cHCC-CC.

Keyword(s) : Combined hepatocellular carcinoma and cholangiocarcinoma, US, CEUS, cHCC-CC, contrast-enhance ultrasound

1157 - Predictive Model for Targeted Combination Immunotherapy Efficacy in Unresectable

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Hepatocellular Carcinoma Patients Based on Multimodal MRI Radiomics (MC hccAI 007) A Multicenter Study

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Background / **Hypothesis:**
Targeted combination immunotherapy has improved outcomes in unresectable hepatocellular carcinoma (HCC), yet the majority of patients fail to respond. Existing clinical staging and biomarker systems offer limited predictive power. Radiomics enables noninvasive quantification of tumor heterogeneity and may enhance therapeutic response prediction.
Materials and **Methods:**
In this multi-center study, 142 patients with unresectable HCC underwent pre-treatment contrast-enhanced MRI. Radiomic features from arterial and portal venous phases were extracted and optimized through multistage selection and LASSO-Cox regression. 117 machine-learning algorithms were compared, and the best-performing model was validated via 10-fold cross-validation. Proteomic profiling of 15 resected samples identified biological correlates of imaging phenotypes. Model interpretability was assessed using SHAP and Grad-CAM analyses.

Results:
Fifteen prognostic features were retained. The Random Survival Forest (RSF) model achieved the best performance (C-index=0.79, 95%CI: 0.757–0.953). RSF score, ALBI score, and performance status were independent predictors of overall survival ($P<0.05$). Stratification based on RSF-derived risk (cutpoint=14.49) revealed significantly improved survival in the low-risk group (HR=0.19, $P<0.0001$). Proteomics identified 24 differential proteins enriched in NK-cell cytotoxicity and FcγR-mediated phagocytosis pathways.

Conclusions:
The multimodal MRI radiomics model provides an interpretable and biologically grounded tool for predicting immunotherapy efficacy in unresectable HCC, enabling personalized risk stratification and treatment optimization.

Keyword(s) : Hepatocellular carcinoma, Immunotherapy, MRI, Predictive model, Machine learning.

1158 - Donor Isolated Hyperbilirubinemia and Post Liver Transplant Survival A Propensity Matched Analysis

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Background / **Hypothesis:**
Donor hyperbilirubinemia is commonly viewed as a sign of hepatic dysfunction and often leads to organ discard, despite limited evidence on its true prognostic meaning. However, mild to moderate bilirubin elevation without accompanying liver enzyme abnormalities—termed isolated hyperbilirubinemia—may represent benign conditions rather than intrinsic hepatic injury. Understanding the impact of such donor characteristics on transplant outcomes is essential to improve organ utilization. This study aimed to evaluate the post-transplant survival of recipients who received grafts from donors with isolated hyperbilirubinemia compared with those from donors with normal bilirubin levels.

Materials and **Methods:**
We conducted a retrospective cohort study including adult liver transplant recipients (≥ 18 years) between January 2020 and March 2022. Donors were categorized according to pre-procurement serum bilirubin levels. Isolated hyperbilirubinemia was defined as total bilirubin between 1.2–5.0 mg/dL, with normal alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase levels, and no evidence of systemic illness. Recipients were propensity score-matched (1:1) for age, sex, and year of transplant, yielding 36 matched pairs. Post-transplant survival was analyzed using Cox proportional hazards models adjusted for potential confounders and validated through stepwise selection procedures. All analyses were conducted using Python 3.11 with appropriate statistical libraries.

Results:
A total of 72 liver transplant recipients (mean age: 53 years; 52.8% male) were included. The median follow-up duration was 5.7 months (interquartile range: 0.3–28.2 months), during which 47 deaths occurred. Remarkably, recipients of grafts from donors with isolated hyperbilirubinemia demonstrated substantially better survival compared to those receiving grafts from donors with normal bilirubin levels. After full adjustment, donor

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isolated hyperbilirubinemia was associated with an 81% reduction in mortality risk (adjusted hazard ratio: 0.19; 95% confidence interval: 0.08–0.45; $p < 0.001$). The observed benefit remained consistent across sensitivity analyses and was independent of donor and recipient clinical profiles.

Conclusions:

Isolated donor hyperbilirubinemia does not appear to indicate poor graft quality. Rather, such donors may safely expand the liver donor pool without compromising—and possibly improving—recipient survival. Recognizing this benign biochemical pattern could prevent unnecessary organ discard and optimize organ utilization. Larger, multicenter studies with longer follow-up are needed to confirm these findings and clarify the mechanisms underlying the observed benefit.

Keyword(s) : Liver transplantation; donor hyperbilirubinemia; graft survival; post-transplant outcomes; donor selection

1159 - Gut Microbiota Derived CoQ8 Improves Hepatic Lipid Metabolism via GPR120

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Background / **Hypothesis:**
Dysbiosis of the gut microbiota has been implicated in modulating host metabolic processes through microbial metabolites; however, the precise underlying mechanisms remain inadequately defined. Our prior investigation revealed a marked reduction in serum levels of coenzyme Q8 (CoQ8) among obese pediatric subjects. We posited that this diminution results from the depletion of specific gut bacterial taxa and contributes to metabolic dysregulation via the gut-liver axis.

Materials and **Methods:**

Serum CoQ8 was analyzed by LC-MS/MS in 80 obese and 40 lean children, correlating levels with metabolic parameters. To trace its gut origin, fecal metagenomic sequencing identified CoQ8-associated bacteria in a subgroup, and their production capacity was confirmed by in vitro culture. Fecal microbiota transplantation from obese or healthy donors into germ-free mice tested effects on serum CoQ8 and liver metabolism. Mechanism studies included SPR binding, STAT3 knockdown, ChIP, and hepatocyte assays. In HFD-fed

mice, treatment with engineered CoQ8-producing bacteria or CoQ8, plus a GPR120 antagonist, evaluated therapeutic efficacy.

Results:

Serum CoQ8 was significantly lower in obese children and inversely correlated with adverse lipid profiles and insulin resistance. Metagenomic analysis revealed a decreased abundance of putative CoQ8-producing bacteria, such as *Prevotella* and *Bacteroides*. In vitro culture confirmed their efficient CoQ8 biosynthesis. Crucially, FMT demonstrated that microbiota from obese donors was sufficient to reduce serum CoQ8 and exacerbate hepatic steatosis in recipient mice, a phenotype rescued by CoQ8 supplementation. Mechanistically, CoQ8 bound to GPR120 with high affinity ($KD=3.2 \mu M$), activating STAT3 signaling. Phosphorylated STAT3 directly bound to the promoter of carnitine palmitoyltransferase 1 (CPT1), upregulating fatty acid oxidation. In HFD-fed mice, supplementation with the engineered CoQ8-producing bacterium replicated the benefits of direct CoQ8 administration, significantly reducing hepatic triglyceride accumulation in a GPR120-dependent manner.

Conclusion:

Our findings define a gut-liver axis wherein obesity-associated depletion of specific gut bacteria reduces circulating CoQ8. This microbial metabolite activates the hepatic GPR120-STAT3-CPT1 pathway to promote fatty acid oxidation and improve lipid homeostasis. Restoring CoQ8-producing bacteria or supplementing with CoQ8 represents a promising therapeutic strategy for obesity-related metabolic disorders.

Keyword(s) : Children Obesity, Lipid Metabolism, CoQ8

1162 - Study on the Mechanism of Samhd1 Mediated NEMO Polyubiquitination in Liver Ischemia Reperfusion Injury

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Background / **Hypothesis:**
Hepatic ischemia reperfusion injury (IRI) is one of the major pathological processes in liver transplantation and various liver surgeries. To date, there is still a lack of effective diagnosis and treatment strategies for hepatic IRI, and one of the root causes is that its mechanism has not been fully elucidated. This study aims to explore the molecular mechanism by which Samhd1 participates in

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the regulation of hepatic IRI, providing a research foundation and theoretical basis for the prevention and treatment of hepatic IRI.

Materials and Methods: This study established a classic 70% liver ischemia-reperfusion injury (IRI) model in mice. The expression and localization of key molecules were detected using methods such as immunoprecipitation, immunofluorescence, quantitative real-time polymerase chain reaction (qRT-PCR), and enzyme-linked immunosorbent assay (ELISA). The pathological damage and liver function of mice in each group were examined through hematoxylin and eosin (HE) staining, terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) assay, and microplate method.

Results:

We have discovered that Samhd1 is a key molecule exacerbating hepatic ischemia - reperfusion injury (IRI). Down - regulation of Samhd1 can significantly alleviate hepatic IRI, while over - expression of Samhd1 yields the opposite effect. Mechanistically, Samhd1 can mediate the polyubiquitination modification of NEMO, induce the activation of the IKK inflammatory complex, and enhance its stability, ultimately leading to the phosphorylation and combined translocation of multiple subunits of the IKK family. Inhibition of IKK can reverse hepatic IRI caused by Samhd1 enrichment.

Conclusions:

In conclusion, this study has revealed the potential functions and roles of Samhd1 in liver ischemia-reperfusion injury (IRI). Targeting Samhd1 may be a potential strategy for treating liver IRI.

Keyword(s) : liver transplantation, liver ischemia reperfusion, interactions between proteins

1163 - One-Pot Assay Based on CRISPR Cas13a Technology for HEV RNA Point-of-Care Testing

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Background / Hypothesis: Hepatitis E virus (HEV) poses a serious threat to both public health and animal food safety, thereby highlighting the demands for rapid, sensitive, and easy-to-use detection. This study aimed to develop a One-Pot assay using CRISPR/Cas13a for detecting HEV RNA, suitable for point-of-care testing (POCT) in resource-limited settings.

Materials and Methods: We established, optimized, and evaluated the methods for HEV RNA detection using CRISPR/Cas13a combined with reverse transcription polymerase chain reaction (RT-PCR) and reverse transcription recombinase-aided amplification (RT-RAA). Additionally, a large cohort of 154 HEV-infected patients and 74 HEV-infected animal specimens were utilized for application validation.

Results:

The RT-PCR/RT-RAA+CRISPR/Cas13a assays for HEV RNA detection (genotypes: HEV-1, HEV-3, and HEV-4) were established, optimized, and validated, achieving a limit of detection (LoD) of 1copy/μL and 100% specificity. In the HEV-infected application validation (ddPCR assay as the gold standard), the positivity rates of patients detected by RT-qPCR, RT-PCR-CRISPR and RT-RAA-CRISPR assays were 70.8%, 98.6%, and 79.2%, respectively, and among animals were 75.8%, 95.2%, and 87.1%, respectively, which were superior to those of RT-qPCR. Furthermore, sample rapid lysis, reagent lyophilization, and the One-Pot device were integrated to construct a One-Pot assay with an LoD of 102copies/μL. The 5-minute rapid lysis of processed specimens (serum and feces) significantly reduces the assay time to 35min, making it easy to perform, minimizing contamination, and meeting the requirements for screening.

Conclusions:

We developed a One-Pot assay for HEV RNA detection using CRISPR/Cas13a, which effectively achieves a "sample in, result out" POCT system, maximizes the potential for POCT implementation, and shows promise as a valuable tool for detecting and monitoring HEV infection.

Keyword(s) : Hepatitis E virus (HEV), CRISPR/Cas13a, Recombinase-aided amplification (RAA), One-Pot assay, Point-of-Care Testing (POCT)

1164 - Study on the Mechanism of TRIM55 Mediating K63 Polyubiquitination of Bcl10 Protein to Aggravate Liver Ischemia Reperfusion Injury

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Background / Hypothesis: Hepatic ischemia-reperfusion injury (IRI) has long been a major concern for liver transplant and various liver surgery patients, severely affecting prognosis. This study aims to clarify the molecular mechanism by which the

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E3 ubiquitin ligase trimeric domain protein TRIM55 regulates liver IRI, providing a research foundation and theoretical basis for the prevention and treatment of liver IRI.

Materials and **Methods:**
We established a 70% liver ischemia-reperfusion injury (IRI) model in classic mice. We detected the expression and localization of key molecules using methods such as IHC, WB, immunofluorescence, qRT-PCR, and ELISA. We also examined the pathological damage and liver function of mice in each group using HE staining, TUNEL assay, and microplate method.

Results:

We have discovered that TRIM55 is an important signal transduction protein that exacerbates liver IRI. Downregulation of TRIM55 can significantly alleviate liver IRI, while overexpression of TRIM55 has the opposite effect. Mechanistically, TRIM55 can mediate the polyubiquitination modification of Bcl10 at the K63 site, inducing the classical and non-classical IKK inflammatory signaling pathways, thereby exacerbating liver IRI. Specific inhibition of IKK can reverse liver IRI caused by TRIM55 enrichment.

Conclusions:

In conclusion, this study has revealed the potential functions and roles of TRIM55 in liver ischemia-reperfusion injury (IRI). Targeting TRIM family proteins may be a potential strategy for treating liver IRI.

Keyword(s) : liver transplantation, liver ischemia reperfusion, TRIM

1167 - SERPINE2 mitigates hepatic ischemia-reperfusion injury via a SUMO-ubiquitin rheostat
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Background / **Hypothesis:**

Hepatic ischemia-reperfusion injury (HIRI) is a prototypical tissue stress-induced injury. Serpin family E member 2 (SERPINE2) has been implicated in tissue remodeling, but its role in HIRI remains undefined. This study aimed to elucidate the biological function of SERPINE2 during HIRI and to evaluate its potential as a therapeutic target.

Materials and **Methods:**
Donor liver biopsies from 50 transplants were profiled for SERPINE2 and correlated with recipients'

postoperative day-3 (POD3) biochemistry. A murine partial-liver HIRI model was used in wild-type, hepatocyte-specific Serpine2 knockout (Serpine2-HKO), and AAV8-mediated hepatocyte SERPINE2- or TRIM28-overexpressing mice. SERPINE2 expression was significantly reduced after reperfusion in both human liver graft biopsies and murine livers, with donor hepatic SERPINE2 inversely correlated with recipients' POD3 ALT/AST.

Results:

Relative to controls, Serpine2-HKO mice exhibited aggravated liver injury, inflammatory infiltration, hepatocellular apoptosis, and early fibrotic remodeling after HIRI, whereas hepatocyte-targeted SERPINE2 overexpression mitigated these phenotypes. Mechanistically, SERPINE2 enhanced the expression of TRIM28, which directly interacted with NRF2, promoting its SUMOylation while constraining its ubiquitination, thereby stabilizing NRF2 and sustaining its antioxidant transcriptional program. Reconstitution of TRIM28 in Serpine2-HKO mice partially reversed biochemical and histologic injury and restored NRF2 downstream signaling.

Conclusions:In conclusion, SERPINE2 confers protection in HIRI by modulating TRIM28 to promotes NRF2 SUMOylation and attenuates its ubiquitin-mediated degradation, thereby maintaining NRF2 proteostasis and antioxidant capacity. These findings provide new insights into potential therapeutic strategies for mitigating HIRI.

Keyword(s) : Hepatic ischemia-reperfusion injury ; SERPINE2 ; SUMOylation

1168 - Peginterferon alpha 2b and nucleos(t)ide analogs combination therapy for chronic hepatitis B patients with high HBsAg level at the end of treatment (52 weeks) from ongoing CREATED Excellent Study

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Background / **Hypothesis:**
Background and **Aims:**
Chronic hepatitis B (CHB) patients with HBV DNA < LLOQ, HBeAg negative and HBsAg <1500 IU/mL after prior nucleos(t)ide analogs (NAs) treatment were the preferred population for clinical cure (PPCC). Peginterferon alpha (PegIFN α)-based therapy enables clinical cure in this population. The purpose of this study aims to observe the efficacy of NAs combined with PegIFN α -2b in treating CHB patients with HBsAg >1500 IU/mL (Non-preferred population for clinical cure, NPPCC).

Methods:

The CREATED EXCELLENT study (ChiCTR2400081073) is a multi-center, prospective, non-randomized, open-label, positive-controlled study. CHB patients with NAs treated for ≥ 3 months and HBsAg >1500 IU/mL were enrolled and divided into two groups. The NAs monotherapy group continued to receive their original NAs (ETV/TDF/TAF/TMF) treatment, while the PegIFN α -2b+NAs group received PegIFN α -2b (180 μ g/week) subcutaneously in addition to NAs. The total treatment period was 52 weeks, and all participants were followed up for 24 weeks after the end of treatment (EOT).

Results:

A total of 682 patients (NAs monotherapy group, n=330; PegIFN α -2b+NAs group, n=352) were included. At baseline, there were 170 patients with HBsAg between 1500 IU/mL and 3000 IU/mL (NAs group: 21.81%, PegIFN α -2b+NAs group: 27.84%), 133 patients with HBsAg between 3000 IU/mL and 6000 IU/mL (NAs group: 18.23%, PegIFN α -2b+NAs group: 20.68%), 90 patients with HBsAg between 6000 IU/mL and 9000 IU/mL (NAs group: 12.11%, PegIFN α -2b+NAs group: 14.24%); and 289 patients with HBsAg greater than 9000 IU/mL (NAs group: 47.91%, PegIFN α -2b+NAs group: 37.23%). The baseline average HBsAg level in the NAs group was 18767.23 $\pm$ 5623.33 IU/mL and in the PegIFN α -2b+NAs group was 17346.91 IU/mL (P > 0.05). After 52 weeks of treatment, the average HBsAg level of the NAs group was 16963.92 $\pm$ 5127.89 IU/mL and the PegIFN α -2b+NAs group was 3589.40 $\pm$ 1223.47 IU/mL (P<0.001). The stratification ratio of patients with HBsAg<1500 IU/mL, 1500<HBsAg<3000 IU/mL, 3000<HBsAg<6000 IU/mL, 6000<HBsAg<9000 IU/mL and more than 9000 IU/mL were 46.51%, 14.11%, 25.66%, 4.73% and 9.32%, respectively, in the PegIFN α -2b+NAs group; there rates were 20.00%, 30.15%, 26.72%, 3.32%, and 20.22% in the NAs group, respectively. At week 52 of EOT, 2 participants in NAs

group had HBsAg loss, compared with 9 participants in PegIFN α -2b+NAs group.

Conclusions: Compared with NAs monotherapy, PegIFN α -2b combined with NAs therapy for the treatment of NPPCC (HBsAg>1500 IU/mL) rapidly reduced HBsAg levels, and increased the number of PPCC (HBsAg<1500 IU/mL), thereby providing a new treatment strategy for CHB patients with high-level HBsAg to achieve clinical cure.

Keyword(s) : Chronic hepatitis B (CHB), Nucleos(t)ide analogs (NAs), Peginterferon alpha (PegIFN α), Clinical cure

1170 - Nurse led lifestyle modification intervention to improve self efficacy illness perception self management behaviours and liver steatosis among adults with metabolic dysfunction associated steatotic liver disease A pilot randomised controlled trial

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Background / **Hypothesis:**
Metabolic dysfunction associated steatotic liver disease (MASLD) is a growing global and local health burden linked to progressive liver injury, cardiovascular complications, and increasing healthcare costs. Despite clinical guidelines underscoring lifestyle modification as the cornerstone therapy, many patients struggle to adhere to recommended changes due to low self-efficacy, poor illness perception, inconsistent information, and limited structured support. Evidence-based, theory-informed, and patient-centred programme development is urgently needed, particularly in regions where structured interventions are lacking. This study hypothesised that a nurse-led lifestyle modification intervention, developed through systematic evidence appraisal and qualitative exploration, would improve self-efficacy, illness perception, self-management behaviours, and liver steatosis among adults with the disease.

Materials and **Methods:**
A three-phase study was undertaken. Evidence from a systematic review and a qualitative study informed the design of a nurse-led lifestyle modification intervention grounded in Bandura's self-efficacy theory. A single-

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centre, assessor-blinded pilot randomised controlled trial (RCT) was then conducted. Sixty-four adults with imaging-confirmed disease were randomised 1:1 to either the intervention group or a usual-care group. The three-month intervention included lifestyle counselling, behavioural strategies, structured education, goal setting, and psychological support delivered through face-to-face and telephone sessions. Outcomes were measured at baseline and week twelve. The primary outcome was self-efficacy, and the secondary outcomes were illness perception and self-management behaviours. The clinical outcome was steatosis grade. Feasibility and acceptability indicators included recruitment, retention, fidelity, attendance, and participant satisfaction. Repeated-measures analysis of variance was used to assess time-by-group effects.

Results:

In Phase III pilot RCT, 64 participants were recruited and demonstrated significant time-by-group interaction effects across self-efficacy, illness perceptions, self-management behaviours, and steatosis grade (all $p < .001$). High feasibility of the RCT was reflected by a recruitment rate of 76.4%, a retention rate of 98.4%, and session attendance exceeding 81%. Intervention acceptability was high, as evidenced by strong participant satisfaction ratings.

Conclusions:

The study demonstrated that a nurse-led lifestyle modification intervention is effective significantly improved self-efficacy, illness perceptions, self-management behaviours, and steatosis grade among MASLD patients, with high feasibility and acceptability. Developed from systematic evidence and stakeholder input, this intervention addresses critical gaps in MASLD management. These findings support the implementation of a full-scale RCT and underline the potential of nurse-led behavioural interventions in managing MASLD in Hong Kong and beyond.

Keyword(s) : MASLD, Metabolic dysfunction associated steatotic liver disease, lifestyle modification, nurse-led intervention, self-efficacy, CAP, steatosis

1172 - EMMPRIN Loss Promotes Fatty Acid Transport Protein FATP2 Lactylation in MAFLD via the MCTs lactate Axis

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Background / Hypothesis: Although metabolic-associated fatty liver disease (MAFLD) is a growing global health concern, its molecular mechanisms remain unclear. This study explores EMMPRIN-mediated metabolic reprogramming in hepatic steatosis.

Materials and Methods: The expression of hepatic EMMPRIN was examined in MAFLD murine models and steatotic cells models. Genetic manipulation of EMMPRIN, including both ablation and overexpression, was conducted in murine models to evaluate its impact on lipid accumulation and inflammation. Multi-omics techniques, including transcriptomics, 4D-FastDIA lacylproteomics, and co-immunoprecipitation, were employed to identify EMMPRIN's interaction partners and lactylation targets. Lipidomics was utilized to quantify changes in fatty acid profiles.

Results: EMMPRIN expression was found to be elevated in MAFLD, contributing to the development of steatohepatitis phenotypes. Its genetic ablation resulted in decreased lipid accumulation and inflammation, whereas its overexpression intensified the disease. Mechanistically, EMMPRIN was shown to interact with monocarboxylate transporters (MCT1/4), influencing lactate shuttling and intracellular lactate concentrations. The knockout of EMMPRIN resulted in the lactylation at lysine 174 (K174) of fatty acid transport protein 2 (FATP2), a modification mediated by CREBBP, thereby influencing subsequent lipid metabolic processes and preferentially directing activated fatty acids. Lipidomic analyses substantiated the role of FATP2 lactylation in determining hepatic fatty acid profiles.

Conclusions: The study findings demonstrated that EMMPRIN drove MAFLD progression through regulation of lactate shuttling and protein lactylation. The EMMPRIN-MCTs-FATP2 axis was identified as offering new therapeutic targets for fatty liver disease.

Keyword(s) : MAFLD; EMMPRIN; Lactylation; FATP2; Lipid metabolism; Fatty acid metabolism.

1173 - Unfolding Clinical Research Landscape on Bacterial Infections in Cirrhosis Trends Hotspots and Insights

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Background / Hypothesis: Bacterial infection is the most common complication in cirrhosis patients. This study evaluated the research hotspots and trends of bacterial infections in cirrhosis through bibliometric analysis, and provides guidance for future research on bacterial infection in cirrhosis.

Materials and Methods: We extracted publications on bacterial infections associated with cirrhosis from the Web of Science Core Collection, covering the period from January 2000 to May 2025. CiteSpace and VOSviewer were used for bibliometric analysis.

Results: Publications on bacterial infections in cirrhosis reflect an upward trend, with 69 countries/territories contributing to research, of which United States ranks first with 324 publications. The Journal of Hepatology is the prime publication on liver cirrhosis associated with bacterial infections, Fernandez Javier being the most-cited author. Cirrhosis with bacterial infection is the most popular research topic regarding mortality, infection, ascites and sepsis. The research hotspots of the future are systemic inflammation, acute-on-chronic liver failure and chronic liver failure.

Conclusions: The hotspots and trends of related research are discussed, and the research results are presented objectively through knowledge maps. The research directions in this field are analyzed to provide a reference for subsequent clinical research.

Keyword(s) : Cirrhosis, Bacterial infections, Bibliometric, Data visualization

1174 - A Ten Year Comparative Analysis of Open vs. Laparoscopic Anatomical Hepatectomy for Right Sided Complex Hepatocellular Carcinoma A Large Sample Nationwide Multicenter Real World Study

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Background / Hypothesis: The optimal surgical approach for complex right-sided hepatocellular carcinoma (HCC), particularly regarding long-term oncologic outcomes between laparoscopic anatomical hepatectomy (LAH) and open anatomical hepatectomy (OAH), remains undefined. This large-scale, multicenter real-world study aimed to compare the perioperative outcomes and long-term survival of LAH versus OAH for right-sided HCC across different tumor stages and anatomical locations.

Materials and Methods: In this nationwide retrospective cohort study, 1,914 patients who underwent curative-intent right-sided AH (Lap-group: 766; Open-group: 1,148) between March 2015 and May 2020 across five high-volume Chinese centers were analyzed. Patients were stratified by Barcelona Clinic Liver Cancer (BCLC) stage 0–A (n=1,342), B (n=309), and C (n=263). Baseline characteristics were balanced using inverse probability of treatment weighting (IPTW) and propensity score matching (PSM). Primary endpoints included overall survival (OS) and disease-free survival (DFS).

Results: Following IPTW adjustment, the Open-group demonstrated significantly longer median DFS in BCLC stage 0–A (47.0 vs. 45.0 months; HR=1.14, 95% CI: 1.01–1.30, P=0.02) and stage B (36.0 vs. 28.0 months; HR=1.45, 95% CI: 1.01–1.31, P<0.01). Conversely, in the stage C cohort, Lap-group achieved superior median OS (39.0 vs. 33.0 months; HR=0.56, 95% CI: 0.36–0.86,

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P<0.01). Subgroup analyses indicated that the DFS advantage of Open-group was confined to CNLC Ib/Ia stages and resections involving the right anterior section. Lap-group was associated with a lower bile leak rate in stage 0–A (3.73% vs. 7.44%, P=0.016) and a higher frequency of repeat resection after recurrence. Microvascular invasion (MVI) grade emerged as the strongest independent prognostic factor for both OS and DFS across all cohorts.

Conclusions:

The selection between LAH and OAH for right-sided HCC should be individualized based on tumor stage and location. OAH provides superior DFS in specific early–intermediate subgroups, whereas LAH confers favorable survival in selected advanced-stage cases, potentially owing to enhanced feasibility of salvage therapy.

Keyword(s) : Right-sided HCC, Laparoscopic anatomical hepatectomy, Inverse probability of treatment weighting, Real-World study

1176 - METTL16 drives MAFLD progression by regulating lipid synthesis and immune responses

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Background / **Hypothesis:** Methyltransferase-like 16 (METTL16) is a newly identified m6A RNA methyltransferase involved in RNA methylation and gene expression regulation. Although its oncogenic roles have been described in hepatocellular carcinoma, the contribution of METTL16 to metabolic dysfunction-associated fatty liver disease (MAFLD) remains unclear. This study aimed to elucidate the expression pattern, functional significance, and molecular mechanism of METTL16 in MAFLD. **Materials** and **Methods:** Methylation-related gene expression profiles were analyzed in liver tissues from 206 MAFLD patients and 10 healthy controls. Palmitic acid-induced cell models and diet-induced MAFLD mouse models were used to evaluate the biological functions of METTL16. Gene silencing was achieved using siRNA or AAV-mediated shRNA delivery. Hepatic steatosis, insulin resistance, and fibrosis were assessed by histological staining, biochemical assays, and immunoblotting. Bioinformatic analyses were conducted to examine immune cell infiltration, signaling pathways activation, and to predict potential transcriptional regulators and small-molecule inhibitors of METTL16. **Results:**

METTL16 expression was markedly upregulated in MAFLD livers and showed a strong correlation with lipid metabolism-related pathways. Knockdown of METTL16 significantly alleviated hepatic steatosis, insulin resistance, and fibrosis in high-fat diet-fed mice. Mechanistically, cell death-inducing DFF45 like effector family members A (CIDEA) was identified as a downstream mediator of METTL16-driven steatosis. Elevated hepatic METTL16 expression was associated with enhanced infiltration of innate immune cells, activation of immune-related pathways, and increased expression of pro-fibrotic genes. Database analyses further indicated that METTL16 might regulate immune-related gene expression at both transcriptional and post-transcriptional levels. Moreover, ERG1 and MYC were identified as potential upstream transcriptional regulators of METTL16, and eleven small molecules were predicted to bind to and inhibit METTL16 activity. **Conclusions:**

METTL16 promotes hepatic steatosis and immune-mediated fibrogenesis in MAFLD, providing novel mechanistic insights into m6A-dependent metabolic regulation. Targeting the METTL16-CIDEA axis or pharmacologically inhibiting METTL16 activity may represent a promising therapeutic strategy for MAFLD.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease; RNA methylation; Methyltransferase-like 16; Cell death-inducing DFF45 like effector family members A; Immune response

1179 - Association of Urinary Spot Sodium Potassium Ratio with Acute Kidney Injury in Decompensated Cirrhosis

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Background

Acute Kidney Injury (AKI) is a frequent and severe complication in patients with decompensated cirrhosis, contributing significantly to morbidity and mortality. Early recognition of renal dysfunction is crucial for timely intervention. The urinary sodium-to-potassium (Na/K) ratio is a simple, non-invasive, and readily available bedside marker reflecting renal sodium handling and circulatory function. This study aimed to evaluate the association between urinary spot Na/K ratio and AKI in patients with decompensated cirrhosis.

Materials and **Methods:** This cross-sectional observational study was conducted at the Department of General

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Medicine, Atal Bihari Vajpayee Institute of Medical Sciences and Dr. Ram Manohar Lohia Hospital, New Delhi, from February 2024 to June 2025. A total of 122 adult patients with decompensated cirrhosis were enrolled. Clinical, biochemical, and urinary parameters were assessed, and the urinary Na/K ratio was calculated. AKI was defined using ICA-AKI/KDIGO criteria. Receiver Operating Characteristic (ROC) curve analysis was performed to determine the diagnostic performance of urinary Na/K ratio in predicting AKI.

Results:

Of 122 patients, 61 (50%) developed AKI. The mean urinary Na/K ratio was significantly lower in patients with AKI compared to those without. A cut-off value of ≤ 0.945 predicted AKI with 45.9% sensitivity and 95.1% specificity (AUC = 0.770). The ratio showed a strong correlation with serum creatinine rise ($\Phi = 1.000$). Lower Na/K ratios were also associated with severe ascites, higher bilirubin, hyponatremia, and advanced hepatic encephalopathy.

Conclusions: The urinary spot sodium-to-potassium ratio is a reliable, rapid, and non-invasive biomarker for early identification of AKI in decompensated cirrhosis. Incorporating this marker into routine evaluation may help clinicians identify high-risk patients earlier, allowing timely intervention and improved clinical outcomes.

Keyword(s) : Decompensated cirrhosis, Acute kidney injury, Urinary sodium-potassium ratio,

1185 - Perioperative Management Ethical Concerns and Outcomes of Pediatric Domino Liver Transplants from Maple Syrup Urine Disease Donors

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Background / Hypothesis: Domino liver transplantation (DLT) expands the donor pool by using structurally normal livers from patients with metabolic disorders like Maple Syrup Urine Disease (MSUD). This study presents our pediatric DLT experience, focusing on ethical issues, family perspectives, perioperative care, and outcomes.

Materials and Methods: This retrospective study included all children who underwent DLT between January 2019 and September 2025, building on our earlier work (Kumar et al., 2023) with a larger cohort and extended follow-up. Each child with MSUD donated to a recipient with end-stage liver disease or another metabolic disorder. Families received detailed counselling on the procedure, ethical considerations, and social implications before giving informed consent. All liver transplants (LT) were approved by the Transplant Board and Ethics Committee.

Results: Eighteen MSUD patients underwent living donor LT, and their explanted livers (domino allografts) were transplanted into 20 recipients. Two older children with MSUD underwent split LT, benefiting four recipients. Half of the DLT recipients' families initially feared the potential for new genetic conditions but consented after thorough counselling. All three surgeries in the DLT were performed simultaneously through coordinated teamwork, with three synchronized surgical teams carrying out liver extractions in parallel; the MSUD donor hepatectomy was straightforward. The MSUD children followed a branched-chain amino acid restricted diet pre-transplant and an unrestricted normal diet post-transplant, with normalized amino acid levels and no symptoms. The post-LT ICU and hospital stay durations were 12 (8–16) days and 34 (22–51) days, respectively. Patient survival among DLT recipients was 65% at a mean follow-up of 25 (15–31) months. No long-term vascular, biliary, graft-related complications, or MSUD occurrence were seen in DLT recipients, and there was no mortality in the MSUD donor cohort (Table attached).

Conclusions: MSUD livers are safe for transplant and should be offered with proper counselling. A multidisciplinary approach supports long-term success and helps address organ shortages.

Keyword(s) : Domino liver transplant, Maple syrup urine disease, Living donor

1189 - VISTA deficiency exacerbates the development of acute on chronic liver failure by promoting Tc1 Differentiation

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) has a high early mortality, and there is abundant evidence that persistent inflammatory response and immune activation are the core pathogenesis of ACLF. However, the mechanism of immune disorder has not been fully clarified. Previous studies have shown that deficiency of V-type immunoglobulin domain-containing suppressor of T-cell activation (VISTA) exacerbates the progression of inflammatory diseases. Moreover, the immune response mediated by CD8⁺T cells can directly cause liver damage and is closely related to the dysregulation of inflammatory response in ACLF patients. Our study was designed to investigate the role of VISTA expression on CD8⁺T cells in the pathogenesis of ACLF.

Materials and Methods: We assessed the expression and function of VISTA on CD8⁺ T cells in blood samples from healthy controls, patients with stable cirrhosis, and ACLF patients. Transcriptome sequencing was performed to determine the relevant pathways, which was then verified by WB. Inhibitors of relevant signaling pathways were added to the CD8⁺ T cells to explore the key signal mechanism.

Results: In ACLF patients, the expression of VISTA was significantly reduced in the CD8⁺ T cells compared with healthy subjects and stable cirrhosis patients, especially in the effector memory and terminally differentiated effector memory cells with high CD45RA expression subsets. The expression of VISTA on CD8⁺ T cells was associated with disease severity and prognosis. VISTA downregulation contributed to the activation and proliferation of CD8⁺ T cells, and enhanced the differentiation of type 1 cytotoxic T cells (Tc1), as well as heightened inflammatory cytokine secretion. This effect was mediated through the activation of the extracellular signal-regulated kinase 1 and 2 (ERK1/2) signaling pathway.

Conclusions: Our study reveals that VISTA downregulation drives ACLF progression by amplifying Tc1-mediated inflammation. This insight not only advances the mechanistic understanding of ACLF pathogenesis but also nominates VISTA as a potential candidate for therapeutic intervention.

Keyword(s) : acute-on-chronic liver failure, immune checkpoint, V-type immunoglobulin domain-containing

suppressor of T-cell activation, type 1 cytotoxic T cells, inflammation, prognosis

1190 - Macrophage Nrf1 Foxo1 KLF6 Axis Drives Mitochondrial Dysfunction and Inflammatory Progression in Liver Fibrosis

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Background / Hypothesis: Liver fibrosis is a progressive condition driven by chronic injury, ultimately leading to cirrhosis. Immune dysregulation, particularly macrophage polarization, plays a crucial role in fibrosis progression. Pro-inflammatory M1 macrophages worsen inflammation and fibrosis, with polarization influenced by metabolic shifts between glycolysis and oxidative phosphorylation. Nuclear factor erythroid 2-related factor 1 (Nrf1), a key redox-sensitive factor essential for mitochondrial homeostasis, has been extensively studied in hepatocytes but remains largely unexplored in macrophages in the context of liver fibrosis. This study aims to elucidate the immunoregulatory role of macrophage Nrf1 in liver fibrosis, potentially identifying novel therapeutic targets to mitigate or reverse fibrosis progression.

Materials and Methods: Myeloid-specific Nrf1-knockout (Nrf1 M-KO) mice and Foxo1 knockout (Foxo1M-KO) mice were developed to investigate the role and underlying mechanisms of macrophage Nrf1-Foxo1 axis across three murine models of liver fibrosis induced by a high-fat diet, carbon tetrachloride injection or bile duct ligation. Bone marrow-derived macrophages (BMDMs) were isolated from conditional mice, then stimulated with lipopolysaccharide (LPS, 100 ng/mL, 6 hours). CRISPR/Cas9 and siRNA approaches were used to assess downstream targets.

Results: Nrf1 expression was significantly reduced in macrophages from fibrotic liver tissues, as confirmed by qRT-PCR and immunofluorescence staining in both human and murine models. Nrf1M-KO mice exhibited exacerbated liver fibrosis, characterized by elevated serum ALT/AST levels, increased collagen deposition, and upregulated fibrotic markers (TGF- β , Col1a1, TIMP1). Loss of Nrf1 promoted M1 macrophage polarization, enhanced inflammatory responses (elevated TNF- α , IL-1 β , iNOS), and impaired

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mitochondrial function (reduced ATP production and increased mitochondrial ROS). In contrast, Foxo1M-KO mice exhibited attenuated fibrosis, reduced inflammation, and improved mitochondrial integrity. Mechanistically, Nrf1 directly interacted with Foxo1 to regulate the transcription of KLF6, a key mediator of mitochondrial function and macrophage polarization. These findings underscore the pivotal role of the Nrf1-Foxo1-KLF6 axis in macrophage-driven inflammation and fibrosis, highlighting potential therapeutic targets for liver fibrosis.

Conclusions:

Our study highlights the functional properties of macrophage Nrf1-Foxo1 axis in controlling mitochondrial reprogramming and liver fibrosis progression.

Keyword(s) : Liver Fibrosis, Macrophage, Nrf1, Mitochondrial Dysfunction, Inflammatory

1195 - MMP 9 Suppresses the Anti HBsAg B Cell Response During Chronic HBV Infection

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Background / Hypothesis: Chronic Hepatitis B (CHB) infection poses a significant threat to global health, and current treatment regimens are insufficient for achieving a cure for the disease. Virus-specific T-cell and B-cell responses are crucial in the clearance of Hepatitis B Virus (HBV) infection. Our previous research indicated that Matrix Metalloproteinase (MMP)-9, together with MMP-2, are involved in the regulation of intrahepatic anti-HBV effector T cell responses in acute self-limiting HBV infection. However, it remains unclear if MMP-9 also plays a role in regulating anti-HBV immune responses and mediating HBV clearance in the scenario of chronic HBV infection.

Materials and Methods: We addressed this question by employing MMP-9 gene knockout (KO) and RNA interference techniques in chronic HBV replication mouse models.

Results: Our results showed that the absence of MMP-9 in FVB mice led to significantly accelerated clearance of

Hepatitis B surface antigen (HBsAg) in peripheral blood and increased production of Hepatitis B surface antibody (HBsAb) in pAAV-HBV1.2 or BPS plasmids hydrodynamic injection (HDI) mouse models. Adoptive transfer of spleen immune cells from MMP-9 KO mice to wild-type mice could also accelerate serum HBsAg clearance and enhance HBsAb production in the pAAV-HBV1.2 HDI mouse model, suggesting that the anti-HBV effect of MMP-9 KO is mainly mediated by immune cells. RNA-seq analysis showed that MMP-9 KO significantly induced enrichment of genes related to the B-cell receptor signaling pathway in the splenocytes of mice post pAAV-HBV1.2 HDI compared to wild-type mice. MMP-9 KO mice also showed significantly enhanced B cell activation, as well as increased numbers of germinal B cells and follicular helper CD4+ T cells infiltration in the liver post pAAV-HBV1.2 HDI compared to wild-type mice. Importantly, in vivo knockdown of MMP-9 expression by RNAi also resulted in significantly accelerated serum HBsAg clearance in wild-type FVB and C57BL/6 mice post pAAV-HBV1.2 HDI.

Conclusions: MMP-9 may play a role in negatively regulating anti-HBV B cell responses during chronic HBV infection. Targeting MMP-9 could serve as a novel strategy for the treatment of chronic HBV infection.

Keyword(s) : Chronic Hepatitis B, Matrix Metalloproteinase-9, Adaptive Immune Response, B cells, Follicular Helper T cells

1196 - Prevalence and severity of chronic atrophic gastritis in cirrhotic patients versus dyspeptic controls

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Background / Hypothesis: Chronic atrophic gastritis represents a crucial step in the Correa cascade leading to gastric malignancy. In patients with cirrhosis, premalignant gastric lesions are reported in nearly one-third of cases, independent of portal hypertension severity. Cirrhosis is thought to increase the risk of chronic atrophic gastritis by promoting gastric mucosal injury and inflammation. This study aimed to compare the frequency of chronic atrophic gastritis in individuals with cirrhosis versus a dyspeptic control population.

Materials and Methods:

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A total of 115 patients with cirrhosis (mean age: 54.6 ± 13.4 years) and 510 patients with dyspeptic symptoms (mean age: 55.8 ± 14.6 years) were included. Demographics, comorbidities, endoscopic findings, and histopathological biopsy results were assessed. The presence and severity of chronic atrophic gastritis as well as *Helicobacter pylori* status were recorded. Gender distribution was comparable between group

Results:

The prevalence of chronic atrophic gastritis was significantly higher in cirrhotic patients (51.3%) compared with controls (31.7%, $p < 0.001$). The severity of atrophy was also significantly greater in the cirrhosis group. *Helicobacter pylori* positivity was lower in patients with cirrhosis (17.3%) compared with controls (26.7%, $p < 0.05$).

Conclusions:

Patients with cirrhosis exhibit a significantly higher frequency and greater severity of chronic atrophic gastritis compared with individuals presenting with dyspepsia. These findings reinforce the association between cirrhosis, premalignant gastric mucosal changes, and increased gastric cancer risk. The presence of more severe and frequent chronic atrophic gastritis highlights its role as a potential key factor in gastric carcinogenesis in cirrhosis. Routine endoscopic surveillance with targeted biopsy sampling should therefore be considered an essential preventive strategy in this high-risk patient population.

Keyword(s) : premalignant gastric lesions, chronic atrophic gastritis, cirrhosis

1197 - Enhancement of Liver Regeneration in Mice through DDX3X CD36 Pathway Mediated Promotion of Hepatic Lipid Accumulation

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Background / Hypothesis:

The liver possesses a unique regenerative capacity, which is indispensable for the recovery process following liver resection and the management of various liver diseases. Accumulation of lipids in hepatocytes has been recognized as a pivotal factor during liver regeneration, yet the intricate mechanisms underlying this process remain elusive. This study endeavors to elucidate the mechanism through which DDX3X mediates lipid accumulation during liver regeneration.

Materials and Methods:

We collected serum samples from patients at various time points post-hepatectomy. A liver regeneration model in C57BL/6 mice was established by performing a two-thirds partial hepatectomy (PH). Hepatocyte-specific DDX3X knockout (DDX3X Δ hep) mice were generated using CRISPR/Cas9/Cre-LoxP technology to investigate the pivotal role of DDX3X in liver regeneration. High-throughput RNA sequencing was conducted to dissect the downstream signaling pathways in DDX3X Δ hep mice during liver regeneration. Oil Red O staining was utilized to visualize liver lipid accumulation. qPCR and Western blotting were employed to assess the expression levels of target genes and proteins, respectively.

Results:

Patients undergoing PH exhibited a progressive decrease in serum DDX3X levels. In the mouse liver regeneration model, hepatic DDX3X expression initially surged (0-12 hours) and subsequently declined (24-96 hours). Following two-thirds PH, DDX3X Δ hep mice demonstrated a significantly augmented regenerative capacity compared to controls, accompanied by a marked increase in liver lipid levels. RNA sequencing revealed enhanced fatty acid uptake in DDX3X Δ hep mice during liver regeneration. Notably, knockdown of the key fatty acid receptor CD36 significantly reduced hepatocyte lipid accumulation and impeded liver regeneration in DDX3X Δ hep mice.

Conclusions:

During liver regeneration, hepatocyte DDX3X deficiency promotes hepatocyte proliferation by augmenting liver lipid accumulation through CD36 regulation. Our findings offer novel insights into the mechanisms underlying liver regeneration and identify potential targets for accelerating post-surgical recovery in patients.

Keyword(s) : liver regeneration, lipid accumulation, DDX3X, CD36

1201 - Outcomes of Living Donor Liver Transplantation in Patients with or without Acute on Chronic Liver Failure A Single Centre Experience

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) is a life-threatening syndrome in patients with chronic liver disease (CLD), marked by multi-organ failure and high mortality. Living donor liver transplantation (LDLT) is critical in donor-scarce regions. This study compares baseline characteristics, perioperative complications, and long-term survival in recipients with versus without ACLF, focusing on etiology, ACLF grading, and graft factors

Materials and Methods: Data were derived from a prospective local registry with 591 adult LDLT recipients (2018–2023). ACLF was defined using EASL-CLIF (multi-organ failure, grades 1–3) and APASL (jaundice/coagulopathy with complications) criteria, assessed at initial liver transplant evaluation and within 24 hours of LDLT

Results: Patients with ACLF (n=101, 17.1%) had higher MELD-Na (27 vs. 20, $p<0.001$), bilirubin (6.84 vs. 1.75 mg/dL, $p<0.001$), creatinine (108 vs. 70.5 $\mu\text{mol/L}$, $p<0.001$), metabolic/genetic etiologies (9.9% vs. 2.8%, $p=0.001$), chronic kidney disease (23.7% vs. 8.1%, $p<0.001$), and lower Hepatocellular carcinoma (HCC) prevalence (11.8% vs. 29.6%, $p<0.001$). Graft-to-recipient weight ratio (GRWR) was lower in ACLF (0.59 vs. 0.66, $p=0.10$). Patients with ACLF had higher rates of infection (27.7% vs. 10.4%, $p<0.001$), bleeding (14.9% vs. 6.3%, $p=0.004$), and biliary complications (15.8% vs. 7.8%, $p=0.010$). They had longer intensive care unit (5 vs. 3 days, $p<0.001$) and hospital stays (33.66 vs. 20.7 days, $p=0.036$). At 5-year follow-up, overall survival was lower in ACLF (log-rank $p=0.001$), and worse in higher ACLF grades (EASL-CLIF grade 3: 55% vs. 86% for no ACLF, $p=0.002$). Graft survival was lower in ACLF (75% vs. 85%, $p=0.02$). In multivariable analysis, chronic kidney disease independently predicted mortality (HR 2.09, 95% CI 1.11–3.95, $p=0.023$).

Conclusions: LDLT in ACLF incurs significant perioperative morbidity and reduced long-term survival compared to non-ACLF, with outcomes declining sharply with ACLF severity. Chronic kidney disease is a key independent predictor of mortality. Timely LDLT remains a life-saving option in donor-scarce settings.

Keyword(s):

1204 - Sequential therapy with anti PD 1 antibody and pegylated interferon alpha promoted HBsAg reduction and enhanced T cell response in patients with chronic hepatitis B

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Background: This study aimed to investigate the safety, efficacy and immunological mechanisms of sequential therapy with anti-PD-1 antibody ($\alpha\text{PD-1}$) and pegylated interferon-alpha (Peg-IFN α) in patients with chronic hepatitis B (CHB).

Methods: In this non-randomized, prospective, open-label, ongoing study, hepatitis B e antigen (HBeAg)-negative virally suppressed patients with hepatitis B surface antigen (HBsAg) levels between 100 and 600 IU/mL were assigned to one of the following regimens: nucleos(t)ide analogue (NA) monotherapy for 48 weeks (NA group); NA plus Peg-IFN α for 48 weeks (NA+Peg-IFN α group); NA plus $\alpha\text{PD-1}$ for 24 weeks followed by NA alone for 24 weeks (NA+ $\alpha\text{PD-1}$ group); or NA plus $\alpha\text{PD-1}$ for 24 weeks followed by NA plus Peg-IFN α for 24 weeks (NA+ $\alpha\text{PD-1}$ /Peg-IFN α group). $\alpha\text{PD-1}$ (sintilimab, 100mg) was administered intravenously every 3 weeks.

Results: A total of 62, 26, 25, and 10 patients in the NA, NA+Peg-IFN α , NA+ $\alpha\text{PD-1}$, and NA+ $\alpha\text{PD-1}$ /Peg-IFN α groups, respectively, completed the 48-week follow-up. The incidences of overall adverse events (AEs) (100.0% vs. 100.0%) and severe AEs (SAEs) (30.8% vs. 30.0%) were comparable between the NA+Peg-IFN α and NA+ $\alpha\text{PD-1}$ /Peg-IFN α groups. In contrast, the NA+ $\alpha\text{PD-1}$ group exhibited numerically lower rates of AEs (88.0%) and SAEs (16.0%), though these differences were not statistically significant ($P>0.05$). Baseline HBsAg levels were comparable across all groups ($P=0.906$). At week 24, the mean changes in HBsAg from baseline were -0.03, -1.35, -1.01 and -0.66 log₁₀ IU/mL in the NA, NA+Peg-IFN α , NA+ $\alpha\text{PD-1}$ and NA+ $\alpha\text{PD-1}$ /Peg-IFN α groups, respectively. By week 48, the greatest mean HBsAg reduction was observed in the NA+ $\alpha\text{PD-1}$ /Peg-IFN α group (-3.08 log₁₀ IU/mL), followed by the NA+Peg-IFN α (-1.68 log₁₀ IU/mL), NA+ $\alpha\text{PD-1}$ (-0.96 log₁₀ IU/mL) and NA (-0.09 log₁₀ IU/mL) groups. Furthermore, at week 48, NA+ $\alpha\text{PD-1}$ /Peg-IFN α group also achieved the highest HBsAg loss rates (50.0%), significantly exceeding those in the NA (0%), NA+Peg-

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IFN α (23.1%), and NA+ α PD-1 (16.0%) groups ($P < 0.001$). During α PD-1 treatment, T cell activation and enhanced HBV-specific T cell responses were observed ($P < 0.05$). After sequential Peg-IFN α therapy, HBV-specific IFN- γ spot counts decreased, whereas CD38 expression on T cells continued to elevate ($P < 0.05$), and the Th2 cell frequencies showed an increasing trend. Conclusions: In a 48-week treatment course, sequential therapy with α PD-1 and Peg-IFN α showed an acceptable safety profile, and appeared to induce stronger HBsAg reductions and higher HBsAg loss rates than either agent alone in NA-treated CHB patients. These findings provide new insights into achieving CHB functional cure.

Keyword(s) : HBV, HBsAg, PD-1, Peg-IFN α , T cell

1209 - Therapeutic Effects of Qinggan Huatan Huoxue Prescription on Metabolic Dysfunction Associated Steatohepatitis via Modulation of the CYP4a32-PPAR γ -CD36 Signaling Axis

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Background / Hypothesis:
Therapeutic Effects of Qinggan Huatan Huoxue Prescription on Metabolic Dysfunction-Associated Steatohepatitis via Modulation of the CYP4a32-PPAR γ -CD36 Signaling Axis

Materials and Methods:
Twenty-five male mice fed a high-fat diet were randomly assigned to five groups: normal control, model, positive control, low-dose QHHP, and high-dose QHHP. Mice received QHHP, atorvastatin, or saline for four consecutive weeks. Liver lipid accumulation and inflammatory infiltration were evaluated by hematoxylin-eosin (HE) staining, lipid content was quantified by Oil Red O staining, and liver fibrosis was assessed using Masson's trichrome staining. Molecular docking and molecular dynamics simulations were performed to evaluate the binding affinity and stability of QHHP active compounds with the CYP4a32-PPAR α -CD36 signaling axis. The expression of CYP4a32, PPAR α , and CD36 was assessed by immunohistochemistry, immunofluorescence, qPCR, and Western blotting.

Results:

QHHP treatment improved hepatic lipid accumulation and inflammation in mice, accompanied by improved liver function and serum lipid profiles in both mice and human subjects. In vitro, Nile Red immunofluorescence in Huh7 and AML12 cells confirmed the therapeutic effect of QHHP on MASH. RNA-seq analysis of mouse livers revealed enrichment in the PPAR γ signaling pathway, and PCR and Western blot assays demonstrated increased hepatic expression of Cyp4a32 and CD36 and decreased expression of PPAR γ . Mass spectrometry identified 10 active compounds in QHHP, and network pharmacology, molecular docking, and molecular dynamics simulations suggested interactions between these compounds and the targets Cyp4a32, PPAR γ , and CD36. Further in vitro experiments validated that QHHP exerts its therapeutic effects on MASH through modulation of the Cyp4a32-PPAR γ -CD36 axis.

Conclusions:

QHHP ameliorates hepatic lipid metabolism and inflammatory responses in MASH through modulation of the CYP4a32-PPAR γ -CD36 signaling axis. These findings provide mechanistic evidence supporting the multitarget therapeutic potential of TCM prescriptions in the management of MASH.

Keyword(s) :

1215 - Oxysterol Binding Protein Related Protein 2 Drives Sterol Accumulation and Neutrophil Dysfunction in Sepsis Associated with Alcoholic Cirrhosis

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Background / Hypothesis: Patients with alcoholic cirrhosis often develop sepsis with high mortality. Neutrophil dysfunction, marked by impaired pathogen clearance, excessive inflammation, and metabolic dysregulation, is common, yet its underlying mechanisms remain poorly understood.

Materials and Methods: Untargeted proteomic and metabolomic profiling were performed on neutrophils from healthy controls, non-septic alcoholic cirrhosis, and septic/septic shock patients. A total of 185 patients were randomized (70:30) into training (n=150) and test (n=35) cohorts. Proteins and metabolites were quantified using LC-MS/MS and UHPLC-HRMS, and

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pathway enrichment, fold change, t-tests, and AUC analyses, along with neutrophil functionality analysis.

Results: Multi-omics analysis revealed significant alterations in neutrophil function and metabolism in cirrhotic sepsis. A total of 9,115 proteins and 1,414 metabolites were identified, with 6,488 proteins ($FC > 1.5$, $p < 0.05$) and 729 metabolites ($FC > 1.5$, $p < 0.05$) significantly dysregulated compared to non-septic cirrhosis. Pathway enrichment analysis highlighted dysregulation in lipid metabolism, sterol homeostasis, and mitochondrial function. Key findings include the upregulation of OSBPL2 ($FC > 3892$, $AUC = 0.93$) and LDLR ($FC > 4.47$, $AUC = 0.92$), which promote increased uptake of fats, contributing to sterol accumulation. Elevated levels of cholesterol ($FC > 6$), 25-hydroxycholesterol ($FC > 500$), and cortisone ($FC > 25$) confirmed this metabolic shift.

Sterol overload induced lipid droplet formation, reprogramming neutrophils into lipid-laden cells. The increased lipid uptake caused by OSBPL2 and LDLR led to membrane instability, increased cell lifespan, and disrupted neutrophil functions. This metabolic shift triggered mitochondrial and ER stress. Mitochondrial dysfunction, marked by increased expression of THEM4 ($FC > 5$) and HIF1A ($FC > 7$), indicated impaired oxidative phosphorylation and elevated ROS production. ER stress markers, including HSPA1A ($FC > 3$) and AMFR ($FC > 189$), reflected disrupted protein folding.

These structural disruptions resulted in defective neutrophil degranulation, with altered exocytosis proteins such as KLC1 ($FC > 59$) and ITGB2 ($FC > 11$), indicating secretory exhaustion. Neutrophils also showed impaired phagocytosis and compromised antimicrobial response, despite elevated pro-inflammatory cytokines like IL6 ($FC > 29$) and ICAM1 ($FC > 6$). Furthermore, neutrophils in sepsis exhibited impaired chemotaxis and migration, as indicated by reduced CXCR2 ($FC > 2.5$) and ITGB2 ($FC > 11$), reflecting a diminished ability to respond to inflammation.

Conclusions- This study shows metabolic and functional reprogramming of neutrophils in cirrhotic sepsis, driven by sterol accumulation and organelle stress. Lipid buildup and mitochondrial dysfunction impair neutrophil functions, including degranulation, phagocytosis, and antimicrobial responses. Enhanced NETosis and dysfunctional chemotaxis worsen immune dysregulation. Biomarkers like OSBPL2 and LDLR offer potential targets for diagnostic and therapeutic

interventions, improving patient

Keyword(s) : Sepsis, Alcoholic Cirrhosis, Neutrophils, Proteomics, Metabolomics, Lipid Metabolism, Cholesterol, Biomarkers, Mitochondrial Stress, Immune Dysfunction.

1226 - Metabolic dysfunction associated steatotic liver disease by fibrosis stage in China and Japan a 20 year model

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Background / **Hypothesis:**

As metabolic dysfunction-associated steatotic liver disease (MASLD) continues to grow worldwide, China and Japan are similarly impacted. MASLD is linked to increased prevalence of cirrhosis, hepatocellular carcinoma, and a myriad of other life-threatening complications. Documenting the current and projected number of individuals with MASLD by fibrosis stage in the Chinese and Japanese adult populations provides an understanding how MASLD may evolve over time, which is vital to counteracting the burden of the disease.

Materials and **Methods:**

A Markov model was used to estimate the current and projected number of individuals with MASLD by fibrosis stage in the adult populations of China and Japan. The model incorporated prevalence, natural history, and mortality. Published estimates were used to build and validate the model projections, including the prevalence of MASLD, obesity, diabetes, and liver transplantations specific to each country.

Results:

The model projected total cases in China increasing from 400,903,100 to 429,100,700 (7%) in the next ten years, to 412,929,300 cases in 2045. F2-F3 cases, however, increased from 17,412,900 to 30,051,600 (73%) from 2025 to 2045. Further, F4 cases more than doubled from 1,572,300 to 3,484,300, and advanced liver disease cases, which includes decompensated cirrhosis, hepatocellular carcinoma, and liver transplantation, simultaneously increased by 128% from 209,200 to 477,900.

Japanese total cases declined by 15% in the twenty-year period from 26,669,100 in 2025 to 22,612,300 in 2045. This can be attributed to the country's slowing of obesity rates which directly influences MASLD incidence, as well as the falling population numbers. Yet, as seen in

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China, the danger of higher fibrosis stages burgeoning persisted. In ten years, F2-F3 cases increased from 1,993,100 to 2,071,900 (4%). In twenty years, F4 cases increased from 245,100 to 287,000 (17%), and advanced liver disease cases increased from 35,300 to 42,700 (21%).

Conclusions:

The use of modeling provides insights into the current and future disease burden of MASLD in China and Japan and establishes a framework for public health discussion to facilitate best policies and practices for optimal management of MASLD. The effects of later stages of MASLD rising appear to be perspicacious, posing morbid long-term outcomes. Effective therapies halting the progression of fibrosis will markedly benefit the rising disease burden of later stages of MASLD in China and Japan.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease, disease burden, fibrosis stage, Markov model, China, Japan

1229 - PD 1 is required for the early expansion of HBV specific CD8+ T cells and does not suppress their effector function during the acute phase of HBV infection

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Background / **Hypothesis:**
Programmed cell Death Protein 1(PD-1) is one of the key inhibitory receptors that regulates CD8 T cell exhaustion during chronic viral infection. However, the role of PD-1 in modulating effector T cell differentiation and function during acute hepatitis B virus (HBV) infection is remains poorly defined.

Materials and **Methods:**
We analyzed PD-1⁺ and PD-1⁻ CD8⁺ T cell dynamics in

acute self-resolving (AR) and chronic (CH) HBV mouse models using flow cytometry and RNA sequencing. Wild-type (WT) or PD-1 knockout (KO) HBV core antigen-specific TCR transgenic (C93-TCRtg) CD8⁺ T cells were adoptively transferred into naïve mice prior to HBV challenge.

Results:

Acute HBV infection induced sustained PD-1 upregulation on intrahepatic/splenic CD8⁺ T cells, predominantly exhibiting an effector phenotype. PD-1⁺ cells displayed enhanced proliferation, cytotoxicity, and effector cytokine production compared to PD-1⁻ counterparts. Transfer of PD-1 KO C93-TCRtg CD8⁺ T cells resulted in significantly impaired early expansion in both spleen and liver during acute infection, alongside reduced granzyme B expression but unaffected IFN- γ , TNF- α , or IL-2 production. Transcriptomics revealed PD-1 deficiency promoted apoptosis gene signatures. In chronic HBV infection, PD-1 KO cells failed to expand long-term and exhibited exacerbated exhaustion (loss of IFN- γ production).

Conclusions:

PD-1 is essential for the early expansion and survival of HBV-specific CD8⁺ T cells during acute infection and does not suppress their effector cytokine function. Its absence impairs proliferation and promotes exhaustion of HBV-specific CD8⁺ T cell in chronic settings.

Keyword(s) : PD-1, hepatitis B virus, CD8 T cell exhaustion.

1232 - Preoperative Viral Hepatitis Screening Evaluation of the Consultation Chain

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Background / Hypothesis: Chronic hepatitis B virus (HBV) and hepatitis C virus (HCV) infections are often asymptomatic, and delayed diagnosis may adversely impact clinical outcomes. Preoperative screening with HBsAg and anti-HCV in surgical patients offers an opportunity to assess occupational risk for healthcare workers and identify undiagnosed cases requiring follow-up. However, referral of patients with positive results to infectious diseases specialists is inconsistent. This study evaluates referral practices for patients screened positive in the orthopedics clinic and examines surgical team awareness on the issue.



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Materials and Methods: This cross-sectional descriptive study was conducted by reviewing the hospital information system records of patients who presented to the orthopedics outpatient clinic between July 1, 2024, and October 1, 2025, and underwent HBsAg and anti-HCV testing as part of the preoperative assessment. For patients with positive results, referral to infectious diseases or gastroenterology clinics, receipt of antiviral therapy, and history of prior chronic hepatitis follow-up were recorded. Additionally, a structured 14-item anonymous questionnaire was administered to 81 orthopedic physicians to evaluate the perceived purpose of preoperative screening, referral practices, and clinical attitudes. Data were analyzed using descriptive statistical methods.

Results: A total of 20,690 patients were screened; HBsAg positivity was detected in 161 (0.78%) and anti-HCV positivity in 38 (0.18%) patients. Among HBsAg-positive individuals, 17 were already under follow-up and 7 were receiving antiviral therapy, while 144 patients (89.4%) were identified as newly positive. Of these, 34% were referred to infectious diseases or gastroenterology clinics. Among anti-HCV-positive patients, 32 were newly diagnosed, and 37.5% of them were referred for specialist follow-up (Table 1).

Of the 81 surveyed physicians, 68% reported having received three doses of the HBV vaccine, and 70.4% stated that their anti-HBs level was ≥ 10 IU/L. Preoperative screening was reported to be routinely ordered in 91.3% of cases. Referral frequency varied; 33.3% of physicians reported always referring positive patients, whereas 27.2% reported frequently doing so. The proportion of physicians who reported feeling uneasy about operating on HBsAg-positive patients was 39.5%, compared to 74.1% for anti-HCV-positive patients. Surgical postponement due to hepatitis positivity was low (7.4%) (Table 2).

Conclusions: Although new hepatitis diagnoses are infrequent in preoperative screening, low referral rates suggest a missed opportunity for linkage to care. Despite high vaccination coverage, disparities exist in physician attitudes and referral behaviors, especially toward HCV. These findings underscore the need for better education on hepatitis management and standardized referral protocols to improve care continuity.

Keyword(s) : Preoperative screening, Hepatitis B virus, Hepatitis C virus, HBsAg, anti-HCV, referral practices, surgical awareness

1233 - Predictors of response to ursodeoxycholic acid treatment and non invasive identification of autoimmune hepatitis tendencies in primary biliary cholangitis

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Background / Hypothesis: Up to 40% of primary biliary cholangitis (PBC) patients may have a poor response to ursodeoxycholic acid (UDCA) treatment, and those with concurrent autoimmune hepatitis (AIH) tendencies require combined treatment with glucocorticoid/immunosuppressive drugs. Therefore, early diagnosis, assessment of concurrent AIH tendencies, and standardized treatment of PBC patients are particularly important for their prognosis.

Materials and Methods: A total of 154 PBC patients who underwent liver biopsy and received at least one year of UDCA monotherapy were included. The subjects were divided into the UDCA well-response group and the poor-response group based on treatment response. Univariate and multivariate logistic regression were used to evaluate the predictive value of clinical and pathological indicators for treatment response. Kaplan-Meier univariate analysis and Cox proportional hazards regression model were constructed to screen the independent predictors of patient survival prognosis. The included PBC patients were further divided into two groups based on the presence or absence of AIH tendency. Univariate and multivariate logistic regression were used to evaluate the predictive diagnostic value of hematological indicators for PBC with AIH tendency.

Results: Compared with the UDCA well-response group, the baseline ALP, GGT, AST, TBIL, IgG, GLB and APRI in the poor-response group were significantly higher. In terms of liver pathological manifestations, the proportion of patients with III-IV stage fibrosis, moderate to severe interface hepatitis, hepatocyte bile stasis, bile duct absence, and pseudolobule formation in the poor-response group was significantly higher than that in the well-response group. The results of logistic univariate regression and multivariate regression model analysis showed that baseline ALP, AMA-M2, GLB, and bile duct absence were independent risk factors affecting the response to UDCA. The multivariate COX regression model showed that UDCA treatment poor response, higher APRI, hepatocyte bile stasis, and III-IV stage fibrosis were independent risk factors for the poor prognosis of PBC patients. Univariate logistic regression analysis and multivariate logistic regression analysis

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showed that serum globulin, AST, total bilirubin, and IgG were important predictors of the AIH tendency in PBC patients.

Conclusions: PBC patients with severe hepatocyte cholestasis or bile duct absence had a lower response rate to UDCA treatment. The degree of liver fibrosis is a key factor affecting the prognosis of UDCA treatment. PBC patients with poor UDCA treatment response are more likely to have AIH tendencies. This study also identified non-invasive hematological indicators for predicting the AIH tendency of PBC patients, this will be helpful in guiding the proper treatment of these patients.

Keyword(s) : Primary biliary cholangitis, liver tissue pathology, ursodeoxycholic acid, AIH tendency, prognosis

1237 - Novel Trends in Liver Transplantation for MASLD and ALD Emerging Epidemiology Allocation & Post Transplant Challenges

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Background / Hypothesis:
The prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as nonalcoholic fatty liver disease (NAFLD), has increased worldwide. By 2019, 38% of adults in North America or Australia had MASLD, and 5% had metabolic dysfunction-associated steatohepatitis (MASH), formerly known as nonalcoholic steatohepatitis (NASH). People with MASLD or MASH are at increased risk of adverse liver-related health outcomes, and MASH is projected to soon become the leading indication for liver transplant (LT) in the US. The new nomenclature of MASLD was adopted to replace NAFLD in 2023. While their definitions differ, approximately 99% of patients with NAFLD would be classified as having MASLD. The aim of the study to review and summarize recent trends and emerging challenges in liver transplants for MASLD (metabolic dysfunction-associated steatotic liver disease) and ALD (alcohol-related liver disease).

Materials and Methods:
A focused narrative synthesis of registry data, epidemiologic modeling, and cohort studies (2013–2025) evaluating LT indication trends, donor/recipient characteristics, and post-LT outcomes in MASLD and ALD.

Results:

The proportion of liver transplant candidates with ALD increased from ~23% in 2013 to nearly 50% in 2022, while MASLD rose from 19% to 27%. MASLD recipients frequently present with obesity, diabetes, and cardiovascular comorbidities, contributing to higher peri- and post-transplant metabolic risk. De novo or recurrent steatosis in grafts is increasingly reported, though long-term graft survival appears preserved. Allocation disparities and differing pre-transplant requirements (sobriety for ALD vs metabolic optimization for MASLD) highlight policy gaps. Advances include machine-perfused and marginal graft use, metabolic risk-stratified recipient selection, and early post-LT weight and glycemic interventions.

Conclusions:

MASLD and ALD now dominate liver transplant indications. Novel strategies in candidate selection, allocation policy, and metabolic management are critical to improving outcomes. Future research should focus on recurrence of graft steatosis, cardiovascular mortality, and metabolic precision medicine in post-LT care.

Keyword(s) : MASLD/MASH, ALD, rising LT indications, machine-perfused marginal grafts, metabolic comorbidities

1238 - Awareness of Primary Biliary Cholangitis Among Turkish Physicians An Online Survey

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Background / Hypothesis:

Primary biliary cholangitis (PBC) is a chronic, rare, autoimmune liver disease characterized by non-suppurative cholangitis of the small bile ducts. This study aimed to assess the awareness of physicians in Türkiye, who are likely to encounter PBC patients.

Materials and Methods:

An online survey consisting of 28 questions related to the disease was sent to physicians via email and mobile phone groups. In addition to collecting participants' general demographic data, the survey included

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questions on their medical specialty, years of practice, and knowledge about the epidemiology, pathology, diagnosis, treatment, and follow-up of PBC.

Results:

Of the 615 physicians who were sent the survey, 270 answered the questions fully. Of the participants, 48% were women, and 28.8% had been practicing medicine for over ten years. Regarding specialties, 56% were internal medicine specialists or residents, 11.4% were gastroenterology specialists or residents, 9% were general practitioners, 8% were general surgery specialists or residents, 9% were family medicine specialists or residents, and 6% were other subspecialists. The rate of correct answers was lowest among general practitioners (64.5%) and highest among gastroenterologists (90%). There was a significant difference in correct response rates between specialists (77%) and non-specialists (71%) ($p=0.003$). The awareness of the need for response evaluation in first year of UDCA treatment was lowest among the family medicine and general practitioner group (36%).

Conclusions:

PBC is a relatively rare disease with low awareness not only in the general population but also among physicians. Delayed diagnosis and lack of treatment response evaluation are associated with poor outcomes. The results clearly indicate the need to raise awareness of PBC among physicians.

Keyword(s) :

1239 - Study on the Value of Antithrombin III Activity Combined with COSSH ACLF Score in Predicting the Prognosis of HBV ACLF Patients in the Intensive Care Unit

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Background

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Hypothesis:

To explore the predictive value of antithrombin III (AT-III) activity combined with the Chinese Society of Hepatology-ACLF (COSSH-ACLF) score for the mortality risk of patients with hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF) admitted to the intensive care unit (ICU).

Materials

and

Methods:

A total of 196 patients with severe HBV-ACLF admitted to the ICU of the Fifth People's Hospital of Wuxi from

January 1, 2020 to January 1, 2025 were retrospectively included. The baseline AT-III activity levels at admission were recorded. The area under the receiver operating characteristic curve (AUROC) was used to compare the efficacy of the European Chronic Liver Failure Consortium-ACLF (CLIF-C ACLF) score, COSSH-ACLF score, COSSH-ACLF II score, and Model for End-Stage Liver Disease (MELD) score in predicting the 28-day and 90-day mortality rates of HBV-ACLF patients.

Results:

Multivariate Cox regression analysis showed that AT-III activity (HR = 0.961, 95% CI 0.937–0.986, $P = 0.002$), INR (HR = 1.468, 95% CI 1.130–1.909, $P = 0.004$), and the presence or absence of sepsis (HR = 0.391, 95% CI 0.240–0.636, $P < 0.001$) were independent predictors of 90-day survival outcomes in HBV-ACLF patients. The AUROC of AT-III activity for predicting 28-day mortality was 0.745 (95% CI 0.677–0.804, $P < 0.001$), with an optimal cut-off value of 20%; for predicting 90-day mortality, it was 0.725 (95% CI 0.657–0.787, $P < 0.001$), with the same cut-off value of 20%. The AUROC of COSSH-ACLF score for predicting 28-day and 90-day mortality was 0.760 (95% CI 0.694–0.819, $P < 0.001$) and 0.738 (95% CI 0.671–0.798, $P < 0.001$), respectively, with a corresponding cut-off value of 90.6 for both. When AT-III activity and COSSH-ACLF score were combined, the AUROC for predicting 28-day and 90-day mortality increased to 0.780 and 0.757, respectively. Further analysis revealed that the AUROC of AT-III activity on the 7th day of admission for predicting 28-day and 90-day mortality was 0.799 and 0.773, respectively; the corresponding AUROC of COSSH-ACLF score was 0.898 and 0.872, respectively; and the AUROC of the combined prediction was 0.928 and 0.897, respectively.

Conclusions:

The activity of AT-III is closely related to the prognosis of HBV-ACLF patients. Either alone or in combination with the COSSH-ACLF score, it can effectively predict the 28-day and 90-day mortality risk of HBV-ACLF patients treated in the ICU. The predictive efficacy is further enhanced when the 7th-day indicators are dynamically monitored.

Keyword(s) : Antithrombin III Activity ; COSSH-ACLF Score ; Prognosis of HBV-ACLF ; Intensive Care Unit

1243 - Liver Transplantation Outcomes in Primary Sclerosing Cholangitis Patients With and Without Ulcerative Colitis A Comparative Analysis

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Background / **Hypothesis**
Primary sclerosing cholangitis (PSC) is a rare immune-mediated cholestatic disease characterized by progressive inflammatory fibrosis, strictures, and eventual cirrhosis. Up to 60–80% of PSC patients have inflammatory bowel disease (IBD), mainly ulcerative colitis (UC). PSC is a major indication for liver transplantation (LT), but post-transplant outcomes are influenced by rejection, recurrent PSC, malignant risk, and IBD-related immune activation. Recurrence occurs in nearly 20% of patients, with active IBD, male sex, and intact colon as risk factors. The effect of UC on graft and patient survival remains uncertain. This study evaluated the impact of UC on survival and post-transplant complications in PSC patients undergoing LT.

Materials and **Methods**
We performed a retrospective cohort study (January 2002–March 2023) at Imam Khomeini Hospital Complex (IKHC), Tehran. PSC was confirmed by explant pathology and UC by colonoscopy and histopathology. Patients younger than 18 years, re-transplants, those with hepatocellular carcinoma or cholangiocarcinoma, incomplete records, or loss to follow-up were excluded. In total, 167 PSC patients undergoing LT were included. Follow-up every three months included cholangiography and laboratory tests. Primary outcomes were overall survival and rejection-free survival; secondary outcomes were biliary stricture, hepatic artery thrombosis (HAT), and renal failure (RF). Statistical analysis included Chi-square, Fisher's exact test, Wilcoxon rank-sum test, Kaplan–Meier curves, Cox regression, and time-dependent covariates.

Results
Among 167 patients, 116 had UC. Baseline characteristics were similar except for higher INR in UC patients ($p=0.003$) and differences in ABO blood type. UC severity was mild in 61%, moderate in 22%, and severe in 17%. Overall survival was 75%. UC patients had higher rejection incidence (42% vs. 25%, $p=0.039$). Overall survival showed a borderline advantage in UC ($p=0.061$), while rejection-free survival showed a borderline decrease ($p=0.086$). Severe UC was

associated with increased HAT (HR 5.38, $p=0.028$). Tacrolimus use correlated with fewer biliary strictures ($p=0.015$). Higher BMI and MELD were associated with reduced renal-failure-free survival. Baseline creatinine predicted overall survival ($p=0.013$), and elevated CEA correlated with shorter rejection-free survival ($p<0.001$). Post-transplant renal failure strongly predicted mortality (HR 3.12, $p<0.001$). In multivariable analysis, UC showed a borderline survival advantage ($p=0.083$).

Conclusions

PSC patients with UC experienced higher rejection rates, increased HAT in severe UC, and worse outcomes in those with renal dysfunction. Pre-transplant creatinine, BMI, MELD, INR, and CEA were significant predictors. These findings underscore the importance of IBD control, renal protection, surgical optimization, and cancer surveillance in PSC patients undergoing LT.

Keyword(s) : Primary Sclerosing Cholangitis, Ulcerative Colitis, Liver Transplantation, Post-transplant Complications, Acute Rejection

1245 - Combined Application of Serum Alpha Fetoprotein and PIVKA II Levels for Prognostic Stratification in Patients With Hepatocellular Carcinoma

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Background / **Hypothesis:**
Protein induced by vitamin K absence or antagonists-II (PIVKA-II) and alpha-fetoprotein (AFP) are widely used biomarkers for the diagnosis and surveillance of hepatocellular carcinoma (HCC). However, their combined prognostic utility has not been fully clarified. This study aimed to evaluate the prognostic performance of combining serum AFP and PIVKA-II to determine the survival of HCC patients.
Materials and **Methods:**
The retrospective study enrolled 598 treatment-naïve HCC patients diagnosed from 2020 to 2023. Patients were categorized based on their serum PIVKA-II (cut-off value 1500 mAU/mL) and AFP (cut-off value 20 ng/mL) levels at diagnosis and stratified into three groups: high AFP and high PIVKA-II, low AFP and low PIVKA-II, and others. Cox proportional hazards models were used to

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identify independent predictors of overall survival (OS). Kaplan–Meier (KM) analyses were performed to compare survival across groups. Results:

Significant differences in tumor characteristics, including tumor stage, size, multiplicity, vascular invasion, and extrahepatic spread, as well as liver functional reserve were observed across the three groups. After a median follow-up of 16.0 months (interquartile range 5.0-22.0), 141 patients had died, with a 3-year OS rate of 63.0%. The 3-year OS rates for the three groups (high AFP & high PIVKA-II, either AFP/PIVKA-II elevated, and low AFP & low PIVKA-II) were 36.5%, 69.3%, and 85.0%, respectively. A multivariate analysis revealed co-existence of elevated serum AFP and PIVKA-II levels, lower serum albumin level, higher serum creatinine level, higher serum alkaline phosphatase levels, larger prothrombin time international normalized ratio (PT INR), presence of vascular invasion, and non-curative treatment as independent risk factors for poor OS. KM analysis confirmed clear prognostic separation between the three biomarker groups ($p < 0.05$) (Figure). Conclusions:

AFP and PIVKA-II are independent predictors of OS in HCC patients, and their combined assessment offers superior prognostic stratification. Integration of both biomarkers can effectively classify patients into distinct risk categories and may guide clinical decision-making and individualized treatment planning.

Keyword(s) :

1250 - Identifying MASLD Patients with MASH or Significant Fibrosis The Role of Lower ALT Thresholds

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Background / Hypothesis: Although ALT is a marker for detecting MASH and significant fibrosis in patients with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), some individuals with normal ALT levels still exhibit these histological changes, leading to underdiagnosis. Liver biopsy is often recommended for MASLD patients with elevated ALT, yet its routine use remains limited. This study aimed to determine the prevalence of MASH and significant fibrosis in MASLD patients with normal ALT and to identify predictive factors.

Materials and Methods: In this cross-sectional study, 404 MASLD patients without concurrent viral hepatitis

who underwent liver biopsy at Tianjin Second People's Hospital between October 2012 and July 2024 were included. Liver histology served as the gold standard for assessing inflammation and fibrosis. Patients were categorized using both conventional and lower ALT thresholds. Univariate and multivariate analyses were performed to identify factors associated with MASH and fibrosis in patients with normal ALT.

Results: Among the 404 patients with MASLD, 284 (70.3%) had MASH and 271 (67.1%) had significant fibrosis. Of those with conventionally normal ALT levels ($n=130$), the prevalence of MASH and significant fibrosis was 49.23% and 50.00%, respectively. Using a lower ALT threshold (30 U/L for men, 19 U/L for women), patients above the cutoff had significantly higher rates of significant fibrosis (56.18% vs. 36.59%, $P=0.002$, Table 1), along with elevated ferritin, CAP, and APRI levels ($P < 0.05$). The incidence of MASH also showed a decreasing trend in the lower ALT group (53.93% vs. 39.02%, $P = 0.082$). There was no significant difference in the prevalence of MASH or significant fibrosis between patients aged <50 and ≥ 50 years ($P > 0.05$). Female patients in the higher ALT subgroup had significantly higher rates of both MASH and significant fibrosis. Multivariate analysis confirmed that the lower ALT threshold was an independent predictor of MASH (OR=10.089, $P < 0.001$) and significant fibrosis (OR = 5.718, $P = 0.006$, Table 2).

Conclusions: Current conventional ALT ULNs result in the missed diagnosis of a substantial number of MASLD patients with histologically significant disease. Implementing lower ALT upper limites (30 U/L for men, 19 U/L for women) significantly improves risk stratification and could prevent missed diagnoses of MASH and significant fibrosis.

Keyword(s) : Metabolic Steatohepatitis; Fibrosis; Pathology; Missed Diagnosis

1252 - Clinical Trajectories of Acute Decompensation in Cirrhosis The First Prospective PREDICT Based Study from India

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Background / Hypothesis: Acute decompensation (AD) of cirrhosis is a heterogeneous clinical state with variable short-term

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outcomes. The PREDICT study identified three phenotypes—Stable Decompensated Cirrhosis (SDC), Unstable Decompensated Cirrhosis (UDC), and pre-Acute-on-Chronic Liver Failure (pre-ACLF)—driven largely by systemic inflammation. This study aimed to evaluate the clinical trajectories of AD based on the PREDICT framework in an Indian cohort and to identify inflammatory and biochemical predictors of progression to ACLF and 6-month mortality.

Materials and Methods: This prospective observational study included 105 consecutive adults with cirrhosis admitted with AD to a tertiary-care center over 12 months. AD was defined by new or worsening ascites, hepatic encephalopathy (HE), gastrointestinal bleeding, or bacterial infection. Patients with ACLF at presentation, immunosuppression, pregnancy, or post-transplant status were excluded. All participants were followed for 90 days to classify outcomes as SDC, UDC, or pre-ACLF, and for 6-month survival. Demographic, clinical, and laboratory parameters—including Hs-CRP, TNF- α , IL-6, and urinary NGAL—were recorded at baseline. Group comparisons were performed using appropriate statistical tests, and variables with $p < 0.1$ on univariate analysis were entered into multivariate logistic regression for prediction of ACLF and mortality. Prognostic accuracy of Hs-CRP was assessed using ROC analysis.

Results:

Of 105 patients (mean age 46.6 ± 10.9 years; 82.9% male), 57 (54.3%) were classified as SDC, 24 (22.9%) as UDC, and 24 (22.9%) as pre-ACLF at 90 days. Pre-ACLF patients had significantly higher prevalence of HE (54%), bacterial infections (46%), SBP (50%), sepsis (38%), renal dysfunction (creatinine 1.47 ± 0.33 mg/dL), and markedly elevated inflammatory markers (Hs-CRP 38.1 ± 14.6 mg/L; TNF- α 157 ± 50 pg/mL; IL-6 126 ± 45 pg/mL; all $p < 0.001$). Thirty-one patients (29.5%) died by 6 months. Non-survivors showed significantly higher baseline inflammation, organ dysfunction, and severity scores (MELD-Na 22.4 ± 5.8 vs 16.6 ± 5.6 , $p < 0.001$). On multivariate analysis, Hs-CRP independently predicted both ACLF (OR 1.057; $p = 0.04$) and 6-month mortality (OR 1.111; $p = 0.03$). Hs-CRP demonstrated excellent prognostic accuracy (AUC 0.92), with an optimal cut-off of 10.8 mg/L (90% sensitivity, 95% specificity).

Conclusions:

In this first Indian prospective validation of the PREDICT

classification, inflammatory phenotype strongly determined clinical trajectory and survival in AD. Pre-ACLF was characterized by severe systemic inflammation and high early mortality. Hs-CRP emerged as a simple, robust, and independent predictor of ACLF and 6-month mortality. Routine CRP assessment may greatly improve early risk stratification in acutely decompensated cirrhosis.

Keyword(s) : Acute decompensation, Acute-on-chronic liver failure, PREDICT classification, Systemic inflammation, Hs-CRP, Prognosis, Mortality

1257 - Si Ni San a traditional Chinese herbal formula ameliorates cholestatic liver injury by targeting the calcium sensing receptor (CaSR) MAPK pathway
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Background / Hypothesis: Cholestatic liver injury (CLI) is a common hepatic disorder characterized by bile acid accumulation, leading to hepatocyte damage and inflammation. Si-Ni-San (SNS), a traditional Chinese medicine formula, has shown potential in CLI treatment, yet its molecular mechanisms remain unclear. This study aimed to explore the protective effects and mechanisms of SNS in CLI.

Materials and Methods: The primary active components of SNS were identified using high-performance liquid chromatography (HPLC). A network pharmacology approach was used to predict the protective mechanisms of SNS against CLI. In vivo and in vitro experiments were performed to verify the network pharmacology findings. Molecular docking was used to explore the interactions between the active compounds of SNS and the calcium-sensing receptor (CaSR).

Results:

HPLC identified 4 main active components of SNS, and the network pharmacology predicted that SNS exerts its therapeutic effects on CLI by modulating the calcium and MAPK signaling pathways. The result of animal experiments revealed that SNS significantly alleviated hepatocellular injury, reduced inflammatory infiltration, restored bile acid homeostasis. Additionally, SNS inhibited CaSR expression and lowered Ca^{2+} concentrations, leading to the downregulation of p-p38

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MAPK and p-JNK in both in vivo and in vitro models. Molecular docking further revealed strong binding affinities between SNS active ingredients and CaSR, supporting its role in regulating the CaSR/MAPK pathway in CLI treatment.

Conclusions:

SNS alleviated CLI by regulating bile acid homeostasis, reducing inflammatory infiltration, and lowering Ca^{2+} concentrations, likely through inhibition of the CaSR/MAPK pathway.

Keyword(s) : cholestatic liver injury; Si-Ni-San; network pharmacology; bile acid; calcium-sensing receptor

1263 - The Paradox of Size A Non Linear Relationship Between Liver Size and Fibrosis Stage in Acute Autoimmune Hepatitis

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Background/Hypothesis: No conventional radiological characteristic for fibrosis is defined for autoimmune hepatitis (AIH). In acute hepatitis, hepatomegaly is often attributed to inflammation. Conversely, advanced fibrosis is associated with splenomegaly. This study aimed to investigate the correlation between liver size and histological fibrosis stage in patients presenting with acute AIH.

Material and Methods In this single-center retrospective study (2015-2025), 63 patients with acute AIH (IAIHG criteria) were included. Other liver disease etiologies were excluded. CT or MRI measured liver size. Data included labs and liver biopsy results (Modified Ishak staging). The relationship between liver size and fibrosis stage was analyzed using the Kruskal-Wallis test and ordinal logistic regression. Patients were categorized as non-fibrotic (Stage 0-2) and advanced fibrosis (Stage 3-4) and compared using non-invasive scores (APRI, FIB-4) and portal hypertension indicators. Results: Of 63 patients (77.8% female, with a mean age of 42.98), 30.1% (n = 19) had advanced fibrosis (Stage 3-4) at diagnosis. A significant relationship was found between liver size and the stage of fibrosis ($p < 0.05$). Median liver size exhibited a non-linear pattern, increasing from Stage 0 (150 mm) to Stage 3 (180.5 mm) and then decreasing at Stage 4 (148.5 mm). In multivariate logistic regression (adjusting for age, sex, and diabetes), liver size was the only independent variable associated with fibrosis stage ($p = 0.013$). When

comparing non-fibrotic (S0-2) vs. advanced fibrosis (S3-4) groups, standard non-invasive scores (APRI, FIB-4) and portal hypertension indicators (e.g., spleen size) showed no significant difference (all $p > 0.05$). However, liver size was significantly larger in the advanced fibrosis group (median 180 mm vs 157 mm, $p < 0.05$). Conclusion: Standard non-invasive scores (APRI, FIB-4) and portal hypertension signs fail to predict advanced fibrosis in acute AIH, likely due to the inflammatory nature of the disease. Our study demonstrates a novel, non-linear relationship: liver size increases with fibrosis up to Stage 3, then decreases at Stage 4 (cirrhosis) due to fibrotic retraction. This suggests hepatomegaly may be a radiological indicator of progressive fibrosis (especially Stage 3), not just inflammation. Liver size is a simple, accessible, non-invasive marker that may aid in estimating fibrosis when a biopsy is not feasible.

Keyword(s) : Autoimmune Hepatitis, Hepatomegaly, Liver Fibrosis, Liver Cirrhosis, Acute Hepatitis

1265 - Risk factors for advanced liver fibrosis in psoriasis patients with metabolic dysfunction associated steatotic liver disease (MASLD)

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Background:

Psoriasis is a risk factor for steatotic liver disease (SLD), and the prevalence of SLD among psoriasis patients is high. Most previous studies have been retrospective. We aim to identify clinical risk factors for predicting advanced liver fibrosis in patients with psoriasis and metabolic dysfunction-associated steatotic liver disease (MASLD) in a prospective Asian cohort.

Materials and Methods:

Adult psoriasis patients at National Taiwan University Hospital (NTUH) were screened for SLD using FibroScan®. Patients with controlled attenuation parameter (CAP) ≥ 248 dB/m were prospectively recruited. Advanced fibrosis was defined as liver stiffness measurement (LSM) >8 kPa. Patients with excessive alcohol consumption were excluded. A questionnaire was administered to collect past medical history, alcohol consumption, smoking, and exercise habits. Laboratory tests for metabolic dysfunction were performed. Psoriasis-related history was collected from the electronic medical record system at NTUH. Clinical

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risk factors for advanced fibrosis were investigated and the performance of established non-invasive scores for predicting advanced fibrosis (FIB-4, APRI, and SAFE) was also examined.

Results:

From April to October 2025, a cohort of 109 patients was prospectively recruited, all of whom met the criteria for MASLD. The prevalence of advanced fibrosis was 28%. The cohort had a high burden of metabolic risk factors, including obesity (71%), prediabetes or diabetes (79%), and dyslipidemia (78%). Twelve patients (11%) had chronic hepatitis B virus (HBV) infection. Univariable analysis identified body mass index (BMI) (odds ratio [OR]: 1.15 per unit [kg/m²] increase, 95% confidence interval [CI]: 1.04-1.28), waist circumference (OR: 1.06 per cm increase, 95% CI: 1.02-1.10), high waist-to-height ratio (WHtR ≥ 0.6) (OR: 5.67, 95% CI: 2.17-14.82), and high insulin resistance (homeostatic model assessment for insulin resistance [HOMA-IR] > 2.9) (OR: 2.67, 95% CI: 1.12-6.38) as significant risk factors for advanced fibrosis. Psoriasis severity and disease duration were not associated with advanced fibrosis. Multivariable analysis identified WHtR ≥ 0.6 (OR: 5.50, 95% CI: 1.95-15.54), chronic HBV infection (OR: 5.58, 95% CI: 1.29-24.20), and cumulative methotrexate dose > 1500 mg (OR: 3.41, 95% CI: 1.22-9.55) as independent risk factors for advanced fibrosis. FIB-4 performed poorly (area under the receiver operating characteristic curve [AUROC]: 0.594), while the SAFE score (cutoff < 0) was effective for ruling out advanced fibrosis (negative predictive value [NPV]: 90%).

Conclusions:

In this cohort of patients with concurrent psoriasis and MASLD, an elevated waist-to-height ratio (≥ 0.6), chronic HBV infection, and a cumulative methotrexate dose > 1500 mg were independent risk factors for advanced fibrosis.

Keyword(s) : MASLD, Advanced fibrosis, Psoriasis, Waist-to-height ratio, HBV, Methotrexate

1266 - The quality and reliability of short videos about liver cirrhosis on Bilibili and TikTok Cross sectional study

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Background / Hypothesis: Cirrhosis is a major global public health burden, with a high prevalence in China.

Short video platforms such as TikTok and Bilibili have become key channels for disseminating cirrhosis-related health information, but concerns remain regarding the quality and reliability of such content. Materials and Methods: A cross-sectional study was conducted. Using "cirrhosis" as the keyword, 157 eligible videos (74 from TikTok, 83 from Bilibili) were selected. Video quality and reliability were assessed using the Global Quality Score (GQS), modified DISCERN (mDISCERN), and JAMA scoring tools. Demographic characteristics, content categories, and user engagement metrics were analyzed with nonparametric tests and correlation analysis. Results: The median GQS, mDISCERN, and JAMA scores were 3 (IQR 3-4), 2 (IQR 2-2), and 3 (IQR 3-3), respectively. TikTok videos had higher user engagement (median likes: 1987 vs. 51) but shorter duration (57s vs. 328s) and lower GQS (3 vs. 4) than Bilibili. Videos by hepatologists and other specialists showed significantly higher quality scores than those by TCM practitioners and non-professionals. No significant correlation was found between user engagement and video quality. Conclusions: Cirrhosis-related short videos on TikTok and Bilibili have suboptimal scientific quality. Bilibili provides more in-depth content, while TikTok has higher popularity. Professional background of uploaders is a key determinant of content quality. Platforms should strengthen qualification review and content supervision to improve health information equity.

Keyword(s) : Keywords: Cirrhosis; short-video; cross-sectional study; health education; quality evaluation

1272 - Comparing two intervention strategies to improve hepatitis B virus deoxyribonucleic acid testing rate among hepatitis B surface antigen positive hospitalized patients

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Background / Hypothesis: Hepatitis B surface antigen (HBsAg) screening is fundamental for chronic hepatitis B management, while hepatitis B virus deoxyribonucleic acid (HBV-DNA) testing is crucial for determining the viral replication status and initiating antiviral therapy. However, the HBV-DNA testing rate among inpatients is generally low. This study aimed to analyze the HBsAg positivity

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rate and HBV-DNA testing among inpatients at Ningbo Second Hospital and to evaluate the effectiveness of two intervention measures—publishing departmental rankings on the hospital intranet and conducting on-site educational sessions in departments—in improving the HBV-DNA testing rate.

Materials and Methods:1. Retrospectively collect data from inpatients between August 2024 and November 2025 and calculate the monthly HBsAg positivity rate and HBV-DNA testing rate.2. Starting from November 2024, departmental HBV-DNA testing rate rankings were published monthly on the hospital intranet. Changes in the testing rate over the subsequent 5 months (December 2024 to April 2025) were evaluated.3. Starting from May 2025, on-site educational sessions emphasizing the clinical significance of HBV-DNA testing were conducted in the 10 departments with the lowest testing rates. Changes in the testing rate over the following 6 months (May to October 2025) were evaluated. A t-test was used to compare testing rates before and after the interventions, with a P-value < 0.05 considered statistically significant.

Results:1. During the baseline period (August–November 2024), a total of 40,255 inpatients were included. The HBsAg positivity rate was 7.8%(3,139/40,255), and the mean HBV-DNA testing rate was 28.4%.2. During the period of intranet ranking publication (December 2024–April 2025), a total of 48,176 inpatients were included. The HBsAg positivity rate was 7.3% (3,506/48,176), and the mean HBV-DNA testing rate was 28.4%, showing no significant change compared to the baseline (P = 0.94).3. During the period of on-site educational sessions (May–October 2025), a total of 61,512 inpatients were included. The HBsAg positivity rate was 7.4% (4,531/61,512), and the HBV-DNA testing rate increased to 32.6%, showing a significant improvement compared to the previous period (P < 0.05).

Conclusions: Publishing testing rate rankings on the intranet alone did not effectively improve the HBV-DNA testing rate. However, targeted on-site educational sessions in departments significantly increased the HBV-DNA testing rate. Proactive, face-to-face medical education holds important practical value in enhancing HBV-DNA testing among inpatients.

Keyword(s) : HBV,HBsAg,HBV-DNA,Whole-Process Management of Hepatitis B

1277 - Evaluation of Treatment Response in Chronic Hepatitis C Patients Receiving Sofosbuvir Velpatasvir Voxilaprevir A Multicenter Real World Experience from Türkiye

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Background / Hypothesis:The combination of sofosbuvir/velpatasvir/voxilaprevir (SOF/VEL/VOX) is recommended as a salvage therapy for treatment-experienced chronic hepatitis C (CHC) patients. However, it is used in our country for treatment-naïve and treatment-experienced patients. This study aims to present real-world data from Türkiye on CHC patients treated with SOF/VEL/VOX. The present study was conducted by the Viral Hepatitis Study Group of the Turkish Society of Clinical Microbiology and Infectious Diseases (KLİMİK).

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Materials and Methods: It was a multicenter, retrospective, observational study. The data were collected from patients receiving SOF/VEL/VOX therapy at 12 medical centers in Türkiye between 1 June 2022 and 31 December 2024. The patients had received the treatment for 8 to 12 weeks.

Results: Of the 139 patients enrolled, 63.3% (n = 88) were male, with a mean age of 54.4 years. Most patients were non-cirrhotic (94.2%, n = 131) and treatment-naïve (92%, n = 128); 49.6% (n = 69) were infected with genotype 1b. Early virologic response (EVR) could be assessed in 126 patients, with an EVR rate of 82.5% (n = 104). End-of-treatment data were available for 113 patients, all achieving an end-of-treatment response. Among the 80 patients for whom week-12 post-treatment data were available, 97.5% sustained virologic response at week 12 (SVR12). Significant improvements were observed in AST, ALT, and platelet levels, along with reductions in APRI and FIB-4 scores (p = 0.001). No serious adverse events leading to treatment discontinuation were reported. Mild adverse events included pruritus (2.1%, n = 3), fatigue (2.1%, n = 3), and nausea (1.4%, n = 2).

Conclusions: The SOF/VEL/VOX combination is a highly effective and well-tolerated treatment option in treatment-naïve CHC patients, achieving an SVR12 rate of 97.5%.

Keyword(s) : sofosbuvir/velpatasvir/voxilaprevir, hepatitis C treatment, real-world data, Türkiye

1278 - Small lesion oriented early hepatocellular carcinoma segmentation via a hybrid attention deformable U-Net

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Background : / **Hypothesis:**
Early detection of hepatocellular carcinoma (HCC) on contrast enhanced CT is hindered by tiny, low contrast lesions, motion/phase artifacts, and cross scanner variability. We hypothesized that coupling 2.5D hybrid attention with a boundary aware deformable decoder would enhance small lesion recall and boundary fidelity without prohibitive computation.

Materials and Methods:
We developed a Hybrid Attention Deformable U-Net comprising a 2.5D encoder that interleaves depthwise separable convolutions with windowed self-attention (shifted tilings) and cross scale gated fusion, and a decoder with modulated deformable convolutions plus an auxiliary contour head trained on signed-distance targets. A composite objective (Focal-Tversky, boundary-IoU, Soft-Dice) with online hard example mining and motion/phase-aware augmentation (synthetic respiratory blur, slice misregistration) was used. The Liver Tumor Segmentation (LiTS) dataset (n=131) was standardized with liver ROI extraction; five-fold, patient disjoint cross validation was performed. Inference used sliding window with overlap and test time augmentation.

Results:

Across folds, the method achieved Dice $78.6 \pm 1.5\%$, HD95 11.2 ± 2.1 mm, and thin band boundary IoU $61.0 \pm 1.5\%$, with lesion wise sensitivity $75.9 \pm 2.3\%$ and small lesion (<10 mm) sensitivity $56.3 \pm 3.0\%$. It consistently outperformed five strong 2D/3D and hybrid baselines under an identical pipeline. Under motion/phase perturbations, Dice decreased by only 1.4 points (vs. 2.9 – 4.5 for comparators), indicating improved robustness; the model uses 12.9M parameters with ≈ 2.3 s/volume single GPU inference.

Conclusions:

The approach improved early HCC segmentation on contrast enhanced CT, particularly for sub-centimeter and subcapsular lesions, while maintaining practical efficiency. Multi-center, multi-phase validation and semi- or weakly supervised learning with domain adaptation will be pursued.

Keyword(s) : Hepatocellular carcinoma, Liver tumor segmentation, Hybrid attention, Deformable convolution, 2.5D segmentation

1280 - Tislelizumab plus lenvatinib versus tislelizumab monotherapy as adjuvant therapy for hepatocellular carcinoma patients at high risk of recurrence a prospective multicenter cohort study

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Background / Hypothesis:Accumulating clinical evidence has shown that adjuvant PD-1 inhibitor therapy confers prognostic benefits in hepatocellular carcinoma (HCC) patients at high risk of recurrence after curative hepatectomy. However, the necessity of combining with molecular targeted agents remains controversial. This study aims to evaluate the efficacy of tislelizumab (a PD-1 inhibitor) plus lenvatinib (a molecular targeted drug) versus tislelizumab monotherapy in this patient cohort.

Materials and Methods:This is a prospective multicenter cohort study. HCC patients who underwent curative resection and had at least one high-risk factor for recurrence were screened and recruited from 13 centers in China between January 16, 2019, and December 26, 2024. All enrolled patients were assigned to receive adjuvant tislelizumab (200 mg) monotherapy or tislelizumab plus lenvatinib. The primary endpoint was recurrence-free survival (RFS), with secondary endpoints including overall survival (OS) and safety.

Results:A total of 167 patients were enrolled, with 91 (54.5%) receiving adjuvant tislelizumab plus lenvatinib and 76 (45.5%) receiving adjuvant tislelizumab monotherapy. During follow-up, the median RFS was 26.4 months (95% CI: 18.6–34.1) in the tislelizumab monotherapy group versus 22.4 months (95% CI: 17.0–27.8) in the tislelizumab plus lenvatinib group, with no statistically significant difference observed (HR: 0.81; 95% CI: 0.53–1.24), and this trend persisted after propensity score matching. Median OS was not reached in either cohort, and there was no significant difference between the adjuvant tislelizumab monotherapy and tislelizumab plus lenvatinib groups, both prior to (HR: 0.95; 95% CI: 0.43–2.07) and following propensity score matching. The rate of grade 3 or 4 adverse events was significantly higher with tislelizumab plus lenvatinib, particularly for hand-foot syndrome, rash, and hypertension.

Conclusions:The addition of lenvatinib did not significantly improve the prognosis of patients receiving adjuvant PD-1 inhibitor therapy. Further large-scale randomized controlled trials are required to evaluate the potential of adjuvant combination therapy.

Keyword(s) : Adjuvant, curative resection, hepatocellular carcinoma, PD-1 inhibitor, lenvatinib

1281 - Short Term Gains Long Term Challenges A Pilot Randomized Controlled Trial for Time Restricted Feeding Versus Caloric Restriction in MASLD

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Background:

Weight loss remains the cornerstone therapy for Metabolic dysfunction associated steatotic liver disease (MASLD), for which caloric restriction (CR) is an established strategy. Recently, time restricted feeding

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(TRF) which aligns food intake with circadian rhythms have shown promise in improving metabolic outcomes. However, comparative data on adherence and weight loss between conventional CR and TRF in MASLD remain limited.

Methods:

We conducted a single-centre pilot randomized controlled trial comparing TRF with conventional CR in adults with significant MASLD fibrosis ($\geq 7\text{kPa}$). Forty participants were randomized 1:1 to either daily CR (control) or TRF with an 8-hour feeding window (8:00–16:00) while maintaining equivalent caloric restriction. Dietary adherence was tracked through digital food logs and structured nutritional reviews. Compliance was defined as $\geq 70\%$ adherence to the prescribed caloric intake (and TRF window for the intervention group). Participants were followed up to 12 months with serial anthropometric and clinical chemistry measurements.

Results:

Forty participants (mean age 48.2 ± 12.2 years; 67.5% male, 72.5% Chinese) were enrolled. Baseline metabolic and anthropometric parameters were similar between the two arms (BMI 31.2 vs 30.7 kg/m², $p = 0.77$; waist circumference 104.6 vs 102.3 cm, $p = 0.59$, CAP 315.7 vs 311.2 dB/m, $p = 0.68$, HbA1c 6.2% vs 6.1% , $p = 0.77$). Comorbidities were balanced (diabetes 30%, hypertension 50%, dyslipidaemia 67.5%).

TRF participants achieved greater weight loss than control from Month 1 through Month 6 ($p < 0.01$), with a mean percentage loss at 6 months of $-7.7 \pm 5.0\%$ versus $-2.0 \pm 4.9\%$ ($p = 0.01$). The difference attenuated at 12 months ($-3.8 \pm 4.4\%$ vs $-1.6 \pm 5.3\%$, $p = 0.19$), reflecting partial weight gain after the intervention period. Early caloric adherence was comparable between the groups but declined in the TRF arm after 3 months. In the multivariable analysis, TRF independently predicted greater weight loss ($p < 0.01$). Higher caloric adherence correlated with incremental weight reduction (-5% adherence for each 10% weight increase, $p < 0.01$) though the relationship differed by the intervention arm (interaction $p < 0.01$).

Conclusion:

TRF led to significantly short-term weight loss compared with standard caloric restriction, and was feasible among adults with significant MASLD. However, adherence waned over time, enforcing that sustained behaviour support is essential to maintain long-term benefits.

Keyword(s) : Metabolic dysfunction associated steatotic liver disease, time restricted feeding, caloric restriction, weight loss, liver fibrosis

1283 - Diagnostic Performance of Non Invasive Serum Biomarkers for Fibrosis in Hepatocellular Carcinoma Patients With Metabolic Dysfunction-Associated Steatotic Liver Disease

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Background

Non-invasive serum biomarkers (NISB), such as the Fibrosis-4 (FIB-4) and aspartate aminotransferase-to-platelet ratio index (APRI), are widely used to estimate liver fibrosis and cirrhosis. However, recent studies indicate that their diagnostic performance may be altered in patients with hepatocellular carcinoma (HCC), particularly those with metabolic dysfunction-associated steatotic liver disease (MASLD). This study aimed to evaluate the correlation between various NISBs and histological fibrosis severity and to compare their accuracy in predicting advanced fibrosis in HCC patients with concurrent MASLD.

Materials

A total of 469 HCC patients who underwent curative resection at Taipei Veterans General Hospital and had available pathology slides and complete clinical datasets were included in the study. Spearman correlation coefficients were used to assess associations between ISHAK fibrosis scores and five NISBs: FIB-4, APRI, non-alcoholic fatty liver disease fibrosis score (NFS), BMI-AST/ALT ratio-diabetes mellitus (BARD) score, and AST/ALT ratio (AAR). Diagnostic performance for predicting advanced fibrosis (ISHAK ≥ 4) was evaluated using receiver operating characteristic (ROC) analyses and compared across biomarkers. Subgroup analyses were conducted for viral versus non-viral etiologies. Optimal single cutoffs were determined using Youden's

Methods:



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index; rule-out and rule-in thresholds corresponded to values with sensitivity >90% and specificity >80%, respectively.

Results:

Among the cohort, 252 patients (53.7%) had advanced fibrosis. Compared with those without advanced fibrosis, these patients exhibited more aggressive tumor features, including larger tumor size, more multifocal disease, and higher prevalence of vascular invasion and extrahepatic spread, while liver functional reserve remained comparable. Among the NISBs, FIB-4 and APRI demonstrated the strongest correlation with ISHAK fibrosis stage. FIB-4 showed marginally superior discriminatory ability for advanced fibrosis (AUROC 0.735; 95% CI 0.690–0.779; $p < 0.001$), similar to APRI (AUROC 0.731; 95% CI 0.686–0.776; $p < 0.001$). The optimal single cutoff of FIB-4 was 2.39, with rule-out and rule-in thresholds of 1.31 and 2.45, respectively. Subgroup analyses showed consistent performance of NISBs and similar cutoff values across viral and non-viral etiologies.

Conclusions:

In HCC patients with concurrent MASLD, NISBs such as FIB-4 and APRI demonstrated modest correlation with histological fibrosis and comparable diagnostic accuracy across different etiologies. The FIB-4 rule-out threshold of 1.3 aligned with current guideline recommendations, while the rule-in threshold identified in this cohort was slightly lower than those reported in earlier development studies.

Keyword(s) : Hepatocellular Carcinoma (HCC), Metabolic dysfunction–Associated Steatotic Liver Disease (MASLD), Fibrosis, Cirrhosis, Non-invasive Serum Biomarkers (NISB)

1284 - Antifibrotic Efficacy and Renal Safety of Pemafibrate in MASLD A Multicenter Randomized Controlled Study

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Background

The prevalence of metabolic dysfunction–associated steatotic liver disease (MASLD) is rapidly increasing worldwide, yet choice of pharmacologic therapy is limited. Pemafibrate is a novel selective PPAR α modulator (SPPARM α) with greater activity and selectivity than conventional PPAR α agonists. Previous studies have shown improvements in alanine aminotransferase (ALT) without increasing serum creatinine. We aimed to evaluate the efficacy and safety of pemafibrate in patients with MASLD.

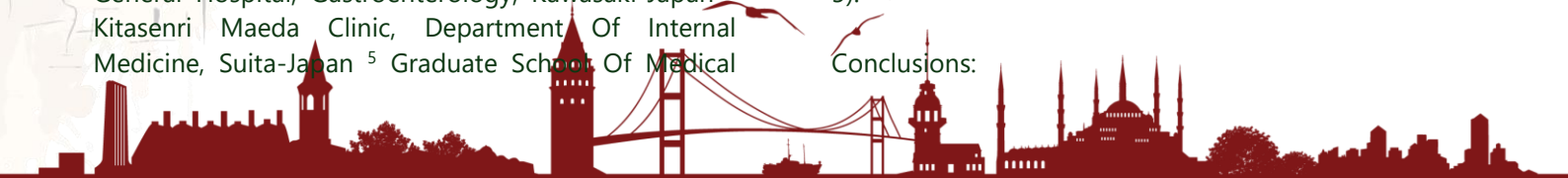
Materials

This multicenter, open-label, randomized controlled trial enrolled MASLD patients aged 20–80 years who met inclusion criteria. Participants were randomly assigned to receive pemafibrate 0.4 mg/day (PEM-H), pemafibrate 0.2 mg/day (PEM-L), or fenofibrate (FENO) for 48 weeks. The primary endpoint was the change in ALT after 24 weeks. Secondary endpoints included changes in fibrosis markers and, in selected centers, liver stiffness assessed by magnetic resonance elastography (MRE) at week 48.

Results:

Among 360 enrolled patients, 356 were analyzed. Mean ALT changes at week 24 were -24.0 ± 23.0 U/L (PEM-H), -14.8 ± 27.6 U/L (PEM-L), and -4.2 ± 35.9 U/L (FENO), showing significantly greater reductions with pemafibrate compared with fenofibrate. Percent changes in Mac-2 binding protein glycosylation isomer (M2BPGi) at week 48 were $-21.0 \pm 23.0\%$ (PEM-H), $-17.8 \pm 25.6\%$ (PEM-L), and $-3.2 \pm 27.3\%$ (FENO), respectively ($p < 0.05$ vs FENO). Liver stiffness by MRE decreased by $-9.2 \pm 14.0\%$ (PEM-H), $-8.4 \pm 9.9\%$ (PEM-L), and $-3.6 \pm 21.4\%$ (FENO), respectively ($p < 0.05$ vs. FENO). Percent changes in eGFR was $-3.7 \pm 10.0\%$ (PEM-H), $-0.8 \pm 10.3\%$ (PEM-L), and $-10.1 \pm 10.2\%$ (FENO), indicating less renal function decline with pemafibrate. Treatment-related adverse events occurred in 8 patients (PEM-H), 8 patients (PEM-L), and 18 patients (FENO), respectively; 15 cases involved myalgia or rhabdomyolysis. Liver or biliary disorders occurred only in the FENO group ($n = 5$).

Conclusions:



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Pemafibrate 0.4 mg/day and 0.2 mg/day significantly reduced ALT and improved both fibrosis markers and liver stiffness compared with fenofibrate, with superior renal safety and tolerability. These findings demonstrate that pemafibrate exerts antifibrotic effects and represents an effective and well-tolerated therapeutic option for MASLD.

Keyword(s) :

1286 - Clinical characteristics of diabetic patients with limited improvement in elevated ALT following tirzepatide initiation

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Background / **Hypothesis:**
Tirzepatide acts on both GIP and GLP-1 receptors and improves glucose metabolism, and is therefore expected to reduce elevated ALT levels in diabetic patients with steatotic liver disease. However, some patients do not show such improvement. This study aimed to identify the clinical characteristics of diabetic patients in whom ALT reduction was not achieved after tirzepatide administration.

Materials and Methods:
Among 100 diabetic patients treated with tirzepatide for at least 12 weeks at our institution, 51 patients with baseline ALT >30 U/L were analyzed. Cases were defined as "effective" if ALT decreased to <30 U/L or showed a ≥30% reduction from baseline, and "ineffective" otherwise. Clinical factors associated with ineffectiveness were retrospectively examined.

Results:

Of the 51 patients (35 men and 16 women; median age 52 years), 21 were classified as effective and 30 as ineffective. Univariate logistic regression analysis revealed significant associations between ineffectiveness and lower platelet count (odds ratio [OR] 1.117 per 10,000/ μ L decrease; $P=0.0379$), lower serum albumin (OR 21.41 per 1 g/dL decrease; $P=0.0464$), and prior use of GLP-1 receptor agonists (OR 4.400; $P=0.0285$). Switching from GLP-1RA/insulin combination therapy was not significant ($P=0.7176$). Multivariate analysis identified serum albumin and prior GLP-1RA use as independent predictors. ROC analysis of

albumin levels ($n=27$, excluding 24 cases with missing data) yielded a cutoff of 4.3 g/dL (AUC=0.750). Patients with albumin ≥ 4.3 g/dL and no prior GLP-1RA monotherapy ($n=7$) had an odds of ineffectiveness of 0.167, compared to 4.0 in other cases ($n=20$), with an OR of 24.0 ($P=0.0019$).

Conclusions:

ALT reduction with tirzepatide therapy may be less likely in patients previously treated with GLP-1 receptor agonists or those with baseline serum albumin levels below 4.3 g/dL.

Keyword(s) : GIP, GLP-1 receptor agonists, albumin

1289 - Computed tomography based hepatosplenic volumetry can predict all cause and liver related mortality in patients with cirrhosis

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Backgrounds: Although traditional noninvasive prognostic models for cirrhosis are widely used, their predictive performance remains limited. Previous studies have identified computed tomography (CT)-based hepatosplenic volumetry as a predictor of portal hypertension, decompensation of cirrhosis, and liver fibrosis staging. This study aimed to evaluate the prognostic value of CT-based hepatosplenic volumetry in predicting mortality among cirrhotic patients. **Materials and Methods:** This retrospective study included adult patients diagnosed with cirrhosis at our hospital from 2008 to 2022. All patients underwent portal-venous or equilibrium-phase CT imaging. Total liver volume (TLV), spleen volume (SV), and liver to spleen volume ratio (LSVR) were obtained. The primary endpoint was all-cause mortality, and secondary endpoint was liver-related mortality, which was subcategorized into infectious, portal hypertensive, oncologic, and other hepatic causes. Univariable and multivariable logistic regression analyses were performed to identify predictors of mortality, and Kaplan-Meier survival curves were constructed to compare survivals between patients stratified by the median LSVR value.

Results: 525 cirrhotic patients were enrolled, with a

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mean follow-up of 4.74 ± 2.22 years. The primary etiologies of cirrhosis were viral (50.3%) and alcoholic (32.0%). 361 (68.8%) patients had decompensated cirrhosis. 182 (34.67%) patients died during the follow-up period (Table 1). The univariate analysis identified age, MELD score, LSVR, and TLV/SV as predictors of all-cause mortality. The multivariate analysis pinpointed age (OR =1.041; 95% CI: 1.022-1.060; P=0.000), MELD (OR =1.058; 95% CI: 1.022-1.095; P=0.001), LSVR (OR =3.091; 95% CI: 1.265-7.555; P=3.091) and TLV/SV (OR =0.863; 95% CI:0.770-0.969; P=0.012) as independent predictors for all-cause mortality (Table 2). Age, MELD score, LSVR, and TLV/SV were associated with liver-related death in the univariate analysis. The multivariable analysis indicated that age (OR =1.025; 95% CI: 1.008-1.043; P=0.004), MELD score (OR =1.053; 95% CI: 1.018-1.090; P=0.003) and LSVR (OR =2.377; 95% CI: 1.012-5.581; P=0.047) were independent predictors for liver-related mortality (Table 3). Kaplan-Meier survival curves demonstrated significant differences in both overall survival (Figure 1) and liver-related survival (Figure 2) between high and low LSVR groups (Log-rank test, P<0.05). Subgroup analyses revealed that LSVR (OR =4.815; 95% CI, 1.140-20.332; P=0.032) were independent predictors for infection-related mortality. LSVR (OR =5.461; 95% CI, 1.809-16.488; P=0.003) and TLV/SV (OR =0.739; 95% CI, 0.571-0.956; P=0.020) were independent predictors of oncology-related mortality. Conclusions: LSVR can independently predict both all-cause and liver-related mortality in cirrhotic patients, supporting its potential role as a complementary prognostic tool for risk stratification and clinical decision-making in the health care of cirrhosis.

Keyword(s) : liver to spleen volume ratio (LSVR), total liver volume (TLV), spleen volume (SV), computed tomography, cirrhosis, all-cause mortality, liver-related mortality

1305 - Fenofibrate and ursodeoxycholic acid in treatment naïve primary biliary cholangitis a pilot randomized trial with integrated multi omics analysis

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Background / Hypothesis: Primary biliary cholangitis (PBC), a chronic autoimmune liver disease, involves the destruction of interlobular bile ducts, resulting in cholestasis and liver fibrosis. Currently, ursodeoxycholic acid (UDCA) is the sole first-line therapy for PBC, and refractory patients can be treated with a supplementary fenofibrate therapy.

However, it remains unclear whether fenofibrate monotherapy can directly improve the biochemical indicators of patients with PBC. This study conducted a preliminary investigation of the efficacy and safety of fenofibrate monotherapy in treatment-naïve patients with PBC and analyzed the metabolic changes and potential mechanisms associated with different treatments.

Materials and Methods: Thirty treatment-naïve PBC patients were enrolled in a 6-month, randomized, parallel-controlled, open-label pilot trial and randomly assigned in a 1:1 ratio to two groups: (1) UDCA (13–15 mg/kg/d) and (2) fenofibrate (once daily, 200 mg) monotherapy. The primary endpoint was the normalization rate of alkaline phosphatase (ALP). The secondary endpoints were changes in pruritus severity, liver stiffness, and prognostic scores. Adverse events during the treatment period were also recorded. Plasma samples collected from patients at baseline, 1, and 6 months post-treatment and from 16 healthy controls underwent multi-omics analysis, including the Olink Reveal assay, bile acid quantification, and untargeted metabolomics.

Results:

After 6 months of treatment, 86.7% patients in the fenofibrate group achieved a normal ALP level compared to only 28.6% patients in the UDCA group (P < 0.001). The two groups showed no significant differences in pruritus severity, liver stiffness, or prognostic scores. Adverse events were detected in 73.3% and 80% of patients in the fenofibrate and UDCA groups, respectively, with no occurrence of severe adverse events. Fenofibrate treatment significantly increased the plasma FGF21 level. Moreover, UDCA treatment increased the levels of 7 bile acid (BA) components, whereas fenofibrate exhibited a minimal effect on BA. Additionally, fenofibrate and UDCA altered the levels of 71 and 62 lipid components, respectively.

Conclusions:

Fenofibrate substantially increased the ALP normalization rate in PBC patients, with an incidence of adverse events similar to that following UDCA treatment. Furthermore, fenofibrate more profoundly influenced metabolism-related indicators, while UDCA more significantly affected BAs. Thus, fenofibrate monotherapy appears to be a promising option for treating PBC patients.

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Keyword(s) : primary biliary cholangitis, fenofibrate, Olink, untargeted metabolomics

1308 - KDM5A Driven Epigenetic Reprogramming of IL1B⁺ CD14⁺ Monocytes Mediates Resistance to Anti-PD 1 Therapy in Advanced Hepatocellular Carcinoma by Suppressing T Cell Mediated Antitumor Immunity

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality globally, with anti-PD-1 immunotherapy emerging as a promising therapeutic strategy. However, the clinical efficacy of immune checkpoint blockade remains limited, primarily due to complex and poorly understood immune evasion mechanisms. In particular, circulating immune signatures involving monocytes may represent critical but underexplored determinants of therapeutic responses. This study aims to characterize circulating immune signatures in HCC patients treated with anti-PD-1 immunotherapy, elucidate the role and mechanisms of IL-1β⁺ CD14⁺ monocytes in tumor immune evasion, and identify novel targets to improve HCC immunotherapy outcomes.

Materials and Methods: Peripheral blood from 10 HCC patients undergoing anti-PD-1 treatment was analyzed via single-cell RNA sequencing (scRNA-seq). Paired tumor tissue samples from 4 patients receiving targeted immunotherapy were examined using multiplex immunofluorescence staining and spatial transcriptomics. To explore mechanisms, we integrated ATAC-seq analysis, in vitro monocyte-T cell co-culture experiments, and murine tumor models, combining single-cell and spatial omics with functional validation to comprehensively assess systemic immune responses to therapy.

Results: Responders exhibited a higher abundance of CD8⁺ T cells and fewer CD14⁺ monocytes compared to

non-responders. The accumulation of IL-1β⁺ CD14⁺ monocytes suppressed CD8⁺ T cell function and contributed to resistance. Spatial transcriptomics revealed increased infiltration of IL-1β⁺ CD14⁺ macrophages in non-responders, which interacted with T cells to inhibit antitumor activity—a pattern confirmed by multiplex immunofluorescence. Mechanistically, in non-responders, CD14⁺ monocytes showed synergistic upregulation of the immunosuppressive SOCS3 and pro-inflammatory IL-1β, leading to M1 macrophage reduction and CD8⁺ T cell dysfunction. These gene expression patterns were epigenetically regulated by KDM5A. In murine HCC models, combined IL-1β blockade and anti-PD-1 therapy decreased immunosuppressive factors, enhanced T cell infiltration, and significantly suppressed tumor progression and improved therapeutic efficacy.

Conclusions: IL-1β⁺ monocytes play a key role in suppressing CD8⁺ T cell-mediated antitumor immunity. Targeting these monocytes enhances T cell responses and synergizes with anti-PD-1 therapy, offering a promising strategy to improve HCC immunotherapy outcomes.

Keyword(s) : Hepatocellular carcinoma; Single-cell RNA sequencing; IL-1β⁺ CD14⁺ monocytes; Prognosis; Biomarker

1310 - Creb1 regulated slc39a1 attenuates alcoholic liver disease by orchestrating autophagy and mitophagy

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Background / Hypothesis:

Alcoholic liver disease (ALD) remains a leading cause of liver-related morbidity globally, yet its molecular mechanisms are incompletely defined. Zinc transporter SLC39A1, implicated in ion homeostasis, is hypothesized to play a critical role in ALD pathogenesis. This study aims to decipher a hitherto unrecognized SLC39A1-dependent pathway that confers protection against ALD.

Materials and Methods:

We established hepatocyte-specific Slc39a1 knockout (CKO) mice and employed Lieber-DeCarli ethanol feeding in vivo and ethanol-stimulated primary

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hepatocytes in vitro. Liver injury, steatosis, cell autophagy and mitophagy were assessed. Transcriptional regulation of SLC39A1 by CREB1 was evaluated via ChIP and luciferase assays. Mechanistic insights into SLC39A1-DRP1 interaction and mitophagy were explored through genetic and pharmacological interventions.

Results:

Analysis of the GEO database and human ALD samples revealed a significant upregulation of SLC39A1, which negatively correlated with severity of liver injury—evidenced by reduced liver damage, steatosis (HE), inflammation (TGF- β), and fibrosis (Masson), suggesting a protective role in patients. Consistent with human data, SLC39A1 was also significantly elevated in the livers of ethanol-fed mice and associated with improved survival. Slc39a1 CKO mice developed exacerbated ethanol-induced liver injury and steatosis compared to wild-type littermates. Mechanistically, SLC39A1 deficiency enhanced autophagy, as evidenced by increased LC3IIB and Atg5 expression and elevated autophagic flux—effects reversible upon autophagy inhibitor chloroquine (CQ). Importantly, CQ treatment in Slc39a1 CKO mice attenuated liver injury, reduced steatosis, and improved survival, underscoring the pathogenic role of excessive autophagy in this context. Further investigation identified CREB1 as a direct transcriptional regulator of SLC39A1 via ChIP-seq data and validated by chromatin immunoprecipitation, showing enhanced promoter binding upon ethanol challenge. Furthermore, SLC39A1 physically interacted with DRP1, promoting mitochondrial fission and Fundc1-mediated mitophagy. Either genetic or pharmacological inhibition of DRP1 suppressed SLC39A1-driven mitophagy, indicated by abolished Fundc1 induction. Conversely, DRP1 overexpression rescued impaired mitophagy and attenuated apoptosis in SLC39A1-silenced hepatocytes.

Conclusions: Our findings unveil a cytoprotective axis in which CREB1-driven SLC39A1 curbs alcohol-induced liver injury through dual regulation of autophagy and DRP1-dependent mitophagy. This mechanistic insight not only delineates a novel signaling pathway in ALD pathogenesis but also nominates SLC39A1 as a compelling therapeutic target for clinical intervention in alcoholic liver disease.

Keyword(s)

CREB1, SLC39A1, DRP1, autophagy, mitophagy

1311 - Liver Centric Therapeutic Profiling of 2 β Hydroxybetulinic Acid 3 β Caprylate (HBAC) A Phyto Derived Modulator of Hepatic Redox Balance Gluconeogenesis and Lipotoxic Stress in Experimental Diabetes

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Background / Hypothesis: Natural triterpenoids are promising antidiabetic agents with hepatoprotective potential. We evaluated HBAC (2 β -hydroxybetulinic acid 3 β -caprylate), isolated from Euryale ferox seeds, for antidiabetic, anti-oxidant and hepatic metabolic effects in streptozotocin (STZ)-induced diabetic rats.

Materials and Methods: HBAC was purified by column chromatography and characterized by NMR/IR/MS (designated HBAC). Male Wistar rats received STZ (60 mg/kg i.p.) to induce diabetes. HBAC was administered orally at 20, 40 and 60 mg/kg once daily for 45 days. Assessments included fasting plasma glucose, plasma insulin, glycosylated hemoglobin (A1c), HOMA-IR, hepatic gluconeogenic enzymes (G6Pase, F-1,6-BPase, G6PD, hexokinase), lipid profile (TC, LDL, HDL, TG), antioxidant markers (SOD, CAT, GPx, GSH, MDA) and histopathology of pancreas, liver and kidney. (n=5/group).

Results: HBAC produced dose- and time-dependent reductions in fasting glucose and A1c, with the 60 mg/kg dose restoring glucose and A1c toward normal and significantly increasing plasma insulin versus diabetic controls. HOMA-IR was significantly improved, indicating reduced insulin resistance. HBAC restored disturbed hepatic gluconeogenic enzyme activities and increased hexokinase and G6PD activities (p<0.01). Lipid abnormalities ($\uparrow$ TC, $\uparrow$ LDL, $\uparrow$ TG, $\downarrow$ HDL) were ameliorated, and antioxidant status (SOD, CAT, GPx, GSH) was significantly restored while MDA decreased (p<0.01). Histology showed partial regeneration of pancreatic islets and reversal of STZ-induced liver/kidney damage in HBAC-treated groups. Effects were comparable to/ exceeded those of conventional reference drug at the highest HBAC dose.



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Conclusions: HBAC (2 β -hydroxybetulinic acid 3 β -caprylate) exhibits potent antidiabetic activity in STZ rats by improving glycemic control, reducing insulin resistance, normalizing hepatic gluconeogenesis and restoring antioxidant defenses, with supportive histopathology. These data suggest HBAC is a promising candidate for further preclinical development as an antidiabetic agent with hepatic protective properties.

Keyword(s) : HBAC (2 β -Hydroxybetulinic Acid-3 β -Caprylate), Hepatoprotection, Triterpenoid, Euryale Ferox Salisb.

1312 - Multiparametric magnetic resonance imaging to risk stratify patients with chronic liver disease **Descriptive results from the ELEGANCE study**

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Background / **Hypothesis:**
The ELEGANCE study is a prospective, multi-centre, cohort study enrolling 2000 participants with chronic liver disease, at risk of developing hepatocellular carcinoma (HCC) in Singapore. Participants undergo standard-of-care 6-monthly ultrasound imaging and alpha-fetoprotein (AFP) testing for HCC surveillance, and provide clinical biochemistry and bio-samples for metabolomic, microbiome and miRNA profiling. A sub-cohort undergoes advanced phenotyping using non-contrast, multiparametric MRI (mpMRI) with preliminary results presented herein.

Materials and **Methods:**
mpMRI were acquired on clinical scanners and

processed with LiverMultiScan and Hepatica softwares (Perspectum, UK). LiverMultiScan provides fibro-inflammation (from iron corrected T1, cT1), liver fat content (LFC, from proton density fat fraction [PDFF]) and liver iron content (from T2* mapping). Hepatica returns whole liver volume and volume for each Couinaud segment. Spleen volume is also calculated. Descriptive statistics report mpMRI measurements across different aetiologies. A logistic regression model was developed to evaluate the utility of mpMRI for identifying cirrhosis.

Results:

At the time of analysis, N=732; 124 with metabolic dysfunction associated liver disease (MASLD), 328 chronic hepatitis B (HBV), 7 chronic hepatitis C (HCV), and 273 clinically diagnosed cirrhosis of varying aetiologies.

In MASLD (mean age 59 years, 40% female, mean BMI 26), mean liver cT1 was 770ms, LFC 14%, liver iron 0.97mg/g and volume 1.4L. The prevalence of lean-MASLD (defined as LFC $\geq$ 5% and BMI<23) was 17%, the prevalence of MASH (defined as cT1 $\geq$ 800ms) was 31% and fibrotic-MASH (cT1 $\geq$ 875ms) was 6%. In HBV (mean age 61 years, 44% female, mean BMI 24) mean liver cT1 was 717 ms, LFC 6%, liver iron 0.91 mg/g and volume 1.1L. 41% of the HBV patients had co-morbid MASLD as defined by LFC $\geq$ 5%. In the cirrhotic group (mean age 65 years, 40% female, mean BMI 26), mean liver cT1 was 773ms, LFC 6%, liver iron 0.9mg/g and volume 1.3L. A logistic regression model to classify cirrhosis using all the mpMRI variables distinguished those with or without cirrhosis with an area under the receiver operator curve (AUC) of 0.88 in the MASLD sub-group and 0.80 for the entire cohort (Figure 1).

Conclusions:

mpMRI enabled the identification of participants along the continuum of liver disease from simple steatosis to MASH and cirrhosis and aided in the identification of co-morbid MASLD and viral hepatitis. Accurately phenotyping patients with chronic liver disease for disease severity may aid in identifying those at highest risk of developing subsequent liver related events including cancer.

Keyword(s) : chronic liver disease, multiparametric MRI, cancer surveillance

1327 - VDAC1 ACSL3 mediated aberrant lipid droplet mitochondria crosstalk to promote HCC in CHB and MASLD coexistence

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Background / Hypothesis: Chronic hepatitis B (CHB) and metabolic dysfunction-associated steatotic liver disease (MASLD) are constituting a growing global health burden. Notably, their co-occurrence is linked to dramatically worsened clinical outcomes, including rapid fibrosis progression and a heightened incidence of hepatocellular carcinoma (HCC), pointing to a vicious synergy between viral infection and metabolic dysregulation. However, the core mechanisms underlying this convergence, particularly how MASLD reshapes the CHB liver to drive oncogenesis, are poorly defined. Our study seeks to decipher these key mechanistic drivers, focusing on dysregulated inter-organelle communication and metabolic reprogramming.

Materials and Methods: Liver biopsies from patients with simple MASLD (n=7), CHB (n=10), CHB complicated by MASLD (n=12), and controls (n=5) were subjected to spatial transcriptome sequencing. A mouse model of CHB concomitant with MASLD was established via rAAV8-1.3 HBV hydrodynamic injection followed by high-fat, high-cholesterol diet (HFHC) or high-fat methionine-restriction choline-deficient diet (HFMRCd) feeding. Proteins mediating lipid droplet-mitochondria tethering were profiled by LC-MS/MS. VDAC1-ACSL3 interaction and USP9X-mediated deubiquitination were confirmed by IP-MS and site-directed mutagenesis. An orthotopic HCC model using RIL-175 cells expressing wild-type or K365A-mutant ACSL3 were established to assess the tumorigenesis capacity.

Results: Spatial transcriptomics identified pronounced mitochondrial dysfunction and a distinct immune landscape, marked by significant enrichment of tumor-associated macrophages in livers of patients with CHB complicated by MASLD, even during the immune-

tolerant phase. Mechanistically, hepatocytes in this disease context exhibited upregulated expression and oligomerization of the mitochondrial outer membrane protein VDAC1. VDAC1 directly interacted with acyl-CoA synthetase long-chain family member 3 (ACSL3), whose protein stability was enhanced through K365-dependent deubiquitination by USP9X. The formation of the VDAC1-ACSL3 complex facilitated aberrant lipid droplet-mitochondria contact, leading to exacerbated oxidative stress, severe mitochondrial impairment, and metabolic reprogramming characterized by excessive lactate production, thereby fueling a procarcinogenic niche. Crucially, the ACSL3 K365A mutation disrupted USP9X-dependent deubiquitination and led to increased ACSL3 degradation, reduced ACSL3-VDAC1 interaction, normalized mitochondrial function, attenuated lactate generation and potently suppressed tumor formation in an orthotopic model. Furthermore, VDAC1 oligomerization triggered mitochondrial DNA release, activating the cGAS-STING pathway and subsequent interferon-stimulated gene responses, which contributed to the suppression of HBV replication.

Conclusions: Our findings identify the VDAC1-ACSL3-mediated lipid droplet-mitochondria tethering as a novel pathogenic mechanism that drives metabolic stress and microenvironment reprogramming in CHB complicated by MASLD. This axis represents a promising therapeutic target to mitigate hepatocarcinogenesis in this high-risk patient population.

Keyword(s) : CHB, MASLD, lipid droplet-mitochondria tethering, mitochondrial dysfunction, metabolic reprogramming, VDAC1, ACSL3

1328 - CRISPR Prime Targeting of Interleukin 1 Receptor Type 1 (IL1r1) in Stellate Cells Identifies a Pathogenic Inflammation to Fibrosis Axis in the Liver
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Background / Hypothesis: Liver fibrosis, a central driver of chronic liver disease, arises from maladaptive activation of hepatic stellate cells (HSCs) and excessive extracellular matrix deposition. Although transforming growth factor beta (TGF β) is a well-established fibrogenic signal, the interplay between inflammatory cues and TGF β -driven fibrogenesis is incompletely understood. We developed a novel mouse model of fibrosis and analysed livers using a NanoString nCounter Fibrosis Panel. Potential

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genetic targets, including interleukin-1 receptor type 1 (IL1r1), were identified and their role in fibrosis and modulating TGF β response examined.

Materials and Methods:

We generated a novel CRISPR HSC model with heterozygous and homozygous knockout of IL1r1. Successful IL1r1 disruption was confirmed by immunofluorescence, flow cytometry, and functional loss of Interleukin-1 beta (IL1 β) responsiveness. Fibrotic activation was probed in two-dimensional monocultures and physiologically relevant three-dimensional co-cultures, quantifying alpha-smooth muscle actin (α -SMA) and collagen type I. Pharmacologic modulation with the antioxidant N-acetylcysteine (NAC) assessed the contribution of redox-sensitive pathways.

Results:

IL1r1 deficiency markedly attenuated TGF β -induced myofibroblastic transformation, with homozygous knockouts exhibiting the largest reductions in α -SMA and collagen I expression ($p < 0.01$). NAC further suppressed fibrotic marker induction across genotypes, implicating oxidative stress as an amplifier of TGF β signalling downstream or in parallel to IL1r1. Collectively, these data establish the major role of IL1r1 linking inflammatory and profibrotic signalling.

Conclusions:

This study introduces a novel CRISPR-engineered HSC model and identifies IL1r1 as a critical modulator of fibrosis. These findings reveal an inflammation fibrosis axis and highlight IL1r1 as a potential therapeutic target for liver disease.

Keyword(s) :

1331 - Evaluation of Hepatitis B Serology Vaccination Rate and Reactivation in Hepatitis C Patients Treated with Direct Acting Antivirals

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Background / Hypothesis: In patients infected with HCV treated with DEAs, HBV reactivation may occur during or after treatment. Therefore, HBV serology should be

checked before treatment, treatment should be given to patients requiring HBV treatment, and patients at risk of reactivation should be monitored. HBV vaccination is also recommended for patients susceptible to HBV. Our study aimed to retrospectively screen patients diagnosed with HCV and treated with DAAs to determine hepatitis B screening rates and serology in these patients, as well as to determine hepatitis B vaccination rates and hepatitis B reactivation rates based on the screening results.

Materials and Methods: 170 patients were retrospectively reviewed. The patients' demographic characteristics, laboratory findings, treatments received, treatment responses, HCV genotype, HBV serology, HBV reactivation rates and HBV vaccination rates were investigated. Groups at risk for HBV reactivation were compared. Statistical analysis of the data obtained from the patients was performed using IBM SPSS Statistics for Windows version 26.0. Statistical data were evaluated using the Kolmogorov Smirnov, Student t, Mann Whitney U and Chi-square tests. A significance level of $p < 0.05$ was accepted.

Results: When the demographic characteristics of the included patients were analysed, 94 (55.3%) were male and 76 (44.7%) were female. The mean age was found to be 63 ± 14.3 years. When the serology of 170 patients were analysed, HBsAg positive; 3 patients (1.8%) and HBsAg negative; 167 patients (88.2%) were found. In HBsAg negative patients, HBsAg negative/anti HBs negative; 114 patients (68.2%), HBsAg negative/anti HBs positive; 53 patients (31.8%). HBsAg-negative patients were divided into subgroups according to their serological characteristics for vaccination and reactivation evaluation. HBsAg negative/anti-HBs negative/anti HBc IgG negative 75 patients (44.9%), HBsAg negative/anti-HBs negative/anti HBc IgG positive 15 patients (9%), HBsAg negative/anti-HBs negative/anti HBc IgG unknown 24 patients (14%), HBsAg negative/anti-HBs positive/anti HBc IgG negative 11 patients (6.6%), HBsAg negative/anti-HBs positive/anti HBc IgG positive 42 patients (25.1%). In terms of reactivation, HBV reactivation was not detected in 3 HBsAg positive patients, while HBV reactivation was detected in 1 (1.75%) of 57 anti-HBc positive patients. In our study, HBV vaccination was initiated in a total of 23 patients diagnosed with CHC. It was observed that 20.2% (23/114) of HBsAg negative/anti-HBs negative patients and 20% (15/75) of HBsAg negative/anti-HBs negative/anti-

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HBc IgG negative patients were vaccinated.

Conclusions: HBV reactivation may be observed during HCV treatment with DEA drugs. Therefore, HBV serology should be checked before treatment, treatment should be given to patients who require it, patients at risk of reactivation should be identified and closely monitored, and patients who are susceptible to HBV should be vaccinated. In our study, no reactivation was observed in HBsAg-positive patients, while reactivation was observed in one anti-HBc IgG-positive patient. Therefore, when screening patients who will receive DEA for HBV, anti-HBc IgG should be checked along with HBsAg.

Keyword(s) : Hepatitis B, hepatitis C, reactivation, serology, vaccination

1334 - Time Restricted Feeding Improves Hepatic Fibrosis in Advanced MASLD Preliminary Results from a Randomised Controlled Trial

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Background:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is among the most prevalent chronic liver diseases globally, driven by metabolic syndrome, obesity, and insulin resistance. Progressive MASLD can advance to steatohepatitis, fibrosis, and cirrhosis, contributing to significant liver-related morbidity. Lifestyle modification remains the mainstay of therapy, with weight loss through caloric restriction shown to improve hepatic steatosis and fibrosis. However, adherence to continuous calorie restriction is difficult. Time-restricted feeding (TRF), a form of intermittent fasting that restricts daily caloric intake to a fixed window is a potentially sustainable approach. This study compared the effects of TRF versus standard medical therapy on hepatic and metabolic outcomes in advanced MASLD.

Methods

In this prospective, randomised, single-centre trial, adults with advanced MASLD were assigned 1:1 to standard medical therapy (control) or TRF (8-hour feeding window). This abstract presents interim results from data collected over the first 8 weeks from 40 participants. We analysed the change in controlled attenuation parameter (CAP, dB/m) and liver stiffness (kPa) by transient elastography, changes in liver enzymes (ALT, AST, ALP, GGT, bilirubin) and metabolic indices (HbA1c, LDL, triglyceride levels). Between-group comparisons were made using Welch's t-test or Mann-Whitney U test ($p < 0.05$).

Results:

Of the 40 enrolled participants, complete paired data were available for 17 control and 17 TRF participants for primary elastography analysis. Steatosis (CAP): Both groups showed minimal changes from baseline to week 8. Mean Δ CAP was 0.65 ± 33.4 dB/m in controls versus 3.35 ± 45.9 dB/m in TRF ($p = 0.85$), indicating no statistically significant difference in hepatic fat regression. Fibrosis (kPa): A significant improvement was seen in the TRF group (-3.12 ± 3.48 kPa) compared with controls (-0.02 ± 2.39 kPa), with a between-group difference of 3.10 kPa (95% CI 1.01–5.19, $p = 0.005$). This represents a statistically significant fibrosis regression between the TRF and the control arm. Liver Enzymes: Both groups showed reductions in ALT, AST, and GGT, but differences were not significant (all $p \geq 0.46$). Metabolic Indices: Mean changes in HbA1c, LDL, and triglycerides were small and comparable (all $p > 0.27$).

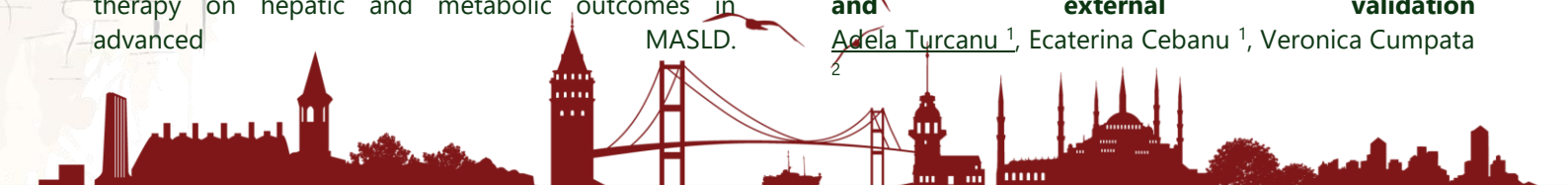
Conclusions:

TRF led to significant improvement in liver fibrosis compared with standard caloric restriction, despite minimal differences in steatosis, liver enzymes, or metabolic indices. These interim findings support TRF as a promising dietary strategy with potential antifibrotic effects. Ongoing recruitment and longitudinal follow-up will determine whether these effects are sustained and reproducible in a larger cohort.

Keyword(s) : Metabolic dysfunction-associated steatotic liver disease, time-restricted feeding, intermittent fasting, liver fibrosis, metabolic indices

1335 - A 7 item discharge risk index predicts 12 month decompensation in HDV cirrhosis derivation and external validation

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Background / **Hypothesis:**
HDV cirrhosis progresses rapidly, and is a frequent reason for hospitalization involving complex medical needs and carries a high risk of death in hospital. Therefore, a practical, discharge-time tool is needed to stratify risk of early decompensation in these patients. Aim of the study: to derive and externally validate a 7-item Decompensation Risk Index (bedside score) for HDV cirrhosis.

Materials and **Methods:**
Two real-world cohorts with HDV cirrhosis patients were analyzed: Derivation cohort (DC) - 104 patients and Validation cohort (VC) - 101 independent HDV cases. Exclusion criteria were: other associated liver diseases (autoimmune hepatitis, cholestatic liver diseases, hereditary liver diseases, HCV), HIV, malignancies. Twelve-month decompensation was defined as any of ascites, hepatic encephalopathy, or variceal hemorrhage. The Index assigns points for: MELD-Na ≥ 15 (+2), Child-Pugh ≥ 7 (+2), albumin < 30 g/L (+2), sodium < 130 mmol/L (+2), platelets $< 50 \times 10^9$ /L (+2), any baseline complication (+1), male sex (+1). Predefined categories: < 5 points = low risk, 5–7 = medium, ≥ 8 = high. Discrimination was assessed by AUC-ROC; calibration by calibration-in-the-large/slope and Brier score; validation category event rates and pragmatic thresholds were reported.

Results:

DC - 104 HDV-cirrhosis patients, median age 56 years; 51.5% male, AUC 0.907, PPV 64%; NPV 100%. In VC - median age 55.7 ± 8.8 years; 48 % male; AUC = 0.933, indicate excellent discrimination of patients with/without decompensation. Pragmatic thresholds: ≥ 1 point: Sensitivity 100%, Specificity 86.9%; ≥ 3 points: Sensitivity 53.8%, Specificity 98.4%. The results support the integration of the score into the discharge form for triage of follow-up frequency (1–3 months for high risk, 3 months for medium, 6 months for low).

Conclusions:

A discharge-ready, 7-item Index shows robust discrimination and acceptable calibration for predicting 12-month decompensation in HDV cirrhosis, with actionable thresholds to tier surveillance intensity. Prospective implementation and impact evaluation are

warranted.

Keyword(s) : decompensation, liver cirrhosis, HDV
1336 - Can poor sleep signal fatty liver disease Evidence from a rural population in North India
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Background / **Hypothesis:**
Metabolic-dysfunction associated fatty liver disease (MAFLD) is a major emerging liver disorder, including in lean populations. While metabolic risk factors are well-studied, rural populations remain underrepresented in such research, despite their high burden of unrecognized MAFLD. Poor sleep may influence hepatic steatosis via insulin resistance, inflammation, and circadian rhythm disruption. The study hypothesized that poor sleep quality and short duration are independently associated with high MAFLD risk, and that sleep parameters could serve as non-invasive, scalable risk markers.

Materials and **Methods:**
This community-based cross-sectional study recruited 550 adults (≥ 18 years) residing in catchment area of a primary health centre in North India. Sleep quality and duration were assessed using the Pittsburgh Sleep Quality Index (PSQI); a global score > 5 indicated poor sleep. Sleep duration was categorized as < 6 h (short), 6–8 h (normal), > 8 h (long). MAFLD risk was evaluated using a validated non-laboratory 15-point Self-Assessment Score (≥ 8 = high risk). Descriptive statistics, chi-square, and multivariable logistic regression were used. Additional analyses included ROC curve for predictive validity of PSQI, test for trend across sleep duration categories, and interaction analysis with BMI and sex.

Results:

Among 550 participants (mean age: 42.6 ± 13.5 years; 56.4% female), 38.2% had poor sleep quality and 31.6% had high MAFLD risk. High MAFLD risk was observed in 47.9% of poor sleepers vs 22.1% of good sleepers ($p < 0.001$). MAFLD risk was also elevated in short sleepers (44.6%) vs normal (28.5%) and long sleepers (23.8%) ($p = 0.002$; p -trend < 0.01). On multivariable regression, poor sleep quality (AOR=2.31; 95% CI: 1.58–3.38) and short sleep (AOR=1.76; 95% CI: 1.15–2.69) were independently associated with high MAFLD risk. ROC analysis yielded an AUC of 0.71 (95% CI: 0.65–0.77)

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for PSQI in predicting high MAFLD risk. Significant interaction was noted between poor sleep and elevated BMI (p-interaction=0.03), indicating amplified risk in overweight individuals.

Conclusions:

Poor sleep quality and short sleep duration are strong, independent predictors of MAFLD risk, with moderate discriminatory ability on ROC analysis. These findings suggest that sleep assessment could serve as a low-cost, non-invasive surrogate marker for hepatic risk stratification in primary care. Integrating sleep-focused screening into MAFLD preventive strategies may improve early detection and intervention, especially in resource-constrained settings.

Keyword(s) : metabolic-dysfunction associated fatty liver; MAFLD; Sleep quality; Sleep duration; Rural population; community-based

1337 - Analysis of Factors Associated with Liver Fibrosis in Type 2 Diabetes Patients and the Predictive Value of CHI3L1 for Liver Fibrosis

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Background / Hypothesis:

Type 2 diabetes mellitus (T2DM) is the most common chronic metabolic disease globally, with its comorbidity with metabolic dysfunction-associated steatotic liver disease (MASLD) becoming increasingly prevalent. The prevalence of MASLD is significantly increased in patients with Type 2 diabetes mellitus (T2DM), elevating the risk of cirrhosis and hepatocellular carcinoma. Therefore, early diagnostic assessment of liver fibrosis in T2DM patients is critically important. Our study investigated the factors associated with liver fibrosis in T2DM patients with comorbid MASLD and evaluated the predictive value of chitinase-3-like protein 1 (CHI3L1) for liver fibrosis in these patients.

Materials and Methods:

This is a retrospective study that enrolled T2DM patients who received care at Tianjin Third Central Hospital between January 2023 and August 2024 as research subjects. Liver fibrosis was assessed in both the total T2DM population and in T2DM patients with comorbid MASLD using liver stiffness measurement (LSM) and non-invasive assessment indicators including APRI, FIB-4, and CHI3L1. The LSM value ≥ 8 kPa was established as the threshold for significant liver fibrosis

(SLF). Correlation analysis was applied to evaluate the relationships between LSM, CHI3L1, and other indicators. Univariate and multivariate logistic regression analyses were conducted to identify risk factors associated with liver fibrosis, and receiver operating characteristic (ROC) curve analysis was used to assess the predictive value of CHI3L1. Results:

This study included 236 patients with T2DM, of whom 176 had comorbid MASLD (prevalence rate: 74.6%) and 89 had significant liver fibrosis (prevalence rate: 37.7%). Among T2DM-MASLD patients, 74 had significant liver fibrosis, with a prevalence rate of 42%. In the T2DM-MASLD population, patients with significant liver fibrosis demonstrated significantly higher levels of BMI, TSH, AST, GGT, LDH, CHI3L1, APRI, LSM, and CAP compared to the non-significant fibrosis subgroup, with all differences being statistically significant ($P < 0.05$). Conversely, the significant fibrosis subgroup exhibited significantly lower levels of PLT, LDL-C, and PWR compared to the non-significant fibrosis subgroup ($P < 0.05$). CHI3L1 showed positive correlation with LSM ($r = 0.48$, $P < 0.001$). Multivariate logistic regression analysis identified CHI3L1, APRI, and CAP as potential independent risk factors for significant liver fibrosis in T2DM-MASLD patients. The area under the ROC curve (AUC) for CHI3L1 in predicting significant liver fibrosis was 0.717 (95% CI: 0.638-0.796, $P < 0.05$), with an optimal cutoff value of 96.6 ng/mL. Conclusions:

The prevalence of MASLD among T2DM patients was 74.6%, while the prevalence of significant liver fibrosis was 37.7%.

Keyword(s) : Type 2 diabetes mellitus ; fibrosis ; chitinase 3-like protein

1338 - Validation of the 2025 Global Expert Panel Consensus Definition of ACLF in Real World Hospitalized Patients With Cirrhosis

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Background / Hypothesis:

The Defining Organ Failures in Patients With Cirrhosis: Consensus Statements (hereafter referred to as the Global Expert Consensus, GEC) proposed a new diagnostic framework for acute-on-chronic liver failure (ACLF) and introduced updated criteria for the definition of organ failures. However, this definition has not

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undergone systematic evaluation in independent clinical cohorts, and its evidence base remains limited. This study aimed to assess the clinical applicability and short-term prognostic discrimination of the GEC definition of ACLF.

Materials and Methods: We retrospectively included patients with cirrhosis and acute hepatic deterioration (defined as TB $\geq$ 5 mg/dL and INR $\geq$ 1.5) admitted to Beijing You'an Hospital between 2020 and 2025. ACLF was classified according to both the GEC and EASL-CLIF definitions, while organ failures were assessed using GEC criteria. The primary outcomes were 28-day and 90-day transplant-free survival (TFS).

Results:

A total of 410 patients were consecutively enrolled. The proportions of patients fulfilling the GEC and EASL-CLIF definitions of ACLF were 44.9% (184/410) and 46.3% (190/410), respectively. Compared with non-ACLF patients, those identified by the GEC criteria had a higher incidence of infection (89.7% vs. 77.4%, $P = 0.001$) and significantly lower TFS at both 28 and 90 days (56.0% vs. 91.6% and 40.8% vs. 87.2%, respectively; all $P < 0.001$). When compared with EASL-CLIF, the GEC definition demonstrated higher AUCs for predicting 28-day and 90-day TFS (28-day: 0.744 vs. 0.685, $P = 0.056$; 90-day: 0.760 vs. 0.682, $P = 0.015$), indicating overall superior risk stratification and short-term prognostic discrimination. In addition, the GEC showed better concordance (C-index: 0.705 vs. 0.641, $P = 0.003$). Sensitivity analyses showed a stepwise decline in TFS as the number of organ failures increased from 0 to ≥ 4 (all $P < 0.001$).

Conclusions: The GEC definition of ACLF shows robust short-term prognostic performance in hospitalized patients with cirrhosis and acute deterioration. Moreover, GEC-based organ failure stratification further refines assessment of disease severity. To our knowledge, this study represents the first clinical validation of the GEC definition in hospitalized cirrhotic patients, supporting its value as a more effective tool for short-term prognostic evaluation.

Keyword(s) : Acute-on-chronic Liver Failure; Global Expert Panel Consensus; Transplant-free Survival; Organ Failure; Prognostic Discrimination.

1341 - Screening of new active molecules derived from ursodiol in the treatment of primary biliary cholangitis and ursodeoxycholic acid

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Background / Hypothesis: Primary biliary cholangitis (PBC) is a chronic autoimmune cholestatic liver disease that primarily affects the biliary system, leading to progressive hepatocellular damage and fibrosis. Despite ursodeoxycholic acid (UDCA) being the first-line and standard therapy that improves biochemical parameters and delays histological progression, approximately 20–40% of patients show suboptimal or no response to treatment. Moreover, UDCA does not sufficiently alleviate clinical symptoms in some cases. Therefore, the discovery and design of novel ursodiol-derived compounds with enhanced therapeutic efficacy and improved pharmacokinetic profiles represent a promising research direction for optimizing PBC management.

Materials and Methods: In this study, a series of ursodiol derivatives were virtually screened using in silico computational approaches to identify potential candidates with improved binding affinity and pharmacological properties. Molecular docking analyses were performed to evaluate interactions with key PBC-related molecular targets, followed by ADME profiling to assess drug-likeness and pharmacokinetic behavior. Additionally, MM-GBSA calculations were conducted to estimate binding free energies and refine compound selection. The results of this comprehensive in silico screening will be presented, providing insights into the structural determinants that may guide the development of next-generation UDCA-based therapeutics for PBC treatment. Total 118 organic compounds were taken into consideration. The whole compounds were downloaded from PUBCHEM. Maestro (Version 13.7.125) was used all calculations.

Results: All results were compared with that of ursodiol. Three different protein structures were used in this study which PDB ID's are 1OSH, 3DCT and 4QE6. In molecular docking analyses, four different organic compounds were found as potential due the having better results than that of ursodiol. Their PUBCHEM ID's are 10133, 11281160, 11731984, and 16217615. Their ADME, MM-GBSA and CYP2C9 p450 cytochrome analyses were

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completed.

Conclusions: In conclusion, all of these compounds were found to be good candidates to replace ursodiol and may be utilized to optimize the management of PBC. However, further and more comprehensive studies are required in this direction.

Keyword(s) : Primary biliary cholangitis, Ursodiol derivatives, In silico screening, Molecular docking, UDCA alternatives

1344 - A single VRON 0200 prime dose induced sustained and or continued HBsAg declines up to one year post dosing and rapid HBsAg declines with addition of antiviral agents in chronically HBV infected patients

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Background / Hypothesis: Current and investigational HBV Functional Cure treatments have been limited by their inability to restore anti-HBV immunity, leading to off treatment viral rebound. VRON-0200 is an immune-modulator containing a checkpoint modifier, with HBV core and pol (but not S) antigens, designed to enhance, broaden, and prolong T cell responses. Here, we report safety, immunogenicity, and HBsAg data from chronically-HBV infected patients(pts) that received VRON-0200 alone, and in combination with investigational antivirals, when added to ongoing NUC therapy.

Materials and Methods: CHB pts, NUC-suppressed with HBsAg <500IU/mL(Cohorts 1(C1) and 2(C2)), or <1,000IU/mL(Cohort 3(C3)), were randomized to receive i.m. VRON-0200 low(C1) or high(C2) dose prime on D1, w/wo VRON-0200 boost on D91; C3 receive VRON-0200 prime, followed by 6 mthly doses of elebsiran plus tobevibart starting on D28, alone, or with a VRON-0200 boost on D196. Safety, virologic, and immunologic assessments were performed. HBsAg (ELISA; LLOD:0.05IU/mL), T cell frequencies (IFN γ ELISpot), core,

pol,S antigen)), and neutralizing antibody responses(NAbs) were measured.

Results: As of November 17,2025, 35 pts were included: 28(80%)male, 30(86%)Asian, median age:46 yrs, and median BL HBsAg,179 IU/mL(range:10–1169). In 11,959 pt safety days, 38 TRAEs were reported: 4-GR2,34-GR1, with no tx-related SAEs or D/Cs. In C1/2, 23 of 27 had reached EOS(D360): of those,19/23(83%) had sustained and/or continued HBsAg declines(9/19>50% declines), 4 pts had >1log10 IU/mL; a boost dose did not enhance responses, and declines were independent of BL HBsAg levels. In C3, within 7 days of the first antiviral dose, rapid and profound HBsAg declines were observed(100% <2 IU/mL; 1 LLOD) and were independent of BL HBsAg levels. Additional antiviral doses improved declines with 3 of 6 pts at D140 achieving HBsAg loss. IFN γ ELISpot responses were not associated with HBsAg declines; immunogenic responses to VRON-0200 were observed in all pts, regardless of pre-existing NAb status. Additional data to be reported.

Conclusions: VRON-0200 alone, or in combination, had no observed safety concerns, and was well tolerated. Despite not targeting HBsAg, a single VRON-0200 dose was able to induce broad anti-HBV immunity in the majority of pts, that was sustained and/or continued to decline up to one-year post dosing. When combined with antivirals, rapid and profound HBsAg declines were observed in all patients. This novel approach where anti-HBV immunity is "sparked" (i.e. primed) with VRON-0200, and then "fanned" (i.e. boosted) by HBV removal, could position VRON-0200 to be a foundational backbone agent for future HBV Functional Cure regimens.

Keyword(s) : HBV, Functional Cure, VRON-0200, HBsAg, Checkpoint Modifer, Therapeutic Vaccine

1346 - A Ductless Dilemma Pembrolizumab Induced versus Paraneoplastic Vanishing Bile Duct Syndrome

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Background / Hypothesis: To present a case of a 72-year-old male, known Urothelial Carcinoma Stage IV, Prostate Adenocarcinoma who developed progressive jaundice seven weeks after receiving his eighth cycle of Pembrolizumab

Materials and Methods:



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Patient was admitted for a two day history of beginning icteresia progressing to jaundice. Initial diagnostics showed conjugated hyperbilirubinemia; with SGPT and Alkaline Phosphatase both 13x and 6.7x the upper limit of normal, respectively. Workup for obstruction with Abdominal Ultrasound, Whole Abdominal CT-Scan, MRCP and ERCP were negative, showing only signs of patchy hepatic steatosis with no mass lesions. Hepatitis, infectious and autoimmune workup were also negative, and was managed initially as a case of immune-related adverse event from Pembrolizumab. Oral prednisone was initiated; however, due to increasing bilirubin and liver enzymes, shifted to pulse dose steroid therapy for 3 days and again maintained on oral prednisone, azathioprine and ursodiol afterwards. Serial monitoring showed a gradual but fluctuating decline in liver parameters. Liver biopsy was eventually pursued, revealing a cholestatic pattern of injury with associated 70% bile duct loss suggestive of Vanishing Bile Duct Syndrome (VBDS). Medications were continued with gradual decrease in liver enzymes and bilirubin levels; however, after a few days, values plateaued. Paraneoplastic syndrome was then considered, and the patient underwent Transurethral Resection of Bladder Tumor and Prostate. Steroids and Immunosuppressant were maintained post procedure and liver parameters eventually reached their lowest values after 2 weeks. Patient was discharged with close follow up recommended afterwards

Results:

With the clinical picture, diagnostics and histopathology obtained, Vanishing Bile Duct Syndrome secondary to Pembrolizumab versus a paraneoplastic process were considered. Paraneoplastic VBDS is an extremely rare phenomenon mostly associated with hematological malignancies, particularly Hodgkin's lymphoma rather than solid tumors. With this, a diagnosis of Pembrolizumab induced VBDS was still heavily favored. Management included prompt discontinuation of Pembrolizumab and the initiation of targeted supportive therapy, including ursodiol, systemic corticosteroids, and immunosuppressant

Conclusions:

Pembrolizumab-induced VBDS is a rare adverse event documented primarily through isolated case reports with no exact incidence rates available. Diagnosis relies heavily on histopathology findings of ductopenia defined as a loss of more than 50% of bile ducts with evaluation of at least ten portal tracts. Currently, no

standardized treatment regimen exists, however prompt discontinuation of the offending drug and supportive care with ursodiol, steroids and immunosuppressants have been suggested. Prognosis is variable, ranging from gradual recovery to progressive liver failure and in severe cases, mortality.

Keyword(s) :

1349 - Enhanced detection of hepatocellular carcinoma using combined biomarker panels including alpha fetoprotein alpha fetoprotein L3 des gamma carboxyprothrombin and the galad score

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Background / Hypothesis:Hepatocellular carcinoma (HCC) remains a major global health burden, with early detection being crucial for improving outcomes. However, conventional diagnostic methods, including imaging and alpha-fetoprotein (AFP), have limited sensitivity and specificity. Emerging biomarkers such as AFP-L3 and des-gamma-carboxy prothrombin (DCP/PIVKA-II), along with composite models like the GALAD score, have shown potential in enhancing HCC detection. Evidence on the diagnostic performance of GALAD in diverse populations, particularly in Southeast Asia, remains scarce. This study evaluated the diagnostic accuracy of AFP, AFP-L3, DCP, and GALAD, individually and in combination, for detecting HCC. Materials and Methods:A cross-sectional study was conducted at Tam Anh General Hospital, Ho Chi Minh City, Vietnam, from March to October 2025. A total of 83 patients with histologically confirmed HCC and 83 patients with chronic hepatitis or cirrhosis were enrolled. Serum AFP, AFP-L3, and DCP were quantified, and GALAD scores were calculated. Diagnostic performance was assessed using sensitivity, specificity, and area under the receiver operating characteristic curve (AUC). Biomarkers were compared individually and in combination, with subgroup analyses stratified by BCLC stage.

Results:AFP >8.5 ng/mL and AFP-L3 >3.85% yielded moderate sensitivity (72.3% and 77.1%) and high specificity (91.6% and 86.7%). DCP >33.5 mAU/mL and GALAD >2.19 demonstrated superior diagnostic accuracy, with sensitivities of 91.6% and 85.5%, specificities of 92.8% and 95.2%, and identical AUCs of

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0.957. Both DCP and GALAD significantly outperformed AFP and AFP-L3 ($p < 0.05$), with no significant difference between them ($p > 0.05$). Combined biomarker panels further improved performance, with the AFP–AFP-L3–DCP panel achieving the highest sensitivity (95.2%). In advanced HCC (BCLC $\geq B$), GALAD (AUC 0.939) outperformed AFP and AFP-L3 and was comparable to DCP. None of the biomarkers reliably differentiated very early (BCLC 0) from early-stage HCC (BCLC A). Conclusions: DCP and GALAD exhibit superior diagnostic accuracy compared with AFP and AFP-L3, particularly in advanced HCC. Combining multiple biomarkers further enhances detection, although none effectively distinguish very early from early-stage disease. These findings support the clinical utility of DCP and GALAD and underscore the need for improved strategies to identify very early-stage HCC.

Keyword(s) : AFP, AFP-L3, DCP, GALAD, HCC

1350 - Metabolic Syndrome is Independently Associated with 1 Year Readmission of Patients with Alcohol Associated Cirrhosis

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Background / Hypothesis:
To explore the relationship between metabolic syndrome and the 1-year readmission rate in patients with alcohol-associated cirrhosis, and to uncover potential factors that influence this relationship.

Materials and Methods:
This retrospective analysis encompassed 379 patients with alcohol-associated cirrhosis who were hospitalized from January 2012 to December 2023. Patients were categorized in two groups according to whether they had metabolic syndrome (MetS) or not, and comparisons were made regarding their baseline characteristics, liver function indicators and complications. Multivariate regression analysis was conducted to determine the independent factors influencing the 1-year readmission rate. In addition, stratified analysis was conducted to investigate how various factors might influence the relationship between MetS and the 1-year readmission rate.

Results:

Of the 379 patients with alcohol-associated cirrhosis, 40 (10.6%) patients had MetS, who exhibited a reduced occurrence of ascites (40.0% vs. 62.5%, $P = 0.006$) but a

higher rate of 1-year readmission (65.0% vs. 44.5%, $P = 0.014$) compared to those without MetS. In total, 177 patients (46.7%) were readmitted within 1 year. The incidence of MetS (65.0% vs. 44.5%, $P = 0.014$), hepatic encephalopathy (19.2% vs. 10.9%, $P = 0.023$) and variceal bleeding (13.6% vs. 5.0%, $P = 0.003$) was also considerably higher in individuals who were readmitted within 1 year compared to those not readmitted. Multivariate analysis indicated that MetS, hepatic encephalopathy, variceal bleeding and ALT levels were independent predictors of 1-year readmission. Stratified analysis showed no significant interaction between MetS and 1-year readmission rate across different subgroups stratified by age, BMI, hypertension, diabetes, hyperlipidemia, ALT levels, ALP levels and TBIL levels.

Conclusions:

Metabolic syndrome is one of the independent predictive factors for 1-year readmission in individuals with alcohol-associated cirrhosis, which influences the risk of readmission in conjunction with factors such as hepatic encephalopathy and variceal bleeding.

Keyword(s) : metabolic syndrome; alcohol associated cirrhosis; alanine aminotransferase; readmission; MELD score

1352 - Evaluation of Liver Fibrosis Using FIB 4 and ARFI in Non Diabetic NAFLD Patients

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Background / Hypothesis: Background and Aims: Non-alcoholic fatty liver disease (NAFLD) is a growing cause of liver fibrosis, even in non-diabetic populations. Non-invasive methods such as the Fibrosis-4 (FIB-4) index and Acoustic Radiation Force Impulse (ARFI) elastography are increasingly utilized, yet data in non-diabetic patients remain limited. This study aimed to evaluate the diagnostic performance of FIB-4 and ARFI in detecting liver fibrosis among non-diabetic NAFLD patients.

Materials and Methods: We conducted a cross-sectional study involving 107 non-diabetic patients diagnosed with NAFLD at Tam Anh General Hospital, Ho Chi Minh City, between January 2023 and October 2024. The FIB-4 index was calculated using a standardized formula. Liver stiffness was measured by ARFI-based shear wave

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velocity (SWV). Correlation analyses, ROC curves, and multivariate logistic regression were applied to determine associations with significant ($F \geq 2$) and advanced fibrosis ($F \geq 3$). Results: The mean FIB-4 score was 1.46 ± 1.82 and mean SWV was 1.29 ± 0.44 m/s. The prevalence of fibrosis stage $F \geq 2$ and $F \geq 3$ was 15.0% and 11.3%, respectively. FIB-4 showed high diagnostic performance, with AUROC of 0.903 for $F \geq 2$ and 0.949 for $F \geq 3$. Independent predictors of $F \geq 2$ included age, platelet count, AST, and GGT, while age, AST, and total cholesterol were associated with $F \geq 3$. Among prediabetic patients (47.7%), 50% had $F \geq 2$ fibrosis and 41.7% had $F \geq 3$. Conclusions: FIB-4 and ARFI are effective non-invasive tools to assess liver fibrosis in non-diabetic NAFLD patients. Their application may facilitate early identification and management of at-risk individuals.

Keyword(s) : NAFLD, liver fibrosis, FIB-4, ARFI, non-diabetic, liver stiffness

1353 - 30% of Inactive HBsAg Carriers Achieved Clinical Cure at 48 Weeks with Pegylated Interferon Alpha 2b Therapy A Multicenter Real World Study (STARHB Project in China) 3 years Data Update

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Background and Aims: This study aimed to investigate the efficacy and safety of achieving functional cure (HBsAg loss) using Pegylated Interferon alpha-2b (PEG IFN α -2b) treatment in inactive HBsAg carriers (IHC). Method: The STARHB Project (The STUDY ON TREATMENT FOR INACTIVE CARRIERS OF HEPATITIS B) is a multicenter real-world study in China focused on the functional cure of IHC, aiming to onboard approximately 25,000 patients (ChiCTR2200061541). Based on patient preferences, participants were assigned to a control group (without antiviral treatment) or treatment groups (NAs group and PEG IFN α -2b $\pm$ NAs group). The basic treatment duration was 48 weeks, extendable to 96 weeks as needed. Results: As of Oct. 2025, 24,758 individuals were screened, and 20,788 eligible IHC patients were included in the mITT population baseline analysis. This cohort was 63.9% male with a mean age of 43 years; 5.6% were under 30 years old, and 39.4% had fatty liver. The mean HBsAg level was 265 IU/ml, with 49.3% below 100 IU/ml. The mean HBV DNA level was 265 IU/ml, with 62.1% having undetectable HBV DNA at baseline. The mean ALT was 21 U/L. Liver biopsies were performed on 231 patients at baseline, with 35.9% showing significant liver necroinflammation and/or fibrosis ($\geq G2$ and/or $\geq S2$). At present, 3.58 patients have completed the 48-week follow-up. In the control group (n=43), 24 achieved virological response (VR), with 4 achieved HBsAg loss. In the NAs group, the VR rate at 48 weeks was 92% (93/101), with 5 achieved HBsAg loss. Among those treated with PEG IFN α -2b $\pm$ NAs, the VR rate at 48 weeks was 90.17% (2578/2860), with an HBsAg loss rate of 30% (852/2860). ROC analysis indicated that baseline HBsAg levels, 12-week and 24-week HBsAg levels, and 12-week AST levels could predict HBsAg loss at 48 weeks. Conclusion: PEG IFN α -2b therapy can effectively achieve functional cure in IHC patients, with a 48-week HBsAg loss rate of 30%. Lower baseline, 12-week, and 24-week HBsAg levels, along with higher 12-week AST levels, are predictors of functional cure at 48 weeks. Thus, the IHC population is a favorable group for pursuing clinical cure for hepatitis B.

Keyword(s) : PEG IFN α -2b;inactive HBsAg carriers;HBsAg loss;antiviral therapy

1356 - Severe Hepatic Steatosis Impairs PEG IFN Response in HBsAg Clearance Favorable Chronic

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Hepatitis B A Retrospective Cohort Study

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Background/Objectives: The impact of hepatic steatosis (HS) severity on pegylated interferon-alpha (PEG-IFN α) efficacy in chronic hepatitis B (CHB) patients ideally suited for HBsAg clearance remains undefined. We aimed to investigate this relationship in a well-defined, favorable-population. **Methods:** This multicenter, retrospective cohort study enrolled 499 HBeAg-negative CHB patients with HBsAg <1500 IU/mL who completed 48 weeks of PEG-IFN α -based therapy. Patients were stratified by controlled attenuation parameter (CAP) values into no-HS (CHB, n=250), mild (n=90), moderate (n=41), and severe HS (n=118) groups. The primary endpoint was HBsAg clearance at week 48. **Results:** The overall HBsAg clearance rate was 39.1% (195/499). While no significant difference existed between the composite CHB+HS and CHB groups (37.0% vs. 41.2%, $P>0.05$), clearance rates demonstrated a severity-dependent gradient. Notably, patients with severe HS had significantly reduced clearance (24.6%) compared to the CHB group (41.2%, $P<0.01$) and to mild or moderate HS subgroups (45.6% and 53.7%, respectively; $P<0.01$). After adjustment for confounders using inverse probability of treatment weighting (IPTW), severe HS remained an independent negative predictor of HBsAg clearance (adjusted HR=0.577, 95% CI: 0.345–0.964; $p=0.04$). **Conclusion:** Severe hepatic steatosis is independently associated with a significantly attenuated response to PEG-IFN α in HBsAg-low CHB patients, highlighting the critical need for incorporating steatosis severity assessment into clinical decision-making.

Keyword(s) : chronic hepatitis B; hepatic steatosis; pegylated interferon; HBsAg clearance; treatment response; controlled attenuation parameter; severity

1361 - L Taurine for Fatigue in Decompensated Cirrhosis A Randomized Controlled Trial

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Background / Hypothesis: Chronic liver disease (CLD), including cirrhosis, affects over 1.5 billion people worldwide. Cirrhosis can manifest with diverse symptoms. Fatigue, despite its substantial impact on

quality of life, often goes under-recognized and inadequately managed. No universally effective treatment exists. This study explored whether taurine supplementation improved fatigue in patients with decompensated cirrhosis.

Materials and Methods: We conducted a randomized controlled trial from July 1st 2023 to December 31st, 2024, at an urban tertiary care center in South India. Individuals with decompensated cirrhosis presenting to the outpatient unit and reporting fatigue were screened and enrolled after obtaining informed consent. Fatigue was objectively quantified using the Fatigue Assessment Scale (FAS) score. Participants were block randomized to the study group (standard care plus 1000 mg L-aurine for 12 weeks), while the control group received standard care alone for the same duration. The primary outcome was change in FAS score at 12 weeks. Secondary outcomes included assessment of adverse drug events and changes to quality of life.

Results: A total of 382 patients were screened for enrolment. After applying exclusion criteria, 162 were excluded. Finally, 220 participants (110 in each group) underwent randomization and were enrolled. Eighteen patients were excluded from the final analysis (8 in the study group and 10 in the control group) due to reasonable causes. Both groups were comparable at the baseline with regards to demographics and disease severity, except for a statistically significant difference in FAS scores. Taurine use was associated with a greater median reduction in fatigue scores that did not reach statistical significance. Additionally, there was no significant improvement in quality of life compared to standard of care. Subgroup analysis revealed that among non-anemic patients (Hemoglobin ≥ 12 g/dL), L-aurine use showed statistically significant, greater reduction in fatigue scores, with a mean Δ FAS of -11.9 ± 4.04 compared to -4.57 ± 9.23 in the control group ($p = 0.01$). No serious adverse events were notable between groups.

Conclusions: L-Taurine did not produce a statistically significant improvement in fatigue. Baseline imbalances in fatigue scores appeared to be influenced by anemia, highlighting the importance of controlling for this confounder in future research. Nonetheless, the addition of L-aurine could effectively reduce fatigue in non-anemic individuals which requires further validation in a larger well-defined multi-center cohort (Clinical Trials Registry India number CTRI/2023/06/054455)

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Keyword(s) : ascites, CLD, liver disease, portal hypertension, cirrhosis

1362 - Cell Microcapsule Loaded Potent Nanoenzymes Alleviate Drug Induced Liver Injury via Dual Strategies

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Background / Hypothesis: Drug-induced liver injury (DILI) primarily results from the metabolic activation of drugs such as acetaminophen or anti-tuberculosis agents, leading to the accumulation of toxic intermediates that impair mitochondrial function. These toxins disrupt oxidative phosphorylation, reduce ATP production, and promote excessive reactive oxygen species (ROS) formation. Furthermore, Kupffer cell activation and disrupted redox homeostasis, particularly through suppression of antioxidant pathways like Nrf2, exacerbate DILI, providing a mechanistic basis for designing multi-target therapeutic strategies. This study aims to investigate whether cell-encapsulated nano-antioxidants can alleviate hepatic DILI and elucidate its specific mechanisms.

Materials and Methods: A liver-targeted oral delivery system with high safety was designed for DILI treatment. CeO₂ nanoparticles and silibin were encapsulated within cellular vesicles to generate highly biocompatible anti-inflammatory nanoparticles. These nanoparticles resist gastric acid degradation, are absorbed via intestinal M cells, and undergo translocation for targeted delivery to the liver. To validate the safety and targeting efficacy of the material, an APAP-induced liver DILI mouse model was employed. Oral administration occurred one day prior to model establishment and concurrently with APAP injection. Mice were euthanized 24 hours later. Liver tissue was collected for histopathological examination, measurement of common inflammatory cytokine levels, and molecular analyses including Western blotting.

Results: Yeast cell microcapsules loaded with nanoenzymes demonstrated excellent ROS scavenging capacity, stability in the intestinal environment, and biocompatibility with cells and mice. Furthermore, yeast cell microcapsules effectively targeted macrophages in the liver. In the drug-induced mouse model, nanoenzyme-loaded yeast cell microcapsules effectively alleviated weight loss, hepatic fibrosis, and hepatocyte necrosis while reducing inflammatory cytokines.

Additionally, yeast cell microcapsules remodeled the gut microbiota, increasing SCFA levels by suppressing harmful bacteria and enhancing beneficial bacteria.

Conclusions: This natural product-targeted oral delivery system offers a novel therapeutic approach for drug-induced liver injury through anti-inflammatory, antioxidant, and gut microbiota-modulating effects. Furthermore, detailed experimental findings elucidate the precise mechanism by which nano-antioxidant agents alleviate drug-induced liver injury. Overall, this study provides a comprehensive account of the effects of nano-antioxidant agents on drug-induced liver injury, laying the groundwork for future research into treating this condition with nano-therapeutic materials.

Keyword(s) :

1375 - Multidimensional analysis of diagnosis and treatment status of chronic hepatitis B

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Background / Hypothesis: The aim was to analyze key indicators for the diagnosis and treatment of chronic hepatitis B (CHB), including virus detection rate, standardized treatment rate, and loss to follow-up rate, in order to provide a basis for optimizing diagnosis and treatment plans, improve the diagnosis and treatment level of CHB, and improve patient prognosis.
Materials and Methods: Patients with CHB admitted to Guangzhou Eighth People's Hospital Affiliated to Guangzhou Medical University from January 2024 to January 2025 were enrolled. The data were collected and organized using Excel. Statistical analysis was conducted using SPSS 26.0 software, with a focus on evaluating core indicators such as virus detection rate, standardized treatment rate, and loss to follow-up rate.
Results:

The positive rate of hepatitis B surface antigen (HBsAg) in nonspecific patients was 28.95%, the antiviral treatment rate in specialized patients was 90.78%, and the standardized antiviral drug conversion treatment rate in low-level viremia (LLV) patients was 61.45%. The standardized antiviral drug conversion treatment rates for high-risk patients with combined kidney/bone injuries were 72.75% and 74.40%, respectively. The overall dropout rate was 10.47%, with a dropout rate

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of 13. 80% for LLV patients.

Conclusions:

The antiviral treatment coverage rates in CHB patients were over 90%, but in certain groups (such as LLV patients and those with kidney or bone injuries), the standardized treatment rates were still low and loss to follow-up rates were high, suggesting the need to improve HBV screening, treatment for special populations, and patient adherence.

Keyword(s) : Chronic hepatitis B ; Treatment ; Follow up ; Low-level viremia

1378 - Leucine rich alpha 2 glycoprotein 1 turnover imbalance impairs regenerative signaling in Alcohol associated Hepatitis

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Background / Hypothesis: Severe alcohol-associated hepatitis (SAH) is characterized by intense hepatic inflammation, impaired regeneration, and rapid clinical deterioration. We hypothesized that disrupted protein turnover reflects the failure of regenerative mechanisms in SAH and may identify biomarkers predictive of liver injury and recovery potential.

Materials and Methods: SAH patients, healthy controls, and liver donors received 70% D₂O, and plasma samples were collected at baseline, Days 1, 3, 5, and 7. LC-MS/MS analysis quantified turnover of pathology- and regeneration-related proteins, which correlated with disease severity. Key regeneration markers were validated in the ALD model and abstinent rats, and further confirmed in platelets of SAH patients, controls, and post-hepatectomy donors.

Results:

SAH plasma showed significantly higher protein turnover (0.38/day) compared to controls (0.23/day,

p<0.05). Turnover of 37 proteins was elevated, primarily involving inflammatory, complement, platelet activation, and ECM remodeling pathways, while 14 proteins involved in HDL assembly and BCR regulation were reduced (FC>1.5, p<0.05). ECM and fibrosis-related turnover cluster correlated with histological severity ($r^2=0.98$, p<0.05). Liver donors exhibited increased turnover of 50 proteins linked to platelet degranulation, complement activation, and HDL remodeling. Notably, LRG1 turnover and LRG1-mediated regenerative potential were significantly reduced in SAH patients (35%) compared to liver donors (100%, p<0.05). Alcohol abstinence in a preclinical ALD model restored hepatic LRG1 expression, its receptors (IL6r, IL4r, TGF β 2r, EGFr), and downstream survival/proliferation signaling (FC>1.5, p<0.05). Platelet degranulation, a source of LRG1, was impaired in SAH, contributing to compromised LRG1-mediated signaling and regeneration. Western blot analysis demonstrated higher plasma LRG1 in donor regeneration compared with SAH, and functional blockade of LRG1 delayed wound healing, validating its pro-regenerative role.

Conclusions: Leucine-rich alpha-2-glycoprotein 1 turnover, particularly from plasma or platelets, supports liver regeneration and may serve as a novel indicator of liver repair and regenerative capacity in alcohol-associated liver disease.

Keyword(s) :

1380 - The SAFE DILI Model for the Dynamic Prediction of Liver Transplantation or Death in Idiosyncratic Drug Induced Liver Injury

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Background

&

Aims

decision

making.

Idiosyncratic drug induced liver injury (DILI) exhibits a highly variable clinical course, ranging from spontaneous recovery to liver transplantation (LTx) or death. Although existing models assist in risk stratification, they rarely provide individualized, time dynamic predictions. This study aimed to develop and externally validate a simple, clinically applicable model to dynamically estimate the risk of LTx or death in patients with DILI.

Methods

We included 2,170 adjudicated DILI cases from two Chinese cohorts (derivation cohort, n=938; validation cohort, n=741) and two independent international cohorts, the Spanish DILI Registry (n=344) and the Latin American DILI Network (n=147). The primary outcome was LTx or death. A Cox regression model incorporating baseline laboratory variables was developed and internally evaluated using Harrell's c index, time dependent area under the curve (AUC), calibration, and decision curve analysis. External performance was assessed across validation and confirmation cohorts.

Results

During follow up, LTx or death occurred in 38 patients in the derivation cohort and in 55, 12, and 9 patients in the validation and confirmation cohorts, respectively. The SAFE DILI model included total bilirubin, albumin, and international normalized ratio. The c index in the derivation cohort was 0.85; time dependent AUCs were 0.94, 0.90, 0.86, and 0.82 at 30, 90, 180, and 270 days, respectively. Both calibration and decision curve analyses demonstrated consistent agreement and clinical utility. High risk patients (SAFE DILI >1.05) had significantly lower transplant free survival at 30, 90, and 180 days compared with low risk patients in both the derivation and validation cohorts. SAFE DILI showed higher discriminatory ability than established prognostic tools such as nR-based Hy's law, the Spanish DILI prognostic algorithm and MELD, across all validation cohorts.

Conclusion

SAFE DILI offers a clinically practical and time dynamic approach for predicting transplant free survival in patients with DILI. Its robust validation in diverse, multicenter, international cohorts underscores its value in facilitating early risk stratification and personalized

Keyword(s) : Drug-induced liver injury; Hepatotoxicity, Prognosis, Risk stratification, Transplant-free survival

1384 - Hepatitis A and E Guidelines created by the Japan Agency for Medical Research and Development (AMED) HAV and HEV Study Group
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Background / Hypothesis: There have been no clinical practice guidelines for hepatitis A virus (HAV) and hepatitis E virus (HEV) infection in Japan. Materials and Methods: Therefore, in 2024, the Japan Agency for Medical Research and Development (AMED) HAV and HEV Study Group created and published "Recent advances in hepatitis A virus research and clinical practice guidelines for hepatitis A virus infection in Japan" and "Recent advances in hepatitis E virus research and the Japanese clinical practice guidelines for hepatitis E virus infection." Results: We will introduce these guidelines, which seem useful for the research and clinical practice for hepatitis A and E. Conclusions: These guidelines for HAV and HEV infection could shed light on their research and clinical practice in Japan as well as in Asian-Pacific countries.

Keyword(s) : HAV; HEV; ALF; Acute-on-chronic liver failure; Japan; AMED Guidelines

1385 - Direct chemical reprogramming of urine derived stem cells from acute on chronic liver failure patients into hepatocyte like organoids to attenuate liver injury

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) has extremely high short-term mortality, while access to liver transplantation remains limited, creating an urgent need

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for safe, expandable autologous cells for bioartificial liver (BAL) support and cell therapy. Urine-derived stem cells (USCs) are easily and non-invasively obtained and show strong proliferative and immunomodulatory capacity. ACLF is characterized by TRF1/telomerase decline, PGC1- α downregulation and telomere-mitochondrial dysfunction with enhanced cellular senescence and apoptosis. We hypothesized that direct chemical reprogramming of ACLF-patient USCs (LF-USCs) into hepatocyte-like organoids (3D CiHep organoids) could restore the TRF1-PGC1 α -mitochondrial axis and thereby ameliorate liver injury in ACLF.

Materials and **Methods:** LF-USCs were isolated from urine of ACLF patients, expanded to $\leq P6$, and characterized for surface markers and tri-lineage mesenchymal differentiation. A three-stage small-molecule/cytokine protocol—definitive endoderm, hepatic progenitor cells, and hepatic differentiation/maturation—was used to generate chemically induced hepatocyte-like cells and 3D CiHep organoids, whose metabolic, detoxification, and secretory functions were evaluated. Concanavalin A (ConA)-induced acute liver injury and Fah-KO (M-NSG) liver failure mouse models were employed to assess intraperitoneal transplantation of N-USC- and LF-USC-derived 3D CiHep organoids, and telomere-mitochondrial parameters, senescence, injury, and apoptosis markers were quantified.

Results:

LF-USCs displayed typical “rice-grain” morphology, expressed CD73/CD90/CD105, and retained tri-lineage mesenchymal differentiation. LF-USC-derived 3D CiHep organoids were dominated by a mature hepatocyte phenotype, incorporated biliary epithelial elements, exhibited correct hepatocyte polarity with organized bile canaliculi networks, and showed near-adult liver function in albumin synthesis, ammonia/urea metabolism, CYP450-mediated drug metabolism, and bile acid/bilirubin handling. In both ConA and Fah-KO (M-NSG) models, LF-USC-derived 3D CiHep organoids substantially reversed ACLF-associated abnormalities, restoring TRF1, telomerase activity, PGC1- α , mtTFA, mtDNA content, and ETC I-V, while reducing SOD2, β -galactosidase, KIM-1, CYP2E1, and cleaved-caspase3/caspase3. Functionally, 3D CiHep organoids significantly lowered serum ALT/AST and pro-inflammatory cytokine levels, reduced hepatocellular necrosis and TUNEL-positive cells, and improved short-term survival compared with vehicle controls.

Conclusions:

Direct chemical reprogramming of LF-USCs into hepatocyte-like organoids yields an expandable

autologous liver cell source and restores the TRF1-PGC1 α -mitochondrial axis, improving telomere integrity and mitochondrial function while limiting senescence and apoptosis. These benefits translate into meaningful hepatoprotection in preclinical ACLF/liver failure models, supporting 3D CiHep organoids as a promising individualized BAL and cell-based.

Keyword(s) : Acute-on-chronic liver failure, Bioartificial liver, Urine-derived stem cells, Cell-based therapy, Direct chemical reprogramming

1387 - The transcription factor PRDM5 is a novel pro fibrotic factor in liver fibrosis

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Background / Hypothesis: Hepatic stellate cells (HSCs) are the key cellular drivers of liver fibrosis, promoting the excessive production of the extracellular matrix (ECM). PRDM5, a PR-domain transcription factor, is crucial for ECM homeostasis, and its deficiency is known to reduce collagen production in fibroblasts. However, the role of PRDM5 in HSCs and liver fibrosis remains unknown. We hypothesized that PRDM5 plays a pro-fibrotic role by activating HSCs and driving the progression of liver fibrosis.

Materials and Methods: The role of PRDM5 was investigated using both loss-of-function (shRNA-mediated knockdown) and gain-of-function (overexpression) approaches in primary human HSCs. HSC activation phenotypes, including proliferation, migration, contraction, and collagen production, were examined. RNA sequencing was performed on PRDM5-knockdown cells to identify regulated pathways. The direct DNA binding of PRDM5 to target genes was demonstrated using a chromatin profiling assay (CUT&RUN) in HSCs. In vivo, PRDM5-knockout mice were generated and subjected to fibrosis induction using the CDAHFD (a model mimicking human MASLD for 8 weeks) and CCl₄ models (4 weeks). Fibrosis was assessed by Sirius red and Masson trichrome stainings, and liver function was examined by measuring blood serum aminotransferase, albumin, and total bilirubin levels. Finally, PRDM5 expression was investigated in human liver tissues with a spectrum of fibrotic levels from normal to cirrhosis, and its correlation with ECM gene expression was determined. PRDM5 expression was also investigated in human samples from other fibrotic diseases.

Results:

Knockdown of PRDM5 in human HSCs was found to suppress proliferation, migration, contraction, and

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collagen production, while PRDM5 overexpression yielded the opposite results. Transcriptomic analysis indicated that the depletion of PRDM5 markedly influenced ECM-related pathways, resulting in the downregulation of several ECM genes. Mechanistically, the CUT&RUN assay confirmed the presence of PRDM5 at the promoter of ECM genes. In vivo, PRDM5-knockout mice showed resistance to fibrosis in both the CDAHFD and CCl₄ models, accompanied by improved liver function. Clinically, PRDM5 was observed to be upregulated in human fibrotic liver tissues and demonstrated a strong positive correlation with ECM gene expression. Similar upregulation patterns were also observed in other fibrotic organs, including lung, heart, and skin.

Conclusions:

We show PRDM5 activates HSCs and drives ECM gene transcription through direct promoter binding, leading to enhanced fibrosis across diet- and chemical-induced liver injury models. This is the first study to show the critical role of PRDM5 in fibrotic diseases and suggest its potential as a novel therapeutic target.

Keyword(s) : PRDM5, Liver fibrosis, Hepatic stellate cells, Extracellular matrix, Transcriptional regulation

1389 - Variability of Presentation of Cholelithiasis in Relation to Etiology and Stages of Cirrhosis of Liver in a Tertiary Care Hospital in Dhaka City

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Background: In patients with cirrhosis of liver, cholelithiasis has been recognized to have a higher incidence associated with high morbidities. The treatment of the cholelithiasis in cirrhotic patients has become a challenging issue. To manage these groups of people more effectively, we need better understanding between the relation of cholelithiasis and cirrhosis of the liver.

Aim and objective: The aim of this study is to see the demographic and clinical profile of cholelithiasis among cirrhosis of liver disease.

Materials and methods: This cross-sectional study was carried out at the Department of Gastroenterology and Hepatology of BIRDEM, Dhaka, Bangladesh. A total of 261 patients were enrolled. Among them, 131 were patients with cirrhosis of liver and the remaining 130 served as controls. Purposive type sampling was used to select study. Relevant history, physical examination,

laboratory investigations were recorded. Data were analyzed using the software SPSS 25. Result: In this study, cholelithiasis was significantly high in cirrhotic patient (34.4%) than in control (7.7%) ($p < 0.001$). Also found more often in male cirrhotic patients (68.9%) than in female (31.1%) ($p = 0.001$). A significant association was found between family history of cholelithiasis and the presence of gallstones in cirrhotic patients ($p = 0.003$). The stages of liver disease, as assessed by the Child- Pugh classification, cholelithiasis was found more often in decompensated liver disease (80%) than in compensated (20%) but this is not statistically significant. Cirrhosis with cholelithiasis significantly associated with HBV related etiology ($p = 0.001$) where non B non C etiology is significantly associated with cirrhosis without cholelithiasis group ($p = 0.038$). CBC, liver enzymes, and serum bilirubin did not show significant differences. Among patients with cholelithiasis, 31.1% were symptomatic, while 68.9% were asymptomatic. No significant association was observed between the presentation of cholelithiasis (symptomatic vs. asymptomatic) and common etiology of liver cirrhosis ($p = 0.614$) and also there were no significant associations with liver disease severity ($p = 0.156$).

Conclusion: Cholelithiasis is not uncommon among patients with cirrhosis of liver, among them a considerable portion have asymptomatic cholelithiasis. Presence of cholelithiasis in cirrhosis is significantly associated with male gender, having family history of gallstones and HBV related etiology. But there were no significant association between the presentation of cholelithiasis (symptomatic vs. asymptomatic) with common etiology of liver cirrhosis and stages of liver disease. These results emphasize the need for targeted screening and management strategies for this large group of patients.

Keyword(s) : Cirrhosis of Liver, Cholelithiasis

1393 - Optimal timing of hepatectomy after conversion therapy and stratification by risk of recurrence in patients with hepatocellular carcinoma a multicenter retrospective investigation (GUIDANCE005)

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Background / Hypothesis: Conversion therapy can render initially unresectable hepatocellular carcinoma (HCC) amenable to potentially curative resection. No consensus guidelines exist about what criteria should guide when such surgery should be performed and how risk of postoperative recurrence should be assessed. The present study explored potential criteria for these purposes.

Materials and Methods: This multicenter retrospective study included 464 patients with initially unresectable HCC who underwent conversion therapy followed by hepatectomy at 24 medical centers in Guangxi, China. Recurrence-free and overall survival were determined using Cox regression and restricted cubic spline models for patients stratified according to radiological response rate and tumor size after conversion therapy, based on version 1.1 criteria for Response Evaluation Criteria in Solid Tumors (RECIST) or on modified RECIST criteria.

Results: Of the 2,219 patients screened for enrollment, 464 (403 men) aged 52.0 ± 10.5 years were analyzed, among whom conversion therapy led to complete response in 2 (0.4%) based on RECIST 1.1 or 78 (16.8%) based on mRECIST. Regardless of the choice of RECIST criteria, radiological response $\geq 40\%$ was associated with significantly longer recurrence-free and overall survival. Maximization of the log-rank statistic identified cut-off rates of 34.0% based on RECIST 1.1 and 42.0% based on mRECIST for optimal stratification of patients according to survival risk. Tumor downstaging after conversion therapy, which occurred in 142 patients (30.6%) based on RECIST 1.1 and 206 (44.4%) based on mRECIST, was associated with significantly longer recurrence-free and overall survival. Pathological complete response, which occurred in 122 patients (26.3%), was also associated with significantly longer recurrence-free survival (hazard ratio 0.45, 95% confidence interval 0.26–0.78) and overall survival (hazard ratio 0.28, 95% confidence interval 0.12–0.69).

Conclusions: Radiological response rate and tumor size following conversion therapy may be useful prognostic determinants for HCC recurrence. Predicting recurrence

risk following hepatectomy should also take into account whether pathological complete response was achieved.

Keyword(s) : hepatocellular carcinoma, conversion therapy, hepatectomy, radiographic response, tumor size

1396 - Exploring the efficacy of Systemic Inflammatory Markers in predicting outcomes post Transarterial Chemoembolization (c TACE & deb TACE)

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Background / Hypothesis: Tumour inflammation is the driving force behind tumour progression in hepatocellular carcinoma. Various prognostic markers can predict connection between inflammatory response and tumour microenvironment. Systemic inflammatory markers like lymphocyte monocyte ratio, systemic inflammatory index, neutrophil lymphocyte ratio can offer insight into the inflammatory milieu, which may predict the response to locoregional therapy.

Materials and Methods: This is a retrospective, single centre study done from January 2023 to March 2025 encompassing 43 patients with intermediate stage Hepatocellular carcinoma, undergoing transarterial chemoembolization (cTACE & debTACE) for tumour downstaging. Patients were evaluated for biochemical and inflammatory panel, serum AFP levels, imaging prior to undergoing transarterial chemoembolization. Post TACE follow up imaging was done at one month and three months to identify response using modified response evaluation criteria in solid tumours (mRESIST) criteria. Patients having severe liver or renal dysfunction, active infection were excluded from the study.

Results:

Total 43 patients fulfilling the inclusion criteria were included in the study and evaluated for response after TACE and median survival at 3 and 6 months. The median age of the participants was 55.84 ± 9.87 years consisting of 25.5% females and 74.4% males. The most common etiology was Chronic Hepatitis B, followed by Metabolic dysfunction associated steatotic liver disease & chronic Hepatitis C amongst others. 27.9% patients

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underwent drug eluting bead TACE and 72.09% patients underwent conventional TACE. Higher SII (p= 0.041)and lower LMR (p= 0.024) were associated with a poor tumour response. The ROC analysis for the Lymphocyte-to-Monocyte Ratio demonstrated a discriminative ability to predict treatment outcome in patients with HCC with a cut-off value of ≥ 2.2 [AUC- 0.892,(95% CI0.619 -0.976)]having better prognosis. AFP levels also correlated with the outcome to TACE (p=0.033) There was no significant difference in the outcome with debTACE as compared to cTACE.

Conclusions:

Peripheral inflammatory markers can unveil the dynamics between the tumour biology, the tumour stromal microenvironment and the immune response, serving as valuable prognostic indicators, predicting recurrence, disease progression and response. This study helps identify systemic inflammatory markers that can be incorporated prior to TACE, paving way for an enhanced therapeutic precision.

Keyword(s) : Hepatocellular Carcinoma, Systemic Inflammatory Markers, TACE

1399 - Trajectory Based Multidimensional Risk Scoring in a Single Center Retrospective Cohort Exploratory Development for Predicting Poor HBsAg Seroclearance Response in Chronic Hepatitis B

Functional Cure
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Background / Hypothesis:Chronic hepatitis B functional cure is associated with multiple markers—with HBsAg levels being particularly critical—yet methods for comprehensive dynamic multi-marker assessment to predict poor cure outcomes remain lacking. This single-center retrospective longitudinal study thus characterizes trajectories of multiple indicators—including HBsAg, ALT, AST, and Plate—in CHB patients on functional cure regimens (starting with pegylated interferon) and explores their links to cure outcomes.

Materials and Methods:A retrospective study included chronic hepatitis B (CHB) patients pursuing functional cure at Sichuan Provincial People's Hospital (2022–2025). Dynamic data of indicators (e.g., HBsAg, ALT) were collected. CHB Trajectory Clustering, multidimensional dynamic risk scoring, and treatment

failure probability model were used for grouping and analysis. Binary logistic regression explored associations between risk factors and treatment failure.

Results:A total of 166 chronic hepatitis B (CHB) patients were included in the analysis. Patients were classified into four trajectory types based on indicators including hepatitis B surface antigen (HBsAg): rapid high-clearance (22.8%, n=38), delayed high-clearance (26.5%, n=44), fluctuating (34.3%, n=57), and slow low-clearance (16.3%, n=27). Treatment failure rates were calculated for each trajectory type: 8.3% in the rapid high-clearance type, 15.6% in the delayed high-clearance type, 32.7% in the fluctuating type, and 68.2% in the slow low-clearance type. A comprehensive risk scoring system was further established, integrating five key dimensions: baseline HBsAg score, HBsAg reduction score, transaminase stability score, early response score, and trajectory type score. Additionally, a time-weighted risk prediction model for calculating treatment failure probability was developed, with the formula: $P_{failure} = \min(0.95, P_{baseline}(t) + \sum_{i=1}^n W_i \times I_i)$ (where $P_{baseline}(t)$ represents the time-dependent baseline failure probability, W_i denotes the weight of the i-th adverse risk signal, and I_i is the indicator variable for the presence of the i-th risk signal)

Conclusions:This study established an integrated system combining trajectory clustering and dynamic risk scoring, identifying four CHB treatment response trajectory types and quantifying their failure risks, with slow low-clearance patients having the highest failure risk. Five factors (including baseline HBsAg, early HBsAg reduction, ALT stability) correlated with treatment failure, with trajectory type as the strongest independent predictor (HR=8.48), highlighting the value of dynamic HBsAg monitoring in clinical decision-making.

Keyword(s) : Chronic Hepatitis B; Trajectory Clustering; Dynamic Risk Scoring; Treatment Outcome Prediction

1401 - Effects of the secondary metabolites of Terminalia arjuna on non alcoholic fatty liver disease in experimental mice model

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Background

Hypothesis:

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Fatty liver diseases, including non-alcoholic fatty liver disease (NAFLD) and NASH, are significant global health issues that can lead to severe conditions. Terminalia arjuna, a medicinal plant, is being studied for its potential liver-protective properties. The aim is to isolate its compounds and study their effectiveness in treating NAFLD.

Materials and Methods:

To create the NAFLD mouse model, Swiss albino male mice were injected with 200µg streptozotocin (STZ) and given a high-fat diet (HFD) for three months starting at one month of age. The NAFLD-induced mice were treated with four isolated secondary metabolites of T. arjuna using an oral dose of 50 mg/kg of body weight daily for one month starting from three months of age, while still being fed the HFD. After four months of age, all mice were sacrificed for further analysis. The histological changes and the percentage of fibrosis were quantified using hematoxylin & eosin (H&E) and Masson trichrome (MT) staining in the liver tissues, respectively. The dried powdered bark of T. arjuna was extracted using ethyl acetate. This extract was separated through chromatography and fractions rich in triterpene acid derivatives were purified by HPLC. The structures of the purified metabolites were elucidated using high-resolution ¹H- and ¹³C-NMR experiments.

Results:

The isolated secondary metabolites were arjunic acid, arjunoglucoside-1, arjungenin, and arjunolic acid. It was found that the body weight of NAFLD mice treated with the vehicle was significantly reduced, whereas the liver index and blood glucose levels were markedly elevated when compared to the normal mice. The blood glucose level and liver index were improved in all treatment groups. The NAFLD group showed significantly increased levels of glucose, creatinine, total cholesterol, triglycerides, and hepatic enzymes ALT, AST, and ALP in serum in comparison to the normal group. These parameters were significantly reduced in mice treated with arjunic acid, arjunoglucoside-1, arjungenin, and arjunolic acid compared to the untreated NAFLD mice. The NAFLD activity score (NAS) and percentage of fibrosis were significantly increased in the NAFLD group but significantly reduced in most treatment groups.

Conclusions:

The study findings indicate that all four secondary metabolites have hepatoprotective effects against NAFLD. Arjunic acid and arjungenin are particularly

effective. Further research is needed to elucidate the molecular mechanisms behind this effect.

Keyword(s) : arjunolic acid; arjunic acid, arjungenin, arjunoglucoside-1; fibrosis; non-alcoholic fatty liver disease; Terminalia arjuna

1408 - Predicting Recompensation and Exploring the Prognostic Benefit of its Durability in patients with HBV related decompensated cirrhosis

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Background / Hypothesis: Although a subset of patients with HBV-related decompensated cirrhosis can achieve recompensation following nucleos(t)ide analogue (NAs) therapy, the impact of recompensation durability on liver-related clinical outcomes remains insufficiently elucidated. Furthermore, the determinants of recompensation, particularly the prognostic value of virological response indicators and baseline decompensation complications, require further investigation.

Materials and Methods:

This retrospective-prospective cohort study enrolled patients with HBV-related decompensated cirrhosis receiving NAs therapy. Based on whether recompensation was achieved within the initial 2 years, patients were stratified into a recompensation group and a non-recompensation group. Those in the recompensation group were further divided into a long-term recompensation group and a non-long-term recompensation group based on maintaining recompensation for over 2 years. All patients were subsequently followed until the occurrence of clinical outcomes—including hepatocellular carcinoma (HCC), acute-on-chronic liver failure (ACLF), or liver-related death—or until the end of the study period in December 2024. We compared the cumulative incidence of clinical outcomes among these groups. Furthermore, we investigated the determinants of recompensation and developed a predictive model.

Results:

A total of 813 patients were enrolled with a median follow-up of 6 years. By year 2, 25.5% achieved recompensation. The recompensation rate was inversely correlated with the number of baseline complications (33.3% vs. 23.5% vs. 14.7% for patients with one, two, and three complications, respectively). Among patients

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with a single complication, the recompensation rate was highest in those with ascites (40.6%), followed by bleeding (23.8%) and hepatic encephalopathy (15.7%). Among those who achieved recompensation, 85.1% maintained long-term recompensation. The long-term recompensation group had lower cumulative incidence of HCC than the non-long-term group (5-year: 8.7% vs. 36.8%; 10-year: 18.3% vs. 43.9%) and also exhibited a reduced incidence of liver-related death. Virological response within 6 months (OR = 2.390, 95% CI: 1.671–3.458, $p < 0.001$) was an independent predictor for 2-year recompensation, and a prediction model incorporating this factor demonstrated good performance.

Conclusions:

Virological response within 6 months is a strong predictor of 2-year recompensation. Recompensation rates were lower in patients with more baseline complications or with hepatic encephalopathy as a single complication. Over 80% of patients maintained long-term recompensation and exhibited lower 5- and 10-year cumulative incidence of HCC and liver-related death than the non-long-term recompensation group, underscoring the necessity for continued clinical monitoring and risk assessment even after initial recompensation.

Keyword(s) : Keywords: Hepatitis B; Liver Cirrhosis; Decompensation; Recompensation; Carcinoma, Hepatocellular; Predictive Models

1410 - Characterization of Serum Cytokine Profile and Its Early Warning Value in Elderly Patients with Hepatitis E

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Background / Hypothesis:Hepatitis E virus (HEV) is one of the public health problems threatening human health. The mortality rate of elderly people infected with HEV can reach 25% to 75%. With the intensification of global population aging, it is of great significance to study the HEV infection mechanism of this special population. This study aims to analyze the serum cytokine levels of elderly patients with hepatitis E and

explore the value of serum cytokine levels in predicting the severity and prognosis of elderly patients with hepatitis E.

Materials and Methods:This case-control study collected 35 elderly patients with acute hepatitis E (AHE), 40 elderly patients with acute liver failure related to hepatitis E virus (HEV-ALF), and 43 healthy elderly patients. First, the levels of 12 cytokines in the three groups of subjects were analyzed using immunofluorescence technology. Secondly, the serum cytokine levels of patients in the healthy elderly group and the HEV-infected group (AHE elderly group and HEV-ALF elderly group) were compared. The serum cytokine levels of patients in the AHE elderly group and the HEV-ALF elderly group were compared. According to the prognosis, 40 HEV-ALF patients were divided into the survival group (n=33) and the death group (n=6). Finally, univariate Logistic regression analysis, receiver operating characteristic (ROC) curve, and decision curve analysis (DCA) were used to evaluate the predictive and decision-making power of serum IL-4 level for prognosis.

Results:IL-8 was significantly highly expressed in the elderly HEV infection group. However, there was no statistically significant difference in the expression of the two cytokines, IL-5 and IL-12P70, between the healthy elderly group and the HEV infection group. The expression of serum cytokines is also correlated with the severity of hepatitis E progression. The AUC of serum TNF- α level for predicting severe hepatitis E was 0.747, with a sensitivity of 58.97% and a specificity of 82.86%. The AUC of serum IL-4 level for predicting death within 30 days in elderly patients with HEV ALF was 0.793, with a sensitivity of 100.00% and a specificity of 57.58%. DCA found that the serum IL-4 level has a good decision-making power for death within 30 days in elderly patients with HEV-ALF.

Conclusions:The level of serum TNF- α has a good predictive efficacy for the severe illness of elderly patients with AHE. The higher the level of serum IL-4, the worse the prognosis of patients with HEV-ALF.

Keyword(s) : Elderly patients with hepatitis E infection; Cytokine; Liver failure; Severe exacerbation; Prognosis

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1414 - Multi omics integration analysis identifies Molecular Signatures of Liver Stress in preclinical model of Acute Hypoxia

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Background / Hypothesis: Hypobaric hypoxia, as encountered at high altitudes, imposes systemic stress with significant yet understudied effects on the liver, a central hub for metabolic regulation. Short-term hypoxia may initiate harmful molecular changes, disrupting hepatic homeostasis. This study aimed to characterize temporal liver-specific proteomic and metabolic perturbations under acute hypoxic exposure.

Materials and Methods: Male Sprague-Dawley rats (n = 6 per group) were exposed to simulated 25,000 ft altitude for 1, 3, or 7 days using a hypobaric chamber. Liver tissues were harvested and subjected to untargeted high-resolution proteomics and metabolomics. Integrated analysis identified time-dependent molecular alterations linked to hepatic injury and stress adaptation.

Results: A total of 313 metabolites were upregulated by hypoxia ($p < 0.05$, $FC > 1.5$) associated with redox and mitochondrial pathways (glutathione, cysteine/methionine, thiamine metabolism, TCA cycle) reflecting stress response. key nitrogen and amino acid metabolism pathways such as ammonia recycling, histidine, beta-alanine were significantly suppressed, indicating impaired detoxification and antioxidant capacity in liver due to hypoxia. Proteomic profiling revealed 444 upregulated proteins related to stress signaling, lipid handling ($p < 0.05$, $FC > 1.5$). 586 proteins were downregulated associated with pathways involved in protein and amino acid metabolism, lipid metabolism, membrane trafficking, neurotransmitter transport, fatty acid oxidation, biological oxidations, proteasome degradation, and calcium signaling. The downregulation of these pathways indicates impaired hepatic biosynthesis, energy metabolism, detoxification, and protein turnover. Integration analysis showed that

significant increase in energy metabolism (TCA cycle) directly correlates to increase in Inflammatory (tryptophan catabolism, nucleotide metabolism and GPCR signaling) cascade ($R^2 > 0.7$, $p < 0.05$). Together, these alterations reflect compromised liver function and molecular disruptions contributing to metabolic stress, redox imbalance, and potential progression toward hypoxia-induced liver injury.

Conclusions: Short-term hypobaric hypoxia induces early hepatic molecular injury, marked by compromised nitrogen metabolism, proteostasis, and antioxidant defense, despite concurrent activation of redox and mitochondrial pathways. These findings underscore the liver's vulnerability to acute altitude exposure and highlight potential molecular targets for mitigating hypoxia-induced hepatic stress.

Keyword(s) : Hypobaric hypoxia, Proteomics, metabolomics, integration, Machine learning

1416 - PPAR α Activation Restores GSDME Pyroptosis to Reverse Anti-PD 1 Resistance in HCC

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Background / Hypothesis: Resistance to anti-Programmed cell death protein 1 (anti-PD-1) remains a major clinical obstacle in hepatocellular carcinoma (HCC). The fibroinflammatory liver microenvironment (FILM), characterized by excessive extracellular matrix deposition and immunosuppressive inflammatory cell infiltration, has been implicated in anti-PD-1 resistance; however, the underlying mechanisms remain poorly understood.

Materials and Methods:

Clinical HCC cohorts, FILM-induced syngeneic mouse models, and lipidomic, transcriptomic, and single-cell analyses were used to evaluate how FILM regulates tumor-cell pyroptosis and immunotherapy efficacy. Mechanistic assays including co-culture systems, ChIP-qPCR, rescue experiments, and ROS/caspase activity measurements were performed to delineate the FILM-PPAR α -PDK4-ROS-caspase-3-GSDME signaling axis. The therapeutic effects of PPAR α agonists were

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assessed in multiple mouse models and a retrospective patient cohort.

Results:

FILM selectively suppressed GSDME cleavage—rather than total GSDME abundance—resulting in diminished tumor-cell pyroptosis, impaired dendritic-cell activation, reduced cytotoxic T-cell infiltration, and anti-PD-1 resistance. Mechanistically, FILM-derived cancer-associated fibroblasts recruited and polarized NOS2⁺ macrophages, whose nitric-oxide production induced S-nitrosylation-dependent inhibition of the transcription factor SP1, thereby reducing PPAR α transcription. Reduced PPAR α activity led to decreased transcription of PDK4, suppressed mitochondrial ROS generation, attenuated caspase-3 activation, and ultimately blocked GSDME-mediated pyroptosis. Pharmacological PPAR α activation (GW7647 or fenofibrate) restored the PDK4-ROS-caspase-3-GSDME cascade, enhanced immunogenic tumor-cell death, reprogrammed the FILM-immunosuppressive niche, and synergized with anti-PD-1 therapy in multiple HCC models. Retrospective clinical analyses showed improved tumor shrinkage and progression-free survival in patients receiving fenofibrate plus anti-PD-1, particularly in tumors with preserved PPAR α expression.

Conclusions:

This study identifies PPAR α activation as a promising strategy to overcome FILM-induced anti-PD-1 resistance in HCC by restoring GSDME-mediated pyroptosis. Combining PPAR α agonists with immune checkpoint inhibitors represents a potential therapeutic approach for HCC patients, particularly those with FILM.

Keyword(s) : anti-PD-1 resistance, PPARA, Pyroptosis, GSDME

1417 - The Clarity Trial Prospective evaluation of pemafibrate therapy in MASLD

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Background

The Clarity trial was designed to prospectively evaluate the metabolic and hepatic effects of pemafibrate in metabolic dysfunction-associated steatotic liver disease (MASLD). Although pemafibrate exerts potent triglyceride-lowering effects, prospective studies integrating paired biochemical and histological

endpoints remain limited. We hypothesized that pemafibrate-based therapy would improve clinical, biochemical, and particularly histological markers in MASLD with hypertriglyceridemia.

Materials and Methods
Clarity (UMIN000052895) is a single-center prospective study enrolling adults with biopsy-proven MASLD and hypertriglyceridemia who required longitudinal histological assessment. Pemafibrate 0.2 mg/day and nutritional counseling were initiated after baseline evaluation. The primary endpoint was paired histological change at week 52, including fibrosis stage, steatosis, lobular inflammation, ballooning, and NAFLD activity score (NAS). Secondary endpoints included biochemical, metabolic, and elastography-based changes. This interim analysis included 14 patients completing ≥ 52 weeks of follow-up with paired biopsies (median age 60 years, IQR 45–71; 7 men and 7 women). Median (IQR) differences were assessed using the Wilcoxon signed-rank test.

Results

The primary histological endpoint demonstrated significant improvement. Fibrosis stage decreased from 3 (2–3) to 2 (1–3) ($p = 0.015$), with 7 of 14 patients showing fibrosis regression. Lobular inflammation improved from 2 (1–2) to 1 (1–1) ($p = 0.005$). Steatosis grade remained stable at 1 (1–2) at both time points ($p = 0.41$), while hepatocellular ballooning showed a modest reduction. NAS decreased from 4 (4–5) to 3 (2–4) ($p = 0.0076$). Regarding secondary endpoints, body weight decreased from 75.1 (70.0–89.4) to 71.3 (67.4–87.2) kg ($p = 0.0067$), and triglycerides from 143 (125–198) to 96 (82–119) mg/dL ($p = 0.013$). Liver enzymes decreased significantly: AST from 55.0 to 30.5 U/L ($p = 0.0067$), ALT from 77.5 to 27.5 U/L ($p = 0.0085$), GGT from 80.0 to 30.5 U/L ($p = 0.0166$), and ALP from 271.5 to 172.0 U/L ($p = 0.0107$). Liver stiffness improved from 12.7 (8.8–20.3) to 7.5 (6.1–10.5) kPa ($p = 0.0088$), while controlled attenuation parameter remained unchanged. No pemafibrate-related adverse events occurred.

Conclusions

This interim analysis of the Clarity trial shows significant improvement in the primary histological endpoint—particularly fibrosis and lobular inflammation—together with favorable biochemical and metabolic changes in MASLD with hypertriglyceridemia. These findings suggest potential hepatic benefit of pemafibrate, although confirmation in the full cohort is required before therapeutic implications can be established.

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Keyword(s) : MASLD, pemafibrate, paired biopsy, prospective study, hypertriglyceridemia, fibrosis regression

1418 - Efficacy of Sulfasalazine in Patients with Primary Biliary Cholangitis and Incomplete Response to Ursodeoxycholic Acid

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Background / Hypothesis: Approximately 40% of primary biliary cholangitis (PBC) patients exhibit incomplete response to ursodeoxycholic acid (UDCA), leading to increased disease progression risk and limited second-line therapeutic options. Our preliminary animal studies suggested sulfasalazine (SASP), which is a classic drug for inflammatory bowel disease, alleviates cholestatic liver injury via activation of peroxisome proliferator-activated receptor α (PPAR α). This study aimed to evaluate the efficacy of SASP combined with UDCA in UDCA-nonresponsive (UDCA-NR) patients and to further investigate its underlying mechanisms of action.

Materials and Methods: This single-center, prospective, single-arm exploratory study enrolled 8 UDCA-NR patients. UDCA-NR was defined as a serum ALP level $>1.67 \times \text{ULN}$ after stable UDCA therapy (≥ 12 months). All patients maintained UDCA at 13–15 mg/kg/day and received SASP at 1.5 g/day for 48 weeks. The primary endpoint was the composite biochemical response rate at week 48, defined as meeting all the following criteria: (1) serum ALP $<1.67 \times \text{ULN}$ (specifically <225 U/L for females and <208 U/L for males); (2) a $\geq 15\%$ decrease in ALP from baseline; and (3) serum total bilirubin $\leq \text{ULN}$ ($28 \mu\text{mol/L}$). Metagenomic sequencing, fecal bile acid profiling, and flow cytometry were used to investigate the specific mechanisms underlying the therapeutic effects of SASP.

Results: Following 48 weeks of SASP combined therapy, composite biochemical response rate was 62.5% (5/8), with ALP normalization rate of 25% (2/8). Serum ALP, γ -GGT, ALT and AST decreased significantly compared

with baseline; no serious adverse events were reported. Metagenomic sequencing showed SASP increased relative abundance of the genera *Blautia* and *Bifidobacterium*, while decreasing that of *Alistipes putredinis*, *Parasutterella*, and *Klebsiella*. Fecal bile acid profiling revealed that, although total bile acid levels showed no significant change pre- versus post-intervention, SASP elevated UDCA, GUDCA, and TUDCA, and reduced the toxic secondary bile acids DCA and LCA. Flow cytometry indicated SASP ameliorated peripheral Th17 (T helper 17 cell)/Treg (regulatory T cell) immune imbalance and increased double-negative (IgD⁻CD27⁻) B cells. Subgroup analysis identified high baseline relative abundance of *Escherichia coli*, *Ruminococcus gnavus*, *Streptococcus* spp. was associated with non-response to SASP, while high baseline *Blautia obeum* associated with treatment response and more substantial Th17/Treg balance improvement.

Conclusions: This study demonstrates that SASP effectively elicits a biochemical response with a favorable safety profile in UDCA-NR PBC patients. Its mechanism of action involves modulation of the "gut microbiota-bile acid-immune" axis. These findings indicate that SASP may serve as an effective second-line treatment option for UDCA-NR PBC patients, which warrants further investigation.

Keyword(s) : Primary biliary cholangitis, Sulfasalazine, Ursodeoxycholic acid nonresponse, Gut microbiota, Bile acids

1419 - Evodiamine alleviates liver fibrosis by targeting the ANXA2 PI3K AKT CCND1 axis in hepatic stellate cells

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Background / Hypothesis: Liver fibrosis is a pivotal pathological process in chronic liver disease progression, characterized by hepatic stellate cell (HSC) activation and excessive deposition of extracellular matrix. Currently, no highly effective anti-fibrotic drugs are available for clinical use. This study aimed to screen natural compounds derived from clinically used

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traditional Chinese medicine (TCM) anti-fibrotic formulations for their HSC-targeting activity and to investigate their mechanisms of action.

Materials and Methods: Using network pharmacology combined with ADME screening, potential core active components were screened from Anluo Huaxian Pills, Fuzheng Huayu Formula, and Biejia Ruangan Formula. The inhibitory effects of candidate compounds on HSC activation were evaluated in a TGF- β 1-induced LX-2 cell model. The in vivo efficacy of evodiamine (EVO) was comprehensively assessed in three liver fibrosis models: CCl₄-induced hepatotoxic, DDC diet-induced cholestatic, and high-fat diet combined with CCl₄ (HFD+CCl₄)-induced NASH-related liver fibrosis. Direct molecular targets were identified using DARTS, biolayer interferometry (BLI), and cellular thermal shift assay (CETSA). Further mechanistic insights were gained through gene silencing, overexpression, and pathway intervention studies.

Results: Among the 23 candidate compounds, evodiamine significantly inhibited the expression of TGF- β 1-induced HSC activation markers (TGF- β , COL1A1, and α -SMA) and effectively suppressed HSC proliferation. All three liver fibrosis models confirmed that evodiamine (50 mg/kg/day) markedly ameliorated liver injury (ALT, AST), reduced HSC activation and collagen deposition, and showed no observable organ toxicity. Additionally, it improved cholestatic parameters (ALP, Tbil, TBA) in the DDC model and alleviated metabolic disturbances (dyslipidemia and insulin resistance) in the NASH model. Mechanistic studies revealed that Annexin A2 (ANXA2) is a direct target of evodiamine in HSC. ANXA2 was highly expressed in activated HSCs and fibrotic liver tissues, with levels correlating positively with fibrosis severity. Evodiamine promoted ANXA2 ubiquitination and proteasomal degradation, inhibited the downstream PI3K/AKT signaling pathway, and downregulated CCND1 expression, thereby blocking HSC activation and proliferation. Functional assays confirmed that ANXA2 inhibition replicated the anti-fibrotic effects of evodiamine, while its overexpression attenuated the drug's efficacy.

Conclusions: This study successfully identified the natural compound evodiamine from traditional Chinese medicine formulations as a potent inhibitor of HSC activation. We demonstrate for the first time that evodiamine exerts its anti-fibrotic effects by directly targeting ANXA2/PI3K/AKT/CCND1 axis in HSCs. These findings provide an experimental basis for developing evodiamine as a promising candidate for the treatment of liver fibrosis.

Keyword(s) : Evodiamine, liver fibrosis, hepatic stellate cells, Annexin A2, PI3K/AKT

1422 - Unveiling the pathogenesis and early warning biomarkers of acute liver failure associated with disulfidptosis through bioinformatics analysis and experimental validation

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Background / Hypothesis: Acute liver failure (ALF) represents a clinical syndrome with rapid onset and high mortality. Nevertheless, the pathogenesis, early warning and treatment of ALF remain to be elucidated. Disulfidptosis is a newly identified form of regulated cell death. The potential role and clinical value of disulfidptosis in ALF has not been explored yet. **Materials and Methods:** We employed bioinformatics analysis, machine learning, and experimental validation to investigate this hypothesis. Four microarray datasets (GSE14668, GSE38941, GSE62029 as training datasets, and GSE96851 as the independent validation cohort) were retrieved from the Gene Expression Omnibus (GEO) database using the search terms 'acute liver failure' and 'ALF'. ALF-related modules were identified using WGCNA. ALF-specific disulfidptosis-related genes (ALF-DRGs) were screened by intersecting module genes, differentially expressed genes (DEGs), and disulfidptosis-related genes (DRGs). Hub genes were identified through machine learning algorithms, and their early warning power was evaluated. Expression levels were validated in ALF mice and patients using qPCR and immunohistochemistry (IHC). Immune infiltration analysis was performed, and transcription factor (TF) and miRNA regulatory networks were predicted.

Results: Eight ALF-DRGs were screened out, of which, four genes including SLC7A11, IQGAP1, GYS1 and PDLIM1 were identified as hub genes. DCA, calibration curve and ROC analysis exhibited their high early warning power for ALF. The mRNA and protein levels of SLC7A11 and IQGAP1 were significantly elevated in the

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livers of ALF mice and patients. Immune infiltration analysis revealed significant correlations between hub genes and various immune cells. Furthermore, TF and miRNA prediction identified potential upstream regulators of hub genes. Conclusions: Disulfidptosis probably holds a pivotal place in the pathogenesis of ALF, and four disulfidptosis-related hub genes exhibit high early warning power for ALF. Our work provides novel insights into the pathogenesis, early warning and treatment of ALF.

Keyword(s) : Acute liver failure; Disulfidptosis; Machine learning; Early warning; Immune infiltration

1423 - Characterisation of people living with chronic hepatitis B virus infection in China and stratification by HBsAg levels

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Background / **Hypothesis:**
Chronic hepatitis B virus (HBV) infection is a significant global public health concern, particularly in China where prevalence is high, leading to serious outcomes such as cirrhosis and hepatocellular carcinoma (HCC). Hepatitis B surface antigen (HBsAg) levels impact treatment response, with low levels predicting better outcomes. However, quantitative HBsAg (qHBsAg) testing is not routinely conducted, leading to limited published data. This study describes patients' characteristics, clinical outcomes and HBsAg loss according to different qHBsAg levels measured at the index date among adults with chronic HBV infection in mainland China.

Materials and **Methods:**
This retrospective cohort study used Tianjin regional electronic health records from 82 hospitals in China

(January 1, 2016–December 31, 2023). Adult patients with a documented diagnosis of chronic HBV infection and at least one qHBsAg measurement were eligible. The index date was the date of the first recorded qHBsAg test, which was required to occur within 12 months before or after initial chronic HBV infection diagnosis. Analyses' baseline was set at one year prior to the index date; patients' demographic and clinical characteristics were assessed during the baseline period. A sub-cohort of patients without major liver adverse outcomes (MALO), including compensated or decompensated cirrhosis, HCC or liver transplant at baseline, was identified to estimate rates of subsequent clinical and virological outcomes during follow-up.

Results:

A total of 33,826 patients were included (mean [SD] age 43.3 [13.4] years; 57.8% male). At baseline, 14.4% patients had qHBsAg ≤100 IU/mL, 21.8% >100–≤1000 IU/mL, 21.6% >1000–≤3000 IU/mL and 42.2% >3000 IU/mL. HBeAg positive rates increased with HBsAg levels (8.5% for ≤100 IU/mL to 55.6% for >3000 IU/mL). Among a sub-cohort of 28,731 patients without MALO at baseline (mean [SD] age 41.7 [12.9] years; 55.9% male), 5380 (18.7%) were ever treated with nucleos(t)ide analogues (NAs) and 388 (1.4%) with interferon; 13.4% developed compensated cirrhosis, 8.6% decompensated cirrhosis, 3.9% HCC, and 1.6% died in-hospital or underwent liver transplant during an average follow-up of 4.5 years; 656 (2.3%) achieved HBsAg loss (262 [6.7%] with baseline qHBsAg ≤100 IU/mL, 113 [1.9%] with >100–≤1000 IU/mL, 79 [1.4%] with >1000–≤3000 IU/mL and 202 [1.5%] with >3000 IU/mL). Among those without MALO at baseline who were treated with NAs, 122 (2.3%) experienced HBsAg loss.

Conclusions: This large chronic HBV infection cohort reveals the distribution of qHBsAg levels in real-world clinical settings and may help identify patients more likely to achieve functional cure with emerging therapies.

Funding: GSK (study 219254).

Keyword(s) : chronic HBV infection, quantitative HBsAg, long-term outcomes, HBsAg loss, patient characteristics, China

1426 - Double plasma molecular adsorption system improves 90 day survival in patients with acute on chronic liver failure

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) is a clinical syndrome with high mortality, characterized by rapid hepatic deterioration triggered by an acute insult in individuals with underlying chronic liver disease. As a liver replacement therapy, artificial liver support systems are now extensively employed in the management of ACLF. However, the survival benefits of the double plasma molecular adsorption system (DPMAS) and its combined modalities remain incompletely elucidated and warrant further investigation.

Materials and Methods: Clinical data of hospitalized ACLF patients admitted to Beijing You'an Hospital, Capital Medical University, from January 2022 to June 2025 were prospectively collected, with 90-day follow-up. ACLF was diagnosed according to at least one of the following: Asian Pacific Association for the Study of the Liver (APASL), European Association for the Study of the Liver (EASL), or the 2025 Chinese guideline. Kaplan-Meier analysis evaluated survival, and univariable and multivariable Cox regression assessed the prognostic impact of DPMAS.

Results: A total of 624 ACLF patients were enrolled and divided into the DPMAS group (n=235) and control group (n=389) according to DPMAS therapy (alone or combined). After 1:1 propensity score matching (PSM) based on MELD, MELD-Na, and ACLF grades, 235 patients remained in each group. Post-matching, 90-day transplant-free survival was higher in the DPMAS group than in controls (58.3% vs. 43.4%, $P < 0.05$). Stratified analysis by ACLF grade showed a significant survival benefit of DPMAS in grade 0-1 patients (64% vs. 48%, $P < 0.05$), but not in grades 2 or 3. When stratified by MELD-Na, DPMAS conferred a survival advantage in patients with MELD-Na ≥ 25 (53% vs. 29%, $P < 0.05$), but not in lower tiers. In patients with ACLF grade 0-1 and $15 \leq \text{MELD-Na} < 25$, 90-day survival was higher in the DPMAS group (60% vs. 43%, $P < 0.05$). Multivariate Cox analysis in this subgroup indicated that DPMAS independently reduced 90-day mortality by 44% (HR = 0.56, 95% CI: 0.37–0.89, $P < 0.05$).

Conclusions: DPMAS therapy was associated with a significant reduction in mortality among ACLF patients, with the most pronounced benefit observed in those with ACLF grades 0-1 and $15 \leq \text{MELD-Na} < 25$.

Keyword(s) : DPMAS, ACLF, Prognosis, ACLF grades, MELD-Na

1431 - Persistent sarcopenia confers a cumulative risk of fibrosis progression and liver related events in biopsy proven metabolic dysfunction associated fatty liver disease

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Background / Hypothesis: Baseline sarcopenia is associated with fibrosis progression and liver-related events (LREs) in patients with metabolic dysfunction-associated fatty liver disease (MAFLD), yet the prognostic value of its dynamic status remains unclear. We aimed to investigate the effect of dynamic changes in sarcopenia status on LREs and fibrosis progression characterized by liver stiffness measurement (LSM) increasing across risk thresholds.

Materials and Methods: Patients with biopsy-proven MAFLD were enrolled from a prospective cohort with follow-up every 6 months for longitudinal assessment of LSM and appendicular skeletal muscle mass (ASM). ASM was measured via bioelectrical impedance analysis (BIA). Sarcopenia was defined as ASM/body weight $< 29.0\%$ in men and $< 22.9\%$ in women, or an ASM/BMI ratio < 0.789 in men and < 0.512 in women. Primary endpoints were liver fibrosis progression (defined as an upward LSM transition from < 10 kPa to 10–15 kPa, or from 10–15 kPa to ≥ 15 kPa), non-invasive diagnosis of clinically significant portal hypertension (CSPH, Baveno VII criteria) and LREs (decompensated cirrhosis, liver transplantation, hepatocellular carcinoma, and liver-related death). Patients were stratified by dynamic sarcopenia status into non-sarcopenia (no occurrence) and sarcopenia (any occurrence) groups; the latter was subdivided into persistent (present at all visits) and non-persistent sarcopenia (incident or reversed sarcopenia). Prognostic risk was quantified using conventional and time-dependent Cox regression analyses.

Results: A total of 239 patients were included. Sarcopenia was identified in 67 (28.0%) patients at a median age of 53 years (IQR: 39–60), with 65.7% being female. During a median follow-up of 36 months, disease progression occurred in 34 (14.2%) patients. Progression rates were significantly higher in the sarcopenia group compared to the non-sarcopenia group (22.4% vs 11.1%, $P = 0.024$; Log-rank $P = 0.025$). In multivariate Cox regression adjusted for age, baseline diabetes, creatinine, PLT, and ALB, sarcopenia remained an independent risk factor for disease progression (HR=2.153, 95% CI: 1.067–4.343, $P=0.032$). Subgroup analysis revealed that persistent sarcopenia accounted

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for the principal risk of disease progression compared to the non-sarcopenia group (26.3% vs 11.1%, $P = 0.014$), whereas non-persistent sarcopenia showed no significant risk. Time-dependent Cox analysis confirmed the significant dynamic risk of sarcopenia in both univariate (HR=1.039, 95% CI: 1.006-1.073, $P = 0.019$) and multivariate analysis (HR=1.036, 95% CI: 1.003-1.071, $P = 0.032$). Conclusions: Dynamic sarcopenia status is significantly associated with the risk of disease progression, and persistent sarcopenia confers a substantial cumulative risk of disease progression, independent of metabolic comorbidities.

Keyword(s) : Sarcopenia, Dynamic status, MAFLD, Liver fibrosis progression, Liver-related events.

1436 - HBV Inhibits Stress Granule Formation by Hijacking SG Core Proteins into the Nucleocapsids

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Background / Hypothesis: Hepatitis B virus (HBV) infection is the leading cause of chronic hepatitis B, cirrhosis, and hepatocellular carcinoma (HCC) worldwide. Due to the incomplete understanding of the mechanisms of virus-host interactions, there is currently a lack of effective therapeutic drugs for chronic hepatitis B. Stress granules (SGs) are membrane-less cellular compartments that form upon exposure to various environmental stressors including viral infection, and play a critical role in the antiviral response of host cells. Many viruses have developed distinct strategies to suppress SG assembly for survival. However, the relationship between HBV and SG formation remains unknown.

Materials and Methods: In this study, we performed immunofluorescence staining of G3BP1 and used confocal microscopy to examine the impact of HBV replication on SG formation. The role of viral proteins in SG assembly was investigated by overexpressing assay. To determine whether the suppression of SGs is mediated by encapsidation process, we treated cells with CpAMs or mutated key packaging sites mutations (polymerase R105A or core Y132A). Meanwhile, polysome profiling and ribosome sequencing were performed to investigate the effect of SGs on viral translation and replication.

Results: Our study indicated that although HBV infection and replication induce phosphorylation of eIF2 α -the trigger of SG formation, no SGs were observed in HBV-replicating cells. Conversely, HBV infection and replication significantly inhibit SG formation triggered by various stimuli. This inhibitory effect was abolished by treatment with the CpAMs, and the mutation in key packaging sites, indicating that encapsidation process is essential for HBV-mediated SG suppression. Mechanistically, proteomic analysis of nucleocapsid-associated proteins revealed the sequestration of SG core components within viral capsids. Overexpression of these SG proteins rescued SG formation, confirming their functional hijacking by HBV. On the other hand, we confirmed SGs mediated the arrest of HBV translation, thereby suppressing viral replication. Conclusion: HBV replication subverts SG formation by sequestering core SG components into capsids, sustaining viral replication by evading translational arrest, which offers critical insights into the HBV-host interaction and viral replication strategy.

Conclusions: HBV replication subverts SG formation by sequestering core SG components into capsids, sustaining viral replication by evading translational arrest, which offers critical insights into the HBV-host interaction and viral replication strategy.

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Keyword(s) : HBV, stress granule, viral replication

1447 - N acetyl cysteine plus corticosteroid versus corticosteroid alone for treatment of acute severe autoimmune hepatitis a randomized clinical trial

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Background / Hypothesis: Corticosteroids are mainstay of treatment in patients with acute severe autoimmune hepatitis (AS-AIH). However, a proportion of these patients will need liver transplantation due to liver failure and its complications. While N acetyl cysteine (NAC) has been used in patients with acute liver failure, there is no data about its usefulness in patients with AS-AIH. In this study, we aimed to investigate the impact of NAC in treatment of patients with AS-AIH.

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Materials and Methods:
In a prospective controlled trial, patients diagnosed with AS-AIH were randomized to receive prednisolone alone or prednisolone plus N acetyl cysteine (150 mg/kg/hour then 12.5 mg/kg/hour for 4 hours and 6.25 mg/kg/ hour for 16 hours). Intravenous NAC (600 mg twice daily) was continued for the next 3 days. Prednisolone was started with a dose of 60 mg per day for the first 10 days and tapered with 10 mg every 10 days. Patients were followed for 6 months. Primary outcome was liver transplant free survival and secondary outcomes were time to recovery and overall survival. Recovery was defined as decreasing INR below 1.5 (INR <1.5) with declining aminotransferase and bilirubin. AS-AIH was defined based on the international autoimmune hepatitis group score as acute presentation of AIH with an international normalized ratio (INR) ≥ 1.5 and without liver cirrhosis and hepatic encephalopathy.

Results:

A total of 57 patients diagnosed with AS-AIH and mean age of 31.03 ± 8.95 years were included. There were 47 women (82.5 %) and 10 men (17.5 %). Thirty one patients were assigned to receive NAC and prednisolone and 26 patients were assigned to receive prednisolone alone. Liver transplant free survival was 93.4 ± 4.5 % in patients who received NAC and 72.7 ± 8.8 % in patients who did not receive NAC (log rank, $P=0.025$). In a multivariable cox regression model treatment with NAC was an independent predictor of liver transplant free survival at 6 months follow-up (HR: 8.84; 95 % CI: 1.17 – 66.66; $P=0.035$). Overall survival was 93.5 ± 4.4 % in patients who received NAC and 88.5 ± 6.3 % in patients who did not receive NAC(log rank, $P=0.47$). Time to recovery was shorter in patients who received NAC compared to those who did not receive NAC (OR: 4.86; 95 % CI: 1.49- 15.79; $P=0.007$).

Conclusions:

Treatment with NAC added to corticosteroid was associated with improved transplant free survival and will shorten time to recovery in patients with AS-AIH.

Keyword(s) : acute severe autoimmune hepatitis; N acetyl cysteine; liver transplantation; Corticosteroid

1456 - Determination of Ankyrin Repeats Domain (ANK) of RNASE L Gene in Hepatitis C Patients and its effects on Viral Load

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Background / Hypothesis:

: Hepatitis C virus (HCV) is one of the major global causes of death. Different types of gene are involved as Ankyrin repeat domains of RNASE L gene. It performs a significant role in antiviral response, regulated by interferon, and involved in cleavage of RNA. Therefore, aim of this study was to identify Ankyrin repeat domain expression in Hepatitis C positive patients and correlate it with viral load of Hepatitis C.

Materials and Methods:

: In this study, a total of 80 HCV positive patient's whole blood samples were investigated. RNA was extracted from plasma followed by Real Time PCR for quantization of HCV viral load and genotypic analyses. DNA was also extracted from these samples followed by PCR amplification of Ankyrin repeat domain of RNASE L gene. Data was analyzed using SPSS

Results:

All of the patients (n=80) included in study had HCV infection. Mean age of patients was 50.86 ± 14.84 years. Among them, 48(63.8%) were males and 32 (36.1%) were females. Majority of patients were males and belonged to age group 58-73 years age. All HCV infected individuals 36 (45%) had HCV genotype 3 and had viral loads mean range 837404.21 ± 1302 . Therefore, Ankyrin repeats domain of RNASE L gene expression was high in HCV patients sample with viral load of 17.00 ± 15.1 .

Conclusions:Ankyrin repeat domain expression was observed in Hepatitis C patients and its significant correlation with viral load of Hepatitis C. Ankyrin repeat domain of RNASE L gene in conjunction with therapeutic intervention are required for establishing better strategies for controlling HCV infection.
Keywords: Hepatitis C Virus; Ankyrin Repeat; Ribonuclease L; Polymerase Chain Reaction (PCR).

Keyword(s) : Keywords: Hepatitis C Virus; Ankyrin Repeat; Ribonuclease L; Polymerase Chain Reaction (PCR).

1459 - Revisiting HepaScore New Evidence for Non Invasive Fibrosis Assessment in Indonesian Patients with Chronic Hepatitis B

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Background / Hypothesis:
Liver fibrosis assessment is essential in the management and prognosis of chronic hepatitis B (CHB). Although liver biopsy remains the gold standard, its invasiveness and risks highlight the need for accurate non-invasive alternatives. This study aimed to re-evaluate HepaScore — a novel fibrosis index — and compare its diagnostic performance with APRI and FIB-4 using transient elastography (TE) as the reference standard. To our knowledge, this is the first study to apply HepaScore in an Indonesian CHB cohort.

Materials and Methods:
This cross-sectional diagnostic study included 90 CHB patients, equally divided into fibrosis (n=45) and non-fibrosis (n=45) groups based on TE results. Univariable and bivariate analyses, along with Receiver Operating Characteristic (ROC) curve testing, were performed to assess diagnostic accuracy. Metrics including AUC, sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and likelihood ratio (LR) were calculated for HepaScore, APRI, and FIB-4.

Results:
HepaScore demonstrated the highest diagnostic performance with an AUC of 0.860 (95% CI: 0.771–0.924; $p < 0.001$), outperforming APRI (AUC 0.796, 95% CI: 0.698–0.874) and FIB-4 (AUC 0.623, 95% CI: 0.515–0.723). When combined with APRI, HepaScore achieved an optimal AUC of 0.895 (95% CI: 0.813–0.950; $p < 0.001$). The HepaScore-APRI combination improved diagnostic accuracy to 86.7% (sensitivity 84.4%, specificity 88.9%).

Conclusions:
HepaScore shows superior diagnostic accuracy compared with APRI and FIB-4 in detecting liver fibrosis among CHB patients. Combining HepaScore with APRI yields the most reliable non-invasive approach, particularly valuable in resource-limited settings where TE is unavailable.

Keyword(s) : HepaScore; APRI; FIB-4; chronic hepatitis B; liver fibrosis; non-invasive diagnosis; transient elastography.

1461 - A Retrospective Study on PD 1 CTLA 4 Bispecific Antibody Therapy Promoting Partial Functional Cure of Hepatitis B in Patients with HBV Related Hepatocellular Carcinoma
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Background:
Immunocheckpoint inhibitors (ICIs), particularly those targeting programmed death-1 (PD-1) and cytotoxic T-lymphocyte-associated protein 4 (CTLA-4), have shown potential in reversing T-cell exhaustion in chronic hepatitis B (CHB). While PD-1 monotherapy trials demonstrate preliminary efficacy, the role of CTLA-4 targeting in CHB remains unclear. This study evaluated the impact of PD-1/CTLA-4 bispecific antibody therapy on hepatitis B surface antigen (HBsAg) clearance in patients with HBV-related hepatocellular carcinoma (HCC).

Materials and Methods:
In this retrospective cohort study, 78 HCC patients receiving PD-1/CTLA-4 bispecific antibody therapy at the Fifth Medical Center of the Chinese PLA General Hospital (September 2022–October 2024) were screened. After applying inclusion criteria, 20 HBV-HCC patients comprised the PD-1/CTLA-4 group. Using propensity score matching (1:2), a control group (n=39) receiving PD-1 inhibitor monotherapy was matched for gender, age, HBV DNA load, and HBsAg titer. Factors associated with HBsAg loss or >1 log decline were analyzed via competing risk Cox regression.

Results:
The baseline characteristics of the two groups showed no significant differences. With a median follow-up time of 17.8 months, one patient (5.8%) in the PD-1/CTLA-4 group achieved HBsAg loss, while no patients in the PD-1 group reached this endpoint. The incidence of HBsAg decline >1 log was 20.00% (5/20) in the PD-1/CTLA-4 group compared to 7.69% (3/39) in the PD-1 group (Fig.1 A). Competing risk Cox regression analysis revealed that the cumulative incidence of HBsAg loss or decline >1 log was significantly higher in the PD-1/CTLA-4 group than in the PD-1 group ($P = 0.017$) (Fig.1 B). After adjusting for competing risks of mortality, multivariate analysis confirmed that PD-1/CTLA-4 therapy (hazard ratio [HR] 5.119; 95% CI 1.337–19.603; $p = 0.017$), HBV DNA ≥ 1000 IU/mL (HR 0.998; 95% CI

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0.996-1.000; $p = 0.032$), and total bilirubin (TBIL) level (HR 0.859; 95% CI 0.740-0.997; $p = 0.046$) were significantly associated with HBsAg loss or decline >1 log (Fig.1 C).

Analysis of overall survival (OS) showed no significant difference between the PD-1/CTLA-4 group and the PD-1 group (Fig.1 D). Furthermore, HBsAg loss or decline was not correlated with OS in both groups (Fig.1 E). However, a trend toward improved OS was noted in PD-1/CTLA-4 responders (Fig.1 F-G).

Conclusions:

PD-1/CTLA-4 bispecific antibody therapy significantly enhances HBsAg clearance compared to PD-1 monotherapy in HBV-HCC patients, supporting further investigation of dual ICI strategies for CHB functional cure. Larger prospective studies are needed to validate these findings.

Keyword(s) : Immunocheckpoint inhibitors; Chronic hepatitis B; PD-1/CTLA-4 bispecific antibody; Functional cure

1463 - Real World Outcomes of Durvalumab Plus Tremelimumab (STRIDE) for Unresectable Hepatocellular Carcinoma in Japanese Patients

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Background / Hypothesis: Atezolizumab plus bevacizumab has been widely adopted as first-line therapy for unresectable hepatocellular carcinoma (uHCC). In Japan, the durvalumab plus tremelimumab regimen (STRIDE) was approved in December 2022. STRIDE is distinct from prior systemic therapies for HCC in that it consists solely of immune checkpoint inhibitors without VEGFR inhibition. Notably, STRIDE has demonstrated durable long-term survival, with reported 5-year and 6-year survival rates of 19.4% and 17.1%, respectively.

However, concerns remain regarding early disease progression and the comparatively high incidence of immune-mediated adverse events (imAEs), and the optimal patient population for STRIDE remains unclear. This study aimed to evaluate real-world treatment outcomes and identify clinical indicators that may guide therapy selection.

Materials and Methods: We retrospectively analyzed 65 patients with uHCC who received STRIDE and were registered in a multi-center cohort study of hepatobiliary malignancies (BLOOM study) up to October 2025. Patient characteristics, objective response rate (ORR), disease control rate (DCR), progression-free survival (PFS), and imAEs were evaluated.

Results:

The median age was 68 years (range 15–86), and 55 patients (84%) were male. Etiology included HCV in 8 patients (12%), HBV in 8 (12%), and non-viral disease in 49 (76%). ECOG performance status was 0/1 in 53/12 patients. Child–Pugh scores were 5/6/ ≥ 7 in 30/21/14 patients, and the median ALBI score was -2.36 (range -3.8 to -1.26). BCLC stage was B/C in 19 (29%)/46 (71%). STRIDE was administered as first-line therapy in 43 patients (64%) and as second-line or later in 22. Among 58 evaluable patients, ORR was 20% (12/58), and DCR was 50% (29/58). Median PFS was 6.1 months (95% CI: 3.7–not reached). STRIDE as initial treatment showed numerically higher ORR (25% vs. 13%; $P = 0.329$) and significantly longer PFS compared with later-line use (not reached vs. 2.7 months; HR, 0.31; 95% CI, 0.15–0.60; $P < 0.01$). IMAEs occurred in 24 patients (36%), including 11 with ≥ 3 events. The most frequent severe toxicities were colitis, adrenal insufficiency, and dermatologic events (each $n=2$).

Conclusions:

Real-world data suggest that STRIDE provides superior PFS when used as initial treatment, supporting the clinical importance of tremelimumab single priming at treatment initiation. Early-line use may maximize therapeutic benefit in patients with uHCC.

Keyword(s) : hepatocellular carcinoma,STRIDE

1466 - A new prediction model based on plasma ammonia and the Chinese Group on the Study of Severe Hepatitis B ACLF II score for patients with Hepatitis B virus related acute on chronic liver failure.

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Background / **Hypothesis:**
Early risk prediction is essential to reduce the mortality
of patients with hepatitis B virus-related acute-on-
chronic liver failure (HBV-ACLF). We aim to establish a
new short-term prognosis (28 days) model for patients
with HBV-ACLF.

Materials and **Methods:**
We designed a new model (COSSH II -AMM) based on
the serum ammonia (AMM) and the Chinese Group on
the Study of Severe Hepatitis B-ACLF- II score (COSSH-
ACLF II s) by multivariate COX regression analysis in
the deriving cohort of 408 patients with HBV-ACLF from
October 2012 to August 2024. External validation was
carried out using data from another 92 patients with
HBV-ACLF.

Results:
The serum ammonia/reference laboratory upper limit of
normal for ammonia (AMM-ULN) levels were
significantly elevated in non-survivors compared to
survivors (1.2 vs. 0.88, $P < 0.001$). The cut-off of AMM-
ULN was 1.6 determined by using X-tile. With an area
under the area under the curve (AUROC) of 0.875 for 28-
day mortality prediction, the COSSH II-AMM model
showed significantly better performance than existing
prognostic models (Child-Pugh: AUROC=0.725; MELD:
AUROC=0.820; AARC: AUROC=0.833; CLIF-C ACLF:
AUROC=0.853; COSSH-II-ACLF: AUROC=0.860). The
cut-off of COSSH II-AMM was 6.4 determined by using
X-tile. Kaplan-Meier analysis revealed significantly
divergent survival curves between patients with COSSH
II-AMM scores < 6.4 and those with scores ≥ 6.4 ($P <$
0.0001). The COSSH II-AMM scoring system
demonstrated significantly improved concordance
between predicted and observed overall survival (OS)
outcomes in the derivation cohort when compared with
conventional prognostic models.

Conclusions:

Our study substantiated the prognostic value of blood
ammonia levels in predicting 28-day mortality among
HBV-ACLF patients. The novel prognostic score (COSSH
II-AMM) significantly improves survival prediction
accuracy in patients with HBV-ACLF.

Keyword(s) : Hepatitis B virus; Acute-on-chronic liver
failure; Ammonia; Prediction model; Mortality.

1468 - Multicenter evaluation of the efficacy and safety of rechallenge with immune checkpoint inhibitors in unresectable hepatocellular carcinoma

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Background / Hypothesis: Immune checkpoint inhibitors
(ICIs) are a standard treatment for unresectable
hepatocellular carcinoma, but sometimes they must be
discontinued because of resistance or immune-related
adverse events. The present study explored whether
later resuming ICI therapy in such patients
("rechallenge") can improve their prognosis.

Materials and Methods: Data were retrospectively
analyzed for patients treated between March 2019 and
May 2024 at 12 medical centers in China. Patients had
unresectable hepatocellular carcinoma and were initially
treated with ICIs, which were discontinued due to
resistance or severe immune-related adverse events and
then later resumed. Event-free survival, objective
response and disease control based on RECIST 1.1
criteria, as well as incidence and severity of immune-
related adverse events, were assessed separately during
first- and second-line ICI therapy.

Results: The final analysis included 272 patients (247
men) with a median age of 50 (IQR 43-58) years old at
enrollment; first-line ICIs were discontinued in 253
patients (93.0%) because of tumor progression. Both
first- and second-line therapies lasted a median of 3.0
months and, for approximately 80% of patients,
involved the combination of ICIs and targeted agents
rather than ICI monotherapy. During first- or second-
line therapy, respective median event-free survival times
were 5.3 (95% CI 4.7-5.9) months and 4.4 (95% CI 3.8-
5.0) months, objective response rates were 23.2% and
18.4%, disease control rates were 69.9% and 48.2%, and
rates of immune-related adverse events of grades 3-4
were 18.4% and 19.9%. Objective response was
observed in 26 patients (9.6%) during both therapy lines,
while 10 (12.2%) suffered disease progression during
first-line therapy yet achieved partial response during
second-line treatment. No deaths due to immune-
related adverse events occurred during the study.

Conclusions: ICI rechallenge following tumor

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progression or severe immune-related adverse events can benefit some patients with unresectable hepatocellular carcinoma without posing additional safety risks.

Keyword(s) : Hepatocellular Carcinoma, Immune Checkpoint Inhibitors, Rechallenge, Tumor Progression

1469 - Targeting NEK9 in hepatocellular carcinoma to remodel immune microenvironment and enhance immunotherapy

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Background / **Hypothesis:**
Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality, often associated with immune evasion that hampers the effectiveness of therapies. This study explores the role of NEK9, a kinase involved in regulating immune microenvironment remodeling in HCC, and its potential as a therapeutic target to enhance the efficacy of immune checkpoint inhibitors, such as PD-L1 blockade.

Materials and **Methods:**
We analyzed NEK9 expression in clinical HCC samples and its correlation with prognosis. Genetic manipulation and pharmacological inhibition of NEK9 were employed in HCC cell lines and mouse models. The effects of NEK9 on immune cell infiltration, PD-L1 expression, and tumor progression were assessed. The combination of NEK9 inhibition and PD-L1 blockade was tested to evaluate its therapeutic potential.

Results:
NEK9 expression was significantly elevated in HCC tissues and correlated with poor prognosis. Mechanistically, NEK9 facilitated the stabilization of NF- κ B2 by phosphorylating TRIM28 and USP46, leading to increased PD-L1 expression and myeloid-derived suppressor cell (MDSC) accumulation. Inhibition of NEK9 reduced PD-L1 levels, restored CD8⁺ T cell infiltration, and improved the response to PD-L1 blockade, demonstrating enhanced antitumor effects both in vitro and in vivo.

Conclusions:
NEK9 plays a crucial role in immune evasion in HCC by stabilizing NF- κ B2 and promoting PD-L1-mediated immune suppression. Targeting NEK9 can effectively remodel the tumor immune microenvironment, enhancing the efficacy of immunotherapies, particularly in combination with immune checkpoint inhibitors.

Keyword(s) : Hepatocellular carcinoma, kinase, immunotherapy

1471 - Biomolecular Mapping of Circulating Extracellular Vesicles to Develop AI Driven Prognostic Panels for Stratifying Acute Liver Failure Patients at Risk of Early Mortality

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Background / Hypothesis: Acute liver failure (ALF) is a severe condition with high mortality, marked by microbial and metabolic factors in the blood. Plasma-derived exosomes, reflecting inflammation, offer a non-invasive tool to identify ALF patients at high risk of early mortality.

Materials and Methods: Plasma samples from 320 subjects (270 ALF, 50 healthy) were analyzed for EV isolation using PROSPR, characterized by Transmission Electron Microscopy (TEM), Dynamic light scattering (DLS) and Nano particle tracking analysis (NTA) and Proteomics. Meta-proteomics and metabolomics were performed on 50 ALF and 20 controls. EV data were correlated with clinical parameters and ALF complications. A diagnostic metabolite panel was identified and validated in a separate cohort (220 ALF; 153 non-survivors, 67 survivors) using HRMS and machine learning.

Results: Plasma-derived exosomes (120–200 nm) were characterized with a concentration of $3.25 \times 10^{11} \pm 0.55$ particles/mL, zeta potential of -30.67 ± 3.6 mV, and >20 EV-specific proteins (Vesiclepedia). Metabolomics and meta-proteomics of exosome cargo identified over 1000 metabolites and bacterial peptides from >15 phyla and >25 genera. More than 800 metabolites were differentially expressed in ALF exosomes, with greater differences in non-survivors ($FC > 1.5$, $p < 0.05$). Non-survivor specific metabolites showed increased Sugar metabolism and Beta oxidation of long chain fatty acid, Primary bile acid synthesis linked to advanced glycation end products and inflammation ($p < 0.01$). Bacterial-peptide based alpha/beta diversity was higher in ALF more so in Non-survivors corresponding to increase in Bacteroidota, Actinomycetota functionally linked to energy and xenobiotic metabolism ($p < 0.05$). A strong correlation ($r^2 > 0.91$, $p < 0.05$) between Shannon-metabolome, Shannon-bacterial diversity, functionality,

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and clinical parameters suggest that EV metaproteomic cargo drives metabolomic cargo and its functionality in Non-survivors ($p < 0.05$). Higher EV levels of *Enterococcus faecium* and *Klebsiella pneumoniae* in non-survivors correlated with metabolites and severity ($r^2 > 0.8$, $p < 0.05$). The panel of 5 key EV metabolites (4-Amino-2-methyl-5-phosphomethylpyrimidine (10.9-logFolds), Deoxycholic 3-glucuronide (8.8-logFolds), 2'-Deoxyinosine (4.78-logFolds), D-erythro-1-(Imidazol-4-yl)glycerol 3-phosphate (4.17-logFolds) and Palmitoyl sphingomyelin (3.8-logFolds), showed diagnostic accuracy $> 95\%$ (AUC=0.98) with Hazard Ratio (17.58). In panel of key Metabolites, significant increase in Deoxycholic-3-glucuronide level showed highest normalized importance and predicted early-mortality with $> 95\%$ accuracy/sensitivity/specificity using ANN and ML. Interestingly, beta glucuronidase upregulated (> 2 -fold), leading to a notable rise in conjugation of Deoxycholic Acid into Deoxycholic-3-glucuronide and downregulation of ABCB3 (MRP3) transporter leads regulation of intracellular accumulation of bile acids and activity of MAPK signaling which contributed to hyper-inflammation and oxidative in ALF-non-survivors.

Conclusions: Plasma-derived exosome metabolite cargo panel indicates hyper-inflammation and advanced glycation specific to non-survivors, potentially identifying ALF patients at risk of early mortality.

Keyword(s) : Liver failure, Exosomes, omics, Metabolome, DILI, Bile Acid

1472 - Divergent immune resolution and protracted immuno metabolic memory in hepatitis E revealed by cross species multi omics

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Background : Hepatitis E virus (HEV) infection is typically self-limited in immunocompetent hosts but can lead to severe outcomes such as fulminant hepatitis in pregnant women or immunocompromised individuals. Current evidence suggests that liver injury is driven by dysregulated host immunity rather than direct viral cytopathy. To comprehensively delineate the dynamic immune landscape of HEV infection, we applied an

integrated multi-omics and cross-species approach.

Materials and Methods: We performed a longitudinal multi-omics analysis in a rabbit model infected with HEV genotype 3. Liver and serum samples were collected at baseline (HEV-BL), first viremia (HEV-F), peak viremia (HEV-P), and viral clearance (HEV-N) stages. Transcriptomic and proteomic profiles were generated from liver tissues, and metabolomic sequencing was conducted on serum. For cross-species validation, we analyzed public transcriptomic data from HEV gt1- and HCV-infected chimpanzees (GSE22160).

Results:

In rabbits, hepatic transcriptomics revealed phased immune activation with sustained enrichment of adaptive immunity pathways—including T cell activation and antigen presentation—even post-clearance, indicating failure to restore immunological homeostasis. CD8⁺ T cell activity remained suppressed throughout infection, while innate immune populations (eosinophils, neutrophils, Th17 cells) persisted, reflecting a maladaptive state with unresolved inflammation. Proteomic analysis confirmed broad and persistent immune activation, with all differentially expressed proteins upregulated across stages and antigen presentation pathways consistently enriched. Metabolomic profiling identified enhanced T cell-associated pathways (Th1/Th2/Th17 differentiation, NF- κ B signaling) at peak viremia, suggesting metabolic reinforcement of adaptive immunity. Integrated multi-omics highlighted purine metabolism as a cornerstone pathway persistently activated during infection and resolution. Cross-species analysis in chimpanzees demonstrated a potent but transient type I interferon response that resolved rapidly after viral clearance. Critically, comparative profiling with HCV revealed fundamentally distinct immune resolution kinetics: HEV triggered rapid interferon-stimulated gene downregulation post-clearance, whereas HCV maintained sustained immune activation, underscoring a virus-specific host engagement strategy.

Conclusions:

Our findings unveil a protracted immuno-metabolic memory post-HEV clearance, characterized by unresolved innate activation and metabolic reprogramming. This study redefines the paradigm of HEV immunopathogenesis and offers novel insights for biomarker discovery and immunomodulatory interventions in high-risk populations.

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Keyword(s) : HEV; multi-omics; anti-virus; Cross-species validation

1473 - Dominant Neoantigens in Liver Cancer Malignant Ascites

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Background / **Hypothesis:**
Neoantigen-based immunotherapies have shown promising efficacy in liver cancer, driven by the strong immunogenicity of neoantigen-reactive T cells, which are key effectors of immune checkpoint blockade (ICB). However, most patients present with advanced disease, limiting access to tumor tissue for neoantigen profiling. Malignant ascites, enriched with tumor cells, offers a feasible liquid biopsy alternative. This study aimed to determine whether dominant neoantigens exist in malignant ascites from advanced liver cancer patients and to assess their potential as personalized immunotherapy targets.

Materials and **Methods:**
Ascites samples from cirrhosis and advanced liver cancer patients were collected. Using flow cytometry, we isolated the 7-AAD⁻ CD45⁻ CD326⁺ tumor cell subset (P7 population). For samples with <2,000 cells, RNA and DNA were amplified using Smart-seq2 and the Discover-sc WGA kit. Whole-exome and RNA sequencing were performed to define somatic mutations and mutational signatures. Candidate neoantigens were predicted using VEP and NetMHCpan-4.1 based on peptide-HLA binding affinity, after removing redundant sequences and accounting for HLA loss-of-heterozygosity. Tetramer staining, flow cytometry, ELISPOT, and ELISA were used to evaluate neoantigen-specific T cells and immunogenicity.

Results:

We established a workflow enabling rare tumor cell isolation from malignant ascites for sequencing. Liver cancer patients showed significantly higher numbers of P7 tumor cells in ascites than in peripheral blood or cirrhotic controls. Single-cell sequencing revealed partial overlap between mutations in ascites-derived cells and primary or metastatic lesions. In eight patients, C>T/G>A transitions predominated, with mutational signatures corresponding to COSMIC signatures 1, 3, 12, 15, and 30. NetMHCpan analysis identified multiple putative neoantigens derived from TOP3A, TUBB6, NUTM2G, AFAP1, FGD2, NFKBIZ, and CLDND1. Tetramer assays confirmed neoantigen-specific T cells in ascites, characterized by high CD39 expression. Functional assays validated strong immunogenicity of an HLA-A*02:01-restricted AFAP1-derived neoantigen,

suggesting it as a dominant candidate.

Conclusions:

Malignant ascites from advanced liver cancer patients provides a valuable source for identifying dominant neoantigens, offering a practical strategy for discovering actionable targets in individuals lacking tumor tissue for neoantigen-based personalized immunotherapy.

Keyword(s) : liver cancer, ascites, dominant neoantigens, immunotherapy

1476 - Prediction of microvascular invasion in HCC using spatiotemporal radiomics of contrast enhanced ultrasound a deep learning model with tumor microenvironment correlation

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Background / **Hypothesis:**
The presence of microvascular invasion (MVI) in hepatocellular carcinoma (HCC) is a critical prognostic indicator, but it can only be diagnosed by postoperative histopathology. Contrast-enhanced ultrasound (CEUS), with its dynamic microvascular imaging capability, holds promise in prediction of MVI.

Materials and **Methods:**
Leveraging TimeSformer to construct and evaluate the deep learning model MAPUSE for predicting MVI, we utilized a total of 5,148 CEUS videos (>3,300,000 frames) from 1,716 HCC patients across 23 nationwide centers. After training (n=495) and validating (n=213), MAPUSE was tested on two hold-out test cohorts from the same 23 centers using different microbubble agents - Sonovue (n=92) and Sonazoid (n=98), three subgroups (tumor size >5cm, 3-5cm and <3cm) and two prospective cohorts from southern and northern China (n=120 & 130). Additionally, we conducted a transcriptomics analysis on bulk RNA sequencing data from 203 patients and single-cell RNA sequencing data from 12 patients in the training and validation cohorts. We also explored the self-attention mechanism of MAPUSE in the prospective cohort. Using overlap weighting method to mimic RCT, we tested the clinical implications of MAPUSE for immunotherapy in a cohort of 568 HCC patients after ablation, whose MVI status could not be histopathologically confirmed.

Results:
AUCs of MAPUSE in prediction of MVI in Sonovue set, Sonazoid set and both sets were 0.835, 0.858 and 0.851,

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respectively. In subgroup analysis, MAPUSE showed excellent performance in >5cm tumors (AUC=0.978) and good performance in 3-5cm tumors (AUC=0.814) and <3cm tumors (AUC=0.756). In two prospective cohorts, MAPUSE achieved AUCs of 0.886 and 0.847. The transcriptomics analysis revealed a significant association between MAPUSE and immune processes involving infiltration of CD8+ T cells. By exploring the self-attention mechanism, downregulated CD8+ T cells were observed within surgical section corresponding to peri-tumor strong attention areas. Additionally, patients after ablation with positive MVI predicted by MAPUSE can benefit from immunotherapy.

Conclusions:

The MAPUSE model is an AI model that potentially enables real-time ultrasound scanning for predicting MVI while visualizing information of the tumor immune microenvironment such as CD8+ T cell infiltration. It can not only realize the preoperative noninvasive detection of MVI, but also provide clinical decision-making basis for patients who cannot obtain pathological MVI diagnosis.

Keyword(s) : microvascular invasion, hepatocellular carcinoma, contrast-enhanced ultrasound, tumor microenvironment

1481 - Development and validation of brain age clock from psychometric hepatic encephalopathy score for early detection of hepatic encephalopathy

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Background / **Hypothesis:**
Brain age models derived from MRI or EEG are constrained by cost and accessibility. The Psychometric Hepatic Encephalopathy Score (PHES), a neuropsychological battery for hepatic encephalopathy (HE) screening, exhibits a strong age dependency, which complicates its original purpose, suggesting its potential as a convenient functional brain age estimator. We propose and validate machine learning-based methods for estimating brain age, and evaluate whether the resulting brain age gap (the difference between predicted brain age and chronological age) serves as a valid tool for early detection of HE.

Materials and **Methods:**
The brain age model was derived from a multi-regional survey of healthy Chinese adults (20-79 years) that

employed a stratified sampling strategy across geographic regions, urban/rural residence, gender, and age groups. Exclusion criteria included hepatic, neurological, and psychiatric conditions, as well as sensory impairments that affected PHES test performance. Five machine learning algorithms were trained to predict chronological age from education level and PHES subtest scores. For clinical validation, a cohort of cirrhosis patients from Peking University People's Hospital was employed for evaluation, where the model's performance in predicting the 6-month risk of hepatic encephalopathy was quantified by the area under the ROC curve (AUC). The screening performance of the brain age gap was compared to the conventional PHES scoring system.

Results:

Three of five PHES tests (NCT-A, NCT-B, and DST) exhibited significant linear relationships with age ($p < 0.001$), which were also key variables selected by Boruta feature selection. These tests, in addition to education level, were used to construct linear regression, random forest, SVM, and XGBoost models, achieving mean absolute errors of 0.518-7.694 years in predicting age, a level that is comparable to and superior to current standards using the brain image approach. When applied to cirrhosis patients, the median model-derived brain age gap was -1.4-2.2 (IQR: 13.7-27.4), with larger gaps correlated with poorer self-perceived health. The novel age-gap approach outperformed conventional PHES scoring in predicting HE onset within 6 months, AUCs of 0.657-0.772 across models vs. 0.512 using the conventional approach (all $p < 0.05$).

Conclusions:

This study used machine learning models that convert PHES results into a brain age gap, which significantly outperformed conventional scoring in detecting hepatic encephalopathy. The observed gap quantifies the accelerated functional brain aging associated with this condition. By repurposing an existing neuropsychological tool, our approach offers a practical alternative to costly neuroimaging. Further validation in larger cohorts is warranted to confirm its clinical utility.

Keyword(s) : hepatic encephalopathy, psychometric hepatic encephalopathy score, machine learning, risk assessment, age clock

1482 - From Gene Expression Profiles to Functional Networks A Multi Omics Integrative Analysis of Methyl Eugenol Promoting Cholangiocarcinoma Development

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Background / **Hypothesis:** Cholangiocarcinoma is a malignant tumour arising within the biliary tract system. In recent years, its incidence has shown an upward trend, characterised by insidious onset, rapid progression, and poor prognosis. Its aetiology remains incompletely understood; beyond known causes such as viral infections and choledochal cysts, environmental factors are increasingly being identified. Methyl eugenol, a naturally occurring compound found in spices and herbs, was classified as a Group 2B carcinogen by the International Agency for Research on Cancer in 2023. Existing research indicates methyl eugenol promotes the development of hepatocellular carcinoma, though its potential role in promoting cholangiocarcinoma requires further investigation.

Materials and **Methods:** This study obtained cholangiocarcinoma RNA-seq data from the TCGA database. Candidate genes were screened via differential analysis ($P < 0.05$, $|\log_2FC| > 1$). WGCNA was employed to identify gene modules correlated with clinical phenotypes. Methyl eugenol targets were predicted through integrated network toxicology analysis. Key genes were selected by combining protein-protein interaction (PPI) networks (STRING confidence > 0.4) and CytoHubba. Biological functions were elucidated through GO and KEGG enrichment analyses, whilst molecular docking validated the binding activity between methyl eugenol and core target proteins.

Results: The co-expression gene module identified by WGCNA as positively correlated with cholangiocarcinoma (MEturquoise module, $Cor=0.91$, $P=3e-17$) yielded 188 methyl eugenol-cholangiocarcinoma intersection targets. GO analysis revealed significant enrichment of target genes in mitotic regulation (BP, $P=1.57e-07$) and chromosome (CC, $P=5.92e-08$). KEGG analysis indicated their regulation of oncogenic pathways including cell cycle ($P=6.24e-06$) and p53 signalling ($P=0.00014$). Integrated STRING and Cytospace analyses identified five key genes (BUB1, CHEK1, PLK1, AURKB, MAD2L1). Molecular docking showed methyl eugenol binds to PLK1 with an affinity of $-6.3 \text{ kcal/mol}^{-1}$, forming

hydrogen bonds (LEU-221) and hydrophobic interactions (VAL-210/LYS-225/HIS-228) to constitute a stable complex, suggesting it may directly activate oncogenic signalling pathways.

Conclusions: These findings indicate that methyl eugenol exposure may enhance the expression of these genes in cholangiocytes, disrupt the cell cycle and promote cell proliferation, thereby aiding in the prediction of patient prognosis and identifying therapeutic targets.

Keyword(s) : Cholangiocarcinoma , Environmental Oncology

1484 - Exploring the Anticancer Effects of Spirulina Extract in Human Hepatocellular Carcinoma Cells

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is among the most common and aggressive liver cancers, often diagnosed at advanced stages and showing limited response to conventional chemotherapies. The search for safer, natural compounds with therapeutic potential has drawn attention to *Spirulina platensis* (*S. platensis*), a microalga widely recognized for its nutritional and biological value. *Spirulina* contains phycocyanin, phenolic acids, carotenoids, and essential amino acids that collectively exert strong antioxidant and anti-inflammatory effects. Despite increasing interest in its health benefits, the direct antitumor activity of *Spirulina* extract can inhibit cell proliferation and induce apoptosis in human hepatocellular carcinoma cells, possibly through oxidative stress modulation and mitochondrial signaling pathways.

Materials and Methods: Pure *S. platensis* powder (analytical grade) was purchased from a certified commercial supplier. The material was stored at room temperature in a dry, dark container until use. For experimental applications, the *Spirulina* powder was dissolved in DMSO. The human hepatocellular carcinoma cell line Huh7 was grown in DMEM containing 10% FBS, 100 Units/ml penicillin and 100

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µg/mL streptomycin, and incubated with 5% CO₂ at 37°C. The cytotoxic effect of *S. platensis* on Huh7 cells was investigated using colorimetric MTT assay. The concentrations of 0.1, 0.5, 1, 2.5, 5, 7.5, and 10 mg/ml were applied to the cells. Results: *S. platensis* was observed to exhibit dose-dependent antiproliferative properties to the Huh7 cell line, and the IC₅₀ value was determined as 5.3±0.46 mg/ml. The results showed that *S. platensis* has high antiproliferative activity at low cytotoxic doses against human hepatocellular carcinoma cells. Conclusions: *S. platensis* exhibited high cytotoxic activity against hepatocellular carcinoma cancer cells. In the future, it is planned to investigate the effects of *S. platensis* on other anticancer hallmarks (proliferation, invasion, and migration) and its antibacterial properties.

Keyword(s) : *Spirulina platensis*, Hepatocellular carcinoma, Anticancer activity, Huh7

1486 - Serum bile acids profile analysis predicts severity and progression of liver disease

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Background: Bile acids profoundly influence the onset and progression of liver disease through signaling regulation, inflammatory responses, and interactions within the gut-liver axis. During the progression from hepatitis to cirrhosis and ultimately hepatocellular carcinoma, the bile acid profile undergoes dynamic changes. However, the association between specific bile acids and distinct stages of liver disease progression remains unclear. Identifying bile acids with stage-specific signatures holds significant value for elucidating the mechanisms of liver disease progression and establishing early warning systems.

Materials and Methods: This study included 889 patients with liver disease from the Peking University Hepatology Institute, categorized as whether of severe conditions (with irreversible liver fibrosis and cirrhosis that is related to fundamental changes in monitoring frequency, complication risks, treatment regimens, and prognosis) and whether of advanced stages (liver function failure or malignant transformation). Predictive capability of different bile acids was initially assessed by regression model and then confirmed by random forest

algorithm for outcome classification. Ten machine learning algorithms were employed to construct liver disease discriminative models. The training-to-test ratio for each model was 7:3, with 5-fold cross-validation applied to calculate predictive performance.

Results: Among all patients, 185 (20.8%) had severe liver diseases, and 117 (13.2%) were in the advanced stage. Among the 15 bile acids, TCDCA, TCA, GCDCA, GCA, and TUDCA demonstrated the strongest predictive ability for liver disease outcomes. Variable importance derived from the random forest algorithm further validated the robust predictive efficacy of these five bile acids. Naive Bayes demonstrated the highest predictive capability for both classification outcomes, effectively distinguishing patients with severe liver disease and those with advanced liver disease with AUC values of 0.873 (0.821–0.925) and 0.901 (0.854–0.948), respectively.

Conclusions: This study found that the serum bile acid profile composed of specific bile acids can effectively distinguish the severity and advanced stage of liver disease. Abnormal changes are closely associated with key pathological processes such as hepatitis, cirrhosis, and the development of hepatocellular carcinoma. Mechanistic and clinical research targeting these bile acids holds promise as a novel strategy for the assessment and treatment of liver disease.

Keyword(s) : Bile Acids, Cirrhosis, Liver Disease Progression, Biomarkers, Machine Learning

1493 - Clinical determinants of treatment response in Asian hepatocellular carcinoma undergoing lenvatinib therapy the impact of non viral liver etiologies

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Background / Hypothesis: Tyrosine kinase inhibitors remain an important treatment option in the systemic therapy of advanced hepatocellular carcinoma (HCC). Increasing evidence indicates that the etiology of HCC influences response to systemic therapy. We aimed to investigate whether viral etiology impacts the survival in patients with HCC receiving lenvatinib.

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Materials and Methods: Patients with HCC treated with lenvatinib from August 2018 to June 2024 were identified from a Hong Kong population-based cohort. Virus-related HCC was defined by virological or serological evidence of hepatitis B virus (HBV) or C (HCV) virus infection. Propensity score matching (PSM) was performed at a 1:1 ratio (viral vs non-viral HCC) to mitigate intergroup confounding. The primary analysis applied an intention-to-treat design to investigate the association between viral etiology and overall survival (OS). Subgroup analyses were further conducted to assess whether this association varied by sex (male, female), presence or absence of type 2 diabetes (T2D), and status of liver cirrhosis. The OS outcomes were evaluated using the log-rank test and adjusted hazard ratios (HRs) were calculated using the Cox proportional hazards model.

Results: This study included 276 PSM-matched patients (mean age 71.3 (± 10.1) years, 77.5% male) with HCC receiving lenvatinib treatment (138 virus-related HCC with 86.2% HBV-related, 138 non-viral HCC). All covariates were well-balanced after PSM. Cirrhosis was present in 224 (81.2%) patients, while 170 (61.6%) patients had T2D. The viral etiology was associated with an increased risk of all-cause death (HR 1.3; 95% CI 1.0-1.7; $P = 0.035$), with a significantly shorter median OS (mOS) when compared to patients with non-viral etiology (8.1 vs 9.5 months, $P = 0.034$). Subgroup analyses in PS-matched cohorts demonstrated viral etiology remained associated with a higher all-cause mortality risk in patients with compensated cirrhosis (HR 2.1; 95% CI 1.3-3.4; $P = 0.003$) and male patients (HR 1.3; 95% CI 1.0-1.8; $P = 0.049$), but not in non-cirrhotic or female patients. Notably, among patients with T2D, viral-related HCC had significantly poorer OS than non-viral HCC (mOS 8.7 vs 9.7 months, $P = 0.049$), with no such significant difference in those without T2D (mOS 7.5 vs 8.5 months, $P = 0.39$).

Conclusions: Among lenvatinib-treated HCC patients, viral etiology was associated with significantly poorer OS than non-viral etiologies, with this association being particularly pronounced in individuals with compensated cirrhosis, or those with T2D. The presence of a non-viral liver disease etiology may aid clinical decision-making of the suitability of tyrosine kinase

therapy for advanced HCC.

Keyword(s) : hepatocellular carcinoma; tyrosine kinase inhibitor; etiologies; survival outcome

1494 - Distinct Clinical Profile of Wilson Disease Related Cirrhosis Insights from a Propensity Score Matched Multicenter Study

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¹

Background / Hypothesis:

Wilson disease (WD) is a rare inherited metabolic disorder in which cirrhosis represents a common manifestation and a critical determinant of prognosis. This study aimed to identify risk factors associated with WD-related cirrhosis and delineate complication profile.

Materials and Methods: This retrospective study included patients with WD treated at three large general hospitals between January 2008 and December 2024. To compare the clinical characteristics and complication profiles across different cirrhosis etiologies, propensity score matching (PSM) was applied to evaluate differences between WD cirrhosis and cirrhosis secondary to hepatitis B virus (HBV), autoimmune hepatitis, and alcohol.

Results:

The study enrolled 1,227 participants, comprising 201 patients with WD. Patients with WD demonstrated a significantly higher incidence of cirrhosis and elevated non-invasive fibrosis scores compared to other liver diseases. Multivariate logistic regression identified platelet count as a protective factor for WD related cirrhosis (OR = 0.99, 95% CI: 0.97-0.99, $P = 0.018$). A predictive model incorporating variables such as age and PLT demonstrated good discriminatory performance for identifying WD cirrhosis (AUROC = 0.89, 95% CI: 0.83-0.95). Compared to HBV, autoimmune, and alcohol related cirrhosis, WD cirrhosis was associated with a lower incidence of esophagogastric variceal bleeding (EVB) and hepatocellular carcinoma (HCC), but a higher incidence of liver failure and hepatic encephalopathy.

Conclusions:

Although patients with WD exhibit higher liver fibrosis scores, they demonstrate lower risks of EVB and HCC in

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cirrhosis. Exploring the potential mechanisms is crucial for optimizing follow-up in WD and managing progression in common cirrhosis.

Keyword(s) : Wilson disease; Cirrhosis, Hepatitis B virus, Complications;

1497 - A Phase I Clinical Trial Evaluating the Safety and Efficacy of the QH 1A Combination Bio artificial Liver Support System Incorporating Ex vivo Mesenchymal Stem Cells for the Treatment of ACLF patients

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) has a high mortality rate, and artificial liver support represents an important therapeutic approach. Compared with mechanical artificial liver systems, bio-artificial liver (BAL) support more closely resembles native hepatic function. This study evaluated the safety and preliminary efficacy of the QH-1A combination bio-artificial liver support system incorporating ex-vivo mesenchymal stem cells (MSCs) in ACLF patients.

Materials and Methods: This single-center, open-label Phase I clinical trial (approved by the National Medical Products Administration of China) assessed the safety and preliminary efficacy of QH-1A. Patient plasma was separated using a plasma separator and perfused through an MSC-seeded hollow-fiber cartridge, allowing MSC-derived extracellular vesicles (including exosomes) and other bioactive factors to return to the patient. Participants were enrolled sequentially into escalating duration cohorts of 4 hours (n=3), 6 hours (n=3), and 8 hours (n=3). Follow-up visits were conducted immediately after treatment and on days 3, 7, and 28. Escalation decisions were made by an independent Safety Evaluation Committee based on 28-day safety observations.

Results: From May 2025 to October 2025, nine patients with ACLF were enrolled at the Third Affiliated Hospital of Sun Yat-sen University. Eight cases were HBV-related and one was due to alcoholic liver disease. Cirrhosis was present in 77.8% (7/9) of patients. All participants completed treatment. The 28-day transplant-free survival rate following the first treatment was 88.89%; one patient underwent liver transplantation on day 26 due to lack of clinical improvement. A total of 25 adverse

events occurred during treatment; only one serious adverse event (SAE) was reported and was assessed as unrelated to the study treatment. No events led to treatment discontinuation. Improvements were observed in total bilirubin (TBIL), international normalized ratio (INR), and COSSH-ACLF II score after QH-1A treatment (Figure 1).

Conclusions: The QH-1A bio-artificial liver support system demonstrated favorable safety and tolerability in patients with ACLF and was associated with improvements in liver function, coagulation parameters, and overall clinical severity scores. These findings suggest that QH-1A may represent a promising therapeutic option for the management of ACLF. Further research is needed to validate its safety and efficacy.

Keyword(s) :

1507 - Hyperammonemia in pre hepatic and intrahepatic portal hypertension comparable levels different clinical outcomes

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Background / Hypothesis: The role of hyperammonemia as a key factor in the development of hepatic encephalopathy (HE) is well established in patients with intrahepatic portal hypertension (PH). However, this association remains poorly characterized in patients with pre-hepatic PH. Aim: To compare the frequency and severity of hyperammonemia and its association with the development of HE in patients with different types of PH.

Materials and Methods: A cross-sectional study enrolled 69 participants: 25 patients with non-cirrhotic, non-malignant portal vein thrombosis (PVT group), 29 patients with liver cirrhosis (LC group), and 15 patients with a combination of cirrhosis and PVT (LC-PVT group). PVT was confirmed by contrast-enhanced computed tomography. According to transient elastography, liver fibrosis stages were F0-F2 in the PVT group and F4 in all patients with cirrhosis. The clinical diagnosis of overt HE

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was based on the West Haven criteria, whereas minimal HE (MHE) was assessed using psychometric tests. Ammonia concentration was measured in capillary blood using the PocketChem BA PA-4140 analyzer (ARKRAY Factory, Inc., Japan). Results: The frequency of HE was approximately 66% in the cirrhosis groups (LC and LC-PVT), compared with 24% in the PVT group ($p = 0.004$). Furthermore, the cirrhosis groups exhibited both overt HE (34%) and MHE (32%), whereas only MHE was identified in the PVT group. Hyperammonemia was observed in 17 (68%) patients in the PVT group, 21 (72%) patients in the LC group, and 14 (93%) patients in the LC-PVT group ($p = 0.176$). The severity of hyperammonemia did not differ significantly between the groups: the median ammonia concentration was 83 (47–118) $\mu\text{mol/L}$ in the PVT group, 95 (51–139) $\mu\text{mol/L}$ in the LC group, and 102 (63–154) $\mu\text{mol/L}$ in the LC-PVT group ($p = 0.217$). The severity of PH was similar across all groups except for moderate-to-severe ascites, which was detected only in the groups with LC. No significant differences were identified in the prevalence ($p = 0.376$) or grade of esophageal or gastric varices ($p = 0.929$), nor in the history of variceal bleeding ($p = 0.773$). In addition, no statistically significant difference was observed in the prevalence of large (≥ 10 mm in diameter) spontaneous and/or surgically created portosystemic shunts ($p = 0.424$). Conclusions: The frequency and severity of hyperammonemia were comparable across different types of PH. However, patients with pre-hepatic PH exhibited a lower degree of HE, possibly reflecting reduced cerebral susceptibility to the neurotoxic effects of ammonia, which warrants further investigation.

Keyword(s) : liver cirrhosis, portal vein thrombosis, portosystemic shunts, hepatic encephalopathy, minimal hepatic encephalopathy

1516 - Serum HBP as a Mechanistic Predictor of Primary Resistance to Atezolizumab plus Bevacizumab in HCC.

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Background:

Atezolizumab plus bevacizumab (Atezo+Bev) has become a standard first-line therapy for advanced hepatocellular carcinoma (HCC), yet no validated biomarkers can reliably predict therapeutic efficacy before treatment initiation. This study aimed to identify mechanisms of primary resistance to Atezo+Bev and to establish serum biomarkers with immediate clinical applicability.

Methods:

Spatial transcriptomics (VISIUM and VISIUM HD) and single-cell transcriptomics (Xenium 5K) were performed on eight HCC biopsy or surgical specimens to identify molecular features associated with progressive disease (PD). Treatment responses were classified as responders (CR/PR/SD) or non-responders (PD). Proteins identified in tissue analyses were subsequently evaluated in pretreatment serum samples from a training cohort of Atezo+Bev-treated patients and validated in an independent cohort, totaling 99 patients. Results:

Spatial transcriptomics revealed uniformly high intratumoral expression of heparin-binding protein (HBP) exclusively in the non-responder group. Importantly, pretreatment biopsies displayed the same HBP-high signature, indicating that resistance is pre-existing rather than therapy-induced. Xenium 5K analysis demonstrated dense interaction networks between HBP-expressing HCC cells and M2-polarized macrophages, while VISIUM HD identified ITGA-dependent ligand-receptor signaling as the dominant communication axis. In vitro, recombinant HBP significantly induced CD163 and CD204 expression in human macrophages ($p = 0.023$, $p = 0.003$), whereas ITGA knockdown abrogated this effect ($p = 0.005$, $p = 0.014$). Clinically, patients with high pretreatment serum HBP exhibited significantly shorter PFS based on both median stratification ($p = 0.026$) and ROC-derived cutoff ($p = 0.029$). These findings were independently validated in a separate cohort ($p = 0.050$).

Conclusion:

Pretreatment serum HBP establishes an ITGA-mediated immunosuppressive circuit that confers primary resistance to Atezo+Bev and is a robust, mechanism-based biomarker for predicting non-response before treatment initiation. HBP has immediate translational potential for precision immunotherapy and may guide early treatment selection or intensification strategies in HCC.

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Keyword(s) : Hepatocellular carcinoma, Biomarkers, Atezolizumab plus bevacizumab, Immune checkpoint inhibitors, Spatial transcriptomics analysis

1517 - Hepatitis B serology and vaccine response in uveitis patients treated with adalimumab effects of the 1998 national hepatitis B vaccination program in Turkey

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Background / Hypothesis:

Adalimumab is an effective biologic agent for noninfectious uveitis but carries a risk of hepatitis B virus (HBV) reactivation under immunosuppression. In Turkey, hepatitis B vaccination was incorporated into the national childhood immunization program in 1998, and this cohort effect is likely to influence HBV seroepidemiology in adults. In this study, hepatitis B serologic profiles and hepatitis B vaccine responses in uveitis patients treated with adalimumab were evaluated, and the impact of the 1998 vaccination program was examined.

Materials and Methods:

This single-center retrospective cohort included patients aged ≥ 18 years who received anti-TNF therapy for uveitis between November 2021 and August 2025. Adalimumab was the only biologic agent. HBsAg, anti-HBs and total anti-HBc IgG results of the patients were recorded from medical records. Patients were grouped by birth year as born before 1998 or in/after 1998, reflecting inclusion in the vaccination program. Anti-HBs-negative patients were scheduled to receive a 0-1-6 HBV vaccination regimen; post-vaccination anti-HBs ≥ 10 mIU/mL was considered a serologic response. Patients with HBsAg or anti-HBc positivity received tenofovir or entecavir prophylaxis according to national guidelines.

Results:

A total of 197 patients (mean age 40.5 ± 14.3 years; 55.8% male) were included; four were HBsAg positive. Among the remaining 193 HBsAg-negative patients, 49.7% were anti-HBs positive and 20.7% were anti-HBc positive. No anti-HBc positivity was detected in patients born in or after 1998, whereas 26.7% of those born before 1998 were anti-HBc positive ($p < 0.001$). Pre-

treatment anti-HBs positivity was 86.0% in patients born in or after 1998 and 39.3% in those born before 1998 ($p < 0.001$). Among anti-HBs-negative patients who completed a three-dose vaccination schedule, approximately half developed seroconversion.

Conclusions:

In uveitis patients treated with adalimumab, adults who were vaccinated against hepatitis B under the 1998 program exhibit a vaccine-based, low-risk serologic profile, whereas older patients frequently lack immunity or show evidence of past infection (anti-HBc positivity), which carries a risk of HBV reactivation. Planned hepatitis B vaccination is often not completed before anti-TNF initiation, and vaccination started during biologic therapy may elicit a suboptimal immune response. Ensuring that candidates for anti-TNF therapy are immune to hepatitis B and do not have evidence of past infection before immunosuppression may reduce HBV reactivation and prevent poor vaccine responses in this vulnerable population.

Keyword(s) : Hepatitis B virus, Uveitis, Adalimumab, Tumor necrosis factor inhibitors, Vaccination, Hepatitis B surface antibody

1525 - A Multi Sectoral Co Payment Model to Reduce Hepatitis C Treatment Costs and Advance Elimination Among Low Income Populations Experience from Yangjiang China

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Background / Hypothesis:

Although highly effective direct-acting antivirals (DAAs) are widely available in China, financial barriers remain a major obstacle to hepatitis C elimination among low-income populations. To address this challenge, Yangjiang City launched a pilot project that integrates government insurance, philanthropic assistance, and corporate support to provide affordable antiviral therapy for economically disadvantaged patients.

Materials and Methods:

Patients who met Yangjiang's criteria for "low-income households" (annual household income $< \text{RMB } 47,000$) or were classified by the civil affairs department as expenditure-driven impoverished households were eligible. Each patient received a subsidy of RMB 1,875, which, together with standard medical insurance reimbursement, substantially reduced out-of-pocket costs. Depending on insurance category and whether

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care was delivered at a secondary or tertiary hospital, insured residents paid between RMB 0 and 2,221 for a 12-week course of sofosbuvir/velpatasvir. The program was led by the Chinese Association of STD and AIDS Prevention and Control, supervised by the Yangjiang CDC, and jointly implemented with local hospitals and pharmaceutical partners.

Results:

Since the establishment of the national case reporting system in 2004, Guangdong Province has consistently recorded hepatitis C incidence rates above the national average. Yangjiang, located on the southwestern coast of Guangdong, reported 14,738 cumulative hepatitis C cases from 2004 to 2022. Previous recall efforts performed poorly, with an HCV RNA testing rate of approximately 20% and a treatment rate of about 30%, falling far short of WHO elimination targets; economic hardship appeared to be the main contributing factor. The co-payment program began on July 1, 2025. Here we presented the first phase data. As of October 31, 2025, 32 patients met eligibility criteria, most of the patients were farmers and had low income, all 32 initiated treatment, and 31 had completed therapy, pre-treatment genotype testing is not necessary. None had yet reached the post-treatment Week-4 or Week-12 follow-up time points. All enrolled patients started therapy on the same day they were approved for financial support which was prompt, treatment rate reached 100% in this pilot. Baseline characteristics are summarized in Table 1.

Conclusions:

The multi-sectoral co-payment model in Yangjiang effectively reduced financial barriers and enabled rapid treatment initiation once subsidies were granted. This approach improved antiviral treatment coverage and accessibility, embodied a patient-centered strategy, and accelerated progress toward the city's 2030 HCV elimination goal. The experience provides a scalable and replicable pathway for hepatitis C elimination in resource-limited settings, offering insights not only for China but also for other regions.

Keyword(s) : Multi-Sectoral Co-Payment Model, hepatitis C elimination

1526 - Transcriptional Activity of cccDNA in HBsAg Negative Individuals with a History of Hepatitis B Infection

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Background:

A significant proportion of hepatocellular carcinoma (HCC) cases occur in individuals who are hepatitis B surface antigen (HBsAg)-negative. These patients, often perceived as having resolved infection, remain at risk for HBV reactivation during immunosuppression, highlighting the persistence of a viral reservoir. This study aimed to quantify the covalently closed circular DNA (cccDNA) reservoir and investigate its transcriptional activity in the livers of HBsAg-negative individuals to elucidate its link to HCC pathogenesis.

Materials and Methods:

We collected liver tissue and serum samples from 68 HBsAg-negative, anti-HBc-positive patients, categorized into three groups: the HBV-related HCC group (HBV-HCC, n=34), the Treated group (post-antiviral therapy, n=15), and the Natural Clearance control group (n=19). A group of 38 HBsAg-positive patients (<100 IU/mL) with HCC (n=22) or under therapy (n=16) was also enrolled (Table1). For HCC patients, paired samples from both the tumor and the peritumoral tissue were collected. Intrahepatic cccDNA and total HBV DNA (tDNA) levels were precisely quantified using Droplet Digital PCR. Serum pregenomic RNA (pgRNA) was measured to assess cccDNA transcriptional activity.

Results:

- 1.Serum pgRNA was undetectable in all 68 HBsAg-negative patients. In the HBV-HCC group, peritumoral tissues harbored significantly higher cccDNA levels than tumor tissues(Figure1).
- 2.A clear gradient of intrahepatic viral reservoir size was observed: HBsAg-positive > HBsAg-negative (HCC/Treated) > Natural Clearance group. This gradient was consistent for both cccDNA and tDNA in peritumoral(Figure2) and Treated groups(Figure3). The tDNA/cccDNA ratio showed no significant differences among most groups but was higher in the HBsAg-positive and negative Treated groups compared to the Natural Clearance group.
- 3.Notably, among HBsAg-negative individuals, no significant differences in cccDNA, tDNA, or their ratio were found between the HBV-HCC and Treated groups(Figure4).

Conclusions:

Our study demonstrates that a substantial cccDNA reservoir persists in the liver of HBsAg-negative individuals, with the highest levels found in peritumoral

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tissue. The viral load correlates with clinical status, indicating that HBsAg seroclearance does not equate to viral eradication. The undetectable pgRNA and stable tDNA/cccDNA ratio suggest the reservoir is largely transcriptionally silent. The lack of viral load difference between HBsAg-negative HBV-HCC and Treated groups implies that hepatocarcinogenesis may be driven more by past viral integration or indirect oncogenic effects rather than ongoing viral replication. These findings underscore the risk associated with this residual reservoir and question the sensitivity of pgRNA as a sole marker for its transcriptional activity.

Keyword(s) : Hepatitis B virus; Covalently closed circular DNA; HBsAg-negative; Hepatocellular carcinoma; Viral reservoir; Droplet Digital PCR

1527 - Development and Validation of an Early Diagnostic Model to Distinguish Bacterial Infection in Patients with Acute on Chronic Liver Disease (AoCLD) and Systemic Inflammatory Response Syndrome (SIRS)

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Background / Hypothesis: Early differentiation between infectious and non-infectious systemic inflammatory response syndrome (SIRS) in patients with acute-on-chronic liver disease (AoCLD) remains challenging. This study aimed to develop and validate an early diagnostic model to accurately identify infection status in AoCLD patients with SIRS, thereby guiding targeted anti-infective therapy and improving clinical management.

Materials and Methods: Based on two multicenter prospective cohorts from the Chinese Acute-on-Chronic Liver Failure (CATCH-LIFE) study, 515 AoCLD patients with SIRS were randomly divided into a training cohort (n=361) and a validation cohort (n=154). In the training cohort, predictive factors for infection were screened using logistic regression, least absolute shrinkage and selection operator (LASSO) regression, multicollinearity analysis, and stepwise regression. Restricted cubic splines (RCS) were employed to explore nonlinear relationships and interaction effects. A nomogram model was subsequently constructed. The model's performance was evaluated using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA), with internal validation via bootstrap method.

Results: The final model incorporated five clinical indicators: jaundice, ascites, neutrophil count (N), albumin (ALB), and C-reactive protein (CRP). CRP exhibited a significant nonlinear relationship with infection risk ($P < 0.001$) and was included as a spline term, while an interaction term between ALB and jaundice was incorporated. The model demonstrated excellent discrimination, with area under the ROC curve (AUC) of 0.840 (95% CI: 0.788-0.888) in the training cohort and 0.865 (95% CI: 0.803-0.927) in the validation cohort, significantly outperforming single biomarkers. Calibration curves showed high consistency between predicted and observed probabilities. DCA indicated substantial clinical net benefit across threshold probabilities. A clinical decision pathway was developed based on the nomogram's risk prediction and optimal DCA threshold.

Conclusions: The early diagnostic model developed in this study effectively differentiates between infectious and non-infectious SIRS in AoCLD patients, potentially reducing unnecessary antimicrobial exposure and improving clinical outcomes.

Keyword(s) : Acute-on-Chronic Liver Disease (AoCLD), Systemic Inflammatory Response Syndrome (SIRS), Infection, Early Diagnosis, Risk Factors

1528 - Palliative care reduces disease time burden but is under utilized in terminal stage hepatocellular carcinoma

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Background / Hypothesis: Guidelines recommend palliative care (PC) in hepatocellular carcinoma (HCC) patients, especially at the terminal stage. However, the PC utilization patterns and its impact on disease time burden have not been studied.



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Materials and **Methods:** HCC patients diagnosed at the terminal stage (never received any anti-cancer therapy) and who have died between 2003 – 2024 were identified from a territory-wide population-based cohort in Hong Kong. PC utilization was defined as patients who received any specialist PC consultation or inpatient hospice care within the 3 months before death. PC utilization patterns in different time periods were reported. Disease time burden was analyzed by percentage of days at home (%DAH) – defined as percentage of days not requiring healthcare utilization (inpatient stays, emergency department attendance, day procedures, blood tests, and imaging) in the last 3 months of life.

Results:

This study included 12556 patients with HCC at the terminal stage (mean age 68.8 years, 76.5% male, 61.7% with viral hepatitis, 46.2% with cirrhosis, mean MELD score 10.8, mean AFP 42816.1 ng/mL) with an average survival of 142.1 (95% CI 135.4 – 148.9) days after HCC diagnosis. Only 18.0% of the cohort (2254 patients) received PC in the 3 months preceding death. The PC contact rate has remained similar in different time periods (17.9% in 2003 – 2008, 19.0% in 2009 – 2014, 17.5% in 2015 – 2019, 16.1% in 2020 – 2024; all $p > 0.05$). The time interval between PC contact and death also remained static (average 20.6 days in 2003 – 2008, 22.5 days in 2009 – 2014, 22.4 days in 2015 – 2019, 19.3 days in 2020 – 2024; all $p > 0.05$). PC contact led to significantly higher %DAH (45.0% in PC group vs 40.6% in non-PC group; $p < 0.001$), and this difference was mainly driven by reduced time burden on inpatient stay (47.1% in PC group vs 51.7% in non-PC group; $p < 0.001$) and emergency department attendance (0.8% in PC group vs 1.0% in non-PC group; $p < 0.001$). The impact of PC on disease time burden remained robust in propensity-score matched and multivariate analyses, respectively.

Conclusions:

PC significantly reduced disease time burden in terminal HCC in this real-world cohort. However, PC has remained under-utilized in the past 2 decades. Proactive and earlier PC referral may improve quality of life in terminal HCC.

Keyword(s) : Hepatocellular carcinoma, Palliative care, Disease time burden

1529 - CD39 Promotes Angiogenesis and Poor Prognosis in Hepatocellular Carcinoma via Activation of the PI3K AKT Signaling Pathway

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Background / **Hypothesis:** Hepatocellular carcinoma (HCC) is one of the most common and aggressive malignancies worldwide, characterized by rapid progression, a highly angiogenic phenotype, and a high risk of metastasis and recurrence. Accumulating evidence implicates CD39 in the development of multiple cancers through diverse mechanisms. However, the specific role of tumor cell-derived CD39 in HCC progression, particularly in the regulation of angiogenesis and the underlying molecular pathways, remains poorly defined.

Materials and **Methods:** Expression of CD39, CD31, VEGFA and pan-cytokeratin (PANCK) in HCC tissues was evaluated by immunohistochemistry and multiplex immunofluorescence, and the prognostic significance of CD39 expression was assessed using Cox regression analyses. VEGFA expression and secretion in HCC cell lines were quantified by Western blotting, real-time quantitative PCR and ELISA. The effects of tumor cell-conditioned medium on human umbilical vein endothelial cell (HUVEC) proliferation were determined by CCK-8 assays, whereas Transwell migration and invasion assays and tube-formation assays were used to evaluate HUVEC motility and in vitro angiogenic capacity. Phosphorylation of PI3K, AKT and mTOR was examined by Western blotting. A subcutaneous xenograft model in nude mice was established to investigate the impact of CD39 on HCC angiogenesis in vivo.

Results:

CD39 was upregulated in HCC tissues, and high CD39 expression was associated with a shorter 1-year progression-free survival. Public database analyses further revealed a close association between CD39 expression and angiogenesis-related gene signatures. Functionally, conditioned medium from CD39-overexpressing HCC cells enhanced HUVEC proliferation, migration, invasion and tube formation. Mechanistic studies showed that CD39 promoted VEGFA synthesis and secretion by HCC cells, which was corroborated in clinical samples in which CD39 expression in tumor cells positively correlated with CD31 and VEGFA levels. Upregulation of CD39 was accompanied by increased phosphorylation of PI3K, AKT

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and mTOR, together with augmented production and release of VEGFA by HCC cells, indicating that CD39 may drive HCC angiogenesis through activation of the PI3K/AKT/mTOR pathway and subsequent VEGFA upregulation. Consistently, in a nude mouse subcutaneous xenograft model, CD39 expression significantly promoted intratumoral angiogenesis in vivo.

Conclusions:

Our study identifies CD39 as an independent predictor of poor prognosis in patients with hepatocellular carcinoma. We delineate a pivotal role for CD39 in driving HCC angiogenesis and demonstrate that CD39 activates the PI3K/AKT signaling pathway to enhance VEGFA production and secretion by tumor cells, thereby promoting angiogenesis both in vitro and in vivo. These findings highlight CD39 as a promising therapeutic target in HCC, particularly in the context of anti-angiogenic strategies.

Keyword(s) : Hepatocellular carcinoma, CD39, Angiogenesis, Prognosis, PI3K/AKT signaling pathway

1531 - Dynamic changes in gut microbiota associated with HBsAg decline in chronic hepatitis B patients undergoing IFN treatment

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Background / Hypothesis:
Recent studies have demonstrated a close interaction between chronic hepatitis B (CHB) and gut microbiome dysbiosis as well as the effects of antiviral treatment with Nucleos(t)ide analogues (NAs) on gut microbiota dysbiosis. However, the impact of interferon (IFN) treatment on gut dysbiosis remains to be elucidated.

Materials and Methods:
Treatment-naïve CHB patients were prospectively recruited and received PEG-IFN- α 2b (n=26) or NAs (n=50) treatment, with 34 healthy individuals (HC) as controls. All subjects were age-gender matched. Fecal samples were collected at serial treatment time points for metagenomic sequencing.

Results:

Chronic HBV infection is associated with gut microbiota dysbiosis compared to HC, and IFN treatment

significantly impacts gut microbial α - and β -diversity metrics more than NAs treatment. Two distinct enterotypes (C1-Prev/ C2-Bifi) were identified among CHB patients. During IFN treatment, enterotypes changed with HBsAg response (decline >50%). Non-responders shifted from C1-Prev to C2-Bifi, while responders mostly kept the C1-Prev enterotype. Trajectory analysis revealed 81 taxa with opposite temporal trends and 124 taxa with concordant trends between the two treatments. Furthermore, a significantly higher number of differentially abundant bacterial species were observed between IFN responders and non-responders in comparison to the nucleoside analog group (277 taxa vs 76 taxa). Multiple short-chain fatty acid (SCFA)-producing genera decreased in IFN non-responders while remaining stable or increasing in responders.

Conclusions: The study indicates that IFN treatment significantly disrupts the gut microbiota of CHB patients, and SCFA correlate with HBsAg response to IFN treatment.

Keyword(s) : chronic hepatitis B; gut microbiota; antiviral treatment; interferon

1533 - Sex differences in cirrhosis among untreated chronic hepatitis B a 10 year longitudinal cohort study from China

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Background / Hypothesis:

Current guidelines for antiviral treatment of chronic hepatitis B (CHB) are based on age, alanine aminotransferase (ALT) levels, viral load, a family history of hepatitis B virus (HBV)-related cirrhosis or hepatocellular carcinoma, while not considering sex factor.

Materials and Methods:

This retrospective longitudinal cohort study included 1,543 untreated CHB patients (760 males and 774 females) not meeting antiviral treatment criteria in previous guidelines and investigated sex differences in liver disease progression according to the latest Chinese guidelines for the prevention and treatment of CHB (2022).

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Results:

The cumulative incidence of cirrhosis was significantly higher in males compared with females among all untreated CHB patients (23.1% vs 7.9%, $p < 0.001$), treatment-eligible patients (25.3% vs 8.0%, $p = 0.001$), and treatment-ineligible patients (20.6% vs 7.3%, $p < 0.001$). Sex male was strongest risk factor of cirrhosis (HR, 2.474; 95% CI, 1.628-3.760; $p < 0.001$) among treatment-naïve CHB patients. Males showed a relatively high incidence of cirrhosis although not meeting antiviral treatment criteria, while females showed a relative low incidence of cirrhosis although meeting antiviral treatment criteria. In treatment-eligible females, those with abnormal ALT (10.4%) showed a numerically higher incidence of cirrhosis compared with those with normal ALT. In treatment-ineligible patients, male sex and HBeAg-positivity were risk factors of cirrhosis.

Conclusions:

In conclusion, males showed a higher risk of cirrhosis than females in treatment-naïve CHB patients. Expanding antiviral treatment criteria may be considered in male patients, but not in female patients. Initiating antiviral treatment may be stratified by sex and HBeAg status.

Keyword(s) : sex differences; cirrhosis; chronic hepatitis B; treatment-naïve; antiviral treatment criteria.

1542 - Mechanistic Insights into the Protective Effects of Faecalibacterium prausnitzii and Its Metabolite Short Chain Fatty Acids on Biliary Injury in Primary Biliary Cholangitis

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Background / Hypothesis: Primary biliary cholangitis (PBC) is a chronic cholestatic liver disease that primarily affects middle-aged women. Studies suggest that alterations in the gut microbiome are closely associated with the pathogenesis and treatment of PBC, but the underlying mechanisms remain unclear.

Materials and Methods: This study first conducted gut microbiota analysis of fecal samples from PBC patients and healthy controls using metagenomic sequencing, with 16S and other

methods employed to identify differential microbial taxa. Subsequently, a bile duct ligation (BDL) mouse model was established and treated with *F. prausnitzii* (1×10^9 CFU/day), while ductular reaction and liver fibrosis were evaluated through histopathological staining. For in vitro experiments, a GDCD-induced injury model using human primary biliary epithelial cells (HiBECs) was developed, followed by treatment with varying concentrations of short-chain fatty acids (SCFAs). Furthermore, ChIP assays were performed to detect H3K27ac modification levels, elucidating the specific mechanism by which SCFAs exert their effects through epigenetic regulation of the ELF3/PI3K-Akt pathway. All data were statistically analyzed using two-tailed t-tests/ANOVA with FDR correction ($P < 0.05$ considered statistically significant).

Results:

We found that *F. prausnitzii* was significantly reduced in the gut microbiota of PBC patients. Supplementation with *F. prausnitzii* markedly improved BDL-induced biliary injury, including cholangitis, bile duct hyperplasia, and liver fibrosis, while also restoring gut microbial balance and intestinal barrier function. These beneficial effects were linked to short-chain fatty acids (SCFAs) produced by *F. prausnitzii*. Mechanistic studies revealed that *F. prausnitzii*-derived SCFAs enhanced histone H3K27 acetylation, upregulating the transcription factor ELF3 and subsequently activating the PI3K-Akt signaling pathway to promote bile duct cell repair.

Conclusions:

This study demonstrates that *F. prausnitzii* exerts protective effects in PBC. Its depletion in PBC patients and its therapeutic potential in alleviating cholangitis and liver fibrosis, along with restoring gut function, highlight its promising clinical application for PBC treatment.

Keyword(s) : Metagenomics, Primary Biliary Cholangitis, Faecalibacterium prausnitzii, Biliary Response

1551 - Non Invasive Detection of Esophageal Varices in Compensated Cirrhotic Patients

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Background / Hypothesis: Varices are present in 30-40 % of patients with compensated cirrhosis (Child-Pugh class A). Although screening endoscopy for esophageal varices (O.V.) is recommended to all patients with cirrhosis, this recommendation is not a result of

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evidence-based data. We studied the association of (platelet count/spleen diameter ratio, insulin resistance and splenoportalindex) and the presence of O.V. in patients with compensated cirrhosis.

Materials and Methods: 124 patients with compensated liver cirrhosis due to chronic hepatitis C virus were studied. After clinical, laboratory, ultrasound examinations, all patients underwent screening endoscopy and O.V. were reported as present or absent. According to presence or absence of varices; two groups were described: group I without varices and group II with varices.

Results: Among 124 patients with mean age of (51.81 ± 12.94) , 2 groups were described: group I (30 patients) and group II (94 patients) with a male majority (20 patients in group I and 66 patients in group II). In group I and group II: the mean platelet count/spleen diameter ratio was $(1022.6 \pm 73.36, 608.76 \pm 58.44)$ respectively, the mean insulin resistance value was $(2.426 \pm 0.618, 3.081 \pm 0.474)$ respectively. The mean splenoportalindex (SPI) value was $(2.878 \pm 0.870, 6.349 \pm 0.514)$ respectively. For platelet count/spleen diameter ratio and SPI, the sensitivity was (95.7%), specificity (83.3%) and accuracy (92.7%). For platelet count/spleen diameter ratio and insulin resistance (IR), the sensitivity was (70.2%), specificity (86.7%) and accuracy (74.1%). For IR and SPI, the sensitivity was (70.2%), specificity (76.7%) and accuracy (71.7%). For the three predictors combined, the sensitivity was (78.7%), specificity (82.2%) and accuracy (79.5%).

Conclusions: Low platelet count/spleen ratio and high SPI are very useful non invasive predictors for the presence of O.V. that could be used either separately or combined to decrease the number of upper GIT endoscopies needed in cirrhotic patients management. However, IR as a non invasive predictor is still in need for further evaluation.

Keyword(s) : oesophageal varices; Liver; Cirrhosis, Non-invasive

1552 - Multi Omics Integration Identifies a Prognostic Signature Guiding Precision Survival in Advanced HCC

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Background / Hypothesis: Hepatocellular carcinoma (HCC) remains a leading cause of cancer-related mortality globally, with over 70% of patients diagnosed at intermediate or advanced stages (BCLC B/C) where curative treatments are no longer feasible. Current prognostic tools are inadequate to capture the heterogeneity of outcomes in this population, underscoring the critical need for integrated predictive models that incorporate multimodal data.

Materials and Methods: Plasma samples were collected from 242 patients with advanced HCC (BCLC B–D) who received first-line systemic therapy at Beijing Ditan Hospital, Capital Medical University, from January 2020 and December 2023. Plasma proteomic profiling was performed using the DB4000 Deep Blood DIA Proteome platform with data-independent acquisition and Astral mass spectrometry. Proteomic differences between survivors and non-survivors were assessed via principal component and differential abundance analyses. Candidate biomarkers were validated by ELISA, and their cellular origins and prognostic relevance were explored using single-cell RNA sequencing of HCC tissues.

Results:

Among 154 HBV-HCC patients with 1-year follow-up, 47 (30.5%) died. Using mRMR and LASSO regression, we identified 15 proteins associated with 1-year mortality. Multivariate logistic regression further refined a model incorporating four proteins (EGFLAM, HTRA3, ADGRG2, AGXT) and two clinical variables (K+, GGT). The model demonstrated strong predictive performance (AUC: 0.939, accuracy: 0.841, $p < 0.05$) and favorable clinical utility on decision curve analysis. All biomarkers except AGXT were significantly upregulated in deceased patients (FDR-adjusted $p < 0.05$), and ELISA confirmed these trends. The model effectively stratified mortality risk across treatment subgroups (conservative vs. ICI-based). Single-cell transcriptomics revealed predominant expression of these proteins within the tumor microenvironment.

Conclusions:

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This study comprehensively analyzed the plasma proteomic landscape of HCC and provided a promising blood based tool for predicting the risk of death in patients with advanced HCC and precision immunotherapy.

Keyword(s) : Hepatocellular carcinoma, plasma proteomic, survival prognosis, machine learning model

1555 - Concordance Between Ultrasound Steatosis Grading and Elastography CAP Classification A Case Series of Chronic HBV Patients on TAF Therapy

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Background / Hypothesis: Tenofovir alafenamide fumarate (TAF) therapy, along with accompanying metabolic factors, may influence hepatic steatosis in chronic hepatitis B (HBV). While ultrasonographic (USG) steatosis grading is widely used in clinical follow-up, it remains an operator-dependent and subjective modality. This study aimed to evaluate the concordance between steatosis grading by USG and by the more specialized, less accessible Controlled Attenuation Parameter (CAP) measurement performed via elastography, both assessed during the same clinical visit.

Materials and Methods: This single-center, consecutive case series included chronic HBV patients aged ≥ 18 years on TAF therapy who presented to the infectious diseases outpatient clinic and had both USG-based steatosis grading (S0–S3) and elastography-CAP results available from the same visit. Patients with significant alcohol use, concomitant chronic liver diseases (HCV/HDV, autoimmune liver disease, Wilson's disease, hemochromatosis), acute hepatitis flares, or incomplete USG/CAP data were excluded. CAP categories were defined as follows: S0 ≤ 0.63 , S1: 0.64–0.72, S2: 0.73–0.82, and S3 ≥ 0.83 dB/m. Discrepancies between USG and CAP classifications were analyzed, and findings were reported in counts and percentages. **Results:** The median age of the 16 included patients was 57.5 years, with 56.2% being female. Steatosis grading by USG revealed 10 patients with S0, 3 with S1, and 3 with S2, while CAP identified 13 patients as S0, 1 as S1, and 2 as S2. Full concordance between the two methods was observed in 12 patients (75.0%), with a one-stage discrepancy in 2 patients (12.5%), and a greater-than-one-stage discrepancy in 2 patients (12.5%). A heatmap (Figure 1) illustrates concordance between USG and CAP classifications, with diagonal cells indicating perfect agreement.

In 3 patients (18.8%), USG classified steatosis at a higher stage than CAP. All USG-S0 cases matched with CAP-S0. USG-S1 cases were distributed across CAP S0–S2, while 67% of USG-S2 cases were classified as CAP-S0, and 33% as CAP-S2. Regarding lipid profiles: LDL ≥ 130 mg/dL was observed in 7 patients (43.8%), LDL ≥ 160 mg/dL in 1 patient (6.3%), and triglycerides ≥ 150 mg/dL in 6 patients (37.5%). **Conclusions:** There was a high concordance rate between USG and CAP assessments, with clinically significant discrepancies limited to a minority. USG demonstrated a slight tendency to overclassify steatosis. Despite the small sample, these findings support the practical use of USG in steatosis monitoring among HBV patients on TAF, though CAP may be useful in borderline cases.

Keyword(s) :

1559 - SIRPA as a Novel Regulator of T Cell Apoptosis in Hepatocellular Carcinoma Implications for Immunotherapy

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Background / Hypothesis: Immune checkpoint inhibitors (ICIs) were effective in only 20% of advanced hepatocellular carcinoma (HCC), suggesting the presence of additional immune evasion mechanisms.

Materials and Methods: In this study, we employed a CRISPR library to screen key genes associated with T-cell apoptosis and identified differentially expressed genes (DEGs) in tumor-infiltrating lymphocytes (TILs) from HCC patients with high concentrations of interleukin-2 (IL-2). Integration analysis revealed signal regulatory protein alpha (SIRPA) as a novel regulator of T cell apoptosis in HCC, distinct from its classical role as a macrophage inhibitory receptor. In addition, the function of SIRPA was verified in vivo and in vitro using Sirp α ^{-/-} mice and T cells derived from PBMC.

Results:

In vitro, SIRPA deficiency reduced apoptosis in human and mouse T cells, while its overexpression promoted apoptosis. In vivo, the degree of tumor suppression strongly correlated with Sirp α deficiency in T cells, with even haploinsufficient Sirp α exerting anti-tumor effects. After macrophage depletion, Sirp α ^{-/-} mice showed

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significantly increased proportions of functional CD8+ T cells in peripheral blood and tumor tissues, along with elevated granzyme B (GZMB) levels compared to wild-type (WT) mice, confirming that Sirp α deficiency in T cells resists apoptosis. Notably, in adoptive immunotherapy, TCR-T cells overexpressing SIRPA exhibited significantly reduced killing function, and tumor-bearing mice receiving Sirp α -/- CD3+ T cells inhibited tumor growth compared to those receiving T cells from WT. Mechanistically, SIRPA upregulated EGR4 to activate the Bcl-2/Bax pathway, promoting T-cell apoptosis.

Conclusions:

Targeting SIRPA may suppress HCC progression by inhibiting T-cell apoptosis, offering a novel immunotherapeutic strategy.

Keyword(s) : signal regulatory protein alpha; T cell apoptosis; hepatocellular carcinoma; Immune checkpoint inhibitors

1560 - Correlation between transient elastography and liver biopsy in CHB patients with elevated HBV DNA

Dr	with normal ALT	normal ALT
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Background / Hypothesis: Chronic hepatitis B (CHB) is a significant global health burden, leading to liver fibrosis and cirrhosis. Many CHB patients with elevated hepatitis B virus (HBV) DNA levels have persistently normal alanine aminotransferase (ALT), complicating fibrosis assessment. Liver biopsy is the gold standard for fibrosis evaluation but has limitations, necessitating non-invasive alternatives like transient elastography (TE).

Materials and Methods: A cross-sectional study was conducted at the Department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Dhaka, from June 2019 to February 2020. Forty CHB patients (HBsAg positive >6 months, ALT <40 IU/L, HBV DNA >2,000 IU/mL) underwent TE and percutaneous liver biopsy. Fibrosis stages were evaluated using the METAVIR scoring system. TE findings were correlated with histological fibrosis using Pearson's correlation test, with statistical analysis performed via SPSS version 23.

Results: The mean age of patients was 30.20 ± 8.3 years, with a male predominance (75%). TE classified 77.5% of patients as having F0-F1 fibrosis and 22.5% as F2 fibrosis. Histological analysis identified 57.5% with F0-F1 fibrosis and 42.5% with significant fibrosis (F2-F4). TE and biopsy findings showed a positive correlation ($p < 0.001$). The receiver-operating characteristic (ROC) curve for TE demonstrated an area under the curve (AUC) of 0.774, with a cut-off value of 5.9 kPa yielding a sensitivity of 70% and specificity of 91% for detecting significant fibrosis.

Conclusions: TE shows a strong correlation with liver biopsy findings in CHB patients with normal ALT, demonstrating its potential as a reliable, non-invasive alternative for fibrosis assessment. Utilizing TE in clinical settings could enhance early detection and management of liver fibrosis, reducing the need for invasive biopsies.

Keyword(s) :

1564 - Second line Therapy after Immunotherapy for Unresectable Hepatocellular Carcinoma a Multicenter Data Registry Analysis

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Background / Hypothesis: Evidence on second-line treatment in systemic treatment for unresectable hepatocellular carcinoma (uHCC) is limited. This study investigated the efficacy of second-line treatment after first-line immunotherapy using clinical data from seven high-volume institutions

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in Tokyo, Japan.

Materials and Methods: Patients who received first-line immunotherapy for uHCC at the participating between August 2018 and June 2025 were extracted for analysis from a multicenter data registry (BLOOM study). Outcomes of patients receiving second-line treatment with or without immune checkpoint inhibitors (ICIs) were compared.

Results:

Of the 188 patients receiving first-line treatment with atezolizumab/bevacizumab (AB, n = 159) or durvalumab/tremelimumab (DT, n = 29), 83 (44%) went on to receive second-line systemic treatment (AB: 66, 41% vs. DT: 17, 58%, p = 0.067). There was no significant difference in median overall survival (OS) or progression-free survival (PFS) between first-line AB and DT.

Excluding seven patients who participated in second-line clinical trials or with insufficient data, 76 patients undergoing second-line treatment after immunotherapy were analyzed. Of these, 13 received ICIs with or without VEGF inhibitors (ICI group), while 63 were treated solely with VEGF inhibitors (non-ICI group). The median age at the start of first-line therapy was 68 years, and 82% were male. Etiologies were hepatitis B/C/non-viral in 8/12/56 patients. Child-Pugh score 5/6/7/8/9: 55/14/5/0/1. BCLC stage A/B/C: 3/28/45. 21% had macrovascular invasion and 36% had extrahepatic spread. There were no significant differences in these characteristics between groups. Objective response to first-line therapy was higher in the ICI group (42% vs. 13%, p = 0.034). Second-line objective response rates (25% vs. 21%, p > 0.99) and disease control rates (58% vs. 68%, p = 0.73) were comparable. There were no significant differences in median first-line OS (not reached vs. 26.0 months, p = 0.23), median second-line OS (not reached vs. 16.9 months, p = 0.26), or second-line PFS (3.7 vs. 4.1 months, p = 0.49) between groups. Patients with favorable response to first-line immunotherapy were more likely to also have favorable response to second-line therapy (57% vs. 15%, p = 0.027) and had longer median second-line PFS than those who did not (19.7 vs. 7.4 months, p = 0.049), regardless of choice of second-line therapy.

Conclusions:

Choice of second-line therapy after first-line immunotherapy did not affect outcomes in this multicenter cohort. Favorable response to first-line therapy was associated with favorable response to second-line therapy and with longer second-line PFS,

suggesting a possible carryover effect.

Keyword(s) : hepatocellular carcinoma, liver cancer, immune checkpoint inhibitors, second-line therapy, systemic therapy

1565 - The Efficacy and Safety of Pegylated Interferon Alpha Treatment in Patients with HBV Related Cirrhosis and HCC after Radical Surgery (1 Year Data from the TERMINATOR Project)

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Background: In China, most HCC is HBV-related with high post-radical surgery recurrence. We explored the application of Peg-IFN- α combined with nucleoside(tide) analogues(NAs) to evaluate the efficacy and safety of Peg-IFN- α in reducing the incidence of HCC in patients with HBV related cirrhosis and reducing the recurrence of HCC in patients after radical surgery. **Methods:** The analysis was conducted in the data of 1 year from a multi-center, prospective, real-world cohort study (the TERMINATOR Project) from China. The cirrhosis group was consisted of compensated HBV related cirrhosis patients, and the HCC group was consisted of patients with HBV-related HCC who underwent radical surgery. Patients were given Peg-IFN- α combined with first-line NAs, including ETV, TDF, TAF,

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or TMF.
Results: As of November 2025, this study enrolled 426 patients with HBV-related cirrhosis and 97 with HCC. 149 cirrhosis and 54 HCC patients completed 24-weeks treatment. 79 and 28 patients completed 48-weeks treatment, respectively. At baseline, 41.08% of cirrhosis and 12.37% of HCC patients had detectable HBV DNA. After 24 weeks of treatment, only 8.05% of the cirrhosis group and 9.26% of the HCC group had detectable HBV DNA. HBsAg clearance was observed in 4.03% of the cirrhosis group and 5.56% of the HCC group. Clinically, one cirrhosis patient (0.67%) developed HCC, and two HCC patients (3.70%) experienced recurrence. At the 48-week visit, virological response was complete, with 100% of patients in both groups achieving undetectable HBV DNA. HBsAg clearance rates were 7.59% in the cirrhosis group and 3.57% in the HCC group. No cirrhosis patients developed new HCC, and one HCC patient (3.57%) had a recurrence. No decompensated cirrhosis occurred. Thrombocytopenia was the main adverse event.
Conclusions: Using Peg-IFN- α combined with NAs treatment, the incidence of HCC was low in patients with HBV related cirrhosis and the recurrence rate was low in patients with HBV related HCC after radical surgery. The patients were safe and the incidence of decompensated cirrhosis seldomly occurs.

Keyword(s) : hepatitis B virus, liver cirrhosis, hepatocellular carcinoma, peg-IFN- α

1567 - Multi omics Analysis of Bile Acid Dysregulation in Hepatitis E virus Infection

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Background / Hypothesis: Hepatitis E virus (HEV) infection can lead to acute hepatitis and potentially progress to acute liver failure, but the underlying mechanisms of bile acid metabolism disruption remain poorly understood. Bile acid homeostasis is crucial for liver function and is regulated by complex interactions between hepatic enzymes, transporters, gut microbiota, and metabolic pathways. This study aimed to comprehensively characterize bile acid metabolism dysregulation in HEV patients through integrated analysis of serum and fecal metabolomics, gut microbiota composition, and hepatic molecular

expression profiles.

Materials and Methods: We enrolled 15 acute hepatitis E (AHE) patients, 16 HEV-related acute liver failure (HEV-ALF) patients, and 15 healthy controls. Targeted bile acid metabolomics was performed on serum and fecal samples, with measurement of serum fibroblast growth factor 19 (FGF19) and 7 α -hydroxy-4-cholesten-3-one (C4) levels. Gut microbiota analysis was conducted using 16S rRNA sequencing of fecal samples. Liver tissue samples were analyzed using HE staining, immunohistochemical staining for key bile acid metabolism molecules, and quantitative PCR analysis of related gene expression.

Results: Patients with HEV infection exhibited elevated serum levels of total bile acids and primary conjugated bile acids, while fecal levels of total bile acids and secondary bile acids were significantly decreased. In patients with acute hepatic failure caused by hepatitis E, serum FGF19 levels were markedly increased and C4 levels were substantially reduced. Intestinal microbiota analysis revealed a declining trend in the abundance of bacterial genera responsible for bile acid metabolism in hepatitis E patients. Hepatic tissue from hepatitis E patients demonstrated pronounced cholestasis, hepatic inflammation, and necrosis. Immunohistochemical analysis of liver tissue showed significantly decreased levels of farnesoid x receptor (FXR) and bile salt export pump (BSEP), accompanied by a marked elevation in FGF19. Gene expression analysis in hepatic tissue revealed decreased mRNA expression of cholesterol 7 α -hydroxylase (CYP7A1) and sterol 12 α -hydroxylase (CYP8B1), which are responsible for bile acid synthesis, as well as reduced mRNA expression of bile acid transporters BSEP and sodium-taurocholate cotransporting polypeptide (NTCP). Furthermore, the mRNA expression of FXR, a key regulator of bile acid homeostasis, was decreased, while FGF19 mRNA expression was increased.

Conclusions: Our findings reveal comprehensive bile acid metabolism disruption in HEV infection, characterized by impaired hepatic bile acid transport, altered gut-liver axis signaling, and gut microbiota dysbiosis. The severity of metabolic dysregulation correlates with disease progression, providing insights into HEV pathogenesis and potential therapeutic targets.

Keyword(s) : Hepatitis E virus, Acute liver failure, Bile acid

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metabolism, Gut microbiota, Gut-liver axis, Farnesoid X receptor, Fibroblast growth factor 19, Cholestasis

1568 - Role of CD146 High Endothelial Cells in Vascular Remodeling and Fibrosis Progression in Cirrhotic Liver A Single Cell Transcriptomic Study

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Background / **Hypothesis:**
During liver cirrhosis, capillarization of liver sinusoidal endothelial cells (LSECs) and abnormal vascular remodeling drive persistent fibrosis, yet the signaling that links endothelial activation to fibroblast activation remains unclear. CD146 levels in serum and liver tissue increase with cirrhosis severity, suggesting a role in fibrogenesis. This study aimed to define the lineage features of CD146-high endothelial cells and their crosstalk with fibroblasts in human cirrhotic liver using single-cell transcriptomic and histological evidence.
Materials and **Methods:**
The public single-cell RNA-sequencing dataset GSE136103, including liver samples from healthy controls and patients with cirrhosis, was analyzed. After quality control, dimensionality reduction and clustering, major cell types were annotated using canonical markers. Endothelial cells were further subclustered to identify LSEC, arterial, venous and lymphatic subsets and to compare MCAM (CD146), STAB2, VCAM1 and other gene expression patterns. Ligand-receptor analysis was used to explore endothelial-fibroblast signaling associated with MCAM-high endothelial cells. In an independent liver cohort, CD146 and VWF co-immunofluorescence was performed to validate endothelial expression.

Results:
Six major cell populations were identified: Kupffer cells, endothelial cells, hepatocytes, NK cells, T cells and fibroblasts. MCAM expression was significantly upregulated in cirrhotic liver and mainly restricted to endothelial cells. In healthy livers, LSECs showed high expression of STAB2 and CLEC4G, whereas cirrhotic LSECs displayed reduced STAB2 and increased MCAM and VCAM1, consistent with a capillarized LSEC profile. Joint STAB2-MCAM analysis showed cirrhosis-derived cells enriched in an STAB2^{low}/MCAM^{high} population, while healthy cells were predominantly STAB2-high/MCAM-low, indicating an association between CD146-related endothelial activation and sinusoidal-to-capillary-like transition. Immunofluorescence confirmed increased

CD146+/VWF+ endothelial cells in cirrhotic versus normal liver. SPP1-ITGA5-ITGB1 and SPP1-ITGAV-ITGB1 axes were enriched between MCAM-high endothelial cells and fibroblasts, suggesting pathological endothelial-fibroblast crosstalk.
Conclusions:

This study delineates CD146-high endothelial cell lineages in human cirrhotic liver and identifies SPP1-integrin-mediated signaling potentially linking LSEC capillarization to fibroblast activation. CD146 may therefore not only mark cirrhosis progression but also promote fibrosis-related vascular remodeling, providing a potential target for anti-fibrotic strategies disrupting endothelial-fibroblast crosstalk.

Keyword(s) :

1573 - Comparison of ALBI PALBI MELD and Child-Pugh Scores in Chronic Liver Disease Evidence from Bangladesh

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Background / **Hypothesis:**

Accurate evaluation of hepatic reserve is vital for prognosis in chronic liver disease (CLD). Conventional models such as the Model for End-Stage Liver Disease (MELD) and Child-Turcotte-Pugh (CTP) scores are widely used but include subjective parameters and laboratory variability. The Albumin-Bilirubin (ALBI) and Platelet-Albumin-Bilirubin (PALBI) scores are objective indices derived from routinely measured biochemical parameters. The efficacy of the ALBI or PALBI scores has not been validated in Bangladeshi adults with liver cirrhosis. This study aimed to determine the correlation of ALBI and PALBI with MELD and CTP scores and to compare their predictive value in Bangladeshi patients with CLD of mixed etiologies.

Materials and **Methods:**
A cross-sectional study was performed on 105 patients with established CLD. Demographic, clinical, and laboratory data were collected to calculate ALBI, PALBI, MELD, and CTP scores. Correlations were analyzed using Spearman's rank test, while one-way ANOVA and chi-square tests evaluated associations with age and etiology. Hierarchical linear regression models were applied to assess the independent predictive contributions of ALBI and PALBI to MELD and CTP.

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Results:

Both ALBI and PALBI showed significant positive correlations with MELD ($p = 0.539$ and 0.625 , $p < 0.001$) and with CTP ($p = 0.721$ and 0.679 , $p < 0.001$). Neither MELD nor CTP varied significantly with age ($p = 0.526$, 0.733), though etiological causes differed by age ($p < 0.001$). Regression analysis identified PALBI as a stronger predictor of MELD ($\Delta R^2 = 0.085$, $p < 0.001$), while ALBI contributed more uniquely to CTP ($\Delta R^2 = 0.054$, $p = 0.001$).

Conclusions:ALBI and PALBI demonstrated strong correlations with conventional liver severity scores, supporting their utility as objective, simple, and economical indices of hepatic dysfunction. PALBI better reflected MELD-related systemic effects, whereas ALBI correlated more closely with CTP, representing hepatic reserve and function.

Keyword(s) : ALBI, PALBI, MELD, Child–Pugh, chronic liver disease, hepatic reserve, Bangladesh

1576 - Qilingxiaoji formula and the effective ingredients inhibit hepatocellular carcinoma progression by inhibiting miR32 GPR180 signaling pathway

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Background / Hypothesis:Hepatocellular carcinoma (HCC) is one of the most prevalent malignant tumors globally. The Qilingxiaoji formula (QLXJ), traditional Chinese medicine, has been used empirically for treating HCC.To investigate the anti-tumor effects of QLXJ and its active components and elucidate the underlying anti-tumor mechanisms.
Materials and Methods:A retrospective study was conducted to evaluate the clinical effects of QLXJ from 2019.01-2023.07. A mouse subcutaneous model and a diethylnitrosamine (DEN) and carbontetrachloride(CCl4)-induced HCC model were built to definite the therapeutic effects of QLXJ. Differential gene expression in tumors was analyzed by RNA-sequencing. The potential active components of QLXJ were identified through UPLC-Q-TOF-MS combined with network pharmacology. Cell viability

and proliferation were assessed using CCK8 and EdU staining assays, and apoptosis was analyzed by flow cytometry. Knock-down and activation of GPR180 were employed in cell models. Phosphorylation of Smad3 and HDAC4 were inhibited to explore the contribution of the Smad3/HDAC4 signaling pathway in the anti-HCC effects of kaempferol.

Results:QLXJ significantly reduced the tumor recurrence in the 2nd year after operation (40.5% vs 23.0%, $P = 0.002$). In addition, QLXJ inhibited HCC growth in both mouse models. RNA-sequencing and experimental validation revealed that GPR180 may be a potential target of QLXJ. Network pharmacology and UPLC-Q-Orbitrap-MS identified kaempferol as an active component of QLXJ. Kaempferol inhibited the proliferation and induced apoptosis of hepatoma Huh7 and H22 cells. Kaempferol downregulated the expression of GPR180 and decreased phosphorylated proteins of Smad3 and HDAC4. Furthermore, knock-down of GPR180 inhibited the proliferation and induced apoptosis of HCC cells, synergistically with kaempferol. CTHRC1, a ligand of GPR180, attenuated the kaempferol-induced inhibition of proliferation and apoptosis in HCC cells, and increased the levels of p-Smad3 and p-HDAC4. Inhibition of Smad3 and HDAC4 did not increase apoptotic cell death induced by kaempferol. In vivo studies showed that kaempferol significantly could inhibit the tumor growth in subcutaneous model, reducing tumor volume (731.4 ± 169.8 vs 308.8 ± 137.1 mm³, $P < 0.05$) and tumor weight (2.24 ± 1.17 vs 0.96 ± 0.68 g, $P < 0.05$), with a tumor inhibition rate of 57.06%.
Conclusions:QLXJ inhibits tumor growth in both subcutaneous and DEN/CCL4 induced orthotopic HCC models, and kaempferol, as an active component of QLXJ, prevents hepatoma cells proliferation and induces apoptosis via inhibiting miR-32 mediated GPR180/Smad3/HDAC4 signaling pathway.

Keyword(s) : hepatocellular carcinoma, Qilingxiaoji formula, kaempferol, GPR180, miR-32

1580 - Prognostic Evaluation of Hepatocellular Carcinoma Impact of Etiological Factors on Survival Outcomes

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Background / Hypothesis:Hepatocellular carcinoma (HCC) is a highly lethal malignancy that develops in the

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setting of diverse etiological factors. Prognostic evaluation in HCC primarily relies on established scoring systems such as the Barcelona Clinic Liver Cancer (BCLC) staging system, the Model for End-Stage Liver Disease (MELD), and the Child–Pugh classification. More recently, the Albumin–Bilirubin (ALBI) score has been adopted as an additional prognostic indicator reflecting systemic inflammation, hepatic functional reserve, and nutritional status. The objective of this study was to assess the influence of different etiological factors on the prognosis of patients with HCC.

Materials and Methods: A total of 173 patients diagnosed with hepatocellular carcinoma (HCC) and followed at our clinic were retrospectively analyzed. The etiological distribution was as follows: HBV (n=96), HCV (n=11), chronic Delta hepatitis (HDV) (n=20), MAFLD (n=12), and unknown etiology (n=34). For all patients, hemogram and biochemical parameters, BCLC and ALBI scores, MELD and Child–Pugh classifications, survival duration, and mortality outcomes were recorded. Comparisons between etiological groups were performed using the Fisher–Freeman–Halton test for categorical variables and the Kruskal–Wallis test, with a p-value <0.05 considered statistically significant.

Results: No statistically significant difference in mortality was observed among the etiological groups (p = 0.82). Mortality rates were 59.4% in HBV, 45.5% in HCV, 50% in Delta hepatitis, 61.8% in unknown etiology, and 58.3% in MAFLD. A statistically significant difference in mean age was detected across the groups (p < 0.001), with patients in the Delta hepatitis group (58.80 ± 11.71) being significantly younger. No significant intergroup differences were observed regarding survival duration (p = 0.09), ALBI score, BCLC stage, Child–Pugh classification (p = 0.23), or MELD score (p = 0.51).

Conclusions: This study indicates that the underlying etiology of hepatocellular carcinoma does not significantly influence mortality, survival outcomes, or established prognostic scoring systems, including ALBI, BCLC, Child–Pugh, and MELD. Although patients with Delta hepatitis developed HCC at a younger age, this did not translate into a difference in prognosis. These findings suggest that HCC exhibits a comparable prognostic course across different etiological

backgrounds, highlighting the need for larger, multicenter studies to further elucidate the impact of etiology on clinical outcomes.

Keyword(s) : Delta hepatitis, hepatocellular carcinoma (HCC)

1581 - Continuing or Switching Systemic Therapy Combined with Locoregional Therapy Defines a Personalized Strategy for Oligoprogression in HCC A Multicenter Retrospective Study

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Background / Hypothesis: The optimal management of oligoprogression during first-line immune checkpoint inhibitor-based therapy for advanced hepatocellular carcinoma (HCC) remains unclear. This study compared the outcomes between continuing first-line systemic therapy combined with locoregional therapy (FLST+LRT), switching to second-line systemic therapy combined with LRT (SLST+LRT), or switching to SLST alone.

Materials and Methods: This multicenter, retrospective study enrolled 226 patients with oligoprogressive HCC. Patients were stratified into FLST+LRT (n = 87), SLST+LRT (n = 93), and SLST alone (n = 46) groups. Overall survival (OS) and progression-free survival were analyzed. Factors associated with overall were identified.

Results: Median OS was significantly longer in both FLST+LRT (26.4 months) and SLST+LRT (24.9 months) groups compared to SLST alone (18.1 months; p = 0.041 and p = 0.029, respectively). Subgroup analyses demonstrated that among durable responders, FLST+LRT achieved a median OS of 42.5 months, superior to SLST+LRT (19.1 months, p = 0.049) and SLST alone (18.3 months, p = 0.006). In non-durable responders, SLST+LRT resulted in

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longer median OS than both FLST+LRT ($p = 0.030$) and SLST alone ($p = 0.042$). For intrahepatic progression, both LRT-based strategies improved OS compared to SLST alone ($p = 0.004$ and $p < 0.001$, respectively). Multivariate analysis identified ALBI grade (hazard ratio [HR] 1.846, $p = 0.013$), no LRT after oligoprogression (HR 1.683, $p = 0.040$) and AFP > 200 ng/mL (HR 1.795, $p = 0.012$) as independent prognostic factors for OS.

Conclusions: Integrating locoregional therapy for oligoprogressive HCC significantly improves survival compared to switching systemic therapy alone. Treatment should be individualized based on the prior response duration and progression pattern.

Keyword(s) : Hepatocellular carcinoma · Systemic treatment · Immunotherapy · Locoregional therapy · Oligoprogression

1587 - Remodeling of the bile acid connectome across the hepatitis cirrhosis hcc axis network analysis reveals stage specific hubs and potential therapeutic targets

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Background / **Hypothesis:**
While bile acids (BAs) are established players in liver disease progression and are components of predictive models, the dynamics of BA networks across disease stages remain unexplored. We hypothesize that the transition from hepatitis to cirrhosis and hepatocellular carcinoma (HCC) is driven not just by changes in individual BA concentrations, but by a fundamental rewiring of BA interaction networks. This study aims to systematically map and quantify the BA network topology across this disease spectrum to identify hub BAs that may coordinate metabolic dysregulation.

Materials and **Methods:**
We prospectively enrolled 889 patients from Peking University Hepatology Institute in 2025, categorized into six groups: Hepatitis B (HBV), MAFLD, HBV-MAFLD, compensated cirrhosis (CC), decompensated cirrhosis (DC), and HCC. Serum levels of individual BAs were quantified using LC-MS/MS. For each group, a BA co-abundance network $G=(V,E)$ was constructed, where

nodes (V) represent BAs and edges (E) represent strong correlations ($|\text{Spearman's } r| > 0.65$, capturing both synergistic and inhibitory relationships. Network topology was analyzed using density, clustering coefficient, and modularity. Cross-group comparisons of BA profiles and core identities pinpointed hub BAs whose relative changes are linked to global network restructuring.

Results:

We found significant differences in network structure across the Hepatitis-Cirrhosis-HCC Axis. Specifically, network density-reflecting the frequency of interactions between bile acids- increased from 0.09-0.14 (MAFLD, HBV and overlap), 0.15-0.17 (CC and DC), to 0.22 (HCC); clustering coefficient-reflecting the degree of collaboration among BAs within functional modules-rose from 0.58 to 0.74 accordingly. Analysis of network hubs revealed that UDCA as a core BA in MAFLD, where as glycine-conjugated BAs dominated the other groups. That is, GDCA composed 6.4%-8.1% of the BA pool for HBV (with/ without MAFLD; 6.1-7.7 times higher than reference level), and was identified as a driver for progression to CC. GCDCA becomes the hub when further progressing to DC, whereas GCA acts as a consistent hub within the networks throughout the full disease spectrum.

Conclusions:

This work moves beyond individual BA quantification to define the "BA connectome" in liver disease. Identifying the dynamic hub BAs provides novel insights into the critical control points of metabolic dysregulation at each stage. These hubs represent potential biomarkers for disease monitoring and novel therapeutic targets for interrupting progression. Our network analysis framework offers a powerful paradigm for understanding complex metabolic interactions in hepatobiliary diseases.

Keyword(s) : Bile Acids, Liver Disease Progression, Network Analysis, Hepatic Decompensation, Biomarkers, HCC

1588 - Role and Mechanism of TGFβ pSAP Signaling in Regulating Hepatocyte Macrophage Crosstalk in Metabolic Dysfunction Associated Steatohepatitis

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Background / Hypothesis: Metabolic dysfunction-associated steatohepatitis (MASH) is an escalating public health challenge in China, driven by increasing rates of obesity and type 2 diabetes. Dysregulated sphingolipid metabolism has been implicated in disease progression, yet the underlying mechanisms remain poorly defined.

Materials and Methods: Liver tissues from patients with MASH and matched controls were analyzed for GlcCer (18:1/16:0) levels. Hepatocyte-specific Psap knockout (Psap^ΔHKO) and myeloid-specific Gpr37 knockout (Gpr37^ΔMKO) mice were generated and fed a Western diet for 26 weeks to establish experimental MASH models. Targeted lipidomics, spatial transcriptomics, and single-cell transcriptomics were applied to investigate hepatic lipid metabolism, inflammation, and fibrosis-related pathways.

Results: TGFβ signaling was found to induce prosaposin (pSAP) glycosylation in hepatocytes, promoting its extracellular secretion. MASH livers exhibited marked accumulation of GlcCer (18:1/16:0), while extracellular glycosylated pSAP activated GPR37 in macrophages, triggering enhanced TGFβ secretion. This TGFβ-enriched environment further amplified pSAP glycosylation and secretion, driving MASH progression through two mechanisms: (1) accumulated GlcCer competitively inhibited HSC70-PLIN2 interaction, impairing lipophagy-mediated lipid droplet degradation; and (2) glycosylated pSAP activated GPR37 signaling in macrophages, reinforcing TGFβ release and establishing a hepatocyte-macrophage-TGFβ positive feedback loop.

Conclusions: These findings identify the TGFβ/pSAP axis as a key regulator of hepatocyte-macrophage crosstalk in MASH, advancing mechanistic understanding and highlighting novel therapeutic opportunities. Furthermore, circulating glycosylated pSAP may serve as a promising biomarker for MASH diagnosis.

Keyword(s) : Metabolic dysfunction-associated steatohepatitis, Sphingolipid metabolism, TGFβ, Lipophagy, Hepatocyte-macrophage crosstalk

1589 - Intermittent therapy restores anti HBV efficacy of IFN by reducing elevated dysfunctional HLA-DR+CD38hiCD8+ T lymphocytes

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Background / Hypothesis: After long-term interferon (IFN) therapy, some patients experience a stagnation of hepatitis B surface antigen (HBsAg) levels. Only after IFN intermittent therapy do the antiviral efficacy continue. Here, We aimed to explore the immunological mechanisms underlying this phenomenon in patients with chronic hepatitis B (CHB).

Materials and Methods: After conducting a prospective recruitment of CHB patients, IFN therapy was administered to some patients in the Naive group and the nucleoside (nucleotide) analogue (NA)-treated group. For those in the IFN-treated plateau group (abbreviated as Plateau group) who experienced stagnation in HBsAg decline during IFN therapy, IFN intermittent therapy was employed, followed by IFN retreatment. At the corresponding time points, peripheral blood samples were collected to measure clinical indicators. Flow cytometry was used to assess cell phenotype and function, and Luminex multiplex assays were employed to evaluate the levels of soluble mediators in plasma.

Results:

A total of 151 patients were enrolled. The Plateau group exhibited a significant increase in the percentage of HLA-DR+CD38dim subgroup and a significant decrease in HLA-DR+CD38hi subgroup in T lymphocytes after 12-24 weeks of IFN intermittent therapy. After another 12-24 weeks of IFN retreatment, the HLA-DR+CD38dim subgroup markedly decreased while the latter significantly increased. Concurrently, both the HBsAg and the HLA-DR+CD38hi subgroup in T lymphocytes were remarkably lower than the levels of baseline at IFN discontinuation. Additionally, only in CD8+ T lymphocytes, HLA-DR+CD38hi subgroup expressed lower levels of co-stimulatory molecules and cytotoxic functions compared to the activated HLA-DR+CD38dim subgroup. Finally, plasma cytokine results showed IFN-inducible protein (IP)-10 was the strongest correlation with HLA-DR+CD38hiCD8+ T lymphocytes during IFN-based antiviral therapy.

Conclusions:

The emergence of the IFN-treated plateau phase may be associated with an excessively high proportion of

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dysfunction HLA-DR+CD38hiCD8+ T lymphocytes, indicating impaired cytotoxic function. IFN intermittent therapy can reverse this phenomenon. Furthermore, IP-10 may serve as a plasma cytokine marker for predicting antiviral efficacy.

Keyword(s) : chronic hepatitis B, interferon, HLA-DR+CD38+CD8+ T lymphocytes, plateau phase, intermittent therapy

1592 - Fibroscan role in determining the patients who are at high risk for developing HCC in 1000 cirrhotic Egyptian patients.

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Background / Hypothesis:Hepatocellular carcinoma (HCC) is common primary malignancy of the liver and one of the most frequent causes of death in patients with liver cirrhosis. Nowadays, liver stiffness measured noninvasively by transient elastography has been reported to be well correlated with histologically assessed liver fibrosis stage. The aim of this study was to study the role of ultrasound elastography (FibroScan) in early detection of HCC in cirrhotic patients as well as verifying whether it could be used as a tool for identifying cirrhotic patients who are at high risk of developing HCC.

Materials and Methods:

This study was conducted on 1000 patients with cirrhosis of both sexes. The studied patients were classified into: group I which included 500 patients with cirrhosis and HCC diagnosed by ultrasound examination and confirmed by triphasic computed tomography and group II which included 500 patients with cirrhosis and without evidence of HCC diagnosed by clinical, laboratory and ultrasound examination. Patients with morbid obesity, ascites, hepatic metastatic lesions and patients with HCC beyond Milan criteria were excluded from this study. Tumour characteristics were assessed. Tumor staging was done using Okuda, CLIP, VISUM and Tokyo staging systems. Transient elastography was done for all patients and all their results were expressed in kilopascal.

Results:

Patients with HCC had a mean age of 57.3 years old, while cirrhotic non-HCC patients are younger with a mean age of 51.4 years old. Also HCC commonly presented in males (86%) more than females (14%). Liver stiffness was significantly higher in HCC patients compared to cirrhotic patients. The sensitivity and specificity in diagnosis of HCC were 76% and 87% respectively at cut-off of 30.5 kpa with 91.8% accuracy. Fibroscan has a positive significant correlation with tumour size ($P<0.001$), Child-Pugh ($P<0.001$), Okuda classification ($P<0.001$), CLIP staging ($P<0.001$) and Tokyo classification ($P<0.001$) among HCC patients. It was found that likelihood of HCC risk was correlated with increase of liver stiffness. At liver stiffness of 25-30 kpa the probability of HCC is 93% so, these patients should undergo close follow up. Patients with stiffness ≥ 30 kpa had HCC.

Conclusions:

According to this study, fibroscan has an important role in early detection of HCC in cirrhotic patients with cut off 30.5 Kpa. Cirrhotic patients with liver stiffness ≥ 30 kpa are most likely to have HCC. Cirrhotic patients with liver stiffness ≥ 25 kpa need close follow up as they are at high risk to develop HCC.

Keyword(s) : HCC; Fibroscan; Egyptian

1596 - ABT score based clinical model predicts prognosis in unresectable hepatocellular carcinoma treated with TACE combined with Bevacizumab and Sintilimab therapy

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Background / Hypothesis:TACE combined with Bevacizumab and Sintilimab (T-B-S) therapy demonstrated promising treatment effects and improved prognosis in unresectable hepatocellular carcinoma (uHCC). However, biomarkers for predicting the treatment efficacy remains to be further explored. In this study, we aimed to construct a noninvasive and easily assessable clinical model, in order to predict prognosis of uHCC receiving T-B-S therapy. Materials and Methods:156 uHCC patients received T-B-S therapy were retrospectively recruited. Univariate and multivariate Cox regression analyses were used to identify independent prognostic factors for overall

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survival (OS). Model prediction potential was assessed by area under the receiver operating curve (ROC) and C-index. Survival curves were compared by Kaplan-Meier method.

Results: 156 uHCC patients were divided into training and validation cohorts (86 and 70 cases), and among all patients, 43 achieved complete response, 53 showed partial response, 32 had stable disease and 28 had progressive disease. The objective response and disease control rate were 61.5 and 82.1%, respectively. 23 patients underwent sequential resection (12 in training cohort and 11 in validation cohort). Univariate and multivariate Cox regression analyses identified alkaline phosphatase to prealbumin ratio (APR) > 0.96, BCLC-C stage and TBIL >17 µmol/L as independent predictors of poor survival, which consist of the ABT score. The area under the ROC of ABT model for predicting 2-year OS was 0.823 in training cohort and 0.828 in validation cohort. 2-year OS of different risk groups defined by ABT based clinical model were 89.8%, 43.7%, and 12.9%, respectively ($p < 0.0001$).
Conclusions: The T-B-S therapy showed promising treatment efficacy. ABT based clinical model was constructed to predict survival of uHCC receiving T-B-S treatment and the ABT model performed well in risk stratification.

Keyword(s) : Unresectable hepatocellular carcinoma, T-B-S therapy, Prognostic model, Progression-free survival.

1598 - Pneumonia incidence and pathogen spectrum in liver transplant recipients a decade long retrospective analysis comparing pre coronavirus disease 2019 and pandemic era temporal patterns and risk factors

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Background/Hypothesis: Liver transplant recipients face substantially elevated pneumonia risk due to pre-existing immune dysfunction from cirrhosis and intensive post-transplant immunosuppression, contributing significantly to morbidity and mortality. Pneumonia accounts for 15.5% of infection-related deaths in this population. Most studies lack detailed analysis of pre-transplant liver-specific risk factors, including severity scores (MELD, Child-Pugh) and

metabolic status. This study examines whether pneumonia incidence and pathogen distribution vary across post-transplant periods and are influenced by these specific factors. The Coronavirus Disease 2019 (COVID-19) pandemic potentially altered infection epidemiology, and this research will assess whether the shift toward stricter infection control and altered antibiotic use is reflected in measurable changes in multidrug-resistant bacterial and fungal pneumonia rates.

Materials and Methods: This retrospective cohort study includes 812 adult liver transplant recipients (≥18 years) at Acibadem Atakent Hospital from September 2015 to September 2025. Pneumonia diagnosis will follow CDC criteria with clinical and radiological confirmation. Data will be stratified into pre-COVID (September 2015-February 2020) and COVID-19 era (March 2020-September 2025) periods. Variables extracted will include MELD/Child-Pugh scores, liver disease etiology, immunosuppression details, and viral serostatus. Pneumonia episodes will be classified by timing: early (0-30 days), intermediate (31-180 days), and late (>180 days), and by acquisition type (Community-Acquired, Healthcare-Associated, Nosocomial). Incidence rates per 1000 patient-years will be calculated and multivariable Cox regression with time-dependent covariates will be used to identify independent risk factors ($p < 0.05$).

Results: The large cohort of 812 patients will define dynamic shifts in pathogen distribution and antimicrobial resistance patterns across early, intermediate, and late periods. Results will define distinct pathogen profiles for each period, focusing on multidrug-resistant organisms. Key liver-specific risk factors, such as high pre-transplant MELD score and immunosuppressive regimen, will be identified with Hazard Ratios. Dedicated analysis will quantify changes in overall incidence and MDR organism and viral pneumonia rates between pre-COVID and COVID-19 eras.

Conclusions: This study will fill important gaps in understanding post-transplant pneumonia through its decade-long, multi-dimensional analysis and COVID-19 era comparison. Findings will inform organ-specific

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infection prevention protocols, including targeted prophylaxis recommendations (e.g., fungal/viral agents), and refine antimicrobial stewardship programs. Results will guide therapy selection and management, ultimately improving safety and outcomes in liver transplant recipients.

Keyword(s) : liver transplantation, pneumonia, pathogen spectrum, immunosuppression, post-transplant infection

1602 - The Association Analysis of Metabolic Comorbidities and the 10 Year Prognosis of Hepatic Events in Chronic Hepatitis B Patients

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Background / **Hypothesis:**
Chronic hepatitis B (CHB) patients face persistent risk of hepatocellular carcinoma(HCC) and liver-related mortality despite nucleos(t)ide analogue-induced viral suppression. The prognostic significance of metabolic comorbidities (diabetes, hypertension, dyslipidemia) on long-term outcomes remains inadequately quantified. To determine whether metabolic disorders independently predict 10-year liver-related events (HCC, transplantation, mortality) in CHB.

Materials and **Methods:**
This retrospective cohort analyzed 1,717 CHB patients from Beijing Titan Hospital(2010–2020). Metabolic comorbidities were ascertained by chart review. Liver-related events were assessed using Kaplan-Meier analysis and multivariable Cox regression.

Results:
The 10-year cumulative incidence of liver-related events was 9.03%. Metabolic comorbidities significantly elevated risk: hypertension (10.38%), diabetes (16.92%), and dyslipidemia (9.52%) (all p<0.05). Multivariable Cox regression identified ≥2 metabolic comorbidities as an independent predictor of adverse outcomes (adjusted HR 2.41; 95% CI 1.72–3.38; p<0.001), irrespective of baseline MELD score. Noncirrhotic HBeAg-negative patients with low HBV DNA (<2000 IU/mL) but metabolic comorbidities exhibited substantially increased event risk.

Conclusions:

Metabolic comorbidities independently escalate 10-year liver event risk in CHB, particularly with multiple disorders. Integrated management concurrently targeting viral suppression and metabolic pathways is essential, even in virologically controlled patients.

Keyword(s) : Metabolic comorbidities; Chronic Hepatitis B(CHB); Liver-related events; Prognosis.

1603 - FcγR II B mediates CD8+ T cells dysfunction in Hepatocellular Carcinoma

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Background / **Hypothesis:**
Reversing the functional inhibition of CD8+ T cells is key to overcoming challenges of immunotherapy in hepatocellular carcinoma (HCC). FcγR II B is an inhibitory receptor expressed on various immune cells, including B cells, macrophages, and CD8+ T cells. Soluble fibrinogen-like protein 2 (sFGL2), a functional ligand of FcγR II B, has been reported to play an important role in promoting the growth of several cancers. However, the direct impacts of FcγR II B on CD8+ T cells remain to be clarified. Here, our aim is to elucidate the role of FcγR II B, specifically sFGL2-FcγR II B axis, in the direct suppression of CD8+ T cells in HCC.
Materials and **Methods:**
Murine HCC models were established through subcutaneous or orthotopic injection of Hepa1-6 cells in wild-type (WT), fcgr2b^{-/-} or fgl2^{-/-} C57BL/6J mice. Expression of FcγR II B on subsets of CD8+ T cells and immunosuppressive effects of FcγR II B were identified in the murine HCC models by multicolor flow cytometry. Features of FcγR II B expression in CD8+ T cells were also verified in murine models of melanoma (B16), colon cancer (MC38) and glioma (GL261). Murine HCC tissues were analyzed by ELISA, multiplex immunohistochemistry and co-immunoprecipitation to evaluate the expression level of sFGL2 and interactions between sFGL2 and FcγR II B. Functional markers associated with sFGL2-FcγRIIB axis in CD8+ T cells were identified by multicolor flow cytometry.

Results:

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FcγR II B expression was mainly observed on activated CD8⁺ T cells in murine HCC, melanoma, colon cancer and glioma models. Conventional fcgr2b knockout effectively controlled tumor growth (murine HCC models) and increased the number of tumor-infiltrating CD8⁺ T cells. Compared to WT CD8⁺ T cells, the fcgr2b knockout CD8⁺ T cells downregulated expression of Caspase-3 and Caspase-7 and enhanced cytotoxicity. Besides, ELISA confirmed a high sFGL2 expression in murine HCC tissues. Co-immunoprecipitation and multiple immunofluorescences demonstrated an interaction and co-localization between sFGL2 and FcγR II B. Furthermore, compared to FcγRIIB⁻ CD8⁺ T cells, knockout of fgl2 significantly reduced the expression of Caspase-3, Caspase-7 in FcγR II B⁺ CD8⁺ T cells. Conclusions:

Our results identify FcγR II B as a new immune checkpoint of CD8⁺ T cells, which severely limits anti-tumor immunity. Targeting FcγR II B may represent a promising strategy for treatment of HCC.

Keyword(s) : Hepatocellular carcinoma, Tumor immunology, FcγR II B, CD8⁺ T cells, Soluble fibrinogen-like protein 2

1607 - From Dysbiosis to Prediction A Novel Gut Microbiota-Derived Index for Spontaneous Bacterial Peritonitis in HBV Related Cirrhosis
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Background / Hypothesis: Spontaneous bacterial peritonitis (SBP) is a severe complication of hepatitis B virus (HBV)-related cirrhosis, in which gut microbiota dysbiosis plays a pivotal role. This study aimed to characterize microbial alterations, establish a microbiota-derived index (SBP-MI), evaluate longitudinal changes, and develop an SBP risk prediction model (SBP-RP).

Materials and Methods: A total of 135 participants were included: healthy controls (n=40), compensated cirrhosis (n=30), cirrhosis with ascites but without SBP (n=40), and SBP (n=25). Fecal 16S rRNA sequencing and clinical data were obtained. An additional 140 cirrhotic patients with ascites were prospectively followed for 6 months, with SBP occurrence as the endpoint; 40 provided paired fecal samples. SBP-MI was constructed from key microbial shifts, and multivariable Firth logistic regression identified independent predictors.

Results: SBP was characterized by enrichment of pathogenic taxa (Escherichia-Shigella, Klebsiella, Veillonella, Streptococcus) and depletion of short-chain fatty acid producers (Prevotella, Roseburia, Faecalibacterium, Bacteroides), forming the basis of SBP-MI. During follow-up, improved patients had greater microbial diversity and beneficial commensals, whereas progression was linked to Haemophilus expansion. SBP-MI effectively tracked these changes and outperformed the Hepatitis B Cirrhosis Dysbiosis Index. Multivariable analysis identified INR, previous history of SBP, and SBP-MI as independent predictors. The SBP-RP yielded an AUC of 0.91, with calibration that appeared acceptable in this cohort.

Conclusions: Distinct dysbiosis characterizes SBP. SBP-MI captures microbial imbalance and progression, while the SBP-RP model integrating microbial and clinical factors provides promising predictive value for early risk stratification.

Keyword(s) : HBV-related cirrhosis, spontaneous bacterial peritonitis, gut microbiota-derived index, 16S rRNA, prediction model

1608 - Two Year Follow up of a Dose Escalation Trial of Mesenchymal Stromal Cell Therapy for Decompensated Liver Cirrhosis
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Background / Hypothesis: Decompensated liver cirrhosis (DLC) imposes a substantial global health burden with limited therapeutic options. Mesenchymal stromal cell (MSC) therapy has shown potential benefits in DLC; however, optimal dosing strategies and dose-dependent safety and efficacy in humans remain unclear. We conducted a Phase Ia trial (NCT05227846) using a single-arm, dose-

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escalation design to evaluate the safety and tolerability of MSC therapy in patients with DLC and to collect preliminary therapeutic signals over a 2-year follow-up period.

Materials and Methods: Patients were assigned to one of four single-dose MSC cohorts: Cohort I (5.0×10^7 cells), Cohort II (1.0×10^8 cells), Cohort III (1.5×10^8 cells), and Cohort IV (2.0×10^8 cells). Follow-up assessments were conducted at 3 days, 7 days, 14 days, 28 days, and at 3, 6, 9, 12, 15, 18, 21, and 24 months post-treatment. The primary safety endpoint was the incidence of adverse events (AEs). Preliminary therapeutic signals included changes in Child–Pugh and MELD scores, liver function parameters and quality of life. Additional outcomes included DLC-related complications, progression to liver failure and liver transplant-free survival.

Results:

Among 15 participants, 13 experienced a total of 24 AEs (86.67%). In Cohort I, all 3 participants reported 4 AEs; in Cohort II, all 3 participants reported 9 AEs; in Cohort III, all 6 participants reported 10 AEs; and in Cohort IV, 1 of 3 participants reported 1 AE. No grade ≥ 3 AEs leading to withdrawal occurred, and no dose-limiting toxicities were observed. Following infusion, MELD scores showed a decreasing trend from Day 3 onward in Cohorts II, III, and IV, with Cohort IV maintaining values below baseline throughout the 15-month observation period. Child–Pugh scores decreased from Day 3 in Cohorts I, III, and IV, with Cohort IV remaining below baseline throughout the 24-month follow-up. These findings suggest a potential therapeutic advantage at higher dosage.

Conclusions:

MSC therapy demonstrated good safety and tolerability across all cohorts, with no severe AEs, dose-limiting toxicities, or unexpected serious adverse reactions during the 2-year follow-up. All cohorts exhibited early improvements in MELD and Child–Pugh scores, with the most pronounced and sustained improvements observed in Cohorts III and IV. These results support further evaluation in a multicenter controlled trial.

Keyword(s): Decompensated liver cirrhosis; Mesenchymal stem cells; Dosage; 2-year follow-up; Safety; Therapeutic advantage

1609 - Prediction of Liver Related Events Using Non invasive Fibrosis Scores in Patients with Metabolic Dysfunction–Associated Fatty Liver Disease

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Background / Hypothesis: To assess the ability of non-invasive fibrosis scores to predict liver-related events (LREs) in patients with metabolic dysfunction–associated fatty liver disease (MAFLD).

Materials and Methods: We prospectively enrolled patients with biopsy-confirmed MAFLD at Beijing Friendship Hospital from 2018 to 2025 and conducted follow-up. Demographic and laboratory data were collected. Evaluated non-invasive scores included liver stiffness measurement (LSM) by transient elastography, APRI, FIB-4, NFS, Agile 3+, Agile 4, and FAST. LREs were defined as hepatic decompensations, hepatocellular carcinoma, death, liver transplantation, or clinically significant portal hypertension (CSPH) according to the Baveno VII consensus. Patients were stratified into risk categories based on established cut-offs for each score. The cumulative incidence of LREs across strata was compared using Kaplan–Meier analysis, and Cox regression models were used to evaluate the predictive performance of each score.

Results:

A total of 226 patients with biopsy-proven MAFLD were included, of whom 70 (31.0%) were male, with a median age of 54 years (IQR: 41–61) and a median follow-up of 36 months. Twenty-two patients (9.7%) developed LREs. Compared to the non-LRE group, the LRE group had higher baseline levels of total bilirubin, creatinine, LSM, and non-invasive fibrosis scores, as well as a higher prevalence of diabetes, but lower platelet counts (all $P < 0.01$). During follow-up, LSM and all non-invasive scores remained elevated in the LRE group. Risk stratification revealed that patients in the high-risk category of each score had the highest 3-year cumulative incidence of LREs (LSM ≥ 15.0 : 39.4%; APRI ≥ 1.5 : 27.3%; FIB-4 > 2.67 : 31.6%; NFS > 0.676 : 37.8%; FAST ≥ 0.67 : 22.1%; Agile3+ ≥ 0.679 : 27.3%; Agile4 ≥ 0.565 : 35.9%), all significantly higher than in low- and

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intermediate-risk groups ($P < 0.01$). In univariable Cox regression, LSM and all non-invasive scores were significantly associated with an increased risk of LREs (LSM: HR = 2.44, 95% CI: 1.88–3.17; APRI: HR = 1.74, 95% CI: 1.30–2.33; FIB-4: HR = 1.80, 95% CI: 1.45–2.24; NFS: HR = 3.79, 95% CI: 2.44–5.89; Agile3+: HR = 17.66, 95% CI: 4.79–65.07; Agile4: HR = 3.39, 95% CI: 2.24–5.13; FAST: HR = 2.88, 95% CI: 1.63–5.10).

Conclusions:

MAFLD patients with baseline LSM or non-invasive fibrosis scores in the high-risk range are at significantly increased risk of liver-related events.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease; Non-invasive scores; Liver-related events; Liver fibrosis

1615 - Mechanistic Study of Celiac Sympathetic Denervation in the Treatment of Metabolic Dysfunction Associated Steatohepatitis (MASH)

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Background / Hypothesis: Metabolic dysfunction-associated steatohepatitis (MASH) is a complex hepatic disorder characterized by pathological lipid accumulation, chronic inflammation, and hepatocellular injury. Emerging evidence implicates the sympathetic nervous system (SNS) in the pathogenesis of MASH. This study aims to elucidate the regulatory mechanisms of celiac sympathetic denervation in MASH progression.

Materials and Methods: A murine MASH model was established through a high-fat, high-cholesterol, and high-sucrose diet. Sympathetic denervation was achieved via two approaches: chemical ablation using intraperitoneal 6-hydroxydopamine (6-OHDA) and ethanol-based celiac sympathetic denervation. Hepatic norepinephrine (NE) levels, serum biochemical profiles, and histopathological analyses were employed to evaluate the therapeutic effects of sympathetic modulation on MASH progression. The pathways associated with sympathetic nerve modulation in metabolic dysfunction-associated steatohepatitis (MASH) were identified through an integrated multi-omics approach combining hepatic and intestinal metabolomics, proteomics, and metagenomics analyses.

Results: Celiac sympathetic denervation significantly

attenuated hepatic lipid accumulation, reduced inflammatory infiltrates, and improved glucose tolerance in diet-induced MASH mice. Mechanistically, this intervention disrupted sympathetic overactivation, thereby ameliorating metabolic dysregulation and hepatocellular injury. Integrated multi-omics analyses revealed that sympathectomy exerts modulatory effects on multiple hepatic pathways involved in fatty acid oxidation, glucose metabolism regulation, cholesterol metabolism, and bile acid biosynthesis.

Conclusions: Our findings provide preclinical evidence supporting sympathetic neuromodulation as a novel therapeutic approach for MASH, offering insights into its pathophysiological mechanisms.

Keyword(s) : Metabolic dysfunction-associated steatohepatitis (MASH); Sympathetic nervous system (SNS)

1619 - Safety and Effectiveness of Peginterferon Alfa 2b Combined with Tenofovir Amibufenamide in Patients with Chronic Hepatitis B

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Background / Hypothesis: Data on the combination of Peg-IFN α -2b and tenofovir amibufenamide (TMF) remain limited. This study aims to evaluate the safety and effectiveness of Peg-IFN α -2b combined with TMF in patients with CHB. Materials and Methods:

Patients treated with TMF (25 mg/day) and Peg-IFN α -2b (180 μ g/week) from January 2022 to May 2025 were retrospectively enrolled from three hospitals in Henan, China. This study included two groups: the switch combination group, consisting of patients who transitioned from nucleos(t)ide analog (NA) monotherapy to a combination of TMF and Peg-IFN α -2b; and the treatment-naïve combination group, comprising patients who received TMF plus Peg-IFN α -2b as initial therapy. The primary endpoint of this study was the rate of hepatitis B surface antigen (HBsAg) loss at 48 weeks of treatment, the secondary endpoints were

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the rate HBV DNA undetectable (<25 IU/mL) and safety profiles during treatment.

Results:

A total of 180 patients were enrolled, with 120 patients in the switch group and 60 patients in the treatment-naïve group. The treatment period of NA monotherapy in switch group was 67.45 ± 35.36 months. At baseline, the age, HBV DNA, hepatitis B e antigen (HBeAg), HBsAg, alanine aminotransferase (ALT) levels of the patients in the switch group and treatment-naïve group were 38.48 ± 0.89 vs. 37.20 ± 1.28 years ($p=0.413$), 4.1 (2.6, 6.5) vs. 4.9 (3.1, 7.8) log₁₀ IU/mL ($p=0.169$), 255.0 (11.0, 406.6) vs. 304.9 (12.1, 459.7) PEIU/mL ($p=0.536$), 2.63 ± 1.14 vs. 2.79 ± 1.18 log₁₀ IU/mL ($p=0.370$), 34.10 ± 4.31 vs. 50.14 ± 6.62 U/L ($p=0.037$), respectively; At 48 weeks of treatment (Figure 1), the HBV DNA undetectable rates were 99.17% vs. 96.67% ($p=0.537$), the HBeAg clearance rates were 76.67% vs. 42.86% ($p=0.026$), the HBsAg loss rates were 44.16% vs 28.33% ($p=0.040$), in the switch group and treatment-naïve group, respectively. Regarding laboratory abnormalities, the main adverse events during treatment were the ALT elevation (57.50% vs. 33.33%, $p=0.002$), leukopenia (46.67% vs. 31.67%, $p=0.054$), neutropenia (45.83% vs. 28.33%, $p=0.024$), thrombocytopenia (65.83% vs. 40.00%, $p=0.001$), in the switch combination group and treatment-naïve combination group, respectively. Only one patient presented with elevated total bilirubin (39.2 µmol/L). No serious adverse events occurred.

Conclusions:

At week 48 of treatment, the switch combination group exhibited significantly higher rates of HBsAg loss and HBeAg seroconversion compared to the treatment-naïve combination group. Both groups maintained a favorable overall safety profile throughout the treatment period. These findings suggest that initiating therapy with nucleos(t)ide analogs followed by combination with Peg-IFN α may be a reasonable and promising strategy to promote HBsAg

Keyword(s) : Combined treatment; Chronic hepatitis B; Effectiveness; Safety; Tenofovir amibufenamide; Peg-IFN α -2b

1624 - A Noninvasive Model Based on Liver and Spleen Microcirculatory Perfusion Parameters for Predicting High Risk Varices in CHB Related Cirrhosis

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Background

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Hypothesis:

Hepatic and splenic microcirculatory perfusion is closely associated with the development of portal hypertension. This study aimed to evaluate the utility of liver and spleen microcirculatory perfusion parameters in ruling out high-risk varices (HRV) in chronic hepatitis B (CHB) -related cirrhosis patients receiving antiviral therapy.

Materials

and

Methods:

CHB-related cirrhosis patients receiving antiviral therapy who underwent both upper gastrointestinal endoscopy (EGD) and upper abdominal IVIM (Intravoxel Incoherent Motion) imaging between January 2020 and October 2025 were retrospectively enrolled. Based on EGD findings, patients were classified into high-risk and non-high-risk varices groups. Univariate and multivariate regression analyses were performed to identify potential predictors of HRV, and a predictive model was subsequently established using binary logistic regression. The diagnostic performance, spared EGD rate, and safety profile of this model were compared with FIB-4, APRI, Baveno VI criteria ($LSM < 15$ kPa and $PLT > 150 \times 10^9/L$).

Results:

A total of 113 patients were included, with a mean age of 60 ± 9 years, and 71 (62.8%) were male. The alanine aminotransferase level was 32.9 (24.0, 57.0) U/L, and the median liver stiffness measurement was 16.6 (13.0, 20.9) kPa. Among the enrolled patients, 40.7% (46/113) had HRV. Among all clinical and IVIM parameters evaluated, hepatic D* and splenic D* emerged as the only independent predictors, each reflecting microcirculatory perfusion of the liver and spleen, respectively. These two parameters were then incorporated to construct the Hepato-Splenic Microcirculatory Perfusion Model (HS-MPM). Compared with APRI (AUROC: 0.632) and FIB-4 (AUROC: 0.674), HS-MPM exhibited superior diagnostic accuracy (AUROC: 0.897). With an acceptable risk of missing HRV (4.3%), the proportion of endoscopies that could be safely avoided increased from 21.2% using the Baveno VI criteria to 39.8% with HS-MPM.

Conclusions:

HS-MPM, derived from hepatic and splenic microcirculatory perfusion parameters, represents a

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potential noninvasive tool for ruling out HRV and reducing unnecessary endoscopic examinations.

Keyword(s) : Chronic hepatitis B; Cirrhosis; High-risk varices; Microcirculatory perfusion; Noninvasive model

1625 - Alcohol Consumption Accelerates Liver Fibrosis in Chronic Hepatitis B Evidence from Mendelian Randomization and a Prospective Cohort Study

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Background / Hypothesis: Observational studies have suggested that alcohol consumption increases the risk of chronic liver diseases, but the causal role of alcohol consumption for viral hepatitis remains unclear. We used Mendelian randomization (MR) to predict the effect of alcohol consumption on viral hepatitis B. To delineate the impact of alcohol consumption on viral load, liver inflammation, and fibrosis progression in patients with chronic hepatitis B (CHB) through a prospective cohort study. Materials and Methods: Up to 45 single-nucleotide polymorphisms were used as instrumental variables for alcohol consumption. Genetic association estimates for viral hepatitis were obtained from large-scale consortia and UK Biobank. Analyses were conducted using the inverse variance-weighted, weighted median, MR-PRESSO, MR-Egger, and multivariable MR methods. A prospective cohort of hospitalized patients with chronic hepatitis B and a history of alcohol use was enrolled between 2019 and 2024. These patients were followed for 24 months to assess changes in viral load, liver inflammation, and fibrosis progression.

Results:

Genetically predicted alcohol consumption was consistently associated with viral hepatitis B (HBV) across the different analyses. The odds ratios (ORs) per 1-SD increase of log-transformed alcoholic drinks per week were 1.27 ([95% CI, 1.12–1.45] $P=2.87 \times 10^{-4}$) for HBV in the inverse variance-weighted analysis. Compared to the non-drinking group, patients in the moderate-to-heavy drinking group showed no significant difference in HBV DNA levels ($P=0.84$). However, they exhibited significantly lower rates of ALT/AST normalization ($P=0.007$) and faster progression of liver fibrosis ($P=0.003$). This association demonstrated a dose-effect relationship, where even light drinking exerted adverse effects on fibrosis regression, albeit to a lesser extent than moderate-to-heavy consumption.

Conclusions:

This study provides evidence of a causal relationship between higher alcohol consumption and increased risk of HBV. Even light to moderate alcohol intake can act synergistically with viral hepatitis to promote rapid liver disease progression. Therefore, abstinence from alcohol is strongly recommended to protect liver health, particularly for individuals with HBV infection, with the aim of enhancing the clinical efficacy of antiviral therapy and preventing accelerated disease progression in chronic viral hepatitis.

Keyword(s) : alcohol consumption, Hepatitis B virus, Mendelian randomization, Prospective cohort study, Liver fibrosis

1629 - Analysis of Risk Factors and Establishment of a Predictive Model for Liver Disease Severity in Non Alcoholic Fatty Liver Disease

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Background / Hypothesis: To identify risk factors associated with liver disease severity in patients with non-alcoholic fatty liver disease (NAFLD) and develop a risk prediction model. Materials and Methods: Clinical data were collected from NAFLD patients diagnosed via liver biopsy at the Second Department of Hepatology, Beijing Ditan Hospital, Capital Medical University, from January 2018 to December 2022. Patients were initially grouped by ALT levels to analyze its limitations in NAFLD diagnosis. Pathological reports were used to classify patients into non-significant (S/F/G < 2) and significant (S/F/G ≥ 2) groups based on fibrosis (S), inflammation (G), and steatosis (F) severity. Logistic regression identified independent risk factors for liver disease severity, and a predictive model was established. Model performance was validated using the area under the curve (AUC).

Results:

Significant differences were observed between ALT-normal and ALT-abnormal groups in AST, GGT, ALP, BMI, hyperlipidemia prevalence, age, and diabetes prevalence ($P < 0.05$). Univariate analysis revealed age, BMI, AST, ALB, ALP, TG, and high-calorie diet as significant variables ($P < 0.05$) across inflammation, steatosis, and fibrosis subgroups. When stratified by inflammation severity, multivariate analysis identified sex, age, BMI, ALB, and PclII as independent predictors of significant inflammation in NAFLD. High BMI and hyperlipidemia were risk factors for NAFLD steatosis.

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When grouped by fibrosis severity, age, height, BMI, ALB, and PcllI emerged as independent predictors of pathologically significant fibrosis in NAFLD. Patients with $G \geq 2$ and/or $S \geq 2$ were classified as the significant pathology group, while others formed the non-significant group. A logistic regression model was constructed using regression coefficients and constants, $P=1/(1+e^{-Y})$, $Y=-12.486+0.913 \times \text{Sex} + 0.442 \times \text{BMI} + 0.04 \times \text{PcllI} - 0.085 \times \text{ALB}$. The area under the ROC curve was 0.833 (95% CI: 0.797–0.869), with a maximum Youden index of 0.456, corresponding to sensitivity and specificity of 67.3% and 83.3%, respectively.

Conclusions:

Sex, BMI, PcllI, and ALB are closely associated with NAFLD severity. The logistic regression model demonstrates potential clinical utility for predicting significant liver pathology.

Keyword(s) : Non-alcoholic fatty liver disease, ALT, Inflammation, Steatosis, Fibrosis

1634 - Integrated Plasma and Fecal Metabolic Profiling in Alcoholic Cirrhosis Association with Gut Microbiota Dysbiosis

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Background / Hypothesis:

The gut microbiota is critically involved in the progression of alcohol-related liver disease. However, comprehensive research simultaneously characterizing the specific alterations in gut microecology and the corresponding changes in host plasma and fecal metabolites in patients with alcoholic liver cirrhosis (ALC) remains limited, leaving their potential roles and clinical implications not fully understood.

Materials and Methods:

This case-control study enrolled male patients with ALC (n=27) and healthy controls (HC, n=24). Researchers performed 16S rRNA gene sequencing on fecal DNA to profile the gut microbiota. Additionally, untargeted liquid chromatography–mass spectrometry (LC-MS) metabolomics was applied to analyze both plasma and fecal samples. Differences in microbial composition and metabolite profiles were compared, and their associations with each other were evaluated.

Results:

The study revealed significant gut dysbiosis in ALC patients. At the genus level, there was a notable increase in opportunistic pathogens like *Veillonella*, *Lactobacillus*, *Streptococcus*, *Klebsiella*, and *Escherichia*, alongside a decrease in beneficial commensals such as *Ruminococcus*, *Bacteroides*, *Alistipes*, and *Prevotella*.

Metabolically, bile acid and amino acid pathways were highly disturbed. In plasma, primary and secondary bile acids (e.g., CA, DCA) and aromatic amino acids (e.g., phenylalanine, tyrosine) and their derivatives (e.g., indole) were significantly elevated, while beneficial metabolites like taurine and betaine were decreased. Crucially, correlation analysis showed that the enriched pathogenic genera (*Streptococcus*, *Lactobacillus*, *Veillonella*) were positively correlated with these accumulating harmful plasma metabolites and negatively correlated with the depleted commensal bacteria.

In feces, the levels of specific bile acids (DCA, LCA, CDCA, UDCA) were lower, while amino acids were elevated, indicating a systemic metabolic disruption. These fecal metabolites also showed significant correlations with the dysbiotic gut microbiota.

Conclusions: The proliferating opportunistic pathogens are strongly associated with the accumulation of harmful metabolites in the circulation, including specific bile acids and amino acid derivatives. These findings suggest that specific intestinal bacteria contribute directly to the progression of ALC by regulating host bile acid and amino acid metabolism, highlighting the gut-liver axis as a critical therapeutic target.

Keyword(s) :

1640 - The β hydroxybutyrylation modification of ACSL4 inhibits the growth of hepatocellular carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma is one of the main causes of cancer-related deaths. Lysine β -hydroxybutyrylation (Kbhb) is a post-translational modification induced by ketone body metabolites, particularly β -hydroxybutyric acid (BHB). Kbhb plays a crucial role in multiple human

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diseases by regulating multiple biological processes, including gene transcription, enzyme activity, and protein stability. However, the role of Kbh**b** in hepatocellular carcinoma has not yet been fully elucidated.

Materials and **Methods:** Proteomic quantification of lysine β -hydroxybutyrylation was conducted on 16 pairs of human hepatocellular carcinoma tissues and adjacent tissues by mass spectrometry. The key Kbh**b** sites affecting β -hydroxybutyrylation of ACSL4 were analyzed and identified through mass spectrometry, immunoprecipitation (IP), amino acid site mutation, and site-specific antibody construction methods. The enzyme that catalyzes the addition of BHB to ACSL4 Lys702 was determined. The effect of K702- β -hydroxybutyrylated ACSL4 on tumor growth was evaluated in vitro.

Results:

28618 lysine β -hydroxybutyrylation (Kbh**b**) sites in 6621 proteins were identified, among which 26761 sites in 6001 proteins were quantified. Comparing cancerous and adjacent non-cancerous tissues, numerous differentially expressed proteins and Kbh**b** sites were observed, including 2159 up-regulated sites and 586 down-regulated sites. Among them, the ACSL4 protein K150, K231, K383, K576, K587, K702 sites enriched in the fatty acid degradation pathway ranked among the top down-regulated sites in hepatocellular carcinoma. Mutating them into Kbh**b**-deleted mutation (arginine, R) or Kbh**b**-mimicking mutation (glutamine, Q) revealed that K702R significantly reduced the β -hydroxybutyrylation level of ACSL4 and its protein expression compared with wild-type ACSL4, while K702Q increased these indicators. IP assays showed the P300 over-expression led to an increase in the Kbh**b** levels of ACSL4 and Lys702, whereas overexpression of CBP or KAT8 did not significantly alter these levels. Furthermore, ACSL4 K702Q markedly suppressed the proliferation of HepG2 and Huh7 cells compared with wild-type ACSL4. Interestingly, K702R counteracted the inhibitory effect of BHB on the growth of liver cancer cells.

Conclusions:

Our data established the protein lysine β -hydroxybutyrylation landscape of hepatocellular carcinoma and identified that the Lys702 β -hydroxybutyrylation regulated the expression of ACSL4 protein. In addition, P300 mediated the β -hydroxybutyrylation of ACSL4 to inhibit the growth of hepatocellular carcinoma. This study has unraveled a novel mechanism regulating ACSL4, providing potential targets and strategies for the treatment of

hepatocellular

carcinoma.

Keyword(s) : β -hydroxybutyrylation, ACSL4, hepatocellular carcinoma, post-translational modification

1642 - Heme oxygenase 1 inhibiting mitochondrial apoptosis in metabolic associated fatty liver disease

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Background:

Heme oxygenase-1 (HO-1), a key enzyme in antioxidant defense, protects against chronic liver inflammation and fibrosis. Endoplasmic reticulum (ER) stress-mediated necroptosis is known to contribute to hepatic injury. Hence, we investigated the function of HO-1 in the development of MASH and the underlying mechanisms involving endoplasmic reticulum (ER) stress-mediated necroptosis.

Materials and **Methods:** Mice fed the methionine- and choline-deficient (MCD) diet for 4 weeks and cultured human hepatocytes treated with free fatty acids (FFAs) for 24 hours were utilized as in vivo and in vitro MASH models, respectively, pharmacological interventions and genetic modifications were employed to up- or downregulate HO-1.

Results:

MCD-fed mice developed severe hepatosteatosis, hepatocyte ballooning, focal necrosis, and inflammatory cell infiltration, along with swollen or disrupted rough ER and elevated biomarkers of ER stress and necroptosis in the liver. Administration of hemin, a HO-1 inducer, in MCD-fed mice upregulated HO-1 in the liver, downregulated ER stress and necroptosis, ameliorated steatohepatitis, and decreased serum ALT and AST. Meanwhile, the HO-1 inhibitor ZnPP showed the opposite effects. In cultured human hepatocytes, HO-1 overexpression reduced while HO-1 silencing exacerbated FFAs-induced ER stress and downstream necroptosis biomarker expression and steatohepatitis injury. In addition, downregulating ER stress with an ER stress suppressor inhibited hepatocellular necroptosis. Mechanistically, HO-1 suppressed ER stress by downregulating GRP78. Finally, elevated HO-1 levels were detected in the liver and serum of MASH patients.

Conclusions:

HO-1 protects against the development of MASH by inhibiting ER stress-mediated hepatocellular necroptosis. HO-1 may serve as a potential therapeutic

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target for MASH.

Keyword(s) : Heme oxygenase-1, Non-alcoholic steatohepatitis, Endoplasmic reticulum stress, Necroptosis

1646 - Mapping the Global Network of Viral Hepatitis A B C and E Unveiling Disease Specific Epidemic Architectures through Complex Network Analysis

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Background / Hypothesis: The global burden of viral hepatitis remains a formidable public health challenge, with HBV and HCV alone accounting for 1.3 million deaths annually. Traditional surveillance, reliant on national reports and regional aggregates, fails to capture the complex interconnectedness driving these epidemics. This study employs complex network theory to uncover the fundamental architectural patterns governing the global persistence and diffusion of Hepatitis A, B, C, and E.

Materials and Methods: We constructed static and dynamic networks using Global Burden of Disease data from 1990-2021. Static networks comprised high-incidence countries (top 50%) as nodes, connected by edges determined as a high Spearman correlation coefficient (>0.9) in incidence rates. Dynamic networks employed 10-year sliding windows with 1-year steps based on this foundation to capture temporal evolution. Key metrics were calculated to quantify structural characteristics: density (measuring overall connection tightness), efficiency (reflecting global transmission capacity), clustering coefficient (indicating local interconnection strength) and modularity (measuring the degree of network division into separate communities). Degree centrality was employed to identify network hub nations.

Results: We identified distinct topological architectures for hepatitis subtype networks. Hepatitis B exhibited a dense, integrated network (density=0.75, efficiency=0.90) showing significant increases in connectivity (density from 0.372 to 1.000, $ptrend<10^{-7}$) alongside modularity decline (from 0.132 to 0.000,

$ptrend<10^{-6}$), forming a single connected component with emerging community structures. In parallel, hepatitis A demonstrated high connectivity (efficiency=0.91) with strong local clustering (clustering coefficient=0.99), yet displayed progressive fragmentation despite a stable low modularity (0.01). In stark contrast, hepatitis C presented a sparse and fragmented architecture (density=0.22, efficiency=0.55) with the highest modularity (0.22) and five connected components (Figure 1), showing accelerating structural polarization despite gradual density increases (from 0.264 to 0.433, $ptrend<10^{-4}$). Hepatitis E maintained a stable hybrid structure (density=0.37, efficiency=0.93) with increasing modularity (from 0.056 to 0.119, $ptrend<10^{-5}$). Analysis of degree centrality identified Comoros as a critical hub consistently maintaining strong correlations with 40-91 high-epidemic countries across all four diseases networks, while Equatorial Guinea, Eritrea, Benin, Sao Tome and Principe and Lao People's Democratic Republic emerged as pivotal hubs across three different disease networks.

Conclusions: This study reveals disease-specific topological architectures that demand distinct strategies for achieving global elimination goals. For highly integrated networks like HBV's, we recommend establishing coordinated sentinel systems in hub nations to monitor progress and elimination barriers. For fragmented networks like HCV's, the identified hub nations provide critical insights into cluster-specific topological patterns, enabling targeted module-level interventions.

Keyword(s) : viral hepatitis, complex network analysis, epidemic topology, public health, global health

1647 - Antiviral Therapy in Patients with HBV Infection in the HBeAg Negative Indeterminate Phase

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Background / Hypothesis: Emerging data suggest that a substantial proportion of patients with chronic hepatitis B (CHB) do not fall into any of the defined phases and are considered indeterminate. Current practice guidelines do not recommend antiviral therapy for CHB patients in the

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indeterminate phase. However, these CHB patients have a significantly high risk of hepatocellular carcinoma. We aimed to investigate liver pathological changes and assess the effects of antiviral treatment on CHB patients in the HBeAg-negative indeterminate phase.

Materials and Methods: We retrospectively analyzed the outcomes of antiviral therapy for CHB patients in the HBeAg-negative indeterminate phase who underwent liver biopsy and were treated with nucleoside/nucleotide analogues (NAs) for up to 96 weeks compared with HBeAg-negative chronic infection. Phases of natural history were determined according to the AASLD practice guidance.

Results:

A total of 392 HBeAg-negative patients were enrolled in this study. Of these, 192 were in the defined phase and 200 were in the indeterminate phase. Approximately 75% of CHB patients in the indeterminate phase showed hepatic inflammatory necrosis $\geq$ G2 (significant inflammation) and/or fibrosis $\geq$ S2 (significant fibrosis). The rate of undetectable HBV DNA in chronic infection and indeterminate phase groups totally was ~91.1%, ~99.5%, ~100%, 100% or 96.5%, 98.5%, 99.5% 99.5% at week 12, 24, 48 or 96, respectively. The ALT normalization rates were 55.7%, 74.2%, 80.7%, and 83.9% in the defined phase group, contrasted with 37.5%, 50.0%, 64.3%, and 69.6% in the indeterminate phase group at 12, 24, 48, and 96 weeks, respectively. With prolonged antiviral therapy, surface antigen levels gradually decreased in both groups; however, no patient had loss of hepatitis B surface antigen.

Conclusions:

Majority of CHB patients in the indeterminate phase had significant liver histological changes. Antiviral therapy can effectively inhibit HBV replication in these CHB patients regardless of ALT levels, although hepatitis B surface antigen loss were low.

Keyword(s) : CHB; Antiviral Therapy; Indeterminate Phase

1649 - Platelet Albumin Bilirubin (PALBI) Score A non invasive Predictor of Esophageal Varices in Patients with Liver Cirrhosis Compared to Child Turcotte Pugh (CTP) Score

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Background

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Hypothesis:

Esophageal varices are serious consequences of portal hypertension in patients of liver cirrhosis, carrying a high risk of fatal bleeding. Early and non-invasive identification of patients at risk of OV is essential for initiating prophylactic treatment. The established Child-Turcotte-Pugh (CTP) score while widely used for assessing prognosis, relies on subjective parameters (ascites, encephalopathy). Consequently, objective, laboratory-based scores such as the Albumin-Bilirubin (ALBI) score and its derivative, the Platelet-Albumin-Bilirubin (PALBI) Score (incorporating platelet count, a marker of portal hypertension), have emerged as promising non-invasive alternatives for risk stratification and prediction of clinical outcomes.

Materials

and

Methods:

This cross-sectional study was done in the Department of Hepatology, Dhaka Medical College Hospital (DMCH), Bangladesh from January to August 2025. Total 80 patients were included. The PALBI score (calculated as $S.A \times PLT$) and the standard ALBI score were determined. The CTP classification was also recorded. Esophageal varices were expected as "present" when the OV score was ≥ 1 . Diagnostic accuracy was assessed using Receiver Operating Characteristic ROC curve analysis for the PALBI score and the CTP score, and sensitivity and specificity were calculated for selected cutoffs.

Results:

In a total 80 patients, 77 (96.2%) had esophageal varices (OV ≥ 1). The mean ALBI score was -1.788 ± 0.759 and the mean PALBI score was $515.7 \pm 310.8 \times 10^3$. The PALBI score was found to have excellent diagnostic ability to predict patients with and without esophageal varices AUC = 0.926. In comparison, the CTP score showed perfect diagnostic ability AUC = 1.000 in this cohort, while the ALBI score had good ability AUC = 0.853. Furthermore, the PALBI score showed a moderate, statistically significant inverse correlation with the CTP score ($r = -0.486$, $p < 0.001$), confirming its validity as a marker of liver function severity. The sensitivity and specificity for PALBI $\leq 500 \times 10^3$, PALBI $\leq 650 \times 10^3$ were 59.7% and 100.0%, and 72.7% and 100.0%, respectively.

Conclusions:

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PALBI score was found to have excellent ability to diagnose patients with and without esophageal varices, performing significantly better than the ALBI score alone. Although the CTP score achieved perfect discrimination in this specific cohort, PALBI is a simple, objective, and reproducible noninvasive marker that correlates strongly with disease severity and provides high accuracy for risk stratification.

Keyword(s) : PALBI Score, Esophageal varices, CTP Score, Liver Cirrhosis.

1652 - Investigating the Role of Gut Microbiota Derived Valine in Treating MAFLD through PPAR Signaling

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Background / Hypothesis: With its complex pathogenesis, MAFLD has placed gut microbiota-centered therapies, informed by the gut-liver axis, in the spotlight.

Materials and Methods: An MAFLD cell model and high-fat diet (HFD)-fed mice were treated with L-Valine (LV). Hepatic steatosis, insulin resistance, and liver histopathology were assessed. Key proteins (PPAR α , ZO-1) were analyzed by Western blot.

Results: ① LV levels are inversely associated with the severity of MAFLD and LV ameliorated MAFLD's a cellular model (Fig 1-2).

② LV intervention significantly altered body fat content and liver metabolism, and improved HFD-induced insulin resistance in mice (Fig 3-5).

③ LV treatment alleviated intracellular lipid accumulation and improved liver histopathology of mice (Fig 6-7).

④ LV ameliorated MAFLD by activating the PPAR α pathway (Fig 8).

⑤ LV intervention partially reversed the HFD-induced downregulation of ZO-1 protein expression (Fig 9).

Conclusions: Our findings demonstrate that L-Valine

ameliorates MAFLD by activating the PPAR α signaling pathway and Strengthening the intestinal barrier, leading to reduced hepatic steatosis and improved systemic metabolism.

Keyword(s) : L-Valine; MAFLD / MASLD; PPAR α ; gut microbial metabolite

1653 - Efficacy of sofosbuvir velpatasvir in patients with hepatitis C virus genotype 3 infection

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Background: With the widespread clinical application of pangenotypic direct-acting antiviral agents, the cure rate for hepatitis C has significantly improved. However, treating hepatitis C virus (HCV) genotype (GT) 3 remains a major clinical challenge, and real-world data for this genotype in Eastern China remain limited. This study aimed to evaluate the efficacy of sofosbuvir/velpatasvir (SOF/VEL) in achieving a sustained virological response at 12 weeks post-treatment (SVR12) and its impact on related clinical parameters in patients with HCV GT 3 infection.

Methods: A total of 223 patients with HCV GT 3 infection who received SOF/VEL, with or without ribavirin (RBV), at Ningbo Second Hospital and Ma'anshan Fourth People's Hospital from March 2019 to March 2025 were retrospectively enrolled. Clinical data were systematically collected to analyze baseline characteristics, diagnostic and treatment details, and comorbidities. The primary endpoint was the overall SVR12. The prevalence of comorbidities in this population and the impact of SOF/VEL on clinical parameters were also evaluated.

Results: Among the 223 enrolled patients, GT 3a and 3b accounted for 17% (37/223) and 83% (186/223), respectively. Those with a history of drug use, co-infection with HBV, HIV, and syphilis accounted for

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44.84% (100/223), 3.59% (8/223), 4.04% (9/223), and 13.45% (30/223), respectively. Concurrent fatty liver disease, liver cirrhosis, and hepatocellular carcinoma were present in 34 (15.25%), 63 (28.25%), and 7 (3.14%) patients, respectively. Efficacy analysis showed an overall SVR12 rate of 96%, with rate of 92% in HCV GT 3a and 98% in HCV GT 3b. Additionally, the normalization rates of alanine transaminase (ALT) (91.03% vs 18.39%) and aspartate aminotransferase (AST) (89.69% vs 24.22%) were significantly higher at the end of treatment compared with baseline (all $P < 0.001$). At 12 weeks post-treatment, significant improvements from baseline were observed in the levels of total bilirubin ($Z = -4.170$), ALT ($Z = -8.986$), AST ($Z = -8.563$), white blood cell count ($Z = -3.044$), platelet count ($Z = -4.943$), APRI ($Z = -7.454$) and FIB-4 ($Z = -6.027$) (all $P < 0.01$).

Conclusions:The SOF/VEL regimen achieved a high sustained virologic response rate and significantly improved both liver function and fibrosis markers in patients with HCV GT 3 infection.

Keyword(s) : Hepatitis C virus, Sofosbuvir-velpatasvir, Cirrhosis, Genotype, Treatment

1656 - Analysis on the differences of clinicopathological characteristics in drug induced liver injury patients with positive or negative autoantibodies and different clinical types

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Background:To investigate the differences in clinical and pathological characteristics among patients with drug-induced liver injury (DILI) by autoantibody status and R value-based clinical types to inform clinical practice.

Methods:This study included 138 patients hospitalized from January 2016 to July 2023 with biopsy-confirmed DILI. Patients were grouped by autoantibody results (positive/negative) liver histopathological changes (<

G2 and $\geq$ G2, < G3 and $\geq$ G3, < G4 and G4, < S2 and $\geq$ S2, < S3 and $\geq$ S3, < S4 and S4, mild pathological injury [$< G2 + < S2$] and significant pathological injury [$\geq G2$ and/or $\geq S2$] and R values for DILI classification ($R \geq 5$: hepatocellular injury type; $2 < R < 5$: mixed type; $R \leq 2$: cholestatic type), with clinicopathological characteristics compared.

Results:Of the enrolled patients, 79 were autoantibody-negative and 59 were autoantibody-positive, including 59 cases (100.00%) positive for anti-nuclear antibody and 8 cases (13.56%) positive for anti-mitochondrial antibody. Traditional Chinese Medicine (including single herbs, herbal dietary supplements, or compound preparations) accounted for the highest proportion of medications causing DILI (55/138, 39.86%). The autoantibody-positive group exhibited significantly higher levels of serum globulin (GLB) ($t = 2.983$), alkaline phosphatase ($Z = 2.042$), and adenosine deaminase (ADA) ($Z = 2.582$), and a lower albumin (ALB)/GLB ratio ($t = 2.211$) compared to the negative group (all $P < 0.05$). The autoantibody-positive group had a higher mean Ridit value for liver inflammation activity grade (G) ($u = 2.302$, $P = 0.021$) and a higher proportion of $\geq G3$ and G4 ($\chi^2 = 4.471, 6.478$; $P = 0.034, 0.011$). Among the $R \geq 5$, $2 < R < 5$, and $R \leq 2$ groups, significant differences were observed in serum total bilirubin (TBil) ($H = 11.166$), alanine aminotransferase (ALT) ($H = 109.01$), aspartate aminotransferase (AST) ($H = 75.663$), ALT/AST ratio ($H = 23.096$), adenosine deaminase (ADA) ($H = 34.013$), and gamma-glutamyl transferase (GGT) ($H = 9.831$) (all $P < 0.01$). The $2 < R < 5$ group had the highest mean Ridit value for G and the highest proportion of $\geq G3$ grades, with significant differences among the three groups ($\chi^2 = 7.385, 7.163$; all $P < 0.05$). Independent risk factors for G severity were ALB, GGT, and ALT/AST; for advanced fibrosis stage (S) were ALT/AST, platelet count, and ALB; and for combined G/S severity were GGT, ALB, and ALT/AST.

Conclusions:The clinical and pathological features of DILI vary across different disease states. Autoantibody-positive DILI and mixed-type DILI ($2 < R < 5$) are associated with more severe liver inflammation grades.

Keyword(s) : Drug-induced liver injury, Autoantibody, Clinical types, Pathology, Clinical characteristics

1661 - Clinical Characteristics of Autoimmune Hepatitis in Patients with Hepatitis B-D Coinfection

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A	Case	Series
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Background / Hypothesis:

Patients infected with hepatitis B and D coinfection are frequently seen in clinical practice. However, their association with autoimmune hepatitis (AIH) is an unusual phenomenon

Materials and Methods:
We through this case series highlighted six patients having this uncommon association. These patients had presented to our outpatients' department and were thoroughly investigated. Their baseline laboratory parameters, serology, imaging and endoscopic findings were recorded, following which liver parenchymal biopsies were done for most of them.

Results:
Most of them were managed with tablet entecavir along with azathioprine and were kept on close follow-up for any possible deterioration.

Conclusions:
Our case series highlights this rare association, which should be kept in mind in patients with HBV and HDV coinfection presenting with deranged LFTs.

Keyword(s) : Hepatitis B; Hepatitis D; AIH
1662 - Socioeconomic Disparities and Potential Impact of Delayed Presentation on the Burden of Portal Hypertension in a Bangladeshi Tertiary Care Setting
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Background / Hypothesis:

Portal hypertension is a major cause of morbidity in South Asia, yet the influence of socioeconomic disparities and delayed presentation on disease severity remains insufficiently defined. We hypothesized that lower socioeconomic status contributes to advanced-stage cirrhosis, more severe variceal burden, and higher

rates of hepatocellular carcinoma at presentation.

Materials and Methods:

A cross-sectional observational study was conducted in a tertiary care hospital from December 2024 to June 2025. A total of 118 adults diagnosed with portal hypertension based on endoscopic, radiologic, and biochemical criteria were enrolled consecutively. Socioeconomic status was defined using monthly household income and categorized into lower, middle, and higher classes according to standard income-based thresholds. Variceal grades were stratified across socioeconomic groups. Cirrhosis was classified as compensated or decompensated. Categorical variables were compared using chi-square or Fisher's exact test, with significance set at p<0.05.

Results:
The cohort had a mean age of 46.85 ± 13.90 years; 85.6% were males. Most participants belonged to the middle (54.2%) or lower (36.4%) socioeconomic classes. Hepatitis B was the predominant etiology (65.3%). Cirrhosis was present in 94.9% of patients, and 85.6% presented with decompensated disease. Among decompensated cirrhosis patients (n=101), 39 (38.6%) were lower class, 57 (56.4%) middle class, and 5 (5%) higher class, showing a significant association between socioeconomic status and cirrhosis stage ($\chi^2=10.30$, p=0.0058). Variceal severity demonstrated a strong socioeconomic gradient. Grade III varices occurred in 60.5% of lower-class, 71.9% of middle-class, and only 18.1% of higher-class patients, a significant association ($\chi^2=16.22$, p=0.0027). Overall, 77.1% had moderate to large varices. Hepatocellular carcinoma was detected in 24 patients (20.3%). HCC showed significant socioeconomic variation: 13 lower-class, 7 middle-class, and 4 higher-class patients ($\chi^2=7.83$, p=0.0199), indicating disproportionate HCC burden in lower-income groups. Other complications included splenomegaly (82%), anemia (70%), ascites (66%), hematemesis (59%), and hepatic encephalopathy (37%).

Conclusions:
This study highlights that portal hypertension in Bangladesh is predominantly driven by chronic hepatitis B infection, with a striking male predominance and a high rate of decompensated cirrhosis at presentation. Most patients present with advanced disease and complications reflecting delays in diagnosis. Lower

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socioeconomic status is significantly associated with decompensated cirrhosis, severe variceal grades, and higher rates of hepatocellular carcinoma, reflecting major disparities in early detection and access to care. Targeted community screening and strengthened referral pathways are essential to reduce the burden of advanced portal hypertension.

Keyword(s) : portal hypertension, socioeconomic status, delayed presentation, variceal severity, hepatocellular carcinoma, cirrhosis, Bangladesh

1664 - Matrix Stiffness Suppresses Hepatitis B Virus Replication via YAP Mediated Intrinsic Antiviral Signaling

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Background / Hypothesis: Elucidating the intrinsic host mechanisms that constrain hepatitis B virus (HBV) replication is a fundamental question in hepatology. Independent of its biochemical composition, the extracellular matrix provides potent physical cues that govern cell fate and function. Given the physiological variations in liver tissue stiffness, we hypothesized that biomechanical signals represent a previously unexplored layer of regulation in hepatotropic virus-host interactions.

Materials and Methods: Primary hepatocytes were cultured on tunable hydrogels engineered to mimic distinct mechanical microenvironments. The impact of substrate stiffness on the HBV replication cycle and the associated mechanotransduction signaling pathways was systematically dissected. Gain- and loss-of-function studies for the transcriptional co-activator YAP (Yes-associated protein) were conducted to define its precise role. A targeted functional screen identified YAP-responsive genes with anti-HBV activity. The clinical relevance of this mechanosignaling axis was interrogated through single-cell RNA-sequencing analysis of liver biopsies from male patients with active HBV infection. The therapeutic efficacy of targeting this pathway was validated in male HBV-hydrodynamic injection mouse models using a small-molecule YAP

agonist.

Results:

High matrix stiffness, mimicking a fibrotic mechanical environment, potently suppressed HBV replication in primary hepatocytes. This antiviral state was mechanistically linked to the activation and nuclear translocation of YAP. Ectopic YAP activation was sufficient to suppress HBV transcription and antigen production, whereas its inhibition attenuated the stiffness-mediated antiviral effect. Subsequent screening identified several YAP-target genes that confer robust, cell-intrinsic resistance to HBV. Crucially, single-cell resolution analysis of human liver tissues revealed a significant negative correlation between the cell-autonomous YAP signaling signature and the intracellular load of HBV transcripts. Consistent with these findings, pharmacological potentiation of YAP signaling in vivo effectively curtailed viral replication in a male mouse model of HBV infection.

Conclusions:

This study delineates a novel innate antiviral mechanism wherein the biomechanical property of the liver microenvironment is sensed by hepatocytes through YAP activation to orchestrate a cell-intrinsic defense program against HBV. Our data establish YAP-mediated mechanosignaling as a fundamental host pathway that can be harnessed to suppress HBV. This discovery provides a new conceptual framework for understanding liver-virus interactions and identifies mechanosignaling components as potential targets for developing novel antiviral strategies.

Keyword(s) : Matrix Stiffness, Hepatitis B Virus, YAP, Antiviral Signaling

1672 - Hepatic steatosis in Wilson disease phenotype or metabolic comorbidity

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Background / Hypothesis: Hepatic steatosis (HS) is a common manifestation in Wilson disease (WD), primarily related to mitochondrial dysfunction induced by toxic hepatic copper accumulation, which disrupts lipid metabolism. With increased survival and transition

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of patients into adulthood, cardiometabolic risk factors may additionally contribute to the development of metabolic-associated steatosis. Distinguishing copper-induced steatosis from metabolic steatosis is essential for assessing fibrosis progression risk and optimizing patient management. The aim of this study was to determine the prevalence and characteristics of HS in patients with WD.

Materials and Methods: A retrospective descriptive study was conducted, including 53 patients diagnosed with WD between 2016–2024. HS evaluation was based on clinical criteria (body mass index, waist circumference), biochemical parameters (glucose, lipid profile), and imaging (ultrasound or computed tomography, FibroScan). DNA sequencing by the Sanger method was performed using the Taq DyeDeoxy Terminator Cycle sequencing kit (Applied Biosystems) with an ABI-Prism 3100 genetic analyzer (Applied Biosystems)

Results: Cohort analysis identified three clinico-imaging subgroups. Group 1 – 33 patients (53% males), mean age 20.2 ± 6.5 years, without clinical-imaging signs of HS, mean liver stiffness 10.1 ± 6.2 kPa, mean steatosis 241 ± 125.5 dB/m ($p > 0.05$). Group 2 – 16 patients (63% males), mean age 25.7 ± 8.1 years, without clinico-biochemical changes but with imaging findings suggestive of HS, mean liver stiffness 11.2 ± 5.6 kPa, mean steatosis 262 ± 165.1 dB/m ($p > 0.05$). Group 3 – 4 patients (100% males), with mean body mass index 28.83 ± 1.83 kg/m², mean waist circumference 105 ± 3.56 cm, mean HDL-cholesterol 0.825 ± 0.28 mmol/L, mean triglycerides 2.10 ± 0.79 mmol/L, mean liver stiffness 14.7 ± 7.1 kPa, and mean steatosis 285.5 ± 154.1 dB/m ($p < 0.05$). Genetic variants identified in Group 3 included: p.H1069Q, p.G1341D, p.Phe764=, p.Met769Hisfs*26.

Conclusions: HS is present in approximately one-third of patients with WD, and nearly 8% exhibit an associated metabolic component. The mixed phenotype with metabolic involvement appears correlated with age and the accumulation of cardiometabolic risk factors. Systematic integration of metabolic assessment in WD monitoring may improve risk stratification and personalized therapeutic management, with potential benefits for fibrosis progression and long-term outcomes.

Keyword(s) : Wilson disease, hepatic steatosis, cardiometabolic risk

1673 - Towards hepatitis B elimination in Ghana vaccination coverage and its predictors among informal sector workers in Kejetia Kumasi Ghana

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Background / **Hypothesis:** Hepatitis B virus (HBV) infection remains a major public health concern in Ghana, where prevalence is high despite the availability of an effective vaccine. Informal sector workers represent a large proportion of the national workforce but have limited access to preventive health services. This study assessed hepatitis B vaccination coverage and its predictors among informal sector workers in Kejetia market, Kumasi, Ghana.

Materials and **Methods:** A cross-sectional survey was conducted among 809 market workers selected using stratified random sampling across different occupational groups. Data were collected using structured interviewer-administered questionnaires covering sociodemographic characteristics, HBV awareness and knowledge, and vaccination history. Vaccination uptake was categorized as at least one dose (≥ 1) and full

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coverage (≥ 3 doses). Logistic regression analyses were used to identify predictors of vaccination uptake.

Results:

Overall, only 16.9% had completed the three-dose schedule, though 31.6% of participants reported having received at least one dose of HBV vaccine. While 88.8% of respondents had heard of HBV infection, 76.5% reported very little or no knowledge about the disease, and 49.7% did not know its routes of transmission. Vaccination uptake was significantly higher among those who had received HBV-related health education (62.7%) compared to those who had not (15.3%, $p < 0.001$). In the multivariate logistic regression model, ≤ 30 years (aOR = 2.209, 95% CI [1.140-4.282], $p = 0.019$), secondary education (aOR = 2.426, 95% CI [1.220-4.824], $p = 0.012$), and tertiary education (aOR = 4.796, 95% CI [2.121-10.845], $p < 0.045$) were the independent predictors of vaccination status.

Conclusions:

HBV vaccination coverage among informal sector workers in Kejetia Market is alarmingly low despite high general awareness of the infection. Sociodemographic factors and receipt of HBV-related health education strongly influenced uptake. These findings underscore the urgent need for targeted health education and subsidized vaccination programs tailored to informal sector workers in Ghana to achieve national HBV elimination goals.

Keyword(s) : Hepatitis B infection, Ghana, Market, Vaccination, Health Education, Public Health

1676 - Predictive Factors of Tyrosine Kinase Inhibitor Response in Patients with Advanced Hepatocellular Carcinoma

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is the most common primary liver malignancy and carries a poor prognosis in advanced stages. Tyrosine kinase inhibitors (TKIs) are standard systemic therapies, but patient responses vary. Identifying predictive factors may optimize patient selection and individualized treatment. We hypothesized that hepatic function, tumor characteristics, and baseline laboratory

parameters influence TKI response and survival.

Materials and Methods: This retrospective study included 103 advanced HCC patients treated with sorafenib or regorafenib between 2009 and 2025. Demographics, clinical scores (ECOG, Child-Pugh, MELD, ALBI), laboratory markers, imaging findings, toxicity, treatment response (mRECIST), progression-free survival, and overall survival were analyzed. Kaplan-Meier and Cox regression were used.

Results: Among patients treated with sorafenib ≥ 3 months: partial response 13.1%, stable disease 45.9%, progression 41%. Significant OS predictors: sex, Child-Pugh stage, tumor involvement pattern, extrahepatic metastasis, portal vein thrombosis, BCLC stage. Independent predictors of shorter progression-free survival: MELD-Na score, ECOG performance, Child-Pugh C, diffuse tumor involvement, portal vein thrombosis. ALBI score was protective. Hand-foot syndrome was the most common toxicity requiring dose reduction.

Conclusions: Liver reserve (MELD-Na, ALBI), bilirubin levels, performance status, tumor pattern, and portal vein thrombosis strongly influence TKI response and survival. These factors support more accurate patient selection and individualized treatment strategies.

Keyword(s) : Hepatocellular carcinoma; tyrosine kinase inhibitor; sorafenib; regorafenib; predictive factors; survival

1677 - Establishment of the TBRI Biobank for Liver Diseases Advancing Research Translation and Precision Medicine in Hepatology

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Liver diseases including viral hepatitis, nonalcoholic fatty liver disease, autoimmune hepatitis, metabolic disorders, and hepatocellular carcinoma (HCC) remain major global health challenges. In Egypt, the burden is particularly high, with the world's highest prevalence of hepatitis C virus (HCV) and an increasing incidence of HCC. Despite the success of direct-acting antivirals (DAAs), many patients continue to experience serious complications, underscoring the urgent need for early diagnostic and prognostic tools.

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To address this need, establishing an integrated infrastructure that links high-quality clinical data with advanced molecular and genetic technologies is essential. Biobanks play a pivotal role in this effort by enabling the systematic collection, processing, and preservation of human biospecimens that support precision (personalized) medicine. Disease-specific biobanks accelerate biomarker discovery, facilitate targeted drug development, and enhance research quality.

This work focuses on establishing the Theodor Bilharz Research Institute (TBRI) Biobank for liver diseases, which stores blood, serum, buffy coat, and DNA samples from adult patients. To date, 1,000 blood samples have been collected, forming a robust resource for future molecular and clinical investigations. The biobank provides researchers with integrated socio-demographic data, longitudinal clinical records, and high-quality biospecimens to support both retrospective and prospective studies. Through this infrastructure, the TBRI Biobank aims to advance biomarker discovery, foster innovation in hepatology, and ultimately enhance public health and personalized patient care.

Keyword(s) : Liver Diseases- Biobank- Personalized Medicine

1679 - PASSIVE TOBACCO SMOKE INDUCED CHANGES IN THE EXPRESSION OF IRON METABOLISM GENES IN MOUSE PUPS

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Background / Hypothesis:
Tobacco smoke has a global impact, particularly on pregnant women and their newborns. Maternal tobacco smoke exposure is associated with different pregnancy complications and outcomes, viz., less development and dysfunction of vital organs and regulatory pathways. This study focused on the impact of the whole gestation

period passive tobacco smoke exposure (PTS) on the heart and liver functions, and some iron regulatory and inflammatory gene expression in neonates. Materials and Methods: Randomly selected PTS-exposed neonates of the pregnant mice were processed by anesthetizing them hypo-thermally/cryothermally, followed by their dissection to excise the heart and liver under a magnifying glass. All the pups have proceeded for morphometric measurements, cardio, hepatic histological, and gene expression analysis. Results:

A significant reduction was observed in all morphometric parameters: viz litter size, birth weight, crown-rump, and heart and liver weight. Furthermore, PTS induced microarchitectural changes in cardiomyocytes with the formation of edema, hemorrhage, lymphocytic infiltration, and cardiomyolysis while liver histology revealed central canal distortion, hemolysis, sinusoidal space, and fused nuclei. Collagen, glycogen, and iron deposition were also evident in the Masson trichrome, Periodic acid Schiff, and Prussian blue stains. The gene expression analysis of Dmt-1, TfR-1, Hamp, and HO-1 showed upregulation, while some genes, Hfe, Fpn-1, Ferritin H, and HJV showed downregulation. Furthermore, the mRNA expression of TNF- α was upregulated, and the IL-6 expression was downregulated in neonates of PTS-exposed mice. The current study has highlighted that PTS exposure to neonates during gestation can induce changes in hepatocytes and regulatory pathways. Conclusions:

Therefore, such long-term exposure to passive tobacco smoke must be checked for healthy pregnancy outcomes, and implementation of preventable, controllable measures, the revision of legislation, and safety guidelines must be followed to reduce the frequency of congenital defects.

Keyword(s) : Passive tobacco smoke, Neonates, Iron gene expression, Heart, Liver, Toxicology

1690 - Off Therapy Outcomes After Long Term Nucleos(t)ide Analogue Treatment in HBeAg Negative Chronic Hepatitis B

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Background / Hypothesis:

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Long-term nucleos(t)ide analogue (NA) therapy effectively suppresses HBV replication and reduces the risk of cirrhosis, hepatic decompensation, and liver-related mortality in patients with chronic hepatitis B(CHB)(1,2). However, HBsAg seroclearance remains rare during continuous therapy, with annual rates below 0.5%(3,4). Achieving HBsAg loss is considered a key step toward functional cure. APASL guidelines allow NA discontinuation after at least two years of undetectable HBV DNA demonstrated on three measurements six months apart (5,6). The optimal timing for stopping NAs in HBeAg-negative patients remains debated. Increasing evidence suggests that NA withdrawal may enhance HBsAg loss in carefully selected patients(4,7–13). This study evaluated virological relapse, biochemical relapse, sustained off-therapy response, and HBsAg seroclearance after NA cessation in HBeAg-negative CHB patients.

Materials and Methods:

This retrospective study included CHB patients treated with NAs for at least four years. Eligible patients were HBeAg-negative, had undetectable HBV DNA, and normal ALT values at discontinuation. All patients were followed closely with serial laboratory assessments. Virological relapse, biochemical relapse, sustained response (HBV DNA <2000 IU/mL and normal ALT), and HBsAg clearance were recorded.

Results:

Thirty patients were included, with follow-up ranging from nine months to four years. All patients experienced virological relapse shortly after NA discontinuation, while 11 patients(36%) developed biochemical relapse. A sustained off-therapy response (HBV DNA below 2000 IU/L, normal ALT) was achieved in 18 patients (%60). Among these, 4 patients (13%) cleared HBsAg (in the 12th, 18th, 40th and 48th months). 4-year rate of HBsAg clearance of 36% among sustained biochemical/virological responders who stopped NAs treatment. 2 patients(6,6%) with virological and/or biochemical relapses were retreated with oral antiviral agents, without evidence of liver decompensation; HBsAg was not lost in either case. The increase in ALT level occurred either concomitantly with the reappearance of HBV DNA or 1–2 months later. The duration of biochemical relapses was transient in most of the patients. The limitations of this study are the small number of patients and short follow-ups, which increases

heterogeneity. In this study, HbsAg seroclearance improved over a period of 12 to 48 months. Therefore, if patients were followed for longer periods, the HbsAg seroclearance rate would be expected to be over 13%.

Conclusions:

In HBeAg-negative CHB patients, NA discontinuation after four to five years appears safe when accompanied by close monitoring. Sixty percent of patients maintained a sustained response, and 13% achieved HBsAg loss. These findings support the potential role of treatment withdrawal in promoting functional cure in selected patients.

Keyword(s) : HBsAg seroclearance after NA cessation , virological relapse, biochemical relapse, sustained off-therapy response

1694 - Non Invasive Evaluation of Fibrosis Regression in HBV and HCV A Real World Comparative Analysis

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Background

Chronic hepatitis B (CHB) and chronic hepatitis C (CHC) both lead to progressive liver fibrosis, yet fibrosis remodeling may differ according to viral etiology. FIB-4 and APRI are widely used to evaluate fibrosis evolution, but comparative data on fibrosis regression dynamics between CHB and CHC remain scarce. This study aimed to compare fibrosis regression in CHB and CHC using non-invasive biological scores (FIB-4 and APRI) before and after antiviral management.

Materials and Methods:

This retrospective, descriptive, and analytical study was conducted over an 8-year period in a gastroenterology department. FIB-4 and APRI were calculated at baseline and after 48 weeks of follow-up. Normality was assessed using Shapiro–Wilk test. Paired comparisons were performed using the Wilcoxon signed-rank test, and intergroup comparisons of Δ FIB-4 and Δ APRI used the Mann–Whitney U test. Fibrosis evolution was categorized as regressive, stable, or progressive according to predefined score changes.

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Results:

The study included 125 CHB and 165 CHC patients. CHB patients were younger than CHC patients (mean 46.9 vs median 60 years). In CHB, fibrosis stages were as follows: F0 in 14(11.2%) patients, F1 in 76(60.8%), F2 in 22(17.6%), F3 in 2(1.6%), and F4 in 11(8.8%). In contrast, CHC patients presented with more advanced fibrosis, with F1 in 37 patients (29.4%), F2 in 31(24.6%), F3 in 24 (19%), and F4 in 32(25.4%). Baseline fibrosis scores were lower in CHB than CHC (median FIB-4: 1.65 [0.82–2.48] vs 3.19 [1.48–4.35]; APRI: 0.57[0.27–1.11] vs 1.00[0.44–1.93]). In CHB, FIB-4 decreased to 0.90[0.63–1.40] and APRI to 0.25[0.19–0.38]; median Δ FIB-4 was $-0.31[-1.21; 0.11]$ and Δ APRI $-0.18[-0.58; 0.01]$ (both $p < 0.01$). In CHC, FIB-4 decreased to 1.36[0.91–2.16] and APRI to 0.37[0.25–0.51]; median Δ FIB-4 was $-1.26[-2.97; -0.26]$ and Δ APRI $-0.54[-1.52; -0.14]$ (both $p < 0.001$). The decline in fibrosis scores was significantly greater in CHC than CHB (Δ FIB-4 $p < 0.001$; Δ APRI $p < 0.001$). Categorical evolution showed fibrosis regression in 64% of CHB vs 86% of CHC patients, with progression observed in 17% vs 14%, respectively.

Conclusions:

Both CHB and CHC cohorts demonstrated significant fibrosis regression assessed by non-invasive biological markers, but the magnitude of improvement was markedly greater in CHC than CHB. This difference may partly reflect the fact that CHC patients in our cohort were diagnosed at a more advanced stage, with higher baseline fibrosis levels, allowing a larger margin for measurable regression after viral eradication. These findings underline the value of FIB-4 and APRI in monitoring fibrosis evolution and highlight distinct remodeling profiles depending on viral etiology, with more pronounced regression after HCV cure than HBV suppression.

Keyword(s) : Hepatitis C CHC CHB Fibrosis Non invasive test FIB4 Cirrhosis

1698 - Etiological Predictors of Portal Vein Thrombosis in Patients with Porto Sinusoidal Vascular Disease

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Background

Hypothesis:

Porto-sinusoidal vascular disease (PSVD) is an important cause of non-cirrhotic portal hypertension. Portal vein thrombosis (PVT) is a frequent and clinically significant complication in PSVD, but its determinants remain unclear. Whether PVT arises primarily from portal hypertension-related hemodynamic changes or from underlying prothrombotic conditions is debated. This study aimed to identify clinical, biological, and etiological factors associated with PVT in PSVD.

Materials and Methods:

We conducted a single-center, retrospective study of histologically confirmed PSVD patients between January 2018 and April 2025. Patients lost to follow-up or with revised diagnoses were excluded. The cohort was stratified by the presence or absence of PVT. Statistical analyses were performed using Jamovi software; $p < 0.05$ was considered significant.

Results:

Fifty-eight patients were included (median age 42 years; M/F ratio 0.6). PVT was present in 33 patients (56.9%), mostly at initial diagnosis; secondary thrombosis occurred in 5 cases (15.2%). Among PVT cases, 57.6% were complete and 42.4% partial. Univariate analysis showed no association between PVT and age, sex, prothrombin time, collateral venous circulation, portal hypertension decompensation, or endoscopic signs. Interestingly, thrombocytopenia ($p = 0.0033$) and splenomegaly ($p = 0.0023$) were more frequent in patients without PVT. Etiological assessment revealed that all PVT patients had at least one prothrombotic condition, including antiphospholipid syndrome (20.8%), myeloproliferative neoplasms (15%), protein S deficiency (13.2%), protein C deficiency (11.3%), estrogen-progestin contraception (9.4%), pernicious anemia (7.5%), paroxysmal nocturnal hemoglobinuria, Behçet's disease, factor V Leiden mutation, or prior abdominal surgery. In contrast, >70% of patients without PVT had no identifiable prothrombotic factor ($p < 0.0001$).

Conclusions:

In PSVD, PVT is strongly associated with underlying prothrombotic conditions, while patients without thrombosis are predominantly idiopathic. These findings support systematic etiological screening to improve thrombotic risk stratification and guide management.



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Keyword(s) : Porto-sinusoidal vascular disease; Portal vein thrombosis; Non-cirrhotic portal hypertension

1708 - Males are emerging as the primary high risk population for gallbladder and biliary tract cancers

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Background / **Hypothesis:**
Gallbladder and biliary tract carcinoma (GBTC) is a highly lethal malignancy with a considerable global burden. Understanding its sex-specific, age-related, and geographic trends is essential for precision prevention and public health planning. This study assessed the global burden of GBTC from 1990 to 2021, projected trends to 2040, and examined associations with age, sex, geographic location, and the Socio-demographic Index (SDI).

Materials and **Methods:**
All GBTCs were coded as C23-C24.9. Age-standardized incidence rates (ASIRs), mortality rates (ASMRs), disability-adjusted life years (DALYs), and total percentage change were used to quantify the disease burden. Associations between SDI and ASMRs were examined across regions, and temporal trends were projected to 2040 using ARIMA modeling.

Results:
In 2021, 216,768 new GBTC cases and 171,961 deaths occurred, with an ASIR of 2.56 and an ASMR of 2.04 per 100,000. From 1990-2021, global ASIR, ASMR, and DALY rates declined by 11.5%, 24.1%, and 26.2%, respectively, with steeper declines among females. Male incidence surpassed females in 2013 and is projected to remain higher through 2040, particularly among males aged 70-94 years. Furthermore, since 2013, both the incidence and intensity of male predominance increased globally. South Asia showed the largest increase in death percentages, while Latin America and Europe declined, mainly due to reduced female deaths. The highest national ASIRs were observed in Chile.

Conclusions:
Despite global declines, rising male incidence and persistent mortality in high-SDI regions highlight widening disparities. Targeted interventions for older men and high-burden regions are urgently needed to

reduce the future GBTC burden.

Keyword(s) : Gallbladder and biliary tract carcinoma (GBTC); Global burden of disease; incidence; mortality; prediction

1713 - Hepatocyte myb like swirm and mpn domains 1 exacerbates liver fibrosis by stabilizing gelsolin via k29 linked deubiquitination

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Background / **Hypothesis:**
Hepatocyte autophagy acts as a protective brake during liver fibrosis. We found that the deubiquitinase Myb-like SWIRM and MPN domains 1 (MYSM1) is selectively and markedly upregulated in hepatocytes in human cirrhosis and multiple murine fibrosis models. Its precise role in fibrogenesis remains unknown. We hypothesized that MYSM1 suppresses hepatocyte autophagy by deubiquitinating a novel substrate, thereby increasing TGF- β 1 release and driving hepatic stellate cell activation.

Materials and **Methods:**
MYSM1 expression and cellular localization were determined in human fibrotic livers and two murine models (CCl₄ and bile duct ligation) using snRNA-seq and primary hepatocyte isolation. Hepatocyte-specific MYSM1 knockout mice and AAV8-TBG-mediated hepatocyte-specific overexpression/knockdown of MYSM1, gelsolin (GSN), and ATG5 were generated. Autophagic flux was evaluated by mRFP-GFP-LC3, transmission electron microscopy, and LC3-II turnover. MYSM1-GSN interaction and K29-linked ubiquitination were dissected using Co-IP/mass spectrometry, linkage-specific assays, and MPN-domain mutants. Hepatic stellate cell activation was assessed using conditioned medium from manipulated hepatocytes.

Results:
Hepatocyte-specific MYSM1 deletion dramatically attenuated fibrosis in both CCl₄ and BDL models, restored autophagic flux, and reduced TGF- β 1 secretion.

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Conversely, MYSM1 overexpression worsened fibrosis. Interactome and transcriptomic analyses identified GSN as the key MYSM1 substrate. MYSM1 directly bound GSN and removed K29-linked ubiquitin chains via its MPN domain, markedly stabilizing GSN protein. GSN overexpression completely reversed the anti-fibrotic and pro-autophagic effects of MYSM1 knockout, whereas GSN knockdown phenocopied MYSM1 deletion. Simultaneous MYSM1 and GSN inactivation showed additive protection. Blocking autophagy with ATG5 knockdown abolished all benefits. Conditioned medium from MYSM1-deficient hepatocytes induced significantly less hepatic stellate cell activation.

Conclusions:

We delineate a novel MYSM1–GSN–autophagy–TGF- β 1 axis in hepatocytes that drives liver fibrosis progression. MYSM1 stabilizes GSN through K29-linked deubiquitination, inhibits autophagy, and amplifies TGF- β 1-mediated hepatic stellate cell activation. Targeting the MPN-domain deubiquitinase activity of MYSM1 represents a promising hepatocyte-specific therapeutic strategy.

Keyword(s) : Liver fibrosis, MYSM1, Autophagy, Gelsolin, Hepatic stellate cell activation

1715 - THE OUTCOME PREDICTION ROLE OF RED CELL DISTRIBUTION WIDTH IN 1000 HOSPITALIZED CIRRHOTIC EGYPTIAN PATIENTS

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Background / Hypothesis:
A systemic inflammatory response syndrome (SIRS) is linked to red cell distribution width (RCDW), which produces pro-inflammatory signals that act directly on hematopoietic stem cells in the bone marrow. This stimulation may cause alterations in the membrane of red blood cells (RBCs), as assessed by RCDW, which have been linked to increased morbidity and death in a number of systemic disorders. This study aimed to evaluate RCDW as a predictor of outcome in hospitalized cirrhotic patients.

Materials and Methods:
This prospective cross-sectional study was conducted on 1000 patients. The outcome was assessed by days of

hospitalization; mortality in hospitalized patients or during short-term follow-up (3 months) and rehospitalization during follow-up of 6 months.

Results:

Male represented 69.6%. Mean age was 57.67 ± 13.07 years old. Baseline co-morbidities were recorded as the presence of diabetes mellitus (200 patients) and hypertension (400 patients). Hepatitis C virus was the commonest etiology of the diseased liver (90%). Child-Pugh classes A, B and C of studied patients represented (21.2%, 38.8% and 40%). The survived patients during follow-up represented 63.3%. Area under the curve for RCDW was 0.923 (95% CI, 0.904–0.943), 0.910 for C-reactive protein (95% CI, 0.890–0.930), 0.904 for Hb (95% CI, 0.883–0.925) and 0.903 for platelets (95% CI, 0.882–0.924). RCDW cutoff point at 21.35 for predicting survival had sensitivity 93%, specificity 91%, accuracy 92%, positive predictive value 85 and negative predictive value 96. Regression analysis revealed a significant positive association between both RCDW and white blood cells with mortality.

Conclusions:

RCDW could provide useful information for predicting the length of hospitalization and survival in hospitalized cirrhotic patients.

Keyword(s) : RED CELL DISTRIBUTION WIDTH ; outcome; cirrhosis; hospitalized

1717 - A new switch for lactate accumulation in metabolic dysfunction associated steatotic liver disease

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Background / Hypothesis: Lactylation is a novel epigenetic modification that converts lysine ϵ -amino groups into lactyllysine via enzyme-catalyzed reactions using lactate as a donor. Functioning as a "metabolic-epigenetic" switch, lactylation regulates chromatin, protein structure, and signaling pathways, exerting

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multifaceted effects across immune and metabolic systems. Metabolic dysfunction-associated steatohepatitis (MASLD) ranks among the world's most prevalent chronic liver diseases. Its core pathogenesis centers on insulin resistance, which drives abnormal lipid accumulation in the liver. Metabolic dysregulation triggered by insulin resistance establishes lactate as a key substrate for de novo lipid synthesis in hepatocytes. The differential uptake of lactate by hepatocytes and hepatic stellate cells (HSCs) promotes glycolysis, lipid deposition, oxidative stress, and HSC activation. This process determines the progression rate and prognosis of MASLD from simple fatty liver to steatohepatitis and fibrosis. Although drug development holds promise for alleviating MASLD and improving outcomes, researchers face challenges: exploring novel targets to optimize therapeutic strategies is essential for achieving superior efficacy.

Materials and Methods: This review was compiled through systematic literature retrieval. Databases searched included PubMed, Web of Science, China National Knowledge Infrastructure (CNKI), and Wanfang Data, with the search conducted up to November 2025. Chinese search terms included "lactylation," "liver disease," and "MASLD." Inclusion criteria were: (1) formally published Chinese or English literature; (2) Research content related to lactylation, liver disease, or MASLD; (3) Document types included original research, reviews, and high-quality conference papers. Exclusion criteria: Duplicate publications, incomplete data, or documents with low relevance to the topic. Finally, the eligible documents were summarized, synthesized, and analyzed to form the content of this review.

Results: This paper focuses on recent advances in histone and non-histone lactylation research, comparing three novel epigenetic dimensions: L-lactylation, D-lactylation, and N-ε-carboxyethyl lactylation. It also reviews the current status of lactylation studies in MASLD. Finally, it identifies existing research gaps and unresolved issues in lactylation studies within the MASLD context.

Conclusions: Lactylation may serve as a bridge between lipid deposition, inflammation, and fibrosis, thereby holding potential for achieving targeted MASLD

therapy.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease, lactate, lactylation, insulin resistance

1722 - Paraneoplastic Hyperbilirubinemia in Hodgkin's Lymphoma An Uncommon Presentation with Significant Diagnostic and Therapeutic Challenges

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Background: Hodgkin's Lymphoma (HL) can rarely cause paraneoplastic hyperbilirubinemia, creating a diagnostic dilemma when liver dysfunction appears without biliary obstruction or hepatic disease. This presentation often mimics drug-induced liver injury (DILI), making it difficult to distinguish between treatment toxicity and a tumor-related process. Clarifying the true cause is critical, as management and chemotherapy decisions differ significantly between DILI and paraneoplastic syndromes.

Case Presentation: A 36-year-old man with classical HL presented with isolated jaundice and was found to have markedly elevated total, direct, and indirect bilirubin, as well as increased transaminases and alkaline phosphatase, despite no history of liver disease or alcohol use. Extensive infectious and autoimmune testing was unrevealing, and imaging showed no hepatic lesions, biliary obstruction, or abnormal metabolic activity. He was started on corticosteroids and Brentuximab-based therapy, but progressive hyperbilirubinemia raised concerns for DILI, a paraneoplastic process, or possible Vanishing Bile Duct Syndrome (VBDS); liver biopsy demonstrated ductopenia without meeting criteria for VBDS. Given the rising bilirubin, treatment was switched to an AVD regimen (with reduced-dose Adriamycin) while continuing corticosteroids. This modification led to a steady and significant decline in bilirubin levels, accompanied by clinical improvement and PET-CT-confirmed metabolic remission. The patient's response supported a paraneoplastic etiology of his hyperbilirubinemia, responsive to targeted lymphoma therapy.

Conclusion: This case highlights a rare presentation of paraneoplastic hyperbilirubinemia in HL and the need for comprehensive evaluation of liver dysfunction in oncology patients and underscores the importance of flexibility in modifying chemotherapy regimens based

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on evolving clinical status.

Keyword(s) : Hodgkin's Lymphoma, Paraneoplastic Hyperbilirubinemia, DILI

1725 - Suboptimal sensitivity of quantitative HBcrAg assay for managing HBeAg negative chronic hepatitis B

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Background / **Hypothesis:**
HBV core-related antigen (HBcrAg) aids in diagnosis of HBeAg-negative chronic hepatitis B (ENH) and predicting nucleos(t)ide analogues (NAs) cessation. However, serum HBcrAg level in ENH patients is generally low, and the sensitivity of the HBcrAg assay was inconsistently reported in previous studies. The factors contributing to this inconsistent sensitivity remain unclear. We therefore aimed to investigate the sensitivity-related factors of the HBcrAg assay. **Materials** and **Methods:**
ENH patients treated with tenofovir (TDF) for at least two years were included. Serial serum HBcrAg was tested using the Lumipulse HBcrAg quantitative assay. Baseline HBV genotype and core antigen amino acid mutations were detected by Sanger sequencing. Serum HBcrAg, HBsAg, HBV DNA and pre-genomic RNA (pgRNA) levels were dynamically monitored over 2 years of treatment. **Results:**

The functional sensitivity of the HBcrAg assay was 0.96~1.12 kU/ml, and carry-over contamination from anti-HBc assay was identified as a potential interference factor. A total of 78 ENH patients treated with TDF were enrolled. Serum HBcrAg level in ENH patients was low (1.74 ± 1.03 log₁₀ kU/ml) and correlated well with HBV DNA regardless of genotypes (genotype B, $R=0.66$; Genotype C, $R=0.74$; both $P<0.01$). However, genotype affected the correlations of HBcrAg with HBsAg and pgRNA: positive correlations were observed in genotype C (HBcrAg vs HBsAg: $R=0.67$, $P<0.001$; HBcrAg vs pgRNA: $R=0.57$, $P<0.001$), but not in genotype B (HBcrAg vs HBsAg: $R=0.32$, $P=0.18$; HBcrAg vs pgRNA: $R=0.29$, $P=0.22$). Notably, for baseline serum samples, 14.10% (11/78) of patients had underestimated HBcrAg levels, including 4 with false-negative results; these patients harbored more amino acid mutations in the three core epitopes targeted by the Lumipulse assay.

After 2 years of TDF treatment, the reductions of HBsAg, HBcrAg and HBV pgRNA were 0.23 log₁₀ IU/ml (7.43%), 0.96 log₁₀ kU/ml (55.57%) and 1.91 log₁₀ copies/ml (63.46%), respectively. HBcrAg clearance occurred later than pgRNA clearance (23.08% [18/78] vs 37.18% [29/78] of patients with negative results). HBcrAg and HBV pgRNA remained positively correlated in genotype-C patients during treatment. Two patients showed false-negative HBcrAg with persistently positive pgRNA, which was associated with more core antigen mutations.

Conclusions:

The HBcrAg assay has insufficient sensitivity for managing ENH, partially attributed to core antigen epitope mutations. Combining HBcrAg with pgRNA may improve the accuracy of ENH diagnosis and prediction of relapse after NA discontinuation.

Keyword(s) : HBeAg-negative chronic hepatitis B (ENH); HBV core-related antigen (HBcrAg); pre-genomic RNA (pgRNA); assay sensitivity

1726 - Glucagon like peptide 1 receptor agonists and reduced liver and non liver complications in patients with type 2 diabetes and viral hepatitis

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Background / **Hypothesis:**

Glucagon-like peptide-1 receptor agonists (GLP-1RA) are a novel class of anti-diabetic drugs that can treat obesity and type 2 diabetes (T2D) and has been shown to reverse steatohepatitis and reduce cardiovascular risk. T2D and viral hepatitis frequently coexist and worsen liver and non-liver outcomes, but effective pharmacological therapies are limited. We aimed to evaluate the long-term effect of GLP-1RA on liver and non-liver complications among patients with T2D and viral hepatitis in the United States.

Materials and **Methods:**

Patients with T2D and viral hepatitis initiating GLP-1RA were included from 2007 to 2022 in Merative™ MarketScan® Research Databases. Primary outcomes included (1) hepatocellular carcinoma (HCC) and cirrhosis, and (2) cardiovascular disease (CVD), chronic kidney disease (CKD), and non-liver cancer. Propensity

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score matching was applied to balance baseline characteristics and Cox regression models were conducted to estimate hazard ratio (HR) and 95% confidence interval (CI).

Results:

61,164 patients with T2D and viral hepatitis were initially analyzed. Patients with and without GLP-1RA use were 1:4 matched by propensity score for their demographics, comorbidities, and background medications. A total of 9,499 patients with T2D and viral hepatitis (2,133 in GLP-1RA and 7,366 in non-GLP-1RA group; mean age 57.2 years and 41% were female at baseline) were included. Compared with non-GLP-1RA group, GLP-1RA use was associated with 34% lower risk of any liver-related outcomes (HR 0.66, 95% CI 0.53-0.81; $P<0.001$), 50% lower risk of HCC (HR 0.50, 95% CI 0.29-0.88; $P=0.016$), 35% lower risk of cirrhosis (HR 0.65, 95% CI 0.52-0.80; $P<0.001$), 18% lower risk of CVD (HR 0.82, 95% CI 0.66-1.02; $P=0.079$), 34% lower risk of CKD (HR 0.66, 95% CI 0.53-0.83; $P<0.001$), and 28% lower risk of non-liver cancer (HR 0.72, 95% CI 0.53-0.99; $P=0.042$) (Table 1). Compared with dipeptidyl peptidase-4 inhibitors as active comparator, GLP-1RA use was associated with significantly lower risks of any liver-related outcomes (HR 0.70, 95% CI 0.51-0.95, $P=0.024$), HCC (HR 0.48, 95% CI 0.24-0.98, $P=0.045$), cirrhosis (HR 0.68, 95% CI 0.50-0.93, $P=0.016$) and CKD (HR 0.63, 95% CI 0.44-0.89, $P=0.010$). These significant relationships still were observed when we repeated the analysis using inverse probability of treatment weighting for the balance of patient characteristics (HR: 0.53-0.77).

Conclusions:

In a nationwide real-world cohort, GLP-1RA use was associated with a substantially lower risk of liver and non-liver complications than non-GLP-1RA group among patients with T2D and viral hepatitis. Large-scale randomized clinical trials are warranted.

Keyword(s) : Glucagon-like peptide-1 receptor agonists, type 2 diabetes, viral hepatitis, liver outcomes, cardiovascular disease, chronic kidney disease, non-liver cancer

1727 - Diagnosis of the differentials of ascites in low resource settings.

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Background / Hypothesis: A High serum ascites albumin gradient (SAAG) is highly suggestive of portal hypertension. Common causes of low-SAAG ascites are tuberculous peritonitis and peritoneal metastasis, and the differentiation between them is challenging. PCR with ascitic fluid or diagnostic laparoscopy appears to be an ideal tool for diagnosing tuberculous or metastatic peritonitis, but it is not available in all centres.

Materials and Methods: This is an observational study. A total of 25 patients with low SAAG ascites were included. Means are compared with the Independent-sample t-test. Cut-off value, sensitivity and specificity are measured with the ROC curve. This study was carried out at the Gastroenterology Department of BIRDEM General Hospital, Dhaka, Bangladesh.

Results:

Among 25 patients with low SAAG ascites, 12 were due to peritoneal metastasis and 9 were due to peritoneal TB.

SAAG in peritoneal met 08.21 ± 2.99 and in peritoneal TB 06.87 ± 1.34 (p-value 0.219)
Ascitic Fluid Glucose in peritoneal met 07.13 ± 2.38 and in peritoneal TB 04.82 ± 1.55 (p-value 0.016)
Ascitic Fluid Protein in peritoneal met 43.39 ± 13.35 and in peritoneal TB 63.47 ± 10.51 (p-value 0.001)
Ascitic Fluid Albumin in peritoneal met 24.53 ± 7.49 and in peritoneal TB 30.38 ± 5.36 (p-value 0.051)
Ascitic Fluid ADA in peritoneal met 18.04 ± 7.31 and in peritoneal TB 39.61 ± 13.15 (p-value <0.001)
Ascitic Fluid Cholesterol in peritoneal met 99.31 ± 38.51 and in peritoneal TB 107.22 ± 19.53 (p-value 0.573)
Ascitic fluid glucose <5.6 mg/dl suggests Peritoneal TB (p-value 0.016) with 75% sensitivity and 78% specificity.
Ascitic fluid Protein >57.7 gm/dl suggests Peritoneal TB (p-value 0.001) with 87% sensitivity and 78% specificity.
Ascitic fluid ADA >31.5 U/L significantly suggests Peritoneal TB (p-value <0.001) with 94% sensitivity and 78% specificity.

Conclusions: This small sample sized study shows low ascitic fluid glucose (<5.6 mmol/L), high ascitic fluid protein (>57.7 gm/dl), and ADA (>31.5) significantly

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suggest tuberculous peritonitis and exclude malignant ascites in patients with low SAAG ascites. Although the proposed results are not confirmatory, we may apply empirical anti-tuberculous treatment in low-resource settings.

Keyword(s) : Serum ascites albumin gradient-SAAG, Tuberculosis-TB

1729 - The expression of Fmnl1 in monocyte derived macrophages promotes hepatic inflammation in MAFLD

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Background / Hypothesis: Metabolic dysfunction-associated fatty liver disease (MAFLD) is a chronic, progressive liver disorder characterized by complex interactions among immune cells, with macrophage-driven inflammation playing a pivotal role in disease progression. FMNL1, a member of the formin protein family, is involved in cytoskeletal remodeling and cell migration. However, its specific role in the pathogenesis of MAFLD remains unclear.

Materials and Methods: Single-cell RNA sequencing with pseudotime analysis was employed to profile FMNL1 expression during monocyte-to-macrophage differentiation. Myeloid-specific Fmnl1 knockout and control mice were established and subjected to choline-deficient high-fat diet (CDHFD) or western diet with fructose/CCl₄ to model MAFLD progression. Human MAFLD tissues and mouse livers were analyzed by immunohistochemistry and multi-color immunofluorescence for FMNL1 expression and localization. Histopathological assessment included H&E and Sirius Red staining for steatosis, inflammation and fibrosis. Flow cytometry of Percoll-isolated hepatic nuclei characterized monocyte-derived macrophages and M1/M2 populations. *in vitro*, PMA-differentiated THP-1 macrophages with lentiviral-mediated FMNL1 knockdown underwent Transwell migration assays and phalloidin-based F-actin staining. Mechanistic studies included RNA sequencing, co-immunoprecipitation-mass spectrometry, and Cdc42 activity pull-down assays in FMNL1-knockdown cells.

Results: Immunohistochemical analysis revealed a significant

upregulation of FMNL1 in both human MAFLD and CDHFD mouse livers, predominantly localized to monocyte-derived macrophages. Notably, the degree of hepatic inflammation was reduced in myeloid-specific Fmnl1 knockout mice, which also exhibited a decreased number of monocyte-derived macrophages and a reduction in M1 macrophages. FMNL1 expression was markedly induced during PMA-mediated differentiation of THP-1 macrophages and was also increased in bone marrow-derived macrophages (BMDMs) stimulated with MSF. FMNL1 knockdown resulted in impaired F-actin polymerization and significantly reduced macrophage migratory capacity. Transcriptomic profiling of FMNL1-knockdown cells demonstrated significant enrichment in pathways related to GTPase activity and Rho signaling. Additionally, co-immunoprecipitation and mass spectrometry identified Cdc42 as a key binding partner of FMNL1. Importantly, PMA stimulation triggered a time-dependent activation of Cdc42 (in its GTP-bound form), which was associated with enhanced macrophage migration. This Cdc42 activity was essential for FMNL1 upregulation via the MAPK signaling pathway (MEK/ERK). Finally, FMNL1 depletion impaired the M1 polarization capacity of BMDMs.

Conclusions: A novel mechanistic pathway is proposed in which inflammatory signals activate Cdc42, leading to the upregulation of FMNL1 expression through the MAPK pathway. Elevated FMNL1 facilitates actin cytoskeleton reorganization, promoting the recruitment and M1 polarization of monocyte-derived macrophages, thereby exacerbating liver inflammation. Targeting the Cdc42-FMNL1 axis may represent a promising therapeutic strategy for the management of MAFLD.

Keyword(s) : MAFLD, Macrophages, Monocyte-derived Macrophages, FMNL1, Cytoskeleton

1732 - Associations of sweetened beverages consumption with the risk of MASLD and its related outcomes across genetic risk profiles a prospective population based cohort study

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Background / Hypothesis: To examine the associations of the intake of sugar-sweetened beverages (SSBs),

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low/no-sugar-sweetened beverages (LNSSBs), and fruit juices (FJs) with incidence and progression of metabolic dysfunction-associated steatotic liver disease (MASLD), including cirrhosis, liver cancer, and liver-related mortality, and to evaluate whether these associations vary by genetic risk.

Materials and Methods: Of the 502,151 participants in the UK Biobank, 170,918 who completed at least one web-based 24-hour dietary recall (Oxford WebQ) were included in the analyses after excluding individuals with baseline liver-related disease or implausible energy intake. For genetic stratification analyses, individuals of non-European ancestry or with missing genetic data were excluded, resulting in a sub-cohort of 142,065 participants. Analyses were conducted using Cox proportional hazards models after multiple imputation. The primary outcome was incident MASLD. Secondary outcomes were new-onset cirrhosis, liver cancer and liver-related mortality.

Results: During a median follow-up of 14 years among 170,918 participants, 1,904 MASLD cases, 566 cirrhosis cases, 146 liver cancer cases, and 274 liver-related deaths were documented. Consumption of >2 servings/day was associated with higher MASLD risk for both SSBs (hazard ratio [HR]: 1.57, 95% confidence interval [CI]: 1.28-1.93) and LNSSBs (HR: 2.40, 95% CI: 2.03-2.83), while <1 serving/day of FJs was associated with lower risk (HR: 0.82, 95% CI: 0.74-0.90) compared with non-consumption. Among those with low genetic risk, >2 servings/day of SSBs was associated with greater MASLD risk (HR: 1.95, 95% CI: 1.28-2.98) than in those with high genetic risk (HR: 1.17, 95% CI: 0.77-1.76). Similarly, the associations of LNSSBs and FJs with MASLD were more pronounced among individuals with low genetic risk.

Conclusions: Higher consumption of SSBs and LNSSBs was associated with increased risk of MASLD and related outcomes in a dose-dependent manner, whereas moderate FJs intake was inversely associated with these risks. Participants with low genetic risk were more susceptible to the effects of beverage consumption on MASLD and cirrhosis.

Keyword(s) : MASLD, sugar-sweetened beverages, genetic risk, prospective cohort

1735 - Policy Blind Spots and Clinical Realities Determinants of Continued Alcohol Use in Alcohol Associated Liver Disease at India's First Integrated AUD-ALD Clinic

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Background:

Alcohol-associated liver disease (AaLD) is a significant public health concern, particularly in the USA, where alcohol use is on the rise. Despite medical advice to abstain, many patients with liver damage continue to consume alcohol. While external and internal triggers for relapse have been identified, limited research has explored the interplay of dependence severity, harm perception, and motivational readiness in this group.

Methods:

A cross-sectional survey was conducted at the Integrated AUD clinic of Institute of Liver and Biliary Sciences (ILBS), New Delhi. A total of 3759 (over a period of two years (2022-2024) patients with AaLD were referred and categorized as "cases" (n=2932; alcohol use within the last 60 days) and "controls" (n=827; abstinent for more than 60 days). Patients were seen and managed by dedicated addiction specialist and team with Institutional protocols. Data were collected using a semi-structured clinical proforma, the Severity of Dependence Scale (SDS), and the Readiness to Change Questionnaire (RTCQ). Comparisons were made using chi-square and Mann-Whitney U tests to assess differences in clinical variables, dependence severity, harm perception, and motivational stages.

Results:

Participants had a mean age of 43.8 years (SD=19.4), all being male. Most (77%) reported a history of daily or near-daily alcohol use, with over 60% having consumed alcohol for more than 6 years. Compared to controls (Mean SDS=6.41), Cases (Mean SDS=7.10) had significantly higher SDS scores ($p<0.001$), indicating greater dependence, and were more likely to be in the pre-contemplation or contemplation stages rather than action stage of change ($p=0.001$). Compared to controls, cases demonstrated significantly lower awareness regarding the harms of alcohol, its addictive potential, and the importance of quitting after liver disease diagnosis (all $p<0.001$). Biomarker comparisons showed higher bilirubin levels and MELD scores in current users.

Conclusion:

High dependence, low motivation to change, and poor

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harm perception appear to be key contributing factors for continued alcohol use. This is first of its kind integrated AUD-ALD clinic data in India, that combine motivational enhancement, psychoeducation, and dependence treatment to support sustained abstinence cirrhosis patients under the same roof; and provides an opportunity to scale up integrated AUD clinics in the country and globally.

Keyword(s) :

1738 - Combination Therapy with Toll like receptor 7 Agonist and Programmed Death Ligand 1 Antibody Restores HBV Specific CD8+T Cells in Virally suppressed Chronic Hepatitis B
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Background / **Hypothesis:**
In a randomized, controlled phase II trial (OCEANcure05, NCT 04202653), combination therapy with the TLR7 agonist TQ-A3334 and the anti-PD-L1 antibody TQ-B2450 effectively reduced hepatitis B surface antigen (HBsAg) in nucleos(t)ide analogue (NAs)-experienced chronic hepatitis B (CHB) patients, with favorable safety and tolerability. This study aimed to delineate the immunologic features induced by the combination regimen and their relationships with clinical efficacy.

Materials and **Methods:**
24 NAs-experienced CHB patients were randomized into NAs monotherapy, dual therapy with NAs plus TLR7 agonist, or triple therapy with NAs, TLR7 agonist and PD-L1 antibody. After 24 weeks of treatment, patients were followed-up until Week 48. The dynamics of serum cytokine levels from baseline to end of treatment (EOT) were measured by Meso Scale Discovery electrochemiluminescence assay (MSD). The immune status of CD4+T cells, CD8+T cells, and HBV-specific CD8+T cells from PBMC were evaluated by flow cytometry. Single-cell RNA sequencing was performed on PBMC from representative patients. In vitro PBMC-HepAD38 co-culture system was employed for validation.

Results:

In the triple therapy group, serum IFN- γ , IP-10, and sPD-L1 levels were significantly elevated, while sPD-L1 levels were markedly decreased at Week 12. Serum IL-21 levels

were profoundly increased and were higher than those in the dual therapy group at EOT. The triple therapy group demonstrated enriched effector phenotypes of CD4+T and CD8+T cells. Transcriptomic analysis revealed an enrichment of IL-21-related gene sets in CD4+T cells from triple therapy patients at EOT. In vitro, TLR7 agonist plus PD-L1 antibody increased IL-21+CD4+T frequency and supernatant IL-21 level compared to TLR7 alone. The triple therapy group exhibited superior HBV-specific CD8+T cell responses at EOT. The frequencies of core-IFN- γ +, CD107a+, and polymerase-IFN- γ +CD8+T cells increased from baseline and higher than those in dual therapy group. In vitro, PD-L1 antibody plus TLR7 agonist enhanced core-specific CD8+T responses, whereas the addition of IL-21/IL-21R blockade abolished these effects. Triple therapy triggered STAT3 phosphorylation in CD4+T cells and enriched the JAK-STAT signaling pathway. Targeted inhibition of STAT3 in CD4+T cells blocked the triple therapy-induced augmentation of core-specific CD8+T cell response, HepAD38 lysis, and suppression of HBV DNA and HBeAg levels.

Conclusions:

The combination of TLR7 agonist and PD-L1 antibody drives STAT3-dependent IL-21 secretion from CD4+T cells, thereby enhancing HBV-specific CD8+T cell responses and suppressing viral replication via the IL-21/IL-21 receptor pathway. This mechanism may contribute to the enhanced efficacy of triple therapy in NAs-experienced CHB patients.

Keyword(s) : combination therapy, chronic hepatitis B, HBV specific CD8+T, IL-21

1747 - Predictive Biomarkers for Hepatitis E Virus Related Acute Liver Failure Based on Lipidomics and Transcriptomics

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Background: Hepatitis E virus (HEV) is a major cause of acute viral hepatitis globally. Although usually self-limiting in healthy individuals, it can progress to severe hepatitis or acute liver failure (ALF). This study aimed to provide biomarkers for patient with Hepatitis E virus-related acute liver failure (HEV-ALF) using lipidomics and transcriptomics.

Methods: We enrolled 150 participants, including 50 acute hepatitis E (AHE) patients, 50 HEV-ALF patients, and 50 healthy controls (HCs). Lipidomics analysis was

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performed using LC-MS/MS, with differential lipids screened based on a variable importance in projection (VIP) score > 1.0, an absolute fold change (FC) > 1.5, and a p-value < 0.05. Additionally, 43 paired whole blood samples (15 AHE, 15 HEV-ALF, 13 HCs) were collected for transcriptomics. Differentially expressed genes were identified using $p < 0.05$ and $FC > 1.0$. Results: Lipidomics analysis identified 750 significantly altered lipid metabolites. Compared to HCs, the AHE group exhibited 444 differential lipids (190 upregulated, 254 downregulated). Between the HEV-ALF and AHE groups, 306 differential lipids were identified, all upregulated. Machine learning was employed to identify optimal lipid marker panels: the best combination for distinguishing AHE from HCs was Cer(d18:1/23:1); TG(6:1COOH/9:0/9:0); LPC (29:2). Using this panel, the AdaBoost classifier achieved an AUC of 1.0, and the Light Gradient Boosting Machine (LightGBM) achieved an AUC of 0.960. For distinguishing HEV-ALF from AHE, the best marker was DG (21:4/18:0), with the Light Gradient Boosting Machine (LightGBM) yielding an AUC of 0.770. Subsequent validation using multiple algorithms (Decision Tree (DT), Support Vector Machine (SVM), Random Forest (RF), K-Nearest Neighbors (KNN), Naive Bayes (NB), Logistic Regression (LR)) for AHE vs. HCs showed that all AUCs exceeded 0.95. For HEV-ALF vs. AHE, the SVM and NB both achieved AUCs above 0.84. Transcriptomics analysis identified 6093 differentially expressed genes. Compared to HCs, the AHE group had 5114 differential genes (1554 upregulated, 3560 downregulated). Comparing HEV-ALF to AHE revealed 979 differential genes (499 upregulated, 480 downregulated). Finally, integrated bioinformatics analysis of differential lipids and genes was conducted to explore the associated molecular mechanisms. Conclusion: This multi-omics study delineates the molecular landscape of HEV infection and progression. We identified significant lipid metabolic and transcriptomic disruptions in AHE and HEV-ALF patients. Machine learning-derived lipid panels showed exceptional diagnostic potential, while transcriptomics revealed key genes and pathways involved in disease progression. Our findings provide novel insights into HEV pathogenesis and offer a foundation for early diagnosis and targeted interventions.

Keyword(s) : Hepatitis E; Acute Liver Failure; Lipidomics; Transcriptomics; Machine Learning; Biomarkers

1753 - Allogeneic Umbilical Cord Mesenchymal Stem Cell Transplantation in Patients with Cirrhosis Caused by Hepatitis B A Single Center Case Series

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Background:

Chronic hepatitis B is a major cause of cirrhosis and liver failure, with limited treatment options beyond transplantation. Mesenchymal stem cell (MSC) therapy offers regenerative, antifibrotic, and immunomodulatory potential. Umbilical cord-derived MSCs provide a readily available, low-immunogenic source suitable for allogeneic use. This study evaluated the safety and preliminary efficacy of allogeneic umbilical cord MSC transplantation in patients with decompensated cirrhosis secondary to chronic hepatitis B.

Methods:

This phase I-II, open-label, single-arm trial evaluated the safety and preliminary efficacy of allogeneic umbilical cord-derived MSC transplantation in patients with decompensated cirrhosis due to chronic hepatitis B (ClinicalTrials.gov: NCT04357600). Adults aged 18–65 years with Child-Pugh B cirrhosis stable on antiviral therapy were enrolled through consecutive sampling. Exclusion criteria included malignancy, coinfection, major comorbidities, pregnancy, alcohol dependence, or prior transplantation. GMP-grade MSCs were verified by surface markers (CD73+, CD90+, CD105+) and sterility testing. Each patient received 10–100 million MSCs intravenously over 30 minutes after standard premedication and hydration. Hemodynamic parameters were monitored for 24 hours. Follow-up at 1, 3, and 6 months included laboratory tests and clinical evaluation, with liver ultrasonography at 6 months to assess safety and early efficacy.

Results:

All six male patients (aged 37–60 years) with decompensated cirrhosis completed the intravenous administration of MSC infusion without adverse reactions or hemodynamic instability. At one month post-infusion, significant hematologic improvements were observed: hemoglobin, hematocrit, erythrocytes, platelets, and leukocytes all increased ($p < 0.05$), suggesting enhanced marrow activity and immunomodulatory effects. Coagulation parameters remained stable overall, though individual cases showed shortened PT and reduced INR. Liver enzymes (AST, ALT) remained within baseline ranges, and direct bilirubin decreased significantly at three months ($p = 0.048$). Synthetic function improved, with albumin and total

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protein rising significantly at one month ($p=0.018$ and $p=0.023$, respectively), accompanied by an improved albumin/globulin ratio ($p=0.012$). Transient elastography showed marked reductions in liver stiffness in two patients, indicating potential antifibrotic effects. Individual variations were noted, but overall trends favored improved hematologic and hepatic parameters without safety concerns. One patient (Case 5) experienced septic shock resulting in death, deemed unrelated to therapy.

Conclusions:

Allogeneic umbilical cord-derived MSC transplantation was safe and well-tolerated in patients with decompensated cirrhosis secondary to chronic hepatitis B, showing potential regenerative and antifibrotic effects through improvements in hematologic indices, serum albumin, and liver stiffness. These preliminary findings warrant further evaluation in larger, controlled trials to confirm therapeutic efficacy and long-term outcomes.

Keyword(s) : umbilical cord, cirrhosis, hepatitis B, chronic liver disease, mesenchymal stem cells, regenerative therapy

1754 - The mechanism of TMBIM5 in ameliorating alcohol related liver disease by regulating mitochondrial dynamics

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Background / Hypothesis: ALD is a group of metabolic disorders induced by prolonged and heavy alcohol consumption. However, there are many limitations in the treatment of ALD. TMBIM5 has been shown to maintain the normal function of mitochondria in other diseases, but its role in ALD is unclear. This study aimed to investigate its role and mechanism of TMBIM5 in the development of ALD.

Materials and Methods: The mechanism was investigated TMBIM5 in alcohol-induced in vivo and in vitro models. The pathological changes were observed by H&E and Masson staining. Inflammation, apoptosis, and mitochondrial function were evaluated by qPCR, Western blotting, immunofluorescence, ROS production, ATP levels, JC-1 staining, MPTP opening, and oxygen consumption rate trials. And the CO-IP experiments and a series of mutant plasmids of TMBIM5 were constructed to further clarify the molecular mechanism.

Results:

A significant decrease in TMBIM5 protein expression was observed in alcohol-induced groups, while no significant change is observed at the mRNA level. TMBIM5 deficiency further aggravated alcohol-induced liver injury, whereas overexpression alleviated these damage. In addition, we found that TMBIM5 overexpression attenuated alcohol-induced mitochondrial oxidative stress and preserved mitochondrial function. Furthermore, we elucidate alcohol induces post-transcriptional degradation of TMBIM5 by downregulating the expression of mitochondrial fission proteins, while upregulating fusion proteins, ensuring the stability of mitochondrial structure and function, thereby protecting hepatocytes from alcohol damage.

Conclusions:

TMBIM5 can alleviate hepatocyte injury by regulating mitochondrial dynamics, positioning it as a promising therapeutic strategy.

Keyword(s) : Alcoholic liver disease, Hepatocytes, Mitochondrial dynamics, TMBIM5, Inflammation

1757 - Impact of etiology on outcomes with atezolizumab plus bevacizumab in advanced hepatocellular carcinoma A multicenter propensity matched analysis

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Background / **Hypothesis:**
Atezolizumab plus bevacizumab is widely used as first-line therapy for advanced hepatocellular carcinoma (HCC). Etiologic and immunologic heterogeneity in HCC may influence the tumor immune microenvironment and treatment response, although vascular endothelial growth factor (VEGF) inhibition may reduce such variation. We hypothesized that survival outcomes and immune-related adverse events (irAEs) would be comparable between viral and non-viral HCC after rigorous adjustment for baseline characteristics.

Materials and **Methods:**
This real-world, retrospective study included 1,115 patients with advanced HCC treated at nine tertiary centers between 2021 and 2025. Patients were classified as viral (hepatitis B or C virus) or non-viral (alcohol-associated, metabolic dysfunction-associated, or cryptogenic). Propensity score matching was performed using nearest-neighbor matching (1:1 ratio) without replacement and with a caliper of 0.1 of the standard deviation of the logit. A total of 217 matched pairs were analyzed. Overall survival (OS), progression-free survival (PFS), and irAEs, including proteinuria associated with VEGF inhibition, were analyzed. Kaplan-Meier curves, Cox proportional hazards models, and standardized mean differences (SMDs) were used to evaluate covariate balance and outcomes. Differences in irAEs were compared using chi-square tests.

Results:
Baseline characteristics were well balanced after matching (all SMD <0.1). OS did not differ between viral and non-viral groups (log-rank $p=0.92$; HR 0.99, 95% CI 0.78–1.25). PFS was also comparable (log-rank $p=0.91$; HR 0.99, 95% CI 0.80–1.22). Survival curves for both OS and PFS were nearly superimposed, with no clinically meaningful differences observed between etiologic

subgroups. The incidence of irAEs was similar (non-viral 53.6% vs viral 50.9%; $p=0.633$). Proteinuria occurred more frequently in the non-viral group (21.1% vs 13.5%), although this difference was not statistically significant ($p=0.095$).

Conclusions:

In this propensity-matched cohort, etiology did not affect survival outcomes with atezolizumab plus bevacizumab, supporting its use as first-line therapy for advanced HCC regardless of underlying etiology. Additional prospective research is required to confirm these results.

Keyword(s) : Hepatocellular carcinoma; Immunotherapy; Atezolizumab; Bevacizumab; Tumor immune microenvironment

1758 - Predictive Role of Spleen Stiffness in Gastrointestinal Bleeding for Patients with Unresectable Hepatocellular Carcinoma Undergoing Systemic Therapy

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Background / **Hypothesis:**
Systemic therapy has demonstrated efficacy in delaying disease progression among patients with unresectable hepatocellular carcinoma (u-HCC). However, the occurrence of gastrointestinal bleeding (GIB) significantly influences the selection of appropriate systemic treatment regimens. Consequently, predicting GIB is paramount for optimizing clinical decision-making. This study aims to identify risk factors for GIB in HCC patients undergoing systemic therapy.

Materials and **Methods:**
We conducted a retrospective analysis of 412 patients with cirrhosis and u-HCC admitted to Guizhou Provincial People's Hospital between June 2023 and March 2025. Following multiple imputation for missing data, patients were randomly stratified into training and test sets. Predictive features were selected using Least Absolute Shrinkage and Selection Operator (LASSO) regression and subsequently incorporated into machine learning (ML) models. Through feature importance analysis, we identified features common across all models and those demonstrating significant clinical relevance to bleeding risk. These features were then subjected to receiver operating characteristic (ROC) prediction analysis. Additionally, Kaplan-Meier survival curves and log-rank tests were employed to evaluate the predictive utility of these features for bleeding events.

Results:

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Results: The Spleen Stiffness Measurements (SSM) demonstrated significant diagnostic accuracy for GIB, yielding an area under the curve (AUC) of 0.821 (95% CI: 0.767–0.874, $P < 0.001$) with an optimal cutoff value of 45.5. Kaplan-Meier analysis revealed significantly shorter GIB-free survival in patients with $SSM > 45.5$ compared to those with $SSM \leq 45.5$ (log-rank $P < 0.0001$).

Conclusions: SSM, identified via ML models and validated through ROC and survival analyses, serves as a reliable predictor for GIB in u-HCC patients. It facilitates early risk stratification and guides targeted interventions to mitigate bleeding-related morbidity.

Keyword(s) : Spleen Stiffness Measurements, unresectable hepatocellular carcinoma, Systemic therapy

1768 - ALT Trajectories Differentiated by Early Treatment Response Determine Long Term Outcomes in Autoimmune Hepatitis

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Background / Hypothesis: In autoimmune hepatitis (AIH), alanine aminotransferase (ALT) is a central biomarker for monitoring treatment response. Current treatment guidelines establish the normalization of ALT within six months as a key criterion for complete biochemical response, which is linked to improved long-term outcomes. However, this paradigm relies solely on a single-timepoint absolute value and employs an extended observation window that may delay necessary treatment adjustments. The significance of early ALT dynamics and their role in shaping long-term ALT trajectories remain unclear. We aimed to characterize long-term ALT trajectories based on early treatment response and to evaluate the relationship between these longitudinal ALT profiles and subsequent clinical events.

Materials and Methods: This retrospective study included AIH patients with elevated baseline ALT who underwent liver biopsy. Participants were stratified into responders and non-responders according to whether ALT normalized within 3 months of treatment initiation. ALT trajectories were

modeled using a generalized additive mixed model (GAMM), and the joint model was applied to assess the association between longitudinal ALT changes and clinical events.

Results:

Among 41 AIH patients followed for a median of 37 months, 56% achieved ALT normalization within 3 months. A total of 5 clinical events (12.2%) occurred during follow-up. GAMM analysis revealed a significant interaction between time and early treatment response (Pinteraction < 0.001), with markedly different ALT trajectories between groups: responders showed a steep initial decline ($\beta = -0.363$), whereas non-responders exhibited minimal improvement ($\beta = -0.049$). After the initial 3-month period, ALT levels stabilized in both groups, with no significant further change (Pinteraction = 0.29). In the joint model, longitudinal increases in ALT were significantly associated with an increased risk of clinical events (HR 2.8 per 50% increase; 95% CI 1.0–7.9; $p = 0.047$).

Conclusions:

Early non-response to treatment is associated with persistently elevated ALT levels. Our findings suggest that assessing treatment response at 3 months may offer a clinically useful, earlier opportunity to identify complete biochemical response. Furthermore, longitudinal ALT trajectories are significantly linked to clinical events in AIH, underscoring the critical importance of monitoring these dynamic trends throughout the course of treatment.

Keyword(s) : autoimmune hepatitis, Liver biopsy, alanine aminotransferase, outcome

1769 - Admission NLR and APRI as Practical Low Cost Predictors of Severity in Cirrhosis Data from an Indian Tertiary Center

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Background / Hypothesis: Cirrhosis in low and middle income settings often presents at advanced stages, with high rates of decompensation and limited access to elastography, specialized prognostic scoring, or timely imaging. At many centers, clinicians must triage patients using only bedside assessment and routine laboratory tests. This highlights a need for simple, inexpensive marker that can support early severity assessment at the point of

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admission. Inflammation-derived indices and basic fibrosis surrogates obtained from standard blood investigations may offer practical value in such environments.

This study evaluates whether the Neutrophil-Lymphocyte Ratio (NLR) and the AST-to-Platelet Ratio Index (APRI) can meaningfully reflect disease severity and patterns of decompensation in cirrhotic patients presenting to a tertiary care hospital in Mumbai, India, and whether these readily available markers can assist early clinical stratification.

Materials and Methods: This cross-sectional study included 140 adult cirrhotic patients admitted between April 2024 and April 2025. Admission laboratory tests were used to calculate Neutrophil-Lymphocyte Ratio (NLR) and AST-to-Platelet Ratio Index (APRI). Severity was classified using Child-Pugh scores, and decompensation events were documented clinically. NLR and APRI were compared across severity grades and correlated with decompensation using ANOVA and chi-square testing.

Results: Among 140 cirrhotic patients, admission Neutrophil-Lymphocyte Ratio (NLR) and AST-to-Platelet Ratio Index (APRI) rose consistently with disease severity. Mean NLR increased from 3.5 in Child-Pugh A to 6.2 in B and 10.8 in C ($p < 0.001$). APRI showed a similar progression, rising from 1.1 to 1.9 and 3.2 respectively ($p < 0.001$). Elevated NLR (≥ 6) strongly correlated with major decompensation at presentation, including ascites and hepatic encephalopathy. A combined high-risk profile (NLR ≥ 6 plus APRI ≥ 1.5) identified 84 per cent of all patients presenting with at least one significant decompensating event, compared with 38 per cent when both markers were low ($p < 0.001$). These trends remained consistent across etiologies.

Conclusions: NLR and APRI, calculated from universally available admission laboratories, provide meaningful insight into both severity and early decompensation in cirrhosis. Their strong correlation with Child-Pugh class and

clinical instability supports their use as rapid, low-cost indicators at first medical contact. In resource-limited hepatology settings, these markers can help identify high-risk patients before advanced scores, imaging, or elastography are available. Incorporating NLR and APRI into routine initial assessment may strengthen early triage, guide clinical prioritisation, and improve frontline decision-making for cirrhotic patients in everyday practice.

Keyword(s) : Cirrhosis, NLR, APRI, India

1771 - Our Journey Towards a Dedicated Transjugular Intrahepatic Portosystemic Shunt (TIPSS) Service Teamwork Makes the Dream Work
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Background / Hypothesis: Transjugular intrahepatic portosystemic shunt (TIPSS) is a well-established intervention for the management of portal hypertension and its complications. Although widely available in many developed countries, resource limitations, lack of expertise, and infrastructural barriers have historically impeded its implementation in Malaysia. Establishing a dedicated TIPSS service requires strong multidisciplinary collaboration to optimise patient selection, ensure procedural success, and provide coordinated post-procedural care.

Materials and Methods: We describe our centre's initial experience in developing a dedicated TIPSS service, with procedures performed despite significant logistical constraints.

Results: Over an 8-month period, 15 TIPSS procedures were attempted, with 14 successful (technical success rate 93.3%). Indications included variceal bleeding—both salvage and pre-emptive (66.7%)—and refractory ascites (33.3%). Underlying etiologies of portal hypertension were alcohol-related cirrhosis (13%), hepatitis B or C cirrhosis (33.3%), metabolic dysfunction-associated steatohepatitis (27%),

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autoimmune liver disease (7%), cryptogenic cirrhosis (7%), and non-cirrhotic portal hypertension (20%). Technical success was defined as creation of a functional portosystemic shunt with adequate reduction of the portosystemic gradient. Clinical outcomes assessed included bleeding control, ascites response, iatrogenic complications, post-TIPSS hepatic encephalopathy (HE), post-TIPSS heart failure (HF), duration of hospitalisation, and in-hospital mortality. Most patients demonstrated rapid haemodynamic and clinical improvement. No post-TIPSS HE occurred. One patient developed acute HF. Two patients experienced iatrogenic complications: one hepatic artery injury requiring embolisation (TIPSS remained successful), and one portal vein injury in which the TIPSS procedure was abandoned and primary portal vein repair was performed. Overall, 87% of patients were discharged within 7 days post-TIPSS. No in-hospital mortality was reported.

Conclusions:

Our early experience demonstrates that a dedicated TIPSS service—driven by collaborative multidisciplinary teamwork—can be successfully established even within a resource-limited environment. Through collaborative effort, TIPSS has evolved from a distant dream to a practical and impactful treatment option for patients with complex portal hypertension in Malaysia.

Keyword(s) : TIPSS, Liver cirrhosis, Liver fibrosis, portal hypertension

1775 - Longitudinal Analysis Reveals Comparable ALT Trajectories and Achievement of Biochemical Remission Regardless of Baseline Fibrosis in Autoimmune Hepatitis

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Background / Hypothesis:

There is limited data on whether significant liver fibrosis impacts the treatment response in autoimmune hepatitis (AIH). Studies examining the association between liver fibrosis and long-term longitudinal ALT data are lacking. We aimed to investigate the association between longitudinal Alanine aminotransferase (ALT) over time and significant liver

fibrosis in AIH patients.

Materials and Methods: This retrospective review included 41 patients with AIH. All patients underwent liver biopsy at baseline and received standard treatment according to the EASL AIH guidelines. Significant liver fibrosis was diagnosed with a modified Ishak score $\geq$ S3. Patients were categorized into two groups based on their baseline fibrosis status: significant fibrosis and non-significant fibrosis. The association between ALT and clinical types was analyzed using a generalized additive mixed model (GAMM).

Results:

A total of 41 patients (80.4% female, 46.3% with significant fibrosis) were assessed, yielding 744 longitudinal ALT measurements. The GAMM analysis revealed that the longitudinal trajectory of ALT over time did not differ significantly between the two fibrosis groups (p for interaction = 0.819). Both groups had a steeper decline in ALT in the early stage of treatment and were stable thereafter. The monthly decline in Log₁₀(ALT) was -0.022 units (95% CI: -0.031 to -0.014) in the significant fibrosis group, compared to -0.021 units (95% CI: -0.029 to -0.013) in the non-significant fibrosis group.

Conclusions:

Longitudinal analysis via GAMM indicates that significant fibrosis does not impede the achievement of biochemical remission in AIH patients under standard treatment.

Keyword(s) : Autoimmune hepatitis, Liver biopsy, Alanine aminotransferase, Histological disease

1776 - Immune characteristics of peripheral blood cells in inactive hepatitis B surface antigen carriers treated with interferon

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Background / Hypothesis:

Predictors of functional cure in inactive HBsAg carriers treated with pegylated interferon- α -2b (Peg-IFN- α) remain unclear. We investigated early immune signatures using mass cytometry.

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Materials and **Methods:**
Among 240 screened inactive carriers (HBsAg<1000 IU/mL, HBeAg-negative, HBV DNA <2000 IU/mL, normal ALT), 166 were randomized 2:1:1 to continuous Peg-IFN- α weekly for 48 weeks (n=90), intermittent Peg-IFN- α (n=48), or untreated observation (n=28). A balanced subset of 94 patients (47 cured [CR] vs 47 non-responders [NR] at week 48) underwent PBMC analysis at baseline, week 12, and week 48 by mass cytometry (CyTOF) using a 37-marker panel covering T, B, NK, and monocyte subsets.

Results:

Week-48 HBsAg clearance rates were 31.1% (28/90) in the continuous arm, 18.8% (9/48) in the intermittent arm, and 3.6% (1/28) in the observation arm ($P<0.001$). In the immune cohort, baseline CR patients had lower CD4+ naïve T cells ($15.3 \pm 5.6\%$ vs $20.8 \pm 6.9\%$, $P=0.004$) and higher immature B cells ($5.4 \pm 2.5\%$ vs $3.7 \pm 2.0\%$, $P=0.007$). At week 12, CR showed robust T-cell restoration versus NR: CD4+ central memory ($6.4 \pm 3.0\%$ vs $3.1 \pm 1.9\%$), CD4+ effector memory ($19.1 \pm 5.9\%$ vs $11.8 \pm 4.7\%$), CD8+ effector memory T cells ($29.4 \pm 8.2\%$ vs $17.3 \pm 6.8\%$), and reduced PD-1+Eomes+ exhausted CD8+ T cells ($6.6 \pm 2.8\%$ vs $14.1 \pm 4.5\%$) (all $P<0.001$). Week-12 memory T-cell expansion combined with exhaustion reduction predicted HBsAg clearance (AUC=0.937).

Conclusions:

Continuous 48-week Peg-IFN- α achieved the highest functional cure rate (31.1%). Early (week 12) restoration of CD4+/CD8+ memory T cells with reversal of exhaustion is the dominant immune predictor of HBsAg seroclearance. Week-12 CyTOF monitoring may enable response-guided therapy.

Keyword(s) : hepatitis B, inactive carrier, HBsAg clearance, pegylated interferon, mass cytometry, memory T cell, T-cell exhaustion

1781 - An Endothelial Related Gene Signature Derived from Integrative Transcriptomics Predicts HCC in Cirrhosis

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Background

Liver cirrhosis is a major global health challenge and a key risk factor for hepatocellular carcinoma (HCC), a malignancy with high mortality due to late diagnosis. This study aimed to integrate single-cell RNA

sequencing (scRNA-seq) and bulk RNA sequencing (bulk RNA-seq) data, using single-cell data to identify Endothelial-associated transcriptomic changes during the progression from cirrhosis to HCC, and using bulk data to validate these findings in independent cohorts, while developing predictive models for early risk assessment.

Materials and **Methods:**
We integrated scRNA-seq (GSE136103, GSE202642 and GSE242889) and bulk RNA-seq datasets (GSE25097 and GSE14323) derived from cirrhosis and HCC liver tissues. hdWGCNA of Endothelial scRNA-seq data was used to identify Endothelial-associated gene modules whose hub genes significantly overlapped with Endothelial DEGs. Gene expression and network analyses were performed, and candidate genes were used to construct diagnostic models with LASSO regression. Model performance was evaluated using ROC curves.

Results:

We identified eleven Endothelial-associated genes, among which OIT3, FCN2, CXCL12, TYROBP, AKAP12, PDK4, FCN3, ALDOB and ALB were upregulated in HCC, ZFAND2A and GNG11 were downregulated in HCC, indicating an activated state of endothelial cells characterized by immunomodulation, metabolic reprogramming, and a pro-angiogenic niche that collectively fosters tumor progression and immune evasion. Based on these findings, we established predictive models using LASSO, which stratified cirrhotic patients from RNA-seq datasets into high- and low-risk groups according to cutoff values. The model demonstrated high diagnostic performance (AUC=0.998).

Conclusions:

This study highlights the critical role of Endothelial-associated transcriptomic remodeling in liver disease progression. The machine learning-based predictive model offers a promising approach for early diagnosis and clinical decision-making in patients with cirrhosis.

Keyword(s) : scRNA-seq; liver cirrhosis; hdWGCNA; machine learning; predictive model

1782 - An Evaluation of the Effectiveness of Entecavir in Patients with Acute Chronic Hepatitis B liver Failure

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Background:

Entecavir has been shown to be effective in randomized controlled trials in highly selected patients with hepatitis B virus infection. Entecavir does not cure HBV and may not prevent complications of hepatitis B such as cirrhosis of the liver or liver cancer. The dose is double for people who have persistent hepatitis viremia (the presence of virus in the blood) while taking lamivudine or have lamivudine resistance. It is recommended to take entecavir on an empty stomach, two hours before or after a meal. For some people, hepatitis B infection become chronic, meaning it lasts more than six months. Having chronic hepatitis B increases your risk of developing liver failure, liver cancer or cirrhosis a condition that permanently scars the liver. This study is aimed to evaluate the efficacy of Entecavir in chronic hepatitis B patients in the real-world setting.

Methodology:

In this study a total of 32 acute chronic Hepatitis B liver failure patients (age > 18 years with both sexes but male predominant) were included in Hepatology department of Bangladesh Medical University (BMU), Dhaka from January 2013 to December 2015. The patients were randomized into two groups: Entecavir group (N=32) and followed at least for 03 months.

Results:

Altered level of consciousness was present in 14 (43.8%) patients, moderate ascites in 26 (81.3%), and encephalopathy in 16 (50.0%) in the entecavir group. Differences were not statistically significant ($p > 0.05$). Mean laboratory values were: total count $10181.3 \pm 3594.1/\text{mm}^3$, serum bilirubin 22.0 ± 5.7 mg/dl, ALT mean rank 18.0 U/L, AST mean rank 17.8 U/L, prothrombin time 23.1 ± 4.2 sec, INR 2.0 ± 0.3 , serum albumin 2.3 ± 0.5 g/dl, serum creatinine 0.85 ± 0.31 mg/dl, serum sodium 134.1 ± 5.2 mmol/l, serum potassium 3.7 ± 1.0 mmol/l, MELD score 26.5 ± 2.0 , and Child-Pugh score 12.0 ± 1.5 ($p > 0.05$). After three months of entecavir therapy, mean serum bilirubin improved from 22.0 ± 5.7 to 5.1 ± 1.7 mg/dl; INR from 2.0 ± 0.3 to 1.4 ± 0.2 ; serum albumin from 2.3 ± 0.5 to 3.1 ± 0.3 g/dl; Child-Pugh score from 12.0 ± 1.5 to 9.3 ± 0.9 ; and MELD score from 26.5 ± 2.0 to 17.0 ± 2.1 . Negative mean rank ALT was 4.0 U/L (sum of rank 8.0); positive

mean rank and sum of ranks were 0. Differences were statistically significant ($p < 0.05$).

Conclusion:

Entecavir is very potent anti-HBV drug with a high genetic barrier to resistance, highly effective in lamivudine-naïve CHB patients and most promising for their long-term treatment but not very suitable for CHB patients harboring LAM resistant HBV mutants.

Keyword(s) : Entecavir, hepatitis B, HBV

1783 - LLGL1 Loss Attenuates Hippo Signaling and Sustains Pro oncogenic YAP Activity in Hepatocellular Carcinoma Cells

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Background / Hypothesis: Liver cancer is the sixth most frequently diagnosed malignancy worldwide but persists as the third leading cause of cancer-related deaths. Approximately 85% of primary liver tumors are hepatocellular carcinoma (HCC). Dysregulation of the Hippo signaling pathway is a well-established driver of HCC development and progression. LLGL scribble cell polarity complex component 1 (LLGL1), a conserved regulator of epithelial polarity, has been implicated in Hippo pathway modulation across several cancer types; however, its mechanistic role in HCC remains largely undefined. Given the central role of Hippo signaling in controlling proliferation and tissue homeostasis, defining LLGL1-dependent regulatory mechanisms may provide new insight into polarity-signaling crosstalk in HCC. In this study, we sought to investigate how LLGL1 influences Hippo pathway activity in HCC cells.

Materials and Methods: LLGL1 knockout (KO) Huh-7 cells were established via CRISPR/Cas9n-mediated gene disruption. Hippo signaling activation was induced by plating cells at high confluency. Expression levels of core Hippo pathway proteins were analyzed by Western blotting and quantified across biological replicates. All experiments were performed under standardized culture conditions to ensure reproducibility, and statistical analyses were performed with $p < 0.05$ considered significant.

Results: Protein analyses revealed that key molecular markers of Hippo pathway activation [phosphorylated (p)-MOB1 (Thr35), p-LATS1/2 (Ser909/Ser872), p-YAP (Ser127), p-TAZ (Ser89), and p-YAP (Ser397)] were

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significantly upregulated in confluent control cells ($p < 0.05$ for all), confirming robust pathway activation under high-density conditions. LLGL1 deficiency markedly altered this response. Total MOB1 levels were reduced 2.4-fold in LLGL1 KO cells compared with controls ($p = 0.0003$). Furthermore, p-TAZ (Ser89), a post-translational modification that promotes cytoplasmic retention and functional inactivation of TAZ, was reduced 1.4-fold in LLGL1 KO cells compared to controls ($p = 0.03$). In contrast, levels of p-YAP (Ser397), a phosphorylation event associated with proteasomal degradation of YAP, were not significantly altered in LLGL1 KO cells ($p = 0.58$), suggesting impaired YAP turnover and sustained YAP transcriptional activity.

Conclusions: This study provides the first evidence that LLGL1 contributes to Hippo tumor-suppressive signaling in HCC cells. Loss of LLGL1 leads to decreased MOB1 and reduced p-TAZ (Ser89) levels, together with unchanged p-YAP (Ser397), collectively indicating attenuated Hippo pathway activity and persistent pro-oncogenic YAP signaling. These findings position LLGL1 as a potential regulator of Hippo-mediated growth control in HCC and highlight the importance of polarity-Hippo pathway interactions in liver tumor biology.

Keyword(s) : CRISPR/Cas9, Hepatocellular carcinoma, Hippo pathway, LLGL1

1785 - Automatic Detection of Hepatocellular Carcinoma on MRI Using a Lesion Aware Fine Tuning Framework

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Background / Hypothesis: Automated segmentation of hepatocellular carcinoma (HCC) on MRI can substantially improve the efficiency of clinical workflows and imaging research. However, small HCCs (< 3 cm) and atypical HCCs often present low contrast, blurred boundaries, and non-classic enhancement patterns, making them prone to being missed by deep learning models.

Materials and Methods: This study included contrast-enhanced MRI examinations of 263 patients with HCC. An nnUNetV2-based model was developed for automatic liver and tumor segmentation, with a specific focus on improving the recall of small and atypical lesions. An initial model was trained using the full dataset to ensure robust performance for large lesions. Subsequently, 26 cases containing small-volume or

atypical HCCs were used for targeted fine-tuning by employing lesion-centered patch sampling and resampling strategies, while inheriting the weights of the initial model to enhance sensitivity to small tumor foci.

Results: The fine-tuning strategy significantly enhanced the model's detection capability for small and atypical HCC. The overall model Dice score improved from 0.803 before fine-tuning to 0.906, while the tumor category Dice score substantially increased from 0.6369 to 0.8423. After fine-tuning, the tumor IoU rose from 0.576 to 0.765, indicating a notable improvement in the model's recall ability for small lesions. Meanwhile, the liver segmentation performance remained stable, demonstrating that the fine-tuning strategy targeting small lesions did not compromise the segmentation accuracy of the overall structure.

Conclusions: By combining small-lesion resampling, lesion-centered patch augmentation, and nnUNetV2-based weight-inheritance fine-tuning, the proposed model achieves substantially improved recall of small and atypical HCCs while maintaining excellent liver segmentation accuracy. This strategy provides a practical and clinically valuable approach for enhancing early HCC detection on MRI.

Keyword(s) :

1798 - East Asian subgroup analysis of the global Phase 3 GLISTEN study of linerixibat for the treatment of cholestatic pruritus in patients with primary biliary cholangitis (PBC)

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Background / Hypothesis: Cholestatic pruritus in PBC is common, debilitating, and negatively impacts quality of life. In the global Phase 3 GLISTEN study (NCT04950127), a statistically significant

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improvement in pruritus and pruritus-related sleep interference was observed with linerixibat, a minimally absorbed ileal bile acid transporter inhibitor in registration phase for the treatment of pruritus in PBC. Here, we report the results for the East Asian subgroup (EAS; China and Japan) and for all participants in GLISTEN.

Materials and Methods:
In this double-blind, randomised, placebo-controlled study, adult patients with PBC and moderate-to-severe pruritus received oral linerixibat 40 mg or placebo twice daily. Stable background therapy was allowed. Pruritus severity and pruritus-related sleep interference were self-assessed daily using a 0–10 numerical rating scale. Primary endpoint: change from baseline in monthly itch score (worst weekly score of each month) over 24 weeks. Secondary endpoints included change from baseline in monthly sleep score (worst weekly sleep score of each month) over 24 weeks.

Results:

In total, 238 patients were randomised, including 72 in the EAS. Over 24 weeks, linerixibat resulted in significantly greater pruritus improvement than placebo (least-squares [LS] mean change: –2.86 vs –2.15, $p=0.0013$ overall; –2.63 vs –1.71 in EAS). After 24 weeks, the observed mean (SD) changes from baseline in pruritus were –3.66 (2.50) for linerixibat and –2.82 (2.32) for placebo overall, and –3.41 (2.23) versus –2.03 (1.61) in EAS. At Week 24, a ≥ 3 -point reduction in pruritus was achieved by 56% of patients on linerixibat versus 43% on placebo overall, and by 55% versus 34% in EAS. Improvement in pruritus with linerixibat was rapid and superior to placebo (LS mean change at Week 2: –1.78 vs –1.07, $p=0.0002$ overall; –1.56 vs –0.66 in EAS). Linerixibat significantly improved pruritus-related sleep interference over 24 weeks versus placebo (LS mean change: –2.77 vs –2.24, $p=0.024$ overall; –2.18 vs –1.61 in EAS). Gastrointestinal adverse events (AEs) were reported more frequently with linerixibat than placebo; diarrhoea occurred in 61% versus 18% overall and 74% versus 19% in EAS (mostly mild to moderate). Treatment discontinuation due to diarrhoea occurred in 4% of patients on linerixibat overall and 0% in EAS.

Conclusions:

In patients with PBC and moderate-to-severe pruritus, linerixibat rapidly and significantly improved pruritus and pruritus-related sleep interference versus placebo. Although gastrointestinal AEs were more frequent with

linerixibat, they were manageable and rarely led to discontinuation. Efficacy, safety, and tolerability of linerixibat in the EAS were consistent with the overall results.

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Keyword(s) : cholestatic pruritus, East Asian, worst itch, primary biliary cholangitis, sleep interference, numerical rating scale, IBAT, linerixibat, Japan, China

1802 - Hesperidin inhibits HBV via promoting macrophage M1 polarization through activation of the NF κ B CXCL TNF α pathway

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Background / Hypothesis:Chronic hepatitis B (CHB) seriously threat human health worldwide. Although antiviral drugs were commonly used in treatment of CHB clinically, they cannot completely eliminate the virus. Macrophages, as antigen-presenting cells, are an important component of innate immunity. Their polarization towards M1 can inhibit HBV infection by promoting the expression of cytokines such as TNF α . Hesperidin exhibits significant antiviral activity via promoting polarization towards M1 of macrophage.

Materials and Methods:Construct a mouse model of HBV infection by injecting rAAV8-HBV1.3 adenovirus into the tail vein of mice. Enzyme linked immunosorbent assay was used to detect the levels of HBsAg and HBeAg in the serum of HBV infected mice, and real-time qPCR was used to detect the expression levels of macrophage polarization related factors in the liver of HBV infected mice. Application of transcriptomics technology and bioinformatics analysis enrichment analysis to screen and predict potential pathways and targets for anti HBV effects of Hesperidin polarized macrophages. Differential gene expression was validated on THP-1 cell lines using PCR and ELISA. ELISA was used to detect HBsAg and HBeAg levels in HepG2.2.15 cells after differential gene intervention, Western blot was used to validate potential pathways, and PCR was used to validate differential gene expression in vivo.

Results:Enzyme linked immunosorbent assay detected that hesperidin can significantly reduce the levels of HBsAg and HBeAg in the serum of HBV infected mice. Real time qPCR results showed that Hesperidin can promote macrophage polarization towards M1 phenotype and inhibit cell polarization towards M2

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phenotype in the liver of HBV infected mice. Compared with the control group, hesperidin treatment affected the expression of 233 genes, of which 122 genes were upregulated and 111 genes were downregulated. The GO and KEGG pathway analysis results indicate that the IL-17 signaling pathway regulated by chemokines including CXCL and TNF α is significantly enriched. CXCL do not directly reduce the levels of HBsAg and HBeAg, but TNF α can inhibit their production, while CXCL can promote secretion of TNF α in macrophage. At the same time, through positive feedback regulation, CXCL activate NF κ B, further upregulated levels of TNF α . Conclusions: Hesperidin reduces the levels of HBsAg and HBeAg in animal serum and increases the expression of genes related to M1 polarization in macrophages in the liver. Hesperidin increases the levels of CXCL and TNF α production by activating the NF κ B pathway, thereby exerting anti HBV effects.

Keyword(s) : Chronic hepatitis B, macrophage, M1 polarization, hesperidin, CXCL

1804 - LOXL1 G153D Mutation Impairs Liver Fibrosis Regression by Enhancing Extracellular Matrix Stabilization

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Background / Hypothesis: Lysyl oxidase-like 1 (LOXL1) is a copper-dependent enzyme essential for extracellular matrix (ECM) cross-linking, contributing to tissue remodeling and fibrotic processes. Although LOXL1 mutations have been shown to enhance ECM cross-linking in glaucoma, their involvement in liver fibrosis remains insufficiently understood.

Materials and Methods: Genomic DNA from 168 patients with chronic hepatitis B (CHB)-related liver fibrosis was analyzed to identify LOXL1 single-nucleotide polymorphisms (SNPs) and assess their association with fibrosis progression and regression. To validate functional relevance, heterozygous Loxl1 G153D mutant mice were generated and exposed to carbon tetrachloride (CCl₄) to induce liver fibrosis, followed by spontaneous regression after withdrawal. The biological impact of the Loxl1 G153D mutation was further examined using an integrated

multi-omics strategy, including decellularized liver proteomics, transcriptomics, and metabolomics.

Results:

Patients carrying the heterozygous LOXL1 G153D mutation exhibited a significantly lower fibrosis reversal rate after 78 weeks of antiviral therapy (AVT) compared with non-mutant patients (26.3% vs. 52.4%, $p = 0.025$). Post-AVT, mutation carriers showed higher serum ALT and GGT levels, increased liver stiffness measurements (LSM), and elevated Ishak fibrosis scores. In vivo, Loxl1 G153D mice demonstrated greater inflammatory infiltration, lipid accumulation, and pronounced perisinusoidal fibrosis during spontaneous regression following CCl₄-induced injury. Mechanistically, metabolism-related pathways were predominantly activated during fibrosis progression, whereas inflammation-associated pathways were enriched during regression. Decellularized liver mass spectrometry further showed that the Loxl1 G153D mutation enhances ECM cross-linking and stabilization. In hepatic stellate cells (HSCs), the mutation promoted LOXL1 protein translocation to the cell membrane and extracellular compartment, markedly increasing HSC migration and contractility. Collectively, these findings indicate that the LOXL1 G153D mutation enhances extracellular LOXL1 secretion, strengthens ECM cross-linking and deposition, and disrupts fibrosis regression.

Conclusions:

Our findings demonstrate that the heterozygous LOXL1 G153D mutation is associated with impaired liver fibrosis regression following AVT in patients with CHB. This clinical association is supported by mechanistic evidence from Loxl1 G153D mutation mouse models, revealing a previously unrecognized role of LOXL1 in shaping the hepatic fibrosis regression microenvironment.

Keyword(s) : liver fibrosis; regression; extracellular matrix; chronic hepatitis B; hepatic stellate cells

1805 - Clinical Validation and Diagnostic Value Assessment of Plasma GSTM3 as a Novel Biomarker for Liver Fibrosis

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Background: Liver fibrosis is a critical pathological

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process in the progression of chronic liver disease to cirrhosis. Its dynamic reversible nature offers important opportunities for clinical intervention. However, the mechanisms underlying fibrosis reversal remain unclear, and the lack of accurate non-invasive diagnostic biomarkers significantly hinders early intervention and treatment monitoring. Our previous study in a mouse model of liver fibrosis demonstrated that GSTM3 expression was significantly elevated during fibrosis progression and gradually decreased during reversal, suggesting its close association with fibrosis severity and potential involvement in its regulation. This study aims to evaluate the clinical feasibility and diagnostic value of plasma GSTM3 as a novel biomarker for liver fibrosis.

Methods: Liver tissue specimens from 48 patients with different stages of liver fibrosis were collected from Beijing Ditan Hospital and categorized into S0, S1-2, and S3-4 groups based on the modified Scheuer scoring system. Immunohistochemistry was used to detect GSTM3 expression in liver tissues to validate its correlation with fibrosis progression. To further explore its potential as a non-invasive biomarker, plasma samples from 180 liver fibrosis patients and 50 healthy controls were analyzed for GSTM3 levels using ELISA.

Results: Immunohistochemistry revealed a significant positive correlation between GSTM3 expression in liver tissues and fibrosis severity. Weak expression was observed in the S0 group, markedly elevated levels in the S1-2 group, and the highest expression in the S3-4 group, demonstrating a gradient increase with fibrosis progression. Plasma GSTM3 levels also showed a stage-dependent increase: 8.14 ng/ml in healthy controls, 14.97 ng/ml in the S1-2 group, and 36.20 ng/ml in the S3-4 group. This trend of increasing GSTM3 levels with advancing fibrosis remained consistent across individual stages and different grouping methods. ROC curve analysis indicated that plasma GSTM3 levels possess good diagnostic performance for significant liver fibrosis ($S \geq 2$), with an AUC reaching 0.8213, suggesting its considerable potential as a non-invasive diagnostic biomarker for assessing the progression of liver fibrosis.

Conclusion: Clinical data confirm that GSTM3 exhibits a clear stage-dependent increase with liver fibrosis progression, consistent with prior animal experiments. These findings suggest that GSTM3 not only plays a critical role in liver fibrosis development but also holds promise as a non-invasive biomarker for clinical diagnosis and disease monitoring.

Keyword(s): Liver Fibrosis, GSTM3

1812 - Clinical characteristics and influencing factors of porto sinusoidal vascular disease patients with

metabolic dysfunction associated steatotic liver disease

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Background: / **Hypothesis:**
The confluence of porto-sinusoidal vascular disease (PSVD) and metabolic dysfunction-associated steatotic liver disease (MASLD) presents a complex clinical scenario, potentially accelerating disease progression. This study aims to explore the Clinical characteristics and influencing factors of PSVD patients with MASLD.

Materials and Methods:
This retrospective study included patients who underwent liver biopsy were diagnosed with PSVD at Beijing Ditan Hospital as shown in Figure 1. Patients were stratified into two groups: patients with MASLD (PSVD with MASLD group), and patients without MASLD (PSVD without MASLD group). Clinical features and risk factors were analyzed to identify features specific to PSVD with or without MASLD. Nomogram model was used for diagnostic prediction of PSVD patients with MASLD.

Results:
A total of 285 PSVD patients undergoing liver biopsy were enrolled, including PSVD with MASLD group (n=91) and PSVD without MASLD group (n=194). The proportion of females was higher than that of males in PSVD with MASLD group. Compared to PSVD without MASLD group, PSVD with MASLD group exhibited higher levels of NE, hemoglobin (HGB), platelet (PLT) count, alanine aminotransferase (ALT), albumin (ALB), total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL), prothrombin activity (PTA), and controlled attenuation parameter (CAP), while lower high density lipoprotein (HDL), international normalized (INR), FIB-4, and liver stiffness measurement (LSM), as shown in Table 1. Liver pathology findings revealed a higher proportion of nodular regenerative hyperplasia ($p=0.000$) in PSVD with MASLD group, while lower obliterative portal venopathy ($p=0.000$), irregular dilation of hepatic sinusoids ($p=0.000$), and perisinusoidal fibrosis ($p=0.011$) (Table 2). Stratified analysis of the two patient groups showed that PSVD patients with MASLD had significantly higher proportions of males, diabetes, hyperlipidemia, $PLT \geq 150 \times 10^9/L$, $LSM \geq 10$ kPa, and $FIB-4 > 3.25$, compared to PSVD patients without MASLD (Table 3). Univariate and multivariate logistic regression analysis revealed that HGB [OR 1.039, 95%CI (1.019-1.060), $p=0.000$], ALT

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[OR 1.011, 95%CI (1.001-1.020), $p=0.026$], TC [OR 1.925, 95%CI (1.330-2.786), $p=0.001$], HDL [OR 0.166, 95%CI (0.063-0.434), $p=0.001$], CAP [OR 1.011, 95%CI (1.002-1.020), $p=0.019$], and Whether with hyperlipidemia [OR 9.491, 95%CI (4.398-20.481), $p=0.000$], emerged as independent risk factors for PSVD patients with MASLD, as shown in Table 4. Nomogram model revealed that AUC 0.880, 95% CI (0.839-0.921), and the specificity of this model is 0.897 (Figure 2).

Conclusions:

HGB, ALT, TC, HDL, CAP, and hyperlipidemia were independent influencing factors for PSVD patients with MASLD. Nomogram model can effectively predict the disease risk of PSVD patients in the population with MASLD.

Keyword(s) : porto-sinusoidal vascular disease, metabolic dysfunction-associated steatotic liver disease, hyperlipidemia, controlled attenuation parameter, nomogram model

1813 - Unmasking the unknown the unexpected discovery of hepatic epithelioid hemangioendothelioma

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Background / Hypothesis: Epithelioid hemangioendothelioma (EHE) is a rare tumor of vascular endothelial cell origin that is composed of epithelioid and histiocyte-like vascular endothelial cells in mucus or fibrotic matrix. It affects the liver, lungs, mediastinum and other sites, but the liver is the most commonly involved site.

Materials and Methods: A 63-year-old female patient, diagnosed case of Papillary Thyroid Carcinoma s/p total thyroidectomy and radioactive iodine had an incidental finding of hypovascular liver nodules at segments VIII and II on whole abdomen CT scan requested for monitoring of renal cyst Bosniak 1. She was asymptomatic and physical examination was unremarkable. Further investigation was done with PET CT scan and MRI followed by percutaneous needle biopsy of the lesion. She then underwent hepatic resection of the involved segments. After discussion with a multidisciplinary team, no further treatment was given and active surveillance was done.

Results: PET CT scan and MRI were equivocal for metastasis. Histologic examination of percutaneous

needle biopsy specimen revealed a vascular tumor for which hemangioendothelioma is considered. Liver volumetric study was done with noted adequate future liver remnant. Hepatic resection of the involved segments and subsequent histopathologic examination showed 1.5 and 2cm at segment II and VIII, respectively with vascular invasion and mitotic activity that ranges from 0-1/10 high power field. A repeat MRI of the liver with Primovist after 6 and 12 months revealed no new lesion.

Conclusions: Hepatic EHE is an extremely rare tumor that can only be confirmed by histologic examination. Given its rarity, there are no standardized guidelines for its treatment. The treatment options are broad which include chemotherapy/radiotherapy, ablation, surgery, liver transplantation as well as active surveillance. Compared with other malignant tumors of the liver, HEHE has a good prognosis.

Keyword(s) : Hepatic epithelioid hemangioendothelioma; Hepatic resection; Case report

1815 - Efficacy of Silybin Phospholipid Complex in Metabolic Associated Fatty Liver Disease

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Background / Hypothesis: silybin phospholipid complex is a commonly used hepatoprotective agent in clinical practice. In vitro studies have demonstrated its efficacy in alleviating hepatic steatosis and liver fibrosis. This study aimed to clinically evaluate the effectiveness of silybin phospholipid complex in improving liver fat content and liver fibrosis in patients with fatty liver disease.

Materials and Methods:

Patients with metabolic-associated fatty liver disease (MAFLD) admitted to Beijing You'an Hospital and the First Hospital of Lanzhou University between 2020 and 2025 were retrospectively enrolled in this study. They were divided into the treatment group and the control group based on whether they received monotherapy with Silybin phospholipid complex. Changes in liver fat content, liver fibrosis indices, and liver function parameters were analyzed between the two groups. The primary outcome was a reduction in liver fat content, and the secondary outcomes were decreases in liver fibrosis indices and liver function parameters.

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Results:

A total of 176 patients were included, with 122 in the treatment group and 54 in the control group. Overall, after 3 and 6 months of continuous medication, the main liver function indicators in the treatment group—ALT (Diff0-3:-10, Diff0-6:-10) and AST (Diff0-3:-7, Diff0-6:-5)—showed a decreasing trend compared to baseline, and the magnitude of reduction at each time point was greater than that in the control group—ALT (Diff0-3:-5, Diff0-6:-1) and AST (Diff0-3:-2.5, Diff0-6:-0.5). The controlled attenuation parameter values in the treatment group (3M: 268.39 ± 59.92 , 6M: 273.02 ± 51.63) were lower than those in the control group (3M: 289.73 ± 61.82 , 6M: 283.91 ± 47.27), and the difference from baseline in the treatment group showed an overall increasing trend (Diff 0-3M: -12.5, Diff 0-6M: -16.5). The treatment group demonstrated a greater reduction in liver stiffness measurement (LSM) values from baseline at both 3 and 6 months (Diff 0-3M: -0.55, Diff 0-6M: -0.3) compared to the control group (Diff 0-3M: -0.1, Diff 0-6M: -0.1).

Conclusions: Silybin phospholipid complex holds promise for reducing liver fat content and alleviating the severity of liver fibrosis in patients with metabolic-associated fatty liver disease, providing a potential therapeutic option for clinical management of this disease.

Keyword(s) : Silybin phospholipid complex; Metabolic-associated fatty liver disease; Controlled attenuation parameter; Liver stiffness measurement; Liver function; Liver fibrosis.

1816 - The roles of THBS2 SPP1 axis in hepatic stellate cells activation and liver fibrogenesis

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Background / **Hypothesis:** Thrombospondin-2 (THBS2) is markedly upregulated in fibrotic livers and correlates with fibrosis stage, independent of etiology. This study aimed to elucidate the role and underlying mechanisms of hepatic stellate cell (HSC)-derived THBS2 in HSC activation and the progression of liver fibrosis.

Materials and **Methods:**

We generated HSCs-specific Thbs2 knockout mice (Thbs2 Δ HSC) and HSCs-specific human THBS2 knock-in mice (hTHBS2 KI HSC). Liver fibrosis was induced by intraperitoneal injection of carbon tetrachloride (CCl₄) or by bile duct ligation (BDL). The role of the THBS2-SPP1 axis in fibrogenesis was investigated using a dual targeting approach: extracellular SPP1 was neutralized by intraperitoneal administration of an anti-mouse SPP1 antibody (inVivoMAb), while intracellular SPP1 was suppressed using AAV6-GFAP-shSpp1; both strategies were validated in parallel in vitro. Furthermore, we identified and validated a THBS2-specific inhibitor and combined it with an SPP1-targeting small molecule inhibitor to evaluate the therapeutic potential of dual THBS2-SPP1 targeting for the treatment of liver fibrosis.

Results:

Thbs2 Δ HSC mice exhibited significantly reduced hepatic inflammation and fibrosis in both CCl₄- and BDL-induced models. Conversely, hTHBS2KI HSC mice displayed markedly aggravated hepatic inflammation and fibrotic progression. Mechanistic studies revealed that Thbs2 knockdown in HSCs substantially suppressed the expression of the pro-fibrotic effector Spp1, while THBS2 overexpression robustly enhanced both SPP1 expression and its secretion—findings consistently validated in vitro. Notably, molecular interaction assays confirmed that THBS2 directly interacts with SPP1 in a dimeric configuration. Furthermore, we found intraperitoneal administration of inVivoMAb anti-mouse SPP1 neutralizing antibody and AAV6-GFAP-shSPP1-mediated Spp1 silencing significantly attenuated fibrotic progression in CCl₄-treated hTHBS2KI HSC mice, demonstrating distinct anti-fibrotic efficacy through coordinated blockade of extracellular SPP1 signaling and intracellular SPP1 biosynthesis. Additionally, we identified a novel THBS2 inhibitor through integrated virtual drug screening and surface plasmon resonance (SPR) validation. Pharmacodynamic evaluations confirmed the compound's potent anti-fibrotic effects in both CCl₄- and 3,5-diethoxycarbonyl-1,4-dihydrocollidine (DDC)-induced liver fibrosis mouse models, with significant improvements in histopathological features.

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Conclusions:

Our findings demonstrate that HSC-derived THBS2 drives liver fibrogenesis through the regulation of SPP1. Concerted targeting of the THBS2-SPP1 axis presents a promising therapeutic strategy for significantly mitigating liver fibrosis.

Keyword(s) :

1818 - Selective Inhibition of Hepatic Stellate Cell Derived Microfibril associated glycoprotein 4 Attenuates Liver Fibrosis

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Background / **Hypothesis:**
Microfibril-associated glycoprotein 4 (MFAP-4) is a member of the fibrinogen-related protein superfamily and a crucial component of the extracellular matrix (ECM). However, its role in the pathogenesis of liver fibrosis remains unclear.

Materials and **Methods:**
Liver biopsy samples from cirrhotic patients with various etiologies and multiple murine models of liver fibrosis were analyzed to determine the expression pattern and cellular origin of MFAP-4. To investigate the in vivo role of MFAP-4 in hepatic stellate cells (HSCs), Mfap4^{fl/fl} mice were administered with an adeno-associated virus serotype 6 (AAV6) vector encoding lecithin-retinol acyltransferase (Lrat)-Cre, achieving HSC-specific deletion of Mfap4 in the carbon tetrachloride (CCl₄) fibrosis model. In vitro, MFAP4 was either overexpressed or silenced in LX-2 cells to assess its effects on HSC activation and fibrogenic properties. Subsequent immunofluorescence staining and collagen gel contraction assays were performed to delineate the mechanisms by which MFAP-4 regulates liver fibrogenesis. To evaluate the therapeutic potential of MFAP-4 inhibition, AAV6 vectors expressing Mfap4-targeting shRNAs under either the U6 promoter or the HSC-specific glial fibrillary acidic protein (GFAP) promoter were administered via tail vein injection in CCl₄ and bile duct ligation (BDL) mouse models. In parallel, a selective small-molecule inhibitor of MFAP-4 was identified through virtual screening and subsequently validated for its antifibrotic efficacy.

Results:

MFAP-4 was predominantly enriched in activated HSCs

and significantly upregulated in advanced liver fibrosis in both humans and mice. HSC-specific suppression of Mfap4 in Mfap4^ΔHSC mice markedly attenuated fibrosis and intrahepatic inflammation after CCl₄ treatment. Mechanistically, MFAP-4 promoted Collagen I and elastin deposition, as well as gel contraction, thereby enhancing ECM stiffness-mediated HSC activation. Notably, selective knockdown of Mfap4 by either AAV6-GFAP-shMfap4 or AAV6-U6-shMfap4 significantly retards liver fibrosis in CCl₄ and BDL models. BJFSH102, a selective small-molecule MFAP-4 inhibitor, ameliorated liver fibrosis by disrupting MFAP-4 interactions with elastin and collagen I in CCl₄ models.

Conclusions:

Our findings indicate that HSC-derived MFAP-4 drives liver fibrogenesis by promoting elastin and collagen I deposition and reinforcing ECM stiffness-mediated HSC activation. Selective inhibition of HSC-derived MFAP-4 effectively attenuates fibrosis, representing a promising therapeutic strategy for liver fibrosis.

Keyword(s) : Liver fibrosis; Hepatic stellate cells; Microfibril-associated glycoprotein 4; Extracellular matrix remodeling

1819 - Impact of the expanded program on immunization on Hepatitis B elimination efforts Insights from a Turkish reference center

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Background / **Hypothesis:**
Hepatitis B vaccination was introduced globally in the early 1980s. In Türkiye, the first major step toward hepatitis B immunization occurred in 1998, when the vaccine was incorporated into the national childhood immunization schedule within the Expanded Program on Immunization (EPI). Subsequent catch-up campaigns enabled a substantial proportion of individuals born after 1991 to be vaccinated. These efforts are expected to reduce the prevalence of hepatitis B surface antigen (HBsAg) and increase the prevalence of anti-HBs antibodies. This study aimed to assess age-specific patterns of HBsAg and anti-HBs results among individuals tested at Ankara Bilkent City Hospital, a

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major tertiary referral center in Türkiye.

Materials and Methods: Ankara Bilkent City Hospital is a 4,050-bed tertiary reference hospital with a high patient volume. Demographic data and laboratory results of patients who underwent HBsAg or anti-HBs testing between 1 January 2022 and 31 December 2023 were retrieved from electronic medical records. Patients were grouped into 10-year age categories. For each individual, the first available HBsAg (qualitative) and anti-HBs (quantitative) result was recorded. Anti-HBs titers ≥ 10 mIU/mL were considered positive. Age-specific HBsAg and anti-HBs positivity rates were calculated. For anti-HBs titers, the 5th, 10th, 25th, 50th, 75th, 90th, and 95th percentiles were determined.

Results:

Among 443,030 individuals tested for anti-HBs, 2.8% had invalid results; among 558,730 tested for HBsAg, 2.1% were invalid. Of the valid results, 48.0% (206,866/430,832) were positive for anti-HBs, and 2.3% (12,319/546,879) were positive for HBsAg. Age-specific positivity rates are shown in Table 1. The median anti-HBs titer (Q1–Q3) was 7.39 (<3.10–135.39) mIU/mL. Percentile distributions of anti-HBs titers by age group are presented in Table 2.

Conclusions:

A pronounced decrease in HBsAg positivity is observed beginning in the 30–39-year age group, with an even sharper decline in the 0–19-year group, reflecting the impact of universal childhood vaccination after 1998. Although anti-HBs positivity declines rapidly after the first decade of life, this decrease is only modestly reflected in HBsAg positivity until the fourth decade. These findings support the concept that individuals who achieve seroconversion remain protected against hepatitis B despite declining antibody levels, unless they become immunosuppressed. Sustained immunization efforts remain essential for achieving hepatitis B elimination goals in Türkiye, an intermediate-endemic country.

Keyword(s): Hepatitis B, childhood vaccination, anti-HBs titer

1820 - Downstaging by Atezolizumab plus Bevacizumab in Advanced Hepatocellular Carcinoma Identifying Candidates for Conversion Therapy Based on Tumor Biology and Vascular Invasion Patterns

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Background / Hypothesis: Advances in systemic therapy for hepatocellular carcinoma (HCC) have enabled a subset of patients to achieve sufficient tumor response to allow conversion therapy or even cancer-free status. However, the real-world frequency of downstaging and the tumor characteristics associated with successful conversion remain under-investigated. This study aimed to evaluate downstaging outcomes during first-line atezolizumab plus bevacizumab (Atezo+Bev) therapy and to identify clinical factors that contribute to downstaging.

Materials and Methods: We retrospectively analyzed patients with advanced HCC and evaluable intrahepatic lesions who received first-line Atezo+Bev between October 2020 and September 2024. Cumulative downstaging rates were calculated using two criteria: (1) the tumor-based Borderline Resectable (BR) classification, assessing transitions from BR2 to $\leq$ BR1 and from BR1 to resectable (R), and (2) the Milan criteria, evaluating transitions from outside to within the Milan criteria. Factors associated with downstaging were explored using univariable Cox proportional hazards models.

Results:

A total of 178 patients were included. Child–Pugh class A accounted for 157 patients, while BCLC stage B and C were observed in 68 and 94 patients, respectively. Median overall survival for the entire cohort was 15.7 months (95% CI, 13.6–18.5 months). At treatment initiation, 132 patients were classified as BR2 and 30 as BR1. Downstaging from BR2 to $\leq$ BR1 occurred in 25 patients, and BR1 to R in 5 patients. The cumulative BR-based downstaging rates were 14.3% at 6 months and 31% at 12 months. According to the Milan criteria, 170 patients were initially outside the criteria, and 11 achieved downstaging to within the criteria, corresponding to cumulative rates of 7.2% at 6 months and 11% at 12 months.

Univariable Cox analysis identified “solitary intrahepatic tumor with major vascular invasion (Vp4 or Vv3)” as a strong factor associated with successful downstaging

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(HR 3.73, 95% CI 1.59–8.76, $p=0.0025$). In these tumors, downstaging was typically driven not by substantial reduction in tumor size but by decreased arterial enhancement and regression of the tumor thrombus.

Conclusions:

Downstaging during Atezo+Bev therapy was most frequently observed in cases of solitary HCC with advanced macrovascular invasion, where regression of vascular invasion played a dominant role. In contrast, transitions into the Milan criteria were limited, indicating that large-scale tumor shrinkage is uncommon. Solitary tumors with severe vascular invasion may represent promising candidates for conversion therapy during Atezo+Bev treatment.

Keyword(s) : Hepatocellular carcinoma, Downstaging, Atezolizumab plus bevacizumab, Conversion therapy, Macrovascular invasion

1821 - Adolescent MASLD Incidence Trends 1990–2023 Global and National Perspectives

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Background / Hypothesis: Understanding the current burden of adolescent MASLD is essential for effective prevention strategies. This study explores global trends and country-specific trajectories of MASLD incidence among adolescents aged 15–19 years.

Materials and Methods: Age-specific incidence rates (per 100,000) from 1990 to 2023 were obtained from the GBD 2023 database. Country-level incidence rates for 2023 were mapped, and temporal trends were summarized for the top-30 countries, stratified by SDI.

Results:

From 1990 to 2023, the global incidence (both sexes) increased from 866 to 1,085 per 100,000 (+25.2%). Male incidence rose from 1,017 to 1,273 (+25.2%), while female incidence increased from 710 to 887 (+24.9%). A distinct geographic gradient emerged in 2023: the highest incidence levels were found in North Africa and the Middle East, while several Asian countries reported comparatively lower rates. Among the top-30 countries, high-middle SDI nations experienced the steepest and most sustained increases, while many high-SDI countries showed moderate growth with plateaus appearing in the late 2010s. Low and low-middle SDI countries demonstrated gradual but consistent

increases.

Conclusions:

The global incidence of adolescent MASLD has increased significantly since 1990, with the highest rates observed in North Africa and the Middle East and notable growth in high-middle SDI countries. The persistent male excess and widespread upward trends emphasize the urgent need for adolescent-targeted prevention and risk factor control. These trends call for future research to quantify risk contributions and develop comprehensive risk profiles

Keyword(s) : MASLD incidence;GBD;adolescent

1822 - Blood biomarkers for non invasive diagnosis of advanced fibrosis or compensated cirrhosis in chronic hepatitis B

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Background / Hypothesis: Currently, hepatitis B virus infection remains a major driver of the global burden of liver disease, contributing to approximately 30% of liver fibrosis/cirrhosis cases and accounting for one-quarter of liver disease-related deaths. The definitive diagnosis of liver fibrosis/cirrhosis still relies on invasive liver biopsy; however, its application in clinical practice is limited due to invasiveness, poor acceptability, high cost, and significant variability. This study aims to identify reliable blood biomarkers or their combinations for the non-invasive diagnosis of chronic hepatitis B with advanced fibrosis or decompensated cirrhosis.

Materials and Methods: We enrolled 252 patients with hepatitis B virus infection from Peking University People's Hospital, Beijing, China. Among them, 132 patients were initially recruited with histologically confirmed advanced fibrosis or decompensated cirrhosis, while the remaining 120 were HBV-infected individuals without fibrosis. The team measured and calculated nine common clinical variables: age, gender, ALT, AST, TBIL, DBIL, PLT, LSM, FIB-4, as well as three candidate serum biomarkers: Golgi protein 73 (GP73), cytokeratin-18 fragment M30 (CK-18 M30), and cytokeratin-18 fragment M65 (CK-18 M65). Using logistic regression and random forest approaches, potential non-invasive diagnostic biomarkers and their combined panels were identified.

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Results:We found that LSM (AUC: 0.898; 95% CI: 0.859–0.938), GP73 (AUC: 0.832; 95% CI: 0.783–0.881), FIB-4 (AUC: 0.719; 95% CI: 0.655–0.782), AST (AUC: 0.684; 95% CI: 0.619–0.75), CK18-M65 (AUC: 0.676; 95% CI: 0.609–0.743), CK18-M30 (AUC: 0.653; 95% CI: 0.585–0.722), and ALT (AUC: 0.62; 95% CI: 0.551–0.689) exhibited certain discriminative ability for patients with chronic hepatitis B (CHB) and advanced fibrosis or decompensated cirrhosis. A panel comprising three parameters (GP73, LSM, FIB-4), designated as N3, differentiated patients with CHB and advanced fibrosis/decompensated cirrhosis from the control group with an area under the receiver operating characteristic curve (AUROC) of 0.908. Furthermore, a panel incorporating seven parameters (GP73, LSM, FIB-4, ALT, AST, CK18-M30, CK18-M65), referred to as N7, identified patients with advanced fibrosis/decompensated cirrhosis with an AUROC of 0.912.

Conclusions:In summary, we have developed a promising blood-based biomarker and corresponding panels for the non-invasive diagnosis of chronic hepatitis B with advanced fibrosis or decompensated cirrhosis. This development holds the potential to assist clinicians in reducing unnecessary liver biopsies.

Keyword(s) : Chronic Hepatitis B ; Fibrosis ; Cirrhosis ; Blood Biomarkers ; Non-invasive Diagnosis

1824 - Utility of EUS guided Liver Biopsy in Liver Disease.

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Background / **Hypothesis:**
Endoscopic Ultrasound guided Liver Biopsy has many advantages over the current standard of care and appears to have fewer contraindications and adverse events. Percutaneous Liver Biopsy has been associated with side effects like pain abdomen and bleeding seen up to 19 % of cases. Endoscopic ultrasound (EUS) guided liver biopsy (EUS-LB) can overcome many limitations of percutaneous liver biopsy and Trans-Jugular Liver Biopsy. It is commonly performed using 19G EUS fine-needle biopsy (FNB) needles.

Materials and **Methods:**
The primary aim of this study was to detect the adequate biopsy rate with EUS guided Liver Biopsy and to detect rate of successful pathological diagnosis. This is a retrospective observational study. Patients undergoing EUS-LB during the study period of January 2023 to June 2025 were taken up for this study. EUS-guided liver biopsies were performed using linear echo endoscope PENTAX Defina EPK 3000 and 19G FNB needle (Acquire TM, Boston Scientific, United States). Procedure was performed by an experienced endoscopist, and all liver biopsy samples were taken using “wet technique” from left lobe of liver using Trans-gastric route. Tissue accusation was performed with single puncture and 3-4 back and forth motions of needle.

Results:

Total 30 patients were taken up for the study. Median age of study population was 59 years, out of which 14 patients were males and 16 were females (46.6% and 53.3%). Adequate biopsy rate (according to European Association for the Study of Liver Disease criteria) was 70%.

Rate of conclusive diagnosis was 96.6% (29/30). The commonest diagnosis was metastatic adenocarcinoma (23.3%), followed by autoimmune hepatitis (13.3%). Mean CPT (Complete Portal Tract) was 7.5 with SD of 2.94 and mean TSL (Total Sample length) was 1.547cm with SD of 0.667 (CI being 95%). Most common adverse effect encountered was pain abdomen (10%).

Conclusions:

EUS-Liver Biopsy technique is characterized by relatively high diagnostic yield and safety. EUS guided Liver Biopsy can have a high histological yield when performed using proper technique. Considering its safety and high rate of biopsy, it can be used as an alternative procedure to Percutaneous Liver Biopsy.

Keyword(s) :

1832 - An Unexpected Turmoil Hepatitis B Reactivation with Atypical Serology Following Neoadjuvant Capecitabine Oxaliplatin in an Unsuspected Patient with Gastric Adenocarcinoma A Multidisciplinary Management Complexity

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Background / **Hypothesis:**
Hepatitis B virus (HBV) reactivation is a well-recognized adverse event of immunosuppressive therapy, particularly with certain chemotherapeutic agents and biologics. Continued studies have stratified these agents whether they induce low, moderate or high risk for reactivation based on HBsAg and AntiHBc status. Reactivation can lead to acute liver injury, thereby leading to compromise and delay in oncologic and curative surgery. HBV reactivation induced by Capecitabine and Oxaliplatin, however, is rarely reported.

It is therefore hypothesized that even low-risk agents can cause severe HBV reactivation, particularly in patients without prior HBV screening, emphasizing the need for universal pre-chemotherapy screening and prompt antiviral therapy.

Materials and **Methods:**
A 67-year-old male patient newly diagnosed with non-metastatic gastric adenocarcinoma was given neoadjuvant capecitabine-oxaliplatin for a contemplated curative resection. Patient had new onset jaundice and body weakness post treatment prompting workup with liver biochemical tests consistent with acute liver injury, hence, hepatitis work-up was done including HBsAg, anti-HBc, anti-HBs, HBeAg, anti-HBe, anti-HCV, anti-HAV IgM, HBV DNA, along with liver ultrasound. Upon confirmation of HBV reactivation, tenofovir alafenamide was initiated, with serial monitoring of liver biochemistry and HBV DNA levels performed to assess treatment response.

Results:
Workup was done 4 weeks post Capecitabine and Oxaliplatin treatment revealing acute liver injury with elevated AST (1,359 U/L), ALT (827 U/L) and total bilirubin (252 µmol/L). Ultrasound revealed mild parenchymal changes with no focal lesions. Hepatitis serology: HBsAg (+), antiHBc(-), anti-HBs (-), HBeAg (+), anti-HBe (-), anti-HBc IgM (-), and HBV DNA: 8,560,000 IU/mL, consistent with HBV reactivation in the setting of immunosuppression (an atypical serology). Tenofovir was promptly initiated. Surgery was deferred due to recent acute liver injury. Patient had upper GI bleeding controlled with tumor-directed radiotherapy (5 sessions). Subsequent studies showed decreasing trends of AST, ALT and bilirubin, with HBV DNA decreased to 489,000 IU/mL 4 weeks post-treatment. Subsequently, patient had (+)Anti-HBc and (+)anti-HBe on repeat serology. Patient was eventually cleared for curative surgery after nutritional optimization.

Conclusions:
Although considered low-risk, Capecitabine and Oxaliplatin can cause severe HBV reactivation causing acute liver injury, consequently resulting to delayed oncologic or surgical treatment. Early initiation of antiviral therapy can help stabilize liver function and allow timely continuation of curative cancer treatment. This highlights the importance of universal HBV screening before chemotherapy, including use of low-risk agents particularly in endemic regions.

Keyword(s) : Hepatitis B virus (HBV) reactivation; Atypical serology; Capecitabine-Oxaliplatin; Gastric adenocarcinoma

1834 - The national burden of acute liver failure from 2019 to 2022 A Thailand nationwide population based cross sectional time series study
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Background / **Hypothesis:**
Acute liver failure (ALF) is a rare but life-threatening condition with extremely high short-term mortality. Despite its substantial impact on the healthcare system, the national burden of ALF in Thailand remains poorly defined. This study evaluated the prevalence, etiologies, complications, and predictors of 30-day mortality among Thai patients with ALF from 2019 to 2022.

Materials and **Methods:**
We conducted a nationwide, population-based, cross-sectional time-series study using Thailand's National Health Security Office (NHSO) database. Adults (≥18 years) with ALF were identified using ICD-10 codes K72.0 and K71.11; patients with cirrhosis (K74,650,658,659,703,729,766,767, I850-851) were excluded. Baseline characteristics, comorbidities,

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etiologies, complications, and procedure claims were extracted from inpatient and outpatient/emergency encounters. Age- and sex-standardized incidence and 30-day mortality rates were analyzed using descriptive statistics and temporal trend analyses with LOESS smoothing. Predictors of 30-day mortality were assessed using multivariable Cox proportional hazards models.

Results:

A total of 7,957 patients with ALF were identified, of whom 57.3% (n=4,560) died within 30 days. Those who died were older (57.2 ± 19.1 vs 47.9 ± 20.3 years; $P < 0.001$) and more often male (65.6%). Most cases occurred in the northeastern (38.6%) and central (22.2%) regions. Etiologies were largely unspecified (82.3%), followed by acute viral hepatitis infection (7.9%), mushroom poisoning (3.9%), alcohol-associated liver disease (ALD) (1.9%), and autoimmune hepatitis (1.8%); drug-related ALF accounted for 1.0%. Acute renal failure (41.6%) and sepsis (13.2%) were the most common complications. Liver transplantation (LT) was performed in only 0.2% of all cases. In multivariable analyses, prior ALF (within one year) was associated with increased 30-day mortality (aHR 1.51; 95% CI 1.29-1.77; $P < 0.001$). Etiologies associated with higher mortality included drug/mushroom poisoning (aHR 1.77), liver and bile duct cancer (aHR 1.35), ALD (aHR 1.19) and acute viral hepatitis (aHR 1.02). Cerebrovascular disease (aHR 0.81; $P = 0.002$) were unexpectedly associated with lower mortality risk.

Conclusions:

ALF is a major public health challenge in Thailand, with >57% short-term mortality and extremely limited access to LT. High-risk etiologies, including viral, toxin-related, and alcohol-associated ALF, contribute substantially to the national mortality burden. Improved early detection pathways, expanded access to advanced liver support therapies, and increased liver transplantation capacity are urgently needed to reduce ALF-related mortality in Thailand.

Keyword(s) : Acute liver failure; Epidemiology; Mortality; Etiology; Thailand

1836 - Trends in hepatitis B related diseases in Asia 1990 2023 an analysis of the Global Burden of Disease 2023 update

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Background / Hypothesis: Although hepatitis B prevention and control measures have been continuously enhanced and vaccination rates have steadily risen, the disease burden among children and adolescents remains significant. The prerequisite for achieving the WHO's goal of completely eliminating viral hepatitis by 2030 is Asia's accurate understanding of the current disease burden, thereby providing a basis for formulating and optimizing intervention measures.

Materials and Methods: Based on the Global Burden of Disease (GBD) 2023 dataset, we extracted data on the burden of hepatitis B in Asia, including acute and chronic forms, as well as overall disease burden, covering incidence, prevalence, mortality, disability-adjusted life years (DALYs), and their corresponding age-standardized rates. To account for differences in geographic distributions within the population, we calculate age-standardized rates based on the age-specific data extracted from the GBD 2023 dataset. To further assess temporal patterns across different age groups, we estimated the average annual percent change (AAPC).

Results: From 1990 to 2023, the overall age-standardized prevalence rates (ASPR) of the total burden related to hepatitis B in Asia declined (AAPC = -4.84%, 95%CI: -5.12% to -4.55%), along with significant reductions in age-standardized mortality and DALYs. Throughout this period, ASPR was consistently higher in men than in women, although women exhibited a faster rate of decline. Age-specific analysis revealed the steepest decline in prevalence among children aged 5-9 years (AAPC = -6.46%, 95%CI: -7.22% to -5.69%). The prevalence of hepatitis B-related liver cancer declined at the slowest rate and even showed an upward trend in the 15-19 age group (AAPC = 0.19%, 95%CI: 0.07% to 0.30%). In contrast, other hepatitis B-related diseases demonstrated an overall decreasing disease burden. Notably, Bhutan experienced the greatest decline in ASPR (AAPC = -8.18%, 95%CI: -8.65% to -7.71%) but also reported the only increase in the age-standardized DALYs (ASDR) in the region (AAPC = 1.04%, 95%CI: 0.39% to 1.70%).

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Conclusions:The Asia burden and trends of hepatitis B decreased significantly in children and adolescents, exhibiting regional and national variations. The decline in the prevalence of hepatitis B-related liver cancer has been slow, indicating the need for timely and targeted interventions to address emerging risks among adolescents.

Keyword(s) : hepatitis B virus; prevalence; incidence; Asia; epidemiology; public health

1837 - Early Ablation for Sub centimeter Hepatocellular Carcinoma Divergent Outcomes in Primary vs Recurrent Tumors over 10 Years

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Background / Hypothesis:
Background: The optimal management of sub-centimeter hepatocellular carcinoma (HCC), particularly early ablation versus observation, and outcome differences between primary and recurrent tumors, remains unclear.
Purpose: To compared long-term outcomes of ultrasound-guided percutaneous thermal ablation for sub-centimeter (≤ 1.0 cm) versus 1.0-2.0 cm HCC, analyzing primary and recurrent cases separately.

Materials and Methods:
A retrospective review of 850 patients (494 primary, 356 recurrent HCC) treated with ultrasound-guided percutaneous thermal ablation from January 2014 to June 2024. Overall survival (OS) and recurrence-free survival (RFS) were evaluated using Kaplan-Meier and log-rank tests. Sliding diameter-threshold survival analysis was performed.

Results:
Primary patients with ≤ 1.0 cm tumors showed 100% OS across all follow-up points (1-, 3-, 5-, 7-, and 10 years), with the difference from the 1.0-2.0 cm group (98.5%, 85.2%, 70.6%, 59.7%, and 48.6%) becoming significant only at 10 years ($P=0.047$). Conversely, recurrent HCC patients with ≤ 1.0 cm tumors had significantly better OS than larger recurrent tumors at all timepoints (10-year: 78.9% vs 30.4%; all $P<0.05$). Using 1.5 cm cutoff, primary patients with tumors ≤ 1.5 cm showed improved long-term OS, but this was not observed for recurrent HCC. Sliding-threshold analysis identified survival inflection points at 1.2 cm for primary and 0.9 cm for recurrent tumors.

Conclusions:

Primary and recurrent sub-centimeter HCCs respond differently to early thermal ablation. For recurrent ≤ 1.0 cm lesions, immediate ablation offers clear, size-dependent survival benefits. Conversely, the therapeutic benefit for primary sub-centimeter HCC appears delayed. These distinct patterns suggest that immediate treatment is warranted for recurrent sub-centimeter HCC, while a surveillance strategy may be currently acceptable for primary sub-centimeter HCC.

Keyword(s) : sub-centimeter hepatocellular carcinoma, thermal ablation, prognosis

1843 - Gut Liver Axis Mechanisms Underlying Spontaneous Reversal of Liver Fibrosis A Gut Microbiota Metabolomics Analysis

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Background / Hypothesis:Reversing liver fibrosis is crucial for improving the prognosis of chronic liver disease. The gut microbiota-mediated regulation of the liver-gut axis plays a significant role in this process, yet its dynamic changes and underlying mechanisms during spontaneous fibrosis reversal remain unclear. This study aims to systematically reveal the dynamic patterns of the liver-gut axis during spontaneous fibrosis reversal by integrating gut microbiome and metabolomics data from a mouse model of liver fibrosis reversal. It further explores the intrinsic connections between microbiota and metabolism.

Materials and Methods:Intestinal contents were collected from mice in the control group, liver fibrosis model group, and spontaneous reversal groups at 4 and 12 weeks. Gut microbiota structure was analyzed via 16S rRNA gene sequencing. Metabolic profile dynamics were assessed using non-targeted metabolomics. Comprehensive bioinformatics methods were employed for differential analysis, pathway enrichment analysis, and correlation integration.

Results:Analysis of gut microbiota 16S rRNA sequencing revealed significant differences in β -diversity among the model group, the 4-week reversal group, and the 12-week reversal group, indicating that gut microbiota composition continues to evolve throughout different stages of the reversal process. At the phylum level, the relative abundance of Bacteroidetes decreased throughout the reversal process, while that of Firmicutes

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significantly increased in the 4-week reversal group. Genus-level analysis revealed a significant increase in the relative abundance of Akkermansia in the 12-week reversal group, while the family Fusulinaceae reached its peak in the 4-week reversal group. Non-targeted metabolomics analysis showed that multiple metabolic pathways were significantly altered in the reversal groups compared to the model group. The linoleic acid and α -linolenic acid metabolic pathways exhibited higher impact values during the later stages of reversal; Pathways including taurine metabolism, biotin metabolism, and ascorbic acid metabolism remained continuously active throughout reversal. Integrated analysis of gut microbiota and metabolomics revealed significant correlations between changes in specific bacterial groups and metabolite levels, indicating a close association between microbial restructuring and metabolic reprogramming during liver fibrosis reversal.

Conclusions: By integrating 16S rRNA sequencing with non-targeted metabolomics, this study reveals the dynamic evolution of gut microbiota restructuring and host metabolic profiles during spontaneous reversal of liver fibrosis. The reversal of liver fibrosis may depend on microbiota-metabolism coupling regulation processes dominated by specific functional microbial communities. This work provides theoretical support for elucidating the "liver-gut axis" mechanism underlying fibrosis reversal and lays the theoretical foundation for developing gut microbiota-based intervention strategies for liver fibrosis.

Keyword(s) :

1846 - Patient Derived Liver Organoids as a Translational Platform to Validate Targeted Nanoparticles for Image Guided Liver Cancer Surgery

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Background / Hypothesis: Accurate visualization of liver cancer margins remains a major challenge during surgical resection. Glypican-3 (GPC3) is a tumor-associated antigen highly expressed in hepatocellular carcinoma (HCC) and certain metastatic liver cancers but absent in normal hepatocytes. This study established a patient-derived

liver organoid platform to validate the tumor selectivity of a GPC3-targeted nanoparticle (TJ12-NP) and to bridge its application to image-guided liver cancer surgery.

Materials and Methods: TJ12-NP was synthesized and labeled with Cy5 or indocyanine green (ICG) for different experimental settings. Cy5-labeled TJ12-NP was used to evaluate tumor-specific uptake in patient-derived organoids representing normal liver, HCC, and colorectal liver metastasis (CRLM). Organoid identities were confirmed by marker profiling through immunostaining. Confocal microscopy was applied to visualize nanoparticle localization, and live-dead assays assessed cytocompatibility. ICG-labeled TJ12-NP was further tested in vivo in Huh-7-bearing nude mice for fluorescence imaging and fluorescence-guided tumor resection.

Results:

Cy5-TJ12-NP exhibited strong and specific accumulation in GPC3-positive HCC organoids and comparable uptake in CRLM organoids, whereas minimal signal was observed in normal liver organoids, confirming tumor-selective targeting. The nanoparticles were well tolerated by organoid. In vivo, ICG-TJ12-NP enabled high-contrast tumor visualization, achieving a markedly higher tumor-to-background ratio compared with non-targeted controls and facilitating complete fluorescence-guided resection.

Conclusions:

This study establishes patient-derived liver organoids as a robust translational platform for validating molecularly targeted nanoparticles. The integration of organoid based specificity testing with preclinical imaging demonstrates a clinically relevant pathway toward precision, image-guided liver cancer surgery.

Keyword(s) : Liver cancer; Nanoparticles; GPC3; Organoid; Image-guided surgery

1848 - Metastatic Liver Involvement from Primary Adamantinoma like Ewing Sarcoma of the Lung in an Elderly Male A Rare Case Presentation
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Background / Hypothesis: Ewing sarcoma is a malignant small round blue cell tumor that primarily affects bone and soft tissue in adolescents and young adults. Adamantinoma-like Ewing sarcoma (ALES) is a rare

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subtype characterized by epithelial differentiation and immunoreactivity for CD99 and cytokeratins. While conventional Ewing sarcomas have a well-documented pattern of metastatic spread—most commonly to the lungs and bone marrow—primary lung ALES is rare, and hepatic metastasis from this origin is exceptionally uncommon.

This report documents a case of hepatic metastasis from a primary pulmonary ALES in a 64-year-old male. To our knowledge, delayed liver metastasis from lung ALES in an elderly patient is exceedingly rare and has limited representation in published literature.

Materials and Methods:We report the case of a 64-year-old male with a prior diagnosis of Adamantinoma-like Ewing sarcoma of the left lung confirmed by core needle biopsy. Two years after initial diagnosis and treatment, the patient presented with epigastric pain, abdominal bloatedness, and shortness of breath. Physical examination revealed hepatomegaly, and imaging via four-phase dynamic computed tomography (CT) of the abdomen demonstrated a large, hypervascular mass in the right hepatic lobe, raising suspicion for malignancy. Fine needle aspiration (FNA) under ultrasound guidance was performed. Histopathological examination revealed round cells arranged in disorganized sheets and aggregates with mild cytologic atypia, scant cytoplasm, and a mitotic count of 0–1 per high power field. These findings were morphologically consistent with metastatic Ewing sarcoma and matched the patient's prior pulmonary ALES diagnosis.

Results: This case illustrates a rare occurrence of liver metastasis developing two years after initial diagnosis of pulmonary ALES. Most hepatic Ewing sarcomas reported in literature are primary hepatic tumors or part of disseminated disease from bone primaries. Delayed solitary liver metastasis from a lung primary is a particularly rare presentation.

Radiologically, the liver mass mimicked hepatocellular carcinoma due to its vascular profile. However, in patients with prior history of ALES or similar malignancies, metastasis should be included in the differential diagnosis. Histopathological and immunohistochemical correlation is indispensable in confirming the diagnosis.

Conclusions: This case highlights a rare instance of delayed hepatic metastasis from pulmonary Adamantinoma-like Ewing sarcoma in an elderly male. Clinicians should maintain a high index of suspicion for metastatic disease in patients with prior rare malignancies who present with new abdominal symptoms. Comprehensive imaging and tissue diagnosis, including histopathology and immunohistochemistry, remain the cornerstone of

accurate identification and management of such atypical metastatic presentations.

Keyword(s) : Adamantinoma-like Ewing Sarcoma, pulmonary sarcoma, liver metastasis, small round blue cell tumor

1849 - Yangyin Fuzheng Jiedu Prescription Reduces Recurrence Risk in Patients with Small Hepatocellular Carcinoma after Minimally Invasive Treatment a Pragmatic Randomized Controlled Trial

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Background / Hypothesis:Small Hepatocellular Carcinoma (HCC) often has no symptoms, so it is easily overlooked. Minimally invasive treatment of postoperative recurrence of small HCC is still a clinical problem. This study aims to evaluate the clinical efficacy and safety of Yangyin Fuzheng Jiedu Prescription (YFJP) in the treatment of postoperative recurrence of small HCC through a pragmatic randomized controlled trial (pRCT).

Materials and Methods:A total of 300 newly diagnosed small HCC patients were recruited in this study and randomly divided into control group and YFJP group, with 150 patients in each group. The control group received conventional minimally invasive treatment methods, including radiofrequency ablation and hepatic artery chemoembolization. On the basis of conventional treatment, the YFJP group was combined with YFJP treatment, which was added or decreased according to the symptoms. The treatment period is 12 months, and the overall survival (OS) and recurrence free survival (RFS) of the two groups of patients were followed up and observed.

Results:We were pleasantly surprised to find that the average RFS of the YFJP group patients was 8 months, significantly higher than the control group's 3 months ($P < 0.05$). The postoperative recurrence rate of the YFJP group was significantly lower than that of the control group (30% vs. 52.6%, $P < 0.05$). Secondly, YFJP can significantly reduce the levels of AFP, ALB, and TBIL (all $P < 0.05$). We also found that $ALT \leq 50$ IU/L, $AFP \leq 400$ ng/mL, and receiving TACE combined with RFA treatment are the advantageous populations for the treatment of small HCC with YFJP. In addition, there was

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no significant difference in the incidence of treatment-related adverse events between the two groups ($P > 0.05$).

Conclusions:The YFJP treatment has shown good efficacy and safety in improving the postoperative RFS and reducing the recurrence rate of small HCC patients.

Keyword(s) :

1851 - Inhibiting PPM1D facilitates the phosphorylation and oligomerization of VDAC2 to elicit immunogenic tumor destruction by activating cGAS STING IFN response

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Background / Hypothesis: Immune checkpoint blockade (ICB) represents an important therapeutic approach for hepatocellular carcinoma (HCC), while resistance to ICB treatment remains challenging. Here we reported oncoprotein protein phosphatase magnesium-dependent 1 δ (PPM1D) as a novel driver for HCC immunotherapy resistance.

Materials and Methods:In vitro and in vivo experiments were constructed to examine the function of PPM1D on HCC behavior. Immunoprecipitation (IP) and mass spectrometry were used to find the interactor of PPM1D. Co-IP, immunofluorescence and DUOLink PLA were used to verify the interaction. ChIP-PCR and luciferase assays were used to confirm the binding of transcription factor on PPM1D promoter.

Results: We showed that tumor cell PPM1D impaired CD8⁺ T-cell cytotoxicity and contributed to HCC progression. Furthermore, we found that tumor cell elevated PPM1D expression in response to effector CD8⁺ T lymphocyte activation via TNF α -NF- κ B signaling. Mechanistically, PPM1D de-phosphorylated mitochondrial outer membrane component VDAC2 to prevent VDAC2 oligomerization in tumor cells, thereby maintaining mitochondrial homeostasis and limiting mitochondrial DNA leakage-triggered, cGAS-STING activation. Blocking PPM1D-dependent VDAC2 dephosphorylation disrupted mitochondrial integrity,

recruiting Parkin to target PKM2 for K48-linked polyubiquitination and proteasome degradation. Also, inhibiting PPM1D improved the cytotoxicity of infiltrating CD8⁺ T lymphocytes and synergized with PD-1 inhibitor in preclinical HCC models without causing remarkable toxicity.

Conclusions: PPM1D orchestrates mitochondrial homeostasis in HCC cells through reducing VDAC2 oligomerization that limits mtDNA leakage to prevent cGAS-STING-IFN response and anti-tumor immunity, revealing PPM1D as a promising therapeutic target.

Keyword(s) : Liver cance; Oncogene;Checkpoint blockade;Mitochondrial homeostasis

1857 - Therapeutic Role of N Acetylcysteine in Acute Liver Failure Secondary to Anti Tuberculosis Drug Induced Liver Injury Insights From Indonesia

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Background:

Tuberculosis (TB) remains a major public health problem in Indonesia, one of the world's highest burden TB countries. First-line anti-tuberculosis drugs (ATDs) are highly effective, but frequently associated with drug-induced liver injury (DILI). Severe hepatotoxicity may progress to acute liver failure (ALF), a life-threatening condition associated with high mortality. N-acetylcysteine (NAC), a glutathione precursor with antioxidant and vasodilatory properties, is well established in acetaminophen-induced ALF and has shown emerging benefit in non-acetaminophen etiologies, including ATD-induced liver injury (AT-DILI). This case series presents two patients with AT-DILI progressing to ALF who received intravenous NAC.

Case

presentation:

Case 1: A 50-year-old female with meningoencephalitis, spondylodiscitis, sacroiliitis TB, complicated by type 2 diabetes mellitus and rheumatoid arthritis developed syncope, progressive jaundice, pruritus, and rash while on ATDs. Laboratory evaluation showed coagulopathy (PT ratio: 2.8), hyperbilirubinemia (Total: 18.00 mg/dl, direct: 14.10 mg/dl, indirect: 3.90 mg/dl) and mildly elevated transaminases (ALT 50 U/L; AST 19 U/L) consistent with AT-DILI. Intravenous NAC was administered following the standard protocol: a loading dose of 150 mg/kg over 1 hour, followed by 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours. Continuous infusion of 100 mg/kg/day was maintained until

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improvement in encephalopathy and coagulation parameters. NAC therapy resulted in a gradual decline in coagulation parameter (PT ratio: 1.46), bilirubin (Total: 4.80 mg/dl, direct: 3.71 mg/dl, indirect: 1.23 mg/dl) and transaminase level (ALT 34 U/L and AST 16.5 U/L) within ten days, followed by complete clinical recovery. Case 2: A 32-year-old female with pulmonary TB presented with loss of consciousness consistent with grade III hepatic encephalopathy. She appeared pale and jaundice but hemodynamically stable. Laboratory findings showed severe coagulopathy (PT ratio: >5.9), markedly elevated bilirubin (Total: 18.9 mg/dl, direct: 9.62 mg/dl, indirect: 9.28 mg/dl) and transaminase levels (ALT: 311 U/L; AST: 1023 U/L). Intravenous NAC was administered according to protocol. Transaminases improved gradually (ALT 59 U/L and AST 389 U/L), while coagulation parameter (PT ratio: 6.14) and bilirubin levels (Total: 19,60 mg/dl) remained persistently elevated during observation.

Conclusion:

These cases highlight the potential therapeutic role of N-acetylcysteine in the management of ALF secondary to AT-DILI. NAC administration was associated with improvement of clinical symptoms, coagulation, bilirubin, and liver enzyme profiles. In TB-endemic and resource-limited settings where liver transplantation is not widely available, NAC may offer a valuable therapeutic option. Further prospective studies are required to clarify the efficacy, optimal dosage, and timing of NAC administration in non-acetaminophen ALF, particularly in AT-DILI.

Keyword(s) : N-acetylcysteine, acute liver failure, drug-induced liver injury, anti-tuberculosis therapy, hepatotoxicity

1861 - Test Treat! A Small Step For The World A Big Step For Us; Hepatitis C Microelimination Efforts in a Small City in Türkiye

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Background / Hypothesis
Ordu Province, located in northern Türkiye, has a population of 770,000 and a surface area of 5,952 km². As highlighted in the WHO 2024 Global Hepatitis Report, global coverage of viral hepatitis is very low. Until the introduction of DAA regimens in our country,

there were a significant number of patients who did not respond to interferon therapy. DAA therapy was provided to a waiting group, treating a substantial patient pool. In this ongoing process, it became necessary to identify patients requiring new diagnoses and treatment. In this study, we present our efforts to increase diagnosis and treatment rates in our province. Materials and Methods
Our HCV elimination project, which adopted a "Test-Treat" strategy, was launched in early 2022. Key objectives included education, collaboration with local health administrators, testing strategies, and treatment phases.

- Education phase: Hospital clinicians, clinicians administering immunosuppressive therapy, primary care physicians, and the public (social media, media, print publications).
 - Collaboration with local health administrators: Meetings and symposiums, revisions to the Hospital Information Management System (HIS). Joint projects to increase rapid testing capacity in the field.
 - Test!: Testing opportunities, screening of family medicine patient records, a reflex testing strategy in laboratory services, and screening of at-risk groups.
 - Treatment!: Referring patients to treatment, active patient monitoring, and ensuring treatment continuity.
- Results

From 2016 to 2020, 174 patients were treated with genotype-specific DAA (SoF or PROD). Between 2020 and the present, 178 patients have received treatment. A total of 352 patients were treated with DAA. The number of anti-HCV and HCVRNA tests performed over the years is shown in the table (Table 1). Conclusions: The project we are working on aims to increase the number of tests in our province and treat positive cases. We must strive for HCV eradication. This effort must be undertaken in collaboration with primary care physicians and healthcare administrators. Global eradication can be achieved through efforts that begin in the smallest living spaces and grow like an avalanche. Our project is ongoing; we await the completion of our joint point-of-care testing projects with the Health Directorate, and the implementation of our screening strategies continues in the field.

Keyword(s) :

1867 - Identification of benefit subgroups from heterogeneous effects of adjuvant transarterial chemoembolization in hepatocellular carcinoma using causal model

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Background / Hypothesis: Adjuvant transarterial chemoembolization (TACE) after curative resection for hepatocellular carcinoma (HCC) shows conflicting results, likely due to the heterogeneity of treatment effects (HTEs). We aimed to assess HTE and develop a tool to identify patients most likely to benefit from adjuvant TACE.

Materials and Methods: We retrospectively included HCC patients from 10 tertiary centers (real-world cohort, n = 2,630) between 2012 and 2023 and an independent clinical trial (validation cohort, n = 328). After propensity score matching between patients with and without adjuvant TACE, the real-world cohort was defined as the development cohort (n = 1,632). HTE on overall survival (OS) was assessed using (1) subgroup analyses, (2) Cox regression (risk-based), and (3) causal survival forest (effect-based) in the development cohort. Individual treatment effects were used to inform a policy tree (iTACE), which was validated across various cohorts. Survival analyses were performed using Cox regression and log-rank tests.

Results: A total of 2,958 patients were included (median age, 58 years; 83.6% male). After matching, adjuvant TACE showed no significant average treatment effect on OS [hazard ratio (HR) 0.85, 95% confidence interval (CI) 0.70-1.03, P = 0.1061]. Consistent HTEs were observed across subgroup-, risk-, and effect-based analyses. Patients with microvascular invasion, younger age, or corona enhancement on imaging derived greater survival benefits (P < 0.05). The iTACE stratified patients into recommendation subgroups; those in the rec-adjuvant TACE subgroup achieved better survival (HR 0.54, 95% CI 0.43-0.69), while the rec-active surveillance subgroup had poorer outcomes (HR 1.88, 95% CI 1.32-2.67) in the development cohort (P < 0.05). The stratified treatment effect was confirmed in the validation and real-world cohorts (P < 0.05).

Conclusions: Adjuvant TACE demonstrates significant HTE following curative resection for HCC. An interpretable tool enables the individualized identification of patients likely to benefit from adjuvant TACE, providing a framework for postoperative management.

Keyword(s) : Hepatocellular carcinoma; Adjuvant treatment; Transarterial chemoembolization; Heterogeneity of treatment effect; Overall survival; Decision-making tool

1883 - Early Onset PSC With Recurrent Cholangitis Requiring Early Transplant Evaluation A Five Year Case Trajectory and Prelude to the Transplant Course

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BACKGROUND

Primary sclerosing cholangitis (PSC) is a progressive cholestatic liver disease that typically follows an unpredictable course. Early-onset PSC is particularly uncommon and often demonstrates a more aggressive trajectory, especially when associated with ulcerative colitis (UC). Recurrent cholangitis, progressive stricturing, and rising biochemical markers may occur despite optimized endoscopic management, underscoring the need for timely transplant referral. This case documents a five-year clinical course of young-onset PSC culminating in early liver transplant evaluation, serving as the prelude to a forthcoming discussion on the transplant process in this patient.

CASE

A 23-year-old Filipino male was diagnosed with UC and PSC at age 18. Over five years, he developed intermittent abdominal pain and diarrhea, with imaging later revealing cholelithiasis. He underwent ERCP with sphincterotomy, biliary stent placement, and subsequent laparoscopic cholecystectomy. Persistent dominant strictures required scheduled ERCP with stent replacement every two years, unusually frequent for a patient this young and indicative of significant large-duct involvement.

PRESENTATION

His disease course was complicated by recurrent episodes of acute cholangitis, including infections involving *E. coli*, *Enterococcus faecalis*, and *Klebsiella* bacteremia, managed with stent exchange and intravenous antibiotics. During the current admission, he presented with a 1-day history of fever, vomiting, and malaise. Examination revealed mild scleral icterus. Laboratories showed marked hepatocellular injury (>10× ULN in AST/ALT), elevated ALP (271 U/L), GGT

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(418 U/L), and direct hyperbilirubinemia, although blood cultures remained negative.

MRCP revealed no biliary obstruction, yet demonstrated hepatomegaly, capsular retraction, and T2 hyperintense lesions in segments V and VIII, consistent with progressive PSC despite ductal patency. Notably, the patient's PIVKA-II level was elevated, raising concern for occult hepatobiliary neoplasia or advanced cholestatic dysfunction despite the absence of a detectable mass. He responded promptly to piperacillin-tazobactam with resolution of fever.

Given his recurrent cholangitis, progressive biochemical injury, and radiologic features suggestive of advancing disease, he was referred early for liver transplant evaluation at age 23. Once recovered from infection, comprehensive transplant work-ups and preoperative preparation were initiated, including laboratory staging, cardiopulmonary clearance, nutritional assessment, and psychosocial evaluation.

CONCLUSION

This case demonstrates the aggressive natural history of early-onset PSC and the limitations of long-term stent therapy in preventing progression. The combination of recurrent infections, rising liver enzymes without obstruction, and PIVKA-II elevation prompted early transplant referral. This report serves as the clinical foundation for a forthcoming follow-up detailing the patient's transplant work-up and perioperative course.

Keyword(s) : Primary sclerosing cholangitis (PSC), Ulcerative colitis (UC) , early-onset PSC, Recurrent cholangitis, Biliary stenting Progressive cholestatic liver disease

1886 - The role of Alix and VPS4A in autophagy and endosomal pathways and their relation to HBV replication

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Background / Hypothesis: Autophagic and endosomal

pathways coordinately contribute to hepatitis B virus (HBV) production. Alix and VPS4A are two important endosomal sorting complex required for transport (ESCRT) components involved in membrane scission. Previous studies showed that Alix and VPS4A participate in HBV replication and modulate the release of HBV particles. Yet, the mechanisms remain elusive. In the present study, we investigated the roles of Alix and VPS4A in HBV biogenesis in the context of endosomal membrane trafficking and autophagy. Materials and Methods: We examined the production, trafficking, and release of HBV particles by using gene silencing and overexpression of wild-type (WT) and dominant-negative (DN) mutants of Alix and VPS4A in HBV replication cell models. Results: Alix silencing increased the levels of intracellular HBV DNA and subviral particles (SVPs/HBsAg), extracellular HBsAg, and virions, while decreasing the secreted naked capsids. Alix silencing promotes HBsAg secretion along the early endosomes while it reduces HBsAg transported to late endosomes and autophagosomes. Alix silencing impairs autophagosome formation by activating the AKT/MTOR pathway and increasing autophagosome degradation in lysosomes. VPS4A silencing has minimal effects on HBV production, while DN VPS4A significantly blocks HBV secretion. DN VPS4A disrupts endosomal trafficking and HBsAg colocalization within early endosomes and late endosomes, while promoting autophagosome formation and lysosome activity, resulting in HBV degradation.

Conclusions: Our findings demonstrate that the endosomal pathway plays a critical role in HBV progeny secretion, particularly when lysosomal activity is suppressed. Conversely, increased lysosomal function drives HBV degradation through the autophagosome-lysosome pathway.

Keyword(s) :

1889 - Liver Fibrosis Explainable Deep learning Network for Automated Staging by Using Equilibrium Enhanced Phase MR Images

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Background / Hypothesis: Conventional non-invasive diagnostic modalities, including ultrasound-based liver stiffness measurement (LSM) and serological biomarkers, have been widely adopted in clinical practice. However, their diagnostic accuracy remains limited, often falling short in precisely assessing fibrosis severity. MRI-driven deep learning

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approaches has opened new avenues for non-invasive, quantitative evaluation of liver fibrosis. This study aimed to develop an nnU-Net model for automated liver segmentation and propose a novel explainable framework integrating multi-task contrastive learning with attention mechanisms to autonomously identify and visualize the discriminative MRI patches for fibrosis staging.

Materials and Methods: A total of 706 treat-naïve chronic hepatitis B patients were eligible for this retrospective study. 655 patients in center 1 between May 2015 and July 2022 were randomized into the training (424), validation (99) and internal test (132) set. 51 patients in center 2 between March 2018 and May 2023 were collected as an external test set. The reference standard was biopsy obtained within 6 months of the MR examinations. This study employed VGG-16 as the backbone architecture, designed to integrate contrastive learning for automated patch selection while simultaneously enhancing the network's discriminative capability for fibrosis classification. Input data were L2-normalized and included equilibrium enhanced phase MR images and LSM. An extraction function returned the top nine probability-ranked patches from the contrastive learning task along with clinically significant fibrosis classification predictions ($F \geq 2$). Segmentation performance was quantitatively evaluated using the Dice Similarity Coefficient (DSC) to measure spatial overlap between predicted and ground truth liver masks. All case predictions, labels, and AUCs were systematically recorded. The Integrated Discrimination Improvement (IDI) metric quantified the diagnostic enhancement of the patch-based model over LSM alone.

Results:

The nnU-Net model achieved a training set DSC of 0.95 for liver segmentation across 365 cases. For $F \geq 2$ fibrosis staging, the AUCs demonstrated sliding changes when averaging predictions from top-ranked patches in the internal test set: 0.818 (single best patch), 0.799 (top two), 0.779 (top three), 0.753 (top four), 0.723 (top five), with performance plateauing beyond five patches. The network achieved AUC values of 0.853 (training), 0.835 (validation), 0.818 (internal test), and 0.805 (external test) for $F \geq 2$ fibrosis staging using the "top discriminative patch". A significant difference was observed between the "top discriminative patch" model and the LSM-only approach ($P < 0.0001$), yielding an IDI

value of 0.287.

Conclusions: The proposed framework provides an accurate, automated, and explainable tool for liver fibrosis staging, and could add value to elastography.

Keyword(s) : Liver fibrosis; MRI; Deep learning; Diagnosis; Elastography

1890 - Deep Learning CT Signature for Predicting Occult Liver Metastases and Neoadjuvant Therapy Benefit in Pancreatic Ductal Adenocarcinoma A Multicenter Retrospective Study

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Background / Hypothesis: To develop a deep learning (DL) framework integrating pancreatic tumor and liver imaging features to predict occult liver metastases (OLM) in pancreatic ductal adenocarcinoma (PDAC), thereby guiding personalized neoadjuvant therapy (NAT).

Materials and Methods: In this multicenter retrospective study (2015-2022), 1,063 patients with PDAC were included: a training cohort ($n = 450$), internal validation cohort ($n = 113$), external validation cohort ($n = 342$), and an NAT cohort ($n = 158$). We developed a Mamba-based model that integrates multi-phase computed tomography features from both primary tumors and liver regions. Its performance was evaluated using the area under the receiver operating characteristic curves (AUCs). To assess clinical utility, survival outcomes were compared between NAT followed by surgery and upfront surgery groups based on model-defined risk stratification. Finally, radiotranscriptomics analysis was employed to elucidate the underlying biological insights.

Results: The Mamba-based model demonstrated robust performance for predicting the OLM in PDAC, with the AUCs of 0.890 (training), 0.820 (internal validation), and 0.806 (external validation), respectively. Model-defined high-risk patients had significantly shorter progression-free survival (PFS: HR = 1.95, $P < 0.001$) and overall survival (OS: HR = 1.96, $P < 0.001$), independent of clinicopathological factors. Notably, NAT conferred significant OS benefits exclusively in model-defined high-risk patients (17.43 vs. 34.10 months; $P < 0.001$), even after propensity score matching (18.03 vs. 34.10 months; $P = 0.003$), while low-risk patients showed no survival benefit. Furthermore, radiotranscriptomics also

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revealed the relative biological aggressiveness of high-risk patients.

Conclusions: The proposed Mamba framework enabled accurate prediction of OLM and may serve as a clinically actionable tool for identifying PDAC patients most likely to benefit from NAT.

Keyword(s) : Pancreatic ductal adenocarcinoma, Deep learning, Occult liver metastases, Neoadjuvant therapy, Radiotranscriptomics

1895 - Add on traditional Chinese medicine for hospitalized acute on chronic liver disease
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Background / Hypothesis

Acute-on-chronic liver failure (ACLF) carries high short-term mortality. Traditional Chinese Medicine (TCM) is increasingly used as an adjunctive therapy, yet its effectiveness in real-world settings remains insufficiently evaluated. We hypothesized that add-on TCM during hospitalization could improve survival and early clinical recovery in ACLF patients.

Materials and Methods

This retrospective cohort study included hospitalized ACLF patients at Beijing Ditan Hospital in 2023–2024 and 2018–2019. Patients received either standard Western medicine (WM) alone or WM plus TCM. Outcomes were assessed at 4, 12, and 24 weeks. The primary endpoint was short-term mortality; secondary endpoints included changes in liver function, coagulation parameters, inflammatory markers, and MELD and Child–Pugh scores. Baseline imbalances were minimized using inverse probability of treatment weighting (IPTW). Cox proportional hazards models were applied in unmatched and IPTW-adjusted analyses.

Results

Compared with WM alone, add-on TCM was associated with significant reductions in bilirubin levels, inflammatory markers, and severity scores at 4 weeks ($p < 0.05$), while no early improvements were observed in coagulation, liver enzyme, or protein levels ($p > 0.05$). TCM consistently demonstrated survival benefits:

unmatched Cox models showed reduced mortality at 4-, 8-, 12-, and 24-week follow-up ($HR \approx 0.20, 0.36, 0.40$, and 0.38 , respectively). IPTW-adjusted analyses yielded similar hazard ratios ($\approx 0.20, 0.37, 0.41$, and 0.39), indicating a 60%–80% reduction in mortality risk across follow-up periods.

Conclusions

Add-on TCM therapy may significantly reduce short-term mortality and improve bilirubin, inflammatory, and severity-related outcomes in ACLF patients. Consistent findings from both unmatched and IPTW-adjusted Cox models support TCM as a beneficial complementary treatment. Further prospective studies are warranted to confirm these effects.

Keyword(s) : traditional Chinese medicine, acute on chronic liver disease

1897 - Genotype Phenotype Correlation of ATP7B Mutations in Wilson Disease
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Background / Hypothesis:

Wilson disease (WD) is an autosomal recessive disorder of copper metabolism caused by mutations in the ATP7B gene. Patients exhibit significant variability in age of onset, affected organs, and disease severity. Clarifying the relationship between genotype and phenotype is essential for understanding WD heterogeneity.

Materials and Methods:

We performed ATP7B gene sequencing in 289 WD patients recruited from two centers in China, analyzed mutation types and homozygous/heterozygous distributions, and selected the five most frequent mutation combinations for further analysis. Skewed continuous variables were compared using the Kruskal-Wallis test, and categorical data were analyzed with the chi-square or Fisher's exact test. Clinical features—including age at diagnosis, serum ceruloplasmin (CER), 24-hour urinary copper, Kayser-Fleischer (K-F) ring presence, and clinical subtype—were compared among genotype groups. Regression models were used to assess interactions between genotype and age at diagnosis on the risk of cirrhosis and liver failure.

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Survival curves were employed to evaluate the prognostic differences of loss-of-function (LOF) mutations across distinct age subgroups.

Results:

The independent effect of genotype on cirrhosis risk approached statistical significance (LOF vs Missense mutation, $p=0.051$). Among diagnosis age <12 years old, patients carrying two 2-LOF mutations had the shortest median survival time (2.0 years), which was significantly worse than the 0-LOF group (7.0 years, $p=0.010$). However, in patients ≥ 12 years old, no significant differences in survival rates were observed among groups, suggesting that the negative impact of LOF mutations is more pronounced in early-onset patients. Patients carrying the p.T1106M/p.R778L combination showed significantly later age at diagnosis, higher CER levels, and slightly lower urinary copper, suggesting a milder clinical phenotype. K-F ring positivity and clinical subtype differed among genotype groups. Interaction analysis revealed that the rate of age-related cirrhosis progression in the p.T1106M/p.R778L group was slower than in the p.R778L/p.R778L reference group, whereas interactions with liver failure risk were not significant.

Conclusions:

The interaction between genotype and age at diagnosis has a regulatory effect on cirrhosis risk, but shows limited impact on liver failure. Loss-of-function mutations significantly negatively affect prognosis in early-onset patients (<12 years), though this effect is attenuated in late-onset patients. The p.T1106M/p.R778L combination can serve as a "mild-type" genetic marker, characterized by delayed disease onset, higher CER levels, and a slower progression to cirrhosis risk.

Keyword(s) : Wilson disease, ATP7B, genotype-phenotype correlation, loss-of-function mutations, diagnosis age

1899 - Exploring the value of imaging indicators in predicting TACE resistance in hepatocellular carcinoma based on contrast enhanced CT

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Objective: There have been disagreements among various societies regarding the criteria for TACE resistance in the past, and there is currently a lack of reliable models for predicting outcomes and risk

stratification in TACE candidate patients who meet clinical practices in China. Our study aims to establish a TACE resistance prediction method for unresectable HCC patients based on tumor burden and radiomics features, and evaluate its value in early identification of TACE resistance.

Method: A retrospective study was conducted on 255 HCC patients who received initial TACE treatment from three medical centers between January 2013 and January 2024. Among them, 110 cases were used as the training set, 47 cases were used as the internal testing set, and 98 cases were used as the external testing set. Using logistic regression model, extract radiomics features from tumor areas in preoperative enhanced CT images and establish an radiomics model. Perform univariate and multivariate logistic regression analysis on clinical data and semantic features to identify independent risk factors affecting patients' resistance after 3 TACEs, establish clinical and semantic prediction models, and then combine patient individual radiomics scores to establish a joint prediction model. Calculate the area under the operating characteristic curve (AUC) of the subjects in the training set, internal and external test sets. And use log rank test to evaluate the difference in overall survival (OS) between low and high-risk TACE resistance in HCC patients predicted by the model. And validate the calibration degree and clinical value of the model using calibration curves and decision curves. Result: Regression analysis showed that tumor growth type was an independent risk factor affecting patients' resistance after 3 TACEs. The AUC of the joint model are 0.81 for the training set, 0.79 for the internal test set, and 0.78 for the external test set. The median OS differences among the three cohorts of low and high-risk HCC patients with TACE resistance determined by the joint model were statistically significant ($P<0.05$). Meanwhile, patients who developed treatment resistance after three TACEs had significantly shorter median survival times and statistical differences. The calibration curve and decision curve show that the joint model has a good calibration degree ($P<0.05$), which can bring net benefits to patients. Conclusion: This study developed and validated a combined model for HCC patients receiving TACE treatment, which can effectively predict the TACE resistance of patients after 3 surgeries and provide support for subsequent treatment strategies.

Keyword(s) : Hepatocellular carcinoma ; TACE refractoriness ; Radiomics ; Prognosis

1903 - Performance of FIB 4 and MAFLD Fibrosis Score vs. Transient Elastography in a Filipino Cohort

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Background: The Asian Pacific Association for the Study of the Liver recommends a two-step risk assessment in the diagnosis of metabolic dysfunction-associated fatty liver disease (MAFLD). This study aims to evaluate the performance of non-invasive tests Fibrosis-4 index (FIB-4) and MAFLD Fibrosis score (NFS) in relation to the risk for advanced fibrosis by liver stiffness measurement (LSM) on vibration-controlled transient elastography (VCTE).

Methodology: Adult Filipino patients at risk for liver fibrosis from MAFLD, with available data from FibroScan and laboratory tests used to compute FIB-4 and NFS, and without other causes of liver disease (e.g., chronic viral hepatitis, alcoholic liver disease), were included. Data from November 2024 to September 2025 was used.

Results: The study included 1057 patients at risk for MAFLD. Of the 764 (72.3%) patients stratified as low risk by FIB-4, 92 (12%) were misclassified by LSM. For NFS, which included 713 (67.4%) patients from the total cohort, 232 (32.5%) were stratified as low risk. However, 31 (13.3%) were misclassified by LSM. There were less misclassified patients with FIB-4 compared to NFS. By independent samples t-test, misclassified patients were more likely to have higher weight, body mass index, waist circumference, aspartate transaminase, alanine aminotransferase, and glycohemoglobin A1c, and lower albumin levels.

Conclusion: Additional patient characteristics, such as comorbidities (especially diabetes), waist circumference, and BMI, should be considered when using non-invasive tests for risk stratification for MAFLD to minimize the possibility of misclassification by LSM.

Keyword(s) : Non-invasive Tests, FIB-4, MAFLD Fibrosis Score, Vibration-controlled Transient Elastography, Fibroscan, MAFLD

1906 - A Fe Curcumin based Strategy to Reinvigorate CAR T Cells by Reversing Exhaustion and Senescence in Liver Cancer

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Background / **Hypothesis:**
Chimeric antigen receptor-T (CAR-T) therapy has demonstrated remarkable efficacy in hematological malignancies. However, its effectiveness against hepatocellular carcinoma (HCC) remains limited by T cell exhaustion, senescence, and the immunosuppressive tumor microenvironment (TME). Curcumin, a natural anti-tumor compound, exhibits limited efficacy as a monotherapy. This study innovatively constructs an Fe-curcumin (FC) nanoplatform designed to remodel the immunosuppressive TME and confer a superior "memory-persistence" phenotype upon CAR-T cells, thereby aiming to overcome these critical barriers in HCC treatment.

Materials and Methods:
We developed ROS-scavenging Fe-curcumin (FC) nanoparticles for co-delivery with CAR-T cells. Their impacts on CAR-T cell apoptosis, proliferation, exhaustion, and memory phenotypes were profiled via flow cytometry and RNA-seq. In vivo antitumor efficacy and TME modulation were assessed in murine models using MRI and immunohistochemistry.

Results:
The FC nanoparticles effectively induced tumor cell apoptosis without exerting cytotoxicity on T cells or CAR-T cells. Furthermore, they alleviated CAR-T cell senescence and exhaustion by suppressing the p53 signaling pathway, thereby enhancing cytotoxicity and proliferative capacity. RNA-Seq and flow cytometry confirmed an upregulated memory phenotype (e.g., CCR7) alongside downregulated exhaustion markers (e.g., PD-1, Tim-3) in FC-treated CAR-T cells. In vivo, the FC/CAR-T combination demonstrated a significant suppression of tumor growth and an extension of survival. The nanoparticles also remodelled the TME by alleviating hypoxia (reduced HIF-1 α) and acidosis (increased pH), promoting M2-to-M1 macrophage repolarization, and enhancing endogenous T/NK cell infiltration.

Conclusions:
This study demonstrates that FC nanoparticles reinvigorate CAR-T cells by mitigating exhaustion and senescence via p53 pathway inhibition, while concurrently remodeling the immunosuppressive TME. Our findings provide a novel and potent combinatorial strategy to overcome the key limitations of CAR-T

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therapy in solid tumors, highlighting its significant clinical translation potential.

Keyword(s) : Liver cancer , CAR-T therapy , Tumor microenvironment , P53 signaling pathway

1908 - Performance of Baveno VII Criteria in Predicting Clinically Significant Portal Hypertension in Burned Out MAFLD

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Background / Hypothesis:

Burned-out MAFLD represents an advanced stage of metabolic liver disease in which hepatic steatosis disappears (CAP S0) despite substantial underlying fibrosis. This phenotype often reflects progressive disease biology and is associated with a higher likelihood of developing portal hypertension. Although non-invasive tools are widely used to assess the risk of clinically significant portal hypertension (CSPH), including liver stiffness measurement (LSM), platelet count, and the Baveno VII criteria, their performance specifically in burned-out MAFLD remains poorly defined. The Child-Pugh classification provides an assessment of hepatic functional reserve, but its role in predicting portal hypertension—particularly in the burned-out phenotype—remains unclear. Therefore, this study aimed to evaluate the application of the Baveno VII criteria for predicting CSPH in patients with burned-out MAFLD.

Materials and Methods:

A retrospective single-center pilot study was conducted. Liver stiffness measurement (LSM), platelet count, and Child-Pugh class were collected at baseline. Burned-out MAFLD was defined as steatosis grade S0 (CAP <248 dB/m) with significant fibrosis (LSM ≥ 8 kPa). CSPH was defined non-invasively as LSM ≥ 25 kPa. Baveno VII categories were stratified into rule-out (LSM <15 kPa and platelets $>150 \times 10^9/L$), grey zone, and rule-in (LSM ≥ 25 kPa). Subgroup analyses were performed for patients with burned-out MAFLD.

Results:

Among 44 patients (median age 59.5 years; 61% male), burned-out MAFLD accounted for 16 cases (36.4%). Non-invasive CSPH was identified in 50% of the overall cohort. In the burned-out subgroup, CSPH prevalence

was higher (62.5%) compared with non-burned-out MAFLD (42.9%). Based on the Baveno VII classification, most burned-out patients were categorized as rule-in (62.5%), followed by grey zone (31.2%), with only 6.2% meeting rule-out criteria. Within the burned-out group, CSPH occurred in 55.6% of Child-Pugh A, 80.0% of Child-Pugh B, and 50.0% of Child-Pugh C patients. Platelet count and Child-Pugh class demonstrated limited discriminatory performance for CSPH (AUC ≈ 0.50 and 0.59 , respectively).

Conclusions:

Burned-out MAFLD demonstrates a high prevalence of non-invasive CSPH, with the majority of patients fulfilling Baveno VII rule-in criteria. While the Baveno VII criteria remain useful for identifying high-risk individuals, their ability to spare endoscopic screening appears limited in the burned-out phenotype. These findings highlight the need for refined non-invasive stratification tools tailored specifically to burned-out MAFLD and warrant validation in larger prospective studies.

Keyword(s) : Burned-out MAFLD; clinically significant portal hypertension; CSPH; Baveno VII; non-invasive assessment; liver stiffness measurement.

1909 - A Diagnostic conundrum in a Case of Obstructive Jaundice in Biliary Hamartoma

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Background / Hypothesis:

Hilar masses pose a significant diagnostic challenge, as differentiating malignant conditions like hilar cholangiocarcinoma and benign biliary strictures is often difficult. Biliary hamartomas are rare benign condition

caused by malformation of the intrahepatic bile ducts. Most biliary hamartomas are clinically asymptomatic and often found incidentally. We hereby present a case of hilar mass which was eventually diagnosed as biliary hamartoma.

Materials and Methods:

A 61-year-old male, with no known comorbidities, presented with jaundice for two years. Prior interventions included ERCP with stenting and subsequent percutaneous transhepatic biliary drainage

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for recurrent obstructive episodes. Several attempts to obtain tissue diagnosis was done but did not yield any histopathologic diagnosis. Initial bile GeneXpert MTB/RIF assay detected trace Mycobacterium tuberculosis (MTB); however, repeat testing was negative. A PET scan study revealed hypermetabolic ill-defined mass at the hepatic hilum extending to both hepatic lobes, with intrahepatic duct dilatation, highly suggestive of Klatskin tumor. Due to persistent diagnostic uncertainty and atypical clinical progression, liver biopsy was done prior to oncologic treatment. Histopathologic analysis revealed multiple irregularly dilated bile ducts embedded in fibrous stroma, consistent with biliary hamartomas. Following confirmation of this benign diagnosis, aggressive oncologic or surgical interventions were avoided. The patient instead continued supportive biliary decompression with subsequent clinical improvement.

Results:

Biliary hamartoma are uncommon benign lesions found in the liver. In this case, the patient presented with repeated episodes of jaundice, fevers, abdominal distension and right upper quadrant pain. However, clinical presentation is not compatible with hilar cholangiocarcinoma hence tissue correlation was recommended. Biopsy of liver proved to be benign biliary hamartoma which has better clinical outcome.

Conclusions:

This case highlights the diagnostic complexity of hilar masses and emphasizes the importance of tissue confirmation when clinical, radiologic, and metabolic findings are discordant. Biliary hamartomas, though rare, can closely mimic cholangiocarcinoma—including PET avidity—leading to potential misdiagnosis. Thus, key clinical and histopathologic confirmation is essential to avoid diagnostic pitfalls or unnecessary therapy, and to guide appropriate patient-centered management.

Keyword(s) : obstructive jaundice, hilar mass, biliary hamartoma, cholangiocarcinoma

1912 - Association Between Fatty Liver and Risk of Liver Failure in Patients with Acute Hepatitis B A Retrospective Cohort Study

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Background / Hypothesis: Fatty liver (FL) is a common comorbidity associated with adverse outcomes in various liver diseases, but its impact on the prognosis of acute hepatitis B (AHB) remains unclear. This study aimed to evaluate the influence of FL on liver-related outcomes in hospitalized patients with AHB.

Materials and Methods: We retrospectively analyzed hospitalized patients diagnosed with AHB between 1 January 2010 and 30 December 2023. Based on imaging and laboratory findings, patients were categorized into AHB with FL (AHB-FL) and AHB without FL (AHB-no FL). Demographic and clinical data were collected. The primary outcome was acute liver failure (ALF); secondary outcomes included hepatitis B surface antigen (HBsAg) loss and seroconversion. Multivariable regression models were used to assess the independent association between FL and clinical outcomes. Kaplan-Meier analysis was performed to compare time to HBsAg loss and seroconversion.

Results:

A total of 200 patients were included, with 29 (14.5%) in the AHB-FL group and 171 (85.5%) in the AHB-no FL group. The incidence of ALF was significantly higher in the AHB-FL group than in the AHB-no FL group (34.5% vs. 15.8%, $P=0.02$). Rates of HBsAg loss (75.9% vs. 82.5%, $P=0.40$) and seroconversion (44.4% vs. 53.1%, $P=0.40$) were comparable between groups. Kaplan-Meier curves showed no significant differences in time to HBsAg loss ($P=0.07$) or seroconversion ($P=0.43$). In multivariable analysis, FL remained an independent risk factor for ALF (odds ratio 4.61; 95% CI 1.26-16.86; $P=0.02$), but was not independently associated with HBsAg loss (hazard ratio [HR] 1.60; 95% CI 0.92-2.78; $P=0.10$) or seroconversion (HR 1.69; 95% CI 0.80-3.57; $P=0.17$). Subgroup analysis revealed a stronger association between FL and ALF among rural patients (P for interaction=0.02), suggesting that regional factors may modify clinical risk and management.

Conclusions:

In hospitalized patients with AHB, FL substantially increases the risk of ALF but does not significantly affect HBsAg loss or seroconversion. Early identification and

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active management of FL in this population are essential to reduce acute liver-related complications and improve clinical outcomes.

Keyword(s) : liver failure; acute hepatitis B; fatty liver; HBsAg loss; seroconversion; prognosis

1916 - Restoration of ATP7B Trafficking and Biliary Function in Wilson Disease Hepatocytes through CRISPR Cas9 Mediated Gene Correction

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Background / Hypothesis:The ATP7B R778L mutation, prevalent in Asian Wilson disease (WD) patients, is hypothesized to disrupt copper-induced protein trafficking to bile canaliculi—a fundamental process in hepatocyte polarity. This study systematically evaluates the functional efficacy of CRISPR/Cas9-mediated genetic correction in restoring ATP7B localization and copper-responsive dynamics using patient-derived hepatocyte-like cells.

Materials and Methods:Urine-derived stem cells (USCs) from a WD patient (ATP7B R778L homozygous) were non-integratively reprogrammed into induced pluripotent stem cells (iPSCs). The pathogenic mutation was precisely corrected to wild-type (WT) using CRISPR/Cas9, generating an isogenic control. Corrected (ATP7BWT/WT), uncorrected (ATP7BR778L/R778L), and healthy control iPSCs were differentiated into hepatocyte-like cells (HLCs) via a defined protocol. The polarization status and bile canaliculi formation were rigorously assessed by confocal microscopy for apical markers (MRP2/ABCC2, BSEP) and tight junctions (ZO-1). ATP7B localization was evaluated by immunofluorescence under copper-chelated and copper-loaded conditions.

Results:HLCs from all groups developed mature hepatocyte characteristics, forming extensive, branching networks of bile canaliculi, as evidenced by the exclusive apical localization of MRP2 and BSEP, delineated by ZO-1. In uncorrected WD-HLCs, the mutant ATP7B R778L

protein was predominantly retained in the Golgi apparatus and failed to traffick to the canalicular membrane upon copper challenge. In stark contrast, HLCs derived from the genetically corrected iPSCs displayed a complete functional rescue: ATP7B properly localized to the trans-Golgi network under basal conditions and underwent robust, copper-induced redistribution to the bile canaliculi, a phenotype identical to HLCs from healthy controls. Quantitative analysis confirmed a significant restoration of ATP7B canalicular signal post-correction.

Conclusions:This study provides definitive evidence that CRISPR/Cas9-mediated correction of the ATP7B R778L mutation restores not only the genetic sequence but, critically, the dynamic copper-responsive trafficking essential for ATP7B function in polarized hepatocytes. By rescuing the mutant protein's capacity for copper-induced canalicular redistribution—a polarity-dependent process fundamental to hepatic copper homeostasis—we establish that genetic repair at the stem cell level effectively reverses a core molecular pathology of Wilson disease. These findings provide a robust scientific foundation for developing autologous cell-based therapies as a potential curative strategy for this inherited metabolic disorder.

Keyword(s) : Wilson disease, ATP7B, CRISPR/Cas9, hepatocyte-like cells, copper homeostasis

1918 - Solving a Recurrent Hepatic Abscesses Through Discovery of a Cholecystoduodenal Fistula resulting from an Over the Scope Clip closure of Duodenal Perforation as a complication of Microwave Ablation for HCC

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Background / Hypothesis: A 75-year-old male with hepatocellular carcinoma (BCLC A) on a background of compensated hepatitis C-related cirrhosis presented with recurrent liver abscess. He has hypertension and cholelithiasis. Four years earlier, he received eight cycles of Atezolizumab-Bevacizumab for tumor downsizing, followed by microwave ablation. Post-ablation he developed a duodenal bulb perforation, successfully closed with an endoscopic Over-the-Scope Clip (OTSC). One month post-clipping, he developed a liver abscess, followed by another episode four months later. Both were managed with percutaneous drainage, pigtail

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catheter insertion, and culture-guided antibiotics (*Klebsiella pneumoniae* and *E. coli* respectively), leading to full recovery. Six months prior to admission, he developed a 5.4 × 4.8 × 3.9 cm (52 cc) subhepatic abscess, which was drained pigtail catheter and treated with a six-week course of ciprofloxacin (*Streptococcus pneumoniae* sensitive), resulting in improvement and subsequent catheter removal.

One month prior to admission, patient presented with fever and anorexia. Triphasic CT scan reveals a subdiaphragmatic abscess formation posterior to the right hepatic lobe is measuring 7.5 x 5.2 x 4.9 cm (100 cc), stable gallbladder stones (1.5cm) with air pocket levels within the lumen, a dilated common bile duct (1.0 cm), and punctate distal lithiasis.

Materials and Methods:
Triphasic CT scan of the abdomen, ERCP, surgery

Results:

ERCP showed an erythematous duodenal bulb with a fistulous tract seen at the anteromedial wall with purulent discharge, the same area where the OVESCO clip was deployed. Fistulography opacified the gallbladder, common hepatic duct (10mm) and common bile duct (12mm). Cholangiography showed a dilated CBD with distal filling defect. Sphincterotomy and balloon sweeps removed bile sludge, and a 10 Fr × 7 cm biliary stent was placed. The patient underwent exploratory laparotomy with cholecystectomy, takedown of a cholecystoduodenal fistula, repair of duodenal perforation, and drainage of abscesses. His postoperative course was uneventful, and no further abscess recurrence was noted on follow-up.

Conclusions:

A rare sequence of duodenal perforation, OTSC closure, cholecystoduodenal fistula and recurrent abscess highlights the need for high index of suspicion in unusual causes based on patient's clinical profile and history. Recurrent liver abscess complicates MWA in 1.6% of cases. Cholecystoduodenal fistula at the site of an OTSC closure of duodenal perforation underscores how thermal injury plus clip-mediated tissue traction may create a persistent bilioenteric connection, promoting recurrent pyogenic abscess via contamination from biliary or enteric flora. This required multidisciplinary diagnosis (CT, ERCP, fistulography) and definitive surgical repair.

Keyword(s) : recurrent liver abscess, cholecystoduodenal fistula, duodenal perforation, OTSC clip, Microwave ablation

1920 - A New Noninvasive Marker Predicting Liver Fibrosis in Patients with Chronic Hepatitis B
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Background: Chronic hepatitis B virus infection (CHB) is a significant health problem affecting more than 250 million people and causing approximately 900,000 deaths each year due to cirrhosis and hepatocellular carcinoma. The infection can be silent; fibrosis and even cirrhosis can develop without symptoms or liver enzyme abnormalities. Numerous non-invasive, easily applicable methods and scoring systems have been developed to detect disease progression. This allows for early antiviral treatment to improve disease outcomes. This study aims to develop an effective formula for determining fibrosis by determining fibrosis values obtained from liver biopsies of patients with CHB, along with biochemical variables and risk factors.

Materials and Methods: Patients who were followed up for CHB and underwent liver biopsy at Samsun Training and Research Hospital were included in the study. Patients under the age of 18, those with additional viral hepatitis disease and other liver-related diseases and those with insufficient data in retrospective file scans and laboratory tests were not included in the study. Patient demographics, pathology results and biochemical data were recorded as retrospective data. These data were compared with the Ishak fibrosis stage and their diagnostic validity in predicting liver fibrosis was statistically evaluated and a new serological marker was developed.

Results: In our study, 204 patients were grouped as having advanced fibrosis (F4-6) and low-grade fibrosis. The proportion of HBeAg(+) patients and ALP were higher in the advanced fibrosis group. Univariate analysis of variables predicting advanced fibrosis revealed that HBeAg, AST, ALT, ALP, GGT, platelet count and INR were significant variables. Multivariate analysis revealed that HBeAg(+), ALP and platelet count were independent risk factors. In HBeAg(-) patients, univariate analysis revealed ALP, GGT, lymphocyte count, platelet count and INR were significant variables. Multivariate analysis revealed only ALP as an independent risk factor. In previous studies, the effectiveness of many markers that have proven to be powerful in detecting

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liver fibrosis was compared with the ASLI([AST+ALT]xALPxINR/PlateletxAlbumin) formula that we developed in showing advanced fibrosis; according to this, API, Fibro-Q were found to be statistically insignificant in detecting advanced fibrosis, while AAR,APRI, FIB-4,King and ASLI formulas were found to be statistically significant. Conclusions: In our study, the higher prevalence of advanced fibrosis in the HBeAg(+) group suggests that patients were in an "immune tolerant" phase, leading to delayed treatment and progression of fibrosis. The ASLI score we developed has a higher predictive power than other markers, particularly in distinguishing low fibrosis levels. We believe it will be beneficial for early diagnosis and monitoring of fibrosis. Further studies are needed in other populations.

Keyword(s) : Alkaline Phosphatase , HBeAg , Hepatitis B, Liver Fibrosis , Non- invasive tests

1923 - Women's Perspective on Non Antibiotic Interventions for rUTI Prevention Meta Analysis of Efficacy Safety and Clinical Implications

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Background / **Hypothesis:**
Recurrent urinary tract infection (rUTI) affects up to 50% of adult women annually and represents a substantial global health burden. This burden is further aggravated by rising antimicrobial resistance. Non-antibiotic strategies, such as cranberry products, D-mannose and probiotics, have emerged as promising alternatives; however, their comparative efficacy and safety versus placebo remain insufficiently characterized, particularly from the perspective of women's health across different menopausal stages.

Materials and **Methods:**
We conducted a systematic review and meta-analysis in accordance with PRISMA guidelines. We searched PubMed, Embase, and Web of Science up to October 2025 for studies comparing non-antibiotic prophylaxis to placebo in adult women with rUTIs. The primary outcomes were the recurrence of symptomatic or culture-confirmed rUTIs (measured by relative risk [RR]) and adverse events. Random-effects models were employed to account for high heterogeneity, as assessed by I². Publication bias was evaluated using Egger's test.

Results:
We included 17 studies with a total of 2749 female participants, of whom 1444 received non-antibiotic therapies. In preventing recurrent urinary tract infection

(rUTI), non-antibiotic therapies were significantly more effective than placebo (RR 0.45, 95% CI [0.32-0.64]). Among them, cranberry (RR 0.33, 95% CI [0.16-0.70]) and fosfomycin trometamol (RR 0.09, 95% CI [0.05-0.17]) demonstrated better efficacy, while D-mannose did not show a significant trend due to the limited number of studies (Figure 1). The studies also indicated that both cranberry and hyaluronic acid and chondroitin sulphate(HA/CS) significantly extended the time to the first occurrence of UTI after treatment (Figure 2). When compared to antibiotic regimens, HA/CS also showed comparable efficacy (RR 0.80, 95% CI [0.66-0.97]) (Figure 3). The safety of non-antibiotic therapies was comparable to that of placebo, with no adverse events leading to treatment discontinuation. Conclusions:

Non-antibiotic interventions significantly reduce the risk of recurrent urinary tract infections (rUTI) in adult women without increasing adverse events, with cranberry and hyaluronic acid combined with chondroitin sulphate showing particularly strong effects. For women with frequent infections or concerns about antibiotic resistance and hepatotoxicity, these non-antibiotic therapies may be effective alternatives to antibiotics. Future research should focus on larger randomized controlled trials to determine the optimal doses, treatment duration, and long-term outcomes, as well as differences in quality of life and cost-effectiveness.

Keyword(s) : Recurrent Urinary Tract Infections; Non-Antibiotic Prophylaxis; Cranberry; Probiotics

1926 - Hepatitis and HIV Seroprevalence in Individuals with Substance Use Disorders A 25 Year Retrospective Analysis

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Background / Hypothesis: Injection drug use and related high-risk behaviors significantly contribute to the transmission of Hepatitis A (HAV), Hepatitis B (HBV), Hepatitis C (HCV), and Human Immunodeficiency Virus (HIV). World Health Organization (WHO) and the United Nations Office on Drugs and Crime (UNODC) guidelines recommend screening individuals who use drugs not

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only for HBV and HCV, but also for HIV and other sexually transmitted infections. However, screening rates remain inadequate. Our study aims to determine the seroprevalence of HAV, HBV, HCV, and HIV among individuals with substance use disorder, and evaluate their association with substance use.

Materials and Methods: In our study, the medical records of patients with substance use disorder at the Department of Psychiatry of a tertiary hospital between June 1, 2000, and July 1, 2025, were retrospectively reviewed. The participants' sex and serological test results (HBsAg, anti-HBs, anti-HCV, anti-HAV IgG, anti-HIV) were evaluated. The types of substances used by the patients were categorized based on the route of administration as oral, parenteral, and inhalational. The data were analyzed using descriptive statistical methods, and differences between groups were assessed with the chi-square test.

Results: Median age was 33 years (IQR:25-45), 85.6% were male. A total of 8,145 patient records were reviewed for the specified period, and 2,478 patients (30.4%) who underwent serological screening were included in the study. HIV serology was performed in 2,343 patients, and 20 patients (0.85%) were positive. HCV serology was performed in 2,422 patients, and 47 patients (1.94%) were positive. Of these patients, 16 (34.04%) received treatment, and HCV RNA negativity was documented in 22 patients (46.80%). HBV serology was performed in 2389 patients, and 56 (2.3%) were positive. Among 56 patients, 7 (12.50%) received treatment and achieved HBV DNA negativity. Among 44 patients with isolated anti-HBc IgG positivity, only 11 underwent HBV DNA testing; however, none were positive. Other serological findings are summarized in Table 1. The distribution of substance types used by patients with positive HBsAg and Anti-HCV results is presented in Table 2 below.

Conclusions: In our study, only 30.4% of patients were screened, with an HBsAg positivity rate of 2.34% and an anti-HCV positivity rate of 1.94%. These rates are higher compared to the values reported for the general population in the Aegean Region, where HBsAg positivity ranges between 1–1.5% and anti-HCV positivity between 0.1–0.4%. The elevated seroprevalence observed in our cohort highlights the need to strengthen infection control strategies among high-risk individuals.

Keyword(s) : hepatitis, drug use disorder, seroprevalance

1934 - Comprehensive Evaluation of Demographic Endoscopic and Ultrasonographic Characteristics in First Episode Variceal Bleeding

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Background / Hypothesis: Esophagogastric variceal hemorrhage (EGVH) is one of the most severe complications of portal hypertension and remains a major cause of morbidity and mortality among patients with liver cirrhosis. Identifying demographic and clinical factors associated with first bleeding episodes is essential for improving risk stratification and guiding individualized management.

Materials and Methods: This retrospective, single-center cohort study included 67 consecutive cirrhotic patients presenting with their first episode of EGVH between January 2020 May 2025. Demographic data, etiology of cirrhosis, laboratory parameters, ultrasonographic vascular measurements, endoscopic findings, medication history, clinical complications during hospitalization, and early outcomes were evaluated. Comparisons between sexes were performed using appropriate statistical tests, and $p < 0.05$ was considered statistically significant.

Results: The study cohort consisted of 26 females (39%) and 41 males (61%) with a mean age of 61.7 ± 13.9 years. Cryptogenic cirrhosis (31.3%) and HBV-related cirrhosis (22.4%) were the most common etiologies. Red spots and nipple signs were observed in 92.5% and 73.1% of patients, respectively. Endoscopic band ligation was performed in 85.1% of cases, whereas N-butyl-2-cyanoacrylate (NBCA) injection was applied in 16.4%. At the time of presentation, ascites was present in 37.3% of patients, whereas hypotension was observed in 20.9%. In-hospital mortality was 7.5%. Most demographic, laboratory, and ultrasonographic parameters; including length of hospital stay, hemoglobin, platelet count, ALT, ALP, bilirubin, albumin, potassium, BUN, creatinine, INR, ammonia levels, as well as spleen size and both portal

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and splenic vein diameters were comparable between genders. In contrast, several variables demonstrated significant gender related differences: male patients were younger and exhibited higher AST and GGT levels, whereas female patients had higher serum sodium concentrations.

Conclusions: Patients presenting with first variceal bleeding demonstrated moderate liver dysfunction, high prevalence of endoscopic bleeding stigmata, and substantial early complication rates. NSBB prophylaxis did not fully prevent first bleeding episodes, suggesting the need for optimized dosing or combined strategies. These findings enhance the understanding of risk profiles among patients experiencing their first episode of EGVH and may support more individualized clinical decision-making.

Keyword(s) : Esophagogastric variceal hemorrhage, portal hypertension, cirrhosis

1935 - Neuropilin 1 alleviates metabolic dysfunction associated fatty liver disease by promoting lysosomal degradation of toll like receptor 4 in macrophages

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Background / **Hypothesis:**
The prevalence of metabolic dysfunction-associated fatty liver disease (MAFLD) is increasing year by year and has become one of the leading causes of end-stage liver disease worldwide. Macrophage-mediated chronic hepatic inflammation plays a pivotal role in the progression MAFLD. Neuropilin-1 (Nrp1) acts as an immune regulator, but its specific role in hepatic macrophages during liver steatosis remains unclear. This study aims to explore the role and mechanism of Nrp1 in MAFLD.

Materials and **Methods:**
Myeloid -specific Nrp1 knockout mice (Nrp1MKO) were generated by crossing Lyz2-Cre mice with Nrp1-flox/flox mice. Nrp1MKO and littermate controls were fed a western diet (WD) for 16 weeks or a methionine-choline-deficient (MCD) diet for 8 weeks to induce MAFLD. Single-cell RNA sequencing datasets from patients with nonalcoholic steatohepatitis (NASH) were analyzed. In vitro experiments were conducted in bone

marrow-derived macrophages (BMDMs) and THP-1 cells using lentiviral overexpression and knockdown of Nrp1. Mechanistic interactions were assessed through RNA-seq, western blotting, flow cytometry and co-immunoprecipitation.

Results:

Nrp1 expression was significantly upregulated in hepatic macrophages from patients with steatohepatitis and MAFLD mouse models. In vivo, Nrp1MKO mice exhibited exacerbated hepatic steatosis, inflammation, and liver injury compared with controls under WD or MCD feeding. In vitro, Nrp1 deficiency in macrophages promoted polarization toward a pro-inflammatory phenotype and increased the secretion of cytokines and chemokines, including IL-1 β , IL-6, TNF- α , and CCL2. RNA-seq and gene set enrichment analysis revealed a significant enrichment of the toll-like receptor signaling pathway in Nrp1-deficient macrophages. Notably, while Tlr4 mRNA levels remained unchanged, western blotting showed markedly accumulated toll-like receptor 4 (TLR4) protein in Nrp1-deficient macrophages. Mechanistically, we demonstrated that Nrp1 directly binds to TLR4 and facilitates its lysosomal degradation. Consequently, Nrp1 deficiency impaired TLR4 turnover, leading to sustained activation of the downstream nuclear factor (NF)- κ B signaling pathway. Conversely, Nrp1 overexpression accelerated TLR4 degradation and suppressed NF- κ B activation.

Conclusions:

Nrp1 acts as a critical negative regulator of hepatic inflammation by forming a Nrp1/TLR4 complex to promote the lysosomal degradation of TLR4, thereby inhibiting NF- κ B signaling. Targeting the macrophage Nrp1-TLR4 axis may represent a potential therapeutic strategy for MAFLD intervention.

Keyword(s) : MAFLD, Neuropilin-1, macrophages, inflammation, TLR4

1938 - Prediction of Esophageal Varices in Primary Biliary Cholangitis What Is the Role of Non Invasive Tests

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Introduction

Screening for esophageal varices (EV) in patients with

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primary biliary cholangitis (PBC) is essential to reduce the morbidity and mortality associated with gastrointestinal bleeding. Current research increasingly explores non-invasive tests that may help limit unnecessary endoscopic procedures. The e-LIFT score, proposed as a non-invasive method for predicting varices, has been evaluated in only one study. The objective of our work was to assess the correlation between endoscopic findings and the e-LIFT score.

Patients	and	Methods
This retrospective, descriptive, and analytical monocentric study was conducted over an eight-year period (2018–2025) in a gastroenterology department. It included patients followed for PBC who underwent an upper gastrointestinal endoscopy (EGD). Data were collected from patient records. EGD was performed in all patients to detect esophageal varices. The diagnostic performance of the e-LIFT score was assessed using ROC curve analysis, including sensitivity, specificity, and negative predictive value.		

Results
A total of 67 patients with PBC were included. The mean age was 50 ± 14.2 years, with a marked female predominance (80.6%, F/M ratio = 4.15). EGD revealed EV in 41.4% (n = 24) of patients, among whom 12.5% (n = 3), 41.6% (n = 10), and 45.8% (n = 11) had grade I, II, and III varices, respectively. The predictive performance of the e-LIFT score was acceptable, with an AUC of 0.745. A cutoff value of 13 showed the best discriminative accuracy, yielding a sensitivity of 82.14%, a specificity of 61.51%, and a negative predictive value of 82.76%.

Conclusion
The e-LIFT score appears to be a simple and reliable non-invasive serum marker for screening esophageal varices in primary biliary cholangitis. An upper endoscopy may potentially be avoided in 82.76% of patients with an e-LIFT score below 13.

Keyword(s) : primary biliary cholangitis esophageal varices e-lift

1940 - Dual SGLT2 DPP 4 Inhibition Synergistically Restores Hepatopancreatic Integrity in Experimental Diabetes

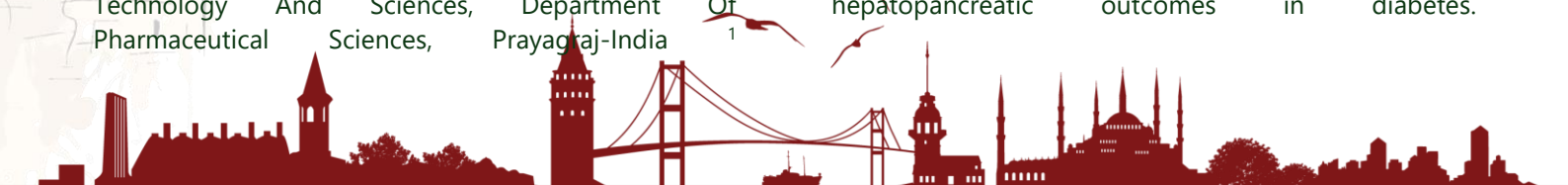
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Background / Hypothesis: Diabetes mellitus, now exceeding 500 million cases globally, underscores an urgent need for metabolic therapies that also preserve hepatic function. Emerging evidence shows that improving hepatic resilience can substantially reduce long-term diabetic complications. Empagliflozin, an SGLT2 inhibitor, provides broad metabolic benefits by lowering glucotoxicity, enhancing fatty acid oxidation, improving mitochondrial efficiency, and restoring healthy adipose–liver communication. Sitagliptin, a DPP-4 inhibitor, further strengthens metabolic control through incretin enhancement, optimizing insulin secretion, reducing glucagon, suppressing lipogenesis, and improving oxidative defense. The complementary nature of these mechanisms suggests a promising synergistic potential for improved hepatic outcomes in diabetes.

Materials and Methods: STZ induced diabetic rats received Empagliflozin (10 mg/kg), Sitagliptin (10 mg/kg), their combination and standard metformin for 28 days. Serum glycemic index, hepatic enzyme levels, lipid profile, and oxidative stress markers (MDA, SOD, CAT) were evaluated. Pancreatic as well as liver histology were performed to assess steatosis, inflammation, β -cell morphology, islet integrity, and hepatocellular architecture. Comparisons were made to identify improvements in pancreatic restoration, hepatic protection, and overall metabolic regulation.

Results: Empagliflozin markedly improved glycemic status and reduced hepatic steatosis, while Sitagliptin enhanced incretin signaling, reduced DNL, preserved β -cell structure, and improved oxidative defense. Combination therapy produced the strongest hepatopancreatic benefit, including $\approx 60\%$ reduction in hepatic triglycerides, reduced MDA, elevated SOD/CAT, and improved islet morphology with greater β -cell preservation. The synergism arose from dual correction of glucotoxicity (via SGLT2 inhibition) and enhanced insulinotropic incretin activity (via DPP-4 inhibition), resulting in superior mitochondrial efficiency, lipid balance, and structural organ protection.

Conclusions: Empagliflozin and Sitagliptin together provide robust metabolic, hepatic, and pancreatic protection, demonstrating synergistic reductions in fatty liver, oxidative stress, and β -cell damage. Their combined mechanism driven actions highlight a promising therapeutic strategy for improving hepatopancreatic outcomes in diabetes.



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Keyword(s) : Hepatopancreatic Protection; Diabetes; SGLT2 Inhibitor; DPP-4 Inhibitor; STZ; Oxidative Stress

1941 - Predictors and Immune Signatures of Baveno VII-Aligned Recompensation in Pediatric Wilson Disease with Decompensated Cirrhosis

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Background / Hypothesis: With growing evidence that etiological therapy may reverse decompensation in chronic liver disease, pediatric data remain scarce, particularly for Wilson disease. We evaluated the incidence, determinants, cytokine signatures, and outcomes associated with Baveno-VII-defined hepatic recompensation in children with decompensated Wilson disease.

Materials and Methods: Ambispective cohort including children <18 years with decompensated Wilson disease (2014–2024) and ≥1-year follow-up. Recompensation required: (i) control of copper overload, (ii) ≥12 months' absence of ascites/HE without supportive drugs and no variceal bleeding, and (iii) normalized bilirubin, albumin, and INR. Longitudinal liver function trends, cumulative recompensation incidence, survival, and re-decompensation were assessed. A subset underwent paired stool–plasma cytokine quantification

Results: Seventy-nine children (median age 9.2 years) were analyzed. Recompensation occurred in 88.6%, mostly within 12–36 months of therapy, sharply rising between year-1 and year-2. Clinical recovery followed a reproducible pattern: ascites → jaundice → INR → albumin normalization. Re-decompensation occurred in 11.4%, predominantly due to non-adherence, but was usually salvageable without transplant.

Baseline smaller spleen size, higher platelet and leukocyte counts, lower liver stiffness, and lower ALT significantly predicted recompensation. Recompensated patients had superior native liver survival (98.6% vs 33.3%, $p < 0.001$).

Cytokine profiling ($n=20$) revealed higher stool IL-6 and

higher plasma IL-10 in those achieving recompensation. IL-10 may reflect enhanced regulatory immune tone, supporting hepatocyte recovery and fibrosis reversal. The elevated gut-localized IL-6 likely reflects beneficial gut–liver axis activation rather than systemic inflammation — possibly promoting mucosal healing, microbiome restoration, and improved portal immune signaling. Together, these cytokine patterns appear to represent an immune microenvironment favorable for hepatic tissue remodeling.

Conclusions:

Recompensation is frequent, physiologically robust, and survival-enhancing in pediatric Wilson disease. Portal hypertension markers and baseline immune state may serve as early prognostic tools. Cytokine signatures highlight gut–liver immune restoration as a potential biological driver of reversibility. Multicenter validation and immune-integrated predictive modeling are warranted.

Keyword(s) : Pediatric Cirrhosis; Hepatic Recompensation; Portal Hypertension; Elastography; Native Liver Survival; Gut–Liver Axis; Cytokines

1943 - Investigation of the Therapeutic Effects of $\beta 3$ Adrenergic Stimulation on Paracetamol Induced Acute Liver Damage

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Background/Hypothesis: Although paracetamol is a well-tolerated and safe analgesic, the annual increase in cases of paracetamol toxicity has become a significant public health concern. Excessive paracetamol intake causes hepatocellular damage by inducing oxidative stress, mitochondrial dysfunction, and inflammation. Recent studies on $\beta 3$ -adrenergic receptors, which are considered to play a key role in our hypothesis, have shown that these receptors exert critical regulatory effects on energy metabolism and mitochondrial function. The roles of $\beta 3$ -adrenergic receptors, in the gluconeogenic pathway and hepatic lipid metabolism suggest that they may also have potential effects on

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inflammatory processes. Our study investigated the effects of Mirabegron, a $\beta 3$ -adrenergic receptor agonist, on liver damage.

Materials and Methods: In this study, Balb/C mice were divided into following groups: healthy control, NAC, paracetamol, Mirabegron 5mg/kg, Mirabegron 10mg/kg, Mirabegron 20mg/kg, and Mirabegron 20mg/kg+healthy control. All groups except the healthy controls received 400mg/kg paracetamol via oral gavage. One hour after paracetamol administration, the NAC group was treated with NAC at a dose of 140mg/kg, and the Mirabegron groups received the target drug Mirabegron at doses of 5, 10, and 20mg/kg via oral gavage. At the end of the experiment, mRNA expression levels of TNF- α , IL-6, IL-1 β , and CYP2E1 were evaluated, and TNF- α and IL-1 β levels were also measured by ELISA method.

Results: TNF α , IL-1 β , and IL-6 mRNA expression levels increased in the toxicity group compared to the healthy group. The 5 and 10 mg/kg doses of mirabegron reduced these markers, whereas, the 20 mg/kg dose did not exert a similar anti-inflammatory effect. ELISA results were consistent with the PCR findings. CYP2E1 mRNA expression was elevated in the paracetamol group and decreased in the 5 and 10 mg/kg mirabegron groups. However, the 20 mg/kg dose was not effective; on the contrary, it further stimulated CYP2E1 expression.

Conclusions: In this study, paracetamol-induced hepatic toxicity led to marked increases in TNF α , IL-1 β , IL-6, and CYP2E1 mRNA expression levels. Treatment with mirabegron at doses of 5 and 10mg/kg effectively reduced pro-inflammatory cytokine expression and suppressed CYP2E1 induction. Interestingly, the 20 mg/kg dose did not exhibit similar protective effects and instead demonstrated a stimulatory impact on CYP2E1 expression, suggesting a dose-dependent response profile for mirabegron. Overall, this study demonstrates that mirabegron, particularly at lower doses, exerts hepatoprotective effects against paracetamol-induced liver injury by modulating inflammatory signaling. These results highlight $\beta 3$ -adrenergic stimulation as a promising therapeutic candidate for xenobiotic-induced acute liver damage and warrant further investigation in future studies

Keyword(s) : Mirabegron, Liver, Paracetamol, $\beta 3$ -adrenergic receptors, CYP2E1

1945 - Impact of Microbiome in Obese vs Lean Steatotic Liver Disease in Children

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Background / Hypothesis: Steatotic liver disease (SLD) in children is influenced by environment, genetics, obesity, and insulin resistance. We aimed to investigate the association between intestinal microbiome profiles and liver steatosis in obese versus lean pediatric patients.
Materials and Methods: The study analyzed fecal microbiota, anthropometric measurements, and biochemical and metabolic tests in children attending Pediatric Gastroenterology and Adolescent Clinics between January and December 2023. The study group (SG) comprised of 52 pediatric SLD patients (41 obese: SGO, 11 lean: SGL), with liver steatosis on ultrasound (US). A control group (CG) of 42 age-matched healthy volunteers (20 obese: CGO, 22 lean: CGL) was also included. Inclusion criteria were ages 6-18 years, no other causes of liver steatosis, chronic diseases or drug use, no gastrointestinal surgery, and absence antibiotic or probiotic use in the previous three months. Medical history, demographics, anthropometric measurements, biochemical parameters, imaging findings, and exercise and dietary habits were reviewed. Stool samples were analyzed using 16S rRNA Amplicon Sequencing to assess gut microbiota diversity and composition. SPSS 28.0 was used for statistical analysis.
Results:

The mean age was 13.0 $\pm$ 3.0 years, without significant difference between groups. Significant sex difference was observed in CGO versus SGO (F/M ratio 1.86 vs 0.64, p=0.05). Mean waist circumference SDS was significantly higher in SGL compared to CGL (1.7 $\pm$ 0.8 vs

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0.8±0.7, p=0.007). Increased alpha diversity in CGO and CGL compared to SGO and SGL suggested a link between reduced alpha diversity and SLD. Genera associated with both obesity and SLD included Coprococcus, Bacteroides, Alistipes, and Lachnospiraceae NK4A136. However, logistic regression identified decreased abundance of Lachnospiraceae NK4A136 and Monoglobus as significant factors in addition to increased waist circumference, for distinguishing patients with SLD. Conclusions: Obesity, liver steatosis, and metabolic syndrome correlate with distinct changes in pediatric gut microbiota. Reduced abundance of NK4A136 and Monoglobus is associated with pediatric SLD, regardless of obesity

Keyword(s) :

1949 - Development and Evaluation of Polyherbal Chitosan Nanoparticles Antioxidant and Cytotoxic Effects on HepG2 Cells
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Background / **Hypothesis:**
Cancer and oxidative stress are pathologically intertwined, driving the search for novel, multi-targeted therapies. Polyherbal formulations can offer synergistic advantages over single compounds. Berberis aristata, Andrographis paniculata, and Thevetia peruviana are individually recognized for their bioactive properties, yet their combined potential remains unexplored. This study investigates this specific polyherbal combination for its integrated antioxidant and anticancer efficacy.
Materials and **Methods:**
A polyherbal combination (2:1:1) was qualitatively and quantitatively analyzed by ultraviolet spectroscopy and subsequently loaded into chitosan nanoparticles (CS-NPs) fabricated by ionic gelation. The developed CS-NPs were characterized using scanning electron microscopy (SEM) and X-ray diffraction (XRD). The cytotoxic effect on HepG2 cell lines and the antioxidant potential (via DPPH assay) were assessed for both the polyherbal combination and its nanoformulation.
Results:

Content of berberine, andrographolide and rosmarinic acid in polyherbal combination (2:1:1) was found to be 2.188, 0.472 and 0.264%w/w. Cytotoxicity of different polyherbal combinations (2:1:1, 1:1:1, 1:1:2 and 1:2:1) and its prepared chitosan nanoparticles (2:1:1) against HepG2 cells were found to be at an IC₅₀ of 13.96, 16.47, 15.20, 16.1 and 15.67 µg/ml

respectively. 2, 2-diphenyl-1-picrylhydrazyl scavenging activity of Polyherbal combination (2:1:1) and its prepared chitosan nanoparticles (2:1:1) showed potent antioxidant potential.

Conclusions: The developed Polyherbal combination (2:1:1) and its prepared chitosan nanoparticles was found to possess antioxidant and anticancer potential.

Keyword(s) :

1950 - Decellularized Liver Matrix from Healthy versus Acute Liver Failure Livers Possesses Distinct Structural and Functional Properties
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Background / **Hypothesis:**
The liver extracellular matrix (ECM) provides a dynamic and instructive microenvironment that is fundamental to cellular homeostasis and function. In acute liver failure (ALF), this complex architecture undergoes profound pathological remodeling. We hypothesize that decellularized ECM scaffolds retain distinct compositional and structural signatures between healthy and ALF liver. Consequently, these scaffolds are predicted to differentially regulate fundamental cellular behaviors.

Materials and **Methods:**
We generated acellular liver scaffolds from healthy mouse livers (L-ECM) and from a murine model of ALF (ALF-ECM) using a standardized tissue-engineering decellularization protocol. The resulting ECM scaffolds were rigorously characterized. Residual DNA was quantified to confirm decellularization efficacy, total protein content was measured via a BCA assay, and the three-dimensional ultrastructure was visualized using scanning electron microscopy (SEM). To functionally compare the bioactivity of the matrices, extract solution of L-ECM and ALF-ECM were extracted for further testing. The concentration of the pivotal pro-regenerative cytokine VEGF within the matrix extracts

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was quantified by enzyme-linked immunosorbent assay (ELISA). Then, conditioned medium for human umbilical cord-derived mesenchymal stem cells (UC-MSCs) was prepared and used for UC-MSCs culturing. Cellular proliferation was assessed using a CCK-8 assay, and migratory capacity was evaluated via a migration assay.

Results:

Both L-ECM and ALF-ECM scaffolds met the critical criterion for effective decellularization, with residual DNA content below 50 ng/mg. However, significant differences were observed in their composition and architecture. Quantitative analysis revealed that L-ECM retained a significantly greater total protein content. SEM imaging further demonstrated that L-ECM preserved a regular, highly porous, and organized fibrillar network, whereas ALF-ECM exhibited a markedly disorganized and dense architecture with irregular pore structures. Although the concentration of VEGF in L-ECM and ALF-ECM were significantly higher than control medium, L-ECM still extracts contained a substantially higher concentration compared to ALF-ECM extracts. Similarly, while both ECMs types supported UC-MSC proliferation superior to control medium culture, the proliferative response was significantly more robust on L-ECM. Moreover, cell migration was markedly enhanced on L-ECM compared to both the control and ALF-ECM groups.

Conclusions:

This study demonstrates that the pathophysiological origin of liver dECM critically determines its composition, structure, and biofunctionality. ALF-ECM, derived from a diseased microenvironment, is characterized by compromised structural integrity and diminished retention of key growth factors like VEGF, which directly translates to its inferior capacity to direct essential cellular processes.

Keyword(s) : Decellularized extracellular matrix; Acute liver failure; Microenvironment; Cell behavior

1954 - Effects of Obinutuzumab on Cecal Ligation and Puncture Induced Septic Liver Injury in Rats

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Background / Hypothesis: Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. The liver, a central organ in the regulation of systemic inflammatory responses, becomes particularly vulnerable and undergoes significant injury during sepsis. Obinutuzumab, a type II anti-CD20 monoclonal antibody targeting B cells, has emerged as a potential therapeutic agent for modulating the immune response. This study aimed to investigate the effects of Obinutuzumab on hepatic inflammatory responses in a rat model of sepsis induced by cecal ligation and puncture (CLP), a well-established model of excessive immune activation.

Materials and Methods: A total of 58 rats were divided into six groups: Healthy, Healthy + Obinutuzumab 10 mg/kg, CLP, CLP + Obinutuzumab 2.5 mg/kg, CLP + Obinutuzumab 5 mg/kg, and CLP + Obinutuzumab 10 mg/kg. Obinutuzumab was administered intraperitoneally 1 hour after CLP. Hepatic proinflammatory cytokines, NF- κ B protein levels, and NF- κ B gene expression were assessed.

Results: Obinutuzumab treatment reduced hepatic proinflammatory cytokines in a dose-dependent manner compared with the CLP group. TNF- α and IL-6 levels were significantly reduced at 2.5 mg/kg ($p < 0.01$) and highly significantly decreased at 5 and 10 mg/kg ($p < 0.0001$). NF- κ B protein levels showed significant reductions at 5 mg/kg ($p < 0.01$) and highly significant decreases at 10 mg/kg ($p < 0.0001$), while NF- κ B gene expression was highly significantly diminished at all doses ($p < 0.0001$). In contrast, IL-1 β levels were significantly reduced only in the CLP + Obinutuzumab 10 mg/kg group ($p < 0.001$), whereas no significant differences were detected at the lower doses ($p > 0.05$).

Conclusions: These findings suggest that Obinutuzumab may protect against sepsis-induced liver injury by modulating the immune response, highlighting its potential as a therapeutic agent to prevent organ damage associated with sepsis.

Keyword(s) :

1965 - Modeling the burden of steatotic liver disease and its subcategories in people living with HIV

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Background / Hypothesis: People living with HIV (PLWH) show a high prevalence of metabolic comorbidities and other partially overlapping liver-related risk factors, including co-infections with hepatitis B (HBV) and hepatitis C (HCV) virus, which increase the risk of steatotic liver disease (SLD) and liver fibrosis, leading to worse outcomes. However, the disease burden of SLD, its different subcategories, and associated risk factors in PLWH under antiretroviral therapy remains to be determined.

Materials and Methods: In this prospectively enrolling cohort study, participants with an HIV infection were non-invasively screened using vibration-controlled transient elastography (VCTE) for SLD (controlled attenuation parameter [CAP] ≥ 248 dB/m) and fibrosis (liver stiffness measurement [LSM] ≥ 8 kPa). The Fibroscan-AST (FAST) score was used to identify those with metabolic-dysfunction associated steatohepatitis (MASH) and significant fibrosis, using a cut-off ≥ 0.67 . Uni- and multivariable logistic regression analysis was used to determine predictors of SLD.

Results: In the entire cohort of 325 participants, 71.4% were male and the median age was 51 (41; 58) years. The median HIV disease duration was 12 (5; 20) years and the majority had a CD4 cell count $\geq 500/\mu\text{L}$ (70.2%). A history of HBV and HCV was detected in 19.4% (n=63) and 5.8% (n=19), respectively. Median BMI (kg/m^2) was 25.3 (22.5; 28.5), and 17.5% (n=57) were obese (BMI ≥ 30 kg/m^2). Insulin resistance was detected in 24.3% (n=79). The overall prevalence of SLD was 50.8% (n=165), with MASLD (42.8%, n=139) showing the highest prevalence as compared to either MetALD (2.5%, n=8) or ALD (0.9%, n=3). Of those with MASLD, 23.7% (n=33) had a history of HBV or HCV coinfection (MASLD-viral). Cryptogenic or other causes (viral, undefined due to missing data) of SLD were identified in 4.3% (n=14). Metabolic comorbidities were overall more prevalent in participants with SLD. Liver fibrosis and MASH were detected in 5.8% (n=19) and 1.8% (n=6), respectively, in those with MASLD. On multivariable analysis, the cardiometabolic criterion with a BMI ≥ 25 kg/m^2 or a waist circumference $\geq 94/80$ cm (male/female) was jointly associated with SLD (OR 5.2, 95% CI 2.5-10.6, $p < 0.001$). In turn, HIV-related parameters showed no effect on the risk of SLD.

Conclusions: The burden of SLD and in particular MASLD

is high in PLWH. Screening for SLD, fibrosis, and associated metabolic comorbidities may reduce the burden of liver disease in PLWH and identify those at higher risk of liver disease progression.

Keyword(s) : PLWH; MASLD; HBV; HCV; VCTE

1967 - Hepatitis C Virus Micro-elimination in the Hospital Setting What Results Have We Achieved

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Background: Hospital-based hepatitis C virus (HCV) screening and linkage-to-care are critical to achieving HCV elimination targets. We evaluated the outcomes of a 3-year, hospital-based HCV micro-elimination model.

Materials and Methods: We conducted a prospective interventional program of HCV case-finding and linkage-to-care at the Second Affiliated Hospital of Chongqing Medical University, Chongqing, China, starting in 2022. All adult patients admitted to the hospital during the study period who tested positive for anti-HCV were included. Anti-HCV-positive patients were referred to an infectious diseases specialist for HCV RNA testing, and HCV RNA-positive patients were offered direct-acting antiviral (DAA) therapy according to international guidelines. Primary outcomes were the proportions of HCV RNA-positive patients who were (1) linked to specialist care, (2) initiated on DAA therapy, and (3) started treatment within 12 weeks of diagnosis. Outcomes were compared between the early phase (2022) and late phase (2023–2024) of the program.

Results: Among 265,645 patients tested for anti-HCV, 4,303 (1.62%) were anti-HCV-positive. Of these, 817 were HCV RNA-positive (mean age 50.4 years; 63.4% male; mean viral load 5.70 log₁₀ IU/mL), and 672 (82.3%) received DAA treatment. The proportion of HCV RNA-positive patients receiving treatment increased significantly from 78.6% in the early phase to 83.7% in the late phase ($P < 0.05$). Among HCV RNA-positive patients managed in non-hepatology and non-infectious diseases departments, the proportion treated increased from 54.4% to 70.8%. Within 12 weeks

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of HCV diagnosis, the proportion of patients initiating DAA therapy rose from 85.5% to 91.1% ($P < 0.05$).

Conclusions: This hospital-based HCV micro-elimination program significantly improved DAA treatment uptake and the timeliness of treatment initiation, including among patients managed outside hepatology and infectious disease departments. It represents an effective model for advancing HCV micro-elimination in the hospital setting.

Keyword(s) : hepatitis C virus; HCV; micro-elimination; direct-acting antivirals; DAA

1968 - Investigation of the Effects of Vasopressin V2 Receptor Inhibition on Aquaporin Expression and Liver Functions in Sepsis Induced Hepatic Injury

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Background / Hypothesis: Sepsis is a severe clinical syndrome characterized by uncontrolled activation of the systemic inflammatory response, ultimately leading to multiple organ dysfunction and high mortality. Given its central role in immune regulation and metabolic homeostasis, the liver is among the first organs in which sepsis-associated cellular and molecular injury becomes evident. Aquaporin channels (AQP2 and AQP5), which are essential for maintaining water homeostasis in hepatocytes, may be influenced by inflammation-related signaling alterations and thereby contribute to hepatic dysfunction. The MAPK signaling pathway, a key regulator of inflammatory processes, is known to be markedly activated during sepsis. Tolvaptan, a vasopressin V2 receptor antagonist, is proposed to exert modulatory effects on dysregulated aquaporin expression and MAPK-mediated inflammatory responses. This study aims to evaluate the potential of tolvaptan to modulate sepsis-associated alterations in AQP2 and AQP5 expression, disturbances in MAPK activation, and the sepsis-induced increases in hepatic IL-1 β and IL-6 levels.

Materials and Methods: Five experimental groups were established: healthy control, LPS control, and tolvaptan-treated groups receiving 15 mg/kg, 30 mg/kg, or 60 mg/kg. The sepsis model was induced by intraperitoneal lipopolysaccharide administration, and tolvaptan was given orally at the specified doses. Liver tissues were collected for the evaluation of AQP2, AQP5, and MAPK gene expression using real-time PCR, and tissue levels of IL-1 β and IL-6 were quantified by ELISA. Data were analyzed using appropriate statistical tests.

Results:

LPS administration resulted in a significant disruption of AQP2 and AQP5 gene expression, accompanied by a marked elevation in MAPK transcriptional activity. Serum IL-1 β and IL-6 concentrations were also significantly increased in the LPS control group. Tolvaptan treatment produced a dose-dependent amelioration in all assessed parameters, with higher doses yielding a pronounced attenuation of MAPK activation. Additionally, substantial reductions in IL-1 β and IL-6 levels were observed following tolvaptan administration.

Conclusions:

V2 receptor inhibition by tolvaptan modulated sepsis-associated disturbances in aquaporin expression and attenuated MAPK signaling activation, leading to a reduction in the systemic pro-inflammatory response. These findings suggest that V2 receptor antagonism may represent a mechanism-based therapeutic approach for sepsis-associated hepatic injury. Nevertheless, further studies are required to elucidate the underlying mechanisms in greater detail and to clarify its translational and clinical relevance.

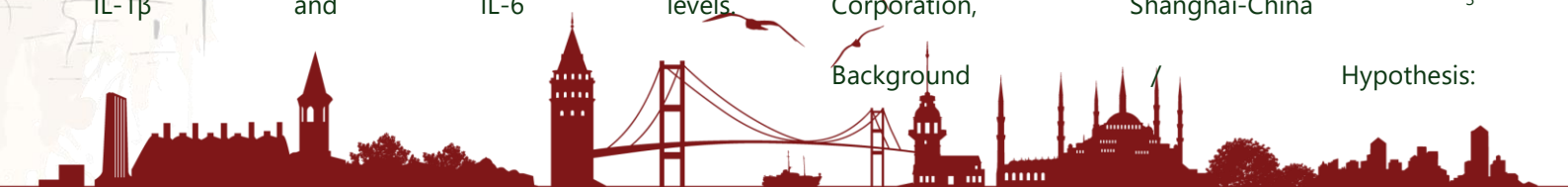
Keyword(s) : Sepsis-induced hepatic injury; Aquaporin; Vasopressin V2 receptor inhibition; Tolvaptan, Inflammation

1973 - Prognostic Utility of baseline and SVR12 24 M2BPGi for Liver Related Events in hepatitis C patients

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Background /

Hypothesis:



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Previous studies on the risk prediction of hepatocellular carcinoma (HCC) or liver-related events using M2BPGi have been conducted primarily in Japanese populations. However, no long-term study has evaluated the association between elevated M2BPGi levels and the development of liver-related events in the Chinese population.

Materials and **Methods:** This study included patients who received treatment with interferon or direct-acting antiviral (DAA) regimens for chronic hepatitis C at Peking University People's hospital between 2011 and 2018. Blood samples were collected at the initiation of treatment and at 12 or 24 weeks after the end of treatment. M2BPGi was measured using a fully automated chemiluminescent enzyme immunoassay system based on a novel sandwich immunoassay method. The diagnostic performance of M2BPGi, FIB-4 and FibroScan at baseline and SVR12/24 for predicting liver-related events was assessed using ROC curve analysis.

Results: ROC curve analysis revealed that the predictive performance of M2BPGi and FIB-4 for liver-related events at baseline and SVR12/24 was significantly superior to that of FibroScan (AUROC = 0.78, 0.83 vs. 0.69; AUROC = 0.75, 0.81 vs. 0.58, respectively). However, when the FIB-4 cut-off value was adjusted to 3.55 at baseline to match the approximately 90% specificity of M2BPGi, the predictive performance of M2BPGi was comparable to that of FIB-4. Combined assessment of M2BPGi and FIB-4 effectively improved the predictive performance for liver-related events (AUROC = 0.85), but no improvement was observed when M2BPGi was combined with FibroScan (AUROC = 0.77). At SVR12/24, M2BPGi and FIB-4 exhibited higher negative predictive values (NPV, 100.00%). A high NPV indicated that M2BPGi (cut-off value: 0.89 COI) could effectively rule out patients at low risk of non-HCC-related events. After 10 years of follow-up, patients with elevated M2BPGi levels (4.85 C.O.I. and 0.89 C.O.I., respectively) and FIB-4 levels (1.78 C.O.I. and 1.83 C.O.I., respectively) at baseline and SVR12/24 had a higher cumulative incidence of liver-related events, confirming that M2BPGi can serve as an important stratification indicator. Considering that the specificity of FIB-4 in the elderly population is susceptible to age, M2BPGi maintains a more stable false-positive rate across age groups, showing stronger age robustness and clinical applicability.

Conclusions: Notably, both before and after treatment, the diagnostic performance of M2BPGi for predicting liver-related events was comparable to that of FIB-4, but both were superior to FibroScan, suggesting its clinical

value as a non-invasive biomarker for identifying high-risk patients.

Keyword(s) : Noninvasive marker; M2BPGi; chronic hepatitis C; HCC; liver-related events;

1984 - Comparative Efficacy of Entecavir and Tenofovir Disoproxil Fumarate in Preventing HBV Reactivation in Patients Receiving Biologic Disease Modifying Antirheumatic Drugs

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Background / **Hypothesis:**

Current guidelines recommend initiating antiviral prophylaxis to prevent hepatitis B virus (HBV) reactivation in patients receiving biologic disease-modifying antirheumatic drugs (bDMARDs). The aim of this study was to compare the efficacy of entecavir (ETV) and tenofovir disoproxil fumarate (TDF) for prophylaxis.

Materials and **Methods:**

Patients who were HBsAg and anti-HBc IgG positive, HBsAg negative, and anti-HBc IgG positive (±antiHBs positive), initiated HBV prophylaxis, and continued prophylaxis for at least 1 year after bDMARDs were discontinued were retrospectively evaluated at a university hospital between 2023 and 2025. Patients receiving antiviral therapy for HBV or related HIV, hepatitis C, or D infections were excluded from the study. Patients receiving ETV (0.5 mg/day) or TDF (245 mg/day) were divided into three risk groups: low, intermediate, and high risk. The distribution of these risk groups was balanced between the ETV and TDF groups. HBV reactivation rates and side effects of the medications were evaluated.

Results:

No significant differences were found between the ETV (n = 240) and TDF (n = 123) groups in terms of demographic data, viral serological parameters, or reactivation risk profiles. HBV reactivation was observed in only one patient in the ETV group. The patient was initially HBsAg positive and HBV DNA negative, but her HBV DNA level was measured as 100 IU/ml and ALT/AST levels were elevated. Thirty-two patients (13.3%) in the ETV group (nausea, diarrhea, insomnia) and 18 patients (14.6%) in the TDF group (headache, diarrhea, rash) experienced side effects (p = 0.84). No patients required

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a change in prophylactic agent.

Conclusions:

ETV and TDF demonstrated similar efficacy in preventing HBV reactivation in patients receiving bDMARDs, and both drugs were well tolerated with similar rates of adverse events. Prospective studies with longer follow-up and detailed safety assessments are needed to better define optimal prophylaxis strategies in this population.

Keyword(s) : HBV reactivation, entecavir, tenofovir disoproxil fumarate, DMARDs

1985 - Effects of Linalool-Regorafenib Combination on Anticancer Activity in HUH 7 Cells

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Background / Hypothesis:

The aim of this study is to evaluate the effect of linalool, a compound with antioxidant properties, on the anticancer activity in HUH-7 cells when administered in combination with regorafenib.

Materials and Methods:

HUH-7 cells were treated with regorafenib and linalool for 48 hours at different concentrations (200, 100, 50, 25, 12.5, 6.25, 3.12, and 1.56 µM). Cell viability was assessed using the CCK-8 assay, and IC₅₀ values were calculated. Drug interactions were analyzed using the Chou–Talalay method, and the Combination Index (CI) was determined (CI > 1 indicating antagonism). Expression levels of EGFR, MAPK, PI3K, NF-κB, Caspase-7, and Caspase-3 were quantified using ELISA. Statistical significance was set at p < 0.05.

Results:

The linalool-regorafenib combination yielded a CI>1, indicating an antagonistic interaction. Regorafenib significantly reduced the expression levels of EGFR, MAPK, PI3K, and NF-κB compared to the control group (p < 0.05). No significant reduction was observed in the combination group (p > 0.05), and expression levels were significantly higher than those in the regorafenib group (p < 0.05). Caspase-7 and Caspase-3 levels were significantly increased in both the regorafenib and combination groups compared to the control (p < 0.05); however, the combination group exhibited significantly lower levels than the regorafenib group (p < 0.05).

Conclusions:

Co-administration of linalool with regorafenib reduces the anticancer efficacy of regorafenib by diminishing its

effects on growth signaling pathways and apoptosis.

Keyword(s) : Regorafenib, Linalool, Antagonism, EGFR, Apoptosis, HUH-7

1989 - Post Polypectomy Bleeding in Patients with Cirrhosis A Systematic Review of Risk Predictors and Procedure Related Factors

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Background / Hypothesis:

: Patients with cirrhosis are considered at increased risk for post-polypectomy bleeding (PPB) due to coagulopathy, portal hypertension, and associated comorbidities. However, the frequency and predictors of bleeding, especially in the modern era of optimized endoscopic practice, remain incompletely characterized. This systematic review aims to quantify the incidence of immediate and delayed PPB in cirrhotic patients, evaluate procedure- and patient-level predictors, and propose practical implications for clinical endoscopy

Materials and Methods:

A comprehensive literature search was performed in PubMed, Google Scholar, Scopus, and Web of Science databases for articles published up to April 2025. Retrospective or prospective studies in adults with a diagnosis of cirrhosis and undergoing colonoscopy were selected. Of these, those reporting outcomes on PPB, immediate (IPB) defined as intra-procedural or delayed (DPB) defined as bleeding within 30-d of procedure. Case reports, conference abstracts, and studies not reporting outcomes / data on interest were excluded. Data were extracted into a standardized Excel framework on patient demographics, liver disease severity (Child-Pugh class, MELD, INR, platelet count), procedural characteristics (polypectomy technique, polyp size, number, location), and clinical outcomes (IPB and DPB, transfusion, hospitalization)

Results:

:: A total of five retrospective cohort studies, encompassing 4,345 cirrhotic patients, met inclusion criteria (Table). IPB occurred in 6.3–7.5%, and DPB in 0.3–5.9% of cirrhotic patients in comparison to the risk of

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30-day PPB rate of < 0.5% to 0.16% in otherwise healthy individuals (Rabeneck et al, Gastro, 2008, Gatto et al, Gastro 2003, ASGE guideline < 1%). Risk of significant bleeding (defined as requiring hospitalization or transfusion) was rare (0.3–2.0%). Risk factors for PPB from different studies included Child-Pugh B/C (OR: 19.7), decompensated cirrhosis (OR: 1.65), ascites (OR: 2.82), polyp size ≥ 10 mm (OR: 1.087 to 8.3). Prolonged INR was associated with increased risk of IPB ($r = 0.61$) and platelet counts was associated with DPB ($r = -0.58$), with increased risk below 50,000. Colonoscopy with endoscopic mucosal resection (EMR) was associated with higher rates of IPB and DPB compared to snare polypectomy. Aspirin use (after propensity score matching) was not independently associated with increased bleeding risk. Use of antiplatelets (such as clopidogrel) within 5 days was associated with increased risk of DPB (RR = 2.42). Conclusion : Rates of PPB of colonoscopy in cirrhotic patients is higher compared to general population ranging from 0.3 to 6.1 % .{ (Kim et al., 2019)

Keyword(s) : post procedural bleed, post polypectomy, EMR, colonoscopy

2000 - Efficacy of Yangyin Fuzheng Jiedu Prescription for Hepatocellular Carcinoma with Portal Vein Tumor Thrombosis An Evaluation Using Five Propensity Score Methods
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Background / Hypothesis:
The selection of adjuvant therapies for advanced hepatocellular carcinoma (HCC) has been a subject of considerable interest. This study aims to investigate the potential value of Traditional Chinese Medicine (TCM) in improving outcomes for HCC patients with portal vein tumor thrombus (PVTT), and to compare the efficacy differences between approved proprietary Chinese patent medicines for HCC and the hospital-prepared formulation, Yangyin Fuzheng Jiedu Formula (YFJP).

Materials and Methods:
A retrospective analysis was conducted on 892 HCC patients with PVTT treated between 2012 and 2022. Based on the actual treatments received, the cohort was divided into a Western medicine (WM) group (n=579) and an Integrated Chinese and Western Medicine (ICWM) group (n=313). Data on TCM usage duration were extracted from electronic medical records.

Propensity score matching (PSM) at a 1:1 ratio and four types of propensity score weighting—Inverse Probability of Treatment Weighting (IPTW), Stabilized IPTW (sIPTW), Overlap Weighting (OW), and Standardized Mortality Ratio Weighting (SMRW)—were employed to adjust for confounding factors. Multivariable Cox proportional hazards models were used to identify independent factors influencing 5-year overall survival (OS). Kaplan-Meier survival curves were plotted to compare survival among different TCM usage patterns, durations, and types. Subpopulation Treatment Effect Pattern Plot (STEPP) analysis was utilized to identify subgroups deriving greater benefit from specific TCM formulations.

Results:

After adjustment using propensity score methods, TCM therapy was identified as an independent protective factor for prognosis in PVTT patients (adjusted HR [95% CI]: 0.4438 [0.3674, 0.5361], $p < 0.001$). Other independent factors influencing 5-year OS included etiology, WM treatment modality, platelet count, creatinine level, gamma-glutamyl transferase level, alpha-fetoprotein level, mean platelet volume, and CD8+ T-cell count. Prolonged duration of TCM administration was associated with a stronger protective effect on prognosis (p for trend < 0.001), indicating greater patient benefit. STEPP analysis revealed that patients with mild to moderate thrombocytopenia, prothrombin activity approaching 70%, or an inflammatory burden index greater than 4 derived significantly more benefit from the YFJP decoction than from CPMs ($p < 0.05$).

Conclusion: Long-term adjunctive therapy with TCM can significantly improve the prognosis of HCC patients with PVTT. The effectiveness of Chinese patent medicines was reaffirmed. Subgroups with relatively better coagulation function or more severe inflammation were more likely to benefit from the YFJP decoction, suggesting potential targets for future mechanistic research.

Keyword(s) : hepatocellular carcinoma, portal vein tumor thrombus, traditional Chinese medicine, platelet , inflammation, priority population

2002 - Acute Hepatitis B Infection Following Long-Acting Injectable Antiretroviral Therapy Drug Switch in HIV
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Background / Hypothesis:

Long-acting injectable antiretroviral therapy (LAI-ART) drug switch is a new modality of treatment option for stable HIV-infected individuals. The current regimen (cabotegravir and rilpivirine) does not have activity against Hepatitis B virus (HBV). This study evaluates cases of acute hepatitis B occurring after LAI-ART initiation and highlights the importance of HBV immunization screening and pre-treatment vaccination.

Materials and Methods:
We conducted a retrospective review of HIV-infected individuals who initiated LAI-ART between 2022-2025 at Princess Margaret Hospital, Hong Kong. Demographic, virologic, and immunization data were extracted from electronic health records. Cases of acute HBV were identified based on serologic criteria (HBsAg and/or HBV DNA positivity with anti-HBcIgM). Clinical outcomes, timing of onset, and management strategies were analyzed.

Results:

A total of 65 cases were reviewed and all cases sustained good HIV virological suppression after drug switch. For pre-treatment HBV immunity, 20 (30.8%) had serum anti-Hbs titre of 10-100 IU/ml and 41 (63%) had titre >100 IU/ml. 4 (6.2%) cases did not have protective immunity (with anti-Hbs <10 IU/ml) and 2 (3.1%) developed acute HBV infection within 6-9 months of LAI-ART initiation. Both cases presented with asymptomatic transient transaminase elevation, anti-HBc IgM and HBsAg positivity. Antiretroviral therapy were switched back to tenofovir-based 3-drug regimen. None progressed to fulminant hepatic failure and both developed s-seroconversion within 9 months of treatment.

Conclusions:

Patients with HIV are at high risk for acquiring HBV due to shared routes of transmission. Our findings underscore the potential risk of acute HBV infection in patients receiving LAI-ART without protective HBV immunity. Routine HBV carriage and immunity screening, timely vaccination should be mandatory before initiating long-acting therapy to prevent

avoidable liver complications and chronic HBV infection.

Keyword(s) :

2006 - Phenotypic Pattern of Acute Liver Injury in Crimean Congo Hemorrhagic Fever Among Patients with Chronic Hepatitis B in an HBV Endemic Region
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Background / Hypothesis:

Crimean-Congo hemorrhagic fever (CCHF) frequently causes acute liver injury through direct hepatocellular damage and immune-mediated mechanisms. In Hepatitis B Virus (HBV)-endemic regions, a proportion of CCHF patients have unrecognized chronic hepatitis B (CHB), yet the phenotypic expression of acute liver injury in this coinfecting population is poorly defined. We aimed to characterize the hepatic injury pattern and clinical outcomes of CCHF in patients with chronic HBV.

Materials and Methods:

We retrospectively reviewed 20 HBV-positive patients with polymerase chain reaction (PCR)-confirmed CCHF followed between 2011-2025 in Northeastern Türkiye. Demographic data, laboratory parameters (Alanin aminotransferase (ALT)-max, International normalised ratio (INR)-max, albumin-min, platelet-min), transfusion requirement and CCHF severity scores were collected. A phenotypic classification of liver injury was defined as:

- Mild injury: ALT <5× Upper Limit Normal (ULN), INR <1.20, albumin ≥3.2 g/dL
- Moderate injury: ALT ≥5×ULN and/or INR 1.20-1.49
- Severe hepatic dysfunction: INR ≥1.5, albumin <3.0 g/dL, and/or classified as severe CCHF.

Clinical outcomes included transfusion requirement, hospitalization duration and in-hospital survival.

Results:

Among 20 CHB-positive CCHF patients (mean age 60.5 years; 80% male), 17 (85%) had mild-to-moderate disease and 3 (15%) met criteria for severe CCHF.

- ALT-max ranged from 22-358 U/L;
- INR-max ranged 0.83-1.61;
- Albumin-min ranged 3.0-3.9 g/dL;
- Platelet levels ranged 22-151 ×10³/mm³.

Phenotypic distribution:

- Mild injury: 11/20 (55%)
- Moderate injury: 6/20 (30%)
- Severe hepatic dysfunction: 3/20 (15%)

Comparison of ALT (max), INR (max), platelet (min) and

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hospital stay lengths of the patients is shown at Table 1. ALT (max) and INR association is demonstrated at Figure 1.

Transfusion was required in 5/20 (25%). Median hospitalization duration was 7 days (IQR 4–10). All patients survived and were discharged in good clinical condition.

Importantly, even patients with known CHB, elevated baseline ALT or high HBV DNA—including one with HBV DNA >1,000,000 IU/mL—did not demonstrate disproportionate hepatic decompensation during CCHF.

Conclusions:

In this HBV-endemic cohort, most HBV-positive CCHF patients exhibited mild-to-moderate acute liver injury, and severe hepatic dysfunction was uncommon despite underlying CHB. These findings suggest that chronic HBV infection alone may not predispose to worsened hepatic outcomes during CCHF, except in cases with advanced fibrosis or severe CCHF phenotype. Prospective studies with larger cohorts and fibrosis-stage stratification are warranted.

Keyword(s) : Crimean-Congo Hemorrhagic Fever, Hepatitis B, Liver injury

2012 - Eliminating Viral Hepatitis by 2030 Goals and Gaps from a Large Scale Community Based Study in Shenzhen China

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Background / Hypothesis:
Aligning with the WHO's goal to eliminate viral hepatitis as a public health threat by 2030, this study evaluates the current epidemiological profile of viral hepatitis in community-based populations in Shenzhen, China and identifies key challenges toward its elimination.

Materials and Methods:
Between September 2023 and July 2025, large-scale community screening was implemented across 95 community health centers in Shenzhen. Testing included hepatitis B surface antigen (HBsAg), surface antibody (HBsAb), and hepatitis C antibody (anti-HCV), alongside the collection of epidemiological data.

Results:

A total of 96,170 tests were performed (51,168 for hepatitis B; 45,540 for hepatitis C). The overall HBsAg positivity rate was 4.32%, with a marked age gradient:

the highest prevalence was among those aged 30–60 years, while children under 5 years had a positivity rate of only 0.15%. Notably, the hepatitis B diagnosis rate (awareness of infection status) was only 9.69% (214/2,209). Among HBsAg-positive individuals, 78.23% were unaware of their family history of hepatitis B, and 80.36% did not know their family history of cirrhosis or liver cancer. The HBsAb seropositivity rate, indicating immune protection, was 28.40%, whereas 66.27% of the population remained susceptible (both HBsAg and HBsAb negative). Among susceptible individuals, 90.77% had an undocumented vaccination history. The hepatitis C infection rate was 0.28%, with the highest prevalence (0.54%) among adults over 60 years, reflecting distinct age clustering.

Conclusions:

Shenzhen has achieved notable success in hepatitis B control, particularly in pediatric vaccination. However, three critical gaps persist: a substantial proportion of cases remain undiagnosed, susceptibility remains high among adults, and risk awareness is critically low in key populations. To meet the 2030 elimination target, efforts must prioritize innovative community screening strategies, catch-up vaccination for susceptible adults, and the establishment of an integrated "screen-diagnose-treat-manage" continuum of care. This study provides a valuable evidence base for tailoring elimination strategies in Shenzhen and comparable urban settings in China.

Keyword(s) : Hepatitis B; Epidemiology; Community screening; Diagnosis rate; Hepatitis elimination; Shenzhen

2019 - Cardiometabolic Risks in Elderly Individuals With Fatty Liver Living in Mid Mountain Regions of Central Asia

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Background / Hypothesis:
Elevated altitude is known to modulate metabolic pathways through chronic hypobaric hypoxia, autonomic activation, and HIF-1 α -mediated inflammatory responses.

Materials and Methods:
An open comparative study was conducted among individuals with fatty liver as determined by ultrasound (n=705) living in low-altitude (750–800 m above sea level, n=349) and mid-altitude (2046–2300 m above sea

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level, n=356) areas of Central Asia. Patients aged 60 years and older were compared.

Results:

We studied the frequency of cardiometabolic risks depending on the altitude of residence, which revealed significant age-related differences. The following parameters were used: waist circumference, blood pressure, triglycerides, HDL, fasting glucose, HbA1c. It should be noted that a waist circumference above 90/80 cm (for men and women) among the elderly with fatty liver reached 72.5% versus younger people (48%). In individuals over 60 years of age living in mid-altitude conditions, compared with residents of low-altitude areas, three or more components of metabolic syndrome were observed. The frequency of a combination of three components exceeded 18% (versus 6-10% in patients under 60 years of age), and four components were more than twice as frequent compared to the younger age category. The presence of five components was rare, but was recorded predominantly in patients in the older group, which emphasizes the severity of metabolic imbalance in the elderly living in hypoxic mid-altitude conditions. Characteristically, one or two components of metabolic syndrome remained dominant in both age groups, consistent with early and subclinical stages of metabolic decompensation. However, in the older patient group, the frequency of a combination of two components was statistically higher (up to 30%), which may reflect accelerated age-related remodeling of metabolic status under chronic hypobaric conditions. Among individuals under 60 years of age, metabolic burden is less pronounced; however, the high proportion of patients with one or two components also indicates an unfavorable background and the potential early development of MAFLD in the region. In elderly residents of mid-mountain areas, fatty liver disease was frequently associated with hypertension (70% vs. 31%), coronary heart disease (48.8% vs. 15.9%), and COPD (8.4% vs. 2.5%), which were more than twice as common among younger patients. OSA occurred in a third of patients. As expected, chronic cholecystitis, pancreatitis, gallstone disease, and chronic kidney disease were common associated. Conclusions. Our data demonstrate an independent association of residential altitude with a more pronounced metabolic phenotype of fatty liver in the elderly.

Keyword(s) : fatty liver, comorbidity, cardiometabolic risks, mid-altitude, low-altitude.

2023 - Impact of Liver Fat Contents on Brain Structure and Cognitive Functions in Patients with MASLD

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Background / **Hypothesis:**
This study aims to investigate the association between liver fat contents using magnetic resonance imaging-proton density fat fraction (PDFF) with brain structure and cognition in adults with metabolic dysfunction-associated steatotic liver disease (MASLD).

Materials and **Methods:**
Cross-sectional relationships of MASLD with cognitive testing of memory, attention, and executive function were assessed, while adjusting for multiple variables including type 2 diabetes, hypertension and smoking status. Neuroimaging outcomes included brain volumes, regional homogeneity, amplitude of low-frequency fluctuations, functional connectivity (FC) and cerebral blood flow using arterial spin labeling (ASL), reflecting cerebrovascular health.

Results:

Eighty-six patients were included (mean age 29.97 ± 11.23 years; 74.42% men; mean body mass index 30.01 ± 5.82 kg/m²). Liver fat contents was negatively associated with the volume in the hippocampus ($\beta = -0.21$, 95% CI -0.087 – -0.008), parahippocampal gyrus ($\beta = -0.269$, 95% CI -0.096 – -0.022) and precuneus ($\beta = -0.299$, 95% CI -0.099 – -0.017). Cognitive function did not correlate with liver fat contents. Dividing 86 patients into 2 groups according to the median of liver fat deposition at 17.73%, increased FC between the right parahippocampal gyrus and the right precuneus was found in group where liver fat deposition > 17.73%.

Conclusions:

Participants with more liver fat may have smaller volumes of cognitive brain regions, particularly those correlating with memory and self-related information processing. Moreover, the FC result showed that functional connectivity of the right parahippocampal gyrus-precuneus was significantly positively associated with liver fat deposition.



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Keyword(s) : Liver Fat Contents, Brain Structure, Cognitive Functions

2028 - **sepsis**

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Background / Hypothesis:

ddv avdav adsf vadgew rgrwe 3rgeag sf vavqa evqaeq
dvf dv

Materials and Methods:

Results:

Conclusions:

Keyword(s) : dvca

2039 - Modelling liver fibrosis and MASH with humanized immune system mice

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Reliable systems that reproduce human liver biology are required for preclinical assessment of human-specific compounds aimed at fibrosis and steatohepatitis. In this study, we characterize two experimental approaches to induce liver pathology using humanized immune system NCG (HIS-NCG) mice.

NCG mice reconstituted with human CD34⁺ hematopoietic stem cells were assigned to one of two injury paradigms. The first involved repeated administration of carbon tetrachloride (CCl₄) for 23 days, whereas the second relied on a 14-day high-fat diet lacking methionine and choline (HFMCD).

Both interventions produced acute deterioration in general health condition and clear biochemical evidence of hepatic damage, as shown by ALT increases, but led to divergent pathological phenotypes. CCl₄ exposure produced severe liver fibrosis, with most mice developing high-grade (F3) fibrosis or cirrhotic architecture without steatosis or human macrophage infiltration. Conversely, the HFMCD regimen induced a broad phenotype consistent with MASH, including a

NAS score of 6, low-grade (F1) fibrosis, inflammatory infiltration by human CD68⁺ macrophages, and increased circulating LGALS3BP.

These two systems provide distinct platforms for examining agents directed at human immune-mediated drivers of liver pathology: the HFMCD model for MASH and the CCl₄ model for advanced fibrosis. An extended configuration incorporating both human immune and hepatic elements is also available for studies that require simultaneous evaluation of immune and metabolic pathways.

Keyword(s) : MASH, fibrosis, humanized mice, preclinical, model

2041 - A Rare Presentation of Salmonella enteritidis Bacteremia With Coronary Mycotic Aneurysm and Non Cirrhotic Portal Hypertension

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Background:

Salmonella enteritidis bacteremia is an uncommon but clinically important cause of systemic infection, with the potential to involve vascular structures and form mycotic aneurysms. Evaluation of fever of unknown origin (FUO) requires a structured, stepwise diagnostic approach utilizing accessible imaging modalities to identify both common and unexpected causes.

Case

Presentation:

A 51-year-old man with immune thrombocytopenia managed on chronic corticosteroids presented in septic shock after treatment of a right gluteal abscess. Initial abdominal ultrasound demonstrated nonspecific parenchymal alterations in a normal-sized liver, along with nodular changes in both lobes, prompting further evaluation with contrast-enhanced CT. CT imaging revealed periportal hypodensities, signs of portal hypertension, and an incidental 6.5 × 4.4 cm non-enhancing hypodense collection adjacent to a coronary artery. Blood cultures subsequently yielded Salmonella enteritidis. Coronary CT angiography confirmed fusiform aneurysms of the right coronary artery with circumferential thrombus and minimal calcific plaque, consistent with a Salmonella-related mycotic aneurysm. Antithrombotic therapy was deferred in the context of underlying thrombocytopenia. The patient received

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culture-directed intravenous antibiotics, resulting in clinical stabilization, and was discharged on a course of outpatient parenteral therapy.

Conclusion:

The combination of predisposing immunologic factors, persistent systemic symptoms, distinctive imaging abnormalities, and positive blood cultures supported the diagnosis of a *Salmonella* mycotic aneurysm. Given the high risk of rupture and substantial associated mortality, timely diagnostic evaluation, appropriate antimicrobial therapy, and consideration of surgical or percutaneous intervention are critical. This case highlights the essential role of sequential imaging and individualized, multidisciplinary management in identifying and treating uncommon vascular complications in immunocompromised patients.

Keyword(s) : non-cirrhotic portal hypertension; salmonella enteritidis infection; mycotic aneurysm

2047 - Controlled expansion stent grafts in TIPS a real world analysis of in vivo diameter change and early hepatic encephalopathy

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Background / **Hypothesis:** Post-TIPS hepatic encephalopathy (HE) remains a major limitation of portosystemic shunting, and stent diameter is a key modifiable determinant of HE risk. The VIATORR® Controlled Expansion (VCX) stent graft was designed to allow tighter control of the functional lumen (8–10 mm) and potentially reduce HE by preventing passive self-expansion seen in conventional VIATORR TIPS stents (VTS). However, clinical data remain conflicting and information regarding the in vivo expansion behavior of VCX in real-world practice is limited. This study compared early HE after TIPS using VCX versus 8-mm VTS and quantitatively characterized VCX expansion and associated hemodynamic changes.

Materials and **Methods:** This single-center retrospective cohort included consecutive cirrhotic patients undergoing TIPS between January 2024 and July 2025, receiving either an 8-mm VTS or VCX stent-graft. VCX stents were initially dilated to 8 mm and further dilated to 10 mm only if

post-procedural portal pressure gradient (PPG) remained >12 mmHg or decreased <50% from baseline. The primary endpoint was new-onset OHE within 6 months. Stent diameter was quantitatively measured along the VCX controlled-expansion mid segment on fluoroscopy at baseline and 3 months. PPG measurements were also obtained at both timepoints.

Results:

Among 153 patients (43 VCX, 110 VTS), OHE occurred in 30 patients (19.6%). The VCX group showed a higher cumulative 6-month OHE incidence than the VTS group (HR = 2.34, 95% CI 1.05-5.20; log-rank $p = 0.032$). Among VCX patients, the mid-segment diameter increased from 8.0 mm to a mean of 8.65 mm (range 8.2–9.1 mm) at 3 months ($p < 0.001$). Post-TIPS PPG increased from 7.7 mmHg to 11.6 mmHg ($p < 0.001$). Stent expansion was not correlated with PPG increase (Spearman $r=0.038$, $p=0.834$).

Conclusions:

VCX stent-grafts demonstrated consistent early in vivo expansion and are associated with a higher incidence of early OHE compared with 8-mm VTS. The observed PPG rise at 3 months underscores the unreliability of immediate intra-procedural PPG as a surrogate for long-term hemodynamic status. These findings support the need for refined VCX-specific dilation strategies—such as staged or intentionally smaller initial dilation—to optimize the balance between portal decompression and HE risk. Prospective studies are warranted to validate individualized diameter-modulation protocols.

Keyword(s) : transjugular intrahepatic portosystemic shunt; hepatic encephalopathy; portal pressure gradient; controlled expansion stent; stent diameter

2065 - Comprehensive multiomics study of ballooned hepatocytes in MASH Insights into cellular adaptation to metabolic overload

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Background/Hypothesis: Ballooned hepatocytes (BHs) are key indicators for MASH diagnosis, treatment, and prognosis. However, the molecular features of BHs remain largely undefined. This study aimed to perform integrated transcriptomic and proteomic analyses of MASH BHs in situ to elucidate their molecular phenotype and role in disease pathogenesis.

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Materials and Methods: Three MASH models were developed using diet-induced mouse groups: wild-type C57 mice on high-fat, high-glucose, and choline-deficient diets; C57 mice on high-fat, high-glucose, and high-cholesterol (HFFC) diets; and leptin-deficient C57 ob mice on HFFC diets, with corresponding normal diet controls. Liver tissues were subjected to laser-capture microdissection to isolate BHs, macrovesicular and microvesicular steatotic hepatocytes (SHs), and normal hepatocytes (NHs). Cell samples were processed and underwent RNA sequencing and mass spectrometry for omics analyses. Data validation was performed via quantitative PCR and immunohistological examination. Clinical relevance was assessed using MASLD cohort databases and surgical MASH liver specimens. **Results:** A total of 62,939 cells underwent transcriptome sequencing, and 122,358 cells underwent mass spectrometry-based proteome analysis, identifying 22,046 protein-coding genes and 7,438 proteins. The crucial markers suggested by omics data were confirmed through morphological and biochemical assays. The clinical relevance of BH proteome was demonstrated through blood proteomics analysis of MASH cohorts and multiplex tissue staining of patient slices. Integrated omics analyses indicated consistent functional alterations in BHs compared with NHs and SHs across the models. Notably, BHs exhibited widespread downregulation of fundamental processes, involving metabolism, immune response, cell death and proliferation, morphogenesis, and cell junctions, as well as diminished competence of mitochondria, ER, and actin cytoskeleton. Cell evolutionary projection suggested that BHs could be transitional forms of SHs. **Conclusions:** BHs appear to be a self-protective, dormant state of hepatocytes in response to damaging metabolic overload. They serve as markers of metabolic stress rather than malignant drivers in MASH progression. This research fills a critical knowledge gap regarding BH molecular features and offers new insights into MASH pathogenesis.

Keyword(s) : Metabolic dysfunction associated steatohepatitis, ballooned hepatocytes, transcriptomics, proteomics, laser-captured microdissection.

2066 - Phenotypic Age is a Hepatocentric Marker of Biological Aging Stronger Associations of Liver Related and Metabolic Outcomes over Cardiovascular Events in Steatotic Liver Disease

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Background / **Hypothesis:** Phenotypic age (PhenoAge) captures physiological dysregulation beyond chronological age. While often viewed as a generic mortality marker, its organ-system specificity remains undefined. We hypothesized that PhenoAge predominantly reflects hepatic and metabolic biological aging rather than vascular senescence, and thus better predicts metabolic dysfunction and liver-related outcomes than cardiovascular disease (CVD), particularly within the growing burden of steatotic liver disease (SLD).

Materials and **Methods:** We analyzed 443,072 UK Biobank participants free of baseline liver or cardiovascular disease. PhenoAge Acceleration (PhenoAgeAccel = PhenoAge – chronological age) was derived from clinical biomarkers. SLD was defined by the Hepatic Steatosis Index. Outcomes included incident T2D, CVD, and liver-related disease (LRD; defined as fatal/non-fatal cirrhosis, decompensation events such as ascites/varices, or hepatocellular carcinoma) ascertained over a median 14.8-year follow-up. Multivariable Cox models, adjusted for major confounders, estimated hazard ratios (HRs) for accelerated versus non-accelerated aging, stratified by SLD status. Predictive utility was assessed by the change in concordance index (Δ C-statistic) over conventional risk factors.

Results:

Among 178,763 participants with SLD, accelerated aging was powerfully associated with incident T2D (HR 2.64, 95% CI 2.55–2.74) and LRD (HR 1.87, 1.56–2.25), but modestly with CVD (HR 1.29, 1.24–1.35). This distinct hepatometabolic signature persisted in non-SLD participants, where the risk magnitude for T2D (HR 2.59) and LRD (HR 2.23) far exceeded that for CVD (HR 1.30). Crucially, adding PhenoAgeAccel to baseline models significantly improved discrimination for LRD (Δ C-index 0.04, $p < 0.001$; C-index increasing from 0.80, 95% CI 0.79–0.82 to 0.84, 95% CI 0.82–0.85), whereas the incremental value for CVD prediction was minimal (Δ C-index < 0.01 ; C-index unchanged at 0.70, 95% CI 0.69–0.71).

Conclusions:

Phenotypic age is not a generic aging marker but a robust indicator of metabolic and hepatic biological

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aging. It substantially enhances risk stratification for liver-related endpoints beyond traditional risk factors, with limited added value for cardiovascular prediction. In the era of precision hepatology, incorporating PhenoAge into clinical risk algorithms may improve early identification of individuals with SLD at highest risk of progression to overt liver disease and metabolic complications.

Keyword(s) :

2067 - Hepatic Arterial Infusion Chemotherapy Combined with Donafenib and PD 1 Inhibitor for Advanced Hepatocellular Carcinoma with Lung Metastases A Multicenter Retrospective Study

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Background / Hypothesis: Advanced hepatocellular carcinoma (HCC) with lung metastasis carries a poor prognosis. Donafenib combined with PD-1 inhibitor has become a standard systemic therapy for advanced HCC. Hepatic arterial infusion chemotherapy (HAIC) demonstrates potent local tumor control. However, the efficacy of adding HAIC to donafenib plus PD-1 inhibitor in this population remains undefined. This study aimed to evaluate the clinical efficacy and safety of hepatic arterial infusion chemotherapy (HAIC) combined with donafenib and programmed cell death protein-1 (PD-1) inhibitor in patients with hepatocellular carcinoma (HCC) and pulmonary metastases.

Materials and Methods: This multicenter retrospective study enrolled treatment-naïve patients with advanced HCC (BCLC stage C) and lung metastases who received donafenib plus PD-1 inhibitor with or without HAIC between March 2019 and June 2023. Propensity score matching (PSM) was performed to minimize selection bias between treatment arms. The primary endpoint was overall survival (OS). Secondary endpoints included progression-free survival (PFS), objective response rate (ORR), and disease control rate (DCR) assessed per RECIST 1.1 criteria, along with treatment-related adverse events (TRAEs).

Results:

Of 180 eligible patients enrolled, 78 received HAIC plus donafenib and PD-1 inhibitor (HDP group) and 102

received donafenib plus PD-1 inhibitor alone (DP group). After 1:1 PSM, 60 patients remained in each cohort. The HDP group achieved a significantly prolonged median OS compared with the DP group (22.8 versus 11.5 months; hazard ratio [HR]: 0.44; 95% confidence interval [CI]: 0.29–0.67; $P < 0.001$). Median PFS was also superior in the HDP group (8.3 versus 5.2 months; HR: 0.68; 95% CI: 0.47–0.98; $P = 0.038$). The HDP group exhibited higher ORR (43.3% versus 16.7%, $P = 0.002$) and DCR (81.7% versus 65.0%, $P = 0.039$). Although TRAEs of any grade and grade ≥ 3 occurred more frequently in the HDP group, all events were clinically manageable with no treatment-related deaths reported.

Conclusions:

The combination of HAIC with donafenib and PD-1 inhibitor demonstrates favorable survival outcomes and acceptable tolerability in advanced HCC patients with lung metastases, warranting further investigation in prospective studies.

Keyword(s) : Hepatocellular carcinoma; Lung metastasis; Hepatic arterial infusion chemotherapy; Donafenib; PD-1 inhibitor

2068 - The impact of implementing the expanded antiviral therapy eligibility for HBsAg positive pregnant women in China a modelling study

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Background / Hypothesis: The World Health Organization's 2024 guideline of hepatitis B virus (HBV) expanded the eligibility for antiviral therapy to eliminate HBV mother-to-child transmission (MTCT), recommending treatment for HBsAg-positive pregnant women who cannot access HBV DNA or HBeAg tests. This study aimed to evaluate the potential impact of implementing expanded interventions in China.

Materials and Methods: We conducted a survey and developed a simulation model incorporating data on the natural history of HBV, prevalence, HBV DNA testing coverage, HBeAg testing coverage, and antiviral treatment coverage. We estimated the impact of existing interventions and expanding interventions for prevention of MTCT. We simulated 810 scenarios. For prenatal intervention in HBsAg-positive pregnant

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women, the existing interventions administers antiviral treatment to those with HBV DNA levels $\geq 200,000$ IU/mL or HBeAg positivity. The expanded interventions extends treatment to pregnant women without access to HBV DNA or HBeAg testing. A decision tree and Markov model was used to evaluate the cost-effectiveness.

Results: A survey indicated that the testing coverage was 52.7% in resource-limited areas and 90.31% in resource-replete areas, while the treatment coverage were 64% and 81%, respectively. Overall, our model demonstrated that expanded interventions would avert substantially more new infections than existing interventions across all scenarios. Specifically, expanded interventions could reduce new infections by 53.04% in resource-limited areas and by 41.56% in resource-replete areas, while simultaneously lowering per-capita costs by US\$1134.28 and US\$294.10, respectively. The expanded interventions were cost-effective across all simulated scenarios, with an incremental cost-effectiveness ratio of -\$16,363.77 per quality-adjusted life year. The probabilistic analysis indicated that at a willingness-to-pay threshold of the three times gross domestic product per-capita in China, the probability of expanded interventions being cost effective was 95.68% in resource-limited areas and 96.58% in resource-replete areas, respectively. One-way deterministic sensitivity analyses showed the robustness of the results.

Conclusions: Compared to the existing interventions, the expanded antiviral interventions could contribute to averting new HBV infections and was cost-effective in China, which would accelerate the elimination of hepatitis B.

Keyword(s) : hepatitis B virus, mother-to-child transmission, antiviral prophylaxis, cost-effective

2081 - Late Presentation and Poor Outcomes of Hepatocellular Carcinoma in a Resource Limited Setting An Observational Study in a Tertiary Care Hospital Bangladesh

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a major cause of cancer-related mortality

worldwide, with a disproportionately high burden in resource-poor countries. Limited access to screening, delayed diagnosis, and restricted availability of curative treatment options contribute to poor outcomes.

Materials and Methods: A medical college hospital-based observational study was conducted among patients diagnosed with HCC from January 2021 to December 2022. Data were collected from the hospital record system. Demographic data, underlying etiology, mode of diagnosis, BCLC stage at presentation, treatment received, and survival outcomes were recorded and analyzed.

Results: A total of 52 patients with hepatocellular carcinoma (HCC) were included, of whom 84.61% were male and 15.39% were female. The most common underlying etiology was HBV-related cirrhosis. None of the patients had been enrolled in an HCC surveillance program prior to diagnosis. Ultrasonography failed to detect HCC in 65.35% of cases, and all diagnoses were confirmed with contrast-enhanced CT (CECT). At presentation, 59.61% of patients were classified as BCLC stage D, and 30.76% as stage C, with no patients presenting in BCLC stage A. Regarding treatment, 61.53% received palliative care, and 21.15% received systemic therapy; no patient received curative treatment. Outcomes were poor, with 53.84% of patients dying within 3 months of diagnosis and the remainder within one year.

Conclusions: HCC in resource-poor settings is characterized by late detection, limited access to diagnostic tools, and restricted therapeutic options, resulting in poor survival. Strengthening screening programs, improving access to antiviral therapy, and establishing affordable treatment pathways are essential to reduce the burden and improve patient outcomes.

Keyword(s) : HCC – Hepatocellular Carcinoma, BCLC – Barcelona Clinic Liver Cancer Classification, CECT – Contrast-Enhanced Computed Tomography

2083 - Targeting STK40 unleashes anti tumor immunity by triggering tumor vulnerability and T

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cell **reinvigoration**

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Background / **Hypothesis:**
Immunotherapy has revolutionized cancer treatment, yet its clinical efficacy in hepatocellular carcinoma (HCC) remains limited, with the underlying mechanisms of resistance poorly defined.

Materials and **Methods:**
The regulators of immune evasion in HCC were identified through in vivo CRISPR screening. Multiple animal models, including subcutaneous tumorigenesis models and spontaneous liver cancer models, were employed to validate the role of immune regulator. In-depth molecular mechanism studies were conducted to elucidate the functional mechanisms of the immune regulator.

Results:

Through an in vivo CRISPR-Cas9 screen, we identify Serine/Threonine kinase 40 (STK40) as a previously unrecognized master regulator of immune evasion in HCC. Functionally, combining Stk40 ablation with PD-1 blockade induces complete tumour regression and durable immune memory in preclinical models. Hepatocyte-specific Stk40 knockout fully abrogates MycOE/Trp53KO-driven HCC tumorigenesis following hydrodynamic delivery. Mechanistically, STK40 acts as a scaffold for the COP1 E3 ubiquitin ligase, promoting interferon gamma receptor 1 (IFNGR1) degradation, thereby rendering tumour cell resistant to IFN- γ -mediated cytotoxicity. Genetic depletion of Stk40 stabilizes IFNGR1, restoring tumour cell sensitivity to T cell cytotoxicity. Concurrently, Stk40 loss triggers autonomous GM-CSF secretion, enhancing the infiltration and activation of conventional type 1 dendritic cells (cDC1s), which promotes antigen cross-presentation and CD8⁺ T cell activation. Notably, inducible Stk40 knockout mice exhibit no overt physiological abnormalities, suggesting that STK40 targeting is therapeutically safe. Pharmacological inhibition of STK40 using antisense oligonucleotides, combined with PD-1 blockade elicits potent anti-tumour responses across multiple cancer types.

Conclusions:

Our findings establish STK40 as a unique dual-function

therapeutic target that simultaneously enhances tumour-intrinsic IFN- γ responsiveness and tumour-extrinsic cDC1-mediated T cell activation, overcoming two major barriers to effective anti-tumour immunity.

Keyword(s) : hepatocellular carcinoma, Serine/Threonine kinase 40, immunotherapy, immune evasion

2087 - CD146 Regulates Hepatocyte Lipotoxicity through Modulation of Mitochondrial Energy Metabolism and Inflammatory Response

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Background / **Hypothesis:** Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide, with its pathogenesis involving a vicious cycle of lipid metabolism dysregulation, mitochondrial dysfunction, and chronic inflammation. Previous studies using a high-fat, high-fructose diet-induced MASLD mouse model demonstrated that CD146 deficiency significantly ameliorates hepatic lipid deposition, reduces pro-inflammatory immune cell infiltration, and promotes the restoration of hepatocyte mitochondrial structure and quantity. Building on these findings, this study aims to elucidate the specific molecular mechanisms by which CD146 regulates mitochondrial function and inflammatory responses in hepatocyte lipotoxic injury.

Materials and Methods: In this study, siRNA-mediated gene silencing was employed to establish a CD146 low-expression model in AML-12 hepatocytes. A lipotoxic stress microenvironment was simulated using combined oleic acid/palmitic acid (OA/PA) treatment. The pathophysiological effects of CD146 deficiency were systematically evaluated through RT-qPCR, western blot analysis, Nile Red staining, and ELISA.

Results: RT-qPCR analysis revealed that CD146 knockdown significantly downregulated the gene expression of key lipid synthesis transcription factor SREBP-1, metabolic regulatory kinase PDK2, and pro-inflammatory cytokine IL-1 β following OA/PA treatment. Protein analysis showed upregulation of the mitochondrial fission protein Drp1, while CD146 silencing alleviated lipid deposition and reduced ROS and ATP levels. Functional assays demonstrated that CD146 knockdown specifically inhibited the activity of

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mitochondrial respiratory chain complex V, with no significant effect on complexes I-IV.

Conclusions: The in vitro experimental results are consistent with previous animal studies, indicating that CD146 likely plays a critical role in hepatocyte lipotoxic injury by coordinately regulating lipid synthesis, inflammatory responses, and mitochondrial energy metabolism. These findings suggest that CD146 may serve as an important molecular target linking metabolic stress and inflammatory responses, providing a new direction for intervention strategies in MASLD.

Keyword(s) : CD146, Lipotoxicity, Mitochondrial Energy Metabolism, Inflammatory Response

2092 - Paris II Outperforms Other UDCA Response Criteria to predict Transplant Free Survival in Asian PBC Patients

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Background / Hypothesis: Biochemical response to ursodeoxycholic acid (UDCA) is the cornerstone of prognostic stratification in Primary Biliary Cholangitis (PBC). While multiple criteria define treatment response, their performance may vary across ethnicities and

regions. We evaluated the comparative prognostic utility of established UDCA response criteria in a large, multi-ethnic South-East Asian (SEA-PBC) cohort.

Materials and Methods: The multicenter SEA-PBC registry included consecutive patients diagnosed between 1980 to 2024 from 13 institutions within the Asia-Pacific regions. We analysed UDCA-treated patients with sufficient data to assess 1-year biochemical response. The primary outcome was transplant-free survival (TFS). The prognostic performance of response criteria (Paris-II, Toronto, POISE, ALP normalization & deep response) was analysed using competing-risk Fine-Gray regression and compared via time-dependent AUC.

Results: Among 1,128 patients (85% female, median age 62), 98.8% received UDCA. One-year response rate varied significantly: Paris-II (54%), Toronto (67%), POISE (32%), ALP normalization (39%), and deep response (16.2%). All criteria significantly predicted TFS ($p < 0.05$). Paris-II demonstrated the strongest prognostic discrimination, with the subdistributional hazard ratio (sHR 0.13, 95%CI: 0.06-0.30, $p < 0.001$) and time-dependent C-statistics (AUC 0.68-0.70). Paris-II responders were typically older, female, and had less severe baseline disease. Among Paris-II non-responder, only 12.6% received second-line therapy (Fibrates 13%, 1% Obeticholic acid).

Conclusions: The Paris-II criteria provide a strong prognostic discrimination for transplant-free survival in SEA-PBC cohort. A striking treatment gap among Paris-II non-responders highlights a critical unmet need for second-line treatment in SEA-PBC cohort.

Keyword(s) : Primary Biliary Cholangitis, Treatment response, Outcome, Survival, SEA-PBC

2094 - Inhibition of Aurora B Induces Senescence and Potentiates Immunotherapy in Hepatocellular Carcinoma

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a global health challenge, with limited treatment options for advanced-stage patients. Although recent approvals of targeted therapies and immune checkpoint inhibitors (ICIs) have expanded the therapeutic landscape, their clinical benefits are often constrained by modest response rates

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and acquired resistance. Despite ongoing efforts to identify new therapeutic targets, only few inhibitors have progressed to clinical trials, benefiting a small subset of patients. These limitations underscore the need for more effective therapeutic strategies.

Materials and **Methods:** We integrated data from DepMap and TCGA to identify HCC-specific vulnerabilities. A kinome-wide CRISPR screen validated essential targets. Pharmacological inhibition (AZD1152) and genetic knockout of Aurora B (AURKB) were employed for functional validation. Senescence was assessed via SA- β -gal staining, transcriptomics, and SASP analysis. Immune interactions were evaluated using co-culture assays with T cells and flow cytometry for MHC-I and immune markers. In vivo efficacy was tested in immunocompetent murine HCC models.

Results:

Aurora B was identified as a top candidate essential for HCC survival, overexpressed in tumors, and correlated with poor prognosis. A kinome-wide CRISPR screen further confirmed its essential role in HCC cell survival. Pharmacologic inhibition or genetic knockout of Aurora B markedly suppressed proliferation of HCC cells and induced robust cellular senescence, characterized by cell-cycle arrest, DNA damage, and SASP. These senescent cells exhibited heightened susceptibility to T-cell-mediated cytotoxicity, associated with increased immunoproteasome activity and upregulated HLA expression. Co-culture assays revealed that senescent tumor cells promoted interferon- γ production and enhanced cytotoxicity in CD8⁺ T cells as well as PD-1 expression. In both in vitro and in vivo models, combining Aurora B inhibition with anti-PD-1 therapy markedly suppressed tumor growth.

Conclusions:

This study identifies Aurora B as a therapeutically actionable vulnerability in HCC, whose inhibition induces senescence and enhances tumor immunogenicity. The combination of Aurora B inhibitor with anti-PD-1 therapy yields synergistic anti-tumor effects, supporting its potential as a promising strategy to improve immunotherapy outcomes in HCC.

Keyword(s) : Hepatocellular carcinoma, Senescence, Immunotherapy.

2100 - From Organ to Surface Perforating Disorders in Chronic Liver Disease
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Background/Hypothesis:

Our article sheds light on vesicular, intensely itchy, scarring skin lesions associated with systemic diseases: Acquired Perforating Dermatositis. Perforating disorders are more commonly seen in chronic renal failure, diabetes, and liver disease to a lesser extent.

We present three real-life cases of end-stage liver disease—due to autoimmune hepatitis, viral hepatitis, and alcoholic liver disease, respectively—manifesting as unusual clusters of itchy skin lesions. Two of these were documented by skin biopsies, while the third case responded to treatment of the underlying liver condition.

This report underscores the need to consider chronic liver disease as a potential cause of perforating disorders, expanding the spectrum beyond traditionally associated conditions mentioned above.

Perforating disorders have a documented prevalence of 10-15% in patients with chronic kidney disease and diabetes mellitus. However, their prevalence in chronic liver disease remains underreported. Emerging case reports suggest a noteworthy association, necessitating further research and clinical awareness.

Materials/Methods/Results:

A 46-year-old female patient with autoimmune liver disease developed a widespread vesicular itchy rash, predominantly affecting her extremities. Despite detailed history and extensive laboratory testing, the results were inconclusive. A dermatological referral and subsequent biopsy confirmed a perforating disorder associated with chronic liver disease, specifically Kyrle disease.

A 51-year-old male patient with decompensated liver cirrhosis due to viral hepatitis presented with similar skin lesions on his back and extremities. Further testing and biopsy confirmed the same histopathological findings of Kyrle's disease.

A 38-year-old male patient with advanced alcoholic liver disease on top of autoimmune hepatitis presented with similar skin lesions on his face. Treatment of the underlying liver conditions, in addition to quitting

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alcohol, healed his lesions.

Conclusions:

These cases underscore that perforating disorders can occur in liver disease. Early recognition enables more targeted and effective treatment, ultimately improving patient outcomes. A multidisciplinary approach is paramount for optimal management.

Keyword(s) : Severe Itching Papules, End-organ damage, Perforating Dermatitis, Perforating Folliculitis, End-stage liver disease.

2102 - Temporal Trends and Outcomes of AMA Negative Primary Biliary Cholangitis in a Multi Centre South East Asian Cohort

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INTRODUCTION: Primary biliary cholangitis (PBC) is a chronic inflammatory liver disease characterized by anti-mitochondrial antibody (AMA) and cholestasis. Early studies suggest AMA negative PBC patients (AMA-) may

associated with delayed diagnosis and poor outcomes. Clinical outcomes of PBC patients from South-East Asia has not been reported. We aim to determine the prognostic implication of AMA positivity among PBC patients.

METHODS: The SEA-PBC registry comprises PBC patients from 13 institutions from the Asia-Pacific regions. Patients not fulfilling diagnostic criteria of PBC, overlap syndrome, and missing outcome data were excluded. Temporal trends in AMA-negative (AMA-) PBC from 2010-2023 were analyzed using centre-adjusted piecewise mixed-effect logistic regression. Primary outcome was transplant-free survival. Transplant-free survival was analysed using Fine-Gray competing regression, with death as competing event, adjusting for established prognostic factors.

RESULTS: Among 1,128 PBC patients, 610 PBC patients (85% female, median age 62 years, 8.5% were AMA-negative) were included for analysis. Compared with AMA-positive patients, AMA- were younger, had higher ALT, lower median FIB-4 score (1.7 [1.1-2.6] vs 2.1 [1.3-3.2], $p=0.008$) and lower proportion of cirrhosis patients (9.5% vs 22.2%, $p=0.029$), consistent with earlier-stage disease at diagnosis. The proportion of AMA- PBC increased steadily over time after adjustment for centre-level clustering (aOR per year: 1.02, 95%CI: 1.016-1.024). Treatment response and symptom burden were comparable irrespective of AMA status. In adjusted competing-risk analysis, AMA positivity did not independently associate with transplant-free survival (adjusted sHR: 1.15, 95%CI: 0.44-2.98).

CONCLUSION: In this large contemporary South-East Asian PBC cohort, AMA negative PBC is increasingly diagnosed and presents at an earlier disease stage. These findings suggest improve disease recognition over time in South-East Asia

Keyword(s) : PBC, AMA, Hepatology, Epidemiology

2104 - A Longer Way to Go Chronic Hepatitis B Burden and Trends Across the Asian Pacific Region

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Background:

The World Health Organization (WHO) aims to eliminate hepatitis B as a public health threat by 2030. However, the Asian-Pacific region, which carries much of the global HBV burden, is still far from reaching these targets. To speed up progress, it is essential to understand the current disease burden and how it is changing over time.

Materials and Methods: We used data from the newly released Global Burden of Disease (GBD) 2023 study, defining chronic hepatitis B (CHB) as the GBD cause "CHB including cirrhosis." For the total population and for children under 5 years, we extracted prevalence rates and numbers of prevalent cases; for the total population, we additionally obtained mortality rates, numbers of deaths, and disability-adjusted life years (DALYs) rates and counts. We described the CHB burden in 2023, analyzed temporal trends and average annual percent changes (AAPC) from 1990–2023, and applied a Bayesian age–period–cohort model to project the burden from 2024 to 2030.

Results:

In 2023, the Asian-Pacific region accounted for 63% of global chronic hepatitis B cases (178 million), 66% of deaths (259 thousand), and 65% of DALYs (8.4 million), with prevalence, mortality, and DALY rates all exceeding global averages; however, it bore only 23% of childhood infections (1,964 thousand), reflecting a lower pediatric prevalence rate (590 vs. 1,325 per 100,000). East Asia dominated absolute disease burden (99 million cases, 104 thousand deaths), while South Asia had the highest pediatric caseload (714 thousand). From 1990–2023, Western Asia showed the steepest annual decline in adult prevalence (–1.99%), and Central Asia in pediatric prevalence (–7.62%); conversely, Southeast and Central Asia exhibited rising mortality trends (+0.18% and +0.78% per year, respectively). Compared with 2015, the prevalence of CHB among

children under 5 years in the Asian-Pacific region is projected to decline by 27.9% by 2030, remaining well below the WHO's target of 90% reduction. Although age-standardized mortality is decreasing, total deaths are expected to rise by 19.6% over the same period.

Conclusions:

Asian-Pacific still carries >60% of global CHB cases and deaths; under-five prevalence is on course to fall only 27.9% by 2030, well below the WHO's target of 90% reduction. East and South Asia account for four-fifths of regional infections and three-quarters of childhood cases, making them the priority for intensified prevention, diagnosis and treatment to meet elimination goals.

Keyword(s) : Chronic Hepatitis B, Global Burden of Disease, Asian-Pacific Region, WHO's Targets for 2030, Hepatitis Elimination

2105 - Breaking the habit Evaluating the routine use of SBP prophylaxis

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Background and Aim: Spontaneous bacterial peritonitis (SBP) is the most common bacterial infection in patients with decompensated cirrhosis and is associated with high mortality and morbidity. Despite advances in medical care, 1-year mortality after SBP ranges from 31% to 93%. SBP prophylaxis is used either as primary prophylaxis (PP) to prevent the initial episode or secondary prophylaxis (SP) to prevent recurrence. The evidence for this is based on outdated data published more than two decades ago. With the emergence of multidrug-resistant organisms (MDRO), the routine use of SBP prophylaxis needs to be re-evaluated. We aimed to investigate the impact of PP and SP on survival and MDRO development.

Methods:

Data were retrospectively collected from patients admitted with cirrhosis-related ascites across three tertiary centres in Australia between January 2022 and December 2023, with follow-up until death or December 2024.

Results:



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A total of 379 patients were included, with 61% being male, with a median (IQR) age of 62(19) years, MELD-Na score of 21(9) and follow-up of 1.2(1.9) years. Alcohol was the predominant aetiology (59%) for underlying cirrhosis, and 73(19.2%) had MDRO at baseline. On index admission, 13(3.4%) patients were already on SP, 21(5.5%) were on PP, and 51(13.5%) patients were diagnosed with SBP. Post index admission, 32(8.4%) and 48(21%) patients were commenced on PP and SP, respectively. During the follow-up, 52(13.7%) patients developed an episode of SBP.

The mean SBP-free survival was similar between those on PP and those meeting criteria but not commenced on PP [2.9 v 2.7years, Chi-square=3.7, p=0.2], and with or without SP [2.2 v 2.0years, Chi-square=1.1, p=0.3]. Overall survival was similar between those not on SP [median (95%CI):1.7 (1.2-2.1) years] and those on SP [median (95%CI):1.8(1.5-2.2) years], Chi-square=0.5, p=0.5.

Overall, the risk of developing MDRO culture-positive SBP during follow-up was significantly higher for those on either PP or SP [27.5 % v 9.4%, Chi-square=18.1, p<0.001]. The risk was only higher for those who had MDRO-positive status at baseline [64.7% v 5.4%, Chi-square=29.6, p<0.001] but not for those with MDRO-negative status at baseline [17.5% v 10.3%, Chi-square=2.5, p=0.1].

Conclusions:

This study shows that in a real-world setting, the use of PP and SP for SBP does not have a survival benefit. In fact, those who have MDRO at baseline have a higher risk of developing culture-positive SBP with MDRO if started on prophylaxis. Future randomised controlled trials are required to justify PP and SP for SBP.

Keyword(s) : Spontaneous Bacterial Peritonitis; Prophylaxis; Multidrug Resistant Organisms; Survival

2107 - COVID 19 pandemic had minimal impact on the diagnostic and treatment uptakes for HCV – a territory wide study

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Background / Hypothesis:
Health inequalities among individuals living with chronic hepatitis C virus (HCV) infection are pressing issues that have not received the attention they warrant. The

COVID-19 pandemic exaggerated the health inequalities. This study evaluated the secular trends in diagnostic and treatment uptake for HCV over the last two decades, contrasting the pre-COVID period with the post-COVID era.

Materials and Methods:
All individuals living with chronic HCV infection who had at least one positive result for HCV antibody (anti-HCV), HCV RNA or HCV genotype between January 2000 and December 2024 were identified. Antiviral treatment for HCV was defined with the prescription of (pegylated-) interferon with or without ribavirin or direct-acting antivirals (DAAs). Annual incidence of HCV diagnosis was summarised overall by age and gender.

Results:

We first identified 520,647 consecutive individuals with HCV markers checked, and 25,207 patients living with chronic HCV infection diagnosed in 2000-2024. Their mean age was 54.3 ± 15.9 years and 17,599 (69.97 %) were male respectively. In men, the diagnostic rate ranged from 0.34% [0.32%, 0.36%] in 2017 to 1.21% [1.12%, 1.30%] in 2024; in women, from 0.15% [0.14%, 0.17%] in 2017 to 0.53% [0.47%, 0.59%] in 2024. By age group, the highest post-COVID diagnostic rate was observed in those aged 40–49 years, increasing from 0.34% [0.29%, 0.38%] in 2017 to 1.34% [1.14%, 1.54%] in 2024. Rates also rose in 50–59 years (0.24% [0.21%, 0.27%] to 1.25% [1.10%, 1.40%]), 30–39 years (0.25% [0.21%, 0.29%] to 0.72% [0.56%, 0.88%]), and ≥70 years (0.19% [0.17%, 0.22%] to 0.57% [0.50%, 0.65%]). The lowest rates were seen in the 20–29 age group, increasing from 0.12% [0.08%, 0.16%] to 0.34% [0.17%, 0.51%].

Conclusions:

The COVID-19 pandemic had minimal impact on the diagnosis of HCV in Hong Kong. It was likely due to the endeavours mentioned in the Hong Kong Hepatitis Action Plan 2020-2024, which may have effectively mitigated the pandemic's potential disruption to hepatitis elimination efforts.

Founding: This work was supported by the Investigator Sponsored Research of Gilead Sciences (Reference: IN-HK-987-7181).

Keyword(s) : HCV, COVID-19, DAA

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2110 - THE RISING THREAT OF MASLD IN YOUNG ADULTS A COMPREHENSIVE METABOLIC AND HEPATIC ASSESSMENT FROM SOUTH ASIA

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Background / **Hypothesis:**
Metabolic dysfunction-associated fatty liver disease (MAFLD) is rapidly increasing in South Asia, with an alarming rise among young adults. This study aimed to determine the prevalence, metabolic determinants, and early hepatic impact of MAFLD in a young cohort presenting to a tertiary care centre

Materials and **Methods:**
This prospective observational study was conducted over a two-year period from June 2023 to June 2025, enrolling 300 consecutive patients presenting with nonspecific gastrointestinal symptoms such as vague abdominal pain, dyspepsia, lethargy, and low energy. Demographic data, BMI, dietary patterns, physical activity, substance use, and family history were recorded. Liver biochemistry, fasting glucose, and lipid profile were assessed in all patients, alongside ultrasonographic evaluation of hepatic steatosis. Gender-based analysis was performed to compare disease burden and associated metabolic risk patterns

Results:

Among the 300 enrolled patients (180 females, 120 males), the mean age was 30.2 ± 2.5 years, reflecting a predominantly young population. Ultrasound-confirmed hepatic steatosis was highly prevalent, observed in 82% of females and 64% of males. The majority reported consumption of energy-dense, high-fat meals, frequent intake of processed and refined carbohydrate-rich foods, and habitual late-night eating, accompanied by a striking absence of structured physical activity. Elevated SGPT levels (55–100 IU/L) were recorded in 104 females and 68 males, with significant hypertriglyceridemia (180–300 mg/dL) noted across the cohort. Fasting hyperglycemia was present in 34 females and 22 males. Overweight and obesity were common, affecting 48 males and 54 females. Cardiometabolic risk was further accentuated by a strong family history of cardiovascular disease, limited awareness of personal cardiac health, and high rates of smoking among males, while moderate alcohol intake

was reported by 25 males.

Conclusions:

MASLD is highly prevalent in this young South Asian cohort, with a disproportionately higher burden among females. The combination of unhealthy dietary behaviours, physical inactivity, metabolic abnormalities, and cardiometabolic risk factors highlights an urgent need for early lifestyle intervention, targeted metabolic screening, and structured long-term follow-up to prevent progression to fibrosis, cirrhosis, and cardiovascular events.

Keyword(s) : masld, young population. risk factors

2113 - Unveiling occult squamous commitment as a clinical fate determinant in cholangiocarcinoma

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Squamous transdifferentiation in intrahepatic cholangiocarcinoma (iCCA) has long been regarded as a rare event (~1-2%), yet its emergence heralds lethal disease progression. Here, we identify occult squamous commitment, a latent state characterized by activation of a squamous transcriptional program without histological transformation, quantifiable by a squamous transdifferentiation potential (STP) index. Approximately 20-30% of patients are classified as STP-high and experience unfavorable outcomes comparable to adenosquamous iCCA. FOSL1 functions as the key squamous commitment initiator, directing a stepwise squamous trajectory through dynamic chromatin-state remodeling. It cooperates with p63 in an enhancer-dependent manner to dysregulate PTGS2/COX-2 expression. The resulting intratumoral PGE2 accumulation undermines T-cell effector function and promotes neutrophil-mediated FOXP3-negative type 1 regulatory T-cell (Tr1) polarization. Clinically, STP-high tumors are predisposed to resistance to combination regimens involving immunotherapy. Celecoxib emerges as a dual-function agent that suppresses squamous plasticity while simultaneously reinvigorating T-cell activity. Our findings delineate a continuum of squamous states in iCCA and provide a framework for treatment tailored to dynamic lineage transition.

Keyword(s) :



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2117 - Clinical Characteristics and Short Term Outcomes of Patients with Cirrhosis and Subclinical Decompensation

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Background / **Hypothesis:**
According to the 2025 Chinese guidelines for the management of cirrhosis, subclinical decompensation is defined as the presence of objective evidence—such as minimal hepatic encephalopathy (MHE), imaging-confirmed small-volume ascites (<2 cm), or portal hypertensive enteropathy (positive fecal occult blood test)—without related clinical symptoms. This study aimed to describe the baseline characteristics of such patients and preliminarily analyze their short-term clinical outcomes.

Materials and **Methods:**
This was a single-center, ambidirectional cohort study. Consecutive patients with cirrhosis who met the diagnostic criteria for subclinical decompensation were enrolled between January and December 2023. Baseline data, including demographics, etiology, Child-Pugh score, and MELD score, were collected. Patients were subsequently followed up regularly. The primary endpoints were progression to clinical decompensation, re-compensation, occurrence of hepatocellular carcinoma (HCC), and death.

Results:

A total of 81 patients were included. Baseline characteristics: mean age was 55.9 ± 11.1 years, and 60.5% were male. The primary etiology was viral hepatitis (60.5%). The majority of patients (81.5%) had well-preserved liver function (Child-Pugh class A), with a mean MELD score of 8.3 ± 3.3 . The most common manifestation of subclinical decompensation was asymptomatic small-volume ascites (61.7%), followed by MHE (24.7%) and a positive fecal occult blood test (13.6%). Outcomes: Among the 52 patients who completed the 96-week follow-up, re-compensation was observed in 22 cases (42.3%), progression to HCC in 5 cases (9.6%), and death in 2 cases (3.8%).

Conclusions:

Patients meeting the criteria for subclinical decompensation are characterized by well-preserved liver function but with established complications of portal hypertension, with asymptomatic ascites being the most frequent presentation. During short-term follow-up, while some patients achieved re-compensation, a considerable proportion still progressed to HCC or death. This population represents

a critical window for intervention, and the clear clinical profile and outcome data provide an important basis for developing individualized management and surveillance strategies

Keyword(s) : Subclinical Decompensation;minimal hepatic encephalopathy;Cirrhosis

2121 - Eliminating Viral Hepatitis in Pakistan A Comprehensive Strategic Framework

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Background / **Hypothesis:**

Pakistan is grappling with a significant public health challenge, bearing a high burden of viral hepatitis B and C. Approximately 3.8 million people are living with hepatitis B virus (HBV) and 9.8 million with hepatitis C virus (HCV). To address this pressing issue, the National Hepatitis Strategic Framework (NHSF) 2024-2030 has been developed, aligning with the World Health Organization's (WHO) Global Health Sector Strategy. The NHSF aims to eliminate viral hepatitis as a major public health threat by 2030. To achieve this goal, the framework outlines a comprehensive approach, focusing on:

1. Strengthening leadership and governance
2. Improving prevention through enhanced vaccination coverage and safe blood transfusions
3. Increasing access to testing and treatment services
4. Building laboratory capacity for diagnosis and surveillance
5. Enhancing surveillance and strategic information systems

Key strategies include scaling up harm reduction services for high-risk populations, integrating hepatitis testing and treatment with primary care services, and promoting community awareness and engagement

Materials and **Methods:**

The NHSF was developed through a systematic and inclusive approach, involving assessment of the current hepatitis situation, stakeholder engagement (healthcare

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professionals, policymakers, community representatives, patient advocacy groups), analysis of global best practices and WHO guidelines, identification of gaps in hepatitis services, establishment of clear objectives and targets and development of a comprehensive framework with implementation plans and monitoring system. This approach ensured the NHSF is evidence-based, inclusive, and tailored to Pakistan's needs, providing a roadmap for eliminating viral hepatitis.

Results:

The NHSF targeted tangible targets, including:

- Reducing new chronic HCV infections to ≤ 5 per 100,000
- Reducing chronic HBV prevalence among 1-5 year-olds to $\leq 0.1\%$
- Reducing HBV and HCV-related mortality to ≤ 6 per 100,000 by 2030

Conclusions:

Pakistan's NHSF provides a roadmap for eliminating viral hepatitis as a major public health threat by 2030. Effective implementation, monitoring, and partnerships will be crucial to achieving the targets and reducing the burden of viral hepatitis in Pakistan. By working together, Pakistan can make significant progress towards eliminating viral hepatitis and improving the health and well-being of its population. The NHSF serves as a guiding document for stakeholders, policymakers, and healthcare professionals to collaborate and strive towards a hepatitis-free Pakistan.

Keyword(s) : Hepatitis Strategy, Elimination, Pakistan, Global Hepatitis Strategy, Hepatitis B, Hepatitis C, Hepatitis D, Hepatitis A, Hepatitis E, Monitoring and Evaluation, Data Collection, Reporting, Monitoring and Evaluation

2124 - GENDER DISPARITIES IN HEPATOCELLULAR CARCINOMA A COMPARATIVE ANALYSIS

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Background / **Hypothesis:**

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy and a major cause of cancer-related mortality worldwide. It shows a clear gender disparity, with males more frequently affected. In

Pakistan, HCC incidence is increasing due to chronic hepatitis B (HBV), hepatitis C (HCV), hepatitis D (HDV), and metabolic dysfunction-associated steatotic liver disease (MASLD; formerly NAFLD/NASH). Limited local data exist regarding gender-based differences in risk factors and complications. This study aims to assess gender distribution, associated risk factors, and complications among HCC patients at Jinnah Postgraduate Medical Centre (JPMC), Karachi.

Materials and Methods:

This cross-sectional observational study was conducted in the Gastroenterology Ward of Jinnah Postgraduate Medical Centre (JPMC), Karachi, from January 2023 to December 2025. All male and female patients aged 18 years and above with a confirmed diagnosis of hepatocellular carcinoma (HCC) were included. Data were collected using a structured proforma, documenting demographics, risk factors (HBV, HCV, MASLD, alcohol use, and co-infections), and complications (upper gastrointestinal bleeding, portal systemic encephalopathy, ascites, and hepatoma rupture). Data were analyzed using SPSS version 26.0. Descriptive statistics were used to summarize variables, and the Chi-square test was applied to assess gender differences, with a p-value ≤ 0.05 considered statistically significant.

Results:

were more prevalent among females ($p < 0.001$). Among complications, upper gastrointestinal bleeding (UGIB) occurred in 31.4% of patients, significantly more in males (37.4%) than females (18.2%) ($p < 0.001$). Portal systemic encephalopathy (PSE) was also more frequent in males (28.1% vs. 18.8%, $p = 0.002$). Ascites (55.8%) and hepatoma rupture (0.2%) showed no significant gender difference ($p > 0.05$).

Conclusions:

: Hepatocellular carcinoma (HCC) showed a marked male predominance, with nearly two-thirds of patients being male. HCV infection emerged as the leading etiological factor, particularly among males, whereas HBV and MASLD were more prevalent among females. Complications such as upper gastrointestinal bleeding and portal systemic encephalopathy were significantly more frequent in males, while ascites and hepatoma rupture showed no gender difference. These findings highlight the importance of gender-specific preventive strategies, emphasizing viral hepatitis control in males and metabolic liver disease screening in females to

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reduce the burden of HCC in Pakistan.

Keyword(s) : Hepatocellular Carcinoma, Gender Disparities, HBV, HCV, HDV, MASLD, NAFLD, Liver Cancer.

2125 - Efficacy and Safety of Magnesium Isoglycyrrhizinate in Checkpoint Inhibitor Induced Liver Injury in Patients with Advanced Solid Tumors An Exploratory Study

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Background / Hypothesis: Immune checkpoint inhibitors (ICIs) have shifted the paradigm of cancer treatment; however, immune-mediated liver injury occurs in >30% of patients receiving ICI therapy and may require cessation of ICI therapy and initiation of immunosuppression. Magnesium isoglycyrrhizinate, a magnesium salt mainly consisting of 18 α -glycyrrhizic acid stereoisomers, possesses hepatoprotective activities and has been shown to be more effective in lessening ICI-induced liver injury when combined with steroid therapy than steroid therapy alone. We undertook this exploratory study to investigate the efficacy and safety of magnesium isoglycyrrhizinate for ICI-induced liver injury in patients with advanced solid tumors on ICI combination therapy. **Materials and Methods:** Patients aged between 18 and 75 years who were to receive first-line ICI therapy in combination with chemotherapy or other antitumor therapy and had normal liver function at screening (alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase and total bilirubin $\leq 1.0 \times$ upper limit of normal) received magnesium isoglycyrrhizinate 200 mg given intravenously once daily for ≥ 3 days for 4 to 6 cycles, given within 2 days of each cycle of ICI combination therapy. The primary endpoint was the incidence of liver injury. The secondary endpoints included the severity of liver injury and safety. **Results:** Between March 17, 2024 and June 28, 2025, 50 patients were screened for eligibility, 12 with hepatocellular carcinoma or liver metastasis and 6 with abnormal liver function at screening were excluded. Thirty-two patients (nonsmall cell lung cancer, 20 patients; gastric cancer, 9; colorectal cancer, 2; esophageal cancer, 1) received magnesium isoglycyrrhizinate (median duration of treatment, 9.9 weeks [IQR 4.1-16.0], median number of cycles, 4 [IQR 2-6]; median cumulative dose, 900 mg [range, 200-3800]). The incidence of ICI-induced liver injury was

15.6% (95% CI 5.3% to 32.8%). Grade 1 ICI-induced liver injury occurred in 5 patients; no grade ≥ 2 ICI-induced liver injury was reported. The median time to onset of ICI-induced liver injury was 6.1 weeks (range, 3.3-10.0) and the median cycle 3 (range, 2-4). Treatment-emergent adverse events (AEs) occurred in 71.9% (23/32) patients, but grade ≥ 3 treatment-emergent AEs were infrequent (6.3%[2/32]). No treatment-related AE was reported. Serious AEs occurred in 1 patient. **Conclusions:** Magnesium isoglycyrrhizinate is safe and effective in reducing the incidence and severity of ICI-induced liver injury in advanced solid tumor patients. The findings support further investigation of magnesium isoglycyrrhizinate as a potential preventative therapy for ICI-induced liver injury.

Keyword(s) : immune checkpoint inhibitor, drug-induced liver injury, magnesium isoglycyrrhizinate, efficacy, safety

2130 - Predictors and impact of complete pathological response to pre transplant immune checkpoint inhibitors in hepatocellular carcinoma Findings from the Global LIFT HCC consortium

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Background / Hypothesis: Achieving complete pathological response (cPR) has been associated with lower hepatocellular carcinoma (HCC) recurrence after liver transplantation (LT). However, Predictors of cPR to immune checkpoint inhibitors (ICIs) remain unclear. Prior studies suggest that strong treatment responses indicate increased immune activation and a higher risk of graft rejection. We aimed to identify factors linked to cPR and assess its effect on overall survival (OS), HCC recurrence, and graft rejection.

Materials and Methods: In the largest multinational retrospective study (Leveraging Immunotherapy for Liver Transplantation in HCC; LIFT-HCC), we included HCC patients who received ICIs before LT and had post-LT follow-up. Logistic regression analyses identified factors linked to achieving cPR. OS, Cumulative incidence of recurrence (CIR), and graft rejection were estimated by cPR status.

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Results: A total of 408 patients (14 countries, 60 centers) were included (mean age 54 ± 10 years, 91% male). At ICI start, 75% were Child-Pugh A, the median (IQR) AFP was 53 (6, 690), and 23% were within Milan criteria. At LT, the median (IQR) AFP was 9 (3, 129), and 43% were within Milan criteria. Pre-LT imaging showed 17.9% had no viable tumor. On explant pathology, 17% achieved cPR. Receiver operating characteristic analysis showed that absence of viable tumor on imaging had 71.7% sensitivity and 92.2% specificity for predicting cPR, with an area under the curve of 0.82 (95%CI 0.76-0.88). Median ICI cycle number (8 vs 3, $p < 0.001$), median ICI washout period (98 vs 65 days, $p = 0.003$), and PD-L1 use frequency (50% vs 8%, $P < 0.001$) were significantly higher among cases with cPR versus those without cPR. In the multivariable model (Table), >4 ICI cycles (OR 2.79, 95%CI 1.1-7.1), a greater decline in delta log-AFP (OR 0.76, 95%CI 0.62-0.93), and no viable tumor on imaging (OR 19.61, 95%CI 7.61-50.55) were independently associated with cPR. During a median post-LT follow-up of 21.2 months (95%CI, 18.9-25.4), 21% died, 26% had HCC recurrence, and 19% experienced allograft rejection. Cases without cPR had significantly lower OS (HR 3.04, 95%CI 1.31-7.06, $P = 0.01$) and higher CIR (SHR 6.32, 95%CI 2.54-15.7, $P < 0.001$), but not graft rejection (SHR 0.86, 95%CI 0.45-1.66, $P = 0.66$) (Figure).

Conclusions: cPR was observed in 1 of 6 patients receiving pre-LT ICIs. Each unit decrease in delta log-AFP and receiving >4 ICI cycles remained independently associated with achieving cPR after adjusting for pre-transplant imaging. cPR was associated with higher OS and lower CIR, but did not affect the risk of rejection.

Keyword(s) : Hepatocellular Carcinoma; Liver Transplantation; Immune Checkpoint Inhibitors; Complete Pathological Response; Recurrence; Rejection; Survival

2132 - Leveraging Decentralization Integration and Task Shifting to achieve Micro elimination of Hepatitis C among prisoners in Northern India

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Background / Hypothesis: Hepatitis C virus (HCV) infection is highly prevalent among incarcerated individuals, yet diagnosis and treatment remain challenging in correctional settings. A decentralized,

integrated, and task-shifting approach can overcome these barriers. This project utilized a novel prison-based model for HCV micro-elimination, leveraging the existing public health infrastructure to streamline care and accelerate treatment initiation.

Materials and Methods: A micro-elimination project was conducted across four major jails in Uttarakhand state of India, from Aug2024- Aug2025, offering HCV self-testing (HCVST). Reactive HCVST results led to RNA testing and liver fibrosis assessment via onsite transient elastography (TE) using portable TE machine. Drones aided sample collection and direct-acting antiviral (DAA) delivery and teleconsultation supported constant handholding. Confirmed active infections were treated with DAA therapy, following NVHCP guidelines. A usability study of HCVST kits was also conducted among 100 inmates (Figure 1).

Results: Of 4050 inmates (96.7% male; mean age: 35 ± 13 years), 40.9% were from Haridwar, 25.1% from Dehradun, 20.4% from Haldwani and 13.6% from Roorkee. Overall, 138 (3.41%) tested positive for HCVST, with 67% (92/132) positives from Haridwar, 14% (20/132) from Dehradun and 18% (26/132) from Roorkee. Key reported behaviors included sharing toothbrushes/shaving tools (38.1%) and unsafe sex (14.6%). HCV positivity was significantly higher among those sharing needles [Risk in exposed = 56.9% (33/58), OR: 5.1; 95%CI: 2.9-9.0], using unprescribed injectable drugs [Risk = 52.1% (37/71), OR: 4.3; 95%CI: 2.3-7.5], unsafe sex [Risk = 15.4% (70/455), OR: 11.9; 95% CI: 7.9-17.8], tattooing (OR: 6.2; 95%CI: 2.3-16.6), and sharing personal items (OR: 3.7; 95% CI: 2.4-5.5). Logistic regression identified unsafe sex (OR: 8.6; 95%CI: 4.9-15.1), needle sharing (OR: 7.2; 95%CI: 2.1-25.0), and injectable drug use (OR: 3.8; 95%CI: 1.2-11.9) as independent predictors ($p < 0.05$). Out of 138 Ant-HCV positive, 117/138 (85%) were tested for HCVRNA, 106/117 (90%) were HCVRNA positive (median HCVRNA-30,245 IU/mL (range 262-625,463)). Significant liver fibrosis (TE > 12 kPa) was observed in 15 cases. 74/106 (70%) individuals were put on DAA treatment, only 21/74 (28%) completed treatment, and the SVR-12 rate was 95% (Figure 2). The only reason for dropouts at different levels of the care cascade was "release from the prison". The overall usability of HCVST kits was 80%, with high acceptability, but some challenges were encountered during sample collection that required assistance. Conclusion: This project demonstrated the feasibility of utilizing HCVST to screen and link inmates to hepatitis C care within a correctional setting, which has high

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burden.

Keyword(s) : HCV Self-testing, Decentralization, Integration, Task-sharing

2134 - Iron Metabolism Defects in Wilson Disease Suspects A Real World Two Phase Molecular Epidemiology Study

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Background / **Hypothesis:**
Clinically, genetic defects in the ATP7B gene alone are often insufficient for diagnosing Wilson disease (WD) or explaining the phenotypic heterogeneity observed in suspected cases. Additionally, we observed that a subset of these individuals exhibited iron metabolism anomalies. Therefore, we hypothesized that genetic defects in iron metabolism may contribute to the unexplained heritability in WD suspects.

Materials and **Methods:**
WD suspects were identified based on the Leipzig criteria. In the first phase, 498 suspects underwent targeted sequencing of the ATP7B gene, and major iron markers were assessed. Serum ferritin levels and iron saturation (FeSat) percentages were compared with those of healthy controls, patients with non-infectious non-liver diseases, and patients with liver diseases, ensuring age and gender matching across groups. In the second phase, a new cohort of 299 suspects underwent exome sequencing, with a focus on genes involved in iron metabolism and potential mediators of copper toxicity.

Results:

In the initial phase, 32.89% of WD suspects had elevated serum ferritin levels, and 41.52% had higher-than-normal FeSat percentages, both significantly higher than in healthy controls ($p < 0.001$ and $p < 0.001$, respectively). Similar trends were observed when compared to the non-infectious non-liver disease group

(ferritin $p < 0.05$, FeSat $p < 0.001$). However, when compared to patients with liver disorders, the significance for ferritin disappeared, while the significance for FeSat remained ($p < 0.05$). These findings were more pronounced in genetically confirmed subgroups of WD suspects. In the second phase, 50% of the suspects with ATP7B genetic defects exhibited rare iron metabolism defects, while this proportion rose to 65% when considering all suspects, regardless of ATP7B status. The most frequent defects involved TFR2 and SLC11A2.

Conclusions:

Our study confirmed the disruption of iron metabolism in WD suspects. Rare genetic defects associated with iron metabolism co-occurred with ATP7B defects, underscoring the interplay between the regulatory pathways of both transition metals.

Keyword(s) : Wilson disease, ATP7B, copper, iron, missing heritability

2137 - Protective and Therapeutic Roles of Vitamin D in Reducing Chlorpyrifos Induced Liver Fibrosis Through Modulation of Fibrotic Pathways

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Background / **Hypothesis:**

Organophosphate pesticides, particularly chlorpyrifos (CPF), induce liver fibrosis through oxidative stress and hepatic stellate cell activation, leading to elevated expression of α -smooth muscle actin (α -SMA), transforming growth factor-beta 1 (TGF- β 1), and Collagen-I. Given vitamin D's established anti-inflammatory and antifibrotic properties, this study investigated its protective and therapeutic effects on CPF induced hepatic fibrosis in male albino Wistar rats. **Objective:**

To determine the prophylactic and therapeutic effects of vitamin D on chlorpyrifos induced liver fibrosis

Materials and **Methods:**
Thirty five male albino Wistar rats were allocated into four groups: control (n=5), CPF-treated (n=10), and two vitamin D-treated groups (D1, n=10; D2, n=10). Hepatic fibrosis was induced via daily intraperitoneal CPF injection (1 ml/kg) for eight weeks. In group D1, vitamin D (0.5 μ g/100g, twice weekly) was administered after fibrosis establishment, while in group D2, vitamin D was given simultaneously with CPF. Assessments included serum liver function tests (ALT, GGT, bilirubin),

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histopathological examination (H&E and Masson's Trichrome staining), and quantitative real-time PCR analysis of fibrogenic markers (α -SMA, TGF- β 1, Collagen-I).

Results:

CPF exposure significantly elevated liver enzymes and bilirubin (ALP: 168.8 ± 10.45 IU/L; GGT: 16.1 ± 1.72 IU/L; bilirubin: 0.46 ± 0.042 mg/dL). Vitamin D treatment dose dependently reduced these parameters, with therapeutic administration (D1) showing moderate improvement (ALT: 155.6 ± 8.11 IU/L; bilirubin: 0.35 ± 0.02 mg/Dl; GGT: 14.4 ± 1.95 IU/L) and prophylactic administration (D2) demonstrating superior efficacy (ALT: 102 ± 7.85 IU/L; bilirubin: 0.2 ± 0.04 mg/Dl; GGT: 4.9 ± 1.66 IU/L). Histopathological analysis revealed that CPF induced severe stage-3 fibrosis, which improved to stage-1 in D1 treatment group and completely resolved to stage-0 in D2 group. Molecular analysis demonstrated marked downregulation of fibrogenic genes in D2 compared to CPF: α -SMA (2.55-fold vs. 0.97-fold), Collagen-I (3.82-fold vs. 1.02-fold), and TGF- β 1 (3.64-fold vs. 1.09-fold), indicating potent antifibrotic activity.

Conclusions:

Vitamin D significantly ameliorates CPF induced hepatic fibrosis across biochemical, histological, and molecular parameters. Prophylactic administration (simultaneous with CPF) demonstrates superior protective effects compared to therapeutic intervention, achieving complete fibrosis reversal and normalization of fibrogenic gene expression. These findings suggest vitamin D as a promising preventive and therapeutic agent against CPF induced liver injury.

Keyword(s) : Liver fibrosis, chlorpyrifos, vitamin D, α -SMA, TGF- β 1, collagen, hepatic stellate cells

2143 - A model screen test and treat hepatitis C elimination project among under served communities in Islamabad the federal capital of Pakistan

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Background

Pakistan faces a significant hepatitis C virus (HCV) burden with limited access to care and treatment. To address this, a same-day testing and treatment initiation model program was launched in Islamabad's marginalized communities (slums) on March 2, 2019.

Materials and Methods

The program, implemented in 17 slums with a population of 50,000, offered free HCV screening, testing, and treatment. Trained community health workers (CHWs) conducted door-to-door screening, testing individuals aged ≥ 12 years using a rapid HCV antibody test. Those testing positive were referred to a nearby clinic for diagnosis of active HCV infection (RNA) by GeneXpert.

Results

A total of 24,216 participants were screened, with 514 (2.12%) testing positive for anti-HCV. Of these, 440 (85.6%) underwent HCV RNA testing, and 323 (73.4%) had detectable RNA. Three hundred eighteen individuals (98.5%) initiated treatment, and 284 (89.3%) completed it. Treatment involved a 12-week sofosbuvir and daclatasvir regimen for non-cirrhotic patients, while those with cirrhosis (APRI ≥ 1.5) were referred to specialists. Patients received counseling, hepatitis B vaccination, and follow-up care. HCV RNA testing at 12 weeks post-treatment showed 96.4% (241/250) achieved viral clearance (cure).

Conclusions

Same day hepatitis C testing and treatment initiation led to minimal patient loss as most patients could be quickly linked to care. CHWs were very effective in reaching "hard-to-reach" populations with limited access to health services. Their involvement led to high rates of linkage to care and adherence to treatment for hepatitis C.

Keyword(s) : Micro elimination, Hepatitis C, Linkage to Care, Community Outreach services, Community Mobilization, Cascade of Care

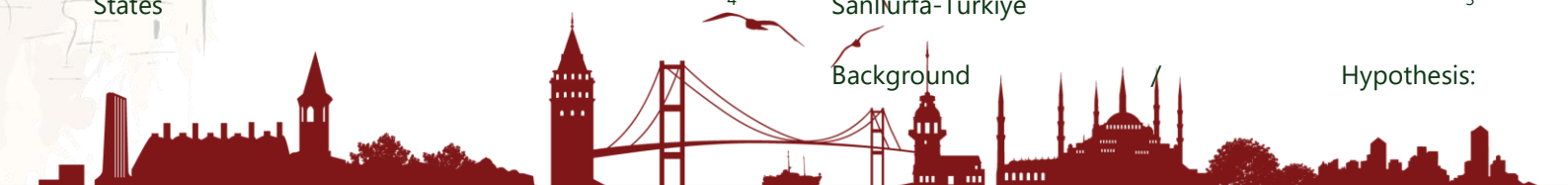
2145 - Gestational cholestasis single center experience

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Background

Hypothesis:



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The incidence of gestational cholestasis (GC) is one in a thousand. The aim of this study is to evaluate the incidence of GC in the Şanlıurfa region and the characteristics of patients diagnosed with GC.

Materials and **Methods:** Births performed at Şanlıurfa Training Hospital in the last year were retrospectively reviewed, and patients diagnosed with GC were included in the evaluation.

Results: A total of 52,717 births were performed between December 2024 and December 2025, and 69 patients were diagnosed with GC. The incidence of GC for this cohort was 0.0013. The mean age of patients with GC was 27.4 ± 5.6 years (range 19-43). All but one patient (97%) had a cesarean section. Thirty-six patients (50.8%) were diagnosed with GC during their first pregnancy, 13 (19%) during their second, 12 (17.5%) during their third, 2 (3.2%) during their fourth, 4 (6.3%) during their fifth, and one patient each (1.6%) during their sixth and seventh births. The mean bile acid level was 28.17 ± 20.59 (2.26-80.19) mg/dl. All patients complained of itching. Three patients (4.8%) had gestational diabetes mellitus, and one (1.6%) had gestational hypertension. No maternal complications or deaths were detected. Fetal complications were detected in a total of 7 babies (10%), including macrosomia in 2 babies, cleft lip in 1 baby, intrauterine growth restriction in 1 baby, and ventricular septal defect (VSD) in 1 baby. Abdominal ultrasonography results were obtained for 44 patients, and cholelithiasis was detected in 4 of them (10%). Of the 59 patients for whom treatment information was obtained, 45 (75%) received UDKA treatment.

Conclusions:

The incidence of gestational cholestasis in the Şanlıurfa region is thirteen per ten thousand. In these patients, the frequency of congenital defects and maternal cholelithiasis is increased, reaching approximately ten percent.

Keyword(s) : gestational cholestasis, incidence, delivery, cesarean section

2148 - B Cell Depletion Therapy Shows Efficacy in Steroid Refractory Autoimmune Hepatitis

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Background

Hypothesis:

Autoimmune hepatitis (AIH) is typically managed with corticosteroids and azathioprine, but a subset of patients are refractory to standard immunosuppression or experience severe side effects, risking progressive liver damage and cirrhosis. B lymphocytes play a critical role in AIH pathogenesis through autoantibody production and antigen presentation. We hypothesized that depleting B cells with rituximab (an anti-CD20 monoclonal antibody) could induce disease remission in steroid-refractory AIH patients by targeting an underlying driver of autoimmunity.

Materials and **Methods:** We conducted an open-label pilot study including 12 adults with biopsy-proven AIH who remained biochemically and histologically active despite conventional therapy. Patients received two rituximab infusions (1 g each, two weeks apart) and were monitored over 12 months. Liver function tests (ALT, AST, bilirubin) and immunoglobulin G (IgG) levels were measured at baseline and regular intervals. Autoantibody titers (ANA, SMA) were tracked, and liver biopsies were obtained at baseline and 12 months post-treatment to assess histological inflammation (HAI score). Clinical outcomes, steroid-sparing effects, and adverse events were also recorded.

Results:

B-cell depletion therapy was associated with significant clinical and biochemical improvements. By 6 months post-rituximab, 9 of 12 patients (75%) achieved complete biochemical remission (normalization of ALT/AST and IgG). The remaining 3 patients showed partial improvement in liver enzymes. Follow-up biopsies at 1 year in 8 patients demonstrated reduced interface hepatitis and lymphoplasmacytic infiltrates, with the HAI inflammation score improving by an average of 4 points compared to baseline. Prednisone requirements were substantially reduced in responders (median dose decreased by 60% at 6 months). Rituximab was well tolerated; no severe infections or infusion reactions occurred, aside from transient mild infusion-related symptoms in two patients.

Conclusions:

B-cell depletion with rituximab appears to be a promising therapeutic option for AIH patients unresponsive to standard therapy. The majority of treated patients achieved biochemical remission and histological improvement, alongside significant steroid sparing. These results suggest that targeting B cells can effectively modulate the autoimmune process in AIH, warranting larger controlled trials to confirm efficacy

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and long-term safety.

Keyword(s) : autoimmune hepatitis; refractory AIH; B-cell depletion; rituximab; immunotherapy

2150 - Early Skeletal Muscle Loss Predicts Poor Outcomes in Advanced Hepatocellular Carcinoma Treated with Hepatic Arterial Infusion Chemotherapy

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Background / **Hypothesis:**
Sarcopenia-related parameters, including the skeletal muscle index (SMI), influence prognosis and treatment tolerance in hepatocellular carcinoma (HCC). However, evidence is limited regarding hepatic arterial infusion chemotherapy (HAIC), particularly the prognostic relevance of early on-treatment changes in skeletal muscle mass. We aimed to clarify the clinical impact of low SMI and early SMI decline on outcomes in patients with advanced HCC undergoing HAIC.

Materials and **Methods:**
We retrospectively analyzed 70 consecutive patients with advanced HCC treated with HAIC. SMI was quantified using computed tomography and reassessed 3 weeks after HAIC initiation. Patients were stratified into a decreased SMI group (n = 27) and a non-decreased SMI group (n = 43) based on early SMI change. Progression-free survival (PFS) and overall survival (OS) were compared between groups. Associations between early SMI decline and nutritional status, liver function, therapeutic response, and adverse events were examined. Cox proportional hazards models were used to identify independent prognostic factors for OS.

Results:
Early skeletal muscle loss was strongly associated with inferior clinical outcomes. Patients in the decreased SMI group experienced significantly shorter PFS and OS compared with those without early SMI decline. Notably, pre-treatment SMI alone did not demonstrate a robust prognostic effect, whereas an SMI decrease at 3 weeks after HAIC initiation emerged as a powerful predictor of survival. Early SMI decline was accompanied by poorer nutritional status and deteriorated liver function during treatment, suggesting vulnerability to catabolic stress and reduced physiologic reserve.

Therapeutic efficacy was also diminished in the decreased SMI group, with poorer treatment response observed compared with the non-decreased SMI group. In contrast, the overall incidence of HAIC-related adverse events did not significantly differ between groups, implying that early muscle loss reflects systemic deterioration rather than simply treatment toxicity. On multivariable analysis, decreased SMI at 3 weeks remained an independent factor associated with worse OS after adjustment for relevant clinical covariates.

Conclusions:

In advanced HCC treated with HAIC, an early decline in SMI—rather than baseline SMI—identifies a high-risk population with significantly shorter PFS and OS, poorer nutritional and hepatic functional status, and reduced therapeutic response. Serial CT-based monitoring of SMI shortly after HAIC initiation may provide a practical, objective biomarker to evaluate general condition, guide supportive interventions, and improve prognostic stratification in routine clinical practice.

Keyword(s) : Hepatocellular carcinoma, Hepatic Arterial Infusion Chemotherapy, Sarcopenia, skeletal muscle index

2152 - Circulating cell free mitochondrial DNA predicts prognosis and triggers macrophage STING dependent inflammation in acute on chronic liver failure

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Background / **Hypothesis:**
Acute-on-chronic liver failure (ACLF) is characterized by excessive systemic inflammation and high short-term mortality, yet the upstream molecular mechanisms underlying this inflammatory cascade remain poorly defined. This study aimed to determine the prognostic value of circulating cell-free mitochondrial DNA (cf-mtDNA) in ACLF and to elucidate its mechanistic role in driving innate immune activation.

Materials and **Methods:**
Plasma cf-mtDNA levels were quantified by quantitative PCR in patients with ACLF and analyzed in relation to disease severity, inflammatory cytokines, and short-term survival. A murine ACLF model was established by chronic carbon tetrachloride (CCl₄) administration followed by acute lipopolysaccharide/D-galactosamine challenge to characterize the temporal relationship

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between cf-mtDNA release, liver injury, and systemic inflammation. Transcriptomic, histological, and molecular analyses were performed to identify activated cytosolic DNA-sensing pathways. The functional role of the cGAS-STING pathway and therapeutic efficacy were evaluated using myeloid-specific STING deletion (STING^{fl/fl}-Lyzmcre), pharmacologic inhibition of cGAS or STING, and enzymatic degradation of circulating cf-mtDNA with DNase I.

Results:

Circulating cf-mtDNA levels were markedly elevated in patients with ACLF, progressively increased with disease severity, and independently predicted 90-day mortality. In murine ACLF, severe hepatocellular mitochondrial damage was accompanied by increased plasma cf-mtDNA and pronounced hepatic inflammation. Notably, longitudinal analyses demonstrated that cf-mtDNA elevation preceded the rise of key inflammatory cytokines, including IL-6 and TNF- α . Transcriptomic analyses revealed significant enrichment of cytosolic DNA-sensing pathways, with prominent activation of the cGAS-STING axis. Importantly, STING expression and activation were predominantly localized to hepatic macrophages in both murine and human ACLF livers. In vitro, mtDNA released from injured hepatocytes directly activated cGAS-STING signaling in macrophages, inducing downstream TBK1-IRF3 and NF- κ B activation. Genetic deletion of STING in myeloid cells or pharmacologic inhibition of cGAS or STING markedly attenuated macrophage activation, reduced hepatic necroinflammation, and improved survival in ACLF mice. Targeting the upstream trigger, degradation of circulating cf-mtDNA by DNase I similarly alleviated liver injury and improved outcomes.

Conclusions:

Circulating cf-mtDNA functions not only as a prognostic biomarker but also as an upstream pathogenic driver of ACLF by activating macrophage cGAS-STING signaling. Targeting this mtDNA-driven innate immune pathway represents a promising therapeutic strategy for mitigating inflammation-driven liver failure.

Keyword(s) : Acute-on-chronic liver failure; cf-mtDNA; cGAS-STING; macrophage; DNase I.

2161 - Circulating Tumor DNA Analysis Enables Early Detection of Hepatocellular Carcinoma in High Risk Patients

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Background / **Hypothesis:**
Early detection of hepatocellular carcinoma (HCC) in high-risk patients (e.g., those with cirrhosis) is critical for curative intervention, yet current surveillance tools such as ultrasound and alpha-fetoprotein (AFP) have suboptimal sensitivity for early-stage tumors. Circulating tumor DNA (ctDNA) analysis offers a non-invasive "liquid biopsy" that can detect tumor-specific genetic and epigenetic alterations in blood. We hypothesized that incorporating ctDNA-based screening would improve the early detection rate of HCC in at-risk patients compared to standard surveillance methods.

Materials and **Methods:**
We conducted a prospective cohort study involving 200 patients with cirrhosis undergoing routine HCC surveillance over 2 years. Patients provided blood samples every 6 months for ctDNA analysis alongside ultrasound/AFP screening. A targeted next-generation sequencing panel was used to detect low-frequency tumor-associated mutations (e.g., TERT promoter, TP53, CTNNB1) and methylation signatures known to be specific for HCC. Test performance (sensitivity, specificity, positive predictive value) for early-stage HCC (BCLC 0/A) detection was calculated for ctDNA, ultrasound/AFP, and the combination of both. Confirmatory imaging (MRI) and biopsy were used to diagnose HCC in cases of positive screening results.

Results:

During the study period, 30 patients were diagnosed with HCC, of which 22 were early-stage. ctDNA analysis detected 19 of 22 early HCC cases, yielding a sensitivity of 86% (vs 59% for ultrasound/AFP, $p < 0.01$), with a specificity of 92% in those who remained tumor-free. Combining ctDNA with ultrasound increased early detection sensitivity to 95%. Notably, ctDNA became positive a median of 4 months (range 2–9) prior to radiological tumor confirmation in 15 cases, enabling earlier intervention. In 3 instances, ctDNA screening identified very small tumors (< 2 cm) missed by ultrasound. False positives were limited (positive predictive value 75%), and no HCC cases were missed when both modalities were used together.

Conclusions:

Implementing ctDNA-based liquid biopsy significantly improves the detection of early-stage HCC in high-risk patients, outperforming traditional ultrasound/AFP surveillance. The ability of ctDNA screening to flag

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emerging tumors months before imaging detection highlights its potential to facilitate timely curative treatments. These findings support the integration of ctDNA analysis into HCC surveillance protocols, pending further validation in larger trials.

Keyword(s) : hepatocellular carcinoma; circulating tumor DNA; early detection; liquid biopsy; surveillance

2168 - Colorectal Carcinoma in Young patients single Centre study from Karachi Pakistan.

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Introduction:

Colorectal cancer (CRC) is on the rise among young adults. It affects the colon, appendix, and rectum. According to GLOBOCAN 2018, CRC is the 4th most common cancer worldwide and the third leading cause of cancer mortality. The global CRC rate is expected to rise by 60% by 2030, leading to 2.2 million new cases and 1.1 million deaths annually. CRC is heterogeneous, with 70% of cases being sporadic, 10-30% familial, and 5-7% due to hereditary diseases.

Objective:

To evaluate the clinicopathological features of colorectal cancer (CRC) in young adults under the age of 30 Years.

Method:

This was a cross-sectional retrospective study conducted at the Gastroenterology and Oncology Department of JPMC, Karachi, from January 2018 to December 2022. All patients under 30 with CRC were included in the study. Diagnosis was made through colonoscopy and histopathology. Data was analyzed using SPSS version 23. Descriptive statistics were calculated using frequencies and percentages.

Results:

A total of 566 CRC patients were analyzed, of which 117 were young patients. Of the young patients, 67 (57.3%) were male and 50 (42.7%) were female. The majority were Urdu-speaking (47 or 40%), followed by Sindhi (40 or 34%), and Pathan and Punjabi (13 or 11%). Smoking was strongly associated with CRC, with 13 (9%) active smokers, and 8 (6%) using Naswar. The most common area of residence was Orangi Town (8 or 6%), followed

by Korangi (3 or 4%). Colonoscopy revealed polypoid growth in 79 (67%) patients, followed by ulcerated lesions in 23 (19.7%). The most common site of CRC was the rectum (48 or 41%), followed by the sigmoid colon (46 or 39.3%). The least common site was the ascending colon (7 or 2.6%). Moderately differentiated adenocarcinoma was the most common histological type, found in 96 (82.5%) patients, with 17 (10%) having the signet ring type. The most common clinical presentations were abdominal pain (64 or 53%), constipation (45 or 38.5%), and bleeding per rectum (42 or 36%).

Conclusion:

CRC is a leading cause of mortality and is increasingly common among young adults, predominantly affecting the left side of the colon. More studies are needed to develop guidelines for CRC screening and surveillance in our population

Keyword(s) :

2172 - The coexistence of MAFLD exacerbates liver fibrosis in patients with chronic hepatitis B.

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Background / Hypothesis: Despite the increasing recognition of metabolic dysfunction-associated fatty liver disease (MAFLD) and Chronic Hepatitis B patients (CHB), their interplay on liver fibrosis remains insufficiently elucidated. This study aimed to assess liver fibrosis in CHB patients with concurrent MAFLD

Materials and Methods: A cross-sectional study included 148 patients with a confirmed diagnosis of CHB infection, categorized according to the MAFLD standard criteria into CHB with MAFLD (CHB/+MAFLD) and without MAFLD (CHB/-MAFLD). Demographic, metabolic, and biochemical parameters were analyzed. Fibrosis was assessed non-invasively using vibration-controlled transient elastography (VCTE). A multivariate logistic regression identified independent predictors of significant fibrosis.

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Results:

Among the CHB cohort, 34.5% fulfilled MAFLD criteria. Using FIB-4 >1.3 for significant fibrosis, the CHB/+MAFLD group exhibited a higher prevalence of significant steatosis (45.1%) compared to 23.7% in the CHB/-MAFLD group, with higher medians in the CHB/+MAFLD group (1.2 vs 0.8, $p < 0.001$). Consistently, liver stiffness measurement (LSM) assesses the severity of fibrosis. The prevalence of significant fibrosis ($F \geq 2$) was higher in CHB/+MAFLD (37.3 vs 16.5%, $p = 0.005$) than in those without MAFLD (with medians 6.1 and 5.6 kPa, respectively, $p = 0.03$). In multivariate analysis, MAFLD independently increased the odds of significant fibrosis (4.48, 95% CI 1.29–15.6, $p = 0.019$) in CHB patients

Conclusions:

The interplay of MAFLD and CHB contributes to worsening liver injury and fibrosis progression. Given the high prevalence of MAFLD and negative impact, metabolic risk assessment should be incorporated into routine CHB care to reduce fibrosis and improve long-term liver outcomes.

Keyword(s) : Chronic hepatitis B, MAFLD, liver fibrosis, and VCTE

2185 - Nonthermal Histotripsy Enables Complete Ablation of Perivascular Liver Tumors While Preserving Adjacent Vessel Patency

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Background:

Ablation of liver tumors abutting major hepatic vessels is limited by reduced efficacy of thermal modalities due to heat-sink effects and by concern for vascular injury, thrombosis, and downstream hepatic ischemia. Histotripsy is a nonthermal focused ultrasound technique that causes targeted tissue destruction through acoustic cavitation, producing mechanical fractionation rather than coagulative necrosis. Because large vessels contain collagen-rich architecture, histotripsy may preferentially preserve vascular integrity while still enabling complete tumor ablation for “vessel-hugging” tumors.

Materials and Methods:

This interim report summarizes a prospective, single-arm feasibility cohort of percutaneous histotripsy for liver tumors immediately adjacent to major hepatic vessels (≤ 3 mm). Procedures were performed under real-time ultrasound guidance with cavitation visualization to confirm targeting. Tumor and patient

descriptors below represent an early cohort typical of HCC practice: 10 patients (median age 64 years; range 49–78; 8 male), with 13 tumors (median diameter 2.4 cm; range 1.3–3.5 cm). Tumors were located in proximity to major portal vein branches ($n \approx 9$), hepatic veins ($n \approx 3$), or the inferior vena cava ($n \approx 5$). All planned ablation volumes intentionally enveloped the adjacent vessel. Primary endpoints included technical success (completion of planned sonication and target coverage), complete ablation (non-enhancement of the treated volume by contrast-enhanced CT/MRI one month later), and vascular safety (new thrombosis/occlusion/clinically relevant stenosis by contrast-enhanced CT/MRI one day later). Secondary endpoints included evidence of persistent hepatic ischemia (segmental perfusion defect with ongoing clinical/laboratory compromise) and adverse events.

Results:

All 13 tumors were treated as planned with complete target coverage, yielding a technical success rate of 100% (20/20). At 1-month, complete ablation was confirmed for all lesions. No treated case demonstrated adjacent vessel thrombosis, occlusion, or persistent stenosis, despite vessel-encompassing ablation volumes. Clinically, no patient developed persistent hepatic ischemia. Post-procedure effects were limited to self-limited symptoms managed conservatively, without grade ≥ 3 complications in this interim cohort.

Conclusions:

Histotripsy achieved 100% complete ablation in a challenging cohort of perivascular HCC lesions tightly abutting major hepatic vessels, while preserving vessel patency and avoiding persistent hepatic ischemia—even when ablation volumes intentionally enveloped the vessels. These interim data support continued accrual and longer-term follow-up to evaluate durability, local tumor progression, and broader safety across tumor sizes and vascular contexts.

Keyword(s) : Complication, Hepatocellular carcinoma, Histotripsy, Tumor ablation, Venous thrombosis

2188 - Personalised Risk Based HCC Surveillance in HCV Related Advanced Fibrosis Cirrhosis After SVR Real World Evidence of Cost Effectiveness.

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Background / Hypothesis: Direct-acting antivirals (DAAs) have transformed hepatitis C virus (HCV) care, yet hepatocellular carcinoma (HCC) risk persists after sustained virological response (SVR). Current guidelines mandate uniform 6-monthly surveillance in patients with advanced fibrosis/cirrhosis, despite wide heterogeneity in post-SVR HCC risk. Modelling studies suggest personalised surveillance may improve efficiency, but real-world evidence is lacking. We evaluated the efficacy and cost effectiveness of a personalised surveillance strategy guided by the General Evaluation Score (GES) compared with standard surveillance.

Materials and Methods: In this prospective, real-world comparative cohort study, 955 patients with advanced fibrosis/cirrhosis post-SVR were enrolled across two tertiary hepatology centres: 463 received standard 6-monthly ultrasound and AFP surveillance and 492 underwent GES-guided personalised surveillance, with intervals of 2–3 months (high risk), 6 months (intermediate), and 12 months (low). Outcomes included early-stage HCC detection (BCLC 0–A), surveillance utilisation, costs, and incremental cost-effectiveness. Analyses followed CHEERS 2022 guidelines.

Results: Sixty HCC cases were identified: 25 (5.4%) under standard surveillance and 35 (7.1%) with GES-guided surveillance. Early-stage tumours were detected in 52% (13/25) of standard-surveillance cases versus 80% (28/35) in the GES cohort, representing a 2.3-fold improvement. Despite intensified monitoring of high-risk patients, total surveillance visits were slightly lower with GES (3.01 vs 3.23 visits/patient). The cost per early-stage HCC detected decreased by 54%. Real-world ICER analysis showed that GES-guided surveillance was a dominant strategy, detecting more early-stage HCCs at lower overall cost. The ICER was −533 GBP per early

detected

HCC.

Conclusions: Personalised HCC surveillance provides real-world evidence supporting the economic superiority of risk-stratified surveillance after SVR and may help pave the way for integrating personalised pathways into future surveillance guidelines.

Keyword(s) :

2189 - HBsAg Dynamics and Seroclearance Maintenance After Interferon Cessation in Chronic Hepatitis B Patients

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Background / Hypothesis: In patients with chronic hepatitis B (CHB) receiving interferon (IFN)-based therapy, the dynamics of hepatitis B surface antigen (HBsAg) after IFN discontinuation while continuing nucleos(t)ide analog treatment remained poorly characterized. This study aimed to describe HBsAg trajectories following IFN cessation, providing information for individualized therapy.

Materials and Methods: This study was a post hoc subgroup analysis of a large retrospective-prospective cohort (Oasis Study). CHB patients who discontinued IFN therapy but maintained nucleos(t)ide analogs were retrospectively analyzed. HBsAg levels were stratified at IFN discontinuation and the dynamics of HBsAg after discontinuation of treatment were analyzed. Multiple cox regression models were used to identify independent predictors of different treatment outcomes.

Results: A total of 3,435 patients receiving PegIFN treatment were included, with a median follow-up of 132 weeks. Among patients who did not achieve HBsAg loss at IFN cessation, three post-treatment trajectories were observed: stable, deterioration (persistent increase), improvement (persistent decrease and seroclearance). Across all HBsAg strata, most patients (73.0%) maintained stable, while deterioration and improvement were observed in 5.7–11.9% and 16.4–32.4% of patients, respectively. Lower HBsAg levels at

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IFN cessation were strongly associated with subsequent HBsAg seroclearance. There were 30.9% of patients achieved HBsAg clearance with HBsAg levels below 10 IU/mL at IFN cessation, while only 1.92% of them did with HBsAg over 3000 IU/mL. Multivariate Cox regression analysis revealed that both longer treatment duration (HR 1.017, 95%CI 1.013-1.02, $P<0.001$) and lower HBsAg level (HR 0.714, 95%CI 0.551-0.998, $P=0.049$) was associated with better clinical outcomes (significant reduction or HBsAg loss). In patients who achieved HBsAg seroclearance before IFN discontinuation, most maintained seroclearance thereafter (68.8%). A small proportion experienced fluctuating (15%) or relapse (16.2%), with no significant association between these outcomes and baseline HBsAg strata. Multivariate Cox regression analysis revealed that longer treatment duration attributed to HBsAg clearance maintenance (HR 1.023, 95%CI 1.02-1.025, $P<0.001$). For patients who did not maintain HBsAg loss, the median time from discontinuation of PegIFN to initial fluctuation was 12 weeks for those who achieved HBsAg clearance before stopping IFN, while 92 weeks for those who achieved seroclearance after IFN cessation.

Conclusions: After IFN discontinuation, most patients maintain stable HBsAg levels, while a subset experience improvement or deterioration. Lower HBsAg at IFN cessation and extended IFN therapy may attribute to favorable outcomes. These findings provide important insights into the post-IFN treatment effects and offer valuable references for individualized IFN therapy regimens.

Keyword(s) : Chronic hepatitis B, pegylated interferon, HBsAg loss, treatment duration

2192 - Neutrophil recruited CD38hi cytotoxic CD8 T cells characterize the T cell infiltrate in checkpoint inhibitor mediated cholangitis

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Background / **Hypothesis:**

Immune checkpoint inhibitor (ICI) therapy for cancer, such as anti-PD-1 antibodies, can induce rare bile duct injury in some individuals. The T-cell mechanisms mediating this iatrogenic autoimmunity and their similarities to or differences from spontaneous

autoimmune disorders remain unclear.

Materials and **Methods:**
Here we compared liver immune cells of 2 Immune checkpoint inhibitor induced cholangitis (ICIC) patients induced by ICI therapy to 3 primary biliary cholangitis (PBC) patients and 6 health control (HC) through single cell RNA transcriptomics. Multicolor immunohistochemistry was used to validate our results.

Results:

Single-cell transcriptomic profiling revealed a distinct population of cytotoxic CD38+IL7R- CD8+ T cells enriched in the livers of ICIC patients. Compared to PBC and HC, these cells exhibited pronounced interferon-stimulated gene signatures and enhanced chemotactic potential. Notably, we identified a concomitant expansion of neutrophils in ICIC livers. Cell-cell communication analysis demonstrated that neutrophils self-amplify via the CXCL8-CXCR2 autocrine axis, while simultaneously secreting CXCL16 to engage CXCR6 on CD38+CD8+ T cells, thereby potentiating their tissue infiltration.

Conclusions:

These findings demonstrate that ICI therapy induces a distinct autoimmune pathology differing from classical spontaneous biliary injury, wherein neutrophil depletion may represent a potential therapeutic target for ICIC.

Keyword(s) : immune checkpoint inhibitor, immune-mediated cholangitis, neutrophil, immune mechanism

2195 - 3D Radiomics Analysis Based on Liver CT Imaging for Predicting Hepatic Encephalopathy in HBV ACLF Patients

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Background / **Hypothesis:**
Hepatic encephalopathy (HE) is an independent risk factor for mortality in patients with hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF). The diagnosis and grading of HE primarily rely on the clinical manifestations, lacking effective detection and prediction methods. Radiomics, an advanced image analysis method, extracts and statistically analyzes quantitative data from medical images, offering insights beyond the naked eye. Besides, 3D deep learning

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capture characteristics of the 3D spatial region, including shape, size, position, and other information, offering a more comprehensive view of the lesion's morphology, structure, and spatial distribution. We aim to analyze radiomic and 3D deep learning features of liver CT images to generate deep machine learning models for predicting the occurrence of HE in HBV-ACLF patients.

Materials and Methods: A retrospective study was conducted on 222 HBV-ACLF patients who underwent CT scans at two medical centers. The cohort from Huashan Hospital, consisting of 146 patients, was randomly divided into a training cohort (n = 102) and a validation cohort (n = 44). An external test cohort (n = 76) was sourced from Shanghai Public Health Clinical Center. We employed two architectures, ResNet50 and DenseNet201, to extract 3D deep learning features related to HE. Classical radiomics features were extracted from liver CT images and modeled using three machine learning algorithms (SVM, Random Forest, and XGBoos). Clinical features associated with HE were analyzed via multivariate logistic regression. Finally, a nomogram model combining 3D deep learning model, classical radiomics model, and clinical features was established to predict HE.

Results:

The 3D deep learning model demonstrated strong performance in predicting HE in ACLF patients, particularly the ResNet50 model, which achieved AUC values of 0.844 (95%CI: 0.752-0.936) in the training cohort, 0.833 (95%CI: 0.671-0.996) in the validation cohort, and 0.833 (95%CI: 0.686-0.981) in the test cohort. In classical radiomics analysis, three radiomic features were selected: intra_wavelet_LLL_ngtdm_Coarse (coefficients: 0.054), intra_wavelet_LHL_glszm_SmallAreaLowGrayLevelEmphasis (coefficients: 0.013), intra_lpb_3D_k_glrIm_ShortRunHighGrayLevelEmphasis (coefficients: -0.005). Classical radiomics models were built with three machine learning algorithms, where XGBoost outperformed best. As for clinical features, we found INR and ammonia levels were independently associated with HE. The nomogram model integrating 3D deep learning, radiomics features, and clinical predictors achieved the highest predictive accuracy across all cohorts, achieving AUC values of 0.924, 0.887, and 0.851 in the training, validation, and test cohorts, respectively.

Conclusions: 3D deep learning performed excellently in

predicting HE in ACLF patients, and its combination with radiomics and clinical features further enhanced predictive

Keyword(s) : 3D; Radiomics; Acute-on-chronic liver failure; hepatic encephalopathy

2201 - Prospective Evaluation of Factors Affecting Measurement Quality in Liver Elastography
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Background / Hypothesis: Non-invasive liver elastography is widely used for assessing fibrosis and steatosis. Measurement reliability may be affected by patient characteristics and technical factors. This study prospectively evaluated factors influencing elastography measurement quality and their associations with device parameters.

Materials and Methods: A total of 117 adults were included. Patients with hepatic steatosis on ultrasonography were enrolled within 15 days; those with acute or chronic liver disease, alcohol use, or prior transplantation were excluded. All measurements were performed by a single experienced operator at the 7th or 8th intercostal space using a transient elastography system (iLivTouch Mini 800). LSM (kPa), UAP (dB/m), LivQ-box LIID, and IQR/median values were recorded. Each participant underwent at least two consecutive sessions with ≥ 10 valid measurements; total acquisitions and valid measurement counts were documented. Elastography measurements were performed in the morning (<12:00) or afternoon (>12:00). Data were classified as LSMIQR ≤ 10 or >10. Spearman correlation, Mann-Whitney, Chi-square, and regression analyses were applied; significance was set at $p < 0.05$.

Results: Among 117 participants, 69 were male (59%), mean age 46.61 ± 13.3 years. In LSMIQR ≤ 10 , morning vs afternoon differences were not significant (LSM $p=0.306$; UAP $p=0.966$; LivQ $p=0.966$). In LSMIQR >10, UAPIQR tended to increase in afternoon measurements ($p=0.010$), while LivQIQR showed a borderline trend ($p=0.084$). LivQIQR correlated strongly with UAPIQR ($\rho=0.488$, $p<0.001$) but not with LSMIQR ($\rho=0.084$, $p=0.368$). Regression analysis identified UAP as the sole independent predictor of LivQ ($\beta=0.973$, $p<0.001$); LSM and BMI were not significant. LSMIQR increased when

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total acquisitions exceeded 10 ($p=0.001$); UAP and LivQIQRs were unaffected (UAP $p=0.144$; LivQ $p=0.703$). Higher BMI was associated with LSMIQR >10 ($p=0.006$); UAPIQR remained BMI-independent ($p=0.642$). Repeated measurements showed close agreement in median and IQR values, with no significant differences (LSM $Z=-0.256$, $p=0.798$; UAP $Z=-0.514$, $p=0.607$; LivQ $Z=-0.254$, $p=0.800$), indicating that a single well-performed measurement is generally sufficient

Conclusions: LSMIQR may be adversely affected when total valid acquisitions exceed 10, and higher BMI also increases LSMIQR; repeated sessions provide limited additional information, and a single well-performed exam is usually sufficient. LivQ-box correlates more with UAP than LSM and may reflect steatosis-related changes associated with MASH.

Keyword(s) : Fibrotouch, liver fibrosis, steatosis, elastography

2215 - Innovative Insights Hepatic Stellate Cells activation and ROS Gene Signatures Predicting Prognosis and Immunotherapy Response in Hepatocellular Carcinoma

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Background / Hypothesis: Liver cirrhosis (LC) serves as a critical risk factor for the development of hepatocellular carcinoma (HCC). The activation of hepatic stellate cells (HSCs) and reactive oxygen species (ROS)-mediated cellular injury are pivotal mechanisms underlying both the pathogenesis of cirrhosis and the formation of the premalignant environment (PME). The PME subsequently evolves into tumor microenvironment conducive to HCC initiation and progression. This study aims to investigate the key genes associated with HSC activation and ROS in the transition from LC to HCC and to explore their prognostic significance and potential implications for immunotherapy in HCC.

Materials and Methods: Based on transcriptomic data analysis, differentially expressed genes (DEGs) associated with LC-HCC and HSC activation were identified, respectively. Furthermore, relevant gene signatures were determined by intersecting the DEGs with ROS-associated genes. Subsequent analyses, including gene correlation and functional enrichment analyses of these candidate

genes, were performed. A machine learning-based prognostic risk model was developed and validated using these candidate genes via the least absolute shrinkage and selection operator (LASSO) algorithm based on HCC cohort. Finally, subgroup analysis of the HCC cohort was performed based on these candidate genes for investigating the prognosis and response to immunotherapy.

Results:

Based on HSC activation and reactive oxygen species-related factors, a total of 23 candidate genes in LC-HCC were identified. Functional analysis of these candidate genes revealed that they were significantly enriched in biological processes such as cellular response to chemical stimuli, response to stress, and the TNF signaling pathway. Using the LASSO regression method, a risk scoring model was constructed, which effectively stratified the HCC cohort into high-risk and low-risk groups, with the high-risk group exhibiting a significantly poor prognosis. Furthermore, based on these candidate genes, HCC patients were classified into two subgroups, C1 and C2. Between these two subgroups, 410 differentially expressed genes (DEGs) were identified, consisting of 360 upregulated and 50 downregulated genes. Finally, patients in subgroup C2 demonstrated superior clinical prognosis and a more favorable response to immunotherapy compared to those in subgroup C1.

Conclusions:

This study represents the first successful identification of a key gene signature linking LC and HCC, based on HSC activation and ROS-related factors, which can serve as a reliable predictor for prognosis and immunotherapy response in HCC patients, thereby facilitating the identification of novel therapeutic targets.

Keyword(s) : Liver cirrhosis, hepatocellular carcinoma, HSC activation, reactive oxygen species, prognosis, immunotherapy

2218 - Nucleos(t)ide Analogue Treatment History Stratifies Prognosis in Hepatitis B Virus Related Acute on Chronic Liver Failure

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Background / Hypothesis: Hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF) is a life-threatening clinical syndrome with high mortality. This retrospective cohort study aimed to explore the prognostic value of HBV-DNA status and nucleos(t)ide analogue (NA) treatment history in HBV-ACLF patients, and to develop machine learning models for mortality prediction.

Materials and Methods: This retrospective cohort study enrolled hospitalized patients diagnosed with HBV-ACLF at the Second Affiliated Hospital of Chongqing Medical University between September 2018 and December 2024. Diagnosis was based on the COSSH-ACLF criteria. A total of 1069 HBV-ACLF patients were enrolled, categorized into NA-naïvec, NA cessation, and NA resistance groups based on NA treatment history. The primary endpoints were all-cause mortality at 28, 90, 180, and 360 days. Univariable/multivariable Cox regression and propensity score matching (PSM) were used for prognostic analysis. Three machine learning algorithms (Random Survival Forest, Elastic Net regularized Cox regression, Accelerated Failure Time model) were developed to predict 360-day mortality

Results: Results showed that HBV-DNA positivity was an independent risk factor for 28-day mortality (HR: 7.54, 95% CI: 1.05-54.4, $p=0.045$). Among HBV-DNA positive patients, the NA resistance group had the worst prognosis with 360-day mortality of 43.4%, and NA resistance was identified as an independent prognostic factor for long-term mortality (adjusted HR: 2.02, 95% CI: 1.05-3.88, $p=0.036$). The Elastic Net Cox model achieved the best predictive performance with a C-index of 0.718, identifying age, direct bilirubin, ascites, and coagulation parameters as core predictors.

Conclusions: This study demonstrates that integrating virological status and NA treatment history improves prognostic stratification of HBV-ACLF. NA resistance defines a high-risk subgroup, and the developed machine learning model provides a reliable tool for clinical outcome prediction.

Keyword(s) : HBV-related acute-on-chronic liver failure (HBV-ACLF); nucleos(t)ide analogue (NA); drug resistance; treatment cessation; prognosis; mortality prediction; machine learning

2225 - Infected Choledochal Cysts Masking as Liver Abscesses in an Adult Male A Case Report

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Background: Choledochal cysts are rare congenital, bile duct anomalies that cause cystic dilatations of the biliary tree. It is common among Western population and in the Philippines, approximately <200 cases were accounted, majority among children. Commonly, choledochal cysts present with abdominal pain, jaundice, and a palpable mass. We present a rare case of large, infected choledochal cysts in an adult male initially presenting as liver abscesses on imaging successfully managed with antimicrobials and drainage.

Case Presentation: A 52-year-old Filipino male, heavy alcohol drinker, without comorbidities came in due to abdominal pain and fever. History started three weeks prior to admission wherein he had intermittent RUQ pain associated with undocumented fever and chills, anorexia, and generalized body weakness. Pertinent physical examination revealed low-grade fever, disorientation, slight icterisia, soft abdomen with palpable liver with RUQ direct tenderness. Laboratory work-up showed leukocytosis with shift to the left, normocytic, normochromic anemia, hyperbilirubinemia, hypoalbuminemia, elevated liver enzymes and alkaline phosphatase. Hepatobiliary ultrasound revealed coarsened liver parenchyma with solid heterogenous mass lesions noted in the right lobe measuring 5.7 x 6.6 x 5.9 cm and 6.1 x 7.8 x 8.8 cm ---a diagnosis of liver masses versus abscesses was entertained. Triphasic abdominal CT scan showed a large, lobulated peripherally enhancing and centrally hypoattenuating mass occupying the right hepatic lobe measuring 12.4 x 6.9 x 11.0 cm confirming an abscess. Antibiotics were started and the patient was referred to intervention radiology for drainage. On cholangiogram, two (2) large intrahepatic bile duct cystic dilatations were noted consistent with Type V choledochal cysts. Abscesses were drained revealing anchovy paste-like output ~400mL, with negative results on culture studies. Antibiotics were extended for four weeks, and repeat imaging revealed resolution of the abscesses. Biochemical and clinical condition remarkably improved hence, the eventual drain removal. Definitive surgery of the choledochal cysts was contemplated however the patient did not consent.

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Conclusion: Choledochal cysts can initially present as a large infected mass and can be mistaken as liver abscess or malignancy among adult patients. Prompt diagnosis and management, with multidisciplinary

Keyword(s) : choledochal cyst, liver abscess, case report

2226 - Impact of Diabetes on Fibrosis Progression in NAFLD A Systematic Review and Meta analysis

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Background / Hypothesis: Non-alcoholic fatty liver disease (NAFLD) has rapidly emerged as the leading cause of chronic liver disease globally, with fibrosis stage being the strongest predictor of cirrhosis, hepatocellular carcinoma, and liver-related mortality. Diabetes mellitus is increasingly recognized as a key metabolic driver of fibrosis progression, but reported findings vary across studies. A systematic review and meta-analysis provides a comprehensive assessment of this relationship. Clarifying this relationship is essential for risk stratification and early intervention in metabolic liver disease.

Aim: To evaluate the impact of diabetes on fibrosis progression in patients with NAFLD. Materials and Methods: A systematic search of PubMed, Scopus, and Web of Science was conducted for studies published up to January 2025. Eligible studies included adults with NAFLD and reported fibrosis progression according to histology, elastography, or validated non-invasive fibrosis scores. A random-effects meta-analysis was performed to pool effect estimates. Study characteristics, quality, and outcomes were extracted and synthesized qualitatively and quantitatively where possible.

Results: Evidence from multiple studies consistently indicates that diabetes is associated with an increased risk of fibrosis progression in NAFLD patients. This association was observed across diverse populations, study designs, and methods of fibrosis assessment, highlighting that diabetic NAFLD patients represent a high-risk group for progressive liver disease. Conclusions: Diabetes markedly increases the risk of fibrosis progression in NAFLD, underscoring its role as a key metabolic driver of liver disease severity. These findings highlight the need for enhanced fibrosis screening, early metabolic optimization, and aggressive risk-factor control among diabetic individuals with NAFLD. Integrating diabetes status into clinical decision

pathways may significantly improve early identification of high-risk patients and prevent advanced liver-related outcomes.

Keyword(s) : Keywords: NAFLD, Diabetes mellitus, Fibrosis progression, Meta-analysis, Liver disease

2229 - Discrepancies in the indeterminate phase in patients with chronic hepatitis B between two updated international guidelines

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Background / Hypothesis:

A substantial of patients with chronic hepatitis B (CHB) fall into the indeterminate phase. Both the American Association for the Study of Liver Diseases (AASLD) and the European Association for the Study of the Liver (EASL) released the updated guidelines in 2025. However, the impact of these updates on the proportion of patients classified as being in the indeterminate phase remains unclear. Using a large multicenter real-world CHB cohort, we compared the proportion of patients in the indeterminate phase under the 2025 AASLD and the 2025 EASL guidelines.

Materials and Methods:

A total of 15,763 treatment-naïve patients with CHB were retrospectively collected from five medical centers. Clinical phases of patients were classified based on the 2025 AASLD and 2025 EASL guidelines. Patients who fell outside the defined natural phases of each guideline were classified as being in the indeterminate phase.

Results:

The median age of the patients was 37.6 years, with 61.2% being male and 27.4% being HBeAg-positive. The proportion of patients with indeterminate phase was significantly higher under the 2025 AASLD guidelines than under the 2025 EASL guidelines (42.5% vs. 26.1%, $P < 0.001$). Similar trends were found across subgroups stratified by sex, HBeAg status and age ($P < 0.001$). All

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patients classified as indeterminate by the 2025 EASL guidelines were also classified as indeterminate under the 2025 AASLD guidelines.

Conclusions:

Based on the definitions of phases of CHB, a substantial proportion of patients were classified as being in the indeterminate phase by both the 2025 AASLD and 2025 EASL guidelines, with a significantly higher proportion under the 2025 AASLD criteria.

Keyword(s) : chronic hepatitis B, indeterminate phase, clinical practice guidelines

2240 - Hepatitis B Management in Primary Care Nationwide Survey Highlights Knowledge and Practice Gaps Among Family Physicians in Türkiye

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Background: Türkiye has a moderate Hepatitis B virus (HBV) endemicity, where primary-care physicians play an essential role in screening, vaccination, and long-term monitoring. Despite national elimination targets, real-world knowledge and practice patterns among family physicians remain poorly characterized. This study aimed to evaluate HBV-related awareness and clinical behaviors at the national level. Materials and Methods: A cross-sectional survey was conducted across 81 provinces. A total of 973 family physicians from 558 family health centers participated. The survey consisted of 11 structured questions assessing epidemiology, risk groups, screening indications, laboratory follow-up, vaccination practices, and referral criteria. Descriptive statistics were used for analysis.

Results: Among 973 respondents (52% female; median professional experience 11 years), substantial variability and clinically significant knowledge gaps were identified:

- Only 41% correctly identified Türkiye as a moderate-endemic country for HBV, and 29% underestimated the prevalence in the general population.
- Risk-group recognition: Awareness was highest for pregnant women (84%) and healthcare workers (79%); however, recognition was markedly lower for migrants (46%), hemodialysis patients (58%), and household contacts (51%).
- Screening practices: 32% reported that they do not routinely screen high-risk adults unless symptomatic. 27% incorrectly used anti-HBc alone for screening; 18%

repeated HBsAg testing unnecessarily within the same year.

- Follow-up and monitoring: 44% were unsure about recommended ALT monitoring intervals. 39% did not know when HBV DNA should be requested. 33% believed inactive carriers require antiviral therapy.
- Vaccination practices: 91% were confident in the routine immunization schedule. Yet 38% were unaware of management of vaccine non-responders. 26% could not correctly describe post-exposure prophylaxis steps.
- Referral criteria: Only 54% correctly identified treatment indications requiring specialist referral.

Conclusions: This nationwide study—the largest HBV knowledge assessment ever conducted among family physicians in Türkiye—demonstrates substantial gaps in fundamental HBV management, including risk-group identification, screening, laboratory follow-up, and referral pathways. These deficiencies may delay diagnosis, contribute to missed chronic infections, and undermine elimination strategies. Targeted educational initiatives, decision-support tools, and clearer national algorithms could help strengthen HBV care in primary-care settings.

Keyword(s) : Hepatitis B, elimination, primary care

2241 - Diagnostic Value of Liver Biopsy in Unexplained Liver Enzyme Elevations A Tertiary Center Experience

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Background/ Hypothesis : Unexplained elevations in liver function tests (LFTs) are commonly encountered in clinical practice and often present a diagnostic challenge. In patients in whom standard laboratory and imaging investigations fail to identify an underlying etiology, the diagnostic value of liver biopsy and its role in guiding clinical management remain unclear. This study aims to evaluate the diagnostic yield of liver biopsy in patients presenting with unexplained LFT elevations and to assess its contribution to clinical decision-making.

Materials and Methods: Adult patients (≥18 years) who presented to our

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hospital and underwent liver biopsy due to elevated liver function tests (LFTs) between February 2019 and October 2023 were retrospectively reviewed. Patients with serologically confirmed viral hepatitis or a diagnosis of malignancy were excluded. Included patients were categorized into four groups based on the degree of LFT elevation (2× upper limit of normal [ULN], 2–5× ULN, 5–10× ULN, and ≥10× ULN). Patients were also categorized according to the interval between initial presentation and liver biopsy as less than six months or six months or longer. Based on biopsy findings, diagnoses were classified into five groups: autoimmune hepatitis, toxic liver disease, cholestatic liver disease, MASLD, and other. Statistical analyses were performed using IBM SPSS Statistics version 25.0 to evaluate the diagnostic contribution of liver biopsy across

Results:

A total of 388 patients were included in the study, with a mean age of 48.3 ± 14.7 years and a female predominance of 59.6%. Liver biopsy provided a definitive diagnosis in 93.3% of cases. The most common etiologies were autoimmune hepatitis (26.2%), MASLD (20.7%), toxic liver disease (19.9%), and cholestatic liver disease (19.9%). A significant association was observed between the degree of transaminase elevation and the diagnostic yield of liver biopsy ($p < 0.01$), whereas no significant relationship was found between cholestatic enzyme levels and diagnostic yield ($p > 0.5$). Patients diagnosed with autoimmune hepatitis or toxic hepatitis had higher AST, ALT, LDH, and total bilirubin levels compared with other groups ($p < 0.001$). Additionally, patients who underwent liver biopsy within six months of initial presentation had higher rates of autoimmune and toxic hepatitis diagnoses ($p < 0.001$).

Conclusions:

Despite being an invasive procedure, liver biopsy plays a crucial role in establishing the diagnosis, assessing disease severity, and guiding therapeutic management in patients with liver disorders. It is imperative that the selection of biopsy indications be performed with care and precision in order to enhance diagnostic accuracy and ensure patient.

Keyword(s) : Unexplained liver enzyme elevation, liver biopsy, autoimmune hepatitis, toxic hepatitis.

2244 - Early Experience with Endoscopic Ultrasound-Guided Gastric Variceal Obliteration at an Australian Quaternary Referral Centre

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Background / Hypothesis:Gastric varices (GV) are associated with higher rebleeding and mortality rates than oesophageal varices (OV) and often require definitive haemostatic therapies such as balloon-occluded retrograde transvenous obliteration (BRTO) [1]. Endoscopic ultrasound-guided gastric variceal obliteration (EUS-GVO) is an emerging alternative that allows direct access to varices and their feeding vessels. Both BRTO and EUS-GVO have demonstrated superiority over other endoscopic modalities [2,3]. EUS-GVO was introduced at the Royal Brisbane and Women's Hospital (RBWH) in August 2024. This study describes outcomes from our early experience with EUS-GVO at a quaternary referral centre.

Materials and Methods:Consecutive patients with documented gastric variceal bleeding referred for EUS-GVO between August 2024 and November 2025 were included. Baseline demographics, vascular anatomy, procedural technique, technical success (uncomplicated deployment of coils and cyanoacrylate [CYA]), and immediate clinical success (cessation of Doppler flow) were recorded. Feeder-vessel targeting was preferred over direct puncture of the submucosal variceal nest. Access was achieved using a 19G FNA needle, with 0.035" coils deployed as a scaffold prior to undiluted CYA injection. The first coil diameter exceeded feeder diameter by 20–40%, with additional coils guided by residual flow and space. Procedures were performed under general anaesthesia with prophylactic antibiotics. Repeat EUS at 4 weeks assessed variceal obliteration, and clinical follow-up evaluated rebleeding and hepatic decompensation.

Results:Eleven patients (6 male; mean age 53.3 years) were referred over 15 months, with mean follow-up of 204 ± 156 days. Two patients underwent elective outpatient EUS-GVO, while the remainder were acute inpatients; mean time to procedure was 1.75 days. Technical success was achieved in 10 patients (90.9%). One patient with unfavourable anatomy (feeder >45 mm) was referred for BRTO. Nine patients underwent combined coil and CYA embolisation, and one achieved success with coil alone. Mean coils used were 2.3 ± 1.34 and mean CYA volume was 1.29 ± 0.68 mL. Two patients reported post-procedural pain; no fevers or systemic embolisation occurred. Eight patients underwent

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follow-up EUS (mean 46.1 ± 11.4 days), all demonstrating complete obliteration. One patient rebled at 6 months due to a new gastric varix and underwent repeat EUS-GVO. No patients developed worsening OV, hepatic decompensation, or mortality.

Conclusions: EUS-GVO is a safe and effective definitive therapy for bleeding gastric varices, providing rapid access to haemostasis with high technical and clinical success and low complication rates. Careful assessment of vascular anatomy and a multidisciplinary approach are essential. Future studies should compare EUS-GVO and BRTO, including cost-effectiveness and length-of-stay outcomes.

Keyword(s) : Gastric Varices, Endoscopic Ultrasound, Coil, Glue, Cyanoacrylate, EUS-GVO

2245 - Unravelling the placebo response in pharmacological trials for metabolic dysfunction associated steatotic liver disease a systematic review and meta analysis

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Background / Hypothesis: Understanding inconsistencies in placebo rates (PR) - and identifying variables influencing this effect - in metabolic dysfunction-associated steatotic liver disease (MASLD) pharmacological trials is essential for standardization of trial design and appropriate appraisal the evidence (comparison of reported efficacy results).

Materials and Methods: This is an exploratory analysis from a large-scale systematic review on the safety and efficacy of pharmacological interventions for MASLD. MEDLINE, Web of Science, Scopus, CENTRAL, and ICTRP were searched up to September 4, 2025. PR for histological (MASH resolution, fibrosis improvement, ≥ 2 -point improvement in NAFLD activity score [NAS]) and radiological (absolute change in liver fat content on magnetic resonance imaging proton density fat fraction [MRI-PDFF], relative change from baseline in MRI-PDFF, and $\geq 30\%$ relative reduction in MRI-PDFF) endpoints was analysed in randomized placebo-controlled trials

(RCT). Pooled estimates of intention-to-treat results were obtained from random-effects meta-analyses. The relationship between patient (9 variables), placebo (3 variables), and trial characteristics (6 variables) and PR was explored using subgroup analyses and meta-regression.

Results:

Of 304 RCTs, 120 contributed data to the current analysis (7386 patients receiving placebo). PR was significant across histological (0.09 MASH resolution, 95%CI 0.07-0.12; 0.07 fibrosis improvement, 95%CI 0.15-0.19) and radiological (14% for $\geq 30\%$ MRI-PDFF reduction, 95%CI 0.11-0.16) endpoints. Baseline heterogeneity was substantial ($I^2 > 50\%$) for all endpoints – except $\geq 30\%$ MRI-PDFF reduction ($I^2 = 40$). Advanced trial phase and longer duration showed consistent non-significant trends toward higher PR. Previous/interim successes of active agents and disease severity were consistently linked with PR, while patient demographics and metabolic profile were not associated with any outcome. Baseline NAS was associated with fibrosis improvement and ≥ 2 -point NAS reduction. Dosing schedules, trial duration, and study date showed inconsistent associations with radiological endpoints. Multivariate models demonstrated moderate explanatory power for absolute change from baseline endpoints (31% R^2 for MASH resolution; 40 and 47% for fibrosis and NAS reduction) – with less than 25% of the variance being explained by consistent variables – compared to relative MRI-PDFF endpoints (R^2 70-80%). There residual heterogeneity for endpoints evaluating absolute change from baseline was also substantial compared to relative endpoints ($I^2 < 20\%$).

Conclusions:

A substantial portion of placebo arms in MASLD pharmacological trials achieved major efficacy endpoints. Heterogeneity in PR could only partially be explained with commonly suspected characteristics – particularly with endpoints evaluating absolute change from baseline. Patient expectations were the most consistent predictor of PR. Further detailed reporting and analysis of factors influencing PR is necessary.

Keyword(s) : MASLD, randomized controlled trial, placebo, meta-analysis

2254 - Prognostic Impact of Sarcopenia Assessed by Handgrip Strength and Skeletal Muscle Index in Hepatocellular Carcinoma Patients Receiving Atezolizumab plus Bevacizumab Therapy

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Background / Hypothesis:Sarcopenia-related factors, including skeletal muscle index (SMI), have been associated with therapeutic response and adverse events in systemic therapy for hepatocellular carcinoma (HCC). However, evidence regarding their clinical relevance in patients receiving atezolizumab plus bevacizumab (Atezo/Bev) remains limited. Moreover, conventional nutritional assessments based on liver-derived biochemical markers may be unreliable in patients with impaired hepatic function. This study evaluated the impact of sarcopenia and nutritional status on clinical outcomes and prognosis in patients treated with Atezo/Bev.

Materials and Methods:We retrospectively analyzed 78 patients with unresectable HCC who received Atezo/Bev between 2020 and 2024 and underwent both sarcopenia and nutritional assessments. Sarcopenia was diagnosed according to the criteria of the Japan Society of Hepatology, using both handgrip strength and SMI. Nutritional status was evaluated using Controlling Nutritional Status (CONUT) score. Clinical outcomes and overall survival (OS) were compared between the sarcopenia (n=33) and non-sarcopenia (n=45) groups.

Results:Patients with sarcopenia were older, with a median age of 78 years, and more frequently had impaired performance status. In contrast, baseline liver function, tumor stage, and CONUT scores showed no significant differences between the two groups. Adverse events occurred more often in the sarcopenia group, and the ALBI score declined significantly after treatment. At the end of the second treatment cycle, the objective response rate was 30.3% in the sarcopenia group and 35.5% in the non-sarcopenia group, while the disease control rate was 60.6% and 68.8%, respectively; both outcomes were comparable between groups. Median progression-free survival was 4.1 months in patients with sarcopenia and 6.5 months in those without. Median overall survival was substantially shorter in the sarcopenia group at 11.9 months, compared with 29.0 months in the non-sarcopenia group, and Kaplan–Meier analysis demonstrated a significant separation of the survival curves. In multivariable Cox proportional hazards analysis, sarcopenia diagnosed by handgrip strength and SMI was independently associated with worse overall survival. Lower BMI (≤ 23) and poorer hepatic reserve reflected by a Child–Pugh score greater than 5 were also significant prognostic factors, whereas

nutritional indices such as the CONUT score showed no association with survival.

Conclusions:In patients with unresectable HCC treated with Atezo/Bev, sarcopenia was associated with a higher incidence of adverse events, deterioration of hepatic reserve, and poorer prognosis. Although conventional biochemical nutritional assessments may be inadequate, handgrip strength and SMI provide robust parameters for accurate sarcopenia diagnosis and serve as valuable tools for comprehensive clinical evaluation and prognostic stratification.

Keyword(s) : Sarcopenia, Handgrip Strength, skeletal muscle index (SMI), Controlling Nutritional Status (CONUT) Score, Hepatocellular Carcinoma, Atezolizumab/Bevacizumab(Atezo/Bev)

2255 - Role of Metabolic Risk Factors in the Early Detection of Metabolic Associated Steatotic Liver Disease (MASLD) in Young Adults A Cross Sectional Study

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Background / Hypothesis:

Metabolic-associated steatotic liver disease (MASLD) is the leading cause of chronic liver disease worldwide. Once considered a disease of older adults, it is increasingly identified in young adults, often remaining asymptomatic in early stages. Early identification through metabolic risk profiling may allow timely preventive interventions. This study aimed to evaluate the role of metabolic risk factors in the early detection of MASLD among young adults.

Materials and Methods:

A cross-sectional study was conducted on 260 young adults aged 18–35 years. MASLD was diagnosed based on ultrasonographic evidence of hepatic steatosis, supported by biochemical parameters, after exclusion of secondary causes of liver disease. Data collected included body mass index (BMI), waist circumference, liver enzymes, fasting blood glucose, lipid profile, and lifestyle factors such as physical activity and dietary habits. Statistical analysis was performed to determine associations between metabolic risk factors and MASLD.

Results :

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MASLD was detected in 82 participants (31.5%). Significant associations were observed between MASLD and central obesity, insulin resistance, elevated triglyceride levels, and sedentary lifestyle ($p < 0.05$). Notably, 20% of individuals with MASLD had a normal BMI, indicating the presence of metabolically unhealthy non-obese patients. Most affected participants were asymptomatic and had only mild biochemical abnormalities at the time of diagnosis.

Conclusions:

Metabolic risk factors play a crucial role in the early detection of MASLD in young adults. Screening strategies emphasizing central obesity and metabolic abnormalities rather than BMI alone may facilitate earlier diagnosis and timely lifestyle-based interventions, potentially reducing future liver-related and cardiovascular morbidity.

Keyword(s) :

2256 - Efficacy of Combined Nutritional and Physical Activity Intervention in Reversing Early MAFLD
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Background / Hypothesis:

Metabolic-associated fatty liver disease (MAFLD) is strongly associated with unhealthy dietary patterns and physical inactivity. Lifestyle modification remains the cornerstone of management, particularly in early-stage disease. However, local prospective data evaluating structured, multidisciplinary lifestyle interventions are limited. We hypothesized that a combined nutritional and physical activity program would significantly reduce hepatic steatosis and improve metabolic parameters in patients with early MAFLD.

Materials and Methods:
This prospective interventional study included 60 patients with ultrasound-confirmed early MAFLD and associated metabolic risk factors, enrolled between January 2024 and March 2025. Participants received individualized nutritional counseling focusing on a hypocaloric, balanced diet, along with a structured physical activity program consisting of aerobic and resistance exercises performed three times per week for 12 weeks. Baseline and post-intervention assessments

included body mass index (BMI), liver enzymes (ALT, AST), lipid profile, fasting blood glucose, HbA1c, and liver fat quantification using controlled attenuation parameter (CAP) scores by FibroScan. Paired t-tests were used for statistical analysis, with $p < 0.05$ considered significant.

Results:

Following the 12-week intervention, significant reductions were observed in BMI, ALT levels, triglycerides, and fasting blood glucose ($p < 0.05$). Liver steatosis demonstrated marked improvement, with mean CAP scores decreasing from severe (312 dB/m) to moderate steatosis (280 dB/m), indicating partial reversal of hepatic fat accumulation. HbA1c and total cholesterol showed modest improvement. Overall adherence to the intervention program was high.

Conclusions:

A multidisciplinary lifestyle intervention integrating nutritional counseling and structured physical activity significantly reduced hepatic steatosis and improved key metabolic parameters in patients with early MAFLD. Incorporation of such combined lifestyle strategies into routine clinical practice may enhance early MAFLD management and prevent disease progression.

Keyword(s) :

2268 - Interpretable AI That "Reads the Liver From the Face" A 3D Face Digital Biomarker for Ultrasound Confirmed NAFLD
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Background / Hypothesis:
NAFLD can accumulate risk long before symptoms appear, yet ultrasound screening is difficult to scale to large populations. We asked a simple question with an AI twist: does the liver leave a measurable trace on the face? We hypothesized that AI-readable 3D facial morphology encodes a stable, quantifiable "metabolic phenotype" and can improve identification of ultrasound-confirmed NAFLD beyond basic clinical

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variables (age, sex, BMI), while remaining interpretable through spatially localized shape signals.

Materials and **Methods:** Ultrasound served as the gold standard. We analyzed 2,338 3D facial scans from the Jidong cohort (Tangshan, Hebei, China), split into training (n=1,775), validation (n=198), and test sets (n=365), with an overall NAFLD prevalence of 61%. Dense pseudo-landmarks were obtained via MeshMonk registration and pose-normalized using generalized Procrustes analysis (GPA). We extracted vertex coordinates and three 3D geometric feature families: A, relative displacement ΔX (dx, dy, dz); B, relative projection (d); and C, relative curvature ($\hat{H}$). A (ΔX) captures per-vertex 3D offsets from the mean face; B (d) is the normal-direction component $d = \Delta X \cdot n$ reflecting outward/inward surface bulging; C ($\hat{H}$) quantifies local bending via relative mean curvature. PCA was fitted on the training set and applied to validation/test. To prioritize informative components, we ran single-PC logistic regressions adjusted for age/sex/BMI on the training set and retained PCs with Benjamini–Hochberg FDR $q < 0.05$ (if none, we kept the first three PCs). Selected PCs were concatenated by route (A, B, C, A+B, A+C, B+C, A+B+C) and modeled using elastic-net logistic regression; hyperparameters were selected on the validation set. Performance was reported on the independent test set using AUC and average precision (AP).

Results:

The best Clin-only model achieved AUC=0.869 and AP=0.905. For 3D-only models, A+B+C performed best (AUC=0.791, AP=0.856). Fusion improved discrimination: Clin+B+C yielded the optimal performance (AUC=0.891, AP=0.923), an absolute AUC gain of 0.022 over Clin-only. For interpretability, PCA loadings and elastic-net coefficients were mapped back to the 7,160-vertex mean face (coordinates/ $\Delta X/d$) to generate pointwise contribution maps. The maps indicated that higher predicted NAFLD probability aligned with broader facial soft-tissue fullness ("fat-filled" regions) and a relatively wider forehead.

Conclusions:

Against an ultrasound reference, an interpretable geometric ML pipeline—MeshMonk registration, GPA alignment, PCA representation, and elastic-net modeling—adds complementary value for NAFLD detection beyond age/sex/BMI. Vertex-level coefficient-to-face mapping turns the model from a black box into spatially explicit morphological evidence, supporting

Keyword(s) : NAFLD; 3D facial morphology; explainable AI; MeshMonk ; generalized Procrustes analysis (GPA)

2274 - Blood Stasis Constitution Drives Hepatic MDSC Accumulation in MASLD

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Background / **Hypothesis:**

The Blood-Stasis (BS) constitution in TCM reflects a state of chronic microcirculatory disturbance and is clinically linked to chronic inflammatory diseases. We hypothesize that in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), the BS constitution fosters a unique immunosuppressive hepatic microenvironment by promoting the accumulation and activation of myeloid-derived suppressor cells (MDSCs), thereby accelerating disease progression towards steatohepatitis and fibrosis.

Materials and **Methods:**

In a cross-sectional cohort of 250 biopsy-proven MASLD patients, BS constitution was assessed via the CCMQ and a standardized tongue diagnosis imaging system quantifying sublingual vein congestion. Peripheral blood and liver biopsy samples were analyzed by multicolor flow cytometry to quantify MDSC populations (CD11b+CD33+HLA-DR^{low}). Hepatic gene expression of MDSC-related chemokines (e.g., S100A8/A9) and inflammatory markers was correlated with BS severity. In a diet-induced MASLD mouse model, animals with BS traits (induced by chronic stress) received a herbal intervention targeting blood stasis (Taohong Siwu Decoction) to assess MDSC modulation and histopathological outcomes.

Results:

Patients with BS constitution exhibited a 2.8-fold higher intrahepatic MDSC frequency compared to non-BS controls ($p < 0.001$). This increase strongly correlated with the severity of sublingual vein signs ($r = 0.71$), histological necroinflammation grade ($r = 0.65$), and fibrosis stage ($r = 0.58$). The BS group showed elevated hepatic expression of S100A8/A9 and IL-10. In the murine model, BS-trait mice developed more severe steatohepatitis with marked MDSC infiltration. Treatment with the blood-activating herbal formula significantly reduced hepatic MDSC accumulation, suppressed pro-fibrotic cytokine levels, and ameliorated liver injury scores ($p < 0.05$ compared to untreated BS mice).

Conclusions:

This study is the first to link the TCM Blood-Stasis constitution to the expansion of immunosuppressive MDSCs in the liver, providing a novel immunologic

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mechanism for its association with aggravated MASLD. The correlation between traditional tongue signs and MDSC levels offers an objective bridging biomarker. Furthermore, the successful modulation of this axis by a targeted herbal intervention demonstrates a actionable pathway for precision therapy. This positions the BS constitution as a critical target for immunomodulatory strategies in MASLD management.

Keyword(s) : Blood-Stasis Constitution, Myeloid-Derived Suppressor Cells (MDSCs), MASLD, Immunosuppression, Tongue Diagnosis, Herbal Intervention

2276 - Long Term Outcomes of Treatment Induced Versus Spontaneously Achieved HBsAg

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Background / Hypothesis:

A serum hepatitis B surface antigen (HBsAg) level <100 IU/mL is recognized as a criterion for partial cure and as an indicator for consideration of antiviral therapy discontinuation in chronic hepatitis B (CHB). Patients who achieve HBsAg <100 IU/mL spontaneously have favorable clinical outcomes with a low risk of hepatocellular carcinoma (HCC). However, whether outcomes differ between patients who achieve HBsAg <100 IU/mL through antiviral treatment and those who achieve it spontaneously remains unclear. We compared the long-term outcomes of patients with treatment-induced versus spontaneously achieved HBsAg <100 IU/mL.

Materials and Methods:

This retrospective study included 886 non-cirrhotic CHB patients from three hospitals in China who achieved HBsAg levels <100 IU/mL, HBsAg negativity, undetectable HBV DNA, and normal ALT levels. Of these,

616 patients were classified in the spontaneous group and 270 were in the treatment-induced group. All patients in the treatment-induced group had received nucleos(t)ide analogue (NA) therapy for at least 6 months at enrollment and continued antiviral treatment during follow-up. The endpoints included HBsAg seroclearance and liver-related adverse events (LRE) (cirrhosis and HCC). Propensity score matching (PSM) was used to balance baseline characteristics between the treated and untreated groups.

Results:

During a median follow-up of 3.0 years, 106 patients achieved HBsAg seroclearance, and 7 patients developed liver-related adverse events. After PSM, there were no significant differences between the spontaneous and treatment-induced groups in the 6-year cumulative incidence of HBsAg seroclearance (19.5% vs. 37.6%, $P=0.270$) or LREs (2.3% vs. 2.1%, $P=0.646$). In the multivariate Cox analysis, baseline HBsAg <15 IU/mL (HR 4.47, $P<0.001$) was associated with a higher likelihood of achieving HBsAg seroclearance. However, antiviral treatment was not significantly associated with achieving HBsAg seroclearance (HR 1.81, $P=0.066$) and the development of LREs (HR 1.49, $P=0.630$).

Conclusions:

Both treatment-induced versus spontaneously achieved HBsAg <100 IU/mL had favorable prognosis with low risk of LREs and high chance of achieving HBsAg loss.

Keyword(s) : chronic hepatitis B, nucleos(t)ide analogue, hepatitis B surface antigen, clinical outcomes

2282 - SIRT2 restrains HDV associated hepatic macrophage pyroptosis by deacetylating NLRP3 and suppressing NF- κ B signaling

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Background / Hypothesis: Hepatitis D virus (HDV) superinfection causes aggressive hepatitis, yet the molecular drivers of HDV-induced liver injury are poorly defined. Inflammatory macrophage activation – especially NLRP3 inflammasome-mediated pyroptosis – is known to exacerbate liver damage. Sirtuin 2 (SIRT2) is an NAD⁺ dependent deacetylase that normally restrains inflammation – for example, by deacetylating NF- κ B p65

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and limiting proinflammatory gene expression, but its role in HDV pathogenesis has not been studied.

Materials and Methods: An AAV-HDV mouse model was established and SIRT2 activity was manipulated in vivo using AAV-SIRT2 (gain-of-function) or pharmacological SIRT2 inhibition (AGK2). Hepatic macrophages were isolated for transcriptomic profiling and mechanistic assays. Liver injury and inflammation were assessed by serum biochemistry and histopathology. NF- κ B signaling and pyroptosis were evaluated by immunoblotting/immunostaining and cytokine quantification, including p65 activation and NLRP3 inflammasome markers (NLRP3, cleaved caspase-1, GSDMD cleavage, IL-1 β /IL-18). Protein interaction and post-translational modification were examined by co-immunoprecipitation and acetylation analyses of NLRP3. Melatonin was administered as an intervention, with pathway readouts assessed in vivo and in HDV-stimulated macrophages.

Results: During HDV infection, SIRT2 was reduced in hepatic macrophages and its decrease correlated with heightened inflammatory responses and aggravated liver injury. Functionally, SIRT2 negatively regulated HDV-associated inflammation and liver damage, accompanied by attenuation of macrophage pyroptosis. Transcriptomic and protein-level analyses indicated that SIRT2 deficiency enhance p65 activation and increase NLRP3 abundance, whereas SIRT2 overexpression produced the opposite effects. Mechanistically, SIRT2 physically associated with NLRP3 and suppressed NLRP3 inflammasome activation by reducing NLRP3 acetylation, thereby limiting caspase-1 activation, GSDMD cleavage, and IL-1 β /IL-18 release. Finally, melatonin upregulated SIRT2, inhibited macrophage pyroptosis, and ameliorated HDV-induced hepatic inflammation and injury, supporting a druggable SIRT2-centered axis.

Conclusions: In summary, SIRT2 is a critical negative regulator of macrophage NF- κ B/NLRP3-mediated pyroptosis during HDV infection. By deacetylating both NLRP3 and NF- κ B p65, SIRT2 limits inflammasome activation and IL-1 β release, protecting hepatocytes from immune-mediated injury. Therapeutic strategies to enhance SIRT2 activity (e.g. melatonin supplementation) may thus mitigate HDV-associated hepatic inflammation.

Keyword(s) : HDV; hepatic macrophage; SIRT2; NF- κ B p65; NLRP3 inflammasome; acetylation; pyroptosis; melatonin

2290 - Coffee Consumption and Hepatocellular Carcinoma An Umbrella Review of Meta Analyses involving Over 12 Million Population

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Background / **Hypothesis:**
Coffee consumption has been extensively studied for its potential impact on hepatocellular carcinoma (HCC) risk, with conflicting results across various meta-analyses. This umbrella review aims to synthesize existing evidence and clarify the association between coffee consumption and the incidence of HCC, providing insights into potential preventive strategies.

Materials and **Methods:**
Following PRISMA guidelines, a systematic search of PubMed, Embase, Scopus, Cochrane Library, and Google Scholar was conducted up to May 2025. We included 12 meta-analyses comprising data from 12,399,664 participants. Pooled relative risks (RRs) with 95% confidence intervals (CIs) were calculated to evaluate the association. Heterogeneity was assessed using the I^2 statistic, and publication bias was examined using Egger's regression test and funnel plot.

Results:
Coffee consumption was associated with a significantly reduced risk of HCC (RR = 0.63; 95% CI: 0.56–0.69; I^2 = 92.25%; p < 0.01), suggesting a 37% lower risk among coffee drinkers. Although substantial heterogeneity was observed, the inverse association remained consistent across various meta-analyses. Subgroup findings from the included studies generally supported this protective effect regardless of population or study design. Egger's regression test indicated no significant publication bias (p = 0.2801), and the forest plot appeared symmetrical, reinforcing the reliability of the pooled estimate.

Conclusions:
This umbrella review consolidates current evidence supporting a substantial reduction in HCC risk associated with coffee consumption. The observed inverse association underscores the potential of coffee as a modifiable factor in HCC prevention strategies.

Keyword(s) : Coffee, Hepatocellular Carcinoma, Liver Oncology

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2291 - The Antagonism between Demographic Shifts and Epidemiological Progress A Decomposition Analysis of the Liver Cancer Burden in the Asia Pacific 1990–2023

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Background:

Despite substantial advances in antiviral therapies, the absolute burden of liver cancer in the Asia-Pacific remains considerable. The relative contributions of epidemiological risk reduction versus demographic forces—population aging and growth—vary markedly across the region. We aimed to disentangle these drivers and quantify how demographic transitions interact with shifting etiological profiles, particularly the transition from viral hepatitis to metabolic liver disease, using data from the Global Burden of Disease (GBD) Study 2023.

Materials and Methods: We analyzed liver cancer incidence, mortality, and disability-adjusted life-years (DALYs) from 1990 to 2023 across six GBD regions and 45 countries and territories in the Asia-Pacific. Five etiologies were examined: hepatitis B virus (HBV), hepatitis C virus (HCV), alcohol use, metabolic dysfunction-associated steatotic liver disease (MASLD), and other causes. Temporal trends in age-standardized rates (ASRs) were assessed using the Estimated Annual Percentage Change (EAPC). Changes in absolute case numbers were decomposed into population growth, population aging, and epidemiological change. Analyses were stratified by etiology.

Results:

From 1990 to 2023, the liver cancer burden in the Asia-Pacific increased substantially. Annual incident cases nearly doubled from 170,279 to 335,716, deaths rose to 293,682, and DALYs increased from 5.02 million to 7.99 million. Mongolia consistently exhibited the highest age-standardized burden. Etiology-specific trends diverged by region. High-income Asia Pacific (DALY EAPC: -2.44; 95% CI: -2.85 to -2.04) and East Asia (-1.79; -1.96 to -1.63) experienced significant declines in HBV-related ASRs, with pronounced reductions among females in East Asia (EAPC: -3.35), reflecting antiviral impact (Fig. 1).

However, population aging largely offset these gains, sustaining a high absolute burden. Conversely, South Asia showed the steepest increase in MASLD-related DALYs (EAPC: 0.97; 95% CI: 0.84 to 1.10), primarily driven by population growth, contributing 41.2% of the increase (Fig. 2). MASLD-related rates showed minimal risk reduction across regions, leading to a net rise in metabolic-related cases.

Conclusions:

Epidemiological gains from viral hepatitis control in the Asia-Pacific are increasingly counterbalanced by demographic momentum. A bifurcated strategy is required: East Asia should strengthen surveillance and geriatric-oriented care for aging viral-exposed cohorts, while South Asia urgently needs population-level interventions targeting metabolic risk factors to curb the rising MASLD burden.

Keyword(s) : Liver cancer; Decomposition analysis; Demographic transition; MASLD; Global Burden of Disease

2292 - Analysis of Clinical Characteristics and Treatment Outcomes in HBV Related Liver Failure Patients with or without Diabetes Mellitus

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Background :

Patients with hepatitis B virus (HBV) - related liver failure (LF) complicated by diabetes mellitus (DM) face more challenging treatment due to immune dysfunction and increased infection risk. This study aimed to compare the clinical characteristics and treatment differences between HBV - LF patients with and without DM.

Materials and Methods: A retrospective analysis was conducted on 91 HBV - LF inpatients admitted to the First Affiliated Hospital of the Army Medical University between April 20, 2017 to June 29, 2025. Patients were divided into two groups based on the presence of DM: the DM group (n = 45) and the non - DM group (n = 46). Demographic characteristics (age, gender), severity of liver disease (proportion of cirrhosis), virological indicators (proportion of HBeAg positivity, HBV DNA positivity), average length of stay, proportion of high - level antibiotic use (including β - lactamase inhibitors, carbapenems), and renal function (serum creatinine levels on admission and discharge) were compared between the two groups.

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Results:

All patients received nucleos(t)ide analog antiviral therapy and insulin for glycemic control, and all were discharged after improvement. There were no significant differences between the two groups in terms of age, gender, proportion of cirrhosis, and HBV DNA positivity (table 1). However, the proportion of high - level antibiotic use in the DM group was significantly higher than that in the non - DM group (86.6% vs 63.3%, $p < 0.05$) (figure 1). Although there was no statistically significant difference, the serum creatinine level in the DM group showed a trend of increase from admission to discharge ($84.1 \pm 70.8 \mu\text{mol/L}$ vs $84.1 \pm 70.8 \mu\text{mol/L}$, $P = 0.06$) (figure 1).

Conclusions:

HBV - LF patients with DM can achieve comparable clinical efficacy to non - DM patients through standardized antiviral and infection management. However, they have a higher infection risk and require more active use of high - level antibiotics and dynamic monitoring of renal function changes.

Keyword(s) : Hepatitis B virus; Liver failure; Diabetes mellitus; Antibiotic therapy; Renal function monitoring

2294 - Comparison of the Effects of Tenofovir Alafenamide and Entecavir on Lipid Profiles in Anti HBc Positive Kidney Transplant Recipients

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Background / **Hypothesis:**
Chronic Hepatitis B virus (HBV) infection in kidney transplant recipients represents a significant concern due to the risk of viral reactivation under immunosuppressive therapy. Therefore, antiviral prophylaxis is recommended for this patient population in clinical guidelines. Entecavir (ETV) and Tenofovir Alafenamide (TAF) are preferred in transplant recipients because of their potent antiviral efficacy and high genetic barrier to resistance. However, data regarding their long-term metabolic effects, particularly on lipid metabolism, remain limited.

In this study, we aimed to compare the effects of TAF and ETV therapies on lipid parameters in anti-HBc-positive kidney transplant recipients receiving antiviral prophylaxis to prevent HBV reactivation.

Materials and Methods:
A retrospective study of 62 anti-HBc-positive kidney transplant recipients on TAF or ETV; lipid and renal parameters were evaluated at baseline and during 2-year follow-up visits.

Results:

A total of 62 kidney transplant recipients were analyzed (27.4% male, 72.6% female; mean age 51.3 ± 12.0 years). Thirty-eight patients received TAF and 24 received ETV. The groups were demographically homogeneous ($p > 0.05$).

Findings in the TAF Group:
• The increase in total cholesterol between baseline and months 6, 12 and 24 was statistically significant (adjusted $p = 0.001, 0.037, 0.010$).

• HDL levels showed significant increases between baseline and months 6 and 24 ($p = 0.003, 0.030$).

• LDL levels exhibited a significant increase only between baseline and month 6 ($p = 0.021$). Additionally, the median GFR increased significantly from 16 to 64.5 ($p = 0.003$).

Findings in the ETV Group
Among the 12 patients included in the analysis, no statistically significant changes were observed in lipid parameters.

• Although time-dependent variations were noted in total cholesterol, HDL and LDL levels, Friedman test results did not reach statistical significance ($p > 0.05$).

• The median GFR increased significantly from 9 to 75 ($p = 0.000$) with a consistent significant rise throughout the follow-up period ($p < 0.001$).

Conclusions:

In this study, the effects of TAF and ETV therapies used to prevent HBV reactivation on lipid profiles were compared among anti-HBc-positive kidney transplant recipients. Our findings demonstrated that patients receiving TAF experienced significant increases in total cholesterol and LDL levels between baseline and month 6, whereas no notable changes were observed in these parameters in the ETV group.
• The most clinically relevant implication of our results is the necessity of lipid profile monitoring during TAF

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therapy. Kidney transplant recipients receiving TAF should undergo regular and long-term lipid monitoring and lifestyle modifications or lipid-lowering therapies should be considered when appropriate. Further prospective studies with larger sample sizes are warranted to evaluate long-term metabolic outcomes in this patient population.

• Limitations of this study include the relatively small sample size and retrospective design. Nevertheless, our findings contribute valuable insights to clinical practice, representing one of the few studies directly comparing the metabolic effects of TAF and ETV in the kidney transplant population.

Keyword(s) : Kidney Transplantation, Tenofovir Alafenamide (TAF), Entecavir (ETV), Lipid Metabolism

2296 - From Campaign to Validation Operationalizing Egypt's National Sustainability Plan for Viral Hepatitis Elimination (2025–2030)

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Background:

Following the historic "100 Million Seha" campaign aimed at hepatitis C virus (HCV) elimination, Egypt achieved the World Health Organization (WHO) "Gold Tier" status on the path to HCV elimination in 2023 and achieved the WHO Eastern Mediterranean Region (EMRO) targets for hepatitis B virus (HBV) control in 2025. To transition from vertical campaigning to permanent disease control, the Ministry of Health and Population developed the National Viral Hepatitis Elimination Sustainability Plan (2025–2030) with the support of the WHO. This study analyzes the operational framework designed to meet the WHO criteria for full validation of the elimination of hepatitis B and C viruses by 2030.

Methods:

We conducted a policy and systems analysis of the 2025–2030 Sustainability Plan, developed through multi-stakeholder consultations involving national committees, UN agencies, and civil society. The plan's five strategic objectives were mapped against WHO validation domains: governance, service integration,

prevention, care optimization, and surveillance. Financial sustainability was assessed using national cost-effectiveness modeling.

Results:

The framework operationalizes elimination through five key pillars:

1. Governance & Finance: Institutionalizing oversight via a National Viral Hepatitis Task Force and securing domestic financing, projected to save USD 4.7 billion in indirect costs by 2030.
2. Service Integration: Shifting testing and treatment into the Universal Health Insurance (UHI) benefits package and primary care family medicine curricula.
3. Prevention: Scaling harm reduction to reach 300 syringes/person/year for people who inject drugs (PWID) and maintaining at least 95% HBV birth-dose vaccination coverage, in addition to sustaining the national indicators of the blood and injection safety measures.
4. Care Optimization: Sustaining the focused efforts targeting the risky groups (e.g., PWID, ...) and deploying "One-Stop" clinics for integrated care.
5. Surveillance: Upgrading the National Electronic Disease Surveillance System (NEDSS) and expanding sequelae surveillance sites to monitor the documented reduction in HCV-related mortality (2018–2025).

Conclusions:

Egypt's roadmap demonstrates how high-burden nations can evolve from mass screening and treatment to sustainable, insurance-integrated elimination models. By prioritizing harm reduction and monitoring sequelae, this framework offers a transferable blueprint for Asia-Pacific nations seeking to operationalize WHO validation criteria within routine health systems.

Keyword(s) : Viral hepatitis elimination; WHO validation; public health policy; sustainability; universal health coverage

2297 - Ischemia Modified Albumin in MASLD Correlation With Ultrasonographic Steatosis Grade and Elastography Based Liver Stiffness

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Background/Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide, and reliable noninvasive tools are essential for disease assessment. Ultrasonography and transient elastography are widely used to evaluate hepatic steatosis and fibrosis. Ischemia-modified albumin (IMA), a marker of oxidative stress and altered albumin metal-binding capacity, has been investigated in various liver diseases, but its role in MASLD remains unclear. We hypothesized that IMA levels may be associated with ultrasonographic steatosis severity and elastography-based liver stiffness.

Materials and Methods: This prospective study included 81 adult patients diagnosed with MASLD. All patients underwent serum IMA measurement, routine laboratory testing, liver ultrasonography, and transient elastography (FibroScan®). Hepatic steatosis was graded on ultrasonography as Grade 1, 2, or 3 according to standard criteria; steatosis grading was available in 80 patients. Transient elastography included controlled attenuation parameter (CAP, dB/m) for steatosis and liver stiffness measurement (kPa) for fibrosis assessment. Laboratory parameters included liver enzymes, bilirubin levels, lipid profile, albumin, IMA/albumin ratio, hemoglobin, platelet count, INR, and C-reactive protein. Statistical analyses were performed using appropriate parametric or non-parametric tests based on data distribution, with $p < 0.05$ considered statistically significant.

Results:

The mean age was 47.35 ± 12.02 years, and 54.3% of patients were female. Ultrasonography demonstrated Grade 1 steatosis in 41.3%, Grade 2 in 47.5%, and Grade 3 in 11.3% of patients. Median IMA levels showed a decreasing trend with increasing steatosis severity but did not differ significantly among grades ($p = 0.255$). The IMA/albumin ratio was also similar between groups ($p = 0.126$). In contrast, AST and ALT levels increased significantly with higher steatosis grades ($p = 0.016$ and $p = 0.022$), while LDL cholesterol levels were significantly lower in Grade 3 steatosis ($p = 0.033$). CAP values increased progressively across steatosis grades and differed significantly among groups ($p < 0.001$). Liver stiffness values were higher in Grade 3 steatosis but did not reach statistical significance ($p = 0.115$).

Conclusions:

IMA levels were not associated with ultrasonographic steatosis severity or elastography-based liver stiffness in MASLD, suggesting limited value as a standalone marker. In contrast, CAP and selected biochemical markers showed clear associations with steatosis

severity, supporting the continued use of imaging-based noninvasive tools in MASLD evaluation.

Keyword(s) : MASLD, ischemia-modified albumin, ultrasonography, transient elastography, controlled attenuation parameter, liver stiffness

2300 - Old Drugs New Targets Repurposing Proton Pump Inhibitors for Autoimmune Liver Diseases Molecular Docking Insights into the TLR4 MD 2 NLRP3 and C5aR1 Axes

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Background/Hypothesis: Autoimmune liver diseases (AILDs) are characterized by loss of immune tolerance and chronic necroinflammatory injury. Current therapies involving corticosteroids and immunosuppressants are limited by severe side effects and non-response rates, necessitating safer, targeted strategies. Drug repurposing offers a rational alternative to de novo drug design. Given the emerging evidence of the pleiotropic immunomodulatory effects of Proton Pump Inhibitors (PPIs), this study aims to elucidate the molecular interaction profiles of PPIs on key hepatic inflammatory targets - Toll-like receptor 4/MD-2 (TLR4/MD-2), NOD-like receptor protein 3 (NLRP3) inflammasome, and Complement C5a receptor 1 (C5aR1) - to provide a structural basis for their repurposing in AILDs.

Materials and Methods: Five PPIs (esomeprazole, omeprazole, pantoprazole, lansoprazole, rabeprazole) were analyzed against crystallographic structures of the TLR4/MD-2 complex (PDB: 3FXI), NLRP3 inflammasome (active: 7VTP; inhibitor-bound: 7PZC), and C5aR1 receptor (allosteric/extra-helical: 6C1R, 5O9H). Molecular docking calculations were performed using Schrödinger Maestro (Glide module). Binding affinities (docking scores), energetic components, and specific ligand-residue interactions were evaluated to assess potential inhibitory mechanisms.

Results: Rabeprazole and Lansoprazole exhibited superior binding affinities across all inflammatory axes compared to other PPIs. Notably, in the inhibitor-bound NLRP3 structure (7PZC), Rabeprazole (-9.10 kcal/mol) and Lansoprazole (-9.04 kcal/mol) demonstrated high-affinity binding, suggesting potent inflammasome

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inhibition. While Rabeprazole showed the highest affinity for the TLR4/MD-2 complex (-7.85 kcal/mol), a distinct "lead switching" was observed in the C5aR1 axis, where Lansoprazole emerged as the lead compound (-6.93 kcal/mol), effectively targeting the allosteric pocket. Conversely, Pantoprazole displayed the weakest interaction profile across all targets.

Conclusions: Although Pantoprazole exhibited lower binding affinity, this study presents novel molecular evidence that PPIs, particularly Rabeprazole and Lansoprazole, may exert dual anti-inflammatory and immunomodulatory activities in autoimmune liver diseases by directly targeting the TLR4, NLRP3, and C5aR1 signaling pathways. Our findings establish a strong foundation for evaluating this drug class as novel therapeutic agents through a "structural repurposing" strategy.

Keyword(s) : Drug Repurposing, Autoimmune Liver Diseases, Molecular Docking, NLRP3 Inflammasome, TLR4/MD-2 Complex, C5aR1

2302 - Stiffness Regression Fundamentally Alters Mortality Patterns and Improves Survival in Advanced Fibrosis After SVR A Cause Specific Outcome Analysis

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Background:

Stiffness regression after sustained virologic response (SVR) reflects structural hepatic improvement, but its impact on survival and cause-specific mortality across fibrosis stages remains unclear. This study evaluated how regression modifies mortality patterns and outcomes in F3 and F4 disease.

Methods:

Longitudinal liver stiffness measurements (LSM) were assessed by estimating individual decay rates; a $\geq 20\%$ LSM reduction defined regression. Overall survival (OS) was prospectively evaluated (mean follow-up 7 years). Outcomes were analyzed separately for F3 and F4. Detailed causes of death were reviewed, and liver-related mortality was compared using Fisher's exact test.

Results:

In F3 (n=134), survival did not differ between the regression (4/87) and non-regression (2/47) groups. Deaths in the regression group were largely non-liver-related (two extrahepatic malignancies, one pneumonia), with only one HCC-related death. In the non-regression group, deaths were due to HCC and heart failure. Liver-related mortality was rare, explaining the absence of survival separation.

In F4 (n=195), regression markedly altered mortality patterns. Among 11 deaths in the regression group (11/107; 10%), only 4 (36%) were liver-related (HCC=2, liver failure=2), while 7 were non-liver-related (extrahepatic malignancy=3, pneumonia, subarachnoid hemorrhage, senescence, graft rejection). In contrast, the non-regression group experienced 19 deaths (19/88; 22%), including 14 liver-related (74%)—HCC (n=12) and liver failure (n=2) (p=0.0626). Other causes included extrahepatic malignancy=2, pneumonia, heart failure, trauma, and senescence. The survival advantage in F4 regression patients reflects a substantial reduction in liver-related mortality.

Conclusions:

In advanced fibrosis, stiffness regression fundamentally shifts mortality patterns and is a major determinant of long-term survival after SVR.

Keyword(s) : Hepatitis C, SVR, Fibrosis, Regression, OS

2306 - Study on Key Inflammatory Proteins of Antiviral Treatment in Children with Chronic Hepatitis B

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Background

Previously study showed that inflammatory proteins play a pivotal role in viral replication and the host immune response. The purpose of this study is to identify which inflammatory protein plays a key role in antiviral therapy in children with chronic hepatitis B (CHB).

Materials

100 children with CHB were randomly recruited into the

Methods:

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study cohort. The participants were stratified based on the initial age and therapeutic efficacy. Using Olink proteomics technology, we detected and analyzed 92 inflammatory proteins in the baseline serum samples of all subjects. 67 patients randomly selected were assigned to the test cohort, while the others constituted the validation cohort. The area under the receiver operating characteristic (AUROC) curve was calculated to construct and evaluate the performance of the prediction model.

Results:

In the test cohort, HBsAg loss rate was 58.0% in children under 3 years old, while it was 47% in children above 3 years old group. Logistic regression showed that SLAMF1 was an independent factor associated with favorable antiviral outcomes in children under 3 years. Additionally, CXCL9 and CCL4 were significantly correlated with HBsAg loss in CHB children aged 1-6 years. The combined model incorporating SLAMF1, CXCL9, and CCL4 yielded the best AUROC of 0.80(95% CI: 0.71-0.967, $p=0.0006$) for predicting HBsAg clearance under 3 years. Similar favorable results were observed in the validation cohort.

Conclusions:

Our findings indicate that SLAMF1 may be a key inflammatory protein playing a critical role in antiviral therapy for children with CHB, which provides an experimental basis for improving their functional cure rate.

Keyword(s) : Chronic hepatitis B; Children; Antiviral therapy; Inflammatory proteins.

2309 - Zonal Restoration and Inter Cellular Signaling in Fibrosis Regression Single Cell Transcriptomics Decodes the Pericentral Hepatocyte HSC Axis

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Background

Single-Cell Fixed RNA Profiling (FLEX) is a novel technology that enables RNA expression profiling from

Hypothesis:

frozen tissues at single-cell resolution. We applied FLEX to a murine model of cirrhosis progression and regression to identify previously unrecognized cellular crosstalk and potential anti-fibrotic targets.

Materials and Methods: Liver fibrosis was induced by intraperitoneal thioacetamide (TAA) administration for 10 weeks, and regression was assessed two weeks after cessation. Livers were fixed, dissociated, and analyzed using FLEX. Molecular validation was performed by immunoblotting, immunohistochemistry, gene silencing and overexpression assays, cytokine/phosphokinase arrays, and analyses of human liver samples.

Results: Approximately 40,000 liver cells were profiled, integrated, and annotated into 10 major cell types using lineage-specific markers. Restoration of pericentral signatures in hepatocytes (HEPs) was the predominant event associated with fibrosis regression. A distinct HEP subcluster, termed recoHEPs, characterized by high expression of Cyp2e1, Cyp1a2, Glul, Oat, Mgst1, and Txn1, exhibited xenobiotic-, metabolic-, and antioxidant-related transcriptional programs. Gene set enrichment analysis (GSEA) and differential expression analysis (DEGs) demonstrated increased activation of central metabolic pathways, including xenobiotic metabolism and antioxidant signaling, in recoHEPs. Immunohistochemistry for pericentral hepatocyte markers (GLUL, CYP2E1, CYP1A2, OAT) confirmed Zone 3 localization, which was diminished during fibrosis progression and restored during regression, whereas Zone 1 markers (CYP2F2, HAL) increased in fibrosis and declined during regression. MultiNicheNet analysis predicted the upregulation of the top 50 ligand-receptor pairs during regression, with Rarres2-Cclrl2 emerging as a prominent axis mediating signals from HEPs to HSCs and among HSCs themselves. Spatial transcriptomics of published human liver datasets showed that RARRES2 is expressed in human pericentral hepatocytes as well as HSCs. Validation experiments demonstrated that RARRES2 expression is high in healthy liver, reduced in fibrosis, and restored during regression in both tissue lysates and serum. In primary mouse hepatic stellate cells (mHSCs), Rarres2 expression was highest in quiescent HSCs and significantly decreased during activation. Functional assays further showed that RARRES2 knockdown using siRNAs in primary human hepatic stellate cells (HHStCs) increased the expression of fibrosis-related genes (COL1A1, COL1A2, COL5A1, FN1, LMCD1, ACTA2, MYL9).

Conclusions: These findings reveal a critical role for pericentral hepatocytes in orchestrating liver fibrosis

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regression through interactions with HSCs. Rarres2 represents a promising therapeutic target for promoting fibrosis resolution.

Keyword(s) :

2310 - NAFLD promotes early hepatocellular carcinoma recurrence via MDSC metabolic reprogramming Insights from a two phase recurrence model in mice

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Background / Hypothesis: Surgical resection remains an important curative therapy for hepatocellular carcinoma (HCC); however, the 5-year recurrence rate still exceeds 70%. The majority of recurrences manifest within the initial 2 years post-surgery, in accordance with the early recurrence criteria. Nonalcoholic fatty liver disease (NAFLD) has been reported to promote early recurrence of HCC. Here, we investigated the role of NAFLD in shaping an immunosuppressive tumor microenvironment after surgical resection, which promotes the early recurrence of HCC.

Materials and Methods: Models of early recurrence were established with luciferase-expressing Hepa1-6 cells in C57BL/6J mice. A randomized, controlled animal experiment was conducted. Single-cell RNA sequencing (scRNA-seq) was performed to examine the immune microenvironment in the liver in the context of NAFLD. Multiplex immunofluorescence staining and flow cytometry were used to validate immune cell population dynamics. Lipidomics was used to delineate the metabolic landscape of NAFLD.

Results: We discovered that the NAFLD microenvironment promoted the expansion of CD11b+Gr1+ myeloid-derived suppressor cells (MDSCs), which concurrently promoted the exhaustion of tumor-specific CD8⁺ T cells. This expansion was positively correlated with the early recurrence of HCC. Furthermore, lipidomic analysis of tumor tissues from NAFLD mice revealed 22 upregulated metabolites. Pathway enrichment analysis revealed a predominant association between these metabolites and glycerophospholipid metabolism. Infiltrating MDSCs enhanced choline metabolism through the significant upregulation of the key rate-limiting enzyme choline kinase alpha (CHKA), consequently activating the PI3K-AKT-mTOR axis. Interrupting the CHKA-PI3K-AKT-

mTOR axis with CHKA inhibitor MN58b or AKT inhibitor capivasertib significantly mitigated tumor progression induced by NAFLD and restored CD8⁺ T cell function. Intercellular interaction analysis suggested that MDSCs reciprocally communicate with T/NK cells to remodel unfavorable conditions via the CEACAM1-CD8a, CD86-CTLA4/CD28, and IL34-CSF1R tumor-promoting signaling pathways.

Conclusions: NAFLD drives early HCC recurrence by remodeling the immunosuppressive niche via the CHKA-PI3K-AKT-mTOR axis. Targeting this pathway represents a novel therapeutic strategy against NAFLD-related HCC recurrence.

Keyword(s) : Hepatocellular carcinoma; Nonalcoholic fatty liver disease; Early recurrence; Myeloid-derived suppressor cells; Choline catabolism

2311 - LOXL1 Regulates Lipid Metabolism to Promote Hepatic Stellate Cell Activation via Ubiquitin Mediated FASN Degradation

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Background / Hypothesis:

Activation of hepatic stellate cells (HSCs) is a central event in liver fibrosis and is accompanied by a marked loss of intracellular lipid droplets. Our previous work showed that deletion of lysyl oxidase-like 1 (LOXL1) in HSCs attenuates stellate cell activation; however, the underlying mechanism remains unclear. Given our observation that LOXL1 deficiency leads to abnormal lipid metabolism in HSCs, we aimed to investigate a novel metabolic mechanism by which LOXL1 regulates HSC activation during liver fibrosis.

Materials and Methods:

LratCreLoxl1^{fl/fl} mice were subjected to CCl₄-induced liver fibrosis. Primary HSCs were isolated for RNA sequencing to delineate metabolic pathway alterations associated with LOXL1 deficiency, and quantitative PCR was performed to analyze lipid metabolism-related gene expression. In vitro, primary HSCs were treated with β -aminopropionitrile (BAPN) to examine intracellular lipid droplet accumulation together with total cholesterol (TC) and triglyceride (TG) levels. Immunoprecipitation followed by mass spectrometry

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(IP-MS) was used to identify LOXL1-interacting proteins. LOXL1 was overexpressed in LX-2 cells to evaluate its effect on fatty acid synthase (FASN) expression. MG132 and cycloheximide (CHX) treatments were applied to assess FASN protein stability, and FASN ubiquitination was analyzed by co-immunoprecipitation.

Results:

In CCl₄-induced fibrotic mice, primary HSCs from LratCreLoxl1^{fl/fl} mice showed significantly reduced expression of fibrogenic genes. RNA-seq identified lipid metabolism as a prominently enriched pathway. Expression of Ppara, Acc, Scd1, and Scd2 increased by 56%, 106%, 134%, and 112%, respectively, with a concomitant increase in intracellular TG levels. In vitro, inhibition of HSC activation was associated with increased lipid droplet accumulation, as shown by BODIPY staining, along with elevated TC and TG contents (89% and 66% increases, respectively). IP-MS identified FASN as the most abundant lipid-related protein interacting with LOXL1. LOXL1 overexpression did not alter FASN mRNA levels but reduced FASN protein abundance by approximately 2-fold. MG132 treatment restored FASN protein levels, while CHX chase assays demonstrated time-dependent accumulation of LOXL1-associated FASN protein. Co-immunoprecipitation confirmed enhanced ubiquitination of FASN in LOXL1-overexpressing LX-2 cells.

Conclusions:

Hepatic stellate cell-derived LOXL1 suppresses stellate cell activation by preserving intracellular lipid metabolic homeostasis. Mechanistically, LOXL1 directly interacts with FASN and promotes its ubiquitin-mediated proteasomal degradation, thereby linking lipid metabolic regulation to HSC activation during liver fibrosis.

Keyword(s) : Liver fibrosis; Hepatic stellate cell; LOXL1; Lipid metabolism;FASN

2314 - Gilbert syndrome increases the risk of gallstone disease a cross sectional study based on health examination data from 338 372 adults

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Background / Hypothesis:Gilbert syndrome (GS) is characterized by mild unconjugated hyperbilirubinemia and may be associated with an increased prevalence of gallstone disease. This study aimed to investigate the relationship between bilirubin levels and gallstone risk.

Materials and Methods:This retrospective study collected data from 338,372 adults at the Beijing Physical Examination Center between 2015 and 2024. Participants were categorized based on GS diagnostic criteria (at least two examinations over 6-month intervals meeting: $1 \times \text{ULN} < \text{total bilirubin [TB]} < 102.6 \mu\text{mol/L}$, direct bilirubin [DB]/TB < 0.5 , without elevated alanine aminotransferase [ALT] or aspartate aminotransferase [AST], and excluding organic hepatobiliary diseases and hemolysis). The relationships between GS status, TB quartiles, and gallstone prevalence at the last examination were analyzed using multivariable logistic regression. Restricted cubic splines were applied to explore the dose-response relationship between TB levels and gallstones.

Results:The prevalence of gallstones was significantly higher in the GS group than in the non-GS group (8.44% vs. 5.66%, $P < 0.001$). After adjusting for age, sex, and body mass index (BMI), both GS status and the highest TB quartile (Q4) were independent risk factors for gallstones (GS: adjusted odds ratio [OR]=1.20, 95% confidence interval [CI]: 1.11–1.30; TB Q4: adjusted OR=1.16, 95% CI: 1.10–1.23). Sex-stratified analysis using restricted cubic splines revealed a linear dose-response relationship in males (Q4 vs. Q1 OR=1.26, P for trend < 0.001 , P for nonlinear=0.187), while a nonlinear association was observed in females (P for nonlinear < 0.001 , P for trend=0.063), with gallstone risk primarily driven by age and BMI.

Conclusions:Gilbert syndrome and elevated TB levels are associated with gallstone disease. The association between bilirubin and gallstones exhibits significant sexual dimorphism: linear and incremental in males, but nonlinear and largely explained by age and BMI in females.

Keyword(s) : Gilbert syndrome, bilirubin, gallstone disease, cross-sectional study

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2318 - Gut Microbiota Transfer and Integrated Omics Identify Streptococcal Collagen Like (Scl1) Protein as a key fibrogenic driver in Alcohol associated liver disease.

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Background / Hypothesis: Alcohol modulated gut microbiota (AGMs) contributes to liver injury. we aimed to investigate how AGMs alters hepatic kinome signaling and mediate inflammation, fibrosis in alcohol-associated liver disease (ALD).

Materials and Methods: Liver kinome and metaproteome changes were studied in rats (n=6/group) colonized with stool of severe alcohol-related hepatitis patients (SAH→healthy-rats) or healthy human-donors (HD→ALD-rats). AGM-associated liver kinome changes were cross-correlated with bacterial genera. A bacterial protein; Streptococcal collagen-like protein 1 (Scl1) was identified with affinity for TGFβR1. Streptococcus pneumonia and pyogenes were screened in stool of SAH patients (n=10). Scl1 was screened in patient stool (n=24) and liver tissue (n=19) samples. Scl1 and streptococcus levels were checked pre and post FMT

Results: Stool metaproteomics showed significant increase in 10 bacterial genera (Streptococcus, Staphylococcus, and Clostridium) in SAH→Healthy-rats, mirroring changes seen in ALD-rats (FC>1.5, p<0.05). Liver kinome profiling showed 85 upregulated kinases in SAH→Healthy-rats, with 34 overlapping with ALD-rats, associated with inflammation (Mapk14, Map3k10 and others), fibrosis (Tgfb1, Igf1r, and others), lipid metabolism (Cdk14, Cdk18) and regeneration (Met, Bmpr1a and others). FMT from healthy human-donors to ALD-rats reversed the expression of Streptococcus (10-fold), Staphylococcus (3-fold), and Clostridium (5-fold), along with 18 kinases associated with inflammation (Btk, Camk4, Nuak1) and fibrosis (Tgfb1, Col4a1, Fgfr2 and others). Strong association ($r^2>0.9$, p<0.05) was seen between Streptococcus abundance

and fibrosis. Level of Scl1 was significantly high in SAH and ALD-rat stool and liver samples (FC>2, p<0.05). It showed strong affinity for TGFβR1, as validated by molecular docking (>86% confidence) and immunoprecipitation assays (>100FC, p<0.05), indicating it as a potential ligand for TGFβR1. Concordantly, expression of Scl1 was highest amongst all the known ligands for TGFβR1 (p<0.05) suggesting Scl1 is a major contributor for TGFβR1 activation and its downstream signalling in these patients. Following fecal microbiota transplantation, expression of Scl1 in plasma and Streptococcus levels in stool were significantly reduced by 2.1-folds and 1.9 folds respectively in SAH patients. Further, Scl1 levels SAH plasma is capable of predicting poor therapeutic response and 30-day mortality with high accuracy (AUC=0.92, cutoff >70 normalized abundance).

Conclusion: Alcohol induced gut dysbiosis alters the liver kinome, promoting fibrosis. Streptococcal Scl1 is identified as a bacterial protein mimicking human collagen and capable of activating fibrotic signalling pathway through TGFβR1 in liver and is capable of predicting poor response in SAH. FMT from healthy donor restores microbial balance and mitigates harmful host-microbe interactions.

Keyword(s) : Kinases, Alcohol-related Liver Disease, Alcohol associated gut microbiota, Scl protein, Streptococcus, Fecal microbiota transplant

2320 - Liver meta proteome and kinome reveal Pseudomonas aeruginosa –pPKCη axis as a driver of steroid resistance in severe alcohol related hepatitis

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Background / Hypothesis: Severe alcohol-related hepatitis (SAH) is a critical condition where corticosteroids remain the primary therapy, but with limited efficacy (~40%) in many patients. The molecular mechanisms underlying steroid non-response are not fully elucidated. This study investigated these mechanisms using global phosphoproteomics and metaproteomic profiling of liver biopsies, focusing on microbial modulation of glucocorticoid signalling.

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Materials and Methods: Liver biopsies from 19 SAH patients (10-responders, 9-non-responders) on prednisolone therapy (cohort-1) were analyzed via phosphoproteomics and metaproteomics. Findings were validated in 20 patients (cohort-2) via IHC and in 32 patients (cohort-3) using RNA-seq. Pathway and kinase enrichment analyses were conducted to identify molecular drivers of steroid resistance. A key glucocorticoid-modulating kinase was functionally validated in vitro and in vivo.

Results: Metaproteomic analysis identified 9,080 bacterial peptides from 257 species. Non-responders had significantly reduced alpha diversity (Shannon index, $p < 0.01$) and enrichment of *Streptococcus pneumoniae*, *Staphylococcus aureus*, *Mycoplasma pneumoniae*, and *Pseudomonas aeruginosa* ($p < 0.05$). Phosphoproteomics revealed 11,172 phosphoproteins and 372 kinases ($p < 0.05$). Non-responders exhibited significant up-regulation of TH17 signalling, metabolic stress, CREB phosphorylation, JAK-STAT signalling, and glucocorticoid receptor (GR) inhibitors ($p < 0.05$), with down-regulation of HSP90 activity, DUSP3, ERK inhibition, and ECM maintenance ($p < 0.05$). Kinome profiling showed increased activity of CDK7, PRKCH, PRKAA1, and MINK1. PRKCH was the most differentially activated kinase ($p < 0.001$), correlating with *P. aeruginosa* abundance ($r^2 > 0.7, p < 0.01$) already known to induce PRKCH. Plasma pyocyanin levels ($r^2 > 0.7, p < 0.01$) were elevated in non-responders and enzymes involved in pyocyanin production such as PhZS were significantly increased in non-responder's liver. PRKCH inhibition significantly restored prednisolone sensitivity in vitro ($p < 0.01$). In the NIAAA binge model, staurosporine plus prednisolone reduced inflammatory and metabolic pathways more significantly than prednisolone alone documenting benefit of combination therapy ($p < 0.01$).

Conclusions: Bacterial dysbiosis and PRKCH-driven kinase signalling mediate steroid resistance in SAH. Targeting PRKCH restores steroid sensitivity and enhances anti-inflammatory activity this offers a new therapeutic strategy for steroid resistance.

Keyword(s) : Phosphoproteomics, Alcohol-related Liver

Disease, Kinases, prednisolone, NR3C1, phosphorylation, non-response, staurosporine, PRKCH.

2326 - Asia Pacific expert consensus on GAAD for hepatocellular carcinoma surveillance

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Background/Hypothesis

Hepatocellular carcinoma (HCC) presents a critical global health challenge, with the Asia-Pacific region accounting approximately 72% of the disease burden. Current surveillance standards, primarily ultrasound (US) with or without alpha-fetoprotein (AFP), are limited by suboptimal sensitivity especially for early-stage disease and operator dependence with ultrasound. The biomarker-based GAAD algorithm—incorporating Gender, Age, AFP, and PIVKA-II—has emerged as a standardized surveillance option. This expert consensus aimed to synthesize evidence regarding the diagnostic performance and cost-effectiveness of GAAD to provide

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expert recommendations tailored for clinical practice in the region.

Materials & Methods
A panel of 15 liver cancer experts conducted a literature review focusing on the clinical and economic utility of GAAD. Twelve preliminary statements were drafted and subsequently refined through two consensus meetings held in Beijing and virtually. The panel evaluated statements using a modified Delphi approach with a five-point Likert scale. Consensus was defined as $\geq 75\%$ of respondents selecting "accept completely" or "accept with minor reservations," and evidence was graded according to the 2011 Oxford Centre for Evidence-Based Medicine Levels of Evidence.

Results

The expert panel achieved 100% consensus on 11 final statements, endorsing GAAD as a validated tool for HCC surveillance.

Diagnostic Accuracy: In validation cohorts, GAAD demonstrated high diagnostic accuracy for differentiating early-stage HCC from benign chronic liver disease, achieving a sensitivity of up to 72%, specificity of approximately 90%, and an area under the curve (AUC) of up to 90.7%. Its performance was consistent across diverse regions and viral etiologies, comparable to the more complex GALAD score (AUC 79–86%) without requiring AFP-L3.

Combination Strategy: Combining GAAD with US significantly enhanced surveillance sensitivity. In comparative studies, GAAD plus US increased early-stage detection rates to 69.7%, compared to 57.6% for GAAD alone and significantly outperforming US alone or US plus AFP.

Economic Utility: Health economic modeling identified GAAD as economically advantageous due to its simpler biomarker composition. In high-risk populations, GAAD plus US is a cost-effective strategy, whereas GAAD alone serves as a dominant, cost-efficient strategy in resource-limited settings where US expertise is scarce.

Clinical Application: While GAAD offers a scalable solution for resource-limited settings, the panel noted that results must be interpreted alongside clinical context, specifically screening for confounders such as warfarin use or vitamin K deficiency which affect PIVKA-II levels.

Conclusions

The Asia-Pacific consensus endorses GAAD as a validated, cost-efficient surveillance tool that addresses current diagnostic gaps. Particularly effective for high-risk populations when combined with US and serves as

a viable standalone option in HCC early detection.

Keyword(s) : HCC; GAAD; GALAD; Surveillance; Liver cancer; APAC; Consensus

2328 - Fatty Liver as a Key Risk Modifier for Adenoma Detection in FIT Positive Young Adults A Systematic Review of the Available Evidence

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Background / Hypothesis:
Early-onset colorectal neoplasia is increasing, and risk stratification in younger adults (<50 years) remains poorly defined. Fatty liver disease—now highly prevalent in younger populations—has emerged as a potential synergistic risk factor for colorectal adenomas, particularly among individuals with positive fecal immunochemical test (FIT) results.

Materials and Methods:
A systematic search identified studies evaluating the association between FIT status, fatty liver, and adenoma detection in adults younger than 50. Only one eligible study was found and included for narrative synthesis.

Results:

In a retrospective cohort of 3,307 adults aged 30–49 years who underwent FIT and colonoscopy, individuals with both fatty liver and a positive FIT result demonstrated significantly higher adenoma and advanced adenoma detection rates compared with those without fatty liver. Fatty liver conferred an adjusted odds ratio of ~2.69 for adenoma detection, independent of metabolic syndrome and other covariates. FIT-positive individuals with fatty liver represented the highest-risk subgroup for clinically significant neoplasia.

Conclusions:

Evidence—though limited to a single study—suggests that fatty liver substantially amplifies the neoplastic yield of colonoscopy among FIT-positive young adults. Fatty liver may serve as an important triage factor for early colonoscopic evaluation in younger populations.

Keyword(s) :

2329 - Multi omics analyses identify a novel hepatocellular carcinoma subtype with better response to immune checkpoint inhibitor therapies

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Background / Hypothesis: Hepatocellular carcinoma (HCC) present persistent therapeutic challenges due to molecular heterogeneity and unpredictable immune checkpoint inhibitor (ICI) responses.

Materials and Methods: In this study, whole transcriptome sequencing was performed on tumor and adjacent non-tumor tissues across 242 patients with HCC and 51 patients with intrahepatic cholangiocarcinoma (ICC). Whole exome sequencing was performed in selected samples of each cancer subtype. Single-cell RNA sequencing was performed on fresh primary tumor samples from 15 HCC patients and 8 ICC patients. Whole genome sequencing was performed on tumor and adjacent non-tumor tissues across 90 HCC patients.

Results: Through integrative multi-omics analyses, we identified a novel ICI-sensitive HCC subtype (IS-HCC) characterized by a pre-dysfunctional microenvironment and distinct genomic drivers (TP53, CBX3, FBN2, etc.), exhibiting aggressive baseline features but achieved favorable response to ICI therapies. While bulk transcriptomic profiles revealed partial similarity with intrahepatic cholangiocarcinoma (ICC), single-cell RNA sequencing and spatial proteomic resolutions uncovered unique immune dynamics in IS-HCC, marked by enriched transitional CD8⁺ T cells co-expressing exhaustion (PD1, TIM-3) and activation (GZMB, IFNG) markers. By validation in a prospective ICI-treated HCC cohort (n=90), this pre-dysfunctional T cell state, rather than tumor mutational burden, predicted superior ICI response (OR=4.595, 95%CI: 1.725 to 12.022), challenging neoantigen-centric paradigms. Furthermore, whole-genome data suggested that somatic mutations may influence intratumoral metabolic reprogramming in IS-HCC, particularly in pathways related to glycometabolism and hypoxia, potentially driving the formation of a functionally pre-dysfunctional immune microenvironment through tumor-associated macrophage induction, suggesting metabolic-immune crosstalk via glycometabolism and

hypoxia pathways might be actionable targets. Furthermore, we developed and validated a 29-gene signature across major global HCC cohorts that stratified IS-HCC patients. Clinically, this biomarker-driven framework transcends current selection paradigms and addresses a critical unmet need by enabling precise ICI deployment selection.

Conclusions: Our findings bridge molecular discovery to clinical translation, with potential to double ICI efficacy in stratified populations and spare non-responders from futile therapies, advancing precision immuno-oncology in liver cancer.

Keyword(s) : Hepatocellular carcinoma (HCC); Immune checkpoint inhibitors (ICIs); Pre-dysfunctional CD8⁺ T cells; Biomarker-driven stratification; Immunotherapy resistance

2336 - Lean MAFLD in Chronic Hepatitis B Clinical Characteristics Fibrosis Risk and antiviral response in a Pakistani Cohort
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Background / Hypothesis: The coexistence of metabolic dysfunction-associated fatty liver disease (MAFLD) in chronic hepatitis B (CHB) is increasingly recognized and both are highly prevalent in Pakistan. However, the burden and clinical impact of lean MAFLD in CHB patients remain poorly defined, particularly in Pakistan, where metabolic risk factors are prevalent despite normal body mass index (BMI). Aim of this study is To determine the prevalence, clinical characteristics, fibrosis risk associated with lean MAFLD in patients with chronic hepatitis B.
Materials and Methods: This observational study included adult patients with CHB presenting to a tertiary-care hepatology center in Pakistan between 2014 and 2024. MAFLD was diagnosed based on imaging evidence of hepatic steatosis with metabolic risk factors. Lean MAFLD was defined as BMI <25 kg/m². Liver fibrosis was assessed using transient elastography and the FIB-4 score. Statistical analysis was performed using SPSS version 26. Logistic regression was used to identify predictors of significant fibrosis (≥F2).
Results:

A total of 1080 patients with CHB were included;454 (42.1%) had coexisting MAFLD. Among MAFLD patients, 175 (38.6%) met criteria for lean MAFLD. Lean MAFLD patients had higher rates of insulin resistance (HOMA-IR ≥2.5: 61.8% vs 34.2%, p<0.01) and dyslipidemia

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compared with non-MAFLD patients. Median liver stiffness was significantly higher in lean MAFLD patients (8.2 vs 6.1 kPa, $p=0.003$). Complete virological response at 12 months was lower in lean MAFLD patients (74.6%) compared with non MAFLD patients (86.9%, $p=0.01$). ALT normalization was also less frequent (62.3% vs 81.4%, $p<0.01$). On multivariable logistic regression analysis, lean MAFLD independently predicted significant fibrosis (adjusted OR 2.04, 95% CI 1.21–3.46; $p=0.007$) after adjustment for age, sex, ALT, and HBV DNA levels and incomplete virological response (adjusted OR 1.89, 95% CI 1.03–3.45; $p=0.04$).

Conclusions:

Lean MAFLD is a common and clinically important phenotype among patients with chronic hepatitis B in Pakistan and is independently associated with increased fibrosis risk and suboptimal antiviral response. Normal BMI does not exclude metabolic liver disease in CHB. Routine metabolic screening should be incorporated into hepatitis B management strategies.

Keyword(s) :

2340 - Metabolic Fatty Liver Masks Hepatitis B Activity Implications for ALT Based Treatment Decisions a study from PAKISTAN

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Background & Aim: Metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly recognized in patients with chronic hepatitis B (CHB). Elevated alanine aminotransferase (ALT) is a key marker for initiating antiviral therapy in CHB. However, the presence of MAFLD may independently elevate ALT, potentially masking underlying HBV activity and complicating treatment decisions. This study aimed to evaluate the impact of MAFLD on ALT interpretation and antiviral treatment eligibility in CHB patients.

Methods:

We conducted a cross-sectional analysis of 500 CHB patients. Clinical, biochemical, and imaging data were collected. MAFLD was defined based on the presence of hepatic steatosis plus metabolic risk factors. Patients were categorized by ALT levels and HBV DNA activity. Multivariable logistic regression was used to assess the

independent association of MAFLD with ALT elevation, adjusting for HBV viral load and other confounders.

Results:

Among 500 CHB patients, 200 (40%) had coexisting MAFLD. Patients with MAFLD had significantly higher ALT levels compared to those without MAFLD (median ALT: 48 vs. 32 U/L, $p<0.001$), independent of HBV DNA levels. Notably, 35% of patients with MAFLD and low HBV replication had ALT above the conventional treatment threshold, potentially leading to unnecessary antiviral therapy. In multivariable analysis, MAFLD independently predicted ALT elevation (OR 2.1, 95% CI 1.4–3.2, $p<0.001$).

Conclusion:

MAFLD can independently elevate ALT in CHB patients, masking the true viral activity and potentially leading to misclassification for antiviral treatment. Clinicians should consider metabolic fatty liver when interpreting ALT and assessing treatment eligibility, highlighting the need for HBV DNA-guided treatment strategies rather than ALT-based decisions alone.

Keyword(s) :

2347 - Semaglutide demonstrates efficacy in improving liver fibrosis across diverse subpopulations in part 1 of the ESSENCE trial – outcomes in Asian and non Asian populations

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Background / Hypothesis: Part 1 of the ESSENCE trial in participants with metabolic dysfunction-associated steatohepatitis (MASH) and stage 2/3 fibrosis (F2/F3) showed positive effects of once-weekly subcutaneous semaglutide 2.4 mg on liver histology and non-invasive tests (NITs). This post hoc analysis evaluated cardiometabolic and liver-related semaglutide treatment effects across Asian and non-Asian participants.

Materials and Methods: In part 1, the first 800 randomized ESSENCE (NCT04822181) participants were stratified by diabetes status, fibrosis stage and Asian/non-Asian race. Endpoints included change from baseline to week 72 in body weight, waist circumference (WC), waist-to-height ratio (WtHR), HbA1c, high-sensitivity C-reactive protein (hsCRP), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and N-terminal propeptide of type III collagen (ProC3); and proportions of participants with NIT responses (VCTE-LSM $\geq 30\%$ and $\geq 25\%$; ELF score ≥ 0.5). Subgroups statistical analysis used the same approach as the main trial. No formal between-race comparisons were performed.

Results: At baseline, in Asian versus non-Asian participants, respectively, median (interquartile range) body mass index (BMI) was 29.7 (25.9–33.4) versus 35.1 (31.4–39.7) kg/m², WC was 101.5 (92.0–109.0) versus 115.0 (105.5–125.0) cm, mean WtHR was 0.63 versus 0.69, and mean (SD) hsCRP was 3.1 (4.7) versus 6.3 (6.2) mg/L. Baseline mean LDL, HDL and HbA1c were similar in both groups. At week 72, semaglutide improved outcomes versus placebo in both groups. WC reduction with semaglutide was lower in Asian versus non-Asian participants (estimated treatment difference [ETD] versus placebo [95% CI]: -3.7 [-6.7; -0.6] vs -6.8 [-8.3; -5.2] cm). ETDs versus placebo (95% CI) in WtHR were -0.022 (-0.041; 0.003) and -0.041 (-0.050; -0.032) in Asian and non-Asian participants, respectively. For body weight, HbA1c, hsCRP, LDL, HDL and ProC3, semaglutide treatment benefits versus placebo were comparable between groups. For Asian versus non-Asian participants, VCTE-LSM responder proportions with semaglutide were similar ($\geq 30\%$: 53.7% and 51.5%; $\geq 25\%$: 58.5% and 59.9%, respectively). For VCTE-LSM $\geq 30\%$, the estimated difference in responder proportions (EDP) versus placebo (95% CI) in Asian and

non-Asian participants were 23.1 (8.1;38.1) and 21.2 (11.3;31.1), respectively. A similar proportion of Asian versus non-Asian participants receiving semaglutide had ELF score reduction ≥ 0.5 (58.2% and 55.0%). The EDP versus placebo (95% CI) was 35.1 (22.3;48.0) and 28.4 (20.0;36.8), in Asian and non-Asian participants, respectively.

Conclusions: Despite differences in baseline characteristics, including BMI, semaglutide demonstrated consistent efficacy across Asian and non-Asian participants in several cardiometabolic and non-invasive liver-related outcomes, reinforcing its utility in people with MASH with F2/F3.

Keyword(s) : MASH, Asian and non-Asian populations, semaglutide, GLP-1RA

2349 - Study on the Anti Liver Cirrhosis of Luteolin Targeting the PPAR α Axis to Regulate Macrophage Fatty Acid Metabolic Reprogramming
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Background / Hypothesis: At present, there are no first-line small molecule drugs for anti-liver cirrhosis. The development of potentially effective small molecule drugs is extremely urgent. Some studies have reported that the luteolin has anti-fibrotic and anti-inflammatory effects. However, whether it has anti-liver cirrhosis effects and its mechanism remain unclear. This study will evaluate the efficacy of luteolin against liver cirrhosis and explore its mechanism.

Materials and Methods: The pharmacological effects were validated using both in vivo and in vitro models of liver fibrosis. Mechanistic

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exploration was conducted by integrating single cell transcriptomics, metabolomics, 16S rRNA microbial profiling, liquid-phase chip and experimental verification. Furthermore, the potential targets of Luteolin were investigated through molecular docking, molecular dynamics simulation, surface plasmon resonance, small molecule-protein pull down, and small molecule-protein truncation experiments. Finally, mechanistic validation was performed in macrophage-depletion models, PPAR α inhibitor models, and PPAR α knockout cell models.

Results:

Both in vivo and in vitro, Luteolin alleviated liver fibrosis. It was found to modulate the PPAR α pathway, fatty acid oxidation (FAO), and macrophage polarization. Moreover, the interaction target of Luteolin was identified as PPAR α , with its ligand-binding domain (LBD) serving as the binding site. The mechanisms were further corroborated by target- and target-cell-knockout experiments.

Conclusions:

Luteolin has an anti-liver cirrhosis effect. The mechanism may be that it binds to the LBD domain of PPAR α to regulate the PPAR α /PGC-1 α /CPT1A axis and mitochondrial homeostasis, which promotes the FAO to induce M2 polarization in macrophages. Luteolin is a newly discovered PPAR α agonist, which is expected to provide a reference for the development of potential small molecule drugs against liver cirrhosis.

Keyword(s) :

2350 - Generation of Macro Scale Engineered Biliary Trees with P gp Efflux Function and Enhanced Injury Resistance

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Background / Hypothesis:

The reconstruction of functional biliary trees (FBT) is crucial in the development of bioengineered liver. Decellularized liver scaffolds (DLS), a common platform for FBT, suffer from mechanical instability, resulting in luminal collapse and consequent hypoxic stress that severely impairs FBT function. Additionally, current engineered biliary constructs lack the necessary injury resistance to endure the pathological microenvironment associated with end-stage liver disease.

Materials and Methods:

This work aimed to construct macro-scale FBT with enhanced injury resistance using rat DLS optimized by genipin and mouse primary cholangiocytes. The newly developed FBTs were systematically evaluated through functional assays, transcriptomic analysis, and hepatotoxic drug-induced injury testing to validate their structural stability and injury resistance.

Results:

The FBT exhibited an optimized microenvironment with enhanced structural stability and fluid mechanical properties, which facilitated cholangiocyte adhesion and alleviated hypoxic stress, as evidenced by downregulation of the HIF pathway. Morphological characterization showed that the FBT recapitulated key features of native biliary networks as early as day 7, including multi-level branch architecture, proper apical-basal polarity as well as various mature metabolic and transport functions. Scanning electron microscopy identified the elongated primary cilia orienting toward the bile duct lumen. Functionally, the FBT exhibited enhanced bile acid transport, marked by the upregulation of Slc51a/Slc51b (encoding OST- α /OST- β) and Abcc3 (encoding MRP3), along with enhanced retinoic acid synthesis capacity. Compared to 2D monolayers, the FBT exhibited a superior polarity barrier and enhanced efflux capacity for toxic substances, supported by the upregulation of Abcb1a, which encodes the key efflux transporter P-glycoprotein (P-gp). Notably, such FBT maintained homeostasis and exhibited unique resistance to hepatotoxic injury. This toxic resistance was linked to P-gp-mediated efflux function in FBT, as demonstrated by the impairment of this protection following verapamil-mediated activity inhibition of P-gp.

Conclusions:

The efflux transporter P-gp plays a critical role in the injury resistance of FBT. Our macro-scale FBT, with enhanced toxic efflux function and injury resistance, provides a promising strategy for bioengineered liver construction and clinical translation.

Keyword(s) : biliary trees, decellularized liver scaffolds, P-glycoprotein

2351 - A HOC Study on Patients Under Real time Surveillance for Liver Cancer in Mongolia

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Background / **Hypothesis:**
In Mongolia, liver cancer is a major health issue and has a high prevalence due HBV, HCV and alcohol abuse. This study analyzed the age distribution, tumor status, cirrhosis status, treatment modalities, surveillance outcomes, and causes of death among patients under surveillance for liver cancer.

Materials and **Methods:**
The study was conducted retrospectively. A total of 1,108 cases were selected and analyzed from the registries of liver cancer patients at Happy Veritas Hospital, the Mongolian National University of Medical Sciences, NCC, Second General Hospital. Patients were classified by age group, tumor type (primary/recurrent), cirrhosis status, treatment modality, and surveillance outcome (alive, deceased, lost to follow-up), and actual counts and percentages were calculated. Graphs were generated from the actual data for comparison.

Results:

The data of a total of 1,108 patients included in the study were analyzed. The results are presented categorized according to the graphs. The age distribution of patients under surveillance is shown in actual numbers. The majority of patients fall into the 60–85 age group. The presence or absence of liver cirrhosis was compared in actual numbers and percentages. 59.3% of patients with cancer have liver cirrhosis. Tumor types were classified as primary or recurrent.

Conclusion: The majority (61.7%) of patients under surveillance have recurrent cancer. Patients who received treatment and those who did not were compared. The vast majority (97.5%) of patients have received some form of treatment. Patients who survived, died, or were lost to follow-up during the surveillance period are shown in actual numbers and percentages. 83.4% of patients under surveillance are alive. The main cause of death is liver cancer (85.5%). Various treatment modalities were compared in actual numbers and percentages.

Conclusion

From the study of citizens under surveillance for liver cancer in Mongolia, it is evident that the majority of patients belong to older age groups and recurrent cancer cases are numerous. Treatment outcomes are

good (97.5% received treatment), but the primary cause of death remains the cancer itself. Since the risk is high in patients with cirrhosis, there is a need to improve early detection

Keyword(s) : A-Hoc, HCC, follow-up

2353 - Analysis of Aggressive Phenotypes in Hepatocellular Carcinoma Using Transcriptomic Data A Machine Learning Framework for Biological Signature Discovery

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Background

Hepatocellular carcinoma (HCC) is a biologically heterogeneous malignancy with high mortality largely driven by intrahepatic spread and portal vein invasion. Clinically, solitary tumors, multifocal disease, and HCC with portal vein tumor thrombosis (PVTT) represent distinct yet progressive phenotypes associated with aggressive tumor behavior and poor prognosis. Accurate molecular stratification of these phenotypes is essential for risk assessment and biomarker-driven clinical decision support. However, identifying stable and biologically meaningful gene signatures from high-dimensional transcriptomic data remains challenging. In this study, we applied machine learning and ensemble-based approaches to classify invasive and metastasis-prone HCC phenotypes using a previously established transcriptomic dataset.

Methods

Analyses were conducted on a publicly available gene expression dataset comprising solitary HCC, multifocal intrahepatic HCC, and HCC with portal vein tumor thrombosis. Six machine learning classifiers representing linear, tree-based, and kernel-based approaches, along with two ensemble learning strategies, were systematically evaluated using 10-fold stratified cross-validation. To enhance robustness and biological interpretability, a consensus-driven feature selection strategy was applied based on feature importance scores derived from the three top-performing models. Two reduced gene sets were generated: an intersection set containing genes consistently selected across models and a union set including genes identified as important by at least one

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model. Both sets were independently re-evaluated.
Results

XGBoost (AUC = 0.927) and Random Forest (AUC = 0.919) demonstrated the highest classification performance. Reanalysis using reduced feature sets further improved predictive accuracy, with the Random Forest model trained on the intersection set achieving an AUC of 0.94 and the XGBoost model trained on the union set achieving an AUC of 0.95. Across cross-validation folds, averaged feature importance scores consistently highlighted major histocompatibility complex class I (MHC-I) genes as the most discriminative features among top-performing models.

Conclusion

Our findings demonstrate that ensemble learning combined with consensus-based gene selection enables accurate and biologically interpretable classification of invasive and metastasis-prone HCC phenotypes. This reproducible and extensible framework provides a robust foundation for molecular risk stratification and biomarker-driven modeling in aggressive HCC and has potential applicability in translational hepatology research

Keyword(s) : Hepatocellular carcinoma, portal vein tumor thrombosis, intrahepatic metastasis, machine learning, ensemble learning, transcriptomics

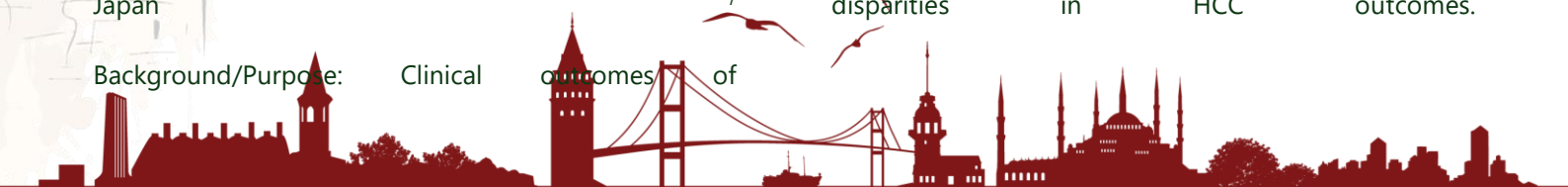
2359 - Stage Specific Survival in Hepatocellular Carcinoma A Multiregional Analysis Across Japan vs Non Japan Cohorts in the Asia Pacific Region

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hepatocellular carcinoma (HCC) vary substantially across the Asia-Pacific region. These differences may reflect heterogeneity in underlying etiologies and healthcare systems, as well as variation in stage-specific survival. To clarify regional diversity in HCC prognosis, we evaluated Barcelona Clinic Liver Cancer (BCLC) stage-specific overall survival (OS) using data from the Asia-Pacific Association for the Study of the Liver (APASL) Hepatology/Oncology Consortium (A-HOC) registry. Methods: This retrospective study used the A-HOC database, which includes newly diagnosed HCC cases from 25 institutions across seven Asian countries. Among 6,580 patients diagnosed between 2013 and 2024, 5,756 patients with available BCLC stage and follow-up data were analyzed. Baseline characteristics, including age, sex, etiology, Child-Pugh, tumor burden, AFP levels, and BCLC stage, were compared among Japan, Taiwan, Turkey, and China. To adjust for interregional differences in background characteristics, inverse probability weighting (IPW) was applied, and BCLC stage-specific OS was compared across regions. Results: Patients in Japan were older at diagnosis (median 73 vs. 61 years), had a slightly lower proportion of males (73.0% vs. 76.8%), and showed a distinct etiologic profile, with higher hepatitis C virus prevalence (37.3% vs. 16.4%) and lower hepatitis B virus prevalence (11.3% vs. 60.6%) than non-Japanese cohorts. The distribution of BCLC stages 0/A, B, and C was 57.5%, 19.4%, and 19.8% in Japan; 46.7%, 27.6%, and 24.1% in Taiwan; 36.7%, 28.3%, and 25.6% in Turkey; and 28.8%, 23.7%, and 46.1% in China, respectively. In the overall cohort, median OS was 10.1 years for stage 0, 7.6 years for stage A, 3.3 years for stage B, 1.3 years for stage C, and 3.3 months for stage D (log-rank $p < 0.01$). Before adjustment, median OS differed among countries (Japan 5.1 years, Taiwan 6.3 years, Turkey 2.1 years, and China 4.3 years). After IPW adjustment for baseline characteristics, no significant differences in OS were observed among Japan, Taiwan, and Turkey for BCLC stages 0/A, B, or C. In contrast, stage-specific differences were observed between Japan and China, with better survival in Japan than in China for BCLC stage 0/A, and better survival in China than in Japan for BCLC stage B and stage C. Conclusions: Although substantial regional variation in HCC prognosis exists across the Asia-Pacific region, much of this variation can be explained by differences in diagnostic stage and patient characteristics. Standardizing surveillance practices and promoting earlier detection may help reduce interregional disparities in HCC outcomes.

Background/Purpose: Clinical outcomes of



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Keyword(s) : hepatocellular carcinoma

2360 - Amlodipine activates an adaptive endoplasmic reticulum stress response in Huh 7 hepatocellular carcinoma cells

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Background / Hypothesis: Amlodipine is a widely used calcium channel blocker. Although generally considered safe, rare cases of amlodipine-associated liver injury have been reported, and the underlying mechanisms remain unclear. Endoplasmic reticulum (ER) stress in hepatocytes is characterized by activation of the unfolded protein response, including upregulation of glucose-regulated protein 78 (GRP78) and, under severe conditions, induction of the pro-apoptotic marker C/EBP homologous protein (CHOP). This study aimed to investigate whether amlodipine induces ER stress in Huh-7 hepatocellular carcinoma cells.

Materials and Methods: Huh-7 hepatocellular carcinoma cells were treated with increasing concentrations of amlodipine or control for 24 hours. Cell viability was measured by the CCK-8 assay, and IC₅₀ values were subsequently determined. Protein expression levels of GRP78 and CHOP were evaluated by Western blot analysis, with GAPDH used as a housekeeping protein for normalization.

Results: Amlodipine treatment caused a dose-dependent reduction in cell viability in Huh-7 cells, with an IC₅₀ value of 63.34 µM. Western blot analysis demonstrated a noticeable increase in CHOP protein expression in amlodipine-treated cells compared to control cells. In contrast, GRP78 protein levels showed no significant change following amlodipine exposure. GAPDH-normalized band intensities suggest that amlodipine exposure is associated with activation of CHOP-related pro-apoptotic ER stress-associated signaling rather than an adaptive GRP78-mediated unfolded protein response.

Conclusions: These findings suggest that amlodipine induces endoplasmic reticulum stress in Huh-7 hepatocellular carcinoma cells, characterized by upregulation of the pro-apoptotic ER stress marker

CHOP without a concomitant increase in the adaptive chaperone protein GRP78. This pattern indicates a predominance of maladaptive ER stress and apoptotic signaling rather than a protective unfolded protein response. Such activation of CHOP-dependent pathways may contribute to amlodipine-induced cytotoxicity in hepatocellular carcinoma cells. Further studies are required to elucidate the upstream ER stress pathways involved and to clarify the role of CHOP-mediated apoptosis in amlodipine-associated hepatocellular injury.

Keyword(s) : Amlodipine, Endoplasmic reticulum stress, GRP78, Hepatocellular carcinoma, Huh-7 cells

2361 - L canavanine selectively inhibits hepatocellular carcinoma cell proliferation and induces apoptosis in vitro

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Background / Hypothesis: Hepatocellular carcinoma is associated with high mortality and limited therapeutic options, highlighting the need for novel antiproliferative agents. L-canavanine, a structural analogue of arginine, has been suggested to interfere with protein synthesis and to preferentially induce cell death in malignant cells. We hypothesised that L-canavanine would decrease cell viability and promote apoptosis in hepatocellular carcinoma cells.

Materials and Methods: Human Huh7 hepatocellular carcinoma cells and murine L929 fibroblast cells were cultured under standard conditions. After 24 hours of attachment, cells were exposed to L-canavanine (12.5–800 µM) for 48 and 72 hours (vehicle control). Cell viability was assessed by XTT assay and expressed as a percentage of control. In Huh7 cells, apoptosis was evaluated by a flow cytometric Annexin V binding assay; the proportion of Annexin V positive cells was taken as a marker of apoptotic cell death. Data were analysed using one-way analysis of variance followed by appropriate post hoc tests; p<0.05 was considered statistically significant.

Results: L-canavanine produced a marked, dose-

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dependent reduction in Huh7 cell viability at both 48 and 72 hours. Concentrations from 25 to 800 μM significantly decreased viability compared with control ($p < 0.05$), with higher doses lowering viability to approximately 20–40% of control, whereas 12.5 μM partially preserved viability. In L929 cells, XTT revealed only modest but statistically significant decreases in viability at the higher concentrations ($p < 0.05$), and overall survival remained substantially higher than in Huh7 cells at equivalent doses. In Huh7 cells, Annexin V flow cytometry showed a clear, dose-dependent increase in Annexin V positive cells, indicating prominent induction of apoptosis; Annexin V positivity was significantly elevated in L-canavanine treated groups compared with control ($p < 0.05$).

Conclusions: L-canavanine exerts pronounced cytotoxic and pro-apoptotic effects in Huh7 hepatocellular carcinoma cells, while causing comparatively limited loss of viability in L929 fibroblast cells. The combination of reduced viability and increased Annexin V positivity supports apoptosis-driven cell death and suggests that L-canavanine may represent a promising candidate for further preclinical evaluation as a selective antitumour agent in hepatocellular carcinoma.

Keyword(s) : L-canavanine, hepatocellular carcinoma, Huh-7, apoptosis, flow cytometry

2364 - Hepatic Involvement and Drug Induced Liver Injury in Brucellosis

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Background / Hypothesis: Brucellosis is a systemic zoonosis with frequent reticuloendothelial involvement. While liver involvement can range from mild transaminase elevation to granulomatous hepatitis, data on the incidence of drug-induced liver injury (DILI) during prolonged antibiotic therapy remains scarce. This study aims to evaluate the incidence of baseline hepatitis and the development of DILI in patients diagnosed with brucellosis during follow up.

Materials and Methods: This retrospective study included patients diagnosed with brucellosis at Ege University and Manisa Celal Bayar University between 2017 and 2025. Diagnosis was confirmed by compatible clinical findings combined with positive serology (Rose Bengal, Wright agglutination test $\geq 1/160$) or blood culture isolation. Baseline hepatic involvement was defined as the elevation of aminotransferases (AST or ALT) above the upper limit of normal, accompanied by clinical findings such as hepatomegaly or splenomegaly, consistent with the literature on brucellar hepatitis. DILI was defined according to international consensus criteria during the follow-up. Normality of distribution was assessed using the Shapiro-Wilk test. Continuous variables were expressed as median (minimum-maximum) for non-normally distributed data and compared using the Mann-Whitney U test. Statistical analysis was performed using the Chi-square test and Fisher's exact test to compare clinical features between groups. A p-value of < 0.05 was considered statistically significant.

Results: A total of 122 patients were included in the study. The median age of the patients was 48 (18-82) years, and 76 (62.3%) were male. Demographic characteristics and baseline clinical findings are presented in Table 1. Based on the strict definition, 25 (20.5%) of the cohort presented with hepatitis. Patients with clinical hepatitis exhibited a higher rate of bacteremia compared to those without clinical hepatitis (Blood culture positivity: 10/17, 58.8% vs. 16/44, 36.4%), although this difference did not reach statistical significance ($p = 0.15$). The detailed comparison of clinical characteristics between groups is presented in Table 2. During the treatment course, 13 (10.7%) of patients developed DILI (Table 3). The incidence of DILI was numerically higher in the clinical hepatitis group compared to the non-hepatitis group (4/25, 16.0% vs. 9/97, 9.3%, $p = 0.46$). No significant association was found between pre-existing liver disease and DILI development (2/15, 13.3% vs. 11/107, 10.3%, $p = 1.00$).

Conclusions: Clinical hepatitis represents a distinct phenotype in brucellosis associated with significant thrombocytopenia and cholestasis. Furthermore, the potential for treatment-related hepatotoxicity underscores the critical necessity for strict and regular liver function monitoring throughout the prolonged antibiotic course, regardless of the patient's baseline hepatic status.

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Keyword(s) : Brucellosis, Hepatitis, Hepatosplenomegaly, Drug-Induced Liver Injury

2368 - Dynamic Metabolic Reprogramming Drives Adipose Fibrosis in a High Fat Diet-Induced Obesity in Mice Model

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Background / Hypothesis: Adipose tissue undergoes significant metabolic and structural change during high-fat diet (HFD)-induced obesity in mice, resulting in increased fat mass, adipocyte hypertrophy, inflammation, oxidative stress, and metabolic dysfunction. Extracellular matrix (ECM) deposition, inflammation, and eventually fibrosis are the results of this progressive change. Finding the metabolic milestones that propel fibrotic remodeling requires an understanding of the successive metabolomic changes that occur during this transition. The objective is to delineate stage-specific metabolic signatures associated with adipose dysfunction and fibrotic progression. **Materials and Methods:** C57BL/6J mice (6–8 weeks old) were fed a standard chow and a high-fat diet consisting of 10% and 50–60% kcal from fat, respectively, till 24 weeks. We performed a comprehensive time-resolved untargeted metabolomics analysis of visceral adipose tissues of control and HFD mice at weeks 8 and 24, coupled with histology, and fibrotic markers. Metabolites from tissue were extracted and analysed using by ultra-performance liquid chromatography-high resolution mass spectrometry (UPLC-HRMS) and metabolite module specific to pathway analysis correlation. **Results:**

We developed and characterized a model of obese fibrosis mice brought on by a high-fat diet. In both comparison groups, high fat feeding led to a substantial increase in body weight and liver weight as well as other biochemical markers such liver enzymes (serum ALT, AST) and lipid profiles (TC, TG, LDL-C, and HDL) ($p > 0.05$). Steatosis and fibrosis are measured by histology and Sirius Red at a later stage (24 weeks). Sequential integration analysis revealed three primary metabolic nodes that contribute to fibrosis: (1) mitochondrial TCA dysregulation/inadequate β -oxidation; (2) glycolysis; and (3) bile acid dysregulation. Early-stage (8 weeks) metabolomics showed a distinct shift toward mitochondrial stress and decreased β -oxidation, as seen by a lower TCA cycle. Significantly lower levels of

tricarboxylic acid (TCA) cycle intermediates (citrate, α -ketoglutarate, and malate) further suggested early-stage oxidative metabolism suppression. Adipose tissue showed a chronic dysfunction pattern with substantial oxidative stress, ECM stabilization, and decreased metabolic flexibility following 24 weeks of HFD. The levels of DCA, TDCA, and 12 α -hydroxylated BAs (such as taurocholic acid) showed significant changes at 24 ($p > 0.05$). We found significant changes in the TCA cycle, β -oxidation, and mitochondrial stress during the transition period of 8 to 24 weeks, but not in glycolysis. **Conclusions:**

Our results provide a thorough metabolomic study linking obesity and adipose fibrosis. Potential metabolic indicators and therapeutic targets for early intervention of adipose tissue fibrotic remodeling in metabolic disease.

Keyword(s) : High fat Diet, Metabolomics, mass spectrometry, Obesity

2369 - AGIUS Score Predicts Re bleeding Risk after Endoscopic Variceal Ligation in Cirrhotic Patients Introduction

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Background / Hypothesis:

Re-bleeding remains a major challenge after endoscopic variceal ligation (EVL) in cirrhotic patients. This study aimed to investigate the predictive value of the Acute Gastrointestinal Injury Ultrasonography (AGIUS) score for both one-year re-bleeding and treatment-resistant re-bleeding (TR-rebleeding, defined as ≥ 3 episodes per year) post-EVL.

Materials and Methods: In this single-center retrospective cohort study, 120 cirrhotic patients who underwent EVL were included. Patients were stratified based on re-bleeding events within one year. Baseline characteristics, AGIUS scores, endoscopic findings, and hemodynamic parameters were analyzed. Multivariate logistic regression was used to identify independent predictors. The predictive performance was evaluated using receiver operating characteristic (ROC) curve analysis.

Results:

Among the 120 patients, 44 (36.7%) experienced re-

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bleeding, with 29 (24.1%) meeting the criteria for TR-rebleeding. Multivariate analysis identified the AGIUS score (adjusted odds ratio [aOR] = 1.558, 95% CI: 1.217–1.996, $p < 0.001$) and high-grade esophagogastric varices (aOR = 13.763, 95% CI: 4.308–43.971, $p < 0.001$) as independent predictors for any re-bleeding. For TR-rebleeding, the AGIUS score (aOR = 1.403, 95% CI: 1.113–1.767, $p = 0.004$) and high-grade varices (aOR = 6.602, 95% CI: 2.391–18.228, $p < 0.001$) remained independent predictors. The combined model (AGIUS score + variceal grading) demonstrated excellent predictive power for any re-bleeding (AUC = 0.853) and for TR-rebleeding (AUC = 0.836).

Conclusions:

The AGIUS score, particularly when combined with endoscopic variceal grading, is a robust and non-invasive predictor for re-bleeding and treatment-resistant re-bleeding within one year after EVL. This novel tool holds significant promise for improving risk stratification and guiding personalized management in cirrhotic patients.

Keyword(s) :

2374 - Non Invasive Assessment of Steatosis and Stiffness in MASLD Links to Body Composition and Dyslipidemia in High Risk Metabolic Groups
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Background

Obesity, prediabetes, and type 2 diabetes are rising globally, with metabolic dysfunction-associated steatotic liver disease (MASLD) as the most prevalent liver disorder. This study investigates the grade of liver steatosis and stiffness in patients with MASLD across these conditions and their associations with metabolic parameters and body composition via bioelectrical impedance analysis (BIA).

Materials

and

Methods:

This prospective cross-sectional study included 40 obese, 40 prediabetic, 40 type 2 diabetic patients with MASLD (diagnosed by ultrasonography and/or shear wave elastography within the past six months), and 40 healthy controls. Body measurements, blood

parameters, elastography, and Tanita BIA assessments were recorded. Statistical analyses used ANOVA with post-hoc tests; $p < 0.05$ was significant

Results:

Body mass index (BMI), fat percentage, fat mass, and visceral fat ratio were significantly lower in non-steatotic individuals compared to grade 1 and 2 steatosis ($p < 0.001$). Liver elastography (kPa), liver size (mm), and pancreatic steatosis grade increased with steatosis severity ($p < 0.001$; Table 1). LDL-cholesterol, triglycerides, and total cholesterol were elevated in steatotic groups versus controls ($p < 0.001$), but not between grades. Diabetes group showed highest elastography values (7.6 ± 2.6 kPa) versus others ($p < 0.001$), with larger liver sizes and higher pancreatic steatosis in all patient groups versus controls ($p < 0.001$).

Conclusions:

Increasing liver steatosis correlates with higher BMI, adiposity, and visceral fat, alongside stiffness suggesting fibrotic progression. Pancreatic steatosis underscores metabolic syndrome's multi-organ impact. Comprehensive metabolic evaluation is crucial for MASLD management.

Keyword(s) : MASLD, obesity, prediabetes, type 2 diabetes, elastography, bioelectrical impedance

2375 - Achieving Recompensation with Low dose Diuretics in HBV related Cirrhosis An Expanded Baveno VII Strategy Yields Similar Long Term Prognosis in A Multicenter Cohort Study
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Background

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Hypothesis:

Baveno VII consensus proposed the definition of recompensation with resolution of decompensation events and discontinuation of respective treatments (except non-selective beta-blockers) for 12 months. However, the impact and necessity of discontinuation of minimal decompensation treatment in the definition of recompensation was unknown.

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Materials and Methods: Patients with hepatitis B virus (HBV)-related decompensated cirrhosis were enrolled at the time of first decompensation from three multicentre cohorts. Recompensation was defined according to Baveno VII criteria. An expanded recompensation criteria were evaluated for patients on low-dose diuretics (anti-aldosteronic drugs ≤ 200 mg/day and/or furosemide ≤ 25 mg/day) and/or lactulose/rifaximin with resolution of decompensated events for 12 months. Propensity score-matching (PSM) was performed in 1:1 or 2:1 matched for sex, age, platelet, albumin and total bilirubin at the time of achieving recompensation between patients meeting Baveno VII criteria or those meeting expanded recompensation criteria. Re-decompensation was defined as the recurrence of ascites, variceal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis or hepatorenal syndrome after recompensation. The cumulative incidence of re-decompensation, liver-related death/liver transplantation (LT) and hepatocellular carcinoma (HCC) was estimated using Kaplan-Meier curves, with subgroup comparisons performed by the log-rank test.

Results:

A total of 1,008 patients were enrolled for analysis, with a median follow-up of 44.8 (IQR: 21.3-64.2) months. Among them, 368 (36.5%) patients achieved recompensation of Baveno VII criteria, and 100 (9.9%) patients met the expanded recompensation criteria. During follow-up, the 5-year cumulative incidence of re-decompensation was comparable between recompensated patients meeting Baveno VII criteria and expanded criteria (22.8% vs. 26.4%, $P = 0.305$), using the time of achieving recompensation as time zero. Moreover, the 5-year cumulative incidence of liver-related death/ liver transplantation was also similar between patients meeting Baveno VII criteria and expanded criteria (5.4% and 11.1%, $P = 0.111$), respectively. However, the 5-year cumulative incidence of hepatocellular carcinoma (HCC) was higher in patients meeting Baveno VII criteria compared with those achieving expanded recompensation criteria (18.8% vs. 18.0%, $P = 0.021$). After performing propensity score-matching, no significant difference was observed in the cumulative incidence of re-decompensation and liver-related death/ liver transplantation, as well as the occurrence of HCC after recompensation ($P > 0.05$).

Conclusions: The continued use of minimal decompensation treatment in expanded

recompensation criteria identifies recompensated patients with similar prognosis, comparable to those meeting the recompensation criteria proposed in the Baveno VII consensus.

Keyword(s) : Recompensation; Re-decompensation; Anti-HBV therapy; Low-dose Diuretics

2377 - Efficacy Differences of Interferon Combined with Nucleos(t)ide Analogs in Treating HBeAg Positive Chronic Hepatitis B Stratified by Baseline Liver Fibrosis Indicators

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Background / Hypothesis:

The impact of baseline liver fibrosis degree on the efficacy of pegylated interferon (Peg-IFN) combined with nucleos(t)ide analogs (NAs) in treating HBeAg-positive chronic hepatitis B (CHB) has not been fully elucidated. This study aims to evaluate the efficacy differences of this combination regimen in patients with varying degrees of baseline liver fibrosis based on a retrospective clinical cohort.

Materials and Methods:

A total of 172 HBeAg-positive CHB patients treated with Peg-IFN combined with NAs were included in this study. Patients were divided into three groups based on baseline non-invasive fibrosis indicators (APRI and FIB-4): Group 1 (no significant fibrosis, $n=75$), Group 2 (significant fibrosis, $n=70$), and Group 3 (early cirrhosis, $n=27$). The primary endpoint was the HBeAg clearance rate at 24 months of treatment, while secondary endpoints included HBsAg clearance rate, virological response (HBV DNA < 20 IU/mL), and liver fibrosis outcomes.

Results:

After 24 months of treatment, there were no significant differences among the three groups in terms of HBsAg clearance rate and undetectable HBV DNA rate. However, significant differences were observed in HBeAg clearance: the cumulative HBeAg clearance rates for Group 1, Group 2, and Group 3 were 16.00%, 30.00%, and 40.74%, respectively, with Group 3 showing a significantly higher clearance rate compared to Group 1 and Group 2 (Log-rank $P = 0.027$). Multivariate analysis revealed that after adjusting for other variables, the odds ratio for achieving HBeAg clearance in Group 3 (early cirrhosis) was 5.234 times that of Group 1 (95% CI: 1.452-18.871, $P = 0.011$), indicating that baseline liver

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fibrosis degree is an independent predictor of HBeAg clearance.

Conclusions:In HBeAg-positive chronic hepatitis B patients, the degree of baseline liver fibrosis is an important factor influencing the efficacy of Peg-IFN combined with NAs treatment. Compared to patients without significant fibrosis, those with significant fibrosis or early cirrhosis at baseline achieved higher HBeAg clearance rates, suggesting that this combination therapy may provide greater clinical benefits for patients with more advanced fibrosis.

Keyword(s) : Chronic hepatitis B, HBeAg, influencing factors

2378 - Metabolic Dysfunction–Associated Liver Disease and Lung Injury A Decade of Progress Toward Mechanistic Integration and Translational Insight

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Background / Hypothesis:Metabolic dysfunction-associated fatty liver disease (MAFLD) and its progressive form, metabolic dysfunction-associated steatohepatitis (MASH), are increasingly recognized as conditions with prominent systemic immunometabolic features rather than liver-confined disorders. Accumulating evidence suggests that the lung may represent a vulnerable extrahepatic target organ in MAFLD/MASH, with diverse pulmonary abnormalities reported. Here, we propose a conceptual framework in which disease-specific mechanisms—chronic metabolic inflammation, liver-derived mediators, and inter-organ crosstalk—contribute to heightened pulmonary vulnerability, with potential implications for risk stratification and therapy. **Materials and Methods:**We performed a comprehensive narrative, non-systematic review of clinical, epidemiological, and experimental studies retrieved from PubMed and Web of Science between 2010 and 2025. The review focused on the clinical spectrum of pulmonary involvement in MAFLD/MASH, evidence for dose–response and potential causal relationships, key pathophysiological mechanisms (immunometabolic dysregulation, hepatic extracellular vesicles, and the gut–liver–lung axis), and emerging translational

approaches.

Results:MAFLD/MASH is associated with a broad range of pulmonary manifestations, including pulmonary vascular complications (hepatopulmonary syndrome and portopulmonary hypertension), impaired lung function, increased susceptibility to acute lung injury and acute respiratory distress syndrome, and interstitial lung disease or pulmonary fibrosis. These associations persist after adjustment for major shared risk factors and demonstrate dose–response relationships with MAFLD/MASH severity. Emerging Mendelian randomization analyses suggest potential causal links between hepatic metabolic dysfunction and adverse lung outcomes. Three interconnected mechanisms are proposed to contribute to pulmonary vulnerability: neutrophil extracellular trap–mediated amplification of lung injury, hepatic extracellular vesicles as mediators of liver–lung communication, and the gut–liver–lung axis as a chronic immunometabolic modifier. Advances in multi-omics profiling, integrated disease models, and composite biomarkers facilitate refined mechanistic investigation and risk stratification. **Conclusions:**MAFLD/MASH may be conceptualized as a state of heightened pulmonary vulnerability mediated by systemic immunometabolic dysregulation and inter-organ crosstalk. Standardized assessment of MAFLD/MASH severity, particularly fibrosis stage, combined with mechanism-informed biomarkers may improve pulmonary risk stratification. Translational strategies targeting shared metabolic, immunomodulatory, and antifibrotic pathways hold promise for integrated management of liver and lung outcomes in this growing patient population.

Keyword(s) : Metabolic dysfunction–associated fatty liver disease (MAFLD); Metabolic dysfunction–associated steatohepatitis (MASH); Lung injury; Pulmonary fibrosis; Inter-organ crosstalk; Extracellular vesicles (EVs); Neutrophil extracellular traps (NETs); Gut–liver–lung axis

2385 - HECTD3 Mediates PRDX4 Stabilization via K63 Linked Ubiquitination to Ameliorate Hepatic Steatosis Inflammation and Fibrosis in MASH

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Background / Hypothesis:Metabolic dysfunction-associated steatohepatitis (MASH) poses a significant global public health challenge, involving complex pathogenesis related to metabolic syndrome and systemic inflammation. HECTD3, an E3 ubiquitin ligase, is known to regulate

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various inflammatory and fibrotic signaling pathways. However, its role in MASH progression remains unclear. This study aims to investigate the function of HECTD3 in MASH and its underlying molecular mechanisms.

Materials and Methods: Hepatocellular HECTD3 were over-expressed in mice via the AAV8 system, and male C57BL/6J mice (six weeks old) were fed either a Choline-deficient, L-amino acid-defined, high-fat diet (CDAHFD) for 10 weeks, or a high-fat, high-fructose diet (HFHFD) diet for 16 weeks to induce MASH. THLE-2 and AML12 cell lines were used for in vitro studies. Multi-dimensional mass spectrometry combined with co-immunoprecipitation (co-IP) was employed to identify HECTD3-interacting proteins.

Results: HECTD3 protein expression was significantly downregulated in the livers of CDAHFD- or HFHFD-fed mice and in palmitic acid (PA)-stimulated hepatocytes. Compared with controls, hepatocyte-specific overexpression of HECTD3 markedly alleviated MASH progression, including reduced lipid accumulation and significantly attenuated fibrosis. Consistently, HECTD3 overexpression decreased lipid deposition in PA-treated hepatocytes. Co-IP combined with mass spectrometry analysis suggested PRDX4 as a potential binding partner of HECTD3, which was further confirmed by both endogenous and exogenous co-IP assays. Previous studies indicated PRDX4 protects against MAFLD progression; our in vitro data showed that PRDX4 overexpression significantly reduced PA-induced lipid accumulation and inflammation in AML12 cells. Immunofluorescence and western blot analysis revealed that hepatocyte-specific HECTD3 overexpression increased PRDX4 protein levels in the livers of MASH model mice. Consistent with in vivo findings, in vitro experiments further demonstrated elevated PRDX4 protein levels in HECTD3-overexpressing hepatocytes, and cycloheximide (CHX) chase assays indicated that HECTD3 overexpression prolonged the half-life of PRDX4 protein. We further detected that HECTD3 overexpression significantly increased the total ubiquitination level of PRDX4, particularly the level of K63-linked polyubiquitin chains.

Conclusions: HECTD3 interacts with PRDX4 and stabilizes its protein expression by mediating its ubiquitination, specifically via K63-linked polyubiquitination. As a crucial positive regulator of PRDX4, HECTD3 plays a protective role in the development and progression of MASH and may

represent a novel potential therapeutic target for MASH treatment.

Keyword(s) : Metabolic dysfunction-associated steatohepatitis (MASH);HECTD3;PRDX4;Ubiquitination
2386 - Immunosuppressive Medication Non adherence in Liver Transplantation Adult Recipients in Vietnam

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Background / Hypothesis:Long-term graft survival after liver transplantation depends heavily on strict adherence to immunosuppressive therapy. Non-adherence remains a major cause of graft dysfunction, rejection, and unplanned hospitalization, yet data from Southeast Asia, particularly Vietnam, are limited.

Materials and Methods:A cross-sectional study was conducted among 136 adult liver transplant recipients followed at the Hepato-Biliary and Pancreatic Surgery Department, 108 Military Central Hospital (2017–2025). Adherence was assessed using the validated BAASIS questionnaire. Clinical, demographic, and psychosocial factors were analyzed for associations with non-adherence.

Results:Mean follow-up after transplantation was 25.7±17.4 months; 83% were male and 98.1% received living-donor grafts. The overall non-adherence rate was 14.6%. Significant determinants of poor adherence included transplant indication (p=0.027), lack of encouragement or feedback from healthcare staff during follow-up (p=0.038), and dissatisfaction with immunosuppressant therapy (p<0.001). Patients with acute liver failure demonstrated the highest adherence rates.

Conclusions:Immunosuppressive adherence among liver transplant recipients in Vietnam is relatively high, though gaps remain. Strengthening post-transplant counseling, structured follow-up communication, and patient-centered medication education could further reduce non-adherence. These findings highlight modifiable behavioral and healthcare-system factors

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and support the need for multidisciplinary interventions in low- and middle-income transplant settings.

Keyword(s) : liver transplantation, adherence, immunosuppression, BAASIS, Vietnam

2389 - Towards Individualized Interferon Therapy in Hepatitis B HBsAg Kinetics and Seroclearance upon Baseline HBsAg Levels and Treatment Duration

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Background / Hypothesis: A standard 48-week course of pegylated interferon (Peg-IFN) is recommended for chronic hepatitis B (CHB). However, in clinical practice, patients with low baseline HBsAg might achieve seroclearance rapidly, while those with high HBsAg may require extended therapy. Real-world evidence to guide individualized IFN treatment duration based on quantitative HBsAg is lacking.

Materials and Methods: This study was a post hoc subgroup analysis of a large retrospective-prospective cohort (Oasis Study). CHB patients receiving Peg-IFN-based therapy were included in this analysis. Patients were stratified by baseline HBsAg: <100, 100-1000, 1000-3000, and ≥ 3000 IU/mL. We analyzed cumulative HBsAg seroclearance rates, kinetics of HBsAg decline, and the association between actual treatment duration (median 48 weeks, range 24-242 weeks) and outcomes across these strata.

Results: A total of 3623 patients were included in this analysis, with median follow-up duration of 3.0 years. A strong inverse relationship was observed between baseline HBsAg level and cumulative seroclearance, with all inter-stratum differences being statistically significant (Table 1).

We presented the dynamic patterns of median HBsAg levels over time. There was a rapid initial drop across all baseline strata. Among patients with baseline HBsAg <100 IU/mL, median levels plummeted to a low plateau by week 24, and in those with higher baseline levels, the decline was more gradual and required a longer time to stabilize, exceeding 72 weeks in patients with baseline HBsAg >3000 IU/mL. For patients who achieved HBsAg loss, the median time of HBsAg loss was 24 weeks in patients with baseline HBsAg <100 IU/mL, 48 weeks in baseline HBsAg between 100-1000 IU/mL, 84 weeks in 1000-3000 IU/mL, and 96 weeks in baseline HBsAg over 3000 IU/mL (Table 1).

We also examined the relationship between IFN

treatment duration and HBsAg loss in patients. When stratified by baseline HBsAg level, patients with HBsAg <3000 IU/mL who received only 24 weeks of IFN had significantly lower cumulative seroclearance rates than those with longer courses, while in patients with baseline HBsAg over 3000 IU/mL, extending IFN therapy beyond 48 weeks associated with improvement in cumulative seroclearance rates (Figure 1).

Conclusions: This study provides a data-driven framework for individualizing IFN therapy. Among patients with low baseline HBsAg, rapid seroclearance suggests even a shortened course may be highly effective. For those with higher levels, extended therapy beyond 48 weeks could be considered to improve outcomes. The identified time-to-clearance benchmarks offer practical guidance for tailoring IFN treatment duration in real-world practice.

Keyword(s) : Chronic hepatitis B, Pegylated interferon, HBsAg seroclearance, Treatment duration, Individualized therapy

2390 - Ultrasonographic evaluation of gallbladder motor function in the diagnosis and prognosis of intrahepatic cholestasis of pregnancy

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Background / Hypothesis: Intrahepatic cholestasis of pregnancy (ICP) is characterized by skin pruritus and impaired liver function. Several previous studies have revealed that the fasting and ejection volumes of the gallbladder in cholestasis of pregnancy were greater than in normal pregnancy. The goal of this study was to explore the specific ultrasonic features of the gallbladder and evaluate the diagnostic and prognostic values of ultrasound in ICP.

Materials and Methods: We induced a retrospective cohort of 60 ICP patients from January 2021 to December 2022, and compared their data with those from healthy pregnant women (n=60). Gallbladder volume was evaluated by real-time ultrasound examination after an overnight fast and 30, 60, 120 and 180 min after a liquid test meal of 200 mL, and ejection fraction was calculated. Diagnostic and prognostic values of volume and ejection function of gallbladder were analyzed by receiver operating characteristic (ROC) curve.

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Results:

Pregnant women in the ICP group had significantly higher gallbladder basal volume ($43.49 \pm 1.34 \text{ cm}^3$ vs $26.66 \pm 0.83 \text{ cm}^3$, $p < 0.01$) and higher ejection fraction compared with the healthy pregnant women. Ejection fraction (120 min) $> 54.55\%$ might be used to predict ICP diagnosis, while Gallbladder volume (60 min) $> 12.52 \text{ cm}^3$ might predict ICP severity.

Conclusions:

Our results indicate abnormal volume and ejection function of gallbladder in patients with ICP. Ejection fraction (120 min) can be used to predict ICP diagnosis, and Gallbladder volume (60 min) can be used to assess ICP severity.

Keyword(s) : Intrahepatic cholestasis of pregnancy, Ultrasound, Gallbladder motor function,

2394 - Demystifying primary hepatic Langerhans Cell Histiocytosis (LCH) From pattern recognition to molecular diagnosis

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Background / Hypothesis:

Langerhans Cell Histiocytosis (LCH) is a rare clonal proliferative disorder of dendritic cells. Hepatic involvement, although uncommon, represents a severe and prognostically adverse form of the disease in the pediatric age group. Though liver involvement in multi system LCH is reported, Primary hepatic LCH had been hardly reported in literature. Hence this study aimed to analyze the patterns of clinic pathologic and immunophenotypic characteristics in children diagnosed as Langerhan Cell Histiocytosis based on primary hepatic manifestations. To identify diagnostic pitfalls and propose a pattern-based diagnostic algorithm to improve early recognition and management.

Materials and Methods:

Retrospective review of the records of children diagnosed with primary hepatic LCH managed at ICH, Egmore, from January 2023- October 2025. Children developing hepatic LCH after being diagnosed as systemic LCH were excluded. Clinical profiles, biochemical data, imaging findings (USG/MRI),

histopathology (H&E, special stains), immunohistochemistry (CD1a, Langerin, S100), and molecular studies (if available) were analysed.

Results:

We present here seven children diagnosed with primary hepatic LCH. The median age at initial presentation was 20 months (IQR 12-24 months). All cases 100% (n=7) presented initially with short duration fever, cholestatic jaundice, pale stools, abdominal distension, hard hepatomegaly with predominant left-lobe enlargement. GGT (Gamma-glutamyl-transferase) elevation (> 10 times) and serum-alkaline-phosphatase (SAP > 4 times) was seen in all cases (n=7) (sensitivity-100%). MRCP showed Sclerosing cholangitis pattern in one child (n=1). ANA, ASMA was positive in 28.5% (n=2). Bone marrow was normal and Mantoux, CBNAAT was negative in all cases. IHC from liver biopsy was negative for LCH in 6/7 (85.7%) cases. Median period of decompensation was 6 months from initial presentation (IQR 3-10 months). Diagnosis was confirmed by immunohistochemistry of nail-matrix biopsy, skin biopsy and liver explant in the initial 3 cases. Average time from initial presentation to diagnosis was three months, diagnosed only after decompensation. Cases 4-7: Skeletal survey and X-ray-skull were normal. Considering institutional experience where similar presentation of hard hepatomegaly with high SAP, GGT which turned out to be LCH, PET-CT was done to find metabolically active area. Showed multiple metabolically active hypodense hepatic lesions. Last two cases none of the investigations were conclusive, diagnosed with BRAF V600E mutation, and MAP Kinase pathway defects.

Conclusions:

Primary Hepatic LCH does exist in children. Suspect early, do not miss window period for definitive chemotherapy. Since liver biopsy often have poor yield in establishing the diagnosis due to the patchy involvement and later stages at diagnosis, further investigations like PET scan and molecular tests for BRAF V600 mutations would help in arriving at the diagnosis early before other system involvement

Keyword(s) : Primary Hepatic Langerhans Cell Histiocytosis, BRAF V600 mutation, early recognition

2395 - Multicohort fecal metagenomes analysis contributes to non invasive diagnosis for cholangiocarcinoma

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Background / **Hypothesis:** Cholangiocarcinoma (CCA) is a highly lethal malignancy with limited early diagnostic options. While emerging evidence implicates the gut microbiome in CCA tumorigenesis, its potential as a non-invasive diagnostic biomarker remains unexplored. This study aimed to characterize CCA-associated gut microbial alterations and evaluate their diagnostic utility across diverse Chinese populations.

Materials and **Methods:** Metagenomic sequencing was performed on fecal samples collected from 643 individuals across three geographically distinct regions in China (East, Central, and Northwest). We analyzed microbial community composition and functional pathways to identify CCA-specific dysbiosis patterns. Diagnostic models were constructed using both species-level taxonomic markers and functional pathway-based features. Model performance was evaluated in the discovery cohort and validated in two independent cross-regional cohorts. Additionally, we assessed the model's ability to differentiate CCA from hepatocellular carcinoma (HCC) and examined disease specificity using 6,847 samples from 31 published studies spanning 11 disease conditions.

Results: CCA patients exhibited distinct gut microbial dysbiosis characterized by significant enrichment of lipopolysaccharide-producing bacteria (*Klebsiella aerogenes*, *Citrobacter koseri*) and depletion of butyrate-synthesizing organisms (*Faecalibacterium prausnitzii*, *Roseburia intestinalis*). Species-level diagnostic models demonstrated superior discriminatory capacity compared to pathway-based approaches, achieving area under the curve (AUC) values of 98.63% and 99.42% in the East China discovery cohort. Importantly, robust cross-regional validation yielded AUCs of 80.89% in Central China and 80.43% in Northwest China, confirming model generalizability. The model effectively distinguished CCA from HCC (AUC=95.58%), addressing a critical clinical differential diagnosis challenge. Meta-analysis of public datasets revealed moderate disease specificity for the CCA microbiome signature, with performance influenced by biomarker overlap across different pathological conditions.

Conclusions:

This study establishes gut microbiome metagenomic profiling as a promising non-invasive diagnostic tool for cholangiocarcinoma, demonstrating robust performance across geographically diverse populations and effective discrimination from other hepatobiliary malignancies.

Keyword(s) : cholangiocarcinoma, gut microbiome, metagenome, diagnostic model, liver cancer

2398 - Etiology Specific Patterns of Liver Stiffness and Steatosis Assessed by Transient Elastography in a Tertiary Referral Center in Southern Philippines

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Background:

Chronic liver disease (CLD) in the Philippines represents a heterogeneous etiologic spectrum shaped by the coexistence of metabolic dysfunction, alcohol use, chronic viral hepatitis, and parasitic infections. In Southern Philippines Medical Center, a tertiary referral center, schistosomal hepatopathy remains a clinically relevant but under-characterized contributor to portal hypertension and advanced fibrosis. Vibration-controlled transient elastography (VCTE) enables non-invasive, simultaneous assessment of hepatic steatosis via the controlled attenuation parameter (CAP) and fibrosis via liver stiffness measurement (LSM). However, comparative real-world data describing etiology-specific CAP–LSM patterns in mixed-disease populations remain limited.

Materials and **Methods:** We conducted a retrospective cross-sectional study of adult patients who underwent clinically indicated VCTE at a tertiary referral hospital in Southern Philippines between May 2023 and November 2025. Demographic variables (age, sex, body mass index) and VCTE-derived parameters (CAP, LSM, steatosis grade, fibrosis stage) were extracted. Liver disease etiologies were categorized as metabolic dysfunction–associated steatotic liver disease (MASLD), viral hepatitis, alcohol-related liver disease, schistosomiasis-related liver disease, or drug-induced liver injury. Non-parametric tests were used to compare CAP and LSM across etiologies. Multivariable logistic regression identified independent predictors of significant steatosis (S2–S3) and advanced fibrosis (F3–F4), while linear regression and cluster analysis explored continuous stiffness

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patterns and phenotypic groupings.

Results:

A total of 482 patients were included. CAP and LSM values differed significantly across etiologies (both $p < 0.001$). MASLD demonstrated the highest steatosis burden, with elevated CAP values and relatively lower median LSM, consistent with early to intermediate fibrotic disease. In contrast, schistosomiasis-related liver disease exhibited a distinctive phenotype characterized by low CAP but markedly elevated LSM, reflecting fibrosis and portal hypertension disproportionate to steatosis. Alcohol-related liver disease showed high and widely variable LSM values, suggesting heterogeneous fibrotic trajectories. On multivariable analysis, significant steatosis was independently associated with BMI, CAP, and MASLD etiology, whereas viral hepatitis and schistosomiasis were inversely associated with steatosis severity. Advanced fibrosis was most strongly predicted by viral hepatitis and schistosomiasis, with age and CAP also contributing, while BMI was not independently associated after etiology adjustment. Cluster analysis revealed clinically interpretable CAP–LSM phenotypes aligning with underlying disease mechanisms.

Conclusions:

VCTE reveals distinct, etiology-specific steatosis and fibrosis patterns in a mixed CLD population from Southern Philippines. Schistosomiasis-related liver disease is characterized by high liver stiffness despite minimal steatosis, underscoring the importance of etiology-aware interpretation of VCTE parameters. These findings support the role of VCTE as a valuable, non-invasive tool for risk stratification and clinical decision-making in regions with diverse and overlapping liver disease etiologies.

Keyword(s) : VCTE; Transient Elastography; CAP; liver stiffness; steatosis; MASLD; viral hepatitis; alcohol-related liver disease; schistosomiasis; Philippines.

2404 - Development and validation of a dynamic nomogram for predicting significant liver injury in chronic hepatitis B patients with indeterminate phase

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Background

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Hypothesis:

A large number of patients with chronic hepatitis B (CHB) are classified into the indeterminate phase, yet a substantial proportion may have significant liver injury and require antiviral treatment. However, few practical non-invasive tools are available for accurately identifying the presence of liver injury in this population. Using a multicenter cohort of CHB patients who received liver biopsy, we developed and validated a dynamic nomogram to predict significant liver injury in CHB patients with indeterminate phase.

Materials

and

Methods:

Patients with CHB in the indeterminate phase based on the AASLD 2018 guideline who underwent liver biopsy were retrospectively collected from five hospitals in China and randomly divided into a training cohort ($n=248$) and a validation cohort ($n=123$) with the ratio of 2:1. Significant liver injury was defined as liver inflammation of grade $\geq G2$ and/or liver fibrosis of stage $\geq S2$ according to the Scheuer scoring system. Variables associated with significant liver injury were selected using least absolute shrinkage and selection operator (LASSO) regression, and a dynamic nomogram was developed. The performance of the nomogram was evaluated using area under the receiver operating characteristic (AUC), calibration curves, decision curve analysis (DCA).

Results:

A total of 371 CHB patients were included, with a median age of 41.0 years and 17.8% were HBeAg positive. 70.2% and 69.9% of patients had significant liver injury in the training cohort and validation cohort, respectively. Hepatitis B e antigen (HBeAg), gamma-glutamyl transpeptidase (GGT), prothrombin time (PT), and alkaline phosphatase (ALP) were identified as independent predictors of significant liver injury in the

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training cohort, and a web-based nomogram was constructed accordingly based on these variables. The nomogram demonstrated good discriminatory capacity and calibration, with AUCs of 0.745 (IQR: 0.679-0.811, $P < 0.001$) and 0.728 (IQR: 0.630-0.827, $P < 0.001$) in the training and validation cohort, respectively; both were significantly higher than that of alanine aminotransferase (ALT). Decision curve analysis indicated superior net clinical benefit of the nomogram.

Conclusions:

We proposed a novel non-invasive dynamic nomogram based on routine clinical parameters with good performance in predicting significant liver injury in CHB patients with indeterminate phase. This tool may assist risk stratification and clinical management of CHB patients with indeterminate phase.

Keyword(s) : chronic hepatitis B, indeterminate phase, significant liver injury, predictive model, dynamic nomogram

2406 - Benefit of treatment expansion A Comparative Analysis of EASL 2025 AASLD 2025 and APASL 2026 Clinical Practice Guidelines for Management of Chronic Hepatitis B

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Background / **Hypothesis:** Thresholds for initiating antiviral therapy for chronic hepatitis B (CHB) vary substantially across major regional guidelines, resulting in substantial variation in treatment coverage. The APASL 2026 CHB clinical practice guidelines (CPGs) introduce a simplified, more inclusive "treat-all" strategy, yet its population-level impact has not been quantified. This study intended to estimate the eligibility, HCC and mortality outcomes under recently updated CHB CPGs.

Materials and **Methods:** Three guideline-specific treatment algorithms were constructed according to APASL 2026, EASL 2025, and

AASLD 2025 CPGs. A synthetic cohort of 100,000 treatment-naïve adults with CHB was generated using publicly available epidemiological datasets. Treatment eligibility under each guideline was assessed deterministically. A state-transition Markov model projected HCC incidence, liver-related mortality, and direct medical costs from 2025–2050. Transition probabilities were derived from large prospective Asian cohorts and meta-analyses evaluating antiviral effectiveness across fibrosis stages in both treated and untreated populations. Model uncertainty was assessed using probabilistic sensitivity analysis (1,000 iterations).

Results:

Treatment eligibility was highest under APASL (85%, range 77–99.99%), followed by EASL (62%, 59–97.9%) and AASLD (47%, 46–58%). Compared with EASL and AASLD, the APASL "treat-all" approach prevented 5,843 (95% UI: 216–22,660) and 9,534 (373–33,322) additional HCC cases, and 4,875 (151–18,337) and 7,955 (220–26,804) liver-related deaths by 2050, respectively. Long-term healthcare costs were also lower under APASL, saving USD 231 million (95% UI: 2.1–1,740 million) versus EASL and USD 377 million (3.1–2,630 million) versus AASLD over 25 years.

Conclusions:

A treat-all approach markedly expands treatment eligibility and yields significant reductions in HCC and liver-related mortality. These findings support APASL's simplified treatment strategy as a practical and impactful pathway toward HBV elimination.

Keyword(s) : Chronic Hepatitis B; Guidelines; Treat-all; APASL; AASLD; EASL

2410 - Hepatitis B Virus Core Antibody Seroconversion and Risk of Hepatitis B Reactivation after Allogeneic Hematopoietic Stem Cell Transplantation

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Background: Anti-HBc positivity is a serological marker of previous HBV infection. Patients with positive anti-HBc are at risk of HBV reactivation during immunosuppressive therapy, chemotherapy, or monoclonal antibody therapy. This study aimed to compare the effects of different immune clearance and reconstitution therapies—including allogeneic

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hematopoietic stem cell transplantation (allo-HSCT), autologous hematopoietic stem cell transplantation (auto-HSCT), and anti-CD20 monoclonal antibody therapy (represented by Rituximab, RTX)—on anti-HBc seroconversion and HBV reactivation risk in patients with previous HBV infection, providing evidence for clinical monitoring and management strategies. **Methods:** This was a single-center, retrospective nested case-control study. Patients who received allo-HSCT/auto-HSCT/RTX treatment at Huashan Hospital, Fudan University from 2020 to 2023, with positive anti-HBc and negative HBsAg before treatment, were enrolled. 10 patients who underwent allo-HSCT served as the observation group, and 10 patients each in the auto-HSCT and RTX groups were selected as control groups based on the follow-up time of anti-HBc after transplantation or RTX initiation. Kaplan-Meier curve and Log-rank test were used to compare cumulative anti-HBc seroconversion rates at 24 months post transplantation of RTX initiation between groups. **Results:** Within 24 months, 7 patients in allo-HSCT group achieved anti-HBc seroconversion, with a median seroconversion time of 11 months post-transplantation. Only 1 patient in auto-HSCT group had anti-HBc seroconversion at 18 months, while no patients in RTX group showed seroconversion. There was a significant difference in the cumulative 24-month anti-HBc seroconversion rates among three groups ($P = 0.035$). In allo-HSCT group, 1 patient experienced HBV reactivation after anti-HBc seroconversion: a 41-year-old male did not receive preventive anti-HBV treatment after allo-HSCT. His anti-HBc disappeared at 9 months. HBV DNA load of 2.45×10^2 IU/mL was detected at 12 months. Then he developed positive HBsAg, HBeAg and anti-HBc, with ALT/AST rose to above ULN at 13 months post-transplantation, indicating HBV reactivation. HBV DNA load dropped below the detection limit after 3 months of entecavir treatment. **Conclusions:** This study observed that allo-HSCT significantly promotes anti-HBc seroconversion, which may be related to the clearance of HBV-specific immune memory by immune reconstitution after allo-HSCT. The low anti-HBc seroconversion might be due to the presence of B cells that secrete anti-HBc during the autologous hematopoietic stem cell isolation process. It is recommended that anti-HBc-positive patients receive preventive antiviral therapy after allo-HSCT; close monitoring for HBV reactivation is needed even after anti-HBc seroconversion. For patients who become anti-HBc-negative after allo-HSCT and require subsequent immunosuppressive therapy, a detailed history of pre-transplant anti-HBc positivity should be obtained.

Keyword(s) : Hepatitis B; HBV Reactivation; Anti-HBc; Allogeneic Hematopoietic Stem Cell Transplantation

2418 - AFP trajectories in HCC after ablation treatment a 15 year retrospective study

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Background / **Hypothesis:**
As a potential curative treatment for HCC, Ablation treatment for HCC as part of the locoregional therapy is becoming more accepted by patients and specialists in various centers and is now being recognized as a potential curative treatment. There is a need to understand whether the prognostic value of AFP applies to patients receiving ablation treatment and its relationship to tumor recurrence and patient's survival.

Materials and **Methods:**
Data of 250 newly diagnosed HCC patients who underwent ablation treatment (RFA/MWA) were analyzed. A total of 717 ablation procedures were included. AFP response were categorized to 4 groups: >20% increase from the baseline with tumor recurrence in imaging, <20% increase with HCC recurrence, >20% with no recurrence in imaging and <20% with no recurrence. The independent risk factors of relapse and mortality were determined by Cox proportional hazard model. The cumulative 1-, 3-, 5-year RFS, OS and RDS rates were computed by Kaplan-Meier method and were compared between groups using the log-rank test. P value <0.05 was considered to be statistically significant. IBM SPSS version 27.0 (SPSS Inc., Chicago, IL, USA) was used to process the data involved.

Results:
AFP elevation $\geq 20\%$ at 3 months post-ablation significantly predicted worse RFS ($p=0.001$) and OS ($p=0.006$) but not RDS ($p=0.288$). Multivariate Cox regression analysis showed that compared to the reference group (i.e., patients with AFP increase <20% and no recurrence), patients with AFP increase $\geq 20\%$ and recurrence had 2.48 times the risk of recurrence (HR=2.477, 95% CI: 1.16-5.28, $p=0.018$) and 2.60 times the risk of death (HR=2.596, 95% CI: 1.14-5.89, $p=0.022$). Patients with AFP increase <20% and recurrence also demonstrated elevated risks with HR=2.866 ($p<0.001$) for recurrence and HR=2.013 ($p=0.004$) for mortality. Kaplan-Meier survival analysis demonstrated

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significantly divergent survival curves between AFP trajectory groups for both RFS and OS (log-rank $p < 0.001$ and $p = 0.004$, respectively). However, AFP trajectories did not significantly predict post-recurrence survival.

Conclusion:

An AFP increase of $\geq 20\%$ at 3 months following ablation is a strong independent predictor of both recurrence and overall mortality in patients with HCC. The lack of association with post-recurrence survival suggests that AFP dynamics primarily reflect initial treatment response. However, AFP trajectories did not significantly predict post-recurrence survival.

Keyword(s) : AFP, post ablation

2422 - Diagnostic Accuracy of Portal System Duplex Sonography Findings Among Filipinos with and Without Liver Cirrhosis; The DupLiCs Study

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Background / Hypothesis: Portomesenteric duplex ultrasonography (PDUS) provides hemodynamic information not captured by liver elastography. Despite its accessibility and safety, its diagnostic role in cirrhosis remains underexplored in Filipino populations. This study aimed to determine the diagnostic accuracy of PDUS parameters in detecting liver cirrhosis among Filipinos.

Materials and Methods: We conducted an analytical cross-sectional study at a tertiary center in Quezon City. Adult Filipinos with elastography-confirmed cirrhosis were recruited as cases and matched 1:1 with controls without liver injury. All participants underwent standardized PDUS. Diagnostic performance was evaluated through sensitivity, specificity, predictive values, likelihood ratios, and overall accuracy. ROC analysis was used for multivariate models.

Results:

The portal vein congestion index emerged as the

strongest single predictor, demonstrating 82.4% diagnostic accuracy (95% CI: 71.2–93.5) and a significant association with cirrhosis. A multivariate model integrating the portal vein congestion index, hepatic vein damping index, and hepatic artery resistivity index achieved an AUC of 0.858 (95% CI: 0.763–0.952), indicating good discriminatory ability.

Conclusions:

PDUS offers reliable hemodynamic markers for cirrhosis detection. The portal vein congestion index shows the highest diagnostic accuracy, while a composite model of portal, hepatic vein, and arterial parameters provides robust discriminatory performance in Filipinos. PDUS may complement elastography in cirrhosis assessment, especially in resource-limited settings.

Keyword(s) : #PDUS #Liver Cirrhosis

2426 - Association of Immune Nutritional Status with Histological Stage and Mortality in Patients with Metabolic Dysfunction Associated Steatotic Liver Disease A Population Based Cohort Study

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Background / Hypothesis: Nutritional-metabolic dysregulation and chronic inflammation are central mechanisms in the progression of metabolic dysfunction-associated steatotic liver disease (MASLD). The Prognostic Nutritional Index (PNI) reflects both the nutritional status and immune function. This study aimed to investigate the associations of PNI with histological severity and mortality in patients with MASLD.

Materials and Methods: The cross-sectional cohort included 969 individuals with MASLD who underwent liver biopsy, from 7 medical centers across China from 2004 to 2024. Logistic regression and restricted cubic splines were used to investigate the association between PNI levels and significant steatosis, moderate-to-severe inflammation and advanced fibrosis. The relationship between PNI and all-cause and cardiovascular-related mortality were subsequently assessed by Cox regression analysis in the NHANES III follow-up cohort.

Results: Univariable and multivariable logistic regression analyses showed that higher PNI levels were

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independently associated with the presence of significant steatosis. When analyzed as a continuous variable, PNI exhibited a linear relationship with the risk of developing significant steatosis (P for nonlinear =0.209). In contrast, lower PNI levels were significantly associated with moderate-to-severe inflammation, with risk increasing linearly as PNI decreased (P for nonlinear =0.052). Although lower PNI levels were significantly associated with advanced fibrosis in univariable analyses only, RCS analyses indicated a linear increase in the risk of advanced fibrosis with decreasing PNI levels (P for nonlinear =0.068). In the NHANES III cohort, Lower PNI categories were significantly associated with an increased risk of all-cause mortality among patients with MASLD, particularly in those aged over 45 years, with concomitant diabetes, and with a FIB-4 score ≥ 1.3 . In contrast, higher PNI categories were associated with an increased risk of cardiovascular mortality in obese patients with MASLD.

Conclusions: Higher PNI levels were associated with significant steatosis, whereas lower PNI levels were linked to hepatic inflammation, fibrosis, and poorer clinical outcomes in patients with MASLD.

Keyword(s) : Metabolic Dysfunction-Associated Fatty Liver Disease; Prognostic Nutritional Index; steatosis; inflammation; fibrosis

2427 - Endoscopic Ultrasound-Guided Portosystemic Pressure Gradient Measurement To Determine Candidacy For Simultaneous Liver-Kidney Transplantation Vs Renal Transplant Alone In End Stage Renal Disease Patients With Compensated Advanced Chronic Liver Disease or Cirrhosis

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Background:

In patients with ESRD and suspected cACLD or cirrhosis, assessment of clinically significant portal hypertension(CSPH) is crucial in guiding clinical decisions regarding transplant. Portosystemic pressure gradient(PPG) assessment makes EUS an effective alternative to TJLB and HVP. Since the proprietary digital manometer is unavailable in India, we describe the technical feasibility, efficacy and safety of EUS-PPG using modified non-proprietary setup (after its ex-vivo validation) in ESRD patients [Figure 1].

Materials and Methods:
Data of patients with ESRD and suspected cACLD(post-dialysis LSM>10 kPa) or cirrhosis who underwent EUS-PPG assessment over 3-years were reviewed. EUS-PPG was performed using a modified setup consisting of 25G EUS-FNA needle, pressure transducer and multiparametric monitor with pressure module [Figure 2]. Primary outcome was technical success (at-least 3 concordant readings of systemic and portal pressures). Secondary outcomes included safety of procedure and correlation of PPG with post-dialysis LSM and histological fibrosis.

Results:

EUS-PPG was performed in 29 ESRD patients[males: 79.3%, age: 31.5 (22-39) years]. The mean post-dialysis LSM was 18.33 (15.5-20.3) kPa. Technical success was 100%. Mean PPG was 4.73 ± 3.9 mm of Hg. Portal hypertension was present in 6 patients with CSPH in 3 patients. On histology F1, F2, F3 and F4 fibrosis was present in 2, 21, 3 and 3 patients, respectively. One patient with CSPH (PPG: 20 mm of Hg) had PSVD. After excluding this patient, PPG showed good correlation with fibrosis stage ($\rho: 0.75$, $p<0.001$)[Figure 3]. Based on histology and PPG, 2 patients were allocated to SLKT and 27 patients for renal-transplant. None of the patients had any procedure-related adverse event.

Conclusions:

EUS-PPG measurement using a non-proprietary setup is technically feasible, safe and offers valuable guidance in deciding between SLKT and kidney-only transplantation.

Keyword(s) : Endoscopic ultrasound, Portosystemic pressure gradient, Compensated advanced chronic liver disease, End stage renal disease

2432 - Endoscopic Ultrasound Guided Ablative Therapies For Hepatocellular Carcinoma

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Background

Hypothesis:

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Endoscopic ultrasound (EUS) has emerged as an important modality in endohepatology, offering high-resolution, real-time imaging with therapeutic capabilities. While percutaneous ablation remains the standard approach for hepatocellular carcinoma (HCC), lesions located in anatomically challenging regions such as the caudate lobe, left lateral segments, or periportal and retroperitoneal lymph nodes may be difficult or unsafe to access. This study aimed to evaluate the feasibility, safety, and efficacy of EUS-guided ablation in patients with hepatocellular carcinoma, focusing on technically challenging lesion sites and individualized therapeutic approaches.

Materials and Methods: We retrospectively reviewed data of all HCC patients who underwent EUS-guided thermal or non-thermal ablation at a tertiary care center between 2023 and 2025. The decision to perform EUS-guided ablation was made through multidisciplinary discussion. Patients with CTP >7B, ascites, platelet count <50×10⁹/L, INR > 2.0 were excluded. EUS was performed using linear-array EUS under anesthesia support, with lesion characterization aided by contrast-enhanced EUS. Radiofrequency ablation or ethanol injection was selected based on lesion characteristics and proximity to vascular structures. The primary outcome was radiological response at one month, assessed using mRECIST 1.1 and LI-RADS criteria [Figure 1,2]. Secondary outcomes included technical success, safety, and clinical outcomes.

Results: Five patients underwent EUS-guided ablation during the study period [Table 1]. Two patients underwent radiofrequency ablation for caudate lobe lesions, one patient received ethanol ablation for a segment I lesion, and two patients underwent ethanol ablation for abdominal lymph nodal metastases. Technical success was achieved in all cases (100%). Follow-up imaging demonstrated complete radiological response in four patients, accompanied by biochemical response with normalization of alpha-fetoprotein levels. One patient was lost to follow-up. No peri-procedural or post-procedural adverse events were observed. During follow-up, one patient developed pulmonary metastasis and was initiated on systemic therapy.

Conclusions: EUS-guided ablative therapy appears to be a feasible, safe, and effective option for carefully selected patients

with HCC located in anatomically challenging regions. Although limited by small sample size, these findings support EUS-guided ablation as a valuable complementary approach to percutaneous techniques, warranting further evaluation in larger prospective studies.

Keyword(s) : Endoscopic ultrasound, Radiofrequency ablation, Ethanol ablation, Hepatocellular carcinoma

2433 - Diagnostic performance of non invasive fibrosis scores in treatment naïve patients with autoimmune hepatitis

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Background / Hypothesis: Autoimmune hepatitis is a chronic immune-inflammatory disease of the liver. The most important stage indicating the progression of chronic liver diseases to these complications is the development of fibrosis in the liver. Due to the invasive nature of liver biopsy and the risk of complications, the use of non-invasive fibrosis markers has become prominent in recent years. Due to its rarity, the number of studies comparing the predictive power of these markers for liver fibrosis in autoimmune hepatitis is extremely limited.

Materials and Methods: Liver biopsy specimens of patients diagnosed with autoimmune hepatitis in our clinic were included in the study. Patients with radiological or laboratory findings suggestive of overlap syndromes were excluded. All liver biopsies were evaluated in a blinded manner according to the METAVIR fibrosis scoring system by an expert hepatopathologist. Laboratory parameters obtained concurrently with liver biopsy were retrospectively collected from the patients' electronic medical records. The APRI, FIB-4, GPR, and eLIFT scores were calculated using standard formulas. The diagnostic performance of these non-invasive scores and serum ferritin levels for predicting the presence of significant fibrosis (METAVIR ≥F2) and advanced fibrosis (METAVIR ≥F3) was assessed using receiver operating characteristic (ROC) curve analysis

Results:



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A total of 161 patients were included in the study. The mean age was 44.9 ± 1.4 years, and 77.6% of the patients were female. Histopathological examination revealed significant fibrosis in 113 patients (70.2%), while advanced fibrosis was present in 53 patients (32.9%).

In correlation analyses, FIB-4, APRI, GPR, and eLIFT scores, as well as ferritin levels, showed statistically significant correlations with METAVIR fibrosis stage (all $p < 0.05$). However, the strength of these correlations was modest, with Spearman's rho coefficients ranging between 0.20 and 0.33. ROC analysis demonstrated that the eLIFT score had a higher diagnostic accuracy than FIB-4, APRI, and GPR for the detection of significant fibrosis, with AUC values of 0.68, 0.65, 0.63, and 0.62, respectively ($p < 0.05$). In contrast, FIB-4 showed the highest diagnostic performance for detecting advanced fibrosis, with an AUC of 0.66 ($p < 0.05$).

Conclusions:

eLIFT demonstrated higher diagnostic accuracy, particularly for the detection of significant fibrosis. In contrast, the AUC values of FIB-4, APRI, and GPR were lower than those reported in studies involving patients with chronic viral hepatitis. This discrepancy may be explained by the distinct pathophysiological characteristics of autoimmune hepatitis, in which fluctuating necroinflammatory activity can substantially influence serum-based fibrosis indices.

Keyword(s) : autoimmune hepatitis, non-invasive, eLIFT, fibrosis

2438 - Long term Serial Liver Stiffness Monitoring Histological Fibrosis and HVPG as Determinants of Disease Progression and Decompensation in MASLD

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Background / Hypothesis:
The impact of disease progression in metabolic dysfunction associated steatotic liver disease (MASLD) on liver outcomes remains poorly understood. We aimed to investigate MASLD progression using longitudinal liver stiffness measurements (LSM) by serial transient elastography (TE), histological fibrosis stage, HVPG and splenic stiffness measurement (SSM) and to evaluate their association with adverse liver outcomes.

Materials and Methods:
All adult patients with MASLD undergoing at least two serial LSM, spaced ≥ 5 years apart (from first to last), were identified from our institutional database for clinical evaluation. Progression and regression were defined as $\geq 19\%$ or $\leq -19\%$ change from baseline LSM, respectively, per QIBA consensus. Associations between baseline LSM-defined MASLD stages (Baveno VII: no cACLD, cACLD, CSPH), biopsy-proven cirrhosis (F4), and Δ LSM with clinical decompensation (CD) after final LSM stratified by HVPG and splenic stiffness response to carvedilol were assessed using time-to-event analysis.

Results:

1119 MASLD participants underwent serial LSMs (76% male, median age 47 years, 28% diabetic, baseline F4 fibrosis, Fib4 and NFS 30.7%, 2.36 and -1.315 respectively). The median time between first to last LSM was 62.3 (49.4–72.1) months. MASLD progression and regression occurred in 388(38%) and 293 (28.7%) participants respectively. Among cACLD participants ($n=634$; 56.7%), 47.7% had cirrhosis (F4 fibrosis) and 73.2% had advanced ($\geq F3$) fibrosis. Participants with F4 fibrosis was associated with more frequent CSPH (Baveno VII criteria) (25.8% vs. 20.6%; $p=0.04$) and lower Δ LSM regression (28.7% vs. 43.8%; $p=0.02$). In MASLD without cirrhosis ($\leq F3$) at baseline, cirrhosis development occurred in 25% of LSM progressors and 11.1% of non-progressors ($p=0.05$) over a median duration of 44(17–78) months. The incidence of CD after the last LSM was higher in participants with baseline CSPH (42.2%) as compared to cACLD alone (37.4%) and no cACLD (4.8%) ($p<0.05$). Participants with incident CD had higher baseline HVPG (14.7 ± 2.8 vs. 12.1 ± 2.9 mmHg; $p<0.001$) and SSM (66.2 vs. 45 kPa; $p<0.001$). A hemodynamic response to carvedilol, defined by a $\geq 20\%$ reduction in HVPG (achieved in 42.7% of patients; median follow-up 21.1 months), but not by SSM criteria, was associated with a significantly lower incidence of variceal bleeding (7.1% vs. 18.1%; $p=0.042$) and new-onset ascites (7.1% vs. 27.7%; $p<0.001$).

Conclusions: Serial liver stiffness measurements over long-term follow up capture disease dynamics in MASLD. Baseline histological fibrosis stage remains a key determinant of portal hypertension severity and subsequent progression. HVPG is the strongest predictor of decompensation and therapeutic benefit.

Keyword(s) :

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2439 - A silent threat A case of infiltrative cholangiocellular carcinoma developing without detectable mass

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Background:

Cholangiocellular carcinoma (CCA) is an aggressive malignancy arising from the biliary epithelium and typically follows a silent clinical course until advanced stages. Patients present with non-specific symptoms often or isolated laboratory abnormalities. In the infiltrative type, is not detected mass usually on imaging studies. In a patient presenting with ascites, rapid differential diagnosis of malignant causes is critical for early diagnosis and treatment.

Case:

A 64-year-old male complained abdominal distension and pain. Physical examination revealed abdominal distension and generalized tenderness. Laboratory analysis showed elevated CRP (112 mg/L) and CA 19-9 levels (700 U/mL). The high protein content of ascites fluid and low (0.6) serum ascites-albumin gradient (SAA) were consistent with malignancy. Cell count detected 275 PMNL/mm³. Empirical treatment for spontaneous bacterial peritonitis was initiated. PMNL of ascites fluid dropped to 20/mm³. ARB, tuberculosis PCR, and culture were negative. Cytology of the ascite revealed signet-ring like malignant epithelial cells. Immunohistochemically, CK7, MOC31, BERP4, CEA, calretinin were positive; CK20, CDX2, GATA3, TTF-1, PAX8 were negative. Gastroscopy and colonoscopy were normal. Contrast-enhanced abdominal CT showed no mass, but there was widespread peritoneal fluid and increased mesenteric density. PET-CT showed increased FDG uptake (SUVmax 5.3) in the peritoneal surface and omentum. Adenocarcinoma was identified on pathological re-evaluation of the cholecystectomy specimen from one year ago. The patient considered as unresectable CCA, was started on Gemcitabine + Cisplatin chemotherapy.

Discussion:

CCA, is an aggressive malignancy with diagnostic difficulties due to its heterogeneous clinical and radiological features. In this case, the absence of a mass on contrast-enhanced CT, the presence of widespread

fluid in the peritoneum, and increased mesenteric density are typical features of infiltrative CCA, these are significant factors contributing to diagnostic delay. In patients with ascites, low SAAG and high ascites protein level are strong indicators of malignancy. Cytological and immunohistochemical examinations are important in identifying the malignancy. Especially, CK7 positivity and CK20/CDX2 negativity are associated with pancreatobiliary adenocarcinoma. Confirming the diagnosis of malignancy with re-evaluation of cholecystectomy specimen from one year ago, shows insidiousness of CCA. Peritoneal involvement indicates advanced disease and poor prognosis.

Conclusion:

This case is important because it shows the importance of a multidisciplinary approach in patients with CCA with peritoneal involvement and emphasizes the contribution of re-evaluating past pathological samples to the diagnostic and treatment process.

Keyword(s) : Cholangiocellular carcinoma, ascites, serum ascites-albumin gradient

2440 - Clinical outcomes and treatment response in primary biliary cholangitis with and without autoimmune hepatitis features a Thai multicenter 10 year cohort

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Background

Hypothesis: Primary biliary cholangitis is a chronic cholestatic liver disease in which a subset of patients presents with features of autoimmune hepatitis, which is termed PBC-AIH overlap. In such patients, inflammatory activity is more aggressive, treatment requirement is different, and the outcomes might be worse. Data from Southeast Asia remains limited. We aimed to compare treatment

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patterns, 12-month biochemical response, and long-term clinical outcomes between PBC patients with and without AIH features in a Thai multicenter cohort.

Materials and Methods: We performed a retrospective cohort study across Chiang Mai University, Prince of Songkla University, and Khon Kaen University, including all patients diagnosed with PBC from 2015–2024 (n=255). Overlap AIH–PBC was defined by the Paris criteria. The primary outcome was biochemical response at 12 months by AASLD-listed criteria and POISE definition. Secondary outcomes of interest were liver decompensation, HCC, and death. Survival analyses were performed using Kaplan–Meier plots and Cox proportional hazards models.

Results:

Of 255 patients, 104 (41%) had PBC with AIH features (PBC+AIH). These patients were younger, had higher baseline ALT/AST levels, and were more frequently ANA-positive. UDCA was used in 98.8%, and fibrates in 9.1% (13.0% in PBC+AIH vs 6.8% in PBC; $p=0.12$). Biochemical response at 12 months was significantly lower in PBC+AIH by Paris II (72% vs 85%; $p=0.024$) and POISE (40% vs 54%; $p=0.043$). During long-term follow-up, decompensation occurred in 19 patients per group (HR 1.49, 95% CI 0.79–2.81; $p=0.221$), and the incidence of HCC was similar (HR 1.14, 95% CI 0.25–5.11; $p=0.864$). Mortality was higher in PBC+AIH (20 vs 12 patients), with a significantly increased risk (HR 2.77, 95% CI 1.29–5.97; $p=0.009$). In multivariable analysis, independent predictors of decompensation, HCC, or death included PBC+AIH (HR 2.77; $p=0.009$), cirrhosis (HR 2.57; $p=0.011$), elevated INR (HR 5.17; $p<0.001$), and baseline ascites (HR 7.37; $p<0.001$). A biochemical response was predicted to favor outcomes across the Paris I/II, Rotterdam, Toronto, and POISE criteria (HRs 0.19–0.32; all $p<0.05$).

Conclusions:

In this Thai multicenter cohort, PBC with AIH features showed a lower 12-month biochemical response and significantly higher mortality, despite similar risks of decompensation and HCC. Early identification of overlap features and strict assessment of UDCA response at 12 months are critical to guide timely therapy escalation and improve long-term outcomes.

Keyword(s) : primary biliary cholangitis, autoimmune hepatitis overlap, biochemical response, ursodeoxycholic acid, fibrates, Thailand, HCC

2441 - MACHINE LEARNING MODELS IMPROVES MORTALITY AND ANTIMICROBIAL RESISTANCE

PREDICTION IN CULTURE PROVEN SPONTANEOUS BACTERIAL PERITONITIS IN CIRRHOSIS A MULTICENTER COHORT STUDY

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Background / Hypothesis: Spontaneous bacterial peritonitis (SBP), particularly when caused by multidrug-resistant organisms (MDROs) and carbapenem-resistant organisms (CROs), is associated with high short-term mortality in cirrhosis and often necessitates broad-spectrum empiric therapy. Conventional tools (e.g., MELD) do not explicitly incorporate resistance risk, limiting precision in prognostication and antimicrobial decision-making. We developed and validated machine-learning (ML) models to predict (i) mortality, (ii) MDRO infection, and (iii) CRO infection among patients with culture-positive SBP.

Materials and Methods: We conducted a retrospective, multicenter study across 13 centers including consecutive adults admitted with acute decompensation of cirrhosis and culture-positive ascitic fluid (SBP) between 2021–2025 (n=319).

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Candidate predictors included baseline clinical variables, laboratory parameters, and organ failure markers at diagnosis. Outcomes were 30-day mortality, MDRO SBP, and CRO SBP, defined using international criteria. Models were developed using Extreme Gradient Boosting (XGB) with hyperparameter optimization and 10-fold cross-validation. Discrimination was quantified using AUC (95% CI) and calibration using Brier score. Feature importance and top predictors were identified. Mortality model performance was compared against MELD using the DeLong test.

Results:

Among 319 patients, 39% died within 30-days. MDRO SBP occurred in 53% and CRO SBP in 24%. For mortality prediction, XGB achieved an AUC of 0.880 (0.839–0.916) (Brier score:0.13), outperforming MELD (AUC: 0.766; $p<0.05$), and stratified patients into high- vs low-risk groups with mortality prevalence of 71% vs 13% (risk ratio: 5.46). For MDRO prediction, XGB achieved an AUC of 0.705 (0.573–0.759) (Brier score:0.24), stratifying MDRO prevalence as 73% vs 43% (risk ratio: 1.69). For CRO prediction, XGB achieved an AUC of 0.683 (0.570–0.808) (Brier score:0.17), stratifying CRO prevalence as 33% vs 15% (risk ratio: 2.2). Top mortality predictors included SIRS, CLIF-C OF, ALP, MELD, prothrombin time, MAP, bilirubin, urea, SPO2/FPO2, SOFC, ACLF grade, and respiratory failure; MDRO/CRO predictors included type of SBP, SOFC, HE grade, respiratory failure, CVS failure, public hospitalization, CLIF-C OF, bilirubin, SBP prophylaxis, INR, MELD, and rifaximin.

Conclusions:

In this multi-center cohort, XGB provided reasonable prediction of mortality and MDRO/CRO SBP, exceeding MELD for mortality and enabling resistance risk stratification to support antimicrobial decisions and trial enrichment.

Keyword(s) : Spontaneous bacterial peritonitis, Multi-drug resistance, Cirrhosis, Machine learning

2443 - Histological severity is not associated with clinical presentation or outcome in children with autoimmune hepatitis

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Background / Hypothesis: Pediatric autoimmune hepatitis (AIH) presents with heterogeneous clinical phenotypes ranging from acute liver failure to chronic liver disease. The relationship between histological severity, clinical presentation, treatment response, and outcomes remains unclear. We evaluated whether histological features correlate with presentation patterns and short-term outcomes in children with AIH.

Materials and Methods: We performed a retrospective analysis of prospectively collected data of children diagnosed with AIH between January 2011 and July 2025. AIH was diagnosed based on compatible clinical and laboratory features, elevated IgG, simplified AIH score ≥ 6 , supportive histology, and exclusion of other liver diseases. Liver biopsies ($n=235$) were reviewed and histological features compared across clinical presentations: acute liver failure/acute-on-chronic liver failure (ALF/ACLF; category 1), decompensated cirrhosis (DCLD; category 2), and acute or chronic hepatitis (AH/CH; category 3). Outcomes assessed included death or liver transplantation and difficult-to-treat (DTT) disease.

Results: Of 235 biopsies analyzed, 69 were from ALF/ACLF, 50 from DCLD, and 116 from AH/CH patients. Baseline fibrosis stage was comparable across groups (mean ~ 2.7 ; $p=0.996$). Most histological parameters—including interface hepatitis, lymphoplasmacytic infiltrate, lobular inflammation, emperipolesis, pseudo-rosette formation, and patterns of necrosis (spotty, bridging, confluent, multiacinar, and pan-acinar)—were similarly distributed across clinical categories (all $p>0.05$). Biliary features, including ductular reaction, bile duct injury or loss, cholangiolitis, and periductal fibrosis, did not differ significantly. Cholestasis showed a non-significant trend toward higher frequency in ALF/ACLF. Overlap features were significantly less frequent in ALF/ACLF compared with DCLD and AH/CH ($p<0.01$). Comparing biochemical characteristics, children with ALF/ACLF were characterized by greater biochemical severity (high PELD/MELD scores, high bilirubin, INR, and a lower albumin), while histological and elastographic parameters remained similar across presentations. At 12 months, 30 patients were lost to follow-up; among 202 evaluable children, 9 underwent LT and 28 died. ALF/ACLF patients had higher necro-inflammatory activity index (HAI) ($p=NS$), however this severity did not correlated with outcomes. Remission rates were highest in the AH/CH group ($p=NS$), despite lower baseline HAI. Difficult to treat (DTT) rates were comparable across ALF/ACLF, DCLD, and AH/CH groups (13%, 12%, and

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13%, respectively; p=NS).

Conclusions: Baseline histological severity does not correlate with clinical phenotype or outcomes in pediatric autoimmune hepatitis. These findings highlight that liver histology reflects disease activity but has limited value in predicting disease course or prognosis.

Keyword(s) : histology, AIH, pediatric

2450 - A rare case Sporadic Burkitt lymphoma presenting with high serum ascite albumin gradient and low ascite glucose

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Background:

Burkitt lymphoma (BL) is an aggressive B-cell lymphoma with three clinical variants: endemic, sporadic, and immunodeficiency-associated. Endemic BL commonly involves the jaw, abdomen, and central nervous system, whereas abdominal involvement occurs in 60-80% of sporadic cases. Ileocecal BL may mimic Crohn disease, Behçet disease, intestinal tuberculosis.

Case:

An 18-year-old male presented with abdominal pain, nausea, vomiting and diarrhea. He had no fever, night sweats, weight loss. His father had history of Hodgkin lymphoma. Physical examination revealed only diffuse abdominal tenderness. Laboratory tests showed elevated serum LDH (486 U/L) and uric acid (10.8 mg/dL), with other parameters were normal. Ascite analysis demonstrated marked leukocytosis (WBC: $52 \times 10^9/L$), LDH 1627 U/L, extremely low glucose level (simultaneous serum glucose: 69 mg/dL), total protein 3.2 g/dL, albumin 2.1 g/dL. Acid-fast bacilli, tuberculosis PCR, culture were negative. The serum-ascite albumin gradient (SAAG) was 1.5 (>1.1), consistent with mixed ascite. Contrast-enhanced abdominal CT showed widespread free intraperitoneal fluid. Portal Doppler ultrasonography and gastroscopy were normal. Colonoscopy revealed a nodular, fragile mucosal lesion at the cecal base causing ileocecal obstruction. Histopathology showed atypical lymphoid cells positive for CD10, CD20, BCL-6, c-MYC, and negative for CD3, CD5, BCL-2, EBV. Ki-67 100%. The patient diagnosed BL with intestinal involvement.

Discussion:

BL is a rare, aggressive malignancy in adults that frequently involves the abdomen. The patient with nonspecific gastrointestinal symptoms and diffuse ascite, had no classic B symptoms. Although peritoneal lymphomatosis is typically associated with a low SAAG, a high SAAG may occur due to sinusoidal and periportal infiltration, leading to occult portal hypertension. In this case, despite a SAAG ≥ 1.1 , elevated ascite protein and LDH levels were consistent with a mixed ascite pattern. Markedly low ascite glucose level commonly suggests tuberculous peritonitis; however, in this patient, the extreme low level of ascite glucose was attributed to BL and is related to high proliferative activity and Warburg effect. This case highlights that peritoneal lymphomatosis may be overlooked on imaging and emphasize the diagnostic value of ascite analysis.

Conclusion:

BL should be considered in young adults presenting with nonspecific gastrointestinal symptoms and unexplained ascite. Even in the absence of definitive imaging findings, the coexistence of a SAAG ≥ 1.1 and a mixed ascite pattern may indicate peritoneal lymphomatosis with occult portal hypertension. Markedly low ascite glucose should be recognized as a feature not only of tuberculous peritonitis but also of high-turnover aggressive lymphomas.

Keyword(s) : Burkitt lymphoma; mixed ascites; low ascite glucose; peritoneal lymphomatosis; Warburg effect

2454 - Anticancer Effects of Bromelain on HUH7 Hepatocellular Carcinoma Cells through Modulation of the EGFR PI3K Pathway

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Background

Hypothesis: Background: The aim of this study was to evaluate the anticancer effects of bromelain on human hepatocellular carcinoma HUH7 cells and to investigate its potential molecular mechanisms involving the EGFR, PI3K, and apoptotic signaling pathways.

Materials

and

Methods:

HUH7 hepatocellular carcinoma cells were cultured under standard conditions. The cells were treated with

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increasing concentrations of bromelain (25 to 800 μ M), and cell viability was assessed using the CCK-8 assay. The IC₅₀ value of bromelain was calculated based on the cell viability data. Following treatment with the IC₅₀ concentration, the expression levels of EGFR and PI3K, as well as the apoptotic markers caspase-3 and caspase-7, were analyzed and compared to the control group. Statistical significance was set at $p < 0.05$.

Results:

According to the CCK-8 assay, the IC₅₀ value of bromelain in HUH7 cells was determined to be 423 μ M. Treatment with bromelain at this concentration resulted in a significant decrease in EGFR and PI3K expression levels compared to the control group ($p < 0.05$). Conversely, the expression levels of caspase-3 and caspase-7 were significantly increased following bromelain treatment ($p < 0.05$). These findings indicate that bromelain suppresses proliferative signaling pathways and induces apoptosis in HUH7 cells.

Conclusions: This study demonstrates that bromelain reduces cell viability in HUH7 hepatocellular carcinoma cells by inhibiting the EGFR/PI3K signaling pathway and promotes apoptotic cell death through the activation of caspase-3 and caspase-7. These results suggest that bromelain may serve as a potential anticancer agent for the treatment of hepatocellular carcinoma.

Keyword(s) : Bromelain, EGFR, Apoptosis, HUH-7

2457 - Impact of the national Hepatitis B vaccination program on HBsAg seroprevalence in pregnant women and comparison with migrant pregnant women

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Background / Hypothesis: Hepatitis B virus (HBV) is an important infectious agent that may lead to serious outcomes for both the mother and the newborn during pregnancy. In Turkey, routine neonatal HBV vaccination has been performed since 1998. This study aimed to compare HBsAg positivity rates between pregnant women born during the vaccination era (1998 and later) and those born before the introduction of the vaccine (pre-1998). In addition, the prevalence of HBsAg positivity among migrant pregnant women was evaluated.

Materials and Methods: This retrospective descriptive study was conducted using the data of pregnant women who underwent HBsAg testing at single center. Repeated test results from the same individuals, as well as records with missing data, were excluded from the study. Turkish pregnant women were divided into two groups according to birth year: those born in 1998 or later and those born before 1998. HBsAg positivity rates were compared between these groups. Although country-of-origin data were unavailable, HBsAg seroprevalence among migrant pregnant women was evaluated separately and compared with that of Turkish pregnant women.

Results: A total of 66456 pregnant women were included in the study, and HBsAg positivity was detected in 625 individuals (0.94%). Among Turkish pregnant women, the overall HBsAg positivity rate was 0.81%. This rate was 0.41% in those born in 1998 or later and 0.99% in those born before 1998. HBsAg positivity was significantly lower among women born in or after 1998 ($p < 0.001$) (Table 1). Among migrant pregnant women, the HBsAg positivity rate was 1.95%, with 151 positive cases detected among 7758 screened individuals. HBsAg positivity was significantly higher in migrant pregnant women compared with Turkish pregnant women ($p < 0.001$) (Table 2).

Conclusions: HBsAg positivity was found to be significantly lower among pregnant women born in 1998 or later, following the inclusion of the HBV vaccine in the national vaccination program. These findings demonstrate that the effectiveness of the national vaccination program in Turkey. However, the detection of HBsAg positivity, albeit at low rates, even among vaccinated cohorts highlights the continued importance of HBV screening and vaccination strategies in women of reproductive age. The higher HBsAg positivity rate observed among migrant pregnant women further underscores the necessity of maintaining universal HBsAg screening during pregnancy.

Keyword(s) : pregnancy, hepatitis B virus, vaccination

2460 - Comparative Efficacy of Lenvatinib Versus Sorafenib in Advanced Hepatocellular Carcinoma A Meta Analysis of Meta Analyses



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Background / Hypothesis: Advanced hepatocellular carcinoma (HCC) remains a major therapeutic challenge, with tyrosine kinase inhibitors such as lenvatinib and sorafenib forming the backbone of systemic treatment. Although multiple meta-analyses have compared the efficacy of lenvatinib versus sorafenib across various clinical outcomes, their results show considerable variability. This meta-analysis of meta-analyses aims to synthesize the highest level of available evidence to comprehensively evaluate the comparative efficacy of lenvatinib and sorafenib in patients with advanced HCC.

Materials and Methods: A systematic literature search of PubMed, Embase, Scopus, and the Cochrane Library was conducted up to November 2025. Six eligible meta-analyses encompassing a total of 17,144 participants were included. Pooled hazard ratios (HRs) and odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were extracted for overall survival (OS), progression-free survival (PFS), objective response rate (ORR), and disease control rate (DCR). Statistical heterogeneity was assessed using the I^2 statistic. Publication bias was evaluated using Egger's regression test, which demonstrated no evidence of significant bias.

Results: Lenvatinib was not associated with a statistically significant improvement in overall survival compared with sorafenib (HR = 0.89; 95% CI: 0.69–1.08; I^2 = 96.18%; p < 0.01), with substantial heterogeneity across studies. In contrast, lenvatinib demonstrated a significant benefit in progression-free survival (HR = 0.70; 95% CI: 0.58–0.83; I^2 = 92.64%; p < 0.01). Moreover, lenvatinib showed markedly higher objective response rates (OR = 6.55; 95% CI: 5.75–7.35; I^2 = 98.67%; p < 0.01) and improved disease control rates (OR = 3.14; 95% CI: 1.99–4.28; I^2 = 99.74%; p < 0.01) compared with sorafenib, despite high heterogeneity.

Conclusions: This meta-analysis of meta-analyses consolidates current evidence and indicates that lenvatinib offers superior efficacy over sorafenib in terms of progression-free survival, objective response

rate, and disease control rate in advanced hepatocellular carcinoma, while overall survival appears comparable between the two agents. These findings support the preferential use of lenvatinib in selected patients and underscore the need for further high-quality studies to clarify survival benefits amid substantial heterogeneity.

Keyword(s) : Hepatocellular carcinoma, Lenvatinib, Sorafenib, Meta-analysis

2461 - Early GI Hepatology Subspecialty Access in Cirrhosis and Survival A Retrospective Cohort Analysis

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Background / Hypothesis: Cirrhosis is a common, high-mortality chronic liver disease, and its outcomes depend heavily on timely guideline-based care, management of complications, medication optimization, transplant evaluation when appropriate, and coordination of preventive services, such as hepatocellular carcinoma (HCC) surveillance. However, early access to GI/hepatology care is uneven and may be constrained by patient-, provider-, and system-level barriers. However, real-world evidence on the effects of early access to GI/hepatology subspecialty care and patient outcomes remains limited. Therefore, we sought to (i) quantify the rates of early GI/hepatology subspecialty care among patients with cirrhosis and (ii) estimate the effect of early GI/hepatology subspecialty care on all-cause mortality in cirrhosis.

Materials and Methods: This retrospective cohort study utilized the Ohio State

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University PCORnet electronic health record database to identify adults with cirrhosis between 2011 and 2023. We defined early GI/hepatology subspecialty access as ≥ 1 GI/hepatology encounter occurring before or on the index date of cirrhosis diagnosis or within 90 days after diagnosis. Follow-up was defined as the period from 90 days after cirrhosis diagnosis to the date of death, the last encounter date, or the end of the study, whichever occurred first. Overlap Propensity Score Weighting (OPSW) was applied to account for confounding. Crude and adjusted Cox proportional hazard models with robust standard errors were used to assess the association between GI/hepatology access and survival.

Results:

Among 8,013 patients newly diagnosed with cirrhosis, the mean (STD) age was 55.5 (13.1) years, 55.4% were male, and 77.7% self-identified as non-Hispanic White. Only 37.3% of the patients had access to GI/hepatology care. The median (Q1, Q3) follow-up time was 1.8 years (0.6-4.0 years). The mortality risk was 17 deaths per 100 person-years. GI/hepatology access was significantly associated with lower mortality rates (12.0 vs. 20.7 deaths per 100 person-years; risk difference (RD) = 8.5 deaths per 100 person-years [95%CI: 7.4 to 9.6 per 100 person-years]). On crude analysis, GI/hepatology access was associated with a 41% lower risk of all-cause mortality than no GI/hepatology access (aHR: 0.59; 95%CI: 0.55 to 0.63). GI/hepatology access was associated with a 9% lower adjusted risk of all-cause mortality compared with no GI/hepatology access (aHR: 0.91; 95%CI: 0.84 to 0.98).

Conclusions: In this large cohort of patients with newly diagnosed cirrhosis, timely care from GI/hepatology subspecialists was low. After adjusting for confounders, GI/hepatology access remained associated with a significant reduction in all-cause mortality. These findings position early subspecialty engagement as a potentially modifiable lever to improve outcomes.

Keyword(s) :

2463 - Association of Long Term Glycemic Control with the Risk of Developing Cirrhosis in Type 2 Diabetes Mellitus A 5 Year Multi Center Retrospective Cohort Study of Liver Outcomes and Predictive Factors

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Marie

Li

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Background

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Hypothesis:

The liver is increasingly being recognized as a target of end-organ damage in patients with longstanding Type 2 diabetes mellitus (T2DM). Uncontrolled DM, commonly defined by hemoglobin A1c (HbA1c) $\geq 7.0\%$ is known to cause several micro- and macrovascular complications however its propensity to cause liver cirrhosis lacks long-term real-world evidence, especially in Filipino populations. This study investigated the effect of glycemic control on the incidence of cirrhosis over a 5-year follow-up period. To date, there are no Philippine studies that have explored this relationship.

Materials

and

Methods:

The study recruited T2DM outpatients from two tertiary-level hospitals – Cardinal Santos Medical Center and Fatima University Medical Center. 551 diabetic patients were identified through clinic records and databases then individually screened using defined inclusion and exclusion criteria, including absence of cirrhosis or significant fibrosis at baseline and absence of other disease processes known to cause liver fibrogenesis. 70 patients qualified for inclusion in the study. A standardized data collection tool was used wherein HbA1c, liver imaging (ultrasound, CT scan, MRI, vibration-controlled transient elastography/VCTE), body mass index, fasting blood sugar, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, platelet count, total bilirubin, international normalized ratio, albumin, globulin, lipid profile, blood urea nitrogen, creatinine, and microalbuminuria were recorded over a 5-year follow-up period. Data analysis was performed using Stata SE, Version 13. The association between glycemic control and cirrhosis was assessed using logistic regression. Changes in lab parameters from baseline to the end of follow-up were analyzed using paired t-tests.

Results:

Two out of three (66.7%) patients who developed cirrhosis within the follow-up period had uncontrolled DM while 21 out of 67 (31.3%) patients who did not

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progress to cirrhosis had uncontrolled DM. This translates to an odds ratio of 4.38 ($p=0.238$). Comparing the aforementioned lab parameters taken at baseline with those taken at the end of the follow-up period, none were significantly different between the controlled and uncontrolled DM groups. Moreover, none of the patients developed liver-related complications.

Conclusions:

The results show that the odds of developing cirrhosis over a period of at least 5 years are 4.38 times higher in patients with uncontrolled DM compared to those with controlled DM. The authors therefore recommend interval monitoring of HbA1c for all diabetic Filipino outpatients, with imaging for liver fibrosis, ideally VCTE, done periodically for those with uncontrolled HbA1c ($\geq 7.0\%$) in order to detect and prevent progression to cirrhosis and its complications.

Keyword(s) : type 2 diabetes mellitus, glycemic control, HbA1c, fibrosis, cirrhosis

2483 - Endoscopic treatment for esophagogastric variceal bleeding in acute on chronic liver failure patients

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Background / **Hypothesis:**
Endoscopic interventions are vital for managing esophagogastric variceal bleeding (EGVB) in cirrhotic patients with portal hypertension. The role of endoscopic injection sclerotherapy (EIS) remains limited in patients with acute-on-chronic liver failure (ACLF). This study aims to investigate the prognostic factors for patients with EGVB accompanied by ACLF.
Materials and Methods:
In this single-center retrospective cohort study, 106 ACLF patients experiencing EGVB episode between January 1, 2021, and June 30, 2024 were included (Figure A). Patients were assigned to an endoscopic treatment group or a non-endoscopic treatment group. Interventions were performed within 12–24 h after hemodynamic stabilization. The primary endpoints were 6-week mortality, transjugular intrahepatic portosystemic shunt (TIPS) placement, and liver transplantation. Risk factors for early rebleeding (72 h–6 weeks), 6-week mortality, and hemorrhage-related death were assessed by logistic regression.
Results:
Early rebleeding occurred in 36.8% (39/106) of patients.

Moderate-to-severe ascites (OR 3.38, 95%CI 1.38–8.30, $P = 0.008$) and absence of endoscopic treatment (OR 0.27, 95% CI 0.11–0.66, $P = 0.004$) independently predicted early rebleeding (Figure B). Six-week mortality was 46.2% (49/106), which was associated with moderate-to-severe ascites (OR 2.59, 95%CI:1.04–6.42, $P = 0.040$), higher total bilirubin (OR 1.004, 95%CI:1.001–1.008, $P = 0.012$) and non-first episode of gastrointestinal bleeding (OR 0.304, 95%CI:0.12–0.77, $P = 0.012$)(Figure C). Endoscopic treatment did not significantly affect overall survival ($P = 0.734$) but reduced hemorrhage-related mortality (OR 0.186, 95%CI: 0.04–0.86, $P = 0.031$). Among the 49 deceased patients, 20 deaths were attributed to gastrointestinal hemorrhage and 29 to other causes. Besides, patients with portal vein thrombosis (PVT) were more prone to bleeding-related death (OR 8.262, 95%CI:1.51–45.09, $P = 0.015$) (Figure D).

Conclusions:

This study indicates that moderate-to-severe ascites and absence of endoscopic treatment are independent risk factors for early rebleeding in ACLF patients with EGVB, while moderate-to-severe ascites, elevated total bilirubin, and non-first gastrointestinal bleeding increase 6-week mortality. Endoscopic treatment lowered hemorrhage-related mortality; however, it did not significantly improve overall survival.

Keyword(s) :

2484 - CT based Vasomics model for noninvasive diagnosis of Porto sinusoidal vascular disease in patients with portal hypertension

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Background / **Hypothesis:**
Porto-sinusoidal vascular liver disease (PSVD) is often misdiagnosed as cirrhosis, while liver histopathology remains the diagnostic gold standard—making accurate noninvasive differentiation a key unmet need. This study aims to develop a CT-based vasomics model for noninvasive diagnosis of PSVD.
Materials and Methods:
Patients diagnosed with PSVD were retrospectively

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recruited from our center from January 2018 to December 2023. The control cohort enrolled patients with cirrhotic portal hypertension recruiting consecutively during the same period with a sample size matching ratio of 1:2. Patients were randomly divided into the training cohort and the validation cohort. Using vasomics technique, 64 vascular parameters can be extracted for each patient from contrast-enhanced abdominal CT angiography, covering features of the portal vein, hepatic veins, inferior vena cava, hepatic artery, and abdominal aorta. Parameters showing statistically significant differences between patients with PSVD and those with cirrhosis were first identified by univariate analyses. These variables were then entered into regression analyses to develop a noninvasive diagnostic model for PSVD in the training cohort. The diagnostic performance of the vasomics model for PSVD was evaluated in the validation cohort

Results:
A total of 228 patients were included, of which 76 were PSVD patients and 152 were cirrhosis patients. The training cohort comprised 40 patients with PSVD and 80 with cirrhosis, while the validation cohort included 36 patients with PSVD and 72 with cirrhosis. A CT-based vasomics model for noninvasive diagnosis of PSVD was developed based on 2 vascular features from hepatic artery, and abdominal aorta. The vasomics model a good performance in differentiation of PSVD with the AUCs of 0.762 (95%CI: 0.664-0.861) and 0.819 (95%CI: 0.722-0.917) in the training and validation cohorts, respectively (Figure 1).

Conclusions:

CT-based liver vasomics technique is feasible for PSVD and enables extraction of quantitative vascular biomarkers. A CT-derived vasomics score can support noninvasive differentiation of PSVD from cirrhosis, potentially reducing reliance on biopsy and improving diagnostic confidence in patients presenting with portal hypertension.

Keyword(s) : Vasomics; Porto-sinusoidal vascular liver disease; Idiopathic non-cirrhotic portal hypertension; Computed Tomography; Noninvasive diagnosis

2488 - SAFETY OF MIDAZOLAM AND NALBUPHINE IN CIRRHOTIC PATIENTS WHO UNDERGO UPPER GASTROINTESTINAL ENODSCOPY

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Materials and Methods:

ABSTRACT

Introduction: - Cirrhosis of liver and its complication are the major health problem, due to very high prevalence of hepatitis B and hepatitis C in our community and it is the common cause of mortality among Pakistan population and frequent cause of admission to hospital in Pakistan. Cirrhosis is the result of progressive destruction and regeneration of the liver parenchyma due to chronic liver disease (CLD). This study is to design to measure the safety of midazolam and nalbuphine in cirrhotic patients who undergo upper gastrointestinal endoscopy in our population.

Objective: - To compare the safety of midazolam and nalbuphine in cirrhotic patients who undergo upper gastrointestinal (GI) endoscopy, at a tertiary care Hospital.

Setting: Inpatients and out patients Department

Duration: Six months from January 2019 till to date.

Study design: Randomized controlled trial

Subject and Methods: - A total of 150 patients having cirrhosis of liver were randomly allocated into two groups. 75 were in group A and treated with midazolam and 75 in group B were treated with nalbuphine. Patients in both the groups was observed for side effects of these drugs for one and half hour after EGD, safety of each drug was achieved if no side effects of these drugs was observed. All demography, clinical history was recorded on a predesigned Performa.

Results: - The average age of the patients was 44.91 ± 11.38 years. There were 82(54.7%) male and 68(45.3%) female. Rate of desaturation, bradycardia, hepatic encephalopathy, clamminess and nausea/vomiting were significantly high in group B than groups A. Comparison of the safety of midazolam and nalbuphine in cirrhotic patients who undergo upper gastrointestinal (GI) endoscopy. Midazolam was significantly safe treatment than nalbuphine in reducing the side effect.

Conclusion: - The efficacy of midazolam in cirrhotic patients undergoing endoscopy is superior to those of nalbuphine. In safety and efficacy issues, midazolam is better than nalbuphine in GI endoscopy, especially in patients with liver cirrhosis.

Background

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Hypothesis:

patients

with

liver

cirrhosis.

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Results:

Conclusions:

Keyword(s) : Key Words: Cirrhosis of liver, Midazolam, Nelbuphine, GI endoscopy

2491 - Hepatocellular Carcinoma in Armenia Liquid Biopsies and Primary Assessment of Molecular Alterations Prevalence

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Background:

Hepatocellular carcinoma (HCC) exhibits significant geographical variation in incidence, largely driven by differences in underlying risk factors. Among these, mutagenic compounds such as aflatoxin B1 (AFB1), benzopyrene, and aristolochic acid have been linked to specific mutational signatures detectable through DNA sequencing. According to Globocan data, Armenia has the highest incidence of HCC in a vast continental region from Eastern Europe to China.

Materials and Methods:

We focused on detecting risk factors of hepatocellular carcinoma in the Armenian population and major mutations in two key driver genes, TP53 and TERT, in individuals with either HCC (n=112) or liver cirrhosis (LC, n=169).

The study included 112 patients diagnosed with HCC or mixed HCC-CCC between 2018 and 2021, and 169 patients with liver cirrhosis as the control group. Inclusion criteria required a diagnosis of HCC based on clinical, imaging, and biochemical evidence. The methods included DNA and RNA extraction, reverse

transcription, nested PCR, sequencing, droplet digital PCR, phylogenetic analysis, and statistical analysis performed using Prism version 9.3.1.

Results:

Our results showed frequent accumulation of tumor risk factors. Only 15.9% of HCC patients had a single risk factor, while approximately 36% presented with at least 4 risk factors. The predominant genotype among HCV-infected HCC patients was 3a. A particularly high rate of Occult B infection (OBI) was observed among HCC patients (46.6%, $p = 1.058 \times 10^{-5}$). Our results revealed a high frequency of TERT promoter mutations in HCC cases (24%), along with the presence of AFB1-associated TP53 mutations (3%) in free-circulating DNA (fcDNA). In comparison, mutations were significantly less common in LC patients (3% and 7%, respectively, $P=0.0057$). Additionally, the mutation burden (mutated copies/mL) was significantly higher in HCC patients than in those with LC ($P=0.0063$). Among HCC patients, the prevalence of mutations was positively associated with anti-HCV positivity and elevated AFP levels, but inversely correlated with the Hepatic Steatosis Index. These findings suggest a high mutation burden in Armenian HCC patients.

Conclusions:

Circulating mutations in plasma are promising tools for the diagnosis of HCC and for monitoring the treatment of patients in Armenia, and potentially in other populations.

A small proportion of Armenian patients with severe liver disease appears to have been exposed to Aflatoxin B1 (TP53 R249S), suggesting that food products may occasionally be contaminated with Aspergillus species.

Keyword(s) : Hepatocellular carcinoma, hepatitis B virus, hepatitis C virus, tumor mutations, aflatoxin B1, liquid biopsy

2496 - Development and validation of an artificial intelligence driven convolutional neural network for automated stool colour classification to facilitate early diagnosis of biliary atresia

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Background / Hypothesis:

Early recognition of acholic (pale) stools in infants with cholestasis is critical. Misclassification of stool colour by parents and frontline community health workers remains common. To address this gap, we developed and validated an AI-driven convolutional neural network (CNN) capable of automated stool colour classification. The long-term goal is to deploy this tool widely so that caregivers can upload a stool image and receive immediate guidance on whether the stool is pale, prompting earlier medical evaluation.

Materials and Methods:

Pale stools from confirmed biliary atresia patients (2023-2025) were included. Pigmented stools were obtained from healthy infants. Images were captured using parent-owned mobile phones with varying resolution, lighting, and clarity. Stool color was confirmed by 2 pediatric hepatologists. After excluding severely blurred images, 74 pale and 110 pigmented images formed the training set. AI model used a DenseNet121 architecture. A validation cohort of 37 images was labelled by a pediatric hepatologist (gold standard) and compared with classifications by a parent, intern, nurse and the AI model.

Results:

AI model achieved perfect agreement with experts, identical to the nurse ($\kappa = 1.00$; Sensitivity 100%; Specificity 100%; AUROC 1.00). This was followed by stool classification by the intern ($\kappa = 0.94$; Sensitivity 100%, Specificity 93%). Parents showed only moderate agreement ($\kappa = 0.62$) with Sensitivity 74% and Specificity 94%, misclassifying 7/37 stools. AUROC values were 1.00 (AI model and nurse), 0.97 (intern), and 0.84 (parent). Fisher's exact testing confirmed strong association with expert classification for AI model and nurse ($p = 1.6 \times 10^{-10}$) and intern ($p = 3.9 \times 10^{-9}$), with weaker association for parents ($p = 1.1 \times 10^{-4}$)

Conclusions:

Parents show suboptimal correlation with expert, underscoring the need for an automated tool. The DenseNet121 model performed at expert level and can be deployed through QR codes on immunisation cards,

enabling caregivers to upload stool images and receive immediate guidance

Keyword(s) : Biliary atresia, DenseNet121 convolutional learning network, early pale stool identification

2499 - Wilson disease mutation pattern with genotype phenotype correlations in paediatric cohort in south India

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Background / Hypothesis:

Wilson disease (WD) is an autosomal recessive disorder of copper metabolism caused by pathogenic variants in the ATP7B gene. India shows marked genetic heterogeneity, and data focusing on paediatric cohorts from South India remain limited. Understanding regional mutation patterns and genotype-phenotype correlations is important for early diagnosis, family screening, and management.

Objectives:

To describe the spectrum of ATP7B gene mutations and evaluate genotype-phenotype correlation in children diagnosed with Wilson disease in South India

Materials and Methods:

Paediatric patients (<12 years) with a confirmed diagnosis of Wilson disease based on clinical, biochemical, and ophthalmologic criteria were included. Genomic DNA was analyzed using ATP7B gene sequencing. Identified variants were classified as homozygous or compound heterozygous. Clinical phenotype (hepatic, neurological, mixed), age at onset, biochemical parameters, and disease severity were correlated with genotypes.

Results:

Total(n=12)WD patients underwent ATP7B gene analysis. Mean age=10 years, 60% preseted with DCLD. 70% consanguinity present. Pathogenic or likely pathogenic variants were identified in all patients. mutation spectrum demonstrated marked heterogeneity, involving multiple exons across the ATP7B gene, with a predominance of missense variants. Most patients harbored homozygous mutations, reflecting a high rate of autosomal recessive inheritance, while compound heterozygosity was observed in a smaller proportion. Missense mutations constituted the majority of variants,

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followed by nonsense and frameshift mutations. A single intronic variant of uncertain significance was also identified in our cohort. Recurrent mutations were noted in specific exons. c.3182G>A (p.Gly1061Glu) mutation in exon 14 was among the most frequently observed variants, detected both in homozygous and compound heterozygous states.

Another recurrent pathogenic mutation was c.813C>A (p.Cys271Ter) in exon 2, a stop-gain variant leading to premature protein truncation. Variants affecting exon 19, particularly c.4021G>A (p.Gly1341Ser) involving the splice region, were identified in multiple patients, predominantly in the homozygous state. Additional pathogenic or likely pathogenic missense mutations included c.2906G>A (p.Arg969Gln) in exon 13 and c.3236G>T (p.Cys1079Phe) in exon 14. A frameshift mutation, c.528del (p.Ser177HisfsTer25) in exon 2, was identified in the homozygous state and classified as likely pathogenic.

One patient exhibited compound heterozygosity involving variants in exons 13 and 14. An intronic variant c.2731-3T>G in intron 11, classified as a variant of uncertain significance (VUS) and occurred in the homozygous state.

Conclusions:

Overall, no single founder mutation was identified. Despite the presence of recurrent variants, there was no consistent genotype-phenotype correlation across unrelated patients. However, the predominance of homozygous pathogenic variants highlights the importance of genetic testing and family screening in this population.

Keyword(s) : Paediatric wilson disease and genetics, Mutation in paediatric wilson , Genetic Wilson disease in children

2504 - Long term outcomes of patients with Hepatitis D recurrence after liver transplantation

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Background / Hypothesis: Turkey is considered an endemic country for hepatitis D virus (HDV), and liver transplantation (LT) remains an important rescue treatment. Guidelines recommend indefinite hepatitis B immune globulin (HBIG) and nucleos(t)ide analog (NA) treatment to prevent hepatitis D virus (HDV) recurrence after LT. We aimed to present LT outcomes for HDV in Turkey from 6 liver transplantation centres (2010 to 2025).

Materials and Methods: HDV recurrence was defined as becoming HBsAg and HDV-RNA positive after LT. Demographic, serologic, clinical, and virologic parameters were assessed. Patients had received livers from HBsAg negative, anti-HBc-negative donors except for one case who received a HBsAg (+) after talking to patient who accepted the risk. Except one centre (Ege University) at which HBIG was given only for one year all other centres offered HBIG + NA treatment of indefinite duration.

Results: During the 15-year period a total of 4345 LTs were performed (558 cadaveric and 3787 living related). HBV and HBV/HDV associated liver disease were the etiology in 1743 (40%) and 305 (7%) of 4345 LTs, respectively. Post-transplantation HDV recurrence (Post-txR) occurred in 18 patients (5.9%); (median age: 49 [18-66], 16 men, 2 women). LT indication was decompensated liver disease (DLD) in 10 and hepatocellular cancer (HCC) in 8. Post-txR occurred after a median of 27.5 months (1-54 months). HBIG was administered for median 10.5 months (0- 36 months). Except one patient (HBsAg+ donor), post-txR occurred after HBIG discontinuation in all cases. Of 18 patients with post-txR, 10 patients enjoyed a mild disease course despite lack of antiviral treatment. Eight patients died during median 92.5 months follow up (4-158 months). Causes: liver-related (n=2, including one with severe HDV recurrence with ALT >5000U/mL, >100 million and 1 million IU/nmL of HDVRNA and HBVDNA, respectively), biliary stenosis-linked sepsis (n=2), B-cell lymphoma (n=1), COVID-19 (n=1), earthquake (n=1), unknown (n=1). Notably, Koç University had zero HDV recurrence among 11 HDV-related LTs (all living donor, 2018-2025) using their risk-stratified HBIG/NA protocol.

Conclusions: HBsAg recurrence is rare after LT for HDV but can occur with HBIG/NA non-compliance, potentially leading to HDV recurrence. HDV recurrence

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appears to run a mild clinical course in line with early reports from Italy and France (1, 2), though severe recurrence with graft failure may also occur. Latent HDV infection of the liver allograft may be rescued by HBV superinfection up to 4 years after LT as suggested previously (3).

Keyword(s) : Hepatitis D virus, Liver transplantation, Recurrence, HBIg, Prophylaxis

2505 - Lonafarnib as finite treatment for chronic hepatitis D Long term follow up data

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Background / Hypothesis: Hepatitis delta virus (HDV) infection leads to the most severe form of viral hepatitis. New treatment options are urgently needed. The farnesyl transferase inhibitor lonafarnib (LNF) may be an option for finite treatment as it possesses a dual mode of action: it interferes with virion assembly (Glenn et al Science 1992) and also has immunomodulatory properties (Lempp et al, Nat Commun 2019). The aim of this study was to assess if finite treatment with a LNF-based regimen is able to prevent hard endpoints such as complications of liver disease and death from liver disease on long-term follow-up (LTFU).

Materials and Methods: All patients subject to this study had received LNF-based treatment in two phase 2 (LOWR 1 and 2) and one phase 2 (LIFT) studies in Ankara (TR) and NIH (USA), respectively, and one phase 3 (DLIVR) study in Istanbul (TR). LOWR 1 and 2 studies contained 21 and 55 patients, respectively, the LIFT study 26 and the phase 3 Istanbul study contained 27 patients with a LNF-based treatment. Start date used for LTFU (median 79.5 months (0-134, n=97) and survival analysis was EOT date of LNF-based treatment. End of follow-up (EOFU) was set to either the last date of data

collection (March 2025), or the date of liver-related event, the start date of a new potential treatment against HDV or loss to follow-up. Short-term viral response (STVR) was defined as undetectable HDV RNA or a ≥ 2 log decline of HDV RNA compared to baseline and biochemical response (BR) as ALT normalization at off treatment month 6. BR and VR were the co-primary endpoints. Cumulative probabilities of any event occurrence were estimated by the Kaplan-Meier method and compared with the log-rank test. Uni- and multivariate logistic regression models with forward Wald deletion were used to estimate the effect of various on and off-treatment variables on clinical outcome.

Results: STVR was achieved in 12 patients. On LTFU none of these patients developed a clinical event in up to 10 years of follow up. Out of 85 patients without VR+BR, 17(20%) developed an event (Figure, $p < 0.05$ by the log rank test).

Conclusions: Patients achieving a 2 log drop in HDV RNA from baseline and normal ALT at 6 months after stopping any finite lonafarnib-based treatment regimen have no major clinical events (liver decompensation, liver cancer, or death) in up to 10 years of follow up.

Keyword(s) : Hepatitis Delta Virus, Lonafarnib, Chronic Hepatitis Delta, Viral Response, HDV RNA

2506 - Evaluation of anti HCV positivity HCV RNA testing and treatment status among individuals presenting to the hospital

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Background / Hypothesis: Hepatitis C virus (HCV) is an infectious disease that can lead to chronic liver disease, cirrhosis and hepatocellular carcinoma (HCC). It can now be easily treated with direct-acting antivirals, yielding high response rates. However, it is known that some patients remain undiagnosed and therefore unable to receive treatment. Identifying and treating these cases is crucial for microelimination programmes. The aim of this study was to evaluate the test results of individuals who underwent anti-HCV testing for various reasons at a tertiary care hospital, and to assess the status of HCV RNA testing and the initiation of treatment in patients who tested positive.

Materials and Methods: Between October 2023 and December 2025, the test results of individuals who applied to Izmir City Hospital and requested anti-HCV

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testing were examined using an automated system. Anti-HCV results were classified as positive, borderline and negative. Duplicate data were excluded from the analysis. The distribution of test results by age was examined, and the treatment status of patients with positive anti-HCV results was evaluated by reviewing HCV-RNA test requests and reports.

Results: During the study period, anti-HCV testing was requested for 213,023 individuals aged 18–114 years. Anti-HCV positivity was detected in 914 individuals (0.43%). Anti-HCV testing was most frequently requested by surgical departments (405/914, 44.3%), followed by internal medicine clinics (320/914, 35%), gastroenterology (84/914, 9.2%), and infectious diseases clinics (75/914, 8.2%). Overall seropositivity was low but increased with age, particularly among individuals aged ≥ 60 years. HCV RNA testing was requested for 1,248 individuals, with most requests originating from internal medicine departments (1,024/1,248, 82%) and the remainder from surgical departments (224/1,248, 18%). Within internal medicine departments, HCV RNA testing was most frequently requested by infectious diseases clinics (248/1,024, 24.2%), followed by gastroenterology (163/1,024, 16%). Of the 1,248 individuals tested for HCV RNA, 85 (6.8%) were positive. HCV RNA positivity was higher among individuals aged ≥ 46 years and showed a progressive increase with advancing age, with a modest secondary increase observed in the 26–35 age group. Treatment status was determined through a review of the hospital information system and reports, revealing that antiviral treatment had been initiated in 56 patients (65.9%) and it was provided by infectious diseases clinics for 39 patients and by gastroenterology clinics for 17 patients.

Conclusions: Anti-HCV seropositivity was 0.43% and increased with age. Anti-HCV tests were most frequently requested by surgical departments, while HCV RNA testing was mainly requested by internal medicine clinics, likely related to routine preoperative screening. Although treatment was initiated in most HCV RNA-positive patients (65.9%), a proportion remained untreated. These findings indicate losses in the transition from diagnosis to treatment and suggest that reflex HCV RNA testing and effective referral systems may be critical for achieving HCV microelimination.

Keyword(s) : Hepatitis C , microelimination, HCV-RNA, Direct-acting antivirals

2507 - High Treatment Adherence Reduces HCC in Cirrhosis with Additional Benefit from TAF Running

title Adherence Lowers HCC Risk in Cirrhosis

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Background: Adherence to antiviral therapy is essential for viral suppression in chronic hepatitis B (CHB), yet its effect on hepatocellular carcinoma (HCC) remains uncertain. We examined the association between nucleos(t)ide analogue (NA) adherence and HCC risk, focusing on tenofovir alafenamide (TAF) versus entecavir (ETV).

Methods: Treatment-naïve adults with CHB initiating TAF or ETV were identified from the Korean National Health Insurance Service database. Adherence was measured using the medication possession ratio, with high adherence defined as $\geq 90\%$. The primary outcome was HCC incidence.

Results: A total of 28,448 patients were included, of whom 19,001 (66.8%) showed high adherence and 9,447 (33.2%) low adherence. In the overall cohort, HCC incidence was comparable between high- and low-adherence groups (14.89 vs. 13.76 per 1,000 person-years; IRR 1.08, 95% CI 0.95–1.24). Among cirrhotic patients, however, high adherence was associated with substantially lower HCC incidence (32.92 vs. 49.59 per 1,000 person-years; IRR 0.66, 95% CI 0.57–0.78). Multivariate analysis confirmed high adherence as an independent protective factor (HR 0.77, 95% CI 0.66–0.90). Notably, within the high-adherence group, TAF was linked to a significantly lower HCC risk than ETV (HR 0.54 vs. 0.89; log-rank $p < 0.001$), whereas low adherence to either agent conferred no protection. Logistic regression further identified ETV use as a strong predictor of poor adherence (OR 2.80, 95% CI 2.65–2.96).

Conclusions: High adherence to antiviral therapy reduces HCC risk in cirrhotic CHB patients. TAF showed superior outcomes to ETV, reflecting both greater adherence and a potential intrinsic advantage.

Keyword(s) : Chronic hepatitis B; Hepatocellular carcinoma; Antiviral adherence; Tenofovir alafenamide; Entecavir

2508 - Midodrine with albumin improves ascites control in Acute on chronic liver failure A Double Blind Randomized trial (MIDAS trial)

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Background: Ascites is common in patients with acute-on-chronic liver failure (ACLF) and can complicate the course of ACLF due to circulatory dysfunction and hemodynamic compromise. Oral midodrine has been variably reported to improve hemodynamics and reduce complications of ascites in patients with decompensated cirrhosis, but its role in high Model for End-Stage Liver Disease (MELD) ACLF patients remains unknown, which we, thus, aimed to assess.

Materials and Methods: In this single-centre, double-blind, randomised trial, patients with ACLF were assigned 1:1 to receive midodrine (5-7.5 mg TID) or a placebo for 30 days, alongside standard care. Transplant-free survival (TFS) at 1 month was the primary outcome, while secondary outcomes included TFS at 3 months, the proportion of patients achieving ascites control, changes in mean arterial pressure (MAP) and plasma renin activity (PRA) at 1 month, the incidence of cirrhosis complications, and diuretic-related complications. A sub-group analysis of patients who received albumin was performed.

Results: One hundred and thirty ACLF patients were enrolled. Midodrine use was not associated with improvement in TFS at 1 (hazard ratio [HR], 0.99 [95% CI, 0.37-2.65]) and 3 months (HR, 0.93 [95% CI, 0.48-1.8]). Ascites control at day 30 (placebo: 9.2% [95% CI, 3.5-19] vs. (midodrine: 20% [95% CI, 12.3-33.5]; $P=0.08$) and 90 (placebo: 44.6% [95% CI, 32.2-57.5] vs. (midodrine: 53.8% [95% CI, 41-66.3]; $P=0.055$) was comparable. MAP increased significantly in midodrine arm at day 30 (1.9 mmHg vs. 6.9 mmHg; $P=0.003$) with a reduction in PRA levels (delta change: 0.22 ± 1.6 vs. -1.6 ± 2.2 ; $P=0.09$). Any complication occurred in 74% of patients in the placebo arm and 81.5% in the midodrine arm ($P=0.4$), while diuretic-induced complications were seen in 29% and 24.6%, respectively ($P=0.55$). In the subgroup analysis, 25 patients received only placebo, 40 received only albumin, 26 received midodrine alone, and 39 received a combination of midodrine and albumin. Survival was similar across the four groups. However, on Kaplan Meier analysis, control of ascites was noted in 40% (10/25) in placebo only arm, 47.5% (19/40) in albumin-only arm, 46.5% (12/26) in midodrine only arm and 59% (23/39) in midodrine and albumin arm at day 90 (Log rank $P=0.08$). Further, addition of albumin to midodrine reduced the need of ascitic tapping (15.4% vs. 68% in

placebo).

Conclusions: Midodrine in patients with ACLF can increase MAP but has no clinically relevant TFS benefit at day 30 and 90. However, the addition of albumin to midodrine reduced the need for paracentesis in patients with ACLF.

Keyword(s): ACLF, midodrine, APASL, Ascites

2510 - Efficacy of PegIFN α Add on Therapy in Treatment Experienced CHB Patients with Low Level Viremia A Large Prospective Cohort Study
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Background: / **Hypothesis:** Low-level viremia (LLV) is increasingly recognized in chronic hepatitis B (CHB) patients undergoing long-term nucleos(t)ide analog (NA) therapy. Despite achieving partial suppression, these patients remain at a heightened risk for liver fibrosis, cirrhosis, and hepatocellular carcinoma (HCC). Effectively managing LLV to achieve complete virological response and potential functional cure remains a critical clinical challenge.

Materials and Methods: In this prospective cohort study, we consecutively enrolled 2,048 treatment-experienced CHB patients with LLV (defined as serum HBV DNA ≥ 10 IU/mL and $< 2,000$ IU/mL) between December 2021 and January 2025. Patients were categorized into three treatment strategy groups: (1) NA Monotherapy (ETV, TDF, or TAF); (2) NA Combination therapy (ETV+TDF or TAF+ETV); and (3) Add-on Peg-IFN α -2b therapy. Clinical outcomes, including virological response and HBsAg/HBeAg clearance, were monitored over a longitudinal follow-up period.

Results: Baseline analysis showed that higher HBV DNA levels correlated positively with HBsAg levels. Interestingly, the highest FIB-4 scores were observed in the 10-100 IU/mL subgroup, suggesting persistent inflammatory progression even at very low viral loads. HBeAg-negative patients achieved significantly higher virological response rates than HBeAg-positive patients. Among the three regimens, the Peg-IFN α -2b add-on group demonstrated superior efficacy, reaching the highest rates of virological response, HBeAg seroconversion, and HBsAg loss. Furthermore, in

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patients aged >35 years, the Peg-IFN α -2b group still showed significantly higher virological response rates compared to both NA monotherapy and combination groups.

Conclusions:

For CHB patients experiencing LLV during NA treatment, switching to or adding Peg-IFN α -2b is more effective than continuing NA monotherapy or switching to NA combination therapy in achieving virological and serological goals. This strategy appears robust across different age groups, suggesting that age should not be a limiting factor when considering interferon-based therapy to mitigate the risks associated with LLV.

Keyword(s) : CHB, NAs, LLV, Virological Response

2511 - Diagnostic accuracy of blood based non invasive tests for screening of varices in children with portal hypertension A systematic review and meta analysis

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Background / **Hypothesis:**
Portal hypertension (PHT) in children is associated with a high risk of esophageal/gastric varices (EV), which may result in life-threatening upper gastrointestinal bleeding, particularly in those with clinically significant varices (CSV). Upper gastrointestinal endoscopy (UGIE) is the reference standard for variceal detection, but is invasive, resource-intensive, and often requires anaesthesia in children. Non-invasive imaging modalities, such as liver and splenic stiffness measurements, show promise but have limited availability and feasibility, particularly in resource-limited settings. Blood-based non-invasive tests represent an attractive alternative; however, their diagnostic performance in children has not been comprehensively synthesised. Therefore, this study was planned to systematically evaluate and meta-analyse the diagnostic accuracy of blood-based non-invasive tests for predicting EV and CSV in pediatric PHT.

Materials and **Methods:**

We conducted this systematic review and diagnostic accuracy meta-analysis following PRISMA-DTA guidelines. PubMed, Embase, and Scopus were searched from inception to October 2025. Studies evaluating APRI, platelet count, Clinical Prediction Rule (CPR), Varices Prediction Rule (VPR), or King's Variceal Prediction Score (K-VaPS) against UGIE in children (≤ 18 years) with PHT were included. Hierarchical summary receiver operating characteristic (HSROC) models were used to pool sensitivity, specificity, diagnostic odds ratios (DOR), and area under the curve (AUC). Subgroup, sensitivity, and publication bias analyses were performed. Risk of Bias was assessed using QUADAS-2 criteria.

Results:

Twenty-four studies were included [APRI-17 studies (N=1355), Platelet count-11 studies (N=1040), CPR-12 studies (N=944), VPR-7 studies (N=655), K-VaPS-5 studies (N=315)]. APRI demonstrated pooled sensitivities & specificities of 0.77 & 0.71 for EV and 0.72 & 0.61 for CSV, with corresponding AUCs of 0.795 and 0.776, respectively. Platelet count showed pooled sensitivities & specificities of 0.69 & 0.77 for EV and 0.66 & 0.72 for CSV, with AUCs of 0.834 and 0.714, respectively. Among prediction scores, VPR showed highest accuracy for CSV prediction (AUC 0.864), followed by K-VaPS (0.727) and CPR (0.717). Substantial heterogeneity was observed across most of the analyses. Diagnostic accuracy was consistently higher in biliary atresia-exclusive cohorts and Asian populations. A high risk of bias was observed in the flow & timing domain of eight studies. The certainty of evidence ranged from moderate to very low.

Conclusions:

Blood-based non-invasive tests demonstrate moderate accuracy for predicting varices in pediatric PHT and may serve as adjunctive triage tools; however, standardised pediatric thresholds and prospective validation are urgently needed.

Keyword(s) : Pediatric; Portal Hypertension; Platelet count; Aspartate aminotransferase to platelet ratio index; Liver cirrhosis; Varices; Clinically significant varices; Non-invasive screening

2514 - Long term Clinical Outcomes of Delta Hepatitis in A Single Center Experience

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Background / Hypothesis:

Delta hepatitis represents the most aggressive form of chronic viral hepatitis and is associated with rapid progression to cirrhosis, a high risk of hepatocellular carcinoma (HCC), and increased mortality. Real-world long-term data are essential to better understand the natural history of the disease and the clinical impact of antiviral treatment strategies. This study aimed to evaluate long-term clinical outcomes, including hepatocellular carcinoma development and mortality, in patients with delta hepatitis followed at a single center.

Materials and Methods:

This retrospective study included 132 patients with delta hepatitis who were followed at a single center over a fifteen-year period. Demographic characteristics, cirrhosis status at diagnosis, development of cirrhosis and HCC during follow-up, histopathological findings, mortality and non-invasive fibrosis scores were analyzed. Appropriate parametric and non-parametric statistical tests were used according to data distribution.

Results:

The median age of the patients was 54.0 years, and 66.7% were male. Cirrhosis was present at diagnosis in 80.5% of patients. HCC was detected in 5.3% of patients at diagnosis and developed in 12.9% during follow-up, resulting in an overall HCC rate of 18.2%. Patients with cirrhosis at diagnosis had a significantly higher rate of HCC compared with those without cirrhosis (21.9% vs 3.7%, $p=0.027$). When cirrhosis subgroups were evaluated, HCC rates increased significantly from non-cirrhotic patients (3.7%) to compensated cirrhosis (19.8%) and reached the highest level in decompensated cirrhosis (75.0%, $p=0.003$). A total of 98 patients received pegylated interferon therapy. Among patients who received pegylated interferon therapy, mortality was significantly lower compared with those who did not receive pegylated interferon therapy, regardless of virological response (12.2% vs 41.2%, $p<0.001$).

Conclusions:

This single-center long-term follow-up study demonstrates that delta hepatitis is associated with a

high burden of cirrhosis, HCC, and mortality. The risk of HCC increases dramatically with advancing cirrhosis stage. Pegylated interferon therapy was associated with a significant reduction in mortality, emphasizing the importance of early diagnosis, close surveillance, and effective treatment strategies in this high-risk patient population.

Keyword(s) : delta hepatitis, cirrhosis, hepatocellular carcinoma, pegylated interferon, mortality

2518 - Antinuclear Antibodies Positivity Indicates a High inflammatory Phenotype in Primary Biliary Cholangitis A Multicenter Retrospective Analysis

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Background / Hypothesis:

To investigate the impact of antinuclear antibodies (ANAs) status on the clinical and pathological characteristics, short-term treatment response, and long-term prognosis of patients with primary biliary cholangitis (PBC).

Materials and Methods:

A retrospective analysis was performed on 690 PBC patients admitted to eight tertiary medical centers, including Nanjing Second Hospital, from January 2017 to October 2023. Patients were divided into an ANAs-negative group ($n = 96$) and an ANAs-positive group ($n = 594$). Propensity score matching (1:1) was used to balance age and sex between groups. Laboratory parameters, histopathological features, short-term biochemical response, and hepatic decompensation events were compared.

Results:

After matching, each group included 96 patients. Baseline aspartate aminotransferase (AST), γ -glutamyl transpeptidase (GGT), alkaline phosphatase (ALP), and immunoglobulin G (IgG) levels were significantly higher in the ANAs-positive group than in the ANAs-negative group (all $P < 0.05$). The prevalence of gp210 and/or sp100 positivity was also significantly higher in ANAs-positive patients ($P = 0.005$). Histologically, the ANAs-positive group showed a higher incidence of epithelioid granuloma ($P = 0.028$) and more severe interface hepatitis ($P < 0.001$) as well as higher Nakanuma hepatitis activity scores ($P < 0.001$). No significant differences were observed in the 12-month biochemical response rate or hepatic decompensation incidence

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between groups (both $P > 0.05$). Multivariate Cox regression analysis identified platelet count $\leq 0.3 \times \text{ULN}$, albumin $\leq 0.6 \times \text{ULN}$, and fibrosis stage $S \geq S3$ as independent risk factors for hepatic decompensation in PBC patients.

Conclusions:

ANAs-positive PBC patients exhibited a high inflammatory burden phenotype, suggesting that ANAs status may serve as a potential serological marker of disease activity. Its prognostic and therapeutic implications require further confirmation in prospective long-term studies.

Keyword(s) : Primary biliary cholangitis ; Antinuclear antibody ; Pathological characteristics ; Propensity score matching ; Prognosis

2519 - The long term results of EUS guided coil application for fundal varices

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Background / Hypothesis: Chronic liver disease and the associated development of portal hypertension and portal vein thrombosis are important causes of morbidity and mortality. As a result of both portal hypertension and portal or splenic vein thrombosis, esophageal and gastric varices may develop. If these varices are not treated, they can cause bleeding with a high risk of mortality. Band ligation of esophageal varices is a well-established method; however, the treatment of fundal varices is difficult and can only be performed using techniques available in advanced centers.

Materials and Methods: In our study, we aimed to investigate the data and long-term follow-up outcomes of 31 patients who underwent coil application for fundal varices at a single center between 2022 and 2025. Results: A total of 31 patients were included in the study. The mean age of the patients was 59.87 ± 12.23 years. Of the cases, 21 (67.7%) were male and 10 (32.3%) were female. When the etiological distribution was analyzed, the most common causes were metabolic dysfunction associated liver disease (MASLD) and portal vein thrombosis (PVT), each identified in 9 patients (29%). This was followed by alcohol-related liver disease in 8 patients (25.8%) and hepatitis B virus (HBV) infection in 5 patients (16.1%). Twenty patients (64.5%) had a prior history of gastrointestinal bleeding. Patients were

followed up median-range 78 (34-112) weeks. The median MELD score of the patients was calculated as 10 [8-12]. The median number of coils applied was 6 [4-7]. During the follow-up period, 30 patients (96.8%) could be evaluated under follow-up. When laboratory parameters were evaluated, the median urea level was 32 [26-47] mg/dL, creatinine level was 0.98 [0.82-1.13] mg/dL, and albumin level was 3.20 [3.10-3.30] g/dL. The median total bilirubin level was 1.03 [0.61-1.74] mg/dL. Upon examination of hematological parameters, the median neutrophil count was $3.49 [2.21-6.09] \times 10^3/\mu\text{L}$, lymphocyte count was $1.00 [0.68-1.71] \times 10^3/\mu\text{L}$, and platelet count was $99 [68-173] \times 10^3/\mu\text{L}$. The median neutrophil-to-lymphocyte ratio (NLR) was 3.10 [2.26-6.08]. At the end of follow-up period one patient expired due to hepatocellular carcinoma and remaining 30 (96%) patients are still going on follow-up. Conclusions: As demonstrated by the results of our study, endoscopic ultrasound-guided coil application to fundal varices is a safe and effective method that protects patients from the potentially severe mortality risk associated with fundal variceal bleeding. When performed by experienced endoscopists in appropriately selected patients, it represents an effective preventive treatment against the possible risks.

Keyword(s) : Coil, Chronic liver diseases, EUS, Fundal varices

2525 - Adherence to an Admission Care Bundle and Its Impact on Outcomes in Decompensated Cirrhosis A Multicenter Analytical Cross Sectional Study

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Background: Decompensated liver cirrhosis is associated with high inpatient mortality and frequent complications. The British Society of Gastroenterology and British Association for the Study of the Liver (BSG/BASL) admission care bundle aims to standardize early inpatient management. However, real-world outcome data from low and middle-income settings like the Philippines remain limited.

Materials and Methods: This study included 146 adult admissions for decompensated cirrhosis at two tertiary referral hospitals between January 2022 and May 2025. Compliance with the BSG/BASL admission care bundle comprising seven evidence-based interventions implemented within 24 hours of admission. Outcomes

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included in-hospital mortality, length of hospital stay (LOS), acute kidney injury (AKI) and nosocomial infection. Disease severity and inflammatory burden were assessed using the MELD-Na score and the systemic immune-inflammatory index (SII). Multivariable logistic regression was used for mortality, AKI, and infection, and linear regression for LOS, adjusting for age and bundle compliance. MELD-Na and SII were modeled separately.

Results: 17.9% achieved full compliance and 82.1% partial compliance. On multivariable analysis adjusting for age and MELD-Na, full compliance was independently associated with improved in-hospital survival compared with partial compliance (OR 4.26, 95% CI 1.13–16.01, $p = 0.032$). MELD-Na was independently associated with mortality (OR 1.05 95% CI 1.00–1.10, $p = 0.048$). In a separate model adjusting for SII, full compliance remained independently associated with survival (OR 5.29, 95% CI 1.42–19.74), while higher SII was associated with mortality (OR 1.0003, 95% CI 1.00002–1.00058). MELD-Na was independently associated with both AKI and nosocomial infection, whereas bundle compliance was not independently associated with these complications after adjustment. Neither MELD-Na nor SII was independently associated with LOS.

Conclusion: Full adherence to the BSG/BASL admission care bundle is independently associated with improved in-hospital survival in decompensated cirrhosis, while AKI and infection appear primarily driven by underlying disease severity.

Keyword(s) : Decompensated cirrhosis; BSG/BASL; MELD-Na; Systemic immune-inflammatory index

2527 - Prognostic Stratification of Non Ischemic Acute Liver Injury in the Emergency Department A Retrospective Cohort Study

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Background / Hypothesis: Patients presenting to the emergency department (ED) with acute liver injury (ALI) represent a heterogeneous group with variable outcomes. While prognosis is well-defined for specific etiologies like paracetamol toxicity or viral hepatitis, the

early risk stratification of undifferentiated ALI remains challenging. Specifically, the utility of complex scoring systems (e.g., MELD-Na) versus simple static markers in the initial ED assessment is debated. We aimed to evaluate the etiology, clinical characteristics, and predictors of in-hospital mortality in a tertiary cohort of patients with non-ischemic ALI.

Materials and Methods: We performed a retrospective observational study of adult patients presenting to a tertiary referral center (2023–2024) with acute liver injury. Inclusion criteria were broadly defined as ALT > 5x ULN to capture early-stage injury, encompassing both Drug-Induced Liver Injury (DILI) Grade 3 criteria and acute viral hepatitis. To focus on primary hepatic injury, patients with ischemic hepatitis ("shock liver") and obstructive jaundice were strictly excluded. The primary outcome was in-hospital mortality. Univariate and multivariate logistic regression analyses were performed to identify independent predictors of mortality.

Results: The final study cohort included 115 patients (median age 45 years; 51% male). The etiology was viral hepatitis in 36.5% (n=42), drug-induced/toxic liver injury in 25.2% (n=29), and indeterminate/seronegative in 20.0% (n=23). The overall in-hospital mortality rate was 13.0% (n=15). Mortality was significantly higher in patients with toxic/drug-induced etiology (24.1%) compared to viral hepatitis (7.1%). In univariate analysis, non-survivors were significantly older (median 67 vs. 41.5 years, $p < 0.001$) and had higher admission INR (1.32 vs. 1.06, $p < 0.001$). Notably, admission ALT levels were significantly lower in non-survivors (234 U/L) compared to survivors (560 U/L, $p = 0.03$), reflecting extensive hepatocellular necrosis ("burned-out" phenotype) in the most severe cases. In ROC analysis, Age (AUROC 0.83) and INR (AUROC 0.78) were robust predictors of mortality. The MELD-Na score (AUROC 0.74) did not demonstrate superior prognostic accuracy compared to INR alone in this cohort. In multivariate analysis, Age (OR 1.09, 95% CI 1.04–1.15) and INR (OR 6.4, 95% CI 1.6–25.4) remained independent predictors of mortality.

Conclusions: In this cohort of non-ischemic acute liver injury, etiology and synthetic function are the primary determinants of outcome. Drug-induced and indeterminate etiologies carry a significantly higher

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mortality risk than viral hepatitis. We confirm that in the acute ED setting, simple admission markers, specifically Age and INR provide robust prognostic information that is comparable to more complex scoring systems like MELD-Na. Clinicians should remain vigilant in elderly patients with coagulopathy.

Keyword(s) : Acute Liver Injury, emergency, coagulopathy

2531 - Evaluation of Leptospirosis Cases in the Şanlıurfa Region

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Background / Hypothesis: Leptospirosis is a globally prevalent zoonotic disease caused by *Leptospira* spirochetes. It usually manifests as a self-limiting febrile illness, but severe Weil's disease occurs in 5–10% of cases. This study aims to assess the etiology, clinical features, and complications of leptospirosis in the Şanlıurfa region to enhance awareness and reduce morbidity and mortality.

Materials and Methods: Between January 2020 and January 2025, medical records of 40 patients diagnosed with leptospirosis and followed in the Gastroenterology Clinic of Harran University Hospital were retrospectively reviewed. Demographic characteristics, occupational and environmental exposure histories, clinical symptoms, laboratory findings, imaging results, treatment protocols, and prognostic data were analyzed. Statistical evaluations were performed using SPSS version 24.0

Results: The median age of 40 patients included in the study was 43 years (18–78), and 85% (n=34) were male. Most cases lived in rural areas with a history of agricultural or contaminated water exposure. The most common symptoms were abdominal pain (60%), fever (47.5%), fatigue (47.5%), and myalgia (40%). Jaundice was detected in 30% of patients, while renal failure (15%) and alveolar hemorrhage (22.5%) were the main complications in severe cases. Intensive care was required in 18 patients (45%), and despite all interventions, 2 patients died.

Conclusions: Leptospirosis peaks in summer–autumn in the Şanlıurfa region, posing a major health concern linked to agriculture and well water. Elevated bilirubin, renal failure, alveolar hemorrhage, and thrombocytopenia were mortality factors. Early diagnosis and preventive measures in farmers and breeders are vital to lowering disease burden. Further multicenter studies are needed to define its true incidence.

Keyword(s) : Leptospirosis, Şanlıurfa, Weil's disease, Spirochet

2533 - Cross study analysis of HBsAg rebound following treatments of elebsiran BRIL 179 in combination with peginterferon alfa

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Background / Hypothesis: siRNA elebsiran and therapeutic vaccine BRIL-179 are being developed for HBV functional cure. In the randomized phase 2 ENSURE study, elebsiran + peginterferon alfa (PEG-IFN α) achieved higher HBsAg loss rate 24 weeks post end-of-treatment (EOT) compared with PEG-IFN α alone in patients with chronic HBV infection receiving nucleos(t)ide analogues (NAs). In another randomized, phase 2 study (BRIL-179-002), BRIL-179 + PEG-IFN α improved EOT and sustained HBsAg loss compared with PEG-IFN α alone. Eligible patients who met NAs discontinuation criteria (HBsAg loss at two consecutive visits, HBV DNA < LLOQ, ALT < 2 $\times$ ULN and HBeAg negative) in both studies discontinued NAs after a consolidation period (12–24

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weeks) and were followed further.

Materials and Methods: This analysis summarizes post-EOT HBsAg rebound (≥ 0.05 IU/mL) in pooled elebsiran/BRIL-179 + PEG-IFN α cohorts and PEG-IFN α alone cohorts from ENSURE (through 12 weeks off-NAs) and BRIL-179-002 (through 48 weeks off-NAs). Fifty-five patients with HBsAg loss at/after EOT were included: 25 patients received elebsiran + PEG-IFN α , 17 patients received BRIL-179 + PEG-IFN α , and 13 patients received PEG-IFN α alone.

Results:

Among the 55 patients who achieved HBsAg loss, HBsAg rebound occurred in 16 of 42 (38.1%) patients receiving elebsiran/BRIL-179 + PEG-IFN α combinations and 6 of 13 (46.2%) patients receiving PEG-IFN α alone. HBsAg rebound occurred at a similar rate during NAs consolidation and discontinuation periods. The magnitude of HBsAg rebound was limited: the majority of the rebounders in combination cohorts (15/16, 93.8%) had maximum HBsAg level < 10 IU/mL (one at 11.7 IU/mL), and 3/6 rebounders in PEG-IFN α alone cohorts had maximum HBsAg < 10 IU/mL (remaining 3 between 10-100 IU/mL). Across all cohorts, HBsAg rebound accompanied with HBV DNA $\geq$ LLOQ is infrequent (6/55, 10.9%): 5/6 had maximum HBV DNA < 100 IU/mL and normal ALT, 1/6 had maximum HBV DNA of 1210 IU/mL with mild ALT elevation to 93 U/L, and none met protocol-defined NAs retreatment criteria.

Conclusions:

Patients achieving HBsAg loss with elebsiran/BRIL-179 plus PEG-IFN α showed favorable off-treatment clinical outcomes, with most HBsAg rebounds < 10 IU/mL and infrequent HBV DNA rebound without significant ALT elevation. These observations suggest long-lasting post-treatment immunological control and further support safe NAs discontinuation in regimens with novel therapeutic modalities in combination with PEG-IFN α . The clinical profiles of rebounds during NAs consolidation versus post NAs discontinuation were similar, suggesting that potentially shortening or eliminating NAs consolidation may be possible.

Keyword(s) : HBV; HBsAg loss; peginterferon; siRNA; therapeutic vaccine

2534 - ASSESSMENT OF ACCURACY OF PLATELET COUNT SPLEEN DIAMETER RATIO IN PREDICTING THE PRESENCE OF ESOPHAGEAL VARICES IN FILIPINO PATIENTS WITH CIRRHOSIS AT SLMC QC

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Background / Hypothesis: Noninvasive evaluation of esophageal varices (EV) may improve the management of patients with cirrhosis and reduce both the medical and financial burden related to screening. In this single center cross-sectional study, our aim was to retrospectively validate the use of the platelet count/spleen diameter (PC/SD) ratio for the noninvasive diagnosis of esophageal varices.

Materials and Methods: A total of 101 cirrhotic patients underwent screening endoscopy for esophageal varices. In all participants, the platelet count-to-spleen diameter ratio ((N/mm³)/mm) was measured, and its diagnostic accuracy was determined. A cutoff value of 1206 for the platelet count/spleen diameter ratio was calculated in this study using the ROC curve. The diagnostic accuracy of this ratio was also analyzed across subgroups defined by disease severity and etiology.

Results:

The prevalence of esophageal varices was 48.5%. In this study, the calculated cutoff value of 1206 had a sensitivity of 87.76% (95% CI, 75.23-95.37%), 84.62% specificity (95% CI 71.92-93.12%), 84.31% positive predictive value, 88% negative predictive value, 5.70 positive likelihood ratio, and 0.14 negative likelihood ratio for the diagnosis of esophageal varices. Accuracy of the platelet count/spleen diameter ratio was maintained for both severity and etiology of disease subgroups.

Conclusions:

The platelet count/spleen diameter ratio may be a useful noninvasive tool for diagnosing esophageal varices among cirrhotics when endoscopy is unavailable and resources are limited. However, the evidence is insufficient to replace endoscopy as the gold standard for diagnosing esophageal varices in all cirrhotic patients.

Keyword(s) : esophageal varices, platelet count spleen diameter ration, cirrhosis

2541 - Alcoholic Liver Transplants Performed Due to Liver Disease A Single Center Experience

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Background / **Hypothesis:** This study aimed to analyze the demographic, clinical, biochemical, and survival characteristics of patients who underwent liver transplantation (LT) due to alcoholic liver disease, with a particular emphasis on the differences between acute alcoholic hepatitis and cirrhosis.

Material and methods: The clinical and laboratory data, imaging findings, and postoperative outcomes of a total of 124 LT recipients were retrospectively evaluated. Patients were first divided into two groups according to international diagnostic criteria [acute alcoholic hepatitis (AAH; n=34; 27.4%) and cirrhosis (n=90, 72.6%)]. Then, based on the opinions of our center's KN team and a radiologist experienced in the hepatobiliary system, they were reclassified as AAH and cirrhosis and compared. Statistical analyses were performed using the Mann-Whitney U, Chi-square, and Kaplan-Meier survival tests.

Results: Most patients were male (96.8%) and the median age was 53 years. Living donor liver transplantation accounted for 93.5% of cases, and the median MELD-Na score was calculated as 19. In the AAH group, total bilirubin, AST, AST/ALT ratio, GGT, and lymphocyte levels were significantly higher. According to the classification recommended by the transplant team and radiologist, the radiological volume of livers in the cirrhosis group and the explant liver weight were found to be significantly smaller. Survival rates at 1, 3, and 5 years after LT were calculated as 76.6%, 68.9%, and 66.1% (Figure 1a), respectively. Interestingly, the rate of alcohol initiation after LT was lower in the group that developed mortality (p=0.001). The main causes of mortality after LT were pulmonary (n=43) and biliary (n=23) complications. Although no correlation was found between the three classification systems used for AAH and cirrhosis, similar survival results were obtained (Figure 1b, 1c).

Conclusions: LT performed due to alcoholic liver disease yields acceptable long-term survival outcomes. When evaluated in conjunction with literature data, we believe that since the long-term outcomes of patients defined as AAH and cirrhosis are similar, it is appropriate not to base patient evaluation for KN primarily on alcohol

consumption and cessation periods.

Keyword(s) :

2543 - Prevalence and risk factors of esophageal and gastroesophageal varices in myeloproliferative neoplasm patients with portal hypertension in a tertiary government hospital a 5 year cross sectional study

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Background / Hypothesis: Myeloproliferative neoplasms (MPNs) are an established cause of non-cirrhotic portal hypertension and may lead to gastroesophageal varices with significant clinical consequences. However, local data on the burden of varices and associated risk factors among MPN patients remain limited.

Materials and Methods: This cross-sectional study included 40 adult patients diagnosed with MPNs who underwent esophagogastroduodenoscopy at a tertiary government hospital between 2021 and 2025. Sociodemographic, clinical, laboratory, and endoscopic data were obtained through chart review. The prevalence of gastroesophageal varices was determined. Logistic regression analyses were performed to identify clinicodemographic factors associated with the presence of varices.

Results: Varices were identified in 23 patients (57.5%), including esophageal varices alone in 27.5%, gastric varices alone in 2.5%, and combined esophageal and gastric varices in 27.5%. Varices were most prevalent among patients with primary myelofibrosis (78.9%), followed by polycythemia vera (45.5%) and chronic myeloid leukemia (30%). Variceal size differed significantly across MPN subtypes (p=0.001), with larger varices observed in polycythemia vera and primary myelofibrosis. In univariate analysis, age 40–59 years (OR: 0.06; 95% CI: 0.01–0.57; p=0.015) and >60 years (OR: 0.03; 95% CI: 0.00–0.38; p=0.006) were associated with lower odds of varices compared with younger patients. Splenic vein diameter was positively associated with varices (OR: 1.38; 95% CI: 1.05–1.80; p=0.020), while platelet count showed an inverse association (OR: 0.99; 95% CI: 0.99–1.00; p=0.004). On multivariate analysis, only platelet count remained independently associated with varices (adjusted OR: 0.99; 95% CI: 0.98–1.00; p=0.020).

Conclusions: Esophageal and gastroesophageal varices are highly

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prevalent among MPN patients with portal hypertension, particularly in primary myelofibrosis. Thrombocytopenia is an independent factor associated with variceal presence and may serve as a practical marker to guide endoscopic surveillance in this high-risk population.

Keyword(s) : Myeloproliferative Neoplasm, Esophageal varices

2553 - EUS guided liver biopsy for diffuse liver diseases is an emerging alternative to percutaneous liver biopsy

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Background and Hypothesis : EUS guided liver biopsy for diffuse liver diseases is an emerging alternative to ancillary percutaneous one. We compared the efficacy and adverse events of this new technique in 157 patients against a historical cohort of 1870 patients who had percutaneous biopsy in gastroenterology clinic. **Methods:** Beginning five years ago we suggested EUS-guided biopsy instead of percutaneous one in candidates. We explained advantages for the sake of cost. 157 out of 201 accepted. All EUS examinations were performed using a linear echoendoscope (EG-3870UTK and EG38-J10UT Linear-Array Ultrasound Gastroscope, Pentax Europe GmbH, Hamburg, Germany). For 34 we used 22 G FNB and for the rest (123), 19 G FNB. We performed dry-technique with capillarization method. We did only one pass. For percutaneous biopsy, we performed prior intercostal transabdominal ultrasound examination to choose the site of biopsy in expiration and to look for intervening structures as well as focal liver lesions. After local cleaning, we inject local anesthetic for cutaneous anesthesia in supine position and then we make certain of the site we puncture. Afterwards we made a millimetric cut with scalpel and we introduce either 16 or 18 G needle. We apply negative pressure to the syringe and ask the patient to take a breath, exhale and hold. Finally we push the needle into the liver and withdraw quickly. We disattach the syringe and inject saline to push the tissue, if present, into the bottle filled with formaline. We discard everything if enough tissue is obtained. If not, we repeat the procedure. For EUS-guided biopsy, we performed linear EUS examination in deep sedation. We visualized left lobe of the liver. We chose a site without significant vascularity and we made one pass with either 22 or 19 G FNB

needle (EUS-FNB Trident™ needle; Micro-Tech Medical, Nanjing, China).

Results: The median portal area number was 9 (None to 18) in percutaneous and 57 (range, 8-102) in EUS-guided biopsy. While the number of patients with insufficient tissue was 4% (76/1871) in percutaneous biopsy, it was 3 (all with 22 G needle) in 157 EUS-guided biopsies. Adverse effects happened in 143 out of 1871 percutaneous biopsies (7.6 %): 133 Pain, 5 intraabdominal bleeding, 3 bile peritonitis, 1 pneumothorax, 1 hemobilia. No adverse event happened in EUS-guided biopsies. The number of patients who were admitted overnight was 221/1871 (11%) in percutaneous biopsies while it was none in EUS-guided biopsy.

Conclusion: In comparison to percutaneous one, EUS-guided liver biopsy cutting needle is significantly more yielding and have less (actually none) side effects and 19 G needle should be preferred over 22.

Keyword(s) : EUS guided liver biopsy, Diffuse liver diseases, Percutaneous liver biopsy, Tissue adequacy

2554 - A Preclinical study on Protective Effects of Atorvastatin Against Methotrexate Induced Hepato Renal Toxicity Role of Oxidative Stress and Inflammation

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Background / Hypothesis: Methotrexate (MTX) is widely used as an immunosuppressive and anticancer agent; however, its clinical utility is often limited by dose-dependent hepatotoxicity and nephrotoxicity. Accumulating evidence suggests that oxidative stress and inflammation are central to MTX-induced organ injury. Atorvastatin, a commonly prescribed HMG-CoA reductase inhibitor, exhibits pleiotropic antioxidant and anti-inflammatory properties beyond lipid lowering. The present study investigated the potential hepatoprotective and nephroprotective effects of atorvastatin in an experimental model of MTX-induced toxicity and compared its efficacy with silymarin, a standard reference drug.

Materials and Methods: Wistar albino rats were divided into six groups (n=6): normal control, atorvastatin alone (10 mg/kg), MTX control (20 mg/kg, single intraperitoneal dose), MTX plus atorvastatin (3 mg/kg), MTX plus atorvastatin (10 mg/kg), and MTX plus silymarin (300 mg/kg). Atorvastatin and silymarin were administered orally for 7 days prior to and 21 days following MTX

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administration. Hepatic and renal function was assessed using serum biochemical markers. Oxidative stress parameters (TBARS, GSH, SOD) and inflammatory mediators (nitrite, MPO, TNF- α , PGE₂) were evaluated in liver and kidney tissues. Histopathological examination was performed to assess structural damage.

Results:

Methotrexate administration resulted in significant hepato-renal injury, evidenced by deranged serum biochemical markers, increased oxidative stress, elevated inflammatory mediators, and pronounced histopathological alterations in both liver and kidney tissues. Atorvastatin treatment significantly attenuated MTX-induced inflammatory responses, as demonstrated by reduced tissue levels of TNF- α , MPO, COX-2, and PGE₂ in both organs. However, in the liver, atorvastatin was associated with a paradoxical increase in oxidative stress, accompanied by persistent elevation of liver function parameters and disruption of redox homeostasis. In contrast, renal tissues showed improvement in oxidative stress markers and functional indices following atorvastatin treatment. Histopathological evaluation supported the biochemical findings, revealing partial restoration of tissue architecture, with more pronounced protection observed in the kidney. Collectively, these results indicate that atorvastatin exerts organ-specific effects in MTX-induced toxicity, providing anti-inflammatory benefits while potentially promoting pro-oxidant effects in the liver under conditions of MTX-associated oxidative stress.

Conclusions:

These findings demonstrate that methotrexate induces significant hepato-renal toxicity mediated by oxidative stress and inflammation. Atorvastatin exhibits marked anti-inflammatory effects in both liver and kidney; however, its influence on oxidative homeostasis is organ-specific. While clear renoprotection is observed, atorvastatin may exacerbate hepatic oxidative stress and functional impairment under methotrexate-induced conditions. This context-dependent response warrants caution when considering atorvastatin as an adjunct to methotrexate therapy and highlights the importance of organ-specific safety evaluation.

Keyword(s) :

2559 - Evaluation of a FIB 4–Based Clinical Pathway for Fibrosis Assessment in Patients with Coronary Disease

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Background / Hypothesis: A stepwise clinical pathway using the FIB-4 index as a first-line screening tool, followed by vibration-controlled transient elastography (VCTE), has recently been recommended in a Circulation consensus statement for metabolic dysfunction–associated steatotic liver disease (MASLD) screening among patients with cardiovascular disease. However, real-world validation of this strategy remains limited. We aimed to evaluate the applicability and potential limitations of this FIB-4–guided clinical pathway in patients with coronary heart disease (CHD).

Materials and Methods: Between December 1 and December 30, 2025, consecutive CHD patients hospitalized in the Department of Cardiology at Beijing Anzhen Hospital, China, were prospectively recruited. All patients underwent concurrent FIB-4 calculation and VCTE assessment (iLivTouch, Hisky Medical, China). According to the recommended pathway, patients with low FIB-4 were classified as low risk and not prioritized for elastography, whereas those with elevated FIB-4 were referred for further VCTE evaluation. The proportion and characteristics of patients potentially misclassified by this pathway were analyzed.

Results: Among 346 CHD patients, 71.1%, 25.4%, and 3.5% were classified as low, intermediate, and high risk by FIB-4, respectively. Among patients with intermediate and high FIB-4 risk, 9.0% and 14.0% were confirmed as having intermediate or high fibrosis risk by VCTE. Notably, among patients classified as low risk by FIB-4, 9.35% and 6.5% were reclassified as intermediate and high risk by VCTE, suggesting potential underestimation of fibrosis when relying solely on initial FIB-4 screening. Compared with concordant low-risk patients, these discordant patients were more likely to be aged ≥ 70 years (OR 3.03, 95% CI 1.30–7.06; $p=0.010$) and to have BMI ≥ 28 kg/m² (OR 4.47, 95% CI 1.55–12.89; $p=0.006$).

Conclusions: The FIB-4–guided clinical pathway effectively stratifies fibrosis risk in the majority of CHD patients; however, a clinically meaningful minority may

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be misclassified. Selective incorporation of VCTE in low FIB-4 patients with advanced age or obesity may improve fibrosis detection. These findings support refinement, rather than replacement, of current stepwise assessment strategies for MASLD in CHD populations.

Keyword(s) : MASLD; coronary heart disease; FIB-4; fibrosis assessment; clinical pathway

2560 - Predictive potential of hypermethylation of RASSF1A and CCND2 promoters in hepatitis B virus induced hepatocellular carcinoma

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Background / **Hypothesis:**
Hepatitis B virus (HBV) infection is a major cause of hepatocellular carcinoma (HCC) globally; however, the epigenetic mechanisms driving chronic hepatitis' progression to cirrhosis and HCC remain elusive. Hypermethylation of the promoters of the cell cycle regulators RASSF1A (tumour suppressor gene) and CCND2 (proto-oncogene), leading to their dysregulation, has been correlated with uncontrolled cell proliferation and detected in various cancer types. Since research examining a possible link between promoter methylation of these genes and HCC in HBV-infected patients is sparse in Bangladesh, this study aimed to bridge that gap and investigate a potential correlation between promoter hypermethylation of RASSF1A and CCND2 and HBV-induced HCC.

Materials and **Methods:**
A total of 140 participants, including patients with different stages of HBV-driven liver diseases: chronic hepatitis, liver cirrhosis, and HCC, along with healthy controls, were recruited, with each group having 35 individuals. Methylation-specific PCR of RASSF1A and CCND2 promoters was performed using methylation- and unmethylation-specific primers, followed by gel electrophoresis. Afterwards, their mRNA expression levels were quantified by qPCR. Differences in methylation levels of these two genes between the groups were subsequently analysed using Fisher's Exact Test. Bonferroni's Post-Hoc analysis was conducted to evaluate the differences in the expression levels between the groups. Finally, the diagnostic performance of RASSF1A and CCND2 promoter methylation for distinguishing HCC from liver cirrhosis and chronic hepatitis was evaluated by a Receiver Operating

Characteristic curve. $P < 0.05$ was considered statistically significant.

Results:

The study found that 37% of the HCC patients and 26% of the cirrhotic individuals had methylation in the RASSF1A promoter ($p < 0.001$), while it was 29% and 17%, respectively, in the promoter of CCND2 ($p < 0.001$). Moreover, RASSF1A in HCC patients was significantly downregulated compared to healthy controls (0.12 ± 0.10 versus 1.00 ± 0.94 , $p < 0.001$). Contrastingly, CCND2 expression increased with disease severity (6.17 ± 5.35 in HCC versus 1.00 ± 0.79 in healthy controls, $p < 0.001$). Interestingly, RASSF1A-methylated samples consistently exhibited lower expression levels than unmethylated counterparts in both HCC and cirrhosis, whereas CCND2 mRNA levels remained high irrespective of methylation status in patients. Both genes' promoter methylation correlated with elevated alpha-fetoprotein (AFP, a standard tumour marker) levels; however, individually offered limited diagnostic specificity (AUC 0.557-0.686 vs AFP AUC > 0.949).

Conclusions: These results highlight hypermethylation of RASSF1A and CCND2 promoters as epigenetic drivers of HBV-associated liver diseases and support their potential inclusion in multi-marker panels for early HCC detection.

Keyword(s) : Hypermethylation, Promoter, RASSF1A, CCND2, Hepatitis B Virus, Hepatocellular Carcinoma, Gene expression

2562 - Successful Endoscopic Ultrasound-Guided Ethanol Ablation of a 7 mm Non Functional Pancreatic Neuroendocrine Tumor in a High Risk Surgical Candidate

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Background / **Hypothesis:**

Pancreatic neuroendocrine tumors (p-NETs) are frequently asymptomatic and are often discovered incidentally or during evaluation of nonspecific constitutional symptoms. The decision-making process in managing p-NETs (< 2 cm) concerning whether to follow up or intervene is difficult, especially in those patients who have significant medical comorbidities and therefore have an increased risk of surgical complications. 5% of low-grade tumors NETs

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metastasize early to distant organs on follow-up. Endoscopic Ultrasound (EUS)-Guided Ethanol Ablation offers a minimally invasive therapeutic approach to treating these patients along with biopsy in the same sitting.

Case Presentation
We describe the case of a 70-year-old man diagnosed with Type II Diabetes Mellitus; he had also recently received an automated implantable cardioverter defibrillator (AICD) due to tachy-brady arrhythmias. He presented to our institution over the course of approximately 8 months with progressive unexplained weight loss and generalized fatigue. His laboratory studies were notable for a mild elevation in his CA 19-9 (62.9 U/mL → 68.4 U/mL); all endocrine studies were normal, further supporting that this represented tumor on contrast-enhanced computed tomography (CT) scan of the abdomen demonstrated a 7.8 x 7 mm arterially enhancing mass in the pancreatic tail; this finding was suggestive of a p-NET. EUS confirmed a well-defined hypoechoic lesion without vascular invasion away from the main pancreatic duct. Fine needle biopsy (FNB) was performed using a single-pass 22-gauge FNB, and 2.5 mL of absolute ethanol was injected into the lesion in the same sitting for a diagnostic and therapeutic approach.

Intervention and **Outcome**
Following the ablative therapy, post-ablation imaging studies demonstrated intralesional hyper-echogenicity consistent with coagulative necrosis. Additionally, the patient reported subjective improvement in fatigue and a weight gain of approximately 1.5 kg within a week. However, given the nonspecific nature of these symptoms and potential confounders like the use of semaglutide, a causal relationship cannot be established. There were no complications associated with the procedure, and repeat imaging studies in the form of a GA-68 DOTATE PET/CT scan one month after the procedure reported no hypermetabolic lesion.

Conclusions:
This case study demonstrates the feasibility and clinical benefit of EUS-guided ablation as a safe and organ-preserving alternative to surgical treatment of selected patients with small p-NETs. The early symptomatic improvement experienced by this patient suggests that minimally invasive ablative therapies may represent a viable option for patients at high risk for surgical complications or who prefer non-surgical treatment

strategies.

Keyword(s) : Pancreatic Neuroendocrine Tumor; Endoscopic Ultrasound; Ethanol Ablation.

2564 - The relationship between the systemic immune inflammation index and hepatitis C virus infection NHANES 2005 to 2012
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Background / **Hypothesis:**
The systemic immune-inflammation index (SII), a contemporary biomarker indicative of inflammatory processes, captures the localized immune reactions and systemic inflammatory responses within the human body. Evidence has established correlations between SII and the incidence as well as the gravity of a range of hepatic pathologies. Nonetheless, the extant literature presents a paucity of studies exploring the SII's role and its clinical significance in the context of hepatitis C virus (HCV) infection. The objective of this research is to scrutinize the correlation between SII and HCV infection.

Materials and **Methods:**
Our research utilized data extracted from the National Health and Nutrition Examination Survey (NHANES). Owing to the asymmetric distribution of SII values, a natural logarithm transformation was implemented to achieve a more normal distribution. A Logit regression analysis was employed to assess the relationship between the natural logarithm of the SII (LN SII) and HCV infection. This cross-sectional analysis included 29,546 participants from 4 cycles of NHANES data.

Results:
The results of the Logit model indicated that lower values of LN SII were linked to a higher probability of HCV infection. This relationship remained significant in the fully adjusted model (OR = 0.455; 95% CI: 0.381-0.543, P <.001). Furthermore, the analysis of the smoothly fitted curve unveiled a relationship resembling an inverted U-shape between LN SII and HCV infection, featuring a turning point value of 6.12.

Conclusions:
This study established a link between lower LN SII levels

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and the occurrence of HCV infection. To validate these findings, further large-scale and prospective studies are required.

Keyword(s) : NHANES; hepatitis C; systemic immune-inflammation index.

2572 - Short Course Adjuvant Tislelizumab plus Tyrosine Kinase Inhibitor Following Curative Ablation for Early Stage Hepatocellular Carcinoma A Prospective Cohort Study

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Background / Hypothesis: Hepatocellular carcinoma (HCC) patients at high risk of recurrence after curative ablation currently lack a standard adjuvant therapy. This study aimed to evaluate the efficacy and safety of a short-course, 3-month adjuvant regimen combining tislelizumab and a tyrosine kinase inhibitor (TKI). **Materials and Methods:** In this prospective, parallel-controlled study, 59 consecutive patients with high-risk early-stage HCC following curative ablation were assigned to receive either a 3-month short-course of adjuvant tislelizumab (200 mg intravenously every 3 weeks for 4 cycles) combined with a TKI (n=29) or active surveillance (n=30) based on patient preference. The primary endpoint was recurrence-free survival (RFS). Secondary endpoints included recurrence rates and safety.

Results: At the time of the primary analysis (November 1, 2025), the median RFS was significantly prolonged in the tislelizumab plus TKI group compared with the active surveillance group (Not reached [95% CI, 21.8–NR] vs. 8.5 months [95% CI, 5.4–11.7]; HR 0.43 [95% CI, 0.20–0.91], P = 0.022). Recurrence rates at 1 and 2 years were 24.1% vs. 60.0% (P = 0.008) and 37.9% vs. 63.3% (P = 0.069) in the tislelizumab plus TKI vs. active surveillance groups, respectively. Subgroup analyses showed that patients aged >60 years, with α -fetoprotein <20 ng/mL, BCLC stage 0/A, or microvascular invasion particularly benefited from tislelizumab plus TKI in terms of RFS. The treatment effect was generally consistent across all pre-specified subgroups (all P interaction > 0.05). Regarding safety, the most common adverse events (AEs) in the adjuvant group were leukopenia (41.4%), thrombocytopenia (37.9%), increased AST (37.9%), hypertension (27.6%), and rash (27.6%). Grade 3–4 AEs occurred in 27.6% of patients, primarily involving thrombocytopenia (17.2%), leukopenia (10.3%), and increased AST (10.3%). No treatment-related deaths were reported.

Conclusions: Short-course adjuvant therapy with tislelizumab plus a TKI significantly reduced the risk of recurrence while demonstrating a manageable safety profile in patients with early-stage HCC following ablation. This regimen represents a novel and promising strategy for HCC adjuvant therapy. Further validation in large-scale, multi-center randomized controlled trials is warranted.

Keyword(s) : Hepatocellular Carcinoma; Adjuvant Therapy; Tislelizumab; Tyrosine Kinase Inhibitor; Recurrence-free Survival

2574 - Plasma microRNA based non invasive stratification of clinically significant and advanced fibrosis in metabolic dysfunction associated steatotic liver disease among Arab adults

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Background / Hypothesis: Metabolic dysfunction–associated steatotic liver disease (MASLD) is a major cause of chronic liver disease and fibrosis in low- and middle-income countries, including Arab settings with high burdens of obesity and type 2 diabetes. Identifying clinically significant fibrosis ($\geq F2$) and advanced fibrosis ($\geq F3$) is difficult where access to liver biopsy is limited and non-invasive scores are not consistently implemented. We hypothesize that circulating plasma microRNA (miRNA) signatures, combined with routine clinical data, can provide a non-invasive multi-marker score to stratify $\geq F2$ and $\geq F3$ fibrosis in MASLD and reduce the need for liver biopsy.

Materials and Methods:

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This two-phase cross-sectional study is conducted over 24 months in Egypt, Palestine and the United Arab Emirates. Adults aged 25–60 years, native to the participating country, are recruited from hepatology, internal medicine and metabolic clinics. Fibrosis stage of MASLD is classified using standardized non-invasive algorithms (including FIB-4, NAFLD Fibrosis Score, elastography and liver biochemical profile). In Phase I, plasma from 30 participants (10 per country; MASLD with $\geq$ F2, MASLD with F0–F1 and healthy controls) undergoes whole-transcriptome RNA sequencing to identify differentially expressed mRNAs and miRNAs associated with $\geq$ F2 and $\geq$ F3. In Phase II, top candidate miRNAs are quantified by qRT-PCR in 150 Egyptian adults (50 MASLD with $\geq$ F2, 50 MASLD with F0–F1, 50 healthy controls). Multivariable logistic models and receiver operating characteristic (ROC) analyses will internally validate composite scores for: (1) $\geq$ F2 vs F0–F1/healthy and (2) $\geq$ F3 vs F2 within the $\geq$ F2 subgroup. Phase I is funded by the Arab Research & Innovation Co-Funded Alliances (ARICA) Initiative under the Federation of Arab Scientific Research Councils and Phase II is funded by the Academy of Scientific Research and Technology of Egypt.

Results:

Recruitment and laboratory analyses are ongoing. Expected outputs include: (1) a ranked panel of fibrosis-associated plasma miRNAs differentiating $\geq$ F2 and $\geq$ F3 from lower-stage disease and healthy controls; (2) ROC-based estimates of discrimination (area under the curve, sensitivity and specificity) for miRNA–clinical composite scores targeting $\geq$ F2 and $\geq$ F3; and (3) comparison of these scores with established non-invasive fibrosis indices to inform their added value in clinical practice.

Conclusions:

This fibrosis-focused liquid biopsy approach aims to deliver simple, blood-based tools to detect clinically significant and advanced fibrosis in MASLD in Arab adults. If diagnostic performance is confirmed, the miRNA-based scores could support earlier risk stratification, guide intensive follow-up, and complement existing non-invasive algorithms in resource-constrained hepatology services.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease, fibrosis, microRNAs, liquid biopsy, non-invasive diagnosis, Arab populations

2576 - Point of care HBcrAg testing to identify high viral replication in chronic hepatitis B a tool for supporting the WHO simplified treatment strategy

in resource limited settings
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Background / Hypothesis:
In 2024, the World Health Organization (WHO) expanded and simplified treatment eligibility criteria for chronic hepatitis B, including recommendations to reduce reliance on HBV DNA testing. However, in many high-burden settings, limited molecular diagnostic capacity results in some individuals initiating lifelong antiviral therapy without virological confirmation. There is therefore an urgent need for simple, accurate, and scalable tools to identify individuals at risk of high viral replication. We evaluated the diagnostic performance of a rapid test for hepatitis B core-related antigen (HBcrAg-RDT) as a point-of-care alternative to laboratory-based antigen and HBV DNA testing.
Methods:

We assessed the diagnostic accuracy of HBcrAg-RDT in two independent adult cohorts in Japan and France. Among 1,246 enrolled participants, 516 were hepatitis B surface antigen (HBsAg)-positive, including 309 untreated individuals. HBcrAg-RDT and hepatitis B e antigen enzyme-linked immunosorbent assay (HBeAg-ELISA) were compared with reference chemiluminescent immunoassays (CLIA/CLEIA) for HBeAg detection (n = 1,240). Secondary analyses evaluated the ability of HBcrAg-RDT to identify individuals with high viral loads (HBV DNA $\geq$ 5.3 log IU/mL; n = 305). Concordance across serum, plasma, and whole blood was also examined to assess point-of-care applicability.

Results:

HBcrAg-RDT demonstrated high sensitivity (97.9–98.5%) and specificity (97.6–98.0%) for detecting HBeAg. When benchmarked against HBeAg-CLIA/CLEIA, accuracy was comparable to that of HBeAg-ELISA. In key clinical

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populations, such as HBsAg-positive individuals, HBcrAg-RDT achieved statistically higher area under the receiver operating characteristic (AUROC) values than HBeAg-ELISA, despite modest absolute differences, indicating a diagnostic advantage. For identifying high-viral-load infections, sensitivity was 96.9% and specificity 93.1% (AUROC 0.95). Performance was preserved in both HBeAg-positive and HBeAg-negative infections, with complete concordance across serum, plasma, and whole blood.

Conclusions:

HBcrAg-RDT provides a highly accurate, practical, and operationally feasible method to identify individuals with high viral replication. Its compatibility with whole blood supports true point-of-care use in resource-limited settings. These findings suggest that HBcrAg-RDT could meaningfully support implementation of the 2024 WHO simplified treatment strategy in adult HBV care by enabling rational treatment decisions where virological testing is unavailable.

Keyword(s) : Chronic hepatitis B; Hepatitis B core-related antigen rapid diagnostic test (HBcrAg-RDT); Point-of-care diagnostics; Treatment eligibility; the World Health Organization (WHO) simplified guidelines; Low-resource settings.

2577 - Using Rose to Improve the Accuracy of Diagnosis of Biliary Tract Diseases through ERCP guided Biopsy

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Background / Hypothesis:
To evaluate the diagnostic efficacy of rapid on-site evaluation (ROSE)- enhanced endoscopic retrograde cholangiopancreatography (ERCP)- guided biopsy for biliary strictures, and develop a predictive model for diagnostic accuracy.

Materials and Methods:
This retrospective study included 200 patients undergoing ERCP- guided biopsy (ROSE cohort, n=100; NON-ROSE cohort, n=100). Diagnostic performance metrics (accuracy, sensitivity, specificity, positive predictive value [PPV], negative predictive value [NPV]), specimen adequacy, biopsy attempts, procedure duration, and complications were compared between

groups. Logistic regression identified independent predictors of diagnostic accuracy. A prediction model was developed and internally validated through receiver operating characteristic (ROC) curve analysis, calibration curves, decision curve analysis (DCA), and constructed as a nomogram.

Results:

The ROSE cohort demonstrated significantly higher accuracy (92.0% vs. 78.0%), sensitivity (97.2% vs. 74.7%), and NPV (91.6% vs. 48.7%) (all $P < 0.05$). ROSE required fewer biopsy passes (2.43 vs. 3.61; $P < 0.001$), shorter sampling time (2.71 vs. 4.33 min; $P < 0.001$), without increased complications ($P > 0.05$). Multivariate analysis confirmed ROSE as an independent predictor (OR=4.0, $P=0.032$).

Conclusions: The validated predictive model showed good discrimination (AUC=0.80) and clinical utility. ROSE significantly enhances diagnostic yield and procedural efficiency in ERCP- guided biopsy for biliary strictures. The developed nomogram provides an evidence-based tool for individualized prediction, supporting clinical decision-making.

Keyword(s) : Biliary stricture, Endoscopic retrograde cholangiopancreatography (ERCP), Rapid on-site evaluation (ROSE), Biopsy

2579 - YTHDF1 high cancer associated fibroblasts derived kynurenine promotes tumor progression by supplying an immunosuppressive niche

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Background / Hypothesis:
Dysregulation of epigenetic RNA modifications is a hallmark of cancer; however, how these alterations shape anti-tumor immunity within the tumor microenvironment (TME) remains poorly understood. Cancer-associated fibroblasts (CAFs) are key stromal components that actively regulate immune suppression.

Materials and Methods:
CAF subsets were characterized based on YTHDF1 expression. Epigenetic regulation of YTHDF1 was

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investigated under lactate-enriched conditions. RNA methylation-dependent translational control was examined using m6A profiling and ribosome association analyses. Protein interactions and ubiquitination were assessed biochemically, and metabolic and immune consequences were evaluated in vitro and in vivo. Therapeutic targeting was tested alone and in combination with programmed cell death protein 1 (PD-1) blockade.

Results:

CAFs with high YTHDF1 expression promoted the differentiation of immunosuppressive regulatory T cells (Tregs). Lactate accumulation in the TME induced YTHDF1 upregulation in CAFs through histone H3 lysine 9 lactylation. Elevated YTHDF1 enhanced the translation of ovarian tumor domain-containing protein 5 (OTUD5) by binding to m6A-modified OTUD5 mRNA. OTUD5 directly interacted with indoleamine 2,3-dioxygenase 1 (IDO1) and inhibited its K48-linked ubiquitin-mediated degradation, resulting in enhanced tryptophan catabolism and kynurenine accumulation, thereby reinforcing an immunosuppressive TME. Pharmacological inhibition of YTHDF1 synergized with PD-1 blockade to enhance antitumor immune responses in multiple models.

Conclusions:

YTHDF1high CAFs remodel the tumor microenvironment through epigenetically regulated tryptophan metabolism, fostering immune suppression. Targeting stromal YTHDF1 represents a promising strategy to enhance the efficacy of cancer immunotherapy.

Keyword(s) : Tumor microenvironment, RNA methylation, ubiquitination, lactylation, immunotherapy

2580 - PYGL A novel non invasive biomarker for diagnostic and prognostic of HEV ALF patients

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Background

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Hypothesis:

Hepatitis E virus (HEV) is a major cause of acute viral hepatitis globally. Although usually self-limiting in healthy individuals, it can progress to severe hepatitis or

acute liver failure (ALF). This study aims to evaluate the prognostic value of glycogen phosphorylase L (PYGL) in patients with HEV-related acute liver failure (HEV-ALF).

Materials

and

Methods:

A total of 280 subjects were enrolled, including 100 patients with acute hepatitis E (AHE), 100 with HEV-ALF, and 80 healthy elderly controls. Serum PYGL levels were measured by ELISA. To assess the diagnostic and prognostic value of GP73, receiver operating characteristic (ROC) curves, orthogonal partial least squares-discriminant analysis (OPLS-DA), and decision curve analysis (DCA) were employed to evaluate its efficacy in diagnosing HEV-ALF and predicting 30-day mortality. The correlation between serum PYGL levels and the number of organ failures and disease progression in patients.

Results:

Serum PYGL levels were significantly elevated in patients with HEV-ALF compared to those with AHE and healthy controls (13.84 (12.93, 14.64) vs. 9.13 (6.97, 10.99) vs. 3.04 (2.54, 3.98), $P < 0.001$). In distinguishing HEV-ALF, PYGL demonstrated an area under the curve (AUC) of 0.911, with 89% sensitivity and 85% specificity. Decision curve analysis confirmed its consistent net clinical benefit across a broad range of threshold probabilities. Over a 30-day follow-up, non-survivors exhibited significantly higher PYGL levels than survivors (17.62 (15.10, 18.33) vs. 13.65 (12.83, 14.31), $P < 0.001$). PYGL also showed strong prognostic performance for mortality, with an AUC of 0.859 (sensitivity 77.3%, specificity 92.3%), supported by favorable clinical decision curve findings. Furthermore, PYGL levels were significantly higher in patients with >3 organ failures compared to those with ≤ 3 failures ($P < 0.05$), and in patients with disease progression compared to those with stabilization or improvement ($P < 0.05$).

Conclusions: PYGL is a potential diagnostic and prognostic biomarker of HEV-ALF patients, and its high level suggests poor prognosis, which provides a new basis for early intervention.

Keyword(s) : patients with hepatitis E infection; Acute liver failure; glycogen phosphorylase L; Biomarker; Prognosis

2583 - Head to Head Comparison Between MRI and A Novel Portable VCTE Device (Liverscan) A Pilot Study

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Background / **Hypothesis:**
Vibration-controlled transient elastography (VCTE) is widely used for liver fibrosis assessment. While the diagnostic accuracy of standard VCTE, particularly Fibroscan, has been extensively compared to magnetic resonance elastography (MRE), Liverscan is a novel, portable, phased array transducer-based VCTE system whose diagnostic performance remains to be rigorously validated. This study aimed to perform a head-to-head comparison between this novel portable VCTE device vs. the MRE for liver stiffness measurement and Liverscan-derived multi-domain attenuation parameter (MAP) vs. MRI-proton density fat fraction (PDFF) for fat measurement, specifically investigating physiological factors driving measurement discordance.

Materials and **Methods:**
We performed a cross-sectional analysis of 48 participants, including 22 metabolic dysfunction-associated steatotic liver disease (MASLD) patients and 26 healthy controls (HC), who underwent concurrent liver stiffness measurement (LSM) using the portable Liverscan (Eieling Technology Limited, Hong Kong, China) and MRE (uPMR 790, United Image, Shanghai, China). Liver fat quantification was also compared between the Liverscan-derived MAP and MRI-PDFF. Agreement was assessed using Spearman's correlation.

Results:

The correlation between LSM by Liverscan and MRE was significant ($r_s = 0.62$, $p < 0.001$). Similarly, liver fat quantification demonstrated strong concordance with MRI-PDFF ($r_s = 0.76$, $p < 0.001$). Importantly, this diagnostic performance was sustained in the MASLD subgroup, where LiverScan maintained significant correlations with MRE for stiffness ($r_s = 0.57$, $p = 0.0051$) and with MRI-PDFF for fat quantification ($r_s = 0.45$, $p = 0.037$).

Conclusions:

Liverscan demonstrates robust diagnostic concordance with MRE and MRI-PDFF, providing a reliable point-of-care solution for the simultaneous assessment of liver fibrosis and steatosis. However, due to the limited sample size and heterogeneous etiologies of

participants, the results are preliminary and inconclusive. Future research should compare the diagnostic accuracy of MRI-based techniques against Liverscan for the diagnosis of liver fibrosis and steatosis using histology as the reference standard.

Keyword(s) : Liverscan, vibration-controlled transient elastography, magnetic resonance elastography, multi-domain attenuation parameter, MRI-proton density fat fraction

2590 - The Predictive Value of Platelet Derived Growth Factor for Evaluating Antiviral Therapy Response and Liver Stiffness Trajectory in Chronic Hepatitis B

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Background / **Hypothesis:**
Chronic hepatitis B virus (HBV) infection is a major contributor to progressive liver fibrosis and cirrhosis. Although nucleos(t)ide analogues (NAs) are effective in suppressing viral replication, a portion of patients exhibits primary non-response, increasing the risk of fibrotic progression. Platelet-Derived Growth Factor-BB (PDGF-BB), a key mediator in hepatic fibrogenesis, has shown potential as a biomarker for fibrosis severity, yet its role in predicting treatment response remains underexplored. This Study want to assess the predictive value of PDGF-BB for identifying primary non-response and tracking liver stiffness changes in patients with chronic hepatitis B undergoing antiviral therapy.

Materials and **Methods:**
This prospective observational study involved 40 treatment-naïve patients with chronic HBV infection monitored over 48 weeks of antiviral therapy. Serum PDGF-BB levels and liver stiffness measurements (LSM) using FibroScan® were recorded at baseline and 6-months follow-up. Fibrosis was staged from F0 to F4, and primary non-response was defined as $<1 \log_{10}$ IU/mL decline in HBV DNA at week 12. ANOVA was used to analyze differences in PDGF-BB across fibrosis stages.

Results:

PDGF-BB levels increased progressively with higher fibrosis stages ($p = 0.000$). The mean PDGF-BB in F0 patients was 208.5 ± 110.2 pg/mL, significantly lower than in F4 patients (1582.3 ± 206.6 pg/mL). A stepwise

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elevation was observed from F1 (486.4 ± 111.2 pg/mL) to F3 (1278.9 ± 72.2 pg/mL), indicating a strong correlation between PDGF-BB levels and fibrosis severity. Median PDGF-BB values also rose across stages, from 188.5 pg/mL in F0 to 1516.2 pg/mL in F4. For predicting primary non-response, ROC analysis showed PDGF-BB levels had sensitivity 85% and specificity 78%, with an AUC of 0.82. Non-responders exhibited significantly higher baseline PDGF-BB levels (1367.2 ± 215.7 pg/mL) compared to responders (783.5 ± 130.3 pg/mL).

Conclusions:

PDGF-BB demonstrates strong potential as a prognostic biomarker for identifying antiviral treatment non-response and predicting liver stiffness progression in chronic hepatitis B patients. Its integration into clinical monitoring could support earlier intervention and individualized therapy adjustments.

Keyword(s) : Platelet-Derived Growth Factor-BB, chronic hepatitis B, fibrosis severity, primary non-response, antiviral therapy, liver stiffness, prognostic biomarker, fibrosis progression, sensitivity, specificity.

2593 - Spermidine suppresses liver fibrosis by remodeling the communication signal between liver sinusoidal endothelial cells and hepatic stellate cells
Running title Spermidine suppresses liver fibrosis by downregulation of LSEC derived BGN

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Background / Hypothesis: Hepatic fibrosis represents a crucial pathological stage in the progression of chronic liver disease, where reversible liver injury evolves into irreversible decompensated liver dysfunction. Liver sinusoidal endothelial cells (LSECs) play a pivotal role in the regulation of hepatic stellate cells (HSCs) activation through paracrine signaling. Therefore, preserving the physiological function of LSECs is essential for preventing the development of fibrosis. Spermidine (SPD) has emerged as a potential antifibrotic agent, but the specific mechanisms by which SPD modulates LSEC function and how it exerts its antifibrotic effects remain poorly understood.

Materials and Methods: To investigate the relationship between SPD intake and liver fibrosis, we utilized data from the National Health and Nutrition Examination Survey (NHANES) and identified an inverse correlation between dietary SPD intake and the risk of fibrosis. In vitro and in vivo models were used to assess the impact of SPD on LSEC function and fibrosis progression. Integrative analyses using proteomics, single-cell RNA sequencing data and machine learning-based prioritization were conducted to identify SPD targets in LSECs.

Results: Our results showed that SPD significantly improved LSEC function and inhibited fibrosis progression. Biglycan (BGN), an extracellular matrix protein from LSECs, was identified as a key target of SPD. Overexpression of BGN diminished the effect of SPD in restoring LSEC dysfunction and promoted HSC activation. Mechanistically, SPD activated NRF2 to increase UBE2G2 expression, thereby enhancing UBE2G2-dependent ubiquitination and degradation of BGN. UBE2G2 knockdown abrogated SPD-induced BGN downregulation and attenuated the protective effects of SPD against LSEC capillarization and HSC activation. Additionally, BGN overexpression in the CCl₄-induced mice model also markedly attenuated the ability of SPD to improve LSECs dysfunction and its antifibrotic efficacy.

Conclusions: This study uncovers a novel mechanism by which SPD ameliorates LSEC dysfunction and inhibits fibrosis progression through modulation of LSEC-derived BGN. These findings suggest that targeting LSEC-derived BGN may represent a promising therapeutic strategy for hepatic fibrosis.

Keyword(s) :

2594 - Risk factors for low level viremia in Tenofovir Disoproxil Fumarate (TDF) treated patients with chronic hepatitis B complicated by advanced hepatic fibrosis compensated liver cirrhosis

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Background

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Hypothesis:

Patients with chronic hepatitis B (CHB) complicated by advanced hepatic fibrosis/compensated cirrhosis are at

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a high risk of disease progression.. Even with first-line nucleos(t)ide analog therapy, a subset of these patients still develops low-level viremia (LLV). Moreover, LLV is associated with the progression of liver fibrosis and an elevated risk of hepatocellular carcinoma. Currently, clinical research on precise predictive markers for LLV in this patient population remains relatively scarce. Therefore, this study aims to identify the high-risk factors associated with LLV in patients with CHB complicated by advanced hepatic fibrosis/compensated cirrhosis.

Materials and Methods:

A retrospective cohort study was conducted on 141 patients with CHB complicated by advanced hepatic fibrosis/compensated cirrhosis who were treated with Tenofovir Disoproxil Fumarate (TDF). According to the virological response at week 48 post-treatment, the patients were divided into two cohorts: LLV (HBV DNA 20–2000 IU/mL) cohort and complete virological response (CVR) (HBV DNA < 20 IU/mL) cohort. Baseline clinical characteristics were extracted from electronic medical records. Univariate and multivariate logistic regression analysis were performed to evaluate the indicators associated with the development of LLV.

Results:

A total of 141 patients were enrolled, male patients accounted for 74.5% (105/141); the median age was 43 years; the mean HBV DNA level was 5.67 Log₁₀ IU/mL. Among them, 17.0% (24/141) experienced LLV. Significant differences were observed between the LLV and CVR groups in terms of baseline HBV DNA, HBV RNA, hepatitis B e antigen (HBeAg) positivity, alanine aminotransferase (ALT), ALT exceeding the upper limit of normal (ULN), aspartate aminotransferase (AST), total bilirubin (TBIL) > ULN, direct bilirubin (DBIL) > ULN, HBV DNA ≥ 5.0 Log₁₀ IU/mL, HBV DNA ≥ 6.0 Log₁₀ IU/mL, HBV DNA ≥ 7.0 Log₁₀ IU/mL, HBV RNA ≥ 5.0 Log₁₀ copies/mL. Multivariate logistic regression analysis showed that HBV DNA ≥ 7.0 Log₁₀ IU/mL (OR = 0.078, 95% CI: 0.024-0.252, p = <0.001), HBV RNA ≥ 5.0 Log₁₀ copies/mL (OR = 0.107, 95% CI: 0.013-0.898, p = 0.039) and TBIL > ULN (OR = 0.154, 95% CI: 0.034-0.695, p = 0.015) at baseline were independently associated with the development of LLV.

Conclusions:

In TDF-treated patients with CHB complicated by advanced hepatic fibrosis/compensated liver cirrhosis, HBV DNA ≥ 7.0 Log₁₀ IU/mL, HBV RNA ≥ 5.0 Log₁₀

copies/mL and TBIL > ULN are the high-risk factors associated with the development of LLV.

Keyword(s) : chronic hepatitis B, fibrosis, cirrhosis, low-level viremia

2596 - Baseline Determinants of Inadequate Ursodeoxycholic Acid Response in Primary Biliary Cholangitis A POISE Based Real World Cohort Study

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Background and Aims: Ursodeoxycholic acid (UDCA) is the first-line therapy for primary biliary cholangitis (PBC); however, a substantial proportion of patients fail to achieve adequate biochemical response and remain at risk for disease progression. Identification of baseline predictors of inadequate response is essential for early risk stratification.

Methods: Electronic records of 453 patients with PBC treated between January 2011 and September 2025 were reviewed. After exclusions, 392 patients initiated on UDCA were included. Biochemical response at 12 months was assessed using POISE criteria (alkaline phosphatase ≤ 1.67× upper limit of normal with ≥ 15% reduction from baseline and total bilirubin ≤ upper limit of normal). Baseline demographic, biochemical, elastographic, bone density, serologic, and histologic variables were compared between POISE responders and non-responders. Independent predictors were identified using multivariable logistic regression, and discrimination assessed using receiver operating characteristic analysis.

Results: At 12 months, 212 of 392 patients (54%) achieved POISE-defined biochemical response, while 180 (46%) were non-responders. The cohort was predominantly female (91.8%) with a mean age of 57.6 ± 13.6 years. Non-responders were older (68.1 ± 5.6 vs 48.7 ± 11.9 years; p < 0.001) with higher baseline liver stiffness (median 17.2 vs 13.9 kPa; p = 0.016). Cholestatic enzymes were higher among non-responders, including alkaline phosphatase (583.6 ± 150.9 vs 299.5 ± 168.1 U/L; p < 0.001) and gamma-glutamyl transferase (539.7 ± 191.8 vs 210.7 ± 167.0 U/L; p < 0.001), with lower platelet counts (100.2 ± 30.5 vs 186.8 ± 86.5 × 10⁹/L; p < 0.001). Transaminases, bilirubin, albumin, international normalized ratio, renal function, and immunoglobulin G

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levels were comparable. Osteopenia was present in 40.8% and osteoporosis in 26.8%, more frequent among non-responders (32.2% vs 22.2%). Extra-hepatic comorbidities and autoantibody profiles, including antimitochondrial antibody positivity (93.4%), were similar. Histology showed more advanced disease among non-responders across Scheuer, Ludwig, and Nakanuma systems, with greater fibrosis, bile duct loss, and orcein-positive copper deposition (all $p < 0.001$). On multivariable analysis, younger age (OR 0.747, 95% CI 0.682–0.818), lower alkaline phosphatase (OR 0.990, 95% CI 0.986–0.994), and lower gamma-glutamyl transferase (OR 0.992, 95% CI 0.989–0.996) independently predicted POISE response (all $p < 0.001$). ROC analysis confirmed excellent discrimination for age (AUC 0.931; specificity 100%), alkaline phosphatase (AUC 0.905; 98.3%), and gamma-glutamyl transferase (AUC 0.902; 100%) (all $p < 0.001$). Conclusions: Nearly half of patients with PBC show inadequate biochemical response to UDCA. Baseline age, SAP, and GGT reliably identify those at high risk of non-response, enabling early risk stratification.

Keyword(s) :

2600 - Hepatitis B virus recurrence in liver transplant recipients receiving subcutaneous hepatitis B immunoglobulin

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Background

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Hypothesis:

Prophylaxis with hepatitis B immunoglobulin (HBIG) combined with nucleos(t)ide analogues (NUCs) remains the standard approach to prevent hepatitis B virus (HBV) recurrence after liver transplantation. Subcutaneous HBIG (sc HBIG) allows outpatient self-administration and sustained immunoprophylaxis; however, real-world data regarding serological dynamics and recurrence rates remain limited. This study aimed to evaluate HBV recurrence in liver transplant recipients receiving sc HBIG.

Materials

and

Methods:

This retrospective, single-center study included 302 adult patients who underwent liver transplantation for

HBV-related liver disease and received sc HBIG in combination with NUC therapy. Baseline demographic and clinical variables included age, sex, pre-transplant HBV DNA status, presence of hepatocellular carcinoma (HCC), hepatitis D virus (HDV) serology and RNA status, MELD-Na score, donor type, and antiviral regimens. Serological markers, including hepatitis B surface antigen (HBsAg) and anti-HBs titers, were assessed at baseline and at months 3, 6, 12, and 24 following transplantation. Post-transplant positive HBsAg and/or HBV-DNA levels were considered HBV recurrence.

Results:

The mean age of the cohort was 57.6 ± 9.2 years, and 78.5% of patients were male. Pre-transplant HCC was present in 42.1%, while anti-HDV or HDV RNA positivity was observed in 59.6% of patients. The majority of transplants were from living donors (97%). The frequency of HBV recurrence at 12 and 24 months post-transplant was 2% and 4.8%, respectively.

Conclusions:

Our findings suggest that sc HBIG administration is an effective method for preventing HBV recurrence after liver transplantation.

Keyword(s) : Hepatitis B virus; liver transplantation; subcutaneous hepatitis B immunoglobulin; recurrence; serologic response

2601 - Diagnostic Accuracy and Long Term Prognostic Value of FIB 4 Index and De Ritis Ratio in Chronic Hepatitis C Patients A Comparative Kaplan Meier Analysis with MELD 3.0

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Background / Hypothesis: Accurate non-invasive assessment of cirrhosis and long-term prognosis is crucial in chronic hepatitis C (HCV) management. We aimed to evaluate the diagnostic and prognostic value of the FIB-4 index and De Ritis ratio in HCV patients and compare their performance with the MELD 3.0 score.

Materials and Methods: In this retrospective study, 303 chronic HCV patients (86 cirrhotic, 217 non-cirrhotic) were followed for a median of 1,906 days (extending up

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to 13 years). Diagnostic accuracy was assessed using ROC-AUC analysis. Long-term survival was analyzed using Kaplan-Meier curves and Cox proportional hazards regression models.

Results:The FIB-4 index and De Ritis ratio showed high diagnostic accuracy for cirrhosis (AUC: 0.871 and 0.803, respectively; $p < 0.001$). For predicting long-term mortality, the FIB-4 index (AUC: 0.641) performed comparably to the MELD 3.0 score (AUC: 0.654). Kaplan-Meier analysis revealed significantly lower survival rates in patients with a baseline FIB-4 score > 6.08 (Log-rank $p = 0.049$). Univariate Cox regression analysis demonstrated that a FIB-4 score > 6.08 was associated with a 2.03-fold increase in the hazard of long-term mortality (HR: 2.031, 95% CI: 1.06–3.87, $p = 0.031$). Multivariate analysis identified FIB-4 as an independent predictor of cirrhosis, while age and albumin remained independent predictors of survival.

Conclusions:The FIB-4 index is a reliable and cost-effective tool that demonstrates prognostic discrimination comparable to MELD 3.0 for long-term mortality risk stratification.

Keyword(s) : Hepatitis C, FIB-4, De Ritis ratio, MELD 3.0, Cirrhosis, Cox regression, Survival analysis.

2602 - Effect of Midodrine on Short Term Mortality and Liver Function in Patients with Cirrhosis and Ascites A Systematic Review and Meta analysis

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Background / **Hypothesis:** Midodrine, an $\alpha 1$ -adrenergic agonist, has been proposed as a vasoconstrictor therapy to counteract circulatory dysfunction in patients with cirrhosis and ascites. Its potential impact on clinical outcomes, particularly mortality and liver function, remains under active investigation.

Materials and **Methods:**

A systematic search was conducted for randomized controlled trials comparing midodrine (with or without rifaximin) to standard medical therapy or albumin in cirrhotic patients with ascites. RevMan 5.4 was used to conduct meta-analyses of mortality (binary outcome)

and MELD scores (continuous outcome). Heterogeneity was evaluated using I^2 statistics. Patient baseline characteristics are summarized in Table 1.

Results

Four RCTs, including a total of 729 patients (464 midodrine, 265 control), met the inclusion criteria. Midodrine significantly reduced short-term (12 weeks) mortality compared to SMT (OR: 0.22; 95% CI: 0.11–0.44; $p < 0.0001$; $I^2 = 29\%$). Midodrine was also associated with a greater reduction in MELD score (MD: -2.49; 95% CI: -2.74 to -2.25; $p < 0.00001$; $I^2 = 0\%$). Heterogeneity was low for both outcomes. No serious adverse effects were reported. Mild symptoms such as thirst and abdominal discomfort were noted in two studies. Midodrine was generally used at 7.5–12.5 mg TID. One study combined midodrine with rifaximin.

Results:

Four RCTs, including a total of 729 patients (464 midodrine, 265 control), met the inclusion criteria. Midodrine significantly reduced short-term (12 weeks) mortality compared to SMT (OR: 0.22; 95% CI: 0.11–0.44; $p < 0.0001$; $I^2 = 29\%$). Midodrine was also associated with a greater reduction in MELD score (MD: -2.49; 95% CI: -2.74 to -2.25; $p < 0.00001$; $I^2 = 0\%$). Heterogeneity was low for both outcomes. No serious adverse effects were reported. Mild symptoms such as thirst and abdominal discomfort were noted in two studies. Midodrine was generally used at 7.5–12.5 mg TID. One study combined midodrine with rifaximin. **Conclusions:**Midodrine is associated with significant improvements in both short-term mortality and liver function, as assessed by MELD score, in patients with cirrhosis and ascites. These findings support the consideration of midodrine as a therapeutic adjunct in selected patients. Further large-scale, multicenter trials are warranted to validate these results.

Keyword(s) : midodrine, cirrhosis, liver

2604 - Research on artificial liver support system for non hepatocellular carcinoma patients with severe immune checkpoints related liver injury

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Background:

As immunotherapy is widely applied in various tumors, immune-related liver injury, as an important part of drug-induced liver injury, has attracted increasing attention. The majority of patients recover liver function after glucocorticoid treatment, but patients with poor response to hormone therapy pose a challenge for

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clinicians.

Materials and Methods:

This study reviewed 18 patients with liver injury caused by immune checkpoint inhibitors who were admitted to Tianjin Third Central Hospital after October 2020. We have meticulously documented the patients' anti-tumor medication usage, clinical biochemical indicators, and artificial liver conditions, and will continue to follow up until December 31, 2025.

Results:

There were 17 males and 3 females with an average age of 62.20 ± 11.18 years. The tumors included 8 cases of lung cancer, 6 cases of gastric cancer, 1 case of esophageal cancer, 1 case of colon cancer, 1 case of soft tissue malignancy, and 1 case of bile duct cancer. Among them, there were 2 patients with chronic viral hepatitis B and 1 patient with liver metastasis. The immune checkpoint drugs used included Tislelizumab, Indilimumab, Carilimumab, Tremelimumab, and Soolilimumab. Upon admission, the patients' liver function tests showed ALT $148.21(25.07, 271.35)$ U/L, AST $194.47(92.66, 296.29)$ U/L, ALP 487.95 ± 291.49 U/L, GGT 637.35 ± 440.93 U/L, TBIL 221.89 ± 98.84 μ mol/L, total bile acid 265.41 ± 150.95 μ mol/L, BUN 7.20 ± 4.44 mmol/L, Cr 73.79 ± 45.92 μ mol/L, NR 1.13 ± 0.44 , IgG 17.41 ± 6.39 g/L, ANA 1:100 in 5 cases, and ANA 1:320 in 1 case. The patients did not respond well to glucocorticoid treatments and were all given artificial liver support system. The modes of artificial liver support system included plasma exchange, plasma adsorption, dual plasma molecular adsorption, and dual plasma molecular adsorption combined with plasma exchange. The number of treatments ranged from 1 to 20. Among them, 12 patients died, 8 patients survived, and 3 patients resumed immunotherapy.

Conclusions:

Immune checkpoint-related liver injury is a significant adverse reaction affecting the survival of cancer patients. On the one hand, patients may have underlying chronic liver disease that is not screened before cancer treatment. On the other hand, various combination therapies such as chemotherapy and targeted therapy may exacerbate liver injury. Therefore, glucocorticoid therapy is ineffective, and patients may even progress to liver failure, with a high mortality rate. Given that cancer patients often do not have the opportunity for liver transplantation, artificial liver support system may become an important treatment method for rescuing immune-related liver injury.

Keyword(s):

2605 - Mobile Ablation for Secondary Hypersplenism in Hepatocellular Carcinoma Patients

with Liver Cirrhosis A Safety and Efficacy Study

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Background / Hypothesis: Secondary hypersplenism is common in patients with hepatocellular carcinoma (HCC) and liver cirrhosis, leading to cytopenia, immune dysfunction, and limited access to antitumor therapies. Although splenectomy can improve hematologic parameters, it carries substantial perioperative risk in cirrhotic patients. Minimally invasive splenic interventions have therefore emerged as alternatives. This study aimed to further evaluate the efficacy and safety of mobile ablation combined with splenic artery balloon occlusion for cirrhosis-related secondary hypersplenism.

Materials and Methods: Between August 2023 and June 2025, 47 patients with HCC and cirrhosis complicated by secondary hypersplenism were prospectively enrolled. Preoperative assessment included routine laboratory tests and liver function grading (Child-Pugh class A, n = 27; class B, n = 20). All patients underwent temporary splenic artery balloon occlusion followed by percutaneous splenic ablation. Treatment outcomes were assessed by ablation volume, dynamic changes in peripheral blood cell counts, postoperative complications, and changes in splenic volume on imaging during follow-up.

Results: A total of 47 ablation procedures were successfully performed, targeting approximately 60–70% of total splenic volume. Fixed dual-electrode ablation was used in 7 patients, while 40 patients underwent single-electrode mobile ablation. Preoperative platelet count was $(51.24 \pm 16.45) \times 10^9$ /L. Platelet levels increased significantly after ablation, reaching a peak at postoperative day 7 (211.57 ± 147.08) $\times 10^9$ /L. Although platelet counts gradually declined at 3, 6, and 12 months, they remained significantly higher than baseline ($P < 0.05$). Leukocyte

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and erythrocyte counts showed no significant long-term differences compared with baseline ($P > 0.05$). Lymphocyte percentage and absolute counts demonstrated an initial decrease followed by recovery, with significant differences compared with preoperative values ($P < 0.05$). Contrast-enhanced ultrasound showed a mean immediate ablation volume of $(72.81 \pm 9.86)\%$ of total splenic volume. MRI follow-up at 6 months revealed a median splenic volume reduction of 67%. No procedure-related mortality occurred. Postoperative complications were mostly mild and manageable, including fever, pain, and hemoglobinuria. Major complications included bleeding ($n = 6$), perisplenic fluid or abscess ($n = 5$), renal failure ($n = 1$), and splenic vein thrombosis ($n = 2$).

Conclusions: Mobile ablation combined with splenic artery balloon occlusion is a safe and effective minimally invasive treatment for secondary hypersplenism in patients with HCC and liver cirrhosis. The technique significantly reduces splenic volume, improves thrombocytopenia, and may contribute to immune function reconstruction, making it a promising alternative for patients unsuitable for splenectomy.

Keyword(s) : Secondary hypersplenism; Hepatocellular carcinoma; Liver cirrhosis; Splenic ablation; Balloon occlusion

2606 - A Simple Five Variable Model Integrating GWAS Variants Predicts Chronic Outcomes in Drug Induced Liver Injury

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Objective:

In clinical practice, progression to chronic liver injury occurs in 6-39% of drug induced liver injury (DILI) cases. However, reliable prognostic tools remain lacking. We aimed to investigate whether genetic variants associated with DILI susceptibility may predict chronic outcomes and thus fill the gap between genetic discovery and clinical translation.

Methods:

Between 2018 and 2023, we prospectively enrolled 338 well characterized DILI patients at Shanghai Ruijin Hospital. Chronic DILI

was defined as persistent liver test abnormalities beyond 6 months. It developed in 167 (49.4%) patients. We analyzed the genotype of two significant single nucleotide polymorphisms (rs72631567, rs28531257), which were combined with routinely available laboratory parameters. These lab parameters included peak alanine aminotransferase, low-density lipoprotein cholesterol, and monocyte count. A light gradient boosting machine learning algorithm was trained on 270 patients and validated independently on 68 patients.

Results:

Both genetic variants at rs72631567 and rs28531257 showed significant associations with chronic DILI progression. The integrated model achieved an area under the curve (AUC) of 0.92 (95% confidence interval 0.89-0.95) in the training cohort and 0.81 (0.70-0.91) in validation, substantially outperforming clinical variables alone (AUC, 0.57, $P < 0.001$). SHAP analysis revealed that rs72631567 contributed the greatest predictive weight, followed by peak transaminase levels.

Conclusion:

The current work demonstrates that susceptibility variants from genome-wide association studies can be prognostic. Our five-variable tool requires only standard blood tests and straightforward genotyping. Early identification of high-risk patients may help determine the intensity of monitoring and therapeutic decisions, thus making precision medicine practical for DILI management.

Keyword(s) : Drug-Induced Liver Injury; Genetic Polymorphism; Machine Learning

2607 - Inhibition of FKBP10 attenuates hepatic stellate cell activation and liver fibrosis via RAS signaling

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Background: Hepatic stellate cell (HSC) activation is a central driving force of liver fibrogenesis. FKBP10, a collagen-associated chaperone involved in extracellular matrix remodeling, has not yet been fully characterized in the context of liver fibrosis. Here, we comprehensively investigated the functional role of FKBP10 and its

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underlying regulatory mechanisms in HSC activation and fibrotic progression.

Methods: The expression and localization of FKBP10 were examined in liver tissues from patients with cirrhosis of various etiologies and in multiple experimental fibrosis models. Gain- and loss-of-function approaches using FKBP10 siRNA and overexpression plasmids were applied in LX-2 cells to assess its effects on HSC activation, apoptosis, and extracellular matrix (ECM) production. The therapeutic potential of FKBP10 inhibition was further evaluated in vivo using AAV6-GFAP-shFKBP10 delivery in fibrotic mouse models. Primary HSCs isolated from CCl₄-induced fibrotic livers were subjected to transcriptomic sequencing to identify downstream pathways regulated by FKBP10. In addition, LC-MS/MS combined with co-immunoprecipitation was performed to identify FKBP10-interacting proteins, and pharmacological inhibitors targeting RAS signaling or VPS4A were employed for mechanistic validation.

Results: FKBP10 expression was markedly upregulated in human cirrhotic livers across multiple etiologies and in several mouse fibrosis models. Receiver operating characteristic analysis based on FKBP10 expression suggested its potential value as an auxiliary diagnostic biomarker for liver cirrhosis. In vitro, FKBP10 silencing significantly suppressed HSC activation, reduced ECM synthesis, and promoted apoptosis, whereas FKBP10 overexpression produced opposite effects. Notably, pharmacological inhibition of RAS signaling effectively reversed FKBP10 overexpression-induced HSC activation and ECM production, indicating that RAS acts as a critical downstream effector of FKBP10. In vivo, AAV6-GFAP-shFKBP10-mediated knockdown of FKBP10 resulted in thinner and more discontinuous fibrotic septa, reduced inflammatory cytokine expression, and overall attenuation of liver injury. Transcriptomic profiling of primary HSCs revealed that FKBP10 deficiency predominantly suppressed the RAS signaling pathway and its downstream profibrotic cascades. Furthermore, LC-MS/MS and co-immunoprecipitation identified VPS4A as a potential binding partner of FKBP10. Importantly, pharmacological inhibition of VPS4A abolished FKBP10-induced RAS activation, suggesting that FKBP10 regulates RAS activity through a VPS4A-dependent mechanism involving RAS membrane-cytoplasmic cycling.

Conclusion: Collectively, these findings demonstrate that FKBP10 promotes HSC activation and liver fibrogenesis by interacting with VPS4A to modulate RAS signaling. Targeting the FKBP10-VPS4A-RAS axis may therefore represent a promising therapeutic strategy for the treatment of liver fibrosis.

Keyword(s) : FKBP10, Liver fibrosis, Hepatic stellate cell activation, RAS signaling pathway, VPS4A, AAV-mediated gene silencing

2608 - Optimizing Pegylated Interferon Consolidation Therapy and Predicting Recurrence After Functional Cure in Chronic Hepatitis B A Study Based on Updated Definitions

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Background / Hypothesis: Despite achieving functional cure (FC), some chronic hepatitis B (CHB) patients experience HBsAg recurrence. This study aimed to identify the optimal consolidation duration and predictors of recurrence based on the updated FC definition.

Materials and Methods: This study enrolled 443 CHB patients treated with Pegylated interferon (PEG-IFN)-based therapy at the First Affiliated Hospital of Nanchang University between November 2020 and May 2023. A total of 113 patients achieved FC, the patients were divided into a training cohort and a validation cohort via time series partitioning method. Patients in each cohort were grouped by recurrence status. Clinical characteristics and longitudinal changes in HBV DNA and HBsAg levels were compared. Independent predictors were identified using multivariate logistic regression. Cutoff values were determined using receiver operating characteristic curve analysis, and the cumulative recurrence rate was estimated by Kaplan-Meier survival analysis. A nomogram was constructed based on independent predictors.

Results:

Consolidation therapy duration, PEG-IFN monotherapy, and end-of-treatment (EOT) HBsAb level were independently associated with recurrence (all $P < 0.05$). The combined model showed high predictive accuracy (AUC=0.913, 95% CI: 0.830–0.964). The optimal cutoff value for consolidation therapy duration was 13.50 weeks. For EOT HBsAb, the cutoff value was 2.03 log₁₀ mIU/mL. The nomogram model demonstrated good predictive performance.

Conclusions:

A consolidation duration of ≥ 13.5 weeks, antiviral therapy with PEG-IFN in combination with nucleos(t)ide

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analogues, and an EOT HBsAb level $2.03 \log_{10}$ mIU/mL were associated with sustained FC. Post-treatment virological monitoring remains essential to allow early detection of HBsAg reappearance and timely intervention. The nomogram model may provide a reference for clinical decision-making and individualized risk assessment.

Keyword(s) : Chronic hepatitis B; Interferon; Functional cure; Consolidation therapy; Dynamic changes

2609 - Acetylation modification of PKM2 promotes macrophage reprogramming and accelerates the progression of MAFLD

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Background / **Hypothesis:** Cholesterol homeostasis is vital, yet its disruption in Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) triggers mitochondrial dysfunction and oxidative stress. Liver macrophages, highly sensitive to cholesterol overload, undergo a metabolic shift from oxidative phosphorylation to glycolysis, driving a pro-inflammatory phenotype that exacerbates hepatic inflammation and fibrosis. As a central glycolytic regulator, Pyruvate Kinase M2 (PKM2) is a key candidate for sensing this stress. However, the role of post-translational modifications (PTMs), specifically PKM2 acetylation, in lipid-induced macrophage dysfunction remains poorly understood. Investigating this mechanism is crucial to elucidating how metabolic reprogramming drives MAFLD, potentially offering novel biomarkers and therapeutic strategies. **Materials** and **Methods:** In this study, RAW264.7 cells and primary PEMs/BMDMs were challenged with 20 μ g/mL cholesterol. siRNA knockdown of ATF2 and LPCAT1, combined with PKM2 mutation (K62R, K305R, K433R), was used for functional analysis. Western blot, IP, qRT-PCR, and ELISA were performed to measure protein, gene, and cytokine levels. Filipin III and BODIPY staining helped detect lipid accumulation and ROS production. The in vivo model involved HFHC-fed mice with macrophage-specific PKM2 knockdown, followed by the adoptive transfer of WT or K433R-PKM2 macrophages. Liver damage and M1 macrophage polarization were assessed as the final endpoints.

Results:

Cholesterol overload triggers macrophage lipid accumulation, oxidative stress, and glycolysis. While total PKM2 remains stable, cholesterol enhances its phosphorylation and acetylation at K433. Mechanistically, the p38-ATF2-LPCAT1 axis drives this modification. In vivo, macrophage-specific PKM2 knockdown mice on HFHC diets showed that WT-PKM2 rescue exacerbates MAFLD pathology, whereas the acetylation-deficient K433R mutant alleviates liver lipid buildup, fibrosis, and M1 polarization. These findings identify PKM2 K433 acetylation as a molecular switch driving metabolic reprogramming, positioning the p38-ATF2-LPCAT1-PKM2 axis as a therapeutic target for MAFLD and lipid-related disorders.

Conclusions

Our study identifies a novel p38-ATF2-LPCAT1-PKM2 signaling axis that drives immunometabolic reprogramming in macrophages under cholesterol stress. We demonstrate that cholesterol overload activates the p38 MAPK pathway, leading to the ATF2-mediated transcriptional upregulation of the acetyltransferase LPCAT1. This cascade enhances the acetylation of PKM2 at the K433 site, acting as a molecular switch that shifts macrophage metabolism toward glycolysis and amplifies pro-inflammatory responses. These findings elucidate a precise mechanistic link between lipid stress-activated signaling and the post-translational modification of metabolic enzymes. By characterizing this regulatory network, our research provides a novel theoretical framework for understanding how cholesterol dysregulation drives macrophage dysfunction and offers potential therapeutic targets for treating lipid-associated inflammatory and metabolic diseases, such as MAFLD.

Keyword(s) : MAFLD, PKM2 Acetylation, Macrophage, P38, ATF2, LPCAT1

2612 - Accuracy of D dimer Levels in Predicting Clinical Outcomes of Patients with Decompensated Liver Cirrhosis in a Philippine Tertiary Hospital

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Background / **Hypothesis:** Decompensated liver cirrhosis is associated with high morbidity, mortality, and prolonged hospitalization, particularly in tertiary public hospitals. Early risk stratification remains challenging in resource-limited settings. D-dimer, a fibrin degradation product reflecting coagulation and inflammatory abnormalities,

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has emerged as a potential prognostic biomarker in cirrhosis; however, local evidence from the Philippine setting remains limited.

Materials and Methods: A prospective cohort study was conducted among 150 adult patients with decompensated liver cirrhosis admitted to East Avenue Medical Center from January to December 2025. Serum D-dimer levels measured within 24 hours of admission were evaluated against two outcomes: in-hospital mortality and prolonged hospitalization (>7 days). Receiver operating characteristic (ROC) curve analysis was used to identify optimal cutoff values. Diagnostic accuracy parameters, including sensitivity, specificity, predictive values, likelihood ratios, diagnostic odds ratio (OR), and area under the curve (AUC), were calculated. Logistic regression was used to determine diagnostic ORs, and ROC curve significance was tested against the null hypothesis of AUC = 0.5.

Results:

A total of 150 patients with decompensated liver cirrhosis were included in the analysis. For predicting in-hospital mortality, an optimal D-dimer cutoff of ≥ 2.38 mg/L yielded a sensitivity of 74.2% and specificity of 60.2%, with an area under the ROC curve (AUC) of 0.691 (95% CI: 0.612–0.770) and a diagnostic odds ratio of 4.35. The positive and negative predictive values were 56.8% and 76.8%, respectively.

For prolonged hospitalization (>7 days), a D-dimer cutoff of ≥ 3.00 mg/L demonstrated 100% specificity and positive predictive value, with a sensitivity of 48.7%. The AUC for prolonged hospitalization was 0.714 (95% CI: 0.635–0.793), indicating good discriminative ability. Elevated D-dimer levels were consistently associated with adverse clinical outcomes.

Conclusions:

Serum D-dimer demonstrates statistically significant and clinically relevant prognostic value in predicting both in-hospital mortality and prolonged hospitalization among patients with decompensated liver cirrhosis. Its good discriminative ability and strong rule-in performance support its use as an adjunctive risk stratification tool in tertiary public hospital settings, particularly where early identification of high-risk patients is critical.

Keyword(s) : Decompensated liver cirrhosis; D-dimer; Prognostic biomarker; In-hospital mortality; Length of hospital stay; Risk stratification; Receiver operating characteristic (ROC) analysis; Tertiary hospital; Philippines

2614 - A Cross Section Study on the Co prevalence of MAFLD and HBV Infection Among Adult Residents in Maojian District Shiyan City.

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Background / Hypothesis: To investigate the epidemiological characteristics of co-occurrence of metabolic associated fatty liver disease (MAFLD) and hepatitis B virus (HBV) infection among adult residents in Maojian District, Shiyan City, Hubei Province, China, and to identify risk factors for the co-occurrence of MAFLD in those with HBV infection.

Materials and Methods: A community-based cross-sectional survey was conducted among residents aged ≥ 18 years in Maojian District. Data on demographics, lifestyle, and dietary habits were collected via an online questionnaire. On-site measurements included measurements of height, weight, and waist/hip circumference. Morning fasting blood samples were obtained for liver function tests, lipid profiles, and hepatitis B surface antigen (HBsAg) detection using colloidal gold method. Hepatic steatosis and fibrosis were evaluated by transient elastography (FibroScan®) to determine liver fat content (controlled attenuation parameter, CAP) and liver stiffness measurement (LSM). Statistical analysis was performed to assess the prevalence, clinical characteristics, and associated risk factors of MAFLD-HBV co-occurrence.

Results: Among 2,105 adults initially surveyed 341 were excluded based on ineligibility criteria; and finally 1,764 participants (mean age: 42.7 ± 17.3 years) were included in the analysis. The prevalence rates of MAFLD, HBsAg positivity, and MAFLD-HBV co-occurrence were 31.7% (535 cases), 4.4% (78 cases), and 1.4% (24 cases), respectively. Compared with HBsAg -positive individuals without MAFLD, those with MAFLD-HBV co-occurrence exhibited significant worse metabolic profiles, including

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higher levels of body mass index (BMI), waist circumference, hip circumference, systolic blood pressure, triglycerides, uric acid, alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), serum creatinine, and cystatin C (all $P < 0.01$). Hypertension (70.83% vs. 11.11%), diabetes (70.83% vs. 22.22%), abdominal obesity (62.50% vs. 12.96%), and insulin resistance (58.33% vs. 22.22%) were also markedly more prevalent in the co-occurrence group (all $P < 0.01$). Multivariate analysis identified suboptimal animal-based food intake as an independent risk factor ($P = 0.012$), whereas regular breakfast consumption served as an independent protective factor ($P = 0.012$) for MAFLD development among HBV-infected individuals.

Conclusions: The prevalence of HBV infection in this community population is lower than the national estimates. The co-occurrence rate of MAFLD and HBV infection is 1.4%, with an increasing trend with age, particularly among males. We recommend assessment of liver steatosis and metabolic parameters in males over 40 years and in individuals with underlying metabolic abnormalities. This study provides insights into the characteristics and potential risk factors for MAFLD-HBV co-occurrence. Future multi-center large-sample studies are needed to clarify risk factors and optimize intervention strategies, including routine metabolic screening and enhanced dietary health management in this population.

Keyword(s) : Hepatitis B virus (HBV); Metabolic-associated fatty liver disease (MAFLD); Comorbidity; Prevalence; Influencing Factors

2617 - Spirulina Prevents Hepatic Steatosis by Promoting Macrophage M2 Polarization via Gut Microbiota Derived 2-Hydroxy-3-Methylbutyrate
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Background / Hypothesis:

Spirulina, a microalgae renowned for its nutrient density, shows promise in modulating glucose and lipid metabolism. However, its efficacy and underlying mechanisms against metabolic dysfunction-associated steatotic liver disease (MASLD) remain unclear. We hypothesized that spirulina exerts its hepatoprotective effects by reshaping the gut microbiome and its associated metabolome.

Materials and Methods:

We conducted an 8-week pilot study in 23 patients with MASLD receiving spirulina supplements (1g TID), assessing changes in liver fat, enzymes, and metabolic parameters. To establish causality and elucidate mechanisms, we employed two distinct murine models of steatohepatitis: a Western diet model and a choline-deficient diet model. Interventions included spirulina gavage, antibiotic-induced microbiota depletion, fecal microbiota transplantation (FMT), and administration of the microbial metabolite 2-hydroxy-3-methylbutyrate (2H3MB). Multi-omics approaches (16S rRNA sequencing, metabolomics, single-cell RNA sequencing) and flow cytometry were utilized.

Results:

Spirulina supplementation significantly alleviated hepatic steatosis in both human subjects and murine models. This was accompanied by a restoration of gut microbial ecology, characterized by enriched beneficial genera (e.g., Akkermansia, Bacteroides, Blautia). Metabolomic profiling identified a marked increase in the gut microbiota-derived metabolite 2H3MB in the feces and liver. Critically, the protective effects of spirulina were abolished in microbiota-depleted mice and could not be transferred via FMT from spirulina-treated donors to naïve recipients, indicating a dependency on an intact and active microbiome. Direct administration of 2H3MB was sufficient to recapitulate the anti-steatotic effects of spirulina in high-fat diet-fed mice. Mechanistically, single-cell RNA sequencing and flow cytometry revealed that 2H3MB reshaped the hepatic immune microenvironment by specifically promoting the M2 polarization of macrophages, which in turn ameliorated lipid accumulation.

Conclusions:

Our findings unveil a novel gut-liver axis pathway through which spirulina prevents hepatic steatosis. Spirulina enhances the production of 2H3MB by specific gut microbes, which then signals to the liver to drive an anti-inflammatory, pro-resolutive M2 macrophage polarization, ultimately mitigating lipid deposition. This work highlights gut microbial metabolites, particularly 2H3MB, as promising therapeutic targets for MASLD.

Keyword(s) : Spirulina, MASLD, Gut-Liver Axis, Macrophage Polarization, 2-Hydroxy-3-Methylbutyrate, Single-Cell RNA Sequencing

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2623 - Transcriptomic Profiling Identifies Anti Inflammatory and Anti Steatotic Programs Mediated by ShenGe Formula in MAFLD

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Background / Hypothesis: Metabolic dysfunction-Associated Fatty Liver Disease (MAFLD) is becoming the most common liver disease worldwide. The discovery of medicines that combine anti-inflammatory and steatosis effects is the hottest area of MAFLD treatment. ShenGe Formula (SGF) is a Traditional Chinese medicine formula for treating MAFLD, with clear clinical efficacy and a Chinese patent. This study aims to investigate the roles of anti-inflammatory and anti-steatotic efficacy mediated by SGF in MAFLD. **Materials and Methods:** The principal constituents of SGF were identified using UPLC-MS. The HFHC-induced murine MAFLD model was generated over 16 weeks, followed by an 8-week treatment phase with SGF. Liver sections were analyzed by histopathology. The primary hepatocytes and liver macrophages were respectively isolated and purified. RNA-seq was used for the transcriptomic analysis of these cells. Candidate mRNA and pathways were verified both in vivo and in vitro through RT-qPCR, Western blot, and immunohistochemistry.

Results: Thirty-nine chemicals were identified in SGF extraction, animal serum, and liver, which are considered to be the key components in treating MAFLD. The hepatic steatosis and inflammation were significantly reduced in the livers of animal models by SGF treatment. Transcriptomic analysis of primary hepatocytes identified 817 different expressed genes (DEGs) associated with SGF treatment, whereas 469 DEGs were identified in primary macrophages. Bioinformatic analysis revealed that SGF improved insulin resistance and lipid metabolism in hepatocytes via the Insulin Receptor and SREBP signaling pathways, while it inhibited the activity of liver macrophages via AMPK and PI3K signaling pathways. Furthermore, Fasn, Scd1, and Tsc2 were identified as potential targets of SGF for reducing lipid accumulation, while Prkaa1, Ppargc1a, and Pik3cb were found as potential targets of SGF for inhibiting the activity of macrophages. Ultimately, the in vivo and in vitro results established that SGF inhibits Fasn expression and elevates Prkaa1 expression to mitigate hepatic lipid accumulation and inflammation in MAFLD.

Conclusions: Our findings confirmed the potential anti-

inflammatory and anti-steatotic programs mediated by ShenGe Formula in MAFLD.

Keyword(s) : MAFLD;TCM;Anti-inflammatory;Anti-steatotic

2625 - Procedural Related Bleeding Among Patients with Liver Cirrhosis in a Tertiary Hospital in the Philippines A Five Year Review

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Background / Hypothesis: Patients with liver cirrhosis are at high risk of bleeding following both surgical and non-surgical procedures. However, there are currently no reliable tests or scoring systems to predict procedure-related bleeding in cirrhotic patients, nor is there consensus on hemostatic thresholds to minimize bleeding risk.

Materials and Methods: This study is a hospital-based retrospective cohort which aimed to determine the relationship between incidence of post-procedural bleeding and patients' baseline demographics, comorbidities, clinical status, liver disease etiology, and liver function. The study included 275 patients diagnosed with liver cirrhosis who underwent at least one procedure during their hospitalization. Medical records were retrospectively reviewed to collect patient characteristics and the incidence of post-procedural bleeding. Data were analyzed using point-biserial correlation and chi-square tests.

Results:

Procedure-related bleeding occurred in 5 of the 275 patients (1.82%). Patients who experienced post-procedural bleeding were more likely to have a lower INR ($r = -0.15$, $p = 0.01$), presence of malignancy ($r = 0.13$, $p = 0.03$), and metabolic dysfunction-associated fatty liver disease (MAFLD) as the etiology of cirrhosis ($r = 0.33$, $p < 0.001$), compared with those without bleeding. No significant associations were found between procedure-related bleeding and age, Child-Turcotte-Pugh or MELD-Na scores, platelet count, use of antiplatelet or anticoagulant therapy, medical thromboprophylaxis, presence of acute or chronic kidney disease, or concurrent infection.

Conclusions:

Procedure-related bleeding was uncommon in this cohort. Although certain factors were associated with bleeding events, the low incidence limits

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generalizability. Larger studies are needed to validate these findings.

Keyword(s) : liver cirrhosis, procedure, bleeding

2626 - Clinical characteristics of colorectal polyps in patients with non alcoholic fatty liver disease in high altitude areas

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Background / Hypothesis: Colorectal cancer has high global incidence and mortality rates. Colorectal polyps are relatively common, with adenomatous polyps having a higher risk of malignant transformation. Non-alcoholic fatty liver disease (NAFLD) has been identified as a risk factor for the development of colorectal adenomas. Here, inpatients with NAFLD from the Second People's Hospital of Xining, in Qinghai Province, and the Second People's Hospital of Tianjin were investigated, comparing the biochemical indicators, colonoscopy findings, and pathological results of polyps between patients from low-altitude (Tianjin) and high-altitude (Qinghai Province) areas. Risk factors associated with the occurrence of adenomatous polyps in NAFLD patients from high-altitude areas were also explored. **Materials and Methods:** A total of 848 patients with NAFLD were enrolled. Of these, 118 underwent colonoscopy between January 2021 and January 2024 at the Second People's Hospital of Tianjin (low-altitude), while the remaining 730 patients were assessed during the same period at the Second People's Hospital of Xining, Qinghai (high-altitude). All enrolled patients met the diagnostic criteria for NAFLD, and the excised colorectal polyps were analyzed pathologically. **Results:** Colorectal polyps were found in 585 cases (80.1%) in the Qinghai cohort and 91 patients (77.1%) in the Tianjin group, indicating a slightly higher incidence in the Qinghai group, although the difference was non-significant ($P > 0.05$). The two groups showed significant difference in the proportion of younger patients ($P < 0.01$). The significant differences were found between the ≤ 0.5 cm and > 1 cm and ≤ 2 cm proportions in both regions ($P < 0.05$). Polyp locations also differed significantly ($P < 0.05$). Patients in Qinghai were more prone to adenomatous polyps, accounting for 89.2% of polyps, compared to those from Tianjin ($P < 0.05$). Patients in Qinghai had a higher incidence of tubular adenomas with low-grade dysplasia, while tubular adenomas with high-grade dysplasia predominated in patients from Tianjin ($P < 0.05$). Multivariate logistic regression indicated that sex (OR = 1.695, 95%CI: 1.131-

2.536), smoking (OR = 0.604, 95%CI: 0.406-0.897), hypertension (OR = 0.683, 95%CI: 0.471-0.991), and white blood cell counts (WBC) (OR = 1.091, 95%CI: 1.015-1.173) were risk factors for the occurrence of adenomatous polyps in patients with NAFLD in high-altitude areas.

Conclusion: Patients with NAFLD from high-altitude regions have a higher incidence of colorectal polyps, with a significantly higher incidence of adenomatous polyps compared to other polyp types. Sex, smoking, hypertension, and WBC are risk factors for adenomatous polyps in NAFLD patients in high-altitude regions.

Keyword(s) : Endoscopic manifestations, Pathological results, Colorectal polyps, Non-alcoholic fatty liver disease, Qinghai region

2627 - ZIP1⁺ CAFs Shape the Immunosuppressive Microenvironment to Drive Hepatocellular Carcinoma Progression

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Background / Hypothesis: The stroma of hepatocellular carcinoma (HCC) is dominated by cancer-associated fibroblasts (CAFs) and collagen deposition. These interconnected CAFs form a robust physical and immunological barrier that not only promotes the exhaustion of anti-tumor effector cells but also recruits immunosuppressive populations, thereby driving tumor progression and metastasis. ZIP1 is a membrane protein responsible for zinc ion transport, known to modulate cell proliferation via intracellular zinc regulation. However, the specific mechanisms by which ZIP1 orchestrates CAF activation and mediates the remodeling of the HCC immune microenvironment remain obscure. This study investigates the role of ZIP1⁺ CAFs in shaping the immunosuppressive landscape of HCC.

Materials and Methods: We analyzed the prognostic significance of ZIP1 in HCC patients using the GEO and TCGA databases. To characterize CAF heterogeneity, we utilized public single-cell sequencing data to identify differentially expressed genes between ZIP1⁺ and ZIP1⁻ CAFs, validated by multiplex immunofluorescence for the activation marker FAP. Spatial transcriptomic

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sequencing was performed on a cohort of HCC patients treated with immunotherapy at our center to analyze the spatial architecture of the immune niche. Furthermore, an orthotopic liver cancer mouse model was established by co-injecting Hepa53.4 cells with ZIP1⁺ or ZIP1⁻ CAFs to evaluate tumor progression and immune cell infiltration in vivo.

Results:
Our results identify ZIP1 as a critical driver of CAF activation and the transition toward an extracellular matrix (ECM)-rich phenotype. ZIP1⁺ CAFs were significantly enriched in HCC tissues and correlated with poorer clinical outcomes. Mechanistically, ZIP1 promotes the expression of cell migration-inducing hyaluronidase (CEMIP), which subsequently functions to generate low molecular weight hyaluronan (LMW-HA). This accumulation of LMW-HA triggers the secretion of C-C Motif Chemokine Ligand 22 (CCL22), a potent chemokine that recruits Regulatory T cells (Tregs) to the tumor region, thereby establishing an immunosuppressive microenvironment. In an orthotopic mouse model, tumors co-injected with ZIP1⁺ CAFs exhibited significantly accelerated progression compared to controls. Consistently, spatial transcriptomics revealed that the presence of ZIP1⁺ CAFs correlates with a dampened response to immunotherapy.

Conclusions:
This study establishes ZIP1 as a critical regulator of CAF activation and a key architect of the immunosuppressive microenvironment in HCC. By fostering Treg accumulation, ZIP1⁺ CAFs facilitate immune evasion and tumor growth. These findings suggest that targeting ZIP1⁺ CAFs holds promise as a therapeutic strategy to remodel the stroma and enhance the efficacy of immune checkpoint inhibitors in hepatocellular carcinoma.

Keyword(s) :

2630 - Single cell transcriptomes reveal the immune characteristics of patients with chronic hepatitis B at different stages of disease progression

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Background / **Hypothesis:**
The progression of chronic hepatitis B (CHB) is closely related to the host immune system. However, traditional techniques are difficult to reveal the highly heterogeneous immune microenvironment of CHB. At

present, the dynamic changes of local and systemic liver immunity in different stages of the natural process of CHB and their interrelations are still unclear.

Materials and Methods:
In this study, single-cell RNA sequencing (scRNA-seq) was performed based on paired liver tissue and peripheral blood mononuclear cell (PBMC) samples of CHB patients in the immune tolerant phase (IT) and immune clearance phase (IC) in public databases. Seurat and other tools were used for quality control, cell typing, reclustering, differential expression, enrichment analysis, depletion score, cell communication analysis, and multiple immunofluorescence technology was used for protein level verification to systematically compare the composition and function differences of intrahepatic and peripheral immune cells.

Results:
A total of 45,890 hepatocytes and 40,143 PBMC cells were analyzed. Liver tissue analysis identified 15 major cell populations. Deep reclustering of T cells identified 12 functional subsets. Compared with the IT group, the proportion of terminally differentiated effector CD8⁺ T cells and Th17 cells was significantly increased, while the proportion of exhausted CD8⁺ T cells was decreased in the liver tissues of the IC group, indicating that a strong antigen-specific immune response occurred in the liver. However, these significant intrahepatic T-cell changes were not observed in paired peripheral blood T cells. The T cells in PBMC were mainly of naive phenotype, and there was no significant difference in each subpopulation between IT and IC groups.

Conclusions:
The antigen-specific T cell response and inflammatory response in the immune clearance phase of CHB are mainly limited to the local liver microenvironment, and peripheral blood monitoring may be difficult to fully reflect their dynamic changes. This finding highlights the necessity of liver tissue analysis to evaluate the immune status and pathological mechanism of CHB, and provides an important theoretical basis for the development of new immunotherapy strategies targeting the local liver microenvironment.

Keyword(s) : Single-cell RNA sequencing; Chronic hepatitis B; Immune microenvironment; T cell subsets; Liver tissue and peripheral blood

2634 - A Novel Murine Model of MetALD Recapitulates the Synergistic and Aggressive Phenotype of Combined Metabolic Dysfunction and Alcohol Consumption

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Background / **Hypothesis:**
The 2023 Delphi consensus defined MetALD as the intersection of metabolic dysfunction and alcohol consumption, highlighting a growing population with poor prognosis. However, the absence of standardized preclinical models mimicking voluntary drinking and metabolic stress limits mechanistic understanding of this "double-hit" pathology. We aimed to establish and characterize a novel murine MetALD model that recapitulates key human disease features.

Materials and **Methods:**
Male and female C57BL/6J mice (8-10 weeks old) underwent a two-bottle choice voluntary alcohol paradigm combined with high-fat diet (HFD) feeding. Behavioral interactions between alcohol preference and caloric intake were monitored. Liver injury, oxidative stress, inflammation, and fibrogenesis were comprehensively evaluated.

Results:
Voluntary alcohol intake and HFD consumption showed reciprocal inhibition, reducing calories from each source. Nevertheless, their combination triggered a synergistic, aggressive phenotype distinct from single-hit controls. MetALD mice exhibited a unique "lean but severe" profile with profound steatohepatitis and advanced fibrosis, marked by elevated ALT/AST, extensive apoptosis, overwhelming oxidative stress (elevated MDA, depleted GSH), intense inflammation (immune infiltration), and accelerated fibrogenesis (upregulated Col1a1/ α -SMA; extensive Sirius Red staining).

Conclusions:
This robust MetALD model demonstrates synergistic acceleration of liver injury, fibrosis, and apoptosis by alcohol and metabolic stress, independent of obesity. It fills a critical translational gap, offering an essential platform for dissecting MetALD pathogenesis and testing targeted therapies.

Keyword(s) : MetALD; Murine model; Synergistic toxicity; Liver fibrosis; Oxidative stress; Steatohepatitis

2635 - Endothelial RGS16 Attenuates Hepatic Fibrosis by Restricting Pathological

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Background / Hypothesis: Liver fibrosis is a leading cause of mortality in chronic liver diseases, yet effective therapies remain limited. Liver sinusoidal endothelial cells (LSECs) orchestrate hepatic homeostasis by maintaining fenestration and releasing nitric oxide (NO) to suppress hepatic stellate cell (HSC) activation. Chronic injury disrupts this balance, triggering LSEC capillarization and angiocrine signaling that drive pathological neovascularization and fibrogenesis. While Regulator of G-protein Signaling 16 (RGS16) modulates GPCR pathways, its specific role in LSEC-mediated vascular remodeling during fibrosis remains undefined. We investigated the mechanistic contribution of endothelial RGS16 to pathological angiogenesis and hepatic fibrosis.

Materials and Methods: We employed complementary in vivo and in vitro approaches. In vivo, LSEC-specific RGS16 knockdown was induced in mice using AAV-Oit3-shRNA vectors, followed by 8 weeks of CCl₄ administration. Fibrosis severity and neovascularization were quantified via qPCR, Western blot, and immunofluorescence. In vitro, C166 endothelial cells were transduced to stably knockdown RGS16. Proliferative, migratory, and angiogenic capacities were assessed using CCK-8, Transwell, and Matrigel tube formation assays.

Results: RGS16 expression was markedly upregulated in LSECs within the CCl₄-induced fibrotic microenvironment, indicating a compensatory protective response. Selective deletion of endothelial RGS16 aggravated liver injury (elevated ALT/AST) and significantly upregulated pro-fibrotic markers (α -SMA, TIMP-1, TGF- β) and pro-angiogenic factors (SDF-1, bFGF). Immunofluorescence confirmed intensified pathological neovascularization in RGS16-deficient livers. In vitro, RGS16 knockdown directly enhanced endothelial proliferation, migration, and tube formation, thereby fueling the angiocrine crosstalk required for HSC activation.

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Conclusions: Endothelial RGS16 functions as a critical protective checkpoint in hepatic fibrogenesis by restraining pathological angiogenesis and subsequent HSC activation. Potentiating RGS16 signaling in LSECs offers a novel therapeutic strategy to normalize the hepatic vascular niche and ameliorate liver fibrosis.

Keyword(s) : Liver fibrosis; LSECs; Pathological angiogenesis; RGS16; Therapeutic target

2639 - Machine learning(ML) based models predict beta blocker response using non invasive tests(NITs) in patients with acute variceal bleed BE RESPONSE STUDY

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Background / Hypothesis: Hepatic venous pressure gradient (HVPG) has prognostic and therapeutic value in patients with acute variceal bleed (AVB). Six-week rebleeding and mortality is close to 20% post-AVB. Non-selective beta-blockers (NSBB) are the mainstay of secondary prophylaxis; however, response is observed in only about 50%. Serial HVPG measurement to assess NSBB response, being invasive, is not feasible. We investigated the diagnostic accuracy of non-invasive tests (NITs) in predicting NSBB response in AVB patients at 3 and 6 weeks using machine learning (ML)-based models

Materials and Methods: This prospective study assessed cirrhosis patients with AVB having HVPG, liver stiffness measurement (LSM), and spleen stiffness measurement (SSM) measured within 5 days of bleed, followed by carvedilol therapy. NITs were measured at 0, 3, and 6 weeks, and HVPG at 0 and 6 weeks, with rebleeding followed up for 90 days (NCT05166317). The primary objective was to determine NIT accuracy in predicting NSBB response at 6 weeks; secondary goals included correlating NITs with HVPG and rebleeding. Associations were determined using Cox proportional hazards regression analysis. A Gradient Boosting classifier was developed using 107 engineered features reduced to 51 via consensus selection. Model performance was assessed using 5-fold stratified cross-validation with bootstrap 95% confidence intervals.

Results:

220 patients with AVB [age 49.56 ± 10.34 years, 91.4% male, predominantly ethanol-related (45%) cirrhosis] with mean MELD 15.13 ± 2.75 , baseline HVPG 18.66 ± 2.2 mmHg, LSM 59.24 ± 15.44 kPa, and SSM 84.06 ± 15.74 kPa were included. At 6 weeks, 86 (39%) were HVPG responders; mean carvedilol dose was 11.91 ± 2.03 mg/d. Rebleeding occurred in 31 (14.1%) patients, predominantly in HVPG non-responders (18.6% vs 7.1%; $p=0.027$). A two-threshold decision strategy was developed: delta SSM at 3 weeks ≥ 5 kPa predicted HVPG response (RULE-IN, PPV 78.7%), while delta SSM < 0 kPa predicted non-response (RULE-OUT, NPV 88.6%). The model achieved AUC 0.858 (95% CI: 0.80–0.91) with MCC 0.60 and high-confidence RULE-IN (PPV 88%) and RULE-OUT (NPV 89%) at extreme probability thresholds. Patients with 3-week SSM > 94 kPa had a response rate of only 7.5% (4/53), ruling out HVPG response at 6 weeks

Conclusions:

ML Driven models based on SSM reliably reflect the HVPG responders in patients with AVB. These models can be used as an alternative to HVPG to make accurate prediction of NSBB response at week 3 itself rather than the standard 6 weeks which would help in patient stratification, dose modification or alternative interventions such as TIPS.

Keyword(s) :

2640 - Necrolytic migratory erythema without glucagonoma An unusual association with chronic pancreatitis and hepatic dysfunction

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Background

Necrolytic migratory erythema (NME) is a rare paraneoplastic dermatosis most commonly associated with glucagonoma, an alpha-cell tumor of the pancreatic islets. It is characterized by superficial epidermal necrosis and erythematous lesions with a centrifugal distribution. More rarely, NME has been reported in association with non-neoplastic conditions, including liver disease, intestinal malabsorption, and pancreatitis. In this report, we describe a case of NME as a rare cutaneous manifestation of chronic pancreatitis with hepatic dysfunction.
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A 57-year-old female patient presented with complaints of redness, dryness, and painful swelling in both legs and ankles, as well as pain and burning of the tongue. The skin lesions appeared as patchy, sharply demarcated discolorations with localized peeling, spreading to the body within one month. Her medical history revealed that she underwent cholecystectomy eight years ago due to gallstones and subsequently developed chronic pancreatitis. She had undergone several ERCP procedures, including common bile duct stent placement, without detection of a tumor. Two years prior, she received a celiac plexus nerve block severe abdominal pain. Physical examination revealed cachexia, subicteric sclera, painful stomatitis, glossitis, angular cheilitis, widespread edema and erythema of the ankles and feet, sharply demarcated brown-red hyperpigmented xerotic lesions on hips and trunk, diffuse macular lesions, generalized xerosis, and erosive intertriginous lesions (Figure 1,2). Other systemic examinations were unremarkable. Laboratory investigations revealed anemia on complete blood count and prolonged PT/INR. Serum albumin levels were decreased. Hepatic enzymes, including ALT, AST, GGT, along with total and direct bilirubin levels, were mildly elevated. Blood glucose, glucagon, and zinc levels were within normal limits. Radiological evaluation demonstrated no evidence of a tumor. Skin biopsy demonstrated hydropic degeneration of keratinocytes in the upper stratum spinosum, consistent with NME (Figure 3). Despite supportive care, the patient died secondary to *Acinetobacter baumannii* sepsis.

Discussion

Necrolytic migratory erythema is a rare and distinctive dermatologic manifestation, most commonly recognized as a paraneoplastic syndrome associated with glucagonoma. More rarely, NME may occur in the absence of a glucagonoma, referred to as pseudoglucagonoma syndrome. Previous reports have described NME in association with intestinal malabsorption, liver cirrhosis, inflammatory bowel disease, chronic pancreatitis, and various malignancies, including small cell lung carcinoma and duodenal neoplasms.

Conclusion

This case emphasizes the importance of recognizing gastrointestinal and systemic diseases that may present with cutaneous manifestations such as NME, a highly rare but clinically significant condition. Early identification of NME may facilitate prompt evaluation for underlying disorders and improve clinical outcomes.

Keyword(s) : Necrolytic migratory erythema, chronic pancreatitis, hepatic dysfunction, pseudoglucagonoma syndrome, cutaneous manifestation, paraneoplastic dermatosis

2641 - Prevalence of Metabolic Associated Fatty Liver Disease in Patients with Rheumatoid Arthritis

A	Cross	Sectional	Study
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Background / Hypothesis:

Rheumatoid arthritis (RA) is a chronic inflammatory disease frequently associated with metabolic and cardiovascular comorbidities. Increasing evidence suggests a link between RA and metabolic-associated fatty liver disease (MAFLD); however, data on the prevalence and the burden of liver fibrosis in RA patients remain limited. The study aimed to determine the prevalence of MAFLD and liver fibrosis in patients with rheumatoid arthritis and to identify associated risk factors.

Materials and Methods:

This cross-sectional study included 154 patients with rheumatoid arthritis recruited between July 2024 and February 2025. All patients underwent detailed clinical assessment, including anthropometric measurements [body mass index (BMI), waist circumference (WC), hip circumference, waist-hip ratio, and waist-height ratio]. Disease activity was assessed using the Disease Activity Score in 28 joints (DAS28). Liver steatosis and fibrosis were evaluated using transient elastography (FibroScan) with controlled attenuation parameter (CAP) and liver stiffness measurement (LSM). Laboratory investigations included liver biochemical profile, lipid profile, glycated hemoglobin (HbA1c), and inflammatory markers

Results:

The median age was 45 years, with a predominance of females (88.3%). MAFLD was identified in 79 patients (51%). Among those with MAFLD, steatosis was graded as mild in 27 (34.2%), moderate in 49 (62%), and severe in 3 (3.8%). Liver fibrosis was detected in 34 patients (22%); most cases were mild (F1: 20.8%), while advanced fibrosis (F2–F4) was rare (1.8%). Univariate analysis showed that age, BMI, WC, triglyceride (TG) levels, and HbA1c were associated with MAFLD. In multivariate analysis, WC (OR = 1.070; 95%

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CI: 1.012–1.132; $p = 0.017$) and TG levels (OR = 1.021; 95% CI: 1.009–1.033; $p = 0.001$) remained independent predictors, whereas HbA1c lost significance. Liver fibrosis was associated with age, BMI, WC, HbA1c, and hepatic steatosis on Univariate analysis; however, after adjustment, only age remained independently associated with fibrosis (OR = 1.050; $p = 0.016$). DAS28 scores were not significantly associated with MAFLD ($p = 0.091$) or liver fibrosis ($p = 0.142$).

Conclusions:

MAFLD is highly prevalent among patients with rheumatoid arthritis, affecting approximately half of the studied population, with predominantly moderate steatosis. Liver fibrosis was present in about one-fifth of patients, mostly mild. Central obesity and hypertriglyceridemia are key independent predictors of MAFLD, while increasing age is the main determinant of liver fibrosis. These findings emphasize the importance of metabolic risk assessment and liver disease screening in patients with rheumatoid arthritis.

Keyword(s) : Metabolic-associated fatty liver disease; Liver fibrosis; Rheumatoid arthritis; Prevalence; FibroScan

2643 - IL 24 attenuates hepatocyte apoptosis and ameliorates metabolic associated steatohepatitis by activating the autophagy lysosome pathway

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Background / Hypothesis:

Impaired autophagy is closely associated with metabolic-associated steatohepatitis (MASH), but its underlying mechanisms remain incompletely understood. This study aimed to investigate the regulatory role and potential mechanisms of the cytokine IL-24 on autophagy and apoptosis during the progression of MASH.

Materials and Methods:

The differentially expressed gene IL-24, closely related to NAFLD, was screened through bioinformatics analysis. A mouse model of MASH was established using a high-fat, high-fructose diet (HFFD), and hepatic overexpression of IL-24 was mediated via adeno-

associated virus vectors. In vitro, lipotoxic injury was simulated by treating AML12 hepatocytes with palmitic acid, and a stable IL-24-overexpressing cell model was established via lentiviral transduction. Histopathological staining, Western blot, TUNEL staining, and mRFP-GFP-LC3 dual-fluorescence autophagy flux analysis were employed to assess the effects of IL-24 on liver pathology, autophagy flux activity, and cell apoptosis.

Results:

IL-24 expression was significantly downregulated in the livers of MASH mice. Exogenous IL-24 intervention effectively alleviated HFFD-induced hepatic steatosis, inflammatory infiltration, and fibrosis. Mechanistic studies revealed that IL-24, by activating the AMPK signaling pathway and inhibiting mTOR activity, promoted the nuclear translocation of the transcription factor TFEB, thereby restoring the overall function of the autophagy-lysosome pathway and significantly reducing hepatocyte apoptosis.

Conclusions: This study demonstrates that IL-24, as a key regulatory factor in MASH progression, activates the AMPK/mTOR/TFEB axis to restore autophagy-lysosome function and inhibit hepatocyte apoptosis, thereby mitigating hepatic lipid accumulation and injury. IL-24 holds promise as a potential novel therapeutic target for the prevention and treatment of MASH.

Keyword(s) : Metabolic dysfunction-associated steatohepatitis; IL-24; Autophagy-lysosome; Metabolism; AMPK/mTOR/TFEB axis

2644 - FKBP10⁺ CAFs Shape the Immunosuppressive Microenvironment to Drive Hepatocellular Carcinoma Progression

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Background / Hypothesis:

The stroma of hepatocellular carcinoma (HCC) is dominated by cancer-associated fibroblasts (CAFs) and collagen deposition. These interconnected CAFs form a robust physical and immunological barrier that not only promotes the exhaustion of anti-tumor effector cells but also recruits immunosuppressive populations, thereby driving tumor progression and metastasis. FKBP10 is an

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important molecular chaperone that primarily functions within the endoplasmic reticulum. It plays a crucial role in the synthesis, folding, and secretion of collagen proteins. However, the specific mechanisms by which FKBP10 orchestrates CAF activation and mediates the remodeling of the HCC immune microenvironment remain obscure. This study investigates the role of FKBP10⁺ CAFs in shaping the immunosuppressive landscape of HCC.

Materials and Methods: We analyzed the prognostic significance of FKBP10 in HCC patients using the GEO and TCGA databases. To characterize CAF heterogeneity, we utilized public single-cell sequencing data to identify differentially expressed genes between FKBP10⁺ and FKBP10⁻ CAFs, validated by multiplex immunofluorescence for the activation marker α -SMA. Spatial transcriptomic sequencing was performed on a cohort of HCC patients treated with immunotherapy at our center to analyze the spatial architecture of the immune niche.

Results: Our results identify FKBP10 as a critical driver of CAF activation and the transition toward an extracellular matrix (ECM)-rich phenotype. FKBP10⁺ CAFs were significantly enriched in HCC tissues and correlated with poorer clinical outcomes. Mechanistically, FKBP10 promotes the expression of cytoskeleton-associated protein 4 (CKAP4), which subsequently functions to connect CAFs with each other via gap junctions. This connection of CAFs by CKAP4 elevates the stiffness of the extracellular matrix, which stimulates hepatocellular carcinoma cells to secrete TGF- β . Then TGF- β functions to activate CAFs to create more extracellular matrix, which forms a positive feedback loop. The increased physical barrier reduces CD8⁺ T cell infiltration, thereby establishing an immunosuppressive microenvironment. In an orthotopic mouse model, tumors co-injected with FKBP10⁺ CAFs exhibited significantly accelerated progression compared to controls. Consistently, spatial transcriptomics revealed that the presence of FKBP10⁺ CAFs correlates with a dampened response to immunotherapy.

Conclusions: This study establishes FKBP10 as a critical regulator of CAF activation and a key architect of the immunosuppressive microenvironment in HCC. By reducing CD8⁺ T cell infiltration, FKBP10⁺ CAFs facilitate immune evasion and tumor growth. These findings suggest that targeting FKBP10⁺ CAFs holds promise as a therapeutic strategy to

Keyword(s):

2647 - Quercetin ameliorates MASLD progression through the inhibition of the hexosamine biosynthesis pathway mediated TAB2 protein O-GlcNAcylation

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a prevalent chronic liver disease spanning MAFL to MASH, an inflammatory stage that accelerates fibrosis and heightens the risk of cirrhosis and hepatocellular carcinoma, yet effective therapies for established MASH remain limited. Dysregulated glucose metabolism—particularly the hexosamine biosynthetic pathway (HBP), which produces UDP-GlcNAc as the donor substrate for protein O-GlcNAcylation—has been implicated in insulin resistance, lipogenesis, inflammation, and fibrogenesis. Based on preliminary evidence that quercetin improves metabolic dysfunction and lowers circulating UDP-GlcNAc, mechanisms were investigated across clinical specimens, animal models, and cellular systems to define quercetin-mediated regulation of the HBP-O-GlcNAc axis and to identify O-GlcNAc-modified proteins relevant to MASLD/MASH progression.

Materials and Methods: Quercetin formulations were quality-controlled by HPLC. Efficacy was evaluated in a MASH mouse model treated with graded oral quercetin, with outcomes assessed by serum biochemistry/ELISA and histopathology. In vitro, quercetin was applied to FFA-induced MIHA cells and MIHA-HSC co-cultures, and lipid accumulation and HSC activation were quantified by Oil Red O staining, ELISA, and RT-qPCR. LC-MS metabolomics interrogated key metabolic pathways, while global protein O-GlcNAcylation and related enzymes were assessed by immunoblotting, sWGA pull-down, and RT-qPCR. Label-free O-GlcNAc proteomics was used to identify differentially modified proteins. TAB2-OGT interaction was validated by IP, co-IP and immunofluorescence. WT and mutant TAB2 were compared for stability, ubiquitination, and downstream signaling, and in vivo efficacy was tested by AAV-mediated TAB2 overexpression followed by quercetin treatment in MASH mice.

Results: Quercetin consistently ameliorated MASLD/MASH in diet-induced MASH mice models and cellular models,



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which was evidenced by improvements in BMI and lipid profiles, along with reductions in markers of liver injury and fibrosis. Quercetin also suppressed hepatic lipid accumulation, inflammation, and fibrosis both in vivo and in vitro. Through metabolomics, the HBP was identified as a critical axis, with reduced levels of UDP-GlcNAc observed in patient serum, MASH liver, and cellular models, accompanied by decreased global O-GlcNAcylation. O-GlcNAc proteomics identified TAB2 as the key O-GlcNAcylated target. TAB2 was O-GlcNAcylated by OGT on Thr180. Mutation of Thr180 led to the destabilization of TAB2 through enhanced ubiquitin-dependent degradation without affecting its localization, and attenuated MAPK/NF- κ B activation. Functionally, TAB2 knockdown resulted in reduced steatosis and hepatic stellate cell activation, which could be rescued by wild-type but not mutant TAB2. In vivo, quercetin or DON alleviated MASH in AAV-Tab2 mice, supporting a mechanism dependent on TAB2 O-GlcNAcylation.

Conclusions: Quercetin ameliorated MASLD through downregulation of the HBP-derived metabolite UDP-GlcNAc-mediated reduced O-GlcNAcylation of TAB2.

Keyword(s) : quercetin, MASLD, hexosamine biosynthesis pathway, O-GlcNAcylation, TAB2

2649 - Microsomal Glutathione S Transferase 2 Links Copper Toxicity to Hepatic Inflammation via Leukotriene C4 in Wilson's Disease

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Background / Hypothesis: Wilson's disease (WD) causes severe liver injury and inflammation due to aberrant copper accumulation. Although oxidative stress plays a central role in WD pathogenesis, the precise molecular mechanisms linking copper overload to specific inflammatory pathways remain unclear. This study aims to identify key molecular mediators of inflammation in WD and assess their therapeutic potential.

Materials and Methods: We analyzed the transcriptome of liver tissues from Atp7b-/- mice to identify key inflammatory mediators. The functional role of the identified target in copper-induced inflammation was assessed using copper-treated hepatocytes. In vivo studies were conducted in both Atp7b-/- mice and an exogenous copper-overload mouse model. Thirty-one WD patients were enrolled in the study to validate clinical relevance using liver biopsies and serum

samples.

Results: We found that microsomal glutathione S-transferase 2 (MGST2) was significantly upregulated in the livers of Atp7b-/- mice. Copper-induced oxidative stress triggered NRF2, which drove the transcriptional upregulation of Mgst2 through direct promoter binding. This upregulation promoted leukotriene C4 (LTC4) biosynthesis, which subsequently activated NOX4 to initiate pro-inflammatory signaling. Importantly, pharmacological blockade of this axis with the CysLT1 receptor antagonist Montelukast or the NOX4 inhibitor GLX351322 significantly attenuated liver injury and inflammatory cell infiltration in both Atp7b-/- mice and exogenous copper-overload mice. Consistently, WD patients exhibited activation of the MGST2/LTC4/NOX4 axis and systemic inflammation.

Conclusions: These findings reveal a novel role for MGST2 in copper-induced hepatic inflammation and provide proof of concept for targeting the MGST2/LTC4/NOX4 axis as a therapeutic strategy in WD.

Keyword(s) : Wilson's disease, MGST2, LTC4, NOX4, Hepatic inflammation

2654 - Comparison of the diagnostic accuracy of magnetic resonance elastography FibroScan and serum fibrosis models for detecting liver fibrosis and cirrhosis in chronic liver diseases a multicohort study across all aetiologies

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Background

Hypothesis:

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Liver fibrosis and cirrhosis of various aetiologies is significant global burden. Although liver biopsy is a gold standard for the diagnosis of liver fibrosis and cirrhosis, it is an invasive procedure with complex process, high price, and poor compliance. We aimed to compare the diagnostic performance of Magnetic resonance elastography (MRE), liver stiffness measurement (LSM) evaluated by FibroScan, the aspartate transaminase-to-platelet ratio index (APRI), and the fibrosis index based on four factors (FIB-4) for detecting liver fibrosis in chronic liver diseases across all aetiologies.

Materials and Methods:

A total of 424 patients of chronic liver disease across various aetiologies were prospectively included from three hospitals in China. All patients underwent liver biopsy, MRE, Fibroscan, and routine laboratory tests. Using liver histology as gold standard, we compared the performance of MRE, Fibroscan, APRI, and FIB-4 for detecting liver fibrosis and cirrhosis.

Results:

For the total cohort of chronic liver disease patients across various aetiologies (424 patients), the AUROCs for MRE, LSM evaluated by FibroScan, APRI, and FIB-4, were 0.93, 0.82, 0.71, and 0.66 for the diagnosis of significant liver fibrosis; and 0.96, 0.87, 0.77, and 0.73 for cirrhosis, respectively. For the subgroup of patients with CHB (128 patients), the AUROCs for MRE, LSM evaluated by FibroScan, APRI, and FIB-4, were 0.91, 0.81, 0.71, and 0.65 for the diagnosis of significant liver fibrosis; and 0.97, 0.86, 0.80, and 0.73 for cirrhosis, respectively. For the subgroup of patients with MAFLD (126 patients), the AUROCs for MRE, LSM evaluated by FibroScan, APRI, and FIB-4, were 0.90, 0.76, 0.70, and 0.66 for the diagnosis of significant liver fibrosis; and 0.97, 0.87, 0.80, and 0.80 for cirrhosis, respectively. For the subgroup of patients with AIH (116 patients), the AUROCs for MRE, LSM evaluated by FibroScan, APRI, and FIB-4, were 0.95, 0.88, 0.71, and 0.70 for the diagnosis of significant liver fibrosis; and 0.96, 0.92, 0.73, and 0.68 for cirrhosis, respectively.

Conclusions: MRE is more accurate than Fibroscan in diagnosing liver fibrosis and cirrhosis in chronic liver disease patients across all aetiologies.

Keyword(s) : Magnetic resonance elastography; liver stiffness measurement; FibroScan; liver fibrosis; cirrhosis

2660 - The Silent Summer Threat Significant Seasonal Worsening of Electrolyte Imbalance in

Cirrhosis Patients in a Tropical Climate Calls for Dynamic Diuretic Dosing

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Background / Hypothesis:

Electrolyte imbalances in cirrhosis, driven by diuretics and disease progression, are key predictors of mortality and hepatic encephalopathy. However, the impact of environmental stress is overlooked. We hypothesized that the persistent heat and humidity of a tropical climate would significantly disrupt electrolyte homeostasis. This study aimed to quantify the seasonal variation of serum sodium and potassium in a large cohort of stable cirrhotic patients.

Materials and Methods: We conducted a prospective, observational study of 300 consecutive cirrhotic patients at the Department of Gastroenterology, Rangpur Medical College Hospital, Rangpur, Bangladesh. Each patient underwent two standardized assessments: once in midsummer and once in midwinter. Serum electrolytes were measured, and the prevalence of hyponatremia ($\text{Na}^+ < 135 \text{ mmol/L}$) and hypokalemia ($\text{K}^+ < 3.5 \text{ mmol/L}$) was compared. Statistical analysis included paired t-tests and ROC curve analysis.

Results: The cohort (mean age 55.5 years, 68% male) included 60% patients on diuretics. We found a dramatic seasonal decline in mean serum sodium (Winter: 135.9 vs. Summer: 133.6 mmol/L, $p < 0.001$) and potassium (4.0 vs. 3.7 mmol/L, $p < 0.001$). This translated into a near-doubling of clinical prevalence:

- Hyponatremia: 22.7% in winter vs. 45.0% in summer ($p < 0.001$).
- Hypokalemia: 8.7% in winter vs. 18.3% in summer ($p < 0.001$).

The effect was profoundly amplified in patients on diuretics, who had significantly lower summer levels than those not on diuretics (Na^+ : 131.9 vs. 136.2 mmol/L; K^+ : 3.5 vs. 4.0 mmol/L). ROC analysis identified $\text{Na}^+ \leq 134.5 \text{ mmol/L}$ and $\text{K}^+ \leq 3.75 \text{ mmol/L}$ as optimal cut-offs for predicting summer-associated depletion.

Conclusion: For the first time, we demonstrate a striking

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"Summer Effect"—a significant seasonal worsening of hyponatremia and hypokalemia in cirrhosis, critically exacerbated by diuretics. This mandates a paradigm shift towards seasonal stewardship in management, including pre-emptive dose adjustments and intensified summer monitoring, to prevent electrolyte-driven complications in vulnerable cirrhotic patients.

Keyword(s) : Cirrhosis, Electrolyte Imbalance, Hyponatremia, Tropical Climate, Diuretic Management

2663 - High Prevalence of MASLD and MASH in Chronic Hepatitis B Patients A Cross Sectional Study

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Background / **Hypothesis:**
Hepatitis B virus (HBV) is a major cause of chronic liver disease worldwide. Recently, metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH) have gained increasing importance among metabolic liver disorders. However, data regarding the prevalence and risk factors of MASLD and MASH in patients with HBV infection remain limited.

Materials and **Methods:**
This study included patients aged ≥ 18 years with chronic hepatitis B (CHB) and documented HBsAg positivity for at least six months. Patients with hepatitis C or D coinfection, excessive alcohol consumption (>30 g/week for men and >20 g/week for women), hepatocellular carcinoma, or pregnancy were excluded. Laboratory, ultrasound, liver biopsy findings, and anthropometric parameters obtained during routine follow-up were evaluated. MASLD was defined as hepatic steatosis with at least one cardiometabolic risk factor. Body mass index (BMI) was categorized as <25 kg/m² (normal/underweight), 25–29.9 kg/m² (overweight), and ≥ 30 kg/m² (obese). Thrombocytopenia was defined as a platelet count $<150 \times 10^9/L$.

Results:

The study encompassed 272 patients of CHB. The prevalence of MASLD detected by ultrasonography was 55.9% (152/272). In patients diagnosed with MASLD; BMI, waist circumference, fasting plasma glucose, plasma triglycerides, low density lipoprotein (LDL)-cholesterol, and aspartate transaminase (AST) were significantly higher, while high density lipoprotein (HDL)-cholesterol was significantly lower than in the MASLD-negative group ($p < 0.005$) (Table 1). Furthermore, MASLD prevalence was significantly higher in obese and overweight patients, those with type 2 DM, those using statins for dyslipidaemia, and anti-HBe-positive patients ($p < 0.005$). MASLD prevalence was significantly lower in patients receiving antiviral therapy ($p = 0.001$) (Table 2). Among patients receiving antiviral therapy, 32 (11.8%) received tenofovir disoproxil fumarate, 31 (11.4%) tenofovir alafenamide fumarate, and 67 (24.6%) entecavir; MASLD rates did not differ according to drug used ($p = 0.324$). The 109 patients who underwent liver biopsy were evaluated for MASH. Twenty-seven cases (24.8%) had MASH. In these patients, triglyceride levels were significantly higher ($p = 0.02$) and HDL levels significantly lower ($p = 0.005$). Moreover, MASH prevalence was significantly higher in patients with MASLD ($p < 0.001$). Multivariate regression analysis identified being overweight and obese, statin use for dyslipidaemia, and AST level as independent risk factors for MASLD, while antiviral therapy was found to be potentially protective (Table 3).

Conclusions:

This study demonstrates that MASLD and MASH are prevalent comorbidities in KHB patients and are closely associated with metabolic risk factors. Comprehensive management of these factors and the administration of antiviral therapy when necessary are considered important.

Keyword(s) : Chronic Hepatitis B, MASLD, MASH

2664 - Integrating genetic omics and functional omics to identify clinically actionable gut microbiota-host interface targets in MASLD

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Background / Hypothesis:

To identify clinically actionable gut microbiota–host interface targets for metabolic-associated steatotic liver disease (MASLD) by integrating human genetic and functional multi-omics evidence.

Materials and Methods:

We intersected TWAS signals for MASLD with curated microbiota–host interaction data from the gutMGene database to identify microbiota-regulated candidate targets. These candidates were further characterized by integrating methylation- and splicing-based QTLs, immune cell–specific single-cell TWAS (scTWAS), and liver single-cell and spatial transcriptomics to delineate their genetic architecture and cellular spatial context. Phenome-wide Mendelian randomization (PheW-MR) and structure-based molecular docking were employed to assess pleiotropic safety and druggability.

Results:

Intersecting MASLD TWAS signals with gutMGene identified 11 microbiota-regulated host targets (CGN, CXCL10, CXCR2, GPBAR1, IL24, MUC1, PTGS2, SIGIRR, IL4, IL6 and TNF). Multi-layer QTL evidence and immune-cell scTWAS supported their genetic involvement and revealed cell type–specific effects. Liver single-cell and spatial transcriptomics showed compartmentalized remodeling of these genes across hepatocyte, myeloid, lymphoid and mesenchymal niches. PheW-wide MR separated broadly pleiotropic hubs (TNF, IL6) from more tractable interface nodes (GPBAR1, CXCR2, PTGS2, MUC1, CGN, SIGIRR), while docking suggested broad druggability for these targets.

Conclusions:

Integrating genetic omics with multi-layer functional omics, we delineated a small set of gut microbiota–regulated host interface targets with convergent evidence, highlighting GPBAR1, CXCR2, PTGS2, MUC1, CGN and SIGIRR as promising, clinically actionable candidates for MASLD and providing a generalizable framework for microbiota-informed target discovery.

Keyword(s) : MASLD, Gut microbiota, Genetic omics, Functional omics, Targets

2666 - Outcomes of cholangiocarcinoma (ICC) or combined hepatocellular cholangiocarcinoma after liver transplantation

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Background / Hypothesis:

To investigate the characteristics and outcomes of patients with cholangiocarcinoma (ICC) or combined hepatocellular-cholangiocarcinoma (ICC-HCC) following liver transplantation.

Materials and Methods:

This study included patients diagnosed with ICC or ICC-HCC who underwent liver transplantation at a tertiary hospital between June 2015 and August 2018. Clinical characteristics and post-transplant outcomes were analyzed.

Results:

Seven patients were enrolled (6 males, 1 female), with a mean age of 55.1 ± 2.2 years. Prior to transplantation, 5 patients were diagnosed with hepatocellular carcinoma (HCC) and 2 with ICC-HCC. The mean maximum tumor diameter was 6.7 ± 1.3 cm, and the median AFP level was 134.2 (IQR 59.8–596.8) ng/mL. Two patients had a single tumor, while five had multiple tumors. Pre-transplant evaluation indicated that 2 patients met the Milan criteria, and 5 exceeded them. All patients had cirrhosis, with no evidence of extrahepatic malignancy. Post-transplant histopathology confirmed ICC in 2 cases and ICC-HCC in 5 cases. All patients exhibited microvascular invasion, and 3 had lymphatic metastasis. During follow-up, all 7 patients experienced tumor recurrence, with a mean recurrence interval of 2.8 ± 0.6 months and a mean overall survival of 6.0 ± 0.9 months. Among the two patients initially assessed as meeting Milan criteria, both were upstaged post-transplant due to lymphatic metastasis, multiple tumors, and vascular invasion. Another patient initially within Milan criteria was found to have multiple tumors with a maximum diameter of 5 cm. One additional case, with multiple tumors (maximum diameter 2.5 cm), recurred first in the lung and retroperitoneal lymph nodes. Recurrence involved multiple organs in all cases: 85.7% (n=6) presented with initial lymphatic metastasis, 71.4% (n=5) with liver recurrence, 42.9% (n=3) with lung

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metastasis, and 14.3% (n=1 each) with adrenal or peritoneal metastasis. Patients received various treatments including radiation therapy, transarterial chemoembolization, and tyrosine kinase inhibitors. All patients died within 11 months after transplantation.

Conclusions: ICC or ICC-HCC is frequently associated with lymphatic metastasis and microvascular invasion, leading to high recurrence rates after liver transplantation. The most common sites of recurrence are lymph nodes, followed by the liver and lungs. Post-recurrence survival remains poor following transplantation.

Keyword(s) : cholangiocarcinoma, liver transplantation, outcomes, survival

2670 - Endoscopic Ultrasound Guided Partial Splenic Embolization in Hypersplenism A Novel Approach Validated in a pilot study

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Objective: To evaluate the safety, feasibility, and preliminary efficacy of endoscopic ultrasound-guided partial splenic embolization (EUS-PSE) in patients with decompensated cirrhosis and hypersplenism.

Materials and Methods: This pilot study enrolled patients with decompensated liver cirrhosis and hypersplenism who underwent EUS-PSE between September 2023 and April 2025 at two tertiary medical centers in China. Demographic and clinical data, changes in hematologic parameters, and perioperative and postoperative complications were recorded and analyzed.

Results: Thirteen patients (8 males, 5 females; mean age 53.5 ± 10.0 years) were included. According to the Child-Pugh classification, 5 (38.5%) were class A, 7 (53.8%) were class B, and 1 (7.7%) was class C. In 10 patients (76.9%), the post-procedural splenic infarction

ratio was <30%. In the remaining 3 patients (23.1%) with infarction ratios >30%, significant increases in platelet and leukocyte counts were observed. The success rate of the operation was 100%. The platelet level of the four patients increased significantly after the operation, and the clinical success rate was 30.7%. 4 patients developed post-procedural complications, including new or worsening pleural effusion and/or ascites. One patient developed a localized splenic abscess. All complications resolved with conservative management.

Conclusions: EUS-PSE appears to be a feasible and safe therapeutic option for managing hypersplenism in patients with cirrhosis. Hematologic improvement is more prominent when the splenic infarction ratio exceeds 30%. EUS-PSE may offer a minimally invasive alternative to conventional transarterial embolization. Further multicenter studies are warranted to confirm long-term efficacy and to determine the optimal infarction range (potentially 50%–70%).

Keyword(s) : Endoscopic Ultrasound-Guided Partial Splenic Embolization; Hypersplenism

2671 - Mechanistic Study of Resmetirom in Treating MAFLD Mouse Model Gut Microbiota Remodeling and Metabolic Regulation

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Background / **Hypothesis:**

The pathogenesis of metabolic associated fatty liver disease (MAFLD) is closely related to gut microbiota, but its specific mechanisms remain unclear. Resmetirom, as the first drug approved for the treatment of MASH with fibrosis, has not yet been studied in relation to gut microbiota. This study aims to investigate the role and mechanisms of gut microbiota in Resmetirom's treatment of MAFLD using multi-omics approaches.

Materials and **Methods:**

We established a MAFLD mice model with a high-fat diet (HFD) and treated mice with Resmetirom for 8 weeks. An FMT model was also constructed. Liver fat and lipid deposition were evaluated with HE and Oil Red O staining. Blood serum indicators were measured, and ileal contents were analyzed for metagenomics and untargeted metabolomics.

Results:



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1. Resmetirom and FMT Alleviate Metabolic Disorders and Hepatic Steatosis

Resmetirom and FMT improved glucose intolerance, insulin resistance (Fig1 A-C), hepatic steatosis, lipid deposition (Fig1 D-E), serum indicators (ALT, AST, TG, TC, LDL-C, HDL-C, Fig1F-K), liver weight, fat distribution, and adipose tissue weights (Fig1 L-Q). They also restored ZO-1 and Occludin levels in intestinal tissues (Fig2 Q-S).

2. Fecal Microbiota Composition and Key Taxa

Significant differences were found among groups (Fig2 A-B). Lefse identified 62 taxa between Control and HFD (Fig 2C), 53 between HFD and Resmetirom (Fig 2D), and 49 between HFD and FMT (Fig 2E). Parabacteroides was enriched in HFD but reduced in Resmetirom and FMT groups (Fig 2F).

3. Fecal Metabolites and Metabolic Pathways

3,475 metabolites across eight classes were identified (Fig 2G). Significant differences in fecal metabolites were observed among the Control, HFD, Resmetirom, and FMT groups (Figure 2H). The metabolic pathways involved in the differential metabolites between the HFD and Resmetirom or FMT groups were distinct (Figure 2I-N). Resmetirom increased butyric acid, indole derivatives, and deoxycholic acid but reduced palmitic and stearic acid (Fig 2O). FMT increased rosmarinic acid, citric acid, and indole-3-methyl acetate but reduced dimethyl hexasulfide, methyl methylthio selenide, and uracil (Fig 2P).

Conclusions: This study explored the role and mechanisms of Resmetirom in treating MAFLD using a HFD mice model and FMT experiments. Resmetirom improved glucose intolerance, insulin resistance, hepatic steatosis, blood lipid profiles, body weight, and fat tissue distribution. Its therapeutic effects were mediated by regulating specific bacterial taxa, restoring gut microbiota balance, and reversing MAFLD progression. Resmetirom also modulated key metabolic pathways linked to gut microbiota, enhancing host metabolic regulation. This study offers new molecular insights and preclinical evidence for Resmetirom in MAFLD treatment.

Keyword(s): MAFLD, Resmetirom,

2672 - Predictors of Post TIPS Persistent Ascites
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Background / **Hypothesis:** TIPS is used to lower portal pressure in patients presenting with acute variceal bleeding or refractory ascites. Some patients who undergo TIPS still require therapeutic paracentesis despite adequate portal pressure reduction and optimum tolerable doses of diuretics. Persistence of ascites post-one month of TIPS placement was defined as patients requiring LVP one month after the procedure, despite maximum tolerable doses of diuretics with no evidence of shunt stenosis. Shunt stenosis was diagnosed by angiographic evaluation showing greater than 50% stenosis, an increase in PSG to over 12 mm Hg or Doppler US criteria such as shunt peak systolic velocity less than 90 cm/sec indicating functional stenosis, absence of flow on color Doppler, main portal vein velocity less than 18-20 cm/sec.

Hypothesis: Determine the persistence of ascites after TIPS and identify variables contributing to its cause.

Materials and Methods: This retrospective cohort study was conducted at ILBS, New Delhi between March 2020 and March 2025, following institutional ethics approval. All cirrhotic patients undergoing TIPS for refractory ascites were screened. Patients who underwent TIPS for other indications, had acute Budd–Chiari syndrome, or lacked adequate follow-up or pre-TIPS data were excluded. Of 384 TIPS procedures, 107 patients met the inclusion criteria. Persistent ascites was defined as the requirement for LVP one month after TIPS despite optimal diuretic therapy and absence of shunt stenosis. Patients were divided into Group A (persistent ascites) and Group B (ascites controlled). Data included demographics, laboratory parameters, echocardiographic indices, renin and NT-proBNP levels, and TIPS procedural details. Statistical analyses were performed using parametric and non-parametric tests, and $p < 0.05$ was considered significant.

Results:

Of 107 patients analysed, 40 (37%) required therapeutic paracentesis at one month post-TIPS. Patients with persistent ascites had significantly higher pre-TIPS MELD scores, indicating more advanced liver disease. Echocardiographic parameters, including E/A ratio, E/e', ejection fraction, and TAPSE were comparable between

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groups, suggesting that overt cardiac dysfunction was not a major determinant. Renal function and synthetic parameters were similar between both groups. Smaller TIPS stent diameter and lesser portosystemic gradient (PSG) reduction were significantly associated with persistence of ascites. Hormonal analysis showed divergent renin responses post-TIPS, while NT-proBNP increased in both groups without statistical significance.

Conclusions:

.Persistent ascites post-TIPS was not related to cardiac diastolic dysfunction (E/A, E/e', TAPSE) or underlying baseline renal parameters
.MELD score and smaller TIPS diameter emerged as key factors influencing persistence of ascites

Keyword(s) :

2673 - Parallel Comparison of Two HBV pg RNA Detection Assays

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Background / **Hypothesis:**
Serum HBV RNA levels have been recognized as an effective biomarker for treating HBV patients. Currently, HBV RNA detection kits from different manufacturers exhibit inconsistent performance. This study aims to evaluate the performance of HBV RNA detection.

Materials and **Methods:**
We included patients treated with tenofovir disoproxil fumarate (TDF) for chronic hepatitis B (CHB) with advanced liver fibrosis/compensated cirrhosis at Peking University People's Hospital from January 2015 to December 2020, as well as patients undergoing HBV RNA serological testing at Peking University People's Hospital from December 2024 to January 2025. We performed performance validation on Reagent A. HBV RNA detection was conducted using both Reagents A and B on fresh samples, frozen samples, and non-concurrent refrigerated samples collected during the same period to assess correlation between the two reagents. Concurrently, levels of HBV DNA, HBsAg, HBsAb, HBeAg, HBeAb, and HBcAb were measured. Reagent A was compared on the ABI 7500 and ABI Q5 amplifiers for 16 low-concentration samples.

Pearson correlation coefficients (R) assessed linear correlation between quantitative results from both reagents. Simple linear regression fitted equations (R^2 evaluated model goodness-of-fit), combined with negative/positive results across sample groups, positive results above detection limits, and positive rates, analyzed detection consistency and detection capability.

Results:

Performance validation of Brand A reagent met all evaluation criteria.

Brand A reagent has a detection limit of 50 copies/mL, while Brand B reagent has a detection limit of 300 copies/mL.

Comparing 133 concurrent fresh samples, the two brands showed a common positive rate of $R \approx 0.9719$, with overall consistency at 88.7%. For samples above Brand B's detection limit, the positive agreement rate between the two brands reached 100%. Among jointly positive samples exceeding 300 copies/mL, the HBeAg positivity rate was 84.2% ($R \approx 0.98$). Samples with inconsistent low detection limits were predominantly HBeAg-negative, while HBeAg-positive samples demonstrated superior consistency and positivity rates. We compared 134 frozen samples. Brand B reagents detected samples above 300 copies/mL, while Brand A detected all such samples ($R \approx 0.9714$), achieving 92.53% overall concordance. For samples above the detection limit of Brand B reagents, the concordance rate between the two brands was 100%. Among 16 low-concentration samples tested on different instruments, the ABI 7500 amplifier demonstrated higher detection rates than the ABI Q5, indicating superior performance for detecting low-concentration HBV RNA.

Conclusions:

Comparative testing of fresh samples and frozen samples from both brands yielded satisfactory results. Reagent A met performance validation standards, and both brands of reagents satisfied testing requirements. The ABI 7500 demonstrated higher accuracy in results.

Keyword(s) : HBV pg RNA, Reagent Comparison

2678 - HBV recurrence in patients with hepatocellular carcinoma and cholangiocarcinoma following liver transplantation

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Background / **Hypothesis:**
To investigate risk factors for hepatitis B virus (HBV) recurrence following liver transplantation in patients with HBV-related hepatocellular carcinoma (HCC) or cholangiocarcinoma.

Materials and **Methods:**
This study included patients with cirrhosis due to chronic HBV infection who underwent liver transplantation for HCC or cholangiocarcinoma between February 2015 and February 2019 at a tertiary hospital. Participants were stratified based on post-transplant HBV recurrence status. Inter-group comparisons for categorical variables were performed using the chi-square test, while continuous variables were analyzed using Student's t-test or the Mann-Whitney U test, as appropriate. Univariate and multivariate Cox proportional hazards regression analyses were conducted to identify independent risk factors.

Results:

Among 112 enrolled patients, 109 had HCC and 3 had cholangiocarcinoma. Compared to patients without HBV recurrence, those with recurrence had significantly higher rates of detectable HBV DNA pre-transplantation (75.0% vs. 27.0%, $P=0.001$), microvascular invasion (100% vs. 67.8%, $P=0.020$), and more advanced TNM stages (Stage III+IV: 58.3% vs. 16.5%, $P=0.001$). They also exhibited larger maximum tumor diameter (6.3 cm vs. 2.5 cm, $P<0.001$) and were more likely to exceed the "Up-to-seven" criteria (83.3% vs. 22.0%, $P<0.001$). No significant differences were found in age ($P = 0.362$), sex ($P = 0.480$), or MELD score ($P = 0.678$). Post-transplantation, HBV recurrence occurred in 12 patients (10.71%) after a median interval of 15.0 months (IQR: 6.5-21.1). Of these, 11 cases (10.1% of HCC patients) followed HCC recurrence, and 1 case (33.3% of cholangiocarcinoma patients) followed cholangiocarcinoma recurrence. Overall, 33 patients experienced tumor recurrence, and 30 died. Among the 12 with HBV recurrence, 11 died due to tumor progression, while 1 remains alive with HCC under treatment. Multivariate analysis identified exceeding the "Up-to-seven" criteria (OR: 7.99, 95% CI: 1.19-53.49, $P=0.032$) and the presence of cholangiocarcinoma (OR: 116.7, 95% CI: 6.06-2250.5, $P=0.002$) as independent risk factors for HBV recurrence.

Conclusions:

HBV recurrence is closely associated with tumor recurrence post-liver transplantation. Patients presenting with larger or multiple tumors, or with cholangiocarcinoma before transplantation, are at significantly higher risk for HBV recurrence. Mortality in this cohort was primarily driven by tumor progression rather than HBV recurrence itself.

Keyword(s) : HBV; HCC; liver transplantation; cholangiocarcinoma

2679 - Outcomes of cholangiocarcinoma (ICC) or combined hepatocellular cholangiocarcinoma after liver transplantation

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Background / **Hypothesis:**
To investigate the characteristics and outcomes of patients with cholangiocarcinoma (ICC) or combined hepatocellular-cholangiocarcinoma (ICC-HCC) following liver transplantation.

Materials and **Methods:**
This study included patients diagnosed with ICC or ICC-HCC who underwent liver transplantation at a tertiary hospital between June 2015 and August 2018. Clinical characteristics and post-transplant outcomes were analyzed.

Results:

Seven patients were enrolled (6 males, 1 female), with a mean age of 55.1 ± 2.2 years. Prior to transplantation, 5 patients were diagnosed with hepatocellular carcinoma (HCC) and 2 with ICC-HCC. The mean maximum tumor diameter was 6.7 ± 1.3 cm, and the median AFP level was 134.2 (IQR 59.8–596.8) ng/mL. Two patients had a single tumor, while five had multiple tumors. Pre-transplant evaluation indicated that 2 patients met the Milan criteria, and 5 exceeded them. All patients had cirrhosis, with no evidence of extrahepatic malignancy. Post-transplant histopathology confirmed ICC in 2 cases and ICC-HCC in 5 cases. All patients exhibited microvascular invasion, and 3 had lymphatic metastasis. During follow-up, all 7 patients experienced tumor recurrence, with a mean recurrence interval of 2.8 ± 0.6

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months and a mean overall survival of 6.0 ± 0.9 months. Among the two patients initially assessed as meeting Milan criteria, both were upstaged post-transplant due to lymphatic metastasis, multiple tumors, and vascular invasion. Another patient initially within Milan criteria was found to have multiple tumors with a maximum diameter of 5 cm. One additional case, with multiple tumors (maximum diameter 2.5 cm), recurred first in the lung and retroperitoneal lymph nodes. Recurrence involved multiple organs in all cases: 85.7% (n=6) presented with initial lymphatic metastasis, 71.4% (n=5) with liver recurrence, 42.9% (n=3) with lung metastasis, and 14.3% (n=1 each) with adrenal or peritoneal metastasis. Patients received various treatments including radiation therapy, transarterial chemoembolization, and tyrosine kinase inhibitors. All patients died within 11 months after transplantation.

Conclusions:

ICC or ICC-HCC is frequently associated with lymphatic metastasis and microvascular invasion, leading to high recurrence rates after liver transplantation. The most common sites of recurrence are lymph nodes, followed by the liver and lungs. Post-recurrence survival remains poor following transplantation.

Keyword(s) : Cholangiocarcinoma; Liver transplantation; Histopathology; Hepatocellular-cholangiocarcinoma

2681 - Characteristics of Autoimmune Hepatitis in elderly patients

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Background: The prevalence and incidence of autoimmune hepatitis (AIH) are increasing yearly, and AIH in the elderly is also rising significantly. This study aims to investigate the characteristics of elderly AIH patients and explore the relationship between ALT levels and liver pathology.

Methods: A retrospective analysis was conducted on AIH patients who underwent liver biopsy and were diagnosed with autoimmune hepatitis (with an International Autoimmune Hepatitis Group (IAIHG) score > 15 or a simplified score > 6) at Tianjin Second People's Hospital from January 2011 to June 2025. Based on the preliminary results of the 2020 decennial

population census in China and the "World Health Statistics" published by the World Health Organization, patients were divided into two groups according to their age at diagnosis, using 60 years as the cutoff. Exclusion criteria included concurrent infections such as hepatitis B or C, alcoholic liver disease, drug-induced liver injury, non-alcoholic fatty liver disease, primary biliary cholangitis, primary sclerosing cholangitis, and others. Clinical data were retrieved from electronic medical records, including gender, age at diagnosis, presence of cirrhosis, and timing of liver biopsy. Laboratory test data, such as liver function parameters, collected within one week before the liver biopsy were also recorded. Patients with an onset age of ≥ 60 years were analyzed and compared with those aged <60 years. Results: A total of 356 patients were included in this study. The elderly group comprised 131 cases (36.8%), while the young and middle-aged group comprised 225 cases (63.2%). At the time of AIH diagnosis, liver pathology indicated cirrhosis in 27.5% (36/131) of the elderly group and 8.4% (19/225) of the young and middle-aged group, with the former being significantly higher than the latter ($\chi^2 = 54.674$, $P = 0.000$). Concurrent extrahepatic immune diseases were present in 22.1% (29/131) of the elderly group, which was higher than the 17.8% (40/225) in the young and middle-aged group, but the difference was not statistically significant ($\chi^2 = 1.007$, $P = 0.316$). Compared to the young and middle-aged group, the elderly group had lower levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) ($P = 0.012$, $P = 0.021$). Conclusion: In elderly patients with AIH, elevated serum ALT levels are often not significant, while liver histopathology may reveal more pronounced interface hepatitis and fibrosis. Relying solely on ALT levels to assess disease severity can easily lead to an underestimation of disease activity, thereby delaying diagnosis and intervention. Therefore, it is recommended to actively consider liver biopsy.

Keyword(s) : Autoimmune hepatitis, elderly, serum alanine aminotransferase, interface hepatitis, liver fibrosis

2690 - TACE plus Lenvatinib and Envafolimab for Conversion Therapy in Unresectable HCC A Prospective Phase II Study

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Background / **Hypothesis:**
Most patients with hepatocellular carcinoma (HCC) are diagnosed at intermediate or advanced stages, when curative resection is not feasible. Conversion therapy aiming to downstage tumors and enable surgical resection has emerged as a potential strategy. We conducted a prospective phase II study to evaluate the efficacy and safety of transarterial chemoembolization (TACE) combined with lenvatinib and the subcutaneous PD-L1 inhibitor envafolimab in patients with unresectable HCC.

Materials and **Methods:**

This single-center, open-label, single-arm phase II trial enrolled patients with Barcelona Clinic Liver Cancer stage B or C unresectable HCC between April and September 2024. Patients received conventional TACE combined with oral lenvatinib and subcutaneous envafolimab until surgical conversion, disease progression, unacceptable toxicity, or death. The primary endpoint was the conversion rate to curative-intent resection. Secondary endpoints included objective response rate (ORR), disease control rate (DCR), pathological response, progression-free survival (PFS), overall survival (OS), and safety.

Results:

Fifteen patients were enrolled. According to mRECIST criteria, the ORR was 53.3% and the DCR was 86.7%. Nine patients (60.0%) achieved sufficient tumor downstaging to undergo curative-intent surgery, and all achieved R0 resection. Pathological complete or major response was observed in five resected patients (55.6%). After a median follow-up of 16 months, the estimated median PFS was 12.0 months. One patient died during follow-up, yielding a 1-year OS of 100% and an 18-month OS of 93.3%. Treatment-related adverse events were manageable, with no unexpected safety signals.

Conclusions: TACE combined with lenvatinib and envafolimab demonstrated encouraging antitumor activity, a high conversion-to-surgery rate, and acceptable safety in unresectable HCC. These findings support further evaluation of this multimodal regimen as a conversion therapy strategy.

Keyword(s) : Hepatocellular carcinoma; Conversion therapy; Transarterial chemoembolization; Lenvatinib; Envafolimab

2691 - Splenic Stiffness Measurement to PREdict Decompensation in Patients with Compensated Advanced Chronic Liver Disease in India (SPRED c ACLD study)

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Background / **Hypothesis:**
Splenic stiffness measurement (SSM) is an emerging non-invasive modality for stratifying patients with compensated advanced chronic liver disease (c-ACLD). Herein, we aimed to determine the utility of SSM to predict one-year decompensation in patients with c-ACLD

Materials and **Methods:**
In this prospective study, all patients with c-ACLD (Baveno VII) were recruited from 2022 to 2024 at a tertiary care centre in north India. Subsequently, they underwent baseline SSM by vibration-controlled transient elastography method using a 100 Hz spleen-dedicated probe. They were followed up for a year to document decompensation and death.

Results:

Over three years, a total of 271 patients were recruited, and 40 were excluded. 231 patients were included in the final analysis. The mean age was 45.18 ± 9.73 years, and the majority were women (58.4%). The most common etiology of c-ACLD was chronic hepatitis C infection [75 (32.5%)]. One year- decompensation and death rate were 8.26% (19 patients) and 2.17% (five patients), respectively. The patient who decompensated had higher SSM, LSM, APRI, and FIB-4 (69.61 ± 16.27 kPa vs 43.99 ± 14.81 kPa, $p < 0.001$, 30.77 ± 11.26 kPa vs 22.68 ± 7.02 kPa, $p < 0.001$; respectively). In the Cox proportional hazards model, SSM predicted one-year decompensation [HR: 1.073 (1.049 - 1.097), $p < 0.001$]. With a cut-off 57.5 kPa, SSM has the highest sensitivity (82%), specificity (87.5%), positive predictive value (98.26%), negative predictive value (36.21%) and diagnostic accuracy (82.61%) for predicting decompensation in patients with c-ACLD. SSM did not predict the mortality in patients with c-ACLD

Conclusions:

SSM is a useful tool in predicting decompensation in patients with c-ACLD. A cut-off of 57.5 kPa has a sensitivity and specificity of 82% and 87.5%,

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respectively, for predicting one-year decompensation in these patients.

Keyword(s) :

2693 - Muscle Matters Impact of Sarcopenia on Mortality and Clinical Outcomes in Pediatric Liver Transplantation

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Background / Hypothesis: The impact of sarcopenia on outcomes following pediatric liver transplantation (LT) remains poorly defined. This study evaluated the association between the psoas muscle index (PMI) and post-transplant outcomes, including mortality, serious infections, surgical complications, and hospital length of stay (LOS), in pediatric LT recipients.

Materials and Methods: We performed a retrospective analysis of 293 pediatric LT recipients from January 2017 to January 2026. Sarcopenia was assessed using PMI (cm²/m²) calculated from preoperative CT imaging at the L3–L4 vertebral level. Patients were stratified into PMI quartiles. Outcomes analyzed included mortality, serious infections, surgical complications, and LOS. Multivariate logistic regression was used to identify independent predictors of adverse outcomes, adjusting for potential confounders including age and Pediatric End-Stage Liver Disease (PELD) score. Receiver operating characteristic (ROC) analysis was performed to identify the optimal PMI cut-off for mortality prediction.

Results: Valid PMI measurements were available for 255 patients. An inverse association was observed between muscle mass and adverse outcomes. Patients in the lowest PMI quartile (Q1, severe sarcopenia) had higher mortality compared with those in the highest quartile (Q4) (9.3% vs. 2.8%). The incidence of serious infections was also greatest in Q1 (75.0%) compared with Q4 (58.3%). Lower PMI was associated with longer hospital LOS (median 24 days in Q1 vs. 20 days in Q4). ROC analysis identified a PMI threshold of 4.89 cm²/m² for mortality prediction. In multivariate analysis, PMI below this threshold was associated with increased odds of mortality (OR 2.66; 95% CI 0.55–13.7; p=0.20), although statistical significance was not reached, likely due to low

overall mortality.

Conclusions: Low PMI is associated with increased infection rates, prolonged hospitalization, and higher mortality in pediatric LT recipients. A PMI cut-off of <4.89 cm²/m² may help identify high-risk children who could benefit from targeted pre-transplant nutritional intervention. Further studies examining long-term outcomes are ongoing.

Keyword(s) : Pediatric liver transplantation, Sarcopenia, Psoas muscle index, Post-transplant outcomes, Pediatric End-Stage Liver Disease

2695 - Candidemia in Liver Transplant Recipients A

Ten	Year	Retrospective	Study
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Background

Candidemia is one of the most severe forms of invasive fungal infections in liver transplant recipients and is associated with high morbidity and mortality. This study aimed to determine the incidence of candidemia in liver transplant recipients, to analyze the distribution of causative Candida species, to evaluate clinical risk factors and antifungal treatment approaches, and to assess the impact of candidemia on clinical outcomes and mortality.

Materials and Methods

The medical records of 1,880 adult patients who underwent liver transplantation at our center between January 2015 and January 2025 and were regularly followed were retrospectively reviewed. A total of 80 patients with Candida species isolated from blood cultures and diagnosed with candidemia were included in the study. Demographic, clinical, and laboratory data were obtained from patient records. Identification of Candida species was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). Antifungal treatment strategies were evaluated according to culture and antifungal susceptibility results.

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Results

The incidence of candidemia was found 4.25%. Of the patients, 67.5% were male, and candidemia most frequently developed during intensive care unit hospitalization. Non-albicans *Candida* species accounted for 62.5% of the isolates, with *Candida glabrata* and *Candida parapsilosis* being the most commonly identified species. Early initiation of antifungal therapy was significantly associated with a shorter time to blood culture clearance ($p = 0.003$) and lower mortality ($p = 0.032$). The overall mortality rate during candidemia was found to be 31.25% among patients with candidemia and no significant relationship was observed between mortality and causative candida agent.

Conclusions

Candidemia remains a serious complication associated with a high risk of mortality in liver transplant recipients. The predominance of non-albicans *Candida* species and the significant impact of early antifungal therapy on clinical outcomes highlight the need for continuous monitoring of center-specific epidemiological data and the implementation of risk-based empirical antifungal treatment strategies.

Keyword(s) : liver transplantation; candidemia; non-albicans *Candida*; MALDI-TOF MS; antifungal therapy

2699 - RAPID Procedure Enables Safe Use of Small Left Lobe Grafts A Two Center Experience

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Background

Resection and Partial Liver Segment Transplantation with Delayed Hepatectomy (RAPID) has been introduced as an innovative surgical strategy to enable liver transplantation using small grafts in carefully selected recipients. By combining staged hepatectomy with partial graft implantation, RAPID aims to overcome conventional graft size limitations while minimizing the risk of small-for-size-related complications. This study presents a two-center experience evaluating the feasibility and outcomes of RAPID transplantation using living-donor left lobe grafts.

Hypothesis:

Materials

and

Methods:

Between 2019 and 2026, 11 patients who underwent RAPID transplantation at two transplant centers were retrospectively analyzed. The median recipient age was 58 years (range: 23–64), with 5 female and 6 male patients. The median MELD-Na score was 18 (range: 8–29). All recipients received living-donor left lobe grafts, with graft weights ranging from 257 to 609 g. Recipient hepatectomy consisted of left lobectomy in 3 patients and left lateral sectionectomy in 8 patients. Biliary reconstruction was achieved using duct-to-duct anastomosis in 3 patients and hepaticojejunostomy in 8 patients. The actual graft-to-recipient weight ratio (GRWR) ranged from 0.35 to 0.58, with a median value of approximately 0.45. Inflow modulation strategies, including shunt narrowing, somatostatin infusion, and closure of splenorenal shunts, were selectively applied during the first stage to optimize portal inflow.

Results:

During the postoperative course, graft function demonstrated progressive adaptation and stabilization following the staged RAPID protocol. Portal inflow modulation strategies effectively supported graft perfusion and regeneration in selected recipients with low GRWR values. Across the cohort, no cases of small-for-size syndrome or early allograft dysfunction were observed. The interstage period allowed for controlled graft hypertrophy and functional recovery, facilitating safe progression within the RAPID framework. Overall, postoperative outcomes supported the technical feasibility of RAPID transplantation using small left lobe grafts under carefully controlled surgical and hemodynamic conditions.

Conclusions: RAPID transplantation represents a feasible and highly promising approach for extending the use of small left lobe grafts in liver transplantation. The absence of small-for-size syndrome and early allograft dysfunction underscores the reliability of this staged strategy when combined with meticulous surgical planning and tailored inflow modulation. RAPID has the potential to redefine graft size thresholds, expand the living donor pool, and provide a valuable alternative for complex transplant scenarios. Further prospective multicenter studies with larger cohorts are warranted to establish standardized indications and long-term outcomes.



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Keyword(s) : liver transplantation, RAPID procedure, left lobe graft

2700 - Does Primary Biliary Cholangitis and Autoimmune Hepatitis Increase the Risk of Precancerous Colon Polyps

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Background / Hypothesis: To evaluate the relationship between primary biliary cholangitis (PBC), autoimmune hepatitis (AIH), and colonic polyps.

Materials and Methods: The study included patients aged 18–65 with noncirrhotic primary biliary cholangitis and autoimmune hepatitis who underwent consecutive colonoscopies from January 2014 to July 2024. Patients were divided into three groups: those with PBC, those with AIH, and a control group. Patients with three or more adenomas, high-grade intraepithelial neoplasia, ≥ 10 mm tubular adenomas, or tumors with a villous component were classified as having high-risk polyps.

Results: The study included 42 patients with PBC and 42 with AIH. 142 consecutive patients who underwent screening colonoscopies comprised the control group. The average age at the time of colonoscopy was 58 years (51–63) in the control group, 53 years (44–60) in the AIH group, and 54.5 years (45–60) in the PBC group. Adenomatous polyps were detected in 40.5% of PBC patients, 16.7% of AIH patients, and 21.1% of the control group. The adenomatous polyp rate in PBC was statistically higher than in all other groups ($p=0.015$). When analyzing polyp location, polyps in PBC were more likely to be located in the transverse colon compared to those in AIH ($p<0.026$), while no differences were observed between the control group. Multiple adenomas were more prevalent in the control group compared to the other groups ($p=0.016$). Large adenomas (≥ 20 mm in diameter) were more frequently detected in the AIH group ($p<0.001$). High-risk polyps were present in 23.8%, 7.8%, and 2.4% of AIH, control group, and PBC patients, respectively, demonstrating a statistically significantly higher frequency in the AIH group ($p=0.002$).

Conclusions: Personalizing screening protocols to

account for individual risk factors may be a crucial step in enhancing screening effectiveness. The current study revealed an increased incidence of adenomatous polyps in patients with PBC and a higher risk of high-risk polyps in those with AIH. Future large-volume prospective studies will help inform decisions about screening colonoscopy at earlier ages and possibly at more frequent intervals in patients with AIH and PBC.

Keyword(s) :

2706 - A Novel Non invasive Model to Identify High Risk Varices in Patients with HBV related Cirrhosis

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Background / Hypothesis: Early detection of high-risk varices (HRV) in cirrhosis is critical for prognostic improvement. Over the past decade, several non-invasive diagnostic models of HRV in patients with advanced chronic liver disease have been developed. However, these models can only be used to exclude HRV patients but cannot effectively rule in HRV patients. This study aimed to develop a non-invasive model, for ruling out and ruling in HRV in patients with HBV-related cirrhosis.

Materials and Methods: This prospective study enrolled 616 consecutive patients with compensated HBV-related cirrhosis from July 2021 to April 2024 and followed until December 2025. Variables selected through least absolute shrinkage and selection operator regression were subsequently analyzed by multivariable logistic regression to construct the model.

Results:

Participants were randomly divided into training and validation cohorts (7:3 ratio). The model consisting of spleen stiffness measurement@100Hz, platelets, spleen diameter, spleen thickness, and sex, named PASS model, performed robustly in training and validation cohorts (AUROC: 0.94 and 0.92). Dual optimal cut-off values (4.84, 78.28) were selected for discriminating HRV. For ruling out HRV, PASS model spared 53.47% (231/432) unnecessary esophagogastroduodenoscopy (EGD) with a HRV missed rate of 1.11% (1/90), with sensitivity and negative predictive value were 98.89% and 99.57%, respectively. For ruling in HRV, 92.50% (37/40) of positive patients were correctly classified (specificity 99.12%, positive predictive value 92.50%). These results



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were then confirmed in validation cohort. The PASS model can also stratify the risk of decompensation events ($p < 0.001$). The 4-year cumulative incidences of liver decompensation were 0.93%, 4.59%, 21.93% in the HRV, gray zone, non-HRV groups, respectively.

Conclusions:

The PASS model is an accurate, novel, and non-invasive HRV diagnostic criterion with dual cut-off values in patients with compensated HBV-related cirrhosis. It is based on five routine laboratory and clinical parameters. PASS can identify patients with an extremely low probability of HRV (HRV misclassification rate $< 5\%$), allowing them to safely avoid EGD. At the same time, it can also accurately identify HRV patients (PPV $> 92\%$), enabling the timely initiation of primary prevention treatment. We also have provided evidence that the PASS model could accurately stratify the risk of decompensation.

Keyword(s) : chronic hepatitis B, liver cirrhosis, high-risk varices, PASS model, spleen stiffness measurement@100Hz

2711 - Related Factors for Low Level Viremia and the Comparative Efficacy of Management Strategies in Treatment Naïve Chronic Hepatitis B Patients Receiving Entecavir or Tenofovir Amibufenamide A Real World Study

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Background / Hypothesis: We aimed to investigate the related factors for low-level viremia (LLV) in treatment-naïve chronic hepatitis B (CHB) patients receiving entecavir (ETV) or Tenofovir Amibufenamide (TMF) and compare the efficacy of subsequent management strategies.

Materials and Methods: A total of 743 treatment-naïve CHB patients (467 receiving ETV and 276 receiving TMF) were enrolled. After 48 weeks of follow-up, patients were categorized into complete virological response (CVR) or LLV. Multivariate logistic regression was used to identify related factors for LLV. LLV patients then received different management strategies. Patients with LLV after ETV monotherapy (ETV-LLV) received one of three interventions: continued ETV monotherapy, switched to tenofovir

alafenamide/tenofovir amibufenamide (TAF/TMF) monotherapy, or add-on TAF/TMF. Patients with LLV after TMF monotherapy (TMF-LLV) received either continued TMF monotherapy or add-on ETV. Antiviral treatment effects were compared after 48 weeks of follow-up.

Results: After propensity-score matching (PSM), the week-48 complete virological response (CVR) rate was significantly higher in the TMF group than in the ETV group (76.80% vs. 62.50%, $P < 0.001$). Multivariate analysis of the matched cohort ($n=544$) identified that HBV DNA (aOR=1.209, 95%CI=1.034–1.412, $P=0.017$), HBsAg (aOR=3.675, 95%CI=2.242–6.025, $P < 0.001$), HBeAg (positive with high COI) (aOR=8.149, 95%CI=3.188–20.831, $P < 0.001$), liver cirrhosis (aOR=2.823, 95%CI=1.287–6.194, $P=0.010$), and fatty liver (aOR=3.485, 95%CI=1.936–6.270, $P < 0.001$) at baseline were independent risk factors for LLV. Conversely, initial therapy with TMF (vs. ETV) was a significant protective factor (aOR=0.236, 95%CI=0.135–0.413, $P < 0.001$). Among patients with ETV-LLV, the add-on TAF/TMF strategy achieved a higher CVR rate than continued ETV (72.3% vs. 40.4%, $P=0.003$), with significantly greater reductions in HBV DNA and HBsAg (both $P < 0.05$). For patients with TMF-LLV, adding on ETV did not significantly improve the CVR rate compared to continuing TMF (67.6% vs. 50.0%, $P=0.167$), nor did it enhance reductions in viral markers. No significant changes in lipid profiles or renal function were observed with any strategy.

Conclusions: Elevated baseline HBsAg, fatty liver, cirrhosis, higher HBV DNA, and high HBeAg COI are independent risk factors for LLV, while initiating therapy with TMF (vs. ETV) is a protective factor. For patients developing LLV on ETV, add-on TAF/TMF therapy significantly improves virological response and is a preferred strategy. In contrast, add-on ETV offers no clear benefit for TMF-LLV patients, highlighting the need to explore alternative management approaches.

Keyword(s) : Chronic hepatitis B; Low-level viremia; Entecavir; Tenofovir Amibufenamide; Antiviral therapy; Management strategy; Add-on therapy

2714 - Exposure to Legacy and Replacement PFAS Is Associated with Metabolic Dysfunction-Associated Steatotic Liver Disease in Women

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Background / **Hypothesis:**
Endocrine-disrupting chemicals (EDCs) are increasingly recognized as metabolic risk factors, yet their contribution to metabolic dysfunction-associated steatotic liver disease (MASLD) in women, particularly for emerging chemical substitutes, remains unclear. We applied a stepwise translational framework to investigate the epidemiologic association, phenotypic relevance, and mechanistic pathways linking EDC exposure to MASLD in women.
Materials and Methods:
First, we analyzed 476 women from NHANES 2017–2018 (217 MASLD cases; 259 controls) to examine associations between circulating EDCs and MASLD using multivariable logistic regression and Bayesian kernel machine regression. Second, an independent hospital-based female cohort (N = 74) was used to confirm exposure–phenotype concordance, with targeted measurement of PFOA, PFPeA, and HFPO-DA. Third, female C57BL/6J mice were exposed to PFOA and its replacements PFPeA and HFPO-DA to interrogate hepatic injury and metabolic pathway perturbations. Finally, data-independent acquisition–based serum proteomics in the clinical cohort was used to validate exposure-linked metabolic mechanisms in humans.
Results:

In NHANES, higher serum PFOA concentrations were associated with increased odds of MASLD (adjusted odds ratio, 1.73; 95% confidence interval, 1.01–2.97), and mixture analyses demonstrated a positive overall exposure–response relationship dominated by PFOA. In the clinical cohort, women with MASLD exhibited higher circulating levels of PFOA, PFPeA, and HFPO-DA. In mice, PFOA and HFPO-DA induced hepatomegaly, inflammatory activation, fibrotic marker elevation, and transcriptomic disruption of fatty acid and bile acid metabolism, whereas PFPeA elicited weaker but consistent pathway alterations. Serum proteomics in women identified differential abundance of lipid- and bile acid-related proteins (FASN, FABP5, SULT1A1, SULT1A3, ALDOB) that correlated with PFAS exposure.
Conclusions:

Using a human–animal–human translational framework, we demonstrate that both legacy and replacement PFAS are associated with MASLD in women through disruption of lipid and bile acid metabolic pathways, highlighting that chemical substitution does not necessarily eliminate hepatic metabolic risk.

Keyword(s) : Metabolic dysfunction–associated steatotic liver disease; Per- and polyfluoroalkyl substances; Endocrine-disrupting chemicals; Women; Lipid and bile acid metabolism

2729 - Lactate triggers HBV Specific CD8+ T cell exhaustion through mitophagy suppression mediated mitochondrial dysfunction

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Background / **Hypothesis:**
Hepatitis B virus (HBV) -specific T cells exhibit an exhausted phenotype associated with glycolytic metabolic disruption and perturbation of mitochondrial homeostasis. However, their relationship and underlying mechanisms remain elusive. Since lactate, a key glycolytic metabolite that can modulate immune response, we investigated its role in HBV-specific T cell dysfunction during chronic HBV infection.

Materials and Methods:
We recruited 242 patients with chronic HBV infection and 78 health controls. The metabolic characteristics of peripheral blood mononuclear cells (PBMCs) were comprehensively analyzed using RNA-seq, metabolomics and scRNA-seq. Additionally, we systematically evaluated the glucose uptake, mitochondrial function, autophagy flux, and mitophagy activity of HBV-specific CD8+ T cells using flow cytometry from both PBMC and liver mononuclear cells (LMCs). To investigate lactate-mediated T cell exhaustion, we performed exogenous stimulations with Bay-876, lactate, Oligomycin, and Midivi-1, and assessed through multiparametric cytokine profiling and functional assays.

Results:

The glucose metabolic status in PBMC was significantly activated during HBV infection, accompanied by the accumulation of lactate. Specifically, HBV-specific CD8+ T cells exhibited more dramatically increased Glut1 expression and 2-NBDG uptake than global T cells, especially in intrahepatic HBV-specific CD8+ T cells. Restraining glucose uptake via Bay-876 improved HBV-specific T cell function, while lactate accelerated their dysfunction. Mechanistically, lactate elicited aberrant mtROS production and mitochondrial membrane

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potential via suppressing mitophagy dysfunction. Meanwhile, reversing mitochondrial dysfunction by MitoQ and MitoTempo restored the HBV-specific T cells function. However, aggravating mitophagy impairment by Midivi-1 blunted the production of IFN- γ and TNF- α in HBV-specific T cell. Concurrently, dysfunctional autophagy also significantly contributed to HBV-specific T cells dysfunction, but this effect was independent of the lactate-mediated exhaustion pathway.

Conclusions: Lactate drives HBV-specific T cell exhaustion by disrupting mitochondrial function via restraining mitophagy. These results highlight lactate metabolism as a potential therapeutic target for chronic HBV infection.

Keyword(s) : HBV-Specific CD8⁺ T cell; Lactate; Mitophagy; Mitochondrial dysfunction

2731 - Tbx1 alleviated hepatic encephalopathy by promoting meningeal lymphangiogenesis and drainage of senescent astrocytes via a CCL21 dependent manner

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Background / Hypothesis: Emerging evidence suggested that meningeal lymphatic vessels (mLVs) were involved in intra-cranial drainage including senescent astrocytes in CNS disorders. Enhanced lymphangiogenesis was reported in hepatic encephalopathy (HE), when astrocyte senescence was potentially related to irreversible cognitive decline in HE. Therefore, based on single-cell RNA sequencing (scRNA-seq) of meninges, we aimed to investigate the role of mLVs in HE. We hypothesized that mLVs could drain senescent astrocytes from HE brain, when enhancing lymphangiogenesis and drainage by Tbx1 would be a potential therapeutic target to help reversing the cognitive impairment in HE.

Materials and Methods: scRNA-seq was performed on meninges from the rat model of HE induced by common bile duct ligation (BDL). mLVs and senescent astrocytes were visualized by immunofluorescence (IF) staining. To trace the drainage pathway, fluorescent labeled senescent astrocytes were injected into hippocampus. Intra-cranial lymphatic drainage efficiency was evaluated by OVA-FITC distribution in deep cervical lymph nodes (dCLNs).

Results:

scRNA-seq analysis suggested significant lymphangiogenesis and glial cell migration in HE meninges. In vivo study demonstrated that HE rats exhibited significant hippocampal accumulation of senescent astrocytes, which were further detected in mLVs and dCLNs. Exogenous tracer confirmed the direct drainage of senescent astrocytes from hippocampus to dCLNs through mLVs. Ligation of dCLN impeded senescent astrocyte efflux via mLVs and exacerbated HE. Tbx1 overexpression enhanced meningeal lymphangiogenesis and senescent astrocyte drainage, improving cognitive impairment and neuroinflammation, which were partially reversed by CCL21 neutralization.

Conclusions:

Our findings demonstrated that mLVs mediated drainage of hippocampal senescent astrocytes to dCLNs in HE rats, which played a vital role in ameliorating cognitive decline and neuroinflammation in HE. Tbx1 emerged as a novel regulator of mLVs, attenuating HE by promoting senescent astrocyte clearance through a CCL21-dependent manner.

Keyword(s) : Tbx1, Hepatic encephalopathy, Lymphangiogenesis, Meningeal lymphatic system, Astrocyte senescence, CCL21

2733 - Joint Association of C Reactive Protein and the Triglyceride Glucose-Chinese Visceral Adiposity Index with Incident Chronic Liver Disease A National Cohort Study

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Background / Hypothesis:

Chronic liver disease (CLD) is a major global health burden driven by the interplay of metabolic dysfunction and chronic inflammation. However, the long-term co-evolution of these processes—captured by the joint trajectories of biomarkers such as the triglyceride glucose-Chinese visceral adiposity index (TyG-CVAI) and C-reactive protein (CRP)—and its link to incident CLD risk remain unknown.

Materials and Methods:

In 4,078 participants from the China Health and Retirement Longitudinal Study (CHARLS) who were free of CLD at baseline, we identified joint CRP and TyG-CVAI trajectories using Kml3D clustering. Associations with

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incident CLD were assessed with generalized estimating equations (GEE), while generalized additive mixed models (GAMMs) examined nonlinear associations and interactions. Annual CLD incidence densities were calculated, and restricted cubic splines (RCS) within Cox models were used to determine risk thresholds for the time-weighted average TyG-CVAI.

Results:

Three distinct trajectory groups were identified: Low Inflammation–Metabolic Fluctuation (LI-MF), Low Inflammation–Persistently High Metabolism (LI-PHM), and Transient High Inflammation (THI). The LI-PHM was independently associated with a 33% increased CLD risk (OR=1.33, 95% CI: 1.05–1.6). Nonlinear relationships and a significant interaction between CRP and TyG-CVAI were confirmed by GAMMs ($p=0.043$). This group also showed the highest annual CLD incidence (peak 53.37/1000 person-years in 2018). RCS indicated two risk thresholds for time-weighted TyG-CVAI: HR=1.3 at 1193.08 and an inflection point at 1801.98 (HR=2.35). Subgroup analyses revealed the LI-PHM–associated risk was strongest in older adults, those with lower socioeconomic status, and sedentary individuals.

Conclusions:

Our findings demonstrate that sustained elevation of TyG-CVAI, even in the context of low-grade inflammation, is a significant driver of incident chronic liver disease, underscoring the need for early risk stratification and intervention based on long-term metabolic profiling.

Keyword(s) : Chronic liver disease, Triglyceride glucose, Chinese visceral adiposity index, C-reactive protein

2736 - Thalidomide reduces transfusion requirements due to gastrointestinal vascular malformations in patients with liver cirrhosis

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Background / Hypothesis: Gastrointestinal vascular malformations (GIVM), such as gastric antral vascular ectasia (GAVE) and gastrointestinal angiodysplasia (GIAD) are important

causes of chronic, transfusion-dependent bleeding in patients with liver cirrhosis. Standard endoscopic therapies often have limited success and high recurrence rates. Thalidomide, an anti-angiogenic agent, is a potential treatment, though evidence regarding its use in cirrhotic populations is limited. We aimed to evaluate the clinical response and safety of thalidomide in reducing transfusion requirements in cirrhotic patients with GIVM-related, transfusion-dependent anemia.

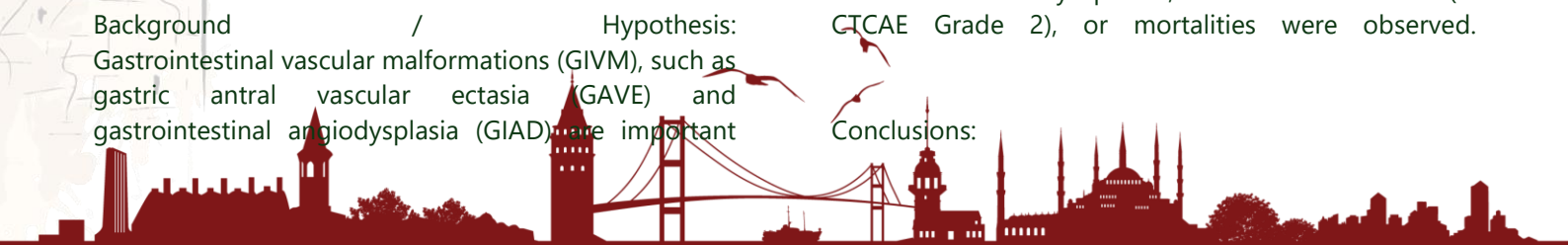
Materials and Methods:

This retrospective, single-center study included adults with liver cirrhosis and transfusion-dependent anemia due to GIVM who received thalidomide for at least three months. The primary endpoint was the change in packed red blood cell (PRBC) transfusion requirements following thalidomide therapy. Secondary endpoints included hemoglobin levels, endoscopy rates, endotherapy sessions, hospitalization rates, and treatment-related adverse events.

Results:

Fifty-seven patients with liver cirrhosis and transfusion-dependent anemia due to GIVM were included, most of whom were Child-Turcotte-Pugh (CTP) Class B (68.4%). Thalidomide was initiated at 50 mg/day and titrated to a maximum of 100 mg/day. The predominant etiologies were GAVE (59.6%) and GIAD (31.6%). Among the 54 patients with paired data, median PRBC units over a 3-month period decreased from 2.0 units (IQR 2.0–4.0) at baseline to 0.5 units (IQR 0.0–2.0) at the endpoint ($p < 0.001$). Mixed-effects negative binomial regression model demonstrated a 69% reduction in transfusion rate at 12 months (IRR 0.31, 95% CI 0.23–0.42; $p < 0.001$). This benefit was consistent across most subgroups, although it did not reach statistical significance in patients with Child C cirrhosis. Significant increase in mean hemoglobin levels was noted $\{7.32 \pm 1.38$ g/dL to 8.57 ± 1.53 g/dL ($p < 0.001$)}. Thalidomide therapy was also associated with a significant reduction in endoscopy procedures, daycare admissions and argon plasma coagulation sessions during the one-year post-treatment period. Peripheral neuropathy necessitating treatment cessation occurred in 5 patients (8.8%). No treatment-related cytopenias, serious adverse events ($> \text{CTCAE Grade 2}$), or mortalities were observed.

Conclusions:



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Thalidomide appears to be an effective and well-tolerated therapy for reducing transfusion requirements in cirrhotic patients with GIVM, even when administered at low doses.

Keyword(s) : "Thalidomide", "Liver Cirrhosis", "Gastrointestinal Hemorrhage", "Gastric Antral Vascular Ectasia", "Gastrointestinal Angiodysplasia", "Transfusion-Dependent Anemia"

2739 - D TACE Combined with HAIC Versus D TACE in BCLC Stage C HCC A Randomized Controlled Trial

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Background / Hypothesis: While both drug-eluting bead transarterial chemoembolization (D-TACE) and hepatic arterial infusion chemotherapy (HAIC) are recommended treatments for advanced hepatocellular carcinoma (HCC), definitive evidence on whether their combination improves survival in patients with Barcelona Clinic Liver Cancer (BCLC) stage C HCC remains uncertain. To compare the efficacy and safety of D-TACE combined with HAIC (D-TACE+HAIC) versus D-TACE alone in patients with BCLC stage C HCC

Materials and Methods: This multicenter, open-label, phase III randomized clinical trial was conducted across five hospitals in China. From April 2023 to April 2024, 220 patients with unresectable BCLC stage C HCC were included. Patients were randomized (1:1) to receive either D-TACE (oxaliplatin 100 mg or raltitrexed 4 mg, followed by embolization with 100-300 or 300-500 µm CalliSpheres® beads preloaded with 40-60 mg of doxorubicin or epirubicin) plus HAIC (oxaliplatin 85 mg/m², leucovorin 400 mg/m², 5-fluorouracil 400 mg/m² each infused over 2 hours, and 5-fluorouracil 2400 mg/m² infused for 46 hours) or D-TACE. The primary endpoint was progression-free survival (PFS). Secondary endpoints included overall survival (OS), objective response rate (ORR), disease control rate (DCR), and adverse events (AEs).

Results:

Median PFS was 6.4 months (95% confidence interval [CI], 5.8-8.4) with D-TACE and 10.2 months (95% CI, 9.2-13.5) with D-TACE+HAIC (hazard ratio [HR], 0.63; 95% CI, 0.47-0.85; P = 0.003). Median OS was 14.0 months

(95% CI, 12.4-17.7) versus 20.5 months (95% CI, 15.8-not reached), respectively (HR, 0.60; 95% CI, 0.42-0.85; P = 0.004). ORR was higher in the combination group (67.3%, 95% CI, 57.7%-75.9%) than in the D-TACE group (50.9%; 95% CI, 41.2%-60.6%, P = 0.014). Elevated transaminases were more frequent with D-TACE+HAIC, but other AEs were similar. No treatment-related deaths occurred.

Conclusions:

Compared with D-TACE alone, D-TACE+HAIC improved survival and tumor response in BCLC stage C HCC with a comparable safety profile.

Keyword(s) : hepatocellular carcinoma; drug-eluting bead transarterial chemoembolization; hepatic arterial infusion chemotherapy; efficacy; safety

2742 - PREDICTION OF NOSOCOMIAL INFECTION DEVELOPMENT REMAINS SUBOPTIMAL DESPITE USING MACHINE LEARNING TECHNIQUES IN A PROSPECTIVE GLOBAL COHORT OF HOSPITALIZED CIRRHOSIS PATIENTS

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Background / Hypothesis: Nosocomial infections (NI) are associated with high rate of mortality in inpatients with cirrhosis and vary globally. However, prediction of NI is hampered due to poor global representation and is unsatisfactory using traditional regression. This study aimed to develop NI predictive models using machine learning (ML) in a global inpatient cirrhosis cohort.

Materials and Methods: The CLEARED consortium includes globally-derived cirrhosis inpatientpatients with globally derived cirrhosis admitted non-electively.

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Demographics, cirrhosis details, inpatient course including NI development across income levels (high income, upper middle, low/low-middle income countries: HIC, UMIC, & LMICs) were recorded. Patients with/without NI were compared. Three ML models [Random Forest (RF), XGBoost & Neural Networks (NN)] were used for NI prediction. Data was split randomly 80/20 for training/testing. Model performance was assessed using AUC and compared with logistic regression (LR).

Results: A total of 8,263 patients were included from 127 centers and 37 countries (55.9 yrs, 64% men, 41% alcohol); 34% were from HIC, 42% UMIC & 24% L/LMICs. 19% had infections in 6 months prior to admission, 21% were admitted for an infection. 10.5% pts developed NI; these pts were younger (55 vs 57, $p<0.001$), from U/LMIC vs HICs (70 vs 64%, $p=0.03$) & alcohol etiology (45 vs 40%, $p=0.02$). NI pts had higher prior ascites/infections, SBP prophylaxis (15 vs 12%), admission MELD-Na (25 vs 19), albumin (2.7 vs 2.9 g/dl) & WBC (7.6 vs 5.9/mm³); all $p<0.001$). Admission with infections (30 vs 20%, $p<0.001$), especially SBP (13 vs 8%) & respiratory infections (8 vs 5%), and liver-related admissions (45 vs 35%) all $p<0.001$ were higher in those who developed NI. Machine-Learning Analysis: 6610 pts were in the training & 1653 pts were in the testing sets. 75 variables were included in the model. The models for NI prediction were suboptimal, but RF (AUC: 0.69 [0.65-0.74]) performed the best vs XGB (0.66 [0.62-0.70]) & NN (0.66 [0.62-0.70]), & was significantly stronger versus LR (AUC: 0.66 [0.61-0.70], $p=0.043$, Fig 2A). Prominent variables are in Fig 2B.

Conclusions: Nosocomial infections were found in 10% of cirrhosis inpatients in a prospective global cohort. Despite baseline differences, day of admission variables did not satisfactorily predict the NI development despite machine learning techniques, with the highest AUC of 0.69. However, these were significantly better than logistic regression. The suboptimal day of admission model performances shows that all inpatients with cirrhosis are at risk for nosocomial infections and should be monitored aggressively.

Keyword(s) : machine learning, CLEARED consortium, nosocomial infections, cirrhosis, disparities

2748 - A Retrospective Cohort Study on the Adherence to American Association for the study of Liver Diseases (AASLD) Quality Measures in Cirrhosis for Variceal Bleeding and its Association with Clinical Outcomes

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Background / Hypothesis: Acute variceal bleeding is a major cause of morbidity and mortality in patients with decompensated liver cirrhosis. The American Association for the Study of Liver Diseases (AASLD) has established evidence-based quality measures to guide management, yet real-world adherence and its effect on outcomes remain variable. The objective is to evaluate adherence to AASLD quality measures in the management of acute variceal bleeding and to assess its association with short-term clinical outcomes.

Materials and Methods: This retrospective cohort study included adult patients with decompensated cirrhosis admitted for endoscopy-confirmed acute variceal bleeding at a tertiary referral center from January 2021 to December 2025. Adherence to five AASLD quality measures—early antibiotic prophylaxis, vasoactive therapy, early endoscopy, endoscopic hemostasis, and nonselective beta-blocker prescription for secondary prophylaxis—was assessed. Clinical outcomes included inpatient mortality, infectious complications, early rebleeding within five days, and length of hospital stay.

Results:

A total of 152 patients were analyzed (mean age 52.3 ± 15.2 years; 59.2% male), predominantly Child–Pugh class B (80.9%). Adherence to individual measures was high for antibiotic prophylaxis (92.8%), vasoactive therapy (88.8%), endoscopic hemostasis (99.3%), and secondary prophylaxis (94.7%), but lower for early endoscopy within 12 hours (73.7%). Full adherence to all measures was achieved in 61.8% of patients. Inpatient mortality was 10.5%, early rebleeding occurred in 1.3%, and infections in 18.4%. Full adherence was not significantly associated with mortality, infection, early rebleeding, or length of hospital stay. Ceftriaxone and octreotide use independently predicted full guideline adherence.

Conclusions:

Adherence to AASLD quality measures was generally high, though timely endoscopy remains a key area for improvement. Early initiation of cornerstone therapies may serve as a surrogate marker for comprehensive guideline-centered care.

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Keyword(s) : Cirrhosis, variceal bleeding, adherence, ceftriaxone, octreotide

2753 - Clinical and biochemical predictors of Hepatocellular Carcinoma A Five Year Cohort Study from a Tertiary Care Center in Pakistan

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) has become one of the major causes of cancer related morbidity and mortality globally, especially in countries where chronic liver disease is highly prevalent. The Timely diagnosis, accurate staging, and appropriate treatment of the disease can be achieved by the early identification of clinical and laboratory predictors within the context of disease progression. This study evaluates the demographic, clinical, and biochemical profiles of patients diagnosed with HCC and to identify independent predictors associated with advanced-stage disease based on the Barcelona Clinic Liver Cancer (BCLC) classification.

Materials and Methods:

A five-year prospective study was conducted at Jinnah Postgraduate Medical Centre, Karachi, involving 516 patients diagnosed with HCC. Data were collected on demographic profiles, clinical features, laboratory investigations, imaging findings, tumor characteristics, and staging. Logistic regression analysis was performed to identify independent predictors of advanced-stage HCC (BCLC stage C-D).

Results:

The mean age of participants was 52.2 ± 9.6 years, with a male predominance (68%). The most common etiology was hepatitis C virus infection (61.8%), followed by hepatitis B (21.7%). Among patients, 43.4% had ascites, and 46.5% had portal vein thrombosis. A significant proportion (32%) presented with terminal-stage disease (BCLC D). Multivariable logistic regression analysis identified longer duration of risk factor exposure ($p = 0.010$), lower hemoglobin levels ($p < 0.001$), higher platelet counts ($p < 0.001$), reduced serum albumin ($p < 0.001$), elevated total bilirubin ($p < 0.001$), and higher serum potassium levels ($p < 0.001$) as independent predictors of advanced-stage HCC (BCLC C-D).

Conclusions: This study highlights key clinical and laboratory predictors associated with advanced hepatocellular carcinoma. The findings underscore the importance of early risk factor identification and routine monitoring of hematological and biochemical parameters for timely diagnosis and staging. These insights may assist clinicians in optimizing screening strategies and improving disease outcomes in resource-limited settings.

Keyword(s) : Hepatocellular carcinoma, BCLC staging, clinical predictors, laboratory markers, liver cancer, Pakistan

2756 - Locoregional Therapy Combined with Continuing or Switching Systemic Therapy for Intrahepatic Oligoprogression in Hepatocellular Carcinoma A Multicenter Retrospective Study

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Background / Hypothesis: The optimal management of intrahepatic oligoprogression during first-line immune checkpoint inhibitor-based therapy for advanced hepatocellular carcinoma (HCC) remains unclear. This study compared the outcomes of continuing first-line systemic therapy combined with locoregional therapy (FLST+LRT), switching to second-line systemic therapy combined with LRT (SLST+LRT), or switching to SLST alone.

Materials and Methods: This multicenter, retrospective study enrolled 168 patients. Patients were stratified into FLST+LRT ($n = 67$), SLST+LRT ($n = 64$), and SLST alone ($n = 37$) groups. Overall survival (OS) and progression-free survival were analyzed. Factors associated with OS were identified.

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Results: The median OS was significantly longer in both the FLST+LRT (31.7 months) and the SLST+LRT (37.2 months) group compared to SLST alone (11.2 months; $p = 0.004$ and $p = 0.001$, respectively). Subgroup analysis demonstrated that among durable responders, the median OS was significantly longer in the FLST+LRT group (not reached, $p = 0.004$) and the SLST+LRT group (27.5 months, 95% CI, 15.0 - 40.1, $p = 0.048$) than in the SLST alone group (15.6 months, 95% CI, 5.8 - 25.4). In non-durable responders, SLST+LRT resulted in longer median OS than both FLST+LRT ($p = 0.040$) and SLST alone ($p = 0.006$). Multivariate analysis identified that the absence of LRT following intrahepatic oligoprogression (HR 2.173, $p = 0.007$), increased ALBI grade (HR 2.979, $p = 0.006$), and AFP > 200 ng/mL (HR 2.208, $p = 0.006$) were independent risk factors for poorer OS.

Conclusions: Integrating locoregional therapies for HCC patients with intrahepatic oligoprogression significantly improves survival compared to switching systemic therapy alone.

Keyword(s) : Hepatocellular carcinoma · Systemic treatment · Immunotherapy · Locoregional therapy · Oligoprogression

2759 - α KB downregulates miRNA 363 3p and targets SERCA2b to alleviate hepatic endoplasmic reticulum stress and steatosis in MAFLD

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Background: Metabolic-associated fatty liver disease (MAFLD) has become a global health concern due to its high prevalence and extensive health consequences. Our previous study revealed that the serum levels of α -ketobutyrate (α -KB), a key metabolite in the transsulfuration pathway, are significantly reduced in MAFLD patients. However, the therapeutic potential and underlying mechanisms of α -KB in MAFLD remain largely unexplored.

Methods: HepG2 cells exposure to lipid mixture (LM) were used as vitro model. In vivo, MAFLD mouse model was induced by HFD intervention. The therapeutic effects of α -KB on MAFLD was evaluated through physiological, biochemical, and pathological analyses.

RNA sequencing was used to identify key regulatory pathways, which were then validated using real-time PCR (RT-PCR), immunofluorescence, and Western blotting. Prediction algorithms, dual luciferase reporter assays, adeno-associated viruses, siRNA, or inhibitors were used to verify the regulatory relationships of this pathway. Moreover, both in vivo and in vitro experiments were conducted to confirm the regulatory effect of α -KB on this pathway. Results: α -KB significantly alleviates hepatic steatosis and lipid metabolism disorders in both in LM-induced HepG2 cells and HFD-fed mice. Transcriptome sequencing, combined with in vitro and in vivo validation, identified the "protein processing in endoplasmic reticulum" pathway is the core pathway through which α -KB exerts its protective effects, with sarcoplasmic reticulum calcium ATPase 2b (SERCA2b) acting as its key regulatory target. α -KB treatment not only upregulated SERCA2b expression but also enhanced its ATPase activity. Mechanistically, SERCA2b was identified as a direct target of miR-363-3p. In the MAFLD model, α -KB suppressed miR-363-3p expression, thereby relieving its post-transcriptional inhibition of SERCA2b, which in turn alleviated endoplasmic reticulum stress and improving lipid metabolism abnormalities. The causal role of this axis was further confirmed by the findings that inhibiting miR-363-3p expression mimicked the protective effects of α -KB, while knockdown of SERCA2b largely abolished these therapeutic effects.

Conclusions: This study reveals for the first time that α -KB alleviates MAFLD by modulating the miR-363-3p/SERCA2b axis to mitigate ER stress and lipid metabolism abnormalities. These findings not only expand our understanding of the physiological functions of TSP but also provide a novel therapeutic target for MAFLD intervention.

Keyword(s) : Metabolic-associated fatty liver disease, α -ketobutyrate, transsulfuration pathway, sarcoplasmic reticulum calcium ATPase 2b, miR-363-3p

2760 - Real world Biochemical Responses to First line Azathioprine versus Mycophenolate and Upon Switching from Azathioprine to Mycophenolate in Autoimmune Hepatitis Patients

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Background

Hypothesis:

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Mycophenolate mofetil (MMF) has been found to be comparable to azathioprine (AZA) in achieving biochemical remission in autoimmune hepatitis (AIH). We aimed to compare real-world biochemical responses of first-line AZA and MMF, and responses after switching from AZA to MMF in AIH patients.

Materials and Methods: We analyzed a retrospective cohort of AIH patients treated with AZA or MMF. Patients received concomitant steroids 0-14 days prior to AZA or MMF. Biochemical responses were defined as: complete response (CR) = alanine aminotransferase (ALT) normalization; partial response (PR) = $\geq 50\%$ ALT reduction but above normal upper limits (ULN); non-response (NR) = $< 50\%$ ALT reduction or ALT increase. We analyzed a subset who switched from AZA to MMF.

Results:

Overall, 67 (AZA) and 12 (MMF) patients were included. CR progressively increased after 3 (AZA=52% vs. MMF=55.6%; $p=0.236$), 6 (AZA=61.7% vs. MMF=100%; $p=0.760$), and 12 months (AZA=76.9% vs. MMF=100%; $p=1.0$) of treatment, with no difference in NR rates (AZA=5.1% vs. MMF=0%; $p=1.0$) after 12 months. Despite similar response rates, AZA demonstrated greater median ALT reduction at 3 months vs. MMF (Δ ALT -111.8 vs -1.0 U/L; $p=0.008$). The reasons for AZA to MMF switch were AZA unavailability ($n=16$; 76.2%) and side effects to AZA ($n=5$; 23.8%). Following switching, most patients maintained biochemical stability after 6 months (87.5%). However, biochemical flare ($\text{ALT} \geq 2 \times \text{ULN}$) occurred in 3 (14.3%) patients, prompting reversion to AZA.

Conclusions:

Our data support the effectiveness of MMF as first-line therapy. However, switching from AZA to MMF may be associated with biochemical flare in a subset of patients, highlighting the need for cautious use in clinical practice with close biochemical monitoring.

Keyword(s) : AIH; Mycophenolate mofetil; Azathioprine
2762 - The Effects of Nomenclature Change on the Epidemiology of Fatty Steatotic Liver Disease
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Background / Hypothesis: The transition from non-alcoholic fatty liver disease (NAFLD) to metabolic dysfunction-associated steatotic liver disease (MASLD) reflects the growing recognition of metabolic drivers of this disease. However, the effects of this change on local epidemiology are not well-studied.

Materials and Methods: To describe the distribution and compare the clinical characteristics of NAFLD, MASLD, and metabolic and alcohol-associated liver disease (MetALD) patients. This retrospective study reviewed outpatient records of consecutive patients diagnosed with NAFLD, MASLD, or MetALD from January 2002 to August 2025. Demographic data, metabolic parameters, and noninvasive fibrosis measures including Fibrosis-4 (FIB-4), NAFLD Fibrosis Score (NFS), and FibroScan were collected.

Results:

Total of 1449 patients were included, 80% had MASLD, 11% NAFLD, and 9% Met-ALD. Mean age was similar across groups (≈ 51 years). MASLD and Met-ALD patients had higher mean BMI (27.79 and 27.31 kg/m²) compared with NAFLD (20.69 kg/m²). Liver enzymes were highest in the Met-ALD group. Mean ALT levels were 65.51 IU/L in Met-ALD, compared with 59.57 IU/L in MASLD and 46.52 IU/L in NAFLD. A similar pattern was seen with AST (48.46 IU/L in Met-ALD, 41.75 IU/L in MASLD, and 44.48 IU/L in NAFLD). Mean platelet counts were comparable across groups (MASLD $258 \times 10^9/L$, NAFLD $248 \times 10^9/L$, Met-ALD $241 \times 10^9/L$). Mean albumin levels were slightly lower in Met-ALD (4.26 g/dL) compared with MASLD (4.45 g/dL) and NAFLD (5.06 g/dL). For fibrosis markers, FIB-4 scores were lower in MASLD (1.23) than in NAFLD (1.51) ($p=0.039$) and Met-ALD (1.58) ($p=0.016$). In contrast, NFS indicated higher fibrosis risk in MASLD (-2.20) compared with NAFLD (-3.34) ($p=0.004$) and similar risk to Met-ALD (-1.46) ($p=0.055$). FibroScan values were numerically higher in MASLD (8.35) and Met-ALD (8.80) than in NAFLD (6.28), but differences were not statistically significant.

Conclusions:

This study shows that a multimodal assessment combining liver enzymes, synthetic function markers, metabolic risk profiling, and more than one noninvasive fibrosis tool may better identify patients at risk for progression who need closer follow-up, early intervention, and timely referral to hepatology.

Keyword(s) : metabolic dysfunction-associated steatotic

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liver disease; non-alcoholic fatty liver disease; metabolic dysfunction and alcohol-related liver disease; epidemiology

2765 - Disability Adapted Digital Therapeutics as a Precision Strategy for MASLD Risk Modulation Longitudinal Neuromuscular in Lower Limb Amputees

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Background / Hypothesis: Metabolic dysfunction associated steatotic liver disease (MASLD) is strongly influenced by skeletal muscle inactivity, insulin resistance, and autonomic imbalance. While structured exercise is a recognized strategy for metabolic liver risk reduction, conventional programs are primarily designed for the general population and often do not accommodate the biomechanical and functional constraints of individuals with physical disabilities. Lower limb amputees represent a metabolically vulnerable yet underserved group in hepatology research. We developed a disability-adapted mobile health (mHealth) digital therapeutic specifically engineered to align with amputee biomechanics and functional capacity, aiming to restore adaptive muscle activation and metabolic demand.

Materials and Methods: Twenty two adults with unilateral lower limb amputation completed a 12 week structured, disability specific digital exercise program integrating progressive strength conditioning and functional adaptation. Longitudinal muscle strength was assessed using Manual Muscle Testing (MMT), and exercise capacity was quantified by total repetitions performed. Quality of life (WHOQOL) was evaluated pre and post intervention. Repeated measures were analyzed using non-parametric methods (Friedman and Wilcoxon tests).

Results: Muscle strength increased significantly over time (Friedman $X^2 = 80.99$, $p < 0.001$; Kendall's $W = 0.74$), indicating sustained neuromuscular adaptation. Training load rose more than threefold from a median of 10 repetitions at week 2 to 36 at week 10 ($X^2 = 110.00$, $p < 0.001$; $W = 1.00$) demonstrating progressive physiological conditioning. Total quality of life improved

significantly (median 55.9 to 67.4; $p = 0.001$; $r = 0.66$).

Conclusions: A disability adapted digital therapeutic program successfully induced sustained neuromuscular remodeling and increased exercise capacity in a high risk amputee population. By aligning exercise design with biomechanical limitations, this precision mHealth strategy re-engages skeletal muscle as a metabolic regulator, targeting key drivers of MASLD. These findings support inclusive, translational approaches to metabolic liver disease prevention in populations historically underrepresented in hepatology research.

Keyword(s) : MASLD, Digital therapeutics, Exercise intervention, liver axis

2773 - Mechanistic Role of MFN2 FSP27 Mediated Lipid Droplet-Mitochondria Interactions in Metabolic Dysfunction associated Steatohepatitis

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Background / Hypothesis: Lipid metabolism disorder and mitochondrial dysfunction are key drivers of metabolic dysfunction-associated steatohepatitis (MASH), but the mechanisms by which lipid droplets (LDs) and mitochondria coordinate lipid metabolism remain unclear. This study aimed to investigate the role of the MFN2-FSP27 complex in mediating LD-MT interactions in fatty acid oxidation and metabolic reprogramming in MASH.

Materials and Methods: Liver tissues from MASH patients and controls were analyzed using immunofluorescence, confocal microscopy, and transmission electron microscopy to observe LD-MT contact sites. Western blot and co-immunoprecipitation were performed to assess MFN2 and FSP27 expression and complex formation. In vitro adipocyte and hepatocyte models with MFN2 or FSP27 knockdown/overexpression were used to examine fatty acid oxidation and metabolic reprogramming using metabolic assays and metabolomics.

Results: MFN2 expression was significantly downregulated in MASH liver tissues, accompanied by reduced LD-MT contacts and impaired FSP27-MFN2 complex formation. Restoration of the MFN2-FSP27 complex in vitro enhanced LD-MT interactions, promoted fatty acid delivery to mitochondria, and significantly increased fatty acid oxidation. Conversely,

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complex dysfunction led to abnormal fatty acid oxidation, metabolic reprogramming, and exacerbated lipid accumulation and inflammatory responses. Conclusions: The MFN2-FSP27 complex is crucial for LD-MT interactions, maintaining fatty acid oxidation and metabolic homeostasis. Its dysfunction contributes to lipid metabolism disorder and metabolic reprogramming in MASH, providing a potential target for therapeutic intervention.

Keyword(s) : metabolic dysfunction-associated steatohepatitis (MASH), lipid droplet mitochondria interactions, membrane contact site (MCS), fatty acid oxidation, MFN2

2774 - Adipose derived FABP5 drives myeloid bias in hematopoietic stem cells and promotes tumor progression.

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Background / Hypothesis: While obesity is a well-established risk factor for increased cancer incidence and mortality, the mechanisms driving this association, including how soluble factors or adipokines exacerbate tumorigenesis, remain elusive. Materials and Methods: We analyzed a clinical cohort of patients with hepatocellular carcinoma (HCC) to evaluate the association between body mass index (BMI) and overall survival (OS). To investigate the causal impact of obesity on tumor progression, diet-induced obesity (DIO) mouse models were combined with orthotopic liver tumor implantation. Flow cytometry, single-cell RNA sequencing (scRNA-seq), and bone marrow proteomics were used to characterize obesity-associated alterations in hematopoietic stem and progenitor cell (HSPC) fate and tumor progression. Subsequent functional validation of Fabp5 was conducted through bone marrow transplantation assays, Seahorse metabolic flux analysis, transgenic mouse models, and pharmacological inhibition to delineate the underlying regulatory mechanisms and define its functional role. Results: Clinical analyses revealed significantly poorer outcomes in obese patients with HCC. In mouse models, obesity accelerated HCC progression, accompanied by marked expansion of myeloid cells—particularly PMN-MDSCs—in peripheral blood, bone marrow, and tumors, along with impaired CD8⁺ T-cell responses. Obesity profoundly remodeled hematopoiesis, characterized by reduced hematopoietic stem cells (HSCs) and

multipotent progenitors and expansion of myeloid progenitors, indicating a strong myeloid bias. Single-cell transcriptomic and functional analyses showed that HSPCs under obese conditions exhibited metabolic reprogramming, enhanced endoplasmic reticulum stress, and loss of stemness. Proteomic profiling identified Fabp5 as markedly upregulated in the bone marrow niche and hematopoietic cells, with circulating levels positively correlating with obesity severity. Functionally, exogenous FABP5 promoted myeloid-biased differentiation, metabolic activation, and stress responses in HSCs, thereby accelerating tumor progression, whereas adipose- or hematopoietic-specific deletion of Fabp5 alleviated hematopoietic remodeling, immunosuppression, and tumor growth. Mechanistically, FABP5 activated the TLR4-MAPK/NF- κ B signaling axis, reprogramming the transcriptional and epigenetic landscape of HSCs to drive inflammatory, myeloid-skewed hematopoiesis under metabolic stress. Conclusions: This study identifies a systemic mechanism by which obesity promotes HCC progression through adipose tissue-derived circulating signals that remodel bone marrow hematopoiesis and the immune microenvironment. Fabp5 emerges as a key molecular hub linking metabolic dysregulation, inflammatory signaling, and tumor-associated immunosuppression across tissues. These findings provide new mechanistic insights into obesity-related hepatocarcinogenesis and highlight systemic metabolic-immune crosstalk as a promising therapeutic target.

Keyword(s) : Obesity; Adipose tissue; FABP5; Bone marrow niche; Hematopoietic stem and progenitor cells; Immune suppression

2779 - Re evaluation of Liver Biopsy Specimens Performed for Presumed Chronic Hepatitis B and Investigation of the Prevalence of MASLD

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Background / Hypothesis: The interaction between chronic hepatitis B (CHB) and metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as a clinically relevant entity in the era of rising metabolic burden. However, MASLD may remain under-recognized in CHB patients, even when liver biopsy is performed. We aimed to determine the histological prevalence of MASLD in CHB patients and evaluate both pathological reporting patterns and

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patient

awareness.

Materials and Methods: In this retrospective study, 100 consecutive CHB patients who underwent liver biopsy between January 2018 and November 2021 were included. Clinical, anthropometric, metabolic, and laboratory parameters were recorded. All biopsy specimens were blindly re-evaluated by two pathologists for hepatic steatosis and scored using the NAFLD Activity Score (NAS). MASLD diagnosis was defined according to current metabolic dysfunction criteria.

Results:

Of the patients, 52% were female and 17% were HBeAg-positive. Steatosis was identified in 32% of cases in the initial pathology reports. Upon re-evaluation, MASLD was detected in 34% of patients, with no statistically significant difference between the two assessments ($p=0.764$). However, steatosis was not explicitly stated in the diagnostic section of the original pathology reports. Patients with MASLD were significantly older and had higher body weight, BMI, waist and hip circumferences, and waist-to-hip ratio (all $p<0.01$). HbA1c, triglycerides, total cholesterol, LDL cholesterol, GGT, and uric acid levels were significantly higher in the MASLD group. Metabolic comorbidities—including diabetes, hyperlipidemia, hypertension, chronic kidney disease, metabolic syndrome, and obesity—were more prevalent in patients with MASLD. Notably, 97% of patients with biopsy-confirmed MASLD were unaware of their condition.

Conclusions:

MASLD affects approximately one-third of patients with CHB and is strongly associated with metabolic risk factors. Despite biopsy-based detection, it is frequently underreported and clinically unrecognized. Systematic assessment and explicit reporting of steatosis in CHB patients are essential to improve risk stratification and long-term management.

Keyword(s) : Chronic hepatitis B; MASLD; liver biopsy; metabolic dysfunction; steatosis awareness

2781 - Dual noninvasive prediction of esophageal varices in cirrhosis using transient elastography and echocardiographic parameters an interim machine learning analysis

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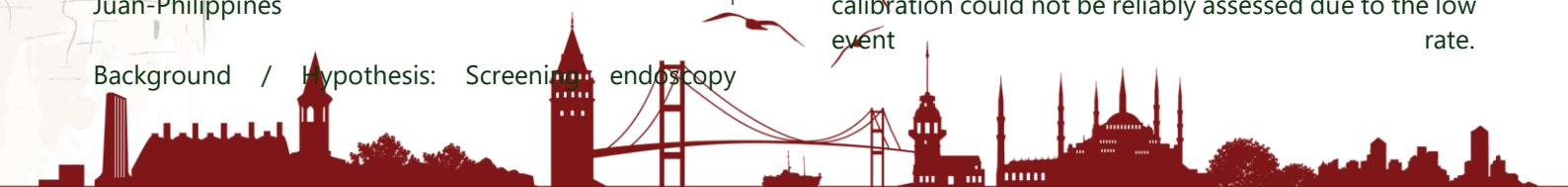
Background / Hypothesis: Screening endoscopy

remains the standard for detecting esophageal varices (EV) in cirrhosis, although many patients undergo unnecessary invasive procedures. The Baveno VII consensus recommends transient elastography (TE) combined with platelet count to identify patients at low risk of varices requiring treatment (VRT), allowing avoidance of screening endoscopy in selected cases. However, portal hypertension reflects both structural liver stiffness and systemic hemodynamic alterations that may not be fully captured by TE alone. We aimed to evaluate whether integrating echocardiographic parameters with TE improves noninvasive prediction of EV.

Materials and Methods: We prospectively analyzed adults with cirrhosis who underwent TE, transthoracic echocardiography, and screening endoscopy ($n=106$). EV prevalence was 6.6% (7/106). Data were divided into training and independent test datasets. To address class imbalance, synthetic minority oversampling was applied to the training dataset only. Logistic regression, naïve Bayes, random forest, and artificial neural network models were developed. Model performance in the independent test dataset was evaluated using sensitivity, specificity, accuracy, predictive values, likelihood ratios, and area under the receiver operating characteristic curve (AUC). Feature contribution was assessed using mean decrease in impurity and Shapley additive explanations.

Results: Patients with EV demonstrated significantly higher liver stiffness (24.37 ± 23.72 vs 6.12 ± 5.09 kPa, $p=0.008$), higher international normalized ratio ($p<0.001$), lower serum albumin ($p=0.024$), hyponatremia ($p=0.026$), and greater prevalence of portal hypertensive gastropathy ($p<0.001$).

In interim analysis, the random forest classifier demonstrated complete separation in both training and independent test datasets, achieving 100% sensitivity, 100% specificity, 100% accuracy, and AUC 1.00. Naïve Bayes showed good discrimination with 92.1% test accuracy and AUC 0.895. Logistic regression and neural network models demonstrated poor minority-class detection with 0% test sensitivity. Liver stiffness consistently emerged as the most influential predictor, demonstrating the highest feature importance and greatest Shapley contribution. Model discrimination remained stable across probability thresholds, although calibration could not be reliably assessed due to the low event rate.



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Conclusions: A dual noninvasive model integrating TE and echocardiographic parameters demonstrated promising predictive performance for EV. This multidimensional approach may complement Baveno VII criteria by refining risk stratification—particularly among patients outside established rule-out thresholds—and may help reduce unnecessary screening endoscopy while maintaining diagnostic safety. However, given the small event number and interim design, results must be interpreted cautiously. Prospective validation, calibration analysis, and external multicenter evaluation are required prior to clinical implementation.

Keyword(s) : Cirrhosis; Esophageal varices; Portal hypertension; Transient elastography; Echocardiography; Machine learning

2784 - The Effect of HALP Score on Predicting Post Transplant Outcomes in Patients with Hepatocellular Carcinoma

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Background:

This study evaluated the utility of the hemoglobin-albumin-lymphocyte-platelet (HALP) score in predicting disease-free survival (DFS) and recurrence in patients with hepatocellular carcinoma (HCC) undergoing liver transplantation (LT), and assessed its potential role as a prognostic biomarker in this population.

Methods:

A total of 476 patients who underwent LT for HCC between 2006 and 2024 were retrospectively analyzed. HALP scores were calculated, and a cut-off value of 29 for recurrence was determined by ROC analysis. Patients were stratified into HALP ≥ 29 and HALP < 29 groups. DFS and recurrence rates were compared. Prognostic performance was evaluated using the concordance index (C-index) and area under the ROC curve (AUC). Outcomes of patients with HALP ≥ 29 were also compared with those meeting the Milan Criteria and the Expanded Malatya Criteria.

Results:

Among 476 patients, 335 had HALP ≥ 29 and 141 had HALP < 29 . The 5- and 10-year DFS rates were significantly higher in the HALP ≥ 29 group than in the

HALP < 29 group (67.1% vs. 58.5% and 49.5% vs. 33.5%, respectively; $p < 0.001$). Recurrence rates were 14% (14/335) in the HALP ≥ 29 group and 31.9% (45/141) in the HALP < 29 group ($p < 0.001$) (Figure 1a). Patients within the Milan Criteria showed superior 5- and 10-year DFS (78.4% and 63.8%) and lower recurrence (3.3% [8/243]) compared with the HALP ≥ 29 group ($p \leq 0.017$) (Figure 1b). Similarly, patients meeting the Expanded Malatya Criteria demonstrated higher 5- and 10-year DFS (76.3% and 63.8%) and lower recurrence (4.9% [16/325]) than those with HALP ≥ 29 ($p \leq 0.037$) (Figure 1c). HALP ≥ 29 was associated with lower LTD, TTD, NLR, PLR, platelet count, Child, and MELD scores (Table 1), indicating more favorable tumor biology and hepatic functional reserve. However, C-index and AUC analyses showed inferior prognostic discrimination of HALP compared with established transplant criteria (Table 2).

Conclusion:

The HALP score predicts disease-free survival and recurrence after transplantation without including tumor-specific variables such as tumor size, number, or AFP levels. However, because its discriminative performance is lower than the Milan and Extended Malatya Criteria, it is not yet suitable for routine use as a selection criteria for liver transplantation.

Keyword(s) :

2785 - Metabolic Dysfunction Increases the Risk of Liver related Events in Virologically Suppressed HBV Related Compensated Cirrhosis Refining Prognostication with the Baveno VI SSM Model

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Background

The independent prognostic role of metabolic dysfunction (MD) in virologically suppressed HBV-related cirrhosis remains unclear. This study aimed to evaluate the prognostic value of MD and its components by combining them with the Baveno VI-SSM model for risk stratification of liver-related events (LREs; hepatic decompensation and hepatocellular carcinoma [HCC]).

Hypothesis:

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Materials and Methods:

Patients with HBV-related compensated cirrhosis and etiology control were prospectively enrolled and post-hoc analyzed to explore the potential impact of MD components on cumulative incidence of LREs. The MD component combined with the Baveno VI-SSM model was further adopted to stratify the risk of LRE in these etiology-controlled cirrhosis patients.

Results:

A total of 855 patients were enrolled, of whom 75.8% (n=648) had at least one MD component. Overall, 49 LREs occurred during a median follow-up of 31.0 months (IQR 26.5–43.5), including 12 decompensation events and 37 hepatocellular carcinoma (HCC) cases. Patients with MD had a higher cumulative LRE incidence than those without MD (7.1% vs. 1.4%; HR 5.02, log-rank $P=0.003$). All 12 decompensation events occurred in patients with MD ($P=0.035$), who also exhibited a higher incidence of HCC than those without MD. MD independently increased the risk of LRE (adjusted HR 3.48, 95% CI 1.07–11.33). Risk escalated dose-dependently by glycemic status (HR for prediabetes: 2.46, 95% CI 2.17–5.17; $P=0.007$; HR for diabetes: 4.01, 95% CI 1.63–9.89; $P<0.001$). By 48 months, the cumulative incidence of LRE was higher in patients with both MD and unfavorable Baveno VI-SSM criteria than in those with MD and favorable Baveno VI-SSM criteria (13.9% vs. 2.4%, HR 6.24, $P<0.001$).

Conclusions:

Metabolic dysfunction, particularly glycemic abnormalities (encompassing prediabetes and diabetes), independently predicts liver-related events in patients with etiology-controlled HBV-related compensated cirrhosis. Integrating metabolic assessment with the Baveno VI-SSM model further enhances risk stratification.

Keyword(s) : Metabolic Dysfunction; Baveno VI-SSM; Compensated cirrhosis; Liver-related events; Chronic hepatitis B

2789 - Comparison of the Safety and Efficacy of Tailored versus Fixed Dose Albumin in Cirrhosis with Sepsis Associated Acute Kidney Injury A Randomised Controlled Trial

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Background / Hypothesis: Sepsis-associated acute kidney injury (SA-AKI) in cirrhosis is marked by circulatory dysfunction and frequent cardiopulmonary complications. Fixed-dose albumin may cause more pulmonary complications. We compared a point-of-care ultrasound (POCUS)-guided tailored albumin strategy with fixed-dose albumin in cirrhosis patients with SA-AKI.

Materials and Methods: In this single-centre, open-label, randomised controlled trial, adults with cirrhosis and SA-AKI with proven or suspected sepsis were randomised 1:1 to POCUS-guided tailored 25% albumin or fixed-dose albumin (1 g/kg/day). The primary outcome was AKI resolution at 48 hours without cardiopulmonary complications.

Results: Altogether 100 patients were analysed (tailored, n = 50; fixed, n = 50). Baseline characteristics were comparable, including age (years: 53.4 ± 12.3 vs 52.6 ± 12.5 ; $p = 0.821$), MELD-Na (28.9 ± 5.25 vs 28.8 ± 5.23 ; $p = 0.796$), SOFA score (8.38 ± 1.48 vs 7.86 ± 1.58 ; $p = 0.12$), lactate (2.05 ± 0.44 vs 2.10 ± 0.64 ; $p = 0.62$), and proven sepsis ([86%] vs [84%]; $p = 0.98$). The majority had spontaneous bacterial peritonitis (58% vs 52%; $p = 0.64$), and nosocomial acquisition was seen in 30% vs 28% ($p = 0.89$). AKI resolution (complete/partial) at 48 hours without cardiopulmonary complications was significantly higher with tailored dosing (42% vs 14%; $p = 0.03$). By day 7, AKI recovery remained superior in the tailored group (78.0% vs 52%; $p = 0.02$). Cumulative albumin dose was significantly lower in the tailored arm at 48 hours (107.1 ± 35.5 g vs 147.0 ± 24.8 g; $p < 0.001$) and at day 7 (162.1 ± 77.5 g vs 228.4 ± 80.8 g; $p < 0.001$). Cardiopulmonary complications were markedly reduced with tailored dosing (22% vs 78%; $p < 0.001$). On multivariate analysis, POCUS-guided tailored strategy (OR = 4.81, 0.94–24.62), lower SOFA score (OR = 0.53, 0.30–0.95), and NT-proBNP (OR = 0.997, 0.995–1.000, and lower urea (OR = 0.94, 0.90–0.98), and Δ change in inferior vena cava diameter (OR = 0.34, 0.16–0.73) independently predicted AKI resolution. The patients who needed oxygen support were significantly lower in the tailored-dose versus fixed-dose group (22% vs 80%; $p < 0.001$). The time to initiation of vasopressor

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therapy was delayed in the tailored-dose group [80 h (78–86) vs 42 h (29–50); $p < 0.001$].

Conclusions: POCUS-guided, tailored albumin therapy reduces the need for albumin infusion, leading to early AKI resolution and a significantly lower incidence of cardiopulmonary complications and dialysis in cirrhosis with SA-AKI. Lower SOFA score and NT-proBNP, the

Keyword(s) : SA-AKI, POCUS, CIRRHOSIS, NT-proBNP,

2793 - 4 1BB Costimulation Restore Tfh1 Function to Augment HBV Specific Humoral Immunity in Chronic Infection

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Background / Hypothesis:Chronic HBV infection is closely associated with dysregulated humoral immunity. Although our study demonstrated that Mother-to-child transmission (MTCT) could be dramatically reduced by the comprehensive interventions among HBV-infected pregnant women (Nature Medicine,2024), how intrauterine HBV antigen exposure shapes host humoral immunity remains incompletely understood. Here, we investigated the humoral immune profile in cord blood from neonates born to HBV-infected mothers to identify potential therapeutic targets and validate whether targeting these pathways could restore HBV-specific humoral immunity through clinical cohorts with persistent chronic infection.

Materials and Methods:We analyzed cord blood samples from 73 neonates born to HBsAg-positive mothers and 24 counterpart controls (HBsAg-negative mothers) within our previously reported clinical cohort(NCT05172453). B cell and CD4+ T cell phenotypes and transcriptomes were analyzed by flow cytometry and RNA-seq. HBV-specific B cells were quantified via ELISPOT and ELISA. Additionally, peripheral blood from 85 untreated chronic HBV patients (aged 1–65 years) was evaluated by full-spectrum flow cytometry to determine Tfh1-cell functional dominance and age-related distribution.

Results:Cord blood from HBV antigen-exposed

neonates exhibited enhanced HBV-specific B cell responses along with significantly elevated frequencies of type 1 follicular T helper cells (Tfh1) compared to controls. In vitro, HBsAg could induced naïve CD4+ T cells to differentiate into Tfh1 cells, but not the other T helper cells (Th1/Th2/Th17) or alternative follicular T helper cells (Tfh2/Tfh17) subsets. These Tfh1 cells secrete both IFN- γ and IL-21, which synergistically enhanced HBsAb production by B cells via the activation of mTORC1-STAT1/STAT3 pathway. These data demonstrate that primary HBV antigen exposure preferentially induces a Tfh1-polarized immune response that initiates HBV-specific humoral immunity. To assess Tfh1 dynamics in chronic infection, we performed full-spectrum flow cytometry on a clinical HBV cohort, revealing an age-dependent decline in both Tfh1 frequency and 4-1BB expression, which positively correlated with HBsAb-secreting B cell counts. Strikingly, 4-1BB stimulation in vitro restored Tfh1 function, rescuing IFN- γ and IL-21 production and enhancing B cell help.

Conclusions:Tfh1-derived IFN- γ and IL-21 potentiate HBsAb production by activating mTORC1-STAT1/STAT3 signaling in B cells. The age-dependent decline in Tfh1 frequency and 4-1BB expression suggests progressive immune dysfunction in chronic HBV infection. Targeting 4-1BB may rejuvenate Tfh1 activity, offering a novel strategy to restore HBV-specific humoral immunity.

Keyword(s) : HBV, 4-1BB, Humoral immunity

2796 - Interferon α programs HBsAg specific humoral immunity through the monocyte IL6 Tfh cell axis to facilitate functional cure of hepatitis B

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Background / Hypothesis: Achieving a functional cure for chronic hepatitis B virus (HBV) infection remains an unmet challenge. Our recent clinical trial demonstrated the combination of siRNA

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with peg-IFN α resulted in a higher rate of functional cure compared to siRNA monotherapy (New Engl J Med. 2024). This finding substantiates an important role of IFN α in mediating functional cure, however, its underlying mechanisms remain poorly defined, particularly in how IFN α shaping antiviral humoral immunity. Herein, we aimed to elucidate how IFN α regulates B cell immunity to facilitate functional cure.

Materials and Methods: We longitudinally followed CHB patients receiving Peg-IFN α therapy and stratified them based on virological outcomes at week 72: HBeAg-non-seroconversion (eAg-NSC, n = 11), HBeAg-seroconversion (eAg-SC, n = 7), and HBsAg seroconversion (sAg-SC, n = 6). In addition, 3 CHB patients who achieved HBsAg seroconversion after five years of nucleos(t)ide analogues therapy (NAs-sAg-SC) were enrolled for comparison. Peripheral blood was collected for scRNA-seq and flow cytometry. The effects of IFN α on immune subsets and HBV-specific B cell responses were further examined through in vitro cytokine stimulation assays and in vivo validation using IL6 $^{-/-}$, IL21 $^{-/-}$, Ccr2 $^{-/-}$ chimeric, and Cxcl13 $^{-/-}$ HBV mouse models with IFN α plasmid.

Results: scRNA-seq and flow cytometry revealed that Peg-IFN α selectively expanded Tfh and plasma cells specifically in sAg-SC patients. However, in vitro stimulation of purified CD19 $^{+}$ B cells with IFN α showed minimal effects indicating the involvement of intermediary cells. Given the robust upregulation of IL6-related gene programs in monocytes, we further investigated IFN α 's effects on these cells and confirmed that IFN α enhanced humoral immunity by augmenting monocyte-derived IL6 secretion. Intriguingly, while IL-6 failed to directly activate B cells, subsequent analyses revealed that IFN α enhanced IL6-mediated Tfh cell interactions and promoted IL6 ST -high Tfh subsets with elevated IL21 expression in sAg-SC patients. In vitro IL6 stimulation increased IL21 production in CXCR5 $^{+}$ CD4 $^{+}$ T cells, whereas IL21 induction was abolished in IL6 $^{-/-}$ or monocyte-deficient mice. Transcriptomic profiling of IL21-stimulated B cells showed significant enrichment of humoral response. scRNA-seq further revealed elevated IL21-IL21R signaling between Tfh and GC-like/plasma B cells exclusively in IFN α -treated sAg-SC patients. Finally, spatial analysis demonstrated that IFN α promoted GC formation via CXCL13-CXCR5 signaling. Notably, GC development was abrogated in IL6 $^{-/-}$, Ccr2 $^{-/-}$ chimeric, and Cxcl13 $^{-/-}$ mice.

Conclusions:

IFN α promotes anti-HBV humoral immunity via a monocyte-Tfh-B cell axis, strategically bridging innate and adaptive immunity to facilitate HBV cure. These findings provide mechanistic insights and identify potential targets to optimize IFN α -based therapeutic strategies.

Keyword(s) : Interferon α , HBV, functional cure, humoral immune, germinal center

2799 - Cathepsin K Attenuates Liver Fibrosis by Suppressing TGF β Smad Signaling via T β R I Degradation

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Background & Aims: Liver fibrosis, driven by hepatic stellate cell (HSC) activation, lacks effective therapy. Cathepsin K (CTSK) has context-dependent roles in fibrosis, but its function in liver fibrosis remains unclear. This study investigated the role and underlying mechanisms of CTSK in liver fibrosis. **Methods:** CTSK expression was analyzed in human fibrotic livers via transcriptomics and confirmed in murine fibrosis models. Its function was investigated in primary HSCs and LX-2 cells by assessing the effects of CTSK overexpression on cell activation and signaling pathways. The therapeutic potential was evaluated using AAV8-mediated CTSK overexpression in two etiologically distinct murine fibrosis models. **Results:** CTSK was upregulated in activated HSCs and fibrotic livers. Mechanistically, CTSK mediated a negative feedback loop that inhibited TGF- β /Smad signaling by promoting Smad7/Smurf2-dependent T β R-I degradation, thereby suppressing HSC activation. Notably, AAV8-mediated CTSK overexpression attenuated liver fibrosis in multiple murine models. **Conclusions:** CTSK functions as an endogenous protective factor that attenuates liver fibrosis by negatively regulating the TGF- β pathway via T β R-I degradation. These findings identify CTSK as a promising therapeutic target for liver fibrosis.

Keyword(s) : Liver fibrosis; Hepatic stellate cells; TGF- β /Smad signaling; Cathepsin K

2809 - Long Term Survival in De Novo Liver Metastatic Colorectal Cancer A Descriptive Cohort Study

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Background / Hypothesis: Historically, the prognosis for metastatic colorectal cancer (mCRC) has been poor. Nevertheless, modern systemic therapy and aggressive local treatment strategies have enabled a subgroup of patients to achieve long-term survival. The objective of this study was to describe clinical, molecular, and treatment characteristics of patients with de novo liver-metastatic mCRC who achieved long-term (>5 years) survival.

Materials and Methods: We performed a retrospective descriptive cohort analysis of patients with histologically confirmed colorectal adenocarcinoma who were diagnosed at a tertiary cancer center from January 2015 to September 2025, had de novo liver-metastatic disease at diagnosis, and achieved overall survival >5 years. Clinical, molecular, and treatment data were collected from electronic health records. Variables were summarized using descriptive statistics (median [IQR] and n [%]).

Results: A total of 27 patients were included. The median age at diagnosis was 56.9 years (IQR 52.2–64.3), and 63.0% were men. Primary tumor location was 25.9% right colon, 55.6% left colon, and 18.5% rectum. At diagnosis, 44.4% had distant lymph node involvement; peritoneal metastasis and lung metastasis were uncommon (3.7% each). According to hepatic metastatic burden classification at diagnosis, 44.4% of patients had oligometastatic liver disease, and 55.6% had multiple liver metastases. Primary tumor resection was performed in 92.6% of patients, and metastasectomy in 70.4%. 61.5% of patients had RAS wild-type tumors, whereas RAS mutations were detected in 38.5%; MSI-high status was identified in 3.8%. First-line chemotherapy was most commonly FOLFOX (74.1%), followed by FOLFIRI (11.1%). Median number of systemic therapy lines in the metastatic setting was 2 (IQR 1–3). Regorafenib was used in 18.2% of patients. Metastatic disease-directed local therapy was administered in 44.4% of patients; among locally treated patients, the most common modalities were

radiotherapy (66.7%) and radiofrequency ablation (50.0%), with combined local therapy (≥2 modalities) used in 33.3%. Median follow-up from diagnosis to last contact was 75.4 months (IQR 63.9–106.0), and 75.0% of patients were alive at last follow-up.

Conclusions: Long-term survival beyond 5 years is achievable in selected patients with de novo liver-metastatic mCRC. High rates of surgical management and the use of local therapies—including multimodality approaches—support the potential value of an aggressive, multidisciplinary treatment strategy in this population.

Keyword(s) : Metastatic colorectal cancer, Liver metastases, Long-term survival

2810 - Sodium Glucose Co Transporter 2 Inhibitors Use in Diabetic Patients with Cirrhosis Improves Liver related Outcomes and Survival

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Background / Hypothesis:

Sodium glucose cotransporter-2 inhibitors (SGLT2is) are widely used to treat type 2 diabetes mellitus (T2DM). The health outcomes of SGLT-2i in T2DM patients with cirrhosis have not been studied.

Materials and Methods:

We performed a retrospective analysis from 1/1/2010 to 12/31/2025 by utilizing two databases.

Adults with T2DM aged 18-90 with history of cirrhosis, for at least 90 days and newly started on SGLT-2i were identified in two separate databases. The two cohorts (T2DM with cirrhosis on SGLT-2i and T2DM with cirrhosis without SGLT-2i) were compared at baseline and after using propensity score matching (PSM). The primary aim was to compare adverse liver-related outcomes (ALO) among patients with T2DM with cirrhosis on SGLT-2i (intervention group) with those without SGLT-2i (control group).

Clinical outcomes were reported as incident rate ratios (IRRs), and compared using a Poisson regression model

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for binary variables. Survival probability was analyzed using Kaplan-Meier (KM) survival curves and hazard ratios (HR) calculated using Cox proportional hazards regression. Statistical significance was determined using a priori p-value of < 0.05.

Results:

The intervention group consisted of 14,231 SGLT-2i users and the control group consisted of 249,746 non-SGLT-2i users. After 1:1 PSM, use of SGLT-2i was associated with reduced composite outcome of hepatic decompensation compared to control group at 1 year (9.9% vs. 12.5%, $p=0.001$), at 3 years (16.4% vs. 18.1%, $p<0.001$) and 5 years (19.9% vs 21.9%, $p<0.001$). HCC rates were significantly lower in the SGLT-2i users at 1 year (2.3% vs. 3.5%, $p=0.001$), at 3 years (2.9% vs. 4.2%, $p=0.001$) and at 5 years (3.4% vs. 4.8%, $p=0.001$). There was a 31% reduction in mortality in the SGLT-2i arm (HR: 0.69; CI: 0.56- 0.83, $p<0.001$), as compared to the non-SGLT-2i arm.

These findings were validated in the EVERSANA database.

Conclusions:

Use of SGLT-2i was associated with a reduced composite outcome of hepatic decompensation and mortality among patients with T2DM and cirrhosis compared with non-SGLT-2i users across two independent datasets. Prospective randomized controlled trials are needed to confirm these findings.

Keyword(s) : sodium glucose co-transporter 2 inhibitors, cirrhosis, hepatic decompensation, hepatocellular carcinoma, mortality.

2814 - Etiological distribution clinical phenotypes and six month outcomes of drug induced liver injury

a single center cohort study
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Background / Hypothesis:
Drug-induced liver injury (DILI) is a leading cause of acute liver dysfunction with variable outcomes. Identifying high-risk phenotypes and common causative agents may improve early detection and management. We aimed to evaluate etiological distribution, clinical phenotypes, disease severity, and

six-month outcomes in patients with DILI.

Materials and Methods:
Between 2021 and 2024, 5012 inpatients and outpatients from internal medicine and gastroenterology clinics were retrospectively screened using ICD codes R94.5 (abnormal liver function tests) and K71 (toxic liver disease). Patients meeting laboratory-based American Association for the Study of Liver Diseases (AASLD) criteria for drug- or herbal-induced liver injury were identified. Drug or herbal/dietary supplement exposure within six months was assessed, and cases with Roussel Uclaf Causality Assessment Method (RUCAM) scores ≥ 3 that were concordant with LiverTox data were included ($n=198$). Patients with viral hepatitis, biliary obstruction, sepsis, heart failure, autoimmune or metabolic liver disease were excluded.

Results:

Fifty-four percent of patients were managed as outpatients, while 46% required hospitalization. According to RUCAM grading, 3% were possible, 86% probable, and 11% highly probable. Nearly half of the patients (44%) were asymptomatic at diagnosis. Hepatocellular injury was the predominant pattern (62%), followed by mixed (24%) and cholestatic (14%) patterns. Diabetes mellitus and higher serum glucose levels were more frequent in the cholestatic pattern, whereas multiple-drug exposure was more common in the mixed pattern. Anti-infective agents were the most common exposures (41%), followed by musculoskeletal (30%) and nervous system drugs (27%). Most cases were mild (82%; Drug-Induced Liver Injury Network [DILIN] severity grade 1), whereas 13% were moderate (DILIN severity grades 2–3) and 5% were severe or fatal (DILIN severity grades 4–5). Hy's law criteria were met in 8.6% of patients and were confined to moderate and severe cases. Six-month mortality was 4%, and chronic liver injury developed in 7% of patients.

Conclusions:

DILI remains a diagnosis of exclusion and should be suspected in patients with unexplained liver test abnormalities. Widely prescribed medications—particularly anti-infective, musculoskeletal, and nervous system drugs—were the predominant causes, often in the context of multi-drug exposure. Although most cases were mild, clinically significant presentations occurred. Careful drug history and structured causality assessment are essential to guide management and improve patient outcomes.

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Keyword(s) : drug-induced liver injury, herbal-induced liver injury, RUCAM, DILIN

2815 - Associations Between Dietary recommendations diet quality and Steatotic Liver Disease Prevalence and Severity in U.S. Adults 2017–2023

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Background / Hypothesis:

The 2025–2030 Dietary Guidelines for Americans (DGA-2025) introduced substantive changes to national nutrition guidance, including expanded emphasis on total protein intake and omission of prior alcohol recommendations. We evaluated how individual dietary components—derived from both the Healthy Eating Index-2020 (HEI-2020) and energy-adjusted z-score models—relate to steatotic liver disease (SLD), fibrosis, and cirrhosis in U.S. adults.

Materials and Methods:

We analyzed data from 10,944 adults (NHANES 2017–2023) with complete 24-hour dietary recall and liver assessment via vibration-controlled transient elastography (VCTE). Dietary exposures included HEI-2020 component scores and z-scores derived from food group densities aligned with DGA-2025 structure. Alcohol intake was assessed using a 6-point Alcohol Adherence Score, reflecting thresholds previously established in DGA-2020. Multivariable logistic regression and stepwise selection models were adjusted for age, sex, race/ethnicity, education, poverty-income ratio, diabetes, obesity, caffeine intake, and NHANES survey weights.

Results:

Across both HEI-2020 and z-score models, several components were consistently associated with liver outcomes. High intake of total fruit, whole fruit, vegetables, greens and beans, whole grains, and seafood/plant protein was associated with significantly lower prevalence of SLD and fibrosis. In contrast, higher intake of red and processed meats, sodium, total protein, saturated fat, and added sugars was associated with greater disease prevalence. Alcohol adherence was independently protective across all outcomes. In stepwise models, components retained as independent predictors of liver disease included greens and beans, seafood and plant protein, saturated fat, and alcohol adherence. Notably, whole grain and sodium—both retained in DGA-2025—were associated with favorable liver outcomes. However, components with consistent protective associations, such as greens and beans, seafood and plant protein, and alcohol moderation, were omitted or de-emphasized in the updated guidelines. Conversely, total protein—now strongly promoted—was independently associated with higher fibrosis and cirrhosis prevalence.

Conclusions:

Multiple dietary components associated with liver health are either omitted or misaligned in the DGA-2025 framework. While several elements—such as whole grains and sodium—remain appropriately emphasized, the removal of alcohol guidance and de-emphasis on protein source may obscure key risk-reducing behaviors for steatotic liver disease and fibrosis. These findings underscore the need for liver-specific dietary guidance that distinguishes protein quality, reinforces sodium and saturated fat limits, and explicitly integrates alcohol-related risk.

Keyword(s) :

2818 - Updated results of the observational study to assess virological effectiveness safety and tolerability of NVR RTV+SOF combination in HCV infected people living with HIV

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Background / **Hypothesis:**
Narlaprevir (NVR) is a NS3 protease inhibitor of HCV used for hepatitis C therapy in combination with ritonavir (RTV) and other direct-acting antivirals (DAAs). The effectiveness of NVR/RTV + Sofosbuvir (SOF) once-daily for 12 weeks was assessed in a large population of Russian people who are infected with HCV (GT 1) and living with HIV (PLWH). HIV/AIDS clinical centers from 8 Russian Regions were involved in the study.

Materials and **Methods:**
Adults treated with NVR 200 mg co-administered with

RTV 100 mg + SOF 400 mg for 12 weeks were included in this prospective observational study. All HCV patients reaching Week 12 post-treatment were assessed for SVR12. All PLWH initiated antiretroviral therapy before starting therapy with NVR/RTV + SOF.

Results:

Overall, 1472 patients with co-infection HIV/HCV who received at least one dose of NVR/RTV+SOF were included in this analysis: 61.55% male, 63.62% with fibrosis F0-F2 (METAVIR score), 57.95% with HIV viral load < 50 copies/ mL; 71.54% with HIV stage 3 (WHO clinical staging), 48.5% current/former alcohol or intravenous drug use. Most patients were white men (99.73%). Median age was 42 years (range 38–47 years).

Conclusions: According to Russian clinical guidelines patients with HIV coinfection are considered a priority population for the treatment of hepatitis C with DAAs. Genotype-specific treatments for HCV are being actively employed in national plan for eliminating HCV infection in Russia. NVR/RTV + SOF in HIV/HCV co-infection was highly effective and tolerable in clinical practice, drug use is a potential barrier to reach HCV elimination in PLWH.

Keyword(s) :

2832 - Immune Excluded CD4⁺ and CD8⁺ T Cell Depletion Predicts Early Recurrence in Hepatocellular Carcinoma with Microvascular Invasion

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Background / Hypothesis: Early recurrence within six months after resection continues to pose a major clinical challenge in hepatocellular carcinoma (HCC) patients, occurring in approximately 31.9% of patients. This study aims to investigate whether reduced immune cell infiltration across intratumoral and peritumoral compartments with early HCC recurrence among

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patients exhibiting high angiogenic activity and microvascular invasion.

Materials and Methods: A multicenter nested case-control study was conducted using liver tissue samples from 49 patients with HCC who underwent surgical resection. Clinical data were retrieved from medical records. Pathological and immunological parameters were assessed using hematoxylin-eosin [H&E] staining for tumor-infiltrating lymphocytes (TILs), and immunohistochemistry for VEGF, CD4, CD8, and regulatory T cells. The analytical approach included ROC curve analysis, Kaplan-Meier survival estimation, and multivariate Cox regression.

Results: Early recurrence occurred in 11 of 49 (22.45%) patients, all showing high VEGF and most with microvascular invasion. Patients with early recurrence had markedly lower intratumoral CD4⁺ T-cell density and reduced peritumoral CD8⁺ T-cell, while peritumoral CD4⁺ and overall TILs remained high than intratumoral. Multivariable analysis identified low intratumoral CD4⁺ (HR 7.98; 95% CI: 1.63–39.07) and decreased peritumoral CD8⁺ (HR 4.98; 95% CI: 1.14–21.70) as independent predictors. Intratumoral and peritumoral Treg densities showed no significant association.

Conclusions: Low intratumoral CD4⁺ and peritumoral CD8⁺ infiltration were identified as independent predictors of early HCC recurrence, reflecting an immune-excluded tumor phenotype characterized by high peritumoral CD4⁺ and TILs. In this context, angiogenesis-driven stromal and vascular barriers impede effective T-cell trafficking into the tumor core, facilitating early recurrence, particularly in tumors with microvascular invasion. These findings underscore the need for intensified post-resection surveillance and may define a high-risk subgroup for future evaluation of adjuvant therapies.

Keyword(s) : angiogenesis, CD4, CD8, early recurrence, hepatocellular carcinoma, microvascular invasion, TILs, Treg

2833 - Hyperinsulinemia Establishes a Pathogenic Feedback Loop between Pyroptotic Hepatocytes and MDSCs in MAFLD

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¹

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Background and Scientific Question: Metabolic dysfunction-associated fatty liver disease (MAFLD) is a prevalent chronic liver disorder whose progression can lead to cirrhosis and even hepatocellular carcinoma. Hyperinsulinemia is a common clinical feature in MAFLD patients; however, whether and how it directly drives disease progression, along with its underlying molecular mechanisms, remains unclear.

Main Results and Conclusion: This study demonstrates that hyperinsulinemia is a key driver of MAFLD exacerbation. Mechanistically, hyperinsulinemia downregulates the demethylase FTO, increases m6A modification of the antioxidant transcription factor NRF2 mRNA, and promotes its degradation. The loss of NRF2 subsequently activates the NLRP3/Caspase-1/GSDMD signaling pathway, inducing hepatocyte pyroptosis. The pyroptosis-related cytokines IL-1 β and IL-18 released from hepatocytes promote the aberrant generation and hepatic recruitment of myeloid-derived suppressor cells (MDSCs). These MDSCs further exacerbate hepatocyte pyroptosis, thereby forming a pathogenic feedback loop of "hepatocyte pyroptosis-MDSC expansion" that continuously drives hepatic inflammation and injury.

Significance: This study is the first to reveal a novel mechanism by which hyperinsulinemia induces hepatocyte pyroptosis via the "FTO/m6A-NRF2 axis" and forms a vicious cycle with the immunosuppressive microenvironment. These findings provide a new perspective for understanding MAFLD pathogenesis and identify FTO, NRF2, and MDSCs as potential therapeutic targets, laying an important theoretical foundation for developing precise treatment strategies against MAFLD.

Keyword(s) : Hyperinsulinemia; N6-methyladenosine (m6A); Pyroptosis; Myeloid-derived suppressor cells (MDSCs)

2834 - Epidemiologic and Clinical Trends in Hepatocellular Carcinoma A Glimpse Through the Lens of Systemic Therapy Availability

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Background / Hypothesis: Recent global shifts in epidemiology and diversity of treatment options for Hepatocellular Carcinoma (HCC) may affect its prognosis. Documenting these changes

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may help address gaps in the care of HCC patients. We aimed to analyze temporal trends in epidemiology and prognosis across three treatment eras: the pre-tyrosine kinase inhibitor (TKI) era (Period 1= <2008), the post-TKI era (Period 2= 2008–2019), and the immunotherapy era (Period 3= >2019).

Materials and Methods:

Consecutive HCC patients diagnosed between 2000-2025 from a single hepatology database were included. Demographics, tumor and treatment characteristics, and survival data were recorded

Results:

A total of 1068 patients were included (Period1=202 vs. Period 2=633 vs. Period 3=233). There was an increase in age of patients ($p<0.001$) and number of tumors/patient ($p<0.001$) over time. More patients presented with Child-Pugh A and Barcelona Clinic Liver Cancer (BCLC) 0-B in the latter years ($p<0.05$). There was a shift in the etiology with viral hepatitis decreasing (Period 1=62.2% vs. Period 2=29.3% vs. Period 3=33.5%) and steatohepatitis increasing (Period 1=1.0% vs. Period 2=27.3% vs. Period 3=40.5%) over the years. Less patients were left untreated (Period 1=42.6% vs. Period 2=31.4% vs. Period 3=23.3%) while more patients received systemic therapy (Period 1=6.5% vs. Period 2=9.7% vs. Period 3=43.6%) through the years. Encouragingly, median overall survival (OS) improved (Period 1=43.4weeks vs. Period 2=118.9weeks vs. Period3=109.9weeks; $p<0.001$). OS improvement was seen in BCLC 0-A but not in BCLC B-D patients.

Conclusions:

We have demonstrated a remarkable shift in etiology of HCC, with steatohepatitis becoming the most prevalent. The shift in lower BCLC stage and earlier use of systemic therapy may be contributing to the increased survival rate.

Keyword(s) : Hepatocellular Carcinoma, Epidemiology, Clinical Trends

2839 - CAN EARLY CHANGES IN ALPHA FETOPROTEIN PREDICT RADIOLOGIC RESPONSE AND OVERALL SURVIVAL IN IMMUNE CHECKPOINT INHIBITOR BASED THERAPY OF HEPATOCELLULAR CARCINOMA

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Background / Hypothesis: On-treatment alpha-fetoprotein (AFP) dynamics have been proposed as early biomarkers of response in hepatocellular carcinoma (HCC) treated with immune checkpoint inhibitors (ICIs). We aimed to determine the prognostic significance of AFP response in HCC patients treated with ICIs.

Materials and Methods: We conducted a retrospective study of consecutive patients with HCC treated with ICIs with at least 1 follow-up imaging from January 2021 to December 2025. AFP response was assessed at 4 and 12 weeks and categorized as responder (>20% decrease) versus non-responder (<20% decrease). Radiologic responses using RECIST 1.1 were classified as complete response(CR), partial response(PR), stable disease(SD), and progressive disease(PD). Overall survival (OS) was estimated using Kaplan-Meier method.

Results:Majority (n=27;87.1%) received atezolizumab-bevacizumab while the rest received durvalumab or pembrolizumab-based treatment. At 4 weeks, there was a trend for better radiologic response in AFP responders vs. non-responders (PR=28.6% vs. 17.6%; SD=57.1% vs. 41.2%; PD=14.3% vs. 41.2%; $p=0.272$), although this was not statistically significant. A similar trend was observed with AFP response at 12 weeks ($p=0.138$). There was a trend for OS to be associated more with radiologic response ($p=0.09$) than AFP response at 4 ($p=0.894$) and 12 ($p=0.280$) weeks. None of the baseline characteristics predicted OS.

Conclusions:AFP responses at 4 and 12 weeks were neither significant predictors of radiologic response nor OS in HCC patients treated with ICIs. The lack of a significant association between radiologic response and OS points to the need for a bigger study to better elucidate the relationship between AFP and radiologic responses with survival.

Keyword(s) : Hepatocellular carcinoma, AFP, Immune checkpoint inhibitors

2841 - PLPP2 drives liver repair and regeneration via remodeling phosphatidylethanolamine metabolism after acute liver injury

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Background / **Hypothesis:**
Acute liver injury is commonly caused by overdose-drug, toxins, or ischemia. The liver possesses a remarkable regenerative capacity, wherein metabolic remodeling plays a crucial role. However, the specific metabolic shift that initiates hepatocyte regeneration remains unclear. We aimed to identify the key metabolic process driving the transition from liver injury to regeneration.

Materials and **Methods:**
We employed multi-time-point proteomic and metabolomic profiling to identify the key metabolic pathway and associated regulatory genes. We analysed liver samples from DILI patients and employed gene-modified mice. Transcriptomic and metabolomic were applied to validate the underlying mechanism.

Results:
Multi-time-point multi-omics analyses demonstrated that phosphatidylethanolamine undergoes a dynamic metabolic reprogramming, with PLPP2 exhibiting synchronous changes among the enzymes regulating PE metabolism. Liver biopsy samples from patients with drug-induced liver injury corroborated the PLPP2 alterations. In murine models, hepatocyte-specific deletion of PLPP2 demonstrated its protective effect against ALI. Mechanistically, the resolution of ER stress was accompanied by a robust upregulation of PLPP2 during the regenerative phase. This shift promotes liver repair and regeneration by initiating the PE biosynthesis. PE, upon binding to PEBP1, modulated its affinity and strengthened its interaction with GLI1, thereby disrupting the association between ITCH and GLI1 and preventing the ubiquitination and degradation of GLI1. Additionally, PE promotes autophagy by facilitating the lipidation of LC3B. The increase of in ER-localized PE mediated by PLPP2 also contributed to alleviation of ER stress, thus establishing a positive feedback loop. Pharmacologic activation of PLPP2 may represent a promising therapeutic strategy for ALI.

Conclusions:
The transition from reduction to elevation of PE maps to the pathological shift of liver from injury to regeneration following ALI, wherein PLPP2 serves as a switch initiating PE biosynthesis. Thus, targeting PLPP2 may represent a promising therapeutic strategy for ALI.

Keyword(s) : Multi-omics; liver regeneration; phosphatidylethanolamine; ER stress; Hedgehog signaling; autophagy

2844 - AARS1 promotes tumor progression and immune evasion via ATF6 lactylation-mediated tryptophan metabolism in hepatocellular carcinoma
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Background / **Hypothesis:**
Hepatocellular carcinoma (HCC) remains a leading cause of cancer-related death, and many patients derive limited benefit from systemic and immunotherapies owing to a strongly immunosuppressive tumor microenvironment. Regulatory T cells (Tregs) are central to this state and correlate with poor prognosis and resistance to immune checkpoint blockade, yet how tumor-intrinsic metabolic and stress programs are converted into stable Treg-promoting signals in HCC is unclear. Alanine-tRNA synthetase 1 (AARS1) was recently identified as a lactate-sensitive protein lactyltransferase, revealing a noncanonical role in coupling glycolysis to protein modification. However, its clinical relevance in HCC and how its lactylation activity shapes tumor biology and Treg-mediated immune evasion are unknown. The tryptophan-catabolizing enzyme TDO2 is a key inducer of Treg accumulation in HCC, but its upstream regulation by tumor lactylation has not been defined. This study aims to clarify the role of AARS1 in HCC progression by (i) defining its clinical and functional impact, (ii) determining how AARS1-mediated ATF6 lactylation drives TDO2-dependent tryptophan metabolism, (iii) establishing how this axis promotes Treg accumulation and tumor growth, and (iv) testing whether targeting the AARS1-ATF6-TDO2 pathway provides therapeutic benefit in experimental HCC models.

Materials and **Methods:**
1. Single-cell and spatial RNA-seq from HCC patients defined AARS1 expression patterns and associated programs. 2. HCC was induced in hepatocyte-specific Aars1/Atf6/Tdo2 knockout mice by DEN/CCl₄ and Myc/Ras. 3. Tumor glycolysis was assessed by micro-PET/CT and ECAR in HCC and tumor-derived cells. 4. AARS1 lactyltransferase activity was evaluated by PPi/AMP and ATF6 K424 lactylation. 5. Immune subsets were profiled by flow cytometry and CyTOF.

Results:
AARS1 was upregulated in HCC and associated with glycolysis, Treg accumulation and poor prognosis. Mechanistically, AARS1 lactylated ATF6 K424 to drive TDO2-kynurenine signaling and Treg expansion, which

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in turn enhanced glycolysis. β -alanine inhibition of AARS1 broke this loop, restrained HCC and improved anti-PD-1 response.

Conclusions:

We identify AARS1 as a lactylation-dependent hub linking tumor glycolysis to ATF6–TDO2–kynurenine signaling and Treg-mediated immunosuppression in HCC. Disrupting this AARS1–ATF6–TDO2 axis with β -alanine remodels the tumor microenvironment, restrains HCC progression, and enhances anti-PD-1 efficacy, highlighting a tractable metabolic–epigenetic therapeutic target.

Keyword(s) : AARS1, lactylation, Treg, ER stress, tryptophan metabolism, immune evasion

2848 - Physician–Patient Dual Track Follow Up Care Bundle in Hepatology A Multidisciplinary Practice Model Integrating Clinical Care and Research Coordination

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Background / Hypothesis:

Long-term follow-up is essential for patients with chronic liver disease, yet structured systems that support both patients and physicians in routine practice are not well described. We developed a physician-led multidisciplinary follow-up care bundle to improve coordination of patient management and clinical research activities. To describe the development and real-world implementation of a Physician–Patient Dual-Track Follow-Up Care Bundle in a specialized hepatology program.

Materials and Methods:

A physician-led team including physician assistants, nurses, and dietitians established a structured follow-up workflow within an infectious disease–based hepatology service. The care bundle included five core components: standardized follow-up coordination, patient navigation, physician-directed supportive care with dietitian-assisted nutritional assessment,

structured patient–physician communication support, and clinical trial screening coordination.

Results:

Since February 2025, the care bundle has supported follow-up of 785 patients with chronic liver disease over one year. The structured workflow helped centralize follow-up coordination and streamline communication between patients and clinicians. From January 2026 onward, the program assisted two patients in entering clinical trial screening. These experiences suggest that the bundle is feasible in routine practice and may help align clinical care with research activities.

Conclusions:

The Physician–Patient Dual-Track Follow-Up Care Bundle provides a practical, physician-led multidisciplinary approach to organizing long-term hepatology follow-up. This model may offer a structured way to support both patient care and research coordination in specialized clinical settings.

Keyword(s) : Physician–Patient Dual-Track Follow-Up Care Bundle

2851 - From Synthetic to Clinical The Effect of Domain Shift on Decision Support Systems

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Background / Hypothesis: Chronic liver diseases, especially cirrhosis and hepatocellular carcinoma, pose a severe clinical burden. Early risk stratification strictly dictates the treatment process. Artificial intelligence models trained on synthetic datasets often show high internal validity. However, their clinical translation is frequently hindered by domain shift due to heterogeneous real-world data. This proof-of-concept study evaluates the domain shift effect. It assesses

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hybrid decision support systems in a pilot clinical setting
to explore feasibility.

Materials and Methods: This feasibility study analyzed a dataset of 301 patients. The cohort included 284 synthetic cases and a critically small pilot sample of 17 clinically validated real-world cases. Classical machine learning models were trained on synthetic data. They underwent external testing on the real-world cohort without retraining to observe domain shift. Controlled data augmentation preserving distribution variance was applied due to the limited clinical sample. Probabilistic risk analyses from a MedGemma-based foundation model were compared with classical model outputs. Furthermore, a multimodal architecture combining MRI and laboratory data was evaluated.

Results: Classical models exhibited a significant performance drop during external testing. Accuracy fell to approximately 53% on the 17 real cases. This quantitatively demonstrated the domain shift effect. Controlled data augmentation successfully reduced these performance fluctuations. The foundation model showed 84.62% concordance with clinical evaluations in fibrosis prediction. In the multimodal benchmarking for hepatocellular carcinoma, the hybrid architecture produced consistent results in 14 of 17 cases (82.3%). The segmentation module achieved a 92% Dice score. The fibrosis staging module reached 93.13% accuracy. However, a 17–18% error rate persisted in morphologically atypical cases.

Conclusions: This pilot study demonstrates the critical impact of domain shift. Models trained solely on synthetic data struggle significantly in real-world clinical environments. High internal validation performance does not guarantee clinical readiness without real-patient testing. Hybrid benchmarking and multimodal integration offer a more balanced decision framework. However, algorithm capacity cannot substitute for actual data representativeness. Expanding to larger, multicenter datasets is strictly essential to achieve robust clinical integration.

Keyword(s) : Hepatocellular Carcinoma, Liver Fibrosis, Domain Shift, External Validation, Multimodal Artificial

Intelligence, Clinical Decision Support Systems, Medical Foundation Models, Benchmarking

2859 - Profile of Hepatocellular Carcinoma in India

A Five Year Prospective Study

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Background / Hypothesis: Hepatocellular carcinoma (HCC) represents the most common primary liver malignancy and is an emerging public health concern in India. Viral hepatitis was considered dominant cause of HCC. Prospective Indian data describing demographic, etiological, and staging characteristics remain limited which we aimed to describe in this study.

Materials and Methods:

This prospective observational study included consecutive patients diagnosed with HCC between January 2020 and December 2025. Demographic parameters, Comorbidities, underlying etiology, cirrhosis status, Child–Pugh class, Albumin-Bilirubin (ALBI) score, and Barcelona Clinic Liver Cancer (BCLC) stage were recorded. Continuous variables were expressed as median with interquartile range (IQR), and categorical variables as frequencies and percentages.

Results: A total of 1028 patient included. The median age at diagnosis was 60 years (IQR 52–67). A majority of patients were men (N=880, 85.6%). Among the 1028 HCC patients, 524 (51%) had diabetes, 403 (39.2%) had hypertension, and 83 (8.1%) had hypothyroidism. MASLD was the most common etiology, observed in 438 patients (42.6%), followed by hepatitis B virus infection in 258 (25.1%), hepatitis C virus infection in 113 (11%), ethanol-related liver disease in 60 (5.8%), cryptogenic liver disease in 145 (14.1%), and other causes in 14 (1.4%) patients. Underlying cirrhosis was present in 654 patients (63.6%), while 374 (36.4%) had non-cirrhotic HCC. Based on Child–Pugh classification, 280 patients (42.8%) were class A, 300 (45.9%) class B, and 74 (11.3%) class C. Among cirrhotic Child A patients, the median ALBI score was –2.33 (IQR –2.66 to –2.05) and in median ALBI score among Non Cirrhosis -2.28 (-2.75 - -2.01). According to BCLC staging, among the cirrhosis patients, 17 patients (2.6%) were stage 0, 195 (29.8%) stage A, 224(34.3%) stage B, 166 (25.4%) stage C, and 52 (8%) stage D.

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Conclusions: MASLD is now the most common cause of HCC in India, surpassing viral hepatitis. Every second individual had diabetes as a concomitant comorbidity. These findings highlight the rising role of metabolic risk factors and the need for better screening and early detection in high-risk populations.

Keyword(s) : HCC, MASLD, BCLC, CHILD PUGH, ALBI

2860 - Advances in Artificial Intelligence for Liver Disease Research: Algorithmic Applications and Technical Challenges

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Background / Hypothesis: Artificial intelligence (AI) has rapidly expanded in hepatology, offering novel solutions for data integration, imaging interpretation, and risk prediction. With the growing complexity of liver disease diagnostics, AI-driven algorithms provide opportunities to enhance accuracy and efficiency. This review summarizes recent algorithmic advances in AI applications for liver disease research and diagnostics.

Materials and Methods: A structured literature review was conducted using PubMed, Web of Science, and IEEE Xplore (2012–2024). Eligible studies focused on machine learning (ML), deep learning (DL), and natural language processing (NLP) models applied to liver disease datasets. Methods were categorized into imaging analysis (CNN-based models), clinical data modeling (logistic regression, random forest, XGBoost), and text mining approaches. Performance metrics including accuracy, AUC, sensitivity, and F1-score were comparatively evaluated.

Results: Deep learning models, particularly convolutional neural networks, consistently achieved high diagnostic performance in imaging-based liver disease classification (AUC commonly >0.90). Ensemble learning methods such as XGBoost demonstrated superior predictive stability compared to traditional statistical models. NLP-based systems improved automated

information extraction from electronic health records. However, variability in datasets and limited model interpretability remain significant challenges.

Conclusions:

AI technologies show strong potential in liver disease analytics and knowledge synthesis. Future development should emphasize explainable AI, standardized datasets, and cross-institutional validation to ensure reproducibility and clinical integration.

Keyword(s) : Artificial Intelligence; Machine Learning; Deep Learning; Hepatology; Natural Language Processing

2871 - Genetic Variants in Bile Transporter Genes in Prolonged Cholestatic Jaundice Associated with HAV and Drug Induced Liver Injury

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Background / Hypothesis:

Cholestatic jaundice results from impaired bile formation or secretion, leading to retention of bile acids and bilirubin. While many cases are attributable to identifiable causes such as Hepatitis A virus (HAV) infection and drug-induced liver injury (DILI), particularly due to complementary and alternative medicine (CAM) use, only a subset of patients develop prolonged cholestasis. This variability suggests an underlying genetic susceptibility. Genes involved in bile acid transport, including ABCB11, ABCB4, and ATP8B1, play a critical role in maintaining bile acid homeostasis. Variants in these genes may impair bile salt export pump function and phospholipid transport, predisposing individuals to persistent cholestasis following hepatic insults. We hypothesized that patients with prolonged cholestatic jaundice secondary to HAV infection or DILI may harbor polymorphisms in bile transporter genes that contribute to disease persistence.

Materials and Methods:

This observational study was conducted at the Asian Institute of Gastroenterology, Hyderabad. Adults (>18 years) with cholestatic jaundice persisting for more than 12 weeks were included. Patients with mechanical biliary

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obstruction were excluded. Peripheral blood samples were collected after informed consent. Genomic DNA was extracted and analyzed using next-generation sequencing targeting a PFIC gene panel comprising ABCB11, ABCB4, and ATP8B1. Clinical data including demographics, biochemical parameters, virological status, and history of drug or CAM intake were recorded and correlated with genetic findings.

Results:

Thirty-four patients were included. The mean age was 26.9 years, with a male predominance (82%). HAV-related hepatitis was identified in 60% of patients (20/34), among whom 7 patients (20% of total cohort) had concurrent CAM exposure. Drug-induced liver injury accounted for 40% (14/34), and 70% of these DILI cases (10/14) were attributed to CAM use. Genetic analysis revealed that 79.4% of patients (27/34) harbored at least one primary gene variant. The most frequent variant was p.Val444Ala (rs2287622) in the ABCB11 gene, detected in both homozygous and heterozygous states. Variants in ATP8B1 and ABCB4 were less common.

Conclusions: A substantial proportion of patients with prolonged cholestatic jaundice secondary to HAV infection and drug-induced liver injury, particularly CAM-related hepatitis, carried variants in bile transporter genes. Screening for these genetic variants may help identify individuals at risk of developing prolonged cholestasis following viral or drug-related hepatic injury.

Keyword(s) :

2874 - Dynamic evolution of the gut microbiome predicts therapeutic response in liver cancer A large scale longitudinal study with a cross cohort meta analysis

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Background / Hypothesis: The gut microbiota modulations influence therapeutic efficacy in liver cancer, yet high-resolution longitudinal evidence remains limited. We hypothesized that specific, dynamically evolving microbial signatures could serve as

robust predictors of treatment response across different clinical settings.

Materials and Methods: A total of 315 consecutive patients with liver cancer (HCC 88%, ICC 12%; HBV-dominant, 64%) receiving systemic therapies (targeted therapy and immunotherapy) were prospectively enrolled. Patients were categorized into Responders (n=175) and Non-responders (n=140). Fecal metagenomes and clinical data were analyzed at baseline (T0) and multiple treatment timepoints (T1, T2). Differential taxa at each timepoint were identified using LEfSe analysis ($P < 0.05$, absolute LDA score > 1.5). To ensure robustness, a timepoint trend meta-analysis was performed by integrating our data with multiple public HCC microbiome databases to identify consensus response-associated taxa.

Results: Our cross-cohort meta-analysis independently validated that specific butyrate-producers, including *Anaerobutyricum hallii* (Beta: 1.45), *Eubacterium rectale* (Beta: 1.25), and *Faecalibacterium SGB15346* (Beta: 1.08), were universally enriched in responders across different cohorts and timepoints. Longitudinally, non-responders demonstrated a rapid and progressive loss of these beneficial taxa, accompanied by the expansion of opportunistic species such as *Escherichia coli* and *Enterococcus faecium*. Spearman correlation analysis revealed that the abundance of these validated beneficial species was significantly positively associated with liver function markers, specifically albumin, prealbumin, and choline esterase, while exhibiting strong negative correlations with alpha-fetoprotein, C-reactive protein, and Child-Pugh Score. Notably, the dynamic decline of meta-analysis-validated taxa provided an early warning signal of resistance that preceded clinical and radiological progression.

Conclusions: By integrating a large-scale longitudinal HCC cohort with a multi-database meta-analysis, we identified a robust microbial signature centered on butyrate-producers that predicts therapeutic response. These findings highlight the potential of using cross-cohort validated microbial markers for early clinical monitoring and personalized therapeutic intervention in liver cancer.

Keyword(s) : Liver cancer, Gut microbiome, Meta-analysis, Longitudinal cohort, Therapeutic resistance, Butyrate-producing bacteria

2876 - Immune Checkpoint Inhibitor-Induced Hepatitis in HCC Poor Response to Immunosuppression.

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Background / Hypothesis: Immune checkpoint inhibitors (ICIs) are increasingly used in India for the treatment of advanced hepatocellular carcinoma (HCC). Immune Checkpoint Inhibitor-Induced Hepatitis is a recognized, though uncommon, immune-related adverse event and can result in significant morbidity and in severe cases mortality.

Materials and Methods: We report four patients who developed ICI-induced hepatitis at our center.

Results: Three were males (75%) and one female (25%), with a mean age of 62 years (range 60–66). Etiologies included NASH in three patients (75%) and HBV in one (25%). Most patients had advanced HCC (BCLC stage C in 75%). ANA was positive in three patients (75%), with one showing markedly elevated IgG (4650 mg/dL). Mean total bilirubin was 6.7 mg/dL, direct bilirubin 3.8 mg/dL, SGOT 394 U/L, SGPT 294 U/L, total protein 7.5 g/dL, and albumin 3.2 g/dL. All patients received immunosuppression with steroids $\pm$ mycophenolate; however, two patients (50%) were non-responders, one patient (25%) responded, and one patient (25%) died due to complications.

Conclusions: ICI-induced hepatitis in HCC can lead to poor outcomes, with a significant proportion showing inadequate response to immunosuppressive therapy. Further studies are needed to identify predictors of Immune Checkpoint Inhibitor-Induced hepatitis and optimize monitoring strategies during immunotherapy.

Keyword(s): ICI-induced hepatitis

2877 - The Effect of Portal Vein Tumor Trombosis on Survival in Patients with Hepatocellular Carcinoma Treated with Different Therapies

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Background / Hypothesis: Portal vein tumor thrombosis (PVTT) is common among patients with advanced hepatocellular carcinoma (HCC) and its presence is associated with dismal prognosis with median overall survival ranging between 2 and 6 months, and 3-years survival of around 5% in untreated patients. Although international guidelines recommend systemic treatments as the standard of care, a tremendous differences exist between East and West in the management of these patients. In clinical practice many patients receive various treatments, including hepatic resection, locoregional treatments, and even liver transplantation. Here we investigated the impacts of PVTT scope and the treatment modalities on patient survival.

Materials and Methods: This study included 954 patients with HCC and PVTT between October 2015 and January 2024. PVTT was classified according to Cheng's classification as follows: Type I, tumor thrombus in the segmental branches of the PV or above; type II, thrombus extending to the right or the left PV; type III, thrombus extending to main PV; type IV, thrombus involving the superior mesenteric vein. Median overall survival was analyzed according to both Cheng's classification and treatment modalities using Kaplan-Meier survival test.

Results:

The mean age was $62,54 \pm 11,95$ years, and the median follow-up period was $10,05 \pm 17,22$ months. The median

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OS according to treatment approaches was as follows: resection $12,10 \pm 12,10$ months, transplantation $38,90 \pm 41,51$ months, RF ablation $31,53 \pm 25,02$ months, sorafenib $6,78 \pm 6,84$ months, TACE $11,79 \pm 10,60$ months, TARE $8,09 \pm 5,16$ months, immunotherapy $11,75 \pm 8,44$ months, others $5,03 \pm 5,88$ months. Median OS was significantly longer in transplantation, and ablation groups compared to other treatment groups in patients with segmental PVTT ($p < 0.001$).

Conclusions:

Treatment modalities for HCC with PVTT should not be limited to systemic therapy. The extend of PVTT should be considered when deciding on treatment alternatives. In patients with segmental PVTT, surgical techniques should be evaluated.

Keyword(s) : Hepatocellular carcinoma; Portal vein tumor thrombosis; Cheng's classification; Median overall survival, Locoregional therapies

2878 - From Stool to Personalized Screening Approach A Comprehensive Meta analysis of Metagenomic Gut Microbiota Assessment in Patient with MAFLD and Control Cohort

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Background / **Hypothesis:**
The global prevalence of metabolic dysfunction-associated fatty liver disease (MAFLD) is increasing, yet no effective personalized screening approach exists to prevent its progression. Emerging evidence regarding the gut-liver axis indicates a correlation between gut dysbiosis and MAFLD, highlighting the potential for microbiome-based screening. This study evaluates genus-specific gut microbiome abundance in individuals with MAFLD compared to controls to assess their potential utilization for MAFLD screening and diagnosis.

Materials and **Methods:**
Metagenomic projects were identified from the NCBI SRA database using the keywords ("MAFLD" OR "Fatty Liver" OR "NAFLD") AND ("Gut Microbiome" OR "stool" OR "microbiota"). Inclusion criteria comprised human cohort studies, MAFLD-diagnosed samples, and 16S

rRNA analysis targeting the V3 or V4 region. Selected projects underwent preprocessing with the QIIME2 pipeline, followed by taxonomic identification using the SILVA database. Outcomes included community-level differences assessed by alpha- and beta-diversity analyses, study-level genus performance evaluated by random forest area under the receiver operating characteristic curve (AUROC), and genus-level meta-analysis. Metadata of interest included disease status and sample geographic region (Asia or Europe).

Results:

Six metagenomic projects, comprising 297 MAFLD patients and 133 control samples, were included in the analysis. The MAFLD group exhibited lower Shannon diversity compared to controls (SMD: -0.29; 95% CI: -0.6 to 0.17, $p = 0.03$). Bray-Curtis PERMANOVA showed that MAFLD accounted for at most 11% of the variation in gut community composition within a single cohort ($R^2 = 0.11$, $p < 0.05$). Further PCoA analysis showed stronger clustering by geographic region than by disease status. Genus-level microbiome profiles effectively discriminated MAFLD from controls (AUROC 0.85–1) in five cohorts. Genus-level meta-analysis identified several genera as more abundant in the MAFLD population, including *Hartmannibacter* sp., *Streptomyces* sp. and *Sphingosinithalassobacter* sp. in Asia region, while *Oribacterium* sp. is found in Europe region. On the other hand, *Papilibacter* sp., *Merdibacter* sp., and *Peptococcus* sp. were depleted in both Asia and Europe.

Conclusions:

This meta-analysis supports the potential use of gut microbiota analysis for personalized MAFLD screening. However, due to the absence of a large, consistent global change in diversity and the microbiome's genus profile, further studies are required. Future investigations that consider MAFLD severity, sex, country, age, and comorbidities are recommended to enable a more personalized approach.

Keyword(s) : MAFLD, gut microbiome, gut-liver axis, metagenomic, dysbiosis

2879 - AARC score predicts survival in patients with Acute on Chronic Liver Failure due to Severe Alcoholic Hepatitis undergoing Plasma Exchange

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Background / Hypothesis: Plasma exchange has emerged as a therapeutic treatment modality in individuals with acute-on-chronic liver failure (ACLF) due to severe alcoholic hepatitis (SAH) who are ineligible for corticosteroid therapy and are not candidates for liver transplantation. The aim of this study was to identify predictors of non-survival in these individuals.

Materials and Methods: It a retrospective study in which the electronic medical records of the patients admitted with acute on chronic liver failure due to severe alcoholic hepatitis and had undergone plasma exchange was analysed. Individuals receiving steroids or eligible for liver transplantation were excluded. The study period was between January 2015 to April 2025.

Results: There was total 126 patients. The mean age was 40.77 ± 6.9 years and 38.7 ± 6.9 years in the non-survivor and survivor group. The mDF (Maddrey's Discriminant Function) was higher in the non-survivor group 92.2 (73-113.2) as compared to the survivors 90.5 (72-112.5), $p:0.83$, and was not statistically significant. The number of Plasma Exchange was similar between both the groups, 3 ± 1.8 and 3.2 ± 1.5 , $p:0.45$ respectively. Mortality was seen in 31 (24.6%) individuals. Baseline Platelet count 122.7 ± 70.7 and 158.01 ± 93.6 , $p:0.03$ and AARC score was 10.7 ± 1.6 and 9.2 ± 1.4 , $p:0.01$ respectively in the non-survivor and survivor group and were the statistically significant predictor of mortality. Serum bile acid was statistically not significant between the two groups. On multivariate analysis AARC score with HR of 2.241, CI:1.316-3.816, $p:0.003$ was predictor of mortality.

Conclusions: Plasma exchange is a therapeutic treatment modality in patients with ACLF due to severe alcoholic hepatitis. A higher baseline AARC score independently predicts mortality in these patients and may guide in risk stratification and management decisions.

Keyword(s) : ACLF - Acute on chronic liver failure, PLEX - Plasma exchange, SAH - Severe alcoholic hepatitis.

2881 - Impact of an AI Supported Triage System on the Diagnostic Workflow of Focal Pancreatic Lesions A Real World Clinical Utility Analysis

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Background / Hypothesis: Accurate and early differentiation of focal pancreatic solid lesions (FPSLs) in the outpatient setting remains a significant clinical challenge. Benign inflammatory conditions, such as focal autoimmune pancreatitis (fAIP) and mass-forming chronic pancreatitis (MFCP), often present similarly to pancreatic ductal adenocarcinoma (PDAC) in terms of clinical features and conventional imaging findings, leading to diagnostic uncertainty and potential unnecessary pancreaticoduodenectomy. Current serum biomarkers lack accuracy, and invasive diagnostic procedures are limited by sampling variability, highlighting the need for a reliable, non-invasive triage tool suitable for outpatient care. Venous phase contrast enhanced computed tomography (CECT) effectively captures pancreatic parenchymal and lesional enhancement patterns, and radiomics derived from this phase can quantify subtle, visually imperceptible differences in enhancement homogeneity, tissue heterogeneity, and periductal parenchymal remodelling.

Materials and Methods: we first summarized the current evidence, including serological markers, imaging findings, histopathology, novel biomarkers, and liquid biopsy techniques, for differentiating benign lesions (particularly fAIP) from PDAC. We then designed a retrospective study, involving 307 FPSLs outpatients (51 MFCP, 67 fAIP, and 189 PDAC cases) who underwent venous-phase CECT. The cohort was split into a training set and a validation set at a 7:3 ratio. Whole-pancreas segmentation was performed automatically, and radiomics features were extracted from CECT images. Clinical variables, including demographics and laboratory indices, were also collected. After multi-step feature selection, six machine learning classifiers, including logistic regression, support vector machine, random forest, XGBoost, k-nearest neighbors, and Extra Trees, were developed and evaluated in both training and validation sets. Calibration curves and decision curve analysis were applied to assess potential clinical utility. The SHAP method was applied to the best-performing model to assess feature importance and enhance interpretability. Furthermore, we proposed a practical three-stage diagnostic algorithm based on our data as well as the present guidelines.

Results:

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The final model included five clinical variables and six wavelet texture features. In the validation cohort, the Extra Trees model achieved the best performance, with an AUC of 0.840. The sensitivity was 0.825 and the specificity was 0.694. SHAP analyses revealed that CA19-9 was the most significant contributor to differentiation, while wavelet texture features and age were also important variables.

Conclusion:

A multimodal machine learning model combining radiomics with clinical variables exhibited robust diagnostic performance in distinguishing between benign and malignant FPSLs. Based on our stepwise workflow, the model, with its high sensitivity and moderate specificity, is well-suited for risk stratification in equivocal FPSL cases, offering valuable insights prior to further invasive evaluations.

Keyword(s) : focal autoimmune pancreatitis; pancreatic ductal adenocarcinoma; Artificial intelligence ; machine learning models; Focal Pancreatic Lesions

2886 - Comparative Analysis of Epidemiological Research Output Metabolic Dysfunction Associated Steatotic Liver Disease vs. Alcohol Associated Liver Disease

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) and alcohol-associated liver disease (ALD) are leading causes of chronic liver disease. Comparative analysis of their epidemiological research can reveal knowledge gaps and guide resource allocation. We conducted a comprehensive bibliometric comparison of epidemiological research on MASLD and ALD, published between 2005-2026.

Materials and Methods: Epidemiological studies on MASLD and ALD were systematically identified and reviewed in PubMed/MEDLINE and Google Scholar to identify relevant studies. Search strategies employed combinations of Medical Subject Headings (MeSH) terms and keywords related to MASLD, ALD, and study design terms. Studies were included if they were original epidemiological research, human studies, focused on

prevalence, incidence, risk factors, mortality, or disease burden, published in peer-reviewed journals, and published in English language. Extracted variables included publication year, geographical origin, study design, study population, World Bank income classification, WHO region, journal quality (SCImago quartile), data source type, citation metrics, and declared funding. Statistical analyses included chi-square tests, independent samples t-tests, and compound annual growth rate (CAGR) calculations.

Results:

A total of 324 studies were included: 292-MASLD, 32-ALD, and 9-classified under both. MASLD research volume substantially exceeded ALD (292 vs. 32); however, growth rates were not significantly different between fields ($p=0.35$), reflecting high variance in both series. Significant geographical disparities were observed: MASLD research was predominantly region-specific, whereas ALD studies more frequently spanned multiple regions (58% of multi-region studies were ALD; $\chi^2=35.93$, $p<0.001$). ALD research was more concentrated in high-income countries (72% vs. 59%), with no ALD studies from low-income countries; the USA contributed disproportionately to ALD output (31% vs. 17% for MASLD), while China dominated MASLD (16% vs. 3%). ALD papers were more likely to have declared funding (31% vs. 16%; $p=0.04$) and to be published in Q1 journals ($\chi^2=6.67$, $p=0.083$). ALD studies also made greater use of secondary data and the Global Burden of Disease database, whereas MASLD studies more commonly used primary data ($\chi^2=29.98$, $p<0.001$). Raw citation counts were higher for MASLD (mean 27.73 vs. 11.27; $p=0.006$), but this difference was not significant after adjustment for publication year ($p=0.28$).

Conclusions:

Despite greater overall volume, MASLD research exhibits significant geographical and methodological gaps, particularly in low- and middle-income countries. ALD research, while more limited in quantity, demonstrates broader international scope, stronger funding support, and higher publication in top-tier journals. Both conditions are markedly underrepresented in low-income settings. These findings underscore the need for targeted research investment in under-resourced regions where disease burden may be highest.

Keyword(s) : MASLD; NAFLD; alcohol-associated liver

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disease; ALD; bibliometric analysis; epidemiological research; health research priorities; knowledge gaps

2888 - Evaluation of selected Plasma Long Non Coding RNAs as predictive Biomarkers in HBV Related Hepatocellular Carcinoma

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Background:

Long non-coding RNAs (lncRNAs) have come to light as promising biomarkers for the early detection of hepatocellular carcinoma (HCC). This study focused on estimating the plasma levels of selected lncRNAs—highly upregulated in liver cancer (HULC), metastasis-associated lung adenocarcinoma transcript 1 (MALAT1), long intergenic non-coding RNA 152 (LINC00152), and urothelial carcinoma-associated 1 (UCA1)—in patients with hepatitis B virus (HBV)-related chronic liver diseases and HCC in Bangladesh.

Materials and Methods:

In this cross sectional study, diagnosed cases of HBV-infected patients with chronic hepatitis, cirrhosis, and hepatocellular carcinoma were included. Total 160 participants were enrolled: 40 with chronic hepatitis, 40 with cirrhosis, 40 with HCC, and 40 healthy controls. Plasma RNA was isolated, reverse-transcribed into complementary DNA (cDNA), and expression of four lncRNAs—HULC, MALAT1, LINC00152, and UCA1—were quantified using GoTaq qPCR Master Mix (Promega, USA) on a QuantStudio 5 Real-Time PCR System (Applied Biosystems, USA). Results of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alpha-fetoprotein (AFP), HBV DNA, HBsAg, viral load, and other biochemical parameters were obtained from prior laboratory reports conducted within one month of enrollment.

Results

In this study, in patients with HCC, serum ALT and AST levels were significantly higher ($p < 0.001$). HBV DNA viral load ($\log_{10}$ copies/mL) was highest in chronic hepatitis patients, followed by HCC and cirrhosis ($p < 0.001$). Alpha-fetoprotein (AFP) levels were markedly elevated in HCC patients compared with the other groups ($p < 0.001$). The expression of HULC, MALAT1, LINC00152, and UCA1 was markedly elevated in patients with HCC compared to those with cirrhosis, chronic hepatitis, and healthy controls. Although increased expression of these lncRNAs was also observed in cirrhosis and chronic hepatitis groups, but the differences were not statistically significant. Receiver

operating characteristic (ROC) analysis indicated that all four lncRNAs achieved area-under-the-curve (AUC) values above 0.8 in discriminating HCC from healthy individuals. HULC exhibited the highest diagnostic accuracy (AUC = 0.910, sensitivity = 92.5%, specificity = 85.0%). The other lncRNAs (MALAT1, LINC00152, and UCA1) also showed sensitivity of 95.0%, 92.5%, and 90.0% and specificity of 80.0%, 85.0%, 77.5% respectively. Compared with alpha-fetoprotein (AFP), HULC and MALAT1 demonstrated superior diagnostic accuracy in distinguishing HCC from cirrhosis.

Conclusions:

Plasma HULC, MALAT1, LINC00152, and UCA1 are potential noninvasive biomarkers for HBV-related detection of HCC. The inclusion of lncRNA profiling within established diagnostic frameworks, combined with AFP and other biomarkers, has the potential to strengthen early diagnosis and monitoring strategies.

Keyword(s) : Hepatocellular Carcinoma; Chronic Hepatitis B; Cirrhosis; Long Non-Coding RNA; HULC; MALAT1; LINC00152; UCA1; Biomarkers

2894 - Splenic probe (100 KPa) is superior to liver probe (75 KPa) in assessment of liver stiffness and predicting outcomes in Acute on chronic liver failure patients

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Background

Early identification of acute on chronic liver failure (ACLF) patients with a high risk of mortality is important to prioritize the need for ICU monitoring and liver transplantation (LT). In ACLF, inflammation and an acute rise in portal pressure drive the outcome and can raise liver (LSM) and spleen stiffness (SSM). Liver stiffness measurement (LSM) using the liver probe of transient elastography is increasingly explored for prognostication in ACLF. However, the conventional liver probe(LP) has a ceiling effect with an upper limit of 75 kPa only. The splenic probe(SP) with an upper limit

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Hypothesis:

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of 100 kPa may overcome this limitation. We prospectively evaluated the prognostic performance of liver stiffness measured using the splenic probe versus the conventional liver probe using the Fibro-scan 630 expert model at baseline and during early follow-up in ACLF patients

Materials and Methods: Patients of ACLF between 18 to 70 years of age were included. The study aimed to compare the prognostic utility of the acute change in LSM measured using SP versus LP on days 7 and 14 for predicting transplant-free survival at day 90 in patients with ACLF.

Results: Study included 56 patients (mean age 40.9 ± 8.6 yrs, males 88.9%, alcohol associated 83.76 %) with ACLF. At day-90, 38 (68.4%) patients had LT free survival, with 15 (26.78%) deaths and 3 (1.68%) LT. Mean baseline AARC score among survivors and non-survivors was 7.68 ± 1.37 and 10.14 ± 1.25 , respectively ($p < 0.001$). Baseline LSM measured through SP (82.67 ± 17.7 kPa) was significantly higher than LP (70.95 ± 9.13 kPa) among survivors ($p = 0.005$) and non-survivors (SP = 90.96 ± 12.79 vs LP = 73.56 ± 9.13 kPa, $p < 0.001$). On day 7, mean change in LSM by SP (-7.09 ± 3.48) was significantly higher than LP (-0.69 ± 0.63) among survivors ($p < 0.001$) and non-survivors (SP = -2.46 ± 1.29 vs. LP = -0.11 ± 0.3 kPa) ($p < 0.001$). Similarly, on day 14, the mean change in LSM by SP (-13.14 ± 7.21 kPa) was significantly higher than LP (-1.83 ± 0.95) among survivors ($p < 0.001$) and non-survivors (SP = -5.89 ± 1.2 vs. LP = -0.08 ± 0.12 , $p < 0.001$).

Conclusions: LSM measured using a splenic probe provides a larger range of stiffness measurement, required for assessing inflammation inherent in ACLF patients. The splenic probe was found to be superior to the conventional probe in stratifying high-risk ACLF patients. Serial assessment of LSM using the splenic probe during the first two weeks reliably segregates ACLF patients at high risk of short term mortality.

Keywords: Transient elastography, Liver stiffness, Liver probe, Splenic probe, Prognosis, ACLF

Keyword(s):

2897 - Integrative Metabolomic and Cytokine Profiling Reveals a "Metabolic Inflammatory" Signature in Chronic Hepatitis B Combined with Metabolic Dysfunction Associated Fatty Liver Disease

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Background / Hypothesis: The coexistence of chronic hepatitis B (CHB) and metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly common and worsens liver outcomes. Noninvasive biomarkers that distinguish CHB-MAFLD from CHB alone are lacking. We aimed to identify integrated cytokine and metabolomic signatures that differentiate CHB-MAFLD from CHB.

Materials and Methods: In this cross-sectional study, 406 participants were enrolled: healthy controls (HC, n=30), CHB alone (CHB, n=149), and CHB with MAFLD (CHB-MAFLD, n=227). CHB was defined by HBsAg positivity >6 months with detectable HBV DNA; MAFLD was diagnosed by imaging proven steatosis plus metabolic dysfunction. Exclusion criteria included other viral hepatitis, malignancy, significant organ dysfunction, systemic disease, and pregnancy. Demographics, clinical labs, serum cytokines (IL-17, TNF- α ; Luminex), and non-targeted serum metabolomics (LC-MS/MS) were compared across groups. Differential metabolites were identified and subjected to KEGG pathway enrichment. **Results:** Compared with CHB alone, CHB-MAFLD patients had higher ALT, AST, GGT, BMI, and uric acid ($P < 0.05$). Serum IL-17 and TNF- α were significantly elevated in CHB-MAFLD versus CHB alone ($P < 0.05$). Metabolomic profiling identified 110 differential metabolites between CHB-MAFLD and CHB groups; the top altered metabolites were Ethyl (S)-3-hydroxybutyrate glucoside, Indole-3-propionic acid, and Hippuric acid. Branched-chain amino acid L-Leucine and L-Tyrosine were decreased in CHB-MAFLD. KEGG enrichment highlighted perturbations in ketone body metabolism, tryptophan metabolism, branched-chain amino acid metabolism, and purine metabolism.

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Conclusions: CHB-MAFLD is characterized by a distinct “metabolic-inflammatory” signature reflecting mitochondrial energy reprogramming, gut microbiota related metabolite shifts, and proinflammatory cytokine activation. Serum IL-17, TNF- α , Ethyl (S)-3-hydroxybutyrate glucoside, Indole-3-propionic acid, and L-Leucine warrant further validation as noninvasive biomarkers to distinguish CHB-MAFLD from CHB alone and to guide precision management.

Keyword(s) : chronic hepatitis B; metabolic dysfunction-associated fatty liver disease; metabolomics; cytokines; biomarkers; metabolic-inflammatory signature

2904 - Clinical Analysis of Liver Transplantation in Patients With Severe Liver Disease

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Background / Hypothesis:
To evaluate the clinical efficacy of liver transplantation in patients with severe liver disease, analyze the risk factors for short-term postoperative mortality, and assess the value of modified piggyback liver transplantation and preoperative and intraoperative continuous renal replacement therapy (CRRT) in improving patient outcomes.

Materials and Methods:
A retrospective analysis was conducted on adult patients who underwent liver transplantation at our center between July 2013 and December 2025. A total of 1,534 patients were included, comprising 143 patients with severe liver disease and 1,391 with non-severe liver disease. Clinical characteristics and postoperative outcomes were compared between the two groups. Logistic regression analysis was performed to identify risk factors for death within 90 days after transplantation in patients with severe liver disease.

Results:
Compared with patients with non-severe liver disease, those with severe liver disease were younger and had a higher proportion of alcohol-related chronic liver disease. They experienced greater intraoperative blood loss, required larger volumes of plasma transfusion, had longer ICU stays, higher rates of early postoperative complications, and lower survival rates within 25 days

(93.01%) and 90 days (91.61%) after transplantation ($P < 0.05$). However, there were no significant differences in 1-year and 3-year patient survival rates or graft survival rates between the two groups. Risk factors for death within 90 days after transplantation in patients with severe liver disease included older age (OR 1.097), longer operative time (OR 1.369), greater intraoperative blood loss per 100 mL (OR 1.053), and uncontrolled postoperative infection (OR > 1.965). Compared with the conventional surgical technique, the modified surgical approach significantly reduced postoperative ICU stay ($P < 0.05$). Patients receiving a three-dose basiliximab induction regimen showed a significantly higher rate of effective infection control compared with those who did not receive the three-dose regimen ($P < 0.05$). Patients who received CRRT preoperatively and intraoperatively had a significantly shorter ICU stay compared with those who did not receive CRRT ($P < 0.05$), while infection control rates were not significantly different between the two groups.

Conclusion:

Liver transplantation is an effective treatment for patients with severe liver disease, providing long-term survival benefits comparable to those of non-severe patients. Risk factors for postoperative mortality include advanced age, excessive intraoperative blood loss, prolonged operative time, and uncontrolled postoperative infection. The use of a staged three-dose basiliximab induction regimen, modified piggyback liver transplantation with optimized surgical techniques, and CRRT for patients with preoperative infection can effectively improve postoperative outcomes.

Keyword(s) : Severe liver disease; liver transplantation; clinical efficacy

2905 - A clinical study on the reduction of donor lymphocyte chimerism rate in recipient tissues after liver transplantation by extracorporeal perfusion of anti thymocyte globulin via portal vein

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Background / Hypothesis:

This study aimed to determine whether ex vivo perfusion of donor livers with antithymocyte globulin (ATG) could reduce donor lymphocyte chimerism in recipients after liver transplantation, and to evaluate the safety of this approach.

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Materials and Methods:

A retrospective cohort of 35 liver transplant recipients who underwent ex vivo ATG perfusion of the donor liver via the portal vein was included. A matched control group was generated using 1:1 propensity score matching. Postoperative liver and renal function parameters, as well as infectious events, were compared between the two groups. In addition, lymphocyte subsets (CD3⁺, CD4⁺, and CD8⁺) in donor liver tissue were assessed, and donor-derived CD8⁺ T-cell chimerism in recipients' peripheral blood was quantified. Statistical comparisons were performed using the Mann–Whitney U test.

Results:

Postoperative liver and renal function, as well as the incidence of infectious complications, were comparable between the ATG-treated group and the matched control group. In contrast, ex vivo ATG perfusion was

associated with a pronounced reduction in CD3⁺, CD4⁺, and CD8⁺ lymphocyte populations within donor liver tissue. Correspondingly, donor-derived CD8⁺ T-cell chimerism in the peripheral blood of recipients was significantly lower in the ATG-treated group.

Conclusions:

In this study, ex vivo ATG treatment of donor livers was not associated with an increased risk of postoperative complications. At the same time, it effectively suppressed donor lymphocyte activity. These findings indicate that ATG perfusion may serve as a feasible and clinically meaningful strategy for the prevention of graft-versus-host disease (GVHD) following liver transplantation.

Keyword(s) : Ex vivo perfusion; antithymocyte globulin; donor lymphocyte chimerism



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1002 - Assessing the Utility of Serum Kallistatin in the Diagnosis of Liver Cirrhosis A Meta analysis

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Background / Hypothesis: Kallistatin, which protects organs and cells against inflammation, fibrosis and oxidative stress, is mainly synthesized and secreted in liver. As it is a negative acute-phase protein, its blood level decreases with a decline in liver function. Although liver biopsy is the gold standard for the assessment of liver fibrosis in cirrhosis, it has its own limitations. Therefore, noninvasive methods to detect liver fibrosis are widely preferred. However, they also have their own limitations. Thus, there is always a need to extend the battery of serum-based assays. Meta-analysis of clinical data on serum kallistatin level in patients with liver cirrhosis and its potential association with the pathophysiology of is lacking. Thus, this meta-analysis evaluates the levels of serum kallistatin as a diagnostic marker in liver cirrhosis.

Materials and Methods: From the inception to May 2025, the PubMed, Google Scholar, and Embase were searched for studies that assessed serum kallistatin in liver cirrhosis patients and matched healthy controls. Results comprised the standardized mean difference (SMD) in the kallistatin level in the serum of the two groups with a 95% Confidence Interval (CI). According to the heterogeneity, the serum kallistatin values were analyzed using the Revman 5.1 program using either a fixed or random effect.

Results: In this meta-analysis, five studies were included. 370 had cirrhosis and 159 were healthy controls. Participants were adults. The analysis revealed a significantly lower serum kallistatin in liver cirrhosis patients as compared to healthy controls (SMD: -3.30, CI: -5.34 to -1.26, I²=98%, P value 0.002). Furthermore, the serum kallistatin level was even lower in the patients with hepatocellular carcinoma as compared to the liver cirrhosis and healthy controls groups.

Conclusions: The meta-analysis revealed significantly decreased serum kallistatin level in liver cirrhosis

patients. Therefore, further studies to investigate the role of the kallistatin may be valuable for the development of future diagnostics or therapeutics of liver cirrhosis. The low levels of serum kallistatin in cirrhosis can be considered as a diagnostic marker for liver cirrhosis and liver cancer. Incorporating serum kallistatin screening into routine check-ups for patients with hepatitis, non-alcoholic fatty liver, and autoimmune diseases could aid in the early detection and prevention of cirrhosis-associated complications.

Keyword(s) : Kallistatin, Liver cirrhosis

1003 - Is drug induced autoimmune hepatitis being underdiagnosed

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Background: Drug-induced autoimmune hepatitis (DIAIH) is a rare but important clinical entity that mimics idiopathic autoimmune hepatitis (AIH) both clinically and histologically. It is triggered by certain medications in genetically predisposed individuals and poses diagnostic and therapeutic challenges due to its overlapping features with classical AIH and drug-induced liver injury (DILI). At present, there are still few reports on this condition both globally and in Vietnam. **Introduction:**

DIAIH represents a subset of liver injury that shares features with idiopathic AIH but is precipitated by drug exposure. Common causative agents include minocycline, nitrofurantoin, hydralazine, and statins. The exact mechanisms underlying DIAIH remain unclear. It is hypothesized that certain drugs act as haptens or modify self-antigens, triggering a loss of immune tolerance in susceptible individuals

Clinical and Histological Features:

Patients with DIAIH often present with non-specific symptoms. Histologically, DIAIH is indistinguishable from idiopathic AIH, showing interface hepatitis, plasma cell infiltration, and sometimes rosetting of hepatocytes.

Diagnosis:

The diagnosis of DIAIH is based on a combination of clinical history, laboratory findings, and liver biopsy. Key elements include the temporal relationship between drug exposure and onset of hepatitis, presence of autoantibodies (ANA, ASMA), elevated IgG, and histological findings.

Management:

Initial management involves prompt discontinuation of the offending drug. Corticosteroid therapy may be

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required in more severe cases or when there is no improvement after drug withdrawal. The long-term need for immunosuppressive therapy is less common than in idiopathic AIH.

Keyword(s) : Keywords: drug - induced autoimmune hepatitis hepatitis, liver disease,

1004 - An acute flare of chronic hepatitis B in a patient with eosinophilic ascites and diabetic ketoacidosis represents a clinical challenge

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Background / Hypothesis: Eosinophilic ascites is an uncommon clinical presentation, most frequently associated with eosinophilic gastroenteritis or parasitic infections. Diagnosis requires careful exclusion of other causes of ascites, especially in patients with underlying chronic liver disease. The treatment of an acute flare of chronic hepatitis B is challenging, particularly in patients with eosinophilic ascites and diabetic ketoacidosis.

Objective: We report a case of eosinophilic ascites in a young man with chronic hepatitis B, newly diagnosed diabetic ketoacidosis, and positive serology for Toxocara exposure, illustrating the diagnostic complexity and successful therapeutic approach.

Materials and Methods: clinical case report and literature review

Results: Corticosteroids remain the cornerstone of treatment for eosinophilic gastroenteritis and associated eosinophilic ascites. In patients with chronic hepatitis B, immunosuppressive therapy poses a risk of HBV reactivation, warranting concurrent antiviral prophylaxis. High-dose corticosteroid therapy in poorly controlled diabetes mellitus cases is also a precipitating factor for recurrent diabetic ketoacidosis. Our patient responded favorably to combined corticosteroid and antiviral therapy, with complete resolution of symptoms and normalization of eosinophil counts.

Conclusion: This case highlights the diagnostic challenges of eosinophilic ascites in a patient with overlapping chronic hepatitis B, diabetic ketoacidosis, and parasitic exposure. Early recognition and appropriate management can lead to favorable outcomes.

Materials and Methods:

Results:

Conclusions:

Keyword(s) : Keywords: Eosinophilic ascites, Eosinophilic gastroenteritis, Chronic hepatitis B, Diabetic ketoacidosis, Parasitic infection

1005 - Drug induced autoimmune hepatitis three case reports.

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Background: Drug-induced autoimmune hepatitis (DIAIH) is a rare but important clinical entity that mimics idiopathic autoimmune hepatitis (AIH) both clinically and histologically. It is triggered by certain medications in genetically predisposed individuals and poses diagnostic and therapeutic challenges due to its overlapping features with classical AIH and drug-induced liver injury (DILI). At present, there are still few reports on this condition both globally and in Vietnam.

Objective: We report three cases of drug induced autoimmune hepatitis diagnosed at Tam Anh General Hospital, Ho Chi Minh city.

Materials and Methods: clinical case reports and literature review

Results: Three patients were diagnosed with drug induced autoimmune hepatitis and their conditions are stable during follow up period

Conclusion: DIAIH should be considered in patients with compatible clinical features

and a history of recent drug exposure. Early recognition, withdrawal of the offending agent, and timely immunosuppressive therapy when needed are key to favorable outcomes. Given the limited number of reported cases worldwide and particularly in Vietnam, further research is essential to enhance understanding of its pathogenesis, improve diagnostic criteria, and guide therapeutic strategies

round / Hypothesis:



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Materials and Methods:

Results:

Conclusions:

Keyword(s) : Keywords: drug - induced autoimmune hepatitis hepatitis, liver disease

1008 - Efficacy and Safety of Terlipressin versus Octreotide for esophageal variceal bleeding A retrospective study at a tertiary hospital in the UAE

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Background / Hypothesis:

Esophageal variceal bleeding (EVB) is a severe complication of cirrhosis with significant morbidity and mortality. Pharmacologic therapy, including terlipressin and octreotide, is commonly used alongside endoscopic variceal ligation (EVL) to control bleeding. However, the comparative effectiveness of these agents remains uncertain. This study evaluates the efficacy and safety of terlipressin versus octreotide in patients with EVB in a real-world clinical setting

Materials and Methods:

We conducted a retrospective cohort study at a tertiary hospital in the UAE, analyzing patients with EVB who received either terlipressin or octreotide as adjuvant therapy to EVL. Patient data were extracted from electronic health records, and outcomes included in-hospital mortality, 30-day mortality, and the need for repeat endoscopy within five days.

Results:

A total of 275 patients were included (terlipressin: 153, octreotide: 122). There were no significant differences in 30-day mortality (24% vs. 16%, $p=0.144$), in-hospital mortality (24% vs. 20%, $p=0.371$), or the need for repeat endoscopy (11% in both groups, $p=0.941$). Cardiac events occurred at similar rates between

groups (14% vs. 18%, $p=0.808$).

Conclusions:

Terlipressin and octreotide demonstrated comparable efficacy and safety as adjuncts to EVL in EVB management. Given the lack of significant differences in clinical outcomes, treatment selection should consider patient-specific factors, cost, and drug availability. Further prospective studies are needed to confirm these findings.

Keyword(s) : Esophageal variceal bleeding , Terlipressin , Octreotide, Endoscopic variceal ligation

1012 - Thromboangiitis obliterans in a Non Smoker Complicated by Budd Chiari Syndrome A Rare Case of Visceral Thrombosis

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Background / Hypothesis: Thromboangiitis obliterans is a rare inflammatory vascular condition that usually affects young male smokers, with symptoms limited to the limbs. Visceral involvement is extremely rare, especially in non-smokers

Materials and Methods: We present the case of a 20-year-old male with no history of smoking, who was evaluated for symptoms including digital ischemia, ulcers, and dry gangrene. A comprehensive workup ruled out atherosclerosis, autoimmune diseases, and other forms of vasculitis, leading to a diagnosis of thromboangiitis obliterans. The patient received medical therapy and wound care for symptom management.

Results: After nearly a year, the patient was readmitted with signs indicative of hepatic congestion, ascites, and spontaneous bacterial peritonitis. Imaging studies revealed hepatic vein outflow obstruction, leading to a diagnosis of Budd-Chiari syndrome. No thrombophilic disorders were identified, although the patient was found to have glucose-6-phosphate dehydrogenase deficiency.



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Conclusions: This case highlights a rare, severe form of thromboangiitis obliterans in a non-smoker, complicated by visceral thrombosis. It underscores the importance of considering systemic involvement even in atypical presentations and maintaining close follow-up in patients at risk of disease progression.

Keyword(s) : Thromboangiitis obliterans, Buerger's disease, non-smoker, Budd-Chiari syndrome, visceral thrombosis, glucose-6-phosphate dehydrogenase deficiency, spontaneous bacterial peritonitis, hypercoagulability, peripheral ischemia, atypical vascular presentation.

1014 - Validation of the CLIF SIG score in patients with HBV related acutely decompensated cirrhosis

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Background / Hypothesis:The CLIF-SIG (Chronic Liver Failure-Systemic Inflammation Gene) score has been proposed to quantify systemic inflammation and predict outcomes in acutely decompensated (AD) cirrhosis, primarily validated in alcohol-related cases. However, its applicability to hepatitis B virus (HBV)-related cirrhosis remains uncertain, given the distinct clinical profile of HBV-ACLF (acute-on-chronic liver failure). We hypothesized that CLIF-SIG is a valid prognostic tool for HBV-related AD cirrhosis.

Materials and Methods:Peripheral blood mononuclear cell (PBMC) transcriptomes from 220 HBV-related AD patients and 29 healthy controls were analyzed. CLIF-

SIG scores were calculated based on a 28-gene panel. Patients were stratified into clinical subgroups (stable decompensated cirrhosis [SDC], unstable decompensated cirrhosis [UDC], pre-ACLF, and ACLF). The prognostic performance of CLIF-SIG was compared with MELD and MELD-Na scores for predicting 90-day transplant-free mortality and AD-ACLF progression. **Results:**Expression analysis revealed significant upregulation of 24 inflammatory genes and downregulation of 4 NK cell-related genes in AD/ACLF patients, with a stepwise increase in CLIF-SIG scores across disease stages. The CLIF-SIG score effectively distinguished between SDC, UDC, pre-ACLF, and ACLF groups. Using a cut-off of 0.386, high-score patients exhibited markedly higher 90-day mortality ($p < 0.0001$). The area under the ROC curve (auROC) for CLIF-SIG in predicting 90-day mortality was 0.821 in AD and 0.73 in ACLF, outperforming MELD and MELD-Na. Furthermore, CLIF-SIG surpassed traditional inflammatory markers in assessing disease severity and progression. Combining CLIF-SIG with MELD further enhanced prognostic accuracy.

Conclusions:The CLIF-SIG score reliably quantified systemic inflammation and predicted clinical outcomes in HBV-related AD cirrhosis, demonstrating robust stratification across clinical phenotypes and outperforming conventional models. These findings support its generalizability beyond alcohol-related cirrhosis, underscoring systemic inflammation's pivotal role in HBV-related cirrhosis progression.

Keyword(s) : CIRRHOSIS; LIVER FAILURE; HEPATITIS B; INFLAMMATION

1015 - Gut Microbial Succession and Escalating Antibiotic Resistance from Chronic Hepatitis B to Acute on Chronic Liver Failure A Metagenomic Study

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Background / Hypothesis:Disruption of the gut microbiota is increasingly recognized as a pivotal driver of chronic liver disease progression, promoting systemic inflammation, immune dysregulation, and bacterial translocation. We performed a comprehensive metagenomic analysis to characterize the ecological and functional shifts in the gut microbiota across

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patients with chronic hepatitis B(CHB), acute decompensation (AD), and acute-on-chronic liver failure (ACLF).

Materials and Methods:Fecal samples (n = 90) collected from patients with CHB, AD, and ACLF were subjected to whole-genome shotgun metagenomic sequencing. Microbial diversity was assessed using alpha and betadiversity metrics, principal coordinate analysis (PCoA), and non-metric multidimensional scaling(NMDS). Differential taxa were identified using linear discriminant analysis effect size (LEfSe).Functional annotation of antibiotic resistance genes (ARGs) and virulence factors (VFs) was performed using the Comprehensive Antibiotic Resistance Database (CARD) and the VirulenceFactors Database (VFDB).

Results:Alpha diversity decreased progressively from CHB to ACLF, while beta diversity demonstrated significant group-wise separation (Kruskal-Wallis test, $p < 1 \times 10^{-100}$). Symbiotic genera, including Blautia, Ruminococcus, and Coprococcus, were enriched in AD, whereas pathobionts such as Escherichia coli and Enterococcus faecium dominated in ACLF. ARG profiling revealed a substantial increase in resistance burden in ACLF, characterized by the enrichment of β -lactamases, efflux pumps, and multidrug resistance elements (e.g., ARO:3005041, ARO:3000833).VFDB analysis showed upregulation of virulence determinants including type III secretion systems, lipopolysaccharides (LPS), and β -hemolysins. Functional analysis indicated a shift from a commensal-driven resistance profile in CHB and AD to a pathogen-dominated profile in ACLF, largely mediated by Enterococcus, Klebsiella, and Streptococcus species.

Conclusions:Progression from CHB to ACLF is associated with a stepwise ecological succession of the gut microbiota, culminating in pathogen-driven dysbiosis and heightened antimicrobial resistance. These findings highlight the gut microbiota as a potential biomarker for disease staging and a target for microbiota-directed therapeutic interventions in advanced liver disease.

Keyword(s) : CIRRHOSIS; LIVER FAILURE; HEPATITIS B; ACUTE-ON-CHRONIC LIVER FAILURE; GUT MICROBIOTA

1016 - Single cell analysis identifies RETN+ monocyte derived resistin as a therapeutic target in hepatitis B virus related acute on chronic liver failure

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Background and aims:Acute-on-chronic liver failure (ACLF) is characterized by intense systemic inflammation and high short-term mortality, yet effective targeted therapies are lacking. We aimed to explore monocyte heterogeneity in hepatitis B virus-related ACLF (HBV-ACLF) to identify specific subsets and associated therapeutic targets.

Materials and Methods:Peripheral blood mononuclear cells from healthy controls (n=4), patients with acute decompensation (AD, n=5), and ACLF patients (n=9) underwent single-cell RNA sequencing (scRNA-seq). Findings were integrated with hepatic scRNA-seq, bulk transcriptomics, multiplex immunohistochemistry, and in vitro functional assays. The in vivo roles of candidate targets were validated in two murine ACLF models.

Results:We identified a distinct RETN+ monocyte subset that was expanded in the circulation and liver of HBV-ACLF patients. These RETN+ monocytes displayed a transcriptional program enriched for anti-inflammatory and pro-resolving signatures, including MS1-B-like features. Interleukin-6 (IL-6) promoted the expansion of RETN+ monocyte via the JAK/STAT3/RUNX1 signaling axis. In murine ACLF models, neutralization of Resistin exacerbated liver injury, increased systemic cytokine levels, and augmented hepatocyte apoptosis. Conversely, administration of recombinant Resistin mitigated liver damage and diminished levels of inflammatory cytokines, and improved survival. Mechanistically, Resistin activated PI3K/AKT signaling in hepatocytes, preserved the Bcl-2/Bax balance, and suppressed intrinsic apoptosis. Clinically, plasma Resistin levels in HBV-ACLF patients inversely correlated with systemic inflammatory markers, liver injury, ACLF severity scores (MELD, CLIF-C ACLF), and short-term mortality.

Conclusions:RETN+ monocytes constitute a distinct immunoregulatory subset enriched in HBV-ACLF, and their product, Resistin, exerts significant hepatoprotective and anti-inflammatory effects. These findings highlight RETN+ monocytes and Resistin as promising immunotherapeutic targets for HBV-ACLF.

Keyword(s) : Liver failure; Single-cell analysis; Inflammation; Cirrhosis

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1024 - Comparison of effectiveness of mycophenolate mofetil associated with standard dosage or reduced dose tacrolimus for liver transplantation immunosuppression

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Background / Hypothesis:

Liver transplantation necessitates lifelong immunosuppressive therapy to prevent graft rejection. This systematic review evaluates the effectiveness of mycophenolate mofetil (MMF) in combination with standard-dose (SD) versus reduced-dose (RD) tacrolimus in liver transplant recipients.

Materials and Methods:

A systematic search was conducted across databases including PubMed, Cochrane, and Embase for studies published from 2000 to 2024. Inclusion criteria focused on randomized controlled trials (RCTs) and observational studies involving human subjects aged ≥ 18 . Primary outcomes included graft survival, acute rejection rates, and patient survival, while secondary outcomes encompassed renal function, infection rates, and adverse effects.

Results:

Out of 85 initially identified studies, 81 were excluded based on predetermined criteria. Ultimately, four studies involving a total of 255 patients (standard-dose: $n = 131$; reduced-dose: $n = 124$) were included for analysis. The findings indicated that mycophenolate mofetil (MMF) combined with tacrolimus, whether standard-dose or reduced-dose, allowed for a significant reduction in tacrolimus dosage, effectively minimizing nephrotoxic effects. Graft and patient survival rates were comparable between the MMF-tacrolimus regimen using standard and reduced doses. Additionally, the MMF group exhibited lower rates of acute rejection and showed improvements in renal function, with safety and efficacy profiles being similar across both dosage groups.

Conclusions:

The combination of MMF with either standard-dose or reduced-dose tacrolimus is an effective immunosuppressive strategy for liver transplant recipients. Both approaches facilitate a reduction in tacrolimus dosage while maintaining similar safety and efficacy. The MMF-tacrolimus regimen contributes to lower rates of acute rejection, with comparable graft and patient survival rates. Further research is warranted

to optimize immunosuppressive protocols and enhance patient outcomes.

Keyword(s) : Liver transplantation, Immunosuppression, Tacrolimus, Mycophenolate mofetil (MMF)

1026 - EXPLORING LABORATORY VARIATIONS IN CIRRHOSIS ETIOLOGIES A COMPARATIVE ANALYSIS OF CBC AND LIVER SYNTHETIC FUNCTION BETWEEN PATIENTS WITH ALCOHOLIC LIVER DISEASE AND OTHER ETIOLOGIES

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Background / Hypothesis: Cirrhosis remains a major cause of global morbidity and mortality, with varying clinical and laboratory features depending on etiology. Alcoholic liver disease (ALD) is one of the most prevalent causes of cirrhosis, globally. This study aimed to compare laboratory parameters—complete blood count (CBC) markers (hemoglobin [Hb], white blood cell count [WBC], platelet count [PLT]) and liver synthetic function tests (albumin and prothrombin time [PT])—between patients with ALD and those with other cirrhosis etiologies (e.g., MASLD, HCV, PBC) at the time of hospital admission.

Materials and Methods: We retrospectively analyzed the data of 160 cirrhotic patients admitted to Mikayelyan University Hospital, Yerevan, Armenia, between 2021 and 2024, categorized into ALD ($n=70$) and non-ALD ($n=90$) groups. Patients with hepatocellular carcinoma (HCC) were excluded. The patients had nearly the same age distribution, with male predominance in the ALD group. Laboratory data (albumin, PT, WBC, PLT, Hb) and clinical characteristics (including diabetes, hypertension, and substance use history) were recorded. Statistical comparisons between groups were performed using t-tests and Mann-Whitney U tests, as appropriate.

Results: Significant differences were found in multiple laboratory parameters between ALD and other cirrhosis groups. Patients with ALD exhibited significantly lower albumin levels (mean 2.95 g/dL) and higher PT (mean 18.5 seconds) compared to other cirrhosis patients (albumin: 3.35 g/dL, PT: 15.2 seconds) ($p<0.01$). In addition, ALD patients showed more severe thrombocytopenia (mean PLT: $107 \times 10^3/\mu\text{L}$) and a

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higher WBC count (mean $8.9 \times 10^3/\mu\text{L}$) compared to those with other cirrhosis etiologies (PLT: $129 \times 10^3/\mu\text{L}$, WBC: $7.3 \times 10^3/\mu\text{L}$) ($p < 0.05$). No significant difference was noted in Hb levels between the two groups (mean Hb for ALD: 12.4 g/dL vs. 12.3 g/dL for other cirrhosis). Additionally, patients with ALD demonstrated a higher prevalence of ascites and splenomegaly (both 70% vs. 55%, $p < 0.05$), reinforcing the severity of liver dysfunction and portal hypertension in this cohort. Conclusions: This study highlights clinically significant differences in laboratory markers between patients with ALD and other etiologies of cirrhosis. ALD was associated with more severe liver synthetic dysfunction (lower albumin, higher PT), thrombocytopenia, and a higher WBC count. These findings suggest that ALD has a unique clinical profile that may have implications for diagnosis and treatment strategies. Specifically, ALD patients may require more aggressive management of a complication such as ascites. Further prospective studies are warranted to validate these findings and investigate how they could inform therapeutic decisions, potentially improving patient outcomes in ALD and cirrhosis.

Keyword(s) :

1027 - Long term follow up of HCV patients who treated with DAA [Direct Acting Antivirals]

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Background: Our country ranks 5th in the world in terms of HCV infection prevalence, after Gambia, Mongolia, Egypt and Uzbekistan [WHO 2017]. 5.4% of the adult population is HCV RNA positive. The highest infection rates [15-24%] occur in men aged 30-49 years [NCDC]. Since 2015, the "Hepatitis C Elimination Program" has been launched in Georgia, within the framework of which antiviral treatment has been provided to more than 65 000 infected people. Five years after the start of the program, the prevalence of HCV infection was already reduced by 2 times in a repeated epidemiological study conducted by NCDC

Material/methods: The aim of the study was longitudinal follow-up of patients who achieved a sustained virological response [SVR] after antiviral therapy with DAA in 2016-17yy. The patients had been treated with ledipasvir/sofosbuvir [Harvoni] or sofosbuvir/velpatavir for 12 weeks. 200 patients with

past-HCV were included in the study. Of these, 100 patients had minimal liver fibrosis - F 1-2 [5-7 kPa] - I group, 100 patients - significant fibrosis - F 3-4 [10-12 kPa] - Group II.

result: After 8 years of antiviral treatment, 15 patients in the first group of patients developed fibrosis to F3, and no ultrasound or laboratory signs of hepatocellular carcinoma [HCC] were detected. 9% had type 2 diabetes mellitus, 45% had hyper- and dyslipidemia. All patients had fat accumulation in the liver parenchyma [S2-S3] - pronounced fatty liver disease [FLD]. 1/3 of patients in group II had progressive liver damage with the development of liver cirrhosis [Child-Pugh score Class A and Class B]. Cofactors for the development of liver cirrhosis were: alcoholic liver disease, type 2 diabetes mellitus, and obesity [BMI 34 and more]. 4 patients were already undergoing treatment for HCC. HCC was detected in 5 more patients during the study. [Total 9 patients]

conclusion: The most important measures to reduce the prevalence of HCV infection are total screening of the population and treatment of infected people with DAA. Treatment is highly effective in the early stages of liver damage. In addition, past-HCV individuals are more likely to develop FLD, which requires medical management. As for patients with significant liver damage, continuous monitoring is necessary to improve the quality of their lives and improve survival rates, for timely detection of damage progression and complications and adequate treatment.

Keyword(s) : HCV, DAA, treatment, FLD, LC

1028 - Chronic viral hepatitis [HCV HBV] course and outcome in patients with severe COVID 19]

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Background / Hypothesis: Chronic viral hepatitis [HCV, HBV] is a significant burden on the country's healthcare system. According to a study conducted by WHO [1917], 5.4% of the adult population is infected with HCV, more than half of whom have liver damage at the stage of significant fibrosis and cirrhosis. As for HBV infection, according to a study conducted by NCDC, the prevalence of HBsAg in the country was 2.9% [3.4% in men, 2.5% in women]. The highest prevalence was

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observed in the age groups of men: 30-39 and 40-49 years. The global COVID 19 pandemic of 1920-24, which was naturally also noted in our country, significantly affected this cohort of patients.

Material/Methods: The aim of the study was to study the course and outcome of chronic viral hepatitis [HCV, HBV] in patients with severe COVID 19 who were treated in the emergency and intensive care unit of the Bochorishvili Clinic. 112 patients were under observation. A - who had received antiviral treatment within the framework of the State Hepatitis C Elimination Program and achieved SVR, and patients with chronic HBV infection who were treated on an outpatient basis with antiviral treatment [with tenofovir or entecavir], and B - patients with chronic viral hepatitis [HCV, HBV] who had relapsed after infection with SARS CoV2 and hospitalization in a hospital.

Results: It was established that the more severe course of COVID 19, prolonged fever in the viremia phase, high rate of lung parenchymal damage on CT scan, the need for intensive care, including artificial ventilation of the lungs, and the unfavorable outcome of the disease did not depend on the etiological factor of liver damage. Worse prognosis, severe hypoalbuminemia, severe coagulopathy, thrombocytopenia The prevalence of bacterial sepsis, a high incidence of complications and a 2-fold higher mortality rate were observed in patients with liver damage in the stage of decompensated cirrhosis. In patients who had undergone antiviral treatment [past-HCV], or HBV was controlled with antiviral treatment, During the severe course of COVID 19, no high risk of complications and mortality was observed.

Conclusions: Therefore, in order to achieve a positive outcome in the further effective management and treatment of patients with chronic liver disease in the emergency department, it is necessary to immediately determine the stage of liver damage and carefully monitor the clinical and laboratory parameters of patients and correct impaired liver functions using appropriate medications.

Keyword(s) : HCV, HBV ,LC, COVID 19 ,complications.

1030 - A case of drug induced liver injury caused by salt fleshed psoralea corylifolia

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Background / Hypothesis:

Drug-induced liver injury (DILI) is one of the most frequent and serious adverse drug reactions and may progress to liver failure. Although traditional Chinese medicines (TCMs), natural medicines (NMs), health products (HPs), and dietary supplements (DSs) are often regarded as safe, their potential to cause DILI has gained increasing global attention. *Psoralea corylifolia* (Bu Gu Zhi) is a commonly prescribed TCM. Here, we describe a case of DILI associated with salt-processed *Psoralea corylifolia*.

Case Report:

A 61-year-old woman was admitted with abnormal liver function persisting for more than 20 days. Following lung cancer surgery, she had been taking herbal decoctions.

Prescription 1 contained Pinellia, Pericarpium Citri Reticulatae, Glycyrrhiza, Ostrea, Panax notoginseng powder, Scutellaria, Houlttuynia, and others.

Prescription 2 included Curcuma, Corydalis, Astragalus, Codonopsis, Atractylodes, Lycium, and Poria. Both prescriptions were taken twice daily. Liver function remained normal after one month.

Subsequently, Prescription 2 was modified by removing Corydalis and adding 15 g of salt-processed *Psoralea corylifolia*. One month later, liver tests showed ALT 135 U/L and ALP 122 U/L; after two months, ALT further increased to 541 U/L and ALP to 131 U/L. The patient reported no fatigue, anorexia, jaundice, pruritus, abdominal pain, or weight loss, and had no history of viral hepatitis, chronic liver disease, or drug/food allergies.

On examination, there was no jaundice, palmar erythema, or spider angiomas; the liver and spleen were not palpable. Viral hepatitis serology, autoimmune antibodies, HIV, CMV, EBV, and ceruloplasmin tests were negative. Renal function was normal, INR was 0.95, and abdominal ultrasonography was unremarkable.

DILI was strongly suspected. The herbal therapy was discontinued, and hepatoprotective agents (tiopronin and polyene phosphatidylcholine) were administered. After 15 days, ALT decreased to 77 U/L and ALP to 105 U/L.

Final diagnosis: drug-induced liver injury, hepatocellular type (ALT 10.8 × ULN, R = 10), acute; Roussel Uclaf Causality Assessment Method (RUCAM) score 10 (highly probable, salt-processed *Psoralea corylifolia*), severity grade 1.

Conclusions:

Salt-processed *Psoralea corylifolia* is frequently

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combined with other herbs to manage lumbar strain, osteoarthritis, and disorders attributed to liver–kidney deficiency or blood stasis. Clinical use of prescriptions containing *Psoralea* requires close monitoring of liver function, ideally at monthly intervals. Once abnormalities are detected, the agent should be discontinued promptly, and hepatoprotective therapy considered.

Keyword(s) : Drug-induced liver injury; Salt-fleshed *psoralea corylifolia*

1031 - Idiopathic Inflammatory Myopathy Presenting as Hepatic Injury A Case Report

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Background / Hypothesis:

Idiopathic inflammatory myopathies (IIM) are heterogeneous disorders characterized by proximal muscle involvement, with polymyositis (PM) and dermatomyositis (DM) as the most common subtypes. They are rare, frequently misdiagnosed in the early stage, and often associated with elevated serum enzymes; liver enzyme abnormalities occur in over 70% of patients. We describe a case of DM initially misattributed to hepatic injury.

Case Report:

A 65-year-old woman presented with 2 weeks of proximal muscle pain and weakness, unrelieved by rest or medication, followed by progressive left shoulder pain. Laboratory evaluation revealed ALT 348 U/L, AST 518 U/L, negative hepatitis B/C serology, WBC $8.95 \times 10^9/L$, neutrophils 82.5%, and ESR 35 mm/h. Hip MRI demonstrated diffuse inflammatory changes, vascular ultrasound revealed bilateral atherosclerosis with venous thrombosis, and shoulder MRI indicated rotator cuff injury. Because liver enzymes exceeded $10 \times ULN$, she was admitted with a presumptive diagnosis of hepatic injury.

On admission, she appeared fatigued with forehead and periorbital erythema, a V-sign rash, neck weakness, proximal muscle tenderness, impaired leg elevation (MRC grade III), and limited arm lifting. Cardiac enzyme showed AST 476 U/L, LDH 1600 U/L, CK 9798 U/L, CK-MB 570 U/L, and troponin I 1.01 ng/mL.

Autoantibodies and autoimmune liver markers were

negative.

Dermatomyositis was diagnosed. She was treated with intravenous methylprednisolone, cyclophosphamide, and tacrolimus, alongside hepatoprotective, anticoagulant, and supportive therapy. Her condition improved rapidly: rash resolved, muscle pain subsided, and repeat testing showed ALT 304 U/L, AST 321 U/L, CK 3013 U/L. She was discharged on outpatient follow-up.

Conclusions:

This case highlights that elevated liver enzymes in PM/DM often reflect muscle rather than hepatic injury. Given the heterogeneous and frequently atypical presentation, particularly in elderly patients, early misdiagnosis is common. Recognition of characteristic rashes and muscle weakness, combined with serum muscle enzyme testing, is essential for timely diagnosis and management.

Keyword(s) :

1032 - Drug Induced Liver Injury from Shenbao Tablets in an Elderly Patient A Case Report

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Background

Drug-induced liver injury (DILI) related to traditional Chinese medicines (TCMs), natural medicines (NMs), health products (HPs), and dietary supplements (DSs) has become a growing global concern. In China, elderly patients frequently use such products in an unregulated manner. Shenbao Tablets, a kidney-yang-tonifying preparation, are commonly prescribed in this population but carry a potential risk of hepatotoxicity. We report a severe case of Shenbao Tablet-induced DILI in an elderly man.

Case Report

A 65-year-old man presented with fatigue and dark urine for 10 days. He had been self-medicating with Shenbao Tablets for 20 days. Past history included hypertension treated with nimodipine and benazepril.

Examination revealed marked jaundice of skin and sclera without stigmata of chronic liver disease. Abdomen was soft with no hepatosplenomegaly or

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ascites. No peripheral edema was noted. Laboratory tests: TBil 199 $\mu\text{mol/L}$, DBil 118 $\mu\text{mol/L}$, albumin 34 g/L, ALT 1006 U/L, AST 1128 U/L, GGT 270 U/L, ALP 242 U/L; INR 1.13, prothrombin activity 74%. Viral hepatitis serology and autoimmune markers were negative. Ultrasound showed normal liver morphology, dense parenchymal echoes, and gallstones without biliary dilatation.

Shenbao Tablet-related DILI was suspected. The drug was discontinued, and hepatoprotective therapy (compound glycyrrhizin, polyene phosphatidylcholine, Ganshukang, Wuzhi capsules) was initiated. Despite treatment, jaundice progressed: one week later, TBil 294 $\mu\text{mol/L}$, DBil 151 $\mu\text{mol/L}$, ALT 535 U/L, AST 592 U/L, GGT 396 U/L, ALP 391 U/L. After 50 days of therapy, liver function normalized. The patient remained symptom-free during 2 years of follow-up without re-exposure.

Final diagnosis: drug-induced liver injury, hepatocellular type (ALT 20.1 $\times$ ULN, R=10), acute; RUCAM score 9 (highly probable, Shenbao Tablets); severity grade 3 (severe).

Discussion and Conclusions

Shenbao Tablets contain 22 herbal ingredients, including Epimedium, Psoralea, Polygonum multiflorum, and Cnidium, all reported to cause hepatotoxicity. Although marketed as a tonic, its safety is not assured. In conclusion, health supplements are not universally safe, particularly in elderly patients. Their use should be guided by professionals, and unexplained symptoms during administration warrant immediate medical attention.

Keyword(s) :

1033 - A Rare Case of Peptic Ulcer Perforation Associated with Lenvatinib in Hepatocellular Carcinoma

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Introduction: Lenvatinib is a type I tyrosine kinase inhibitor with antiangiogenic activity, approved as first-line treatment for patients with advanced, unresectable hepatocellular carcinoma (HCC). Gastrointestinal (GI) adverse effects are most commonly observed within the first six months of treatment initiation. Among these, gastrointestinal perforation is a rare but life-

threatening complication, occurring in approximately 1.3% of patients receiving lenvatinib.

Case Presentation: We report the case of a 54-year-old Asian male with unresectable HCC who presented to the emergency department with acute, severe abdominal pain after two months of lenvatinib therapy (8 mg daily). An esophagogastroduodenoscopy (EGD) performed prior to treatment initiation revealed no abnormalities. Physical examination showed diffuse abdominal tenderness with guarding. Laboratory results were notable for elevated serum creatinine, hyponatremia, and prolonged INR. A non-contrast CT scan of the abdomen revealed massive pneumoperitoneum with extraluminal air surrounding the stomach and diffuse thickening of the gastric fundus wall. Empiric antibiotic therapy with piperacillin-tazobactam was initiated for suspected peritonitis secondary to GI perforation. An emergent exploratory laparotomy revealed a 2 $\times$ 2 cm perforated peptic ulcer on the lesser curvature of the stomach. Modified Graham patch repair was performed, and biopsy specimens were negative for *Helicobacter pylori*. Histopathology showed chronic inflammation, hemorrhage, and ulceration. The patient recovered well postoperatively and was discharged in stable condition.

Conclusion: This case highlights the importance of clinician awareness regarding the potential for serious, albeit uncommon, gastrointestinal complications associated with lenvatinib therapy. Early recognition and timely surgical intervention are critical to improving patient outcomes and reducing the risk of morbidity and mortality.

Keyword(s) : "lenvatinib", "perforated peptic ulcer", "unresectable hepatocellular carcinoma"

1036 - Two cases of drug induced liver damage in the elderly caused by Tusanqi (Gynura japonica)

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Background:

Herbal medicine-related drug-induced liver injury (DILI) has become increasingly recognized in the elderly, partly due to widespread use of health supplements. Tusanqi (*Gynura japonica*), a traditional herb containing hepatotoxic pyrrolizidine alkaloids, is

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easily mistaken for Sanqi (*Panax notoginseng*), which is non-toxic. We report two elderly patients who developed severe chronic liver injury after prolonged ingestion of Tusanqi.

Case Reports:

The first patient, an 80-year-old man, presented with progressive abdominal distension for three months and jaundice for one month. He had ingested self-prepared Tusanqi powder daily for over three years. Laboratory tests revealed marked hyperbilirubinemia, hypoalbuminemia, and coagulopathy, with negative viral and autoimmune markers. CT imaging confirmed cirrhosis with reduced liver volume. He was diagnosed with Tusanqi-induced DILI (RUCAM score 10), decompensated cirrhosis, and hepatic encephalopathy. Despite cessation of the herb and supportive therapy, his condition deteriorated, leading to liver failure and death after 40 days of hospitalization.

The second patient, his 75-year-old wife, had consumed Tusanqi in the same manner. Approximately six months after her husband's diagnosis, she developed confusion, behavioral changes, and hepatic encephalopathy. Laboratory findings showed hypoalbuminemia and mild hyperbilirubinemia, while ultrasonography revealed cirrhotic changes. She was likewise diagnosed with Tusanqi-induced DILI (RUCAM score 10), decompensated cirrhosis, and hepatic encephalopathy. Her encephalopathy responded to branched-chain amino acid therapy, but due to poor adherence she experienced repeated relapses, resulting in 10 hospital admissions over three years. She remains alive with persistent hepatic dysfunction.

Discussion:

Unlike the acute hepatic sinusoidal obstruction syndrome (HSOS) commonly associated with pyrrolizidine alkaloids, both patients developed progressive cirrhosis and chronic liver failure after long-term use. This difference may relate to ingestion method, cumulative exposure, comorbidities, and individual susceptibility. The male patient's rapid deterioration contrasted with his wife's relatively milder course, suggesting variability in host response.

Conclusions:

These cases highlight the hepatotoxic risk of Tusanqi, especially in the elderly. Misidentification with Sanqi contributes to accidental ingestion and delayed recognition of toxicity. Clinicians should maintain vigilance for chronic DILI in patients with unexplained cirrhosis, particularly with a history of herbal use.

Keyword(s) :

1038 - Comparative Analysis of Epidemiological Characteristics of Hepatitis A and Hepatitis E in Beijing from 2015 to 2024

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Background: Although both hepatitis A and hepatitis E are primarily transmitted through the ingestion of contaminated food or water, the differences in their epidemiological characteristics in Beijing remain poorly understood.

Materials and Methods: thods were used to characterize the epidemiological fDescriptive epidemiological meeasures of HAV and HEV cases reported in Beijing from 2015 to 2024. Chi-square tests or t-tests were employed to compare differences in time, region, and population distribution between the two diseases.

Results: From 2015 to 2024, a total of 1,123 HAV cases (977 confirmed, 87.00%) and 3,321 HEV cases (2,972 confirmed, 89.49%) were reported in Beijing. The proportion of confirmed cases was lower for HAV than for HEV ($\chi^2 = 5.265$, $P = 0.022$). Temporally, both diseases showed an initial increase (2015–2018), followed by a decline (2019–2022), and then a resurgence (2023–2024). Urban areas accounted for 61.44% of HAV cases and 61.55% of HEV cases. The proportion of HAV cases in suburban areas (35.17%) was higher than that of HEV (30.38%), while the proportion in rural areas was lower for HAV (3.38%) than for HEV (8.07%), with a statistically significant difference ($\chi^2 = 32.863$, $P < 0.001$). Both diseases were more common in males, but HEV had a higher male predominance (65.70% vs. 51.91% for HAV, $\chi^2 = 67.876$, $P < 0.001$). The mean age of onset for HAV patients (51.0 years) was younger than that for HEV patients (54.9 years), with a statistically significant difference ($t = -7.018$, $P < 0.001$). The top five occupations among HAV cases were homemakers or unemployed (297 cases, 26.45%), retirees (293 cases, 26.09%), office workers (193 cases, 17.19%), commercial service workers (80 cases, 7.12%), and farmers (63 cases, 5.61%). For HEV, the top five were retirees (1,193 cases, 35.92%), homemakers or unemployed (827 cases, 24.90%), office workers (475

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cases, 14.30%), farmers (233 cases, 7.02%), and workers (172 cases, 5.18%). The occupational distribution differed significantly between the two diseases ($\chi^2 = 69.730$, $P < 0.001$).

Conclusions: The incidence trends of HAV and HEV in Beijing were generally consistent. Both diseases were predominantly reported in urban areas, but their distributions in suburban and rural areas differed. The main affected populations for both diseases were retirees, homemakers or unemployed, and office workers. HEV cases had a higher proportion of males and an older age of onset.

Keyword(s) : Hepatitis A, Hepatitis E, epidemiological characteristics, Beijing

1040 - Small Intestinal Bacterial Overgrowth A Missing Link in Gut–Liver Axis Dysregulation of MASLD

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Background / Hypothesis:

Small intestinal bacterial overgrowth (SIBO) may implicate in the progression of metabolic dysfunction-associated steatotic liver disease (MASLD) by disrupting the gut microbiota, compromising intestinal barrier integrity, and elevating levels of endogenous metabolites. Our study aims to explore the relationship between both, specifically focusing on the role of gut microbiota composition and its metabolic byproducts in the pathogenesis and progression of MASLD.

Materials and Methods:

We included patients who underwent abdominal scans and lactulose hydrogen methane breath tests at our hospital. Independent sample t-tests were conducted for basic statistical analyses of the MASLD population. Stratified analyses of MASLD patients were then performed, and the relationship between MASLD and SIBO was assessed using chi-square tests. To identify factors associated with MASLD, a multivariate logistic regression model was applied. Additionally, stool

samples from MASLD patients were collected for 16S rRNA gene sequencing to assess the impact of SIBO status on the distribution of intestinal microbiota and metabolic characteristics in MASLD patients.

Results:

A total of 350 participants were included (97 MASLD [27.7%], 253 non-MASLD [72.3%]). SIBO was highly prevalent (300/350, 85.7%) and differed significantly between MASLD and non-MASLD groups. Multivariate analysis identified SIBO as an independent risk factor for MASLD, with methane-positive SIBO showing a stronger association. The characteristic flora in the MASLD group was primarily distributed within the phylum Firmicutes, with significant enrichment of genera such as Lactobacillales and Bacilli. The SIBO group was predominantly characterized by Proteobacteria, with a notable increase in the Burkholderiaceae family and related genera, such as Dialister, Limnobacter, and Acidovorax. Differential metabolic pathways between the groups were largely concentrated in metabolic processes.

Conclusions:

SIBO contributes to MASLD development, particularly methane-positive SIBO. Distinct microbial signatures were observed, with Firmicutes enrichment in MASLD and Proteobacteria in SIBO patients. Altered gut metabolites mainly involved metabolic pathways, highlighting the potential role of gut microbiota and its metabolites in MASLD pathogenesis.

Keyword(s) : MASLD; Small intestinal bacterial overgrowth; SIBO; Gut–liver axis; Gut microbiota;

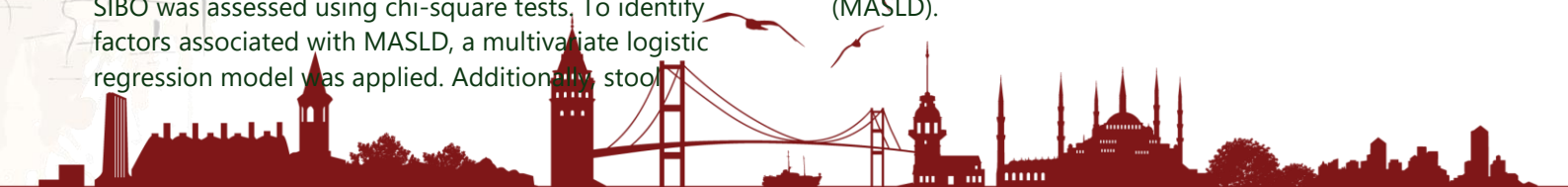
1041 - Controlled Attenuation Parameter Factors and Steatosis Grading in Metabolic Dysfunction associated Steatotic Liver Disease

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Background / Hypothesis:

To identify factors influencing liver-controlled attenuation parameter (CAP) and establish its optimal diagnostic thresholds for hepatic steatosis grading in metabolic dysfunction-associated steatotic liver disease (MASLD).



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Materials and Methods:

758 prospectively enrolled MASLD patients (Zhongshan Hospital, Sep 2022-Jun 2023) were randomized into training (n=619) and validation (n=139) cohorts, stratified by BMI (normal-weight: BMI<25 kg/m²; overweight: BMI≥25 kg/m²). Demographics, laboratory parameters, CAP, and MRI proton density fat fraction (PDFF) were recorded. Using MRI-PDFF as the reference, ROC analysis established CAP thresholds for steatosis grades.

Results:

In the training cohort, multivariate analysis showed CAP correlated with uric acid (b=0.11, P=0.073), glucose (b=13.76, P<0.001), and HDL (b=-45.47, P=0.020) in normal-weight patients, and with ALT (β=0.164, P=0.004), total cholesterol (β=8.41, P=0.006), and BMI (β=2.47, P=0.006) in overweight patients. Overall cohort CAP thresholds were: S1 (≥5%) 283.5 dB/m (AUC=0.880), S2 (≥33%) 311.5 dB/m (AUC=0.712), S3 (≥66%) 328.5 dB/m (AUC=0.799). Normal-weight thresholds: S1 259.5 dB/m (AUC=0.922), S2-3 323.5 dB/m (AUC=0.779). Overweight thresholds: S1 304.0 dB/m (AUC=0.829), S2 311.5 dB/m (AUC=0.677), S3 328.5 dB/m (AUC=0.767). Validation confirmed robust diagnostic performance (Youden index 0.82 overall, 0.83 normal-weight, 0.81 overweight).

Conclusions:

CAP correlates significantly with metabolic factors (uric acid, glucose, HDL) in normal-weight MASLD patients and with liver markers/metabolic factors (BMI, ALT, TC) in overweight patients. CAP demonstrates good diagnostic accuracy for grading hepatic steatosis severity in MASLD, particularly for mild to moderate steatosis.

Keyword(s) : control attenuation index; metabolic dysfunction-associated steatotic liver disease; normal weight; overweight; correlation analysis

1043 - Non invasive detection of occult yet significant liver pathology in ALT normal chronic hepatitis B a multicenter study

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Background / Hypothesis:

Individuals with chronic hepatitis B (CHB) may harbor occult yet significant liver pathology despite normal alanine aminotransferase (ALT) levels, representing a major diagnostic challenge. We aimed to identify and validate non-invasive biomarkers for detecting significant liver pathology among these population.

Materials and Methods:

This multicenter study screened 3,258 CHB cases from 2013-2023, enrolling 137 treatment-naïve HBeAg+ and 253 HBeAg- infection patients with normal ALT (≤40 U/L). All participants underwent liver biopsy (gold standard), liver function and novel biomarker measurement (including CK18-M30, CK18-M65, GP73, IL-10, and IL-2R). Significant liver pathology was defined as inflammation grade (G) and/or fibrosis stage (S) ≥2. Sixteen CK18-M65-centered biomarker combinations were evaluated using eight machine learning algorithms with multicenter stratified validation.

Results:

Significant liver pathology was observed in 45.2% of HBeAg+ infection cases. CK18-M65, CK18-M30, and GP73 strongly correlated with pathology severity (G stage: 0.757, 0.688, and 0.453, respectively; p<0.0001), independent of ALT levels. CK18-M65 demonstrated optimal diagnostic performance (AUC=0.934, 95% CI: 0.896-0.973) with 85.5% sensitivity and 88% specificity, especially in those with low-normal ALT. High CK18-M65 levels conferred >40-fold increased risk of severe liver injury (adjusted OR=40.64). The optimal biomarker combination (CK18-M65+CK18-M30+GP73) achieved AUROC of 0.942, significantly outperforming LSM (0.824), APRI (0.783), and FIB-4 (0.745; all p<0.01), with enhanced clinical utility. HBeAg- infection cases

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showed 46% significant pathology but weaker diagnostic performance.

Conclusions:

CK18-M65-centered biomarker models provide promising non-invasive tools for detecting occult liver pathology and risk stratification of ALT-normal HBeAg+ CHB infection, potentially reducing unnecessary biopsies while identifying candidates for early intervention.

Keyword(s) : chronic hepatitis B (CHB); CK18-M65; non-invasive diagnosis; ALT-normal; liver pathology

1044 - Multifactorial Anemia Unveiling Gastric Antral Vascular Ectasia in a Stage IV Breast Cancer Survivor Endoscopic Resolution with Argon Plasma Coagulation

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Background / Hypothesis:

Gastric Antral Vascular Ectasia (GAVE), or "watermelon stomach," is an uncommon cause of upper gastrointestinal bleeding and chronic anemia. While typically linked to cirrhosis or autoimmune disorders, GAVE may occur in non-cirrhotic elderly patients, especially those with prior oncologic treatments that compromise mucosal integrity. We report a unique case of GAVE in a non-cirrhotic, elderly Filipino female with metastatic breast cancer, successfully managed with argon plasma coagulation (APC).

Materials and Methods:

An 85-year-old female with a remote history of bilateral mastectomy and recent diagnosis of Stage IV invasive ductal carcinoma presented with transfusion-dependent anemia. Laboratory and imaging findings revealed no overt gastrointestinal bleeding. Endoscopy uncovered classic GAVE with linear vascular streaks in the antrum and associated Forrest III gastric ulcers. APC was performed in two sessions with excellent endoscopic and hematologic response. Argon plasma coagulation (APC) was applied in two sessions targeting ectatic vessels in the antrum. Narrow-band imaging aided lesion demarcation. Treatment settings were 40W, 1.5 L/min.

Results:

Follow-up endoscopy revealed marked resolution of lesions. Hemoglobin stabilized, transfusions ceased, and the patient remained asymptomatic. This underscores the importance of considering GAVE in multifactorial anemia, even in non-cirrhotic oncology patients.

Conclusions:

This case highlights GAVE as a reversible cause of anemia in elderly cancer survivors, particularly those with prior chemotherapy and radiation. It also reinforces the safety and efficacy of APC in frail patients. Serial endoscopy provides a compelling narrative for diagnosis and therapeutic success.

Keyword(s) :

1047 - Comparative Analysis of Epidemiological Characteristics and Co infection of Hepatitis B and Hepatitis C in Beijing from 2010 to 2024

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Background:

Through continuous prevention and control effect, the incidence rate of viral hepatitis has been significantly reduced in Beijing. There are similarities in transmission routes between hepatitis B and hepatitis C, the difference in epidemic characteristics between them and the status of coinfection remain to be determined.

Materials and Methods:

Descriptive epidemiological methods were used to analyze the characteristics of HBV and HCV cases in Beijing from 2010 to 2024. The prevalence of co-infection in different populations was calculated. Chi-square tests or rank-sum tests were employed to compare case distributions or differences in age of onset between groups.

Results:

From 2010 to 2024, a total of 45,728 HBV cases and 15,769 HCV cases were reported in Beijing. The annual number of cases decreased from 4,724 (HBV) and 1,671 (HCV) in 2010 to 2,556 (HBV) and 945 (HCV) in 2016, rebounded slightly in 2017–2018, and then increased significantly to 2024(3,546 HBVs and 1,137 HCVs). Both HBV and HCV cases were concentrated in urban areas. The proportion of male patients was higher in HBV (64.14%) than in HCV (56.24%, $P < 0.001$). Retirees accounted for a higher proportion of HCV cases

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(26.54%) than HBV cases (14.88%). The median age of onset for HBV (47 years) was younger than for HCV (53 years). Among HBV patients, 76.04% were aged 20–59 and 22.81% were ≥60 years old, compared to 64.16% and 34.68% for HCV, respectively ($P < 0.001$). 201 cases of HBV/HCV co-infection were reported. 143 (71.14%) were diagnosed with both infections within one month, 34 (16.92%) were first diagnosed with HBV, and 24 (11.94%) were first diagnosed with HCV. The co-infection rate among HBV cases was 0.44% (201/45,728), with no significant difference between genders. The rate was higher in urban areas (0.58%) than in suburban (0.41%) and rural areas (0.21%) ($P < 0.001$). The median age of co-infected patients (52 years) was higher than that of HBV-only patients. The co-infection rate among HCV cases was 1.27% (201/15,769), with a higher rate in males (1.58%) than females (0.88%) ($P < 0.001$). No significant differences were observed among regions. The median age of co-infected patients (52 years) did not differ significantly from that of HCV-only patients (53 years) ($P = 0.112$). Conclusion: The reported cases of HBV and HCV in Beijing exhibited similar temporal trends, with both diseases showing a higher proportion of male cases and urban clustering. The proportion of co-infection was low, and differences in co-infection rates were observed across genders and regions.

Keyword(s) : Beijing, Hepatitis B, Hepatitis C, epidemiological characteristics, Coinfection

1048 - Analysis of the Epidemiological Characteristics of Hepatitis B in Beijing from 2008 to 2024

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Background:

Beijing has made significant achievements in controlling hepatitis B through vaccination programs, but there may be differences in the epidemiological patterns of hepatitis B in specific regions and populations in recent years.

Materials and Methods:

Descriptive epidemiological methods were used to analyze the temporal, spatial, and population distribution characteristics of hepatitis B cases in Beijing from 2008 to 2024.

Results:

A total of 56,694 hepatitis B cases were reported in Beijing from 2008 to 2024, including 4,736 acute cases (8.36%), 48,841 chronic cases (86.22%), and 3,072 unclassified cases (5.42%). The annual number of hepatitis B cases showed a declining trend from 2008 to 2016 and from 2018 to 2022, with minor increases in 2017–2018 and 2023–2024 (primarily chronic cases). In 2024, 3,546 cases were reported (including 66 acute cases), representing a 40.70% decrease compared to 2008 (5,980 cases, including 824 acute cases), with a 91.99% decline in acute cases. No obvious seasonal trend was observed in monthly case reports.

Geographically, urban areas reported more cases (24,743 cases, 43.68%) than suburban (21,190 cases, 34.71%) and rural areas (9,181 cases, 16.21%), with 1,535 cases from unspecified regions (all reported before 2015). Acute cases declined significantly across all regions (all decreased by 90%), while chronic cases decreased markedly in urban and suburban areas (decreased by 44.75% and 33.44% in 2024 compared to 2008, respectively) but only slightly in rural areas (7.05%). Males accounted for 63.85% of all cases.

Among acute cases, males accounted for 69.17%, with the proportion gradually decreasing from 72.33% in 2008 to 59.09% in 2024. The mean age of acute and chronic cases increased from 37.4 and 44.0 years in 2008 to 51.0 and 51.3 years in 2024, respectively (median ages increased from 36 and 43 to 50 and 51 years, respectively). The top five occupations among reported cases were household workers/unemployed (29.40%), farmers (16.09%), retirees (14.31%), office workers (10.93%), and laborers (8.25%). Students and healthcare workers accounted for 1.71% and 0.42% of cases, respectively. Compared to 2008, the number of new cases in 2024 decreased significantly across all occupational groups except household workers/unemployed and retirees.

Conclusions:

The overall number of hepatitis B cases in Beijing has shown a declining trend, with a more pronounced decrease in acute cases. However, the decline in new chronic cases has been slower in rural areas, and the age of new cases has gradually increased. Cases among homemakers/unemployed and retirees have decreased more slowly.

Keyword(s) : Beijing, Hepatitis B, Epidemiological Characteristics

1049 - Rifaximin to reduce the risk of overt hepatic encephalopathy in patients undergoing TIPS real

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world experience in a diverse population

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Background / Hypothesis:

Bureau et al. (Ann Intern Med, 2021) demonstrated that rifaximin reduces the risk of overt hepatic encephalopathy (HE) in patients with cirrhosis undergoing TIPS when started prior to and continued after TIPS. Eighty-six per cent of the study population had ALD.

This practice has been incorporated into EASL CPG. We reviewed current practices and real world outcomes of TIPS in our institution, in a more diverse population.

Materials and Methods:

A retrospective analysis of all adults undergoing TIPS at our hospital between 2/2/21 and 7/5/24 was performed. Primary outcome was rifaximin prescription.

Seventy-six patients were identified, of whom three were excluded due to being on broad-spectrum antibiotics at repatriation and six patients were excluded due to incomplete records. Sixty-seven patients were included.

Results:

Fifty-four patients (80.6%) were cirrhotic. Median UKELD score was 50 (IQR = 47-53) and median MELD-Na was 12 (IQR = 9-17).

Primary indication for TIPS was to treat variceal bleeding in 38.8%, diuretic-intractable ascites in 23.9%, hydrothorax +/- ascites in 3%, and for non-cirrhotic indications (e.g., Budd-Chiari syndrome) in 14.9%. 53.7% had alcohol-related cirrhosis.

Fourteen patients (20.9%) had documented HE prior to TIPS (excluding those patients whose HE had occurred historically, prior to liver transplantation), of whom eight (57.1%) were on rifaximin prior to TIPS. All eight patients continued on rifaximin post-TIPS, of whom four (50%) developed HE; two patients (25%) did not develop HE, and data was unavailable for two patients. Of the remaining six patients, three (50%) were commenced on rifaximin prior to hospital discharge, of whom one (33.3%) developed HE. Of the three patients not prescribed rifaximin prior to hospital discharge, two (66.7%) developed HE; the one patient who did

not was not cirrhotic.

Overall, 47 patients (70.1%) were prescribed rifaximin prior to hospital discharge. Of these, 22 (46.8%) are known to have developed HE, 18 (38.3%) did not develop HE, and data is unavailable for seven patients (14.9%).

Overall, 20 patients (29.9%) were not prescribed rifaximin prior to hospital discharge. Of these, eight (40%) are known to have developed HE, 10 (50%) did not develop HE, and data is unavailable for two patients (10%).

Conclusions:

Rifaximin prescription is usual post-TIPS.

Despite rifaximin being commonly used, HE incidence remains high.

Excluding unknowns, HE incidence post-TIPS was unrelated to UKELD ($p = 0.9937$), however, it approached significance for MELD-Na ($p = 0.0605$), and the small sample sizes (22 HE post-TIPS, 18 no HE post-TIPS) may explain the lack of statistical significance.

Keyword(s) : hepatic encephalopathy, rifaximin, TIPS

1052 - Pan cancer analysis identifies hemopexin as a tumor suppressor in HCC via TNF alpha mediated mitochondrial apoptosis

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Background / Hypothesis: Fibrinolysis, a crucial physiological and pathological process responsible for fibrin degradation in the vascular system, serves as a pivotal regulatory mechanism in coagulation homeostasis. Despite its significance, the pan-cancer biological role of fibrinolysis remains largely unexplored. This study investigates the functional characteristics of fibrinolysis across cancers, with particular focus on its pivotal role in hepatocellular carcinoma (HCC). We aimed to establish a novel molecular classification and risk signature for HCC and identify a key gene influencing its occurrence and progression.

Materials and Methods: Firstly, the differential expression of fibrinolysis-related genes was analyzed in pan-cancer. Subsequently, the non-negative matrix factorization method was employed to identify the fibrinolysis-related molecular subtypes in HCC, and a risk signature was developed. The key gene was

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determined by this risk signature. Further in vivo and in vitro experiments were performed to elucidate the role and underlying mechanism of this gene in the occurrence and progression of HCC.

Results: Fibrinolysis is highly expressed in liver tissues and serves as a protective factor for HCC across pancreatic cancers. Three different molecular subtypes of HCC were successfully distinguished based on the characteristics of fibrinolysis, which were correlated to the prognosis, ImmuneScore, StromaScore, and ESTIMATEScore of HCC patients. A prognostic signature including ACAT1, GRHPR, HPX, PCK2, IYD, and PON1 was developed based on the weighted gene co-expression network analysis (WGCNA) and least absolute shrinkage and selection operator (LASSO) analysis. With the largest absolute value of log2foldchange, hemopexin (HPX) was determined as key gene and for further in vivo and in vitro validation. HPX showed an anti-tumor phenotype via clone formation, EdU assay, wound healing, transwell and xenograft tumor model. Furthermore, RNA-seq results suggest that HPX is associated with apoptosis and TNF- α signaling pathways, flow cytometry apoptosis assay, mitochondrial membrane potential assay and TUNEL staining and were performed to validate these findings above. Western blot and immunohistochemistry showed HPX could upregulate Bax/Bcl-2 expression by activating of TNF- α signaling pathway.

Conclusions: This study identified novel molecular subtypes of HCC. A risk signature with clinical application value was constructed, and the key gene HPX in HCC was further identified. It provides a new direction for the precise treatment of HCC patients.

Keyword(s) : Fibrinolysis, Hepatocellular carcinoma, Hemopexin, Apoptosis

1053 - Development and validation of the FAAP model for prognostic stratification in HCC patients treated with TACE sintilimab plus bevacizumab a multicenter study

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Background / Hypothesis: Although the transcatheter arterial chemoembolization (TACE) combined with sintilimab and bevacizumab improves outcomes in

unresectable HCC (uHCC), predictive tools are lacking. This study developed and validated a prognostic model for triple therapy efficacy.

Materials and Methods: A multicenter study enrolled uHCC patients receiving TACE-sintilimab-bevacizumab. Overall survival (OS) was the primary endpoint; a Cox model was developed and validated.

Results: This study enrolled 147 patients (training cohort: n = 92; validation cohort: n = 55). The optimal cutoff value for the fibrin degradation product-to-cholinesterase ratio*1000 (FCR) was determined as 0.8. Univariate and multivariate Cox regression analyses identified FCR, AST, AFP, and PVTT as independent OS predictors. These variables were integrated to establish the FAAP scoring system, which demonstrated robust discriminative performance with AUC of 0.804 (95% CI: 0.703-0.893) and 0.799 (95% CI: 0.67-0.911) in the training and validation cohorts, respectively. Patients were stratified into three risk groups based on FAAP scores: low (FAAP < 0.7), intermediate (0.7 $\leq$ FAAP < 2.2), and high (FAAP $\geq$ 2.2). Kaplan-Meier analyses revealed significant prognostic stratification for both OS and progression-free survival (PFS) across groups. Subgroup analyses confirmed the prognostic relevance of FAAP scores in key clinical subsets, including age, gender, extrahepatic metastasis status, viral hepatitis etiology, PVTT presence, and Child-Pugh stage.

Conclusions: The FAAP scoring system effectively predicted survival outcomes in HCC patients receiving TACE-sintilimab-bevacizumab therapy, which suggests its clinical utility for treatment benefit evaluation. Further large prospective studies are required for external validation.

Keyword(s) : hepatocellular carcinoma; immunotherapy; transcatheter arterial chemoembolization; prognostic model

1057 - Serum N glycomics for non invasive detection of significant liver pathology across clinical phases of treatment naïve chronic hepatitis B

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Background / Hypothesis:

Early identification of significant liver pathology is crucial for timely antiviral intervention in individuals with chronic hepatitis B (CHB) infection. Current non-invasive methods show limited accuracy in detecting occult liver damage, particularly in those with normal ALT. This study evaluated serum N-glycan profiles for diagnosing significant liver pathology in treatment-naïve CHB patients across clinical phases.

Materials and Methods:

This cross-sectional study analyzed 626 treatment-naïve CHB patients confirmed by liver biopsy, classified according to 2025 EASL guidelines. Serum N-glycan profiles were determined using DNA sequencer-assisted fluorophore-assisted carbohydrate electrophoresis. Significant liver pathology was defined as inflammation grade $\geq$ G2 and/or fibrosis stage $\geq$ S2. Multivariate logistic regression models were developed and compared with traditional non-invasive markers.

Results:

Among 626 CHB patients, 66.0% had significant inflammation and 58.9% had significant fibrosis. Patients with significant pathology showed characteristic alterations, with elevated P1, P3, P6, P7, P11 peaks and decreased P0, P5, P8, P10 peaks (all $p < 0.0001$). Compared to infection phases, hepatitis phases showed P1 increases of 19.6%/36% and P11 increases of 82.4%/73.4% in HBeAg(+)/HBeAg(-) patients, while P0 decreased by 20.3%/27.6% and P10 decreased by 21.6%/20.3%. In patients with significant pathology (G and/or S $\geq$ 2), P1 increased by 27% on average, reaching 58.7%/48.7% in G4/S4 stages. In ALT-normal patients, particularly HBeAg(+) infection phase, P1 increased by 80.2%/65.8% in G4/S4 stages, with P2 also increasing by 54.1%/45.2%. Multivariate analysis identified P11 as strongest risk factor (OR=3.84, 95%CI: 1.74-8.45, $p=0.0008$), followed by P1 (OR=2.04, 95%CI: 1.57-2.64, $p < 0.0001$) and P7

(OR=1.75, 95%CI: 1.31-2.34, $p=0.0002$), while P2 (OR=0.07, 95%CI: 0.02-0.26, $p < 0.0001$) and P0 (OR=0.30, 95%CI: 0.12-0.79, $p=0.0140$) served as protective factors. The glycomics combined model (AUC=0.876) achieved superior performance outperformed the clinical model (0.818), LSM (0.817), APRI (0.830), and FIB-4 (0.672), with 78.7% sensitivity and 83.2% specificity. The optimized model reached AUC=0.917 with accuracy 84.2%, with 78.7% sensitivity and 94.6% specificity. Both glycol-based models maintained diagnostic capability in ALT-normal patients particularly in HBeAg(+) infection.

Conclusions:

Serum N-glycomics demonstrates promising potential for non-invasive identification of significant liver pathology in treatment-naïve CHB patients, providing an alternative approach for early treatment decisions, especially in ALT-normal patients with occult liver damage.

Keyword(s) : Chronic hepatitis B; Treatment-naïve; N-glycomics; Liver pathology; Non-invasive diagnosis

1058 - Serum N glycomics for non invasive detection of significant liver pathology across clinical phases of treatment naïve chronic hepatitis B

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Background / Hypothesis:

Early identification of significant liver pathology is crucial for timely antiviral intervention in individuals with chronic hepatitis B (CHB) infection. Current non-invasive methods show limited accuracy in detecting occult liver damage, particularly in those with normal

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ALT. This study evaluated serum N-glycan profiles for diagnosing significant liver pathology in treatment-naïve CHB patients across clinical phases.

Materials and Methods:

This cross-sectional study analyzed 626 treatment-naïve CHB patients confirmed by liver biopsy, classified according to 2025 EASL guidelines. Serum N-glycan profiles were determined using DNA sequencer-assisted fluorophore-assisted carbohydrate electrophoresis. Significant liver pathology was defined as inflammation grade $\geq$ G2 and/or fibrosis stage $\geq$ S2. Multivariate logistic regression models were developed and compared with traditional non-invasive markers.

Results:

Among 626 CHB patients, 66.0% had significant inflammation and 58.9% had significant fibrosis. Patients with significant pathology showed characteristic alterations, with elevated P1, P3, P6, P7, P11 peaks and decreased P0, P5, P8, P10 peaks (all $p < 0.0001$). Compared to infection phases, hepatitis phases showed P1 increases of 19.6%/36% and P11 increases of 82.4%/73.4% in HBeAg(+)/HBeAg(-) patients, while P0 decreased by 20.3%/27.6% and P10 decreased by 21.6%/20.3%. In patients with significant pathology (G and/or S $\geq$ 2), P1 increased by 27% on average, reaching 58.7%/48.7% in G4/S4 stages. In ALT-normal patients, particularly HBeAg(+) infection phase, P1 increased by 80.2%/65.8% in G4/S4 stages, with P2 also increasing by 54.1%/45.2%. Multivariate analysis identified P11 as strongest risk factor (OR=3.84, 95%CI: 1.74-8.45, $p=0.0008$), followed by P1 (OR=2.04, 95%CI: 1.57-2.64, $p < 0.0001$) and P7 (OR=1.75, 95%CI: 1.31-2.34, $p=0.0002$), while P2 (OR=0.07, 95%CI: 0.02-0.26, $p < 0.0001$) and P0 (OR=0.30, 95%CI: 0.12-0.79, $p=0.0140$) served as protective factors. The glycomics combined model (AUC=0.876) achieved superior performance outperformed the clinical model (0.818), LSM (0.817), APRI (0.830), and FIB-4 (0.672), with 78.7% sensitivity and 83.2% specificity. The optimized model reached AUC=0.917 with accuracy 84.2%, with 78.7% sensitivity and 94.6% specificity. Both glycol-based models maintained diagnostic capability in ALT-normal patients particularly in HBeAg(+) infection.

Conclusions:

Serum N-glycomics demonstrates promising potential for non-invasive identification of significant liver

pathology in treatment-naïve CHB patients, providing an alternative approach for early treatment decisions, especially in ALT-normal patients with occult liver damage.

Keyword(s) : Chronic hepatitis B; Treatment-naïve; N-glycomics; Liver pathology; Non-invasive diagnosis

1059 - The predictive value of different frailty assessment tools for the prognosis of hospitalized patients with liver cirrhosis

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Background / Hypothesis:

Patients with cirrhosis are often accompanied by frailty. The lack of consensus on the clear definition and assessment tool limits the clinical practice of frailty. This study attempts to analyze the predictive value of six frailty tools for mortality in patients with cirrhosis.

Materials and Methods:

200 patients with cirrhosis were enrolled in this prospective cohort study, which the median age was 55 years (IQR 49-64), and the prevalence of frailty ranged from 16.0% to 52.0%. The ROC curve was used to compare the predictive value of six frailty tools. All six frailty tools could predict the mortality of patients with cirrhosis at 1, 3, 6, and 12 months. The predictive efficacy of LFI, FFP, SPPB, and FRAIL was similar, while CFS and EFS was slightly worse in predicting 1- and 3-month mortality. The predictive efficacy of LFI, FFP, and SPPB was similar, followed by FRAIL, while CFS and EFS were poor in predicting 6-month mortality. LFI and FFP had the best predictive efficacy, followed by FRAIL and SPPB, which CFS and EFS were poor in predicting 12-month mortality. The survival analysis revealed that the 12-month survival rate of the frailty group was significantly lower ($P < 0.05$).

Results:

The predictive efficacy of LFI, FFP, and SPPB was similar, followed by FRAIL, while CFS and EFS were poor in predicting 6-month mortality. LFI and FFP had the best predictive efficacy, followed by FRAIL and SPPB, which CFS and EFS were poor in predicting 12-month mortality. The survival analysis revealed that the 12-month survival rate of the frailty group was significantly lower ($P < 0.05$).

Conclusions: LFI and FFP are superior to other frailty assessment tools in identifying the risk of poor prognosis. The applicability of FRAIL and EFS to mortality prediction was first demonstrated in cirrhosis. All six frailty assessment tools can effectively stratify

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the death risk of liver cirrhosis.

Keyword(s) : frailty, frailty index, prognosis

1060 - Transitional Hepatocytes and Mortality Associated Gene Programs Drive NASH Associated Liver Cancer Revealed by Single Cell Transcriptomics

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Background / Hypothesis: Non-alcoholic fatty liver disease (NAFLD), particularly its progressive form non-alcoholic steatohepatitis (NASH), has become a leading cause of hepatocellular carcinoma (HCC). Mortality from NASH-associated liver cancer (NALC) is disproportionately higher in older adults and in populations from low- and middle-income countries (LMICs), but the molecular basis of this poor prognosis remains unclear. This study aimed to identify transitional hepatocyte states and mortality-associated gene programs that underpin progression from NASH to HCC.

Materials and Methods: Publicly available single-cell RNA sequencing datasets of liver tissue from healthy, NASH, and HCC samples were integrated using the Harmony algorithm to correct for batch effects and preserve rare cell populations. Dimensionality reduction, clustering, and marker-based cell annotation were conducted with Seurat, supported by classical markers and differential expression profiles. Pathway activation was quantified with Vision using curated MSigDB gene sets. CopyKAT was employed to distinguish malignant from non-malignant hepatocytes via inferred copy number variation. Cell-cell communication networks were reconstructed with NicheNet to identify ligand-receptor interactions between hepatocytes, macrophages, and stromal cells.

Results: We identified a population of transitional hepatocytes bridging normal and malignant states. These cells expressed mortality-associated genes including MKI67, PCNA, STMN1, TOP2A, MALAT1, FABP5, CDK1, and HSP90AA1, which have been linked to poor survival and aggressive HCC phenotypes. Metabolic variants such as PNPLA3 and TM6SF2, as well as immune regulators like TREM2 and GDF15,

were also enriched, providing a mechanistic link between metabolic injury, immune suppression, and high mortality risk. Progressive activation of HIF1A signaling suggested hypoxia as a key driver of malignant transformation. Concurrently, macrophages polarized into immunosuppressive TREM2⁺ states, recruited by hepatocyte-derived signals via the CCL15-CCR1 axis, reinforcing a suppressive microenvironment.

Conclusions: Transitional hepatocytes and their mortality-associated gene programs, together with immunosuppressive macrophages, drive the progression of NASH to liver cancer. These findings highlight high-risk molecular targets (MALAT1, TOP2A, PNPLA3, TREM2, GDF15) that may explain the elevated mortality observed in older adults and LMIC populations, and provide potential avenues for early intervention in NASH-associated liver cancer.

Keyword(s) : NASH, hepatocellular carcinoma, single-cell RNA sequencing, mortality, pathology

1061 - Integrative AI Based Multi Omics Approach to Uncover Endothelial Diversity and Predict Cholestatic Liver Disease Across Vulnerable Populations

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Background / Hypothesis: Cholestatic liver diseases disproportionately affect vulnerable populations, including neonates with congenital disorders, women with primary biliary cholangitis or intrahepatic cholestasis of pregnancy, men with primary sclerosing cholangitis, and critically ill patients with sepsis- or drug-induced cholestasis. Despite its clinical importance, the molecular mechanisms underlying cholesterol accumulation and its predictive potential in these populations remain poorly characterized. This study aimed to leverage multi-omics data and AI-driven analytics to uncover key regulators of cholesterol saturation and develop predictive models of cholestatic liver disease, with an emphasis on vulnerable patient groups.

Materials and Methods: We integrated transcriptomic, epigenomic, and proteomic data from human liver tissues and in vitro hepatic models. Unsupervised deep

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learning algorithms were applied to identify molecular profiles associated with cholesterol homeostasis. SHAP and LASSO regression were used for feature selection. A machine learning classifier was trained and validated using clinical datasets stratified by age, sex, and disease subtype, incorporating serum cholesterol, bile acid profiles, and histology.

Results: Distinct molecular signatures were identified across vulnerable groups. Elevated hepatic cholesterol saturation correlated with upregulation of SREBF2 and HMGCR, and downregulation of ABCG5/8. Epigenetic modifications near NR1H3 and CYP7A1 were linked to disrupted bile acid synthesis and cholesterol export. The predictive model demonstrated high diagnostic performance (AUC = 0.93; 95% CI: 0.89–0.96). Stratified analyses highlighted differential molecular signatures in neonates, women with autoimmune or pregnancy-related cholestasis, and men with IBD-associated PSC. Pathway analysis revealed dysregulation of cholesterol biosynthesis, FXR/RXR signaling, and ER stress responses.

Conclusions: Our integrative AI-based multi-omics approach reveals key molecular drivers of hepatic cholesterol saturation and provides a robust predictive tool for cholestatic liver disease. By highlighting mechanisms across vulnerable populations, this study offers novel opportunities for early diagnosis, risk stratification, and targeted therapeutic strategies.

Keyword(s) : artificial intelligence, multi-omics, endothelial heterogeneity, cholestasis, vulnerable populations

1062 - GALNT7 Mediated Glycosylation of TAZ at Serine 307 Promotes Gallbladder Cancer Progression via Stabilization and Feed Forward Activation

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Background / Hypothesis: Gallbladder cancer is a highly aggressive malignancy with limited therapeutic options and poor prognosis. Post-translational modifications,

particularly glycosylation, have emerged as key regulators of tumor progression. We hypothesized that the glycosyltransferase GALNT7 may drive gallbladder cancer progression through modulation of the Hippo pathway effector TAZ.

Materials and Methods: Clinical tissue samples, including gallbladder cancer and adjacent non-tumor tissues, were analyzed using proteomics, immunohistochemistry, and quantitative polymerase chain reaction (qPCR). Functional assays including cell proliferation, migration, and invasion were performed using gallbladder cancer cell lines with genetically modified GALNT7 and TAZ expression. Protein interactions were assessed by co-immunoprecipitation and mass spectrometry. To investigate transcriptional regulation, luciferase reporter assays and chromatin immunoprecipitation followed by qPCR were employed to assess TAZ and TEAD1 binding to the GALNT7 promoter. In vivo tumor growth and metastasis were evaluated using mouse xenograft and lung metastasis models. Peptides targeting glycosylation were synthesized and tested for therapeutic efficacy.

Results: GALNT7 was significantly overexpressed in gallbladder cancer tissues and correlated with advanced tumor stage and poor survival. Silencing GALNT7 reduced cell proliferation, migration, and invasion, while overexpression enhanced these malignant behaviors. Mechanistically, GALNT7 interacted with TAZ and induced its glycosylation at serine 307, which inhibited ubiquitination and prolonged protein stability. This modification facilitated the recruitment of the deubiquitinase USP7, further stabilizing TAZ. Glycosylation-defective TAZ (serine 307 to alanine) failed to restore oncogenic properties even in the presence of GALNT7. Additionally, TAZ and its partner transcription factor TEAD1 transcriptionally upregulated GALNT7, forming a self-amplifying loop. Targeting this glycosylation site with a cell-penetrating peptide effectively suppressed tumor growth and metastasis in preclinical models.

Conclusions: Our findings reveal a novel glycosylation-dependent mechanism that stabilizes TAZ and drives gallbladder cancer progression. The GALNT7-TAZ feed-forward loop represents a promising therapeutic target, and inhibition of TAZ glycosylation may offer a new strategy for treating this aggressive cancer.

Keyword(s) : Gallbladder cancer, GALNT7, TAZ, glycosylation, USP7, therapeutic peptide

1063 - Paraspinal Vein Shunt A Potential Key Factor Leading to Hepatic Myelopathy

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Background / Hypothesis:

Hepatic myelopathy (HM) is a rare neurological manifestation in patients with chronic liver diseases. It has been previously reported to be associated with a surgical or spontaneous portosystemic shunt. However, the specific shunt leading to HM occurrence remains unclear. This study tried to identify a key shunt to explore therapeutic approach for HM.

Materials and Methods:

In this retrospective study, 54 patients with portal hypertension (including 19 patients with HM) were enrolled into the analysis of independent risk factors for HM, who had undergone biochemical testing and abdominal enhanced computed tomography (CT) examinations. Three kinds of venous shunt including the splenorenal shunt (SRS), the gastro-renal shunt (GRS), and the paraspinal vein shunt (PVS) were evaluated using CT examinations.

Results:

The PVS, ascites and hepatic encephalopathy were identified as independent risk factors for patients with HM. PVS exists in wide range of patients with HM, which is not an isolated case. The symptoms in patient with HM were substantially reversed by further embolization of the PVS, while only embolizing the GRS or SRS did not improve the symptoms in patients with HM.

Conclusions:

PVS, as one of independent risk factors of HM, is a key shunt among the extensive collateral circulation for HM occurrence. The embolization of PVS is a potentially treatment strategy for HM.

Keyword(s) : Hepatic Myelopathy, Paraspinal Vein Shunt, Portal Hypertension, Key Shunt, Ascites, Hepatic Encephalopathy

1064 - Analysis of clinical characteristics and risk factors of liver injury in treatment naïve patients with lung cancer

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Background / Hypothesis:

Anti-cancer drug-induced liver injury (DILI) is a

common adverse event in anti-cancer therapy, with rising incidence amid widespread use of novel antineoplastic agents and combination regimens.

Materials and Methods:

A retrospective analysis was conducted on the clinical data of patients diagnosed with primary lung cancer at The First Affiliated Hospital of Nanchang University between January 1, 2023, and December 31, 2023. A total of 709 patients who met the study's inclusion criteria and received standardized anti-cancer therapy were enrolled, and 243 developed anti-cancer DILI. Relevant clinical data was collected, including age, gender, smoking/alcohol consumption history, comorbidities, and a history of pre-existing liver diseases. Statistical analyses were then performed.

Results:

Among the 243 patients with liver injury, the majority (65.02%, 158/243) were elderly (aged ≥ 60 years), with a male predominance (male-to-female ratio: 2.92:1). Liver injury occurred within 8 weeks of initial treatment (71.19%, 173/243). A total of 42 patients met the diagnostic criteria for acute DILI, predominantly presenting with hepatocellular injury (45.24%, 19/42). The remaining 201 cases who did not meet the criteria for acute DILI presented with drug-induced liver biochemical abnormalities, mainly of cholestatic injury (46.77%, 94/201) and mixed injury (46.27%, 93/201), with most showing only mild elevations in liver enzymes. The cure rate of acute DILI was significantly lower than that of drug-induced liver biochemical abnormalities (33.33% vs. 46.77%, $P = 0.036$). 28 patients developed chronic DILI, with cholestatic injury as the main clinical phenotype. Multivariable logistic regression analysis identified pre-existing liver disease as an independent risk factor for anti-cancer DILI (odds ratio [OR] = 1.51, 95% confidence interval [CI]: 1.01-2.26, $P = 0.043$). Compared with immunotherapy alone, chemotherapy combined with immunotherapy (OR = 4.38, 95% CI: 1.28-14.95, $P = 0.018$), chemotherapy combined with targeted therapy (OR = 4.93, 95% CI: 1.40-17.42, $P = 0.013$), and chemotherapy alone (OR = 5.56, 95% CI: 1.53-20.16, $P = 0.009$) were all significantly associated with an increased risk of liver injury.

Conclusions:

A history of pre-existing liver diseases and antineoplastic treatment regimens are independent



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risk factors for DILI in treatment-naïve patients with lung cancer. Acute DILI (especially hepatocellular type) carries higher fatal risk, while cholestatic injury tends to chronize. Close monitoring of liver function (within 8 weeks of initial treatment) and early intervention are essential in clinical practice to mitigate adverse outcomes.

Keyword(s) : Drug-induced liver injury, lung cancer, risk factors, anti-cancer therapy, hepatotoxicity.

1066 - Efficacy of laparoscopic assisted Microwave Ablation for treating giant hepatic hemangioma a retrospective controlled study

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Background / Hypothesis:

Hepatic hemangioma (HH) are usually benign, but surgery is recommended for symptomatic cases or when the tumor diameter is ≥ 10 cm, defined as giant hepatic hemangioma (GHH). Although laparoscopic hepatectomy (LH) is effective, it often involves considerable surgical trauma and high costs. The role of transarterial embolization (TAE) combined with laparoscopic microwave ablation (LMWA) as a minimally invasive alternative has not been well established, and little is known about how tumor location might influence recovery. This study evaluated the safety and effectiveness of TAE+LMWA for GHH, compared its perioperative outcomes with LH, and explored, in patients treated with TAE+LMWA alone, whether tumor location (parenchymal vs. capsular) affects postoperative liver and kidney function.

Materials and Methods:

We retrospectively reviewed 118 patients with GHH treated between November 2014 and October 2024: 60 underwent TAE+LMWA and 58 underwent LH. Perioperative outcomes were compared between the two groups. We also analyzed 42 additional patients with HH who underwent TAE+LMWA alone and were discharged in September 2025, dividing them into parenchymal (n=19) and capsular (n=23) groups to assess postoperative hepatic and renal function.

Results:

Compared with the LH group, patients in the TAE+LMWA group had shorter operative times, less blood loss, lower hospitalization costs, and faster recovery ($p < 0.05$). Cure rates were similar between groups ($p > 0.05$). During follow-up, the ablation zone shrank significantly in the TAE+LMWA group. Among

the 42 patients treated with TAE+LMWA alone, liver and kidney function markers (ALT, AST, Cr) did not differ significantly between the parenchymal and capsular groups ($p > 0.05$). However, in subgroup analysis by tumor size, patients with GHH (n=21) had significantly higher TB and DB levels compared with those with large hepatic hemangioma (LHH, n=21; $p < 0.05$).

Conclusions:

TAE combined with LMWA appears to be a safe, cost-effective, and minimally invasive option for managing GHH. Postoperative recovery is influenced mainly by tumor size rather than tumor location.

Keyword(s) : Hepatic hemangioma, Microwave ablation, Laparoscopy, Transarterial embolization, Outcome comparison, Safety and effectiveness

1068 - Gut Microbiota Derived 3 Methylxanthine Regulating T Cell Immune Response to Inhibit Viral Replication and Liver Injury in Chronic Hepatitis B comorbid with Metabolic Dysfunction associated Fatty Liver Disease

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Objective:

The incidence of chronic hepatitis B (CHB) complicated by metabolic dysfunction-associated steatosis liver disease (MASLD) is increasing, posing a significant threat of progressive liver injury/fibrosis, yet underlying mechanisms remain unclear. Gut microbiota (GM) and metabolites are linked to CHB/MASLD progression, but their role in CHB-MASLD is undefined. This study explored GM's role and mechanisms in CHB-MASLD.

Methods:

CHB-MASLD models were established by hydrodynamic injection of pAAV/HBV1.2 into mice fed a choline-deficient, L-amino acid-defined, high-fat diet (CDAHFD). GM and metabolites were analyzed via 16S rRNA sequencing and untargeted metabolomics; intrahepatic T cell responses by flow cytometry.

Results:

Compared with single CHB or MASLD models, CHB-MASLD mice exhibited significantly exacerbated liver

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inflammation and fibrosis, evidenced by marked elevations in hepatic enzymes (ALT, AST), proinflammatory cytokines (TNF α , IL-1 β , IL-6), and fibrosis markers (α -SMA, Collagen I). Paradoxically, serum HBsAg, HBeAg, and HBV DNA levels were substantially reduced in CHB-MASLD mice, suggesting suppressed viral replication despite severe liver injury. Further analyses revealed reduced gut microbial α -diversity and altered community structure in CHB-MASLD mice. Notably, the abundance of the opportunistic pathogen Parabacteroides was significantly increased, accompanied by a marked decrease in the microbial metabolite 3-methylxanthine (3-Mx). 3-Mx levels were negatively correlated with both Parabacteroides abundance and ALT/AST elevations. Concomitantly, CHB-MASLD mice showed enhanced intrahepatic CD4⁺ Granzyme B⁺ T cell responses, reflecting heightened cytotoxic activity. Supplementation with 3-Mx in CHB-MASLD mice delayed HBsAg/HBeAg clearance, reduced ALT/AST levels, and promoted intrahepatic CD4⁺ T cell exhaustion (upregulation of CTLA4 and PD1) while suppressing effector phenotypes (Granzyme B). These findings highlight 3-Mx as a key modulator of viral control and liver inflammation in CHB-MASLD.

Conclusion:

Increased Parabacteroides in CHB-MASLD depletes 3-Mx, thereby promoting intrahepatic CD4⁺ Granzyme B⁺ T-cell-mediated viral clearance but exacerbating liver injury. This study provides new insights into the GM-driven pathogenesis of CHB-MASLD and identifies potential therapeutic targets for this emerging comorbidity.

Keyword(s) : Gut Microbiota, 3-Methylxanthine, Chronic Hepatitis B, Metabolic Dysfunction-associated Fatty Liver Disease, T-Cell Immune Response

1070 - Analysis of Epidemiological Characteristics and Risk Factors of Liver Function Impairment Among Middle Aged and Older Community Residents in Beijing

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Background / Hypothesis:

With socioeconomic development and changes in

lifestyle, non-viral liver diseases have become increasingly prominent and are now common liver conditions in China. Liver function impairment is a critical marker of liver disease progression, and its dynamic changes can reflect the evolution of liver health status. However, there is a lack of dynamic observational data on liver function impairment, particularly regarding its trends and related factors among middle-aged and older community-dwelling adults.

Materials and Methods:

This study utilized data from the Beijing Air Pollution and Population Health Cohort. Participants aged 45-75 years at baseline who provided blood samples during surveys in 2017, 2019, and 2023 were included. Liver function impairment was defined as AST >35 U/L or ALT >40 U/L for females, and AST >40 U/L or ALT >50 U/L for males. The incidence and changes in liver function impairment were analyzed. Logistic regression was employed to identify potential risk factors associated with liver function impairment.

Results:

A total of 1,906 community residents were included, of whom 1,119 (58.71%) were female, with a mean age of 61.85 years. The prevalence rates of liver function impairment in 2017, 2019, and 2023 were 7.66%, 7.08%, and 8.55%, respectively. From 2017 to 2023, the proportions of participants who never experienced, or experienced 1, 2, or 3 episodes of liver function impairment were 82.53%, 13.01%, 3.10%, and 1.36%, respectively. Among the 146 individuals with liver function impairment in 2017, 96 (65.75%) returned to normal levels by 2019. Similarly, among the 135 individuals with impairment in 2019, 91 (67.41%) normalized by 2023. Logistic regression analysis identified that liver function impairment was associated with age (55-64 years: OR=0.64, 95% CI [0.42-0.99]; 65-75 years: OR=0.44, 95% CI [0.27-0.71]), obesity (OR=1.46, 95% CI [1.05-2.02]), diabetes (OR=1.34, 95% CI [1.00-1.79]), and coronary heart disease (OR=1.53, 95% CI [1.05-2.23]). No significant associations were found with gender, educational level, smoking, alcohol consumption, high-intensity exercise or work, sleep duration, sedentary time, hypertension, allergic diseases, stroke, or tumors.

Conclusions:

The overall prevalence of liver function impairment among middle-aged and older adults in Beijing remained stable at approximately 8%, with a low proportion of persistent impairment. At the individual

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level, liver function status exhibited considerable variability over time, with about two-thirds of individuals with impairment returning to normal within a follow-up period. Factors potentially associated with liver function impairment include age, obesity, diabetes, and coronary heart disease.

Keyword(s) : Liver Function Impairment; Epidemiological Characteristics; Risk Factors

1074 - Mediating role of BMI in alcohol linked liver enzyme elevation among adults at a tertiary care hospital in South India

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Background / Hypothesis:

Excessive alcohol consumption is a major risk factor for liver disease, with significant variations in its impact across populations. Unlike prior studies that examine alcohol-induced liver dysfunction and obesity-related liver conditions in isolation, our research integrates these aspects to provide a comprehensive understanding of their interaction. Body mass index (BMI) has been identified as a potential mediator in alcohol-related liver damage. This study aims to examine the association between alcohol consumption and liver function and to explore the mediating role of BMI in a population from India, where both are rising public health concerns.

Materials and Methods:

A cross-sectional study was conducted using data from adult participants. The study included all eligible adults aged 30 years and above who attended the Annual Health Checkup Clinic and underwent routine liver function testing as part of their check-up. Liver function was assessed using serum levels of gamma-glutamyl transferase (GGT), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). Alcohol consumption was self-reported, and BMI was calculated from height and weight measurements. Multiple linear regression models were used to evaluate the relationship between alcohol consumption and liver enzymes, while adjusting for BMI as a mediator. Statistical significance was set at $p < 0.05$.

Results:

The study included a total of 706 participants, comprising 335 (47.5%) males with a mean age of 47.9 years (SD: 10.7). Alcohol use was noted in 37 (11.4%) participants, predominantly among males (36, 22.4%). In the multivariable analysis, alcohol consumption showed a significant association with elevated liver enzyme levels, including GGT ($\beta = 5.9$), ALT ($\beta = 6.2$), and AST ($\beta = 6.1$). BMI was found to mediate this relationship, with individuals having higher BMI showing a greater increase in liver enzyme levels in response to alcohol consumption. However, no significant association was observed for ALP. BMI also independently correlated with higher levels of GGT, ALT, and AST.

Conclusions:

The study findings suggest that individuals with higher BMI are more susceptible to alcohol-related liver damage, as evidenced by elevated levels of GGT, ALT, and AST. These results underscore the need for integrated public health strategies that address both alcohol consumption and obesity to mitigate the rising burden of liver disease, particularly in South Asia. Clinicians should consider both BMI and alcohol consumption when assessing liver function, and interventions targeting both lifestyle factors may have significant public health benefits.

Keyword(s) : Liver enzymes, Alcohol, Body Mass Index, Mediation Analysis

1077 - Performance of BAVENO VI and BAVENO VII Criteria for Excluding High Risk Esophageal Varices in Compensated Cirrhosis with Hepatocellular Carcinoma

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Background / Hypothesis:

High-risk esophageal varices (HRV) are commonly found in patients with hepatocellular carcinoma (HCC). The BAVENO VI criteria, which use liver stiffness measurement (LSM) < 20 kPa and platelet count $> 150 \times 10^9/L$, have been applied for risk stratification. Moreover, the BAVENO VII criteria, which include spleen stiffness measurement (SSM) ≤ 40 kPa, further improve the identification of patients at risk for HRV. However, the use of these criteria in patients with HCC remains limited.

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Objective:

To evaluate the diagnostic performance of BAVENO VI and BAVENO VII in ruling out HRV in compensated cirrhosis with HCC.

Materials and Methods:

This cross-sectional study included patients with compensated liver cirrhosis and HCC who had not received non-selective beta blockers or endoscopic band ligation. Liver stiffness measurement was performed using a 50 Hz shear wave frequency, and spleen stiffness measurements were performed using a 100 Hz shear wave frequency. Endoscopic findings were used as the reference standard.

Results:

Among the 44 patients included in the study, the mean age was 61 years, and the majority were male (84%). Hepatitis C virus infection was the most common etiology (54%). The distribution of Barcelona Clinic Liver Cancer (BCLC) stages 0, A, B, and C were represented by 2 (4.5%), 16 (36.4%), 18 (40.9%), and 8 (18.2%) patients, respectively. Most patients (84.1%) were treatment-naïve for HCC at the time of enrollment. HRV were identified in 19 patients (43.2%). The BAVENO VI criteria demonstrated the best diagnostic performance, with a sensitivity of 100% (95% CI: 82.4–100%), a negative predictive value of 100% (95% CI: 63.1–100%), an endoscopy-sparing rate of 18.2%, and no cases of HRV were missed (HRV miss rate of 0%). BAVENO VII and SSM ≤ 40 kPa were identical in diagnostic performance, with a sensitivity of 57.9% (95% CI: 33.5–79.7%), a negative predictive value of 70.4% (95% CI: 49.8–86.2%), and associated with a significantly higher endoscopy-sparing rate of 43.2% compared to the BAVENO VI criteria ($p = 0.02$). However, these algorithms resulted in a significantly higher HRV miss rate of 42.1% ($p < 0.01$).

Conclusions:

The BAVENO VI criteria demonstrated excellent performance in ruling out HRV in patients with compensated cirrhosis and HCC. The addition of SSM did not enhance diagnostic performance in this population. Further studies are needed to validate the applicability of these criteria across different etiologies of cirrhosis and various stages of HCC.

Keyword(s) : Esophageal varices; Hepatocellular carcinoma; Liver stiffness; Spleen stiffness; BAVENO VI; BAVENO VII; Risk stratification.

1078 - "When Treatment Triggers Disease Hepatic Abscess as a Paradoxical Reaction to Antitubercular Therapy"

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Background / Hypothesis:

Paradoxical reactions during antitubercular therapy (ATT) represent clinical or radiological worsening of existing lesions or the appearance of new ones after initial improvement. Although more often observed in patients with immune dysfunction, such responses can also occur in those without identifiable immunosuppression. Hepatic abscess as a paradoxical manifestation of tuberculosis (TB) is exceedingly rare and poses significant diagnostic and therapeutic challenges.

Materials and Methods:

A 55-year-old male with sputum and GeneXpert-confirmed pulmonary tuberculosis was treated with ATT. After seven weeks of therapy, he developed jaundice, high-grade fever, abdominal pain, and shortness of breath. The diagnostic evaluation included biochemical tests, imaging studies, pleural fluid analysis, and a liver biopsy. ATT was temporarily discontinued due to drug-induced liver injury and replaced with supportive therapy. Once liver function improved, ATT was cautiously reintroduced in conjunction with corticosteroids.

Results:

Investigations revealed elevated liver enzymes, hyperbilirubinemia, and a large hepatic abscess associated with ascites and right-sided pleural effusion. Microbiological studies, including bacterial culture, GeneXpert, and PCR for Mycobacterium tuberculosis from the abscess aspirate, were negative. Liver biopsy demonstrated granulomatous hepatitis consistent with tuberculosis. Following gradual reintroduction of ATT and corticosteroid therapy, the patient became afebrile, regained appetite, and showed radiological resolution of the abscess with normalization of liver function.

Conclusions:

This case demonstrates a rare hepatic manifestation of tuberculosis presenting as a paradoxical reaction during ATT. Recognizing paradoxical responses is crucial to distinguishing them from treatment failure or drug resistance. Careful monitoring, continuation, or cautious reintroduction of ATT, and adjunctive corticosteroid therapy can result in favorable clinical outcomes.



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Keyword(s) : Paradoxical reaction; Hepatic abscess; Tuberculosis; Granulomatous hepatitis; Anti-tubercular therapy

1081 - Risk factors and outcomes of carbapenem resistant enterobacteriaceae infection in liver transplant patients a systematic review and meta analysis

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Background / Hypothesis:

Infectious complications are the major source of mortality following liver transplantation. Over the past decade like Carbapenem Resistant Enterobacteriaceae (CRE) has-infection have evolved as a serious healthcare problem. The aim of this study was to analyze the risk factors for CRE infection and their outcomes in liver transplant patients following liver transplantation.

Materials and Methods:

Medical databases like PubMed, Embase, and Google Scholar databases were systematically searched until April 2025. Any observational studies reported on risk factors and outcomes of CRE-infection among adult liver transplant patients were included. The risk factors studied were pre-transplant MELD score, pre-transplant CRE colonization, pre-transplant ICU stay, HCC, undergoing re-transplantation, combined transplantation or re-operation, intraoperative bleeding, post-transplant need of prolonged mechanical ventilation (MV), renal replacement therapy (RRT), post-transplant CRE colonization and biliary complications. Likewise, outcomes like 6-months survival, 1-year survival and length of hospital stay were also analyzed.

Results:

Eleven studies (n=3927) were analyzed. Pre-transplant MELD score (OR=2.92, 95% C.I.= 1.81-4.70, p<0.001), pre-transplant CRE colonization (OR=4.86, 95% C.I.=1.68-14.06, p<0.001), undergoing re-transplantation (OR=2.73, 95% C.I.=1.23-6.04, p=0.01), combined transplantation (OR=2.52, 95% C.I.=1.63-3.91, p<0.001) or re-operation (OR=3.68, 95% C.I.=2.09-

6.48, p<0.001), significant intraoperative bleeding (OR=2.06, 95% C.I.=1.11-3.84, p=0.02), post-transplant need of prolonged MV (OR=3.95, 95% C.I.=3.06-5.09, p<0.001), RRT (OR=3.91, 95% C.I.=2.32-6.60, p<0.001) and biliary complications (OR=2.24, 95% C.I.=1.13-4.44, p=0.02) were associated with increased risk of CRE-infection. However, Pre-transplant ICU stay (OR=1.49, 95% C.I.=0.69-3.20, p=0.31), HCC (OR=0.88, 95% C.I.=0.70-1.12, p=0.30) and post-transplant CRE colonization (OR=2.69, 95% C.I.=0.88-8.25, p=0.08) were not significantly associated with CRE-infection. Likewise, patients with CRE-infection had significantly lower 6-months (OR=0.12, 95% C.I.=0.06-0.24, p<0.001) and 1-year survival (OR=0.09, 95% C.I.=0.06-0.14, p<0.001) and were likely to have a longer hospital stay (Hedge's g=2.00, 95% C.I.=0.73-3.28, p<0.001).

Conclusions:

This systemic review and meta-analysis found several risk factors like higher pre transplant MELD score, pre transplant CRE colonization, re-transplant, combined transplant or reoperation, intraoperative bleeding, prolonged MV, RRT and biliary complications for CRE infection after liver's transplantation and CRE infection was associated with poor outcomes when compared to no CRE infection.

Keyword(s) : Carbapenem resistant enterobacteriaceae, Liver transplantation

1082 - Prediction of Hepatic Functional Reserve Using a Novel "Severity Index" on Hepatobiliary Phase Gadoteric Acid-Enhanced MRI

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Background / Hypothesis:

Accurate evaluation of hepatic functional reserve is crucial for risk stratification in chronic liver disease (CLD). Existing biochemical scores such as Child-Pugh,

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MELD-Na, ALBI, and FIB-4 provide systemic information but fail to capture spatial or transporter-level hepatic function. We hypothesized that a novel MRI-derived biomarker—the “Severity Index”—which integrates parenchymal uptake and vascular retention on hepatobiliary phase (HBP) gadoxetic acid-enhanced MRI, could noninvasively predict hepatic functional reserve across disease stages.

Materials and Methods:

This retrospective study included 193 individuals (122 CLD, 71 controls) who underwent 1.5T gadoxetic acid-enhanced liver MRI between 2021 and 2023. The Severity Index was defined as (Aortic SI × Portal Vein SI) / Liver SI, measured on 20-minute HBP images. Group comparisons were performed using the Mann-Whitney U test, correlations with MELD-Na, ALBI, and FIB-4 were analyzed via Spearman’s correlation, and diagnostic accuracy was assessed with ROC analysis. Intra- and inter-reader reproducibility were evaluated using the intraclass correlation coefficient (ICC).

Results:

The Severity Index was significantly higher in CLD patients than in controls (median 162.0 vs. 90.6; $p < 0.001$) and effectively differentiated decompensated from compensated disease (AUC = 0.848). It also identified subclinical dysfunction in clinically compensated patients (AUC = 0.800; sensitivity = 90.1%). The index correlated strongly with MELD-Na ($r = 0.503$, $p < 0.001$) and moderately with ALBI ($r = 0.389$, $p < 0.001$), but not with FIB-4 ($p = 0.224$). Reproducibility was excellent (intra-reader ICC = 0.961; inter-reader ICC = 0.863).

Conclusions:

The Severity Index provides a physiologically grounded, reproducible, and noninvasive marker of hepatic functional reserve. By integrating both hepatocellular uptake and vascular clearance, it complements traditional biochemical scores, enables early detection of subclinical dysfunction, and offers a promising imaging biomarker for precision risk stratification in advanced chronic liver disease.

Keyword(s) : Chronic liver disease; hepatobiliary phase

MRI; gadoxetic acid; liver function; Severity Index; MELD-Na; ALBI; noninvasive biomarker

1083 - Functional Liver Imaging Score on Gadoxetic Acid-Enhanced MRI Correlation with MELD Na ALBI FIB 4 and Clinical Status in Chronic Liver Disease

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Background / Hypothesis:

Accurate, noninvasive assessment of hepatic functional reserve is crucial in CLD. Biochemical scores (MELD-Na, ALBI, FIB-4) may miss spatial and transporter-level dysfunction. We hypothesized that the semi-quantitative Functional Liver Imaging Score (FLIS)—integrating hepatobiliary phase parenchymal enhancement, biliary excretion, and portal-vein signal on gadoxetic acid-enhanced MRI—correlates with liver function indices and clinical decompensation and shows high reproducibility across readers.

Materials and Methods:

This single-center retrospective study included 107 adults with CLD who underwent 1.5T gadoxetic acid-enhanced liver MRI (20-min hepatobiliary phase) between 2021–2024. Three radiologists (25, 8, and 6 years’ experience) independently scored FLIS (0–6) blinded to clinical data; one reader repeated scoring after one month. Correlations between FLIS and MELD-Na, ALBI, and FIB-4 were analyzed using Spearman’s rho. Diagnostic performance was evaluated by ROC analyses for ALBI grade 3, MELD-Na <15, and decompensation status. Reproducibility was assessed via two-way random, absolute-agreement ICCs.

Results:

Mean age was 52.4 ± 10.8 years; 67.3% male. Etiologies

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included HBV (59.8%), autoimmune hepatitis (10.3%), HCV (5.6%), cryptogenic (5.6%), PBC (1.9%), and others (16.8%). FLIS demonstrated strong negative correlations with MELD-Na ($r = -0.780, -0.780, -0.816$; all $p < 0.001$) and ALBI ($r = -0.643, -0.644, -0.665$; all $p < 0.001$); correlations with FIB-4 were weaker ($r = -0.235$ to -0.283 ; $p = 0.003-0.015$). For identifying ALBI grade 3, $FLIS \leq 3$ yielded $AUC = 0.939-0.943$, sensitivities 0.75–0.85, specificities 0.931–0.943. For predicting $MELD-Na < 15$, $FLIS \geq 5$ achieved $AUC = 0.903-0.918$, sensitivities 0.875–0.906, specificities 0.800–0.853. For distinguishing decompensated from compensated disease, $FLIS \leq 4$ produced $AUC = 0.829-0.842$. Reproducibility was excellent: intra-observer $ICC = 0.96$ (95%CI 0.94–0.98); inter-observer ICCs 0.96–0.97.

Conclusions:

FLIS on gadoxetic acid-enhanced MRI strongly correlates with MELD-Na and ALBI, shows high diagnostic accuracy for adverse clinical strata, and demonstrates excellent reproducibility. As a fast, cost-efficient, and vendor-agnostic visual score, FLIS can complement biochemical indices for noninvasive functional assessment and risk stratification in CLD and cirrhosis.

Keyword(s) : Chronic liver disease; hepatobiliary phase MRI; gadoxetic acid; FLIS; MELD-Na; ALBI; liver function; decompensation

1084 - Long Term Virological Response Dynamics and Clinical Outcomes in HBV Related Cirrhosis A Real World Cohort Study

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Background / Hypothesis:

The long-term prognostic value of dynamic virological response in HBV-related cirrhosis receiving nucleos(t)ide analogue (NAs) therapy remains inadequately characterized. This study aimed to evaluate its association with clinical outcomes, with stratification by baseline cirrhosis compensation status.

Materials and Methods:

This was a retrospective-prospective cohort study enrolled HBV related cirrhotic patients receiving NAs

treatment from January 2009 to December 2019.

According to the serum HBV DNA level during 2 years of follow-up, The patients were classified into complete virological response (CVR) or partial virological response (PVR) groups. Patients in the CVR group were stratified into maintained virological response (MVR) or virologic breakthrough (VBT) groups based on their dynamic HBV DNA levels throughout the extended follow-up. The primary endpoint were HCC, liver failure (LF) or liver-related death. Secondary endpoints were recompensation or progression to decompensation.

Results:

Among 1,869 patients followed for a median of 7 years, MVR (65.4%) was associated with the most favorable outcomes. In decompensated cirrhosis, the MVR group demonstrated a slow accumulation of HCC risk, with a 10-year cumulative incidence of 25.7%. In contrast, the PVR and VBT groups showed early and sustained elevations in risk, reaching 32.2% and 53.2% at 10 years, respectively ($P < 0.001$ for overall difference). A similar pattern was observed for liver failure and liver-related mortality, with the MVR group consistently exhibiting the lowest risk profile throughout the follow-up (all $P < 0.001$). Furthermore, the MVR group showed the highest rate of clinical recompensation (34.2% vs 22.6% in the VBT group, $P < 0.001$). Among compensated patients, the MVR group demonstrated a significantly lower 10-year HCC risk (14.2% vs 33.6%; $P < 0.001$) and the lowest rate of disease progression to decompensation (16.1% vs 37.0%; $P < 0.001$) compared to patients with suboptimal response. In addition, Within the MVR cohort, HBsAg levels < 100 IU/mL identified a subgroup with a substantially lower HCC risk.

Conclusions:

MVR is crucial for favorable prognosis in HBV-related cirrhosis, especially in patients with HBsAg level less 100 IU/ml. However, about 26.5% of long-term NAs antiviral therapy patients have VBT and poor outcomes. These findings not only highlight the critical role of long-term viral suppression in optimizing clinical prognosis, but also underscore the necessity of long-term strictly monitoring for VBT after achieving CVR.

Keyword(s) : Hepatitis B virus; Cirrhosis; Outcome; Hepatocellular carcinoma; Liver failure; mortality ; Recompensation; Decompensation; Hepatitis B surface antigens ;Antiviral therapy;Cohort

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1086 - COVID 19 Pandemic's Impact on the Global Disease Burden of HBV related Hepatocellular Carcinoma in the Elderly

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Background / Hypothesis:

With the advancing aging population, the disease burden in the elderly has become a critical public health issue. This study utilized data from the Global Burden of Disease Study (GBD) 2021 to evaluate the global, regional, and national disease burden of hepatitis B virus (HBV)-related hepatocellular carcinoma (HCC) in the elderly, with particular focus on trend changes before and after the COVID-19 pandemic. The study further analyzed differences across Socio-Demographic Index (SDI) categories and between genders to provide evidence for public health policymaking.

Materials and Methods:

Based on GBD 2021 data, we analyzed age-standardized incidence and mortality rates of HBV-related HCC in the elderly from 1990 to 2021, with emphasis on comparing trends between 2019 and 2021. Joinpoint regression was used to assess trend changes, while age-period-cohort (APC) analysis examined the effects of age, period, and birth cohort. Decomposition analysis quantified contributions of population aging, epidemiological changes, and population growth to disease burden changes. The association between age-standardized rates and estimated annual percentage change was explored using nonlinear regression models.

Results:

From 1990 to 2021, the global age-standardized incidence rate (ASIR) and mortality rate (ASDR) of HBV-related HCC in the elderly showed an overall declining trend, though with significant regional heterogeneity. African regions maintained higher rates, while some American regions had lower rates. After the COVID-19 pandemic, the global decline rate accelerated, though some countries experienced rising trends. Males consistently showed higher ASRs than females across most regions, with a faster post-pandemic decline in male rates. Decomposition analysis indicated epidemiological changes were the primary drivers of incidence and mortality trends, while aging's

contribution to incidence increased after 2019. APC analysis revealed that HCC risk initially increased then decreased with advancing age, with significant differences across birth cohorts.

Conclusions:

Despite recent progress in global HBV-related HCC control in the elderly, evidenced by declining overall incidence and mortality rates, significant regional disparities persist, with elderly males bearing a heavier disease burden. The post-pandemic period has seen accelerated declines in most regions but rising trends in some countries. Future prevention and control strategies should prioritize elderly populations, particularly males, through enhanced health education, screening, and personalized treatment, while optimizing public health measures and establishing comprehensive surveillance systems to effectively reduce the global disease burden.

Keyword(s) : Global Burden of Disease;Hepatitis B virus;Hepatocellular carcinoma;COVID-19

1087 - Bibliometric Analysis and Research Trends of Specialty Theses on Cirrhosis in Turkey

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Background / Hypothesis:

Liver cirrhosis is a major global health concern, caused by factors such as obesity, non-alcoholic fatty liver disease, alcohol abuse, viral hepatitis B/C, autoimmune and cholestatic diseases, as well as iron or copper overload(1). Clinical management varies by etiology and disease stage. This study aims to analyze cirrhosis-related theses in Turkey to identify research trends.

Materials and Methods:A descriptive retrospective study was conducted by searching the Higher Education Council (YÖK) National Thesis Center database for theses with "Cirrhosis" in the title. Ninety-eight accessible theses published between 2011 and 2025 were evaluated. Parameters included publication year, language, institution, department, advisor academic title, publication status, time to publication, journal index, and citation count.

Results:



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Most theses (98.97%, n=97) were written in Turkish; only one was in English. The peak year for theses was 2022 (n=14, 14.28%), followed by 2020 (n=11, 11.22%), 2014 (n=10, 10.20%), and 2024 (n=9, 9.18%) Figure 1. Health Sciences University produced the highest number (n=16, 16.32%), followed by Ege, Atatürk, and Harran Universities Figure 2. The majority of theses were from Gastroenterology (57.14%, n=56) and Internal Medicine (19.38%, n=19) departments Figure 3. Regarding advisors, 58.16% (n=57) were professors, 31.63% (n=31) associate professors, and 10.20% (n=10) assistant professors. Only 23.46% (n=23) of theses were published as articles, with a median time to publication of 2 years (range 1–12). Published articles appeared mostly in SCI/SCI-E (34.78%, n=8), followed by other international (26.08%, n=6), national (21.73%, n=5), and ESCI-indexed journals (17.3%, n=4). Median citation count was 2 (range 0–78). Statistical analysis showed no significant association between thesis year, department, institution, or advisor title and publication status ($p > 0.05$), but a significant link was found between thesis type and publication ($p=0.008$), with all subspecialty theses (n=3) published.

Conclusions:The fact that the highest number of theses were submitted in 2022 indicates that academic interest in the field of cirrhosis has increased in recent years. The fact that the majority of theses were submitted at Health Sciences Universities demonstrates a strong research infrastructure. The fact that most advisors are professors reflects the influence of experienced academics in the studies. The low rate of theses in English limits international visibility. Only 23.46% of theses were published, suggesting the need for support during the publication process.

Keyword(s) : bibliometric, cirrhosis, academy

1088 - The cGAS STING IFITM3 Axis Drives Monocyte Phenotypic Shift and Immune Dysregulation in Acute on Chronic Liver Failure

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Background / Hypothesis:Acute-on-chronic liver failure (ACLF) is a lethal syndrome characterized by intense systemic inflammation. Monocytes are pivotal in ACLF immunopathology, particularly the pro-inflammatory CD16+ subset, but their upstream drivers remain elusive. Our preliminary single-cell RNA sequencing of

ACLF patients identified IFITM3 as a hub gene upregulated during both CD16+ monocyte differentiation and disease progression. We hypothesize that hepatocyte-derived DAMPs activate the cGAS-STING pathway in monocytes, leading to IFITM3 upregulation, which subsequently drives CD16+ phenotypic shift and enhances hepatic chemotaxis, thereby exacerbating immune imbalance in ACLF.

Materials and Methods:In silico, we re-analyzed single-cell data from HBV-ACLF patients to validate IFITM3's central role. In vitro, IFITM3 was knocked down or overexpressed in human monocyte lines (THP-1, Mono Mac 6). Phenotype (CD14/CD16) was assessed by flow cytometry, and chemotaxis towards liver sinusoid endothelial cell (LSEC) or hepatocyte conditioned media was evaluated via Transwell assays. Key chemokine receptors (CCR2, CX3CR1) and ligands (CCL2, CX3CL1) were measured by qPCR, flow cytometry, and ELISA. In vivo, a preventive strategy using Ifitm3-shRNA lentivirus was employed in a CCl₄-induced ACLF mouse model to assess its impact on monocyte subsets, liver injury, and inflammation.

Results:Our single-cell analysis confirmed IFITM3 as a core gene during monocyte transition to CD16+ subset in progressive ACLF. In vitro, IFITM3 knockdown reduced the CD16+ population, while its overexpression promoted it. Furthermore, IFITM3 knockdown significantly impaired monocyte chemotaxis towards LSEC and hepatocyte signals, correlating with reduced surface expression of CCR2 and CX3CR1. Preliminary data successfully demonstrated efficient knockdown and overexpression of IFITM3 in monocytic cells.

Conclusions:The cGAS-STING-IFITM3 axis is a novel signaling pathway driving CD16+ monocyte skewing and enhancing their liver homing in ACLF. IFITM3 represents a potential key node linking initial injury to sustained immune dysregulation, making it a promising therapeutic target for mitigating inflammation in ACLF.

Keyword(s) : Acute-on-Chronic Liver Failure; Monocyte; IFITM3; cGAS-STING; Immune Dysregulation

1089 - CEP290 a primary cilium related gene as the positive feedback regulator of Nrf2 that modulates liver cancer cells ferroptosis

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Background / Hypothesis:

Though dysfunction of primary cilium is associated with cancers, the molecular basis by which activates signal pathway against ferroptosis are still ambiguous.

Materials and Methods:

Here, in The Cancer Genome Atlas Liver Hepatocellular Carcinoma(TCGA-LIHC) expression data, we utilized weighted gene co-expression analysis (WGCNA) combined with molecular subtype clustering model to prioritize potential novel oncogenes, and validated them in vitro and in vivo.

Results:

We found that the expression of a centrosomal protein 290 (CEP290) increased within liver cancer cells and tissues, and its repression promoted ciliogenesis, iron and malondialdehyde (MDA) accumulation. Mechanically, CEP290 binds nuclear factor erythroid 2-related factor 2 (Nrf2) to displace kelch like ECH associated protein 1 (Keap1), thus inhibiting Nrf2 ubiquitination, stabilizing Nrf2 protein and activating Nrf2 signal transduction. Interestingly, expression of CEP290 was also regulated by recruiting Nrf2 to its promoter. The directly interplay between CEP290 and Nrf2 formed a positive feedback loop. Moreover, inhibition of Nrf2 significantly enhanced ferroptotic effects of CEP290 knockout in vivo.

Conclusions:The above results shed more lights on the important part of CEP290 in connecting ciliogenesis with ferroptosis, and present an important implication for liver cancer treatment.

Keyword(s) : Ciliogenesis, CEP290, Nrf2, ubiquitination, ferroptosis

1090 - Hermansky Pudlak syndrome (HPS)

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Background / Hypothesis:

Hermansky-Pudlak syndrome (HPS) is a rare, autosomal recessive disorder characterized by visual impairment, decreased pigmentation with nystagmus

(oculocutaneous albinism), and blood platelet dysfunction accompanied by prolonged bleeding. Hermansky-Pudlak syndrome (HPS) was diagnosed in a patient presenting with nausea and vomiting.

Materials and Methods:

Case: A 23-year-old female patient presented with nausea and vomiting. Endoscopy revealed no gross pathology to explain the nausea and vomiting. The patient's history revealed HPS. Because there were no bowel (colitis) complaints, a colonoscopy was not recommended.

Results:

The patient was referred to a specialized center for hematology, genetics, and neurology follow-up.

Conclusions:HPS is a hereditary disease with autosomal recessive transmission. Numerous genetic tests are available for diagnosis. Some individuals with this disease may also present with lung fibrosis, inflammatory bowel disease, immunodeficiency, or abnormal deposition of a fat-like substance (seroid lipofuscin) in various body tissues. Hermansky-Pudlak syndrome should be considered in cases characterized by inflammatory bowel disease, albinism, and nystagmus.

Keyword(s) :

1092 - Triple A (Allgrove) Syndrome

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Background / Hypothesis:

Allgrove syndrome (AS), or 3A syndrome (AAA), is characterized by the triad of adrenocorticotrophic hormone (ACTH)-resistant adrenal insufficiency, alacrimia, and achalasia, accompanied by progressive neurological impairment with or without mild mental retardation. A rare case of Allgrove (3A) Syndrome is presented here.

Keywords: Triple A syndrome, 3A syndrome, Allgrove syndrome

Materials and Methods:

Case: The patient was a 30-year-old female living in Oman. Endoscopy revealed signs of achalasia and adrenal insufficiency during examinations performed for difficulty swallowing. Initially, endoscopy was

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performed for achalasia, followed by a bougie dilation and then a Heller myotomy. The difficulty swallowing persisted, albeit mildly.

Results:

Treatment was initiated for adrenocortical insufficiency.

Conclusions: Triple A, or Allgrove syndrome, is a hereditary syndrome characterized primarily by achalasia, alacrima, and adrenocorticotropin (ACTH) resistance-addisonianism. In cases of achalasia, triple A syndrome should not be overlooked, and adrenal insufficiency should be investigated.

Keyword(s) :

1093 - Towards HCV Elimination Time for Clinical and Laboratory Collaboration

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Background / Hypothesis:

Achieving HCV elimination requires identifying and addressing diagnostic gaps. This study aimed to detect undiagnosed HCV cases, evaluate the level of clinical awareness in the diagnostic process, and explore strategies to reduce the number of undiagnosed patients.

Materials and Methods:

Anti-HCV-positive patients identified at our hospital between January 1, 2022, and December 31, 2023, were retrospectively reviewed. Patients who tested anti-HCV-positive but had no HCV RNA test ordered were contacted and invited to the hospital. Those who attended were subsequently tested for HCV RNA.

Results:

A total of 78,769 anti-HCV tests were performed, with 555 patients (0.7%) testing positive. HCV RNA testing was requested for 307 (55.3%) of anti-HCV-positive patients during follow-up visits (Table 1). The rate of HCV RNA testing requests among anti-HCV-positive patients was significantly lower in surgical clinics compared with other departments ($p < 0.001$) (Table 2). Telephone calls were made to 248 patients without prior HCV RNA testing, of whom 114 (46%) could not

be reached. An anti-HCV S/Co ratio above 10.38 predicted HCV RNA positivity with 100% sensitivity and 89% specificity (AUC: 0.95, $p < 0.001$) (Table 3, Figure 1).

Conclusions:

Increasing awareness of HCV diagnosis among surgical clinics and implementing reflex testing algorithms to ensure diagnosis at the patient's first hospital visit will substantially support HCV elimination efforts. In addition, developing reflex testing algorithms based on the identified anti-HCV S/Co cutoff value of 10.38 may enable a more targeted and cost-effective approach to detecting previously undiagnosed HCV cases.

Keyword(s) : HCV screening, HCV RNA, reflex test

1094 - Oxidative Stress-Immunotherapy Gene Signatures Define Prognosis and Immune Subtypes in Hepatocellular Carcinoma

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Background: Hepatocellular carcinoma (HCC) exhibits complex interactions between oxidative stress and immune regulation that critically influence tumor progression and treatment response. This study aimed to identify oxidative stress- and immunotherapy-related genes (OSIRGs) associated with HCC prognosis, to construct a predictive model, and to explore novel immune subtypes relevant to immunotherapeutic sensitivity.

Methods: Transcriptomic and clinical data for HCC were obtained from TCGA-LIHC and GSE14520 cohorts. Differentially expressed genes (DEGs) were identified using DESeq2, and intersected with curated oxidative stress and immunotherapy gene sets to obtain OSIRDEGs. Functional enrichment (GO, KEGG, GSEA), WGCNA, and Cox-LASSO regression were applied to construct and validate a prognostic risk model. Consensus clustering defined molecular subtypes, which were further analyzed by TIDE, immune checkpoint, ICD, and HLA profiling, as well as immune infiltration (CIBERSORT and ssGSEA). Somatic mutation, copy number variation, and drug susceptibility analyses were performed to elucidate genomic and therapeutic correlations.

Results: Twenty-four OSIRDEGs were identified,

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primarily enriched in cell cycle regulation and mitotic pathways. WGCNA and LASSO analyses yielded six key genes (BUB1B, CDKN2A, CENPE, HMMR, PTTG1, SPP1), forming a robust prognostic model validated across independent cohorts ($p < 0.001$). Based on these genes, HCC samples were divided into two distinct subtypes (Cluster 1 and Cluster 2) with significantly different survival outcomes ($p < 0.001$). Cluster 2 exhibited lower TIDE scores and higher expression of immune checkpoint and ICD-related genes, suggesting greater potential responsiveness to immunotherapy. Immune infiltration analyses revealed distinct immune cell compositions and correlations across subtypes. Somatic mutation and CNV analyses indicated recurrent alterations in CDKN2A, CENPE, and HMMR. Drug sensitivity screening linked key model genes to multiple targeted agents. qPCR and western blotting confirmed upregulation of all six genes in HCC cell lines compared with normal hepatocytes. Conclusions: We identified and validated an oxidative stress-immunotherapy-related prognostic model that effectively stratifies HCC patients by survival and immune characteristics. The six-gene signature provides insights into tumor immune heterogeneity and may serve as a biomarker set for predicting immunotherapeutic efficacy and guiding personalized HCC management.

Keyword(s) : Hepatocellular carcinoma; Oxidative stress; Immunotherapy; Prognostic gene signature; Immune subtypes; Biomarkers

1095 - Efficacy and safety of postoperative adjuvant HAIC combining lenvatinib with or without PD 1 inhibitors in solitary large HCC A multicenter retrospective study

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Background: Solitary large hepatocellular carcinoma (HCC; >5 cm) remains prone to early recurrence despite curative resection. Adjuvant strategies that integrate hepatic arterial infusion chemotherapy (HAIC) with systemic therapy are increasingly explored. HAIC achieves high intrahepatic drug exposure, while lenvatinib and PD-1 inhibitors have shown efficacy in advanced HCC. Whether adding PD-1 inhibitors to postoperative adjuvant HAIC plus lenvatinib can further

reduce postoperative recurrence in solitary large HCC warrants evaluation.

Methods: A total of 183 patients with solitary large HCC who underwent curative resection and subsequent PA-HAIC plus lenvatinib (HAIC-L, $n = 108$) or PA-HAIC combined with lenvatinib and PD-1 inhibitors (HAIC-L-P, $n = 75$) were enrolled from three centers between April 2021 and April 2023. Propensity score matching (PSM) was applied to balance baseline characteristics. Disease-free survival (DFS) and overall survival (OS) were analyzed using the Kaplan-Meier method and Cox proportional hazards models, while treatment-related adverse events (TRAEs) were compared between groups.

Results: The HAIC-L-P group demonstrated significantly improved DFS compared to the HAIC-L group both before (HR: 0.570; $P = 0.007$) and after PSM (HR: 0.518; $P = 0.018$). In contrast, no statistically significant difference was observed in OS between the two groups. Multivariate analysis identified elevated AFP (≥ 400 ng/mL), microvascular invasion, and treatment strategy (HAIC-L vs. and HAIC-L-P) as independent predictors of DFS. Additionally, the overall safety profiles were comparable, with similar incidences of TRAEs and no significant increase in hepatic toxicity with PD-1 inhibitor addition.

Conclusion: In patients with solitary large HCC after curative resection, adding PD-1 inhibitors to adjuvant HAIC plus lenvatinib significantly prolongs DFS with acceptable safety, although an OS benefit was not observed within current follow-up. These findings support tri-modality adjuvant therapy as a promising postoperative option. Further prospective, biomarker-driven trials are warranted to validate these findings and optimize patient selection.

Keyword(s) : Solitary large hepatocellular carcinoma; Postoperative adjuvant therapy; Hepatic arterial infusion chemotherapy; PD-1 inhibitors; Combined therapy

1097 - The Roadtrip PEERS AND MEDICAL STAFF IS THE BEST MATCH TO TEST AND TREAT DRUG USERS FOR HEPATITIS C

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Background / Hypothesis:

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To achieve the goal of eliminating hepatitis C by 2030, it is essential to prioritize screening among key populations: people who use drugs, those experiencing homelessness or social precarity, incarcerated individuals, and people attending day shelters, accommodation centers, or asylum seeker centers. The process must be simplified and shortened as much as possible, while building trust with a population that can be difficult to reach. This has been the mission of the "Roadtrip" team since 2019.

Materials and Methods:

The team consists of former drug users cured of their hepatitis C infection, a nurse, and a physician.

Together, they offer hepatitis C screening within various facilities that serve these populations.

Four objectives:

Screening: Perform rapid diagnostic tests (RDTs) for HCV. When an RDT is positive, confirm with a point-of-care RNA test (POCT) and assess liver stiffness using Fibroscan®. If viral load is confirmed and there is no significant fibrosis, treatment is prescribed on the same day by the physician.

Confirmation of cure: Confirm sustained virological response (SVR) in individuals who have not done so for various reasons (lack of health insurance, damaged venous access, etc.).

Detection of reinfection: Verify that no reinfection has occurred and reintegrate patients into care pathways if necessary.

Liver assessment: Perform Fibroscan® for all participants to assess liver health.

Results:

A total of 411 people accepted testing, and 394 HCV (Rapid Diagnostic Tests) were performed. 42 were positive. After POCT confirmation, 30 individuals were RNA positive (7.2%), consistent with published data. All had low Fibroscan® scores, meaning no significant fibrosis, and were therefore eligible for treatment prescribed directly by a general practitioner (in France, any GP can prescribe hepatitis C treatment in the absence of advanced fibrosis). Among these 30 people who use drugs, one case was a reinfection and another was a treatment failure; the latter was referred to a specialized center. The POCT also confirmed cure in 24 individuals.

Beyond hepatitis C, 5 HBsAg-positive cases were detected in asylum seeker centers, and 6 suspected

cirrhotoses were identified in shelters for people in precarious situations. The Roadtrip has just begun activities in prisons; it is too early to present concrete results.

Conclusions:

These Roadtrips, led by peer workers trained in screening tools and supported by a patient association, appear to be an effective way to reach marginalized populations who are often reluctant to seek care. A 7.3% HCV positivity rate and immediate access to treatment demonstrate the effectiveness of this outreach approach.

Keyword(s) : hepatitis ; rapid diagnostic test ; fibroscan ; POCT; Ag Hbs

1100 - MRI differential diagnosis of hepatic metastasis of choroidal melanoma in the patient with hepatitis B cirrhosis

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Choroidal melanoma is a primary intraocular malignant tumor with a propensity for metastasis to the liver. However, it is uncommon in areas with a high incidence of hepatitis B and poses a challenge to clinicians' diagnosis in the absence of complete history information. MRI is an important method for the diagnosis of liver diseases, and there has been no study on the differential diagnosis between choroid melanoma liver metastasis and hepatitis B related hepatocellular carcinoma. Here, we present a case of choroidal melanoma liver metastasis in a patient with HBV cirrhosis and discuss the specificity significance of MRI in differential diagnosis of hepatic metastasis of choroid melanoma and HBV related hepatocellular carcinoma. This suggests that clinicians should consider the possibility of liver tumors other than hepatocellular carcinoma according to their clinical and imaging findings in areas with high incidence of hepatitis B.

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Keyword(s) : Choroidal melanoma, Hepatic metastasis, Hepatitis B cirrhosis, Magnetic resonance imaging

1105 - Comparative Efficacy of NSAIDs and Opiates in Alleviating Pain Associated with Mild to Moderate Pancreatitis A Randomized Controlled Trial

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Background / Hypothesis:

While both non-steroidal anti-inflammatory drugs (NSAIDs) and opioids are utilized for pain relief in acute pancreatitis, the preferred analgesic for mild to moderate cases remains uncertain. We evaluated Opioid and NSAID for analgesic efficacy and side effect profile, in mild to moderate acute pancreatitis.

Materials and Methods:

In parallel arms single-blind randomized clinical trial, mild to moderate acute pancreatitis patients were randomized into NSAID (ketorolac) and Opioid (tramadol) groups. The primary outcome was effectiveness in a reduction in pain at 2-hours of intervention. The secondary outcomes were a reduction in pain at 4-hours, 6-hours, 8-hours, and 24-hours, reduction in hospital stay, and an improvement in patient satisfaction.

Results:

A total of 60 patients were randomized into NSAID (n = 30) and Opioid group (n = 30) in a 1:1 pattern. Both drugs significantly reduced the pain score over 24-hours (p = 0.02). However, there was no significant difference in reduction in pain at 2-hours (Difference: -0.18, 95% CI: -0.70 – 1.03). In addition, regarding patient satisfaction and length of stay in the hospital, both drugs showed almost similar effectiveness. The opioids were most associated with sedation (p = 0.03) and epigastric pain (p = 0.04) compared to the NSAIDs

Conclusions:

The current study demonstrated that both NSAIDs and opioids are equally effective in managing pain in patients with mild to moderate acute pancreatitis. No significant differences were found between the two drug classes regarding pain relief, patient satisfaction, or length of hospital stay. However, opioids were

associated with a higher incidence of adverse effects, including sedation and epigastric pain, compared to NSAIDs.

Keyword(s) : Acute Pancreatitis, Opioid, NSAID, pain management, RCT

1106 - BEDSIDE NUTRITIONAL ASSESSMENT IN PATIENTS WITH LIVER CIRRHOSIS USING MINI NUTRITIONAL ASSESSEMENT PROFORMA

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Background / Hypothesis:

Patients suffering from advanced liver cirrhosis often exhibit compromised nutritional status as a result of reduced appetite and significant metabolic disruptions. These nutritional deficiencies contribute to further complications and an increased risk of death. Early detection of such deficits is essential for optimizing patient management. In this study, we applied the approved Mini Nutritional Assessment (MNA) tool to assess the nutritional condition of individuals who have received a diagnosis with cirrhosis.

Materials and Methods:

This investigation employed a cross-sectional design conducted within the Department of Gastroenterology at HMC Peshawar, encompassing a sample of 330 adult patients aged 18 years and older, all of whom had an established diagnosis of liver cirrhosis. Patients concurrently diagnosed with cancer or with severe liver encephalopathy (grade 3& 4) were excluded. Anthropometric measurements i.e. Body Mass Index (BMI), Mid-Arm Circumference (MAC), and Triceps Skin-fold thickness (TSF) were recorded. Nutritional status was determined using MNA scores, while liver disease severity was categorized according to Child-Pugh classifications. Statistical methods including logistic regression analysis were used to explore the relationships between nutritional parameters and clinical features.

Results:

The study had mean MNA score of 19.03 ± 4.43 , mean age of 54.73 ± 11.79 years, and a mean BMI of $22.41 \pm$

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2.76. We found that malnutrition exhibited a significantly higher prevalence in the older patients ($p=0.001$), in those with hepatitis B ($p=0.002$), and among patients with advance cirrhosis (Child B and C, $p=0.001$). Logistic regression analysis identified ascites and hepatic encephalopathy as significant predictors of nutritional decline.

Conclusion: The findings indicate that malnutrition is widespread among patients with liver cirrhosis, particularly in cases involving advanced disease and complications like ascites. This underscores the need for regular nutritional evaluation using MNA tool to enable timely interventions, which could improve clinical outcomes in this high-risk group.

Keyword(s) : Liver Cirrhosis, Nutritional Assessment, Malnutrition, Hepatic Encephalopathy

1107 - Serum fibroblast growth factor 21 as a novel biomarker of cachexia in chronic liver disease

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Background / Hypothesis:

Cachexia significantly worsens chronic liver disease (CLD) prognosis, and reliable predictive markers are lacking. We investigated clinical factors and biomarkers associated with cachexia in CLD.

Materials and Methods:

Among 526 patients with CLD, 356 with complete clinical cachexia information and preserved serum samples were included. A discovery cohort ($n=240$; August 2014–June 2023) underwent retrospective serum fibroblast growth factor (FGF) 21, interleukin (IL)-6, and tumor necrosis factor (TNF)- α measurements. Independent predictors and optimal biomarker cutoff values were identified by logistic regression and receiver operating characteristic analyses. Results were validated in an independent cohort ($n=116$; July 2023–May 2025).

Results:

Of all patients (median age 68 [range: 19–90] years), 65.8% were male, and 24.6% had cachexia, independently associated with poorer overall survival (hazard ratio: 1.64; 95% confidence interval [CI]: 1.03–2.62; $p=0.038$). Patients with cachexia had significantly higher serum FGF21 than those without (median: 292 vs. 177 pg/mL, respectively; $p=0.002$), while IL-6 and TNF- α levels did not differ. Serum FGF21 level was the sole independent biomarker for cachexia (odds ratio: 1.71; 95% CI: 1.10–2.66; $p=0.016$). Other clinical predictors included advanced Child–Pugh class (B/C vs. A; $p<0.05$) and lower platelet count. An FGF21 cutoff of 426 pg/mL discriminated cachexia prevalence in the discovery (38.3% vs. 20.0%; $p=0.006$) and validation cohorts (40.7% vs. 15.7%; $p=0.014$).

Conclusions:

Serum FGF21 is a novel, clinically relevant biomarker for predicting cachexia in CLD, enabling earlier detection and intervention to improve prognosis.

Keyword(s) : cachexia, fibroblast growth factor, liver cirrhosis, chronic liver disease, biomarker

1108 - Comparison of Liver Biopsy with Shear Wave Elastography and Other Non Invasive Fibrosis Markers in Chronic Hepatitis B

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Background / Hypothesis: Liver fibrosis severity is a key determinant of disease prognosis and treatment strategy in chronic hepatitis B (CHB). Although liver biopsy (LB) remains the gold standard for fibrosis assessment, it is invasive, costly, and carries potential complications, which limit its routine use.

Consequently, several non-invasive methods such as biochemical indices—Fibrosis-4 (FIB-4) and Aspartate Aminotransferase to Platelet Ratio Index (APRI)—and radiological techniques like transient elastography (TE) and magnetic resonance elastography (MRE) have been developed. Shear-wave elastography (SWE) is a relatively new, ultrasound-based, non-invasive method that quantitatively measures liver stiffness without requiring high-cost or regularly calibrated devices. This study aimed to compare fibrosis scores obtained by LB with those derived from SWE, FIB-4, and APRI in treatment-naïve CHB patients.

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Materials and Methods: Seventy patients aged 18–70 years, diagnosed with CHB and scheduled for liver biopsy at Atatürk University Faculty of Medicine, were included. None had received prior antiviral therapy. Shear-wave elastography (SWE) measurements were performed during routine abdominal ultrasonography. Laboratory parameters, including AST, ALT, and platelet count, were recorded to calculate APRI and FIB-4 indices. Histopathological fibrosis stages obtained from liver biopsy were then compared with SWE, APRI, and FIB-4 results to determine diagnostic accuracy and predictive performance.

Results: Among 70 participants, 27 (38.6%) were female and 43 (61.4%) male, with a mean age of 43.2 ± 14.9 years. The area under the receiver operating characteristic curve (AUC) for SWE in predicting significant fibrosis (Metavir $F \geq 2$) was 0.784 ($p < 0.001$). The optimal SWE cutoff value for identifying significant fibrosis was determined as 7.28 kPa, yielding 88% sensitivity, 72% specificity, and a high negative predictive value of 98%. The AUCs for APRI and FIB-4 were 0.81 and 0.78, respectively, demonstrating comparable diagnostic accuracy. A statistically significant correlation was observed between SWE, serum fibrosis indices, and histological scores obtained from liver biopsy.

Conclusions: SWE and serum-based fibrosis markers (APRI and FIB-4) demonstrated strong concordance with liver biopsy findings in patients with chronic hepatitis B. Given their simplicity, reproducibility, low cost, and non-invasive nature, these approaches—alone or in combination—may serve as practical and reliable alternatives to liver biopsy for the evaluation and follow-up of hepatic fibrosis in clinical practice.

Keyword(s) : Hepatitis B, Liver fibrosis, Shear-Wave Elastography

1109 - Stereotactic body radiation therapy as an add on to targeted agents in patients with locally advanced hepatocellular carcinoma

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Background / Hypothesis:

Stereotactic body radiation therapy (SBRT) delivers high-precision, ablative radiation doses to tumors in few fractions, minimizing exposure to normal tissue. In patients with locally advanced hepatocellular carcinoma (HCC), SBRT achieves local control comparable to ablation and is not limited by tumor location. Preclinical and clinical data suggest that SBRT may induce immunogenic cell death, enhance antigen release, remodel the tumor microenvironment, and potentiate the effects of targeted agents or immunotherapy. This systematic review and meta-analysis aimed to assess the efficacy and safety of combining SBRT with targeted agents compared with targeted therapy alone in patients with locally advanced HCC.

Materials and Methods:

A systematic search of PubMed, Embase, and Cochrane CENTRAL was conducted through March 2025. Eligible studies included randomized controlled trials (RCTs) involving adult patients with locally advanced HCC treated with SBRT plus targeted agents versus targeted therapy alone, reporting efficacy and safety outcomes. Risk ratios (RRs) were pooled using a random-effects model.

Results:

Four RCTs using with 381 patients were included. Sorafenib or apatinib were the targeted agents in the included studies. SBRT significantly reduced one-year mortality (RR = 0.47, 95% CI: 0.28–0.78, $p < 0.01$) and two-year mortality (RR = 0.68, 95% CI: 0.51–0.91, $p < 0.01$) compared with targeted therapy alone. The combination achieved a higher overall response rate (RR = 1.53, 95% CI: 1.19–1.96, $p < 0.01$) and disease control rate (RR = 1.31, 95% CI: 1.11–1.54, $p < 0.01$). Adverse event (AE) rates were comparable between the two groups (RR = 1.02, 95% CI: 0.97–1.07, $p = 0.88$).

Conclusions:

SBRT in combination with targeted agents such as sorafenib or apatinib significantly improves survival and tumor response without increasing AE rate compared with targeted therapy alone in patients with locally advanced HCC. These results support integrating SBRT into multimodal treatment strategies for locally advanced HCC and warrant validation in large-scale prospective trials.



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Keyword(s) : Stereotactic body radiation, sorafenib, apatinib, hepatocellular carcinoma, locally advanced HCC

1111 - Effectiveness and safety of Fibrate Therapy in the treatment of adults with Primary Sclerosing Cholangitis A Systematic Review and Meta Analysis

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Background / Hypothesis:

Primary sclerosing cholangitis is characterized by the stricturing of the bile ducts, either intrahepatic or extrahepatic bile ducts, and co-existence of inflammatory bowel disease, of which it is usually found in around 70% of patients. The global prevalence of primary sclerosing cholangitis is estimated to be 16 per 100,000 individuals, and in the Philippines, the prevalence of primary sclerosing cholangitis was around 1%. The risk for complications in the presence of primary sclerosing cholangitis could lead to liver failure, development of cholangiocarcinoma, and possible colon malignancy. This study compared the fibrate therapy and placebo in terms of serum liver biomarkers and safety of patients with primary sclerosing cholangitis

Materials and Methods:

Meta-analysis was the research design used. Specifically, the main author obtained the research design, the methodology of these investigations, interventions used, results and outcomes of these respective studies, among many others. Two research studies of de Vries and colleagues and Hatami and colleagues were included in the meta-analytical assessment.

Results:

The investigation yielded statistical difference with 95% CI between placebo and fibrate therapy in terms of serum biomarkers specifically alkaline phosphatase (399.24 ± 173.57 vs. 261.85 ± 191.92 respectively, $p .050 < .05$). On the other hand, there was no statistical difference between placebo and fibrate therapy in terms of aspartate aminotransferase (66.02 ± 43.95 vs. 59.29 ± 39.86 respectively, $p .390 > .05$), alanine aminotransferase (72.09 ± 62.32 vs. 65.14 ± 42.37 respectively, $p .630 > .05$), total bilirubin (11.07 ± 10.17 vs. 10.52 ± 10.64 respectively, $p .390 > .05$), and serum albumin (22.27 ± 1.80 vs. 21.89 ± 3.23 respectively, $p .460 > .05$).

Conclusions:

Fibrate therapy could potentially improve the outcomes of primary cholangitis of patients. In this

regard, the initiation of fibrate therapy could be beneficial in improving the biochemical markers of the aforementioned disease, better prognosis, and may also alleviate pruritus in patients.

Keyword(s) : Fibrate Therapy, Liver Disease, Primary Sclerosing Cholangitis

1114 - Detection of HBV and HCV seroprevalence using rapid point of care tests among earthquake survivors in Southeastern Türkiye

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Background / Hypothesis: Türkiye has a moderate seroprevalence of HBV infection in its general population, with the Southeastern Anatolia Region exhibiting the highest rate. In contrast, the seroprevalence of HCV infection in Türkiye is low. The study aimed to determine the prevalence of HBV and HCV in the communities affected by the earthquake in Southeastern Türkiye.

Materials and Methods: This was a cross-sectional study in Southeastern Türkiye in December 2023, eleven months after the 2023 Türkiye-Syria earthquakes. HBsAg and anti-HCV were investigated using rapid screening tests on blood samples from participants selected from 251 containers, where 1,213 people resided.

Results: Out of 251 samples tested, 13 (5.2%) were positive for HBV, while none (0.0%) tested positive for HCV. When comparing the risk factors for HBV infection between the positive and negative groups, earthquake survivors who tested positive for HBsAg were significantly more likely to be male sex ($p=0.018$) and to have a family history of HBV ($p<0.001$). There were no significant differences ($p > 0.05$) in age, number of household members, education level, marital status, history of surgery or dental procedure, tattoo-piercing, and history of blood transfusion.

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Conclusions:In Southeastern Türkiye, 5.2% of earthquake survivors tested positive for HBsAg using rapid point-of-care tests. More comprehensive studies are needed to determine the prevalence of HBsAg and anti-HCV in the general population and to identify risk factors.

Keyword(s) : HBV, HBsAg, anti-HCV, point-of-care tests, prevalence.

1115 - Effectiveness and Follow up Challenges of Sofosbuvir Velpatasvir in HCV HIV Co infected Patients A Real world Experience from a Chinese Cohort

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Background / Hypothesis:HCV/HIV co-infected patients represent a unique population with complex clinical needs and potential socioeconomic vulnerabilities, which may impact treatment adherence and follow-up. This study aimed to evaluate the real-world effectiveness of sofosbuvir/velpatasvir (SOF/VEL) in HCV/HIV co-infected individuals and analyze challenges in sustaining post-treatment virologic monitoring.

Materials and Methods:We retrospectively analyzed 35 HCV/HIV co-infected patients treated with SOF/VEL for 12 weeks between 2021 and the present. All lost to follow-up at end-of-treatment (EOT) were contacted for 12-week virologic reassessment with free HCV RNA testing.

Results:Among 35 patients, baseline median HCV RNA was 1.83×10^6 IU/mL; 6 of 31 with Fibro Scan data had F3 or higher fibrosis, and 2 were treatment experienced. At EOT, 4 patients loss to follow up, 29 patients tested with an assay (LLOQ 1000 IU/mL) all had undetectable HCV RNA or levels below LLOQ. Of 3 patients tested with a high-sensitivity assay (LLOQ 15 IU/mL), 2 had low-level viremia (<1000 IU/mL). At post-treatment week 12, 14 patients returned for free testing (LLOQ 15 IU/mL), all achieving SVR, including the 2 with low-level viremia at EOT. Two additional patients reported negative self-test results from external labs. The remaining 15 declined follow-up. The

SVR12 rate was 100% among those retested (16/16). However, high loss-to-follow-up rates at EOT (11.4%) and during SVR12 assessment (57.1% attrition/refusal) highlighted significant retention challenges. No significant adverse event or drug to drug interaction was identified.

Conclusions:SOF/VEL demonstrated high efficacy in HCV/HIV co-infected patients. The substantial attrition underscores the need for tailored follow-up strategies in this special population, where socioeconomic factors, stigma, and complex clinical dynamics may hinder long-term monitoring. Proactive retention measures are critical to ensure complete outcome ascertainment in real-world settings.

Keyword(s) : HIV/HCV coinfection, sofosbuvir/velpatasvir, sustained virologic response, retention challenges

1119 - Metabolic Associated Fatty Liver Disease in Obese and At Risk Children and Adolescents in Türkiye A Meta Analysis of Prevalence

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Background / Hypothesis:

Metabolic associated fatty liver disease (MAFLD), formerly referred to as non-alcoholic fatty liver disease (NAFLD), is the most common chronic liver disease worldwide, affecting approximately 30% of the adult population, with its burden expected to increase further in the future. In Türkiye, prevalence has been reported to reach up to 50% in certain regions. Among children, the overall prevalence of MAFLD is estimated at around 7%, and early manifestation is associated with a more severe prognosis. Despite this growing burden, no systematic analysis of pediatric MAFLD prevalence in Türkiye has yet been conducted. Here, we aimed to estimate the pediatric MAFLD prevalence in Türkiye.

Materials and Methods:

A comprehensive literature search was conducted for

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studies published in English and/or Turkish between 2005 and 2023 using the databases MEDLINE, PubMed, EMBASE, and UlakBim. The search strategy included the following terms: 'Turkey AND NAFLD AND Pediatric' and 'Turkey AND NAFLD AND Prevalence AND Children AND Adolescent.' The studies identified through this literature review were incorporated into the meta-analysis.

Results:

The pooled overall prevalence of MAFLD across the 56 studies, from which 51 studies included obese children, was estimated at 44.6% (95% CI: 39.5–49.7).

Heterogeneity among studies was considerable, with an I^2 value of 96%, indicating substantial variability. Subgroup analysis showed a pooled prevalence of 54.9% in boys (95% CI: 48.6–61.1) and 41.2% in girls (95% CI: 35.6–46.9). Regionally, the prevalence was 49.0% (95% CI: 41.2–57.1) in the Marmara and Aegean regions, and 42.2% (95% CI: 35.3–49.1) in the Black Sea, Mediterranean, and Central Anatolian regions.

Conclusions:

The results of this study indicate a markedly high prevalence of MAFLD among children and adolescents, which is expected to substantially contribute to the future disease burden. These findings should serve as an alert for policymakers to adapt healthcare strategies aimed at preventing childhood obesity and MAFLD.

Keyword(s) :

1120 - Research Trends of Targeted Drugs for Nonalcoholic Fatty Liver Disease A Bibliometric Analysis (2004–2024)

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Background / Hypothesis:The incidence of nonalcoholic fatty liver disease (NAFLD) increases annually; therefore, it is the leading cause of liver-related morbidity and mortality worldwide. No targeted drugs are available for the treatment of NAFLD. We conducted a bibliometric analysis of publications in the field of NAFLD-targeted drug research to identify research hotspots and trends over the past two decades to provide insights that allow rapid grasping of research priorities and guide future investigations.

Materials and Methods:We retrieved all English-language publications about NAFLD-targeted drug research from the Web of Science Core Collection database available between January 1, 2004, and June 7, 2024. Data were exported and stored as Excel files and plain text. Then, they were analyzed and visualized using Microsoft Office Excel 2021, VOSviewer, CiteSpace, and the R package "bibliometrix."

Results:A total of 1,393 publications were included in this study. The analysis revealed a significant increase in research activity over the past 20 years and accelerated growth in publications and citations after 2020. The field of NAFLD-targeted drug research is characterized by the dominance of China and the United States (USA). China produced the most publications, whereas the USA had the most citations. Scholars and institutions in the USA emerged as global academic hubs through extensive international collaboration; however, research in China remained domestically focused. Research themes have evolved from traditional hepatology to interdisciplinary integration (molecular biology and pharmacology). Keywords and citation analyses identified three major research directions: disease mechanisms and pathophysiology, molecular targets and drug development, and multi-omics/technological integration.

Conclusions:We elucidated the evolving research trends of NAFLD-targeted therapeutics and provided a reference framework for exploring novel directions in NAFLD-targeted therapeutics.

Keyword(s) : nonalcoholic fatty liver disease; drug development; bibliometrics; research

1124 - Single arm meta analysis and systematic review on clinical cure of chronic hepatitis B after hematopoietic stem cell transplantation immunoregulatory mechanisms and clinical translational insights

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Background / Hypothesis: The quest for a functional cure for chronic hepatitis B (CHB) remains an elusive objective. Hematopoietic stem cell transplantation



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(HSCT) may elicit viral clearance by immunological remodelling in recipients with positive-HBsAg; however, the factors that determine its effectiveness are yet unknown. The aim of the study is to assess HSCT-induced hepatitis B surface antigen (HBsAg) seroclearance rates and identify immunological mechanisms and combination therapy strategies through meta-analysis and systematic review.

Materials and Methods: In this study, nine trials (308 patients) were subjected to single-arm meta-analysis (R4.4.2 "meta" package) in accordance with PRISMA/Cochrane criteria. Heterogeneity was addressed via random-effects models ($I^2 = 87.1\%$). Subgroup analyses were performed to explore the influence of donor and recipient immune status on the HBsAg clearance rate. The Newcastle-Ottawa Scale (NOS) and the Risk Of Bias In Non-randomized Studies of Interventions (ROBINS-I) tool were used to evaluate the quality and bias of the included studies.

Results: The total HBsAg clearance rate was 26% (95% CI:14% to 39%), greater than that of standard therapies. Notably, HBsAb-positive donors had significantly enhanced clearance (55% vs. 8%, $p < 0.01$). HBeAg-negative recipients fared better (29% vs. 2%). In patients who achieved HBsAg loss, HBsAb-positive donors and HBeAg-negative recipients are the most prevalent types (10/11). Sensitivity analyses confirmed robustness and publication bias was nonsignificant (Egger's $p = 0.271$). We propose a two-phase immunomodulation model for HSCT-induced HBsAg seroclearance. In phase I (passive immunomodulation period), donor-derived HBsAb passively neutralizes the existing pool of circulating HBsAg, lowering the antigenic burden of HBsAg and interrupting the cycle of antigen overload-induced immunosuppression. In phase II (active immune reconstitution period), donor T cells are progressively differentiated into HBV-specific effector T cells that can directly kill cccDNA-positive hepatocytes and induce epigenetic suppression of viral transcription.

Conclusions: In this study, we found that HSCT, especially with HBsAb-positive donors and HBeAg-negative recipients, may present a promising approach toward achieving a functional cure in CHB patients. Although HSCT is clinically challenging due to its procedures and high risks involved, it provides valuable information regarding the immunological mechanisms underlying HBsAg clearance. These findings may guide the design of targeted immunotherapies that can

achieve the same efficacy as HSCT while minimizing potential risks, thereby advancing the safer and more feasible pursuit of functional cures for CHB in response to the WHO's 2030 elimination goal. Nevertheless, substantial clinical heterogeneity among included studies and limited follow-up duration are limitations of this study. Future studies

Keyword(s) : Chronic Hepatitis B, Hematopoietic Stem Cell Transplantation, Functional Cure, Immune Reconstitution, Meta-Analysis

1128 - Zinc alpha 2 glycoprotein (ZAG) emerges as a diagnostic biomarker for Hepatitis B virus related acute on chronic liver failure (HBV ACLF)

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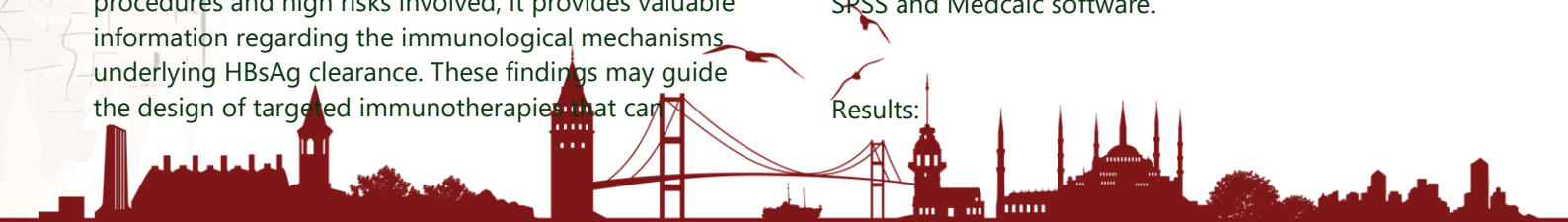
Background / Hypothesis:

Hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF) is a critical syndrome with high mortality, and the absence of reliable prognostic biomarkers poses a significant challenge to its clinical management. The pathogenesis involves viral replication, excessive inflammation, and dysregulated lipid metabolism. Emerging evidence indicates that zinc-alpha 2-glycoprotein (ZAG) modulates these pathways and exerts protective effects in nonalcoholic fatty liver disease (NAFLD). Given these mechanistic parallels, this study sought to determine whether ZAG expression in patient serum and liver tissue could serve as a prognostic biomarker for HBV-ACLF.

Materials and Methods:

A cross-sectional survey involving 72 subjects, including negative controls, Chronic Hepatitis B (CHB) patients, patients with liver cirrhosis (LC), LC-AD, and ACLF was performed. And a prospective longitudinal cohort study was conducted at the First Affiliated Hospital of Zhejiang University and the First Affiliated Hospital of Nanchang University involving 355 patients with HBV-ACLF to assess the prognostic capacity of ZAG. ZAG levels were measured using a commercial ELISA kit, and statistical analyses were conducted using SPSS and Medcalc software.

Results:



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The results demonstrated that ZAG levels decreased progressively with the advancement of liver disease severity ($P<0.001$). Moreover, the ZAG levels in non-survivors were lower than those in survivors ($P<0.001$). Multivariate Cox regression analysis revealed that ZAG was an independent prognostic factor for HBV-ACLF. Patients with lower ZAG levels had a higher mortality rate of 31.7%, while those with higher ZAG levels had a mortality rate of 11%. When patients were stratified based on their cirrhosis status, ZAG remained an independent prognostic factor, regardless of whether they had cirrhosis. As the number of failing organs increased, the ZAG levels decreased correspondingly ($P<0.001$), which was closely associated with coagulation dysfunction and renal failures. At the end of the assessment period (14th day after admission or discharge), the ZAG levels increased significantly in the improvement group ($P<0.001$), decreased significantly in the deterioration group ($P=0.035$). We developed a novel prognostic model (ZAG3TIH) incorporating ZAG. This model demonstrated superior performance for predicting 30-day mortality, achieving an AUROC of 0.881 and significantly outperforming conventional scores such as Child-Pugh, MELD and CLIF-SOFA.

Conclusions:

In conclusion, ZAG demonstrates promising potential as an independent prognostic biomarker for HBV-ACLF. Sequential monitoring of circulating ZAG levels may be valuable for tracking disease progression. It is expected to improve current commonly used ACLF prognostic models. Further studies are needed to explore

Keyword(s) : Zinc-alpha 2-glycoprotein; biomarker; Prognostic model; Dynamic monitoring

1129 - Efficacy of albumin infusion for hypoalbuminemia in post operative period in living donor liver transplantation – An open label randomized controlled trial

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Background / Hypothesis:

Albumin is commonly administered for hypoalbuminemia in immediate post-liver transplant period to improve oncotic pressure and recovery, but evidence is limited. Hypoalbuminemia is often due to capillary-leak rather than reduced synthesis. This study aimed to compare surgical drain output on postoperative day (POD) 5 in patients receiving or not receiving albumin infusion.

Materials and Methods:

In this single-center, prospective, open-label randomized trial (CTRI/2024/07/071234), adult living donor liver transplant (LDLT) recipients were randomized to receive either albumin infusions (target serum albumin ≥ 3.0 g/dL) or no albumin, continued through POD-5. Outcomes including drain output, fluid balance, biochemical markers, and complications were assessed till POD-5 and at discharge.

Results:

Eighty-six adult LDLT recipients were randomized into two equal groups ($n=43$ each) to receive either albumin infusions or no albumin. Baseline demographics, surgical details, and graft characteristics were comparable. On POD-5, serum albumin levels were significantly higher in the albumin arm (3.1 vs 2.4 g/dL, $p<0.005$). The no-albumin group demonstrated a significantly lower median surgical drain output on POD-5 (450 vs 1100 mL, $p=0.043$), representing a 59% reduction compared to the albumin arm. Although urine output was higher in the albumin group (1555 vs 1210 mL, $p=0.022$), the overall cumulative fluid balance remained more positive (5685 vs 4798 mL, $p=0.049$), suggesting greater fluid retention and relative volume overload (Table attached). Despite this, renal function indices were comparable between groups. Notably, the incidence of sepsis was significantly higher in the no-albumin group (27.9% vs 7%, $p=0.021$). Renal function markers, inotropic support, lactate levels, tacrolimus dosing and trough levels, body weight, and time to drain cessation were comparable between groups. Tacrolimus toxicity occurred more frequently in the no-albumin arm (15 vs 5 cases, $p=0.020$). ICU stay (7 [6–14] vs 7 [6–11] days, $p=0.351$), and hospital stay (21 [20–33] vs 22 [19–35] days, $p=0.785$), were comparable, with no mortality in either group.

Conclusions:

Omitting albumin infusion in the immediate post-LDLT period reduced surgical drain output and fluid

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accumulation on POD-5 without adversely affecting renal function, ICU stay, hospital stay, or mortality, although higher sepsis rates and tacrolimus toxicity were observed.

Keyword(s) : Liver transplantation; albumin infusion; fluid balance; postoperative outcomes.

1131 - Dietary Total Antioxidant Capacity and Metabolic Dysfunction Associated Steatotic Liver Disease a cross sectional study

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Background: Metabolic dysfunction associated fatty liver disease (MASLD) is a serious public health problem. Dietary antioxidant intervention is an effective measure to manage diseases. Dietary total antioxidant capacity (TAC) is an index to evaluate antioxidant capacity. There are limited data on its association with MASLD.

Objective: This study aimed to investigate the association between dietary TAC and MASLD in US adults.

Methods: We analyzed cross-sectional data from 8,470 participants of the National Health and Nutrition Examination Survey (NHANES) 2017–2023. Dietary TAC was estimated using 24-hour dietary recall data.

Weighted logistic regression models and restricted cubic splines (RCS) were employed to evaluate the association between dietary TAC and MASLD.

Results: Among the participants, 3165 had MASLD, of whom 517 were identified as having at-risk MASH. After adjusting for confounders, dietary TAC was inversely associated with the prevalence of MASLD (OR=0.90, 95% CI: 0.84–0.97, P = 0.009). Compared to the lowest quartile (Q1), participants in the highest quartile (Q4) of log-transformed dietary TAC had 33% lower odds of MASLD (OR=0.67, 95% CI: 0.54 - 0.83, P <0.001), with a significant dose-response trend (P for trend = 0.002). Dose-response analysis showed that dietary TAC was negatively and non-linearly associated with the risk of MASLD, which was mainly driven by vitamin C, vitamin E, β -carotene, and lutein-zeaxanthin. No significant association was found between dietary TAC and a high risk of at-risk MASH.

Conclusion: Higher dietary TAC may be protective against MASLD, while the evidence is insufficient for its role in at-risk MASH.

Keyword(s) : Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD); Dietary Total Antioxidant Capacity (TAC); Dietary Antioxidants; NHANES (National Health and Nutrition Examination Survey);

1135 - Impact of Low Dose Rifaximin on Gut Antibiotic Resistance Genes in Patients with Decompensated Cirrhosis

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Background / Hypothesis:

Decompensated cirrhosis patients are prone to bacterial infections and frequent antibiotic exposure, leading to elevated gut antibiotic resistance gene (ARG) burden. Rifaximin, a broad-spectrum non-absorbable antibiotic recommended for hepatic encephalopathy (HE) prevention, raises concerns about potential ARG enrichment despite its clinical benefits. We aimed to clarify the impact of low-dose rifaximin on gut resistome dynamics in decompensated cirrhosis.

Materials and Methods:

This secondary analysis utilized fecal metagenomic data from a multicenter randomized trial (NCT02190357). 42 patients with decompensated cirrhosis were enrolled; 26 received rifaximin (400 mg twice daily) for 6 months. Gut resistomes were profiled using shotgun metagenomic sequencing and annotated against the Comprehensive Antibiotic Resistance Database (CARD). ARG diversity, resistance mechanisms, key antibiotic-resistant taxa, mobile genetic elements (MGEs), and phage-mediated horizontal gene transfer were systematically analyzed.

Results: Baseline profiling identified 564 antibiotic resistance ontologies (AROs) spanning 120 AMR gene families, predominantly conferring resistance to fluoroquinolones, macrolides, and tetracyclines via efflux pump mechanisms. 17 core taxa—principally *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Escherichia fergusonii*—harbored the majority of ARGs. Low-dose rifaximin significantly reduced resistome α -diversity (ACE index: 440.7 ± 72.8 vs. 350 ± 27.2 , $p < 0.001$) and selectively depleted fluoroquinolone ($p = 0.003$), macrolide ($p = 0.007$), and streptogramin resistance genes ($p = 0.012$). The intervention achieved dual suppression of ARG reservoirs: (1) selective depletion of high-ARG-load bacteria (*E. coli*, *K. pneumoniae*, *P. aeruginosa*, *E.*

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fergusonii; all $p < 0.05$); (2) disruption of phage-mediated horizontal gene transfer (MGE-AROs: 86→40, $p < 0.001$; phage-AROs significantly reduced, $p = 0.002$). Crucially, rifaximin did not induce cross-resistance to anti-tuberculosis agents; instead, it reduced ethA ($p = 0.017$) and iniC ($p = 0.032$) abundance.

Conclusions:

Low-dose rifaximin paradoxically suppresses the gut resistome in decompensated cirrhosis via dual mechanisms: targeted elimination of ARG-harboring pathobionts and inhibition of phage-driven resistance dissemination. These findings challenge conventional assumptions about broad-spectrum antibiotic-induced resistance enrichment and support rifaximin's expanded therapeutic role in antibiotic stewardship for vulnerable cirrhotic populations.

Keyword(s) :

1136 - Catastrophic unmasking Fatal tuberculosis associated Immune Reconstitution Inflammatory Syndrome presenting as acute liver failure in pregnancy

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Background / Hypothesis:

Immune Reconstitution Inflammatory Syndrome (IRIS) is a severe, potentially fatal inflammatory condition that can occur in severely immunocompromised patients, such as those with HIV, after initiating antiretroviral therapy (ART). The syndrome is triggered by a restored immune system recognizing and over-reacting to pre-existing opportunistic pathogens. Tuberculosis-associated IRIS (TB-IRIS) poses a significant diagnostic and therapeutic challenge, particularly in pregnancy, where physiological changes and complex health considerations for both mother and fetus add layers of complexity. This report highlights a catastrophic presentation of TB-IRIS as fulminant hepatic failure in a pregnant patient newly started on ART.

Materials and Methods:

The case involves a 30-year-old female at 28 weeks gestation, recently diagnosed with HIV during prenatal screening and initiated on ART. Four weeks after starting treatment, she presented with symptoms of tuberculosis (fever, productive cough, night sweats, significant weight loss) which acutely worsened with the onset of jaundice and altered mental status. On admission, she was febrile and severely ill. Laboratory investigations revealed pancytopenia, severe transaminitis, hyperbilirubinemia, and coagulopathy. Mycobacterium tuberculosis was confirmed from sputum and ascitic fluid, and her CD4 count was critically low at 8%.

Results:

The patient was diagnosed with disseminated tuberculosis. However, both her ART and first-line anti-tuberculosis therapy were discontinued due to rapidly deteriorating liver function. Her clinical condition declined, marked by fetal distress, which necessitated an emergency Cesarean section. Post-operatively, she remained critically ill with persistent fever, worsening coagulopathy, and the development of ascites and encephalopathy, consistent with acute liver failure. The diagnosis of TB-IRIS was made based on the paradoxical clinical deterioration following ART initiation. Despite aggressive supportive care, the patient developed disseminated intravascular coagulation (DIC), progressive hepatic failure, and ultimately suffered cardiac arrest and death.

Conclusions:

This case underscores the diagnostic complexity and devastating potential of TB-IRIS, especially in pregnant women with advanced immunosuppression. The exaggerated inflammatory response can mimic other conditions and lead to catastrophic outcomes like fulminant liver failure. It highlights the critical importance of screening for latent and active opportunistic infections, such as tuberculosis, before initiating ART in high-risk patients. Clinicians should maintain a high index of suspicion for IRIS in patients who show paradoxical worsening after starting therapy, as early recognition is paramount.

Keyword(s) : Acute Liver Failure, Immune Reconstitution Inflammatory Syndrome, Pregnancy, Human Immunodeficiency Virus, Tuberculosis

1138 - Peripheral Single Cell Immune Profiling of HBsAg Loss in HBV Related Hepatocellular



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Carcinoma Receiving Anti PD 1 Therapy

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Background / Hypothesis:

Hepatitis B virus-related hepatocellular carcinoma (HBV-HCC) remains a major clinical challenge, driven by concurrent tumor progression and persistent viral antigenemia. While anti-PD-1 therapy has improved HCC prognosis by reinvigorating antitumor immunity, its role in promoting hepatitis B surface antigen (HBsAg) clearance during chronic HBV infection remains incompletely understood.

Materials and Methods:

Overall survival and HBsAg dynamics were retrospectively analyzed in a cohort of 211 HBV-HCC patients. Peripheral blood mononuclear cells from selected patients underwent single-cell RNA sequencing and T-cell receptor (TCR)/B-cell receptor (BCR) repertoire profiling. Functional validation was performed using cytokine assays and flow cytometry following PD-1 blockade and HBV peptide stimulation.

Results:

Anti-PD-1 therapy significantly prolonged overall survival (HR = 0.21, $p < 0.001$) and promoted HBsAg reduction in HBV-HCC patients ($p < 0.05$). Single-cell transcriptomic profiling revealed that HBsAg-negative patients exhibited enhanced activation signatures in T and NK cells, characterized by upregulated expression of JUN, FOS, CD69, and NFkBIA, coordinated activation of TNF and IL-18 signaling pathways. B cells also displayed upregulation of CD69, CD83, and HLA-DQA. Moreover, scRNA-TCR/BCR analyses uncovered biased V-J gene usage in patients who achieved HBsAg seroclearance. Functionally, PD-1 blockade selectively enhanced IFN- γ and TNF- α production in HBV core-specific T cells.

Conclusions:

Anti-PD-1 therapy enhances both survival and HBsAg decline in HBV-HCC by orchestrating reactivation of T, NK, B, and myeloid cells through IL-18 and TNF- α .

-mediated immune remodeling. This multicellular synergy bridges antiviral and antitumor immunity, revealing a dual mechanism of immune restoration under PD-1 blockade.

Keyword(s) : Anti-PD-1; Single-cell RNA sequencing; HBV-HCC; HBsAg seroclearance; Immune remodeling

1142 - From Naïve to Non Responders under DAA Pressure Unveiling NS5A and NS5B Resistance Associated Substitutions in HCV Genotype 3a from Bangladesh

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Background / Hypothesis:

Hepatitis C virus (HCV) remains a critical global health challenge, infecting approximately 58 million people worldwide and causing chronic liver disease, cirrhosis, and hepatocellular carcinoma. Among the circulating genotypes, HCV genotype 3a predominates in Bangladesh is associated with hepatic fibrosis and comparatively lower treatment success rates. Although DAAs are highly effective, RASs in NS5A and NS5B proteins can compromise sustained virological response (SVR). This study aimed to determine the prevalence and pattern of RASs in the NS5A and NS5B regions of HCV genotype 3a among treatment-naïve and DAA-experienced patients in Bangladesh, to elucidate their role in antiviral resistance and treatment outcomes.

Materials and Methods:

A total of 45 HCV genotype 3a 3a-infected patients were analyzed, of which 25 were treatment-naïve and 20 were DAA-failing individuals. Viral RNA was extracted from plasma samples, followed by cDNA synthesis and nested PCR amplification of the NS5A and NS5B regions using genotype 3a-specific primers. Amplicons were subjected to bidirectional Sanger sequencing, and nucleotide sequences were compared to reference HCV genotype 3a sequences. Amino acid substitutions were analyzed using the Geno2Pheno [HCV] online platform to predict potential resistance to Daclatasvir, Velpatasvir, and Sofosbuvir.

Results:



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RASs were detected exclusively within the NS5A region, while no mutations were found in NS5B. Among patients who failed DAA therapy, the predominant substitution was A62S (73.7%), whereas A62T was most frequent among treatment-naïve individuals (65.2%). Combined substitutions, including A62T + P58S (20%) and A62S + P58S + S98G (5%), were more frequently observed in DAA failure cases. The A62S variant showed susceptibility to both Daclatasvir and Velpatasvir, whereas A62L conferred reduced susceptibility to Daclatasvir. Clinically, patients with RASs showed more frequent liver cirrhosis and higher liver enzyme levels than treatment-naïve patients, indicating a link between RASs and advanced liver disease.

Conclusions:

NS5A-associated RASs, particularly A62S/T, play a key role in DAA treatment failure among patients infected with HCV genotype 3a in Bangladesh. The findings highlight the critical importance of routine molecular surveillance and baseline RAS screening to minimize the risk of resistance emergence. Integrating RAS monitoring into the national strategic plan for control of viral hepatitis program in Bangladesh is essential to optimize therapeutic outcomes and achieve HCV elimination goal by 2030.

Keyword(s) : hepatitis C virus, genotype 3a, direct-acting antiviral, resistance-associated substitutions, sanger sequencing.

1143 - Association between AST/ALT ratio and Hemodynamic Parameters of Carotid Arteries in Patients with Primary Aldosteronism: A Cross Sectional Study

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Background / Hypothesis:

The relationship between the aspartate aminotransferase to alanine aminotransferase ratio (AST/ALT) and hemodynamic parameters of carotid arteries in patients with primary aldosteronism is unclear. Therefore, this study aimed to examine the association between the AST/ALT ratio and carotid arteries peak systolic velocity (PSV), end diastolic velocity (EDV) and the ratio of internal carotid arteries (ICA) PSV to common carotid artery (CCA) PSV in patients with primary aldosteronism (PA).

Materials and Methods:

This research was conducted at the 2nd Affiliated Hospital of Nanchang University and involved PA individuals hospitalized between October 17, 2017 and April 24, 2022. 700 eligible participants were included in the analysis for this research. Doppler spectra of the ICA were obtained 1-1.5 cm distal to the carotid bulb in all subjects. The site 1-2 cm proximal to the carotid bifurcation was defined as the measurement location for the common carotid artery. The study applied smooth curve fitting to examine the linearity of the AST/ALT ratio and ICA PSV, ICA EDV and the ICA PSV/CCA PSV ratio relationship. Multiple logistic regression models to quantify this association, presenting results from both crude and fully adjusted models. Stratified analyses to investigate potential effect modifiers.

Results:

Patients with the lowest level of Lg-AST/ALT ratio (T1), patients in T3 group had a higher level of the ICA PSV ($\beta=5.23$, 95% CI: 1.24-9.21) and a higher level of the ICA EDV ($\beta=2.09$, 95% CI: 0.45-3.72). A key finding is the age-dependent positive association between the Lg-AST/ALT ratio and both ICA PSV and the ICA PSV/CCA PSV ratio. After full adjustment for covariates, each standard deviation increase in the Lg-AST/ALT ratio was associated with a significant increase in ICA PSV ($\beta=26.23$; 95% CI: -0.67-53.14, P for interaction=0.034) and in the ICA PSV/CCA PSV ratio ($\beta=0.59$; 95% CI: 0.20-0.98, P for interaction=0.014), but only in individuals aged 60 and above. Similarly, a significant association was observed for the ICA PSV/CCA PSV ratio ($\beta=0.59$; 95% CI: 0.20-0.98, P for interaction=0.014) exclusively in subjects with a hypertension duration of five or more years.

Conclusion:

Age was identified as a critical effect modifier in the relationship between the Lg-AST/ALT ratio and both ICA PSV and the ICA PSV/CCA PSV ratio, necessitating age-stratified analytical approaches. Furthermore, the duration of hypertension also acted as a significant effect modifier for the association between the Lg-AST/ALT ratio and the ICA PSV/CCA PSV ratio, requiring stratification by hypertension duration in its investigation and application.

Keyword(s) : Primary aldosteronism, Aspartate

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aminotransferase, Alanine aminotransferase, Peak systolic velocity, End diastolic velocity.

1145 - Targeting Cancer Associated Fibroblast Subpopulations and Metabolic Pathways to Enhance Immunotherapy in Hepatocellular Carcinoma

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Background / Hypothesis: Immune checkpoint inhibitors (ICIs) have revolutionized the treatment of hepatocellular carcinoma (HCC), yet significant challenges remain. Our previous research indicated that galacturonic acid (GA) is enriched in ICI responders and correlates with improved therapeutic outcomes, although its underlying mechanism is not fully understood. This study aimed to investigate the effect of GA on cancer-associated fibroblasts (CAFs) and associated metabolic pathways, proposing a CAF-mediated metabolic reprogramming mechanism that modulates the tumor microenvironment (TME) and enhances ICI efficacy.

Materials and Methods: We utilized single-cell RNA sequencing (scRNA-seq) to analyze three exploratory patients from our prior study, aiming to identify key metabolic pathways associated with ICI response. Subsequently, an additional eight patients were included to validate the effect of GA on CAF metabolic programming. The effects of GA on HCC and CAFs were further validated through both in vivo and in vitro experiments.

Results: Analysis of the exploratory cohort revealed significant CAF heterogeneity between responders and non-responders. The validation cohort analysis demonstrated that GA treatment selectively reprogrammed CAF metabolism, leading to a downregulation of extracellular matrix production and an upregulation of oxidative phosphorylation. This metabolic shift resulted in a more immune-permissive TME. In vitro assays confirmed that GA-treated CAFs suppressed HCC cell proliferation, reduced immune evasion markers, and altered cytokine and chemokine signaling. In vivo experiments showed that GA-conditioned CAFs significantly slowed tumor

progression, increased CD8+ T cell infiltration, and decreased immunosuppressive cell populations.

Conclusions: This study establishes a novel GA-CAF-TME axis that modulates ICI response in HCC. By reprogramming CAF metabolism, GA creates a less immunosuppressive TME, thereby enhancing ICI efficacy. These findings highlight GA as a promising biomarker and a potential therapeutic adjunct for HCC immunotherapy.

Keyword(s): hepatocellular carcinoma, immunotherapy, cancer associated fibroblast, galacturonic acid

1146 - Treatment with direct acting antivirals in HCV infected patients after liver transplantation

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Background / Hypothesis:

Chronic hepatitis C virus (HCV) infection remains a major indication for orthotopic liver transplantation (OLTx) worldwide. Direct-acting antivirals (DAAs) have revolutionised the management of HCV, offering highly effective and well-tolerated treatment options, even in populations that are difficult to treat. However, real-world data on the effectiveness and safety of DAA therapy in liver transplant recipients remain limited, particularly from Central and Eastern Europe.

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This study aimed to evaluate the clinical characteristics, effectiveness, and safety of DAA therapy in liver transplant recipients with chronic hepatitis C in a nationwide, real-world cohort.

Materials and Methods:

A retrospective analysis was performed of all consecutive adult patients who underwent OLTx before starting DAA therapy between July 2015 and December 2024 in the EpiTer-2 project, which included 20,586 patients treated because of chronic hepatitis C.

Results:

A total of 141 patients participated in the study, with most being men (66%) and aged 50 years or older. In 81 patients (57%), the reason for transplantation was cirrhosis without liver cancer, in 58 patients (41%), liver transplantation was performed due to liver cancer, and in 2 patients, the reason for the procedure was unknown. Most patients (80%) had comorbidities, and nearly a quarter of the population had cirrhosis of the transplanted liver at the start of antiviral therapy, including 10 patients with decompensated cirrhosis. The predominant HCV genotype was GT1b. The median time from OLTx to initiation of antiviral therapy was 24 months. Tacrolimus-based immunosuppression was used in 86% of patients. The most commonly used DAA regimens were OBV/PTV/r±DSV±RBV and SOF/LDV±RBV. Overall, SVR was achieved in 96.4% of patients in the intention-to-treat analysis and in 98.6% after excluding patients lost to follow-up. Treatment was well tolerated: 57% of patients experienced at least one adverse event, mainly of mild severity. Serious adverse events were reported in five patients, leading to discontinuation of treatment in two patients. During DAA treatment and 12 weeks of follow-up after treatment, two deaths were reported, both in patients who had undergone transplantation due to hepatocellular carcinoma. Subgroup analysis by time from OLTx to antiviral therapy (≤ 24 vs. > 24 months) revealed no differences in effectiveness and safety despite some baseline clinical variations.

Conclusions:

DAA therapy in liver transplant recipients with chronic HCV infection is highly effective and well-tolerated. These results confirm that DAA treatment regimens can be safely and effectively used in transplant patients, reinforcing their role as the standard of care in this high-risk population.

Keyword(s) : chronic hepatitis C, hepatitis C virus, liver transplantation, direct-acting antivirals

1148 - Deciphering the Mechanism of Aristolochic Acid I Driven Hepatocellular Carcinoma through Integrated Network Toxicology and Bioinformatics

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Background / Hypothesis: Aristolochic acid I (AAI) is a known liver carcinogen, but its molecular mechanisms in hepatocellular carcinoma (HCC) remain incompletely understood. This study aimed to systematically elucidate these mechanisms.

Materials and Methods: This study employed a multidisciplinary strategy: assessing AAI carcinogenicity via ProTox-3.0; identifying its targets using PubChem; and screening HCC-related genes from GEO. Core genes were filtered through network toxicology and Lasso-Cox regression. Molecular docking and dynamics simulations analyzed AAI-protein interactions, while GO, KEGG and GSEA enrichment revealed relevant pathways. Key gene expression and clinical relevance were validated via TCGA and HPA databases.

Results: The carcinogenic potential of the AAI was 0.77. 52 potential targets of AAI-induced HCC were identified. Network toxicology analysis identified 12 core genes including EZH2, FABP5, and RXRA. Lasso-Cox regression analysis identified four core genes that drive HCC progression, including EZH2. Functional enrichment analysis revealed that AAI-HCC targets were significantly enriched in lipid metabolism and the PPAR signaling pathway ($p < 0.05$), with the PPAR signaling pathway being significantly activated in HCC. Molecular docking and MD simulations confirmed the high affinity and stable binding of AAI to EZH2, FABP5, and RXRA. Through in silico and database-based validation, biological samples and clinical data confirmed that these genes were significantly overexpressed in HCC ($p < 0.001$) and were correlated with poor prognosis ($p < 0.01$).

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Conclusions: AAI may synergistically activate the PPAR signaling pathway by targeting EZH2, FABP5, and RXRA, driving lipid metabolism reprogramming and promoting the occurrence and development of HCC.

Keyword(s) : Aristolochic acid I, Hepatocellular carcinoma, Network toxicology, Molecular docking, Molecular dynamics simulation; PPAR signaling pathway.

1150 - Evaluation of high sensitivity HBcrAg and HBsAg assays as biomarkers for occult HBV infection and hepatocellular carcinoma risk

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Background / Hypothesis:

High-sensitivity iTACT assays for hepatitis B core-related antigen (HBcrAg) and surface antigen (HBsAg) can detect extremely low levels of viral activity and may better reflect residual intrahepatic HBV replication than conventional assays. This study evaluated whether iTACT-HBcrAg and iTACT-HBsAg are associated with the risk of hepatocellular carcinoma (HCC) in individuals seronegative for HBsAg and anti-HCV (NBNC) in Taiwan.

Materials and Methods:

A total of 129 newly developed HCC cases and 520 age- and sex-matched non-HCC controls were selected from the community-based REVEAL-NBNC cohort. Serum iTACT-HBcrAg and iTACT-HBsAg were quantified at baseline using chemiluminescent enzyme immunoassays with improved analytical sensitivity. Logistic regression models were applied to estimate the odds ratios (ORs) and 95% confidence intervals (CIs) for HCC risk after adjusting for demographic, metabolic, and viral covariates.

Results:

The detection rates of HBcrAg (15.3%) and HBsAg (31.5%) using iTACT assays were higher than those obtained with conventional assays (HBcrAg: ≥ 1000

U/mL: 12.4%) among HCC cases. iTACT-HBcrAg positivity was significantly associated with an increased risk of HCC [adjusted OR (95% CI): 2.9 (1.4–6.2), $P = 0.006$], and detectable iTACT-HBsAg also showed a positive association with HCC risk [adjusted OR (95% CI): 2.0 (1.2–3.4) [adjusted OR (95% CI): 2.9 (1.4–6.2), $P = 0.006$], $P = 0.010$]. Combined positivity for both markers conferred the highest risk [adjusted OR (95% CI): 4.8 (1.9–12.2), $P = 0.001$].

Conclusions:

High-sensitivity iTACT-HBcrAg and iTACT-HBsAg assays can uncover occult HBV activity and improve HCC risk stratification in NBNC individuals. These findings highlight their potential utility as biomarkers to refine HCC surveillance strategies in HBV-endemic populations.

Keyword(s) : iTACT-HBcrAg; iTACT-HBsAg; occult hepatitis B virus infection; hepatocellular carcinoma; NBNC; biomarker.

1151 - HDV Screening Practices in Chronic Hepatitis B Real world Data from a Decision Support App in the Context of Global Guidance

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Background / Hypothesis: In people with chronic hepatitis B, Asian Pacific Association for the Study of the Liver (APASL) guidelines recommend that healthcare professionals (HCPs) systematically look for coinfection with hepatitis delta virus (HDV). Consistent with this, updated WHO 2024 guidelines recommend that people who are Hepatitis B surface antigen (HBsAg) positive undergo anti-HDV antibody testing, with reflex HDV RNA testing (where available) following a positive anti-HDV antibody result. One potential way to measure adherence to these guidelines is by analyzing data from the Hep B Consult app, which delivers point-of-care clinical recommendations based on a patient's clinical data entered by an HCP.

Materials and Methods: We analyzed patient cases entered in the Hep B Consult app between 2024-2025, focusing on chronic hepatitis B cases where HCPs chose APASL as their preferred guideline. We then

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reviewed those cases to identify whether HCPs chose to perform anti-HDV antibody testing and, where applicable, reflex HDV RNA testing

Results:Between April 2024 and October 2025, HCPs entered 4819 HBsAg-positive patient cases in the Hep B Consult app under the APASL guideline framework. Anti-HDV antibody testing was performed in 3909 cases (81.1%) and 910 cases (18.9%) had no antibody testing. Among the 901 cases that were antibody positive, 799 (88.7%) had reflex HDV RNA testing performed and 102 (11.3%) had no RNA testing. These results quantify adherence to WHO 2024 and APASL recommendations for universal anti-HDV antibody screening and reflex RNA testing in chronic hepatitis B.

Conclusions:In this analysis of data from the Hep B Consult app, in most cases, HCPs adhered to WHO 2024 and APASL recommendations for HDV screening and reflex testing for patients with chronic hepatitis B. However, in almost one fifth of cases, HCP failed to screen for HDV antibody, and in more than one tenth of cases, they failed to do reflex HDV RNA testing. This uncovers an opportunity to strengthen adherence guidelines, which recommend screening all people with chronic hepatitis B for HDV.

Keyword(s) : Hepatitis B virus (HBV), Hepatitis D virus (HDV), Screening adherence, Clinical decision support Real-world data

1152 - Clinical Efficacy of Robot Assisted Biliary Enteric Reconstruction in Radical Resection of Hilar Cholangiocarcinoma Single Center Practical Experience

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Background / Hypothesis:To explore the safety, feasibility, and efficacy of robot-assisted biliary-enteric reconstruction during radical resection of hilar cholangiocarcinoma, providing evidence for clinical treatment.

Materials and Methods:retrospective analysis was performed on the clinical data of 27 patients who

underwent robot-assisted radical resection of hilar cholangiocarcinoma with concurrent biliary-enteric reconstruction at our institution from January 2019 to December 2023 (follow-up completed by December 2024). Operative time, blood loss, postoperative complications, hospital stay, 90-day mortality, and short-term survival (6-month and 1-year) were recorded.

Results:All 27 surgeries were successfully completed without conversion to open surgery. The total operative time was (265.3±42.5) minutes, with biliary-enteric reconstruction time of (40.2±10.3) minutes and intraoperative blood loss of (150.5±65.2) ml. The overall postoperative complication rate was 55.5% (15/27), including biliary fistula (7.1%), abdominal infection (4.8%), incisional infection (11.1%), pneumonia (9.3%), and other minor complications (23.2%). All complications were resolved with conservative management or percutaneous drainage. The median postoperative hospital stay was (10.5±2.3) days, and no mortality occurred within 90 days postoperatively. During the follow-up period, the 6-month survival rate was 86.2% and the 1-year survival rate was 72.5%, with no severe long-term complications related to biliary-enteric reconstruction observed.

Conclusions:Robot-assisted biliary-enteric reconstruction in radical hilar cholangiocarcinoma resection is safe and feasible, with less intraoperative bleeding and faster recovery. The preliminary long-term survival data support its potential as an effective surgical option for eligible patients.

Keyword(s) : Robot-assisted surgery, Hilar cholangiocarcinoma, Radical resection, Biliary-enteric reconstruction, Clinical efficacy

1154 - Clinical and epidemiological profile of Hepatitis A outbreak among medical students at Shaheed Ziaur Rahman Medical College hostel A cross sectional study

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Background: Hepatitis A virus (HAV) continues to be a

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significant public health concern, causing acute viral hepatitis outbreaks, particularly in South Asia's overcrowded settings characterized by suboptimal sanitation. In late 2023, a cluster of jaundice cases emerged among residents of the Shaheed Ziaur Rahman Medical College (SZMC) hostel in Bogura, prompting a detailed epidemiological investigation.

Objectives: To investigate the clinical and epidemiological features of the outbreak, identify the source and modes of transmission, assess existing preventive measures, and propose evidence-based strategies for future risk mitigation.

Methods: A descriptive cross-sectional study was conducted from January to June 2025 to investigate the outbreak that occurred in late 2023. Cases were defined as students residing in the hostel with clinical signs of acute hepatitis and laboratory-confirmed IgM anti-HAV positivity via ELISA (98.9% sensitivity, 99.4% specificity). A structured questionnaire captured demographic, clinical symptoms, water and food consumption habits, and personal hygiene data. An environmental survey of the hostel's water sources and sanitation infrastructure was conducted to identify potential contamination points.

Results: A total of 22 confirmed HAV cases were identified. The affected population comprised 62.1% male and 37.9% female students. The mean age of the confirmed students was 23.1 years (SD ± 1.1). Common symptoms included jaundice (100%), fatigue (95.2%), anorexia (85.7%), and nausea/vomiting (81%). Epidemiological investigation revealed a strong association with specific risk factors: a majority of students (77%, n=17) reported consuming water from a tube well found to be adjacent to a cracked underground sewage pipe with evidence of leakage. Furthermore, the college canteen was identified as a highly probable common food source, with 72% (n=21) of affected students reporting consumption from it, and observations noted unhygienic conditions. Suboptimal hygiene practices, including inconsistent handwashing (only 33.3% of students reported consistent hand-washing before meals), were common, and none of the affected students had received HAV vaccination.

Conclusion: The outbreak was most likely a common-source event caused by fecal contamination of the hostel water supply, further amplified by unhygienic conditions in the college canteen and poor personal hygiene practices in communal areas. Targeted

interventions, including water source repair, hygiene promotion, and health education, are crucial for effective control. Regular monitoring of water systems sustained public health awareness campaigns, and consideration of HAV vaccination in high-risk communal settings are strongly recommended for preventing future outbreaks.

Keyword(s) : Hepatitis A virus, waterborne outbreak, hostel, Bangladesh, IgM anti-HAV, contaminated water

1155 - MASLD AND GLYCEMIC IMPROVEMENT AFTER SUSTAINED VIROLOGIC RESPONSE IN TYPE 2 DIABETES PATIENTS WITH CHRONIC HEPATITIS C A THREE YEAR FOLLOW UP STUDY

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Background / Hypothesis:

DAA are the gold standard for chronic HCV treatment to achieve a SVR in nearly all patients. HCV infection can directly influence glucose metabolism and MASLD (metabolic dysfunction-associated steatotic liver disease).

Among patients with both type 2 diabetes (T2D) and HCV, SVR may improve glycemic control and hepatic steatosis, but available studies about fibrosis are limited. Our study herein aims to examine the effects of SVR on long-term glycemic control and MASLD.

Materials and Methods:

We conducted a retrospective cohort study of T2D patients from our hospital who had a positive HCV RNA test and received DAA between 25 Jan, 2019 and 22 Jan, 2020. SVR was defined as non-detectable HCV RNA 12 weeks after DAA therapy. Steatosis was assessed with Attenuation Imaging (ATI) developed by Canon company. Fibrosis was assessed by Liver stiffness measurement (LSM) developed by FibroScan. All parameters were statistically evaluated by Friedman test (SPSS version 24.0).

Results:

In total, 93 patients with both T2D and HCV treated by DAA were enrolled, and all achieved SVR. The mean age was 68 y/o. Patients were divided into 2 groups, group 1 were [HbA1c $\leq 7\%$ (N=51)] and group 2 were [HbA1c $> 7\%$ (N=42)].

For group 1, the HbA1c levels were 6.30, 6.30, 6.50 and

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6.50 at entry, 1, 2, and 3 years FU respectively ($P=0.071$). The ATI levels (dB/cm/MHz) were 0.66, 0.60, 0.60, and 0.60 at entry, 1, 2, and 3 years FU respectively ($P<0.001$). The LSM levels (kPa) were 7.41, 7.40, 7.40 and 7.40 at entry, 1, 2, and 3 years FU respectively ($P<0.001$). SVR did not significantly improve HbA1c but significantly improve MASLD in group 1.

For group 2, the HbA1c levels were 8.20, 7.30, 7.30 and 7.05 at entry, 1, 2, and 3 years FU respectively ($P<0.001$). The ATI levels were 0.89, 0.70, 0.70, and 0.70 at entry, 1, 2, and 3 years FU respectively ($P<0.001$). The LSM levels were 7.90, 7.55, 7.50 and 7.50 at entry, 1, 2, and 3 years FU respectively ($P<0.001$). SVR was strongly associated with HbA1c reduction and MASLD improvement after DAA through 1 to 3 years FU in group 2.

Conclusion: DAA therapy in patients with chronic HCV and type 2 DM leads to significant and durable improvements in both glycemic control and MASLD. HCV clearance not only halts liver injury but also exerts long-term metabolic benefits.

Keyword(s) : MASLD, HCV, T2DM,

1160 - Study on the Mechanism of Samhd1 Mediated NEMO Polyubiquitination in Liver Ischemia Reperfusion Injury

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Background / Hypothesis:

Hepatic ischemia reperfusion injury (IRI) is one of the major pathological processes in liver transplantation and various liver surgeries. To date, there is still a lack of effective diagnosis and treatment strategies for hepatic IRI, and one of the root causes is that its mechanism has not been fully elucidated. This study aims to explore the molecular mechanism by which Samhd1 participates in the regulation of hepatic IRI, providing a research foundation and theoretical basis for the prevention and treatment of hepatic IRI.

Materials and Methods:

This study established a classic 70% liver ischemia-reperfusion injury (IRI) model in mice. The expression and localization of key molecules were detected using methods such as immunoprecipitation, immunofluorescence, quantitative real-time polymerase chain reaction (qRT-PCR), and enzyme-linked immunosorbent assay (ELISA). The pathological damage and liver function of mice in each group were

examined through hematoxylin and eosin (HE) staining, terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) assay, and microplate method.

Results:

We have discovered that Samhd1 is a key molecule exacerbating hepatic ischemia - reperfusion injury (IRI). Down - regulation of Samhd1 can significantly alleviate hepatic IRI, while over - expression of Samhd1 yields the opposite effect. Mechanistically, Samhd1 can mediate the polyubiquitination modification of NEMO, induce the activation of the IKK inflammatory complex, and enhance its stability, ultimately leading to the phosphorylation and combined translocation of multiple subunits of the IKK family. Inhibition of IKK can reverse hepatic IRI caused by Samhd1 enrichment.

Conclusions:

In conclusion, this study has revealed the potential functions and roles of Samhd1 in liver ischemia-reperfusion injury (IRI). Targeting Samhd1 may be a potential strategy for treating liver IRI.

Keyword(s) : liver transplantation, liver ischemia reperfusion, interactions between proteins

1161 - Recurrence and Post Liver Transplant Outcomes in Hepatocellular Carcinoma Experience from the Main Center of Iran

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) recurrence after liver transplantation (LT) remains a major challenge in post-transplant management, significantly impacting patient outcomes. However, the burden of recurrence and its prognostic factors are not fully understood. This study aimed to (i) characterize recurrence patterns and survival outcomes among liver transplant recipients with HCC, and (ii) evaluate the prognostic significance of the Model for End-Stage Liver Disease (MELD) score.

Materials and Methods:



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Between April 2017 and May 2025, 160 patients with HCC underwent LT at Abu-Alisina Hospital, Shiraz, Iran, contributing to 477.4 person-years of follow-up. Post-transplant recurrence and mortality were retrospectively ascertained from medical and registry data. Event rates were expressed per 1,000 person-years. Secondary analyses included age- and sex-matched case-control comparisons (38 deaths vs. 38 survivors, and 7 recurrences vs. 24 non-recurrences) and multivariable logistic regression adjusting for demographic and graft-related variables. All analyses were conducted using Python 3.11 with appropriate statistical libraries.

Results:

During a median follow-up of 2.67 years (IQR: 0.43–4.9), 55 deaths occurred, corresponding to a mortality rate of 115.2 per 1,000 person-years (95% CI: 84.8–145.6). The median time to death was 0.26 years (IQR: 0.04–1.83). Mortality was highest in the early post-transplant period: 31 deaths (56%) occurred within the first year, 6 between 1–2 years, 7 between 2–3 years, and 5 beyond three years. In multivariable analyses, MELD score was not associated with mortality (adjusted OR: 0.97; 95% CI: 0.91–1.05; $p = 0.457$). HCC recurrence was infrequent, affecting 8 recipients (5%), yielding an incidence rate of 16.8 per 1,000 person-years (95% CI: 7.2–34.5). The median time to recurrence was 1.6 years (range: 0.01–3.83). Most recurrences (62.5%) developed within two years—three within the first year—while three (37.5%) occurred beyond two years, including two after the third year. In adjusted models, higher MELD scores were inversely associated with recurrence risk (adjusted OR: 0.78; 95% CI: 0.61–0.99; $p = 0.041$).

Conclusions:

In this cohort of HCC liver transplant recipients, recurrence was relatively rare, but overall post-transplant mortality remained high, particularly during the first year. Most recurrences occurred within the first two years, emphasizing the importance of early, intensive surveillance. Although MELD score did not predict overall mortality, its inverse association with recurrence suggests that pre-transplant liver disease severity may influence tumor behavior or recurrence risk. These findings support individualized post-transplant monitoring strategies and provide hypothesis-generating evidence for further investigation into the biological and clinical mechanisms linking liver function and HCC recurrence.

Keyword(s) : Hepatocellular carcinoma; liver transplantation; HCC recurrence; post-transplant mortality; MELD score; risk stratification

1166 - Endoplasmic Reticulum Stress Mediated PPAR α DDX3X Interaction Modulates Con A Induced Liver Injury in Mice

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Background / Hypothesis:

Dysfunction of endoplasmic reticulum (ER) stress is involved in the pathogenesis of immune-mediated liver injury, but its molecular mechanism has not been fully elucidated. Previous studies have shown that PPAR α and DDX3X play important roles in ER stress-related dysfunction. This study further explored how PPAR α and DDX3X interact to regulate ER stress-related dysfunction in immune-mediated liver injury.

Materials and Methods:

Liver injury was induced in C57BL/6J mice via concanavalin A (Con A). DDX3X hepatocyte-specific knockout (DDX3X Δ Hep) mice and control (DDX3X^{fl/fl}) mice were utilized to investigate the role of DDX3X in liver injury. 4-PBA was used to inhibit ER stress, while siRNA, AAV-shRNA, and LV were used to regulate PPAR α expression. Tunicamycin (TM) was used to induce ER stress in primary hepatocytes, and autoimmune hepatitis (AIH) patients were included for validation.

Results:

Inhibition of ER stress alleviates ConA-induced liver injury by increasing PPAR α expression and reducing both DDX3X levels and phosphorylation in the liver. PPAR α activation attenuates liver injury and suppresses the phosphorylation of DDX3X, which is regulated by ER stress. A phospho-deficient DDX3X mutant (p.T323A) significantly reduces ConA-induced liver injury in mice. Mechanistically, phosphorylation of DDX3X promotes its cytoplasmic–nuclear translocation and hepatocyte apoptosis during ER stress. Moreover, PPAR α interacts with the DDX3X helicase domain in the cytoplasm, masking the phosphorylation site (p.323), to inhibit the nuclear translocation of DDX3X and decrease the downregulation of CHOP expression. Additionally, colocalization of PPAR α and DDX3X is observed in the livers of AIH patients, elevated serum DDX3X levels were linked to AIH-associated liver failure, and sustained reduction of DDX3X was

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correlated with a better prognosis.

Conclusions:

This study highlights the role of the PPAR α -DDX3X interaction in regulating DDX3X phosphorylation during ER stress and promoting DDX3X nuclear translocation and hepatocyte apoptosis. Therefore, targeting the ER stress-PPAR α -DDX3X pathway represents a novel therapeutic approach for immune-mediated liver injury.

Keyword(s) : Liver injury, Endoplasmic Reticulum (ER) stress, DEAD-box helicase 3, X-linked (DDX3X), Peroxisome Proliferator-activated Receptor alpha (PPAR α), Autoimmune Hepatitis (AIH)

1169 - Associations between glycaemic indices and non invasive markers of hepatic steatosis and steatohepatitis in Chinese adults with prediabetes

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Background:

Metabolic dysfunction-associated steatotic liver disease (MASLD) and its progressive form, metabolic dysfunction-associated steatohepatitis (MASH), are leading causes of chronic liver disease in Asia and worldwide. While MASLD and MASH are strongly linked with insulin resistance and chronic hyperglycaemia in type 2 diabetes mellitus, their associations with dysglycaemia in prediabetes remain insufficiently explored. This study investigated associations between glycaemic indices and non-invasive markers of MASLD and MASH risk in a Chinese cohort with prediabetes.

Methods:

This cross-sectional study included 173 adults with prediabetes recruited from a community metabolic screening programme in Hong Kong. Participants underwent an oral glucose tolerance test and transient elastography (FibroScan) to assess glycaemic and hepatic parameters. Glycaemic indices included fasting, 1-hour, and 2-hour plasma glucose levels and HbA1c. Hepatic steatosis and MASH risk were evaluated using the controlled attenuation parameter (CAP) and FibroScan-AST (FAST) score, respectively. Associations between each glycaemic index-liver parameter pair were assessed using bivariate correlation and multivariable linear regression analyses.

models were progressively adjusted: Base model (age, sex, alcohol use) and Model 1 (Base model plus physical and sedentary activity).

Results:

Mean age was 60.5 $\pm$ 7.5 years; 38.2% were male. MASLD was present in 60.0% of participants, with a mean CAP of 262.9 $\pm$ 58.1 dB/m. The median (interquartile range) FAST score was 0.08 (0.09), indicating a low overall MASH risk. For CAP, only 2-hour glucose showed a significant correlation in unadjusted analysis (r = 0.20, p = 0.010). This correlation persisted as an independent association after adjustment for age, sex, and alcohol use (β = 0.23, p = 0.003) and remained significant after further adjustment for physical and sedentary activity (β = 0.18, p = 0.032). Fasting glucose, 1-hour glucose, and HbA1c were not significantly associated with CAP in any model (all p > 0.05). In contrast, none of the glycaemic indices showed significant correlations or adjusted associations with MASH risk assessed by the FAST score.

Conclusions:

In this study cohort, 2-hour post-load glucose showed the strongest association with hepatic steatosis, while no glycaemic index was linked to MASH risk. These findings highlight that post-load dysglycaemia, an established driver of steatosis in diabetes, is already associated with hepatic fat accumulation at the prediabetic stage and may represent an early intervention target. Larger, longitudinal studies are necessary to clarify causality and clinical implications for MASLD prevention.

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Keyword(s) : MASLD, MASH, prediabetes, hepatic steatosis, glycaemic indices, FAST score

1171 - Optimizing Renal and Urological Outcomes in Liver Transplant Recipients A Urologist Driven Peri Transplant Care Pathway at a Tertiary Transplant Center

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Background / Hypothesis:

Background



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Renal dysfunction and urological complications significantly influence post-liver transplantation (LT) morbidity, ICU stay, and long-term graft survival. While transplant hepatology and surgery receive primary focus, structured urology involvement remains under-recognized despite the essential interplay of fluid balance, nephrotoxic immunosuppression, catheter-associated complications, and hepatorenal syndrome (HRS).

Aim

To evaluate the impact of a Urologist-led peri-transplant renal preservation and urological complication prevention protocol on post-LT outcomes.

Methods

A prospective observational cohort of 142 liver transplant recipients between 2021–2024 was analyzed. Patients were divided into:

Group A (n=68): Standard transplant team management

Group B (n=74): Managed under the Urology-Integrated Care Protocol (UICP), which included:

- Pre-transplant renal reserve scoring
- Early CRRT/HRS recognition algorithm
- Strict nephrotoxic drug avoidance chart
- Standardized catheterization & stent handling protocol
- Post-LT obstructive uropathy screening ultrasound algorithm

Primary endpoints: postoperative acute kidney injury (AKI), need for dialysis, urinary infection rate, ICU stay duration, and 90-day survival.

Results

Group B demonstrated:

Reduced AKI incidence: 38.2% → 17.5% ($p<0.01$)

Dialysis requirement: 14.7% → 6.8% ($p<0.05$)

Catheter-associated UTI: 26.4% → 9.4% ($p<0.001$)

Median ICU stay: 9 days → 6 days

90-day survival: 86.7% → 93.2%

No increase in operative time or cost was observed.

Conclusion

Early and continuous involvement of Urology in the peri-transplant phase significantly improves renal preservation, decreases urological complications, and enhances early survival following liver transplantation. This multidisciplinary protocol is easily adoptable and offers a scalable, cost-effective improvement in LT outcomes.

Urologists should be integral members of the liver transplant team rather than optional consultants.

Materials and Methods:

Results:

Conclusions:

Keyword(s) :

1175 - First Clinical Evidence of Temperature sensitive Liquid Embolic Agent TACE Remodeling Immune Microenvironment in Unresectable HCC Multicenter Propensity Weighted Cohorts Study

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Background / Hypothesis:

Transarterial chemoembolization (TACE) remains the standard first-line therapy for intermediate-stage HCC; however, conventional embolic agents (e.g., drug-eluting microspheres) display limited vascular penetration and drug delivery efficiency. The temperature-sensitive liquid embolic agent (TempSLE), a novel phase-transition material, has demonstrated promise in preclinical studies but lacks clinical validation in HCC. This study aims to compare the efficacy, safety, and immunomodulatory effects of TempSLE-TACE (T-TACE) versus drug-eluting bead TACE (D-TACE) in unresectable hepatocellular carcinoma (HCC).

Materials and Methods:

This multicenter retrospective study included 287 patients with unresectable HCC treated with TempSLE-TACE (T-TACE; n=101) or D-TACE (n=186) across five tertiary centers in China (2021-2023). Propensity score matching (PSM; 1:1) yielded 93 matched pairs. Evaluated outcomes included tumor response (mRECIST), overall survival (OS), progression-free survival, and safety. Histopathological and immune microenvironment assessments were performed on resected specimens.

Results:

After PSM, the T-TACE group showed a higher objective response rate (58.06% vs. 43.01%, $P=0.040$) and longer median OS (20 vs. 18 months, $P=0.035$). Patients with China Liver Cancer (CNLC) stage IIb derived the greatest OS benefit (hazard ratio=3.092, $P<0.001$). The incidences of fever (23.96% vs. 56.98%, $P<0.001$) and abdominal pain (44.79% vs. 63.74%, $P=0.003$) were lower in the T-TACE group, whereas hand-foot skin reactions were more frequent (40.59% vs. 23.83%, $P=0.001$). No significant differences in severe adverse events (grade ≥ 3) were observed. TempSLE achieved complete vascular occlusion and uniform tumor necrosis; D-TACE resulted in incomplete

embolization with residual viable tumor cells. TempSLE also enhanced CD8+ T-cell infiltration and dendritic cell activation at tumor margins, suggesting an immunogenic effect.

Conclusions:

T-TACE offers superior tumor control and survival outcomes relative to D-TACE in patients with unresectable HCC—particularly in CNLC stage IIb—and is associated with a distinct safety profile and potential immunomodulatory properties. These results support its clinical adoption as an innovative embolic strategy.

Keyword(s) : Intermediate-stage HCC, Temperature-sensitive liquid embolic agent, TACE, Immune Microenvironment

1180 - Study of Serum Trace Elements (Zn Cu Mg) in Hepatic Encephalopathy

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Background / Hypothesis:

Hepatic encephalopathy (HE) is a severe neuropsychiatric complication of liver cirrhosis, arising from impaired hepatic detoxification and systemic metabolic dysfunction. While ammonia has long been considered the primary neurotoxin, the role of trace elements—particularly zinc (Zn), copper (Cu), and magnesium (Mg)—in HE pathogenesis is increasingly recognized. These elements influence ammonia metabolism, oxidative stress, and neuronal excitability. The present study evaluated serum levels of Zn, Cu, and Mg and their correlation with the severity of hepatic encephalopathy.

Materials and Methods:

A cross-sectional observational study was conducted at the Department of General Medicine, Atal Bihari Vajpayee Institute of Medical Sciences and Dr. Ram Manohar Lohia Hospital, New Delhi, from February 2024 to June 2025. A total of 105 adult patients diagnosed with hepatic encephalopathy were included. Serum Zn, Cu, and Mg levels were measured using colorimetric methods, and HE was graded according to West Haven criteria. Correlation with liver dysfunction was assessed using the Child-Pugh classification. Statistical analyses included Pearson correlation and linear regression.

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Results:

Elevated serum copper was observed in 94.3% of patients and showed a strong positive correlation with HE severity ($r = 0.938$, $p < 0.001$; $R^2 = 0.851$). Zinc deficiency was noted in 64.8%, demonstrating a negative correlation ($r = -0.316$, $p = 0.001$) with HE grades.

Hypomagnesemia occurred in 43.8%, with a strong inverse relationship ($r = -0.834$, $p < 0.001$; $R^2 = 0.662$) to HE severity. Trace element imbalances paralleled worsening Child–Pugh class and biochemical decompensation.

Conclusions: Alterations in serum Zn, Cu, and Mg are significantly associated with the severity of hepatic encephalopathy and liver dysfunction. Routine monitoring and correction of these trace element abnormalities may serve as valuable adjuncts in managing cirrhosis patients at risk of HE.

Keyword(s) : Hepatic encephalopathy, Zinc, Copper, Magnesium, Cirrhosis, Trace elements

1181 - Familial Mediterranean Fever as a cause of hepatic amyloidosis and decompensated liver cirrhosis

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Background / Hypothesis:

Familial Mediterranean Fever (FMF) is an autoinflammatory disorder characterized by recurrent fever and serosal inflammatory episodes, often leading to secondary (AA) amyloidosis. While renal involvement is typical, hepatic amyloidosis is rare. This case highlights that prolonged FMF may result not only in hepatic amyloidosis, but can also rarely lead to liver cirrhosis.

Materials and Methods:

A 71-year-old woman with a 17-year history of genetically confirmed FMF (MEFV gene homozygous mutation M680I/M680I) presented with nausea, loss of

appetite, weight loss, abdominal distension, sleep inversion, and fatigue. She reported recurrent diarrhea over a long period. Physical and laboratory evaluations, imaging studies, and histopathological analyses were performed, including liver and intestinal biopsies, Congo red staining, immunohistochemistry, and serologic tests for viral and autoimmune markers.

Results:

CT imaging revealed ascites and nodular hepatic contours consistent with cirrhosis. Liver biopsy demonstrated steatotic liver disease, cirrhosis, and Congo red–positive deposits showing birefringence under polarized light. Immunohistochemistry confirmed AA-type amyloid. Paracentesis revealed benign ascites with SAAG > 1.1 g/dL, indicating portal hypertension. Immunohistochemistry of gastrointestinal biopsies also showed AA amyloid deposition, explaining the chronic diarrhea. Laboratory findings included Hgb 108 g/L, PLT 169×10^9 /L, ESR 42 mm/h, albumin 26.46 g/L, total protein 61.2 g/L, total bilirubin 49.7 μ mol/L, creatinine 125 μ mol/L, CRP 46.46 mg/L, uric acid 776 μ mol/L, LDL 1.62 mmol/L, HDL 0.38 mmol/L, PT 22.1 s, iPT 47%, INR 1.77, alpha-fetoprotein 2.39 IU/mL. BMI was 22.2 with normal blood glucose, indicating no metabolic risk factors. The reduced HDL level in this patient is explained by impaired hepatic lipoprotein synthesis due to cirrhosis rather than cardiometabolic disease. Viral hepatitis (HBV/HCV/HIV), Wilson's disease, and alpha-1 antitrypsin deficiency were excluded. Despite anti-AMA reactivity, normal IgG/IgM levels and absence of autoimmune histological features excluded AIH and PBC. The colchicine dose was increased to 1.8 mg daily, and rifaximin, diuretics, and albumin were added.

Conclusions:

FMF is common in people of Mediterranean origin but it can affect people in any ethnic group. FMF can rarely lead to hepatic amyloidosis and cirrhosis. When common causes of liver disease are excluded, and a personal or familial history of FMF is present, FMF-related hepatic involvement should be considered in the differential diagnosis of liver cirrhosis. Early recognition and optimization of colchicine therapy may help prevent disease progression.

Keyword(s) : Familial Mediterranean Fever, FMF, Cirrhosis, Amyloidosis

1182 - The NF κ B TNFAIP3 axis attenuates MAFLD via activation of hepatocyte autophagy

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Background / Hypothesis:

Emerging evidence indicates that tumor necrosis factor α -induced protein 3 (TNFAIP3) is markedly upregulated in the livers of patients with metabolic dysfunction-associated fatty liver disease (MAFLD). However, its functional role in MAFLD remains largely unclear.

Materials and Methods:

TNFAIP3 expression was analyzed in several diet-induced MAFLD mouse models using public transcriptomic datasets. In vitro, we assessed the effects of TNFAIP3 overexpression and knockdown in palmitic acid-treated HepG2 cells on lipid accumulation, autophagy, and key metabolic pathways. In vivo, high-fat diet-induced mouse models were used to validate the protective role of TNFAIP3. Autophagy inhibitors were applied to determine whether effects of TNFAIP3 were autophagy-dependent, and NF- κ B signaling involvement was examined using pharmacological inhibitors.

Results:

TNFAIP3 was consistently upregulated in the livers of all tested MAFLD mouse models. In the fructose-palmitate-cholesterol diet-induced model, TNFAIP3 expression was substantially elevated specifically in isolated primary hepatocytes, indicating that the increase occurs predominantly in hepatic parenchymal cells. In palmitic acid-treated HepG2 cells, TNFAIP3 overexpression markedly reduced lipid droplet accumulation, whereas knockdown exacerbated lipid deposition. In vivo, hepatic overexpression of TNFAIP3 alleviated steatosis, improved insulin sensitivity, and modulated lipid metabolism-associated pathways. These protective effects were abolished by autophagy inhibitors, confirming that TNFAIP3 exerts its beneficial role in an autophagy-dependent manner.

Mechanistically, TNFAIP3 upregulation was driven by NF- κ B signaling, as pharmacological inhibition of NF- κ B significantly decreased TNFAIP3 expression.

Conclusions:

The NF- κ B-TNFAIP3 axis regulates hepatic lipid metabolism and protects against liver injury in MAFLD, primarily through autophagy. These findings highlight TNFAIP3 as a potential therapeutic target for MAFLD and related metabolic liver diseases.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease; Autophagy; Tumor necrosis factor α -induced protein 3

1183 - Study on the Mechanism of FBXW5 Inducing MKRN1 Ubiquitination and Degradation in Reducing Liver Ischemia Reperfusion Injury

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Background/Hypothesis:

Hepatic ischemia-reperfusion injury (IRI) has long been a major concern for liver transplant and various liver surgery patients, severely affecting prognosis. The research team previously discovered that FBXW5 significantly alleviated liver IRI, but the mechanism remained unclear. This study aims to clarify the molecular mechanism by which FBXW5 regulates liver IRI, providing a research foundation and theoretical basis for the prevention and treatment of liver IRI.

Materials and Methods:

We establish a 70% liver ischemia-reperfusion injury (IRI) model in classic mice. Detect the expression and localization of key molecules using IHC, WB, immunofluorescence, qRT-PCR, ELISA, etc. Detect the pathological damage and liver function of mice in each group using HE, TUNEL, and microplate methods.

Results:

We have discovered that MKRN1 is an important signal transduction protein that exacerbates liver IRI. The MKRN1-dependent LKB1 ubiquitination is a key step in liver IRI. Mechanistically, FBXW5 can mediate the polyubiquitination and degradation of MKRN1, thereby down-regulating the sterile inflammatory signaling pathway.

Conclusions:

In conclusion, this study has revealed the potential functions and roles of FBXW5/MKRN1 in liver ischemia-reperfusion injury (IRI). Targeting FBXW5 may be a potential strategy for treating liver IRI.

Keyword(s) : liver transplantation, liver ischemia reperfusion, interactions between proteins

1184 - Study on the Mechanism of TRIM69 Binding to CRYAB Promoting TRAF6 Autoubiquitination and Aggravating Liver Ischemia Reperfusion Injury

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Background/Hypothesis:

Hepatic ischemia-reperfusion injury (IRI) is a serious complication that is inevitable in liver transplantation and liver surgical operations. Its pathophysiological process involves a complex network of molecular mechanisms, and its exact mechanism has not yet been fully elucidated. Due to the lack of specific treatment methods, the clinical management of liver IRI still faces significant challenges, directly affecting the prognosis and outcome of surgical patients. Previous studies have shown that α B-crystallin (CRYAB) has a significant protective effect in liver IRI, but its specific molecular mechanism still needs to be further explored. This study aims to systematically reveal the protective mechanism of CRYAB in liver IRI, and focus on exploring the key regulatory role of TRIM69 in the process of liver IRI through regulating CRYAB-mediated TRAF6 ubiquitination modification. This research will provide important theoretical basis for clarifying the molecular regulatory network of liver IRI and developing targeted intervention strategies.

Materials and Methods:

This study employed a standardized 70% liver ischemia-reperfusion injury (IRI) mouse model. The expression changes of target proteins were quantitatively analyzed using Western blot technology. Protein-protein interactions were verified by the co-immunoprecipitation (Co-IP) method. Subcellular localization was analyzed by immunofluorescence staining, and the levels of inflammatory factors were determined by enzyme-linked immunosorbent assay (ELISA). In the pathological assessment stage, the morphological changes of liver tissue were observed through the hematoxylin-eosin (HE) staining system. Cell apoptosis was detected by the terminal deoxynucleotidyl transferase-mediated dUTP nick-end labeling (TUNEL) method. Meanwhile, the activities of serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were determined by the microplate method.

Results:

The study found that TRIM69 interacts with CRYAB, removing the latter's silencing effect on TRAF6's self-ubiquitination, thereby promoting the inflammatory cascade reaction of the IKK pathway. CRYAB functions as a molecular chaperone and plays an important anti-inflammatory role. Meanwhile, TRIM69 can competitively bind to CRYAB, thereby reducing its silencing effect on TRAF6, thereby activating the

classical and non-classical IKK regulatory subunits, leading to the translocation of p65 into the nucleus.

Conclusions:

This study has revealed the potential functions of TRIM69 and CRYAB in liver ischemia-reperfusion injury (IRI), providing supplementary information on the mechanism of liver IRI.

Keyword(s) : liver transplantation, liver ischemia reperfusion, TRIM

1186 - Timing Transplantation versus Medical Treatment in Acute Liver Failure and Associated Outcomes Insights from an Indian High Volume Transplant Centre

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Background / Hypothesis:

Acute liver failure (ALF) is a life-threatening condition in which timely liver transplantation (LT) is crucial for survival. The major challenge lies in determining the optimal timing and selection between continued medical management and transplantation. We present our single-center experience with living donor liver transplantation (LDLT) in the management of ALF.

Materials and Methods:

We analyzed data from 70 ALF patients presenting between June 2017 and February 2025, focusing on clinical features, interventions, and outcomes. Transplant timing was individualized based on the overall clinical course, considering worsening encephalopathy, rising intracranial pressure, multi-organ involvement, increasing inotropic needs, and rising INR, bilirubin, lactate and ammonia levels, rather than any single criterion.

Results:

Among 70 patients with ALF with a median age of 24.5

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years (IQR: 13–35 years), 26 (37%) were women and 28 (40%) were children below 18 years of age. The leading cause of ALF was hepatitis A virus infection in 39 patients (55.7%), followed by indeterminate etiology in 14 (20%). Overall, 40% of the cohort survived with their native liver. Transplant timing was guided by the dynamic clinical course. Patients were categorized into three groups: liver transplant recipients (n=34), survivors with native liver (SNL, n=28), and deceased non-transplanted patients (n=8) (Figure attached). The SNL group had a median hospital stay of 12 days (IQR: 9–20), and 13 of these patients met the King's College Hospital criteria for LT listing. Among the 34 transplant recipients, 10 died postoperatively, most commonly (7/10) due to persistent intracranial hypertension leading to brain death. Patients in the deceased group had multiorgan dysfunction or were deemed too sick for transplantation. On univariate analysis, admission INR, arterial lactate levels, jaundice-to-encephalopathy (J–E) interval, and advanced hepatic encephalopathy predicted the need for LT, while multivariate analysis identified elevated admission arterial lactate and a J–E interval exceeding 7 days as independent predictors for liver transplantation.

Conclusions:

Hepatitis A is the main cause of ALF, with high arterial lactate levels and prolonged J–E interval indicating poor outcomes. Choosing between medical and surgical management in ALF is challenging due to postoperative risks, possible native liver recovery, and unreliable severity scores.

Keyword(s) : Acute liver failure, Liver Transplant, Prognostic indicators

1187 - Optimized Outcomes Using the Medial Reduction Technique for the Left Lateral Segment in Infants Undergoing Living Donor Liver Transplantation

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Background / Hypothesis:

Graft reduction is a crucial technique in pediatric liver transplantation, as the size of the graft significantly impact both short and long-term outcomes. Our center previously published the medial reduction technique,

which was developed to reduce both the weight and thickness of the left lateral segment (LLS). While this technique has shown promise, the short and long-term outcomes need to be further evaluated.

Materials and Methods:

This retrospective study examines infantile living donor liver transplant recipients (<1 year of age) at a single center. Two groups were compared: one group, serving as historic controls, underwent the standard lateral and inferior reduction of the LLS between 2010 and 2016, while the other group received medially reduced left lateral segment (MR-LLS) from 2017 onwards. The study's objective is to compare the immediate and long-term outcomes of these two techniques.

Results:

A total of 88 infants were included: 56 underwent MR-LLS and 32 were historic controls. Median age was 10 months (IQR 8–12) in the MR-LLS group and 6 months (IQR 1–8) in controls ($p < 0.001$); median weight was 7.5 kg (IQR 6.3–8.1) vs. 7.27 kg (IQR 6.2–8.9), respectively ($p > 0.05$). The median PELD score was higher in MR-LLS (24 vs. 19; $p = 0.049$). In the MR-LLS group, the median estimated graft weight was 225 g, reduced weight 59 g, estimated GRWR 3.7%, and actual GRWR 2.95%. Graft thickness was reduced by 21%. In the control group, graft weight and GRWR were higher at 259 g ($p = 0.037$) and 3.25% ($p = 0.178$), respectively. Cold ischemia times were similar, but warm ischemia time was shorter in MR-LLS. ICU stay was also shorter in MR-LLS (17 days [IQR 12–23] vs. 22 days [IQR 21–28]; $p < 0.001$). The MR-LLS group had fewer early postoperative complications. Re-exploration was less frequent (14.3% vs. 34.4%; $p = 0.028$), and 3-month mortality was lower (7.1% vs. 12.5%). Long-term complication rates were similar. Median follow-up was 36 months in MR-LLS and 103 months in controls. Cumulative survival was 89.3% vs. 78.1% ($p = 0.359$) (Figure attached).

Conclusions:

Medial reduction of the left lateral segment effectively reduces graft thickness and has demonstrated excellent short- and long-term outcomes, including shorter ICU stays and fewer immediate surgical complications.

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Keyword(s) : pediatric liver transplantation, medial reduction, left lateral segment graft

1188 - Role and Mechanism of membrane CD100 in Regulating the Activation and Differentiation of HBV Specific CD8 T Cell

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Background / Hypothesis:

CD100/SEMA4D plays pivotal roles in mediating T cell responses as a co-stimulatory molecule. It exists and functions in both membrane-bound and soluble forms. Soluble CD100 (sCD100) promotes the maturation and activation of antigen-presenting cells and enhances effector CD8 T cell responses indirectly, thereby accelerating HBV clearance. However, the direct effector of membrane-bound CD100 in regulating CD8 T cell responses during HBV infection is not well defined.

Materials and Methods:

We examined the impact of mCD100 deficiency on the functional phenotype and differentiation of HBcAg-specific CD8 T cells using separate and co-culture systems in vitro. Furthermore, through adoptive transfer of mCD100-deficient HBV-specific CD8 T cells, we investigated the early impact of mCD100 deficiency on the development of HBV-specific CD8 T cell responses during HBV infection.

Results:

The absence of mCD100 expression significantly decrease HBcAg-specific CD8 T-cell proliferation, activation, effector cytokine production, and expression of Granzyme B and Eomes upon antigen stimulation in vitro. In the acute self-resolving models, the early proliferation and effector differentiation of transferred mCD100 KO TCRtg CD8 T cells were impaired compared to their WT counterparts following HBV challenge. Transcriptomic data revealed that significant downregulation of gene sets associated with the PI3K-Akt, mTOR, NF-κB, and JAK-STAT signaling pathways in CD100KO CD8⁺ T cells.

Conclusions:

Membrane CD100 expressed on resting HBV-specific CD8 T cells may function as a co-stimulatory molecule

by providing a direct stimulatory signal for the early proliferation and effector differentiation of T cells. The observed effects of mCD100 may mediate by activating PI3K-Akt-mTOR, JAK-STAT and NF-κB signaling pathways.

Keyword(s) : CD100, hepatitis B virus, co-stimulatory molecule, CD8 T cell

1191 - Identification of Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) and Risk Factors in People Living With HIV

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Background: HIV infection affects 39.9 million individuals worldwide. With the widespread use of combination antiretroviral therapy (cART) the causes of mortality among people living with HIV (PLWH) have shifted toward comorbidities. Approximately half of PLWH have steatotic liver disease, with 90% of these cases are non-alcoholic fatty liver disease (NAFLD). Metabolic dysfunction-associated steatotic liver disease (MASLD) has recently been adopted as a replacement term for NAFLD. In this study, we aimed to determine the prevalence and risk factors of MASLD in PLWH. Materials and Methods: This single-center, cross-sectional study included PLWH on cART ≥6 months and a control group (CG). Hepatic steatosis and liver stiffness were assessed using transient elastography (FibroScan®, Echosens, Paris). Clinical data were extracted from hospital records and analyzed using IBM SPSS v30.0. Results: A total of 128 PLWH and 127 individuals in the CG were included. MASLD prevalence was 40% overall (46.1% in PLWH and 33.9% in CG (p = 0.046)). Significant fibrosis (≥8 kPa) and steatosis was found in 6.3% and 46.9% respectively in PLWH (Table 1). HIV positivity increased MASLD risk 1.67-fold (OR: 1.67, p = 0.047). Among PLWH, the independent risk factors for MASLD development were LSM (OR: 1,3), HOMA-IR (OR:8), and HIV diagnosis time (OR: 4,4) (p <0,05) (Figure 1). Conclusions: MASLD was observed in 46.1% of PLWH, consistent with previous studies reporting a prevalence of 28.8%–48%. In our study, 6.3% of PLWH had significant fibrosis, while another study reported liver

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fibrosis in 10% of this population. In our cohort, HOMA-IR was associated with an increased risk of MASLD; similarly, other studies have identified HOMA-IR as an independent risk factor for NAFLD. Detailed and routine evaluation of PLWH is recommended to prevent comorbidities and complications.

Keyword(s) : HIV, MASLD, Transient Elastography, Fibrosis, Steatosis

1192 - Incidental Diagnosis of Rotor Syndrome in an Adolescent Presenting with Osteoporosis

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Background / Hypothesis:

Rotor Syndrome (RS) is a rare, benign autosomal recessive disorder causing isolated conjugated hyperbilirubinemia due to defects in hepatic OATP1B1 and OATP1B3 transporters. Distinguishing RS from other hereditary hyperbilirubinemias is essential to avoid unnecessary investigations.

Case Report: An 18-year-old male presented with chronic lower back pain. DEXA revealed severe osteopenia (lumbar Z-score: -5.2; femoral neck: -2.8). Physical examination showed jaundice; other findings were normal. Laboratory tests: total bilirubin 7.75 mg/dL, direct bilirubin 4.62 mg/dL, liver enzymes normal. Imaging and liver biopsy were unremarkable. A 24-hour urinary coproporphyrin test showed elevated type I fraction (52.6%). Genetic analysis confirmed biallelic pathogenic variants in SLCO1B1 and SLCO1B3, establishing RS diagnosis. Osteoporosis was idiopathic; bisphosphonate therapy was initiated.

Discussion: RS results from combined OATP1B1 and OATP1B3 deficiency, impairing hepatic reuptake of conjugated bilirubin. Urinary coproporphyrin pattern and genetic testing differentiate RS from Dubin-Johnson Syndrome. Although benign and non-progressive, recognition of RS avoids unnecessary interventions.

Conclusion: RS should be considered in patients with

isolated jaundice and normal liver function. Early clinical, biochemical, and genetic evaluation facilitates diagnosis, prevents invasive procedures, and reassures patients and clinicians.

Keyword(s) : Rotor syndrome, conjugated hyperbilirubinemia, SLCO1B1, SLCO1B3, coproporphyrin, adolescent

1193 - Magnesium Isoglycyrrhizinate for Preventing Novel Antitumor Drug Induced Liver Injury in Patients with Hematologic Malignancies A Multicenter Retrospective Study

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Background / Hypothesis:

Novel antitumor drugs (targeted agents and immune checkpoint inhibitors) are commonly used in the clinical management of patients with hematologic malignancies. Previous studies have indicated that these novel antitumor drugs readily induce Drug-Induced Liver Injury (DILI), with an incidence ranging from 1% to 60%. Effective prevention of novel antitumor drug-related DILI is crucial for the antitumor treatment process in these patients. This study aimed to evaluate the effectiveness of Magnesium Isoglycyrrhizinate (MgIG) in preventing novel antitumor drug-related DILI in patients with hematologic malignancies, thereby providing evidence-based support for liver injury prevention during antitumor therapy.

Materials and Methods:

This study was primarily conducted at the Institute of Hematology & Blood Diseases Hospital, Chinese Academy of Medical Sciences, with participation from 13 subsidiary centers across China. The study population included adult patients (aged ≥ 18 years) with hematologic malignancies who were receiving novel antitumor drugs and either received MgIG for liver protection or did not receive any hepatoprotective measures. All patients had normal baseline levels of Total Bilirubin (TBiL), Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT), and Alkaline Phosphatase (ALP) prior to antitumor therapy. The primary outcome was the severity of liver injury assessed on day 21, day 30, and day 60 after initiating novel antitumor drug treatment.



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Results:

From October 2023 to May 2024, a total of 639 patients were enrolled. 456 patients received MgIG prophylactic hepatoprotective therapy (Observation group), and 183 patients did not receive any hepatoprotective measures (Control group). The proportion of patients with Grade 1 liver injury severity was significantly lower in the Observation group (24.6% vs. 45.5%, $p=0.013$). On day 30, the proportions of patients with Grade 1, Grade 2, and Grade 3 liver injury severity were all significantly lower in the Observation group (26.0% vs. 42.3%; 5.2% vs. 6.2%; 2.9% vs. 6.2%; $p < 0.001$). On day 60, the proportion of patients with liver injury severity of Grade 2 or higher was significantly lower in the Observation group (8.6% vs. 18.6%, $p=0.022$). Furthermore, when the antitumor treatment regimen contained at least one targeted agent, the proportion of patients experiencing liver injury of Grade 1 severity or higher was significantly lower in the Observation group (46.7% vs. 66.7%, $p < 0.001$).

Conclusions:

The results of this study indicate that prophylactic administration of MgIG can effectively reduce the severity of novel antitumor drug-related liver injury in patients with hematologic malignancies, warranting further investigation and clinical promotion.

Keyword(s) : Hematologic malignancies; Novel Antitumor Drug-Induced Liver Injury; Magnesium Isoglycyrrhizinate (MgIG) ; Prevention

1194 - The Silent Summer Threat Significant Seasonal Worsening of Electrolyte Imbalance in Cirrhosis Patients in a Tropical Climate Calls for Dynamic Diuretic Dosing

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Background / Hypothesis: Electrolyte imbalances in cirrhosis, driven by diuretics and disease progression, are key predictors of mortality and hepatic encephalopathy. However, the impact of environmental stress is overlooked. We hypothesized that the persistent heat and humidity of a tropical climate would significantly disrupt electrolyte homeostasis. This study aimed to quantify the seasonal variation of serum sodium and potassium in a large cohort of stable cirrhotic patients.

Materials and Methods: We conducted a prospective, observational study of 300 consecutive cirrhotic patients at the Department of Gastroenterology, Rangpur Medical College Hospital, Rangpur, Bangladesh. Each patient underwent two standardized assessments: once in midsummer and once in midwinter. Serum electrolytes were measured, and the prevalence of hyponatremia ($\text{Na}^+ < 135 \text{ mmol/L}$) and hypokalemia ($\text{K}^+ < 3.5 \text{ mmol/L}$) was compared. Statistical analysis included paired t-tests and ROC curve analysis.

Results: The cohort (mean age 55.5 years, 68% male) included 60% patients on diuretics. We found a dramatic seasonal decline in mean serum sodium (Winter: $135.9 \text{ vs. Summer: } 133.6 \text{ mmol/L}$, $p < 0.001$) and potassium ($4.0 \text{ vs. } 3.7 \text{ mmol/L}$, $p < 0.001$). This translated into a near-doubling of clinical prevalence:

- Hyponatremia: 22.7% in winter vs. 45.0% in summer ($p < 0.001$).
- Hypokalemia: 8.7% in winter vs. 18.3% in summer ($p < 0.001$).

The effect was profoundly amplified in patients on diuretics, who had significantly lower summer levels than those not on diuretics (Na^+ : $131.9 \text{ vs. } 136.2 \text{ mmol/L}$; K^+ : $3.5 \text{ vs. } 4.0 \text{ mmol/L}$). ROC analysis identified $\text{Na}^+ \leq 134.5 \text{ mmol/L}$ and $\text{K}^+ \leq 3.75 \text{ mmol/L}$ as optimal cut-offs for predicting summer-associated depletion.

Conclusions: For the first time, we demonstrate a striking "Summer Effect"—a significant seasonal worsening of hyponatremia and hypokalemia in cirrhosis, critically exacerbated by diuretics. This mandates a paradigm shift towards seasonal stewardship in management, including pre-emptive dose adjustments and intensified summer monitoring, to prevent electrolyte-driven complications in vulnerable cirrhotic patients.

Table: Seasonal Variation in Electrolyte Imbalance Prevalence

Parameter	Summer (n=300)	Winter (n=300)	P-value
Mean Sodium (mmol/L)	133.6 ± 5.1	135.9 ± 4.8	< 0.001
Hyponatremia Prevalence	45.0% (453)	22.7% (221)	

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<0.001

Mean Potassium (mmol/L) 3.7 ± 0.5 4.0 ± 0.4 <0.001

Hypokalemia Prevalence 18.3% (185) 8.7% (84) <0.001

Hyponatremia (Diuretic Group) 58.2% 32.5% <0.001

Keyword(s) : Cirrhosis, Electrolyte Imbalance, Hyponatremia, Tropical Climate, Diuretic Management

1198 - Multidimensional Burden of Hepatic Encephalopathy and Overt Hepatic Encephalopathy among U.S. Patients with Cirrhosis

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Background / Hypothesis: Hepatic encephalopathy (HE) is a serious complication of cirrhosis characterized by neurocognitive symptoms that impair daily functioning, work ability, and quality of life (QoL). Overt HE (OHE) represents the more severe form, marked by episodes of confusion and disorientation that often require hospitalization and caregiver support. The overall patient-reported burden of HE and OHE, including their impact on daily functioning and QoL, remains poorly characterized.

Materials and Methods: An online survey was conducted (August-November 2025) among US adults with cirrhosis, without severe comorbid conditions or liver transplant, and with sufficient cognitive ability to complete the survey. Patients were classified into non-HE or HE cohorts based on self-reported HE diagnosis or rifaximin use; those reporting an OHE diagnosis, episode, or HE-related hospitalizations formed an OHE subgroup. The survey collected socioeconomic and clinical characteristics, work productivity (WPAI), and health-related QoL (PROMIS-29 v2.0).

Results: As of November 6, 2025, 151 patients participated (74 non-HE; 77 HE, including 22 OHE). Median age was 61 years in the non-HE cohort and 64 years in the HE cohort; 63.5% and 57.1% were male, and 75.7% and 80.5% lived with family, respectively. Median age in the OHE subgroup was 67 years, with 63.6% male. Cirrhosis duration averaged 2.9, 3.3, and

3.9 years for the non-HE, HE, and OHE cohorts, with severe liver disease reported by 0%, 45.5%, and 68.2%. Symptoms requiring medical attention were more common among HE and OHE patients, including fatigue (85.7%; 95.5%), trouble sleeping (68.8%; 81.8%), and confusion (68.8%; 81.8%), compared with 51.4%, 24.3%, and 16.2% among non-HE patients, respectively. Most patients had a caregiver (82.4% non-HE; 94.8% HE; 100% OHE), and paid caregiver use increased with severity (3.3%, 23.3%, and 45.5%, respectively), averaging 8.5, 46.7, and 55.7 hours of care weekly. Employment among working-age patients declined with disease progression (91.3% non-HE; 74.4% HE; 28.6% OHE). HE and OHE patients reported greater financial difficulties, including reduced ability to save and difficulty paying bills. Among those employed, mean work productivity loss in the past 7 days was 32.1% (non-HE) and 67.5% (HE). PROMIS-29 scores indicated poorer QoL across physical function, anxiety, depression, and sleep domains among HE patients, with greater impairment in OHE.

Conclusions: HE and OHE are associated with substantial clinical, socioeconomic, and QoL burden. Interventions that delay or prevent progression to these conditions may meaningfully reduce overall patient burden. Data collection is ongoing and results in a larger sample will be reported.

Keyword(s) : patient-reported outcomes, quality of life, humanistic burden, hepatic encephalopathy

1199 - Efficacy of Endoscopic band ligation versus argon plasma coagulation in Gastric Antral Vascular Ectasia management

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Background / Hypothesis:

Gastric Antral Vascular Ectasia (GAVE) is a capillary-type vascular malformation located mainly in the gastric antrum. It is characterized by dilated, tortuous mucosal capillaries, as well as by dilated, tortuous submucosal veins. Aim of the work: to evaluate the therapeutic effects of EBL (Endoscopic Band Ligation) as a new modality for the treatment of bleeding from GAVE in comparison to APC (Argon Plasma

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Coagulation).

Materials and Methods:

This study was conducted on fifty patients presenting with UGI bleeding that proved by upper GIE to be originating from GAVE at Kafr El Sheikh liver center every Tuesday weekly in the period from December 2022 to December 2023. Informed consent was obtained from each patient.

Results:

In this study hemoglobin levels were significantly raised after each endoscopic session done monthly through three months/four endoscopic follow up sessions in patients who underwent EBL and APC with no significant difference between both. Also, recurrence of bleeding from GAVE was significantly decreasing during follow up period in both groups with non-significant difference between both groups. Both groups showed non significant mild complications like superficial ulcers and gastric hyperplastic polyps with non-significant difference between both. UGI endoscopic follow up after 3 months of treatment showed marked improvement in patients treated with EBL and APC. Two patients in each group showed incomplete eradication of GAVE at fourth endoscopic follow up session requiring further follow up. Single patient showed recurrence of GAVE in EBL group with no recurrences in APC group.

Conclusions:

Both APC and EBL are safe and effective management options for both punctate and watermelon types of GAVE.

Keyword(s) : endoscopic band ligation, argon plasma coagulation, Gastric Antral Vascular Ectasia

1200 - CAD LT Score and Outcomes after Liver Transplantation Insights from a Middle Eastern Cohort

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Background / Hypothesis:

Cardiovascular disease remains a major cause of morbidity and mortality following liver transplantation (LT). The CAD-LT score has been proposed as pre-

transplant risk stratification tool for coronary artery disease (CAD) in LT candidates. However, its predictive value has not been validated in Middle Eastern populations.

Aim: To validate CAD-LT score and evaluate its association with post-liver transplant outcomes including (cardiac events, patient and graft survival)

Materials and Methods:

A retrospective analysis of 725 LT adult recipients transplanted between (2020–2024). Data included demographics, comorbidities, pre/post-transplant cardiac assessments (echocardiography, cardiac catheterization), and CAD-LT scores. Patients were stratified into high-, intermediate-, or low-risk groups; [HR, IR, LR]. Outcomes included cardiac events (myocardial infarction, cardiac death, and rhythm disorder), patient and graft survival. Statistical analyses used chi-square tests, the Wilcoxon test, and Kaplan-Meier survival curves.

Results:

The median age was 57.05 years, and 84.4% were male. The most common comorbidities were diabetes (47.66%), hypertension (33.61%), and obesity (33.88%). Based on the CAD-LT score, 41.7% of patients were classified as high risk, while 58.3% were categorized as intermediate- or low risk. Cardiac interventions were significantly more frequent in high-risk patients than in low/intermediate risk patients (9.3% vs 0.47%, $p < 0.0001$). However, four-year patient survival (85% vs. 84%, $p = 0.91$) and graft survival (85% vs. 83%, $p = 0.69$) did not differ significantly between risk groups. A total of 12 cardiac-related deaths occurred, with no significant difference between the groups (50% vs. 50%, $p = 0.544$).

Conclusions:

This is the first study to validate CAD-LT in Middle Eastern LT recipients. While high-risk patients required more pre-transplant cardiac interventions, long-term patient and graft survival were comparable across groups, highlighting the benefit of systematic cardiovascular screening and early intervention.

Keyword(s) : Liver Transplant, cardiac cath, CAD-LT

1202 - A 10 year Cohort Study of The Overall Survival and Treatment Outcomes in Patients with Primary Biliary Cholangitis A Single Centered Retrospective Cohort Study

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Background / Hypothesis: Primary biliary cholangitis (PBC) is a chronic cholestatic liver disease predominantly affecting women, carrying significant risks of cirrhosis and liver failure. Ursodeoxycholic acid (UDCA) is the current treatment of choice. There have been no previous studies investigating the overall survival and treatment outcomes of patients with PBC in Thailand. This study aimed to determine the overall survival and treatment outcomes in a cohort of Thai patients with PBC.

Materials and Methods: We included 57 PBC patients with complete data who were followed at Srinagarind Hospital, Khon Kaen University (2014–2024). Diagnosis required fulfilling at least two standard criteria (biochemical cholestasis, autoantibodies positive, or histology). The primary outcome was 10-year overall survival (OS); secondary outcomes evaluated UDCA treatment response using the Paris I, Paris II, and Rotterdam criteria, and survival differences based on UDCA dosage.

Results: The cohort predominantly comprised women (80.7%), with a median age at diagnosis of 55.14 years. At the time of initial diagnosis, 31.6% of patients had already progressed to cirrhosis, and 47.4% presented with an overlapping syndrome. All analyzed patients received UDCA treatment. The majority (56.1%) were treated with an over-optimal dose (> 15 mg/kg/day), followed by an optimal dose (13–15 mg/kg/day) in 26.3% of patients, and a suboptimal dose in 17.5%. Regarding UDCA treatment response, when utilizing the Paris I criteria, 77.19% of patients showed a favorable response. A lower proportion of responders was observed using the Rotterdam criteria (73.68%) and Paris II criteria (52.63%), respectively. The primary outcome, overall survival, demonstrated a clear dependency on UDCA response across all criteria. Patients classified as UDCA responders by Paris I criteria exhibited significantly higher overall survival compared to non-responders ($P = 0.0038$). This finding was consistent with both Paris II and Rotterdam criteria, where responders also showed significantly better overall survival ($P = 0.00088$ and $P < 0.0001$, respectively). When stratifying patients by UDCA dosage into optimal, suboptimal, and over-optimal groups, the over-optimal dose group showed a trend

towards higher overall survival compared to the optimal and suboptimal dose groups. However, this difference did not reach statistical significance ($p=0.077$).

Conclusions: Achieving a biochemical response to UDCA is a strong and reliable predictor of improved overall survival in Thai PBC patients. These findings confirm favorable long-term outcomes in this population and highlight that further investigation into the optimal UDCA dosing strategy is warranted.

Keyword(s) : Primary Biliary Cholangitis, Overall Survival, Treatment Outcomes, Thailand

1203 - PIVKA II Monitoring to Predict Response of The First Transarterial Chemoembolization (TACE) in Intermediate Stage of Hepatocellular Carcinoma

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Background: Hepatocellular carcinoma (HCC) is a major cause of cancer-related mortality. Transarterial chemoembolization (TACE) is the standard treatment for intermediate-stage disease. Early prediction of treatment response is crucial for optimizing patient management and guiding therapeutic decisions. Protein induced by vitamin K absence-II (PIVKA-II) has emerged as a potential biomarker for HCC monitoring; however, its role in predicting radiologic response—particularly complete response (CR) following the first TACE—remains unclear. This study aimed to determine the optimal cut-off value and diagnostic performance of the percentage reduction in PIVKA-II levels (referred to as PIVKA-II response) at 2 weeks post-TACE in predicting CR at 8 weeks, as assessed by imaging. Additionally, we compared the predictive accuracy of the PIVKA-II response at 2 and 8 weeks following the first TACE.

Methods: We conducted a prospective diagnostic accuracy study involving 57 patients with intermediate-stage HCC who underwent their first TACE at Srinagarind Hospital, Khon Kaen University, Thailand. Serum PIVKA-II levels were measured at baseline, as well as at 2 and 8 weeks post-TACE. Radiologic response at 8 weeks was evaluated using cross-sectional imaging based on the modified Response Evaluation Criteria in Solid Tumors (mRECIST). The

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predictive value of the PIVKA-II response at 2 and 8 weeks post-TACE was analyzed using the area under the receiver operating characteristic (AUROC) curve.

Results: At 8 weeks post-TACE, 26% of patients achieved CR. The median PIVKA-II response at 2 weeks was significantly greater in the CR group (98%) compared to those with a partial response (79%), stable disease (71%), and progressive disease (65%) ($p = 0.04$). AUROC analysis indicated that a PIVKA-II reduction of $\geq 83.8\%$ at 2 weeks predicted CR with a sensitivity of 80.0%, specificity of 64.3%, positive predictive value (PPV) of 44.4%, negative predictive value (NPV) of 90.0%, and an overall accuracy of 68.4% (AUROC 0.74).

Conclusions: An early reduction in serum PIVKA-II levels following the first TACE correlates with radiologic response, particularly CR, making it a potentially valuable biomarker for treatment monitoring. With an AUROC of 0.74 for predicting CR, the PIVKA-II response at 2 weeks shows promise as an early surrogate marker. This could enable timely therapeutic decisions and potentially improve clinical outcomes.

Keyword(s) : Hepatocellular carcinoma (HCC), Transarterial chemoembolization (TACE), Protein induced by vitamin K absence-II (PIVKA-II), Des-gamma carboxyprothrombin (DCP), Biomarkers, Monitoring, Predict response, Complete response (CR)

1205 - Real World Experience of 25 Years of Lamivudine Therapy Highlighting Favorable Long Term Outcomes in Chronic Hepatitis B

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Background / Hypothesis: Lamivudine, the first oral nucleoside analogue for chronic hepatitis B (CHB), transformed antiviral therapy by enabling durable viral suppression and improving long-term outcomes. However, comprehensive evidence on its long-term clinical impact across diverse treatment patterns is limited.

Materials and Methods: This real-world cohort study analyzed 1,134 CHB patients who initiated lamivudine between 1997 and 2010. Clinical outcomes, including

liver transplantation (LT)-free survival, hepatocellular carcinoma (HCC) incidence, and hepatitis B surface antigen (HBsAg) seroclearance, were evaluated according to treatment continuation, cessation, virologic response, and rescue therapy. Propensity score matching and landmark analyses were applied to minimize bias.

Results: Over a maximum follow-up of 28 years (median, 14.8 years), the 10-year LT-free survival rate was 93.7%, and cumulative HCC incidence was 12.6%. HBsAg seroclearance occurred in 4.8% of patients. Treatment discontinuation significantly increased the probability of HBsAg seroclearance without negatively impacting survival or HCC incidence when performed per stopping criteria. Virologic responders demonstrated superior long-term survival (15-year LT-free survival 96.8%) and higher HBsAg seroclearance rates compared with non-responders. Among these responders, treatment discontinuation did not worsen LT-free survival or HCC risk compared with continued therapy, while achieving higher rates of HBsAg seroclearance in patients maintaining sustained virologic response. Lamivudine resistance was associated with a higher HCC risk (HR 1.95, $P < 0.001$), whereas rescue therapy improved LT-free survival (HR 0.37, $P = 0.007$) and reduced HCC incidence (HR 0.60, $P = 0.038$).

Conclusions: Lamivudine therapy provides durable long-term survival and HCC risk reduction, with safe cessation in selected patients and clear benefits of timely rescue therapy. These findings support optimization of CHB management strategies using current potent antiviral agents

Keyword(s) : lamivudine, hepatitis B virus, hepatocellular carcinoma, mortality

1206 - Neurophysiological neuropsychological characteristics and natural history of liver cirrhotics with minimal and grade 1 hepatic encephalopathy

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Background / Hypothesis:

Minimal and Grade 1 hepatic encephalopathy (HE) represent the earliest stages of cognitive dysfunction in cirrhosis and are associated with adverse outcomes. Differentiating them objectively remains challenging. Neurophysiological tools such as Bispectral Index (BIS) and spectral electroencephalography (EEG) may provide objective markers. This study compared clinical, BIS and spectral EEG characteristics between minimal and grade 1 HE and evaluated their treatment outcomes.

Materials and Methods:

This prospective observational study was conducted in the Department of Gastroenterology, GB Pant Hospital, New Delhi, from November 2023 to April 2025. 150 cirrhotics were classified as no HE (n=50), minimal HE (n=50), or grade 1 HE (n=50) using West Haven criteria and Psychometric Hepatic Encephalopathy Score (PHES). Serum ammonia, Critical Flicker Frequency (CFF), BIS, and spectral EEG (mean dominant frequency [MDF], relative alpha, beta, theta, delta powers) were recorded. Receiver Operating Characteristic (ROC) curves and survival analyses were performed to determine diagnostic utility and assess prognosis.

Results:

Mean BIS declined progressively (No HE = 91.18 ± 3.43 , Minimal = 81.48 ± 3.9 , grade 1 = 72.9 ± 5.49 ; $p < 0.0001$). Optimal BIS cut-offs were ≤ 85 for Minimal (AUC = 0.969, Sensitivity 88%, Specificity 96%) and ≤ 78 for grade 1 HE (AUC = 0.895). MDF also decreased significantly (No HE = 12.9 ± 4.66 Hz, Minimal = 7.89 ± 1.19 Hz, grade 1 = 5.13 ± 1.57 Hz; $p < 0.0001$). MDF ≤ 8.9 Hz predicted Minimal HE (AUC = 0.97) and ≤ 6.6 Hz predicted grade 1 HE (AUC = 0.938). Alpha and beta relative powers declined, while theta and delta increased with HE severity (all $p < 0.0001$). CFF ≤ 39 Hz predicted minimal HE (AUC = 0.92), and ≤ 35 Hz predicted grade 1 HE (AUC = 0.659). Serum ammonia showed poor discrimination (AUC ≈ 0.54). During follow-up, grade 1 HE had higher complication, hospitalization, and mortality rates than minimal HE ($p < 0.05$).

Conclusions:

Minimal and grade 1 hepatic encephalopathy are

distinct clinical entities. Patients with grade 1 HE showed a higher risk of progression, complications, hospitalisation, and mortality than those with minimal HE. BIS and spectral EEG provide reliable, objective differentiation between minimal and grade 1 HE, outperforming serum ammonia. Lower BIS and MDF values correlate with disease severity and adverse outcomes, suggesting potential utility in early detection and prognostication of covert hepatic encephalopathy.

Keyword(s) : hepatic encephalopathy, bispectral index, spectral EEG, minimal hepatic encephalopathy, mean dominant frequency, critical flicker frequency, cirrhosis, neurophysiology

1207 - Comparison of inflammatory markers magnetic resonance spectroscopy findings and outcomes in cirrhotic patients with minimal and grade 1 hepatic encephalopathy a prospective observational study

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Background / Hypothesis: Minimal and Grade 1 hepatic encephalopathy (HE) represent the earliest, often under-recognized spectrum of neurocognitive impairment in cirrhosis. Both are associated with impaired quality of life, increased risk of progression to overt HE, and higher mortality. The overlapping clinical features and lack of uniform diagnostic criteria make differentiation difficult. We hypothesized that distinct inflammatory and metabolic alterations, measured by serum cytokines and magnetic resonance spectroscopy (MRS), could objectively distinguish minimal from Grade 1 HE and predict adverse outcomes.

Materials and Methods: This prospective observational study was conducted at the Department of

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Gastroenterology, GB Pant Hospital, New Delhi, between November 2023 and April 2025. Consecutive cirrhotic adults (n = 150) were categorized as No HE, Minimal HE, or Grade 1 HE (50 each). Minimal HE was diagnosed by Psychometric Hepatic Encephalopathy Score ≤ -5 and Critical Flicker Frequency ≤ 38 Hz; Grade 1 HE was defined by West Haven criteria. Serum TNF- α , IL-6, IL-18, IL-10, IL-12, IL-1 β , and endotoxin were quantified by ELISA. Brain MRS (3.0 T Philips Ingenia) evaluated myoinositol, choline, creatine, and glutamine peaks. Patients were followed for 12 months for progression, complications, and survival. Statistical analysis included ANOVA, ROC, Kaplan–Meier, and Cox regression.

Results: Mean ammonia, inflammatory markers, and MELD/CTP scores increased progressively across groups ($p < 0.001$). Compared with no HE, TNF- α ($25.3 \pm 10.8 \rightarrow 47.1 \pm 14.0 \rightarrow 64.3 \pm 28.4$ pg/mL), IL-6 ($22.7 \pm 16.8 \rightarrow 69.9 \pm 42.2 \rightarrow 113.9 \pm 46.1$ pg/mL), and IL-18 ($171.5 \pm 61.6 \rightarrow 348.0 \pm 86.3 \rightarrow 519.4 \pm 178.7$ pg/mL) rose stepwise from no HE to minimal and Grade 1 HE (all $p < 0.0001$). MRS revealed decreased myoinositol and choline with elevated glutamine in HE patients ($p < 0.001$). ROC analysis identified ammonia > 106 μ g/dL and CFF ≤ 38 Hz as optimal thresholds for minimal HE (AUC 0.91 and 0.94, respectively). One-year follow-up showed greater progression to overt HE and higher complication rates in Grade 1 HE compared to minimal HE ($p < 0.05$).

Conclusions: Minimal and Grade 1 HE constitutes distinct but sequential stages within the covert HE spectrum. Combined evaluation of inflammatory cytokines and cerebral MRS profiles enables objective differentiation and may facilitate early risk stratification and targeted management to prevent overt HE and improve prognosis.

Keyword(s) : hepatic encephalopathy, minimal HE, grade 1 HE, cytokines, IL-6, TNF- α , magnetic resonance spectroscopy, ammonia, critical flicker frequency, cirrhosis, MELD

1208 - Insights from the ELPA European Liver Screening Week 2025

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BACKGROUND AND AIMS: In recent years, the European Union(EU) has witnessed a concerning rise in the incidence of MASLD and MASH, conditions characterised by the accumulation of excess fat in liver cells. Of particular concern is the link between the accumulation of fat in the liver and its potential to serve as a significant risk factor for the development of liver cancer. This highlights the importance of addressing lifestyle-related factors and implementing preventive measures to slow the progression of this condition. This project aimed to raise awareness of how simple and effective early detection of liver disease can be using portable transient elastography(TE) within a community setting.

METHOD: The Liver4Life and SOS Hépatite testing vans (ELPA Members from the UK and France) were present at the European Parliament in September/October 2025 for 5 days, offering testing to the Parliament staff and the general public. ELPA representatives engaged with Members of the Parliament to build awareness of the upcoming event, and invitations were sent to members who regularly engage with the liver health agenda. The vans and gazebos were in the pedestrianised walkway outside the main entrance, and specific messaging was created and displayed on the vans. Trained operators, including some Belgian healthcare professionals, managed the TE devices. A brief lifestyle intervention was used when a high Controlled Attenuation Parameter (CAP) reading was identified. The same approach was used if a high stiffness reading was identified, paired with a self-referral letter inviting the individual to seek medical consultation.

RESULTS: The total number of completed and recorded TE examinations over the 5 days was 560. These were all adults. Of the total examinations, 29 people (5.2%) had stiffness readings above 7.1 Kpa, and these people were given self-referral letters after brief intervention advice. The average stiffness reading was 5.1 Kpa. The study also identified 230 people (41%) with a CAP reading above 235 dB/m. Among them, 80 people (35%) had a CAP reading of 290 dB/m or higher. Individuals with a CAP score of 235 dB/m or higher were provided with brief lifestyle intervention advice. The average CAP reading was 235 dB/m.

CONCLUSION: Most individuals with high CAP readings were unaware they had fat in their liver,

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although many suspected it due to their lifestyles, but hesitated to see the results. These findings highlight the urgent need for public health action to raise awareness.

Keyword(s) : liver screening, early detection, prevention, public health, liver health, awareness, MASH, MASLD, liver cancer, transient elastography

1210 - Prevotella Derived Butyrate Inhibits CD8⁺ T Cell Glycolysis via AKT mTOR signal to Alleviate MASH

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Background / Hypothesis:

Metabolic associated steatohepatitis (MASH) is a critical stage of MAFLD, lacking effective therapies. Gut microbiota and their metabolites, particularly short-chain fatty acids (SCFAs), are increasingly recognized as modulators of hepatic metabolism and immune responses. We investigated whether Prevotella could ameliorate MASH by regulating CD8⁺ T-cell metabolism.

Materials and Methods:

Fecal samples from MASH patients and healthy controls were analyzed by 16S rDNA sequencing and untargeted metabolomics. MASH mouse models, germ-free mice, and ex vivo CD8⁺ T-cell cultures were used to assess the effects of Prevotella and butyrate on glycolysis, PI3K/AKT/mTOR signaling, and liver pathology. Metabolic activity was measured using ECAR, 2-NBDG uptake, and lactate production; molecular changes were evaluated by qPCR, Western blot, and flow cytometry.

Results:

16S rDNA sequencing revealed a significant reduction of Prevotella abundance in MASH patients compared with healthy controls, which was inversely correlated with hepatic steatosis and CD8⁺ T-cell infiltration. In MCD diet-induced MASH mice, oral administration of Prevotella alleviated hepatic lipid accumulation and collagen deposition. In germ-free mice, colonization

with Prevotella similarly improved liver histology. Mechanistically, Prevotella suppressed glycolytic activity in CD8⁺ T cells, as shown by decreased 2-NBDG uptake, ECAR measurements, and reduced expression of glycolytic enzymes (HK2, PKM2, LDHA). Additionally, Prevotella downregulated the PI3K/AKT/mTOR signaling pathway in CD8⁺ T cells, linking its metabolic effects to immune modulation. Notably, the combination of Prevotella with a glycolysis inhibitor did not further enhance its therapeutic efficacy compared with Prevotella alone, indicating that suppression of glycolysis is a major mechanism underlying Prevotella's protective effects.

Conclusions:

Prevotella mitigates MASH by downregulating the PI3K/AKT/mTOR-glycolysis axis in CD8⁺ T cells via butyrate-dependent mechanisms, highlighting its potential as a microbial therapeutic candidate for MASH.

Keyword(s) : Prevotella; MASH; CD8⁺ T cells; Glycolysis; PI3K/AKT/mTOR signaling

1211 - Differential Impact of Antiviral Therapy on Bimodal Recurrence Patterns in HBV versus HCV related Hepatocellular Carcinoma Following Radiofrequency Ablation

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Background: Recent studies have shown that antiviral therapy suppresses recurrence of hepatocellular carcinoma (HCC) after curative treatment. However, the differential effects between hepatitis B virus (HBV) and hepatitis C virus (HCV)-related HCC remain unclear. This study aimed to compare the impact of viral suppression on HCC recurrence patterns between HBV and HCV-related cases following radiofrequency ablation (RFA).

Materials and Methods: We retrospectively analyzed patients who underwent curative RFA for primary HBV- or HCV-related HCC at our institution between January 2000 and December 2022. Sustained virological response (SVR) was treated as a time-dependent covariate, with patients stratified into periods before and after achieving SVR. We performed a secular trend analysis by comparing the hazards of recurrence

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between the SVR and non-SVR cohorts. The recurrence rate (hazards of recurrence) was estimated by computing a nonparametric smoothed estimate of the hazard function based on generalized linear mixed models.

Results: Among 214 HBV-related and 1,210 HCV-related HCC cases, median follow-up was 1.85 (range:0.78-4.41) years (SVR) versus 1.75 (range:0.74-2.29) years (non-SVR) for HCV, and 2.41 (range:0.94-4.05) years (SVR) versus 1.06 (range:0.51-1.96) years (non-SVR) for HBV patients. For HCV-related HCC, the non-SVR group demonstrated persistently high recurrence risk with a bimodal pattern, exhibiting peaks at 1-2 years (early : annual hazard rate 63.1%) and 8-10 years (late: annual hazard rate 69.5%). In contrast, the SVR group showed a significantly reduced recurrence risk (peak: annual hazard rate 9.67%) and did not exhibit a bimodal pattern. For HBV-related HCC, the non-SVR group similarly exhibited bimodal peaks at 1-2 years (early: annual hazard rate 50.5%) and 4-6 years (late: annual hazard rate 50.2%). While the SVR group showed reduced recurrence risk (peak annual hazard rate 26.3%), the reduction was substantially less pronounced than in HCV-related HCC.

Conclusions: Effective antiviral therapy attenuates the late recurrence peak of HCC following curative RFA, with this effect being more pronounced in HCV-related compared to HBV-related HCC. These findings suggest that surveillance remains important, particularly for HBV-related cases despite viral suppression.

Keyword(s) : hepatitis B virus; hepatocellular carcinoma; hepatitis C virus; antiviral therapy

1213 - Treatment Efficiency of Bulevirtide in Chronic Hepatitis Delta Number Needed to Treat Analysis From the MYR301 Phase 3 Trial

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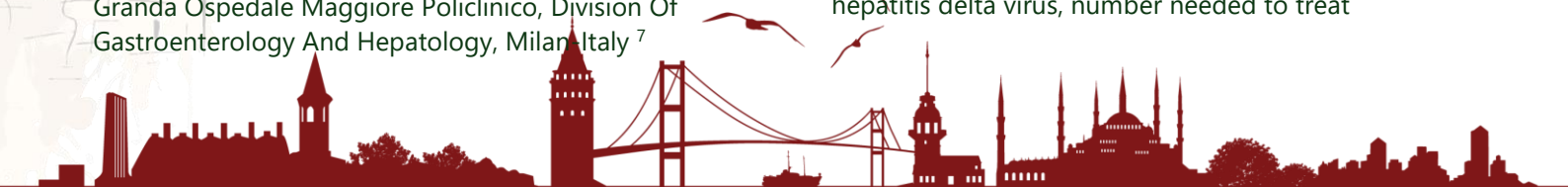
Background / Hypothesis: Bulevirtide (BLV), has demonstrated long-term efficacy with infrequent/low adverse events and treatment discontinuations, in the Phase 3 MYR301 trial for patients with chronic hepatitis delta (CHD). The number needed to treat (NNT) offers a patient-centered measure of clinical benefit, quantifying how many patients must receive therapy for 1 additional patient to achieve a meaningful outcome compared with control. This analysis aimed to estimate the NNT for BLV 2 mg and BLV 10 mg compared with control (delayed treatment) using efficacy endpoints from the MYR301 study at week 48.

Materials and Methods: Data were obtained from the multi-center, open-label, randomized, Phase 3 MYR301 trial in CHD. The efficacy endpoints included in this analysis were combined response i.e., hepatitis delta virus ribonucleic acid (HDV RNA) undetectable or $\geq 2 \log_{10}$ IU per milliliter decline + alanine aminotransferase (ALT) normalization and virologic response. NNT was calculated as the reciprocal of the absolute difference in response rates between arms at week 48 using the intention-to-treat population with 95% confidence intervals (CIs) for each estimate.

Results: At week 48, BLV demonstrated statistically significant improvements vs control across both key efficacy endpoints ($P < .001$). For combined response, the NNT was 3 (95% CI 2–4), corresponding to an absolute risk reduction (ARR) of 43%–46%, indicating that treating 3 patients with BLV 2 mg or 10 mg results in 1 additional responder, reflecting a robust clinical benefit. For virologic response, the NNT was 2 (ARR, 68%–72%), highlighting even greater clinical impact, with only 2 patients needing treatment for 1 additional response.

Conclusions: BLV 2 mg and 10 mg produced statistically significant and clinically meaningful benefits vs control, with NNTs of 2–3 across key efficacy endpoints at week 48. These low NNT values indicate a high treatment efficiency and align with thresholds considered highly favorable in the literature. Ongoing NNT analyses will assess whether these benefits persist beyond week 48. Overall, the combined evidence of favorable efficacy, together with an established safety profile, supports the clinical value of BLV.

Keyword(s) : Bulevirtide, chronic hepatitis delta, hepatitis delta virus, number needed to treat



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1216 - Microbial hippurate promotes GLRA2 activation immune exhaustion and development of shock in ACLF patients

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Background

Critically ill patients with acute-on-chronic liver failure (ACLF) have high sepsis-related mortality. While gut dysbiosis is recognized, the role of microbial metabolites in immune dysfunction and shock is not well understood. This study investigates gut microbiota, circulating metabolites, and their links to immune failure, shock progression, and mortality in ACLF patients.

Methods

In this pilot study, 200 participants were included: 50 healthy controls (HC), 50 ACLF without infection, and 100 critically ill ACLF patients (CAC; 36-sepsis, 64-septic shock) in the discovery cohort. Stool metaproteomics and plasma metabolomics were analyzed, with metabolites validated in 250 ACLF patients (119 survivors, 131 non-survivors) using machine learning (ML). Key microbial and metabolic predictors of septic shock and mortality were identified, and their correlation with neutrophil proteomics and metabolomics was assessed.

Results

Metaproteomic analysis identified distinct microbial signatures in septic shock, sepsis, alcoholic cirrhosis (AC), and healthy controls (HC), with significant separation of septic shock patients ($p < 0.05$). Septic shock had higher microbial diversity ($p < 0.05$), with 300 upregulated and 345 downregulated bacterial species compared to other groups. Key species enriched in septic shock included *Vibrio parahaemolyticus* ($FC > 6$), *Staphylococcus aureus* ($FC > 9$), and *Klebsiella aerogenes* ($FC > 150$). Functional pathways enriched in septic shock included siderophore biosynthesis, amino acid metabolism, and the TCA cycle.

Plasma metabolomics identified 1,236 metabolites, with 374 upregulated and 73 downregulated in septic shock (fold change > 1.5 , $p < 0.05$). Key metabolic pathways involved glycine, phenylalanine, tryptophan, pyruvate, and ammonia metabolism, with elevated hippurate ($FC > 24$, $p < 0.05$) in septic shock. Bacteria-metabolite correlation analysis revealed strong associations between hippurate and 26 bacterial taxa ($R^2 > 0.7$), with hippurate linked to 28-day mortality

($p < 0.05$).

Neutrophil proteomics showed elevated glycine levels in septic shock, with increased expression of glycine-N-acyltransferase (GLYAT1 and GLYAT3) ($FC > 4$, $p < 0.05$). Glycine, converted to hippurate, activated GLRA2, leading to membrane hyperpolarization and calcium signaling disruption, impairing neutrophil function by reducing ROS production and contributing to neutrophil exhaustion. GLRA2 activation also upregulated anti-inflammatory proteins like TGF β 1 ($FC > 1.5$, $p < 0.05$), contributing to immune suppression. Validation with mass spectrometry and ML confirmed hippurate as a strong predictor of septic shock and early mortality, outperforming traditional clinical scores. Cox regression analysis identified hippurate as an independent predictor of 28-day mortality ($HR > 1$). Conclusion

Hippurate and glycine metabolism play a central role in septic shock progression, contributing to neutrophil dysfunction and immune suppression. These findings highlight hippurate as a promising biomarker for early diagnosis and mortality prediction in septic shock.

Keyword(s) : Key Words: ACLF, Sepsis, Metaproteomics, Metabolomics, septic shock, hippuric acid, Hyperglycemia, Immunoexhaustion, Neutrophils

1217 - Integrated Plasma Multi Omics Identifies Fungal Dysbiosis and Lipid Remodelling as Major Determinants of Sepsis and Shock in Alcohol Related Cirrhosis

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Background: Alcohol associated cirrhosis (AC) often succumbs to sepsis progressing to shock and early mortality. While fungal overgrowth and lipid dysregulation are known in liver failure, how increase in fungal infection and associated lipids mediators enhance predisposition to shock and early mortality was evaluated in AC patients

Methods-Plasma and stool samples from AC patients with and without shock ($n = 150$) and healthy controls (HC; $n = 50$) was analysed using high-resolution mass spectrometry-based proteomics, lipidomics, and mycobiome (meta-proteomics) analysis. The identified fungal species (peptides) lipid parameter were cross

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correlated and key lipid species were analysed for the incidence of shock and early mortality in an independent AC cohort (n=250) using machine learning (ML).

Results Shock patients exhibited profound hypolipidemia (low expression >60 subclasses) and reduced lipid diversity as compared to other groups ($P<0.05$). Fungal peptide-based diversity analysis (Mycobiome profiling) revealed expansion of pathogenic fungi including *Aspergillus*, *Candida*, *Yarrowia*, and *Cryptococcus* with over 40 species enriched in shock and non-survivors ($P<0.05$). The fungal-derived 2,5-didehydro-D-gluconic acid showed >91% diagnostic accuracy for predicting predisposition to shock and early mortality. Expression of fungal surveillance proteins (CLEC4G, SCARB1) were decreased which mediated increase in fungal growth and linked lipid mediators which mediate significant increase in mitochondrial beta-oxidation and stress, suggesting exhaustive immune surveillance and bioenergetics collapse in AC patients.

Conclusion: Our study outlines significant dysregulation of fungal-host lipid axis driving mitochondrial dysfunction and bioenergetics collapse mediated shock in AC patients. The identified lipid mediators could be used for early stratification of shock and early mortality in AC patients.

Keyword(s) : Alcohol-associated cirrhosis, Fungal sepsis, Septic shock, Lipidomics, Mycobiome, Mitochondrial stress, Hypolipidemia, 2,5-Didehydro-D-gluconic acid.

1218 - Development and Validation of a Nomogram to Predict Surgical Resection After Conversion Therapy in Initially Unresectable Hepatocellular Carcinoma

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality worldwide, with fewer than 30% of patients being candidates for curative resection at initial diagnosis. Conversion therapy, particularly triple-combination regimens integrating locoregional therapy (TACE/HAIC), tyrosine kinase inhibitors, and immune

checkpoint inhibitors, offers a promising strategy to downstage initially unresectable HCC (uHCC). However, significant heterogeneity exists in patient response, and reliable tools for predicting successful surgical conversion are lacking. We hypothesized that a model incorporating baseline clinical and inflammatory biomarkers could effectively stratify patients according to their likelihood of achieving resection following conversion therapy.

Materials and Methods: We conducted a retrospective analysis of 210 patients with uHCC who received triple-combination conversion therapy at Sichuan Cancer Hospital between January 2022 and January 2025. Patients were randomly allocated to a training cohort (n=147) and a validation cohort (n=63) in a 7:3 ratio. Least absolute shrinkage and selection operator (LASSO) regression was applied to screen 28 potential predictors from clinical characteristics and laboratory indices. Independent factors associated with successful conversion surgery were identified using multivariate logistic regression. A nomogram was constructed and its performance was evaluated by the area under the receiver operating characteristic curve (AUC), calibration curves, and decision curve analysis (DCA).

Results: Among the 210 patients, 47 (22.4%) successfully underwent radical resection after conversion therapy. Multivariate analysis identified four independent protective factors for successful conversion: lower Tumor Burden Score (TBS; OR=0.663, 95% CI: 0.523-0.840), lower Neutrophil-to-Lymphocyte Ratio (NLR; OR=0.572, 95% CI: 0.381-0.860), lower C-reactive protein-to-Albumin Ratio (CAR; OR=0.057, 95% CI: 0.004-0.850), and absence of cirrhosis (OR=0.289, 95% CI: 0.107-0.783). The nomogram demonstrated excellent discrimination, with AUCs of 0.850 (95% CI: 0.784-0.915) in the training cohort and 0.871 (95% CI: 0.783-0.962) in the validation cohort. Calibration plots and DCA indicated robust model fit and favorable clinical utility.

Conclusions: We developed and validated a novel nomogram that integrates TBS, NLR, CAR, and cirrhosis status to accurately predict the probability of surgical resection following triple-combination conversion therapy in uHCC. This practical tool can assist clinicians in early identification of patients most likely to benefit

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from an aggressive conversion strategy, thereby facilitating personalized treatment planning and optimizing resource allocation. Further multicenter prospective studies are warranted to confirm its generalizability.

Keyword(s) : Hepatocellular carcinoma; Conversion therapy; Resection; Predictive model; Nomogram

1220 - Efficacy and Safety of Sofosbuvir Velpatasvir Voxilaprevir for the Retreatment of Chronic Hepatitis C Patients Relapsed after DAA Treatment

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Background / Hypothesis:

Hepatitis C virus (HCV) infection is one of major global public health concerns. Although the overall success rate of direct-acting antivirals (DAAs) therapies is high, a small proportion of patients experience relapse after treatment. This study aims to evaluate the efficacy and safety of Sofosbuvir/Velpatasvir/Voxilaprevir (SOF/VEL/VOX) in chronic hepatitis C (CHC) patients who relapsed following DAAs therapy.

Materials and Methods:

Between March 2022 and August 2024, patients who experienced relapse after DAAs therapy, and then received SOF/VEL/VOX salvage treatment for 12 weeks were included at 24 academic centers in Hunan and Shanxi Province, China. Baseline characteristics, HCV RNA clearance rates, complete blood count, liver function, and other indices were analyzed. The sustained virologic response at off-treatment week 12 (SVR12) was assessed by intention-to-treat analysis and per protocol analysis. The impact of gender, age, cirrhosis status, genotype, and the addition of ribavirin during treatment on SVR12 was assessed. The adverse events during treatment and follow-up were also reported.

Results:

92 patients were included and completed 12 weeks of treatment. Median age was 52, 69.6% were male, 62.0% had cirrhosis and 18.5% had portal hypertension. Of the 50.0% with genotype 3, 80.4% were also cirrhotic. Of note, 93.5% (86/92) had previously been treated with sofosbuvir-based regimen plus NS5A inhibitor and 6.5% (6/92) had been treated with NS3/4A inhibitor-based regimen plus NS5A. The

normalization rates of ALT and AST after 12 weeks of treatment were 54.3% (50/92) and 83.7% (77/92), respectively. By intention-to-treat analysis, SVR12 rate was 93.5% (86/92). For per protocol, the SVR12 was 97.7%, including 100% in genotypes 1b (21/21), 2 (15/15) and 6 (7/7), and 95.5% (43/45) in genotypes 3. And all two non-responders were genotype 3 patients, and had cirrhosis with a FIB-4 index of > 3.25. SVR12 in participants for genotype 3 with cirrhosis was 94.6%. The addition of ribavirin had no effect on SVR12. Further analysis indicated no statistically significant differences in HCV RNA clearance rates based on gender, age, genotype, treatment regimen, or ALT and AST levels. During treatment, one patient who received ribavirin experienced gastrointestinal side effects within one week and discontinued ribavirin on their own. No other severe adverse events were reported.

Conclusions:

SOF/VEL/VOX is highly efficacious and well-tolerated for CHC patients who relapse after DAAs therapy, and those with liver cirrhosis infected by genotype 3 remain the most-difficult-to-treat group.

Keyword(s) : Sofosbuvir/Velpatasvir/Voxilaprevir; Retreatment; Chronic Hepatitis C; Relapse; DAA Treatment

1221 - Neoadjuvant radiotherapy for resectable hepatocellular carcinoma with vascular invasion A systematic review and meta analysis

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Background / Hypothesis: Hepatocellular carcinoma (HCC) with vascular invasion, including portal vein tumor thrombus (PVTT) and microvascular invasion (MVI), is associated with high postoperative recurrence and poor survival. While surgical resection remains a cornerstone treatment, its efficacy is limited by recurrence risks. Neoadjuvant radiotherapy has emerged as a potential strategy to downstage tumors and improve outcomes, but its role in resectable HCC with vascular invasion remains controversial. We hypothesized that neoadjuvant radiotherapy could enhance survival and reduce recurrence in this patient population.

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Materials and Methods: A systematic search of PubMed, Embase, Cochrane Library, CNKI, Wanfang, and VIP databases was conducted up to August 1, 2024, to identify studies comparing neoadjuvant radiotherapy followed by surgery versus surgery alone for resectable HCC with vascular invasion. Four studies (two randomized controlled trials and two prospective studies) involving 362 patients were included. Primary outcomes were 1- and 2-year overall survival (OS) and 1-year disease-free survival (DFS). Pooled risk ratios (RRs) and hazard ratios (HRs) were calculated using random-effects models. Subgroup analyses were performed based on vascular invasion type (PVT vs. MVI).

Results: Neoadjuvant radiotherapy demonstrated a trend toward improved OS (1-year OS RR: 1.62, 95% CI: 0.71–3.69; 2-year OS RR: 2.64, 95% CI: 0.57–12.27) and DFS (RR: 2.68, 95% CI: 0.50–14.43), though not statistically significant. Subgroup analysis revealed significant benefits for PVT patients, with neoadjuvant radiotherapy improving 1-year OS (RR: 1.88, 95% CI: 1.52–2.32) and reducing recurrence (HR: 0.42, 95% CI: 0.31–0.58) and mortality (HR: 0.34, 95% CI: 0.24–0.48). No significant benefit was observed in MVI subgroups. Sensitivity analysis confirmed robustness, and heterogeneity was mitigated by grouping studies by vascular invasion type.

Conclusions: Neoadjuvant radiotherapy is a safe and effective strategy for resectable HCC with PVT, significantly improving survival and reducing recurrence. However, its benefit for MVI remains uncertain. These findings support individualized treatment planning based on vascular invasion type and highlight the need for larger, prospective trials to validate these results.

Keyword(s) : hepatocellular carcinoma, vascular invasion, neoadjuvant radiotherapy, surgical excision, meta-analysis.

1222 - Efficacy and Safety of Radiotherapy Combined with PD 1 Inhibitors and Targeted Therapy for Unresectable Hepatocellular Carcinoma A Real World Multicenter Study

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Background / Hypothesis:

Real-world data concerning the efficacy and safety of triple regimens combining radiotherapy (RT), PD-1 inhibitors and targeted therapy in unresectable hepatocellular carcinoma (uHCC) remains limited. Herein, we aim to evaluate the safety and efficacy of this triple therapy, and identify risk factors associated with survival outcomes.

Materials and Methods:

122 consecutive patients with uHCC who received RT combining PD-1 inhibitors and targeted therapy across 3 medical centers were analyzed. Data on baseline characteristics, treatments regimens, objective response rate (ORR), disease control rate (DCR), overall survival (OS), progression-free survival (PFS) and treatment-related adverse events (TRAEs) were collected. Univariate and multivariate Cox analyses were performed to identify independent risk factors for OS and PFS. Subgroup analysis explored the association between immunotherapy cycles and overall survival across tumor response stratification.

Results:

The median follow-up time was 14.2 months (IQR, 6.9–19.2 months). According to the modified response evaluation criteria in solid tumor (mRECIST), the ORR and DCR were 18.85% (95%CI: 12.34%–26.93%) and 73.78% (95%CI: 65.04%–81.32%), respectively. The median OS was 19.0 months (95%CI: 17.8–23.4 months), and the median PFS was 7.2 months (95%CI: 5.5–10.9 months). TRAEs of any grade occurred in 93 patients (76.23%), with grade 3 or 4 TRAEs observed in 40 patients (32.79%). No treatment-related death was observed. Multivariate analysis identified elevated AFP (HR= 2.747), poor tumor response (HR=2.583) and fewer immunotherapy cycles (HR=0.903) as independent risk factors for shorter OS. Subgroup analysis indicated that fewer immunotherapy cycles was an independent prognostic factor only for patients in SD group (P=0.003). For PFS, higher mALBI grade (HR=1.816) and poor tumor response (HR=3.130) were observed.

Conclusions:

Exhibiting encouraging survival benefits and

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manageable safety profile, the triple therapy that combined radiotherapy plus PD-1 inhibitors and targeted agents provides a promising treatment option for uHCC. For patients within SD, appropriately extending the duration of immunotherapy may improve survival outcomes.

Keyword(s) : Unresectable hepatocellular carcinoma, radiotherapy, PD-1 inhibitors, targeted therapy, multicenter study

1223 - Prevalence and topographic extent of gastric intestinal metaplasia in cirrhosis versus dyspeptic controls Implications for premalignant risk

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Background / Hypothesis:

Liver cirrhosis is a chronic condition associated with high morbidity and mortality. In patients with cirrhosis, premalignant gastric lesions are reported in nearly one-third of cases, independent of portal hypertension severity. In addition, the risk of gastric cancer has been reported to be substantially higher in cirrhotic patients compared with the general population. This study aimed to compare the frequency and topographic extent of gastric intestinal metaplasia between individuals with cirrhosis and a control group of adults presenting with dyspeptic symptoms.

Materials and Methods:

A total of 115 patients with cirrhosis who underwent upper gastrointestinal endoscopy at Baskent University Gastroenterology Endoscopy Unit between 2011 and 2023 were retrospectively evaluated. The control group included 510 adults with dyspeptic symptoms.

Demographic features, comorbidities, endoscopic findings, and histopathological data were reviewed. Gastric intestinal metaplasia prevalence and extent were compared between the two groups.

Results:

Gastric intestinal metaplasia prevalence was 28.7% in patients with cirrhosis and 21.2% in controls ($p = 0.081$). *Helicobacter pylori* positivity was significantly higher in controls than in cirrhotic patients (26.7% vs. 17.3%, $p < 0.05$). Rates of focal gastric intestinal metaplasia were similar between groups (11.4% vs. 13.5%). However, extensive gastric intestinal metaplasia was significantly more common in patients with

cirrhosis compared with controls (16.6% vs. 7.47%, $p = 0.002$).

Conclusions:

This study is the first to evaluate the topographic extent of gastric intestinal metaplasia in individuals with cirrhosis. The findings demonstrate that extensive gastric intestinal metaplasia occurs more frequently in cirrhosis, indicating a higher premalignant potential in this population. According to international clinical guidelines, individuals with extensive or incomplete gastric intestinal metaplasia and those with a family history of gastric cancer require endoscopic surveillance. The present results highlight that patients with cirrhosis may constitute a high-risk group for progression to gastric malignancy. Regular endoscopic follow-up with adequate biopsy sampling is therefore essential for early identification and preventive management.

Keyword(s) : cirrhosis patients, gastric mucosal injury, frequency of atrophic gastritis

1224 - Clinical significance of *Helicobacter pylori* infection in cirrhotic patients Implications for the development of premalignant gastric lesions

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Background / Hypothesis:

Cirrhosis is associated with impaired gastric mucosal defense and an increased risk of gastric cancer, although the mechanisms underlying this elevated risk remain unclear. *Helicobacter pylori* (*H. pylori*) is the most important environmental factor in gastric carcinogenesis; however, its prevalence has paradoxically been reported as lower in cirrhotic patients than in the general population. Chronic atrophic gastritis (CAG) and gastric intestinal metaplasia (GIM), key stages of the Correa cascade, are well-recognized premalignant lesions. This study aimed to investigate the relationship between *H. pylori* infection and the development of GIM and CAG in patients with cirrhosis.

Materials and Methods:

A total of 685 cirrhotic patients and 1817 dyspeptic controls who underwent

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esophagogastroduodenoscopy were reviewed. After applying exclusion criteria, 115 cirrhotic patients (mean age: 54.6 ± 13.4 years) and 510 controls (mean age: 55.8 ± 14.6 years) were included in the analysis. Demographic characteristics, comorbidities, endoscopic findings, and histopathological outcomes were compared. *H. pylori* positivity was determined by histopathology. The presence of GIM and CAG was recorded in *H. pylori*-positive individuals.

Results:

H. pylori positivity was 17.3% in cirrhotic patients and 26.7% in controls ($p = 0.038$). Focal GIM was observed in 11.4% of cirrhotics and 13.5% of controls. Extensive GIM was significantly more frequent in cirrhotic patients (16.6%) than in controls (7.47%, $p = 0.002$). Atrophy was also significantly more common among cirrhotics (51.3% vs. 31.7%, $p < 0.001$). Among *H. pylori*-positive cirrhotic patients, 55% had GIM and 60% had atrophy, compared with 22.7% and 35.2% among *H. pylori*-positive controls, respectively (both $p < 0.05$).

Conclusions:

Although the overall prevalence of *H. pylori* infection was lower in cirrhotic patients than in dyspeptic controls, infected cirrhotic individuals demonstrated significantly higher rates of GIM and atrophy. These findings suggest that *H. pylori* infection accelerates the development of premalignant gastric lesions in cirrhosis and may further elevate their risk of gastric cancer. Identification and appropriate management of *H. pylori* infection should therefore be considered an essential component of gastric cancer prevention strategies in this high-risk population.

Keyword(s) :

1227 - Comparative analysis of serum amino acid levels and their potential as biomarkers in liver cirrhosis A Systematic Review and Meta Analysis

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Background / Hypothesis: Early detection and precise prevention are crucial for reducing complications and mortality in liver cirrhosis (LC). The predictive value of amino acids profiles in monitoring the development of LC lacks consistent and comprehensive conclusions. This study aimed to review existing studies utilizing

metabolomics to detect serum amino acid levels in LC patients.

Materials and Methods: Meta-analysis was performed to identify potential biomarkers predictive of LC. Literature retrieved from eight databases between January 1, 2000 and August 1, 2025 was screened according to the inclusion criteria. The Newcastle-Ottawa Quality Assessment Scale (NOS) was used to assess the risk of bias in the included studies. A random-effects model for amino acid concentrations in the meta-analysis was used to calculate mean differences (MD) and 95% confidence intervals (95% CI). The I² statistic was used to measure study heterogeneity. This study has been registered with the PROSPERO.

Results: A total of 2125 records were retrieved, and 21 studies with 2254 individuals were included after screening, all of which were of high quality. Qualitative analysis of 21 studies including 23 amino acids. The meta-analysis of five studies showed that six amino acids (tyrosine, methionine, ornithine, threonine, citrulline and tryptophan) were significantly increased in the LC group, whereas two amino acids (arginine and valine) were significantly decreased. Subgroup analysis suggested that etiology and detection methods may be sources of heterogeneity in the results.

Conclusions: This manuscript will provide a comprehensive of differential expression of amino acid profiles and provide potential theoretical evidence to subsequently guide the assessment and treatment of LC.

Keyword(s) : liver cirrhosis, amino acids, metabolomics, meta-analysis

1228 - Role of Angiotensin Receptor Blockers in the Treatment of Hepatic Fibrosis in Patients with Metabolic Dysfunction Fatty Liver Disease A Meta Analysis

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Background / Hypothesis: Metabolic dysfunction fatty liver disease (MAFLD) impacts approximately 17% to 51% of adults globally, with a prevalence of 10% to 19% within the local population. Given its increasing incidence and the

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absence of approved pharmacologic therapies, there is an urgent requirement for innovative treatment options. Angiotensin receptor blockers (ARBs), integral components of the renin-angiotensin-aldosterone system (RAAS), have exhibited antifibrotic and anti-inflammatory effects, thereby positioning them as potential candidates for the management.

The objective of the study is to determine the therapeutic efficacy of angiotensin receptor blockers in liver fibrosis of patients with MAFLD liver cirrhosis by conducting a meta-analysis on different experimental studies randomized controlled trials, measuring changes in fibrosis scores, liver enzymes, and lipid profiles, and assessing its safety profile.

Materials and Methods:

A systematic investigation of databases including PubMed, ScienceDirect, PLOS ONE, and Google Scholar up to January 2025 identified 1173 relevant studies. Following the removal of duplicates and a meticulous screening process, five randomized controlled trials (RCTs) involving 642 patients were incorporated in accordance with PRISMA guidelines.

Results:

The meta-analysis indicated that ARB therapy significantly diminished fibrosis scores on liver biopsy compared to control groups (mean difference -0.38 ; 95% CI, -0.65 to -0.11 ; $p=0.006$) and enhanced MAFLD Activity Scores, especially in the domains of lobular inflammation and hepatocellular ballooning. Notable improvements were also recorded in hepatic serologic markers, including decreases in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels. Significantly, the incidence of adverse events did not present significant differences between ARB and control groups, indicating a favorable safety profile.

Conclusions:

These results corroborate the potential role of ARBs in mitigating fibrosis progression and enhancing liver histology in NAFLD. Nonetheless, the relatively modest sample sizes, brief follow-up periods, and diversity among studies highlight the necessity for more extensive, rigorously designed trials with long-term endpoints to conclusively ascertain the efficacy and safety of ARBs as a therapeutic option for NAFLD.

Keyword(s) :

1230 - Treatment outcome of radiotherapy for elderly hepatocellular carcinoma patients with comorbidity

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Background / Hypothesis: The incidence of hepatocellular carcinoma (HCC) is increasing in elderly populations and the management in elderly patients is a globally emerging issue. Elderly patients tend to have several comorbidities affecting treatment tolerance and outcomes, and the optimal management in elderly patients has not been determined. Therefore, we assessed comorbidities in elderly patients and investigated treatment outcomes of radiotherapy (RT) to liver-confined HCC.

Materials and Methods: We retrospectively reviewed 40 elderly patients ≥ 70 years with liver-confined HCC, who were treated with RT between 2015 and 2023. Comorbidity was assessed by using the Charlson comorbidity index (CCI). Survivals were analyzed using the Kaplan-Meier method.

Results: The median age was 75 years (range, 70–87 years). The Barcelona Clinic Liver Cancer (BCLC) stage 0 was in 7 patients, A in 10 patients, B in 9 patients, and C in 14 patients. Most patients (85%) had Child-Pugh class A hepatic function before RT. The CCI scores ranged from 2 to 9 (median, 5). Various RT techniques were used according to patients' condition, tumor burden and treatment aim: 3-dimensional conformal radiotherapy in 4 patients; intensity modulated radiotherapy in 20 patients; and stereotactic body radiotherapy (SBRT) in 16 patients. RT was applied in 30 patients with radical intent and in 10 patients with

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palliative intent. The median biological effective dose (BED) using $\alpha/\beta = 10$ was 53.7 Gy10 (range, 39–134.4 Gy10), when the α/β ratio was assumed to be 10 Gy. The median follow-up period after RT was 18 months. The local progression-free survival and overall survival rates were 74% and 81% at 1 year, and 44% and 52% at 3 years, respectively. The CCI was not associated with survivals. On the other hand, the use of SBRT was the most significant parameter improving survivals.

Conclusions: Although elderly patients had several comorbidities, RT to liver-confined HCC seems to be tolerable, effective, and safe.

Keyword(s) : Charlson Comorbidity Index, Comorbidity, Elderly, hepatocellular carcinoma, radiotherapy

1231 - A Simplified HCV care whole pathway model improves HCV diagnosis and treatment rate in hyperendemic areas experience in Xing'an country from China

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Background / Hypothesis:

Hepatitis C virus (HCV) remains a major health burden, particularly in HCV hyperendemic, remote regions in China. Complicated pre-treatment assessments and follow-up procedures, as well as complex medical-insurance reimbursement processes, are main barriers leading to the low HCV diagnosis and treatment rate. We aims to evaluate the simplified HCV care whole pathway model on improving the HCV diagnosis and treatment rate of HCV-infected patients in Xing'an country which is HCV hyperendemic areas.

Materials and Methods:

A Simplified HCV care whole pathway model for the treatment of all patients infected with hepatitis C virus (HCV) was launched since January 2025. The simplified HCV care whole pathway includes: (i) Treat immediately upon HCV diagnosed by providing Pan-genotypic DAA regimen, without HCV genotype test. (ii) A "green reimbursement channel" was opened to patients undergoing HCV therapy directly supported by government policies. HCV antibodies positive patients in Xing'an country People's hospital will be informed and received education about the simplified HCV care whole pathway model to boost their confidence in subsequent diagnosis and therapy progress and benefit of HCV cure.

Results:

From January 2025 to October 2025, a total of 16585 people were screened, among whom 658 were HCV antibody positive. Encouraged by the simplified HCV care whole pathway model, the HCV RNA testing rate and the treatment rate were significantly improved to 73.7% (485/658) and 90.4% (198/219), respectively, much higher than the rates in the same period of 2024, (see Figure 1). Among the 198 patients who initiated therapy, 93.4% patients were initiated treatment on the day of diagnosis. Thirty-nine patients had decompensated cirrhosis, and 83.8% of HCV infected patients were aged 50–70 years (see Figure 2). All patients received sofosbuvir/velpatasvir for 12 or 24 weeks, The SVR12 were upto 100% (see Figure 3). Overall, the medication's safety and tolerability were good, with no serious adverse events reported.

Conclusions:

The simplified HCV care whole pathway model significantly increased the HCV RNA testing and treatment rate in HCV hyperendemic, remote regions in China. SOF-based regimen could achieve high SVR regardless of genotype testing. This model deserves promotion in remote areas to help accelerate HCV elimination target.

Keyword(s) : HCV ; simplify; hyperendemic ; SOF/VEL

1234 - Associations of PNPLA3 and MBOAT7 Genetic Variants with Risk of Steatotic Liver Disease and Visceral Fat Accumulation in Adolescence

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Background: Steatotic liver disease (SLD), predominantly metabolic dysfunction-associated steatotic liver disease (MASLD), is increasingly observed during adolescence as well as adulthood. Accumulation of body fat, particularly visceral fat, in association with SLD elevates the risk of lifestyle-related diseases and malignancies, underscoring the need to identify aggravating factors before adulthood. Variants in metabolic genes such as Patatin-like phospholipase domain containing 3 (PNPLA3) have been reported to influence SLD and lipid metabolism. Our previous report demonstrated associations between lipid changes and Membrane bound O-acyltransferase domain containing 7 (MBOAT7) variants in young individuals. However, the relationship

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between genetic variants and SLD or visceral fat accumulation in adolescence remains unclear. This study aimed to examine associations between selected metabolic gene variants and SLD as well as visceral fat accumulation using university health check-up data. Materials and Methods: Clinical data from 346 students aged 18–19 years (male: 183; female: 163) who underwent university health check-ups between 2024 and 2025 were analyzed. SLD and body fat were assessed by abdominal ultrasonography. Single-nucleotide polymorphism (SNP) genotyping was performed for PNPLA3 (rs738409), MBOAT7 (rs641738), Peroxisome proliferator activated receptor gamma (PPARG) (rs17036160), Lysophospholipase like 1 (LYPLAL1) (rs12137855), Transmembrane 6 superfamily member 2 (TM6SF2) (rs58542926), and Glucokinase regulatory protein (GCKR) (rs1260326). Associations between these genotypes and metabolic parameters were evaluated after stratification by SLD status. Results: In adolescents, among these 6 gene variants, PNPLA3, TM6SF2, and GCKR variants significantly increased SLD risk, with a stronger effect observed in males. MBOAT7 CC genotype did not show a significant difference in overall SLD risk; however, among SLD cases, CC genotype was associated with greater visceral fat thickness [5.8 mm (IQR: 4.1–8.5) vs 4.2 mm (2.9–5.6)]. PPARG and LYPLAL1 variants were not significantly associated with SLD or visceral fat accumulation, but in non-SLD individuals, PPARG CT genotype was associated with increased subcutaneous fat [3.9 mm (IQR: 2.5–5.2) vs 2.3 mm (1.6–3.6)]. Conclusions: PNPLA3, TM6SF2, and GCKR variants contribute to SLD risk in adolescence, whereas MBOAT7 variants may influence visceral fat accumulation in the presence of hepatic steatosis rather than directly increasing SLD risk. These findings suggest distinct metabolic and genetic backgrounds in adolescence compared to adulthood. Life stage-specific evaluation of SNPs may be critical for establishing precision medicine strategies for SLD.

Keyword(s) : SLD; visceral fat; adolescence; PNPLA3; MBOAT7

1235 - Comparative Efficacy of Emerging Metabolic Therapies for MASLD and MASH A Systematic Review and Network Meta analysis of Randomized Controlled Trials

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Background / Hypothesis:

Metabolic dysfunction–associated steatotic liver disease (MASLD) and metabolic dysfunction–associated steatohepatitis (MASH) represent the fastest-growing causes of chronic liver disease worldwide. Multiple novel pharmacologic agents targeting metabolic and fibrotic pathways have emerged; however, their comparative efficacy remains unclear. A network meta-analysis allows indirect comparisons across drug classes to determine the most effective therapy for steatosis, inflammation, and fibrosis. This study aims to evaluate and rank the therapeutic efficacy of GLP-1 receptor agonists, dual GIP/GLP-1 agonists, SGLT2 inhibitors, FXR agonists, PPAR agonists, and FGF-21 analogues for the treatment of MASLD and MASH.

Methods:

A systematic search of PubMed, Embase, Cochrane CENTRAL, Web of Science, and ClinicalTrials.gov was completed from inception to January 2025. Randomized controlled trials enrolling adults with imaging-defined MASLD or biopsy-confirmed MASH and reporting outcomes on histology or validated non-invasive biomarkers were included. A Bayesian network meta-analysis was performed to compute pooled treatment effects and generate probability rankings (SUCRA values) for each intervention. Primary outcomes included resolution of MASH without fibrosis worsening and fibrosis improvement by at least one stage. Secondary outcomes included MRI-PDFF reduction, ALT and AST improvement, weight change, and glycemic outcomes. Study quality was assessed with the Cochrane RoB-2 tool.

Results:

Forty-six randomized trials involving 12,984 participants were included. Dual GIP/GLP-1 agonists demonstrated the highest probability of achieving MASH resolution without fibrosis worsening and were superior to GLP-1 receptor agonists, FXR agonists, and FGF-21 analogues. FGF-21 analogues ranked highest for fibrosis improvement, followed by PPAR agonists. GLP-1 receptor agonists produced the greatest reductions in MRI-PDFF, weight, and glycemic markers. SGLT2 inhibitors demonstrated modest but consistent biochemical improvements but ranked lower for histologic endpoints. FXR agonists improved biochemical inflammation but were limited by pruritus and lipid-related adverse effects. Treatment rankings remained stable across sensitivity analyses.

Conclusion:

This network meta-analysis demonstrates distinct therapeutic profiles among emerging metabolic agents for MASLD and MASH. Dual GIP/GLP-1 agonists appear most effective for resolving steatohepatitis, whereas



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FGF-21 analogues show superior impact on fibrosis improvement. GLP-1 receptor agonists remain highly effective for metabolic and steatosis-related outcomes, while PPAR agonists and SGLT2 inhibitors offer complementary benefits. These findings provide the most comprehensive comparative evaluation of MASLD pharmacologic therapies to date and may inform individualized, mechanism-based treatment strategies as these agents progress toward clinical implementation.

Keyword(s) : MASLD; MASH; network meta-analysis; GLP-1; dual incretin therapy; FGF-21; FXR agonists; PPAR agonists; SGLT2 inhibitors; fibrosis.

1236 - Multi center analysis of HDV seroprevalence and epidemiological profile in Chinese HBsAg positive inpatients

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Background / Hypothesis:

Hepatitis D virus (HDV) is a defective satellite virus that requires hepatitis B virus (HBV) for replication and exacerbates the severity of viral hepatitis. China carries a heavy burden of HBV, with an estimated 43.3 million infections in 2021. However, the national prevalence and clinical impact of HDV/HBV coinfection remains poorly defined. Accurate epidemiological data are essential to guide clinical and public health responses.

Materials and Methods:

From 2023 to 2025, we screened HBsAg-positive inpatients across nine medical centers representing northern, southern, eastern, and western China. Serum samples were tested for anti-HDV IgM and IgG using ELISA, and seropositive samples were further detected for HDV RNA via quantitative RT-PCR. Clinical and laboratory data were collected and analyzed.

Results:

The final analysis included 13,953 HBsAg-positive patients. Anti-HDV antibodies were detected in 0.57% of the cohort, with 0.29% positive for IgM and 0.31% for IgG. HDV RNA, indicating active viral replication, was detected in 5.06% of seropositive cases. Specifically, HDV RNA was positive in three of four patients with both IgM and IgG antibodies, and in one of 39 with IgG alone. Seroprevalence of HDV exhibited

regional variation ($p < 0.001$), ranging from 0% in Fujian to 0.96% in Guizhou. Higher rates of HDV seropositivity were observed in patients with HBsAb (0.75% vs 0.57%), HCV co-infection (2.47% vs 0.51%) or TP co-infection (0.56% vs 0.48%). Anti-HDV-positive patients were significantly older (57 vs 52 years, $p = 0.016$) and exhibited lower neutrophil (2.56 vs $3.09 \times 10^9/L$, $p = 0.023$) and lymphocyte counts (1.27 vs $1.50 \times 10^9/L$, $p = 0.005$), prolonged prothrombin time (13.1 vs 12.6 s, $p = 0.032$), and reduced fibrinogen levels (2.53 vs 2.74 g/L, $p = 0.004$) in peripheral blood compared to anti-HDV-negative individuals. Additionally, anti-HDV-positive patients had higher HBsAg levels (340.1 vs 250.1 COI, $p = 0.027$) and a non-significant trend toward lower HBV DNA loads (49.9 vs 69.1 IU/mL, $p = 0.101$).

Conclusions:

This multi-centre study provides an updated overview of HDV seroprevalence and active infection rates in China, which are substantially lower than previously reported. The findings highlight geographical disparities and high-risk groups of HDV infection, particularly those HBsAg-positive patients with HCV or TP co-infection or undetectable HBV DNA, providing valuable insights for targeted HDV surveillance and prevention strategies.

Keyword(s) : Hepatitis D virus, hepatitis B virus coinfection, multi-centre study, prevalence

1240 - Impact of Hand Grip Strength on the Clinical Course of Patients with Acute Variceal Bleeding A Single Center Prospective Observational Study

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Background / Hypothesis:

This study aimed to assess handgrip strength (HGS) in patients with acute variceal bleeding (AVB) following endoscopic therapy, compare it with compensated cirrhotic patients, and investigate its association with clinical outcomes, including rebleeding and short-term mortality.

Materials and Methods:

In this single-center, prospective observational study, 34 AVB patients and 21 compensated cirrhotics were enrolled between June 2024 and June 2025. Exclusion

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criteria included hepatocellular carcinoma, hepatic encephalopathy, malignancies, major organ failure, cerebrovascular disease, or neuromuscular disorders. HGS was measured using a digital dynamometer at discharge for AVB patients and during outpatient visits for controls. Baseline demographics, laboratory data, Child–Turcotte–Pugh (CTP) and MELD–Na scores, and 6-week outcomes were recorded. Statistical analyses compared HGS and clinical parameters between groups and evaluated correlations.

Results:

HGS was significantly lower in the AVB group compared with compensated cirrhotics ($p=0.0004$). Within six weeks, nine patients experienced rebleeding and seven patients died. Those with adverse outcomes demonstrated significantly reduced HGS, lower albumin, hemoglobin, and blood pressure, and higher MELD–Na and CTP scores. HGS negatively correlated with prognostic scores, hospital stay, INR, and bilirubin, while positively correlating with albumin.

Conclusions:

Handgrip strength is markedly reduced in patients with AVB and is associated with rebleeding, short-term mortality, and prolonged hospitalization. As a simple bedside measure of sarcopenia and nutritional status, HGS may serve as a valuable prognostic marker. Early identification of low HGS can guide nutritional and rehabilitative interventions to potentially improve outcomes in this high-risk patient population.

Keyword(s) : acute variceal bleeding, mortality, rebleeding, sarcopenia, hand dynamometer, muscle strength

1241 - Liquid Biopsy Based Detection Of TP53 And TERT Gene Mutations In HBV Induced Chronic Liver Disease and Hepatocellular Carcinoma Patients Evidence From Bangladesh

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Background / Hypothesis:

The Tumor Protein 53 (TP53) and Telomerase Reverse Transcriptase (TERT) promoter genes are the most frequently mutated genes contribute to hepatocarcinogenesis in hepatitis B virus (HBV)

patients. Circulating cell free DNA (cfDNA) has emerged as a promising noninvasive 'liquid biopsy' tool for detecting tumor specific genetic mutations and monitoring disease progression. However, there are limited data about these gene mutations in HBV induced chronic liver disease (CLD) and Hepatocellular carcinoma (HCC) patients from Bangladesh that limits the development of precision diagnostics and targeted therapies. Therefore, we aimed to detect TP53 and TERT gene mutations from cfDNA in HBV induced CLD and HCC patients and determine association with liver enzymes and biochemical markers.

Materials and Methods:

This cross-sectional study was conducted among 80 participants where 30 had HCC, 30 CLD and 20 healthy controls. cfDNA was extracted from aseptically collected peripheral venous blood (10 mL) using the QIAamp Circulating Nucleic Acid Extraction Kit. Amplification of the TP53 (pR249S) and TERT (C228T and C250T) genes were performed using selected primers via conventional PCR, followed by visualization on 2% agarose gel with ethidium bromide. Positive samples underwent Sanger sequencing; mutation analysis conducted using MEGA 11 software comparing with the TP53 and TERT reference sequence. Statistical analysis was done using STATA 17.

Results:

TP53 mutations were detected in 3.3% (1/30) CLD and 23.3% (7/30) HCC patients and absent in healthy controls ($p=0.01$). Among HCC patients, the C228T mutation was identified in 3.3% (1/30) and C250T in 6.7% (2/30), with an overall TERT promoter gene mutation frequency of 10% (3/30) and none were found among CLD and healthy controls ($p=0.113$). Among all patients, TERT promoter mutations were significantly associated with low serum albumin (<3.4 g/dL) and elevated bilirubin (≥ 1.2 mg/dL) ($p<0.05$). TP53 mutations were strongly associated with elevated ALT and AST ($p<0.001$), low albumin ($p<0.05$), elevated bilirubin ($p<0.001$), and high AFP levels ($p<0.05$).

Conclusions:

TP53 and TERT gene mutations, detected from liquid biopsy may be used as biomarkers along with liver

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enzymes in HCC patients for early detection, management, and prognosis of HCC. These gene mutations can also be used in precision diagnostics and targeted therapies as personalized treatment plans.

Keyword(s) : TERT promoter mutation, TP53 mutation, cfDNA, liquid biopsy, hepatocellular carcinoma, hepatitis B, Bangladesh

1242 - Hormone therapy and metabolic risk interact to influence metabolic dysfunction associated steatotic liver disease incidence across the menopausal transition a 10 year longitudinal cohort study

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Background / Hypothesis:

Estrogen deficiency after menopause may increase hepatic vulnerability, particularly in women with clustered cardiometabolic risk factors. The hepatic safety of menopausal hormone therapy (MHT) in this context remains uncertain. We hypothesised that menopausal status, MHT use and cardiometabolic risk burden jointly influence the incidence of metabolic dysfunction associated steatotic liver disease (MASLD) and incident significant fibrosis.

Materials and Methods:

We analysed two parallel prospective cohorts of Taiwanese women aged 45–65 years who attended health examinations between 1996 and 2022: a MASLD free cohort (n=9,505) to evaluate incident MASLD, and a significant fibrosis free cohort (n=10,670; Fibrosis 4 [FIB 4] ≤ 1.3 at baseline) to evaluate incident significant fibrosis (FIB 4 > 1.3 during follow up). Menopausal status was classified as premenopausal, postmenopausal without MHT and postmenopausal with long term MHT. MASLD was defined using imaging and metabolic criteria. We estimated adjusted hazard ratios (aHRs) for incident MASLD and significant fibrosis using Cox models, controlling for age, lifestyle, education year, calendar year and cardiometabolic risk factors (body mass index, diabetes, hypertension and dyslipidaemia). Stratified analyses evaluated effect

modification by baseline cardiometabolic profiles.

Results:

Compared with premenopausal women, postmenopausal women without MHT had a higher risk of incident MASLD and a higher hazard of developing significant fibrosis; excess risks rose with the number of cardiometabolic risk factors. Overall, postmenopausal women using MHT showed MASLD and fibrosis risks broadly similar to those without MHT, and we did not observe a consistent protective effect of MHT. Across most cardiometabolic strata, MHT versus no MHT was not significantly associated with incident MASLD or significant fibrosis. However, in postmenopausal women with coexisting diabetes and hypertension, MHT use tended to associate with higher hepatic risk, with approximately 2.4 fold higher MASLD incidence and 2.7 fold higher hazard of significant fibrosis compared with non users, although confidence intervals were wide.

Conclusions:

Postmenopausal status is associated with increased incidence of MASLD and development of significant fibrosis, particularly in women with clustered cardiometabolic risk factors. In this cohort, MHT was predominantly prescribed as oral preparations, which undergo first pass hepatic metabolism. Long term oral MHT did not appear to confer uniform hepatic protection and may be associated with higher MASLD and fibrosis risk in women with combined diabetes and hypertension. These findings support integrating route of administration and cardiometabolic burden into MASLD risk stratification and individualising MHT decisions in midlife women.

Keyword(s) : menopause, menopausal hormone therapy, cardiometabolic risk factors, metabolic dysfunction-associated steatotic liver disease, hepatic fibrosis, cohort study

1244 - Cabozantinib as Third or Later Line Therapy After ICI Failure Real World Outcomes of Reduced Dose Strategies in Advanced HCC

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Background / Hypothesis

Cabozantinib is commonly used as third- or later-line therapy for advanced hepatocellular carcinoma (HCC) following progression on immune checkpoint inhibitor (ICI)-based regimens. In routine practice, many patients begin cabozantinib at reduced doses because

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of impaired liver function or residual toxicities. The therapeutic relevance of these reduced-dose strategies remains unclear. We hypothesized that disease control (DC) could still be achieved at lower doses, and that hepatic reserve would be a key determinant of treatment outcomes.

Materials and Methods

We retrospectively analyzed 33 patients with unresectable HCC previously treated with at least one ICI-based regimen before receiving cabozantinib as third- or later-line therapy. Tumor response was assessed by RECIST v1.1 and categorized into DC (complete response, partial response, or stable disease) or non-DC (progressive disease or not evaluable). Initial dose, early dose modifications, and average daily dose during the first 6 weeks were examined.

Progression-free survival (PFS), overall survival (OS), and factors associated with outcomes were evaluated.

Results

Most patients (91%) initiated cabozantinib at reduced doses (20–40 mg/day), with only three patients starting at 60 mg/day. The objective response rate was 3%, and the DC rate was 52%. Compared with the non-DC group, the DC group had significantly longer median PFS (4.4 vs. 1.2 months; $p < 0.0001$), OS (10.7 vs. 3.0 months; $p = 0.0456$), and treatment duration (134 vs. 22 days; $p < 0.0001$). Time to first dose reduction and mean daily dose in the first 6 weeks were not significantly different between groups. Child–Pugh class A was independently associated with achieving DC and with better survival outcomes.

Conclusions

Cabozantinib is frequently started at reduced doses in later-line settings after ICI-based therapies for advanced HCC. Despite lower starting doses, meaningful and durable disease control (DC) can be achieved—even at approximately 20 mg/day—particularly in patients with preserved liver function. These findings support individualized dose-adjustment strategies and highlight the importance of hepatic reserve in optimizing cabozantinib treatment.

Keyword(s) : Cabozantinib, immune checkpoint inhibitor, hepatocellular carcinoma

1246 - TRENDS IN HEPATOCELLULAR CARCINOMA INCIDENCE IN MONGOLIA A DECADE OF NATIONAL DATA (2014–2024)

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Background / Hypothesis:

Mongolia carries one of the highest liver disease burdens worldwide. Cancer is the second leading cause of death, accounting for nearly one-fifth of all mortality, and hepatocellular carcinoma (HCC) is the most prevalent cancer type, representing approximately 40% of all cancers in the country. The major etiologies of HCC in Mongolia include hepatitis C virus (HCV) infection (46%), hepatitis B virus (HBV) infection (34%), HBV–HCV co-infection (14.4%), and alcohol-related liver disease (5.6%). This study aims to compare the newly reported incidence of hepatocellular carcinoma in Mongolia over the last decade.

Materials and Methods:

This desk review examined reported hepatocellular carcinoma (HCC) cases by utilizing national statistical datasets and reviewing policy documents released by relevant governmental authorities.

Results:

Hepatocellular carcinoma (HCC) in Mongolia remains a major public health challenge. Between 2015 and 2024, the country has maintained some of the highest liver cancer rates in the world. The age-standardized incidence of liver cancer in Mongolia has been reported around 63.2 cases per 100,000 people, markedly higher than global averages. In terms of sex distribution, HCC morbidity is very high at 80.3 per 100,000 men and 60.3 per 100,000 women. Among newly reported liver cancer cases, 2,373 were diagnosed during a recent period, of which 56.4% were men and 43.6% women, making up 32.8% of all cancer diagnoses in the country. Viral hepatitis remains the primary driver: chronic HBV and/or HCV infections account for a large portion of HCC risk. In fact, over 95% of liver cancer cases in Mongolia are attributable to hepatitis B or C. Population-level surveys show a high seroprevalence: for example, HBsAg positivity among adults is around 9–11.8%, and anti-HCV antibodies appear in 11–16% of the general population. Survival remains poor: older reports suggest a 1-year survival rate of ~21–24%, dropping to 6–8% by five years, highlighting late-stage diagnosis as a key problem.

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Conclusions:

Hepatocellular carcinoma remains a critical health burden in Mongolia, with persistently high incidence driven largely by chronic hepatitis B and C infections. Despite national efforts in prevention and policy development, HCC rates have shown little reduction over the past decade, and survival outcomes remain poor due to late diagnosis. Strengthened screening programs, improved antiviral treatment coverage, and earlier detection strategies are essential to reduce HCC morbidity and mortality in the coming years.

Keyword(s) : HCC, cancer, HBV, HCV, incidence

1247 - Clinical characteristics and prognosis of patients with primary biliary cholangitis a single center experience in Lanzhou city China

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Background:Information on the characteristics and prognosis of patients with Primary biliary cholangitis(PBC) in Lanzhou city,Gansu province,China,remains limited.This study aims to investigate the clinical characteristics and prognosis of patients with PBC in a single-center,retrospective cohort.

Materials and Methods:One hundred and nine PBC patients with complete data after one year or more of ursodeoxycholic acid (UDCA) treatment were selected finally.Data were collected at baseline and one year of treatment.Biochemical response was evaluated using the Barcelona criteria.Patients were divided into an IgM elevated group and an IgM control group according to whether the IgM level at baseline was a 15% higher than the upper limit of normal.The difference in prognosis between the two groups was evaluated using the UK-PBC score and GLOBE scores.

Results:The average age was 58.5 ± 10.7 years old,and the female-to-male ratio was approximately 4:1.The median values for the following were also recorded:alkaline phosphatase(ALP),181.0U/L(124.5U/L,265.0U/L),gamma-glutamyl transpeptidase(GGT),93.3U/L(35.8U/L,275.0U/L),alanine aminotransferase (ALT),36U/L (19.7U/L,70.0U/L),total bilirubin(TB),25.1umol/L(17.4umol/L,41.0umol/L),total bile acid(TBA),15.7umol/L(7.4umol/L,51.9umol/L),total cholesterol (TC),3.9 mmol/L(3.1mmol/L,5.0mmol/L),IgM level,2.5g/L(1.5g/L,4.0g/L).A complete biochemical response to UDCA was observed in 64 PBC

patients,accounting for a complete biochemical response rate of 58.7%.Anti-mitochondrial antibody M2 component(AMA-M2)-positive results were found in 72 patients.Fifty patients had elevated IgM, and 59 patients were in the IgM control group. Compared with the IgM control group,the IgM elevated group had higher TBIL,ALP,GGT and TBA, but lower ALB,this difference was statistically significant.($P < 0.05$). Correlation analysis showed that the IgM level was positively correlated with AST, ALP,GGT,TBIL and TBA,and negatively correlated with ALB in PBC patients.After one year of UDCA treatment,the patients in the IgM elevated group had a biochemical response rate than the IgM control group (48% vs 67.8%, $P < 0.05$) and a higher risk of liver transplantation within 5,10,15 years according to the UK-PBC score($P < 0.05$),although there was no significant difference in GLOBE score.

Conclusion: Among patients with simple PBC,elderly women predominate,while the male incidence is on the rise.The biochemical abnormalities of PBC patients with elevated IgM at baseline were more obvious.Current first-line UDCA therapy fails to meet clinical needs,particularly for patients with elevated IgM levels,who exhibit poorer treatment response and prognosis.There is an urgent need for safe and effective second-line medications for PBC in China.

Keyword(s) :

1248 - Progress Toward WHO 2030 Hepatitis B Mortality Target in the Asia-Pacific Region

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Background / Hypothesis:

Hepatitis B virus (HBV) remains a major cause of mortality in the Asia-Pacific region, which bears a substantial proportion of the global HBV-related death burden. Monitoring long-term mortality trends and assessing progress toward the WHO 2030 elimination target—a 65% reduction in HBV deaths from 2015 levels—is essential for guiding regional hepatitis control strategies.

Materials and Methods:

Using Global Burden of Disease (GBD) 2023 estimates,

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we analyzed age-standardised HBV mortality rates (ASMRs) for 26 Asia-Pacific countries from 1990 to 2023 across the High-income Asia Pacific, East Asia, Southeast Asia, and selected South Asian and Oceania regions. "Liver cancer due to hepatitis B" and "Chronic hepatitis B including cirrhosis" were included in the analysis. Temporal trends were assessed using the Estimated Annual Percentage Change (EAPC) derived from log-linear regression. Progress toward the WHO 2030 targets was evaluated by the percentage change in ASMR between 2015 and 2023.

Results:

The regional ASMR declined by 39.8% from 1990 to 2023, decreasing from 15.4 to 9.28 deaths per 100,000 population (EAPC -2.53%, 95% CI -2.78 to -2.27). Mortality continued to fall through 2021 (9.14 per 100,000) but showed a modest increase thereafter, particularly in middle- and high-SDI countries. Progress toward the WHO 2030 target varied substantially across countries. None of the 26 countries was on track for the 2030 goal. Bangladesh, Singapore, Pakistan, India, and Mongolia achieved moderate reductions (mean ASMR decline: 26.8%), whereas 16 countries—including China, Indonesia, Viet Nam, Philippines and Malaysia—showed slow progress (mean decline: 7.22%). New Zealand, Japan, Myanmar, Papua New Guinea, and Brunei Darussalam experienced worsening trends, with ASMR increasing by an average of 3.57% since 2015.

Conclusions:

Although HBV mortality has declined markedly over the past three decades, the Asia-Pacific region remains collectively off track to meet the WHO 2030 mortality reduction target. The recent stagnation and reversals observed after 2021 highlight persistent challenges in HBV prevention and treatment coverage. Strengthening antiviral therapy uptake, maintaining continuity of essential HBV services, and prioritizing investments in under-performing countries will be critical to accelerate progress toward regional HBV elimination.

Keyword(s) : HBV, WHO 2030 target, Mortality

1249 - RE LIVE Study (Re Linkage to Pan Viral Hepatitis) Interim Results from a Multicenter Observational Study on Care Engagement in Chronic Viral Hepatitis

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Background / Hypothesis:

Despite the availability of highly effective antiviral therapies, linkage to care remains a key barrier to the global elimination of viral hepatitis. A considerable proportion of individuals with chronic hepatitis B, C, and D remain undiagnosed, untreated, or lost to follow-up, sustaining the burden of disease and transmission. In Korea, reimbursement criteria for HBV treatment were expanded in 2023; however, a substantial number of eligible patients remain untreated. This study aimed to evaluate systematic re-linkage strategies to re-engage these individuals in the continuum of care, with the goal of improving clinical outcomes and contributing to public health objectives.

Materials and Methods:

A retrospective chart review was conducted at four tertiary academic hospitals in Korea to identify candidates for antiviral treatment. HBV eligibility included (i) non-cirrhotic patients with HBV DNA >2,000 IU/mL and fibrosis stage ≥2, and (ii) cirrhotic patients with any detectable HBV DNA. For HCV, partially diagnosed-but-untreated (DBU) cases were defined as anti-HCV antibody positive with missing HCV RNA data; completely DBU cases were HCV RNA positive without prior antiviral therapy. HBV patients eligible for HDV screening underwent HDV PCR. All identified patients were re-contacted via text or phone for re-evaluation and potential treatment linkage.

Results:

Among 2,192 patient charts reviewed, 33 HBV and 127 HCV patients were enrolled in the call-back program. Among HBV patients, 21.2% (7) responded and agreed to further evaluation; 6.1% (2) initiated antiviral therapy, while 78.8% (26) did not respond to call-back. Of 92 partial HCV DBU cases, 13.0% (12) agreed to evaluation, 3.3% (3) had prior treatment, 3.3% (3) sought care elsewhere, 2.2% (2) refused, and 78.3% (72) did not respond. Among them, 2.2% (2) initiated treatment. Among 35 complete DBU cases, 2.9% (1) agreed to evaluation, 2.9% (1) sought care elsewhere, 5.7% (2) refused, and 88.6% (31) did not respond; only

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2.9% (1) initiated treatment. HDV RT-PCR was performed in 618 chronic HBV patients; 0.1% (1) tested positive but declined further management. In total, 3.1% (5) of 160 patients identified through the call-back program initiated antiviral therapy.

Conclusions:

Although effective antiviral treatments and expanded eligibility criteria are now available, many patients with viral hepatitis remain disconnected from care. Our re-linkage efforts showed limited success in bringing patients back into the treatment pathway. These findings emphasize the need for continued, practical strategies to improve access to care and support national and global goals for viral hepatitis elimination.

Keyword(s) : viral hepatitis, linkage to care, HBV, HCV, elimination

1251 - ANXA4 Deficiency Ameliorates Acetaminophen Induced Acute Liver Injury

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Background: Acetaminophen (APAP) is widely used as an antipyretic and analgesic drug. However, APAP overdose is one of the leading causes of acute liver failure worldwide. Current therapeutic options for APAP-induced acute liver failure are limited. The severity of liver injury depends on the interconnection between metabolic detoxification and inflammatory signaling. Annexin A4 (ANXA4), a calcium-dependent phospholipid-binding protein, acts as a stress-responsive regulator that integrates metabolic and inflammatory pathways through its roles in membrane dynamics and signaling complex organization. This study aimed to elucidate whether ANXA4 deletion protects against acute liver failure and to define the molecular mechanisms underlying this effect.

Materials and Methods: We constructed mice with liver-specific knockout of ANXA4. Wild-type (WT) and ANXA4 knockout mice were administered APAP separately. Liver injury was assessed by serum liver function tests and histological evaluation. Oxidative stress indices and inflammatory mediators were quantified by biochemical assays and qPCR. Hepatic transcriptomic profiling was performed to identify genes associated with protective responses. Primary hepatocytes were used for mechanistic validation. Results: ANXA4 deficiency significantly alleviated APAP-induced liver injury and reduced mortality of

mice, as evidenced by lower serum aminotransferase, and attenuated centrilobular necrosis. The knockout livers exhibited lower reactive oxygen species accumulation and decreased activation of NF- κ B, accompanied by reduced expression of proinflammatory cytokines such as TNF- α and IL-6. Transcriptomic analysis revealed a pronounced increase in flavin-containing monooxygenase 3 (Fmo3), an enzyme involved in xenobiotic detoxification and redox regulation. Elevated Fmo3 expression correlated with improved oxidative balance and suppression of inflammatory signaling. In isolated hepatocytes, ANXA4 deletion enhanced resistance to APAP-induced mitochondrial dysfunction, supporting a direct cytoprotective mechanism linked to coordinated modulation of metabolic and inflammatory pathways. Conclusions: Inhibition of ANXA4 confers a protective effect against APAP-induced acute liver injury, not only by attenuating the immune response but also by effectively activating the APAP detoxification pathway through modulation of drug metabolism. Our findings suggest that ANXA4 may serve as a potential therapeutic target for the treatment of APAP-induced acute liver injury.

Keyword(s) : Liver injury, ANXA4, acetaminophen

1253 - Clinical Trajectories of Acute Decompensation in Cirrhosis The First Prospective PREDICT Based Study from India

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Background / Hypothesis:

Acute decompensation (AD) of cirrhosis is a heterogeneous clinical state with variable short-term outcomes. The PREDICT study identified three phenotypes—Stable Decompensated Cirrhosis (SDC), Unstable Decompensated Cirrhosis (UDC), and pre-Acute-on-Chronic Liver Failure (pre-ACLF)—driven largely by systemic inflammation. This study aimed to evaluate the clinical trajectories of AD based on the PREDICT framework in an Indian cohort and to identify inflammatory and biochemical predictors of progression to ACLF and 6-month mortality.

Materials and Methods:

This prospective observational study included 105 consecutive adults with cirrhosis admitted with AD to a

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tertiary-care center over 12 months. AD was defined by new or worsening ascites, hepatic encephalopathy (HE), gastrointestinal bleeding, or bacterial infection.

Patients with ACLF at presentation, immunosuppression, pregnancy, or post-transplant status were excluded. All participants were followed for 90 days to classify outcomes as SDC, UDC, or pre-ACLF, and for 6-month survival. Demographic, clinical, and laboratory parameters—including Hs-CRP, TNF- α , IL-6, and urinary NGAL—were recorded at baseline. Group comparisons were performed using appropriate statistical tests, and variables with $p < 0.1$ on univariate analysis were entered into multivariate logistic regression for prediction of ACLF and mortality. Prognostic accuracy of Hs-CRP was assessed using ROC analysis.

Results:

Of 105 patients (mean age 46.6 ± 10.9 years; 82.9% male), 57 (54.3%) were classified as SDC, 24 (22.9%) as UDC, and 24 (22.9%) as pre-ACLF at 90 days. Pre-ACLF patients had significantly higher prevalence of HE (54%), bacterial infections (46%), SBP (50%), sepsis (38%), renal dysfunction (creatinine 1.47 ± 0.33 mg/dL), and markedly elevated inflammatory markers (Hs-CRP 38.1 ± 14.6 mg/L; TNF- α 157 ± 50 pg/mL; IL-6 126 ± 45 pg/mL; all $p < 0.001$). Thirty-one patients (29.5%) died by 6 months. Non-survivors showed significantly higher baseline inflammation, organ dysfunction, and severity scores (MELD-Na 22.4 ± 5.8 vs 16.6 ± 5.6 , $p < 0.001$). On multivariate analysis, Hs-CRP independently predicted both ACLF (OR 1.057; $p = 0.04$) and 6-month mortality (OR 1.111; $p = 0.03$). Hs-CRP demonstrated excellent prognostic accuracy (AUC 0.92), with an optimal cut-off of 10.8 mg/L (90% sensitivity, 95% specificity).

Conclusions:

In this first Indian prospective validation of the PREDICT classification, inflammatory phenotype strongly determined clinical trajectory and survival in AD. Pre-ACLF was characterized by severe systemic inflammation and high early mortality. Hs-CRP emerged as a simple, robust, and independent predictor of ACLF and 6-month mortality. Routine CRP assessment may greatly improve early risk stratification in acutely decompensated cirrhosis.

Keyword(s) : Acute decompensation, Acute-on-chronic liver failure (ACLF), Cirrhosis, PREDICT classification, Systemic inflammation, High-sensitivity, C-reactive protein (Hs-CRP), TNF-alpha, IL-6, Urinary NGAL, Prognostic markers, Mortality, Indian cohort

1255 - Chenodeoxycholic Acid Modulation via *Bacteroides intestinalis* AM1 underscores a Novel Approach in Acute Liver Failure

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Background / Hypothesis:

Acute liver failure (ALF) is associated with rapid and progressive hepatocellular injury, and severe metabolic-microbial derangements. We investigated early metabolic markers of non-survival, and a potential microbial intervention using *Bacteroides intestinalis*-AM1, to improve outcomes in ALF.

Materials and Methods: Plasma metabolomics and meta-proteomics were performed in 40 ALF patients and 5 healthy controls (training cohort). A non-survival marker panel was identified and validated in 270 ALF patients (test cohort) using high resolution mass spectrometry and machine learning. It was functionally validated in acetaminophen-induced ALF mouse model. *B. intestinalis*-AM1 was used to study alteration of gut bacteria and amelioration of liver injury.

Results: ALF non-survivors showed a distinct metabolomic signature with elevated primary bile acids, tryptophan, tyrosine, and enrichment of pathways linked to inflammation, cell death, and mitochondrial dysfunction ($p < 0.01$, FDR < 0.01 , FC > 1.5). Non-survivors had higher alpha/beta diversity ($p < 0.05$) with increase in Proteobacteria, Firmicutes, Actinobacteria ($p < 0.05$); functionally associated with energy, amino acid and xenobiotic metabolism ($p < 0.05$). A gut microbiota derangement in converting primary to secondary bile acids was evident as CDCA and cytotoxic metabolites (4-(2-Amino phenyl)-2,4-dioxobutanoate, L-Tyrosine) were higher. Elevated CDCA (logFC > 10) levels correlated with mortality in ALF patients as well as in mouse model. In this case, administration of *B. intestinalis*-AM1 bacteria (10^9 CFU/ml) reduced CDCA and CA levels by upregulating FXR, FGF15, and SLC10A1 gene expression. It also promoted glutathione synthesis through the activity of

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enzymes such as L-histidine amino ligase, GSH synthetase, and others ($FC > 1.5$; $P < 0.05$). As a result, inflammation was suppressed via inhibition of IL-1 β and TLR4 signaling. This treatment further enhanced glutathione-dependent oxidative repair, mitigated mitochondrial dysfunction, and ultimately improved necroptosis outcomes.

Conclusions: Elevated chenodeoxycholic acid (CDCA) levels are associated with poor prognosis in ALF patients. B. intestinalis-AM1, a primary-to-secondary bile acid converter, effectively lowers CDCA levels, activates FXR, enhances glutathione production, and reduces inflammation and mitochondrial dysfunction, thereby protecting hepatocytes and underscoring its therapeutic potential in ALF.

Keyword(s) : acute liver failure, chenodeoxycholic acid, bacteriodes intestinalis AM-1, mitochondrial dysfunction

1256 - Evaluation of Information Quality and Reliability of Chronic Hepatitis B Related Short Videos on TikTok and Bilibili

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Background / Hypothesis:

Chronic hepatitis B (CHB) is a globally prevalent infectious liver disease. The scientific dissemination of knowledge regarding its prevention and management is critical for reducing incidence and improving rates of accurate diagnosis and appropriate treatment. With the widespread adoption of short video platforms, TikTok and Bilibili have become core channels for the Chinese public to access CHB-related information; however, systematic evaluations of the quality and reliability of CHB-focused short videos on these two platforms remain scarce.

Materials and Methods:

A total of 149 valid videos were retrieved from TikTok and Bilibili using "乙肝" as the keyword. Three validated tools—Global Quality Score (GQS), Modified DISCERN, and Journal of the American Medical Association (JAMA) Benchmark Criteria—were employed to assess the information quality of the videos. Video characteristics were analyzed, and differences between the two platforms as well as across uploader identities were compared.

Results:

The overall quality of CHB science popularization short videos on both platforms was low, with the following median (interquartile range) scores: GQS 3 (2, 4), Modified DISCERN 2 (1, 2), and JAMA 2 (1, 2). TikTok outperformed Bilibili significantly in terms of quality scores, and also exhibited advantages in interaction metrics (likes, comments, favorites, shares) and uploader certification rates; in contrast, Bilibili videos had a significantly longer duration. Regarding uploader identity, videos posted by hepatology specialists achieved significantly higher quality scores than those by non-specialist uploaders. Correlation analysis revealed that all interaction metrics exhibited little to no correlation with quality scores.

Conclusions:

The information quality and reliability of CHB-related short videos on TikTok and Bilibili are both poor and need to be improved; platform attributes and uploaders' professional backgrounds are closely related to the quality of short videos; the interaction rate is not proportional to the quality of short videos, and videos with a high interaction rate do not necessarily have good quality and reliability.

Keyword(s) : Chronic Hepatitis B; TikTok; Bilibili; Short Videos; Information Quality; Reliability

1258 - The Liver in Systemic Diseases A Key Metabolic Regulator

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Background / Hypothesis:

The liver plays a central role in systemic metabolism, immune regulation, and detoxification. We hypothesize that liver dysfunction significantly contributes to the progression of extrahepatic diseases, including metabolic syndrome, cardiovascular disorders, and systemic inflammation.

Materials and Methods:

We conducted a retrospective cohort study including 1,200 patients with documented liver conditions (NAFLD, ALD, viral hepatitis) and matched controls. Serum biomarkers, imaging studies, and histopathological data were analyzed. Multivariate regression models were used to assess associations between liver function and systemic disease outcomes.

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Results:

Patients with advanced liver disease showed a higher prevalence of type 2 diabetes (42% vs. 18%, $p < 0.01$), coronary artery disease (28% vs. 12%, $p < 0.05$), and chronic kidney disease (19% vs. 7%, $p < 0.01$). Liver stiffness and elevated ALT levels were independent predictors of systemic morbidity.

Conclusions:

The liver is critically involved in the pathogenesis and progression of multiple systemic diseases. Early detection and management of liver disorders may reduce the burden of extrahepatic complications and improve overall patient outcomes.

Keyword(s) : Liver Involvement, Systemic Diseases, Extrahepatic Manifestations, Metabolic Dysfunction, Biomarkers

1259 - The Central Role of Liver Dysfunction in Systemic Metabolic and Cardiovascular Diseases Remaining characters

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The liver is a pivotal metabolic and immunomodulatory organ, yet its systemic influence beyond classical hepatology is underestimated. We hypothesized that the severity of liver fibrosis, measured non-invasively, is an independent predictor for the incidence and progression of key extrahepatic diseases, including type 2 diabetes (T2D), atherosclerotic cardiovascular disease (ASCVD), and chronic kidney disease (CKD). A prospective, multicenter cohort study was conducted from 2020 to 2024, enrolling 2,850 adults with varying degrees of non-alcoholic fatty liver disease (NAFLD). All participants underwent comprehensive assessment at baseline: clinical evaluation, laboratory tests (including liver enzymes, lipid profile, and HbA1c), vibration-controlled transient elastography (VCTE) to determine liver stiffness measurement (LSM), and abdominal ultrasound. They were followed annually for the primary endpoints: new-onset T2D, major adverse cardiovascular events (MACE), and progression of CKD. Over a median follow-up of 3.5 years, 289 (10.1%) participants developed T2D, 156 (5.5%) experienced a MACE, and 203 (7.1%) showed significant CKD progression. Multivariable Cox regression analysis, adjusted for age, sex, BMI, and traditional risk factors, revealed that elevated baseline LSM was a powerful independent risk factor. Each 1-kPa increase in LSM was associated with a 15% higher risk for T2D (HR 1.15,

95% CI 1.09-1.22), an 18% higher risk for MACE (HR 1.18, 95% CI 1.10-1.27), and a 12% higher risk for CKD progression (HR 1.12, 95% CI 1.05-1.19). Participants with LSM ≥ 10.0 kPa (indicating advanced fibrosis) had a 3.2-fold increased risk for the composite endpoint compared to those with LSM < 7.0 kPa.

Liver fibrosis severity is a robust, independent predictor for the development of critical systemic diseases. Our findings compellingly argue for the integration of liver health assessment, particularly non-invasive fibroscan, into the routine risk stratification for T2D, ASCVD, and CKD. Proactive management of liver disease may not only prevent hepatic complications but also mitigate the global burden of major extrahepatic morbidity and mortality.

Keyword(s) : Liver Fibrosis, Systemic Diseases, NAFLD, Cardiovascular Disease, Type 2 Diabetes, Prognostic Marker

1260 - Mitochondrial Dysfunction in NAFLD A Public Health Perspective Remaining characters

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Non-alcoholic fatty liver disease (NAFLD) is a burgeoning public health crisis, yet the underlying pathological mechanisms driving its progression remain incompletely understood. We hypothesized that hepatic mitochondrial dysfunction is a critical early event in NAFLD pathogenesis, and that quantifying this dysfunction could serve as a novel biomarker for disease progression and systemic metabolic complications, informing public health screening strategies.

We conducted an integrated study combining in vitro and clinical cohorts. In vitro, we exposed human HepG2 hepatocytes to free fatty acids and assessed mitochondrial function via Seahorse Analyzer, oxidative stress markers, and apoptosis. In vivo, we analyzed a prospective community-based cohort of 1,500 individuals with ultrasonographically confirmed NAFLD. Peripheral blood mononuclear cells (PBMCs) were isolated as a surrogate tissue to measure mitochondrial DNA (mtDNA) copy number and respiratory complex activity. All participants were followed for 3 years for the primary outcome of fibrosis progression (measured by FIB-4 index increase ≥ 0.5) and incident type 2 diabetes (T2D).

In vitro, fatty acid-overloaded hepatocytes exhibited significant mitochondrial depolarization, reduced ATP production, and increased ROS, culminating in cell death. In vivo, patients in the lowest tertile of PBMC

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mtDNA copy number at baseline had a significantly higher risk of fibrosis progression (HR: 2.8, 95% CI: 1.9-4.1, $p < 0.001$) and new-onset T2D (HR: 2.1, 95% CI: 1.5-3.0, $p < 0.001$), independent of age, BMI, and HOMA-IR. A combined model integrating low mtDNA copy number and high FIB-4 index showed superior predictive accuracy for 3-year metabolic deterioration (AUC=0.82) compared to either marker alone. Our findings from bench to population demonstrate that mitochondrial dysfunction is a cornerstone of NAFLD pathology. Critically, we establish that a simple, measurable biomarker of this dysfunction (PBMC mtDNA copy number) holds significant prognostic value for predicting liver fibrosis and diabetes risk. This presents a compelling public health opportunity: incorporating mitochondrial health assessment into NAFLD management protocols could enable earlier identification of high-risk individuals, facilitating targeted interventions and alleviating the future healthcare burden.

Keyword(s) : NAFLD, Mitochondrial Dysfunction, Public Health, Biomarker, Disease Progression, Fibrosis

1261 - Single Cell Atlas Reveals Immunometabolic Drivers of NAFLD HCC Transition Remaining characters

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Hepatocellular carcinoma (HCC) arising from non-alcoholic fatty liver disease (NAFLD) is becoming a leading cause of liver-related mortality worldwide. A critical gap exists in understanding the pre-malignant immune microenvironment. We hypothesized that a specific, metabolically reprogrammed CD8⁺ T cell subset accumulates in advanced NAFLD, fostering an immunosuppressive niche that licenses hepatocarcinogenesis.

We employed an integrated approach. A novel murine model of NAFLD-driven HCC (fed a high-fat, high-cholesterol, high-fructose diet) was utilized. Liver immune cells from pre-malignant (6-month) and tumor-bearing (12-month) stages were analyzed using single-cell RNA sequencing (scRNA-seq) coupled with Cellular Indexing of Transcriptomes and Epitopes (CITE-seq). Key findings were validated in a human cohort using multiplex immunofluorescence on liver biopsies from 60 patients with NAFLD, with or without HCC.

scRNA-seq of 45,000 murine liver cells unveiled a trajectory of CD8⁺ T cell exhaustion, culminating in a previously unrecognized subset termed "metabolically

exhausted" CD8⁺ T cells (Tmet-ex). These Tmet-ex cells, enriched in pre-malignant livers, exhibited a unique dual signature: high expression of exhaustion markers (e.g., PD-1, TOX) and a dysregulated metabolic profile with impaired mitochondrial fatty acid oxidation and increased glycolytic dependency. In vivo depletion of this subset significantly reduced subsequent tumor incidence ($p < 0.01$). Crucially, we identified the surface protein Folate Receptor beta (FR β) as a specific marker for Tmet-ex cells. In human NAFLD biopsies, the density of FR β ⁺ CD8⁺ T cells was significantly higher in patients who progressed to HCC within 3 years (AUC=0.88 for prediction).

Our study provides the first single-cell resolution atlas of the immunosuppressive transition from NAFLD to HCC, identifying a metabolically distinct, protumorigenic CD8⁺ T cell subset. The discovery of FR β as a surface marker offers a direct translational path. This opens a novel avenue for public health strategy: targeting FR β ⁺ Tmet-ex cells could serve as a chemopreventive measure for high-risk NAFLD patients, potentially halting HCC development before it begins.

Keyword(s) : NAFLD-HCC, Immunometabolism, Single-Cell Sequencing, T Cell Exhaustion, Cancer Prevention, Biomarker

1262 - Gut Microbiota Derived Metabolites Predict Advanced Fibrosis in MAFLD A Machine Learning Approach Remaining characters

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Metabolic dysfunction-associated fatty liver disease (MAFLD) poses a massive disease burden, with rapid identification of advanced fibrosis being a critical public health priority. The gut-liver axis is implicated in pathogenesis, but its translational potential for community screening remains untapped. We hypothesized that a signature of circulating gut microbiota-derived metabolites, integrated with clinical data via machine learning, could non-invasively and accurately identify patients with advanced fibrosis, outperforming current biomarkers.

We conducted a multi-stage, cross-sectional study. In the discovery phase, untargeted metabolomics was performed on plasma from 400 well-phenotyped MAFLD patients (derivation cohort). Significant metabolites were validated using targeted LC-MS/MS in an independent cohort of 800 MAFLD patients (validation cohort). All participants underwent liver

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stiffness measurement (LSM) by VCTE as the reference standard (advanced fibrosis: LSM ≥ 10.0 kPa). A machine learning model (XGBoost) was trained to predict advanced fibrosis using the metabolite panel and routine clinical variables (e.g., age, BMI, platelets, AST/ALT). Its performance was compared to FIB-4 and NFS.

Untargeted metabolomics identified 12 metabolites significantly associated with advanced fibrosis, primarily belonging to bile acid and aromatic amino acid pathways. Three key metabolites—phenylacetylglutamine (PAGln), a secondary bile acid (isoDCA), and indole-3-propionic acid (IPA)—were consistently validated. The XGBoost model, incorporating these three metabolites and clinical data, dramatically outperformed traditional tests. In the validation cohort, it achieved an AUC of 0.94 (95% CI: 0.91-0.97), with a sensitivity of 92% and specificity of 88%. In contrast, FIB-4 and NFS achieved AUCs of 0.78 and 0.75, respectively. The model also accurately reclassified 35% of FIB-4 indeterminate cases. This study establishes a robust link between specific gut microbiota-derived metabolites and MAFLD severity. More importantly, we demonstrate that a machine learning model leveraging this "metabolic fingerprint" provides a superior, non-invasive tool for detecting advanced fibrosis at the population level. This AI-powered approach represents a paradigm shift in public health strategy, enabling high-throughput, accurate risk stratification that could transform MAFLD screening and direct resources to those at greatest need.

Keyword(s) : MAFLD, Gut-Liver Axis, Machine Learning, Metabolomics, Fibrosis Prediction, Public Health Screening

1264 - Efficacy of Percutaneous Microwave Ablation in Exophytic Hepatocellular carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is the commonest

primary cancer of the liver. HCC location is a critical factor for local recurrence following percutaneous ablation. Exophytic HCC is a challenging location with high risk of tumor rupture. HCCs in these locations are more prone for residual disease or local progression owing to creation of suboptimal ablation volume. The aim of this study was to evaluate the efficacy of percutaneous microwave ablation (MVA) for exophytic (HCC) and outcome after 6 months follow up.

Materials and Methods:

A prospective study was conducted on 40 cases with exophytic HCC. Microwave ablation was performed percutaneously for all patients. Artificial ascites was made in sub-diaphragmatic lesions & Exophytic lesion adjacent to bowel. Patients were followed for 1, 3, and 6 months postoperatively.

Results:

The complications of MVA were reported by 7.5 % of cases and were minor complications. 80 % of cases showed complete ablation while 20 % of cases have incomplete ablation. There was significant decrease in Alpha fetoprotein before and after MVA.

Conclusions:

Percutaneous microwave ablation showed a good result for treating exophytic hepatocellular carcinoma with little complications.

Keyword(s) : Percutaneous microwave ablation, Exophytic hepatocellular carcinoma, Alpha fetoprotein.

1267 - Clinical Characteristics and Prognostic Analysis of Patients with Primary Biliary Cholangitis Complicated with Hepatic Granulomas

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Background / Hypothesis: Primary biliary cholangitis (PBC) is an autoimmune liver disease characterized by intrahepatic small bile duct inflammation, progressive fibrosis, and potential progression to cirrhosis or liver failure. With globally rising incidence and prevalence, its burden is increasing. Ursodeoxycholic acid (UDCA) is first-line therapy but fails to cure, with 30-40% of patients showing inadequate response. The need to optimize PBC management is unmet. Hepatic granuloma is observed in many PBC patients, but the association of hepatic granuloma with PBC and its

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impact remain unclear.

Methods: A retrospective analysis was conducted on 319 patients diagnosed with PBC and undergoing liver biopsy at Beijing You'an Hospital, Capital Medical University, from 2014 to 2024. Histological staging was performed using the Ludwig classification system (Stage I–IV). Serum immunological markers, pathological features, and GLOBE scores were compared between the two groups.

Results: Among the 319 PBC patients, hepatic granulomas were detected in 79 PBC patients (24.8%). Compared with the non-granulomatous group, the granulomatous group exhibited significantly higher globulin (GLB) [(34.40±5.23) g/L vs (32.95±4.81) g/L] and lower total bile acid (TBA) [(11.7±8.5) µmol/L vs (16.0±12.3) µmol/L] (both $P < 0.05$). The positive rate of anti-centromere antibody (ACA) in the granulomatous group (27.9%) was significantly higher than that in the non-granulomatous group (17.2%), with a statistically significant difference ($P=0.028$).

GLOBE scores showed no significant difference between groups [(0.49±0.12) vs (0.58±0.15), $P=0.075$]. Among 79 PBC patients with hepatic granulomas, epithelioid granulomas were observed in 65 cases (82.3%), microgranulomas in 13 cases (16.5%), and mixed granulomas in 1 case (1.3%); 65 cases (82.3%) were located in the portal area, 11 cases (13.9%) in the lobule, and 3 cases (3.8%) in cross-regional distribution. Histological staging revealed that among the 79 PBC patients with hepatic granulomas, 78.5% (62/79) were in stages I–II, which was significantly higher than the proportion of stages I–II in overall PBC patients (63.0%, 201/319). Furthermore, the detection rate of granulomas showed a decreasing trend with advancing disease stage (detection rate of stage I: 32.1%; stage II: 38.5%; stage III: 18.6%; stage IV: 5.9%), with a statistically significant difference ($P < 0.05$).

Conclusion: Hepatic granulomas are more prevalent in the early stages of PBC, diminishing progressively with disease progression, suggesting they may represent a transitional pathological manifestation within the PBC disease course. PBC with hepatic granulomas may denote a distinct disease subtype, and its prognostic implications warrant validation through larger sample cohorts.

Keyword(s) : Primary biliary cholangitis; Hepatic granuloma; Clinical features; Pathological staging; GLOBE score

DATA

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Background / Hypothesis:

Mongolia has implemented several key policies to reduce the incidence of viral hepatitis, including the National Program for the Prevention and Control of Infectious Diseases and the Whole Liver Program (Hepatitis Prevention, Control, and Elimination Program). This study aims to estimate the new incidence of viral hepatitis in Mongolia and evaluate the impact of these policy measures in combating the disease.

Materials and Methods:

This desk review examined reported hepatocellular carcinoma (HCC) cases by utilizing national statistical datasets and reviewing policy documents released by relevant governmental authorities.

Results:

As of 2025, 270 cases of acute hepatitis virus infection have been registered nationwide, equating to 0.8 cases per 10 000 population. Compared to last year, there were 49 additional cases, resulting in an increase of 0.1 case per 10 000 population. In the last decade, the incidence of acute hepatitis virus infection at the national level was highest in 2015, with a rate of 4.0 cases per 10 000 population, and has been steadily decreasing since 2016. As of 2024, the breakdown of hepatitis virus infections is as follows: hepatitis A virus infection accounts for 0.7% of all cases, hepatitis B virus infection for 46.3%, hepatitis C virus infection for 44.5%, and other hepatitis virus infections for 8.5%. In 2024, 54.1% of acute hepatitis virus infection cases were male, and 45.9% were female. Age distribution showed that 30.4% of the cases were individuals aged 20–29 years, and 32.6% were aged 30–39 years. Outpatient services diagnosed 88.9% of all infections, and there were 8 reported deaths due to viral hepatitis infection.

Conclusions:

Last decades, Mongolia has been reducing the

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incidence of new infections through HPCE programs, public awareness campaigns, and new antiviral medicines have been introduced into chronic viral hepatitis treatment.

Keyword(s) : Acute viral hepatitis, HCC, HBV, HCV, Mongolia

1269 - RISK FACTORS AND CLINICAL FEATURES OF ACUTE HEPATITIS D IN MONGOLIA

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Background / Hypothesis:

Hepatitis D virus (HDV) infection is associated with more severe liver disease, including cirrhosis and hepatocellular carcinoma (HCC). Mongolia has one of the highest prevalence rates of viral hepatitis worldwide, with over 15% of the population infected with HCV, more than 10% with HBV, and HDV detected in 75–100% of HBsAg carriers. This study aims to investigate the clinical prognosis and risk factors of acute hepatitis D in Mongolia.

Materials and Methods:

A total of 172 patients diagnosed with acute viral hepatitis D were enrolled in the study, and their demographic, clinical, and laboratory data were collected between 2021 and 2024. Patients were evaluated for risk factors, disease severity, serological markers (anti-HDV IgM and total anti-HDV antibodies), and follow-up outcomes, including viral persistence and HBsAg clearance.

Results:

The mean age of patients was 29.7 ± 7.2 years, with 104 males (60.4%) and 68 females (39.6%). Reported risk factors included unprotected sexual contact (82, 47.7%), hospitalization (84, 48.8%), sharing nail clippers (154, 86%), family contact with hepatitis B (46, 26.7%), dental procedures (22, 12.8%), history of surgery (26, 15.1%), tattooing (16, 9.3%), and acupuncture (24, 13.9%). Serological testing showed that both anti-HDV IgM and total anti-HDV antibodies were positive in 68 of 172 patients. Anti-HDV IgM alone was positive in 130 patients, while total anti-HDV antibodies alone

were positive in 122 patients. During follow-up, among 10 patients with co-infection (HDV + HBV), 3 (30%) achieved HBsAg clearance. In contrast, all 116 patients with superinfection demonstrated persistent HBV and HDV viremia, indicating chronic infection

Conclusions:

Risk factors for hepatitis D virus infection were unprotected sexual contact, admitted hospital, share with nail clipper and family contacts with viral hepatitis. During follow-up, three of 10 (60%) patients with co D infection showed HBsAg loss and 116 patients with super D infection (100%) showed persistent hepatitis B and D viremia.

Keyword(s) : Hepatitis D virus infection, HBsAg, anti-HDV, anti-HDV IgM, Mongolia

1270 - Treatment Outcomes of HCV Genotype 1b with Ledipasvir Sofosbuvir and the Role of Resistance Associated Substitutions in Mongolia

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Background / Hypothesis:

Mongolia ranks among the countries with the highest hepatitis C virus (HCV) prevalence worldwide. Ledipasvir/sofosbuvir (LDV/SOF) has been introduced in Mongolia since 2016 to support national efforts to eliminate HCV. In studies from Western populations, resistance-associated substitutions (RASs) in HCV have been shown to compromise the effectiveness of LDV/SOF therapy in chronic hepatitis C (CHC) patients. This study aims to assess the treatment efficacy of LDV/SOF and examine the impact of RASs on therapeutic outcomes in Mongolian patients with CHC.

Materials and Methods:

HCV genotype 1b (GT1b) patients were prospectively enrolled in Mongolia and treated with a 12-week course of ledipasvir/sofosbuvir (LDV/SOF). Pre-treatment NS5A Y93H resistance-associated substitutions (RASs) in viral quasiespecies were analyzed using next-generation sequencing. Treatment effectiveness was assessed by the sustained virological response at 12 weeks post-treatment (SVR12).

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Results:

A total of 94 patients with chronic hepatitis C (CHC) were included in the analysis. The baseline proportion of the NS5A Y93H resistance-associated substitution (RAS) was <1% in 74 patients, 1–15% in 7 patients, 15–50% in 2 patients, and ≥50% in 11 patients. All participants completed the 12-week course of ledipasvir/sofosbuvir (LDV/SOF), achieving an overall sustained virological response at 12 weeks (SVR12) rate of 90.4%. The likelihood of failing to achieve SVR12 increased with higher baseline Y93H levels: 0% in patients with <1%, 14.3% in those with 1–15%, and 61.5% in patients with ≥15% Y93H (p for trend <0.001). In univariable analysis, treatment failure was associated with older age, baseline alanine transaminase (ALT) <40 U/mL, and a higher proportion of Y93H. However, multivariable analysis identified only a higher baseline proportion of Y93H as an independent predictor of treatment failure ($p = 0.022$).

Conclusions:

LDV/SOF therapy demonstrates a high sustained virological response (SVR) rate in Mongolian CHC patients with genotype 1b who lack baseline Y93H resistance-associated substitutions (RASs). Conversely, a higher baseline proportion of Y93H can substantially reduce the effectiveness of LDV/SOF treatment.

Keyword(s) : Chronic hepatitis C; Resistance-associated substitution; Ledipasvir; Sofosbuvir; Mongolia

1271 - CLINICAL AND LABORATORY CHARACTERISTICS OF CHRONIC HEPATITIS D VIRUS INFECTION IN MONGOLIA

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Background / Hypothesis:

Hepatitis D virus (HDV) infection progresses more aggressively than hepatitis B virus (HBV) mono-infection. Within two years, 10–15% of patients develop severe liver disease, and 70–80% progress to cirrhosis within 5–10 years. The risk of hepatocellular

carcinoma (HCC) is approximately three times higher than in HBV mono-infected patients. This study aimed to evaluate the clinical characteristics and laboratory profiles of patients with chronic HDV infection in Mongolia.

Materials and Methods:

From October 2024 to October 2025, laboratory and clinical data of patients with chronic HDV infection attending the outpatient department of the National Center for Communicable Diseases (NCCD) were analyzed. Data were processed and analyzed using SPSS 25 software.

Results:

A total of 252 patients with chronic HDV infection were included. Among them, 197 (78.2%) had chronic hepatitis, while 55 (21.8%) had liver cirrhosis. The mean age was 48.4 ± 0.66 years, with 151 (59.9%) males and 101 (40.1%) females. Prior HBV antiviral therapy was reported in 112 patients (44.4%), including tenofovir (72.5%), entecavir (27.1%), and adefovir (0.4%). Two patients initiated bulevirtide therapy for HDV in 2023. The mean duration of HBV antiviral therapy was 2.9 ± 0.3 years (range 3 months–10 years). Liver function tests showed elevated transaminase levels: ALT 63.1 ± 5.5 U/L, AST 60.7 ± 38.7 U/L, bilirubin 21.6 ± 3.8 $\mu\text{mol/L}$, and platelet count $194.4 \pm 6.5 \times 10^3/\mu\text{L}$. Serological testing revealed a mean qHBsAg of $7,029.5 \pm 926.6$ IU/mL. Among 67 patients tested for HBeAg, 62 (92.5%) were negative and 5 (7.5%) positive. HBV-DNA levels averaged $11,157,679.2 \pm 7,889,122.6$ IU/mL. HDV-RNA quantification showed 15.3% of patients with levels $>10^7$, 29.6% at 10^6 , 31.6% at 10^5 , 7.1% $<10^4$, and 16.3% with undetectable levels. The mean APRI score for liver fibrosis was 1.0 ± 0.1 .

Conclusions:

Chronic HDV infection in Mongolia predominantly affects middle-aged adults, with 21.8% already presenting with cirrhosis. Laboratory findings indicate elevated transaminases and high viral replication, with 44.9% of patients exhibiting HDV-RNA levels above 10^6 . These findings underscore the aggressive nature of HDV and the need for early diagnosis and therapeutic interventions.

Keyword(s) : HDV, HDV-RNA, APRI score, HCC, treatment



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1273 - Community based interventions for hepatitis C virus elimination among people who use drugs in Shanghai China

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Background:

The prevalence of hepatitis C virus (HCV) infection among people who use drugs (PWUD) in China remains high, yet diagnosis and treatment rates are low. Strategies are urgently needed to achieve the World Health Organization's goal of eliminating HCV as a public health threat by 2030.

Aim: To improve HCV diagnosis and treatment rates among PWUD in Pudong New Area, Shanghai, China.

Materials and Methods:

A retrospective and prospective study was implemented among individuals with substance addiction history in Pudong New Area, Shanghai, China. Three models were explored in our study. In the Self-Referral (SR) model, HCV antibody-positive individuals relied on their own initiative to seek medical services. In the Standard Intensive Referral (SIR) model, managerial supervision was strengthened. The Active Intensive Referral (AIR) model placed greater emphasis on managerial proactivity, enhancing screening and treatment for high-risk populations.

Results:

A total of 3926 PWUD from 36 communities in Pudong New Area underwent rapid HCV antibody testing in the SR model. These numbers were 1973 and 191, respectively, in the SIR and AIR models. Only 18.9% population completed HCV RNA testing in the SR model compared with 100.0% in both the SIR and AIR models ($P < 0.001$). Overall, 70/146 (48.0%) PWUD in the SR model were identified with HCV RNA consistent

with the SIR and AIR models [339/916 (37.0%) and 74/191 (38.7%), $P > 0.05$]. Only 73 (21.5%) patients received DAAs treatment. In terms of treatment, 47/74 (63.5%) of patients in the AIR model initiated treatment, significantly higher than the 15/70 (21.4%) in the SR model and 73/339 (21.5%) in the SIR model ($P < 0.001$). Genotype 3, particularly subtype 3b, was the most prevalent genotype among PWUD in our study, respectively accounting for 23/38 [60.5%] in the AIR model and 112/166 [67.5%] in the SIR model. In the AIR model, all participants received a liver stiffness test. Fifty-two (27.2%) patients had a liver stiffness of more than 12.5kPa. One hundred and eighty-one persons completed the questionnaire in the AIR model. The results revealed that few people had high educational attainment. More than half (52.5%) of the population had no stable marital relationship.

Conclusions:

A multidisciplinary community-based approach significantly improved HCV diagnosis and treatment among PWUD, offering a scalable model for achieving the WHO elimination targets in China.

Keyword(s) : Hepatitis C virus, People who use drugs, Direct-acting antivirals, Community

1274 - Constructing a dual dimensional scoring system by integrating tertiary lymphoid structures and monocyte lymphocyte ratio to optimize recurrence risk stratification in hepatocellular carcinoma a multicenter cohort study

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Background / Hypothesis: The monocyte/lymphocyte ratio (MLR) reflects systemic inflammatory-immune status, while tertiary lymphoid structures (TLS) reflect local anti-tumor immunity. Both markers are associated with hepatocellular carcinoma (HCC) prognosis. This study aims to evaluate a dual-dimensional scoring system integrating TLS and MLR for accurately predicting HCC recurrence risk, and to assess their interaction with adjuvant PD-1 inhibitors.

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Materials and Methods: A total of 1,139 patients who underwent curative resection for HCC without adjuvant therapy were retrospectively enrolled from 4 institutions in China between January 2019 and December 2023. These patients were stratified based on their TLS and MLR status. Additionally, 195 patients who received adjuvant PD-1 inhibitor therapy were prospectively recruited to validate the clinical utility of the scoring system. The X-tile software was employed to determine the optimal cutoff value for MLR (defined as MLR-high if >0.27 and MLR-low otherwise). Recurrence-free survival (RFS) and overall survival (OS) were evaluated. Fifty-five patients in the prospective cohort underwent bulk RNA sequencing to explore underlying mechanisms.

Results: Patients were categorized into four subgroups: 382 (33.5%) TLS+ /MLR-low, 282 (24.8%) TLS+ /MLR-high, 244 (21.4%) TLS- /MLR-low, and 231 (20.3%) TLS- /MLR-high in the retrospective cohort; and 62 (31.8%) TLS+ /MLR-low, 47 (24.1%) TLS+ /MLR-high, 49 (25.1%) TLS- /MLR-low, and 37 (19.0%) TLS- /MLR-high in the prospective cohort. In the retrospective cohort, TLS+ /MLR-low had the best prognosis (median RFS: 25.6 months; median OS: not reached), while TLS- /MLR-high had the poorest (median RFS: 14.5 months; median OS: 41.3 months). Prognoses were comparable between TLS+ /MLR-high and TLS- /MLR-low (median RFS: 19.3 vs. 20.8 months; median OS: 58.0 vs. 61.6 months), with significant differences in RFS and OS across all four subgroups (both $P < 0.001$). In the prospective cohort, TLS+ /MLR-low showed significantly better RFS (median: 43.3 months; $P = 0.043$) and OS (median: not reached; $P = 0.045$) versus other subgroups. Bulk RNA sequencing revealed differentially expressed genes between TLS+ and TLS- cases were primarily associated with the T-cell-inflamed tumor microenvironment and anti-tumor immunity, corroborated by peripheral inflammatory-immune status reflected by MLR. These results indicate the combined score predicts HCC recurrence risk more accurately than either indicator alone.

Conclusions: We developed a novel scoring system refining postoperative recurrence risk stratification, identifying patients more likely to benefit from

adjuvant PD-1 inhibitors, with potential to guide personalized therapy.

Keyword(s): Hepatocellular carcinoma, monocyte/lymphocyte ratio, prognosis, tertiary lymphoid structures

1275 - Development and validation of an explainable machine learning model for predicting the recurrence of adjuvant PD 1 inhibitors in hepatocellular carcinoma patients at high risk of recurrence a multicenter cohort study

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Background / Hypothesis:Identifying the optimal patient subpopulation for benefiting from adjuvant immune checkpoint inhibitors is crucial. This study aims to develop and validate an explainable machine learning model for predicting recurrence in hepatocellular carcinoma (HCC) patients receiving adjuvant PD-1 inhibitors who are at high risk of recurrence after curative resection.

Materials and Methods:This study included retrospective and prospective datasets from 16 medical centers in China, collected from January 2019 to December 2024. Patients who received adjuvant therapy in the prospective cohort were randomly split into the training set and internal validation set, while the retrospective cohort served as the external validation set. Eight machine learning models were employed to predict recurrence in HCC patients. Model performance was evaluated using the area under the receiver operating characteristic curve (AUC), C-index, calibration curve, Brier score, and decision curve analysis (DCA). Additionally, the performance of traditional models was compared to that of time-dependent machine learning models in predicting HCC recurrence. The optimal model was interpreted via SHapley Additive exPlanations (SHAP).

Results:A total of 2,680 patients were included in this study, including 1,649 in the prospective cohort and 1,031 in the retrospective cohort. Adjuvant therapy significantly improved patient prognosis in both cohorts. A total of 910 patients (465 in the prospective cohort and 441 in the retrospective cohort) who received adjuvant PD-1 inhibitor therapy were included in the model analysis. Five key features were selected for model development: tumor size, tumor number, BCLC stage, macrovascular invasion, and microvascular invasion (MVI). Of the eight machine learning (ML) models evaluated, the gradient boosting machine (GBM) model exhibited superior performance. In the external validation cohort, the GBM model achieved 1-, 2-, and 3-year AUC values of 0.831, 0.750, and 0.712, respectively, with corresponding C-indices of 0.800, 0.737, and 0.711—all superior to those of conventional

postoperative recurrence prediction models in predicting recurrence after adjuvant therapy. The GBM model could effectively identify patients with still high recurrence risk after adjuvant therapy; these patients showed no significant difference in prognosis compared with those under active surveillance in both the prospective and retrospective cohorts. Additionally, we developed an online calculator to facilitate easy clinical application.

Conclusions:We constructed a new recurrence risk prediction model for patients with HCC who received adjuvant PD-1 inhibitor therapy after curative resection, which can effectively identify patients who benefit from adjuvant therapy, providing potential guidance to support clinicians in establishing personalized and precision treatment decisions.

Keyword(s) : Adjuvant, curative resection, hepatocellular carcinoma, machine learning, PD-1 inhibitor

1276 - Autoimmune hepatitis triggered by acute Coxiella burnetii infection a case report

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Background:

Q fever, caused by *Coxiella burnetii*, is associated with autoimmune phenomena; however, biopsy-proven autoimmune hepatitis (AIH) developing sequentially after acute Q fever has not been reported to date. We present the first documented case of acute AIH emerging shortly after acute Q fever, suggesting infection-triggered loss of immune tolerance.

Case Presentation:

A 58-year-old livestock farmer presented with nausea, dark urine, jaundice, and acute hepatitis (ALT 552 U/L, AST 352 U/L, direct bilirubin 10 mg/dL). Serologies for hepatitis A–E, EBV, CMV, and other viral causes were negative. *Coxiella burnetii* phase II IgM and IgG were positive; Brucella serology was negative. MRI/MRCP revealed hepatomegaly and periportal edema. Doxycycline was initiated, with initial improvement.

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Following completion of therapy, liver enzymes rose again, raising suspicion for autoimmune hepatitis. Autoimmune workup revealed ANA 1:320 (homogeneous), elevated IgG (25 g/L), with negative ASMA, AMA, and LKM. Liver biopsy demonstrated interface hepatitis, prominent portal mononuclear infiltrates, venulitis, cholestatic pseudorosettes, and rare emperipolesis (Ishak HAI 15; fibrosis stage 2), consistent with acute AIH with cholestatic features.

Treatment and Outcome:

Prednisolone (60 mg/day) followed by azathioprine resulted in rapid biochemical improvement. At 4-month follow-up, ALT decreased to 96 U/L and bilirubin normalized. Clinical course strongly supported infection-triggered AIH.

Discussion:

This case highlights a unique temporal association where acute Q fever appears to precipitate autoimmune hepatitis. Although autoantibody induction during Q fever has been described, progression to biopsy-proven AIH immediately after acute infection has not been previously reported. Proposed mechanisms include molecular mimicry, epitope spreading, IL-10-mediated immune dysregulation, and DAMP-induced loss of self-tolerance.

Conclusion:

Acute Q fever may act as a potent environmental trigger for autoimmune hepatitis, especially when liver tests exhibit a biphasic pattern after antibiotic therapy. Awareness of this rare but clinically significant association is essential for timely diagnosis and management.

Keyword(s) : Autoimmune Hepatitis, Q Fever, Coxiella burnetii, Autoimmunity, Liver Injury

1279 - Multicenter cohort study comparing adjuvant PD 1 inhibitors with active surveillance in hepatocellular carcinoma at high risk of recurrence Benefit in early but not late recurrence

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Background / Hypothesis: Adjuvant PD-1 inhibitors may significantly improve recurrence-free survival (RFS) in hepatocellular carcinoma (HCC) patients at high risk of recurrence after curative resection. This study aims to evaluate the impact of such therapy on early recurrence (defined as recurrence within 12 months after therapy) and late recurrence.

Materials and Methods: This study compared RFS and overall survival (OS) among patients with HCC who received adjuvant PD-1 inhibitor therapy or active surveillance. These patients were prospectively recruited from 14 medical centers in the same province and retrospectively collected from 2 medical centers in other provinces between January 2019 and December 2024. Propensity score matching was used to balance baseline characteristics. Landmark analysis was further performed to evaluate the impact of adjuvant therapy

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on early or late recurrence.

Results: A total of 2,680 patients were included, with 1,649 in the prospective cohort (465 received adjuvant therapy and 1,184 underwent active surveillance) and 1,031 in the retrospective cohort (441 received adjuvant therapy and 590 underwent active surveillance). In the prospective cohort, the median RFS in the adjuvant therapy group was 28.4 months (95% CI 27.4-32.1), significantly longer than the 23.0 months (95% CI 20.3-25.7) in the active surveillance group (HR 0.72; 95% CI 0.69-0.92). Median overall survival was not reached in the adjuvant group, whereas it was 68.3 months (95% CI 54.3-82.3) in the surveillance group, suggesting a significant survival benefit (HR 0.61; 95% CI 0.50-0.75). Landmark analysis further revealed that adjuvant PD-1 inhibitors significantly reduced the risk of early recurrence (HR 0.69; 95% CI 0.58-0.83) but showed limited efficacy against late recurrence (HR 0.97; 95% CI 0.78-1.21). In the retrospective cohort, adjuvant therapy significantly improved RFS (35.6 vs. 21.9 months; HR 0.78; 95% CI 0.66-0.93) and OS (median OS not reached in either group; HR 0.73; 95% CI 0.58-0.91). However, this benefit also appeared to be confined to early recurrence (HR 0.72; 95% CI 0.58-0.90), not late recurrence (HR 0.87; 95% CI 0.67-1.13). Similar results were observed after propensity score matching.

Conclusions: Adjuvant PD-1 inhibitor therapy significantly improves prognosis in HCC patients at high risk of recurrence after curative resection, but its efficacy demonstrates time dependence, with the benefit may be primarily confined to reducing early recurrence.

Keyword(s) : Adjuvant PD-1 inhibitor, curative resection, early recurrence, hepatocellular carcinoma

1282 - Understand Current Treatment Experience Unmet Needs and Attitudes Toward Functional Cure in China Among Patients With Chronic Hepatitis B

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Background/ Hypothesis:

Chronic hepatitis B (CHB) continues to pose a substantial public health burden in China. Although recent years have seen significant advances in screening, diagnosis, and antiviral therapy, aligning with progress toward the WHO 2030 elimination goals. However, limited data are available on patients' perspectives regarding current antiviral treatment experiences and understanding of functional cure. This study aimed to characterize their real-world treatment experiences, perceptions of functional cure, and to explore unmet needs and expectations for novel therapies through a mixed qualitative and quantitative design.

Materials and Methods:

A mixed-methods design was adopted. In the qualitative phase, in-depth interviews were conducted with 10 adult CHB patients to explore their lived experiences, challenges, and motivations regarding current therapies and pursuit of a functional cure. In the quantitative phase, an anonymous online questionnaire using a 6-section questionnaire (covering treatment experience, awareness of functional cure, and new-therapy preference etc.) was distributed to CHB patients residing in mainland China.

Results:

In total, 97 respondents completed the quantitative survey. The mean age was 52.4 years. Educational backgrounds were diverse, with 36.1% holding a bachelor's degree.

Most participants viewed the goal of antiviral therapy as preventing progression to cirrhosis or hepatocellular carcinoma, yet 57.7% had never heard of "functional cure (FC)" and only 10.3% reported clear understanding. Among those who are clear about the definition, patients across all age groups expressed desire for FC, though motivations varied by age: younger patients sought to remove the CHB label and return to a "normal social and family life", while older participants aspired to "live longer and healthier". Although Peg-IFN therapy was perceived as a possible way to FC, its adverse events were described as "unbearable". Patients' perceptions of side effects differed markedly from physicians'; for example, hair loss, considered mild and reversible by clinicians, while was viewed by patients as an outward stigma of illness. With respect to expectations for emerging therapies, patients placed high value on treatment regimens that are shorter, safer, and more predictable in efficacy, while maintaining manageable requirements for clinical monitoring. The majority preferred patient support programs led by healthcare professionals rather than

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peer-led communities.

Conclusions:

This mixed-methods study offers novel insights into the real-world treatment experiences of Chinese CHB patients. Although Awareness and understanding of functional cure remain limited, patients demonstrated strong motivation to achieve HBsAg loss and a strong preference for finite, tolerable regimens under physician guidance. Collectively, these findings underscore the importance of incorporating patient perspectives.

Keyword(s) : Chronic hepatitis B, Chinese CHB patients, Unmet Needs, Functional Cure

1285 - Safety and Outcomes of Allogeneic Hematopoietic Stem Cell Transplantation from HBsAg Positive Donors A Case Control Study from a Tertiary Care Centre.

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Background / Hypothesis:

Hematopoietic stem cell transplantation (HSCT) from HBsAg-positive donors represents a strategy to expand the donor pool particularly in endemic regions. However, this practice may carry risks of HBV transmission and hepatic complications. The available literature is limited, conflicting, and based on low-scale protocols without standardized guidelines for managing such cases. The aim of this study is to evaluate HBV transmission risk, clinical outcomes, and the impact of current prophylactic strategies in recipients of HSCT from HBsAg-positive donors.

Materials and Methods:

We conducted a retrospective matched case-control study. Sixteen HSCT recipients aged >14 years who received grafts from HBsAg-positive donors between 2000 and 2024, were compared with 32 recipient controls matched 2:1 based on age, sex, disease, transplant year, donor type, and HBV status. Detailed donor, recipient, and HBV-related variables were collected, including laboratory data, clinical outcomes, and prophylactic interventions. Hepatic events—including HBV-related hepatitis, hepatic failure, VOD, and hepatic GVHD—were defined using standardized

criteria. Univariate analysis and descriptive analyses were used to identify predictors of HBV transmission and hepatic complications.

Results:

Baseline characteristics were well-matched between the case and control groups. Among the 16 case patients, only two (12.5%) developed post-transplant hepatitis, with both cases being acute. Strikingly, the incidence of hepatitis was lower in the HBsAg-positive donor group (12.5%) compared to the control group (31.2%), though this difference was not statistically significant ($p=0.157$). The absolute risk difference was -18.8% (95% CI -45.1% to +18.1%). Serological analysis revealed Only one (1/8) HBsAg-negative recipients (12.5%, 95% CI) developed de novo persistent HBsAg antigenemia post-transplant. A key finding was the successful implementation of a tailored prophylactic protocol, with the most common regimen being a combination of recipient and donor anti-HBV prophylaxis (50% of patients). An undetectable HBV DNA PCR was achieved in 11/16 pairs (68.8%) post-prophylaxis.

Conclusions:

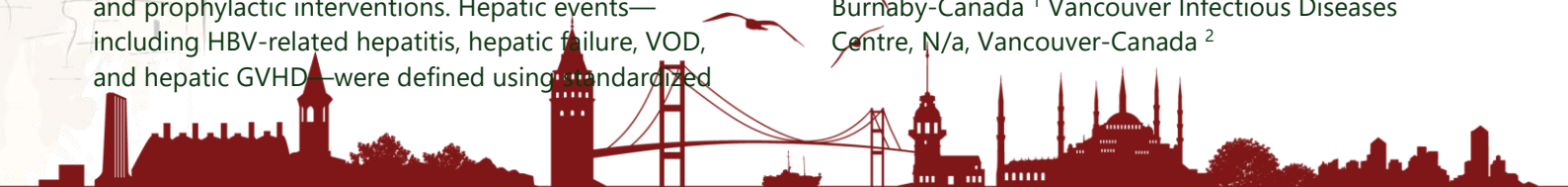
With a structured, multi-modal prophylactic strategy, allogeneic HSCT from HBsAg-positive donors was not associated an apparent increase of HBV transmission, HBV-related hepatitis, or liver-related mortality compared with matched controls. Only one HBsAg-negative recipient (12.5%) developed persistent antigenemia, and two of the sixteen total recipients (12.5%) experienced HBV-related hepatitis. These descriptive results support the cautious use of HBsAg-positive donors in allogeneic HSCT programs which can safely expand the donor pool when managed with vigilant monitoring and comprehensive prophylaxis. However, the small sample size necessitates confirmation in larger studies.

Keyword(s) : Hematopoietic Stem Cell Transplantation, Hepatitis B Virus, HBsAg-Positive Donors, HBV Transmission, Liver Complications

1287 - Using Community Partnerships to Achieve HCV Micro elimination in Vancouver's Inner city

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Background / Hypothesis:

The individuals living in Vancouver's inner city have some of the highest rates of hepatitis C virus (HCV) Canada, and the Vancouver Infectious Diseases Centre (VIDC) is working towards eliminating HCV in this population. The VIDC does this using an approach involving regular community pop-up clinics, or CPCs, at various inner-city locations. At each event, point-of-care HCV antibody testing is offered to up to 30 individuals. Individuals testing positive for HCV antibodies often have their blood drawn on site and those confirmed to be viremic are followed up with by outreach individuals and engaged in treatment under a multi-disciplinary model of care. We aimed to determine whether VIDC is achieving a reduction in the prevalence of HCV viremia in Vancouver's inner-city.

Materials and Methods:

As part of a partnership with Atira housing society, since 2022 CPCs have regularly been held in the common spaces of Atira inner-city buildings with the goal of achieving elimination of HCV infection among the individuals living in these spaces. We determined the proportion of individuals testing positive for HCV antibodies, and those viremic, at CPC events held at 4 different Atira buildings in 2022 through 2024. We then averaged the proportion of individuals testing positive at each location in 2022 and in 2024. We were able to determine if there was a significant reduction in the proportion of individuals residing in these four buildings living with HCV in 2024 relative to 2022.

Results:

We saw a clear reduction in the proportion of viremic individuals tested over time at all four locations. We performed a more detailed analysis of the individuals testing positive at pop-up events at the Luugat and saw that the reduction in viremic individuals over time largely due to the previous treatment of viremic individuals living in that building. Overall, we did not see a reduction in the proportion of individuals with HCV antibodies in these four buildings (32% in 2022 vs. 26% in 2024). However, we saw a clear reduction in the proportion of individuals residing in these residences who were viremic for HCV (14% in 2022 vs. 4% in 2024; $p = 0.1$).

Conclusions:

This study demonstrates that while PWID may be a

particularly difficult demographic to target for HCV treatment, with the help of community partnerships, a multidisciplinary approach to care, and continued testing and follow-up, we are on our way to achieving micro-elimination of HCV among the residents of Vancouver's inner-city.

Keyword(s) : HCV, micro-elimination, PWID, inner-city

1288 - Strategies for reducing the time from HCV diagnosis to treatment initiation Lessons from Vancouver BC Canada

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Background / Hypothesis:

The eradication of HCV infection as a public health concern requires targeted initiatives. In Canada, approximately 35% of people who use drugs are infected with HCV, representing more than 60,000 individuals. The Vancouver Infectious Diseases Centre (VIDC) runs pop-up clinics in Vancouver's inner-city to test for HCV antibodies. Anecdotally, we identify numerous individuals who have documented HCV viremia and who state they have never been offered treatment. There is an urgent need to meaningfully re-engage these individuals in an accessible and timely manner. Reducing the time from HCV antibody diagnosis to treatment initiation is thought to be a key strategy for increasing HCV treatment uptake.

Materials and Methods:

Between August of 2023 and June of 2024, many measures were put in place to help reduce the time from point-of-care testing to initiation of treatment. These included on-site medical records look-up and phlebotomy, immediate special authority (SA) application, assistance attending required medical appointment(s), and partnership with a pharmacy providing daily HCV medication delivery. We measured the effect of these measures on time from initial contact to treatment initiation.

Results:

The median time from initial contact to treatment initiation was reduced from 6 weeks to approximately 3.5 weeks, with a range from 4 days to 57 days. The



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median from initial patient contact to SA application being sent was 2 days (range from 0 to 33 days), and the median number of days for SA approval was 1 day (range from 1 to 22 days). The median number of days from SA approval to prescription being written was 5 days (range from 0 to 40 days), and the median number of days from the prescription being written to the patient receiving their first dose of medication was 7 days (range from 0 to 25 days).

We identified a number of facilitators and barriers to timely treatment initiation. Facilitators included on-site phlebotomy, telehealth assistance for appointments, pharmaceutical delivery, and dispensation with other medications. Barriers included follow-up with individuals without a fixed address, lengthy SA approval times for complex cases, requirement for a fibroscan in some cases, and wait times for HCV medication or pharmaceutical delivery services.

Conclusions:

We have identified many different interventions which have helped to decrease the time between initial contact and treatment start date. We believe that many of these interventions are accessible and can be implemented in similar communities around the world.

Keyword(s) :

1290 - Safety and Tolerability of SIRT and Sorafenib in Advanced Hepatocellular Carcinoma in Mongolia

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is the fifth most common cancer and the third leading cause of cancer-related death worldwide, with over 1 million new cases annually, predominantly in Asia. Most patients are diagnosed at intermediate to advanced stages, limiting curative options such as surgical resection or radiofrequency ablation. Sorafenib, an oral multikinase inhibitor, is the only systemic therapy shown to improve survival, while Selective Internal Radiation Therapy (SIRT) delivers targeted yttrium-90 radiation to tumors. Mongolia has the highest incidence of liver cancer globally, yet data comparing the safety and

tolerability of SIRT and sorafenib in this population are lacking. This study aims to compare adverse events associated with SIRT and sorafenib in patients with locally advanced HCC.

Materials and Methods:

National Cancer Center of Mongolia recruited 39 patients from Mongolia (19 received sorafenib and 20 received SIRT in Singapore) with advanced hepatocellular carcinoma who had not received surgical therapy into a multicenter study involving a total of 360 patients at 27 sites in 11 Asia-Pacific countries. The study was a phase III randomized-controlled clinical trial comparing sorafenib or SIRT.

Results:

Adverse events for patients receiving sorafenib were predominantly grade 1 or 2 in gastrointestinal, constitutional and dermatologic in nature. The most frequently reported drug-related adverse events in patients treated with sorafenib were hypertension, hand-foot skin reaction, diarrhea, alopecia, fatigue. Common procedure-related adverse events were usually mild (grade 1/2) and included nausea and vomiting (27.7% all grades) and abdominal pain (22.1% all grades), with very few grades 3 or greater adverse events.

Conclusions:

In our analysis of 39 patients, we were unable to detect a statistically significant difference in adverse effects between sorafenib and SIRT treatment groups, likely because there were insufficient number patients in our study.

Keyword(s) : Selective Internal Radiation Therapy, Sorafenib, Hepatocellular Carcinoma, Adverse Event, Mongolia

1291 - Comparison between SIRT and Sorafenib in Advanced Hepatocellular Carcinoma in Mongolia

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is the fifth most common cancer and the second leading cause of cancer-related death worldwide, with over 1 million new cases annually. In Mongolia, HCC is the most prevalent cancer, with 2,200–2,400 new cases each year, and nearly 80% of patients present with advanced-stage disease, limiting treatment options and resulting in poor prognosis. Selective Internal Radiation Therapy (SIRT) with yttrium-90 microspheres and the multikinase inhibitor sorafenib are two effective treatments for locally advanced HCC. No randomized trials in the Asia-Pacific region have directly compared their efficacy and safety. This study aimed to evaluate and compare therapeutic outcomes and tolerability of SIRT versus sorafenib in patients with advanced HCC.

Materials and Methods:

At the National Cancer Center of Mongolia, patients were randomly assigned 1:1 to receive either SIRT or sorafenib 800 mg/day orally. Outcomes were assessed through physical examinations, functional assessment, complete blood count, liver function tests, AFP levels, and CT scans every 4, 8, and 12 weeks after treatment initiation.

Results:

Total 39 patients were enrolled: 20 received SIRT and 19 received sorafenib. Median overall survival (OS) and progression-free survival (PFS) did not differ significantly between sorafenib and SIRT arms (OS: 15.56 vs. 9.17 months; HR 0.95, 95% CI 0.46, $p = 0.889$; PFS: 8.51 vs. 5.85 months; HR 1.07, 95% CI 0.53–2.16, $p = 0.842$). Tumor response was higher with SIRT than sorafenib (68.8% vs. 62.5%, $p = 0.033$), including complete response in 2 (10%) patients, partial response in 2 (10%), and stable disease in 7 (43.8%). A total of 165 treatment-emergent adverse events were reported (SIRT 66 vs. sorafenib 99), with fewer grade ≥ 3 events in the SIRT group (83 vs. 115, $p = 0.0964$).

Conclusions:

In locally advanced HCC, overall survival was similar between SIRT and sorafenib. However, SIRT achieved higher tumor response rates and a lower incidence of severe adverse events compared with sorafenib.

Keyword(s) : Hepatocellular carcinoma, SIRT, Sorafenib,

RECIST, Alanine aminotransferase, Aspartate aminotransferase

1292 - Comparative Study of Tenofovir Alafenamide and Tenofovir Disoproxil Fumarate in Chronic Hepatitis B in Mongolia

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Background / Hypothesis:

Chronic hepatitis B (CHB) affects approximately 240 million people worldwide, with Mongolia reporting an 11.1% prevalence among adults. CHB patients can present in different clinical states, including HBeAg-positive or -negative infection, and monitoring relies on HBV-DNA levels, HBeAg status, and liver enzymes such as ALT. Nucleos(t)ide analogs, including tenofovir disoproxil fumarate (TDF) and tenofovir alafenamide (TAF), are standard treatments that effectively suppress viral replication and reduce progression to cirrhosis and hepatocellular carcinoma. Long-term therapy, however, raises concerns about drug resistance, renal impairment, and bone mineral density loss. While TAF has recently been approved in Mongolia, comparative data on its efficacy and safety versus TDF in both HBeAg-positive and -negative CHB patients remain limited. This study aimed to evaluate and compare the short-term efficacy, safety, and tolerability of TAF and TDF in treatment-naïve and treatment-experienced Mongolian adults with CHB.

Materials and Methods:

We performed a randomized, unblinded, non-inferiority study in which patients with compensated cirrhosis (Child-Pugh A and B stage) between 18–70 years old with a positive chronic hepatitis B test were randomized to receive either TAF, TDF, or were switched from TDF to TAF. The primary efficacy endpoint was the proportion of patients with HBV-DNA < 29 IU/ml at week 48.

Results:

The efficacy endpoint, an HBV-DNA < 29 IU/ml at weeks 48, was achieved by 251 (79.9 %) of 314 patients

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receiving TAF, which was not significantly different from the 113 (74.8 %) of 151 patients receiving TDF. After 48 weeks of treatment, patients receiving TAF had significantly smaller bone mineral density reductions than patients receiving TDF. At week 48, the median decrease in eGFR was significantly less in the TAF recipients than the TDF recipients.

Conclusions:

TAF treatment has the same efficacy as TDF treatment. However, TAF treatment had a better safety profile than TDF. Patients receiving TAF had a significantly smaller median decrease in the eGFR by the Cockcroft-Gault equation than patients receiving TDF.

Keyword(s) : Hepatitis B, Tenofovir Alafenamide Fumarate, Tenofovir Disoproxil Fumarate, Adverse Effects, Safety

1293 - Evaluating ALBI Score for Risk Stratification in Acute Hepatitis B and D in Mongolia

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Background / Hypothesis:

Hepatitis D virus (HDV) is a defective virus that requires hepatitis B surface antigen (HBsAg) for replication. Acute HDV infection may occur as co-infection with HBV or as superinfection in chronic HBV carriers, often resulting in more severe disease and higher rates of acute liver failure (ALF) compared to HBV alone. Mongolia has one of the highest rates of HBV and HDV infections globally, although national vaccination programs and viral hepatitis control initiatives have reduced incidence over recent decades. Despite this, acute hepatitis D remains a major cause of ALF, particularly among adults aged 20–44. Prognostic models such as MELD, CTP, and ALBI scores are used to predict liver disease outcomes; however, the utility of the ALBI score in acute hepatitis B with or without HDV has not been evaluated in Mongolia. This study aimed to assess the prognostic value of the ALBI score for clinical outcomes in patients with acute hepatitis B infection with or without HDV.

Materials and Methods:

A total of 114 patients—58 with acute hepatitis B (AHB), 5 with HBV/HDV co-infection, and 51 with HBV/HDV superinfection—admitted to the National Center for Communicable Diseases between 2022 and 2024 were enrolled. Clinical and laboratory data were analyzed to compare outcomes and prognostic markers, including the ALBI score, among patients with and without ALF.

Results:

Compared to the AHB group, patients with HBV/HDV superinfection were slightly older (mean age 28.9 ± 7.4 vs. 25.8 ± 6.5 years, $p = 0.019$) and fewer lived in Ulaanbaatar (62.7% vs. 87.5%, $p = 0.037$). Hospitalization was longer for the HBV/HDV superinfection group (28 ± 13 vs. 23 ± 11 days, $p = 0.022$). Patients with ALF had higher ALBI scores, total bilirubin, transaminases, and INR, along with lower platelet counts, compared to those without ALF. The ALBI score was significantly higher in AHB with ALF than AHB without ALF ($p = 0.001$) and in HBV/HDV superinfection with ALF compared to without ALF ($p = 0.041$). The area under the curve (AUC) for the ALBI score predicting ALF was 0.766, with a cut-off value of -1.71, sensitivity 72.2%, and specificity 75.6%.

Conclusions:

The ALBI score at admission is a useful predictor of survival in patients with acute hepatitis B and acute hepatitis D, helping identify those at higher risk of developing acute liver failure.

Keyword(s) : Acute Hepatitis B, Acute Hepatitis Delta, Acute Liver Failure, ALBI Score

1294 - Clinical Features and Outcomes of Acute Hepatitis B and Delta in Mongolia A One Year Prospective Follow Up Study

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Background:

Mongolia has one of the highest prevalence rates of hepatitis B virus (HBV) and hepatitis D virus (HDV) infections worldwide. Despite the national HBV vaccination program, acute hepatitis D remains a major



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contributor to chronic liver disease. This study aimed to assess the clinical and laboratory characteristics and the progression to chronic infection in adult patients with acute hepatitis B and delta infections in Mongolia.

Materials and Methods:

This prospective cohort study enrolled 182 patients at the National Center for Communicable Diseases in Ulaanbaatar, Mongolia, between 2022 and 2024. All participants provided written informed consent. Patients were categorized into three groups: acute hepatitis B (AHB, n=105), HBV/HDV co-infection (n=5), and HBV/HDV super-infection on chronic hepatitis B (n=72). Clinical symptoms, liver function tests (ALT, AST), and HBsAg status were monitored over a 12-month follow-up period.

Results:

Of the 182 participants, 108 (59.3%) were male. The mean age was 25.2 ± 6.1 years in the AHB group, 28.4 ± 2.2 years in the HBV/HDV co-infection group, and 30 ± 7.6 years in the HBV/HDV super-infection group ($p=0.0001$). Patients with AHB and HBV/HDV co-infection fully recovered clinically, and liver function tests normalized. In the HBV/HDV super-infection group, 95.8% of patients showed clinical recovery, but ALT and AST levels remained significantly elevated compared to other groups (ALT: 85.5 ± 125.8 vs. 18.2 ± 36 vs. 13.8 ± 6.5 ; AST: 63.4 ± 67.1 vs. 18.9 ± 19.2 vs. 19.7 ± 9.6 ; $p=0.0001$). The rate of progression to chronic infection was significantly higher in the HBV/HDV super-infection group. At 12 months post-onset, 6.7% of AHB patients remained HBsAg positive, compared to 40% of HBV/HDV co-infection patients and 94.4% of HBV/HDV super-infection patients ($p=0.0001$).

Conclusions:

Chronic infection developed in 6.7% of AHB patients, 40% of HBV/HDV co-infected patients, and 93.3% of HBV/HDV super-infected patients. Clinical symptoms and liver function normalized in AHB and HBV/HDV co-infection patients. However, in HBV/HDV super-infection patients, liver enzyme levels remained approximately twice the normal range, indicating more severe and prolonged hepatic injury.

Keyword(s) : Acute Hepatitis B, Acute Hepatitis Delta, HBV/HDV Co-infection, HBV/HDV Super-infection, Liver Function, Chronic Hepatitis, Mongolia

1295 - HCC associated with hepatitis B and C Mongoloid and Caucasians of North East Asia

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) accounts for more than half of all cases worldwide in Asian countries, yet data from northeastern Asia, where Mongoloid and Caucasian populations live in adjacent territories, remain limited. This study aimed to compare the clinical and epidemiological characteristics of HCC and to evaluate the diagnostic value of serum cancer markers in Mongoloid and Caucasian ethnic groups.

Materials and Methods:

The studies were carried out in the adjacent territories of Mongolia and the Asian part of Russia in the area of Lake Baikal (Irkutsk region). A total of 300 patients with hepatocellular carcinoma were examined, including 195 Mongols from the city of Ulan Bator and 105 Russians permanently residing in the Irkutsk region.

Results:

In Mongolia 93% of hepatocellular carcinoma cases are associated with viral hepatitis, with hepatitis B accounting for 34.5% of cases, which significantly distinguishes Mongolia from the Asian part of Russia. Excessive alcohol consumption is a significant non-infectious risk factor in Mongolia.

Conclusions:

In connection with the insufficient sensitivity of the determination of alpha-fetoprotein in the ethnic group of Mongoloids, it is necessary to search for new molecular markers. An alternative possibility of using osteopontin and glypican-3 as serum predictors of hepatocellular carcinoma, the diagnostic sensitivity of which is higher than alpha-fetoprotein and does not differ in the ethnic groups of Mongoloids and Caucasians, has been shown.

Keyword(s) : Hepatocellular carcinoma, Mongoloid, Caucasian, Serum markers, Alpha-fetoprotein, Osteopontin, Glypican-3, Viral hepatitis



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1296 - Efficacy and Safety of Tenofovir Alafenamide in the Management of Chronic Hepatitis B in Mongolia

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Background / Hypothesis:

Chronic hepatitis B virus (HBV) infection affects an estimated 250 million people globally, with 1.5 million new infections each year. Mongolia has one of the highest HBV prevalence rates worldwide, with 10.6–11.6% of the population infected. Effective antiviral therapy is essential to reduce disease progression and improve outcomes. This study aimed to evaluate the clinical and virological outcomes of tenofovir alafenamide (TAF) treatment in patients with chronic hepatitis B in Mongolia.

Materials and Methods:

The clinical trials have evaluated TAF in HBeAg positive and HBeAg negative HBV patients. The trials have similar design and randomized, Single-blind, the subjects are unaware of which group they have been assigned to studies. The primary efficacy endpoint was the proportion of patients with HBV-DNA <29IU/ml at weeks 96. Other virological result endpoints were the proportion of patients with HBsAg seroconversion at weeks 96.

Results:

At week 96, the virologic endpoint, defined as HBV-DNA <29 IU/mL, was achieved in 243 patients (79.1%) receiving TAF, compared to 106 patients (78.5%) receiving TDF, demonstrating non-inferiority (95% CI: -9.7 to 2.5; p = 0.26). ALT normalization rates were significantly higher in the TAF group than in the TDF group at week 96 (209 [68%] vs. 82 [60.8%]; p = 0.001). Patients receiving TAF also experienced smaller reductions in bone mineral density (BMD) compared to TDF recipients. Additionally, median changes in eGFR at week 96 were significantly smaller in the TAF group, indicating better renal safety.

Conclusions:

TAF and switching from TDF to TAF are similar efficacy and safety in long-term treatment of TDF.

Keyword(s) : HBV-DNA, qHBsAg, HBeAg, tenofovir alafenamid fumarate, tenofovir disoproxil fumarate

1297 - Clinical Characteristics of HBV and HCV Related Hepatocellular Carcinoma A Comparative Study in Mongolia

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Background:

Hepatocellular carcinoma (HCC) is the fifth most common cancer and the third leading cause of cancer-related deaths worldwide. Chronic hepatitis B (HBV) and hepatitis C (HCV) infections are the major risk factors, responsible for about 54% and 31% of cases. Mongolia has the highest HCC incidence globally, reflecting its high HBV (10.6%) and HCV (9.9%) infection rates. HBV-related HCC is often driven by viral oncogenic mechanisms, whereas HCV-related HCC is strongly associated with chronic inflammation and cirrhosis. Clinical presentation and disease outcomes may differ between HBV- and HCV-related HCC, yet Mongolian data remain limited. This study aimed to compare the clinical and laboratory characteristics of Mongolian patients with HBV- versus HCV-related HCC.

Materials and Methods:

By a single century, we analyzed the clinical characteristics of 23 patients with hepatitis B virus-related hepatocellular carcinoma (HBV group) and 34 with hepatitis C virus-related hepatocellular carcinoma (HCV group).

Results:

Patients with HBV-related HCC were younger (mean 61.1 years) than those with HCV-related HCC (67.1 years). Liver function was better in the HBV group, with lower indocyanine green retention (17.5% vs. 25.4%). More HBV patients presented with stage I disease (55% vs. 44%), but they also had more advanced tumor features, including higher rates of T3–4 tumors (53% vs. 41%), portal vein tumor thrombus grades 2–3 (20.3% vs. 7.1%), and poorly differentiated tumors (7%

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vs. 5%).

Univariate analysis identified HBV infection, younger age (≤ 50 years), higher AFP, more numerous and larger tumors, and portal or hepatic vein tumor thrombus grades 2–3 as poor prognostic factors. However, multivariate analysis showed that only high AFP, tumor number, and portal or hepatic vein thrombus grades 2–3 remained independent predictors of poor prognosis. Logistic regression showed elevated ASAT was significantly associated with hepatitis infection (OR 2.01, 95% CI 1.41–6.91, $p < 0.05$).

Conclusion:

These results suggest that HBV itself is not a stronger prognostic factor than HCV.

Keyword(s) : Hepatitis, Carcinoma, Virus, Neoplasms, Antigens, Blood-Borne Infections

1298 - Long Term Outcomes of Ledipasvir Sofosbuvir Treatment in Hepatitis C Viral Suppression Hepatocellular Carcinoma and Mortality in Mongolia

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Background:

Hepatitis C virus (HCV) infection poses a significant health burden, particularly in Mongolia, where the HCV prevalence is notably high. This study evaluates the long-term outcomes of HCV treatment with ledipasvir/sofosbuvir, focusing on mortality, viral relapse, and hepatocellular carcinoma (HCC) development.

Materials and Methods:

This prospective, longitudinal cohort study initially enrolled patients with chronic HCV in Mongolia between 2020 and 2024, focusing on those who completed the five-year follow-up ($n = 303$). The study measured long-term mortality, HCC development, and viral relapse, employing non-invasive methods to

assess liver fibrosis and liver function.

Results:

At the outset, 98.2% of the patients achieved undetectable HCV RNA levels. Over five years, 6.27% experienced viral relapse and 3.30% developed hepatocellular carcinoma (HCC), with a mortality rate of 5.94%. In a multivariable analysis, the significant predictors for HCC occurrence included age (OR = 1.081, 95% CI = 1.021–1.145), liver cirrhosis (OR = 5.866, 95% CI = 1.672–22.577), and GGT level (OR = 1.011, 95% CI = 1.004–1.018). The independent predictors of mortality included age (OR = 1.083, 95% CI = 1.024–1.147), liver cirrhosis (OR = 6.529, 95% CI = 1.913–22.281), and GGT (OR = 1.011, 95% CI = 1.004–1.017).

Conclusions:

This study demonstrates that ledipasvir/sofosbuvir effectively suppresses HCV initially and maintains low viral relapse rates over the long term. However, it emphasizes the need for continued management to reduce the long-term risk of HCC and mortality, especially in patients with severe liver fibrosis or cirrhosis.

Keyword(s) : viral suppression; liver cirrhosis; chronic liver disease; antiviral therapy

1299 - Hepatitis C virus infection in Mongolia Updated provincial data on prevalence genotype distribution and age specific risk factors

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Background:

Hepatitis C virus (HCV) infection remains a significant public health issue in Mongolia, where both medical and traditional practices contribute to transmission. This study aimed to assess the prevalence, genotype distribution, and risk factors of HCV infection among residents of Arkhangai Province.

Materials and Methods:

A cross-sectional population-based study included 2,304 individuals aged 0–80 years. Serum samples were

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tested for anti-HCV antibodies using ELISA and for HCV RNA using PCR. Positive samples were genotyped, and demographic and exposure data were analyzed using logistic regression to identify independent risk factors.

Results:

The prevalence of anti-HCV antibodies was 12.0%, and HCV RNA positivity was 7.16%. Infection increased significantly with age ($p < 0.001$) and was higher among females (14.6%) than males (8.4%). Genotype 1b predominated (98.2%), followed by 1a (1.2%) and 2 (0.6%). Independent risk factors included cosmetic procedures (OR 2.30, 95% CI 1.51–3.26), cupping therapy (OR 1.68, 95% CI 1.32–1.98), unsafe injections (OR 3.12, 95% CI 2.40–3.81), and shared razor use (OR 1.50, 95% CI 1.08–2.13).

Conclusions:

HCV infection remains prevalent, particularly among older adults and women. Genotype 1b continues to predominate, indicating that the viral genetic distribution has remained largely unchanged over the past decade. Continued education, safe injection practices, and regulation of traditional and cosmetic procedures are essential to reduce HCV transmission and support Mongolia's elimination goals.

Keyword(s) : hepatitis C virus; prevalence; risk factors; Mongolia; genotype 1b; cupping therapy; unsafe injection

1300 - Epidemiology Clinical Features and Laboratory Findings of Acute Hepatitis B and D Virus Infection Among Children Under Five Years of Age in Mongolia

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Background / Hypothesis:

Globally, an estimated 15–20 million people are infected with hepatitis D virus (HDV). Anti-HDV-IgM is found in 2–7% of acute hepatitis cases and 9–50% of chronic hepatitis cases. Among infants born to HBeAg-positive mothers with chronic HBV infection, 90% acquire HBV, and 25–30% progress to chronic infection by age five. Mongolia introduced universal newborn

HBV vaccination in 1991, reducing HBV prevalence from 5.2–9.8% among children born in 1990–2000 to below 1% in those born in 2004–2007.

Materials and Methods:

A retrospective review was conducted using medical records of children aged 0–5 years diagnosed with acute HBV or HDV infection and hospitalized at the National Center for Communicable Diseases from 2020 to 2024. Statistical analysis was performed using SPSS version 26.

Results:

A total of 22 children aged 0–5 years were diagnosed with acute hepatitis B or D; 91% (20) recovered and 9% (2) died. Of these, 59% (13) were under 6 months, 13.6% (3) were 6–12 months, 13.6% (3) were 1–2 years, and 13.6% (3) were 3–4 years old; 59.1% (13) were male and 40.9% (9) female. Hepatitis B vaccination within 24 hours of birth was recorded in 91% (20), while 4.5% (1) were unvaccinated and 4.5% (1) had unknown vaccination status. Maternal chronic HBV infection was present in 86.36% (19), and maternal HBV status was unknown in 13.63% (3).

Clinically, 77.3% (17) had moderate disease, 18.2% (4) severe, and 4.5% (1) were critically ill. The mean hospital stay was 17 ± 10.3 days, while the two fatal cases averaged 4 ± 3.24 days. Common symptoms included jaundice (95.4%), poor appetite (50%), vomiting (50%), fever (31.8%), poor breastfeeding (31.8%), sleep disturbance (18.18%), abdominal pain (4.5%), and irritability (77.3%).

Serology showed acute HBV in 54.5% (12), HBV/HDV co-infection in 41% (9), and one case (4.5%) of HBV/HDV superinfection with acute hepatitis A. ALT and AST were markedly elevated at admission (1055.77 ± 794.46 IU/L and 1137.31 ± 557.87 IU/L) and decreased to 96.04 ± 54.69 IU/L and 60.43 ± 35.28 IU/L at discharge. Total bilirubin dropped from 90.79 ± 25.55 to 25.55 ± 22.37 $\mu\text{mol/L}$, and alkaline phosphatase from 670.34 ± 272.97 to 533.19 ± 169.66 . All decreases were statistically significant ($p < 0.01$). INR improved from 3.79 ± 3.04 to 1.17 ± 0.03 ($p < 0.01$).

Conclusion:

Among the study population, 59% were under 6 months of age, and 86.36% had mothers with chronic HBV infection during pregnancy. Comparison of

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laboratory results showed that cholestatic features

Keyword(s) : Children hepatitis, HBV, HDV, Acute hepatitis, Mongolia

1301 - Clinical and Metabolic Features of MAFLD Among Individuals With Chronic HCV

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Background:

Metabolic dysfunction-associated fatty liver disease (MAFLD) has become a leading cause of hepatic steatosis, particularly in individuals with obesity or insulin resistance. Hepatitis C virus (HCV) infection also promotes liver inflammation and fibrosis, and its coexistence with MAFLD may further increase the risk of steatosis and fibrosis. International studies suggest a synergistic effect between the two conditions. This study aimed to determine the prevalence of MAFLD in patients with chronic HCV infection, evaluate metabolic risk factors, and examine the interaction between viral infection and metabolic dysfunction.

Materials and Methods:

This study included 284 chronic HCV patients treated at the National Center for Communicable Diseases from 2020–2025. Demographic, laboratory, and ultrasound data were collected. Based on MAFLD criteria, 41.9% (117) were MAFLD-positive and 58.1% (167) MAFLD-negative. Statistical analysis was done using SPSS 28, with $p < 0.05$ considered significant.

Results:

The mean age of participants was 46.8 ± 10.5 years, and 58.1% ($n=164$) were male. In the MAFLD group, obesity was present in 76.9% ($n=90$), diabetes mellitus in 32.5% ($n=38$), and hypertension in 69.4% ($n=81$), all significantly higher than in the MAFLD-negative group ($p=0.001$), indicating substantial metabolic abnormalities.

Laboratory results showed higher mean ALT and AST levels in the MAFLD group (55 ± 22 U/L and 48 ± 18 U/L) compared to the non-MAFLD group (35 ± 15 U/L and 30 ± 12 U/L). Additionally, triglycerides (200 ± 70 mg/dL), HDL (40 ± 8 mg/dL), glucose (115 ± 25 mg/dL), and HOMA-IR (3.6 ± 1.2) were markedly worse in the MAFLD group, whereas the control group had

values of 145 ± 45 mg/dL, 50 ± 12 mg/dL, 98 ± 20 mg/dL, and 2.2 ± 0.9 , respectively.

CAP (FibroScan) values were higher in the MAFLD group (288 ± 40 dB/m) than in the non-MAFLD group (250 ± 35 dB/m), indicating mostly moderate-severe steatosis. FIB-4 scores were also elevated in MAFLD patients (2.0 ± 0.7 vs. 1.2 ± 0.5 , $p=0.03$), suggesting more advanced liver injury.

CAP showed moderate correlations with BMI ($r=0.52$), HOMA-IR ($r=0.41$), and triglycerides ($r=0.38$) (all $p=0.001$), while its correlation with HCV viral load was weak ($r=0.12$, $p=0.01$), reaffirming metabolic dysfunction as the main driver of steatosis.

Conclusions:

MAFLD is frequent in chronic HCV patients and is mainly driven by metabolic factors like obesity, insulin resistance, and high triglycerides. Although HCV can aggravate steatosis and fibrosis, its weak link with viral load shows it is not the primary cause. Metabolic dysfunction remains the key determinant.

Keyword(s) : Metabolic dysfunction, hepatitis C, risk factors, viral load, steatosis

1302 - ASSOCIATION BETWEEN METABOLIC RISK FACTORS HEPATIC STEATOSIS AND QUALITY OF LIFE AMONG PATIENTS WITH CHRONIC HCV INFECTION ASSESSED USING THE HCV SIQv4 QUESTIONNAIRE

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Background / Hypothesis:

Globally, the prevalence of metabolic dysfunction-associated fatty liver disease (MAFLD) is rising, making it one of the leading causes of liver disease. International studies suggest that chronic hepatitis C virus (HCV) infection may contribute to metabolic disturbances, hepatic steatosis, and fibrosis. However, no study in Mongolia has comprehensively evaluated the relationship between hepatic steatosis, metabolic indicators, and quality of life in patients with chronic HCV infection. Therefore, this study aimed to assess the associations between these factors.

Materials and Methods:

A total of 284 patients with a confirmed diagnosis of

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chronic HCV infection who received care at the National Center for Communicable Diseases from 2020 to 2025 were included. Demographic data, laboratory results, and FibroScan measurements including CAP for steatosis and FIB-4 scores for fibrosis were analyzed. Using MAFLD diagnostic criteria, participants were categorized as MAFLD-positive 41.9% (n = 117) and MAFLD-negative 58.1% (n = 167). Quality of life was assessed using the HCV-Symptom and Impact Questionnaire version 4 (HCV-SIQv4). Reliability of the tool showed high internal consistency (Cronbach's $\alpha=0.942$). Statistical analyses were performed in SPSS 28, with significance set at $p < 0.05$.

Results:

The mean age of participants was 46.8 ± 10.5 years, and 58.1% (n = 164) were male. A majority reported MAFLD-related symptoms, including weight gain (3.8 ± 1.1 , $p = 0.001$), abdominal discomfort (3.1 ± 1.0 , $p = 0.001$), fatigue (4.3 ± 1.2 , $p = 0.001$), and changes in appetite (2.9 ± 0.9 , $p = 0.001$).

The total HCV-SIQv4 score was significantly higher in the MAFLD-positive group (18.5 ± 5.2 , $p = 0.001$) compared to the MAFLD-negative group (12.3 ± 4.7 , $p = 0.001$), indicating greater impairment in quality of life, likely associated with steatosis and metabolic dysfunction.

The total questionnaire score showed moderate positive correlations with CAP ($r = 0.45$, $p = 0.012$) and HOMA-IR ($r = 0.42$, $p = 0.028$), suggesting that lifestyle- and symptom-related impacts were associated with hepatic steatosis and metabolic abnormalities. A weak correlation was found with HCV viral load ($r = 0.10$, $p = 0.04$), indicating that viral replication itself does not strongly influence MAFLD symptom severity.

Conclusions:

Among chronic HCV patients, MAFLD is strongly associated with impaired quality of life, hepatic steatosis, and metabolic dysfunction. These findings highlight the importance of improving MAFLD screening and monitoring, as well as incorporating quality-of-life assessment into the management of patients with chronic HCV infection to prevent complications and improve patient outcomes.

Keyword(s) : MAFLD, hepatitis C, risk factors, viral load, steatosis

1303 - Impact of Sleep Disturbances and Psychological Distress on Quality of Life in Mongolian Patients with Acute and Chronic

Hepatitis C

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Background:

Hepatitis C (HCV) remains a major global public health challenge, with millions affected worldwide. HCV infection can lead to severe liver damage, including cirrhosis and liver cancer, and significantly reduce quality of life. Sleep disturbances, anxiety, and depression are common in HCV patients and may worsen disease outcomes. This study investigates the impact of sleep disturbances and mental health on quality of life and clinical outcomes in Mongolian patients with HCV.

Materials and Methods:

This cross-sectional study, conducted from October 2024 to March 2025 at the Mongolian National Center for Communicable Diseases, included 187 participants with HCV, whom 82 had acute and 105 had chronic, selected through non-probability purposive sampling. Psychological distress and sleep disturbances were assessed using the General Health Questionnaire-28 (GHQ-28) and the Pittsburgh Sleep Quality Index (PSQI). Data analysis, including regression models, was performed using SPSS version 28, with a p-value <0.05 considered statistically significant.

Results:

Among the 187 participants, the majority were male (58.8%, n=110), 42.8% (n=80) were aged 46–55 years, and slightly over half reported limited income (51.9%, n=97). Lifestyle factors showed that 40.6% (n=76) were smokers and 80.7% (n=151) were overweight. Comparing acute and chronic HCV, chronic patients were more likely to be overweight (85.7% vs 74.4%, $p=0.04$) and have cirrhosis (18.1% vs 3.7%, $p<0.001$). Anxiety and sleep disturbances were higher in chronic patients, with mean GHQ-28 scores of 17.8 ± 4.5 versus 14.2 ± 3.9 and PSQI scores of 8.3 ± 2.6 versus 6.5 ± 2.1 in acute patients (both $p<0.01$). Correlations between anxiety/insomnia and physical health were strong in both groups (acute $r=0.63$; chronic $r=0.72$), while overall physical domains correlated moderately (acute $r=0.48$; chronic $r=0.54$). Regression analysis showed that sleep disturbances and psychological distress significantly predicted poorer quality of life in both

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acute and chronic patients, with low income (OR=2.90, 95% CI: 2.37–3.54, $p=0.001$, OR=1.75, 95% CI: 1.45–2.11, $p=0.001$).

Conclusions:

Chronic HCV patients experience greater anxiety, sleep disturbances, and worse quality of life than acute patients. Sleep disturbances and psychological distress significantly predict poorer outcomes, with low income further worsening quality of life. Early identification and integrated management of mental health and sleep issues are essential to improve patient outcomes.

Keyword(s) : sleep disturbances, insomnia, HCV, PSQI, GHQ-28

1304 - MiR 551a drives cholesterol metabolic reprogramming and sorafenib resistance in hepatocellular carcinoma

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Background / Hypothesis:

Cholesterol metabolic reprogramming has emerged as a critical hallmark of therapy resistance in advanced hepatocellular carcinoma (HCC). However, how specific non-coding RNAs orchestrate cholesterol dysregulation and contribute to sorafenib resistance remains unclear.

Materials and Methods:

We performed in vivo CRISPR/Cas9 knockout screen in sorafenib-resistant HCC models. Hits were validated through sgRNA sequencing, proteomics, CUT&Tag-seq, luciferase reporter assays, and ER-cholesterol imaging, together with functional studies in vitro and orthotopic xenograft models. To evaluate clinical significance, miRNA-specific probes and multiplex protein fluorescence staining were applied to a tissue microarray (TMA) of paired human HCC specimens, enabling quantitative assessment of miR-551a expression, its downstream molecular alterations, and association with clinical outcome

Results:

miR-551a was identified as a leading driver of sorafenib resistance. Its overexpression enhanced tumour growth and survival under sorafenib exposure,

while inhibition of miR-551a restored drug sensitivity. Mechanistically, miR-551a induced a pronounced disruption of cholesterol homeostasis, resulting in intracellular cholesterol accumulation—a metabolic state tightly linked to drug resistance. Importantly, pharmacological targeting of cholesterol metabolism effectively resensitized resistant tumours, confirming the functional relevance of this metabolic rewiring. Clinical TMA analysis further demonstrated that patients with high miR-551a levels exhibited increased cholesterol-dysregulation signatures and significantly poorer outcomes.

Conclusions:

Our study identifies miR-551a as a novel non-coding RNA regulator that reprograms cholesterol metabolism to drive sorafenib resistance in HCC. Integrating mechanistic evidence with TMA-based clinical validation, we highlight the therapeutic potential of targeting cholesterol dysregulation through the miR-551a pathway as a promising metabolic strategy to improve treatment response.

Keyword(s) : Hepatocellular carcinoma, Sorafenib resistance, Cholesterol metabolism, MiR-551a

1306 - MAFLD in Chronic HBV The Dominant Role of Metabolic Dysfunction Over Viral Load

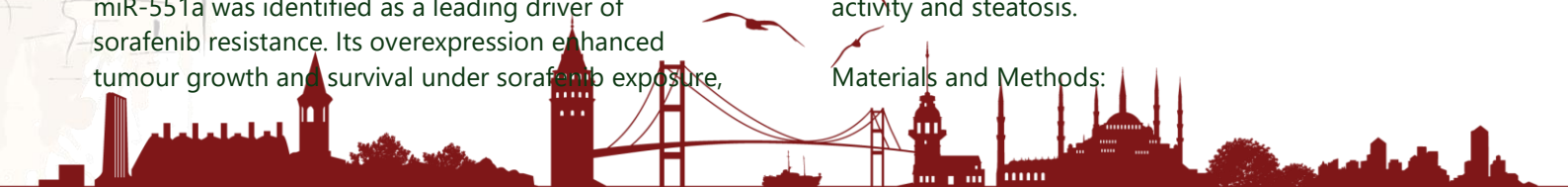
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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) has emerged as a leading cause of hepatic steatosis, particularly among individuals with obesity, insulin resistance, or dyslipidemia. Chronic hepatitis B virus (HBV) infection can cause liver inflammation and fibrosis, but unlike HCV, HBV is not a primary driver of metabolic liver injury. The coexistence of MAFLD with chronic HBV may exacerbate liver disease, but metabolic factors are likely the dominant contributors to steatosis in this population. This study aimed to assess the prevalence of MAFLD among patients with chronic HBV infection, evaluate associated metabolic risk factors, and explore the relationship between viral activity and steatosis.

Materials and Methods:



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This retrospective study analyzed 127 patients with chronic HBV infection who received care at the National Center for Communicable Diseases between 2020 and 2025. Clinical, laboratory, and ultrasound data were collected for all participants. Using established MAFLD criteria, 42% (n=53) were classified as MAFLD-positive, while 58% (n=74) were MAFLD-negative. Statistical analyses were conducted with SPSS version 28, and p-values below 0.05 were considered statistically significant.

Results:

The mean age of participants was 47.1 ± 11.2 years, with 58.1% (n=164) male. Among MAFLD-positive patients, obesity was observed in 75.2% (n=88), diabetes in 31.6% (n=37), and hypertension in 70.1% (n=82), all significantly higher than in the MAFLD-negative group ($p=0.001$). Laboratory findings showed higher ALT and AST levels in the MAFLD group (56 ± 24 U/L and 50 ± 19 U/L) compared to controls (36 ± 16 U/L and 31 ± 13 U/L). Triglycerides (202 ± 72 mg/dL), HDL (41 ± 9 mg/dL), glucose (116 ± 26 mg/dL), and HOMA-IR (3.7 ± 1.3) were also worse in MAFLD patients versus non-MAFLD (145 ± 47 mg/dL, 51 ± 12 mg/dL, 99 ± 21 mg/dL, 2.3 ± 1.0 , respectively). FibroScan CAP values were higher in the MAFLD group (288 ± 41 dB/m vs. 251 ± 36 dB/m), indicating predominantly moderate-severe steatosis. FIB-4 scores were also elevated (2.0 ± 0.7 vs. 1.2 ± 0.5 , $p=0.03$). CAP values showed moderate correlations with BMI ($r=0.51$), HOMA-IR ($r=0.42$), and triglycerides ($r=0.39$) (all $p=0.001$), but a weak correlation with HBV viral load ($r=0.10$, $p=0.02$), confirming metabolic dysfunction as the main driver of steatosis.

Conclusions:

MAFLD is common in chronic HBV patients and mainly driven by metabolic factors like obesity, insulin resistance, and dyslipidemia. HBV's impact on steatosis is minimal, making metabolic management key to preventing liver injury.

Keyword(s) : MAFLD, hepatitis B, metabolic risk factors, steatosis, liver fibrosis

1307 - Assessment of Clinical Status and Liver Function as Predictors of Lenvatinib Efficacy in Hepatocellular Carcinoma A Single Center Study at Dr. Mohammad Hoesin Hospital Palembang (January–December 2025)

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a significant global health challenge with persistently high mortality, especially in countries where limited surveillance and late presentation reduce the opportunities for curative treatment. Lenvatinib is an approved first-line systemic therapy for unresectable HCC and has demonstrated promising progression-free survival across international trials. However, treatment response varies among individuals, largely influenced by liver functional reserve and patient performance status. Identifying reliable clinical and biochemical predictors is therefore essential to optimize patient selection and therapeutic outcomes. This study hypothesizes that clinical performance (ECOG), liver function scores (Child-Pugh and MELD), and baseline laboratory parameters may predict therapeutic response to lenvatinib in HCC patients treated during the 2025 period at Dr. Mohammad Hoesin Hospital (RSMH) Palembang.

Materials and Methods:

A prospective observational analytic cohort study was conducted using data from the national web-based registry RINKAS. Eligible patients were adults diagnosed with HCC and initiated on lenvatinib therapy at RSMH Palembang between January and December 2025. Recorded variables included ECOG performance status, comorbid etiologies, albumin, bilirubin, platelet counts, MELD score, and Child-Pugh class. Descriptive and comparative analyses were performed to explore associations between baseline characteristics and treatment response.

Results:

A total of 20 patients receiving lenvatinib were included. ECOG scores ranged from 0 to 2, with the majority presenting with ECOG 0–1. MELD scores varied between 55.5 and 77.3, while Child-Pugh scores ranged from 4 to 9, predominantly within Child-Pugh A and early B classifications. Preliminary analysis demonstrated that patients with ECOG 0–1 and lower Child-Pugh scores (A5–A6) tended to exhibit more favorable clinical outcomes, whereas higher MELD scores and Child-Pugh B9 were associated with poorer treatment tolerance and reduced response. Trends suggested that preserved liver function at baseline correlated with improved therapeutic benefit from lenvatinib.

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Conclusions:

Baseline clinical performance and hepatic reserve reflected by ECOG status, Child-Pugh classification, and MELD score appear to be important predictors of treatment response to lenvatinib among HCC patients. Patients with better functional status and more preserved liver function demonstrated more favorable outcomes. These findings support the incorporation of liver function stratification into treatment decision-making for lenvatinib therapy and warrant further investigation with larger sample sizes and AFP-dynamic analysis to validate predictive accuracy.

Keywords:

Hepatocellular carcinoma, Lenvatinib, ECOG performance status, Child-Pugh score, MELD score

Keyword(s) :

1309 - Unlocking CTNNB1 hotspot mutations from cell free DNA (cf DNA) in hepatitis B virus induced hepatocellular carcinoma A cross sectional study from Bangladesh

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Background / Hypothesis:

Hepatocellular carcinoma (HCC), the dominant form of primary liver cancer, remains a major global health challenge. Chronic hepatitis B virus (HBV) infection is a significant risk factor of hepatocarcinogenesis driven by genetic and epigenetic alteration. Mutations in the CTNNB1 gene, particularly in exon 3, activate Wnt signaling pathway, leading to uncontrolled transcription and cell proliferation. Early detection of these somatic mutations may serve as a cornerstone of personalized medicine, enabling disease monitoring and tailored therapeutic strategies. Cell-free DNA (cfDNA) is considered a promising alternative source for predicting somatic mutations, offering to track disease dynamics and tailor therapeutic strategies to individuals. This study aimed to detect CTNNB1 gene mutation from cfDNA in individuals with HBV-related chronic liver disease (CLD) and HCC and to assess their

pathogenicity using various bioinformatics tools.

Materials and Methods:

A total of 135 participants were included: 85 with HBV-induced HCC at different stages, 30 with HBV-related CLD and 20 healthy controls. cfDNA was extracted from peripheral blood using the QIAamp Circulating Nucleic Acid Extraction Kit. DNA was amplified by conventional PCR using specific primers, followed by agarose gel electrophoresis and UV visualization. Amplicons were analyzed using Sanger sequencing. Mutation analysis was done using FinchTV software, in silico tools- SNAP, PredictSNP, and I-Mutant were used to predict pathogenicity and protein stability.

Results:

Hotspot mutations were detected in 28.2% (24/85) of HCC patients at the following sites: c.95A>T/G (p.D32V/G; 9.4%), c.110C>A/G (p.S37Y/C; 8.2%), c.133T>C/A (p.S45P/T; 7.1%), c.98C>G (2.4%), and c.121A>G (1.2%). Mutation frequency was significantly higher in late-stage HCC (36.7%) than early-stage (17.4%) ($p < 0.05$; Pearson $r = 0.9961$). Only one mutation (c.133T>C/A) was found in the CLD group (3.3%), and none in healthy controls. In early-stage HCC, serum albumin levels were significantly elevated in mutated cases ($p < 0.05$), while AST levels differed significantly in late-stage HCC between mutated and unmutated groups. Pathogenicity predictions indicated deleterious effects at codons c.95, c.98, c.110, and c.133. I-Mutant analysis revealed reduced β -catenin stability at c.95, c.110, and c.121.

Conclusions:

These findings reflect the potential role of CTNNB1 gene mutations in the pathogenesis and progression of HCC across different stages. In silico prediction tools confirmed the deleterious nature of mutations at critical codons. This non-invasive molecular profiling approach may facilitate personalized treatment strategies and real-time monitoring of tumor dynamics.

Keyword(s) : CTNNB1 gene, Wnt signaling pathway, β -catenin, somatic mutations, hepatocellular carcinoma, Cell-free DNA, Hepatitis B, Bangladesh

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1313 - Combination of radiotherapy and durvalumab gemcitabine cisplatin therapy improve survival in biliary tract cancer (especially extrahepatic bile duct cancer) patients

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Background / Hypothesis:

Durvalumab (Durva) plus gemcitabine/cisplatin (GP) proved better efficacy compared to GP for advanced biliary tract cancer (BTC) patients in TOPAZ-1 trial. Radiotherapy (RT) has immune-stimulatory effect through several mechanism such as releasing tumor associated antigen etc. However, there is no data about synergistic effect and clinical efficacy of RT plus Durva/GP therapy. Therefore we performed study for describing clinical efficacy of combination of RT plus Durva/GP therapy.

Materials and Methods:

The clinical course of patients with advanced BTC who received Durva/GP therapy in single cancer center from April 2023 to July 2025 was assessed. Response rate (partial response [PR], stable disease [SD], progressive disease [PD]) per RECIST v1.1 and median progression free survival (PFS), overall survival (OS) were analyzed.

Results:

Total 21 BTC patients were treated with Durva/GP therapy. Gallbladder cancer (GBC): Intrahepatic cholangiocarcinoma (IHCCC) : Extrahepatic CCC (EHCCC) patients were 6:8:7. Eight patients (RT group) received several types of RT (PULSAR in 6 patients, TARE in 1 patient, carbon beam therapy in 1 patient). The rate of PR:SD:PD in RT versus (vs) non-RT group were 37.5% vs 15.4% : 62.5% vs 61.5% : 0% vs 23.1%. Median PFS in RT vs non-RT group were 10 vs 5 months (p = 0.028) and median OS were 13 vs 10 months (p = 0.012). In only EHCCC patients, median PFS, OS of RT group were better than non-RT group with statistically significance (PFS 10 vs 5 months, p = 0.004; OS 11 vs 6 months, p = 0.027). Almost all patients complained only grade 1/2 nausea and there were no cases with Grade 3/4 adverse effects.

Conclusions:

This study demonstrates that radiotherapy can improve clinical efficacy of Durva/GP therapy in advanced BTC, especially EHCCC patients. Further large scaled prospective studies are needed for proving synergistic effect of radiotherapy and Durva/GP therapy to BTC patients.

Keyword(s) : Biliary Tract Cancer, Durvalumab, Radiotherapy

1314 - SDHA Deficiency in Hepatocellular Carcinoma Promotes Tumor Progression through Succinate Induced M2 Macrophage Polarization

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Background / Hypothesis:Hepatocellular carcinoma (HCC) is an aggressive and lethal malignancy. Metabolic reprogramming dynamically remodels the tumor microenvironment (TME) and drives HCC progression. This study investigated the mechanism through which metabolic reprogramming remodels the TME in HCC.

Materials and Methods:HCC patient transcriptome data were subjected to bioinformatics analysis to identify differentially expressed genes and immune infiltration status. Immunohistochemical analysis was performed to determine the correlation between succinate dehydrogenase complex subunit A (SDHA) expression and M2 macrophage infiltration. SDHA-knockdown or SDHA-overexpressing HCC cells were used for in vitro experiments, including co-culturing, flow cytometry, and enzyme-linked immunosorbent assay. Western blotting assay, functional assays, and subcutaneous tumor model mice were used to elucidate the molecular mechanisms underlying succinate-mediated HCC cell-macrophage interactions in the TME.

Results:Higher infiltration of M2 macrophages correlated with worse prognosis in HCC patients. SDHA was downregulated in HCC tumor tissues and showed a negative correlation with M2 macrophage infiltration. SDHA knockdown promoted M2 macrophage polarization, whereas SDHA overexpression reversed this effect. Mechanistically, SDHA deficiency in HCC cells induced succinate accumulation, which promoted

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M2 macrophage polarization by activating the G protein-coupled receptor 91 (GPR91)/signal transducer and activator of transcription 3 (STAT3) pathway. Concurrently, succinate stimulation enhanced mitochondrial oxidative phosphorylation in M2 macrophages, thereby promoting HCC progression. Serum succinate levels were elevated in HCC patients. The receiver operating characteristic curve analysis indicated that serum succinate is a promising diagnostic marker for HCC (area under the curve = 0.815).

Conclusions:SDHA deficiency leads to succinate accumulation, which promotes M2 macrophage polarization through the GPR91/STAT3 pathway, thereby facilitating HCC progression. Based on these findings, serum succinate could be a promising diagnostic biomarker for HCC.

Keyword(s) : Hepatocellular carcinoma; Metabolic reprogramming; Tumor microenvironment; Succinate; M2 macrophage; Succinate dehydrogenase complex subunit A (SDHA); G protein-coupled receptor 91 (GPR91)

1315 - Clinical characteristics and combined pathogen spectrum of patients with influenza A in winter a retrospective study based on targeted high throughput sequencing detection

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Background / Hypothesis:

To investigate the clinical characteristics, spectrum of co-infecting pathogens, and their associations in patients with influenza A virus infection in a tertiary hospital.

Materials and Methods:

Patients with influenza A virus infection confirmed by targeted next-generation sequencing (tNGS) between December 2024 and February 2025 were enrolled. Clinical data and microbiological test results were collected and analyzed retrospectively.

Results:

A total of 66 patients were included, of whom 54 (81.8%) had co-infections; bacterial co-infection was the most common (34/66, 51.5%). The spectrum of co-infecting pathogens varied across age groups. In infants and young children, *Streptococcus pneumoniae*

and *Staphylococcus aureus* were the predominant pathogens (both 19.2%), whereas *Mycoplasma* spp. accounted for most co-infections in adolescents (40.0%). The rate of fungal co-infection was significantly higher in adults and older adults than in infants and young children (42.9% vs. 2.1%, $P = 0.005$; 57.1% vs. 2.1%, $P < 0.001$, respectively). The proportion of severe disease among adults was 71.4%. In adults with diabetes, the rate of *Aspergillus fumigatus* infection was significantly higher than in non-diabetic adults (100% vs. 0%, $P = 0.048$). All elderly patients with severe disease had fungal co-infections.

Conclusions:

Influenza A is highly prevalent in winter and is frequently complicated by co-infection. tNGS testing facilitates etiological surveillance of co-infecting pathogens. Age-stratified, precise prevention and treatment strategies—focusing on bacterial infections in infants and young children, mycoplasma infections in adolescents, and fungal infections in adults and the elderly—may help reduce the incidence of severe disease and improve clinical outcomes.

Keyword(s) : influenza A; targeted Next-Generation Sequencing; mixed infection; bacteria; fungi; mycoplasma

1317 - Association Between Clinical Predictors And Albumin Billirubin Score In Hepatocellular Carcinoma Patients Receiving Lenvatinib At Dr. Mohammad Hoesin General Hospital Palembang During 2025

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Background / Hypothesis:

Lenvatinib is widely used as a first-line systemic therapy for hepatocellular carcinoma (HCC), but treatment responses vary substantially among patients. Clinical predictors such as hepatic functional reserve and performance status may influence treatment outcomes. This study examined the relationship between Child-Pugh class, ECOG performance status, and Albumin bilirubin (ALBI) score in patients with HCC receiving lenvatinib at a tertiary referral center in Indonesia.

Materials and Methods:

A retrospective review was conducted using complete records of HCC patients treated with lenvatinib in 2025.

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Data collected included Child–Pugh classification, ECOG performance status, and ALBI score. Descriptive statistics were used to summarize patient characteristics. Pearson correlation analysis was applied to assess associations between clinical predictors and ALBI scores.

Results:

Twenty patients were eligible for analysis. Most patients were classified as Child–Pugh A or B, and ECOG performance status was predominantly 0–1. ALBI scores clustered mainly within grades 1 and 2. Better hepatic reserve (Child–Pugh A) and ECOG 0 were consistently associated with more favorable ALBI grades. Significant correlations were observed between Child–Pugh class and ALBI ($r = 0.79$), with a weaker correlation between ECOG status and ALBI ($r = 0.41$). These results indicate alignment between clinical assessments of hepatic function and biochemical liver status.

Conclusions:

Child–Pugh classification and ECOG performance status correlate with ALBI score in patients receiving lenvatinib for HCC. Patients with better preserved liver function and performance status demonstrated more favorable ALBI profiles. These findings support the use of ALBI as an objective complement to established clinical indicators when assessing suitability for lenvatinib therapy and planning treatment strategies.

Keyword(s) : Hepatocellular Carcinoma, Clinical predictors, ALBI score, Lenvatinib.

1318 - Atezolizumab and bevacizumab therapy combined with expanded NK cells significantly prolongs survival in vivo hepatocellular carcinoma model.

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Background / Hypothesis:

Atezolizumab plus bevacizumab (Ate+Beva) proved better efficacy compared to sorafenib for advanced hepatocellular carcinoma (HCC) patients in IMbrave150

trial. However objective response rate of Ate+Beva therapy is around 30%, therefore several trials about combination protocol such as dual immune check point inhibitor plus bevacizumab etc. are ongoing to overcome this unmet need. Natural killer (NK) cell therapy is considered as promising tool that can enhance the efficacy of immunotherapy via several mechanism, however there are no studies about this issue. Therefore we performed preclinical study for efficacy of Ate+Beva therapy combined with expanded NK cell in HCC in vivo model.

Materials and Methods:

SNU-398 cells were subcutaneously injected into the left flank (2.5×10^6) and right thigh (5×10^6) of SCID mice. Four days later, atezolizumab (10 mg/kg) and bevacizumab (5 mg/kg) were administered intraperitoneally, and NK cells (1×10^7) were injected via the tail vein. Tumor size (length $\times$ width) was measured twice weekly. Atezolizumab, bevacizumab, and NK cell treatments were repeated three times at 1-week intervals (Figure 1).

Results:

Total 16 mice were categorized as four groups (Control, NK monotherapy, Ate+Beva, NK+Ate+Beva). The effect about shrinkage of tumor volume & survival rate were maximized in the NK+Ate+Beva therapy group compared to other three groups with significance statistically (Figure 2, 3). There were no meaningful toxicity in NK+Ate+Beva group compared to other three groups.

Conclusions:

This study demonstrates that combination of NK cell with Ate+Beva can improve the efficacy of Ate+Beva therapy in HCC preclinical model. Further clinical prospective studies are needed for proving synergistic effect of NK cell and Ate+Beva therapy to HCC patients.

Keyword(s) : Atezolizumab, Bevacizumab, NK cell, Combination, Hepatocellular Carcinoma

1319 - Integrated Proteomic and Metabolomic Profiling Reveals Distinct Immune and Metabolic Landscapes in Occult Hepatitis B Infection

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Background / Hypothesis:

The pathophysiological distinctions between HBsAg-positive Hepatitis B and Occult Hepatitis B Infection (OBI) remain elusive. This study integrated proteomic and metabolomic analyses to delineate the systemic immune microenvironment in OBI and to identify clinically viable biomarkers for discriminating it from HBsAg-positive HBV.

Materials and Methods:

We analyzed plasma from 20 OBI patients (HBsAg-negative, HBV DNA <200 IU/mL), 20 HBsAg-positive HBV patients, and 10 healthy controls. Mass spectrometry-based proteomics and NMR-based metabolomics were employed. Data analysis encompassed differential expression screening, pathway enrichment, and Weighted Gene Co-expression Network Analysis (WGCNA). Machine learning algorithms were applied to construct a diagnostic model, with candidate biomarkers validated by ELISA in an expanded cohort.

Results:

HBsAg-positive patients exhibited significantly higher ALT levels than OBI patients ($p=0.010$). Multi-omics profiling revealed distinct HBV-induced perturbations in metabolic pathways and inflammatory signatures in both groups. Cellular deconvolution linked HBsAg-positive infection to Kupffer cells and OBI to hepatocytes. Immune phenotyping correlated OBI with CD4⁺ central memory T cells and memory B cells, suggesting a poised mechanism for rapid recall response. A machine learning-derived diagnostic model, built on significantly expressed molecules, robustly differentiated the two patient groups (AUC >0.8). ELISA validation confirmed FGB and FGG as elevated in OBI, nominating them as potential discriminatory biomarkers.

Conclusions:

This study unveils significant differences in the immune and metabolic landscape between HBsAg-positive HBV and OBI. We further present a high-accuracy, non-invasive diagnostic model, offering a promising tool for precise clinical differentiation and patient management.

Keyword(s) : Occult hepatitis B infection, Proteomic, Metabolomic, Biomarkers

1320 - Impact of Long Term Oral Antiviral Treatment on Hepatocellular Carcinoma Risk in Patients With Chronic Hepatitis B Who Are Hepatitis B e Antigen Positive Using the PAGED B Score

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Background / Hypothesis: Chronic hepatitis B (CHB) is a leading cause of hepatocellular carcinoma (HCC) worldwide, and while oral antivirals lower the risk of HCC, they do not eliminate it. Recent studies indicate that moderate hepatitis B virus (HBV) DNA levels may be strongly linked to HCC risk in hepatitis B e antigen (HBeAg)-positive people with CHB. The recently validated PAGED-B (platelets, age, gender, diabetes, and HBV DNA) score, which incorporates HBV DNA, can be used to stratify HCC risk in this population. We used the PAGED-B score to assess HCC risk in HBeAg-positive patients with CHB enrolled in a trial of tenofovir-based therapy.

Materials and Methods: In a Phase 3 multicenter study

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(GS-US-320-0110), patients who were HBeAg positive with HBV DNA $\geq 20,000$ IU/mL and alanine aminotransferase > 60 U/L (males) or > 38 U/L (females) were randomized to receive tenofovir alafenamide (TAF) 25 mg or tenofovir disoproxil fumarate (TDF) 300 mg once daily for up to 3 years. Thereafter, patients received open-label TAF through year 8 (week 384). PAGED-B scores at baseline (BL) and over 384 weeks were generated. Shifts in risk categories (low risk [0 to < 7] and high risk [≥ 7]) were evaluated.

Results: Among 1052 (TAF, $n = 703$; TDF, $n = 349$) HBeAg-positive patients (66% male, 85% Asian [$n = 893$]/Pacific Islander [$n = 4$], 98% HBeAg positive [2% transitioned to HBeAg-negative status between screening and BL visits]), mean (range) BL PAGED-B score was 4.3 (0–11); 885 (84%) and 167 (16%) were low and high risk, respectively. At weeks 240 and 384, mean (range) changes from BL in PAGED-B scores were -0.8 (-4 to 4) and -0.7 (-4 to 2), respectively. Among those in the low-risk category at BL with available data at week 384, the majority remained unchanged; only 6 (1%) shifted to high risk, primarily driven by age and platelet changes. In the high-risk group, most patients improved to low risk (69%), and only 31% remained at high risk. Overall, 12 HCC cases developed over 8 years (0.3%, BL low-risk group; 5.4%, BL high-risk group).

Conclusions: The application of the PAGED-B risk score demonstrates that among HBeAg-positive patients, most (69%) high-risk patients experienced a decrease in risk with long-term treatment, whereas shifts from low risk of HCC at the start of treatment to a higher risk category were rare (1%).

Keyword(s) : Hepatitis B Virus (HBV), hepatocellular carcinoma (HCC), virology, nucleos(t)ide analogs, tenofovir alafenamide (TAF), tenofovir disoproxil fumarate (TDF)

1321 - Predictors of undetectable hepatitis delta virus RNA at 48 weeks after end of treatment with bulevirtide monotherapy in the MYR 301 study

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Background / Hypothesis: Bulevirtide (BLV) is approved for the treatment of chronic hepatitis delta. In the MYR301 study evaluating BLV monotherapy for 2–3 years, a subset of patients who achieved undetectable hepatitis delta virus (HDV) RNA by end of treatment (EOT) maintained undetectable viremia at 48 weeks (W) after EOT (FU48). Here, we present predictors of sustained HDV RNA undetectability through FU48 after 96W or 144W of BLV treatment.

Materials and Methods: Data were analyzed from 149 patients randomized to immediate treatment with BLV 2 or 10 mg/d for 144W, or to 48W of no treatment then BLV 10 mg/d for 96W (DT- > 10 mg). All patients were to be followed through FU48. Logistic regression modeling examined potential predictors of sustained HDV RNA undetectability ($<$ lower limit of quantitation [LLOQ; target not detected]) through FU48 in patients with undetectable viremia at EOT.

Results: Across all cohorts, 65/149 (44%) patients achieved HDV RNA undetectability at EOT, of whom 23/64 (36%) with available follow-up HDV RNA data had sustained undetectability through FU48. Rates of

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sustained undetectability were higher in BLV 2 mg (7/14, 50%) and 10 mg (10/25, 40%) vs DT->10 mg (6/26, 23%) cohorts. Undetectability rates at EOT were higher in patients receiving BLV 10 mg, but the relapse rate was lower in the few patients with HDV RNA <LLOQ in the BLV 2 mg cohort. Baseline predictors of sustained undetectability posttreatment included baseline HDV RNA less than a median of 4.5 log₁₀ IU/mL (odds ratio [OR]: 6.2; 95% CI [1.9, 20.8]; P=.003) and lower baseline hepatitis B surface antigen (HBsAg) level (OR: 0.3 per log₁₀ IU/mL; 95% CI [0.1, 0.8]; P=.019). On-treatment predictors included greater duration of continuous undetectability at EOT (OR per additional week: 1.0; 95% CI [1.0, 1.1]; P <.0001), HBsAg loss or decrease by ≥ 1 log₁₀ IU/mL (OR: 7.2; 95% CI [1.2, 42.3]; P=.030), and W144 antidrug antibody (ADA) incidence (OR: 10.2; 95% CI [1.9, 55.7]; P=.008). The proportion of patients with sustained posttreatment undetectability was 9/10 (90%) in those with ≥ 96 W of undetectability at EOT, 11/22 (50%) with ≥ 48 to <96W, and 3/32 (9%) with <48W. Of patients with ADA incidence by W144, 8/10 (80%) had sustained undetectability vs 15/54 (28%) without. Baseline cirrhosis was not a predictor of sustained posttreatment undetectability; 13/32 (41%) with cirrhosis had sustained undetectability vs 10/32 (31%) without cirrhosis.

Conclusions: Early and sustained HDV RNA undetectability predicted undetectability at EOT and sustained undetectability during follow-up.

Keyword(s) :

1322 - COMPARISON OF SEROLOGICAL FIBROSIS MARKERS WITH LIVER BIOPSY FIBROSIS STAGES IN CHRONIC HEPATITIS B PATIENTS

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Background / Hypothesis: Hepatitis B virus is the most common cause of chronic hepatitis and a global public health problem. It can cause hepatocyte damage, immune cell infiltration and liver fibrosis. Consequently, patients may develop liver cirrhosis and hepatocellular carcinoma. The main step of treatment is to determine

the degree of liver fibrosis. Biochemical, serological and molecular tests, histopathological examination, imaging are the methods used for diagnosis and staging. This study aimed to detect the serological markers closest to the liver biopsy results and to determine the cut-off value by comparing the liver biopsy results and biochemical fibrosis markers of patients followed up with chronic hepatitis B infection. **Materials and Methods:** Four hundred fourteen patients between the ages of 18 and 90 who were admitted to Samsun Training and Research Hospital as outpatients and were followed for Chronic Hepatitis B infection and whose HBV-DNA level was > 2000 IU/ml at least once and who underwent liver biopsy with fibrosis staging, were included in the study. The demographic characteristics, pathology results and biochemical data of the patients were recorded retrospectively as file findings. The evaluated variables and serological fibrosis markers (FIB4, APRI, GPR, AGAP, AAR, NLR, RPR, King's score, FibroQ, API, PLR, AAPRI, APPR, GUCI, Lok score, GAR, FI) were statistically assessed for their diagnostic validity in predicting liver fibrosis by comparing them with the Ishak fibrosis stage, and the cut-off value was determined.

Results: In our study, as a result of the ROC analysis performed to determine the threshold values of factors that can predict advanced fibrosis (F4-6), FIB4, APRI, GPR, AGAP, AAR, RPR, King's score, API, PLR, APPR, GUCI, Lok score, GAR, FI serum tokens were successful. The highest AUROC value was APRI (0.791, p<0.001) and followed by GUCI, APPR, Kings, AGAP, GPR, FIB4, FI (0.784, 0.780, 0.771, 0.748, 0.736, 0.734, 0.701, p<0.001, respectively).

Conclusions: In cases where diagnosis of liver fibrosis takes a long time, is costly, requires special methods-equipment and qualified evaluation, such as biopsy; noninvasive serum markers that are short-term, low-cost, and easily evaluated can make a significant contribution to predicting fibrosis. However, there is no perfect serum biomarker. It is thought that markers with high predictive power such as APRI, GUCI, APPR, Kings score, AGAP, GPR, FIB4, FI can be beneficial in selected cases.

Keyword(s) : Chronic Hepatitis B, Liver Fibrosis, Non-invasive tests

1323 - Final results of MYR301 Phase 3 study evaluating the efficacy and safety of up to 144 weeks of bulevirtide monotherapy for chronic hepatitis delta and 96 weeks of posttreatment follow up

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Background / Hypothesis: Bulevirtide (BLV) 2 mg/day (d) is approved for treatment of compensated chronic hepatitis delta (CHD). In the MYR301 Phase 3 study evaluating BLV monotherapy for 2–3 years, BLV was safe and effective through 144 weeks (W). We present final results through follow-up at 96W (FU96) after end of treatment (EOT)

Materials and Methods: Patients with CHD (N=150) were randomized to immediate treatment with BLV 2 or 10 mg/d for 144W or to 48W of delayed treatment (DT) followed by BLV 10 mg/d for 96W (DT->10 mg) and 96W posttreatment FU. Efficacy endpoints included virologic response (VR; undetectable hepatitis delta virus [HDV] RNA or $\geq 2 \log_{10}$ IU/mL decline from baseline), combined response (CR; VR and alanine aminotransferase [ALT] normalization), ALT normalization, undetectable HDV RNA, and hepatitis B surface antigen (HBsAg) loss.

Results: Baseline characteristics were similar across groups. Most patients (92%) remained in the study at EOT; 72% and 57% completed FU48 and FU96, respectively. CR rates in the 2, 10, and DT->10 mg groups, respectively, declined from 57%, 54%, and 56% at EOT to 24% in each group at FU96. HDV RNA undetectability rates were 29%, 50%, and 52% at EOT and 20%, 22%, and 20% at FU96, respectively. Of 64 patients across all groups with undetectable HDV RNA at EOT and available FU data, 36% had sustained undetectable HDV RNA through FU96, and 64% had viral relapse, which occurred in 93% by FU24 and none after FU48. Sustained posttreatment undetectable HDV RNA was more frequent in patients with longer on-treatment continuous HDV RNA undetectability at EOT: 9/10 (90%) for ≥ 96 weeks, 11/22 (50%) for ≥ 48 to <96 weeks, and 3/32 (9%) for 0 to <48 weeks. Posttreatment HBsAg loss occurred in 3 patients. In the posttreatment period, 14/142 (10%) patients had ALT $> 10 \times$ the upper limit of normal, which occurred by FU24 in most (10/14, 71%). Posttreatment hepatic serious adverse events (SAEs) were reported in 20/142 (14%) patients. The hepatic SAEs resolved in 17/20 (85%) patients, ≥ 16 of whom restarted BLV.

Conclusions: In patients receiving BLV monotherapy for 96W or 144W, response rates decreased after treatment discontinuation. A subset of patients maintained undetectable HDV RNA for 2 years posttreatment, which was associated with longer duration of continuous on-treatment undetectability. Posttreatment viral relapse occurred only in the first year after EOT and may be associated with hepatitis flares.

Keyword(s) :

1324 - Synergistic effects of type 2 diabetes and alcohol consumption on all cause and liver related mortality among patients with steatotic liver

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disease

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Background / Hypothesis:

Type 2 diabetes (T2D) and alcohol consumption have been acknowledged as major risk factors for adverse clinical outcomes for individuals with steatotic liver disease (SLD). However, data about synergistic effects of T2D and alcohol on all-cause and liver-related mortality in SLD remains scarce.

Materials and Methods:

Leveraging data from UK Biobank, we compared risk of all-cause and liver-related mortality across 6 groups stratified by T2D and alcohol consumption in Cox proportional hazards regression: no T2D+no/mild alcohol, no T2D+MetALD, no T2D+ALD, T2D+no/mild alcohol, T2D+MetALD, T2D+ALD. The population attributable fraction (PAF) and relative excess risk due to interaction (RERI) were calculated to exam the interplay between these two factors.

Results:

24,309 all-cause deaths and 1,009 liver-related were recorded for 175,713 participants from UK Biobank. The coexistence of T2D and elevated alcohol intake, particularly when both T2D and severe alcohol consumption, was associated with substantially worse outcomes, including a 1.23~1.64 fold increased risk of all-cause mortality and a markedly higher 1.58~7.32 fold risk of liver-related mortality compared with no T2D+no/mild alcohol. While combination of T2D and

alcohol use accounted for a modest PAF of 6.78% for all-cause mortality, they contributed to 40.52% of liver-related mortality. Significant RERI was exclusively observed for liver-related mortality but not for all-cause mortality.

Conclusions:

Co-exposure to T2D and alcohol intake substantially increased the risk of all-cause and especially the liver-related mortality in SLD individuals. Their interplay conferred significant disease burden, underscoring the importance of abstinence and glycemic control in the management of SLD patients.

Keyword(s) :

1325 - Serum metabolomic characteristics and their predictive value for ninety day prognosis in patients with acute on chronic liver failure

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Background / Hypothesis: Acute-on-chronic liver failure (ACLF) is characterized by severe metabolic disturbances; however, the specific metabolomic features and their predictive value on 90-day prognosis remain unclear. To identify serum metabolomic changes in patients with ACLF with different prognoses to support clinical prediction of outcomes and treatment decisions.

Materials and Methods: This non-interventional, observational case-control study enrolled 58 patients with ACLF. Fasting venous blood samples were analyzed using targeted metabolomics. Univariate and multivariate statistical analyses identified differential metabolites among 18 amino acids, 11 fatty acids, 5 gut microbiota-related metabolites, and 4 bile acid metabolites. Binary logistic regression identified independent mortality risk factors, visualized via forest plots and receiver operating characteristic (ROC) curves.

Results: Significant differences ($P < 0.05$) were observed between the death and survival groups in baseline age, Model for End-Stage Liver Disease (MELD) score, MELD with sodium, neutrophil-to-lymphocyte ratio (NLR), total bilirubin, serum creatinine, blood urea nitrogen, and platelet count. Metabolites, including L-carnitine, creatinine, alanine, arginine (Arg), proline, choline, and oleic acid, also

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showed statistically significant differences between the groups. Multivariate analysis identified age, NLR, and Arg as independent risk factors for 90-day mortality in patients with ACLF. The predictive model, ANA (Age–NLR–Arg) = $-15.481 + 0.135 \times \text{Age} + 0.156 \times \text{NLR} + 0.203 \times \text{Arg}$, with a cutoff of 0.759, achieved an area under the ROC curve of 0.945 with sensitivity of 84.0% and specificity of 87.9%.

Conclusions: The ANA model demonstrates a strong predictive value for 90-day mortality risk in patients with ACLF.

Keyword(s) : Acute-on-chronic liver failure; Metabolomics; Artificial liver blood purification system; Modeling; Prognosis

1329 - Multi center analysis of HDV seroprevalence and epidemiological profile in Chinese HBsAg positive inpatients

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Background / Hypothesis:

Hepatitis D virus (HDV) is a defective satellite virus that requires hepatitis B virus (HBV) for replication and exacerbates the severity of viral hepatitis. China carries a heavy burden of HBV, with an estimated 43.3 million infections in 2021. However, the national prevalence and clinical impact of HDV/HBV coinfection remains poorly defined. Accurate epidemiological data are essential to guide clinical and public health responses.

Materials and Methods:

From 2023 to 2025, we screened HBsAg-positive inpatients across nine medical centers representing northern, southern, eastern, and western China. Serum samples were tested for anti-HDV IgM and IgG using ELISA, and seropositive samples were further detected for HDV RNA via quantitative RT-PCR. Clinical and laboratory data were collected and analyzed.

Results:

The final analysis included 13,953 HBsAg-positive patients. Anti-HDV antibodies were detected in 0.57% of the cohort, with 0.29% positive for IgM and 0.31% for IgG. HDV RNA, indicating active viral replication, was detected in 5.06% of seropositive cases. Specifically, HDV RNA was positive in three of four patients with both IgM and IgG antibodies, and in one of 39 with IgG alone. Seroprevalence of HDV exhibited regional variation ($p < 0.001$), ranging from 0% in Fujian to 0.96% in Guizhou. Higher rates of HDV seropositivity were observed in patients with HBsAb (0.75% vs 0.57%), HCV co-infection (2.47% vs 0.51%) or TP co-infection (0.56% vs 0.48%). Anti-HDV-positive patients were significantly older (57 vs 52 years, $p = 0.016$) and exhibited lower neutrophil (2.56 vs $3.09 \times 10^9/L$, $p = 0.023$) and lymphocyte counts (1.27 vs $1.50 \times 10^9/L$, $p = 0.005$), prolonged prothrombin time (13.1 vs 12.6 s, $p = 0.032$), and reduced fibrinogen levels (2.53 vs 2.74 g/L, $p = 0.004$) in peripheral blood compared to anti-HDV-negative individuals. Additionally, anti-HDV-positive patients had higher HBsAg levels (340.1 vs 250.1 COI, $p = 0.027$) and a non-significant trend toward lower HBV DNA loads (49.9 vs 69.1 IU/mL, $p = 0.101$).

Conclusions:

This multi-center study provides an updated overview of HDV seroprevalence and active infection rates in China, which are substantially lower than previously reported. The findings highlight geographical disparities and high-risk groups of HDV infection, particularly those HBsAg-positive patients with HCV or TP co-infection or undetectable HBV DNA, providing valuable insights for targeted HDV surveillance and prevention strategies.

Keyword(s) : HDV/HBV coinfection, multi-center analysis, seroprevalence, active infection

1330 - Dapsone Syndrome Relapse Unraveling a Complex Case

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Nepal ¹

Dapsone Syndrome Relapse: Unraveling a Complex Case

Abstract

Background

Dapsone syndrome is a rare hypersensitivity reaction marked by symptoms like high fever, maculopapular rashes, hepatitis, lymphadenopathy, and lymphocytosis. Typically, these manifestations occur within the initial 6 weeks of treatment. A reported case involves an individual with borderline tuberculoid leprosy who experienced a relapse of this syndrome.

Clinical significance

A relapsing course of Dapsone syndrome is rare and must be identified early for proper management.

Case Report

A 45-year-old female diagnosed with Boderline Tuberculoid Leprosy was started on combination of antibiotics containing Dapsone who developed fever along with maculopapular rashes all over her body on her 25th day of treatment. Laboratory investigations revealed lymphocytosis with abnormal liver function. She was diagnosed with Dapsone syndrome and her Anti-leprotic therapy were stopped and was started on steroids and other supportive measures. Her liver enzymes showed a declining trend and rashes subsided and steroids was stopped. However, after 2 weeks she experienced a recurrence of the same widespread rashes along with deranged liver function. The patient was again treated with intravenous steroids along with other supportive measures. Rashes and swelling subsided within 4 days of starting high-dose steroids along with gradual improvement in biochemical parameters. She was free of hepatic, dermatological and other parameters in next 4 weeks.

Conclusion

Dapsone stands as the primary treatment choice for all types of leprosy, generally well tolerated by patients. Within the spectrum of rare hypersensitivity reactions, Dapsone syndrome emerges. In the mentioned case, the recurrence of dapsone syndrome was observed, and was treated accordingly.

Keyword(s) : dapsone, DILI, dapsone relapse

1332 - Socioeconomic Factors Among Chronic Liver Disease Patients Experience From Largest Tertiary Care Hospital Of Pakistan

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Background / Hypothesis:

Pakistan has one of the highest global rates of viral hepatitis, contributing significantly to the prevalence of chronic liver disease (CLD). The progression and outcomes of CLD are influenced by social determinants of health (SDOH), including economic disparities, healthcare accessibility, environmental conditions, and public awareness. Understanding these factors will enhance disease prevention and management.

OBJECTIVES : To analyze the economic and social challenges confronting chronic liver disease patients in Karachi,Pakistan.

Materials and Methods:

It is an ongoing cross-sectional study that began in August 2024 at the Jinnah Post Graduate Medical Centre in Karachi. This study included CLD patients and their socioeconomic status, healthcare access, education, social and community context, neighborhood built environment, hospitalizations or readmissions, as well as disease complications, these were among the outcome variables of interest.

Results:The study enrolled 1200 patients with chronic liver disease (CLD), predominantly from Sindh 1179 (94%) and Balochistan 41 (3.2%). Of these, 665 (53%) were males and 586 (47%) were females, with a mean age of 50 ± 13 years. Most patients 1092 (91%) were married. Within Sindh,

Karachi was home to the majority 1043 (83%), particularly the outskirts 213 (17%), followed by Malir 184 (15%), Korangi 174 (14%), Orangi 167 (13%), and Central Karachi 161 (13%).

Etiologies contributing to CLD were HCV 853(81%) followed by HBV 564(47%),HBV+HDV 192(16%) and NAFLD 43(3.6%).A significant proportion 893 (71.4%) had decompensated CLD (DCLD), with a Child-Turcotte-Pugh (CTP-B) score in 767 patients (62%) and



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a MELD score below 15 in 922 (74%).

Stratification by income revealed significant disparities in disease severity and clinical complications. Patients in the low-income group exhibited disproportionately higher rates of Decompensated CLD: 81% vs. 50% (middle-income) and 25% (high-income), CTP class B: 72% vs. 35% and 24% and MELD score <15: 61% vs. 100% and 96%.

Complications were also more frequent among low-income patients, Jaundice: 68% (vs. 48% and 28%), Ascites: 57% (vs. 35% and 16%), Upper GI bleed: 56% (vs. 33% and 21%) Hepatic encephalopathy: 38% (vs. 15% and 8%). All associations were statistically significant ($P < 0.001$). These findings indicate a clear socioeconomic gradient in CLD severity, with the lowest-income patients experiencing the greatest disease burden and complications.

Conclusions: Low-income patients with chronic liver disease experience significantly higher rates of disease severity and complications, underscoring the critical impact of socioeconomic status on clinical outcomes and healthcare disparities. These important elements must not be disregarded to prevent disease-related morbidity.

Keyword(s) : Hepatitis, Socioeconomic factors, chronic liver disease

1333 - Impact of Prior Treatment Response on Survival Outcomes of Subsequent Systemic Therapies Following First line Atezolizumab plus Bevacizumab in Patients with Unresectable Hepatocellular Carcinoma

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Background/Hypothesis: Optimal subsequent treatment strategies following first-line Atezolizumab plus bevacizumab (Atezo/Bev) in unresectable hepatocellular carcinoma (HCC) remain unclear, particularly beyond the second-line treatment settings. We evaluated whether the treatment response to the preceding systemic therapy affects survival outcomes of subsequent treatment lines following Atezo/Bev.
Materials and Methods: In this single-center

retrospective cohort study, we analyzed 353 patients with unresectable HCC, who received Atezo/Bev ≥ 3 cycles (May 2022–December 2023) and underwent subsequent treatment. For each treatment line, patients were stratified into progressive disease (PD) and non-PD groups based on the best response to the prior therapy. Overall survival (OS) and progressive-free survival (PFS) were used as primary and secondary outcomes, respectively.

Results: Among the entire patients, 307 (87.0%) received second-line systemic therapy (lenvatinib or sorafenib in >80% of the patients), and 279 (90.9%) of them underwent the initial response evaluation. OS after second-line therapy did not differ significantly between the first-line PD and non-PD groups (median OS: 7.8 vs. 9.4 months; $P=NS$). Consistently, PFS was also comparable between the two groups (median PFS: 3.7 vs. 4.2 months; $P=NS$). Higher Child-Pugh score at the initiation of second-line treatment emerged as a common risk factor of both poor OS and PFS ($P_s < 0.05$). Interestingly, patients receiving second-line sorafenib or regorafenib showed worse PFS outcomes compared with those treated with lenvatinib ($P_s < 0.05$). In third-line settings (sorafenib or regorafenib in about half of the patients), treatment response of second-line systemic treatment also showed no significant association with subsequent survival outcomes (median OS and PFS of PD and non-PD: 7.4 vs. 6.1 months; and 1.8 vs. 1.6 months, respectively; $P_s=NS$). In addition, the type of prior chemotherapy did not have a significant impact on survival outcomes after initiation of sequential systemic treatment.

Conclusions: The therapeutic response to first-line Atezo/Bev for HCC did not significantly affect survival outcomes of later systemic treatments. Post-Atezo/Bev treatment may not need to depend on prior treatment response in HCC.

Keyword(s) : Atezolizumab plus Bevacizumab; sequential therapy; survival outcome; treatment response

1339 - Clinical Investigation on Hepatocellular Carcinoma Complicated with Esophageal and Gastric Variceal Bleeding

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Background / Hypothesis:

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Hepatocellular carcinoma (HCC) currently ranks as the 4th most common malignant tumor and the 2nd leading cause of cancer-related death in China, posing a severe threat to the health and lives of the Chinese population. Although multiple treatment modalities—including surgery, ablation, transcatheter arterial chemoembolization (TACE), and systemic therapy—have significantly improved the survival of patients with liver cancer in recent years, the complication of esophageal and gastric variceal bleeding (EGVB) in HCC patients has attracted increasing attention. This is primarily because acute EGVB elevates the risk of non-tumor progression-related mortality. Additionally, systemic therapy for HCC may alter the severity of portal hypertension and induce bleeding. To provide patients with comprehensive treatment options, greater emphasis should be placed on the prevention and management of portal hypertension-related complications, particularly EGVB. The objective of this study was to evaluate the clinical characteristics of the first episode of EGVB in patients with HCC.

Materials and Methods:

A retrospective analysis was performed on data from patients admitted to the Department of Gastroenterology/Hepatology, Tianjin Third Central Hospital, between January 2019 and October 2023. All included patients were hospitalized for the first time due to EGVB. The clinical characteristics of HCC patients complicated with EGVB were analyzed.

Results:

During the study period, a total of 5,787 patients were admitted for first-episode upper gastrointestinal bleeding, all of whom were confirmed to have EGVB via gastroscopy. Among these, 598 patients were diagnosed with HCC, accounting for 10.3% of all EGVB patients.

Further analysis focused on these 598 HCC patients with EGVB, revealing the following:

178 cases (29.8%) were complicated with portal vein tumor thrombus;

71 cases (11.9%) had received bevacizumab treatment.

Regarding the etiology of cirrhosis in these HCC patients, the distribution was as follows: 418 cases of hepatitis B-related cirrhosis, 48 cases of hepatitis C-related cirrhosis, 38 cases of alcoholic cirrhosis, 34 cases of primary biliary cholangitis (PBC), and 34 cases of metabolic dysfunction-associated steatotic liver disease (MASLD)-related cirrhosis. Additionally, 175 patients (29.3%) had hypersplenism, and 258 patients (43.1%) had ascites.

Conclusions: For HCC patients—especially those planned to receive combined targeted therapy and immunotherapy—endoscopic examination should be

routinely performed to assess the risk of esophageal and gastric varices. This approach can provide a critical guiding basis for the subsequent management of HCC.

Keyword(s) :

1340 - Akkermansia muciniphila Regulating Cholangiocyte Pyroptosis via the Bile Acid TGR5 Axis

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Background / Hypothesis:

Therapeutic options for Primary sclerosing cholangitis (PSC) remain limited. Given the close association between PSC and gut microbiota, exploring pathogenesis and novel treatments of the disease from this perspective holds significant promise.

Materials and Methods:

A well-established PSC model was induced in mice using a 0.1% 3,5-Diethoxycarbonyl-1,4-Dihydrocollidine (DDC) diet. The temporal dynamics of the gut microbiota and metabolites during disease progression were then profiled using an integrated approach of 16S rRNA sequencing and metabolomics. We further investigated the therapeutic effect of *Akkermansia muciniphila* (AKK) in PSC mice. To this end, we employed an integrated multi-omics strategy, combining 16S rRNA sequencing, transcriptomics, and bile acid metabolomics with pyroptosis pathway analysis, to uncover the underlying protective mechanism.

Results:

During the progression of PSC in mice, the abundance of intestinal AKK gradually decreased, while the intestinal deoxycholic acid (DCA) level also declined. AKK abundance and DCA level were significantly negatively correlated with the degree of biliary inflammation and injury. Furthermore, a positive correlation was observed between intestinal AKK abundance and DCA level in cholestatic mice. Further supplementation with AKK significantly alleviated biliary inflammation and fibrosis in PSC mice. Reduced expressions of hepatic NLRP3, Cleaved caspase-1, and GSDMD-NT, and decreased serum levels of IL-1 β and IL-18 indicated suppression of pyroptosis. Further integrated analysis of 16S rRNA sequencing and bile acid metabolomics revealed that AKK administration, while ameliorating cholestasis and downregulating the biliary pyroptosis pathway, significantly increased the abundance of gut microbiota involved in bile acid biotransformation and elevated the levels of secondary

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bile acids, particularly DCA, in both the liver and feces. Transcriptomic analysis of liver tissue further indicated that AKK intervention upregulated the cAMP signaling pathway and the expression of TGR5 and PKA, implying an activation of the TGR5/cAMP/PKA pathway, which was confirmed at both the mRNA and protein levels. These findings suggested that the DCA-TGR5 axis may be the key molecular mechanism through which AKK regulates biliary pyroptosis.

Conclusions:

Based on the above findings, AKK demonstrated therapeutic potential in alleviating biliary inflammation and delaying the progression of cholestatic disease. The underlying mechanism may involve remodeling the gut microbiota structure, promoting bile acid metabolic reprogramming, stimulating the production of the unconjugated secondary bile acid DCA, and subsequently activating the TGR5 signaling pathway in cholangiocytes. This activation suppressed the GSDMD-mediated pyroptosis pathway, thereby mitigating biliary injury. This work provided a novel strategy for the clinical treatment of PSC by modulating the microbiota.

Keyword(s) : Primary sclerosing cholangitis, Akkermansia muciniphila, Deoxycholic acid, TGR5, pyroptosis

1342 - Exposure to Tenofovir Alafenamide and Tenofovir Disoproxil Fumarate and the Risk of Hepatocellular Carcinoma

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Background / Hypothesis: Nucleos(t)ide analogs have been associated with a reduced risk of hepatocellular carcinoma (HCC) in patients with hepatitis B virus infection. Many studies have suggested that tenofovir-based therapies are more effective than entecavir in reducing HCC risk. Here, we examine HCC risk in patients with chronic hepatitis B using tenofovir alafenamide (TAF) or tenofovir disoproxil fumarate (TDF).

Materials and Methods: Data from four pivotal phase 3 trials were pooled to examine the association between TAF/TDF exposure and HCC occurrence. These included Studies 102 and 103, in which patients were randomized (2:1) to TDF vs adefovir dipivoxil (ADV) for 48 weeks, followed by open-label TDF (up to 8 years' total treatment [384 weeks]), and Studies 108 and 110, in which patients were randomized (2:1) to TAF vs TDF for up to 144 weeks, followed by open-label TAF (up to 8 years' total treatment). HCC occurrence was classified as an adverse event and assessed as part of routine care (hepatic ultrasonography conducted in Studies 108 and 110). Using Cox proportional hazards analyses, we examined the association between exposure to TAF vs TDF and the time to development of HCC. Exposure to TAF vs TDF was the main predictor variable and was captured by the total TAF exposure time/(total TAF exposure time + total TDF exposure time), with values ranging between 0 (TDF only) and 1 (TAF only). HCCs occurring within the first 48 weeks of treatment initiation/switching from ADV to TDF were considered to have pre-existed and were excluded from these analyses to minimize immortal time bias.

Results: In total, 2273 patients (Studies 102/103: n=641; Studies 108/110: n=1632) were included in the pooled analysis. By Year 8, 46 (2.0%) patients had developed HCC (9 cases occurred within the first 48 weeks and were excluded). In the univariate model, sex (hazard ratio [HR]=2.93), age (HR=1.08), cirrhosis (HR=4.32), alanine aminotransferase (ALT) normalization at Week 24 (HR=0.29), and TAF vs TDF

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exposure (HR=0.41) were significantly associated with decreased HCC risk. In the final multivariate analysis, TAF exposure was significantly associated with a 70.0% reduction in HCC risk compared to TDF exposure (HR=0.30). Independently, ALT normalization at Week 24 was also significantly associated with decreased HCC risk (HR=0.29).

Conclusions: Long-term TAF treatment was associated with a greater reduction in HCC risk than TDF treatment. Differences in study design, population, and timing should be considered, and real-world data may help to confirm these findings.

Keyword(s) : Hepatitis B Virus (HBV), Hepatocellular carcinoma (HCC), virology, nucleos(t)ide analogs, tenofovir alafenamide (TAF), tenofovir disoproxil fumarate (TDF)

1343 - Impact of Long Term Tenofovir Based Treatment on Hepatocellular Carcinoma Risk in Patients With Chronic Hepatitis B Using the reREACH B Score

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Background / Hypothesis: Chronic hepatitis B (CHB) is a leading cause of hepatocellular carcinoma (HCC), and oral antivirals can reduce HCC risk. The Risk Estimation for HCC in CHB (REACH-B) score is useful in assessing HCC risk and was updated to incorporate age, gender, platelet count, HBV DNA and alanine aminotransferase (ALT) levels, and hepatitis B e antigen (HBeAg) status. We assessed HCC risk in tenofovir-based clinical trials

using the updated reREACH-B score.

Materials and Methods: In 2 Phase 3 studies, HBeAg-positive (n = 1039) and -negative (n = 593) patients were randomized (2:1) to once-daily tenofovir alafenamide (TAF) 25 mg or tenofovir disoproxil fumarate (TDF) 300 mg for ≤ 3 years (Y), followed by open-label TAF through Y8. reREACH-B scores at baseline and over 8Y (384 weeks [W]) were generated. HCC was assessed throughout and hepatic ultrasonography was introduced at W96 and used every 6 months. Patients were stratified into low (<0.01) and high (≥ 0.01) reREACH-B HCC risk categories, reflecting an annual incidence of 0.2%, the cost-effectiveness threshold for HCC surveillance.

Results: Through 8Y of follow-up, HCC occurred in 26/1632 (1.6%) patients (TAF, 14/1093 [1.3%]; TDF, 12/539 [2.2%]). Nine of 26 (35%) patients had cirrhosis at baseline. Patients who developed HCC were older (median age 52Y vs 39Y; $P<.0001$), more often male (89% vs 65%; $P=.0119$), and more likely to have baseline cirrhosis (FibroTest score ≥ 0.75 ; 35% vs 9%; $P<.0001$). At baseline, 547 and 1084 were categorized as low and high risk. Those at high risk and low risk respectively had median age 43Y vs 32Y; 74% vs 47% were male; 57% vs 77% were HBeAg positive; median platelet counts were 174 vs 233 $\times 10^3/\mu\text{L}$; median HBV DNA was 6.7 vs 8.2 log₁₀ IU/mL; and median baseline ALT levels were 89 vs 70 U/L. Over 8Y, 24/1084 (2.2%) baseline high-risk and 2/547 (0.4%) baseline low-risk patients developed HCC. At Y8, mean (range) change from baseline in reREACH-B score was -0.1 (-1.0, 0.1). Notably, 58% and 52% of baseline high-risk patients with available data at Y5 and Y8 transitioned to low risk; only 5% and 8% shifted from low to high risk at Y5 and Y8.

Conclusions: Application of reREACH-B risk scores to data of patients receiving tenofovir-based therapy supports that few patients at low risk for HCC at treatment initiation developed HCC, while many high-risk patients decreased in risk after long-term treatment.

Keyword(s) : Hepatitis B Virus (HBV), Hepatocellular carcinoma (HCC), virology, nucleos(t)ide analogs,

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tenofovir alafenamide (TAF), tenofovir disoproxil fumarate (TDF)

1345 - Association of MASLD with Prevalence and Severity of GERD in Primary Care

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) is strongly linked to metabolic syndrome, and its association with gastroesophageal reflux disease (GERD) is increasingly reported. Despite shared risk factors, studies directly comparing GERD occurrence and severity in MASLD versus normal liver patients within a single primary care clinic setting using abdominal ultrasound remain limited. This study aimed to investigate the association between MASLD and GERD in a primary care patient cohort.

Materials and Methods: A cross-sectional study was conducted on patients from the Millennium Seoul IM Clinic in Incheon who were diagnosed with MASLD (n=23) by abdominal ultrasound or confirmed to have a normal liver (Control group, n=23). Participants completed a validated questionnaire assessing the prevalence and severity of GERD symptoms. Endoscopic findings were also retrospectively analyzed from medical records.

Results: The MASLD group exhibited a significantly higher overall prevalence of GERD symptoms (60.9% vs. 26.1%, $P=0.020$) compared to the Control group, with significant differences in typical symptoms, including heartburn ($P=0.038$) and acid regurgitation ($P=0.016$). A trend towards greater GERD symptom severity was also noted in the MASLD group ($P=0.014$).

Conclusions: MASLD, diagnosed by abdominal ultrasound in the primary care setting, may represent a significant risk factor for the development of GERD. These findings highlight the need for increased clinical vigilance regarding GERD in MASLD patients and provide foundational evidence for assessing this association in primary care.

Keyword(s) : MASLD, GERD, Primary Care

1347 - Efficacy and safety of tenofovir amibufenamide in patients with chronic hepatitis B An observational study

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Background / Hypothesis:

Tenofovir amibufenamide (TMF) is a newly recommended first-line nucleos(t)ide analogues for chronic hepatitis B (CHB) in China. However, real-world evidence on its safety and effectiveness are limited. This research sought to assess the short-term virological efficacy, biochemical response, as well as safety profile of TMF over 24 and 48 weeks in a real-world cohort of CHB individuals.

Materials and Methods:

In this single-center retrospective analysis, 119 treatment-naïve CHB patients were administered TMF at 25 mg daily. Clinical, virological, and biochemical parameters were gathered at baseline and at weeks 12, 24, and 48 for evaluation. The primary effectiveness endpoint was virological response (VR, HBV DNA <10 IU/mL) after 24 weeks of treatment. Secondary endpoints included VR at other timepoints, alanine aminotransferase (ALT) normalization, and changes in renal parameters and lipid levels.

Results:

At week 24, 68.9% (82/119) of patients achieved VR. Multivariate analysis identified lower baseline HBsAg levels and non-cirrhotic status as independent predictors for achieving VR. The ALT normalization rate was 69.5% at week 24, with lower baseline HBsAg and liver stiffness measurement (LSM) being significant associated factors. One patient achieved HBsAg seroclearance. Regarding safety, no adverse events linked to the medication were observed. There were no statistically significant alterations in serum phosphorus, estimated glomerular filtration rate (eGFR), serum creatinine, or lipid levels (triglycerides, total cholesterol, high-density lipoprotein and low-density lipoprotein) from baseline to week 48.

Conclusions:

This real-world cohort analysis showed that TMF treatment achieved effective virological suppression and high ALT normalization rates by week 24, with its efficacy being influenced by baseline HBsAg levels and liver fibrosis status. Furthermore, TMF exhibited a favorable safety profile, characterized by stable renal parameters and lipid levels over 48 weeks. These findings support TMF as a safe and effective treatment alternative in CHB management.



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Keyword(s) : tenofovir amibufenamide, chronic hepatitis B, HBsAg, functional cure

1348 - Activation of the 5 lipoxygenase in myeloid immune cells is associated with systemic inflammation in acute on chronic liver failure

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Background / Hypothesis:

Profound systemic inflammation is a hallmark of acute-on-chronic liver failure (ACLF), yet upstream inflammatory circuits remain incompletely defined. 5-Lipoxygenase (5-LOX) and its oxylipin products, including 5-hydroxyeicosatetraenoic acid (5-HETE) and leukotrienes, are emerging mediators of myeloid cell-driven inflammation. We examined whether 5-LOX activation and its metabolites link hepatic and systemic inflammation in ACLF.

Materials and Methods:

We enrolled 26 healthy controls (Ctrl), 31 patients with chronic liver disease (CLD), 33 with decompensated cirrhosis (DC) and 55 with ACLF. Bulk RNA-sequencing of whole-blood leukocytes, purified neutrophils and liver tissue, and liver single-cell RNA-seq in 3 controls, 3 DC and 6 ACLF patients were performed. Targeted oxylipin metabolomics quantified 5-LOX-derived lipid mediators in plasma. 5-LOX pathway score were calculated in deconvoluted leukocyte subsets. Intrahepatic co-localization of 5-LOX+ monocytes/macrophages and neutrophils were assessed by multiplex immunofluorescence. The efficacy of zileuton, an inhibitor of 5-LOX, was tested in a mouse model of liver failure induced by lipopolysaccharide plus D-Galactosamine. The effect of 5-HETE on macrophage polarization were assessed in THP-1-derived macrophages.

Results:

Whole-leukocyte and neutrophil transcriptome showed stepwise increases in 5-LOX related gene set (ALOX5, ALOX5AP, LTA4H, LTC4S, LTB4R) and leukotriene receptor genes with worsening disease and being highest in ACLF. GSVA scores for arachidonic acid and leukotriene pathways displayed similar trends. 5-LOX related gene set was enriched in neutrophils and monocytes/macrophages and strongly correlated with activation markers (S100A8/A9), pro-inflammatory cytokines (IL-1B, TNF, CXCL8) and ACLF severity. Plasma 5-HETE and leukotrienes were markedly

elevated in ACLF and correlated with liver injury and systemic inflammation. A significantly higher level of 5-LOX pathway activation was also seen in the liver of patients with ACLF compared to those of DC and controls, as indicated by hepatic transcriptome and validated by multiple approaches including immunohistochemistry, RT-PCR and Western blotting. The liver of ACLF patients also displayed significantly higher number of 5-LOX+CD14⁺/CD68⁺ macrophages and 5-LOX+MPO⁺ neutrophils as shown by multiplex immunofluorescence. In mice of liver failure, hepatic 5-LOX expression and levels of 5-HETE both in liver and serum were up-regulated. Oral administration of zileuton, an inhibitor of 5-LOX, reduced serum 5-HETE, hepatic 5-LOX and IL-6/IL-1 β expression, as well as liver injury markers and histological damage. In vitro, 5-HETE promoted M1-like polarization and pro-inflammatory cytokine production (IL-6, IL-1 β , TNF- α) in THP-1-derived macrophages.

Conclusions:

5-LOX activation in myeloid immune cell is tightly linked to systemic inflammation and disease severity in ACLF. 5-LOX represents a promising therapeutic target for ameliorating systemic inflammation in ACLF.

Keyword(s) : Acute-on-chronic liver failure; 5-lipoxygenase; 5-HETE; Innate immunity.

1351 - Evaluation Of Hepatitis B Recurrence In Liver Transplant Recipients In Vietnam

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Background / Hypothesis:

Hepatitis B virus (HBV) is a global public health challenge and a leading cause of end – stage liver disease requiring transplantation. However, HBV recurrence after transplantation can significantly compromise graft function and patient survival. In the context of limited healthcare resources, evaluating the recurrence rate and the current status of prophylactic strategies is essential.

Materials and Methods:

A cross-sectional descriptive study was conducted at the 108 Military Central Hospital (Vietnam) involving 126 patients who underwent LT due to HBV-related liver diseases. Data collected included pre- and post-transplant laboratory results (HBsAg, HBeAg, Anti-HBs, Anti-HBc IgG/IgM, and HBV-DNA) and information regarding prophylactic regimens (use of Hepatitis B Immunoglobulin [HBIG] and nucleos(t)ide analog [NA]).

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Results:

Male patients accounted for 89.7%, with a mean age of 54.2 ± 10.8 years. The average follow-up duration after transplantation was 34.4 ± 20.1 months. HCC was the most common indication for LT (48.4%). The HBV recurrence rate was 11.1%. Short-term HBIG prophylaxis following transplantation, in combination with long-term NA therapy, has demonstrated effectiveness in preventing HBV recurrence in this patient population. Notably, only 25.4% of patients were Anti-HBs positive, raising concerns about long-term immunological protection. The use of appropriately dosed HBIG in combination therapy, tailored according to high- and low-risk HBV recurrence groups, should be considered to optimize antibody levels in this patient population, as maintaining HBIG therapy remains a major challenge due to financial burden and supply shortages in Vietnam.

Conclusions:

Oral NA therapy remains an effective prophylactic approach for preventing HBV recurrence after LT, particularly in the context of significant challenges associated with HBIG availability in Vietnam. Strategies to enhance post-transplant Anti-HBs titers – such as selective dosing of HBIG administration – should be considered in long-term post-transplant care.

Keyword(s) : Hepatitis B virus, Liver transplantation, Recurrence, Hepatitis B immunoglobulin, Nucleos(t)ide analog, Prophylaxis.

1354 - Prognostic value of alpha fetoprotein and hepatic reserve in hepatocellular carcinoma patients receiving lenvatinib real world evidence from Dr. Mohammad Hoesin Hospital Palembang

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Background

Lenvatinib is an established first-line systemic therapy for advanced hepatocellular carcinoma (HCC). Baseline tumor biology and hepatic functional reserve — commonly assessed using alpha-fetoprotein (AFP), Child–Pugh, and ALBI scores — substantially influence treatment tolerance and early disease trajectory. Evidence describing these prognostic markers in Indonesian HCC populations remains limited. This study aimed to characterize baseline AFP status and hepatic reserve among HCC patients who received lenvatinib at Dr. Mohammad Hoesin Hospital

Palembang in 2025, and to examine the relationship between AFP elevation and other clinical or laboratory predictors. We hypothesized that AFP-positive patients constitute the majority and demonstrate poorer pretreatment profiles.

Materials and Methods

Nineteen consecutive HCC patients receiving lenvatinib between January and December 2025 were included. Extracted variables comprised Child–Pugh score, ECOG performance status, MELD score, ALBI score, and serum AFP level. Data cleaning involved conversion of comma-based decimals and exclusion of clearly non-numeric AFP or MELD entries. AFP positivity was defined as ≥ 200 ng/mL. Descriptive analyses were performed due to the absence of radiologic or survival outcomes in the dataset. MELD values were interpreted cautiously because several entries appeared in a nonstandard numerical format.

Results

Of the 19 patients analyzed, AFP values were available for 16 (84%). All evaluable patients exhibited AFP ≥ 200 ng/mL (16/16, 100%), indicating a fully AFP-positive, high-risk biological profile. Median AFP concentration was approximately 2,820 ng/mL, with extreme right-skewed distribution due to very high outliers (two patients with 80,000 ng/mL and one with 2,365,547 ng/mL). Child–Pugh scores demonstrated broad variation: 4 (n=2, 11%), 5 (n=4, 21%), 6 (n=5, 26%), 7 (n=4, 21%), and 9 (n=4, 21%). Most patients had preserved functional status (ECOG 0: 37%; ECOG 1: 53%). Mean ALBI score was -1.92 , reflecting moderate hepatic impairment across the cohort. Excluding the non-numeric entry, MELD values averaged approximately 65.7, though interpretation was limited by the atypical scale recorded in source data. Overall, patients demonstrated a combination of markedly elevated AFP and variable but frequently diminished hepatic reserve.

Conclusions

This real-world lenvatinib cohort from Dr. Mohammad Hoesin Hospital Palembang was characterized by universally elevated AFP, substantial AFP heterogeneity, and moderate hepatic dysfunction at baseline. These features collectively suggest a high-risk population in whom therapeutic response and survival may be strongly influenced by biomarker-driven tumor biology and hepatic reserve. Future studies incorporating radiologic response, adverse events, and survival outcomes are warranted to clarify the prognostic impact of AFP and liver function indices in

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Indonesian HCC patients treated with lenvatinib.

Keyword(s) : hepatocellular carcinoma, lenvatinib, alpha-fetoprotein, hepatic reserve, Child-Pugh score, ALBI score

1355 - Adipocyte derived Extracellular Vesicles Propagate Hepatic Metabolic Inflammation Remaining characters

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The progression of metabolic dysfunction-associated fatty liver disease (MAFLD) is driven by complex organ crosstalk. While adipose tissue inflammation is a known key driver, the precise mechanisms by which it instigates hepatic injury remain elusive. We hypothesized that adipocyte-derived extracellular vesicles (Ad-EVs) serve as critical "messengers" transporting pro-inflammatory and pro-fibrotic cargos from adipose tissue to the liver, thereby accelerating MAFLD pathogenesis.

We employed a translational approach. In vitro, Ad-EVs were isolated from the supernatant of TNF- α -treated 3T3-L1 adipocytes and applied to primary mouse hepatocytes and hepatic stellate cells (HSCs). In vivo, we utilized a diet-induced obesity mouse model, administering fluorescently labelled Ad-EVs to track their hepatic uptake. A prospective human cohort of 120 individuals with obesity and varying degrees of MAFLD severity (assessed by biopsy/VCTE) was enrolled. Plasma Ad-EVs were isolated, quantified by nanoparticle tracking analysis, and their miRNA cargo was profiled by sequencing.

In vitro, Ad-EVs from inflamed adipocytes were readily taken up by hepatocytes and HSCs. This triggered hepatocyte insulin resistance and apoptosis, and activated HSCs into a pro-fibrotic phenotype. In vivo, injected Ad-EVs preferentially accumulated in the liver. Mice receiving Ad-EVs from obese donors developed significantly worse hepatic steatosis, inflammation, and fibrosis compared to controls ($p < 0.01$). In the human cohort, plasma levels of Ad-EVs were significantly elevated in patients with steatohepatitis (NASH) and advanced fibrosis ($p < 0.001$). miRNA sequencing identified a specific signature, most notably a high miR-155-5p cargo in Ad-EVs, which strongly correlated with histological severity ($r = 0.67$, $p < 0.001$) and was an independent predictor of fibrosis stage in multivariable analysis.

Our findings establish adipocyte-derived extracellular vesicles as a novel and critical pathway in the liver.

adipose tissue axis, directly propagating metabolic inflammation and fibrosis in MAFLD. The identification of a specific Ad-EV miRNA signature, particularly miR-155-5p, offers a novel mechanistic insight and a promising, accessible liquid biopsy biomarker. This discovery paves the way for novel public health strategies targeting inter-organ communication to prevent and halt MAFLD progression in at-risk populations.

Keyword(s) : Extracellular Vesicles, MAFLD, Adipose-Liver Axis, miRNA, Liquid Biopsy, Metabolic Inflammation

1357 - Expression of the FCGBP Gene in Hepatocellular Carcinoma Tissues and Its Role in Prognostic Assessment

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Background / Hypothesis:

[Abstract] Objective: To investigate the expression of the Fc fragment of IgG-binding protein (FCGBP) gene in hepatocellular carcinoma (HCC) and its impact on clinical prognosis, as well as to preliminarily analyze FCGBP variations and molecular biological functions.

Materials and Methods:

Methods: FCGBP expression data and clinical information of HCC patients were obtained from the TCGA database, and the expression levels in tumor, adjacent non-tumor, and normal tissues were compared. Cox regression analysis was used to identify risk factors affecting the prognosis of HCC patients, and the Kaplan-Meier method was applied to analyze the relationship between FCGBP expression levels and prognosis in HCC patients. Log-rank test was used to examine survival differences between high and low FCGBP expression groups. cBioPortal online software was used to analyze FCGBP mutations, and a protein-protein interaction network for FCGBP was constructed using the STRING database, followed by GO and KEGG enrichment analyses to elucidate its molecular biological functions.

Results:

Results: FCGBP expression levels were higher in liver cancer tissues than in adjacent non-tumor and normal tissues ($P < 0.05$); FCGBP levels were also higher in

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hepatitis B virus (HBV)-related liver cancer compared to non-HBV-related liver cancer ($P < 0.05$). FCGBP expression levels were associated with tumor histological grade, vascular invasion, prognostic outcome, and primary tumor stage ($P < 0.05$). Primary tumor stage, tumor size, and FCGBP expression levels were independent prognostic factors for HCC patients, with higher FCGBP expression associated with poorer prognosis (HR=1.47, 95%CI: 1.04–2.08, $P < 0.05$). The mutation rate of FCGBP in HCC patients was 3.4%, with poorer prognosis observed in mutated cases. Enrichment results indicated that FCGBP mainly participates in biological functions such as cell proliferation and division, cell-matrix adhesion, and transcriptional regulation. Immune infiltration analysis showed that FCGBP is associated with the infiltration of various immune cell types.

Conclusions:

FCGBP expression is upregulated in HCC, with notably high expression specifically in HBV-related liver cancer. HCC patients with high FCGBP expression and FCGBP mutations have poorer prognosis. FCGBP may serve as a potential biomarker for poor prognosis in HCC patients.

Keyword(s) : FCGBP; hepatocellular carcinoma; hepatitis B; prognosis

1358 - CORRELATION OF INTERLEUKIN 17 SERUM LEVELS WITH DEGREE OF LIVER FIBROSIS IN CHRONIC HEPATITIS B PATIENTS AT MOHAMMAD HOESIN HOSPITAL PALEMBANG

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Background / Hypothesis:

Chronic hepatitis B (CHB) is a major global cause of progressive liver disease, and immune-mediated hepatic inflammation plays a critical role in driving fibrosis. Interleukin-17 (IL-17), a key cytokine produced by Th17 cells, has been implicated in hepatocellular inflammation, fibrogenesis, and disease progression in chronic liver disorders. However, data on the association between circulating IL-17 levels and liver stiffness in CHB patients remain limited. This study aimed to evaluate the correlation between serum IL-17 levels and the degree of liver fibrosis assessed using

transient elastography

Materials and Methods:

We conducted an analytical observational study with a cross-sectional design among patients with CHB at Dr. Mohammad Hoesin Hospital Palembang. Subjects were enrolled through consecutive non-probability sampling. Serum IL-17 levels were quantified, and liver fibrosis was measured using FibroScan®. Continuous variables were summarized as means and ranges. The correlation between IL-17 serum levels and liver stiffness values was analyzed using Spearman's rank correlation coefficient

Results:

A total of 40 patients were included, with a mean age of 37 years (range 22–60 years). The majority were male (75%). Serum IL-17 levels and liver stiffness values were both treated as numerical variables. Spearman's analysis demonstrated a statistically significant positive correlation between serum IL-17 concentration and liver stiffness ($r = 0.388$; $p = 0.013$).

Conclusions:

Serum IL-17 levels show a significant positive correlation with liver fibrosis severity in patients with chronic hepatitis B. IL-17 may serve as a useful non-invasive biomarker to complement elastography in assessing fibrotic progression, especially in patients in whom liver biopsy is not feasible.

Keyword(s) : interleukin-17; liver fibrosis; chronic hepatitis B; transient elastography; FibroScan

1360 - Gender specific clinical characteristics and care cascade of HCV in east China a multicenter retrospective study

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Background / Hypothesis:

Gender equality and women's empowerment is one of WHO's commitment. Data on the current health care status in HCV-infected women is limited. We aimed to figure out gender-specific clinical characteristics and cascade of HCV care in east China, which is crucial for

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individualized diagnosis and treatment cascade in all HCV patients.

Materials and Methods:

A multicenter, retrospective study was conducted on HCV-infected adults in east China, enrolling patients who tested positive for HCV RNA from three tertiary hospitals between Jan. 2021 and Dec. 2023. Individuals with HBV/HCV co-infection, a history of hepatic decompensation or HCC, other types of carcinomas, severe comorbidities, prior exposure to NS5B/NS5A inhibitors, or incomplete key data were excluded. The characteristics, time interval from diagnosis to treatment, treatment rates, and sustained virologic response at 12 weeks among eligible female and male patients were utilized to analyze the cascade of care.

Results:

A total of 1,052 cases were analyzed: 456 females and 596 males. HCV genotypes were identified in 976 cases: 52.5% of females had genotype 1, while males predominantly had non-1 genotypes, including 29.9% type 3 and 22.5% type 6. The severity of liver disease assessed by the fibrosis-4 score was comparable between genders. Treatment rates were nearly identical, with 96.9% of females and 95.8% of males. Notably, asymptomatic individuals not receiving antiviral therapy were largely unaware of their risk for disease progression. Among those treated, 10.2% of females and 10.3% of males experienced a delay exceeding six months from diagnosis to treatment. Furthermore, the proportion of females who delayed treatment due to specific objective factors—such as childbirth, relapse following interferon therapy, and the COVID-19 pandemic—was higher than that of males (37.8% vs. 15.3%). The incidence of premature therapy discontinuation was lower among females compared to males (2.3% vs. 10.7%). Consequently, the SVR12 rate was higher in females than in males, at 97.3% versus 89.3%. The follow-up rate of post-SVR in specialty clinics and/or health examination centers was also higher among females compared to males (89.8% vs. 79.0%).

Conclusions:

Women with HCV infection can access the same healthcare services as men in east China. The treatment willingness is similar across genders; however, women are more prone to postponing treatment due to specific objective factors. Upon treatment initiation,

women show higher rates of disease control and follow-up compared to men. These findings may indicate differences in health literacy levels, which influence disease outcomes and treatment adherence.

Keyword(s) : HCV infection; gender-specific; care cascade; clinical characteristic; treatment adherence

1363 - Combination therapy with HT 101 and HT 102 achieves rapid and progressive HBsAg loss in HBeAg negative nucleos(t)ide analogues suppressed patients 24 week results from an ongoing multicentre open label phase Ib/IIa study

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Background / Hypothesis:

HT-101 is an N-acetylgalactosamine (GalNAc)–conjugated small interfering ribonucleic acid (siRNA) targeting the S-region of the HBV genome that demonstrated durable reductions in hepatitis B surface antigen (HBsAg) in phase 1 study. HT-102 is a human monoclonal antibody against the antigenic loop of HBsAg that achieved rapid serum HBsAg decline in phase 1. We report 24-week on-treatment data from an ongoing phase Ib/IIa study in China (NCT07183306/CTR20244730) evaluating the safety and efficacy of HT-101 and HT-102 combination treatment, compared with monotherapy.

Materials and Methods:

This multicentre, open-label, partial dose-escalation study enrolled HBeAg-negative patients with stable nucleos(t)ide analogue (NA) background treatment with serum HBsAg 100–3,000 (Median 671) IU/mL. Participants were assigned to five groups: A, HT-101

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400 mg alone; B, Sequential HT-102 300mg followed by HT-101 400 mg; C, HT-101 100mg+HT-102 300mg; D, HT-101 200mg+HT-102 300mg and E, HT-101 400mg+HT-102 300mg. HT-101 and/or HT-102 were administered subcutaneously every 4 weeks for at least 24 weeks. Follow-up planned to 48 weeks post-therapy. Primary endpoint was the incidence of treatment-emergent adverse events (TEAEs). Secondary endpoints included HBsAg kinetics, virological and pharmacodynamics markers, pharmacokinetics, and immunogenicity.

Results:

All participants (Groups A-E) completed 24 weeks of treatment with HT-101 and/or HT-102 (Figure A). At Week 24, HBsAg seroclearance was achieved in 4/20 (20%) participants in group A, 0/8 (0%) in Group B, 5/8 (63%) in Group C, 8/10 (80%) in Group D and 9/10 (90%) in Group E, respectively (Figure B). Among all subjects with combination therapy (Groups C-E), at Week 24 end of treatment (EOT), 22/28 (79%) achieved HBsAg loss. EOT response rates were associated with baseline HBsAg levels: 14/14 (100%) of participants with baseline HBsAg <1,000 IU/mL achieved HBsAg loss, compared with 8/14 (57%) of those with baseline HBsAg ≥1,000 IU/mL, in which HBsAg loss showed a clear dose-dependent effect: Group C: 1/4 (25%), Group D: 2/4 (50%), and Group E: 5/6 (83%). Notably, One additional subject in each of Groups B and C attained HBsAg loss by Week 28. Treatment was well-tolerated: most TEAEs being Grade 1–2 (e.g., injection site reactions). No drug-related serious AEs, grade ≥3 events, ALT flares, or other safety signals were observed.

Conclusions:Combination therapy with HT-101 and HT-102 in CHB patients achieved rapid serum HBsAg reduction and a high rate of HBsAg loss at EOT with a favourable safety profile. These findings support continued development of HT-101 and HT-102 as a promising strategy toward achieving functional cure of HBV.

Keyword(s): HBV, siRNA, neutralizing Ab, combination

1364 - Exploring the Impact of Gut Microbiota on Itch in Cholestasis

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Background / Hypothesis:In cholestatic liver disease, pruritus is one of the most common symptoms in chronic patients, which significantly reduces the quality of life, and even has adverse consequences such as depression and suicide. The role of gut microbiota in this process cannot be ignored. The role of intestinal flora cannot be ignored. The purpose of this study was to explore the potential mechanism of intestinal flora in cholestasis-related pruritus.

Materials and Methods:Fecal samples were collected from 13 patients with cholestasis (6 patients with pruritus and 7 patients without pruritus) for metagenomic sequencing and bioinformatics analysis.

Results:Principal coordinates analysis (PCoA) showed that there was a significant difference in microbial composition between the pruritus group and the non-pruritus group. Compared with the non-pruritus group, the microbial diversity of the pruritus group was significantly increased. Further focusing on the species, we identified two groups of species with significant differences at the species level ($P < 0.05$), and selected the top 20 species with the most significant differences to perform correlation analysis with the patient 's serum total bile acid (TBA). It can be seen that coprococcus sp. Am11-30b is significantly negatively correlated with TBA, while multiple species of veillonella are significantly positively correlated with TBA . In order to clarify the mechanism pathway, we enriched and compared the differential strains of the two groups, and found that the key pathways of bile acid metabolism increased significantly in the pruritus group, which was consistent with the functional spectrum of the identified differential strains.

Conclusions:In summary, in the environment of cholestasis, the intestinal flora of some patients showed structural remodeling and increased diversity, which may be a key factor in the occurrence of pruritus in patients. The correlation between different strains and TBA showed that gut microbiota may lead to pruritus through bile acid regulation, which was supported by the enrichment of bile acid metabolic pathway in pruritus group. Future studies can further

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explore the specific mechanism of these metabolic pathways in the occurrence of pruritus and provide new biomarkers or therapeutic targets.

Keyword(s) : Cholestasis, Itch, Gut Microbiota, Metagenome

1365 - Sequential combination therapy of PD1 antibody and PEG IFN α promotes HBsAg clearance and the restoration of specific T cell response

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Background / Hypothesis:

While PD-1/PD-L1 blockade is a promising therapeutic strategy for chronic hepatitis B (CHB), its efficacy in achieving functional cure remains limited. This study aimed to evaluate the novel combination strategy—PD-1 antibody combined with PEG-IFN α therapy—to promote functional cure through restoration of HBV-specific immune responses.

Materials and Methods:

In this single-arm trial (NCT06457477), 10 CHB patients with HBsAg ≤ 200 IU/ml were received PD-1 antibody monotherapy for 12 weeks. PEG-IFN α was added if HBsAg declined <1 log from baseline, and continued until week 48. The maximum duration of PD-1 antibody treatment was 24 weeks. HBV-specific T-cell responses were assessed ex vivo using three-color FluoroSpot (IFN- γ , IL-2, TNF- α). Plasma inflammation-related proteins were quantified by Olink proximity extension assay.

Results:

A total of 10 patients were enrolled in this study. Among them, 9 patients completed the protocol, while 1 patient withdrew at week 6. Three patients achieved HBsAg loss at week 9 after PD-1 antibody monotherapy (Group A). Six were added PEG-IFN α in treatment at week 12; among them, three achieved HBsAg loss at week 24–30 (Group B), while three remained HBsAg-positive (Group C). Throughout the study period, no fatal adverse events were recorded. In Group A, ALT showed a sharply flare and was associated with HBsAg rapidly clearance or decline during PD-1 antibody monotherapy, and accompanied

by significant increased HBsAg-specific IL-2 response. In Group B and C, PEG-IFN α treatment shows a gradual and sustained ALT elevation, and was not related to HBsAg changes. In Group B, HBV-specific T-cell responses increased during PD-1 monotherapy and were maintained after PEG-IFN α addition, while in Group C, patients showed minimal improvement in HBV-specific T cell response. Plasma cytokine levels were remained stable during PD-1 monotherapy but increased (e.g., TNF, IL-6, IL-10, IL-18) following PEG-IFN α combination.

Conclusions:

PD-1 antibody added in PEG-IFN α treatment enhances HBV-specific T-cell responses and promotes HBsAg loss, providing a promising immunomodulatory strategy toward CHB functional cure.

Keyword(s) : PD-1 antibody, PEG-IFN α , HBsAg clearance, Specific T-cell response

1366 - Spatial Transcriptomics Uncovers Niche Driving Liver Fibrosis Remaining characters

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Liver fibrosis is a reversible yet critical pathological stage in chronic liver disease progression. The complex cellular ecosystem and spatial interactions that dictate the transition from inflammation to fibrosis remain poorly mapped. We hypothesized that a specific periportal inflammatory niche, characterized by unique intercellular communication, is a critical driver of progressive fibrosis and holds actionable targets for intervention.

We integrated spatial transcriptomics (10x Genomics Visium) with single-cell RNA sequencing (scRNA-seq) on liver biopsies from a prospective cohort of 60 patients with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) across the fibrosis spectrum (F0-F4). Cell-cell communication was computationally inferred and validated by multiplex immunofluorescence (mIF) and in situ hybridization. Functional validation was performed in a carbon tetrachloride (CCl₄)-induced murine fibrosis model using targeted cytokine blockade.

Spatial transcriptomics revealed a previously unrecognized "Fibrogenic Niche" enriched in portal tracts of progressive fibrosers. This niche was defined by the colocalization of a specific subset of PDGFR α + fibroblasts, *S100A9+* pro-inflammatory macrophages, and *CXCR6+* T cells. scRNA-seq deconvolution identified a distinct fibroblast

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subpopulation, termed "Niche Anchor Cells (NACs)", which highly expressed COMP and POSTN. NACs were the hub of a pro-fibrotic signaling network, primarily through a strong spatial interaction with macrophages via the *MDK-SORL1* ligand-receptor pair. In the murine model, disruption of this axis using a Midkine (MDK) neutralizing antibody significantly attenuated collagen deposition and reduced myofibroblast activation compared to controls ($p < 0.005$). In our cohort, the spatial signature of this niche strongly predicted 3-year fibrosis progression (AUC=0.91). Our study provides the first high-resolution spatial map of human liver fibrosis, identifying a pathogenic peri-portal niche that acts as an engine for disease progression. We functionally validate the *MDK-SORL1* axis as a novel and targetable driver of fibrogenesis. From a public health perspective, this spatial signature offers a powerful prognostic tool for risk stratification, while the identified targets pave the way for developing precise, niche-directed anti-fibrotic therapies to alleviate the growing burden of chronic liver diseases.

Keyword(s) : Spatial Transcriptomics, Fibrogenic Niche, Cell-Cell Communication, MASLD, Precision Medicine, Anti-fibrotic Therapy

1367 - TCM Constitution Types Predict MAFLD Risk and Pathology Remaining characters

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Traditional Chinese Medicine (TCM) constitution theory postulates that individuals with specific innate body types (e.g., Dampness-Heat, Phlegm-Dampness, Qi Deficiency) are predisposed to certain diseases. We hypothesized that these TCM constitution types can serve as novel, holistic risk indicators for Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), correlating with distinct clinical and pathological features.

A multi-center, cross-sectional study was conducted. 1,200 participants were recruited and classified into one of the nine TCM constitution types using the standardized Constitution in Chinese Medicine Questionnaire (CCMQ). All participants underwent comprehensive assessment: abdominal ultrasonography for hepatic steatosis, liver stiffness measurement (LSM) via VCTE for fibrosis, and detailed serum metabolomic profiling. Multivariate logistic regression was used to analyze the association between constitution types and MAFLD, adjusting for age, sex, and BMI.

The Phlegm-Dampness (odds ratio [OR]=3.52, 95% CI: 2.41-5.15) and Dampness-Heat (OR=2.85, 95% CI: 1.98-4.10) constitutions were identified as strong independent risk factors for MAFLD. Participants with these constitutions exhibited significantly higher LSM values ($p < 0.01$) and a greater prevalence of advanced fibrosis (LSM ≥ 10 kPa) compared to the Balanced (Gentle) constitution. Metabolomic analysis revealed distinct serum profiles: the Phlegm-Dampness type was characterized by elevated branched-chain amino acids and dysregulated bile acid metabolism, while the Dampness-Heat type showed signatures of heightened inflammation and oxidative stress. A model integrating TCM constitution with BMI demonstrated superior predictive power for MAFLD (AUC=0.84) compared to BMI alone (AUC=0.76).

This study provides the first large-scale evidence that TCM constitution types are robustly associated with MAFLD risk, severity, and specific metabolic disturbances. It validates traditional wisdom with modern scientific data, positioning TCM constitution as a valuable, low-cost tool for public health. Integrating constitutional assessment into primary care could enable early, personalized risk stratification and lifestyle interventions for MAFLD prevention, embodying a truly holistic and predictive healthcare model.

Keyword(s) : Traditional Chinese Medicine, Constitution Types, MAFLD, Risk Prediction, Precision Prevention, Metabolomics

1368 - Phlegm Dampness Constitution Alters Gut Microbiome in MAFLD

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The Phlegm-Dampness (PD) constitution in Traditional Chinese Medicine (TCM) is strongly associated with metabolic disorders, including Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD). We hypothesized that the PD constitution harbors a distinct gut microbiome profile that contributes to MAFLD pathogenesis through altered gut-liver axis signaling, providing a mechanistic link between TCM theory and modern pathophysiology.

We conducted a case-control study nested within a prospective cohort of 850 adults with MAFLD. Participants were rigorously classified into TCM constitutions using the Constitution in Chinese Medicine Questionnaire (CCMQ). Fecal samples from 150 PD and 150 Balanced (BL) constitution individuals, matched for age, sex, and BMI, underwent shotgun

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metagenomic sequencing. Serum was analyzed for metabolomics. All participants underwent vibration-controlled transient elastography (VCTE) to assess liver stiffness.

Individuals with PD constitution had significantly higher liver stiffness measurements (6.8 kPa vs 5.1 kPa, $p < 0.001$) and higher rates of significant fibrosis (LSM ≥ 8 kPa) compared to the BL group (22% vs 8%, $p < 0.01$). Metagenomic analysis revealed a robust PD-associated gut microbiome signature characterized by:

1. Enrichment of pro-inflammatory species (*Escherichia coli*, *Klebsiella pneumoniae*).

2. Depletion of beneficial, short-chain fatty acid producers (*Faecalibacterium prausnitzii*, *Akkermansia muciniphila*).

This dysbiotic PD signature was associated with: Reduced microbial gene richness and butyrate synthesis capacity.

Increased serum levels of lipopolysaccharide (LPS) and bile acids.

A machine learning model based on 15 microbial species accurately discriminated PD from BL constitution (AUC=0.82). Mediation analysis confirmed that the gut microbiome composition significantly mediated the association between PD constitution and liver fibrosis severity ($p < 0.05$).

Our findings provide the first evidence that the TCM Phlegm-Dampness constitution is characterized by a pro-inflammatory, dysbiotic gut microbiome that contributes to MAFLD severity via the gut-liver axis. This bridges TCM theory with molecular pathogenesis, identifying the gut microbiome as a potential therapeutic target for individuals with PD constitution. It paves the way for a novel, microbiota-based precision medicine approach to MAFLD management rooted in TCM principles.

Keyword(s) : Traditional Chinese Medicine, Phlegm-Dampness Constitution, Gut Microbiome, MAFLD, Gut-Liver Axis, Precision Medicine

1369 - Qi Stagnation Constitution Primes Pro inflammatory Liver Macrophages Remaining characters

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The Qi-Stagnation (QS) constitution in Traditional Chinese Medicine reflects a state of chronic stress and emotional dysregulation, clinically associated with chronic inflammatory conditions. We hypothesized that individuals with QS constitution exhibit a primed,

hyper-inflammatory state in hepatic macrophages, creating a permissive microenvironment for accelerated liver injury in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). In a multi-center cohort of 680 biopsy-proven MASLD patients, TCM constitution was assessed via the standardized CCMQ. Peripheral blood mononuclear cells (PBMCs) and liver biopsy samples were analyzed using single-cell RNA sequencing (scRNA-seq) from matched QS (n=35) and Balanced (BL, n=35) individuals. Macrophage subsets were characterized, and their inflammatory responses to LPS stimulation were assessed. Serum neuro-immune markers (e.g., substance P, NPY) were quantified.

The QS constitution was independently associated with higher NAFLD Activity Scores (NAS) and more severe lobular inflammation ($p < 0.01$). scRNA-seq of liver tissues revealed a significant expansion of a specific macrophage subcluster, termed "Inflammation-Primed Macrophages" (IPM), in QS individuals. IPMs exhibited a unique transcriptomic signature with upregulated genes in both inflammatory pathways (e.g., NFKB1, IL1B) and neurotransmitter signaling (e.g., TACR1, encoding substance P receptor). In vitro, PBMC-derived macrophages from QS subjects showed heightened IL-6 and TNF- α production upon LPS challenge. Serum levels of substance P were elevated in the QS group and positively correlated with IPM abundance in the liver ($r = 0.72$, $p < 0.001$). A neural-liver axis was suggested by the co-localization of substance P-positive nerve fibers and IPMs in portal areas.

This study provides pioneering evidence that the TCM Qi-Stagnation constitution is linked to a primed, hyper-reactive macrophage phenotype in MASLD, potentially mediated by neuro-immune crosstalk. It mechanistically connects emotional stress-related constitution with liver inflammation, offering a novel biological basis for personalized management strategies that incorporate mind-body interventions and neuro-immune modulation for MASLD patients with this specific constitution.

Keyword(s) : Qi-Stagnation Constitution, MASLD, Liver Macrophages, Neuro-Immunity, Single-Cell RNA Sequencing, Precision Medicine

1370 - Qi Stagnation Constitution Primes Pro inflammatory Liver Macrophages Remaining characters

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The Qi-Stagnation (QS) constitution in Traditional

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Chinese Medicine reflects a state of chronic stress and emotional dysregulation, clinically associated with chronic inflammatory conditions. We hypothesized that individuals with QS constitution exhibit a primed, hyper-inflammatory state in hepatic macrophages, creating a permissive microenvironment for accelerated liver injury in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD).

In a multi-center cohort of 680 biopsy-proven MASLD patients, TCM constitution was assessed via the standardized CCMQ. Peripheral blood mononuclear cells (PBMCs) and liver biopsy samples were analyzed using single-cell RNA sequencing (scRNA-seq) from matched QS (n=35) and Balanced (BL, n=35) individuals. Macrophage subsets were characterized, and their inflammatory responses to LPS stimulation were assessed. Serum neuro-immune markers (e.g., substance P, NPY) were quantified.

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Keyword(s) : Qi-Stagnation Constitution, MASLD, Liver Macrophages, Neuro-Immunity, Single-Cell RNA Sequencing, Precision Medicine

1371 - 消耗流量 707字 单词846 5000 字符 Multi omics analysis reveals key mediators of 90 day prognostic predictive value in patients with acute

on chronic liver failure

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Background/Hypothesis: Acute-on-chronic liver failure (ACLF) is a critical clinical syndrome regulated by many molecules, but the effect of gene and metabolite interaction on 90-day prognosis and predictive value are not clear. In this study, based on multi-omics integration strategy, we screened and validated the expression changes of key molecules in ACLF patients with different prognoses, providing a reference basis for clinical prediction of clinical outcomes and targeted intervention of ACLF.

Materials and Methods: In this study, gene expression Omnibus (GEO) dataset was used to select differentially expressed genes (DEGs) significantly associated with the prognosis of ACLF patients, and untargeted metabolomics was used to detect differentially expressed metabolites (DEMs) in 17 ACLF patients hospitalized in our hospital from January 2023 to December 2024 to construct DEG-DEM co-expression network, and core association pairs and regulatory pathways were identified in combination with functional enrichment analysis. Fasting venous blood was collected from 58 patients with ACLF during the same period. DEG expression was detected by ELISA, and DEM content was quantitatively analyzed by amino acid-targeted metabolomics technique. Patients were divided into survival group and death group according to their survival status at 90 days after ACLF diagnosis, and receiver operating characteristic (ROC) curves were plotted, combined with binary logistic regression to analyze the predictive value of influencing factors for the risk of 90-day death in ACLF patients.

Results: A total of 102 DEGs associated with ACLF prognosis were screened from two public datasets, and 302 serum DEMs were identified by untargeted metabolomics. Combined transcriptome and metabolome analysis confirmed that MAPK14 and L-arginine were differentially expressed in the ACLF group compared with the cirrhotic group and were co-enriched in the TNF signaling pathway. The results of clinical validation showed that 23 of 58 patients (39.66%) with ACLF died within 90 days, and the serum MAPK14 level (18.84 ± 12.87 pg/mL) and L-arginine content (22.81 ± 11.93 μ mol/L) in this group were significantly higher than those in the good prognosis group (AUC = 0.720/0.871, $P < 0.05$), and multivariate analysis confirmed that both were independent risk

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factors for 90-day death in ACLF patients.

Conclusions: MAPK14 and L-arginine are independent predictors of 90-day mortality risk in patients with ACLF, which may be involved in the inflammatory imbalance and disease progression of ACLF by synergistically regulating the function of the funeral pathway.

Keyword(s) : acute-on-chronic liver failure; transcription and metabolomics; prognosis

1372 - Survival Outcomes and Prognostic Nomogram in Primary Hepatic Angiosarcoma Insights From a Population Based Analysis

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Background: Primary hepatic angiosarcoma (PHA) represents an exceptionally rare malignant liver tumor, accounting for nearly 1% of primary hepatic cancers. Its aggressive biological behavior, vague clinical presentation, and rapid progression contribute to its extremely poor prognosis, with median survival typically under six months. Owing to the scarcity of cases, comprehensive survival-related evidence remains limited. This study aims to characterize the demographic and tumor-related features of PHA and to construct a predictive nomogram to estimate overall survival (OS) and disease-specific survival (DSS).

Materials and Methods: Data for patients diagnosed with PHA between 2000 and 2021 were retrieved from the Surveillance, Epidemiology, and End Results (SEER) database. Individuals with incomplete or missing vital-status information were excluded. Patients were stratified based on demographic characteristics, tumor features, and treatment modalities. Kaplan–Meier methods were used to assess differences in OS and DSS, while univariate and multivariate Cox analyses identified independent prognostic variables. A nomogram predicting 6-month and 1-year survival was developed from the final multivariate model.

Results: A total of 316 patients with PHA were included, with a mean age of 62.2 years. Males constituted 35% of the cohort and demonstrated significantly higher 5-year OS compared with females (11.1% vs. 3.4%, $P < .001$). In older adults, survival among patients treated

with combined surgery and chemotherapy was comparable to those receiving chemotherapy alone. However, patients who did not undergo chemotherapy had markedly worse survival than those who did (5-year OS: 3.7% vs. 9.9%, $P < .001$). Sex, age, race, and tumor grade were independently associated with OS and DSS. The constructed nomograms showed reliable discrimination, with C-index values of 0.654 for OS and 0.662 for DSS, supported by well-aligned calibration curves.

Conclusions: PHA continues to carry an extremely poor prognosis. Chemotherapy and surgery appear to offer survival advantages in selected patient groups. The prognostic nomograms developed in this study provide a practical and accurate tool for estimating individualized survival probability and may support clinical decision-making for this rare and aggressive malignancy.

Keyword(s) :

1373 - Constructing and Implementing a New Model for Viral Hepatitis Screening in Guangzhou's Primary Communities

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Guangzhou Eighth People's Hospital, Guangzhou Medical University, Center For Liver Disease, Guangzhou-China ¹ Baiyun Guangzhou Center For Disease Control And Prevention, , Guangzhou-China ²

Background / Hypothesis:

The study aimed to establish and evaluate a novel community-based screening and management model in Guangzhou, focusing on improving early detection and standardized treatment for hepatitis B (HBV) and hepatitis C (HCV). A novel grass-root community screening and management model of hepatitis B (HBV) and hepatitis C (HCV) was developed in order to improve the diagnosis and treatment rate of HBV and HCV in Guangzhou city.

Materials and Methods:

A three-tier collaborative framework [tertiary hospitals-center for disease control and prevention (CDC) -primary care clinics] implemented dual-track screening (fixed-site+mobile units) using rapid screening through HBsAg/HCV-Ab dual testing and structured surveys. Digital closed-loop management integrated screening, referral, and follow-up. Data

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were analyzed via SPSS 26. 0.

Results:

Among 30012 community-dwelling adults screened (Male:Female=1:1.68), overall positivity rate was 5.61% (1,669/30,012), peaking in the 50-59-year cohort ($\chi^2=321.04$, $P<0.001$). Disease distribution revealed 1,565 HBsAg-positive cases (5.21%) and 119 HCV-Ab-positive cases (0.40%), with significant gender disparity (7.48% vs. 4.41%, males vs. females). 363 positive patients were referred to the main hospital. Confirmed cases included 155 HBsAg carriers, 147 chronic hepatitis B patients, 24 with hepatic cirrhosis (including 4 with liver cancer), 10 past HCV infections, 21 chronic hepatitis C patients, 2 with hepatic cirrhosis due to HCV, and 4 with HBV/HCV co-infection. Pre-screening, the standardized treatment rates for HBV and HCV were 33.01% (102/309) and 51.65% (16/29), increasing post-screening to 68.61% (212/309) for HBV and 93.55% (29/31) for HCV. Using the aMAP liver cancer risk scoring system to assess 132 patients, the low-risk group accounted for 43.18%, the medium-risk group for 31.06%, and the high-risk group for 25.76%. Conclusions: The three-tier collaborative model significantly enhanced viral hepatitis diagnosis and treatment efficiency, demonstrating clinical value in liver cancer risk stratification. However, it is necessary to enhance patient compliance management and health education to solve the problem of "untreated patients who should be treated".

Keyword(s) : Viral hepatitis; Community screening; Diagnosis and treatment rate; Prevention and control model

1374 - Prognostic Influence of Histopathological Subtypes and Patient Characteristics in Hepatocellular Carcinoma A Comprehensive Population Based Evaluation

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Background / Hypothesis: Hepatocellular carcinoma (HCC) represents the predominant form of primary liver cancer, accounting for nearly 75–85% of all cases, and remains a major global health burden. Liver cancer ranks among the top causes of cancer-related mortality worldwide. Despite increasing incidence rates, limited comparative evidence exists regarding the clinical outcomes and demographic patterns associated with the different histopathological variants of HCC. This study aims to provide a detailed evaluation of the prognostic implications and clinical profiles of

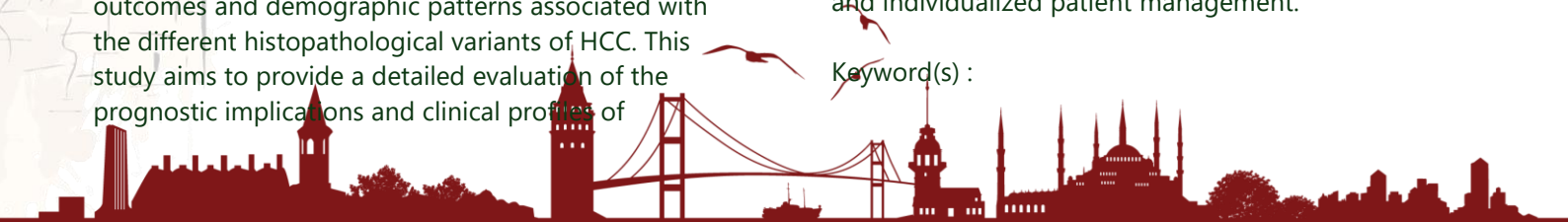
recognized HCC histologic subtypes.

Materials and Methods: Patient data were obtained from the Surveillance, Epidemiology, and End Results (SEER) database, including individuals diagnosed with HCC between 2000 and 2021. Subjects with missing vital-status data or unspecified histological classifications were excluded. Patients were categorized based on demographic factors, tumor histopathology, and treatment modalities. Kaplan–Meier survival analysis and Cox proportional hazards regression were applied to assess overall survival (OS). Independent predictors of mortality were determined using univariate and multivariate analyses.

Results: A total of 1,450 patients with confirmed HCC histological subtypes were included. Clear cell HCC emerged as the most frequently observed subtype, particularly among adult and elderly individuals, with higher representation in white male patients. Clear cell and trabecular variants were more commonly diagnosed at localized disease stages, whereas fibrolamellar HCC demonstrated a greater tendency to present at regional stages. Survival comparisons showed that the pleomorphic subtype was associated with notably poorer OS relative to fibrolamellar HCC (adjusted hazard ratio [AHR] = 4.895; 95% CI: 1.315–18.22; $p = 0.018$). Age also played a significant prognostic role, with individuals older than 60 exhibiting substantially reduced survival (AHR = 2.375; 95% CI: 1.147–4.917; $p = 0.02$). Overall, fibrolamellar and trabecular subtypes demonstrated the most favorable survival outcomes.

Conclusions: Histopathological subtype exerts a substantial influence on survival outcomes in hepatocellular carcinoma, with fibrolamellar and trabecular variants demonstrating the most favorable prognoses. These subtypes also show distinct patterns of tumor spread, underscoring the clinical value of histologic classification. Across all subtypes, age remains a strong and independent predictor of mortality, reinforcing its importance in risk stratification and individualized patient management.

Keyword(s) :



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1376 - GP73 A novel non invasive biomarker for diagnostic and prognostic of HEV ALF elderly patients

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Background / Hypothesis: Hepatitis E virus (HEV) is a significant cause of acute liver failure (ALF), with a very poor prognosis in elderly patients. Against the background of an accelerating aging population and a rising incidence of HEV in China, there is an urgent need for novel biomarkers for early risk stratification and prognostic assessment. This study aims to evaluate the prognostic value of golgi protein 73 (GP73) in elderly patients with HEV-related acute liver failure (HEV-ALF).

Materials and Methods: A total of 123 subjects were enrolled, including 40 elderly patients with acute hepatitis E (AHE), 40 with HEV-ALF, and 43 healthy elderly controls. Serum GP73 levels were measured by ELISA and analyzed for their correlation with clinical indicators. To assess the diagnostic and prognostic value of GP73, receiver operating characteristic (ROC) curves, orthogonal partial least squares-discriminant analysis (OPLS-DA), and decision curve analysis (DCA) were employed to evaluate its efficacy in diagnosing HEV-ALF and predicting 30-day mortality.

Results: Serum GP73 levels were significantly elevated in elderly patients with HEV infection compared to healthy elderly controls (135.02 vs. 22.73 ng/mL, $p < 0.001$). Furthermore, GP73 was markedly higher in the HEV-ALF group than in the AHE group (208.94 vs. 76.69 ng/mL, $p < 0.001$). For diagnosing HEV-ALF in elderly patients, GP73 achieved an AUC of 0.881 (95% CI: 0.81–0.951), with a cutoff value of 138.30 ng/mL, yielding a sensitivity of 77.5% and specificity of 82.5%. Decision curve analysis (DCA) further demonstrated the clinical utility of GP73, showing stable net benefit across a range of threshold probabilities. Correlation analysis revealed that serum GP73 levels were positively associated with key indicators of liver injury and failure, including ALT, AST, TBIL, and INR ($p < 0.05$), highlighting its relevance to disease severity. During the 30-day follow-up, non-survivors exhibited significantly higher GP73 levels than survivors (274.87

± 58.76 vs. 190.52 ± 66.26 ng/mL, $p = 0.006$). GP73 also showed strong predictive performance for mortality, with an AUC of 0.848, sensitivity of 83.3%, and specificity of 85.3%. DCA confirmed its high decision-making value in predicting 30-day mortality.

Conclusions: GP73 is a potential diagnostic and prognostic biomarker of HEV-ALF elderly patients, and its high level suggests poor prognosis, which provides a new basis for early intervention.

Keyword(s): Elderly patients with hepatitis E infection; Acute liver failure; Golgi protein 73; Biomarker; Prognosis

1379 - Synergistic effects of type 2 diabetes and alcohol consumption on all cause and liver related mortality among patients with steatotic liver disease

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Background / Hypothesis:

Type 2 diabetes (T2D) and alcohol consumption have been acknowledged as major risk factors for adverse clinical outcomes in individuals with steatotic liver disease (SLD). However, data about the synergistic effects of T2D and alcohol on all-cause and liver-related mortality in SLD remains scarce.

Materials and Methods:

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Leveraging data from the UK Biobank, we included individuals with liver steatosis diagnosed by a fatty liver index ≥ 60 . Six groups stratified by T2D state and alcohol consumption were further defined: no T2D+no/mild alcohol (no/mild alcohol intake: female: <140 g/week, male: <210 g/week), no T2D+metabolic and alcohol-associated steatotic liver disease (MetALD; with moderate alcohol intake: female: $140\sim 350$ g/week, male: $210\sim 420$ g/week), no T2D+alcohol-associated steatotic liver disease (ALD; with severe alcohol intake: female ≥ 350 g/week, male ≥ 420 g/week), T2D+no/mild alcohol, T2D+MetALD, T2D+ALD. The risks of all-cause and liver-related mortality across these groups were compared by Cox proportional hazards regression, and the population attributable fraction (PAF) and relative excess risk due to interaction (RERI) were calculated to exam the interplay between these two factors.

Results:

24,309 all-cause deaths and 1,009 liver-related deaths were recorded for 175,713 participants with SLD from the UK Biobank. The coexistence of T2D and elevated alcohol intake, particularly in case of severe alcohol consumption, was associated with substantially worse outcomes, including a $1.23\sim 1.64$ fold increased risk of all-cause mortality and $1.58\sim 7.32$ fold risk of liver-related mortality compared with no T2D+no/mild alcohol (Table 1). While combination of T2D and alcohol use accounted for a modest PAF of 6.78% for all-cause mortality, they contributed to 40.52% of liver-related mortality. Significant RERI was exclusively observed for liver-related mortality but not all-cause mortality.

Conclusions:

Co-exposure to T2D and alcohol intake substantially increases the risk of all-cause and especially liver-related mortality in patients with SLD. Their interplay confers significant disease burden, underscoring the importance of abstinence and glycemic control in the management of SLD.

Keyword(s) :

1381 - From Crisis to Control Acute HCV Triggered Acute on Chronic Liver Failure With HRS Successfully Stabilized With Early Sofosbuvir-Velpatasvir and Hemoperfusion A Case Report

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Background / Hypothesis:

Acute-on-Chronic Liver Failure (ACLF) remains a highly fatal syndrome with limited therapeutic options. In Hepatitis C virus (HCV)-related ACLF, direct-acting antiviral (DAA) therapy is conventionally delayed until clinical stabilization, fearing complications from hemodynamic instability and renal impairment. Given that ongoing viral replication drives the systemic inflammation characteristic of ACLF, we hypothesized that early initiation of Sofosbuvir-Velpatasvir (DAA) could accelerate clinical and biochemical recovery by rapidly mitigating the inflammatory burden.

Materials and Methods:

We report the case of a patient presenting with life-threatening HCV-related ACLF Grade 3 (MELD-Na 33) complicated by Hepatorenal Syndrome Type 1 (HRS Type 1), multi-organ failure, and immediate hemodialysis dependence. Sofosbuvir-Velpatasvir was initiated early. The patient received aggressive, multidisciplinary, and supportive care, including terlipressin, albumin, and a series of hemoperfusion (HP) sessions. We monitored the patient's serial laboratory parameters, including creatinine, total bilirubin, AST/ALT, and MELD-Na score, correlating trends with the timing of DAA and supportive interventions.

Results:

Following DAA initiation and intensive support, the patient achieved a rapid and sustained recovery. Key parameters demonstrated dramatic improvement: Creatinine dropped from a peak of approximately 427 micromoles per liter ($\mu\text{mol/L}$) to approximately 52 $\mu\text{mol/L}$ by Day 60, allowing successful weaning from hemodialysis. AST/ALT quickly returned to normal levels (from 1370 U/L and 523 U/L to almost normal by Day 23). The patient was successfully weaned off pressor support and survived the hospital course. The clinical outcome challenges the traditional paradigm of delaying DAAs, showing that early viral suppression, combined with aggressive multi-organ support (including HP), is feasible and associated with rapid organ recovery even in the setting of severe ACLF and HRS-1.

Conclusions:

This case demonstrates the potential role of early Sofosbuvir-Velpatasvir initiation as a supportive strategy in ACLF patients with hepatitis C, including

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those requiring hemodialysis. Early viral suppression may contribute to improved liver function and overall stabilization, although definitive evidence remains lacking. Given the paucity of data and absence of guideline recommendations, controlled studies are urgently needed to clarify the timing and safety of DAA initiation during ACLF and to determine whether early antiviral therapy should be incorporated into future management algorithms.

Keyword(s) : ACLF; Sofosbuvir–Velpatasvir; Direct-acting antivirals; Hepatitis C; Hepatorenal syndrome; Hemodialysis; Early antiviral initiation; Decompensated cirrhosis; Viral suppression; Liver failure outcomes

1382 - SARS CoV 2 Spike Protein Promotes HBV Replication via Autophagy and Enhances Inflammatory Responses in Chronic Hepatitis B Patients with Liver Dysfunction and Metabolic Disorders

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Background / Hypothesis: Autophagy plays a critical role in viral infection and immune regulation, and the SARS-CoV-2 spike (S) protein may influence the pathogenesis of chronic hepatitis B (CHB) by modulating autophagy and immune responses. This study aims to investigate the effects of the S protein on HBV replication and immune function in CHB patients, elucidate the underlying mechanisms, and provide theoretical support for the safety and application of COVID-19 vaccines in CHB patients.

Materials and Methods: This study systematically investigated the effects of the SARS-CoV-2 spike (S) protein on HBV replication and immune function in CHB patients using both in vitro cell experiments and clinical sample analyses. In vitro, HepG2.2.15 cells were engineered to express the S protein, and HBV replication and autophagy-related changes were assessed. Autophagy inhibitors were applied to further validate the underlying mechanism. In addition, peripheral blood mononuclear cells (PBMCs) isolated

from CHB patients were co-cultured with the S protein, and immune cell activation and inflammatory responses were analyzed.

Results: Expression of the S protein significantly promoted HBV replication in HepG2.2.15 cells. Treatment with autophagy inhibitors suppressed HBV replication, indicating a key role of autophagy in S protein-induced enhancement of HBV replication. In clinical samples, S protein stimulation enhanced the inflammatory responses of T cells and monocytes in PBMCs from CHB patients, particularly in those with liver dysfunction and lipid metabolic disorders.

Conclusions: Our findings demonstrate that the SARS-CoV-2 S protein promotes HBV replication through autophagy activation and enhances immune-inflammatory responses in CHB patients. Autophagy inhibitors can reverse the S protein-induced promotion of HBV replication, suggesting autophagy as a potential therapeutic target. Clinical data further indicate that the immune-activating effects of the S protein are more pronounced in CHB patients with liver dysfunction and metabolic disturbances. Targeting the S protein–autophagy interaction may offer new strategies to improve the clinical management of HBV patients with COVID-19 and reduce disease burden.

Keyword(s) : SARS-CoV-2; chronic hepatitis B; spike protein; HBV replication; autophagy; inflammatory response

1383 - Prognostic Impact of Cardiometabolic Kidney Dysfunction on Patients with Metabolic dysfunction-associated steatotic liver disease (MASLD)

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) frequently coexists with cardiometabolic and renal abnormalities. The prognostic impact of advanced cardiometabolic-kidney

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dysfunction (CKM Stage 3) among individuals with MASLD remains unclear. This study evaluated whether CKM Stage 3 is associated with increased mortality in a nationally representative U.S. cohort.

Materials and Methods:

We analyzed adults from NHANES 1999–2018 who met MASLD criteria and linked them to National Death Index mortality data. Participants were stratified into two groups: No CKM Stage 3 and CKM Stage 3. Survey-weighted analyses compared demographic, metabolic, hepatic, and renal biomarkers. Cox proportional hazards models estimated associations with all-cause and cause-specific mortality.

Results:

CKM 3 was present in 5% in the general population. This group demonstrated significantly worse metabolic and inflammatory profiles, including higher fasting glucose, insulin, triglycerides, and HOMA-IR, along with lower HDL cholesterol. Mortality risk was substantially elevated in MASLD participants with CKM Stage 3. Compared with those without CKM Stage 3, the CKM Stage 3 group had markedly higher risks of all-cause mortality (HR 2.77, 95% CI 1.82–4.21, $p < 0.001$), borderline non significant cardiovascular mortality (HR: 1.56, 95% CI 0.86 to 2.82, $p = 0.140$) but not in oncological related mortality.

Conclusions:

CKM Stage 3 more than doubles the mortality risk among U.S. adults with MASLD. These findings underscore the critical interplay between liver, cardiometabolic, and renal dysfunction and support incorporating CKM staging into MASLD risk stratification and clinical management.

Keyword(s) : Metabolic, MASLD, NAFLD, Fatty liver, Steatosis

1386 - Real World Clinical Experience with Long Term Albumin Therapy in Advanced Liver Disease A Case Based Perspective from Asia

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Background:

Long-term albumin therapy (LTA) is recognized as a potential disease-modifying strategy in patients with advanced liver disease. Recent randomized trials and real-world observational studies suggest that LTA may improve patient survival and reduce incidence of liver-related complications. However, real-world clinical practice around LTA in Asia remains heterogeneous, reflecting disparities in patient characteristics, healthcare infrastructure, and access to treatment.

Objectives: This case series evaluated the clinical rationale, therapeutic strategies and longitudinal outcomes of LTA in advanced liver disease across five tertiary centers in Asia.

Methods:

Eight cases were contributed by hepatologists from Indonesia, India and Malaysia, including patients with decompensated cirrhosis or hepatocellular carcinoma (HCC) with metastasis not suitable for liver transplantation who received LTA for ≥ 3 months together with etiological treatment for the liver disease. Demographics and clinical data were collected including liver and renal function, ascites resolution, paracentesis frequency, serum albumin levels and quality-of-life indicators. Each case was analyzed to assess therapeutic rationale, safety, and observed outcomes.

Results:

Patients presented with diverse etiologies for decompensated cirrhosis and HCC (HBV, MASLD, polycystic liver disease), complications (refractory ascites, variceal bleeding, renal dysfunction) and baseline albumin levels (< 30 g/L). LTA dosing regimens varied from fixed doses (60 g/week loading followed 20-40 g/week maintenance) to individualized titration based on clinical response and serum albumin levels. Notably, one patient with HBV received 25 g/month and still experienced clinical benefit. LTA increased serum albumin levels, reduced paracentesis frequency and improved renal and liver function in all seven cases. One patient with MASLD achieved complete

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ascites resolution and discontinued diuretics, while another with polycystic liver disease continued to require biweekly paracentesis and diuretics despite LTA, highlighting variability in LTA response. Ascites control was eased by LTA in one patient with HCC and variceal bleeding, and in three patients with HBV. Quality of life indicators was assessed in two patients with metastatic HCC showed improvements in their quality-of-life with LTA. Safety profile was favourable, with one patient experiencing two episodes of pulmonary edema, treated with diuretics.

Conclusions:

Through individual patient trajectories, this Asian case series provided real-world insights into selection criteria, dosing strategies, and clinical benefits that extend beyond conventional indications supporting the potential role of LTA in improving clinical outcomes in patients with advanced liver disease. While variability in response and treatment regimens limited generalizability of the results, the findings were aligned with clinical trial data and underscore the need of treatment individualization, standardized protocols and further prospective studies in Asian populations.

Keyword(s) : Long -term albumin, liver cirrhosis, hepatocellular carcinoma, quality of life

1388 - External Validation of a Preoperative Microvascular Invasion Risk Prediction Model Using Gadoxetate Enhanced MRI for Predicting Early Recurrence After Surgical Resection in Patients With Single Hepatocellular Carcinoma

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Background / Hypothesis:

Microvascular invasion (MVI) in hepatocellular carcinoma (HCC) is a major risk factor for early recurrence (<2 years) after curative treatment. A preoperative prediction model incorporating tumor markers and gadoxetate-enhanced MRI features has been proposed for estimating MVI risk in patients with single HCC. This study aimed to externally validate the association between the predicted MVI risk and early recurrence after surgical resection.

Materials and Methods:

This study prospectively enrolled at-risk patients with surgically resected single HCC who underwent preoperative gadoxetate-enhanced MRI between July

2019 and June 2023. A previously developed MVI risk prediction model—based on tumor markers (alpha-fetoprotein and protein induced by vitamin K absence-II) and MRI features (peritumoral arterial phase hyperenhancement and peritumoral hepatobiliary phase hypointensity)—was applied to categorize patients into low- and high-risk groups. Early recurrence rates were compared using Kaplan–Meier curves with the log-rank test.

Results:

A total of 102 patients (median age, 61.0 years; 83 men) were analyzed. Based on the model, 75 patients were classified as low-risk and 27 as high-risk for MVI. Pathologically confirmed MVI was present in 17.3% (13/75) of the low-risk group and 92.6% (25/27) of the high-risk group ($P < 0.001$). Early recurrence rates within 2 years were significantly lower in the low-risk group than in the high-risk group (12.0% vs 55.6%, $P < 0.001$).

Conclusions:

The preoperative MVI risk prediction model demonstrated reliable external validity. High predicted MVI risk was strongly associated with pathological MVI and significantly higher early recurrence after resection. This model may help guide preoperative risk assessment.

Keyword(s) : Hepatocellular carcinoma, Microvascular invasion, Tumor marker, Magnetic resonance imaging, Early recurrence

1390 - A study on clinical factors influencing the recurrence of hepatitis B surface antigen following clearance after long term nucleoside analogue therapy in patients with hepatitis B virus infection

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Background / Hypothesis:

In the era of pursuing clinical cure, while some HBV-infected patients achieve HBsAg clearance following effective antiviral therapy, a small proportion still experience reversion to positivity after seroconversion. However, current research findings on HBsAg reappearance have yet to reach a consensus. This study aims to investigate factors associated with the

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recurrence of hepatitis B surface antigen (HBsAg) after clearance in patients with hepatitis B virus (HBV) infection undergoing long-term nucleos(t)ide analogue (NUC) therapy, with a view to reducing the risk of HBsAg recrudescence.

Materials and Methods:

This single-centre, retrospective case-control study followed patients from the longitudinal cohort of HBV-infected individuals at the First Affiliated Hospital of Guangxi Medical University between January 2002 and October 2025. A total of 139 patients were enrolled and divided into an NUC monotherapy group and a NUC plus interferon (IFN) combination therapy group. Cox regression models and Kaplan-Meier analyses were employed to assess the association between clinical factors and HBsAg reversion.

Results:

Among 4219 patients with HBV infection treated with NUCs, the overall HBsAg seroconversion rate was 3.65%. The HBsAg seroconversion rate in the NUC plus IFN group was significantly higher than in the NUC monotherapy group ($P < 0.001$). Negative factors for HBsAg reactivation included first-line NUC regimens, NUC treatment duration exceeding 9 years, NUC consolidation therapy lasting over 1 year post-HBsAg clearance, and post-HBsAg clearance HBsAb levels not below 30 IU/mL. For the NUC plus interferon group, extended consolidation therapy (NUC > 1 year or IFN > 6 months) significantly reduced reactivation risk.

Conclusions:

For patients treated with NUC, first-line NUC regimens, extended treatment duration, and thorough consolidation therapy following seroconversion are critical factors in preventing HBsAg reappearance after clearance. Patients receiving NUC combined with IFN exhibit higher HBsAg seroconversion rates than those treated with NUC alone. However, inadequate duration of IFN or NUC consolidation therapy post-seroconversion may still result in a relatively high rate of HBsAg recurrence.

Keyword(s) : Hepatitis B surface antigen clearance; Nucleos(t)ide analogue; Interferon; Recurrence

**1391 - Integrated Multi omics Liquid Biopsy
Advancing Early Detection and Precision
Management of HCC**

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality worldwide, often diagnosed at late stages due to the limited sensitivity and specificity of current surveillance methods such as ultrasound and alpha-fetoprotein (AFP) testing. Liquid biopsy, which non-invasively analyzes circulating biomarkers, offers a promising alternative. However, single-omics approaches often lack the robustness required for early detection. Multi-omics integration presents a transformative strategy to enhance diagnostic accuracy and enable precision oncology in HCC.

Materials and Methods:

This review synthesizes recent advances in multi-omics liquid biopsy for HCC, incorporating genomic (e.g., TERT, TP53 mutations), epigenomic (e.g., RASSF1A methylation), proteomic/glycomic (e.g., AFP-L3, GP73, exosomal glycans), fragmentomic (cfDNA fragmentation patterns), and cellular biomarkers (CTCs, EVs). Cross-omics integration strategies—such as cSMART, HIFI, and ASDAm-H1—were evaluated alongside emerging technologies including nanopore sequencing, SERS-based single-exosome analysis, and machine learning models (e.g., XAI, GNNs, Bayesian Networks).

Results:

The integration of multi-omics biomarkers has demonstrated transformative diagnostic performance for early HCC. Cross-omics models consistently outperformed single-modality approaches: combined genomic-epigenomic assays achieved sensitivities of 80–90% for early-stage detection, while the fragmentomic-epigenomic HIFI model distinguished HCC from cirrhosis with exceptional accuracy (AUC 0.996, sensitivity 95.42%, specificity 97.83%). Integrated protein-epigenetic panels, such as the ASDAm-H1 index, reached an AUC of 0.96, and longitudinal application of multi-parametric scores like GALAD significantly improved sensitivity to 69.2% for early HCC compared to single measurements (53.8%). Fragmentomics-based models enabled early screening with sensitivities exceeding 96%, and metabolomic

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panels detected preclinical HCC up to one year before clinical diagnosis with 80% sensitivity. Beyond detection, multi-omics liquid biopsy proved vital for precision management: ctDNA mutation profiling enabled dynamic monitoring of targeted therapy response, PD-L1⁺ CTCs and cfDNA copy number variations predicted outcomes to immune checkpoint inhibitors, and machine learning-driven deconvolution of single-exosome SERS fingerprints or cfDNA methylation data provided unprecedented molecular resolution. These approaches not only identified AFP-negative HCC cases but also facilitated early recurrence prediction and molecular subtyping, effectively transitioning liquid biopsy from a passive detection tool to an active component in the dynamic, personalized management of HCC.

Conclusions:

Multi-omics liquid biopsy represents a paradigm shift in HCC management, enabling high-accuracy early detection and dynamic monitoring of disease progression and treatment response. Despite challenges in data integration, standardization, and clinical validation, future directions—including explainable AI, single-cell multi-omics, and closed-loop precision oncology systems—hold great promise. With continued interdisciplinary collaboration and standardization efforts, multi-omics liquid biopsy is poised to become the cornerstone of next-generation HCC care.

Keyword(s) : Hepatocellular carcinoma, Liquid biopsy, Multi-omics integration, Early diagnosis, Clinical translation

1392 - Feasibility of achieving a tumor and drug free state through conversion therapy followed by curative hepatectomy for patients with initially unresectable hepatocellular carcinoma

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Background / Hypothesis:

Whether initially unresectable hepatocellular carcinoma (HCC) patients who receive curative resection after conversion therapy can maintain long-term tumor-free survival in a drug-free state after postoperative drug withdrawal is unclear. This study aimed to compare the effectiveness of adjuvant PD-1 inhibitors versus a drug-free state in these patients.

Materials and Methods:

Data were retrospectively analyzed for patients with initially unresectable HCC who underwent curative hepatectomy after conversion therapy at 12 medical centers in China between January 2019 and June 2023. Tumor-free survival and overall survival were compared between patients who received PD-1 inhibitors and patients in a drug-free state after hepatectomy.

Results:

Relative to the 323 patients in a drug-free state, the 90 who received PD-1 inhibitors showed comparable tumor-free survival (HR 0.77, 95%CI 0.51–1.17) and overall survival (HR 0.59, 95%CI 0.33–1.04). Similar results were obtained for subsets of patients matched to each other through propensity scoring, for the subset of 112 patients who achieved pathological complete response after resection, and for the entire cohort in 12-month landmark survival analysis. An adjuvant PD-1 inhibitor regime lasting at least 6 months led to similar survival as shorter regimes, whether in terms of tumor-free survival (HR 0.89, 95% CI 0.31–2.35) or overall survival (HR 0.92, 95% CI 0.20–4.32). No treatment-related deaths occurred in the entire cohort.

Conclusions: Adjuvant PD-1 inhibitors or in combination with tyrosine kinase inhibitors may not provide survival benefit over a drug-free state to patients with initially unresectable HCC who undergo curative hepatectomy after conversion therapy.

Keyword(s) : hepatocellular carcinoma, conversion therapy, PD-1 inhibitor, adjuvant therapy

1394 - Clinical landscape Mutation mapping Quality of life and Treatment outcomes in Adolescent and Adult Wilsons disease patients in a South Indian cohort.

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Background / Hypothesis:

Wilson's Disease has a perplexing and an unparalleled clinical course presenting as a diagnostic and therapeutic challenge. Inherited in an autosomal recessive manner, copper deposits usually affect liver, brain, eyes, bones amongst other organ systems, having a diverse spectrum and impact on quality of life, morbidity and mortality

Materials and Methods:

This is an ambispective; single centre study done from January 2020 to October 2025 incorporating patients above 13 years of age. The patients were further divided into three groups based on their age. Group A (13-18 years), Group B (18-25 years), Group C (> 25 years)

Quality of life was assessed by SF 36 questionnaire. Genetic analysis was done by Next Generation Sequencing. Patients were followed for changes in MELD and CTP scores, and monitoring side effects of D penicillamine therapy and disease progression

Results:

The study involved 82 cases over 13 years of age encompassing 39 females and 43 males.

Clinical Landscape:

Patients were further divided into Decompensated cirrhosis, Compensated cirrhosis, Acute liver Failure/ACLF, Neuro hepatic Wilsons, and Asymptomatic. 58.3% patients had history of consanguinity. Atypical associations included retinitis pigmentosa (since birth, n=1) sick sinus syndrome requiring pacemaker (n=1) pituitary hypoplasia (n=1) and synovial osteochondromatosis (n=1). 6 patients had successful pregnancies, 2 had recurrent abortions and 2 patients had primary amenorrhoea.

Mutation Mapping:

45.12% of patients underwent genetic analysis. Mutations were divided into Missense variants, Stop Gained variants, Frameshift variants, Synonymous variants amongst others. Most of the patients had homozygous mutations followed by heterozygous mutations. One patient had two heterozygous mutations suggesting biallelic inheritance. The most common variants included chr13:g.52511412C>T followed by chr13:g.52548789del.

Quality of life:

60.9 % patients attempted the SF36 questionnaire to analyse the quality of life. 53.2% experienced better health than prior, 26.8% revealed being worn out

Additional information revealed 54% had completed formal education and 62.8% of patients were employed, while 2 of them experienced bullying at their workplace.

Treatment outcomes:

Out of patients receiving D penicillamine therapy, 4 patients developed worsening of neurological symptoms leading to stopping the drug. One patient developed Steven-Johnson Syndrome. After one year of therapy 14.6% patients had decrease in MELD score. Overall, 4 patients underwent liver transplantation (LDLT=3; DDLT=1) and 7 patients succumbed to the disease

Conclusion:

There is insufficient data on Wilson's disease and its behaviour in adolescence and early adulthood. This paper explores the multifaceted issues and unique challenges in these formative years, seldom addressed in prior studies.

Keyword(s) : Wilson Disease, Mutation mapping, Quality of Life

1395 - Four Year Management of Unresectable Hepatocellular Carcinoma Sustained Tumor Control and Cumulative Multi Organ Toxicities

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Background: Patients with unresectable Hepatocellular Carcinoma (HCC) undergoing long-term interventional therapy combined with systemic antitumor treatment may achieve effective tumor control, yet are at risk of developing therapy-related multi-organ injury during the course of treatment.

Methods: A 59-year-old male was hospitalized four years ago due to epigastric pain. Workup revealed mildly elevated ALT and AST, positive HBsAg, HBV-DNA 1.86×10^5 copies/ml, and AFP 308.10 ng/ml. Contrast-enhanced abdominal CT demonstrated a large right hepatic lobe mass measuring 121 mm × 83 mm, with suspected invasion of the right portal vein and right hepatic vein. The clinical diagnosis was HCC, BCLC stage C. The patient subsequently began long-term treatment involving interventional therapy combined with systemic antitumor therapy: Entecavir 0.5 mg po qd, Lenvatinib 8 mg po qd, Tislelizumab 200 mg ivgtt q3w, along with three sessions of interventional therapy, one session of radiofrequency ablation, one session of Yttrium-90 radioembolization,

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among other treatments.

Results: After four years of antitumor therapy, the patient's HBV-DNA became undetectable, and AFP decreased to 19.96 ng/ml. Follow-up contrast-enhanced abdominal CT showed no increase in the size of the primary tumor and no evidence of intra- or extrahepatic metastasis. However, during this 4-year treatment period, the patient sequentially developed damage to multiple organs and systems, including cardiovascular, gastrointestinal, skin/mucosa, renal, and endocrine systems. Over 3 years ago, the patient developed hypertension, managed long-term with oral antihypertensive agents with adequate control. Also over 3 years ago, recurrent diarrhea began. Endoscopy revealed telangiectasia in the jejunum and colon. 16S gut microbiota testing indicated dysbiosis. Symptoms improved with treatment involving Bifidobacterium and Glutamine. Nine months ago, skin lesions developed, diagnosed by dermatology as bullous pemphigoid, which improved with Methylprednisolone treatment. Over 2 years ago, renal dysfunction was identified. A renal biopsy performed by nephrology 7 months ago indicated IgA Nephropathy - Grade III, with Lenvatinib-induced renal damage not excluded. The condition improved with Methylprednisolone and other therapies. Two months ago, elevated blood glucose, abnormal thyroid function, and low cortisol rhythm were detected and are currently under monitoring.

Conclusions: Interventional therapy combined with systemic antitumor treatment can effectively control the progression of unresectable HCC. However, therapy-related organ injuries occurring during treatment may impede the sequential continuation of antitumor therapy, potentially leading to reduced patient life expectancy. Proactive and effective management of adverse events is crucial to mitigate their severity, achieving an optimal balance between tumor control and organ protection.

Keyword(s) : Hepatocellular Carcinoma, Immune Checkpoint Inhibitors, Lenvatinib

1397 - Ammonia Seeking Zinc Micromotors for Active Ammonia Scavenging in Hepatic Encephalopathy

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Background / Hypothesis:

Hepatic fibrosis, characterized by progressive hepatocyte injury, leads to the systemic accumulation of blood ammonia. This hyperammonemia not only aggravates underlying liver pathology but also crosses the blood-brain barrier, precipitating the neuropsychiatric complications of hepatic encephalopathy. Consequently, the development of targeted therapeutic platforms for the specific clearance of ammonia represents a significant and meaningful pursuit in managing this disease cascade.

Materials and Methods:

This work presents the design and fabrication of a biocompatible zinc (Zn)-based micromotor as a novel platform for active ammonia chemotaxis. The micromotor's propulsion is driven by a water-splitting reaction, generating motive force in an aqueous biological environment. Simultaneously, the dissolution of Zn creates a self-generated Zn^{2+} ion gradient. We demonstrate that this system exhibits robust directional movement towards a source of ammonia ($\text{NH}_3 \cdot \text{H}_2\text{O}$). This chemotactic behavior is governed by a coordination reaction between Zn^{2+} and ammonia molecules, leading to the formation of complex ions such as $\text{Zn}(\text{NH}_3)_1^+$ and $\text{Zn}(\text{NH}_3)_2^+$.

Results:

The coordination-based chemotaxis mechanism proved to be a generalizable strategy for guiding micromotor movement. In an in vivo model, these micromotors displayed a biomimetic collective behavior, enabling them to navigate precisely within the biological milieu. This resulted in a significant reduction of intrahepatic ammonia levels, thereby contributing to the reshaping of the pathological microenvironment. The entire operational mechanism functions in a green and zero-waste manner, enhancing its biocompatibility and facilitating its integration into the domain of sophisticated biological regulation.

Conclusions:

We have successfully established an environment-adaptive micromotor platform that effectively targets and mitigates hyperammonemia at its source. This technology holds considerable promise for the targeted treatment of hepatic fibrosis and associated hepatic encephalopathy. The underlying principles of this strategy, leveraging specific chemical reactions for targeted therapy, are expected to provide valuable

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inspiration for future advancements in personalized and precision medicine.

Keyword(s) : Ammonia, Zinc, Micromotors, Hepatic Encephalopathy

1398 - A Study to assess the relationship of Frailty and Myosteatosis in Cirrhotic patients

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Background / Hypothesis:

Frailty and myosteatosis are emerging as critical, yet underrecognized, predictors of poor outcomes in cirrhosis. Although sarcopenia has been extensively studied, the relationship between muscle fat infiltration (myosteatosis) and clinically measurable frailty remains insufficiently defined, especially using objective MRI-based metrics. Identifying high-risk phenotypes is essential as cirrhotic patients increasingly present with metabolic comorbidities such as diabetes, hypertension, and MASLD.

Aim: To evaluate the prevalence of frailty among cirrhotic patients with MRI-detected myosteatosis and to define a muscle PDFF cut-off predictive of frailty. Secondary goals were to identify independent and shared risk factors for frailty and myosteatosis

Materials and Methods:

In this hospital-based observational analytical study, 100 adult cirrhotic patients underwent comprehensive evaluation. Frailty was assessed using the Liver Frailty Index (LFI); MRI-derived proton density fat fraction (PDFF) at L1–L3 quantified myosteatosis. Demographic variables, comorbidities, Child-Pugh class, MELD-Na, and laboratory parameters were analyzed using chi-square tests, ANOVA, and ROC curves.

Results:: Frailty was strikingly common: 33% were frail and 55% pre-frail, highlighting profound functional impairment even in a mixed inpatient cohort. Muscle fat fraction averaged $15.97 \pm 7.51\%$. ROC analysis identified PDFF $\geq 15.44\%$ as a clinically meaningful threshold predicting frailty (AUC=0.748; 75.8% specificity, 61.8% sensitivity).

Age >60 years increased frailty risk 4.7-fold. Diabetes

($p=0.0001$) and hypertension ($p=0.047$) were strong determinants of both frailty and myosteatosis. NASH/MASLD emerged as the dominant etiology (54%) and conferred significantly higher vulnerability to both conditions compared with alcohol-related disease. Frailty severity correlated with Child-Pugh class ($p=0.0001$) and PT/INR ($p=0.017$), whereas myosteatosis was highest in Child-Pugh C ($p=0.007$). MELD did not correlate with myosteatosis, suggesting muscle quality deteriorates independently of conventional liver severity scores.

Conclusions:

Frailty and myosteatosis are highly prevalent, metabolically driven, and synergistic risk states in cirrhosis. A muscle PDFF cutoff of 15.44% provides an objective, MRI-based biomarker to identify patients at greatest functional decline. These findings underscore the urgent need to integrate routine muscle-quality assessment into cirrhosis evaluation—especially in MASLD and metabolic phenotype patients. Early targeted interventions addressing muscle fat infiltration may significantly alter the clinical trajectory and improve transplant readiness.

Keyword(s) :

1400 - The Silent Risk of Steroid Therapy A Case of HBV Reactivation

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Background / Hypothesis:

Hepatitis B virus (HBV) is a chronic viral infection that causes significant morbidity and mortality worldwide. Immunosuppressive or immunomodulatory agents—particularly corticosteroids—pose a risk for HBV reactivation. In HBsAg-positive individuals, this may result in accelerated viral replication and clinical deterioration, potentially progressing to fulminant hepatitis. Despite this risk, short-term steroid therapy is often considered low risk, leading to the omission of antiviral prophylaxis. Here, we present a case of HBV reactivation following short-term systemic corticosteroid use.

Case Summary

A 50-year-old male with known chronic hepatitis B infection was under observation without antiviral therapy. His viral serology revealed HBsAg positivity, HBeAg negativity, anti-HBe positivity, and HBV DNA of

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6,810 IU/ml. At the three-month follow-up, HBV DNA had increased to 21,500 IU/ml—indicating a ~1 log rise. Upon detailed history-taking, it was discovered that the patient had been taking dexamethasone 4 mg/day for granulomatous mastitis over the past two months. This dose is equivalent to >20 mg of prednisolone. Given the patient's HBsAg-positive status, the therapy represented a high reactivation risk. The clinical picture was consistent with HBV reactivation, and antiviral treatment was initiated.

Discussion

In HBsAg-positive individuals, immunosuppressive treatment—especially moderate to high-dose systemic corticosteroids—is a well-known risk factor for HBV reactivation. Steroids facilitate reactivation by both directly enhancing viral replication and suppressing host immune response. In this case, only two months of dexamethasone 4 mg/day (equivalent to >20 mg prednisolone) led to a ~1 log increase in HBV DNA, indicating reactivation. This underscores that short-term corticosteroid therapy at such doses is not safe in HBsAg-positive patients. The lack of antiviral prophylaxis prior to steroid initiation represents a significant clinical oversight. Although such cases are rarely reported, this case emphasizes the need for HBV reactivation risk assessment and consideration of antiviral prophylaxis, regardless of steroid dose or duration.

Keyword(s) : Immunosuppressive therapy, Hepatitis B reactivation, Corticosteroid use

1402 - Safety Tolerability Immunogenicity and Antiviral Efficacy of GS 2829 and GS 6779 a Novel Arenaviral Vectored Therapeutic Hepatitis B Vaccine Phase 1b Study Results From Virally Suppressed Patients With Chronic Hepatitis B

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Background / Hypothesis: Therapeutic vaccines, such as nonreplicating arenaviral vectors GS-2829 and GS-6779, are a promising approach to achieve durable immune response and functional cure for chronic hepatitis B (CHB) infection. GS-2829 and GS-6779 were engineered to express hepatitis B surface antigen (HBsAg) and core antigen (HBcAg), and highly conserved polymerase reverse transcriptase and ribonuclease H domains. We evaluated the safety, tolerability, immunogenicity, and antiviral efficacy of GS-2829 and GS-6779 in virally suppressed patients with CHB.

Materials and Methods: In this multicenter, randomized (4:1), blinded, placebo-controlled Phase 1b study, alternating intramuscular administrations of GS-2829 or GS-6779 every 4 weeks (ie, 1 cycle = GS-2829 at week 0; GS-6779 at week 4) were evaluated: cohort 5, 2 cycles of low doses (2 × low); cohort 6, 2 cycles of high doses (2 × high); cohort 7, 3 cycles of high doses (3 × high). Safety and tolerability were monitored via treatment-emergent adverse events (AEs), serious AEs, and laboratory abnormalities. Viral efficacy included HBsAg, hepatitis B e antigen (HBeAg), and hepatitis B core-related antigen (HBcrAg). Immunogenicity was evaluated by interferon-gamma ELISpot analysis of hepatitis B virus (HBV)-specific T cells.

Results: Thirty patients (n = 10 per cohort) were enrolled; median age was 51 years, 63% were male, 100% were Asian, 7% had prior interferon use, and genotype B was most common (43%). At baseline, median (minimum, maximum) HBsAg levels for patients in the 2 × low, 2 × high, and 3 × high cohorts who received the vaccine were 1579 (1, 3737), 863 (1, 3725), and 401 (25, 2260) IU/mL, respectively; 0/8, 7/8 (88%), and 4/8 (50%) patients were HBeAg positive. Among 11 HBeAg-positive patients at baseline who received the vaccine, 5 (45%) achieved HBeAg loss. Quantitative declines in HBeAg and HBcrAg were observed in additional HBeAg-positive patients. In 25% (4/16) of patients in high-dose cohorts, HBsAg declined by ~0.5 log₁₀ IU/mL with mild (Grade ≤1) alanine aminotransferase increases. Patients on the

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vaccine showed induced HBV-specific T-cell responses—mainly against HBcAg and polymerase—and 94% of patients in high-dose cohorts had ≥ 3 -fold increases from baseline in T cells for ≥ 1 HBV antigen. All AEs were Grade ≤ 2 and nonserious; none led to discontinuation of study drugs.

Conclusions: Alternating doses of GS-2829 and GS-6779 were safe and well tolerated in virally suppressed patients with CHB. These safety, virology, and immunogenicity data support further investigation of GS-2829 and GS-6779 as CHB treatment.

Keyword(s) : Hepatitis B virus (HBV), virology, HBV cure, clinical trial, therapeutic vaccine

1403 - Developing Blood based Lipid Biomarker for Hepatocellular Carcinoma Risk Prediction in Non cirrhotic MAFLD Patients

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Background / Hypothesis:

The global rise in obesity and type 2 diabetes has positioned metabolic-associated fatty liver disease (MAFLD) as a major risk factor for hepatocellular carcinoma (HCC). While MAFLD contributes to nearly half of all HCC cases, approximately 12% occur in the absence of other liver diseases. Although advanced fibrosis and cirrhosis traditionally guide HCC surveillance, emerging evidence shows that nearly 40% of MAFLD-related HCC arises without cirrhosis, often at early stages and outside routine screening pathways. This highlights a need for early-stage risk prediction tools. Liver plays a central role in synthesising, processing and sorting fats throughout the body. Thus, it is noteworthy to explore the lipid reprogramming in MAFLD and its associated HCC.

This study uses a multi-omic approach to (a) investigate the lipid profiles of non-cirrhotic MAFLD-HCC (ncMAFLD-HCC) and cirrhotic MAFLD-HCC (cMAFLD-HCC) to understand the lipid reprogramming in HCC development, and (b) identify early detection/risk prediction biomarkers for ncMAFLD-

HCC.

Materials and Methods:

Liver biopsies and plasma samples from 221 individuals (140 MAFLD, 66 cMAFLD-HCC, and 15 ncMAFLD-HCC patients) were used in the study. Plasma lipid profiles were performed on 209 samples and 22 liver biopsies. RNA and ATAC sequencing were performed on 18 and 9 liver biopsies respectively. Lipids were extracted and analysed using liquid chromatography-tandem mass spectrometry (LC-MS/MS). Principal component analysis, multivariate statistics, and pathway enrichment analysis were performed using Metaboanalyst v6.0 and R studio.

Results:

Exploratory analysis of lipid profiles demonstrated clear separation among MAFLD, ncMAFLD-HCC and cMAFLD-HCC in both plasma and liver tissue samples. More than 100 lipid species, belonging to diacylglycerols, sphingomyelins, triglycerides, dihydroceramides, and linoleic acid derivatives, were differentially expressed in ncMAFLD-HCC as compared to the MAFLD and cMAFLD-HCC cohort. Logistic regression model with 10-fold cross-validation using a panel of five lipid species achieved 91% and 80% sensitivity and 90% and 93% specificity for panel 1 and panel 2, respectively.

Enrichment analysis linked these lipids to glycerolipid metabolism, G-protein signaling, MAPK signaling, and EGFR tyrosine kinase inhibitor resistance. Additionally, 54 lipid-metabolism-related genes were dysregulated in ncMAFLD-HCC, with enrichment in sphingolipid and glycerophospholipid pathways in liver tissue.

Conclusions:

ncMAFLD-HCC exhibits a distinct lipidomic signature, reflecting profound metabolic dysregulation. These findings are under validation using a bigger cohort and offer a foundation for developing lipid-based biomarkers for HCC risk prediction in non-cirrhotic MAFLD patients. Furthermore, enrichment analysis provide insight into HCC pathogenesis in ncMAFLD-HCC and these pathways are currently studied for their potential for therapeutic targets.

Keyword(s) : Fatty liver disease, MAFLD, HCC, non-cirrhotic HCC



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1404 - A Model for Prediction Hepatic Nodules Development to Hepatocellular Carcinoma in Patients with HBV Related Cirrhosis on Antiviral Therapy

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Background / Hypothesis:

Patients with hepatitis B virus (HBV)-related cirrhosis receiving nucleos(t)ide analogues (NAs) antiviral therapy remain at substantial risk of hepatocellular carcinoma (HCC). However, the surveillance programs for early HCC from hepatic nodules development in patients with HBV-related cirrhosis on antiviral therapy were still not well established. We aimed to develop and validate a nomogram incorporating ultrasound-detected hepatic nodules and routine clinical variables for HCC risk prediction in this population.

Materials and Methods:

The retrospective-prospective real-world cohort included 1,209 treatment-naïve patients with HBV-related cirrhosis who received NAs antiviral therapy for more than 1 year. Patients were randomly assigned to derivation (n = 846) and validation cohorts (n = 363) in a 7:3 ratio. A nomogram model for predicting hepatic nodules development to HCC was developed using multivariable Cox regression models. The predictive performance was assessed using time-dependent receiver operating characteristic (ROC) curve analysis and decision curve analysis. Patients were further stratified into three risk groups based on the total risk scores calculated from the nomogram, and the cumulative incidence of HCC was evaluated using the Kaplan-Meier method. The nomogram model's performance was validated and compared with aMAP, mPAGE-B and REAL-B. Additionally, we conducted a subgroup analysis to evaluate the model's performance in predicting early-stage HCC (Barcelona Clinic Liver Cancer 0-A stage).

Results:

Overall, 323 patients (26.7%) developed HCC during a median follow-up of 7 years, among whom 64.3% diagnosed at early stage. Age, hypertension, decompensated cirrhosis, hepatic nodules and duration of antiviral therapy were independent predictors of HCC, and these five variables were used to develop the prediction model. The nomogram achieved time-dependent AUCs of 0.729-0.762 in the

derivation cohort and 0.742-0.756 in the validation cohort for predicting 1-, 3-, and 5-year HCC risk, significantly outperforming existing models (derivation cohort: aMAP: 0.555-0.598, REAL-B: 0.588-0.636, mPAGE-B: 0.589-0.605; validation cohort: aMAP: 0.588-0.646, REAL-B: 0.601-0.630, mPAGE-B: 0.625-0.665). Furthermore, the nomogram stratified patients into three risk groups with significantly different HCC and early-stage HCC incidences. The 3-year cumulative HCC incidences in the low-, medium-, and high-risk subgroups were 5.2%, 15.2%, and 36.3%, respectively, in the derivation cohort. And the 3-year cumulative incidences of early-stage HCC were 5.0%, 11.8%, and 22.9% across risk groups.

Conclusions:

This nomogram incorporating hepatic nodules demonstrated good discrimination and calibration for HCC risk prediction in patients with HBV-related cirrhosis receiving antiviral therapy, enabling individualized surveillance strategies.

Keyword(s) : Hepatocellular carcinoma, Hepatitis B virus, Cirrhosis, Hepatic nodules; Antiviral therapy, Risk prediction model

1405 - Impact of Smoking on the Longitudinal Progression of Liver Fibrosis

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Background / Hypothesis:

The adverse effects of smoking on liver-related outcomes have been suggested, yet evidence regarding its influence on fibrosis progression is limited. No prior study has thoroughly investigated the temporal relationship between smoking exposure and changes in liver fibrosis indices. We hypothesized that smoking accelerates the progression of liver fibrosis, reflected by longitudinal changes in FIB-4 scores.

Materials and Methods:

We analyzed 46,270 adults who underwent at least two health screening examinations at tertiary hospital. Smoking status was categorized as non-smoker, current smoker, or ex-smoker. Among ever-smokers, subjects were further grouped by cumulative exposure: low (<8 pack-years), intermediate (8–20 pack-years), and heavy (>20 pack-years). Fibrosis progression was assessed using repeated FIB-4 measurements. Linear mixed models (LMMs) were used to evaluate the

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temporal association between smoking status and FIB-4 change, adjusting for sex and weekly alcohol intake.

Results:

After adjustment for confounders, mean FIB-4 scores were significantly higher in current smokers ($\beta \pm \text{SE}$) and ex-smokers ($\beta \pm \text{SE}$) compared with non-smokers (mean $\pm \text{SE}$). In the linear mixed model, both current smoker ($\beta = 0.04565$, $\text{SE} = 0.00844$, $p < 0.0001$) and ex-smoker ($\beta = 0.07067$, $\text{SE} = 0.01976$, $p < 0.0001$) showed a significant increase in FIB-4 compared with non-smoker (mean = 1.1660, $\text{SE} 0.0143$, $p < 0.0001$). Pairwise comparisons showed higher FIB-4 progression in both current and former smokers relative to non-smokers, with no significant difference between current and former smokers.

Among ever-smokers, intermediate ($\beta = 0.02362$, $\text{SE} = 0.01604$, $p = 0.1410$) and heavy smoking ($\beta = 0.0008$, $\text{SE} = 0.00003$, $p = 0.9440$) groups showed slightly higher FIB-4 values than the low smoking group (mean = 1.2930, $\text{SE} = 0.01567$, $p < 0.0001$).

Conclusions:

Smoking is independently associated with accelerated progression of liver fibrosis. Both current and former smokers exhibit greater worsening of FIB-4 scores compared with non-smokers, and fibrosis progression occurs regardless of cumulative smoking exposure.

Keyword(s) : smoking, liver fibrosis, FIB-4, longitudinal analysis, pack-years

1406 - Assessing the Efficacy of donor derived cell free DNA (ddcfDNA) Measurement in Liver Transplant Recipients for Rejection Diagnosis and Therapeutic Response Evaluation A case report

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Background / Hypothesis:

Despite advancements in immunosuppressive therapies, rejection remains a significant challenge for post-liver transplantation. Current diagnostic methods for rejection, such as liver biopsy, are invasive and may not capture rejection episodes in a timely manner. The emergence of circulating biomarkers, particularly donor-derived cell-free DNA (ddcfDNA), presents a promising non-invasive approach for rejection diagnosis and therapeutic monitoring. This case report explores the feasibility and potential benefits of ddcfDNA measurement into clinical practice for liver transplant recipients.

Materials and Methods:

A 45-year-old male patient, about 200 days after deceased-donor liver transplantation for alcoholic liver cirrhosis, visited our hospital for regular follow-up. His liver function has been well maintained during the post-transplant follow-up, but on this day, his total bilirubin level was elevated to 3.02 mg/dL aspartate transaminase (AST) was 469 U/L alanine transaminase (ALT) was 779 U/L and he complained of itching sensation. The tacrolimus level was 11.3 ng/mL, however, the patient reported that he had skipped his immunosuppressant medication while traveling the week before.

Results:

The patient was hospitalized with suspected acute rejection, and a liver biopsy was performed to confirm diagnosis and the first serum ddcfDNA sample was collected. We checked the absolute number of ddcfDNA copies per mL of plasma and fraction of ddcfDNA (ddcfDNA%) and the result was 39.1%. The patient was diagnosed with T-cell mediated rejection, steroid pulse therapy was initiated with an intravenous methylprednisolone dose of 500 mg/day administered for 3 days and then tapered by half every day to 60 mg/day. After that, patient discharged with oral methylprednisolone initiated at 32 mg/day, tapered to 4–8 mg/day within 1–2 weeks. On discharge day, the total bilirubin level was 1.69 mg/dL, AST was 51 U/L, ALT was 164 U/L, and the patient was discharged without any symptoms. And the second serum ddcfDNA sample was collected at patient discharge day, the result was 9.01%, a decrease from the time of admission.

Conclusions:

ddcfDNA serves as a sensitive biomarker for detecting graft injuries in liver transplant recipients. Further large-scale research, which should ideally include protocol biopsies, will be needed to establish the practical value of ddcfDNA measurements in the management of liver transplant recipients.

Keyword(s) : Donor-derived cell-free DNA (ddcfDNA), Graft injury assessment, T-cell mediated rejection, Non-invasive biomarker

1407 - Osteoclast like giant cell tumor of the intrahepatic bile duct a rare neoplasm with a stable

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clinical course A case report

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Background / Hypothesis:

Osteoclast-like giant cell tumors most commonly arise in the bone and are rarely identified within the gastrointestinal tract. Among these locations, the liver is an exceptionally uncommon site. We report a rare case of an osteoclast-like giant cell tumor originating from the intrahepatic bile duct in a 70-year-old woman.

Materials and Methods:

A bile duct mass was incidentally detected during a routine health examination in a 70-year-old woman. Further evaluation with magnetic resonance imaging and positron emission tomography was performed to characterize the lesion and assess operability. The patient subsequently underwent formal left hepatectomy with regional lymph node dissection. Histopathological and immunohistochemical studies were conducted to establish the definitive diagnosis.

Results:

Magnetic resonance imaging demonstrated segmental enhancing wall thickening with intraductal protruding components involving the hilar left bile duct, resulting in separation of the second-order branches and obstructive cholangiohepatitis in the left hepatic lobe. PET imaging showed a hypermetabolic hilar lesion suggestive of cholangiocarcinoma and mildly hypermetabolic lymph nodes in the common hepatic and celiac areas. Surgical resection was completed without evidence of adjacent organ invasion, and pathological examination confirmed an osteoclast-like giant cell tumor. At one-year follow-up, the patient remains disease-free with no evidence of recurrence.

Conclusions:

This report describes the first known case of an osteoclast-like giant cell tumor originating in the intrahepatic bile duct. The case adds to the limited literature on this rare entity and suggests that tumors in this location may demonstrate relatively indolent biological behavior.

Keyword(s) :

1409 - Alcohol Associated Liver Cancer Burden Among Young Adults Comparing China With Global Trends From GBD 2021

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Background / Hypothesis:

To assess temporal trends in alcohol-associated liver cancer burden among young adults (15-49 years) in China during 1990-2021 and compare with global patterns to provide evidence-based recommendations for prevention strategies.

Materials and Methods:

Using the Global Burden of Disease 2021 database, we analyzed age-standardized incidence rates (ASIR), age-standardized mortality rates (ASMR), and age-standardized disability-adjusted life years (DALY) rates for alcohol-associated liver cancer among young adults in China from 1990 to 2021. Temporal trends were evaluated using estimated annual percentage change (EAPC) with 95% confidence intervals, and findings were compared with global estimates.

Results:

In 2021, young adults in China showed lower rates than global levels across all metrics (ASIR: 0.9 vs. 1.1, ASMR: 0.9 vs. 1.1, and DALY: 22.0 vs. 26.4 per 100,000). Over 1990-2021, China demonstrated increasing trends in both ASIR (EAPC=0.62%, 95% CI: 0.42%-0.81%) and ASMR (EAPC=0.16%, 95% CI: 0.00%-0.32%) for alcohol-associated liver cancer among young adults, while the age-standardized DALY rate declined (EAPC=-0.18%, 95% CI: -0.34% to -0.01%). Conversely, global trends showed increases across all metrics: ASIR (EAPC=0.55%, 95% CI: 0.48%-0.62%), ASMR (EAPC=0.31%, 95% CI: 0.25%-0.37%), and age-standardized DALY rates (EAPC=0.12%, 95% CI: 0.06%-0.18%). Notably, young males accounted for the predominant disease burden both in China and globally, exhibiting consistently higher ASIR, ASMR, and age-standardized DALY rates than females.

Conclusions:

Although the burden of alcohol-associated liver cancer among young adults in China remains lower than global levels, the increasing trends warrant heightened public health attention. Targeted interventions for

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young males are urgently needed to mitigate the rising burden of alcohol-associated liver cancer.

Keyword(s) : Alcohol, Liver cancer, Incidence, Mortality, Disability-adjusted life years

1411 - Impact of gut microbiota on the aggravation of NAFLD development in high fat diet mice with circadian rhythm disruption

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Background / Hypothesis: Non-alcoholic fatty liver disease (NAFLD) is closely linked to gut dysbiosis, and circadian rhythm disruption (CRD) can exacerbate this imbalance. However, the interplay between NAFLD, CRD, and gut microbiota remains unclear. This study investigates the impact of gut microbiota on NAFLD progression in high-fat diet (HFD)-induced mice with CRD.

Materials and Methods: Male C57BL/6J mice were divided into three groups: normal diet with normal light-dark cycle (ND-LD), HFD with normal light-dark cycle (HFD-LD), and HFD with disrupted light-dark cycle (HFD-CRD). Mice were monitored for food intake, activity, and body weight. After 14 weeks, samples were collected from euthanized mice. The remaining HFD-LD mice were divided into two subgroups: fecal microbiota donor and normal light-dark cycle-saline gavage (LD-NS). The remaining HFD-CRD mice were divided into two subgroups: disrupted light-dark cycle-normal saline gavage group (CRD-NS) and disrupted light-dark cycle-fecal microbiota transplantation group (CRD-FMT). After a second 14 weeks, samples were collected from euthanized mice. Serum biomarkers, liver pathology, and gut microbiota (16S rRNA sequencing) were analyzed.

Results: HFD induced NAFLD, while CRD exacerbated metabolic dysfunction, liver injury, and gut microbiota alterations. Over time, the impact of CRD on glucose metabolism and liver injury diminished. CRD further altered gut microbiota in NAFLD mice, reducing microbial diversity and increasing pro-inflammatory taxa. FMT from HFD-LD mice partially restored gut microbiota composition, improved lipid metabolism, and reduced liver inflammation.

Conclusions: CRD worsens NAFLD by inducing gut dysbiosis, while FMT from HFD-LD mice mitigates these effects, indicating that gut microbiota plays a regulatory role in the exacerbation of NAFLD induced

by CRD in HFD-fed mice.

Keyword(s) : Non-alcoholic fatty liver disease; Circadian rhythm disruption; Gut microbiota; Fecal microbiota transplantation; 16S rRNA sequencing; High-fat diet

1412 - Lactobacillus acidophilus Modulates the Gut-Liver Axis and Ameliorates MASLD and Type 2 Diabetes-Related Metabolic Dysregulation

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) and type 2 diabetes (T2D) are strongly influenced by gut-liver axis disturbances. Altered microbial communities exert a significant impact on disease progression, yet their mechanistic contribution remains incompletely defined. This study aimed to evaluate whether *Lactobacillus acidophilus* improves hepatic and metabolic abnormalities by modulating gut microbiota and associated metabolic pathways.

Materials and Methods:

Human stool samples were collected from healthy controls, MASLD, and MASH patients (HC, n=39; MASLD, n=35; MASH, n=43) and analyzed by 16S-based microbial taxonomic profiling (MTP) to assess microbial diversity and taxonomic composition. In parallel, a Western diet (WD)-induced MASLD mouse model and ob/ob diabetic mice were used to evaluate the metabolic effects of *L. acidophilus*. *L. acidophilus* was administered 2 times/week at a concentration of 1×10^9 CFU. metformin was used as a positive control to compare the metabolic effects of *L. acidophilus*. Hepatic histology, serum biochemical parameters, glucose metabolism, qPCR and microbiome profiling were performed across models.

Results:

Human microbiome profiling showed that gut microbial imbalance worsened with disease severity, and *Lactobacillus* abundance exhibited a progressive decreasing trend from HC (0.26%) to MASLD (0.23%) and MASH (0.12%).

In WD-fed mice, *L. acidophilus* supplementation reduced hepatic steatosis and inflammatory injury

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(NAS: NCD, 0; WD, 7.1; La, 5.2) and significantly lowered hepatic cholesterol levels ($p = 0.0486$). *L. acidophilus* restored intestinal barrier markers (Occludin and Claudin) and attenuated hepatic inflammation by lowering $Tnf\alpha$ and $Il1\beta$ expression. It also modulated hepatic lipid metabolism by down-regulating *Cd36* and *Srebp1c* while enhancing *Ppar α* expression. Additionally, *L. acidophilus* partially improved WD-induced dysbiosis, reflected by recovery of the Firmicutes/Bacteroidota ratio. In ob/ob diabetic mice, *L. acidophilus* improved glucose homeostasis, reducing OGTT AUC (Con, 45733; La, 36888; Met, 36510). *L. acidophilus* also enhanced hepatic AMPK activation and modulated key metabolic regulators, decreasing *SREBP1c* and *G6pc* expression while increasing *Lpin1* expression, thereby reducing both lipogenesis and gluconeogenesis.

Conclusions:

L. acidophilus improved hepatic steatosis, lipid dysregulation, and glucose homeostasis across multiple MASLD and T2D models. These findings suggest that *Lactobacillus acidophilus* exerts hepatoprotective and antidiabetic effects through AMPK activation and gut microbiota modulation, supporting its potential as a microbiome-based therapeutic candidate.

Keyword(s) : *Lactobacillus acidophilus*; MASLD; Type 2 diabetes; Gut–liver axis; Gut microbiota

1413 - Bile fungal diversity correlated with severity and is predictive of carcinoma in patients with CAGB

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Background / Hypothesis: Gallbladder cancer (CAGB) has poor prognosis. While the bile microbiome and lipidome are associated with CAGB development, the contribution of fungal microbiota is unexplored. Our study aims to investigate the contribution of fungal microbiome and identify top fungal peptides to potentially stratify CAGB patients.

Materials and Methods: Bile samples were collected from patients undergoing cholecystectomy for GS disease ($n=10$), patients undergoing surgery for CAGB ($n=16$) and healthy liver donors ($n=16$) were subjected

to fungal peptide-based diversity analysis and metabolomics. Results were cross-validated on the plasma samples, correlated with clinical parameters, and subjected to machine learning analysis.

Results: Fungal peptide-based diversity analysis identified 6 Phylum and 24 Fungal Species differentially regulated in the bile samples of the study cohort. Fungal diversity (alpha and beta diversity) was significantly higher in CAGB as compared to GS or HC ($p<0.05$). 93 fungal peptides were upregulated and 63 were downregulated in CAGB ($p<0.05$, $FC>1.5$). Linear discriminant analysis significantly segregated CAGB patients with marked increase in *Aspergillus wentii* ($\log_2FC=12.21$), *Nosema bombycis* ($\log_2FC=11.25$), *Saccharomyces* ($\log_2FC=10.89$), *Saccharomyces cerevisiae* ($\log_2FC=10.68$) and *Schizosaccharomyces pombe* ($\log_2FC=10.38$; $p<0.05$). Fungal-metabolite correlation analysis ($r^2>5$, $p<0.05$) identified *Aspergillus wentii*, *Nosema bombycis*, and others were directly associated with lysine biosynthesis, taurine, and hypotaurine metabolism, fatty acid, and other in bile, and cysteine and methionine metabolism, ascorbate metabolism, purine metabolism and other in plasma. Probability of detection (POD) calculated using fungal peptides showed a diagnostic efficiency of 96% and segregated CAGB patients. The identified top 5 fungal peptides were validated using Single reaction monitoring analysis and predicted early mortality with $>90\%$ accuracy/sensitivity and specificity in Machine Learning model.

Conclusions: Bile fungal diversity correlates with CAGB development and identified fungal panel could be used for segregation and early diagnosis of CAGB patients.

Keyword(s) : CAGB- Gallbladder carcinoma, Fungal microbiome, metabolomics, Fungal peptides.

1420 - Machine learning model based on systemic metabolic factor for mortality prediction in acute hepatitis E virus infection development and validation from a dual center cohort

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Background / Hypothesis: Hepatitis E virus (HEV) infection remains a significant

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cause of acute liver failure with high short-term mortality. However, predictive models incorporating systemic metabolic features for mortality risk assessment in HEV patients are lacking. To address this gap, we aimed to incorporate systemic metabolic factors into a machine learning framework with the goal of enhancing the early and accurate prediction of mortality risk in hospitalized HEV patients.

Materials and Methods:

A total of 510 hospitalized HEV patients from two medical centers were retrospectively enrolled and divided into training, internal validation, and external validation cohorts. Metabolic parameters, including total cholesterol, high-density lipoprotein, low-density lipoprotein, and diabetes, were integrated via support vector machine to generate a Metabolism Score (Figure 1A). This score, combined with additional clinical variables, underwent least absolute shrinkage and selection operator regression for feature selection (Figure 1B). Five machine learning algorithms were subsequently developed to predict 28-day and 90-day mortality, and their predictive performance was compared across the three cohorts.

Results:

Ten predictors were identified, including Metabolism Score, infection status, WBC, creatinine, age, platelet count, albumin, TBIL, EGFR, INR, and GGT (Figure 1C). Among the five machine learning models, the logistic regression (LR) model achieved the best performance, with AUROC values of 0.87 (95% CI = 0.83-0.90), 0.98 (95% CI = 0.94-0.99), and 0.84 (95% CI = 0.69-0.94) for 28-day mortality (Figure 1D-I), and 0.86 (95% CI = 0.82-0.90), 0.88 (95% CI = 0.81-0.93), and 0.89 (95% CI = 0.75-0.97) for 90-day mortality in the training, validation, and external cohorts (Figure 1J-L, Figure 2A-C), respectively. Calibration curves showed excellent agreement between predicted and observed outcomes, and decision curve analysis demonstrated a greater net clinical benefit compared with treat-all or treat-none strategies. The LR model also outperformed the MELD score (Figure 2D-I) and was visualized as a nomogram to facilitate clinical use (Figure 2J).

Conclusions:

The LR model integrating systemic metabolic factors effectively predicts short-term mortality in HEV patients. This model provides an interpretable and clinically practical tool to support early risk stratification and personalized management, and may assist clinicians in timely identifying high-risk individuals.

Keyword(s) : Hepatitis E virus, Clinical outcome, Liver

failure, Machine learning, Clinical characteristic, Noninvasive, Predictive model, Prognosis.

1421 - Pharmabiotics *Phocaeicola dorei* ameliorates liver fibrosis by alleviating macrophage efferocytosis of neutrophils

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Background / Hypothesis:

Liver fibrosis results from chronic inflammation, dysregulated immune responses, and persistent activation of hepatic stellate cells (HSCs). Increasing evidence shows that gut microbiota influence hepatic immunity and fibrogenesis. Among these microbes, *Phocaeicola dorei* has been observed to change dynamically during liver disease progression; however, its functional role in fibrosis remains unclear. We hypothesized that *P. dorei* attenuates liver fibrosis by suppressing neutrophil activation and limiting macrophage-mediated efferocytosis of apoptotic neutrophils, thereby reducing downstream HSC activation.

Materials and Methods:

Fecal metagenomic data from 90 individuals (normal, hepatitis, cirrhosis) were analyzed to examine microbial alterations and *P. dorei* abundance. Liver fibrosis was induced in C57BL/6 mice using a DDC diet, followed by oral gavage of *P. dorei* (10^9 CFU, twice weekly). Fibrosis severity, collagen deposition, immune-cell infiltration, and chemokine expression were assessed by histology, Sirius Red staining, immunofluorescence, and qPCR. HSCs were isolated for RNA sequencing to identify pathway changes modulated by *P. dorei*. In vitro assays using HL-60-derived neutrophils, THP-1 macrophages, and LX-2 cells were conducted to evaluate apoptotic body-driven activation and the effects of *P. dorei* supernatant on inflammatory and fibrogenic signaling.

Results:

P. dorei abundance exhibited stage-dependent alterations in human liver disease. In the DDC model, *P.*

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P. dorei supplementation significantly reduced fibrosis, hepatic neutrophil and macrophage infiltration, and expression of CC/CXC chemokines. RNA-seq revealed that *P. dorei* reversed DDC-induced activation of neutrophil degranulation, phagosome, and efferocytosis pathways in HSCs. Expression of key efferocytosis mediators such as Axl, Cd36, Mfge8, and Pros1 was suppressed. In vitro, apoptotic neutrophil bodies stimulated macrophage-HSC activation, increasing COL1A1, ACTA2, and TGF- β , whereas *P. dorei* supernatant markedly decreased these responses. *P. dorei* also shifted the gut microbial composition toward a more anti-inflammatory profile.

Conclusions:

P. dorei protects against liver fibrosis by suppressing neutrophil activation, reducing macrophage efferocytosis, and attenuating HSC activation. These findings highlight *P. dorei* as a promising next-generation pharmabiotic capable of modulating immune-microbiome interactions to limit fibrosis progression.

Keyword(s) : Liver fibrosis, Neutrophil degranulation, Macrophage efferocytosis

1424 - Comparing two intervention strategies to improve hepatitis B virus deoxyribonucleic acid testing rate among hepatitis B surface antigen positive hospitalized patients

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Background / Hypothesis: Hepatitis B surface antigen (HBsAg) screening is fundamental for chronic hepatitis B management, while hepatitis B virus deoxyribonucleic acid (HBV-DNA) testing is crucial for determining the viral replication status and initiating antiviral therapy. However, the HBV-DNA testing rate among inpatients is generally low. This study aimed to analyze the HBsAg positivity rate and HBV-DNA testing among inpatients at Ningbo Second Hospital and to evaluate the effectiveness of two intervention measures—publishing departmental rankings on the hospital intranet and conducting on-site educational sessions in departments—in improving the HBV-DNA

testing rate.

Materials and Methods: 1. Retrospectively collect data from inpatients between August 2024 and November 2025 and calculate the monthly HBsAg positivity rate and HBV-DNA testing rate. 2. Starting from November 2024, departmental HBV-DNA testing rate rankings were published monthly on the hospital intranet. Changes in the testing rate over the subsequent 5 months (December 2024 to April 2025) were evaluated. 3. Starting from May 2025, on-site educational sessions emphasizing the clinical significance of HBV-DNA testing were conducted in the 10 departments with the lowest testing rates. Changes in the testing rate over the following 6 months (May to October 2025) were evaluated. A t-test was used to compare testing rates before and after the interventions, with a P-value < 0.05 considered statistically significant.

Results: 1. During the baseline period (August–November 2024), a total of 40,255 inpatients were included. The HBsAg positivity rate was 7.8% (3,139/40,255), and the mean HBV-DNA testing rate was 28.4%. 2. During the period of intranet ranking publication (December 2024–April 2025), a total of 48,176 inpatients were included. The HBsAg positivity rate was 7.3% (3,506/48,176), and the mean HBV-DNA testing rate was 28.4%, showing no significant change compared to the baseline (P = 0.94). 3. During the period of on-site educational sessions (May–October 2025), a total of 61,512 inpatients were included. The HBsAg positivity rate was 7.4% (4,531/61,512), and the HBV-DNA testing rate increased to 32.6%, showing a significant improvement compared to the previous period (P < 0.05).

Conclusions: Publishing testing rate rankings on the intranet alone did not effectively improve the HBV-DNA testing rate. However, targeted on-site educational sessions in departments significantly increased the HBV-DNA testing rate. Proactive, face-to-face medical education holds important practical value in enhancing HBV-DNA testing among inpatients.

Keyword(s) : HBV, HBsAg, HBV-DNA, Whole-Process Management of Hepatitis B

1425 - Fibroblast activation assessed by PRO C3 and PRO C6 is associated to accumulation of key bile acids A hallmark of fibrosis initiation and mortality in alcohol related liver disease

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Background / Hypothesis: Alcohol-related liver disease (ALD) results from persistent liver damage due to excessive alcohol consumption, often progressing to fibrosis. Disrupted bile acid homeostasis is considered a key feature in liver fibrosis development in ALD. Studies in MASH and PSC patients have shown that antifibrotic therapy reduces both bile acid levels and the fibroblast activity biomarker PRO-C3, hinting at a link between bile acids (BA) and fibrogenesis, which remains unclear. This study investigates the potential role of bile acids in ALD-related fibrogenesis by analyzing associations between fibroblast activity biomarkers, bile acids, and clinical outcomes.

Materials and Methods: Serum levels of PRO-C3 (N-terminal pro-peptide of type III collagen) and PRO-C6 (endotrophin; type VI collagen formation) and plasma concentrations of bile acids obtained through UHPLC-MS/MS were assessed in a cross-sectional cohort of 450 patients with biopsy-proven ALD. Associations between circulating BA concentrations and fibrogenesis biomarkers were analyzed using Spearman correlation. The Wilcoxon test was applied to compare PRO-C3 and PRO-C6 levels in ALD patients stratified by low and high risk of outcomes over a median follow-up period of 5 years using Kaplan-Meier survival analysis obtain survival curves and hazard ratios for mortality (HRM) and decompensation (HRD).

Results: Fibrogenesis markers correlated with glycine conjugated BAs GCA (correlation coefficient of 0.67 for PRO-C3 and 0.45 for PRO-C6) and GUDCA (correlation coefficient of 0.53 for PRO-C3 and 0.34 for PRO-C6) and taurine conjugated bile acid TCA (correlation coefficient of 0.64 for PRO-C3 and 0.46 for PRO-C6) at baseline. GCA, GUDCA and TCA were elevated in ALD patients at risk of all-cause mortality and decompensation (GCA, cutoff of 684 ng/mL, $p < 0.001$ HRM = 3.5 and HRD = 7.6; GUDCA, cutoff of 575 ng/mL, $p < 0.001$, HRM = 2.8 and HRD = 4.1; TCA, cutoff of 720 ng/mL, $p < 0.001$, HRM = 2.6 and HRD = 3.7). Baseline levels of fibrogenesis markers were found to be significantly increased in the higher risk

groups stratified according to BA concentrations of GCA (PRO-C3, $p = 1.15 \times 10^{-28}$; PRO-C6, 8.9×10^{-14}) GUDCA (PRO-C3, $p = 2.67 \times 10^{-12}$; PRO-C6, 1.93×10^{-5}) and TCA (PRO-C3, $p = 2.87 \times 10^{-20}$; PRO-C6, 5.57×10^{-11}).

Conclusions: Increased circulating levels of toxic hydrophobic BA relate to activation of fibroblasts and to poor prognosis in ALD patients, suggesting that bile acids are linked to activation of fibrogenesis, and therefore induce the development of liver fibrosis in ALD.

Keyword(s) : bile acids, fibrogenesis, alcohol-related liver disease, collagens

1427 - Protein tyrosine phosphatase 1B promotes hepatic stellate cell activation via SRC signaling

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Background / Hypothesis:

Liver fibrosis, a major cause of cirrhosis and liver cancer, affects millions globally with limited treatment options. It is a progressive pathological condition marked by excessive extracellular matrix (ECM) deposition that ultimately impairs hepatic function, driven by the activation of hepatic stellate cells (HSCs) into myofibroblasts. Understanding the molecular mechanisms of HSC activation is critical for developing effective therapies. Protein tyrosine phosphatase 1B (PTP1B), which is highly expressed in activated HSCs, has been implicated in collagen overproduction and fibrogenesis, expanding its established roles in metabolic disorders such as obesity and type 2 diabetes. We hypothesized that PTP1B promotes human HSC activation through regulation of pro-fibrotic signaling pathways.

Materials and Methods:

Primary human HSCs were subjected to gain- and loss-of-function experiments to evaluate the effects of PTP1B on HSC activation. Assays included proliferation, migration, and gene expression analyses. Mechanistic studies examined Src signaling, including the effect of the inhibitory tyrosine 530 residue. Pharmacological inhibition of Src and re-analysis of human fibrotic liver tissue datasets were performed to validate the functional and clinical relevance of the PTP1B-Src axis.

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Results:

PTP1B overexpression enhanced HSC activation, increasing cell proliferation, migration, and ECM-related gene expression, while PTP1B knockdown attenuated these effects. Mechanistically, PTP1B promoted Src activation by dephosphorylating its inhibitory Tyr530 residue, driving pro-fibrotic phenotypes. Src inhibition reversed the pro-fibrotic effects of PTP1B. Clinical dataset analyses revealed significant upregulation of both PTP1B and Src in fibrotic human liver tissues, with positive correlations to ECM gene expression.

Conclusions:

These findings identify the novel PTP1B-Src signaling axis that drives human HSC activation and hepatic fibrogenesis, highlighting PTP1B as a potential therapeutic target for liver fibrosis.

Keyword(s) : Liver fibrosis, Protein tyrosine phosphatase 1B (PTP1B), Hepatic stellate cells (HSCs), Extracellular matrix (ECM), Src, Dephosphorylation

1428 - Surgical Resection Versus Non Surgical Treatment for Hepatocellular Carcinoma with Portal Vein Tumor Thrombosis A Comprehensive Systematic Review and Meta Analysis

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Background / Hypothesis: The role of surgical resection (SR) as a primary treatment for hepatocellular carcinoma (HCC) with portal vein tumor thrombosis (PVTT) remains uncertain.

Materials and Methods: A comprehensive search of electronic databases from January 2000 to April 2024 identified studies comparing SR with transarterial chemoembolization (TACE), combined TACE and radiation therapy (TACE-RT), or sorafenib. Meta-analyses were conducted for comparison groups with at least three eligible studies. Hazard ratios (HRs) with 95 percent confidence intervals (CIs) for overall survival

(OS) were extracted or reconstructed from Kaplan-Meier curves.

Results: Twelve retrospective cohort studies involving 4,498 patients were included. SR demonstrated superior long-term OS compared with TACE in Vp1-2 (5-year OS: HR 0.53, 95% CI 0.35-0.79) and Vp3 (5-year OS: HR 0.68, 95% CI 0.61-0.75), with comparable outcomes in Vp4. SR and TACE-RT showed similar 1- and 3-year OS, although SR demonstrated a clear 5-year OS advantage in Vp1-3 PVTT (HR 0.48, 95% CI 0.31-0.74). Compared with sorafenib, SR provided superior OS in Vp1-2 (3-year OS: HR 0.26, 95% CI 0.18-0.38) and comparable OS in Vp3. Sensitivity analyses supported the robustness of these findings, and no substantial publication bias was detected.

Conclusions: Based on current evidence, SR is not inferior to non-surgical treatments including TACE, TACE-RT, and sorafenib in patients with HCC and PVTT and provides meaningful long-term survival benefits, particularly in those with segmental PVTT (Vp1-Vp2). Further comparative studies incorporating immunotherapy are needed in future research.

Keyword(s) : Hepatocellular carcinoma; tumor thrombosis; resection; transarterial chemoembolization; meta-analysis.

1429 - Meta analysis of efficacy and safety of CAR NK cell in the treatment of primary liver cancer

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Background : According to the 2020 Global Cancer Statistics, there are 19.3 million new cancer cases and 10 million cancer deaths annually, highlighting the urgent need for effective cancer treatments. Traditional therapies, including surgery, radiotherapy, and chemotherapy, have limitations in treating hepatocellular carcinoma (HCC). While recent advancements in cancer biology and immunotherapy, particularly the emergence of CAR-NK cell therapy, demonstrate promising therapeutic potential, their efficacy in HCC treatment still faces significant challenges.

Materials and Methods: This study systematically searched the relevant studies in Pubmed, Embase, Cochrane and ClinicalTrial.gov databases up to March 2025, aiming to comprehensively evaluate the clinical efficacy and safety of CAR-NK cell therapy in hepatocellular carcinoma (HCC). The inclusion criteria

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were clinical trials that reported objective response rate (ORR).

Results:After screening, a total of 3 trials involving 49 patients were included. The results showed that NK cell therapy achieved an objective response rate of 72.3% in HCC (usually combined with local treatment), with good safety performance, where fatigue was the most common adverse event.

Conclusions:CAR-NK cell therapy offers a novel therapeutic approach for hepatocellular carcinoma (HCC). Given its promising initial results in HCC treatment, future research should focus on the following aspects. First, larger-scale multicenter clinical trials are required to validate the generalizability and reliability of these findings. Second, deeper exploration of CAR-NK cell therapy mechanisms is essential, including their behavior in the tumor microenvironment and impact on immune evasion. Additionally, research should address strategies to enhance the in vivo proliferation and persistence of CAR-NK cells, as well as reduce treatment-related adverse effects.

Keyword(s) : Natural killer cells; Hepatocellular carcinoma; Chimeric antigen receptor; Meta-analysis

1430 - Fibrosis non invasive test panel (FIB NIT) A novel biomarker panel for investigating fibrosis dynamics through individual components of fibrogenesis (PRO C3) fibrolysis (CTX III) and clinical outcomes (PRO C6) in alcohol related liver disease

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Background / Hypothesis: Persistent and excessive alcohol intake induces damage to the liver, impairing its ability to properly regenerate, potentially leading to alcohol-related liver disease (ALD). Non-invasive tests (NITs) quantifying the dynamics of the extracellular matrix (ECM) remodeling and disease activity may be important for patient prognosis. We aimed to investigate the above-mentioned utility of the serologically assessed FIBrosis Non-Invasive Test panel (FIB-NIT) consisting of three single markers that can

describe fibrogenesis (PRO-C3), patient outcomes PRO-C6) and fibrolysis (CTX-III). All, previously shown to be modulated by interventions in MASH clinical trials.

Materials and Methods: Serum biomarkers of fibrogenesis nordicPRO-C3TM (N-terminal pro-peptide of type III collagen) and for patient outcome nordicPRO-C6TM (endotrophin; type VI collagen formation) as well as fibrosis resolution nordicCTX-IIIITM (MMP degraded fragment of cross-linked type III collagen) were measured in a cross-sectional cohort of 450 biopsy-proven F0-F4 ALD patients. The biomarkers were investigated as single markers to understand disease mechanisms in relation to disease severity and prognosis of outcomes (mortality, liver related events and non-liver related outcomes) over a median 5-yr follow-up.

Results: ALD patients with advanced fibrosis (F3-F4) and cirrhosis exhibited increased fibrogenesis as observed from higher serum PRO-C3 and PRO-C6 ($p < 0.001$) as well as higher fibrolysis as shown by higher serum levels of CTX-III ($p < 0.001$) in comparison to patients with mild-moderate fibrosis (F0-2). Kaplan-Meier curves showed that all three biomarkers provided good prognostic value to stratify ALD patients at risk of all-cause mortality and decompensation (PRO-C3 cutoff of 12.6, hazard ratio for mortality (HRM) of 4.1 and decompensation (HRD) of 10.6; CTX-III cutoff of 15.6; HRM of 2.2 and HRD of 2.6; PRO-C6 cutoff of 11.2; HRM of 3.4 and HRD of 4.8.

Conclusions: The "FIB-NIT" biomarker panel may be utilized for evaluating fibrosis remodeling and risk of patient outcomes in ALD. This panel may offer insights into the underlying mechanisms of fibrosis progression and help understanding the mode of action of novel therapies in ALD as well as other chronic liver diseases.

Keyword(s) : fibrogenesis, fibrosis resolution, clinical outcomes, alcohol-related liver disease, collagens, extracellular matrix remodeling

1432 - Longitudinal Changes in Skeletal Muscle Index Among Patients With Cirrhosis A Mixed Effects Analysis in an Observational Cohort



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Background / Hypothesis: Sarcopenia is a common and severe complication of liver cirrhosis and is closely associated with adverse clinical outcomes. However, precise quantitative data describing the longitudinal rate of change in skeletal muscle index (SMI) based on abdominal CT imaging—and its associations with sex and age—remain limited in cirrhotic populations.

Materials and Methods: This retrospective study included 115 adult patients with cirrhosis (77 men and 38 women) from Beijing You'an Hospital, Capital Medical University. All patients underwent at least two abdominal CT examinations at intervals of 12 ± 3 months. Skeletal muscle area at the L3 vertebral level was measured and used to calculate SMI. A linear mixed-effects model was applied to estimate the longitudinal rate of SMI change over time.

Results: Cirrhotic patients exhibited a significant progressive decline in SMI, with an estimated annual decrease of -1.001 units/year (95% CI: -1.317 to -0.683, $P < 0.001$). Subgroup analyses showed no significant differences in the rate of SMI decline by sex (interaction $P = 0.812$) or age group (≥ 60 vs. < 60 years, interaction $P = 0.736$), indicating that the progression of sarcopenia is a pervasive process affecting patients with cirrhosis irrespective of sex or age.

Conclusions: This study quantitatively demonstrates a progressive decline in SMI during longitudinal follow-up in patients with cirrhosis, with a rate substantially exceeding the physiological age-related decline observed in healthy individuals. These findings underscore that sarcopenia represents a universal and consistent complication throughout the course of cirrhosis. Accordingly, routine longitudinal monitoring of SMI and early targeted intervention are

recommended for all patients with cirrhosis to improve long-term outcomes.

Keyword(s): Liver cirrhosis, Sarcopenia, Skeletal muscle index, Mixed-effects models

1433 - Functional changes following hepatocyte transplantation in mice with liver injury

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Background / Hypothesis: Percutaneous ethanol injection (PEI) is a widely used local therapy for hepatocellular carcinoma due to its minimal invasiveness and strong local tumor control. However, the diffusion of injected ethanol is difficult to control, and oxidative stress and necrosis have been reported to occur in normal hepatocytes surrounding the tumor, which may lead to hepatic dysfunction and poor prognosis. Preventing such injury to normal hepatocytes is for enhancing the safety of PEI. This study aimed to evaluate whether hepatocyte transplantation can suppress PEI induced liver damage in a normal mouse model.

Materials and Methods: C57BL/6 mice were divided into three groups: non-treated control (Control), PEI + PBS (Sham group), and PEI + hepatocyte transplantation (Treatment group). The PEI models were injected 20 μ L of 100% ethanol into the left hepatic lobe, and mice injected either PBS or primary hepatocytes isolated from GFP-transgenic C57BL/6 donor mice via intrasplenic, in the sham and treatment groups, respectively. Serum ALT was measured at 3, 6, 24, and 48 hours and at sacrifice. Mice were sacrificed at 1 and 4 weeks after treatment, and liver tissues were analyzed histologically by H&E staining for morphological assessment and GFP immunohistochemical staining for localization of transplanted cells and genetically by qRT-PCR of inflammation and fibrosis related genes.

Results: Serum ALT levels transiently increased in the treatment group compared with the sham group during the early phase after PEI, but the difference diminished over time until 48 hours. At 1 week, necrotic areas were smaller in the treatment group than in the sham group, indicating reduced hepatic injury. GFP-positive hepatocytes were detected in the liver at both 1 and 4

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weeks after transplantation, though no proliferation was observed between 1 and 4 weeks. qRT-PCR revealed that inflammatory cytokines and fibrosis markers tended to be lower in the treatment group compared to the sham group. In particular, Tgfb and Pai1 were significantly elevated in the sham group compared to the control group, whereas no significant differences were observed between the treatment and control groups. These findings suggest that hepatocyte transplantation may suppress hepatic inflammation and fibrotic responses following PEI.

Conclusions:

In a normal mouse model, hepatocyte transplantation reduced hepatic inflammation and fibrosis after PEI. Hepatocyte transplantation may help preserve normal liver tissue and contribute to improving the safety of PEI therapy.

Keyword(s) : hepatocyte transplantation, PEI, liver injury, liver regeneration

1434 - Awareness and Treatment Status of Patients with Chronic Hepatitis B Results from a Single Center Patient Survey in Hunan province China

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Background / Hypothesis: Chronic hepatitis B (CHB) remains a major public health challenge in China, with significant variations in treatment uptake and disease awareness. This single-center survey aimed to characterize real-world patient profiles, treatment awareness, and barriers to antiviral therapy.

Materials and Methods: A paper-based questionnaire was administered to CHB outpatients under the guidance of trained nurses, covering demographics, diagnosis and treatment history, HBV-related disease burden, and treatment decision factors.

Results: Among 145 respondents, 62.76% (91/145) were male and 82.76% (120/145) were aged 31–60 years; 52.41% (76/145) had a disease duration ≥ 10 years. Overall, 83.45% (121/145) were receiving antiviral therapy, predominantly with tenofovir alafenamide fumarate (TAF) or (entecavir) ETV (66.67%). Of the 24 untreated patients, main reasons for non-treatment included reluctance toward long-term medication (33.33%, 8/24), perception of no symptoms and thus no need for therapy (25.00%, 6/24), and no physician recommendation (25.00%, 6/24).

Although 79.31% (115/145) recognized HBV could lead to cirrhosis or cancer, 14.48% (21/145) mistakenly believed HBV is transmissible via shared utensils. Gender differences were observed: males more often followed physicians' advice (80.22% vs. 64.81%, $p < 0.05$), while females more frequently considered drug efficacy (31.48% vs. 23.08%), cost (16.67% vs. 6.59%), and treatment discontinuation (14.81% vs. 7.69%). Treated patients showed better awareness of transmission and treatment benefits. 85.7% of treated patients recognized the importance of timely treatment in preventing severe complications, compared with 54.5% of untreated patients. Digital channels were widely used for health information, with 40.69% of participants (59/145) selecting hospital official WeChat accounts or apps and 58.62% (85/145) utilizing at least one digital platform.

Conclusions: HBV awareness and treatment rates have improved among Chinese CHB patients, with TAF and ETV being the most common regimens. However, misconceptions about transmission and reluctance toward long-term therapy persist. Digital health platforms show potential in addressing knowledge gaps and supporting adherence.

Keyword(s) : Awareness and treatment; Chronic Hepatitis B; Single Center; Patient Survey

1435 - Individualized nutrition support improves outcomes in hepatitis B virus related acute on chronic liver failure patients at nutritional risk a randomized controlled trial

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Background / Hypothesis:

Patients with acute-on-chronic liver failure (ACLF) are at high nutritional risk, which adversely affects prognosis, yet robust evidence for nutritional interventions is scarce. This study evaluated the safety and efficacy of individualized nutritional support for hepatitis B virus-related ACLF (HBV-ACLF).

Materials and Methods:

In this single-center, open-label randomized controlled trial, HBV-ACLF patients meeting COSSH criteria with NRS-2002 ≥ 3 were randomized 1:1 to individualized nutritional support (energy target $1.2 \times \text{REE}$; protein 1.2 g/kg/day) or control (standard dietary advice). Assessments were performed at baseline, day 30, and day 90. Primary endpoints were 30- and 90-day transplant-free mortality. Secondary endpoints

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included safety, the incidence of adverse outcomes (ascites, gastrointestinal bleeding, infection, hepatic encephalopathy, hepatorenal syndrome, rehospitalization, and ICU admission), liver severity scores (MELD, COSSH-ACLF IIs), anthropometry (mid-arm circumference), muscle function (handgrip strength, gait speed, and SPPB), and quality of life (EQ-5D).

Results:

Between December 2023 and March 2025, 572 patients with HBV-ACLF were screened, 80 were randomized, and 78 (39 in the control group and 39 in the intervention group) were analyzed in the modified intention-to-treat population. Baseline characteristics were generally comparable between groups. Individualized nutrition significantly reduced transplant-free mortality at day 30 (2.6% vs. 20.5%, $p = 0.033$) and day 90 (17.9% vs. 38.5%, $p = 0.044$) compared to controls (Tab. 1). Adverse outcome rates were also significantly lower in the intervention group (day 30: 25.6% vs 56.4%, $p = 0.006$; day 90: 33.3% vs 74.4%, $p < 0.001$), with no excess in serious events, gastrointestinal reactions, complications, or hyperglycemia. The intervention group achieved greater improvements in MELD and COSSH-ACLF II scores, anthropometric indexes, muscle function, adverse clinical outcomes, and EQ-5D scores at follow-up (Fig. 1).

Conclusions:

In HBV-ACLF patients at nutritional risk, individualized target-driven nutritional support is safe, lowers 30- and 90-day transplant-free mortality, reduces adverse clinical outcomes, and improves hepatic function, muscle performance, and quality of life. These findings provide prospective, randomized evidence supporting early, goal-driven, and function-oriented nutrition treatments with dynamic assessments as a practical strategy in ACLF care.

Keyword(s) : Acute-on-chronic liver failure; hepatitis B virus; nutritional risk; Individualized nutrition support;

1437 - Efficacy of Sequential or Combined Tenofovir Alafenamide in Entecavir Treated CHB Patients with Low Level Viremia and Its Impact on Lipid Levels

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Background / Hypothesis:

To compare the efficacy of sequential tenofovir alafenamide (TAF) versus entecavir (ETV) plus TAF in ETV-treated chronic hepatitis B (CHB) patients with low-level viremia (LLV), and their effects on virological response, liver/kidney function, and lipid levels.

Materials and Methods:

A total of 217 CHB patients with LLV after ETV treatment (May 2020 - August 2023) were divided into the TAF group (sequential TAF, $n=180$) and the Combination group (ETV+TAF, $n=37$). After 1:1 propensity score matching (PSM), 37 patients were included in each group. SPSS 26.0 was used to compare HBV DNA undetectable rate, HBeAg seroconversion rate, liver/kidney function indices [LSM, PLT, AST, ALT, Cr], lipid levels [TC, TG, HDL-C, LDL-C], and adverse events.

Results:

After 48 weeks of treatment, the HBV DNA and HBeAg undetectable rates in the Combination group were 86.49% and 59.46%, respectively, higher than 59.46% and 35.14% in the TAF group ($\chi^2=6.852$, 4.391; $P=0.009$, 0.036). Post-treatment, LSM (7.01 ± 1.50 kPa), AST (18.02 ± 2.28 u/L) and ALT (19.85 ± 3.86 u/L) in the Combination group were lower than those in the TAF group (7.90 ± 1.68 kPa, 21.12 ± 2.85 u/L, 22.00 ± 3.90 u/L), with significant differences ($t=2.404$, 5.166, 2.383; $P=0.019$, 0.000, 0.020). PLT ($218.35 \pm 42.60 \times 10^9$) and Cr (70.92 ± 6.54 $\mu\text{mol/L}$) in the Combination group were higher than those in the TAF group ($192.82 \pm 44.13 \times 10^9$, 67.60 ± 6.13 $\mu\text{mol/L}$), with significant differences ($t=2.532$, 2.253; $P=0.014$, 0.027). Post-treatment, TC levels in the TAF group (5.60 ± 0.89 mmol/L) and Combination group (5.45 ± 0.80 mmol/L) were slightly higher than their pre-treatment levels [(5.18 ± 0.85) mmol/L, (5.02 ± 0.83) mmol/L] ($t=2.076$, 2.269; $P=0.041$, 0.026).

Conclusions:

Combined TAF therapy improves virological response, liver function, and fibrosis compared to sequential TAF in ETV-treated LLV patients, whereas sequential TAF has less impact on renal function. Both regimens may

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mildly elevate TC levels.

Keyword(s) : Chronic hepatitis B; Entecavir; Tenofovir alafenamide; Low-level viremia; Lipid levels

1438 - Efficacy and safety of seladelpar in patients with primary biliary cholangitis previously treated with fibrates or obeticholic acid

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Background / Hypothesis: Seladelpar is a first-in-class delpar (selective peroxisome proliferator-activated receptor [PPAR]-delta agonist) indicated for the treatment of primary biliary cholangitis in combination with ursodeoxycholic acid (UDCA) in patients with an inadequate response to UDCA or as monotherapy in patients unable to tolerate UDCA. RESPONSE was a Phase 3, randomised, placebo-controlled clinical trial of seladelpar in patients with inadequate response/intolerance to UDCA. Patients completing RESPONSE were eligible to roll over into ASSURE (NCT03301506), an ongoing, open-label, long-term, Phase 3 safety trial. Here, we describe data from month 18 (month 6 of ASSURE) in patients with or without prior use of fibrates or obeticholic acid (OCA) who rolled over from RESPONSE into ASSURE.

Materials and Methods: Patients received 10 mg seladelpar orally daily or placebo in RESPONSE; patients received open-label 10 mg seladelpar in ASSURE. Fibrates and OCA were prohibited during the study period and a 6-week washout was required prior to entry in RESPONSE. Data were described for patients in ASSURE with or without prior use of fibrates/OCA

and based on whether they received seladelpar (continuous seladelpar patients) or placebo (crossover patients) in RESPONSE. Efficacy included the percentage of patients achieving a composite biochemical response (CBR; alkaline phosphatase [ALP] < 1.67 × upper limit of normal [ULN], ALP decrease ≥ 15%, and total bilirubin ≤ ULN). Safety assessments included adverse events (AEs) and laboratory parameters.

Results: Among patients who continued into ASSURE from RESPONSE (158), 16 continuous seladelpar and 11 crossover patients reported prior use of fibrates/OCA (total, n=27; 17%); 88 continuous seladelpar and 43 crossover patients reported no prior use of fibrates/OCA (total, n=131; 83%). At month 18, among continuous seladelpar patients, 9/15 (60%) patients with prior fibrate/OCA use achieved a CBR vs 54/87 (62%) patients without prior fibrate/OCA use. Among crossover patients, 7/11 (64%) patients with prior fibrate/OCA use vs 32/41 (78%) patients without prior fibrate/OCA use achieved a CBR at month 6 of ASSURE. From ASSURE initiation to month 6, incidence of AEs was similar across continuous seladelpar and crossover patients, regardless of prior OCA/fibrate use; no treatment-related serious AEs were reported.

Conclusions: In this interim analysis of continuous seladelpar and crossover patients from ASSURE, patients who reported prior use of fibrates/OCA achieved a similar sustained biochemical response with seladelpar compared with patients who reported no prior use. Seladelpar appeared safe and well tolerated in this subgroup.

Keyword(s) : fibrates, obeticholic acid, primary biliary cholangitis, seladelpar

1439 - Liver stiffness-based assessment of fibrosis stages in chronic hepatitis B patients in Georgia associations with viral clinical and demographic factors

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Background / Hypothesis: Liver fibrosis is one of the key determinants of prognosis and treatment decisions in chronic hepatitis B (CHB). This study aimed to characterise liver-stiffness-based fibrosis stages in CHB patients in Georgia and evaluate their associations with viral, clinical, and demographic factors, thereby supporting clinical decision-making.

Materials and Methods: A cross-sectional study was conducted among 163 adults with CHB who underwent liver stiffness measurement (LSM) by transient elastography (TE). Variables included HBV DNA, HBeAg, ALT, AST, platelet count, BMI, presence of benign vs malignant liver lesions on abdominal ultrasound (US), antiviral treatment status, age and sex. Fibrosis stages were classified using LSM cut-offs: <7 kPa (F0–F1) and ≥7 kPa (≥F2). Cirrhosis was defined as liver stiffness >12.5 kPa (F4). For association analyses, stiffness was grouped into low <7 kPa (F0–F1) vs high ≥7 kPa (≥F2). Comparisons between low and high stiffness groups used independent-samples t-tests and chi-square tests. Pearson correlations assessed associations between continuous LSM and ALT, AST, platelet count, BMI, and HBV DNA; odds ratios (OR) with 95% confidence intervals (CI) were calculated.

Results: Of 163 patients (58.9% male; 24.1% aged ≥50 years), 69.9% had F0–F1 fibrosis and 30.1% had ≥F2, including 8.0% with F4. HBeAg negativity predominated (88.3%), and HBV DNA categories showed no significant group differences. Abdominal US identified benign liver lesions in 26.4% and hepatocellular carcinoma (HCC) in 1.8%. Patients with high stiffness (≥7 kPa) had higher ALT (64.5 vs 33.6 U/L, $p=0.004$), higher AST (43.4 vs 25.3 U/L, $p<0.001$), and lower platelet counts (210.8 vs $238.0 \times 10^9/L$, $p=0.013$). Age ≥50 years was strongly associated with high stiffness (51.3% vs 22.8%; OR=2.25, 95% CI 1.44–3.52; $p=0.001$). No significant associations were observed with sex, BMI, HBV DNA, HBeAg or ultrasound lesion type. Continuous LSM correlated with

AST ($r=0.439$, $p<0.001$), ALT ($r=0.225$, $p=0.004$), and inversely with platelets ($r=-0.378$, $p<0.001$). Treated patients more often had liver stiffness ≥7 kPa, higher fibrosis stages, HBeAg positivity, and ultrasound-detected HCC.

Conclusions: Higher liver stiffness in CHB patients was associated with older age, elevated transaminases, and lower platelet counts. No significant associations were observed with sex, BMI, HBV DNA, HBeAg or ultrasound lesion type. These findings may help identify patients at greater risk of fibrosis and support more targeted CHB management.

Keyword(s) : Chronic hepatitis B, Liver fibrosis, Liver stiffness measurement, Transaminases, Viral load

1440 - Prevalence and predictors of hepatitis B virus infection among sellers and workers at West Africa's largest market Kejetia Ghana a cross sectional study

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Background / Hypothesis: Hepatitis B virus (HBV) infection remains a major public health challenge in sub-Saharan Africa, including Ghana, despite the infant vaccination programs. Limited data exist on HBV prevalence and predictors in informal sector populations, who may face unique occupational and behavioural exposures. This study assessed the prevalence and predictors of HBV infection among sellers and workers at Kejetia Market, Ghana's largest commercial hub.

Materials and Methods: A cross-sectional study was conducted among 489 adult market workers from December 4, 2024, to January 25, 2025. Participants

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were selected through stratified random sampling across occupational groups. Data on sociodemographic, occupational, and behavioural factors were collected using structured questionnaires. On-site testing for hepatitis B surface antigen (HBsAg) was performed using the Hightop One Step Rapid Test kit. Bivariate and multivariate logistic regression analyses were used to identify independent predictors of HBV infection. $P < 0.05$ was considered statistically significant.

Results: The overall prevalence of HBV infection was 7.36% (36/489), consistent with intermediate-to-high endemicity. Multivariate analysis identified three independent predictors of HBV infection. Female gender (aOR = 0.455, 95% CI: 0.221–0.937; $p = 0.033$) and absence of tattoos (aOR = 0.283, 95% CI: 0.110–0.730; $p = 0.009$) were associated with lower risk of HBV infection, while unvaccinated individuals had 3.37-fold increased odds of getting the infection (95% CI: 1.395–8.142; $p = 0.007$). HBV prevalence declined progressively with increasing vaccine doses, from 9.2% in unvaccinated individuals to 2.3% among those who had completed three or more doses.

Conclusions: HBV infection is common among Kejetia market workers, with prevalence exceeding both continental and global estimates. Gender, tattooing, and vaccination status were significant predictors of infection. Strengthening adult vaccination programs, promoting safe tattooing practices, and implementing male-focused screening and prevention interventions are critical to reducing HBV burden and achieving Ghana's contribution to the WHO goal of eliminating HBV by 2030.

Keyword(s) : Hepatitis B virus; HBsAg prevalence; Vaccination; Predictors; Informal sector workers; Kejetia market; Ghana; Public health; Cross-sectional study

1441 - Disease control as evidenced by transient elastography in the ASSURE study 36 months of seladelpar treatment is associated with stable or improved liver stiffness in patients with primary biliary cholangitis

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Background / Hypothesis: Seladelpar is a first-in-class delpar (peroxisome proliferator-activated receptor [PPAR]-delta agonist) indicated for the treatment of primary biliary cholangitis (PBC) combined with ursodeoxycholic acid (UDCA) in adults with an inadequate response to UDCA, or as monotherapy in patients intolerant to UDCA. While seladelpar's efficacy in improving cholestatic markers is well established, its impact on liver stiffness measurements (LSMs), which typically worsen over time in PBC, remains underexplored. We report longitudinal LSM data in patients treated with seladelpar for up to 36 months (M) in an interim analysis of the ongoing, open-label Phase 3 ASSURE study (NCT03301506).

Materials and Methods: Patients enrolled in ASSURE from the placebo-controlled Phase 3 RESPONSE trial (NCT04620733) or earlier legacy seladelpar studies. LSM assessed by vibration-controlled transient elastography (FibroScan) was collected as an exploratory endpoint with local reads. This analysis included on-treatment LSM from patients with ≥ 1 post-baseline measurement, excluding values with a

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confirmed IQR/LSM ratio >30%. Baseline was defined at the time of seladelpar initiation. Patients were stratified by baseline LSM categories: <10.7, ≥10.7 and <16.9, and ≥16.9 kPa. LSM changes were assessed at 12M, 24M, and 36M, with category shifts assessed at 36M. Due to the non-normal distribution of LSM values, medians were reported. Data cutoff: Jan 31, 2025.

Results: A total of 311 patients were included in the analysis. LSM values at 36M were available in 114 patients. The overall median (IQR) LSM at baseline was 7.5 (5.9, 11.1) kPa. The median change in LSM was -0.2 (-1.7, 1.8) kPa, with a percent change of -2.9% (-22.8%, 25.9%). In patients with baseline LSM of <10.7 kPa, ≥10.7 and <16.9 kPa, or ≥16.9 kPa, median changes in LSM to 36M were +0.1 (-1.3, 1.8) kPa, -0.9 (-3.6, 4.8) kPa, and -5.2 (-10.6, 2.4) kPa, respectively. Similarly, median percent changes from baseline were +2.0% (-18.3, 26.1), -7.4% (-29.5, 38.7), and -29.7% (-51.5, 12.7). For category shifts, most patients (97/114 [85%]) were stable or improved at 36M. Patients whose LSM worsened by 30% or more from baseline were younger ($P < 0.05$), with otherwise comparable characteristics.

Conclusions: In this interim analysis, most patients maintained or improved their LSM category over 36M of seladelpar treatment. In the group at the highest risk for progression (≥16.9 kPa), there was a trend towards improvement with seladelpar.

Keyword(s) : FibroScan, liver stiffness, primary biliary cholangitis, seladelpar

1442 - Combination Therapy Using DPCAS Mode and DPMAS Mode of Artificial Liver Support System for Severe Liver Injury with Sepsis A Case Report

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Background / Hypothesis: Sepsis is characterized by

acute onset and rapid progression, which easily leads to multiple organ failure. The clinical treatment of severe liver injury complicated with sepsis is extremely challenging. The efficacy of the combined DPCAS and DPMAS artificial liver modes in such severe cases lacks verification by single-case studies, and this study aims to explore the clinical efficacy of this combined therapeutic strategy.

Materials and Methods: A 56-year-old female patient with severe liver injury complicated with sepsis (SOFA score of 8 at admission) admitted to our hospital in March 2025 was selected as the research subject. She was given meropenem 1 g intravenously (once every 8 hours) and methylprednisolone 40 mg intravenously (once daily) for anti-infection and anti-inflammatory treatment, and 2 sessions of combined DPCAS-DPMAS artificial liver therapy were performed simultaneously. The patient's clinical data, laboratory test indicators (procalcitonin, C-reactive protein, liver function indexes) and imaging examination results were collected for retrospective analysis.

Results: After treatment, the patient's procalcitonin decreased from 48.8 ng/mL to 0.074 ng/mL, and the levels of C-reactive protein and interleukin-6 returned to normal; liver function indexes such as alanine aminotransferase (909 U/L) and total bilirubin (152.3 μmol/L) all decreased to the normal range. The patient's symptoms such as fever and jaundice completely resolved, the imaging manifestations of pulmonary inflammation improved significantly, she was successfully discharged on the 12th day of admission, and her condition was stable with no abnormal indicators during the 6-week follow-up.

Conclusions: Combined DPCAS and DPMAS artificial liver therapy can effectively alleviate the systemic inflammatory response mediated by sepsis and improve impaired liver function, which is a safe and effective clinical scheme for the treatment of severe liver injury complicated with sepsis.

Keyword(s) : DPCAS; DPMAS; Artificial liver support system; Severe liver injury; Sepsis



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1443 - Seladelpar is associated with sustained reductions in cholestatic markers and a consistent safety profile in patients with primary biliary cholangitis up to 48 months in the ongoing open label ASSURE study

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Background / Hypothesis: Seladelpar is a first-in-class delpar (selective PPAR-delta agonist) indicated for the treatment of primary biliary cholangitis (PBC) in combination with ursodeoxycholic acid (UDCA) in adults with an inadequate response to UDCA, or as monotherapy in patients intolerant to UDCA. Here, we report the latest interim, long-term biochemical efficacy/safety data in a pooled population of patients treated with seladelpar in ASSURE.

Materials and Methods: ASSURE (NCT03301506) is an ongoing, open-label, long-term, Phase 3 trial of seladelpar in patients with PBC rolling over from the placebo-controlled, Phase 3 RESPONSE trial (NCT04620733) or with prior participation in legacy seladelpar trials. The Phase 2 and 3 parent studies for ASSURE required an inadequate response or intolerance to UDCA. Data cutoff: 1/31/2025. Patients were pooled starting with their initiation of seladelpar (at entry into RESPONSE for those randomized to seladelpar; at entry into ASSURE for all others). Patients received blinded, daily oral seladelpar 10 mg in RESPONSE and open-label, daily oral seladelpar 10 mg in ASSURE. Efficacy endpoints included composite biochemical response (CBR; alkaline phosphatase [ALP] $<1.67 \times$ the upper limit of normal [ULN], ALP decrease $\geq 15\%$ from baseline, and total bilirubin $\leq$ ULN), ALP normalization, ALP percent change from baseline, and other laboratory parameters summarized through 36 months (M). Safety was assessed by exposure-adjusted adverse events (AEs) per 100 patient-years of exposure through 48M.

Results: Among 337 patients treated with open-label seladelpar 10 mg, 258 were treated for ≥ 2 years and 117 were treated for ≥ 3 years, with 122 patients reaching their 36M visit by the data cutoff. Most patients achieved a CBR: 69% (225/325) at 12M, 70% (181/258) at 24M, and 67% (82/122) at 36M. ALP normalized in 35% of patients (114/325) at 12M, 37% (96/258) at 24M, and 34% (41/122) at 36M. Treatment with seladelpar resulted in sustained reductions in ALP, with mean percent changes from baseline of -43% at 12M, -43% at 24M, and -40% at 36M. Sustained reductions were also observed in alanine aminotransferase and gamma-glutamyl transferase through 36M. Seladelpar was well tolerated, with overall AEs reported in 83.2 patients per 100 patient-years in year 1 and 62.3 in year 4.

Conclusions: Seladelpar led to sustained reductions in biochemical markers of cholestasis with up to 36M of open-label treatment. Seladelpar also appeared to be safe and well tolerated; no new safety signals were identified with up to 48M of follow-up.

Keyword(s) : primary biliary cholangitis, seladelpar

1444 - Sustained and clinically meaningful improvements in moderate to severe pruritus patients with primary biliary cholangitis treated with seladelpar results from the ASSURE study up to 30 months

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Background / Hypothesis: Pruritus affects up to 80% of patients with primary biliary cholangitis (PBC) and significantly impairs quality of life. Seladelpar (SEL), a first-in-class delpar (selective peroxisome proliferator-activated receptor [PPAR]-delta agonist), is approved for second-line treatment of PBC and has shown efficacy in improving cholestasis and pruritus in the pivotal, placebo (PBO)-controlled RESPONSE study (NCT04620733). We report long-term pruritus outcomes from the RESPONSE study and interim results from its ongoing open-label extension study, ASSURE (NCT03301506), with confirmatory psychometric validation of the pruritus numeric rating scale (NRS).

Materials and Methods: In RESPONSE, pruritus NRS was collected daily for 6 months (M), then weekly for one week each month through 12M. Upon rollover to ASSURE, NRS was collected daily for 6M and then at scheduled clinic visits. Other measures included the 5-D Itch and PBC-40. Patients with moderate to severe pruritus (NRS ≥ 4) at baseline (MSPN) were analyzed based on randomization to SEL 10 mg or PBO in RESPONSE and reported as continuous or crossover SEL 10 mg groups in ASSURE. Regression analysis and Bland-Altman plots assessed agreement between single-visit and weekly averaged NRS scores. Robust psychometric validation of NRS included reliability, convergent validity, and meaningful within-patient change analyses. Change from baseline in NRS, 5-D Itch, and PBC-40 Itch domain scores were assessed in ASSURE with a data cut off of Jan 31, 2025.

Results: Overall, there were 49 SEL and 23 PBO MSPN patients at baseline in RESPONSE. Among them, 33 SEL and 17 PBO patients rolled over to ASSURE, and 27 and 16 patients reached 30M. Daily NRS was strongly correlated with weekly NRS (r [95% CI], 0.93 [0.90, 0.95]). Validation of the NRS demonstrated it was valid and reliable. Reduction in pruritus with SEL in RESPONSE was maintained in ASSURE up to 30M, with clinically meaningful NRS improvement observed in $\geq 50\%$ of patients upon SEL initiation for crossover patients in ASSURE from 15M through 30M. Mean (SE) 5-D Itch total and PBC-40 Itch domain scores were reduced from baseline in continuous SEL (-4.6 [0.7]; -3.3 [0.7]) and crossover SEL (-5.8 [1.1]; -4.3 [1.0])

patients.

Conclusions: SEL led to sustained, clinically meaningful improvement in pruritus among MSPN patients in RESPONSE and with up to 30 months of treatment. Additionally, repeated psychometric validation confirmed the reliability and validity of the NRS as an outcome measure of pruritus in PBC.

Keyword(s) : primary biliary cholangitis, pruritus, seladelpar

1445 - Sirtuin 3 ameliorates acute on chronic liver failure via the glutathione peroxidase 4 mixed lineage kinase domain like protein mediated ferroptosis necroptosis interplay

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) is a clinical syndrome defined by liver failure on preexisting chronic liver disease and is often associated with widespread hepatocyte death involving ferroptosis and necroptosis with high short-term mortality. Ferroptosis and necroptosis are two recently identified forms of non-apoptotic cell death, the role of ferroptosis and necroptosis in ACLF is not fully elucidated, evaluating the mode of cell death in ACLF will provide new insights for developing therapeutic strategies targeting cell death.

Materials and Methods:

Liver samples from six patients with acute-on-chronic liver failure (ACLF) were used. Bioinformatics analysis and establish the ACLF mouse model by combining chronic injury (chronic carbon tetrachloride [CCl₄] injection), acute hepatic insult (injection of a double dose CCl₄), and bacterial infection (intraperitoneal injection of *Klebsiella pneumoniae* [K.P.]), mice were also intraperitoneally pretreated with sirtuin 3 (SIRT3), mixed lineage kinase domain-like protein (MLKL), or glutathione peroxidase 4 (GPX4) inducers and inhibitors. Histological changes in liver tissue were monitored by hematoxylin and eosin staining. Mitochondrial morphology in murine hepatocytes was examined by transmission electron microscopy. Alanine aminotransferase (ALT) and aspartate aminotransferase

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(AST), glutathione, and iron levels were measured using commercial kits. Ferroptosis- and necroptosis-related protein and mRNA expression was detected by western blot and quantitative real-time polymerase chain reaction. SIRT3, GPX4, and MLKL were assessed by immunofluorescence analysis.

Results:

Bioinformatic analysis suggested potential crosstalk between ferroptosis and necroptosis in ACLF, possibly mediated by SIRT3. SIRT3, solute carrier family 7a member 11 (SLC7A11), and GPX4 protein expression was decreased and p53, acyl-CoA synthetase long-chain family member 4 (ACSL4), receptor interacting protein kinase (RIPK) 1, RIPK3, and MLKL levels were elevated in human ACLF liver tissue. In the ferroptosis and necroptosis inhibitor-treated groups demonstrated reduced serum transaminases (ALT, AST), diminished inflammatory mediators, decreased iron accumulation, attenuated hepatocellular injury, elevated GPX4 protein/fluorescence expression, and suppressed MLKL protein/fluorescence. In vitro co-immunoprecipitation confirmed a protein-protein interaction between GPX4 and MLKL. GPX4 overexpression lowered ALT, AST levels and substantially reduced MLKL expression, whereas GPX4 inactivation exacerbated necroptosis. Moreover, SIRT3 agonism alleviated ACLF-related liver injury and iron overload compared with results in the model group, concomitant with GPX4 upregulation and downregulation of p53, RIPK1, RIPK3, and MLKL. SIRT3 knockdown exacerbated both ferroptosis and necroptosis while aggravating hepatic damage in CCl₄/LPS-induced in vivo and in vitro models.

Conclusions:

SIRT3 activation attenuates CCl₄/K.P.-induced ferroptosis and necroptosis by inhibiting the GPX4/MLKL signaling pathway in ACLF.

Keyword(s) : sirtuin 3, ferroptosis, necroptosis, glutathione peroxidase 4/mixed lineage kinase domain-like protein, acute-on-chronic liver failure.

1446 - Metabolic dysfunction associated steatotic liver disease in patients with diabetes mellitus is linked with the severity of metabolic disorders.

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Introduction: MASLD is a metabolically determined disease. Studying the characteristics of various clinical variants of MASLD is important for developing approaches to treatment and prevention. The aim of the study was to identify clinical features in patients with DM with MASLD [MASLD(+)] compared to a control group with DM and without MASLD [MASLD(-)].

Materials and methods: A database of patients (n=5708) formed from 2021 to 2025 was analyzed. For the analysis, a cohort of patients with DM (n = 852) was selected; it included two subgroups: patients with MASLD (n = 686) and those without MASLD (n = 166). Pairs (n=91) of groups MASLD(+) and MASLD(-) were matched for sex, age, BMI, and cardiometabolic risk factors (hypertension, dyslipidemia). Vibration-controlled transient elastography with assessment of the controlled attenuation parameter (CAP) was used for determining the degree of steatosis and stage of liver fibrosis. Blood biochemical parameters characterizing lipid and carbohydrate metabolism were evaluated. The comparison of parameters was performed using the Wilcoxon criterion for continuous parameters and the χ^2 criterion for categorical parameters. The study was funded by a subsidy for the implementation of a state assignment (FGMF-2025-0003) of Federal Research Center of Nutrition and Biotechnology.

Results: The MASLD(+) and MASLD(-) groups did not differ by age (65.0 [58.5–70.0] vs. 64.0 [59.5–69.0] years; p=1.0), BMI (31.5 [29.8–35.8] vs. 31.3 [29.4–34.9] kg/m²; p=0.1), and percentage of women (79.1 vs. 79.1%; p=1.0). In the MASLD(+) group, compared with the MASLD(-) group, there was a larger waist circumference (101.0 [97.0 - 106.8] vs. 97.0 [90.0 - 102.0] cm; p=0.01), waist-to-hip ratio (0.94 [0.90–0.97] vs. 0.87 [0.84–0.94]; p=0.01), LDL (3.2 [2.2 - 3.9] vs. 2.5 [2.0 - 3.7] mmol/L; p=0.02), ALT (25.6 [18.0 - 40.6] vs. 19.6 [15.7 - 24.5] U/L; p<0.0001), total cholesterol (5.1 [4.0 - 5.9] vs. 4.2 [3.5 - 5.2] mmol/L; p=0.01). The two groups matched in fasting glucose (6.3 [5.4 - 7.2] vs. 5.9 [5.3 - 6.9] mmol/L; p=0.4), HbA1C (6.5 [6.0 - 7.3] vs. 6.2 [5.9 - 6.8]%; p=0.1), HOMA-IR (3.1 [2.2 - 4.0] vs. 2.5 [1.6 - 3.5]; p=0.3). In the MASLD(+) group, the MDRD GFR was lower (64.6 [46.9 - 77.0] vs. 71.0 [51.2 - 85.3] mL/min/1.73 m²; p=0.005) and the prevalence of F2-F4 liver fibrosis was higher (33,0 vs. 13,2%; p=0,002). Conclusion: patients with diabetes mellitus and MASLD have more pronounced metabolic disorders compared to the group with diabetes mellitus and without MASLD.

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Keyword(s) : diabetes mellitus, Metabolic dysfunction-associated steatotic liver disease, MASLD, hepatic steatosis, liver fibrosis

1448 - Insights from 10 years of Hepatitis C testing in Vancouver's inner city

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Background / Hypothesis:

Vancouver, Canada's inner-city, or downtown east side (DTES), is known for high rates of poverty, drug use, and Hepatitis C (HCV) infection, with up to 40% of individuals having HCV exposure. The Vancouver Infectious Diseases (VIDC) has been providing accessible point-of-care HCV testing and treatment to individuals living in this neighborhood for more than 10 years.

Materials and Methods:

VIDC provides regular point-of-care antibody testing in various inner-city locations including community centers, shelters, parks, and single room occupancy buildings. Data regarding HCV antibody and viremic status, as well as a range of other demographic and social variables is collected. We used odds ratios to determine characteristics significantly associated with HCV exposure from 2015 until September of 2025 when this analysis was completed.

Results:

A range of characteristics were found to be significantly associated with individuals with HCV exposure (presence of HCV antibodies). The most strongly associated characteristic was having a history of injection drug use. Smoking, poorer health status, being unemployed, having no post-secondary education, and having HIV were also significantly associated with having been exposed to HCV.

We did a separate analysis (students' t-test) in which we determined if the average age of individuals exposed to HCV was different from those not exposed to HCV in 2015, 2020, and 2025. We found no difference in 2015 or 2020, but saw that in 2025, individuals testing positive for HCV exposure were

significantly older than individuals with no exposure (48.6 YO vs. 44.5 YO; $p = 3.16E-07$).

Conclusions:

We identified many factors associated with HCV exposure, all of which are largely consistent with variables previously identified by other research groups. The observation that individuals without HCV exposure were significantly younger in 2025 may indicate that sustained treatment of HCV in this population is reducing the risk of HCV acquisition by individuals who have been using drugs for a shorter period of time. Follow-up analyses will investigate this finding further.

Keyword(s) : HCV, PWID, inner-city, demographics

1449 - Efficacy of low dose versus standard dose terlipressin in patients with variceal hemorrhage systematic review and meta analysis of randomized controlled trials.

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Background: Variceal hemorrhage remains the leading cause of cirrhotic mortality. Current guidelines recommend the use of terlipressin at a dose of 2 mg every 4 to 6 hours for 48 hours, followed by 1 mg every 4 to 6 hours for up to a maximum of 5 days, the minimum effective dose is undefined. Emerging evidence suggests lower doses may offer comparable efficacy to standard protocols, challenging current consensus due to limited data. The aim of this study is to compare the effects of lower doses of terlipressin versus standard doses in the treatment of acute variceal hemorrhage in adults with liver cirrhosis through a systematic review and meta-analysis of randomized clinical trials.

Materials and Methods: We searched Embase, PubMed and Cochrane database from inception to June 26, 2024 to identify randomized controlled trials comparing various doses of terlipressin with standard doses in individuals with cirrhosis for acute esophageal variceal bleeding. We defined standard dosing as a total dose >12 mg. The primary outcome was bleeding control. Secondary outcomes were early rebleeding and mortality. Risk of bias was assessed using the RoB 2 tool. Odds ratios (OR) and 95% confidence intervals

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were calculated, and heterogeneity was determined using I^2 . Registration number: CRD42024546649.

Results: Seven randomized clinical trials were included, with a total of 817 patients: 428 in the low-dose group and 389 in the standard-dose group. Three studies had a low risk of bias and four studies had a high risk of bias. No differences were observed in bleeding control (2 studies; OR 23.07; 95% CI: 0.00–960,436.71; $I^2 = 96.7\%$). Regarding the secondary outcome of early rebleeding, no differences were found between the low-dose and standard-dose groups (5 studies; OR 0.77; 95% CI: 0.34–1.74; $I^2 = 20.2\%$).

Conclusions: In this systematic review and meta-analysis, no differences were observed between low-dose and standard-dose terlipressin in the management of variceal bleeding in patients with cirrhosis.

Keyword(s) : Terlipressin, Variceal hemorrhage, Cirrhosis, Randomized controlled trials, Systematic review.

1450 - The Impact of Hepatic Steatosis on Viral Load and Liver Fibrosis in Patients with Chronic Hepatitis B

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Background: Metabolic dysfunction-associated steatotic liver disease (MASLD) and chronic hepatitis B (CHB) are prevalent chronic liver conditions. This study aimed to investigate the effects of hepatic steatosis on HBV viral load and fibrosis progression in CHB patients, and to identify risk factors for liver fibrosis.

Materials and Methods: A total of 887 CHB patients were divided into CHB-only (n=560) and CHB+MASLD (n=327) groups. The CHB+MASLD group was further stratified into mild (S1), moderate (S2), and severe (S3) steatosis subgroups based on Controlled Attenuation Parameter (CAP) values. The effects of hepatic steatosis on viral load and fibrosis were analyzed. Binary logistic regression was used to identify risk factors for liver fibrosis in CHB.

Results: The mean CAP values in the CHB-only and CHB+MASLD groups were 199.65 ± 42.60 and 264.76 ± 41.13 dB/m, respectively ($p < 0.01$). Compared to the CHB-only group, the CHB+MASLD group had lower serum pgRNA levels (1.88 vs. 2.27; $p = 0.005$) and

lower HBeAg-positive patients (24.3% vs. 30.7%, $p = 0.044$). In HBeAg-negative patients, serum HBsAg levels were lower in CHB+MASLD group (2.73 vs. 2.90, $p = 0.031$), and in HBeAg-positive patients, serum pgRNA levels were also lower (2.66 vs. 4.55, $p = 0.015$). CAP was negatively correlated with pgRNA ($r = -0.117$, $p < 0.01$). Liver fibrosis indices APRI (0.26 vs. 0.30, $p = 0.001$) and FIB-4 (1.05 vs. 1.36, $p < 0.001$) were lower in the CHB+MASLD group than in the CHB-only group. Subgroup analysis revealed that the S1 subgroup of CHB+MASLD had a higher proportion of patients without fibrosis (82.7% vs. 72.1%) and lower proportions with early fibrosis (8.3% vs. 14.8%), advanced fibrosis (5.8% vs. 7.8%), and cirrhosis (3.2% vs. 5.3%) compared to CHB-only group. The distribution of fibrosis stages in S2 subgroup of CHB+MASLD group and CHB-only group was similar (no fibrosis: 71.1% vs. 72.1%; early fibrosis: 15.5% vs. 14.8%; advanced fibrosis: 7.2% vs. 7.8%; cirrhosis: 6.2% vs. 5.3%). However, the S3 subgroup of CHB+MASLD had higher proportions of early fibrosis (16.1% vs. 14.8%), advanced fibrosis (10.3% vs. 7.8%), and cirrhosis (10.3% vs. 5.3%) than CHB-only group. Logistic regression showed that CAP, age, gender, HBV DNA, pgRNA, AST and GGT were independent influencing factors for liver fibrosis. Patients with severe steatosis (S3) had a 5.1-fold higher risk of fibrosis compared to those without steatosis (OR=5.05, 95%CI: 2.48–10.29, $p < 0.001$).

Conclusions: Patients with CHB and MASLD comorbidity exhibit lower hepatitis B viral loads. Mild to moderate steatosis does not accelerate fibrosis progression, whereas severe steatosis does. Hepatic steatosis is an independent risk factor for liver fibrosis in CHB patients.

Keyword(s) : Chronic hepatitis B, hepatic steatosis, liver fibrosis, viral load

1451 - Diagnostic challenge in a young male patient presenting with feeling of upper abdominal lump and recent onset of black terry stool

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Background / Hypothesis:

Non cirrhosis portal hypertension (NCPH) is poorly understood disease. Most of the patient is initially incorrectly labeled as having liver cirrhosis presenting as haematemesis or melaena. This patient came to outpatient department (OPD) with complaints of lump in abdomen and melaena. After evaluation he

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had no etiology of liver cirrhosis in serological investigation and liver biopsy. So we chose to write this case report.

Materials and Methods:

A 19-year-old boy came to out-patient department (OPD) of Hepatology department for evaluation of lump in upper abdomen. The patient complaining 3months history of gradually increasing upper abdominal lump mainly left upper abdomen associated with some abdominal discomfort, fatigue and black terry stool for 3 days. Abdominal lump was not associated with vomiting, abdominal pain, fever, altered bowel habit, weight loss. On query, he had no H/O fever, coughs and jaundice. For evaluation of patient we admitted this patient into hospital and underwent several investigations. His physical examination revealed mild anemia and splenomegaly, otherwise unremarkable in all systemic examinations. After getting esophageal varices we had done endoscopic esophageal ligation (EVL) and searched etiology of liver cirrhosis. As we found no etiology, then we planned for liver biopsy.

Results:

Based on splenomegaly, esophageal varices with normal LFT and liver biopsy report we reached to diagnosis of NCPH to this patient. Investigation reports revealed-

10.08.2022 - 1. CBC: Hb: 9.6gm/dl, WBC: 5500/cumm, Platelets: 115000/cumm; 2. LFT: Bilirubin: 2.8mg/dl, SGPT: 16.8U/L, SGOT: 14U/L, Albumin: 4.3gm/dl, PT: 12sec; 3. USG of W/A: Splenomegaly, mild ascites.

12.08.2022 - 1. Endoscopy of UGIT: Oesophageal varices; 2. HBsAg, Anti-HCV & Anti-Hbc (T): Negative; 3. Hb electrophoresis: Normal.

15.08.2022 - Ceruloplasmin: 32mg/dl; Ferritin: 72ng/ml; 24 hours urinary copper: 7.53ugm; ANA, Anti-LKM & ASMA: Negative.

30.08.2022 - Liver biopsy: Mild steatosis (HAI-2/18, Knodell score: 3/22)

Conclusions:NCPH is rare and diagnosed by exclusion of other cause of liver cirrhosis. Treatment focus on

complication of portal hypertension. But NCPH has better prognosis than cirrhosis of liver.

Keyword(s) : Upper abdominal lump; Non cirrhosis portal hypertension; Splenomegaly; Esophageal varices; Liver biopsy

1452 - Evaluation of Urinary Hepatitis E virus Antigen Colloidal Gold Immunochromatographic Assay in Clinical Diagnosis of Hepatitis E virus Infection

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Background / Hypothesis:Hepatitis E virus (HEV) is a significant public health concern worldwide. Current diagnostic methods for HEV infection have limitations in terms of accessibility and timeliness. To assess the diagnostic performance of the Wantai urinary HEV antigen (Ag) colloidal gold immunochromatographic assay (GICA) in HEV infection.

Materials and Methods:This prospective study enrolled 150 patients with suspected acute hepatitis E and 50 healthy controls. Paired urine, fecal, and serum samples were collected during initial clinical evaluation. Serum and fecal HEV RNA levels were quantified via reverse transcription-quantitative polymerase chain reaction (RT-qPCR), with genotyping performed by nested RT-PCR. Serum anti-HEV IgM/IgG levels were measured by Enzyme-Linked Immunosorbent Assay (ELISA), and urinary HEV antigen was detected using GICA. Diagnostic accuracy metrics were calculated against the reference standard of HEV RNA detection.

Results:HEV RNA was detected in 58% (87/150) of suspected cases. All successfully genotyped cases (69%, 60/87) were HEV genotype 4. All healthy controls tested negative for HEV RNA and urinary HEV Ag. The urinary HEV Ag GICA showed 98.9% sensitivity and 87.6% specificity, with high concordance with RT-qPCR (Kappa=0.85). Longitudinal follow-up revealed viral clearance and liver function normalization in most patients within 3-4 weeks post-symptom onset. 85.7% of patients achieved urinary HEV Ag negative conversion within 6-7 weeks, while anti-HEV IgM

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remained positive in all patients at follow-up conclusion.

Conclusions: Urinary HEV Ag GICA demonstrates high diagnostic reliability for acute HEV infection, offering a practical non-invasive option for resource-limited settings.

Keyword(s) : Hepatitis E virus; Antigen; Colloidal gold immunochromatographic assay; Diagnosis

1453 - The Impact of a Hospital Wide Alert and Referral Program on the Management of Hepatitis B Virus (HBV) Infection in Non Specialist Departments A Before and After Study

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Background / Hypothesis:

A significant proportion of patients with Hepatitis B virus (HBV) infection are initially identified in non-specialist departments. Successful linkage to specialist care is critical for initiating appropriate management. we implemented a hospital-wide "HBV Elimination Program" with an automated SMS (short message system) alert for HBsAg-positive patients and evaluated its impact in non- specialist departments.

Materials and Methods:

A before-and-after study was conducted in a large tertiary hospital. The intervention, initiated in January 21st, 2025, involved sending SMS alerts to all outpatients and inpatients testing positive for HBsAg, advising them to attend a specialist consultation. The primary outcome—including HBV DNA testing rates in HBsAg-positive patients, referral rates, and antiviral treatment rates—were compared between the pre-intervention (February to July 2024) and post-intervention (February to July 2025) periods.

Results:

A substantial proportion (51.9%) of viremic HBV patients were initially identified in non- specialist departments. Following the intervention, the volume of HBsAg tests, HBV DNA tests, and specialist visits in 2025 increased to 168,916, 28430 and 121,913, from 155,882, 22876 and 104,817 in 2024, respectively. The HBsAg positivity rate remained stable (9.1% in 2024 vs. 9.4% in 2025). Critically, the HBV DNA testing rate among HBsAg-positive patients in non- specialist departments improved significantly from 60.7% to

64.7%. Furthermore, the referral rate from non-specialist to specialist departments surged from 27.8% to 35.7% ($P < 0.001$). Most importantly, the antiviral treatment rate for eligible viremic patients in non-specialist departments dramatically increased from 50.7% to 64.9% ($P < 0.001$).

Conclusions:

This study demonstrates that a simple hospital-wide alert system can effectively strengthen the HBV care cascade. This intervention significantly improved rates of HBV DNA testing, specialist referral, and ultimately, antiviral treatment initiation. This model presents a highly effective strategy for closing the gap in HBV management and advancing towards elimination goals.

Keyword(s) : HBV, alert system, referral, elimination.

1454 - Impact of Autoimmune Phenomena on the Clinical Presentation and Prognosis of Wilson's Disease A Retrospective Cohort Study

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Background / Hypothesis:

Wilson's disease (WD) patients often display autoantibody positivity and/or elevated serum immunoglobulins, collectively termed autoimmune phenomena (AP). The clinical relevance of AP in WD, particularly regarding disease severity and prognosis, remains unclear.

Materials and Methods:

We retrospectively enrolled treatment-naïve WD patients (Leipzig score ≥ 4) and classified them as WD with AP (AP-WD) or WD without AP (NAP-WD) based on autoantibody positivity (titer $\geq 1:100$) and/or IgG above the upper limit of normal. Baseline laboratory data, clinical complications, and liver histopathology were compared. Patients received standard anti-copper therapy and were followed longitudinally to evaluate the impact of AP on liver-related outcomes.

Results:

Eighty-six treatment-naïve WD patients were included (48 AP-WD, 38 NAP-WD). At baseline, AP-WD patients exhibited more severe laboratory and clinical features, including lower platelet counts, reduced albumin, prolonged international normalized ratio, higher APRI,

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MELD/PELD, and Child-Pugh scores, and greater prevalence of ascites (all $P < 0.05$). Histopathological analysis demonstrated increased plasma cell infiltration and heightened portal inflammatory activity in the AP-WD group ($P < 0.05$). Longitudinal follow-up revealed that the presence of AP was independently associated with an increased risk of adverse liver-related events, including liver failure, transplantation, or death.

Conclusions:

AP is common in WD and correlate with more severe hepatic dysfunction and poorer long-term outcomes. Screening for autoantibodies and IgG levels in newly diagnosed WD patients may provide important prognostic insight and facilitate early risk stratification, guiding tailored monitoring and management strategies.

Keyword(s) : Wilson's Disease, Autoimmune, Prognosis, Clinical Features, Liver Histology

1455 - Cardiovascular Outcomes of GLP 1 Receptor Agonists vs SGLT2 Inhibitors in Metabolic Associated Steatotic Liver Disease and Type 2 Diabetes A Real World Comparative Effectiveness Study

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Background / Hypothesis:

Patients with metabolic dysfunction-associated steatotic liver disease (MASLD) and type 2 diabetes (T2D) face elevated cardiovascular risk. While both glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and sodium-glucose cotransporter-2 inhibitors (SGLT2is) reduce cardiovascular events, it remains unclear whether GLP-1 RAs confer greater protection than SGLT2is in this specific population. We aimed to compare the cardiovascular outcomes between these

two therapies in patients with MASLD and T2D.

Materials and Methods:

We conducted a retrospective cohort study using the TriNetX global health research network. Adults with T2D and MASLD initiating GLP-1 RA or SGLT2i therapy between 2017 and 2025 were identified. We performed 1:1 propensity score matching to balance demographics, comorbidities, and medications. The primary outcome was major adverse cardiovascular and cerebrovascular events (MACCE), defined as a composite of acute myocardial infarction, ischemic cerebrovascular events, and heart failure exacerbation. Cox proportional-hazards models were used to estimate hazard ratios (HRs).

Results:

After matching, 52,094 patients were analyzed (mean age 58.5 years). Over a median follow-up of 3.0 years, GLP-1 RA therapy was associated with a significantly lower risk of MACCE compared with SGLT2is (HR 0.86; 95% CI 0.80–0.92; $p < 0.001$). Secondary outcomes also favored GLP-1 RAs, including MACE (HR 0.83), heart failure exacerbation (HR 0.80), myocardial infarction (HR 0.68), stroke/TIA (HR 0.82), and all-cause mortality (HR 0.57) (all $p < 0.001$). Results were robust across landmark analyses and E-value estimation.

Conclusions:

In patients with MASLD and T2D, GLP-1 RA therapy was associated with a significantly lower risk of cardiovascular events compared with SGLT2is. These findings support prioritizing GLP-1 RAs in cardiometabolic risk management for this high-risk population.

Keyword(s) : MASLD; Type 2 diabetes; GLP-1 receptor agonists; SGLT2 inhibitors; Cardiovascular outcomes

1457 - Predictors for re hospitalization in liver cirrhosis the first longitudinal cohort study in the Republic of Moldova

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Background / Hypothesis:

Hospital readmissions for cirrhosis are considered an indicator of the quality of healthcare. Strategies

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applied to reduce hospital readmission rates for liver disease have not resulted in a significant decrease in the frequency of rehospitalization among patients with delta viral liver cirrhosis. The few studies involving European cohorts have identified different predictors. Aim: To evaluate rehospitalization as a pragmatic marker of insufficient monitoring in delta viral cirrhosis and to validate the predictors of readmission in the Moldovan cohort.

Materials and Methods:

We prospectively studied 131 patients with delta viral liver cirrhosis, discharged from a tertiary hospital and undergoing surveillance for a duration of ≥ 12 months. Data on demographic, laboratory, and disease-related risk factors were compiled. Twelve-month decompensation was defined as any of ascites, hepatic encephalopathy, or variceal hemorrhage. We used multivariate logistic regression to determine predictors of readmission.

Results:

Mean age of the study population (131 patients with viral delta cirrhosis) was 54.84 ± 9.54 years; 51.5% male. Over a 12-month period, 82% of patients presented at least one episode of decompensation, and 52% required (re)hospitalization. The results showed that male gender was associated with an approximately eightfold higher risk of decompensation compared to female gender (OR = 8.05; 95% CI: 2.01–213.66), portal thrombosis was a major determinant of decompensation (OR = 14.54; 95% CI: 1.03–552.00), and thrombocytopenia was associated with an approximately tenfold higher risk (OR = 10.41; 95% CI: 1.88–232.53). Analysis of the performance of the logistic model, assessed by the ROC curve, revealed a high discrimination capacity (AUC = 0.899), which confirms the value of the selected predictors in estimating the risk of decompensation.

Conclusions:

Rehospitalization is a pragmatic indicator of insufficient monitoring in delta viral cirrhosis. Our study is the first to validate some predictors of hospital readmission in viral delta liver cirrhosis. The results obtained provide useful information to design specific interventions in patients with liver cirrhosis and will help to mitigate readmissions.

Keyword(s) : rehospitalization, decompensation, liver cirrhosis

1458 - DIAGNOSTIC ACCURACY OF PIVKA II IN DETECTING HEPATOCELLULAR CARCINOMA AMONG AFP NORMAL PATIENTS A SYSTEMATIC REVIEW AND META ANALYSIS

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Hepatocellular carcinoma (HCC), which constitutes the majority of primary liver cancers, remains a major public health challenge and ranks among the top causes of cancer-related mortality globally and in the Philippines. Early detection continues to be constrained by the largely silent nature of early-stage disease. Current surveillance relying on ultrasonography with or without alpha-fetoprotein (AFP) offers limited diagnostic precision, with AFP demonstrating only moderate sensitivity and missing up to half of HCC cases. A substantial subset of patients—particularly those with smaller or early-stage tumors—present with normal AFP levels, creating a critical diagnostic blind spot. These limitations heighten the call for more reliable biomarkers capable of strengthening the performance of existing screening frameworks.

Protein Induced by Vitamin K Absence-II (PIVKA-II) has emerged as a high-performance biomarker demonstrating superior diagnostic metrics compared with AFP, especially in AFP-normal individuals who account for 30–50% of HCC presentations. With reported sensitivity and specificity approaching 75% and 92%, respectively, PIVKA-II offers a promising avenue for closing gaps inherent in AFP-based surveillance.

This meta-analysis sought to evaluate the diagnostic accuracy of PIVKA-II relative to AFP in detecting HCC specifically among AFP-normal patients, with emphasis on sensitivity, specificity, and area under the curve (AUC), including for early-stage disease. Four peer-reviewed case-control studies published from 2016 to 2025 and comprising at least 146 AFP-normal patients were systematically identified through PubMed. Inclusion criteria focused on studies assessing diagnostic performance metrics for PIVKA-II. All studies underwent structured quality appraisal using the QUADAS-2 framework. Statistical synthesis was conducted using Meta-DiSc (v1.4), applying a random-effects model to account for interstudy heterogeneity.

Results demonstrated that PIVKA-II consistently



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outperformed AFP across diagnostic endpoints. For any-stage HCC, pooled sensitivity reached 80%, specificity 90%, and the AUC 0.9376, underscoring robust overall accuracy. In early-stage disease, PIVKA-II achieved a sensitivity of 79%, specificity of 82%, and an AUC of 0.888, confirming its strong performance even in the most diagnostically challenging subgroup.

Collectively, these findings reinforce PIVKA-II as a highly effective biomarker for detecting HCC among AFP-normal patients—an area where AFP alone falls short. Its integration into surveillance algorithms offers a strategic opportunity to enhance early detection, accelerate clinical decision-making, and improve long-term survival outcomes. Given its superior diagnostic profile, PIVKA-II warrants strong consideration for routine adoption in multimodal HCC screening and monitoring programs.

Keyword(s) : Alphafeto-protein (AFP)-Normal Hepatocellular Carcinoma, PIVKA-II, Hepatocellular Carcinoma (HCC)

1460 - Dysregulated S100A8 A9–Monocyte Pathway in Cirrhosis Associated Immune Dysfunction and Its Reversal by Mesenchymal Stromal Cells

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Background / Hypothesis: Decompensated liver cirrhosis (DLC) is marked by cirrhosis-associated

immune dysfunction (CAID), characterized by concurrent systemic inflammation and immune deficiency. Monocytes are key drivers of CAID. The calcium-binding proteins S100A8/A9, highly expressed by activated monocytes, promote their recruitment and activation via TLR4 and RAGE, amplifying inflammatory cytokine and chemokine production. Mesenchymal stromal cells (MSCs) exhibit immunomodulatory properties, yet their specific effects on the S100A8/A9–monocyte axis in human CAID remain poorly defined. Materials and Methods: Using single-cell RNA sequencing and CyTOF data from a clinical cohort of MSC-treated DLC patients, we evaluated the effects of MSC infusion on monocyte immunophenotypes and S100A8/A9 expression. Circulating and liver tissue samples from DLC patients and healthy controls were analyzed alongside a CCl₄-induced cirrhotic mouse model. In vitro coculture experiments using patient-derived monocytes further examined MSC-mediated regulation of the S100A8/A9 pathway through RT-qPCR, flow cytometry, and transcriptomic profiling. Results: DLC patients showed increased proportions, absolute counts, and activation of peripheral CD14⁺ monocytes compared with healthy controls. These monocytes exhibited marked overexpression of S100A8/A9 and increased hepatic infiltration of S100A8/A9⁺ monocyte–macrophages in fibrotic regions. Single-cell data revealed a pro-inflammatory S100A8/A9^{high} subset enriched in leukocyte activation and cytokine-secretion pathways, whereas the S100A8/A9^{low} subset was enriched in antigen presentation and interferon- γ responses. Following MSC infusion, peripheral monocyte proportions declined, accompanied by a transcriptomic shift toward a S100A8/A9^{low} state. In vitro, MSCs suppressed monocyte activation markers and reduced S100A8/A9 mRNA expression. In the CCl₄ model, MSC treatment reduced fibrosis and decreased hepatic infiltration of S100A8/A9⁺ monocyte–macrophages. Conclusions: DLC is associated with increased monocyte activation and elevated monocyte-derived S100A8/A9 in the circulation and liver. MSC therapy appears to attenuate these abnormalities by downregulating the S100A8/A9–monocyte axis, providing mechanistic insight into its therapeutic potential in CAID.

Keyword(s) : Decompensated liver cirrhosis; Cirrhosis-Associated Immune Dysfunction; Monocyte; Mesenchymal stromal cells; S100A8/A9

1462 - Effectiveness and safety of tenofovir alafenamide for chronic hepatitis B patients with

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decompensated cirrhosis a retrospective study in Qinghai province

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Background/Hypothesis: An increasing amount of evidence has demonstrated the effectiveness and safety of tenofovir alafenamide fumarate (TAF) in patients with chronic hepatitis B (CHB) and decompensated cirrhosis. However, there is limited long-term data on the effectiveness and safety of TAF. This study aimed to investigate the long-term effectiveness and safety of TAF in CHB patients with decompensated cirrhosis.

Materials and Methods: This single-center, retrospective study enrolled a total of 63 CHB patients with decompensated cirrhosis who were treated with TAF 25mg daily. The follow-up period lasts for 96 weeks. The primary endpoint of this analysis is the proportion of patients with an undetectable HBV DNA level (less than 5 IU/ml) at week 96. Other endpoints included the changes in Child-Pugh (CTP) and Model for end-stage liver disease (MELD) scores, and changes in ascites and hepatic encephalopathy (HE) from baseline. The safety profile, including drug-related adverse events (AEs) or serious adverse events (SAEs), and clinical outcomes such as liver transplantation, hepatocellular carcinoma (HCC) or death were also analyzed, as was renal function.

Results: Sixty-three CHB patients with decompensated cirrhosis were enrolled between January 2023 and February 2025, and were included in the analysis. Of the participants, 79.37% (n=50) were male, with a mean age was 55.24±8.58 years. The median baseline values for HBV DNA and ALT at baseline were 3.45×10⁶ IU/mL and 26.00 U/L, respectively. The mean values for CTP and MELD were 9.19±1.88 and 11.32±3.36, respectively. All 63 patients completed the 96-week follow-up. After 96 weeks of TAF treatment, 92.06% (58/63) patients had undetectable HBV DNA. The mean ALT level remained stable, decreasing from 26.00 U/L at baseline to 22.00 U/L after 96 weeks. The CTP score maintained stable from 9.19 at baseline to 9.49 at week 96 (P>0.05), and the mean MELD score significantly declined from 11.32 at baseline to 6.98 at week 96 (P<0.01). 50.79% (32/63) of patients had mild to moderate ascites at baseline, and this proportion decreased to 42.86% (27/63) by week 96 (P>0.05). HE occurred in 23 patients at baseline and was significantly reduced to 11

patients at week 96 (P<0.01). No drug-related AEs or SAEs were detected during the follow-up period, neither did liver transplantation, HCC, or death. During the 96 weeks of treatment, renal function, as indicated by serum creatinine (Scr) and β 2 microglobulin (β 2-MG), kept stable during the treatment.

Conclusions: This study demonstrated the long-term effectiveness and safety of TAF therapy for CHB patients with decompensated cirrhosis.

Keyword(s) : tenofovir alafenamide fumarate, chronic hepatitis B and decompensated cirrhosis.

1464 - Liver Fibrosis and Cardiovascular Health Jointly Predict Mortality in NAFLD and MAFLD

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Background / Hypothesis: The interaction between cardiovascular health (CVH) and liver fibrosis progression in patients with nonalcoholic fatty liver disease (NAFLD) remains insufficiently elucidated. This study aimed to investigate the joint predictive value of CVH scores and liver fibrosis assessment tools (NFS and FIB-4) for cardiac and all-cause mortality.

Materials and Methods: This cohort study utilized data from the US National Health and Nutrition Examination Survey (NHANES) 2005–2018, including 25,634 participants with NAFLD. Liver fibrosis scores (NFS and FIB-4) were stratified into low, intermediate, and high categories, while CVH was classified as ideal, intermediate, or poor according to the "Life's Essential 8" score. Weighted Cox proportional hazards models were applied to estimate hazard ratios (HRs), with interaction terms incorporated to evaluate the modifying effect of CVH. Study outcomes were all-cause mortality and cardiac mortality.

Results: Higher liver fibrosis scores (NFS and FIB-4) were consistently associated with increased mortality risk. During a median follow-up of 91 months, 2,185 deaths occurred, including 540 cardiovascular deaths. Elevated fibrosis scores showed graded associations with all-cause mortality, and favorable cardiovascular health (CVH) attenuated this risk, whereas poor CVH further amplified it. For cardiovascular mortality, both NFS and FIB-4 remained significant predictors, and the

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interaction between fibrosis scores and CVH was statistically significant ($P < 0.001$). Individuals with high fibrosis scores and poor CVH showed the highest cardiovascular mortality risk.

Conclusions: Liver fibrosis assessed by NFS and FIB-4 was independently associated with both all-cause and cardiovascular mortality. For all-cause mortality, higher NFS and FIB-4 levels showed a significant and graded increase in risk, with notable risk elevation at $\text{NFS} \geq 1$ and $\text{FIB-4} > 1.5$, and these associations were stronger in individuals with poor cardiovascular health. For cardiovascular mortality, NFS and FIB-4 were both significant predictors, with NFS showing early separation in survival and FIB-4 demonstrating risk divergence after approximately 40 months. In MAFLD, FIB-4 and NFS remained consistently associated with cardiovascular mortality, with higher predictive strength compared with non-MAFLD participants.

Keyword(s) : NAFLD; MAFLD; Liver Fibrosis; FIB-4; NFS; Cardiovascular Health; All-Cause Mortality; Cardiovascular Mortality

1465 - Impact of metabolic dysfunction-associated steatotic liver disease on young hepatocellular carcinoma patients following curative resection

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Background / Hypothesis: The prognostic impact of metabolic dysfunction-associated steatotic liver disease (MASLD) in hepatocellular carcinoma (HCC) remains controversial, particularly among younger patients. We investigated the clinicopathological features and survival outcomes of young HCC patients with and without MASLD after curative resection.

Materials and Methods: We retrospectively analyzed 289 treatment-naïve HCC patients aged ≤ 60 years who underwent curative resection between 2016 and 2022. Patients were categorized as non-MASLD ($n=154$) or MASLD ($n=135$) based on non-tumor liver histology.

Overall survival (OS) was the primary endpoint; recurrence-free survival (RFS) was secondary. Survival analyses were performed using Kaplan-Meier and Cox proportional hazards models.

Results: Compared with the non-MASLD group, patients with MASLD had higher body mass index, more cardiometabolic risk factors, lower aspartate aminotransferase (AST) and alpha-fetoprotein levels, earlier tumor stage, and smaller tumor size. After a median follow-up of 59.2 months, 56 deaths were recorded, with a 5-year OS of 76.9% overall. MASLD patients had significantly better 5-year OS (84.2% vs. 70.1%, $p=0.018$) and RFS (51.3% vs. 37.0%, $p=0.010$). However, in multivariate Cox analysis, MASLD was not an independent predictor of OS or RFS. Larger tumor size ($> 5\text{cm}$) independently predicted poorer OS (hazard ratio 2.10, 95% confidence interval 1.14–3.86, $p=0.017$), while elevated AST, larger tumor size, and microvascular invasion were independent predictors of poorer RFS.

Conclusions: Young HCC patients with MASLD presented with more favorable tumor characteristics and showed superior unadjusted OS and RFS after curative resection. However, MASLD itself was not an independent prognostic factor after adjustment, with tumor burden remaining the key determinant of outcomes.

Keyword(s) : MASLD, young HCC

1467 - Clinical and instrumental profile of patients with Cryptogenic steatotic liver disease

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Clinical and instrumental profile of patients with cryptogenic steatotic liver disease

Introduction: In 2023, a revised classification of disorders presenting with hepatic steatosis was introduced, establishing several new diagnostic categories, including cryptogenic steatotic liver disease (CSLD). **Objective:** to determine the prevalence of CSLD

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within the spectrum of steatosis-associated liver diseases and to characterize its clinical and instrumental features.

Materials and Methods. A retrospective analysis was conducted using a database of 4,673 patients who underwent evaluation for liver steatosis and fibrosis. Diagnostic criteria for CSLD and other liver diseases were applied in accordance with the recommendations of the European Association for the Study of the Liver. The degree of steatosis and the stage of fibrosis were assessed using vibration-controlled transient elastography with measurement of the controlled attenuation parameter. Body composition was evaluated by bioimpedance analysis. Laboratory assessments included indicators of lipid and carbohydrate metabolism. The study was funded by a subsidy for the implementation of a state assignment (FGMF-2025-0003) of Federal Research Center of Nutrition and Biotechnology.

Results: The prevalence of CSLD was 0.8% (n = 22) among patients with confirmed hepatic steatosis (n = 2,643). Compared with individuals with metabolic dysfunction-associated steatotic liver disease (MASLD), patients in the CSLD group exhibited significantly lower rates of grade 3 steatosis (S3: 27.3% vs. 67.6%; $p < 0.001$) and advanced fibrosis (F2–F4: 9.1% vs. 29.3%; $p = 0.02$). When compared with a control group without hepatic steatosis and fibrosis (n = 266), patients with CSLD had a higher body mass index (median [Q1–Q3]: 24.1 [22.1–24.5] kg/m² vs. 22.4 [20.7–23.7] kg/m²; $p = 0.008$), primarily due to greater fat mass (19.0 [16.6–26.7] kg vs. 16.4 [12.2–20.5] kg; $p = 0.03$). No significant differences were observed between the CSLD and control group without steatosis and fibrosis in age, sex distribution, lipid profile parameters, or carbohydrate metabolism.

Conclusion: CSLD is a rare condition. Patients with CSLD differ from the control group only in terms of fat mass, despite both groups exhibiting body mass index values within the normal range. Parameters of lipid and carbohydrate metabolism in the CSLD cohort do not differ significantly from those of practically healthy individuals.

Keyword(s) : cryptogenic steatotic liver disease, CSLD, metabolic associated steatotic liver disease, MASLD, hepatic steatosis, liver fibrosis

1470 - Hepatitis E virus orchestrates mitochondrial fusion to drive viral replication through mechanistic coupling of pro viral autophagy and innate immune evasion

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Background / Hypothesis:

Hepatitis E virus (HEV) represents a major cause of acute hepatitis globally, posing a significant clinical threat to immunocompromised individuals and pregnant women. Mitochondrial dynamics—the balance of fission and fusion—are increasingly recognized as central regulators of antiviral responses. This study systematically investigates whether HEV reprograms mitochondrial architecture to facilitate its replication and immune evasion.

Materials and Methods:

We performed immunohistochemistry and immunofluorescence on liver biopsies from HEV-infected patients to evaluate mitochondrial ultrastructure. Acute and chronic HEV infection models were established in hepatoma cell lines. Mitochondrial morphology was assessed using electron microscopy and confocal imaging. Viral replication, autophagic flux, and transcriptomic profiles were analyzed through genetic knockdown, pharmacological inhibition, and RNA sequencing.

Results:

Analysis of human liver biopsies revealed significant mitochondrial aggregation in 65% of acute HEV patients, which correlated strongly with elevated serum aminotransferase levels and disease severity. HEV infection consistently induced pronounced mitochondrial fusion across all cell models, mediated by a biphasic regulatory program: acute infection preferentially upregulated the inner membrane fusion GTPase OPA1, while chronic infection selectively enhanced the outer membrane mediator MFN1. Genetic ablation of either OPA1 or MFN1 reversed mitochondrial elongation and significantly suppressed HEV replication in both acute and chronic models. Integrated transcriptomic analyses demonstrated that depletion of OPA1 or MFN1 disrupted core pathways involved in autophagy and innate immune signaling. Specifically, OPA1 knockdown during acute infection inhibited pro-viral autophagy and enhanced interferon-stimulated gene (ISG) expression, whereas MFN1 knockdown exerted parallel effects in chronic infection. Pharmacological inhibition of autophagy with 3-methyladenine (3MA) substantially attenuated autophagic flux and suppressed HEV replication.

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Furthermore, HEV infection induced G0/G1 cell cycle arrest, which facilitated mitochondrial fusion and enhanced viral replication. Clinically, HEV ORF2 protein levels positively correlated with OPA1 and MFN1 expression in patient liver tissues.

Conclusions:

Our study establishes mitochondrial fusion as a critical host pathway co-opted by HEV to promote viral replication through coordinated activation of pro-viral autophagy and suppression of innate immune responses. These findings reveal a novel viral strategy and highlight mitochondrial fusion machinery as a promising therapeutic target against hepatitis E.

Keyword(s) : HEV; anti-virus; mitochondrial dynamics

1474 - Deciphering the mechanistic underpinnings of hepatitis E severity a tale of T cell exhaustion and maladaptive cDC2 activation at single cell resolution

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Background / Hypothesis:

Hepatitis E virus (HEV) infection is the most common cause of acute viral hepatitis worldwide. Although typically self-limiting in immunocompetent hosts, a subset of patients progresses to fulminant hepatitis, which carries substantial mortality. The immune mechanisms underlying severe HEV infection remain incompletely elucidated. Using single-cell RNA sequencing (scRNA-seq), we sought to delineate the peripheral immune landscape and identify transcriptional programs associated with immunopathology versus protective immunity in severe HEV infection.

Materials and Methods:

We performed scRNA-seq on 20 peripheral blood mononuclear cell samples, including acute hepatitis E (AHE) patients (n=8), HEV-induced acute liver failure (HEV-ALF) patients (n=8,) and healthy controls (HCs) (n=4). Integrated bioinformatic analysis comprise differential expression, pathway enrichment, trajectory inference, and cellular communication mapping to delineate immune heterogeneity and dynamic changes across disease severity.

Results:

Unsupervised clustering identified 23 distinct transcriptomic clusters, corresponding to six major immune lineages. Oxidative phosphorylation was consistently enriched across all immune subsets in HEV-infected patients compared to HCs. Compared to AHE, ALF group exhibited pronounced activation of inflammatory pathways, including NOD-like receptor, TNF, NF- κ B, and IL-17 signaling. Further stratification of T and NK cells revealed nine functionally distinct subsets. IL-17 signaling was uniquely enriched in naïve T cells from HEV-infected patients versus HCs, but became broadly activated across all NK/T cell subsets in ALF. CD8⁺ effector memory T cells (TEM) were expanded in both AHE and ALF. Strikingly, HLA-DR⁺CD38⁺CD8⁺ TEM cells were markedly increased in AHE compared to HCs and ALF. Furthermore, CD8⁺ TEM cells in ALF displayed significant downregulation of cytotoxic effector molecules (GZMA, GZMH, GNLY, PRF1) compared to AHE. Pseudotime trajectory analysis indicated that CD8+TEMRA-lineage cells accumulated terminally in AHE, whereas ALF was characterized by enrichment along a CD8+TEM-differentiated trajectory. Additionally, cDC2 frequencies were elevated in both AHE and ALF groups. Within cDC2, ALF group showed enhanced MHC-II pathway activity and upregulated expression of all eight HLA class II genes, accompanied by the highest module score for HLA-II among myeloid subsets.

Conclusions:

Our findings delineate a pathognomonic immune signature for HEV-induced acute liver failure, characterized by broad inflammatory activation coupled with a state of CD8⁺ TEM cell paralysis and maladaptive cDC2-driven antigen presentation. This specific combination of excessive inflammation and impaired effector function provides a mechanistic basis for disease severity, revealing novel therapeutic targets to restore immune homeostasis in severe hepatitis E.

Keyword(s) : HEV, scRNA-seq, Inflammation, immune response

1475 - Serum cystathionine γ lyase (CTH) as a novel noninvasive biomarker for occult nonalcoholic steatohepatitis

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Background / Hypothesis:In nonalcoholic fatty liver disease (NAFLD), approximately 15%-40% of patients with normal liver function tests actually have occult NASH, whose risks of liver inflammation and fibrosis are often underestimated. There is currently a clinical lack of accurate noninvasive biomarkers for assessing hepatic inflammatory status. We found that cystathionine γ -lyase (CTH) gene expression is specifically upregulated during hepatic inflammation and hypothesized that its protein product, released into the bloodstream, could serve as a specific biomarker reflecting the true severity of liver inflammation.

Materials and Methods:The study enrolled 60 patients with biopsy-confirmed occult NASH and normal liver function. Serum CTH protein concentration was measured using ELISA and correlated with histological inflammatory activity grades (G-scores, 1-4). Traditional inflammatory markers including IL-6, IL-10, and CRP were also detected for comparison. All enrolled patients underwent regular longitudinal follow-up. Serial FibroScan examinations, abdominal CT imaging evaluations, and FIB-4 score calculations were performed to dynamically monitor liver fibrosis progression.

Results:Correlation analysis revealed a significant positive correlation between serum CTH protein levels and the degree of histological liver inflammation (correlation coefficient $r = 0.78$, $P = 0.000$). The diagnostic performance of CTH surpassed that of traditional inflammatory markers such as IL-6, IL-10, and CRP. Longitudinal follow-up data over an average observation period of 24 months showed that 70% of patients with persistently high serum CTH levels exhibited definite liver fibrosis progression. In contrast, no significant deterioration in fibrosis was observed in patients maintaining lower CTH levels.

Conclusions:Serum CTH protein levels are closely associated with the severity of liver inflammation in patients with occult NASH and can effectively predict the risk of fibrosis progression. These findings suggest that serum CTH protein holds promise as a valuable noninvasive biomarker for assessing hepatic inflammatory status and predicting disease outcomes in occult NASH patients, offering a new direction for early identification of high-risk individuals and implementation of personalized interventions.

Keyword(s) : Nonalcoholic steatohepatitis

Cystathionine γ -lyase; Biomarker; Liver inflammation;
Noninvasive diagnosis

1477 - A post authorization study to evaluate the safety and efficacy of sofosbuvir velpatasvir voxilaprevir in the retreatment of chronic hepatitis C patients in Taiwan

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Background / Hypothesis:

Direct-acting antiviral (DAA) agents have been approved for hepatitis C virus (HCV) treatment, providing higher rates of sustained virologic response (SVR) and fewer side effects. However, some patients have experienced treatment failure with prior DAA-based regimens. Vosevi, a pan-genotypic fixed-dose combination of sofosbuvir/velpatasvir/voxilaprevir, is indicated for patients with chronic HCV infection who previously failed DAA therapy. The Taiwan Food and Drug Administration (TFDA) requested a post-

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authorization study (PAS) to evaluate the safety and efficacy of Vosevi in the Taiwanese population.

Materials and Methods:

This multicenter observational study enrolled DAA-experienced patients who received a 12-week course of Vosevi between September 2021 and August 2024. Data were obtained from the Taiwan Association for the Study of the Liver (TASL) HCV Registry (TACR) database. All adverse events (AEs) were collected to evaluate the safety profile of Vosevi, while SVR at 12 weeks post-treatment (SVR12) was used to assess efficacy.

Results:

A total of 116 patients were enrolled across 15 sites in Taiwan. In the safety analysis set, 52 patients experienced 119 AEs (incidence: 44.8%; 95% CI: 36.1–53.9), corresponding to 2.0 events per person-year (95% CI: 1.5–2.7). Most AEs (97.5%) were non-serious. The most common AEs were gastrointestinal disorders (n=33, 28.4%), infections and infestations (n=14, 12.1%), and nervous system disorders (n=12, 10.3%). Two patients (1.7%) experienced serious adverse events (SAEs), and one patient (0.9%) experienced an adverse drug reaction (ADR). No serious ADRs, deaths, hyperbilirubinemia, or drug-induced liver injury (DILI) were reported.

Among 107 patients who completed treatment and follow-up with available SVR12 data, the SVR12 rate was 98.1% (105/107) in the modified full analysis set (mFAS). The SVR12 rates were consistently high across all HCV genotypes and comorbidities, including 100% among patients with genotype 3 or 6, liver cirrhosis, HBV/HIV coinfection, and those who underwent liver transplantation.

Conclusions:

A 12-week regimen of Vosevi (SOF/VEL/VOX) was safe, well-tolerated, and highly effective in DAA-experienced Taiwanese patients with chronic HCV infection. No new safety concerns were identified in this post-authorization study.

Keyword(s) : Hepatitis C virus (HCV), Retreatment

1478 - Unveiling Pyruvate Metabolism A Prognostic and Immunotherapeutic Beacon in Hepatocellular Carcinoma

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Background / Hypothesis:Pyruvate metabolism is crucial in the development and advancement of many types of human cancers. This study sought to investigate the prognostic and immunotherapeutic predictive value of pyruvate metabolism-related genes in hepatocellular carcinoma (HCC).

Materials and Methods:According to The Cancer Genome Atlas (TCGA) database, a machine-learning prognostic risk model was developed using the least absolute shrinkage and selection operator (LASSO) algorithm, based on pyruvate metabolism-gene signatures. The model's predictive accuracy was additionally confirmed with an independent dataset from the International Cancer Genome Consortium (ICGC) database. Consensus clustering was utilized to categorize HCC patients into subgroups according to pyruvate metabolism-gene signatures, with subsequent analyses of differential gene expression, clinical outcomes, and immune characteristics.

Results:In this study, 69 genes related to pyruvate metabolism were identified, and 13 of these genes were screened to create a risk score model through the LASSO regression method. The model effectively categorized HCC cohorts from the TCGA database into high- and low-risk groups, with the high-risk group representing notably poorer prognosis. The model's prognostic accuracy was further confirmed in HCC cohorts from ICGC database. HCC patients were stratified to two subgroups, C1 and C2, according to gene signatures related to pyruvate metabolism. Comparative analysis identified 1,907 differentially expressed genes (DEGs), comprising 1,539 upregulated and 368 downregulated genes. Patients in the C2 subgroup showed enhanced clinical outcomes and a superior response to immunotherapy relative to the C1 subgroup.

Conclusions:This study uncovered that pyruvate metabolism-related gene signatures served as reliable predictors of prognosis and immunotherapy response in HCC patients.

Keyword(s) : Hepatocellular carcinoma, pyruvate metabolism, prognosis, tumor microenvironment, immunotherapy response



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1479 - Disulfidptosis in Acute Liver Failure Roles Molecular Mechanisms and Potential Clinical Applications

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Background / Hypothesis:

Acute liver failure (ALF) is a critical clinical emergency, but its pathogenesis has not been fully elucidated. Cell-free therapeutic strategies based on exosomes have received increasing attention in ALF treatment. Disulfidptosis, a newly discovered cell death modality in 2023, has not been systematically studied in ALF. It is urgent to explore new mechanisms of ALF pathogenesis and develop new effective alternative therapeutic strategies.

Materials and Methods:

D-galactosamine/lipopolysaccharide (D-GalN/LPS) was used to induce BALB/c mice and Alpha mouse liver 12 (AML12) cells respectively to construct in vivo and in vitro ALF models. Subsequently, HYX1-derived exosomes (HYX1-Exos) were administered to treat the ALF models. By detecting glucose and cysteine content, NADPH levels, and using qPCR, Western blot and immunohistochemistry to detect disulfidptosis-related gene and protein expression, the occurrence of disulfidptosis in the in vivo and in vitro ALF models was confirmed and the therapeutic effect of HYX1-Exos was evaluated. Meanwhile, bioinformatics methods were used to screen hub genes closely related to disulfidptosis in ALF patients, and validation was performed in the ALF model group and HYX1-Exos treatment group. In addition, RNA sequencing was performed on HYX1-Exos to screen potential functional miRNAs, their therapeutic effects in the in vitro ALF model were verified by transfecting miRNA mimics, and their in vivo efficacy in the in vivo ALF model was evaluated by tail vein injection of miRNA agomir.

Results:

Multiple indicators showed that disulfidptosis plays an important role in the in vivo and in vitro ALF models. HYX1-Exos had significant protective effects on D-GalN/LPS-induced ALF models, and could effectively inhibit the occurrence of disulfidptosis in vitro and in vivo. Through bioinformatics and machine learning analysis, two key genes, SLC7A11 and IQGAP1, were screened out, and the important role of SLC7A11 and IQGAP1-mediated disulfidptosis in ALF pathogenesis was confirmed. Further studies showed that HYX1-Exos alleviated ALF injury by inhibiting SLC7A11 and IQGAP1-mediated disulfidptosis. miRNA screening

identified miR-4492 as the key miRNA in HYX1-Exos targeting disulfidptosis, which showed significant therapeutic effects in both the in vivo and in vitro ALF models and could effectively inhibit the expression of SLC7A11 and IQGAP1.

Conclusions:

This study revealed that disulfidptosis participates in the pathogenesis of ALF through molecular mechanisms mediated by SLC7A11 and IQGAP1. HYX1-Exos effectively alleviates ALF liver injury by targeting and inhibiting the expression of disulfidptosis-related genes through its carried miR-4492, providing new theoretical basis and potential therapeutic strategies for the clinical treatment of ALF.

Keyword(s) : Acute liver failure; Disulfidptosis; Exosomes; SLC7A11; IQGAP1

1480 - Baseline Screening and Epidemiological Characteristics of First Degree Relatives of HBV Related Hepatocellular Carcinoma Patients A Multicenter Prospective Study in Jiangsu China

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Background / Hypothesis:

First-degree relatives of patients with HBV-related hepatocellular carcinoma (HCC) constitute a high-risk population for chronic hepatitis B virus (HBV) infection. However, real-world screening uptake and antiviral treatment status in this group remain insufficiently characterized. We hypothesize that a structured program integrating screening, diagnosis, treatment, and tailored health education among first-degree relatives of HBV-related HCC patients will increase HBV detection and antiviral treatment uptake, thereby reducing risk of disease progression. This study reports the baseline findings of an ongoing multicenter prospective investigation aiming to establish an integrated screening–diagnosis–treatment–education model to improve HBV management in this high-risk population.



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Materials and Methods:

This multicenter, prospective observational study was conducted across eight medical centers in Jiangsu Province, China. All first-degree relatives of index patients with HBV-related HCC were consecutively enrolled. Baseline assessments included demographic characteristics, HBV serostatus (HBsAg), antiviral treatment status, and family history of HCC. Descriptive statistics and 95% confidence intervals (CI) were calculated. Subgroup analyses were performed by relationship type and gender. The present abstract summarizes baseline data before implementation of the comprehensive intervention package

Results:

A total of 839 first-degree relatives from 221 HBV-related HCC index families were identified. Among them, 378 relatives were deceased or otherwise unreachable, leaving 461 living first-degree relatives eligible for contact. Of these, 411 individuals (89.2%) completed HBV serological testing. Among the tested individuals (411), a total of 161 relatives were HBsAg-positive, yielding an HBV prevalence of 39.2% (95% CI: 34.5%–44.1%), markedly higher than the general population.

Among the tested participants (411), 67 relatives (16.3%) were receiving antiviral therapy. Among HBV-positive individuals (161 relatives), the antiviral treatment uptake was 41.6% (67/161; 95% CI: 34.0%–49.6%).

Subgroup analysis demonstrated a striking pattern of familial clustering. 42.31% of parents (33/78) were HBsAg-positive. The siblings of index patients exhibited elevated HBV prevalence, with rates of 51.75% (59/114) in this group, significantly higher than the general population. 26.14% of children (69/264) were HBsAg-positive, further supporting the clustering of HBV within the family structure. Regarding gender, HBV prevalence was comparable between females (43.2%) and males (56.8%).

Conclusions: This multicenter baseline analysis demonstrates extremely high HBV prevalence and suboptimal antiviral treatment uptake among first-degree relatives of HBV-related HCC patients in Eastern China. The pronounced familial clustering—particularly among parents and siblings—highlights the necessity of targeted public health interventions. The ongoing study will evaluate whether a comprehensive outreach and follow-up program can improve HBV screening

and treatment rates and ultimately reduce long-term HCC risk in this high-risk population.

Keyword(s) : Hepatitis B, Hepatocellular Carcinoma, First-degree Relatives.

1483 - Epidemiological Survey of Liver Cirrhosis in Tianjin Ten Year Data

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Background / Hypothesis:

In recent years, the epidemiological characteristics of liver cirrhosis worldwide have undergone subtle changes. This study analyzed the epidemiological profile of liver cirrhosis over a decade at the largest liver disease center in Tianjin, China.

Materials and Methods:

In this retrospective study, we analyzed LC cases admitted between December 2015 and May 2025. Data on demographics, etiologies, and complications were collected. Etiological changes were assessed annually and compared between two periods (2016–2019 vs. 2020–2024). Continuous variables are expressed as mean $\pm$ SD and categorical variables as percentages. The Student's t-test, χ^2 test, one-way ANOVA, or univariate logistic regression were used for analysis. A P-value <0.05 was considered statistically significant. Analyses were performed using R and GraphPad Prism.

Results:

A total of 20,150 patients were included (65.69% male, 34.31% female). The peak age at diagnosis was 55–59 years. Mean age was 58.1 ± 10.6 years for viral hepatitis-related LC and 65.2 ± 10.5 years for autoimmune LC. Etiology distribution was: viral hepatitis 43.65% (HBV 37.86%, HCV 5.59%, mixed 0.19%), alcohol-related 13.66%, autoimmune 10.92%, mixed 4.25%, and other 26.84%. Viral hepatitis, alcohol-related, and mixed etiology were more common in males (all $P < 0.001$), while autoimmune LC was more common in females ($P < 0.001$). From 2016–2019 to 2020–2024, viral hepatitis decreased from 47.78% to 39.26% ($P < 0.001$), with HBV decreasing from 41.10% to 34.31% ($P < 0.001$). Autoimmune, metabolic, and vascular etiologies increased (all $P < 0.05$), while alcohol-related LC remained stable. Common complications included ascites (28.98%), HCC (30.69%), and UGIB (26.41%). Males had higher rates of HCC (36.83% vs 18.93%) and portal vein tumor thrombosis (5.98% vs 2.29%, both $P < 0.001$). Using HBV as reference, ORs for

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complications were: ascites—alcohol 2.435, autoimmune 1.796; UGIB—HCV 1.318, alcohol 1.812, autoimmune 1.495, mixed 1.412; HCC—HCV 0.651, alcohol 0.133, autoimmune 0.055, metabolic 0.067, vascular 0.089, mixed 0.34; hepatic encephalopathy—alcohol 2.195, autoimmune 1.533.

Conclusions:

Viral hepatitis remains the leading cause of LC in Northern China, although its proportion has decreased significantly, particularly for HBV. Alcoholic LC carries the highest risk of ascites, UGIB, and hepatic encephalopathy. The etiological spectrum is evolving, with a stable burden of alcohol-related LC and a rising contribution from metabolic liver diseases.

Keyword(s) :

1485 - Integrated Multi omics Liquid Biopsy Advancing Early Detection and Precision Management of HCC

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality worldwide, often diagnosed at late stages due to the limited sensitivity and specificity of current surveillance methods such as ultrasound and alpha-fetoprotein (AFP) testing. Liquid biopsy, which non-invasively analyzes circulating biomarkers, offers a promising alternative. However, single-omics approaches often lack the robustness required for early detection. Multi-omics integration presents a transformative strategy to enhance diagnostic accuracy and enable precision oncology in HCC.

Materials and Methods:

This review synthesizes recent advances in multi-omics liquid biopsy for HCC, incorporating genomic (e.g., TERT, TP53 mutations), epigenomic (e.g., RASSF1A methylation), proteomic/glycomic (e.g., AFP-L3, GP73, exosomal glycans), fragmentomic (cfDNA fragmentation patterns), and cellular biomarkers (CTCs, EVs). Cross-omics integration strategies—such as cSMART, HIFI, and ASDAm-H1—were evaluated alongside emerging technologies including nanopore sequencing, SERS-based single-exosome analysis, and

machine learning models (e.g., XAI, GNNs, Bayesian Networks).

Results:

The integration of multi-omics biomarkers has demonstrated transformative diagnostic performance for early HCC. Cross-omics models consistently outperformed single-modality approaches: combined genomic-epigenomic assays achieved sensitivities of 80–90% for early-stage detection, while the fragmentomic-epigenomic HIFI model distinguished HCC from cirrhosis with exceptional accuracy (AUC 0.996, sensitivity 95.42%, specificity 97.83%). Integrated protein-epigenetic panels, such as the ASDAm-H1 index, reached an AUC of 0.96, and longitudinal application of multi-parametric scores like GALAD significantly improved sensitivity to 69.2% for early HCC compared to single measurements (53.8%). Fragmentomics-based models enabled early screening with sensitivities exceeding 96%, and metabolomic panels detected preclinical HCC up to one year before clinical diagnosis with 80% sensitivity. Beyond detection, multi-omics liquid biopsy proved vital for precision management: ctDNA mutation profiling enabled dynamic monitoring of targeted therapy response, PD-L1⁺ CTCs and cfDNA copy number variations predicted outcomes to immune checkpoint inhibitors, and machine learning-driven deconvolution of single-exosome SERS fingerprints or cfDNA methylation data provided unprecedented molecular resolution. These approaches not only identified AFP-negative HCC cases but also facilitated early recurrence prediction and molecular subtyping, effectively transitioning liquid biopsy from a passive detection tool to an active component in the dynamic, personalized management of HCC.

Conclusions:

Multi-omics liquid biopsy represents a paradigm shift in HCC management, enabling high-accuracy early detection and dynamic monitoring of disease progression and treatment response. Despite challenges in data integration, standardization, and clinical validation, future directions—including explainable AI, single-cell multi-omics, and closed-loop precision oncology systems—hold great promise. With continued interdisciplinary collaboration and standardization efforts, multi-omics liquid biopsy is poised to become the cornerstone of next-generation HCC care.



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Keyword(s) : Hepatocellular carcinoma, Liquid biopsy, Multi-omics integration, Early diagnosis, Clinical translation

1487 - Sphingosine Attenuates Steatosis but Exacerbates Fibrosis Risk in Metabolic Associated Fatty Liver Disease (MAFLD) via Hepatic LDLR Modulation

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Background: Dysregulated sphingolipid metabolism contributes to the pathogenesis of glucose intolerance, hepatic fibrosis, and tumor metabolism. However, the role of sphingosine in metabolic-associated fatty liver disease (MAFLD) remains poorly defined.

Methods: After one week of acclimatization, 3-week-old C57/BL-6J mice were fed a high-fat choline-deficient (HCD) diet for 10 weeks, followed by 4 weeks of either continued HCD feeding or HCD with oral sphingosine supplementation. Hepatic steatosis, inflammatory injury, and fibrosis were assessed across normal diet, HCD, and HCD + sphingosine groups. Circulating lipid profiles were also evaluated.

Results: Oil Red O staining revealed a significant increase in the steatosis area in the HCD group (34.924%) compared to the choline-deficient (CD) group (9.062%, $P = 0.004$), which was attenuated by sphingosine supplementation (15.922%, $P = 0.026$). In contrast, Picrosirius red staining indicated a higher fibrosis proportion in the HCD + sphingosine group than in the HCD group (0.577 vs. 0.250, $P = 0.004$). Moreover, the HCD + sphingosine group exhibited elevated LDL-cholesterol and reduced HDL-cholesterol levels, along with decreased membrane localization of the LDL receptor compared to the HCD group.

Conclusions: Sphingosine ameliorates hepatic steatosis but promotes LDL-cholesterol elevation and fibrosis risk in HCD-fed mice, likely through internalization of hepatic LDLR.

Keyword(s) : MAFLD, Sphingosine, Liver fibrosis, LDLR.

1488 - Eicosapentaenoic Acid Prevents the Short term High fat Diet-Enhanced Severity of Concanavalin A-Induced Liver Injury

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Background / Hypothesis:

Metabolic dysfunction-associated steatohepatitis (MASH) represents the progressive, inflammatory stage of metabolic dysfunction-associated steatotic liver disease (MASLD) and is a major driver of cirrhosis and hepatocellular carcinoma. Although early intervention is critical, the molecular events that increase hepatic susceptibility during the initial phase of MASLD remain insufficiently understood. An imbalance in n-3 and n-6 polyunsaturated fatty acids (PUFAs), frequently observed in MASLD, is known to influence hepatic inflammatory responses. Using our short-term high-fat diet (HFD) model, we previously showed that only 4 days of HFD feeding markedly exacerbates concanavalin A (Con A)-induced immune-mediated liver injury, indicating heightened hepatic vulnerability before overt steatosis or metabolic abnormalities appear. To clarify the mechanisms driving this early inflammatory priming, we investigated rapid PUFAs alterations and related immune-inflammatory pathways during the earliest stage of HFD exposure.

Materials and Methods:

Male C57BL/6 mice were fed an HFD for 4 days. Liver and serum samples were collected daily (day 0–4) and analyzed for n-3 and n-6 PUFAs composition using gas chromatography–mass spectrometry. To evaluate preventive effects, mice received eicosapentaenoic acid (EPA, 100 mg/kg) during the short-term HFD period. Con A-induced liver injury was triggered by intravenous administration of Con A (10 mg/kg), and liver damage was assessed by serum transaminase levels and histopathology. Inflammatory and coagulation-related pathways were further examined to elucidate mechanisms underlying early hepatic vulnerability.

Results:

Short-term HFD intake resulted in rapid and dynamic alterations in PUFAs profiles. Notably, hepatic and serum EPA levels significantly decreased, whereas arachidonic acid (AA) levels increased, indicating a shift toward a more pro-inflammatory lipid milieu. Consistent with these biochemical changes, HFD-fed mice exhibited markedly aggravated Con A-induced liver injury. EPA supplementation effectively attenuated the exacerbation of liver injury and reduced inflammatory cytokine expression and hepatic fibrin deposition. These findings suggest that the early decline in EPA contributes to heightened hepatic susceptibility, and restoring EPA levels mitigates both inflammatory and pro-coagulant responses during

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acute immune-mediated liver injury.

Conclusions:

Early exposure to an HFD induces a rapid imbalance of n-3 and n-6 PUFAs, lowering anti-inflammatory EPA while increasing pro-inflammatory AA, thereby heightening susceptibility to immune-mediated liver damage. EPA supplementation during this early phase significantly alleviates liver injury through anti-inflammatory and anticoagulant mechanisms. These results indicate that early EPA intervention may serve as an effective strategy to prevent MASLD progression before clinical symptoms emerge.

Keyword(s) : Eicosapentaenoic acid; Short-term high-fat diet; Concanavalin A-induced liver injury; Metabolic dysfunction-associated steatotic liver disease; Metabolic dysfunction-associated steatohepatitis

1489 - Transcriptomic profiling of liver biopsy tissues reveals $\alpha\beta$ T Cell activation and differentiation are associated with Idiosyncratic drug induced liver injury

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Background / Hypothesis:

Idiosyncratic drug-induced liver injury (iDILI) is a rare liver injury with diverse clinical and pathological manifestations. We know very little about the pathological and physiological processes of iDILI. The lack of specific biomarkers makes it challenging for hepatologists to distinguish iDILI from other types of liver diseases effectively.

Materials and Methods:

Bulk RNA-Seq was performed on liver biopsy tissue from 17 iDILI patients and 13 normal liver tissues. The DESeq2 package and the WGCNA package were used to identify differentially expressed genes (DEGs) and hub modules, respectively. Functional enrichment analysis was performed on the DEGs and co-regulated genes. The results of the above bioinformatics analyses were validated by immunohistochemistry (IHC) on liver tissue.

Results:

We identified 769 DEGs (490 up-regulated genes and 279 down-regulated genes) between iDILI and HC. Go analysis of up-regulated genes enriched in $\alpha\beta$ mononuclear cell differentiation. KEGG analysis showed that the up-regulated genes were mainly enriched in Th17 cell differentiation and Th1 and Th2 cell differentiation. Enrichment analysis of 420 co-regulation genes obtained from DEGs and hub modules were enriched in the regulation of alpha-beta T cell activation. IHC confirmed that the infiltration of alpha-beta T cells in the liver tissue of IDILI manifests in different pathological phenotypes, with varying levels of CD4+ and CD8A+ T cell infiltration. The differentiation and infiltration of $\alpha\beta$ T cells contribute to the differential diagnosis of a variety of liver diseases.

Conclusions: Our study indicates that transcriptomic profiling of liver biopsy tissues reveals a significant association between $\alpha\beta$ T cell activation and differentiation and the pathological processes of iDILI.

Keyword(s) : idiosyncratic drug-induced liver injury, liver biopsy tissues, transcriptomics, $\alpha\beta$ T Cell

1490 - South Asian metabolic phenotype and fibrosis discordance in lean and obese metabolic dysfunction-associated steatotic liver disease insights from a Sri Lankan tertiary cohort

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Background / Hypothesis:

South Asians develop metabolic dysfunction-associated steatotic liver disease (MASLD) at lower body mass indices, yet the fibrosis-risk phenotype of lean MASLD remains poorly characterised. We hypothesised that the South Asian metabolic phenotype may confer disproportionately high fibrosis susceptibility in lean MASLD, resulting in fibrosis indices comparable to obese counterparts despite lower anthropometric burden

Materials and Methods:

A cross-sectional analysis was conducted at a major Sri Lankan hepatology centre between August and October 2025. Adults with ultrasonographic hepatic steatosis and at least one metabolic risk factor were

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included. Height entries provided in non-metric formats were corrected to centimetres prior to recalculation of body mass index (BMI). Patients were categorised as lean (BMI <25 kg/m²) or obese (≥25 kg/m²). Clinical risk factors, metabolic parameters and non-invasive fibrosis markers (fibrosis-4 [FIB-4] index and aspartate aminotransferase-to-platelet ratio index [APRI]) were compared using independent t-tests and chi-square tests

Results:

Forty patients were analysed; 13 (32.5%) had lean MASLD. Despite significantly lower BMI (23.5 ± 0.9 vs 29.5 ± 3.6 kg/m²; $p < 0.001$) and waist circumference (96.7 ± 11.2 vs 109.7 ± 22.5 cm; $p = 0.019$), lean and obese MASLD showed similar metabolic comorbidity burdens including diabetes (30.8% vs 29.6%; $p = 1.00$) and hypertension (23.1% vs 33.3%; $p = 0.77$). Fibrosis markers were comparable: FIB-4 (1.31 ± 1.04 vs 1.17 ± 0.53 ; $p = 0.66$), APRI (0.36 ± 0.22 vs 0.32 ± 0.13 ; $p = 0.54$), and high FIB-4 >1.3 prevalence (38.5% vs 33.3%; $p = 1.00$). These findings demonstrate a fibrosis burden disproportionate to anthropometric profile, supporting a visceral adiposity-driven mechanism in lean South Asians

Conclusions:

Lean MASLD was common and exhibited fibrosis risk comparable to obese MASLD, despite markedly lower BMI. This highlights limitations of anthropometry-based stratification and underscores the need for South Asia-specific MASLD screening pathways incorporating routine fibrosis assessment irrespective of BMI. Findings support urgent multicentre validation to recalibrate fibrosis thresholds for lean South Asian populations

Keyword(s) : Lean MASLD, fibrosis discordance, metabolic phenotype, South Asia, non-invasive fibrosis markers

1491 - Dynamic network collapse in decompensated hepatic cirrhosis

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Background: Alterations in intrahepatic communication networks – driven by fibrotic bridging, vascular shunting, and metabolic dysfunction – serve as latent biomarkers for impending cirrhotic decompensation. However, the dynamic hepatic connectomes and its role in signaling decompensation remain unexplored.

Materials and Methods: We enrolled hepatic cirrhotic patients (with/without HCC) (2022–2025) undergoing gadoxetic acid-enhanced upper abdominal MRI with ≥5 imaging phases (non-contrast, arterial early, arterial late, portal venous, equilibrium). Dynamic contrast-enhanced T1-weighted MRI using a GE Discovery 750 3.0T or Siemens Prisma 3.0T scanner with standardized axial acquisition (FOV = 38 cm), performed prior to and following intravenous injection of 0.1–0.2 mL/kg gadoxetic acid disodium (Bayer AG, Germany). Liver partitioned into 8 segments (I–VIII, Couinaud classification). Representative slices. Circular ROIs placed centrally in Segments I–VIII, avoiding vessels and lesions <5 mm. 837 radiomic features per segment were extracted via PyRadiomics (first-order, GLCM, GLDM, GLSZM, NGTDM, wavelet features). Sliding window (window size=3 phases) was applied to build topological networks. Inter-segmental connectivity quantified by Pearson correlation, with sparsity controlled at 0.2. Small-worldness (sigma, clustering, path length), modularity, hub distribution, nodal centrality (degree, strength, betweenness, closeness, eigenvector) were analyzed.

Results: Significant network reorganization was observed in decompensated compared to compensated livers within the arterial-portal venous-equilibrium window. This included an increase in modularity (indicating functional fragmentation: 0.066 vs. 0.038), sigma (reflecting heightened randomness: 1.514 vs. 1.434), and a decrease in average path length (suggesting impaired information transfer efficiency: 0.786 vs. 0.818); all $P < 0.05$. Segment V dominated the liver network in compensation (20% hub prevalence, consistent with its anatomical centrality). Decompensation triggered hub redistribution: Peripheral Segments II–V & VII–VIII: 12–15%; Marginal Segments I & VI: 8–10%. This indicate collapse of compensatory network. The betweenness centrality of Segment I (measure of its information-bridging role) predicted decompensation risk: 18.2%, 54.2%, and

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83.3% in low, medium and high risk stratus, respectively. High-risk groups showed significantly elevated HRs for onset of ascites, hepatic encephalopathy and variceal bleeding; all $P < 0.05$.

Conclusions: This study pioneers a network paradigm for cirrhosis prognostication, shifting from isolated segmental biomarkers to system-level monitoring. The arterial-portal-equilibrium phase offers a critical window for detecting connectome-driven decompensation signals, with Segment I centrality emerging as a clinically important predictor.

Keyword(s) : Hepatic Cirrhotic, Hepatic Decompensation, Network Analysis, Liver Segments, Complex Network

1492 - Evaluating the accuracy of the histological diagnosis in unexplained liver injury A retrospective study

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Background / Hypothesis:

Liver biopsy is a valuable tool for diagnosing liver diseases with unknown etiology, but it sometimes fails to provide a clear diagnosis. This study aimed to identify the cause of unexplained liver injury and evaluate the accuracy of histological diagnosis.

Materials and Methods:

A retrospective analysis was conducted on the clinical and histological data of patients with chronic liver disease of unknown aetiology at the Second Affiliated Hospital of Nanchang University, China, who underwent liver biopsy between September 2019 and June 2024. Diagnoses were divided into admission diagnosis, histological diagnosis, and final clinical diagnosis groups.

Results:

A total of 104 patients were included, with 64 women (61.5%) and 40 men (38.5%), 96 (92.3%) of whom received a definitive diagnosis through liver biopsy.

The main diseases were autoimmune liver disease (AILD, 49 cases, 47.1%), non-alcoholic fatty liver disease (NAFLD, 26 cases, 25.0%), drug-induced liver injury (DILI, 22 cases, 21.2%), others (11 cases, 10.6%) and unknown etiology (8 cases, 6.9%). AILD was most common in women (40 cases, 55.6%), while NAFLD was most common in men (19 cases, 40.4%). Histological diagnosis showed higher sensitivity, specificity, positive predictive values (PPVs), and negative predictive values (NPVs) compared to admission diagnosis. Venn diagram analysis revealed that 57.7% of cases matched between admission and histological diagnosis, 60.6% matched between admission and final clinical diagnosis, and 92.3% matched between the histological and final clinical diagnosis.

Conclusions:

Histological diagnosis is effective in determining the cause of unexplained liver injury, and liver biopsy remains a crucial tool.

Keyword(s) : Histological diagnosis, liver biopsy, unexplained liver injury.

1495 - Electrolytes as predicting factor on outcome in patient with cirrhosis Study from low socioeconomic south east Asian population.

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Background / Hypothesis:

Patient with cirrhosis have significant abnormalities in their fluid and electrolyte balance, this is mainly manifested by the development of ascites, oedema, neurological symptoms etc. The common disorders of fluid, electrolyte and acid-base metabolism observed in cirrhosis are hyponatremia, hypokalaemia, hyperkalaemia, low albumin and low anion gap, in addition to an excess accumulation of body fluids. out of this hyponatremia is the most prevalent single electrolyte abnormality. The development of hyponatremia in cirrhotic patients is regarded as a poor prognostic sign. in our daily clinical practice, we have observed cirrhotic patients with much less sodium level than patients in same stages of cirrhosis in west. We want to study different electrolytes imbalances in our population of cirrhosis and their impact on the mortality.

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Materials and Methods:

We conducted a descriptive study, which was carried out at The Indus Hospital and Health Network. Data was collected in prospective fashion from April 2023 till October 2025, comparing the different electrolyte imbalances present at the time of admission in hospital with outcome in terms of mortality in patients of cirrhosis. Our study population comprised of one hundred consecutive adult patients, with the diagnosis of cirrhosis. Diagnosis of cirrhosis was based on histology, blood chemistry ultrasonographic features along with classical signs of decompensated cirrhosis such as ascites or oesophageal varices. Patients fulfilling the inclusion criteria were enrolled in the study. Data regarding the aetiology, stage, complications and electrolyte status of the patients at the time of first admission were recorded and then these patients were followed up in next one year for outcome. Our outcome was mortality. In the end data was analysed comparing outcome with initial presentation.

Results:

HCV is the most common cause of cirrhosis (58%). Great majority of patients (90%) have ascites. Among the different biochemical laboratory parameters Child Pugh score, sodium, Creatinine, Total bilirubin, Albumin and Prothrombin time were found to be influencing mortality, and this relationship was statistically significant (P value are <0.001, 0.03, 0.02, 0.01, 0.004, and 0.001 respectively).

Conclusions:

Cirrhosis of liver is associated with different electrolyte abnormalities, most of which clearly influences mortality.

Keyword(s) : ACLD, Electrolyte Imbalance, Low SocioEconomic population, Cirrhosis, Hyponatremia

1496 - Systemic Immune Inflammation Index as a Predictive Biomarker for Non Response to Ursodeoxycholic Acid in Primary Biliary Cholangitis

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China³

Background :

Although ursodeoxycholic acid (UDCA) is the first-line standard treatment for primary biliary cholangitis (PBC), a proportion of patients exhibit an inadequate response, leading to a risk of disease progression. Given the close involvement of inflammatory pathway activation in PBC pathogenesis, the value of the Systemic Immune-Inflammation Index (SII), a novel inflammatory marker, in predicting UDCA non-response remains to be elucidated.

Materials and Methods: To investigate the predictive value of baseline SII levels for UDCA treatment non-response in patients with PBC.

This retrospective cohort study included 238 PBC patients from Qujing First People's Hospital and Qujing Second People's Hospital who received standard-dose UDCA. The SII was calculated using baseline data as follows: $SII = \text{platelet count} \times \text{neutrophil count} / \text{lymphocyte count}$. Treatment response was defined according to the European Association for the Study of the Liver criteria, with good response indicated by an alkaline phosphatase (ALP) level below 1.67 times the upper limit of normal after 12 months of UDCA therapy; otherwise, patients were classified as non-responders. Logistic regression analysis was used to assess the association between SII and UDCA non-response. To verify the robustness of the findings, the following analyses were performed: subgroup analysis stratified by baseline cirrhosis, tertile trend analysis of SII, and a sensitivity analysis excluding patients with baseline chronic diseases.

Results: This study included 238 patients with a median age of 55.5 years (interquartile range: 45.0–65.0), of whom 192 (79.7%) were female. Sixty-eight patients (28.6%) were classified as UDCA non-responders. Univariate analysis revealed significant differences between the non-response and response groups in age, sex, proportion with cirrhosis, ALP level, absolute neutrophil count, absolute lymphocyte count, platelet count, and SII (all $P < 0.05$). Multivariate logistic regression analysis confirmed that after adjusting for potential confounders, elevated SII remained an independent risk factor for UDCA non-response (OR =

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1.07, 95% CI: 1.03–1.11). Furthermore, this association was consistent across various analyses: in the subgroup with baseline cirrhosis (OR = 1.28, 95% CI: 1.10–1.49), in the SII tertile trend analysis (OR = 5.03, 95% CI: 2.08–12.17), and in the sensitivity analysis excluding baseline chronic diseases (OR = 1.07, 95% CI: 1.02–1.11), thereby confirming the robustness of the results.

Conclusions: Baseline Systemic Immune-Inflammation Index (SII) serves as an independent risk marker for predicting UDCA treatment non-response in PBC patients. This indicator holds significant value for the early identification of high-risk individuals and for guiding clinical decision-making toward individualized treatment strategies.

Keyword(s) :

1498 - A Case of Idiopathic Caroli Syndrome Misdiagnosed as Cholangiocarcinoma

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Background / Hypothesis: We aimed to report a case of Idiopathic Caroli Syndrome that was initially misdiagnosed as cholangiocarcinoma, highlighting the diagnostic challenges and emphasizing the importance of comprehensive evaluation including histopathology and genetic testing in such rare liver diseases.

Materials and Methods: The patient was suspected to have autoimmune liver disease or hereditary metabolic liver disease. Histological findings reveal : (1) Fibrous tissue proliferation in the portal areas with the formation of fibrous septa; (2) Marked bile duct proliferation, dilation, and tortuosity (ductal plate malformation) within and around the fibrous septa; (3) Hyperplasia of bile duct epithelial cells without atypia; (4) Absence of bile plug formation. The Genetic testing revealed two heterozygous variants in the PKHD1 gene: c.10630T>A and c.5600+2T>G . The gene encodes fibrillin/multichannel protein, which is associated with ciliopathies.

Results: Based on the above findings, the diagnosis of Caroli Syndrome can be confirmed. Caroli Syndrome is

a rare congenital disorder characterized by cystic dilatation of the intrahepatic bile ducts, congenital hepatic fibrosis, and portal hypertension. It results from ductal plate malformation and typically presents with recurrent cholangitis or portal hypertension-related complications. Diagnosis is primarily based on imaging findings showing characteristic cystic or beaded bile duct dilatations. However, when inflammation persists or a large cyst is present, the characteristic features of cystic bile duct dilatation may disappear. Through liver biopsy, we have ruled out the possibility of cholangiocarcinoma. However, the persistent elevation of liver function indicators and CA19-9 suggests that the patient's disease remains progressive, highlighting the refractory nature of this disease.

Conclusions: Chronic inflammatory manifestations of Caroli syndrome may mimic cholangiocarcinoma. A multidisciplinary approach incorporating imaging, histopathology, and genetic testing is essential for accurate diagnosis and can prevent unnecessary surgical intervention. This case underscores the value of genetic analysis in diagnosing rare congenital liver disorders.

Keyword(s) : Caroli Syndrome; cholangiocarcinoma; PKHD1 gene; autosomal recessive polycystic kidney disease; liver biopsy; genetic testing.

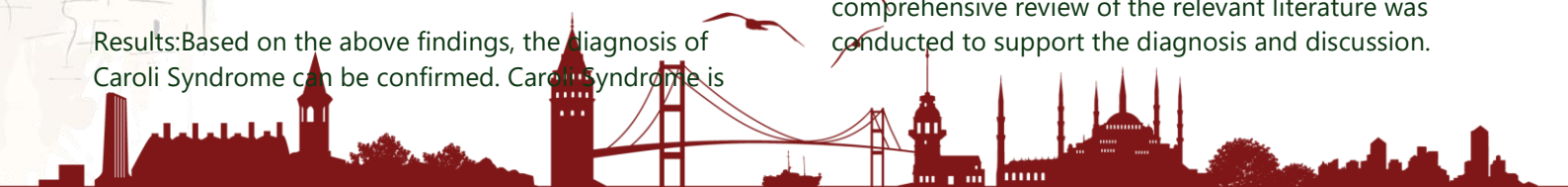
1499 - A case of drug induced autoimmune hepatitis with negative serum markers

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Background / Hypothesis: This study aims to report a case of drug-induced autoimmune hepatitis (AIH) with negative autoantibodies, and to provide clinical evidence and practical experience for the diagnosis and management of similar patients.

Materials and Methods: The clinical, laboratory, imaging, and histopathological data, along with the treatment outcome, of a patient with drug-induced autoimmune hepatitis negative for autoantibodies were systematically collected and analyzed, and a comprehensive review of the relevant literature was conducted to support the diagnosis and discussion.



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Results:Based on the above data, DILI was initially considered. The patient's liver function was still unstable after two hospitalizations, and the cause of liver injury was unknown. Since all patients were negative for autoantibodies, there was insufficient evidence for the diagnosis of AIH. After the third admission, ultrasound-guided liver biopsy was performed to determine the cause. The pathological results showed the characteristic histological manifestations of AIH (moderate to severe interface hepatitis, lymphocyte and plasma cell infiltration, and rosette), and finally the patient was diagnosed with drug-induced autoimmune hepatitis combined with medical history. After diagnosis, prednisone acetate was given for one month, and her liver function indices improved significantly.

Conclusions:This case reconfirms that drugs can induce autoimmune hepatitis that is negative for autoantibodies. Acute or chronic hepatitis in the context of drug exposure history, even if autoantibodies are negative, should be alert to the possibility of DILI-AIH overlap. Liver histopathological examination is the key to the diagnosis. Early recognition and initiation of immunosuppressive therapy (such as glucocorticoids) are usually effective. Clinicians need to broaden their thinking to avoid delays in the diagnosis and treatment of this type of AIH due to negative autoantibodies.

Keyword(s) : drug-induced autoimmune hepatitis; negative autoantibodies; liver biopsy

1500 - Endogenous Endophthalmitis Associated With Liver Abscess Caused By Klebsiella pneumoniae

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Background / Hypothesis:

Endogenous endophthalmitis is a rare but serious complication of liver abscesses, particularly those caused by *Klebsiella pneumoniae*. In this case report, we describe a notable instance of a patient presenting with both a liver abscess and endogenous endophthalmitis concurrently, highlighting the clinical challenge and significance of this association.

Case Description:

Endogenous endophthalmitis is a rare but serious complication of liver abscesses, particularly those caused by *Klebsiella pneumoniae*. Given that the patient had no history of eye trauma or previous eye surgeries, a presumptive diagnosis of endogenous endophthalmitis (EE) in her right eye was made. The patient presented with EE accompanied by abdominal pain, suggesting a possible abdominal source of infection. Abdominal ultrasound and computed tomography scan showed a lesion in her liver, which was suspicious for an abscess. Examination of fluid from the liver abscess, as well as swab samples from the patient's right eye, confirmed the presence of *Klebsiella pneumoniae*. Despite early percutaneous drainage of the liver abscess, the condition of the patient's right eye did not improve, ultimately necessitating an evisceration. The patient was discharged two weeks after admission and experienced no further complications during a three-month follow-up period.

Conclusions:

This case report serves as a reminder that when encountering patients with endogenous endophthalmitis, physicians should consider the possibility of a liver abscess. Physicians should be aware not only of the potential for extrahepatic septic metastases but also of their associated complications. The immediate initiation of a comprehensive multidisciplinary treatment approach is necessary to prevent complications arising from liver abscesses, including endogenous endophthalmitis.

Keyword(s) : liver abscess, *Klebsiella pneumoniae*, Endogenous endophthalmitis

1501 - Are third generation cephalosporin still considered first choice for ascitic fluid infection in decompensated cirrhotic patients Study from largest tertiary care hospital Pakistan.

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Background / Hypothesis:

Ascitic fluid infection (AFI) is a serious complication in patients with decompensated liver disease with a mortality approaching 30%. Third generation cephalosporins (TGC) have been effectively used for this infection. However, resistance to third generation

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cephalosporins has been increasing for other infections. It is therefore imperative to define the effectiveness of Third generation cephalosporins for ascitic fluid infection and factors responsible for the resistance.

Materials and Methods:

This cross-sectional study was conducted on consecutive subjects with ascitic fluid infection who received of injection ceftriaxone from March 2023 till October 2025 at The Indus Hospital and Health Network, Karachi. Ascitic fluid infection was diagnosed based on ascitic fluid Polymorphonuclear count (PMN) of 250 cells/mm³ and or positive ascitic fluid culture. All patients had a repeat diagnostic tap of ascitic fluid 48 hours after the index tap. Failure of reduction of PMN count in the latter tap and/or ascitic fluid bacteria resistant to third generation cephalosporins resistance were considered as nonresponders. Patients receiving any other antibiotic, prior documented resistance to Third generation cephalosporins resistance and patients with hepatoma were excluded.

Results:

Total of 223 patients with ascitic fluid infection received third generation cephalosporins resistance during the study period and were analysed. Out of them 123 (55.2%) were males. Hepatitis C was the commonest aetiology in 151 (67.7%), 185(80.8%) were in Child class C, 175(78%) responded to Third generation cephalosporins while 48(22%) were nonresponders. Ascitic fluid culture was positive in 51(22.9%) patients. The predominant organism found in ascitic fluid was E. coli in 27(53%) of the culture positive group. However, 147(85%) of cases amongst culture negative group responded as compared to 28(55%) of patients in culture positive group. On univariate analysis total bilirubin >6mg/dl, PT >2.2 seconds and positive Ascitic fluid culture (p value 0.04, 0.02 and <0.001 respectively) were associated with non-response to third generation cephalosporins. On multivariate analysis only ascitic fluid positivity was associated with no response to third generation cephalosporins.

Conclusions:

We found 22% resistance to third generation

cephalosporins resistance in Spontaneous bacterial peritonitis, Positive ascitic fluid culture is independently associated with nonresponse to third generation cephalosporins.

Keyword(s) : ACLD, Ascites, SBP, Cirrhosis, Ascitic Fluid Infection, TGC, AFI, Third generation cephalosporin, spontaneous bacterial peritonitis, HCV, Hepatitis C, NBNC CLD

1502 - Chronic hepatitis B with autoimmune hepatitis in a male patient

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Background / Hypothesis: We aimed to present a case of chronic hepatitis B coexisting with autoimmune hepatitis (AIH) in a male patient so as to discuss its clinical manifestations, diagnostic approach, and treatment strategies, thereby providing insights for the clinical management of AIH.

Materials and Methods: Based on the above findings, a definitive diagnosis of "chronic hepatitis B with AIH" was established. However, since the patient did not exhibit typical clinical features of AIH overlapping with chronic hepatitis B, we further explored whether chronic hepatitis B could have acted as a trigger for AIH. Given the relative rarity of AIH in male patients and the necessity for close monitoring of HBV-DNA levels during immunosuppressive therapy, we paid particular attention to balancing the sequence and timing of antiviral and immunosuppressive treatments when formulating the therapeutic plan.

Results: The final diagnosis in this case was "chronic hepatitis B combined with AIH." It is noteworthy that viral infection is a recognized triggering factor for AIH; for instance, various non-specific autoantibodies can be detected in some patients with hepatitis C virus infection. Similarly, chronic hepatitis B can also present with autoantibody positivity and extrahepatic autoimmune phenomena, overlapping with features of AIH. Further research has revealed that viral antigen epitopes share determinant clusters with certain liver autoantigens, potentially inducing cross-reactive immune responses through "molecular mimicry", leading to autoimmune liver injury. This suggests that



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HBV infection or its chronic progression may have acted as an initiating factor for AIH in this patient. Following the initiation of entecavir antiviral therapy, we performed a liver biopsy and administered immunosuppressive treatment. Both the pathological findings and the treatment response confirmed the diagnosis of AIH. At the two-month follow-up, the patient's liver function had normalized, IgG levels had decreased, HBV-DNA had become undetectable, and a recent outpatient review showed that the ANA had turned negative.

Conclusions: This case suggests that chronic hepatitis B may serve as a predisposing factor for AIH. Immunosuppressive therapy can significantly influence HBV-DNA levels; therefore, close monitoring of hepatitis B status during treatment is essential. Careful consideration of the sequence and strategy between antiviral and immunosuppressive therapies is crucial for the diagnosis and clinical management of patients with chronic hepatitis B complicated by AIH.

Keyword(s): chronic hepatitis B; autoimmune hepatitis; liver biopsy

1503 - Clinical Features and Prognostic Assessment of MAFLD Related Cirrhosis Pathological vs. Clinical Diagnosis

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Background / Hypothesis:

In light of the "burn-out" phenomenon in metabolic dysfunction-associated fatty liver disease (MAFLD), an international expert panel has suggested that cirrhosis can be attributed to MAFLD even without typical histological findings, provided there is prior evidence of fatty liver (by histology or imaging) along with metabolic risk factors. This study aimed to compare the clinical characteristics and prognostic scores between patients diagnosed using conventional pathological criteria and those diagnosed clinically.

Materials and Methods:

We retrospectively analyzed 151 patients hospitalized with MAFLD-related cirrhosis at our center between 2022 and 2024. The pathological diagnosis group included patients with cirrhosis and histological evidence of steatosis or steatohepatitis. The clinical diagnosis group was defined according to the APASL

clinical practice guidelines for MAFLD. We compared demographic, metabolic, and laboratory data, as well as prognostic scores between the two groups.

Results:

Of the 151 patients, 38 (25.2%) were diagnosed pathologically and 113 (74.8%) clinically. Compared to the pathological group, the clinical diagnosis group was older (68 vs. 61 years), had longer hospital stays (9 vs. 7 days), and a higher rate of decompensation (73.5% vs. 23.7%, all $p < 0.05$). Laboratory results showed that the clinical group had lower albumin (33.28 vs. 37.13 g/L), triglycerides (1.07 vs. 1.21 mmol/L), total cholesterol (3.40 vs. 4.01 mmol/L), LDL-C (2.08 vs. 2.45 mmol/L), and HDL-C (0.92 vs. 1.17 mmol/L), along with worse coagulation (INR 1.24 vs. 1.20, all $p < 0.05$). This group also had higher serum creatinine (64.30 vs. 55.50 $\mu\text{mol/L}$) and uric acid (342.70 vs. 287.20 $\mu\text{mol/L}$, all $p < 0.05$). Child-Pugh scores, the proportion of Child-Pugh class C (4.42% vs. 0.00%, $p = 0.001$), and MELD scores were all significantly higher in the clinical diagnosis group.

Conclusions:

In our center, most MAFLD-related cirrhosis cases were diagnosed clinically. These patients had more advanced disease, worse liver function, and poorer prognostic scores than those diagnosed pathologically. These findings highlight the importance of early and active clinical diagnosis to allow timely intervention and improve patient outcomes.

Keyword(s): Cirrhosis; MAFLD; Pathological diagnosis; Clinical diagnosis; Clinical features

1505 - The Mechanistic Basis of ASH1L's Oncogenic Role in Hepatocellular Carcinoma

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Background / Hypothesis: Recent studies have suggested that ASH1L promotes tumor progression and contributes to an immunosuppressive microenvironment in hepatocellular carcinoma (HCC). However, the molecular mechanisms by which ASH1L regulates HCC development remain unclear. This study aimed to investigate the oncogenic role of ASH1L in HCC and to elucidate its function in tumor progression.

Materials and Methods: To investigate the oncogenic role of ASH1L in HCC, protein expression was

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examined by western blot to identify cell lines with different endogenous levels. ASH1L expression was silenced in Hep3B and SNU-739 cells using lentivirus-mediated shRNA, followed by puromycin selection for three days to obtain stable knockdown cell lines. The efficiency of ASH1L knockdown was verified using qPCR and western blot. The functional consequences of ASH1L suppression were assessed using viability, colony formation, invasion, and migration assays. In addition, RNA sequencing of ASH1L-silenced cells was conducted to identify ASH1L-regulated genes, followed by validation using qPCR. All experiments were conducted in triplicate, and statistical significance was analyzed using a two-tailed Student's t-test.

Results: ASH1L expression was relatively high in Hep3B and SNU-739 cells, compared to other HCC cell lines. ASH1L knockdown significantly reduced cell viability and colony formation in both HCC cell lines for 3 days post-transfection. In addition, the invasive and migratory capacity was markedly reduced in Hep3B and SNU-739 cells. RNA sequencing using the SNU-739 cell line confirmed that suppression of ASH1L expression led to decreased expression of genes associated with extracellular matrix organization. Consistently, qPCR validation confirmed the downregulation of these ASH1L-regulated genes, supporting the role of ASH1L in promoting extracellular matrix remodeling and tumor progression.

Conclusions: ASH1L promotes HCC progression by regulating cell proliferation, invasion, and migration, likely through transcriptional regulation of extracellular matrix-related genes. These suggest that ASH1L functions as an oncogenic factor in HCC and may be a potential therapeutic target.

Keyword(s) :

1506 - Effect of Semaglutide Therapy on Hepatic Steatosis and Liver Stiffness in Patients with Nonalcoholic Fatty Liver Disease (NAFLD) A Quasi Experimental Study

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Background / Hypothesis:

Non alcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver disease worldwide, with limited pharmacologic treatment options. Semaglutide, a glucagon-like peptide-1 receptor agonist, has shown metabolic benefits and potential effects on hepatic steatosis. This study aimed to compare the effects of Semaglutide therapy and standard medical treatment on hepatic steatosis and liver stiffness in NAFLD patients.

Materials and Methods:

This quasi-experimental study was conducted at the Department of Hepatology, Bangladesh Medical University (BMU), Dhaka, from September 2022 to February 2024. Seventy-four adult NAFLD patients (CAP >234 dB/m; LSM ≥ 7.8 kPa and ≤ 14 kPa) were enrolled. Participants were randomly allocated into a treatment group (n=33), receiving subcutaneous Semaglutide (0.25 mg for first 4 weeks then followed by 0.5mg for next 4 week then escalated to 1 mg weekly for total 24 weeks), and a control group (n=37), receiving standard medical care without pharmacotherapy. Both groups received lifestyle modification with dietary counselling properly. Primary outcomes were changes in CAP and LSM measured by Fibroscan, secondary outcomes included changes in BMI and liver enzymes (ALT, AST). Statistical analysis was performed using SPSS version 27.

Results:

At 24 weeks, the treatment group showed significant reductions in CAP (mean change: 40.91 ± 36.26 dB/m) and LSM (mean change: 2.51 ± 1.46 kPa) compared to the control group (CAP: 8.27 ± 30.99 dB/m; LSM: -0.44 ± 2.48 kPa; $p < 0.001$ for both). Improvements in BMI, ALT, AST, and LDL were also statistically significant in the Semaglutide group ($p < 0.05$). No significant differences were observed in glycemic parameters or other lipid profile components. Semaglutide was well tolerated, with mild gastrointestinal side effects and no severe adverse events reported.

Conclusions: Semaglutide therapy significantly

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improves hepatic steatosis and liver stiffness in NAFLD patients compared to standard medical care. These findings support Semaglutide as a promising therapeutic option for NAFLD, warranting validation in larger randomized controlled trials.

Keyword(s) : Nonalcoholic fatty liver disease (NAFLD); Semaglutide; Hepatic steatosis; Liver stiffness; Controlled attenuation parameter (CAP); FibroScan; Quasi-experimental study

1508 - Minimal Hepatic Encephalopathy in Portal Vein Thrombosis An Underrecognized but Common Complication

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Background / Hypothesis: Type B hepatic encephalopathy (HE) is the least studied of the three types and occurs in patients with portosystemic shunting and no underlying liver disease. Aim: To determine the frequency and risk factors of HE in patients with non-cirrhotic non-malignant portal vein thrombosis (PVT).

Materials and Methods: A cross-sectional study included 44 adult patients with non-cirrhotic, non-malignant PVT, of whom 17 (39%) had childhood-onset PVT. The mean duration of portal hypertension was 92 months. All participants had liver fibrosis stages ranging from F0 to F2, as assessed by transient elastography. Minimal HE (MHE) was identified using psychometric tests. Ammonia concentration was measured in capillary blood using a PocketChem BA PA-4140 analyzer (ARKRAY Factory, Inc., Japan). Multivariable logistic regression models were used to determine factors associated with the presence of HE. Results: No cases of overt HE were identified in the study cohort, while 9 (20.5%) patients were diagnosed with MHE. Hyperammonemia was detected in all patients with MHE, representing a prevalence 1.7 times higher than that observed in patients without MHE (p

$= 0.022$). The median ammonia concentration was significantly higher in the MHE group than in patients without HE (116 [90–132] $\mu\text{mol/L}$ vs. 74 [41–102] $\mu\text{mol/L}$; $p = 0.019$). Using a calculated ammonia cut-off value of 88 $\mu\text{mol/L}$, patients whose levels were above this threshold were 15.3 times more likely to have MHE (odds ratio: 15.3; 95% confidence interval: 1.7–137.4; $p = 0.015$). Compared with patients without MHE, those with MHE had a significantly higher prevalence of large (≥ 10 mm in diameter) spontaneous or surgically created portosystemic shunts ($p = 0.004$). They also had increased concentrations of indirect bilirubin ($p = 0.035$), urea ($p = 0.046$), as well as lower serum sodium levels ($p = 0.037$). Together with sonographic spleen length and the presence of gastric varices, these variables were incorporated into the best-fitting logistic regression models. The models demonstrated an overall accuracy of 88.6–90.5%, a sensitivity of 66.7%, a specificity of 94.3–97.0% and an area under the curve (AUC) of 0.96–0.97. The established cut-off values were 15.3 $\mu\text{mol/L}$ for indirect bilirubin, 4.2 mmol/L for urea, 140.8 mmol/L for sodium and 15.0 cm for splenic length.

Conclusions: One in five patients with non-cirrhotic non-malignant PVT was found to have MHE. Risk factors for its development include large portosystemic shunts, gastric varices, spleen length, hyperammonemia, elevated levels of indirect bilirubin, urea, and low sodium levels.

Keyword(s) : psychometric tests, risk factors, logistic regression model, portosystemic shunts, ammonia

1509 - When Pressure Matters More Than Parenchyma Gut Dysbiosis in Different Types of Portal Hypertension

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Background: Although changes to the gut microbiota

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are usually associated with liver failure in cirrhosis, the exact role of portal hypertension (PH) in causing these changes is unclear. Aim: To determine the impact of different types of PH on gut microbiota composition.

Materials and Methods: A cross-sectional study enrolled 91 participants: 25 patients with non-cirrhotic, non-malignant portal vein thrombosis (PVT group), 29 patients with liver cirrhosis (LC group), 15 patients with combined cirrhosis and PVT (LC-PVT group), and 22 healthy controls. PVT was confirmed by contrast-enhanced computed tomography. According to transient elastography, liver fibrosis stages were F0-F2 in the PVT group and F4 in all cirrhotic patients. Morning stool samples were collected for 16S rRNA gene sequencing using the DNBSEQ-G50 platform (MGI).

Results: In comparisons with controls, the relative abundance of Proteobacteria and Bacilli was significantly increased in the PVT, LC, and LC-PVT groups ($p = 0.033, 0.052, 0.018$ and $p = 0.001, <0.001, <0.001$, respectively), whereas the abundance of Clostridia and Euryarchaeota was significantly decreased ($p = 0.001, <0.001, <0.001$ and $p = 0.045, 0.006, 0.001$, respectively). No statistically significant differences were observed among the PH groups in the abundance of Proteobacteria ($p = 0.590, 0.675$, and 0.980), Euryarchaeota ($p = 0.149, 0.152$, and 0.655), Bacilli ($p = 0.263, 0.069$, and 0.235), or Clostridia ($p = 0.590, 0.823$, and 0.862) for the comparisons PVT vs. LC, PVT vs. LC-PVT, and LC-PVT vs. LC, respectively. The abundance of bacteria from the phylum Actinobacteria was significantly increased only in the PVT group compared with controls ($p = 0.008$), while no significant differences were observed in the cirrhotic groups ($p = 0.816$ and $p = 0.255$). Furthermore, Actinobacteria abundance was higher in the PVT group than in both cirrhotic groups ($p = 0.010$ and $p < 0.001$). A significant reduction in the abundance of the family Porphyromonadaceae was observed in both PVT groups (cirrhotic and non-cirrhotic) compared with controls ($p = 0.003$ and $p < 0.001$), whereas no significant difference was detected in the LC group ($p = 0.089$).

Conclusions: The marked similarity in the alterations to the gut microbiota observed in different types of PH, which are characterized by an increased abundance of Proteobacteria and Bacilli, as well as a decreased abundance of Clostridia and Euryarchaeota, suggests that PH itself, rather than hepatic dysfunction, is a key driver of gut dysbiosis.

Keyword(s) : gut microbiota, portal vein thrombosis, liver cirrhosis, 16S rRNA, Proteobacteria, Clostridia

1510 - INTEGRATED BIOINFORMATIC ANALYSIS OF THE ROLE OF SLC35B2 IN HEPATOCELLULAR CARCINOMA

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Background / Hypothesis:

SLC35B2 is a Golgi-resident transporter of the sulfate donor 3'-phosphoadenosine 5'-phosphosulfate (PAPS), which has been shown to play a crucial role in promoting tumor growth in hepatocellular carcinoma (HCC). However, the precise mechanisms underlying SLC35B2 expression in HCC remain largely unknown. This study aims to elucidate the mechanisms of SLC35B2 expression in HCC through comprehensive bioinformatic analyses.

Materials and Methods:

SLC35B2 expression and its prognostic significance across various cancers were assessed using SangerBox. In HCC, associations with prognosis, immune infiltration, and mutations were analyzed via UALCAN, BoxKaplan-Meier Plotter, GEPIA, Human Protein Atlas, TIMER 2.0, and SangerBox. Co-expressed genes were identified using STRING, and miRNA-gene interactions were explored through the DIANA-MicroT Webserver.

Results:

SLC35B2 expression was significantly elevated in various cancers, including GBM, LGG, UCEC, BRCA, LUAD, ESCA, STES, KIRP, COAD, PRAD, STAD, HNSC, LUSC, LIHC, SKCM, BLCA, OV, PAAD, TGCT, UCS, LAML, and CHOL. In HCC, high SLC35B2 levels correlated with poorer survival and were associated with increased immune cell infiltration, specifically macrophages, monocytes, and CD4 T cells, as well as TP53 mutations. Mechanistic analysis revealed co-expressed genes, including EXT2, B3GAT3, EXTL3, B4GALT7, SLC35D2, SLC35D1, SULT4A1, SLC35D3, RPL15, and TPST2. Additionally, hsa-miR-29a-3p and hsa-miR-29c-3p were identified as potential regulators of SLC35B2 expression.

Conclusions:

Our findings highlight the oncogenic role of SLC35B2 and its prognostic significance across various cancers. In HCC, elevated SLC35B2 expression influences tumorigenesis, progression, and prognosis, suggesting its potential as a diagnostic biomarker and therapeutic target.

Keyword(s) : SLC35B2; HCC

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1511 - A PAN CANCER ANALYSIS OF THE ROLE OF OXCT1 IN HUMAN CANCERS

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Background / Hypothesis:

OXCT1 (3-oxoacid CoA-transferase 1), a rate-limiting enzyme in the process of ketolysis, plays a multifaceted role in hepatocellular carcinoma (HCC) by regulating ketone body metabolism and protein succinylation, thereby promoting tumor progression. However, the expression patterns and functional roles of OXCT1 in various cancers remain largely undefined. This study aims to conduct a pan-cancer analysis to investigate the role of OXCT1 across different human cancers.

Materials and Methods:

Data for this study were obtained from the Cancer Genome Atlas, the Gene Expression Omnibus, and the Clinical Proteomic Tumor Analysis Consortium databases. We employed the R programming language and various online tools to analyze and visualize OXCT1 expression across different cancer types, as well as its associations with tumor molecular subtypes, immune subtypes, diagnosis, prognosis, clinical characteristics, and the immune microenvironment.

Results:

Variation analysis revealed that OXCT1 was differentially expressed across numerous cancer types, including LGG, BRCA, ESCA, STES, KIRP, KIPAN, COAD, PRAD, STAD, LUSC, LIHC, THCA, READ, PAAD, TGCT, UCS, LAML, KICH, and CHOL. Clinical analysis demonstrated that OXCT1 expression significantly correlated with clinical stages or grades in several cancer types. Among different cancer subtypes in the TCGA dataset, high OXCT1 expression was associated with poor prognosis in LAML, LIHC, BLCA, and UVM. Furthermore, we explored the oncogenic role of OXCT1 by analyzing its association with immune infiltration, finding that in most cancer types, OXCT1 expression was positively correlated with the infiltration of macrophages and NK cells. Mechanistic analysis of OXCT1's molecular function indicated that OXCT2, AACS, BDH1, HMGCL1, ACAA2, HMGCL, BDH2, HMGCS2, ACAT1, and ACAT2 may play critical roles in its interactions.

Conclusions:

Our findings underscore the oncogenic role of OXCT1 and its prognostic significance across various cancers, reinforcing its functional activity in cancer types

exhibiting aberrant OXCT1 expression. These studies warrant further molecular assays to elucidate the potential role of OXCT1 in tumorigenesis and provide a foundation for developing novel therapeutic strategies.

Keyword(s) : OXCT1; HCC

1512 - COMPREHENSIVE ANALYSIS OF THE ROLE OF TUMOR ASSOCIATED ANTI LMNB1 AUTOANTIBODY IN HEPATOCELLULAR CARCINOMA

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Background / Hypothesis:

Tumor-associated autoantibodies (TAABs) are produced by cancer patients in response to tumor-associated antigens (TAAs) and have emerged as promising biomarkers for early cancer detection. Recent studies indicate that LMNB1 plays a significant role in the development of hepatocellular carcinoma (HCC). However, detailed information regarding the regulatory mechanisms of HCC progression and the clinically aggressive phenotypes associated with TAABs is lacking. This study conducts a comprehensive analysis to investigate the role of tumor-associated anti-LMNB1 autoantibody in HCC.

Materials and Methods:

Study cases were obtained from the GEO (GSE144269, GSE14520, GSE22058, GSE25097, GSE36376, GSE63898), ICGC, and TCGA_GTEEx databases. Proteomic data were downloaded from PDC000198, PXD008373, and PXD036048. We utilized the R package and the online tool hccTAAb to perform various analyses, including gene and protein expression, survival analysis, immune infiltration, similarity analysis, and differential DNA methylation analysis.

Results:

The expression of LMNB1 in cancer tissues showed a significant increase compared to normal tissues across the GEO, ICGC, and TCGA_GTEEx transcriptomics datasets. Additionally, proteomic data from PDC000198, PXD008373, and PXD036048 also demonstrated elevated LMNB1 expression in HCC cases compared to healthy controls. Survival analysis revealed that higher LMNB1 expression correlates with poorer prognosis. Cox regression analysis for the TCGA-LIHC dataset yielded a hazard ratio (HR) of 1.32 (95% CI: 1.14-1.52, P=0.00122), while the ICGC-LIRI

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dataset yielded an HR of 2.17 (95% CI: 1.48-3.17, $P=0.00092$). Immune infiltration analysis using ssGSEA revealed significant differences in immunological features between high- and low-expression groups, including aDCs, macrophages, and Th2 cells. Similarity analysis identified the top 5 genes (TOP2A, GINS1, TPX2, KIFC1, NCAPG) exhibiting positive correlations, while LDHD, HPD, SLC27A5, CFHR4, and HPR showed negative correlations.

Conclusions:

Our findings underscore the role of tumor-associated anti-LMNB1 autoantibody and its prognostic significance in HCC. These studies warrant further molecular assays to elucidate the potential role of LMNB1 in tumorigenesis and to provide a foundation for future research.

Keyword(s) : LMNB1; HCC

1513 - PAN CANCER ANALYSIS OF HACD FAMILY AND UNRAVELLING THE ONCOGENIC ROLE IN HEPATOCELLULAR CARCINOMA

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Background / Hypothesis:

The HACD (3-Hydroxyacyl-CoA Dehydratase) protein family has demonstrated crucial and diverse roles in various cancers. However, a comprehensive understanding of the distinct contributions of different HACD family members across tumor types remains largely unexplored. This study conducts a systematic analysis to investigate the role of the HACD family in a range of human cancers, with a particular emphasis on hepatocellular carcinoma (HCC).

Materials and Methods:

Study cases were obtained from the Cancer Genome Atlas database, the Gene Expression Omnibus database, and the Clinical Proteomic Tumor Analysis Consortium. We utilized the R package and various online tools to analyze and visualize HACD expression across different cancer types, as well as its associations with tumor molecular subtypes, immune subtypes, diagnosis, prognosis, clinical characteristics, methylation status, and the immune microenvironment. Co-expressed proteins and regulatory miRNAs were identified through mechanistic analysis.

Results:

Our study reveals significant genetic heterogeneity among HACD family members, along with distinct clinical characteristics. Notably, compared to normal samples, the expression of HACD1 is significantly reduced in tumor samples, while the expression of HACD2 and HACD3 is markedly increased, with HACD4 showing no significant change. High expression of HACD2 has been associated with poor survival outcomes in patients with HCC. Mechanistic analysis identified genes co-expressed with HACD2, including HACD3, HACD4, ELOVL4, FADS1, ELOVL2, SCD5, TECR, SCD, HSD17B12, and ELOVL6. Additionally, hsa-miR-376c-3p may play a critical role in the regulatory interaction with HACD2.

Conclusions:

In summary, the findings of this study provide valuable insights to guide therapeutic strategies aimed at harnessing the impact of HACD family genes in cancer treatment. Our research warrants further molecular assays to elucidate the potential role of HACD2 in tumorigenesis and its viability as a therapeutic target in HCC.

Keyword(s) : HACD; HCC

1514 - AN INTEGRATED ANALYSIS OF THE ROLE OF tsRNA Asp 5 0002 IN HUMAN TUMORS ACROSS MULTIPLE CANCER TYPES

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Background / Hypothesis:

tsRNA-derived small RNA (tsRNA) represents a novel class of regulatory small noncoding RNA that plays a significant role in physiological processes as well as in the development of malignant tumors. tsRNA-Asp-5-0002, a 5'-tRNA fragment (tRF) consisting of 18 nucleotides (TCCTCGTTAGTATAGTGG), has recently been identified as a potential biomarker for hepatocellular carcinoma. However, its expression patterns and biological effects across various cancer types remain largely unexplored. This study aims to conduct a pan-cancer analysis to elucidate the role of tsRNA-Asp-5-0002 in different human cancers.

Materials and Methods:

Data for this study were obtained from the Cancer Genome Atlas (TCGA) database. We employed the R programming language along with several online tools to analyze and visualize the expression levels of tsRNA-Asp-5-0002 across 32 different cancer types, as well as

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its association with patient prognosis.

Results:

Analysis using the tsRinCancer module of the tsRFun platform revealed that tsRNA-Asp-5-0002 was differentially expressed in multiple cancer types, including ACC, DLBC, LGG, MESO, OV, READ, SARC, SKCM, TGCT, UCS, and UVM. Univariate Cox regression analysis conducted through the tsRSurvival module indicated that tsRNA-Asp-5-0002 was significantly associated with prognosis in several cancer types: CESC (cervical squamous cell carcinoma and endocervical adenocarcinoma, $P=0.005177$), COAD (colon adenocarcinoma, $P=0.023525$), KIRC (kidney renal clear cell carcinoma, $P=0.029378$), LUSC (lung squamous cell carcinoma, $P=0.023236$), and THYM (thymoma, $P=0.037697$). Furthermore, we identified 16 potential target genes predicted by the bioinformatics database miRDB: C1orf131, KIF24, FYTDD1, ZNF470, BTBD19, SHOX2, PLEKHG1, GATAD2A, CD48, GID4, GORASP2, NOTCH1, ATXN7, CIT, POTEF, and FBLN1.

Conclusions:

Our findings underscore the oncogenic role of tsRNA-Asp-5-0002 and its prognostic significance across various cancers, particularly in CESC, COAD, KIRC, LUSC, and THYM. These results warrant further molecular assays to deepen our understanding of the potential role of tsRNA-Asp-5-0002 and its target genes in tumorigenesis, providing a foundation for the development of novel therapeutic strategies.

Keyword(s) : tsRNA-Asp-5-0002; HCC

1515 - Investigating the Hidden Link How Common Is MAFLD Among Cataract Patients

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is now widely recognized as a multisystem disease. The liver-eye interplay is a developing research area exploring interconnected metabolic and inflammatory pathways. However, the potential association between MAFLD and cataract remains scarcely investigated. we aimed to evaluate the prevalence of MAFLD among patients with cataract and assess its association with clinical, anthropometric, and metabolic parameters.

Materials and Methods:

A cross-sectional study was conducted on 150 Egyptian patients with cataract. Participants were classified into MAFLD and non-MAFLD groups based on the established diagnostic criteria. Fatty liver indices and liver fibrosis scores were evaluated.

Results:

The prevalence of MAFLD among cataract patients was 79.3%. Nuclear cataract was the most prevalent type (64.7%) among MAFLD patients. Compared with non-MAFLD patients, those with MAFLD had significantly higher BMI, waist circumference, hip circumference, hypertension (53.8% vs 25.8%; $p = 0.005$), and HbA1c (6.41 ± 1.1 vs 6.12 ± 1.36 ; $p = 0.004$). HSI, FLI, and FIB-4 scores were all significantly higher in the MAFLD group ($p < 0.001$). Multivariate analysis identified hip circumference (OR = 1.188), HSI (OR = 1.531), and FIB-4 (OR = 2.249) as independent predictors of MAFLD in cataract patients.

Conclusions:

MAFLD is highly prevalent among cataract patients, particularly those with nuclear cataracts. Hepatic steatosis index, FIB-4 score, and hip circumference were significant predictors of MAFLD. These findings emphasize the need for routine liver evaluation in cataract patients to enable early detection of MAFLD and to guide strategies for reducing metabolic, systemic, and ocular complications.

Keyword(s) : Cataract, Fibrosis, Metabolic dysfunction-associated fatty liver disease



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1518 - Performance of Non Invasive Techniques for Quantifying Hepatic Steatosis Limitations of Conventional MRI Compared with FibroScan in a Preliminary Moroccan Cohort

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Background:

Hepatic steatosis is a growing public health concern, closely linked to obesity, diabetes, and dyslipidemia. Its non-invasive assessment relies mainly on transient elastography (FibroScan) and magnetic resonance imaging (MRI). However, conventional MRI without specialized sequences such as Proton Density Fat Fraction (MRI-PDFF) may underestimate steatosis. Among a cohort of 170 patients with hepatic steatosis, only 12 underwent both FibroScan and MRI, allowing preliminary evaluation of concordance. This study also aims to raise awareness among clinicians regarding the non-equivalence of conventional MRI and FibroScan in routine practice, and to inform the development of locally-adapted diagnostic algorithms.

Materials and Methods:

This retrospective preliminary study included 12 patients selected from a total cohort of 170 confirmed hepatic steatosis cases. Demographic parameters (age, sex), clinical features (BMI, metabolic risk factors), laboratory findings (cytolysis, cholestasis, thyroid panel, metabolic markers), virological status (HBV/HCV), and results from FibroScan (fibrosis and steatosis stages) and MRI (qualitative assessment) were collected. Concordance between FibroScan and MRI was defined as the simultaneous presence or absence of steatosis, while discordance indicated conflicting findings. Descriptive statistics and qualitative analyses of discordant cases were performed.

Results:

The subgroup included 9 women (75%) and 3 men, with a mean age of 51.9 years and mean BMI of 32.7 kg/m². Metabolic risk factors were frequent, including diabetes, hypertension, and dyslipidemia. FibroScan identified severe steatosis (S3) in 66% of patients and significant fibrosis in 25%. Conventional MRI detected steatosis in 75% of patients, primarily in a descriptive manner without quantification.

Concordance between FibroScan and MRI was

observed in 8 patients (66%). Discordance occurred in 4 patients (33%): 3 patients had steatosis detected by FibroScan but not by MRI, and 1 patient had steatosis described by MRI but not confirmed by FibroScan. These discrepancies highlight the limitations of conventional MRI sequences for semi-quantitative assessment, particularly in moderate or heterogeneous steatosis.

Conclusion:

This preliminary study demonstrates significant discrepancies between conventional MRI and FibroScan in assessing hepatic steatosis, with a concordance rate of 66%. Conventional MRI is limited by sequence choice and operator dependency. MRI-PDFF, not yet available in Morocco, provides objective, reproducible quantification and is essential for precise monitoring of metabolic liver disease. These findings underscore the need for clinician awareness, the development of locally-adapted diagnostic algorithms, and further larger studies reflecting real-world practice to optimize non-invasive assessment and patient management.

Keyword(s) : Hepatic steatosis, Fibroscan, proton density fat fraction, Magnetic resonance imaging, Non invasive diagnostic methods

1519 - Real world outcomes of atezolizumab and bevacizumab according to clinical risk factors for unresectable hepatocellular carcinoma A multicenter CLCSG cohort study

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Background / Hypothesis:

Atezolizumab plus bevacizumab (Ate/Bev) is the established first-line therapy for advanced hepatocellular carcinoma (HCC), but evidence in real-world high-risk populations remains limited. Real-world cohorts often include high-risk patients, leading to less favorable outcomes compared with IMbrave150. This study aimed to evaluate survival outcomes in high-risk patients to help guide the need for more intensive treatment approaches.

Materials and Methods:

We retrospectively analyzed 792 patients with advanced HCC who received Ate/Bev between 2022 and 2024 at multiple centers in South Korea. In this study, high risk group (n= 261, 32.9%) was defined such as main portal vein invasion (Vp4), tumor involvement $\geq$ 50% of the liver volume, infiltrative type or bile duct invasion.

Results:

In the entire cohort, the median PFS and OS were 6.97 months (95% CI, 6.24–7.69) and 14.39 months (95% CI, 13.01–15.78), respectively. In the high-risk group, the median PFS was 5.92 months (95% CI, 4.73–7.09) and the median OS was 10.7 months (95% CI, 9.7–11.8), whereas in the non-high-risk group the median PFS was 7.85 months (95% CI, 6.69–9.02) and the median OS was 18.0 months (95% CI, 16.3–19.6). Both PFS and OS differed significantly between the two groups (Both p-value < 0.001). High-risk status was not associated with differences in ORR (53.3% vs. 50.6%, p = 0.536) or DCR (75.1% vs. 71.6%, p = 0.292). Variceal bleeding was observed in 5.7% (n=45) of patients and tended to be slightly more frequent in the high-risk group (6.7% (n=24) vs. 4.9% (n=21)), while severe proteinuria ($\geq$ 3+) occurred in 8.8% (n=70) and appeared somewhat more common in the non-high-risk group (10.7% (n=44) vs. 7.2% (n=26)). Neither difference reached statistical

significance (bleeding p = 0.274; proteinuria p = 0.146). Most bleeding events occurred within the first year.

Conclusions:

Real-world experience provides substantial clinical data for Ate/Bev, but treatment responses remain relatively poor in high-risk patients.

Keyword(s) : Hepatocellular carcinoma, real-world study, Atezolizumab-bevacizumab, high risk population

1520 - Hole in the Wall A Rare Case of Lenvatinib Associated Peptic Ulcer Perforation in Hepatocellular Carcinoma

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Introduction: Lenvatinib is a type I tyrosine kinase inhibitor with antiangiogenic activity, approved as first-line treatment for patients with advanced, unresectable hepatocellular carcinoma (HCC). Gastrointestinal (GI) adverse effects are most commonly observed within the first six months of treatment initiation. Among these, gastrointestinal perforation is a rare but life-threatening complication, occurring in approximately 1.3% of patients receiving lenvatinib.

Case Presentation: We report the case of a 54-year-old Asian male with unresectable HCC who presented to the emergency department with acute, severe abdominal pain after two months of lenvatinib therapy (8 mg daily). An esophagogastroduodenoscopy (EGD) performed prior to treatment initiation revealed no abnormalities. Physical examination showed diffuse abdominal tenderness with guarding. Laboratory results were notable for elevated serum creatinine, hyponatremia, and prolonged INR. A non-contrast CT scan of the abdomen revealed massive pneumoperitoneum with extraluminal air surrounding the stomach and diffuse thickening of the gastric fundus wall. Empiric antibiotic therapy with piperacillin-tazobactam was initiated for suspected peritonitis secondary to GI perforation. An emergent exploratory laparotomy revealed a 2x2 cm perforated peptic ulcer on the lesser curvature of the stomach. Modified Graham patch repair was performed, and biopsy specimens were negative for *Helicobacter pylori*. Histopathology showed chronic inflammation, hemorrhage, and ulceration. The patient recovered well

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postoperatively and was discharged in stable condition.

Conclusion: This case highlights the importance of clinician awareness regarding the potential for serious, albeit uncommon, gastrointestinal complications associated with lenvatinib therapy. Early recognition and timely surgical intervention are critical to improving patient outcomes and reducing the risk of morbidity and mortality.

Keyword(s) : "lenvatinib", "perforated peptic ulcer", "unresectable hepatocellular carcinoma"

1521 - Risk Factors for Liver Fibrosis Severity in Patients with Nonalcoholic Steatohepatitis A Study Based on Liver Pathology

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Background / Hypothesis:

In order to determine the independent risk factors associated with the severity of liver fibrosis in patients with nonalcoholic steatohepatitis (NASH), we conducted this study from the perspective of liver pathological results and biochemical indicators.

Materials and Methods:

A retrospective analysis was conducted on 122 patients diagnosed with NASH via liver biopsy at Tianjin Second People's Hospital between December 2014 and December 2024. Clinical, laboratory, and pathological data were collected. Patients were categorized into F1, F2, and F3 stages based on fibrosis scores. Univariate analysis was used to compare variables across fibrosis stages. Spearman correlation was employed to assess associations between variables and fibrosis severity. Ordinal logistic regression with backward stepwise selection was applied to identify independent risk actors, with odds ratios (ORs) and 95% confidence intervals (CIs) calculated.

Results:

The mean age of participants was 41.6 ± 14.2 years, with 66 males (54.1%). According to fibrosis staging, there were 21 cases in F1 stage, 86 in F2 stage, and 15 in F3 stage. Univariate analysis showed that the degree of hepatic steatosis and lobular inflammation, levels of aspartic transaminase (AST) and low-density lipoprotein (LDL), prevalence of diabetes increased with increasing severity of fibrosis; while serum albumin

(ALB) levels and estimated glomerular filtration rate (eGFR) decreased with increasing severity of fibrosis. These differences were statistically significant ($P < 0.05$). Spearman correlation analysis indicated that diabetes, lobular inflammation, hepatic steatosis, AST, and LDL were positively correlated with fibrosis stage ($p > 0$, $P < 0.05$); AST and ALT were positively correlated with lobular inflammation ($p > 0$, $P < 0.05$); AST was positively correlated with the degree of hepatic steatosis ($p > 0$, $P < 0.05$); and AST showed a marginal positive correlation with ballooning degeneration ($P = 0.068$). Ordinal logistic regression identified lobular inflammation (OR = 5.058, 95%CI: 2.153-11.693, $P < 0.001$), hepatic steatosis (OR = 2.768, 95%CI: 1.398-5.474, $P = 0.003$), diabetes (OR=3.815, 95%CI: 1.220-11.929, $P = 0.021$), and elevated LDL levels (OR=1.669, 95%CI: 1.067-2.609, $P = 0.025$) as independent risk factors associated with the severity of liver fibrosis in patients with NASH.

Conclusions:

Lobular inflammation, hepatic steatosis, diabetes, and elevated LDL levels are independent risk factors associated with the severity of liver fibrosis in patients with NASH.

Keyword(s) : Nonalcoholic steatohepatitis; Liver fibrosis; Risk factors; Pathology

1522 - Efficacy and safety of hepatic arterial infusion chemotherapy sequential therapy versus Atezolizumab Bevacizumab in infiltrative hepatocellular carcinoma with portal vein thrombosis A cohort study with Propensity score matching

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Background / Hypothesis:

Infiltrative hepatocellular carcinoma (HCC) is an aggressive and biologically distinct subtype that often carries a poor prognosis. Because of its molecular characteristics—such as immunosuppressive signaling and low immune infiltrates—these tumors derive limited benefit from immunotherapy, highlighting the need for alternative treatment strategies. This study evaluates a hepatic arterial infusion chemotherapy (HAIC)-based sequential approach compared with atezolizumab and bevacizumab (ate/bev) in patients with infiltrative HCC with portal vein thrombosis.

Materials and Methods:

This cohort study included patients with infiltrative HCC with portal vein thrombosis treated with atezolizumab–bevacizumab (n=214) or HAIC sequential therapy (n=38) as initial treatment. HAIC based sequential approach consisted of six HAIC cycles with 4 week intervals using cisplatin 25mg/m² and 5-fluorouracil 750mg/m² for 4 days followed by tailored subsequent therapies including target agents, ate/bev or local therapy. 2:1 Propensity score matching was applied to adjust for potential baseline imbalances.

Results:

After propensity score matching, the median PFS was 8.05 months (95% CI, 5.63–10.47) in the Atezolizumab–Bevacizumab (Ate/Bev) group and 11.31 months (95% CI, 4.15–18.46) in the HAIC plus sequential therapy group (log-rank p = 0.005). The median OS was 15.01 months (95% CI, 11.41–18.62) in the Ate/Bev group compared with 20.27 months (95% CI, 11.97–28.59) in the HAIC plus sequential therapy group (log-rank p < 0.001). The objective response rate (ORR) was significantly higher in the HAIC plus sequential group compared with the Ate/Bev group (68.3% vs. 28.6%, p < 0.001). The disease control rate (DCR) was 92.1% in the HAIC plus sequential group vs. 78.6% in the Ate/Bev group (p = 0.020). Among the 38 patients treated with HAIC-based sequential therapy, grade ≥3 adverse events occurred in 12 patients (31.6%) This rate was not statistically significantly different from 50 of 219 patients (22.8%) observed in the Ate/Bev group (p=0.241).

Conclusions:

HAIC-based sequential treatment can be effective and

safe management alternative to atezolizumab and bevacizumab especially in patients with good liver function and performance status for infiltrative HCC with portal vein thrombosis .

Keyword(s) :

1523 - Anserine promotes Antibacterial Defense in Cirrhosis via the Mup11+ Hepatocyte CD44+ pDC Crosstalk and CCL5 CCR5 Axis Short title Anserine Promotes Antibacterial Defense via CCL5 pDC in Cirrhosis

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Background / Hypothesis: Patients with liver cirrhosis exhibit heightened susceptibility to severe bacterial infections, yet the underlying immune defects remain poorly characterized. We investigated the dysfunctional intercellular crosstalk within the cirrhotic liver that impairs antibacterial defense.

Materials and Methods:Employing integrated multi-omics, we performed single-cell RNA sequencing (scRNA-seq) on livers from control, infected control, cirrhotic, and infected cirrhotic mice. Key cellular interactions were validated using flow cytometry, functional assays (ex vivo/in vivo), targeted metabolomics, adoptive cell transfer, specific antibody blockade, and cell co-culture.

Results:scRNA-seq revealed a significantly depleted CD44+ plasmacytoid dendritic cells (pDCs) subpopulation responsible for impaired antibacterial immunity in cirrhosis. Multicellular cluster modeling of ligand-receptor interactions revealed that Mup11+ hepatocytes play a key immunoregulatory role via interaction with CD44+ pDCs in response to bacterial infection. Mup11+ hepatocytes play a crucial role in the recruitment of CD44+ pDCs via the CCL5-CCR5 interaction. Furthermore, targeted metabolomics and animal experiments identified histamine metabolite anserine increased the percentage of Mup11+ hepatocytes and CD44+ pDCs to decrease the bacterial burden and mortality in cirrhosis with infection. Finally, clinical validation in a national, multicenter and prospective patient cohort revealed that, CCL5, CD44+ pDCs and anserine are significantly correlated with the incidence of infection and outcome in patients with cirrhosis.

Conclusions: Our study uncovers a novel metabolic-

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immune axis wherein anserine potentiates Mup11+ hepatocytes-CD44+ pDCs crosstalk via CCL5-CCR5 to enhance antibacterial defense in cirrhosis. This multi-omics-driven discovery, validated across models and human cohorts, identifies therapeutic targets for a high-risk cirrhotic population.

Keyword(s) : Cirrhosis; Bacterial infection; Plasmacytoid dendritic cells (pDCs); Anserine; CCL5-CCR5 axis.

1524 - Carbamazepine induced cholestatic hepatitis with rapid fibrosis progression and biopsy confirmed pure red cell aplasia in a young woman

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Background / Hypothesis:

Carbamazepine can cause idiosyncratic hepatic and hematologic toxicity, but the combination of persistent cholestatic hepatitis with rapid progression to advanced fibrosis and concurrent pure red cell aplasia (PRCA) is exceptionally uncommon. This case highlights a rare pattern of multisystem toxicity following short-term therapeutic exposure

Materials and Methods:

A 26-year-old previously healthy woman was initiated on carbamazepine 100 mg twice daily for suspected trigeminal neuralgia. Within one week she developed jaundice, pruritus and dark urine. Evaluation included viral serology (hepatitis A, B, C, E; Epstein-Barr virus; cytomegalovirus), autoimmune markers, metabolic studies, abdominal ultrasonography, magnetic resonance cholangiopancreatography and liver biopsy.

A short empiric course of corticosteroids was administered early in the illness without significant biochemical response. The subsequent development of severe anaemia prompted haematologic evaluation including bone marrow trephine biopsy. Causality for drug-induced liver injury (DILI) was assessed using established clinical criteria

Results:

All viral, autoimmune and metabolic aetiologies were excluded. Imaging demonstrated normal biliary anatomy. Liver biopsy revealed preserved lobular architecture with marked canalicular cholestasis and minimal inflammation, consistent with cholestatic DILI. Despite drug withdrawal and supportive care, cholestasis persisted. Transient elastography suggested advanced fibrosis (F3), and follow-up computed tomography at six months showed radiological features of cirrhosis, indicating rapid fibrosis progression.

Three months after presentation she developed profound anaemia. Bone marrow trephine showed complete absence of erythroid precursors with preserved granulopoiesis and megakaryopoiesis, confirming pure red cell aplasia. No marrow fibrosis, infiltrative process or viral trigger was identified. Darbepoetin therapy resulted in gradual haemoglobin normalisation and resolution of transfusion dependence. Liver biochemistry improved but remained cholestatic.

The chronology, exclusion of alternative causes and concurrent hepatic and marrow involvement support carbamazepine-induced multisystem drug toxicity

Conclusions:

Short-term carbamazepine exposure precipitated persistent cholestatic hepatitis with rapid progression to advanced fibrosis and concurrent biopsy-confirmed pure red cell aplasia. This case emphasises the importance of early drug withdrawal and careful monitoring for multisystem involvement in suspected DILI. Erythropoiesis-stimulating therapy may aid recovery in secondary PRCA associated with drug-induced liver injury

Keyword(s) : Drug-induced liver injury, Cholestatic hepatitis, Pure red cell aplasia, Carbamazepine toxicity, Advanced fibrosis

1530 - Integrative lipidomic and transcriptomic analyses reveal glycosphingolipid remodeling in metabolic dysfunction associated fatty liver disease related advanced fibrosis

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Background / Hypothesis:

The prevalence of metabolic dysfunction-associated fatty liver disease (MAFLD)-related advanced fibrosis is increasing, yet effective therapeutic options remain limited. Lipid metabolic dysregulation plays a pivotal role in fibrosis progression. This study integrated lipidomic and transcriptomic analyses to elucidate lipid alterations and underlying mechanisms in MAFLD-related advanced fibrosis.

Materials and Methods:

Paired plasma lipidomic and liver transcriptomic analyses were conducted in 20 biopsy-proven MAFLD patients (40% with advanced fibrosis). Differential lipid species were determined by limma with multivariable adjustment, and differentially expressed genes (DEGs) were determined by DESeq2. Functional enrichment of DEGs was performed via Gene Ontology (GO) biological process terms. Public transcriptomic datasets from human MAFLD and murine liver fibrosis models (high-fat diet [HFD], carbon tetrachloride [CCl₄], and choline-deficient amino acid-defined high-fat diet [CDAA-HFD]) were analyzed for validation.

Results:

Lipidomic analysis quantified 604 lipid species across 33 classes. In patients with advanced fibrosis, substantial alterations in glycosphingolipids were observed, characterized by marked increases in lactosylceramides (LacCer) and significant reductions in monosialodihexosyl ganglioside (GM3). Furthermore, the patients' hepatic transcriptomic analysis revealed upregulation of glycosphingolipid metabolic genes (e.g., UGCG and B3GNT5), alongside downregulation of GM3 biosynthetic genes (e.g., NEU3, NEU4, and ST3GAL5). These alterations were consistently observed in public human and mouse fibrosis datasets.

Conclusions:

MAFLD-related advanced fibrosis exhibits distinct remodeling of glycosphingolipid metabolism, particularly involving LacCer and GM3 pathways. These findings highlight potential lipid metabolic targets for antifibrotic therapy and warrant further *in vivo* validation.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease, Lipidomic, Transcriptomic, Glycosphingolipid metabolism.

1532 - A Rare Case Of Acute Liver Failure (ALF) Secondary To Clozapine Induced Autoimmune Hepatitis

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Background:

A 53yo Chinese woman with longstanding treatment-resistant schizoaffective disorder on long-term Olanzapine 20mg ON was admitted in Jan 2025 for relapse of schizophrenia. Her Liver Function Tests (LFTs) then were normal.

She was initiated on Clozapine 25mg daily on February 27 with dose escalation of 25mg every 3 days, reaching a target of 300mg daily on March 30. Olanzapine was discontinued. While her psychiatric symptoms improved, she developed significant lethargy and drowsiness on April 2.

Her LFTs then showed significant derangement: Bil 76µmol/L, ALT2078 U/L, ALP348 U/L, GGT301U/L, and INR 2.13. A comprehensive screen for viral and metabolic liver diseases was negative. A diagnosis of possible acute liver failure was established, though the patient was deemed unsuitable for liver transplantation.

While standard autoimmune liver markers (ANA, anti-LKM1, ASMA, AMA) returned as negative, serum Immunoglobulin G (IgG) was markedly elevated at 32.5g/L. An urgent liver biopsy was performed on April 4. The patient was initiated on plasma-exchange for 3 days (April 5–7) and concurrently received IV N-acetylcysteine.

Liver biopsy histology demonstrated confluent midzonal and centrilobular necrosis with mixed inflammatory infiltrate of neutrophils, plasma cells, lymphocytes, histiocytes, and eosinophils, without evidence of fibrosis.

IV hydrocortisone 100mg q8-hourly was initiated on April 8 with improvement of her LFTs and mentation (refer to summarized chart). An attempt to wean her to oral prednisolone 50mg daily on April 15 led to rapid LFT worsening, necessitating immediate escalation of

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steroid therapy back to IV hydrocortisone 100mg q6-hourly the following day.

After 37 days of continued IV hydrocortisone, her LFTs gradually improved. She successfully transitioned to oral prednisolone 30mg daily on May 15. Azathioprine 25mg daily was introduced on May 13 and increased to 50mg daily from May 15 onward, while oral prednisolone dose was further weaned.

Her LFTs normalised by May 19 (bil 24 μ mol/L, ALT 35U/L, ALP 125U/L). Concurrently, her IgG levels reached normal limits within three weeks of initiating steroid therapy.

Conclusion:

We present a rare case of ALF secondary to clozapine-induced severe AIH. This condition proved challenging to manage, necessitating a protracted course of high-dose intravenous hydrocortisone. Despite cessation of Clozapine, tapering IV Hydrocortisone dose (100mg q8hrly) to Oral Prednisolone (50mg daily) after 7 days of administration resulted in a rapid, severe biochemical relapse, which further supports our diagnosis of AIH.

While a few cases of clozapine-induced hepatotoxicity have been reported, none have described AIH or detailed a successful treatment outcome with steroids and immunosuppressants, making this case the first reported globally.

Keyword(s) :

1534 - FABP4 Exacerbates Drug Induced Cholestatic Liver Injury via NLRP3 GSDMD Dependent Pyroptosis

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Background / Hypothesis:

Pyroptosis, a pro-inflammatory form of programmed cell death, plays a critical role in amplifying inflammation during drug-induced cholestatic (DIC) liver injury, yet its regulatory mechanisms remain unclear. Fatty acid-binding protein 4 (FABP4) has been implicated in mediating inflammatory responses. This

study aimed to investigate the impact of FABP4 on pyroptosis in DIC and elucidate the underlying molecular mechanisms.

Materials and Methods:

The study employed Fabp4 knockout (Fabp4^{-/-}) and wild-type mice subjected to α -naphthylisothiocyanate (ANIT)-induced cholestasis, with parallel cohorts receiving the NLRP3 inhibitor MCC950. Liver tissues underwent Western blot and immunohistochemistry analysis to quantify pyroptosis executors (NLRP3, GSDMD-N), complemented by transmission electron microscopy (TEM) for ultrastructural characterization of hepatocytes. Serum ALT/AST levels and hepatic bile acid metabolism were assessed as functional indicators. Mechanistic validation included Western blot and immunohistochemical analysis of cholestatic DILI (C-DILI) patient samples and in vitro experiments where primary hepatocytes were treated with taurocholic acid (TCA) and chenodeoxycholic acid (CDCA).

Results:

ANIT challenge triggered significant NLRP3 and GSDMD-N upregulation in wild-type mice, which was markedly attenuated in Fabp4^{-/-} counterparts (Figure A). Immunohistochemistry revealed synchronized NLRP3/GSDMD overexpression in C-DILI patient livers and ANIT-WT mice, while Fabp4^{-/-} models exhibited significant suppression (Figure B-D). TEM confirmed significantly reduced characteristic pyroptotic morphology (large plasma membrane pores, ballooning degeneration) in hepatocytes from Fabp4^{-/-} mice (Figure E). Primary hepatocytes exposed to TCA/CDCA showed dose-dependent NLRP3 induction (Figure F). Crucially, MCC950 treatment significantly improved ANIT-induced liver injury (Figure H-I) and inflammation, mimicking the protective phenotype of ANIT-treated Fabp4^{-/-} mice, although without altering bile acid metabolism (Figure G-L).

Conclusions:

This study demonstrates that FABP4 exacerbates DIC liver injury by promoting NLRP3/GSDMD-dependent pyroptosis, thereby amplifying the inflammatory cascade. Therapeutic targeting of this axis via FABP4 inhibition or MCC950 analogs presents a promising strategy for DIC management, with mechanistic evidence supporting clinical translation.

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Keyword(s) : Drug-induced Cholestasis, FABP4, NLRP3 inflammasome, Pyroptosis

1535 - Multimodal In Hospital Alert Intervention Significantly Improves HBV DNA Testing and Antiviral Treatment Rates among HBsAg Positive Patients in Non Specialty Departments

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Background / Hypothesis:

Expanding screening and treatment is a core strategy for achieving the World Health Organization's goal of eliminating viral hepatitis as a major public health threat by 2030. This study aimed to evaluate the impact of a comprehensive in-hospital intervention on enhancing the HBV-DNA testing rate and subsequent antiviral treatment initiation among patients with a first-time HBsAg-positive result identified in non-infectious disease departments.

Materials and Methods:

Beginning in January 2025, our hospital implemented a comprehensive intervention to improve hepatitis B screening and management. For physicians: a pop-up alert in the Hospital Information System prompts the ordering physician to consider further action whenever an HBsAg-positive result is reported, providing quick options to "Order HBV-DNA test" or "Request Infectious Disease Department Consult." Additionally, an automated SMS reminder is sent to the physician for follow-up. For patients: the laboratory report automatically includes a comment stating "HBsAg Positive - Recommend consultation at the Infectious Disease Clinic for further assessment and HBV-DNA testing," and an SMS reminder is sent to the patient advising a visit to the Infectious Disease Clinic. We conducted a retrospective analysis comparing data from the pre-intervention period (January to September 2024) and the post-intervention period (January to September 2025).

Results:

During the pre-intervention period (Jan-Sep 2024), 14,586 eligible HBsAg-positive patients were identified in non-specialty departments. Among them, 6,957

(47.70%) underwent HBV-DNA testing. Of the 1,887 patients with a positive HBV-DNA result, 1,384 (73.34%) started antiviral treatment. Following the intervention (Jan-Sep 2025), 17,647 eligible HBsAg-positive patients were identified. HBV-DNA testing was performed in 9,254 (52.44%) of these patients. Among the 2,292 patients with detectable HBV-DNA, 2,245 (97.95%) received antiviral treatment. The increases in both HBV-DNA testing rate and treatment rate were statistically significant, $p < 0.001$.

Conclusions:

A multimodal alert Intervention via the hospital information system, SMS and laboratory report targeting both physicians and patients, significantly improved HBV-DNA testing and antiviral treatment rates among first-time HBsAg-positive patients in non-specialist departments. This integrated intervention model represents an efficient and scalable in-hospital management strategy that can contribute to achieving the global goal of eliminating viral hepatitis as a public health threat.

Keyword(s) : Multimodal In-Hospital Alert Intervention, HBV-DNA Testing, Antiviral Treatment Rates, hepatitis B screening and management

1536 - Diagnosis and treatment of occult primary hepatocellular carcinoma presenting with ovarian metastasis as the initial manifestation a case report

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Background / Hypothesis:

Primary extrahepatic hepatocellular carcinoma is a tumor that occurs outside the liver and has a histological morphology consistent with HCC. It is an extremely rare clinical phenomenon. Hepatoid Carcinoma of the Ovary (HCO), which originates in the ovary, is even rarer and has very few literature reports.

Results:

This case report based on a clearly diagnosed case of HCO and its subsequent treatment. This study was a

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single-case in-depth analysis. The research subject was a 52-year-old female who was treated at the First Affiliated Hospital of Zhejiang University School of Medicine in January 2025. After surgical treatment, the patient was diagnosed with HCO through pathological and immunohistochemical analysis. In addition, after diagnosis, the patient received an immune-targeted combination therapy based on tislelizumab combined with lenvatinib and achieved remarkable therapeutic effects, reaching a stable disease (SD) prognosis. Liver function damage related to tumor treatment occurred during the treatment process and improved after treatment.

Conclusions:

HCO can adopt the immune-targeted combination therapy regimen of tislelizumab combined with lenvatinib, which has a good therapeutic effect on tumors. At the same time, liver function damage related to tumor treatment should also be dealt with in a timely manner.

Keyword(s) : Primary Extrahepatic Hepatocellular Carcinoma, Hepatoid Carcinoma of the Ovary, Treatment strategy

1537 - Targeting the Cholangiocytes Vitamin D TXNIP Pathway to Attenuate Ductular Reaction and Liver Fibrosis

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Background / Hypothesis:

While ductular reaction (DR) is a recognized pathological hallmark of progressive liver injury, no targeted pharmacological interventions exist to modulate this process. Epidemiological evidence indicates a high prevalence of vitamin D deficiency in chronic liver diseases, yet the molecular mechanisms underpinning its potential hepatoprotective functions are poorly defined.

Materials and Methods:

We first established a clinical correlation between

plasma vitamin D concentrations and DR severity in patients. The functional impact of vitamin D signaling was then mechanistically dissected in a 3,5-diethoxycarbonyl-1,4-dihydrocollidine (DDC)-induced murine liver injury model. Mice were administered 1,25(OH)2D3, the bioactive vitamin D metabolite. A cholangiocyte-specific Txnip-knockout mouse model was generated to define the cell-autonomous role of this key vitamin D target gene. A combination of in vivo phenotyping, primary cell culture, and molecular analyses was employed to assess DR, inflammation, fibrosis, cellular proliferation/death, and paracrine signaling.

Results:

Clinical data indicated a significant inverse relationship between systemic vitamin D levels and DR magnitude. 1,25(OH)2D3 supplementation in female mice potently suppressed DDC-triggered DR, inflammatory infiltration, and fibrotic deposition. This protection was coupled with the transcriptional induction of TXNIP within the biliary compartment. Genetic ablation of Txnip specifically in cholangiocytes markedly exacerbated the injury phenotype, enhancing DR, inflammation, and fibrogenesis. At the cellular level, Txnip deficiency resulted in a pronounced pro-proliferative and anti-apoptotic phenotype in cholangiocytes. Furthermore, Txnip-null cholangiocytes exhibited enhanced secretion of TNF- α and TGF- β , which in a paracrine manner activated Kupffer cells and hepatic stellate cells, thereby driving inflammation and collagen production. Critically, the protective effects of 1,25(OH)2D3 were entirely abrogated in biliary-specific Txnip-knockout mice, establishing TXNIP as a necessary downstream effector. Conversely, TXNIP overexpression was sufficient to attenuate the DDC-induced pathological cascade.

Conclusions:

This study elucidates a previously uncharacterized mechanistic pathway wherein vitamin D signaling, via the transcriptional upregulation of TXNIP in cholangiocytes, acts as a critical brake on ductular reaction and its associated inflammatory and fibrotic sequelae. Our findings position the vitamin D/TXNIP axis as a fundamental regulator of biliary epithelial cell response and a promising target for molecular interventions in liver fibrosis.

Keyword(s) : Vitamin D, Ductular reaction, Liver fibrosis, Cholangiocytes



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1538 - Development and Validation of a RF LASSO Cox Derived Nomogram Incorporating Inflammatory Markers to Predict Long Term Survival in Early Stage Hepatocellular Carcinoma after Microwave Ablation

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Background / Hypothesis: This study aimed to identify factors influencing overall survival (OS) in patients with hepatocellular carcinoma (HCC) undergoing microwave ablation (MWA) and to develop and validate novel nomograms for predicting 3-, 5-, and 8-year OS.

Materials and Methods: Data from 440 patients who received MWA at Beijing Ditan Hospital were analyzed using LASSO regression, random forest (RF), and multivariable Cox regression to identify independent prognostic factors. A prognostic nomogram was constructed and validated. OS was assessed using Kaplan–Meier curves.

Results: The variables selected by both LASSO and RSF were incorporated into a multivariable Cox regression model, which identified tumor number, tumor size, monocyte-to-lymphocyte ratio (MLR), and diabetes as independent risk factors for OS. The predictive accuracy, reliability, and clinical utility of the model were further confirmed through time-dependent AUC analysis, calibration curves, and decision curve analysis (DCA). Furthermore, risk stratification based on the model effectively differentiated patients into distinct prognostic subgroups.

Conclusions: A nomogram based on LASSO–RF–Cox analysis was developed to predict OS in early-stage HCC patients after MWA. This model may help identify high-risk individuals and facilitate clinical decision-making, particularly in primary care settings due to its simple and readily available predictors.

Keyword(s) : Hepatocellular carcinoma; Microwave ablation; Nomogram; Overall survival

1539 - The FABP4 CD36 FXR Axis Regulates Bile Acid Homeostasis in Drug Induced Cholestasis

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Background / Hypothesis:

Bile acid (BA) dysregulation is the pathological cornerstone of drug-induced cholestasis (DIC), driven primarily by impaired farnesoid X receptor (FXR) signaling—the master regulator of BA synthesis (via CYP7A1, CYP8B1) and transport (via BSEP, MRP2). While FABP4 is established as an adipokine mediating metabolic inflammation, its direct role in disrupting hepatic BA homeostasis remains unexplored. Critically, clinical data link elevated FABP4 to cholestatic severity, yet whether it modulates FXR to exacerbate BA accumulation is unknown. This study aims to elucidate the molecular mechanism by which FABP4 regulates BA homeostasis in drug-induced cholestasis.

Materials and Methods:

Fabp4 gene knockout (Fabp4^{-/-}) mice and the FABP4 inhibitor BMS309403 were applied to an alpha-naphthylisothiocyanate (ANIT)-induced cholestasis model. Hepatic BA profiles were determined by UPLC-MS/MS. To uncover the underlying mechanisms, FABP4-interacting proteins were initially screened by co-immunoprecipitation combined with mass spectrometry (Co-IP/MS), and the resulting data were further analyzed using the STRING database to identify high-confidence interaction partners. Among these, CD36 emerged as a top candidate, and their direct association was corroborated by immunofluorescence co-localization assay. The functional contribution of the FABP4/CD36/FXR axis was ultimately validated in AML-12 cells, HuH7 cells, and primary mouse hepatocytes.

Results:

Genetic ablation of Fabp4 (Fabp4^{-/-}) or its pharmacological inhibition with BMS309403 significantly attenuated ANIT-induced intrahepatic bile acid accumulation and concurrently ameliorated compositional BA profile disturbances, as shown by an altered CA/CDCA ratio (Figure A-C). To explore the underlying mechanism, we first identified FABP4-interacting partners through co-immunoprecipitation

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combined with mass spectrometry (Co-IP/MS), and subsequent STRING database analysis prioritized the fatty acid transporter CD36 as a high-confidence interactor. Their physical association was further visually confirmed by immunofluorescence co-localization in hepatocytes, which appeared to be dependent on FABP4's lipid-binding capacity (Figure D-H). Functionally, FABP4 overexpression inhibited FXR, thereby attenuating its repressive effect on the target genes CYP7A1 and CYP8B1 and consequently promoting BA synthesis. Notably, this effect was substantially rescued by CD36 knockdown (I-L), collectively demonstrating that the FABP4-CD36 interaction serves to inhibit FXR activity and thereby regulate bile acid metabolism.

Conclusions:

This study identifies FABP4 as a key driver of BA dysregulation in DIC, disrupting BAs homeostasis through CD36 interaction to inhibit FXR signaling. Genetic (Fabp4^{-/-}) or pharmacological (BMS309403) inhibition of FABP4 restores FXR signaling, thereby modulating the expression of FXR target genes CYP7A1 and CYP8B1 to regulate bile acid metabolism. The FABP4/CD36/FXR axis represents a conserved therapeutic target, with FABP4 inhibition offering a promising strategy to reactivate FXR and mitigate cholestatic injury.

Keyword(s) : Drug-induced cholestasis, FABP4, FXR, Bile acid dysregulation

1540 - Pruritus and Its Impact on Quality of Life in Primary Biliary Cholangitis A Study of Patients on Long Term Ursodeoxycholic Acid Treatment

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Background:

Primary biliary cholangitis (PBC) is an autoimmune liver disease, and pruritus is one of the most common symptoms. The severity of pruritus significantly impacts patients' quality of life. However, existing studies indicated that the incidence and severity of pruritus in treated PBC patients were under-evaluated, and its relationship with health-related quality of life (HRQOL) remained unclear.

Objective:

To observe the prevalence and influencing factors of pruritus in PBC patients receiving first-line treatment, and to explore the interrelationship between pruritus and perceived quality of life.

Methods:

This study included patients diagnosed with primary biliary cholangitis who visited our hospital between 2022 and 2024. Patients receiving ursodeoxycholic acid (UDCA) as first-line therapy for over 12 months underwent questionnaire follow-up. Included patients completed the Itch Numerical Rating Scale (NRS), EQ-5D-3L, Hospital Anxiety and Depression Scale, and Fatigue Scale Version 4. Statistical analysis assessed the prevalence and severity of pruritus in PBC patients. Multiple linear regression analyzed correlations between pruritus and quality of life/psychological health, while multivariate regression models examined influencing factors for pruritus occurrence.

Results:

This study included 415 PBC patients who had used UDCA for over 12 months. The prevalence of pruritus symptoms in the study population was 39.5% (n=164), with moderate-to-severe pruritus accounting for 11.1% (NRS>4, n=46). Participants were divided into an pruritus group (n=164) and a non-pruritus group (n=251) based on the presence of pruritus (NRS≥1). The prevalence of cirrhosis was significantly higher in the pruritus group than in the non-pruritus group (56.7% vs 37.5%, P<0.001). Compared with the non-pruritus group, the pruritus group showed significantly increased anxiety levels (5[IQR: 5]vs0[IQR: 5], P < 0.001), depression scores were higher (7(IQR:6)vs0 (IQR:4), P<0.001), and fatigue scores were significantly lower (42 (IQR:11)vs48 (IQR: 6), P<0.001). Furthermore, itch severity showed a linear correlation with patients' EQ-5D index scores, with worse scores corresponding to more severe itching (R²=0.10, P<0.001). Multivariate regression analysis identified disease duration (OR=1.05; P<0.05), UDCA response (Paris I criteria OR=0.54; P<0.05), portal hypertension (OR=1.98; P<0.05), and dry eye/mouth symptoms (OR=4.17; P<0.001) were independent factors associated with pruritus occurrence.

Conclusion:

This study demonstrated a high prevalence of pruritus (39.5%) among PBC patients undergoing first-line therapy. Disease duration, portal hypertension, and dry eye/mouth symptoms were independent factors

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associated with pruritus, while favorable UDCA response served as a protective factor. Furthermore, pruritus significantly impacted patients' quality of life; more severe pruritus was associated with greater anxiety, depression and fatigue, highlighting its significant disease burden.

Keyword(s) : PBC Pruritus HRQOL

1541 - Development and Validation of a Nomogram for Predicting Postoperative Survival in Patients with Multifocal Hepatocellular Carcinoma

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is one of the malignant tumors with high incidence and mortality worldwide. It ranks fifth among all cancers and is the third leading cause of cancer-related deaths. Multifocal hepatocellular carcinoma (M-HCC), as a subtype of HCC, exhibits significant heterogeneity in its clinical manifestations and prognosis. Although M-HCC is often regarded as a simple prognostic factor in existing studies, research on its specific prognostic impact is relatively scarce, lacking systematic and in-depth analysis. Therefore, accurate assessment of the prognosis of M-HCC patients, especially in predicting postoperative survival rates, remains of great clinical significance. The objective of this study is to construct a more precise prognostic prediction model for M-HCC patients by integrating multi-dimensional information including clinical, imaging, and laboratory examinations. This aims to provide more targeted guidance for individualized treatment and ultimately improve the overall survival rate of patients.

Methods : We applied Lasso regression, cross-validated Lasso (CV-Lasso), and random forest analysis to identify key prognostic factors. A nomogram was developed by weighting significant factors to predict postoperative survival. Patients were stratified into low/high-risk groups, and survival analysis was performed using Kaplan-Meier curves.

Results: A total of 578 M-HCC patients were enrolled in the study and randomly divided into a training set and a validation set. Through Lasso, CV-Lasso, and random forest analyses, we identified seven factors that significantly affect the prognosis of M-HCC patients.

age, antiviral therapy, Child-Pugh score, liver cirrhosis, white blood cell count (WBC), platelet count (PLT), and thrombin time (TT). Subsequently, based on these seven factors, we constructed a nomogram model to predict the 3-year, 5-year, and 8-year survival rates of patients. The predictive ability of the model was verified by ROC curves, with an area under the curve (AUC) of 0.688 for 5-year survival prediction and 0.784 for 8-year survival prediction, indicating high predictive accuracy. Kaplan-Meier survival curves further showed that the survival time of patients in the low-risk group was significantly longer than that in the high-risk group ($p < 0.0001$), demonstrating the discriminative ability of the model.

Conclusions: Postoperative antiviral therapy significantly improves M-HCC survival by enhancing immunity and protecting liver function. Child-Pugh score, age, and cirrhosis are associated with poor prognosis. Integrating multi-dimensional data to construct a personalized prognostic model enables precise treatment strategies, improving long-term outcomes and overall survival.

Keyword(s) : Multifocal Hepatocellular Carcinoma; Nomogram; Overall survival

1543 - Endothelial RUNX1 Controls Acetaminophen Induced Liver Injury by Regulating LSEC Dysfunction and Pathological Angiocrine Signaling

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Background / Hypothesis:

Acetaminophen (APAP)-induced liver injury (AILI) is a common cause of drug-induced liver injury (DILI). Liver sinusoidal endothelial cells (LSECs) act as essential gatekeepers of hepatic homeostasis and are central regulators of disease pathogenesis. Although Runx-related transcription factor-1 (RUNX1) is a master modulator of hematopoiesis and inflammatory responses, its specific function in LSECs during AILI remains unclear. This study aimed to investigate the role of RUNX1 in LSEC function and to delineate the underlying mechanisms in AILI.

Materials and Methods:

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Primary LSECs were isolated from AILI mice by density gradient centrifugation and adherence-based purification, and then subjected to gene expression analysis by RNA sequencing, qPCR, and western blotting. The role and expression of RUNX1 were further validated in vitro. Endothelial-specific Runx1-deficient mice were established via AAV2/9 delivery, and AILI was induced by APAP administration. Liver injury was evaluated through histology, biochemical assays (ELISA), and immunostaining. To explore the mechanism, we performed RNA sequencing on LSECs from APAP-injured mice to identify key signaling and angiocrine pathways. The correlation between RUNX1 and DILI was assessed by interrogating publicly available scRNA-seq datasets. Finally, the therapeutic effect of the RUNX1 inhibitor (Ro24-7429) was evaluated in vivo in the AILI mouse model.

Results:

We successfully isolated and characterized high-purity primary LSECs. RNA sequencing revealed a significant upregulation of RUNX1 in LSECs from AILI mice, with peak expression at the early injury phase (6 hours post-APAP). Consistent with these findings, RUNX1 expression was increased both in vivo and in vitro under APAP stimulation. Co-immunostaining with CD31 confirmed that RUNX1 expression was predominantly localized to LSECs. Notably, endothelial Runx1-deficient mice exhibited attenuated LSEC dysfunction, along with ameliorated liver injury and inflammation. Mechanistic studies in human immortalized and mouse primary LSECs indicated that RUNX1 deficiency activated pathways regulating LSEC dysfunction. Furthermore, RNA sequencing analysis revealed that RUNX1 knockdown mitigated the production of pro-inflammatory factors via a paracrine signaling mechanism, thereby protecting against hepatocyte injury. Importantly, pharmacological inhibition of RUNX1 effectively alleviated APAP-induced liver injury.

Conclusions:

Endothelial Runx1 deficiency and its pharmacological inhibition attenuate APAP-induced LSEC dysfunction and liver injury by reducing hepatocyte damage via a paracrine mechanism. Therefore, therapeutic targeting of RUNX1 represents a potential strategy for treating DILI.

Keyword(s) : Drug-induced liver injury; Sinusoidal

endothelia; RUNX1; Paracrine signaling; Acute liver injury.

1544 - Alterations in gut microbiota and metabolism are correlated with the severity of PBC.

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Background / Hypothesis:

The study aims to explore the differences in metabolic profiles and gut microbiota between GP210 antibody-positive (GP210_pos) and GP210 antibody-negative (GP210_neg) patients with primary biliary cholangitis (PBC).

Materials and Methods:

This study employed untargeted metabolomics to analyze serum metabolite differences between 31 patients negative for GP210 antibodies and 13 patients positive for GP210 antibodies in PBC. Additionally, 16S rRNA sequencing was used to compare the gut microbiota composition between the two patient groups. We further explored the correlations between differential metabolites and differential bacterial genera using Sankey diagrams and correlation network graphs. Finally, ROC curve analysis was performed for each metabolite to assess its effectiveness in identifying and predicting GP210 antibody-positive PBC.

Results:

In GP210_pos patients, 11 differential bacterial genera were identified, with the relative abundances of 4 genera significantly increased and 7 genera significantly decreased, along with 9 differential metabolites. Among these, the increased genera *Gemmiger*, *Faecalibacterium*, *Haemophilus*, and *Megasphaera* showed significant negative correlations with 1-Peroxyferolide, Steroid O-sulfate, and significant positive correlations with 10,16-Dihydroxyhexadecanoic acid. Conversely, the decreased genera *Streptococcus* and *Salmonella* exhibited significant negative correlations with Synaptotaxis factor K1, Cotylenin F, and Cassaidine. Additionally, ROC curve analysis revealed that Chaksine had the highest AUC value of 0.74, indicating strong

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predictive capability.

Conclusions:

This study identified significant differences in both metabolite profiles and gut microbiota composition in GP210_pos PBC patients. Univariate ROC analysis results specifically highlighted Chaksine as having strong predictive capability (AUC = 0.74) in its predictive model.

Keyword(s) : Primary biliary cholangitis; GP210 antibody ; gut microbiota

1545 - Significant Burden of Liver Related Outcomes in DM MASLD despite Low Fatty Liver Index Results from Two Nationwide Cohort Studies

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Background / Hypothesis: Lean metabolic dysfunction-associated steatotic liver disease (MASLD), defined by hepatic steatosis with cardiometabolic risk factors in non-obese individuals, is relatively common in Asian populations, but may be overlooked when risk assessment relies solely on noninvasive steatosis markers such as the fatty liver index (FLI). We aimed to evaluate the burden of liver-related outcomes (LROs) in MASLD patients classified as having low steatosis likelihood by FLI.

Materials and Methods: A retrospective cohort study was conducted using the Korean National Health Insurance Service (NHIS) database. Patients with diabetes mellitus who underwent health screening examinations between 2009 and 2013 were included. MASLD was stratified according to FLI into control (FLI < 20), suboptimal (FLI 20–30), and definite (FLI > 30) groups, and LROs were evaluated using Fine–Gray competing risk models. In addition, we conducted parallel analyses in participants from the UK Biobank.

Results: Among 986,436 individuals in the NHIS database, 149,682 (15.2%) were classified as having suboptimal MASLD by FLI. The suboptimal MASLD group had a significantly increased risk of composite LRO (subdistribution hazard ratio [sHR] = 1.10, 95%

confidence interval [CI] = 1.06–1.15, $p < 0.001$) compared to the control group, specifically for hepatocellular carcinoma (sHR = 1.17, 95% CI = 1.09–1.26, $p < 0.001$), and liver-related death (sHR = 1.22, 95% CI = 1.12–1.34, $p < 0.001$). The elevated risk persisted in a cause-specific model and across subgroups. In the suboptimal MASLD group, higher AST and ALT were independently associated with increased risks of LRO (AST: sHR = 1.25, 95% CI = 1.22–1.27; ALT: sHR = 1.15, 95% CI = 1.13–1.17; both $p < 0.001$). However, among 391,220 individuals in the UKB database, the suboptimal MASLD group had a comparable risk of LROs to the control group (sHR = 1.06, 95% CI = 0.61–1.81, $p = 0.844$).

Conclusions: MASLD patients with borderline FLI still experienced a significant burden of LROs in the Korean NHIS cohort but not in the UK Biobank cohort. These findings highlight the limitations of steatosis markers for risk stratification and emphasize the need for more comprehensive assessment strategies, especially in populations with lean MASLD.

Keyword(s) : fatty liver; lean NAFLD; liver cancer; NHIS; UK Biobank.

1546 - Tranexamic acid on risks of rebleeding in acute variceal bleeding a retrospective cohort study on local data in Hong Kong

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Background / Hypothesis:

Variceal haemorrhage is considered one of the most severe complications of liver cirrhosis and portal hypertension. It is postulated that the severity of bleeding is aggravated by hyperfibrinolysis. Recent randomized controlled trial by Kumar et al. demonstrated that tranexamic acid reduces the risk of failure to prevent rebleeding in upper gastro-intestinal bleeding in patients with Child-Turcotte-Pugh class B or C. We analysed local data in Hong Kong on the impact of tranexamic acid on rebleeding rates and overall survival in variceal bleeding.

Materials and Methods:

We performed a retrospective cohort study on adult patients with history of acute gastrointestinal variceal bleeding between January 2000 and May 2020

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identified using a territory-wide database in Hong Kong. Patients with hepatocellular carcinoma, previous thrombotic events and dialysis-dependent end stage renal failure were excluded. The primary and secondary endpoints were 6-week rebleeding events with reference to Baveno V criteria and 6-week all-cause mortality respectively. Propensity score matching was applied to the study cohort before endpoint comparison.

Results:

A total of 5225 patients with 6331 variceal bleeding events were analysed. 374 events in patient receiving tranexamic acid within 1 day of presentation were compared to 374 events in propensity score matched control group. Clinical characteristics were comparable between groups. No significant inter-group difference was observed in rates rebleeding events by 6 weeks (20.1% in treatment group vs 17.1% in control, $p = 0.35$) and 6-week all-cause mortality (20.6% in treatment group vs 21.9% in control, $p = 0.64$).

Conclusions:

In conclusion, tranexamic acid was not shown to significantly improve both rebleeding risks and overall survival by 6 weeks in this local retrospective cohort. Further studies are required to examine the role and safety of tranexamic acid in patients with variceal bleeding.

Keyword(s) :

1547 - Micro RNAs (-122 -34a and -99a) as Potential Predictive Biomarkers for MAFLD

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Background / Hypothesis:The development of non-invasive diagnostic tools to diagnose non alcoholic fatty liver remains essential for patient safety rather than invasive techniques. Mi-RNAs are present in almost all body fluids such as serum, plasma and urine. The increased use of circulating mi-RNAs in clinical practice is explained by their ability to accurately reflect the physiological/pathological state of the tissue they are derived from. This case control study was to address the value of circulating miRNAs as potential non invasive biomarker for the diagnosis of MAFLD

and monitoring of disease progression.

Materials and Methods:Routine clinical assessment, laboratory tests, anthropometric study and liver biopsy results reported for 210 patients with MAFLD (124 patients of simple steatosis (SS) and 86 of MASH). Apparently matched age and sex healthy individuals ($n=90$) were included as control group. Exclusion criteria were; presence of hepatitis viral markers (for B and C), autoimmune hepatitis, alcohol consumption, hepatocellular carcinoma, liver cirrhosis or portal hypertension, diabetes mellitus or ischemic heart disease. Serum sample were tested for mirco-RNAs (-122, -34a and -99a) by quantitative-PCR.

Results:By histopathology, 124 of MAFLD group were of SS and 86 patients were of MASH. Both mi-RNA-122 and -34a level were increased in MAFLD compared to healthy controls ($p<0.001$) and at a cut-off=1.261, mi-RNA-122 had 92%-sensitivity, 85%-specificity to differentiate between MAFLD from healthy controls. While mi-RNA-99a were significantly decreased in MAFLD patients with further decreased in disease severity, and at a cut-off=0.46, the miR-99a had 94%-sensitivity and 96%-specificity to discriminate between SS from MASH.

Conclusions:The integration of circulating mi-RNA panel achieved to diagnose MAFLD cases and to discriminate between SS and MASH. Large scale study is still needed to verify the other miRNAs profile and their role in MAFLD pathogenesis and targeting therapy.

Keyword(s) : MAFLD, MicroRNA, liver biopsy, MASH

1548 - The Potential Role of A Gut Hormone in MAFLD

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Background / Hypothesis:Ghrelin is a gut hormone

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with various functions including energy metabolism and inflammation inhibition. Nuclear factor-kappa B (NF- κ B) participates in the initiation and the progression of inflammation, particularly, the cardiovascular and adipose tissue inflammation. To date the combined role of Ghrelin and NF- κ B in the pathogenesis of metabolic non-alcoholic fatty liver disease (MAFLD) is a matter of debate. We investigated whether acyl ghrelin level and NF- κ B could interplay a role in lipid metabolism and inflammatory injury in MAFLD.

Materials and Methods: Ninety three adult participants were included in the study, 28 patients had proved nonalcoholic steatohepatitis (NASH), and 40 patients had simple steatosis, as well 25 healthy subjects, matched for age, gender and Body mass index (BMI) to the patients were included in the study as a healthy control group. Full history and clinical examination, abdominal ultrasonography and liver biopsy were done when indicated. Liver function tests, lipid profile, blood sugar, insulin and C-peptide, fasting insulin, and plasma acyl ghrelin concentrations were measured. Nuclear NF- κ B microRNA expression was measured by quantitative real time polymerase chain reaction.

Results: Fasting insulin, insulin C-peptide, homeostatic model assessment insulin resistance (HOMA-IR), alanine amino-transferases (ALT) and gamma glutamyl transpeptidase (GGT) were significantly increased and high density lipoprotein cholesterol (HDL-C) was significantly decreased in MAFLD group compared to control group. In addition, a significant increase in ALT, GGT, fasting insulin, insulin C peptide and HOMA-IR were detected in the NASH group compared to group of simple steatosis. The plasma levels of acyl-ghrelin was significantly decreased in MAFLD groups compared to normal control group, the lowest level was detected in NASH group as compared to group of simple steatosis. The expression of NF- κ B microRNA was significantly increased in MAFLD groups compared to normal control group and its level was significantly increased in NASH compared to simple steatosis. The NF- κ B mRNA was positively correlated with BMI, HOMA-IR, ALT, fasting insulin, insulin C-peptide and liver histopathology and acyl-ghrelin was inversely correlated with BMI, HOMA-IR, ALT, fasting insulin,

insulin C peptide and liver histopathology. Both were significantly correlated with HDL-C.

Conclusions: Acyl ghrelin attenuated MAFLD-induced liver injury through down regulation of NF- κ B and they are associated with disease progression. Further large scale studies are recommended to consider ghrelin as promising drug for the prevention and treatment of MAFLD.

Keyword(s) : MAFLD; Acyl ghrelin, NF- κ B, Liver Biopsy

1549 - Retrospective Audit of Terlipressin Use in Hepatorenal Syndrome – Acute Kidney Injury (HRS AKI)

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Background / Hypothesis:

Terlipressin is a vasopressin analogue which induces splanchnic vasoconstriction, thereby reducing portal inflow and redistributing circulatory volume from the splanchnic to the systemic circulation, which ultimately improves renal perfusion. It is widely used for treating hepatorenal syndrome – acute kidney injury (HRS-AKI). However, emerging evidence suggests an increased risk of respiratory adverse events (AEs) associated with terlipressin treatment. This study aimed to evaluate the efficacy and safety of terlipressin in a real-world cohort of Asian patients with HRS-AKI.

Materials and Methods:

A retrospective cohort study comprising 51 patients aged ≥ 21 years with chronic liver disease who received terlipressin for HRS-AKI between January 1 2005 and June 30 2023 was conducted. Primary outcomes included treatment response (based on the 2015 criteria), incidence of AEs, and mortality. Multivariate

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logistic regression was employed to assess the associations between relevant covariates and outcomes.

Results:

Among the 51 patients, 15 (29.4%) achieved a complete response to treatment, 5 (9.8%) had a partial response, and 25 (49%) had no response. Reasons for non-response were initiation of renal replacement therapy (RRT) in 9 patients and absence of stage regression in 15 patients. 18 patients (35.3%) experienced AEs of any severity, with 12 (23.5%) having respiratory-related AEs. 41 patients (80.4%) died, while 3 patients (5.9%) underwent liver transplantation. Modality of terlipressin administration (bolus versus continuous) and number of days of albumin exposure prior to treatment were significantly associated with development of any AE. However, no covariates were found to be significantly associated with mortality.

Conclusions:

Terlipressin remains an effective treatment for HRS-AKI; however, its use is associated with a distinct AE profile, particularly respiratory complications. It should therefore be used selectively with careful monitoring, particularly in patients receiving bolus dosing or large volumes of albumin pre-treatment. These factors should be considered when evaluating the benefit-risk profile of terlipressin in clinical decision-making.

Keyword(s) : Cirrhosis, renal impairment, hepatorenal syndrome

1550 - VCTE Radiomics for Non Invasive Assessment of Hepatic Fibrosis in Metabolic Dysfunction Associated Steatohepatitis

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Background / Hypothesis:

Fibrotic metabolic dysfunction-associated steatohepatitis (MASH) is the leading cause of all-cause and liver-related mortality. Radiomics could predict disease progression. We developed a diagnostic model for identifying fibrotic MASH using radiomics analysis based on hepatic vibration-controlled transient elastography (VCTE).

Materials and Methods:

A total of 163 patients with biopsy-proven metabolic dysfunction-associated steatotic liver disease (MASLD)

were enrolled and divided into training (50%) and validation (50%) cohorts. The outcome was defined as Fibrotic MASH (NAS ≥ 4 and fibrosis stage ≥ 2). VCTE images were obtained and extracted 1209 radiomics feature, where 7 features were selected using Least absolute shrinkage and selection operator regression. R Score was established using support vector machines based on selected features. The diagnostic model was constructed using multiple logistic regression based on R Score and clinical data, which performance was compared with previous models (Agile 3+, Agile 4, FAST, FIB-4, FNI, NFS).

Results:

Fibrotic MASH patients presented elder ages, higher AST, and higher liver stiffness measurement (LSM) than Non-Fibrotic MASH patients ($P < 0.05$). The Radiomics Score (R Score), constructed via SVM using selected features, demonstrated significantly different distributions between Fibrotic MASH and Non-Fibrotic MASH patients in both training and validation cohorts (all $P < 0.01$). R Score [odds ratio (OR)=13171.60, 95%CI=93.08-1863829.74, $P < 0.01$], LSM (OR=1.19, 95%CI=1.05-1.36, $P=0.01$), age (OR=1.06, 95%CI=1.02-1.11, $P=0.01$), and AST (OR=1.02, 95%CI=1.00-1.04, $P=0.04$) were independent risk factors for Fibrotic MASH. These factors (AST, R Score, LSM, Age) were incorporated to construct diagnostic model for Fibrotic MASH, which was named ARLA model (Figure 1A). The ARLA yielded the best diagnostic performance for Fibrotic MASH in both the training (AUROC=0.86, 95%CI=0.78-0.94, Figure 1B) and validation (AUROC=0.87, 95%CI=0.78-0.95, Figure 1C) cohorts. The dual cut-off approach was implemented with a sensitivity ≥ 0.85 for ruling out and a specificity ≥ 0.85 for ruling in the disease. Therefore, the cut-off values of ARLA points were 59.3095 and 68.1445 for rule-out and rule-in, respectively, with characteristics detailed in Figure 1D-E. Accordingly, ARLA exhibited highest correct classified proportion of 69% and 70% in both cohorts (Figure 2A-B). The two-step approach based on FIB4 and ARLA exhibited smaller grey zone and higher correct classified proportion than traditional two-step approach based on FIB-4 and LSM (Figure 2C-D).

Conclusions:

The ARLA model based on VCTE radiomics and clinical characteristic yielded the best diagnostic performance for Fibrotic MASH than preexisting scores. It could be a noninvasive and cost-effectiveness tool for early identification of progressive MASH patients for clinical intervention.

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Keyword(s) : Fibrotic metabolic dysfunction-associated steatohepatitis, Metabolic dysfunction-associated steatotic liver disease, Radiomics, Vibration-controlled transient elastography, Machine learning, Diagnosis

1553 - Hepatic G3BP1 Acts as a Critical Role Integrating Autophagy and Lipogenesis to Suppress MASLD Progression

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Background / Hypothesis:

The molecular mechanisms governing the progression of metabolic dysfunction-associated steatotic liver disease (MASLD) to steatohepatitis (MASH) remain incompletely defined. A comprehensive understanding of the regulatory proteins involved in hepatic lipid metabolism and stress response is critical for elucidating disease pathogenesis.

Materials and Methods:

We initially identified a significant reduction in GTPase-activating protein-binding protein 1 (G3BP1) expression in liver specimens from human MASLD/MASH patients. To define the cell-autonomous functions of G3BP1 in hepatocytes, we generated and characterized hepatocyte-specific G3BP1 knockout (G3BP1-HKO) mice under metabolic challenge. An integrated approach, utilizing histopathology, immunoblotting, co-immunoprecipitation, and luciferase reporter assays, was employed to dissect the phenotypic and molecular consequences of G3BP1 loss.

Results:

G3BP1 deficiency in hepatocytes markedly accelerated the development and severity of hepatic steatosis and inflammatory injury in mice. At the mechanistic level, we identified G3BP1 as a novel and essential regulator of autophagic flux. G3BP1 directly interacted with the SNARE proteins STX17 and VAMP8, and this interaction was necessary for efficient autophagosome-lysosome fusion. Independently, G3BP1 ablation enhanced hepatic de novo lipogenesis. We determined that this

pro-lipogenic effect was consequent to impaired nuclear translocation of the transcription factor TFE3, a known repressor of lipogenic genes. G3BP1 was required for the nuclear accumulation of TFE3, thereby establishing a direct molecular link between G3BP1 and the transcriptional control of lipid synthesis.

Conclusions:

This study unveils a dual-protective mechanism orchestrated by G3BP1 in the liver. G3BP1 safeguards against MASLD/MASH progression by concurrently promoting the completion of autophagy via SNARE complex facilitation and suppressing lipogenesis through enabling TFE3-mediated transcriptional repression. These findings position G3BP1 as a central node integrating proteostatic and metabolic pathways, offering profound insights into the fundamental biology of liver steatosis and highlighting a potential target for therapeutic intervention.

Keyword(s) : Autophagy, Lipogenesis, MASLD, G3BP1

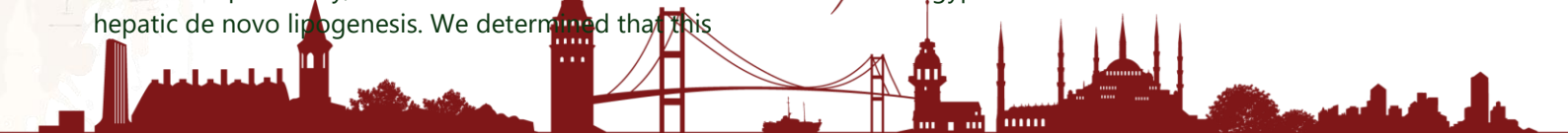
1554 - Micro RNA Gene Polymorphisms in Egyptian Cirrhotics who developed Hepatocellular Carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is the most common type of primary liver cancer worldwide. It is also the fifth cause of death due to cancer in the world. This is mainly due to the fact that HCC is usually being diagnosed at an advanced stage due to a difficult early diagnosis. To save life, new diagnostic and prognostic biomarkers for HCC are of paramount significance. The development and progression of hepatocellular carcinoma (HCC) is a multistage process involving the deregulation of genes that are crucial to cellular processes. Multiple risk factors are correlated with HCC. We determined the association between micro-RNA gene polymorphisms and development of HCC in Egyptian Patients.



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Materials and Methods:The current case-control study included 200 participants, they were recruited from the Outpatient Clinics and Inpatient Department, National Liver Institute-Menoufia University, Egypt, 90 of them were proved as HCC by clinical examination, laboratory and triphasic CT findings. As well as 110 of participants randomly selected from healthy subjects with matched age, sex, risk factors that may affect HCC development as (Diabetes Mellitus, Hypertension and/or smoking) were enrolled in the study as a control group. Cases with chronic hepatitis without HCC, autoimmune hepatitis and chronic inflammatory diseases are excluded by clinical and laboratory investigations. Tumor staging was done using BCLC staging system. Quantification and genotyping of Micro-RNA were performed.

Results:

Among the 200 patients, 2 groups were described: group I included 90 HCC patients with a male majority (72.2%) and 110 controls in group II. Three microRNA SNPs were assayed in both patients and controls. There was a significant association between rs10061133 miR-499b and the risk of HCC. The genotypes GG or G allele were associated significantly to an increased risk of HCC (GG: OR (95% CI)= 2.91 (1.23-4.22), $p = 0.013$; G allele: OR (95% CI)= 1.79 (1.12-2.15), $p = 0.026$) compared with the genotype of AA or AG or A allele

Conclusions:An association between the miRNA499 SNPs and the susceptibility to HCC, aiming to explore some roles and mechanisms of SNPs within miRNA in the occurrence and development of primary liver cancer. To the best of our knowledge, no previous studies elicited such finding. We hope that our efforts and findings will facilitate the use of miRNA SNPs in early detection of HCC and may be targeting for HCC therapy. Further prospective investigations with a large number of cases would allow us to evaluate miRNA-499 polymorphism in a variety of clinical settings to better understand its role in HCC.

Keyword(s) :

1556 - Serum kallistatin level in comparison to other noninvasive panels for potential portal hypertension Evaluation in Egyptian cirrhotics

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Background / Hypothesis:

The development of esophageal varices is an important consequence of portal hypertension. In accordance with the Baveno VI consensus, noninvasive tools are recommended to stratify patients and to identify those who are at risk of high-risk esophageal varices (HREV), for whom endoscopic assessment is warranted. We investigated the diagnostic value of serum kallistatin levels, along with other biochemical panels, in the assessment of portal hypertension among Egyptian individuals with liver cirrhosis.

Materials and Methods:

This cross-sectional research evaluated 90 cirrhotic cases who were admitted to the National Liver Institute's Hepatology and Gastroenterology Department as well as the Endoscopy Unit at Menoufia University by measuring the serum KAL level and comparing it to other panels in order to grade esophageal varices according to the results of GI endoscopy.

Results:

The mean age of the 90 individuals who were enrolled in the study was 59.8 ± 9.8 years; 42.2% of them were female, and 57.8% were male. Patients with varices had a considerably decreased mean level of blood kallistatin. Additionally, it demonstrates a notable decrease in individuals with extensive varices. The presence of EV can be predicted by kallistatin with a sensitivity of 76.5% and a specificity of 98.6% at cut-off levels of 23.1 $\mu\text{g/ml}$.

Conclusions: Serum kallistatin may serve as a potential indicator for identifying esophageal varices, particularly the large and more life-threatening types.

Keyword(s) : oesophageal vaices; Kallistatin; Cirrhosis, Non-invasive

1557 - AADN score Predicting Response to transarterial chemoembolization sintilimab and lenvatinib in patients with hepatocellular carcinoma

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Background / Hypothesis:

Although the triple therapy of transarterial chemoembolization (TACE) combined with immune checkpoint inhibitors and tyrosine kinase inhibitors is becoming an effective treatment for unresectable hepatocellular carcinoma (uHCC). However, there is still a lack of effective tools for predicting therapeutic effects at present.

Materials and Methods:

To develop a predictive tool for the prognosis of uHCC patients treated with TACE, sintilimab and lenvatinib. Based on multicenter data, this study constructed and validated an AADN score as variables to predict overall survival in patients treated with this combination therapy. This study included 188 uHCC cases (training cohort: n = 101, validation cohort: n = 87) from three different hospitals. who treated with TACE, sintilimab and lenvatinib.

Results:

In multivariate analysis, alpha-fetoprotein ≥ 100 ng/mL [hazard ratio (HR) = 2.579, P = 0.010], alkaline phosphatase > 120 U/L (HR 2.234, P = 0.021), direct bilirubin > 7.3 μ mol/L (HR = 2.931), P = 0.007) and neutrophil to lymphocyte ratio > 2.5 (HR = 3.127, P = 0.006) were identified as independent prognostic factors and were used to establish the AADN score. Kaplan-Meier survival curves and time-dependent receiver operating characteristic curves were used to assess the accuracy of the AADN score, with area under receiver operating characteristic curve values of 0.827 (training cohort, 95% confidence interval: 0.743-0.911) and 0.832 (validation cohort, 95% confidence interval: 0.742-0.923). According to the score, the patients were divided into low-risk, intermediate-risk and high-risk groups. Overall survival and progression-free survival were significantly different between groups.

Conclusions:

The AADN score can distinguish the prognostic risk of uHCC patients treated with TACE, sintilimab and lenvatinib, provides a basis for individualized treatment decision-making, and have clinical application prospect.

Keyword(s) : Hepatocellular carcinoma; Transarterial chemoembolization; Sintilimab; Lenvatinib; AADN score

1558 - Detection of Small Fiber Neuropathy Changes by Sudoscan in Hepatitis C Virus Infected Cirrhotics

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Background / Hypothesis: Worldwide, 130-170 million people are infected with hepatitis C virus (HCV), which represents more than 3 % of the world's population. HCV infection is associated with several extrahepatic manifestations, some of which are neurological and mostly related to immune mechanisms. Hepatitis C Virus (HCV) infection can affect the neurological system and neuropathy is one of these manifestations. HCV infection is associated with diabetes mellitus (DM) type II and diabetic patients are at higher risk of acquiring HCV infection. Sweat function has been proposed to assess early autonomic neuropathy. Aim: this study aimed to evaluate small fiber neuropathy in asymptomatic HCV-related cirrhotic patients with or without diabetes mellitus through sweat function assessment by SUDOSCAN test.

Materials and Methods:

three groups were involved: 47 healthy controls, 48 HCV-related cirrhotic patients without DM (Group 1) and 49 HCV-related cirrhotic patients with DM type II (Group 2). All participants were subjected to liver panel tests, renal function tests, cell blood counts, HbA1c, abdominal ultrasound. Sweat function was assessed in all patients and controls by measuring hand and feet Electrochemical Skin Conductance (ESC, μ S) using Sudoscan

Results:

Peripheral neuropathy was detected in none of the controls, 39% of Group 1 patients and in 62% of Group 2 patients (p < 0.0001). The mean feet ESC (FESC) was 88.3 ± 6.8 μ S in controls, 67.2 ± 19.2 μ S in Group 1 and 57.9 ± 19.4 μ S in Group 2 (p < 0.0001). A significant correlation was observed between FESC and bilirubin, albumin, creatinine, international normalized ratio,

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transaminases and splenic size.

Conclusions: the mean FESC are decreased in HCV-related cirrhosis with or without DM when compared to the control group, suggesting small fiber autonomic neuropathy based on sweat function assessment. SUDOSCAN seems a valuable noninvasive method for early detection of small fiber neuropathy in asymptomatic HCV-related cirrhosis with or without DM. Long-term follow-up would be useful to evaluate the progression of neurological dysfunction in this population; and future studies are needed to confirm our results in a larger population of hepatic patients with or without cirrhosis of any etiologies, along with a clinical neurological examination.

Keyword(s) : HCV; Small Fiber; Neuropathy; Sudoscan

1561 - Cell type specific bidirectional modulation of Zfp36l2 by a Chinese herbal formula reverses T cell exhaustion to suppress hepatocellular carcinoma

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Background / Hypothesis:

A major limitation in cancer immunotherapy is the establishment of terminally exhausted CD8⁺ T cells within the tumor microenvironment. We investigated the mechanism of Yangyin Fuzheng Jiedu Formula (YFJD), a traditional Chinese medicine formula, and hypothesized that it counteracts T cell exhaustion through a novel, cell-type-specific mechanism.

Materials and Methods:

The therapeutic efficacy of YFJD was evaluated in multiple immunocompetent murine HCC models, including MYC/CTNNB1/sg-p53 and c-MYC/Mcl1 (hydrodynamic injection), as well as subcutaneous models (sgPten/c-Met and c-MYC-driven HCC5). A critical dependency test was performed by comparing YFJD efficacy in immunocompetent C57BL/6J mice versus CD8⁺ T cell-deficient Balb/c nude mice bearing HCC5 subcutaneous tumors. Mechanistic studies employed high-dimensional flow cytometry, single-cell RNA sequencing (scRNA-seq), in vivo CD8⁺ T cell depletion, and in vitro co-culture systems with siRNA-mediated gene silencing.

Results:

YFJD robustly inhibited tumor growth across all models

and extended survival. This efficacy was strictly dependent on CD8⁺ T cells, as it was abrogated in Balb/c nude mice and upon CD8⁺ T cell depletion in immunocompetent hosts. YFJD remodeled the tumor immune microenvironment, specifically reducing the frequency of PD-1⁺TIGIT⁺TIM-3⁺ terminally exhausted CD8⁺ T cells and enhancing T cell polyfunctionality. scRNA-seq revealed that YFJD bidirectionally regulated the RNA-binding protein Zfp36l2—upregulating it in terminally exhausted CD8⁺ T cells while downregulating it in tumor cells. Functional studies confirmed that Zfp36l2 knockdown in tumor cells abolished YFJD-enhanced, CD8⁺ T cell-mediated tumor killing.

Conclusions:

Our work unveils a novel mechanism of action for YFJD: it orchestrates a cell-type-specific bidirectional regulation of Zfp36l2. By simultaneously targeting this node in both CD8⁺ T cells and tumor cells, YFJD effectively reverses T cell exhaustion and promotes anti-tumor immunity, presenting a sophisticated therapeutic paradigm for HCC.

Keyword(s) : Hepatocellular carcinoma, CD8⁺ T cell exhaustion, Tumor microenvironment, Zfp36l2, Bidirectional regulation, Genetic mouse models

1562 - Evaluation of HBV RNA as a predictive biomarker to determine intrahepatic HBV activity among chronic hepatitis B virus infected patients

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Background: Chronic hepatitis B virus (HBV) infection is a serious global public health concern that can lead to liver failure, cirrhosis, and subsequently hepatocellular carcinoma. HBV DNA has been used to assess the disease progression, but HBV DNA is rapidly undetectable after antiviral therapy while HBV RNA serves as an effective biomarker for monitoring the effectiveness of therapy and predicting disease prognosis. HBV RNA is a direct transcriptional product of hepatitis B virus (HBV) covalently closed circular DNA (cccDNA), playing a crucial role in amplifying and replicating the viral genome. This study was focused to determine the HBV RNA as an alternative marker for reflecting intrahepatic HBV activity and analyzing the correlation of plasma HBV RNA with HBV DNA and HBsAg.

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Materials and Methods: This descriptive cross-sectional study included 20 chronically infected, treatment-naïve patients and 20 patients treated with entecavir for over 48 weeks. Plasma and intrahepatic tissue were collected. HBV DNA and HBV RNA was extracted from the plasma samples and quantified by quantitative reverse transcription PCR (qRT-PCR). cccDNA were extracted by DNeasy Blood and Tissue kit (Qiagen, Netherlands) from the hepatic tissue subsequently was digested by Plasmid-Safe™ ATP-Dependent DNase (PSAD) (Epi-Centre Technologies Corp. Madison, WI, USA). The digested product was used to amplify cccDNA using specific primers by GoTaq qPCR master mix (Promega, Madison, USA). HBsAg levels were measured by chemiluminescence-based immunoassay (CLIA).

Results: HBV DNA and RNA were detected in all the treatment-naïve participants, while HBV RNA was detected in 25% of anti-viral treated patients, though having undetectable HBV DNA. The cccDNA was detected in 75% (15/20) and 70% (14/20) of treatment-naïve and treated patients respectively. HBV DNA and RNA levels were significantly higher in the treatment-naïve group compared to treated patients ($p < 0.001$). On the contrary, no significant difference was observed in HBsAg level between the groups ($p = 0.228$). HBeAg-positive patients showed significantly higher levels of HBsAg, HBV DNA, and RNA compared to HBeAg-negative patients. In addition, results demonstrated a strong positive correlation of HBV RNA with HBV DNA irrespective of anti-viral treatment, HBeAg seropositivity, and cccDNA detectability ($p < 0.001$). Nevertheless, with HBsAg level, no significant correlation was observed. Serum HBV RNA level can predict detected cccDNA with moderate accuracy (65%).

Conclusion: Serum HBV RNA is a promising biomarker for determining viral activity in both treatment-naïve and antiviral-treated chronic hepatitis B patients. Its assessment may improve therapy response monitoring and management, providing a viable alternative to invasive

Keyword(s) : Hepatitis B virus, HBV-DNA, HBV-RNA, cccDNA, quantitative HBsAg, Chronic Hepatitis B patients

1563 - Dynamics of Fibrosis Regression and Long term Liver related Events in Patients with Compensated Cirrhosis A 13 year Prospective

Cohort on Entecavir Therapy

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Background / Hypothesis: Chronic hepatitis B virus (HBV) infection is a leading cause of cirrhosis worldwide. Although nucleos(t)ide analogue (NA) therapy can slow the progression of HBV-related cirrhosis, the extent of achievable fibrosis regression and the long-term incidence of liver-related events (LREs) beyond ten years remain unclear. This study aimed to characterize 13-year dynamics of fibrosis reversal and LREs in a prospective cohort receiving NA therapy.

Materials and Methods: Fifty treatment-naïve patients with chronic hepatitis B and compensated cirrhosis were enrolled from 2012 to 2013 and received entecavir (ETV). Semi-annual assessments included laboratory tests, FibroScan, APRI, FIB-4, ALBI, Child-Pugh and MELD scores. LREs were defined as hepatic decompensation, hepatocellular carcinoma (HCC) or liver-related death. Cumulative incidences were estimated by Kaplan-Meier analysis, and predictors were identified via Cox regression models.

Results: After a median follow-up of 11.0 years (IQR 9.1–12.0), 12 patients developed LREs, including 9 cases of HCC. Kaplan-Meier analysis showed that cumulative LRE incidence rates of 0%, 11.0%, 22.6%, and 33.6% at 1, 5, 9, and 13 years, respectively. Corresponding HCC incidence rates of 0%, 8.8%, 13.5%, and 28.8%. Among 27 patients presented with significant liver fibrosis ($\text{FIB-4} \geq 3.25$) at baseline, cumulative fibrosis reversal rates were 25.9%, 46.5%, 75.2%, and 75.2% at 1, 4, 7, and 11 years, respectively. ALT was elevated in 24 patients at baseline, with a normalization rate of 91.7% (22/24) at year 1. Non-invasive indicators (LSM, APRI, Child-Pugh, ALBI), decreased significantly within the first year and remained stable throughout follow-up (1–11 years). Baseline characteristics did not predict clinical outcomes, however, multivariate analysis of on-treatment data at year 1 identified ALBI score as an independent predictor of subsequent LREs (HR 16.41, 95% CI 1.92–140.15, $P = 0.011$).

Conclusions: Long-term NA therapy leads to sustained

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fibrosis regression in over 75% of patients with compensated HBV-induced cirrhosis. Nonetheless, the risk of LREs persists, affecting nearly one-third of patients by year 13. The ALBI score at one year of treatment is a strong predictor of long-term outcomes, supporting its use for risk stratification and guiding enhanced surveillance in high-risk individuals.

Keyword(s) : Long-term outcomes, Prediction, Liver-related events, HBV-induced cirrhosis, Antiviral therapy

1566 - Enrichment of intratumoral *Cutibacterium acnes* in Advanced Hepatocellular Carcinoma and its promotive role in tumor progression

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Background / Hypothesis:

The significance of tumor-resident intracellular microbiota is increasingly recognized. However, studies on the molecular mechanisms by which intratumoral bacteria promote hepatocellular carcinoma (HCC) development remain scarce. This study aimed to characterize the microbiota signature of HCC tissues and explore its role in HCC progression.

Materials and Methods:

First, we confirmed the presence of microbiota using staining, fluorescence in situ hybridization (FISH), and transmission electron microscopy (TEM). Additionally, some bacteria were cultured from fresh HCC tissues. Subsequently, 16S rRNA sequencing revealed a significant difference in beta diversity between early-stage and advanced-stage HCC.

Results:

Moreover, the abundance of *Cutibacterium acnes* (*C. acnes*) was higher in advanced-stage HCC tissues than in both early-stage HCC tissues and adjacent normal tissues. We further demonstrated that *C. acnes* significantly promotes the proliferation and migration of HCC cells. Consistently, subcutaneous tumor models confirmed that *C. acnes* accelerates HCC development.

Conclusions: Our findings suggest that eliminating *C. acnes* or inhibiting its downstream signaling pathways

may serve as a novel therapeutic strategy for HCC.

Keyword(s) :

1569 - Impact of Thrombophilia on the Extent of Portal Vein Thrombosis at Diagnosis

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Background / Hypothesis:

Portal vein thrombosis (PVT) is the partial or complete obstruction of the portal vein and may extend to intrahepatic branches or the splanchnic venous axis, influencing risks such as portal hypertension and intestinal ischemia. Thrombophilic conditions—including natural anticoagulant deficiencies, antiphospholipid syndrome (APS), and myeloproliferative neoplasms (MPNs)—are known risk factors, but their impact on the extent of PVT is not well defined. Clarifying this relationship is essential to guide anticoagulation and surveillance. This study aimed to evaluate the relationship between major thrombophilic conditions and the anatomical extent of PVT at diagnosis in a Moroccan tertiary care cohort.

Materials and Methods:

We conducted a retrospective study including all patients with imaging-confirmed PVT (recent or chronic with cavernoma) managed at a university hospital between 2010 and 2025. Thrombosis was categorized as:

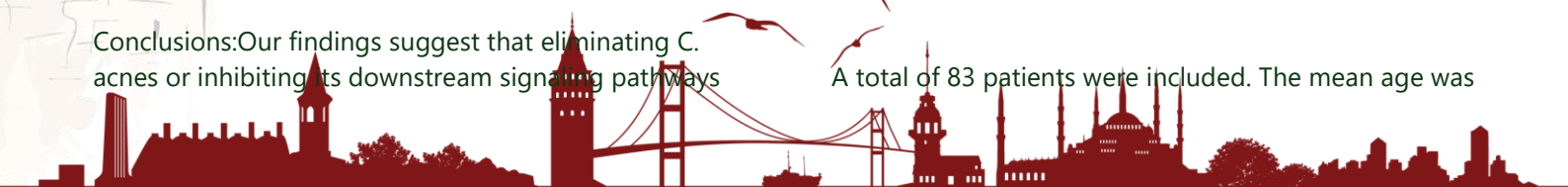
Localized: limited to the main portal trunk

Extended: involving intrahepatic portal branches, the spleno-mesenteric trunk, superior mesenteric vein, and/or splenic vein

Patient demographics, etiology, and extent of thrombosis were systematically collected. Etiologies were classified as natural anticoagulant deficiencies, APS, MPNs, idiopathic, and other causes (mainly inflammatory or infectious). The proportion of extended thrombosis was compared across etiological groups.

Results:

A total of 83 patients were included. The mean age was



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44.9 years (range 19–76), with a strong female predominance (77.1%; sex ratio M/F = 0.3). The most frequent underlying cause was natural anticoagulant deficiency (31.3%, n=26), followed by APS and MPNs (13.3% each, n=11), idiopathic forms (18.1%, n=15), and other causes (24.1%, n=20).

The prevalence of extended thrombosis varied markedly according to etiology:

APS: 72.7% (8/11)

MPNs: 63.6% (7/11)

Anticoagulant deficiencies: 46.2% (12/26)

Idiopathic forms: 20% (3/15)

These data highlight that patients with APS or MPNs are at substantially higher risk for extensive thrombotic involvement, whereas idiopathic cases tend to remain localized.

Conclusions:

In this cohort, thrombophilic conditions, particularly APS and MPNs, were strongly associated with extended PVT at diagnosis. These findings underline the need for systematic thrombophilia screening in all patients presenting with PVT to identify high-risk individuals. Early identification of patients predisposed to extensive thrombosis allows for timely initiation of appropriate anticoagulation, close imaging surveillance, and individualized management strategies. Recognition of the link between specific thrombophilic disorders and thrombus extension is critical for optimizing outcomes and preventing severe complications such as portal hypertension or intestinal ischemia. Future studies should evaluate prospective risk stratification algorithms and local adaptation of therapeutic strategies to improve patient prognosis in Moroccan and regional practice settings.

Keyword(s) : Portal vein thrombosis , anticoagulation , thrombophilia

1570 - Gadoteric Acid-Enhanced MRI Parameters for Functional and Volumetric Assessment in Chronic Liver Disease Stratification

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Background / Hypothesis:

Chronic liver disease (CLD) encompasses a progressive continuum from non-advanced to compensated and decompensated cirrhosis. Accurate staging is essential for prognostication, therapeutic decision-making, and transplant planning. Gadoteric acid-enhanced MRI offers quantitative functional biomarkers of hepatocellular uptake and biliary excretion, while splenic volumetry reflects portal hypertension severity. However, few studies have simultaneously assessed both functional MRI biomarkers and volumetric indicators across clinically defined CLD stages. We hypothesized that hepatocellular function-based indices—Hepatocellular Uptake Index (HUI), Liver-Spleen Index (LSI), Relative Liver Enhancement (RLE), and Contrast Uptake Index (CUI)—combined with spleen volume would demonstrate complementary, stage-dependent behavior, improving non-invasive stratification of CLD severity.

Materials and Methods:

This retrospective single-center study included 140 patients who underwent gadoteric acid-enhanced MRI between March 2020 and June 2025. Patients were categorized into three groups based on EASL 2021 criteria: non-advanced CLD (NA-CLD, n=18), compensated advanced CLD (CA-CLD, n=97), and decompensated advanced CLD (DA-CLD, n=25). Circular ROIs (2.0–5.0 cm²) were placed on segments VI and VIII of the liver, spleen, and paraspinal muscle on hepatobiliary phase (20-minute) and unenhanced images (Figure 1). RLE, CUI, HUI, and LSI were computed using established formulas. Liver and spleen volumes were calculated using validated ellipsoid models. Kruskal-Wallis, Spearman correlation, and Dunn-Bonferroni post-hoc analyses were applied.

Results:

HUI (p=0.006), LSI (p=0.013), RLE (p=0.033), and spleen volume (p<0.001) differed significantly across disease stages, while CUI (p=0.120) and liver volume (p=0.165) did not (Figure 2). HUI and LSI showed consistent decreases from NA-CLD to both CA-CLD and DA-CLD (p<0.05), indicating progressive hepatocellular impairment (Figure 4). RLE exhibited significant decline only between CA-CLD and DA-CLD (p=0.027). Spleen volume was significantly higher in both CA-CLD and DA-CLD compared to NA-CLD (p<0.001). Spearman analysis revealed significant negative correlations

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between disease stage and both HUI ($p=-0.250$, $p=0.003$) and LSI ($p=-0.228$, $p=0.007$), and a positive correlation for spleen volume ($p=0.240$, $p=0.005$) (Figure 5). Strong inter-parameter correlations were found between RLE and CUI ($p=0.821$), and between HUI and LSI ($p=0.756$) (Figure 3).

Conclusions:

HUI and LSI demonstrated the strongest stage-dependent declines and monotonic associations with disease severity, indicating high sensitivity to progressive hepatocellular dysfunction. Spleen volume provided complementary structural insight into portal hypertension. In contrast, liver volume alone was not discriminatory. These findings support a parallel interpretation model integrating functional GA-MRI indices with splenic volumetry (Figures 3–6) to enhance non-invasive staging of chronic liver disease and potentially inform future multiparametric MRI-based risk stratification frameworks.

Keyword(s) : Liver Diseases, Liver Cirrhosis, Magnetic Resonance Imaging, Gadoxetate Disodium, Severity of Illness Index

1571 - Impact of Hepatitis B Virus Infection on Surgical Conversion and Survival in Unresectable Hepatocellular Carcinoma Treated With TACE Targeted Therapy and PD 1 Inhibitors

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Background / Hypothesis: Hepatitis B virus (HBV) infection is a predominant etiological driver of hepatocellular carcinoma (HCC), yet its potential influence on the efficacy of conversion therapy in patients with unresectable HCC (uHCC) remains poorly defined. Triple therapy consisting of transarterial chemoembolization (TACE), targeted agents, and PD-1 inhibitors has demonstrated promising downstaging potential. However, whether the unique viral-immune microenvironment of HBV-related HCC confers differential therapeutic benefit has not been systematically investigated. We hypothesized that HBV infection may modulate tumor immunobiology and angiogenic signaling, thereby enhancing responsiveness to triple therapy.

Materials and Methods: This retrospective single-center cohort study included 190 consecutive uHCC patients treated with triple therapy between February 2022 and February 2025. Patients were stratified into HBV-positive ($n = 133$) and HBV-negative ($n = 57$) groups based on HBsAg status. Propensity score matching (PSM, 1:1, caliper 0.02) was performed to balance baseline characteristics. The primary endpoint was surgical conversion. Secondary endpoints included tumor response (mRECIST), overall survival (OS), progression-free survival (PFS), and treatment-related adverse events (AEs).

Results: After PSM, 32 matched pairs were included. HBV-positive patients achieved significantly higher surgical conversion rates both before PSM (25.6% vs. 12.3%, $P = 0.041$) and after PSM (20% vs. 10%, $P = 0.021$). Tumor response was also superior in the HBV-positive group, with higher ORR (36.7% vs. 20%, $P = 0.043$) and DCR (83.3% vs. 60%, $P = 0.045$). HBV-positive patients exhibited significantly prolonged OS and PFS. After matching, median OS was not reached in the HBV-positive cohort but was 20 months in HBV-negative patients ($P = 0.014$). Median PFS was 28.4 vs. 17.3 months, respectively ($P = 0.030$). Tumor diameter ≥ 7 cm was the only independent predictor of OS (HR 3.64, $P = 0.019$) and PFS (HR 12.66, $P = 0.002$). Grade 3–4 AEs were comparable between groups, and no treatment-related deaths occurred.

Conclusions: HBV-positive patients receiving TACE combined with targeted therapy and PD-1 inhibitors achieve superior surgical conversion, enhanced tumor response, and improved survival compared with HBV-negative patients. HBV infection status may serve as a clinically meaningful factor for selecting candidates to benefit from conversion therapy in uHCC.

Keyword(s) : unresectable HCC; HBV infection; conversion therapy; TACE; tyrosine kinase inhibitor; PD-1 inhibitor; survival outcomes

1572 - Comparison of Long term Efficacy and Renal Safety between Entecavir and Tenofovir Alafenamide Fumarate in Treatment naive Patients with Chronic Hepatitis B

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Background / Hypothesis:

To analyze the long-term virological efficacy and renal safety of entecavir (ETV) and tenofovir alafenamide fumarate (TAF) as first-line antiviral therapies in patients with chronic hepatitis B (CHB).

Materials and Methods:

A retrospective analysis was performed on CHB patients who received ETV or TAF treatment and completed ≥ 192 weeks of follow-up. Propensity score matching (1:1) was used to balance baseline data (age, sex, liver/renal function), resulting in 45 patients in each group. The Kaplan–Meier method was used to compare the cumulative incidences of renal dysfunction, low-level viremia (LLV), HBV DNA suppression, and HBeAg/HBeAb seroconversion. Intergroup differences were analyzed by the Log-rank test, hazard ratios (HR) were estimated by the Cox proportional hazards model, and repeated measures ANOVA was used to evaluate the dynamic changes in quantitative HBsAg and estimated glomerular filtration rate (eGFR).

Results:

After propensity score matching, the two groups were well-balanced in baseline characteristics (age, gender, liver/renal function). With prolonged treatment, the cumulative incidence of renal dysfunction gradually increased in both groups, with parallel survival curves and no significant difference ($\chi^2=0.032$, $P=0.858$; TAF vs. ETV HR=1.083, 95% CI, 0.44–2.66). The cumulative incidence of LLV also increased over time, which was slightly higher in the TAF group but not statistically significant ($\chi^2=0.415$, $P=0.520$; HR=1.233, 95% CI, 0.610–2.494). For HBV DNA suppression, the cumulative rates increased similarly in both groups ($P>0.05$) with a median time of 48 weeks (HR=1.063, 95% CI, 0.693–1.631). No significant differences were observed in HBeAg/HBeAb seroconversion rates between the two groups; the median time to HBeAb seroconversion was 96 weeks in both groups, while the median time to HBeAg seroconversion was 48 weeks in the ETV group and 96 weeks in the TAF group (HR=0.85, 95% CI, 0.51–1.42). HBsAg levels declined significantly over time in both groups ($P=0.001$).

0.001) with no intergroup differences. Baseline eGFR was similar between the two groups (≈ 109 mL/min-1.73m²) and showed a mild linear decline ($F=22.420$, $P<0.001$), with no significant intergroup variations at different time points.

Conclusions:

During approximately 192 weeks of real-world follow-up, both ETV and TAF as first-line antiviral therapies achieved similar HBV DNA suppression rates and HBeAg/HBeAb seroconversion rates, with comparable effects on lowering HBsAg quantitation. There were no significant differences between the two regimens in terms of the cumulative incidence of renal dysfunction and dynamic changes in eGFR, indicating overall favorable renal safety.

Keyword(s) : Chronic Hepatitis B;Entecavir;Tenofovir Alafenamide Fumarate; seroconversion rates; safety

1574 - Aeromonas Infections in Hepatic Hospital Source Specific Distribution and Antimicrobial Resistance Profiles Over Two Decades

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Background / Hypothesis:

Aeromonas species represent a serious threat to patients with hepatic disease, who are particularly vulnerable to bacterial infections due to immunocompromised status. However, comprehensive data focusing on Aeromonas infections specifically in this population remain scarce. This study aimed to investigate the clinical distribution and antimicrobial resistance patterns of Aeromonas species isolated from patients in hepatic hospital.

Materials and Methods:

We conducted a retrospective study at a tertiary liver hospital of Beijing from 2000-2023. Clinical data and microbiological results were extracted from the laboratory information system. Species identification and antimicrobial susceptibility testing were performed following standard protocols. Data analysis was completed using WHONET 5.6 and SPSS 22.0.

Results:



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A total of 408 *Aeromonas* strains were identified between 2000 and 2023. Among 197 patients with clearly documented clinical data, 91.4% (180/197) had underlying liver diseases, including cirrhosis, hepatocellular carcinoma, biliary tract disorders, and HBV/HCV-related liver infections. The predominant species were *A. hydrophila* (36.52%), *A. veronii* bv *sobria* (30.88%), and *A. caviae* (27.45%). Bloodstream infections were the most common (46.6%), followed by ascites (28.1%), bile (8.8%), stool (7.1%), hydrothorax (6.0%), and sputum (3.4%). Notably, *A. hydrophila* was the predominant species in bloodstream infections (47.6%), significantly higher than in ascites, bile, stool, and hydrothorax. In ascites, *A. veronii* bv *sobria* (46.5%) was most frequent, while *A. caviae* dominated in bile (66.7%), stool (51.6%), and hydrothorax (44.0%). Bloodstream isolates exhibited lower resistance rates to most antibiotics compared to ascites isolates. Regarding species-specific AMR, *A. caviae* demonstrated higher resistance to ceftazidime (CAZ), cefepime (FEP), piperacillin/tazobactam (TZP), aztreonam (ATM), and levofloxacin (LVX) than *A. hydrophila* and *A. veronii* bv *sobria*. In contrast, *A. veronii* bv *sobria* showed the lowest resistance rates overall.

Conclusions: In patients with hepatic disease, *Aeromonas* species exhibit distinct clinical distributions and antimicrobial resistance patterns based on infection sources. Source-specific predominance was observed, with *A. hydrophila* in blood, *A. veronii* bv *sobria* in ascites, and *A. caviae* in biliary and abdominal sources. The higher resistance in non-bloodstream isolates and significant inter-species variation underscore the need for tailored antimicrobial strategies in this high-risk population.

Keyword(s) : *Aeromonas*, antimicrobial resistance, liver disease, clinical distribution

1575 - Metastatic Hepatic Pleomorphic Liposarcoma with Ruptured vascular mass A case report

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Background / Hypothesis:

Pleomorphic liposarcoma is a rare subtype of liposarcoma, accounting for less than 5% of all liposarcoma cases and representing one of the most aggressive forms of soft tissue sarcoma.¹ While liposarcomas primarily occur in the extremities and

retroperitoneum, hepatic involvement is exceedingly rare, with only a handful of cases reported in the literature.² The liver, being a vascular organ, presents a unique set of challenges when sarcomas metastasize or originate within it. These tumors are often diagnosed at advanced stages due to their asymptomatic nature in early phases and the nonspecificity of initial symptoms such as epigastric pain, anorexia, and nausea.

Materials and Methods:

This case report describes a 25-year-old male presenting with metastatic hepatic pleomorphic liposarcoma complicated by a ruptured vascular mass and systemic metastases. The patient initially experienced nonspecific symptoms, including epigastric pain, anorexia, and nausea, leading to delayed diagnosis. Imaging revealed a large, vascular hepatic mass with evidence of capsular rupture, while histopathology confirmed a high-grade pleomorphic liposarcoma.

Results:

The management of this case focused on controlling the complications arising from the advanced stage of pleomorphic liposarcoma of the liver, including tumor rupture, obstructive jaundice, and systemic metastases. The primary therapeutic intervention was transarterial embolization (TAE), performed in two separate sessions, to manage the vascular nature of the hepatic tumor and prevent further hemorrhage.

Conclusions: The case underscores the complexities of managing hepatic sarcomas, including the use of transarterial embolization to control vascular rupture and the challenges of addressing systemic metastases. While some studies have explored the role of embolization and surgical interventions in managing liver sarcomas, their effectiveness remains variable.⁷ This case contributes to the growing body of literature on pleomorphic liposarcoma, offering insights into its clinical course, radiological findings, and therapeutic challenges.

Keyword(s) : liposarcoma, liver, TACE

1577 - Long Term HBsAg Kinetics and Prognostic Significant in Patients with Compensated Cirrhosis A 13 year Prospective Cohort on Entecavir Therapy

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Background / Hypothesis: Hepatitis B surface antigen (HBsAg) levels in patients with chronic hepatitis B (CHB) reflect disease activity and are predictive of long-term outcomes. However, most existing studies are limited to approximately 5 years of follow-up and rely on a narrow set of serological markers, making it difficult to characterize the long-term kinetics of HBsAg and its prognostic significance during nucleos(t)ide analogue (NA) therapy. This study established a prospective cohort with up to 13 years of follow-up to evaluate the association between baseline HBsAg levels and clinical prognosis, and to delineate the long-term trajectory of HBsAg over time.

Materials and Methods: From 2012 to 2013, 50 treatment-naïve patients CHB patients with compensated cirrhosis receiving entecavir (ETV) treatment were enrolled and followed semi-annually. Serial data included HBsAg, HBeAg, multiple laboratory and imaging parameters, and records of long-term clinical events (LREs), defined as hepatic decompensation, hepatocellular carcinoma, or liver-related death. HBsAg kinetics were analyzed using a linear mixed-effects model, HBeAg changes with generalized estimating equations, and prognosis with the Kaplan–Meier analysis and Cox regression models.

Results: Over the 13-year period, HBsAg levels demonstrated a steady and continuous decline, with an average annual reduction of 24.4%, representing the most consistent and time-dependent serological markers. Among ? patients who were HBeAg-positive at baseline, HBeAg seroconversion rates were ?% and ?% at years 5 and 10, respectively. Virological response (VR, defined as HBV DNA < 10 IU/mL) was achieved in 67.3% (33/49) of patients at 6 months and 85.7% (42/49) at 1 year. Regarding clinical outcomes, patients with baseline HBsAg < 1000 IU/mL exhibited significantly higher LRE-free survival ($p = 0.013$), indicating that lower initial HBsAg levels are associated with improved long-term clinical stability.

Conclusions: This 13-year ultra-longitudinal study

systematically confirms two key characteristics of HBsAg during antiviral therapy: (1) HBsAg levels demonstrate a sustained decline over time, serving as the most stable kinetic marker of long-term treatment response; and (2) patients with lower baseline HBsAg levels experience better clinical outcomes and higher LRE-free survival. These findings provide an important foundation for individualized risk stratification and treatment efficacy evaluation in CHB patients with compensated cirrhosis.

Keyword(s): Hepatitis B, HBsAg Kinetics, Compensated Cirrhosis

1578 - Construction of a Predictive Model for Adverse Outcomes in Patients with Acute Decompensated Cirrhosis Based on PBMC Transcriptomic Analysis

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Background / Hypothesis:

The PREDICT study described three subtypes of acute decompensated cirrhosis (AD) with different pathophysiology and prognosis: stable decompensated cirrhosis (SDC), unstable decompensated cirrhosis (UDC) and pre-acute-on-chronic liver failure (pre-ACLF). The mortality rates of patients with different subtypes vary. Among them, the one-year mortality rate of SDC patients is the lowest (about 10%), while that of UDC and pre-ACLF patients is as high as 36% and 67% respectively. Therefore, there is an urgent need to identify high-risk AD patients early and intervene for them in a timely manner.

Materials and Methods:

Based on the inclusion and exclusion criteria and the completeness of the 90-day follow-up outcome, a total of 163 AD patients were ultimately included and divided into the recovery group (no readmission due to decompensation during the 90-day follow-up period) and the non-recovery group (readmission due to decompensation or progression to ACLF or death during the 90-day follow-up period). Peripheral blood was collected within 24 hours of their first admission and transcriptomic analysis of PBMCs was subsequently performed between the two groups. The random forest and the Least Absolute Shrinkage and Selection Operator (LASSO) regression were adopted for feature gene screening and to construct the

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prediction model.

Results:

PBMC transcriptome sequencing analysis revealed 165 DEGs between the recovery group and the non-recovery group. Nine key genes (FOXC1, TPD52, NOS1AP, SPRY2, TIFAB, MANSC1, AP3B2, ROBO4, HLA-DQA1, TPST1) were screened out through random forest and LASSO regression. A prediction model with good accuracy (AUC=0.82), calibration and practicability was constructed, which was superior to the traditional MELD-Na (AUC=0.579) and CLIF-C AD (AUC=0.617) scores.

Conclusions:

The predictive model constructed using the 9 key DEGs screened by LASSO regression can effectively guide the risk stratification of AD patients, and this model can be used as an auxiliary tool for evaluating the prognosis of AD patients.

Keyword(s) : Acute Decompensated Cirrhosis; Adverse Outcomes; Predictive Model; Machine Learning

1579 - Comparative Outcomes of Subsequent Line Therapies Following First Line Atezolizumab Plus Bevacizumab for Advanced Hepatocellular Carcinoma in a High Volume Korean Center

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Background: While first-line therapy for advanced hepatocellular carcinoma (HCC) is well established, optimal subsequent-line treatment after immunotherapy remains unclear and varies widely across centers. We analyzed the patterns and outcomes of second- to fourth-line systemic therapies following first-line atezolizumab plus bevacizumab in a high-volume tertiary center.

Methods: We analyzed data from 353 patients with advanced HCC who underwent subsequent treatment after failure of 3 or more cycles of first-line atezolizumab plus bevacizumab initiated between May 2022 and December 2023 at a high-volume tertiary center in Korea. Overall survival (OS) and progression-free survival (PFS) were evaluated using the Kaplan-Meier method, and differences between treatment groups were compared using the log-rank test.

Results: Tyrosine kinase inhibitors (TKIs) constituted the majority of subsequent-line systemic therapies. In the second-line setting, lenvatinib (n=174, 49.3%) and sorafenib (n=114, 32.3%) were most frequently used, with no use of immune checkpoint inhibitors (ICIs). Sorafenib was the leading therapy (n=85, 52.1%) in the third-line setting, followed by regorafenib (n=43, 26.4%), and ICIs (n=11, 6.7%). In the fourth-line setting, regorafenib was most commonly administered (n=40, 44.9%), followed by nivolumab (n=22, 24.7%) and cabozantinib (n=12, 13.5%). Among second-line therapies, lenvatinib showed longer median OS (9.5 vs 8.3 months; HR 1.17, 95% CI 0.87–1.58, p=0.30) and significantly longer median PFS (5.3 vs 2.8 months; HR 1.55, 95% CI 1.19–2.02, p=0.001) compared with sorafenib. In the third-line setting, TKIs (n=136) and ICIs (n=11) demonstrated similar outcomes, with comparable median OS (6.9 vs 4.0 months; HR 1.86, 95% CI 0.80–4.32, p=0.15) and median PFS (1.7 vs 1.8 months; HR 1.05, 95% CI 0.56–1.95, p=0.88). In the fourth-line setting, outcomes for TKIs (n=57) and ICIs (n=21) were likewise similar in median OS (5.6 vs 5.5 months; HR 1.00, 95% CI 0.54–1.85, p=0.99) and median PFS (1.9 vs 1.8 months; HR 0.85, 95% CI 0.51–1.41, p=0.53).

Conclusions: TKIs were the predominant subsequent-line therapies after first-line atezolizumab plus bevacizumab, with lenvatinib showing superior PFS compared with sorafenib in second-line setting. Beyond second line, TKIs and ICIs demonstrated similar survival outcomes. These real-world treatment patterns highlight the need for multi-center prospective studies to guide optimal post-immunotherapy sequencing.

Keyword(s) : Hepatocellular carcinoma, Immunotherapy, Subsequent-line therapy, Treatment sequencing, Real-world data

1582 - Proteomic Profiling and Clinical Correlation of Prognostic Proteins in Advanced Hepatocellular Carcinoma with Hyperbilirubinemia

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Background / Hypothesis:

This study focused on patients with advanced hepatocellular carcinoma (HCC), aiming to analyze differentially expressed proteins and their enriched pathways in the baseline serum of patients with hyperbilirubinemia compared to those with normal

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total bilirubin (TBil) levels, and to investigate the role of key regulatory proteins in HCC progression by integrating prognostic information.

Materials and Methods:

A total of 72 HCC patients receiving targeted immunotherapy were enrolled and stratified into a hyperbilirubinemia group (n=28) and a normal TBil group (n=44) based on TBil levels. Proteomics technology was employed to compare baseline differential proteins between the groups, followed by functional enrichment analysis and protein-protein interaction (PPI) network construction. Cox regression analysis was conducted in the hyperbilirubinemia group, and key proteins were validated through survival analysis.

Results:

Baseline differential protein analysis identified 35 upregulated proteins (including IL-17A) and 1 downregulated protein. KEGG pathway enrichment revealed that these proteins were primarily involved in the cytokine-cytokine receptor interaction pathway. The Cox regression model assessed the prognostic value of IL-17A, and survival analysis demonstrated a significantly shorter median overall survival in the high IL-17A expression group (HR=3.15, 95% CI: 1.17-8.46, P=0.023), indicating that IL-17A is an independent predictor of poor prognosis.

Conclusions: High IL-17A expression is significantly associated with poor prognosis in HCC patients with hyperbilirubinemia, serving as both a prognostic biomarker and a potential therapeutic target.

Keyword(s): Hepatocellular Carcinoma; Hyperbilirubinemia; Proteomics; Prognosis

1583 - Effect Of Polyethylene Glycol Versus Lactulose on Hepatic Encephalopathy In Patients With Liver Cirrhosis A Randomized Clinical Trial (PEG HE Trial)

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Background / Hypothesis:

Hepatic encephalopathy is a prevalent complication of liver cirrhosis being responsible for significant morbidity and mortality. Lactulose remains the gold standard treatment for liver cirrhosis; however, its use

is limited by refractory effectiveness and adverse effects. Polyethylene glycol is emerging to be a potential alternative therapy. This study aimed to compare effectiveness and safety of PEG and lactulose in patients hospitalized with HE.

Materials and Methods:

This randomized controlled trial, with parallel group was conducted at Aga Khan University, Karachi, Pakistan. Eligible patients were randomized to receive either PEG or lactulose. The primary outcome was resolution of HE with ≥ 1 -grade reduction in HESA score at 24, 48, and 72 hours. The secondary outcomes were the length of hospital stay, adverse events, readmissions, and three-months mortality.

Results:

A total of 55 patients were enrolled. Patients receiving PEG and lactulose achieved comparable effectiveness in resolution of HE in 72 hours (p-value = 0.7232). At 24 and 48 hours, both PEG and lactulose achieved similar HESA scores demonstrating comparable effectiveness in resolving HE, although not statistically significant (p-value = 0.2605, p-value = 0.5222). The lower bounds of the 95% confidence intervals for risk differences continuously stayed above the 10% threshold, indicating that PEG was non-inferior to lactulose at all timepoints. No adverse events were reported in both groups, with similar readmissions and three-month mortality.

Conclusions:

PEG was found to be effective in resolving HE with initial improvement in symptoms and safety profile. PEG presents to be a viable alternative to lactulose for HE management, although long-term studies with larger sample sizes are warranted.

Keyword(s):

1584 - Drug related hepatic steatosis associated with antineoplastic drugs A pharmacovigilance study from the FDA adverse event reporting system (FAERS)

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Background / Hypothesis:



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Since drug-induced hepatic steatosis is relatively rare but closely related to adjusting the treatment of the primary disease, preventing liver steatosis and its inflammation. This study investigated the risk factors of drug-induced hepatic steatosis by analyzing a large dataset from the FDA Adverse Event Reporting System (FAERS).

Materials and Methods:

The reporting odds ratios (ROR) and information component (IC) were used to evaluate the reports of hepatic steatosis from the first quarter of 2004 to the third quarter of 2024. Univariate, LASSO and multivariate regression analyses were used to explore the risk factor. Moreover, the time to onset of hepatic steatosis was also assessed.

Results:

Overall, 16,767 cases related to fatty liver disease were found in FAERS. In total, 117 drugs were found to be associated with hepatic steatosis, with antineoplastic drugs (28/117), antiviral drugs (18/117), psychotropic drugs (17/117), and immunomodulatory drugs (12/117) being the most common. Age under 41 years old, a heavier weight and 13 different drugs, including 3 antineoplastic drugs of interest, namely Methotrexate, Pegaspargase, and Tamoxifen, were risk factors for hepatic steatosis. The median time to onset was 123.50 days, while the onset of Pegaspargase was faster, with a median time of only 19 days ($P < 0.001$).

Conclusions:

With the increasing variety of drugs, it's necessary to focus on hepatic steatosis and steatohepatitis caused by drugs, and adjust the therapeutic drugs in a timely manner to reduce the incidence of liver fibrosis, liver cirrhosis and prevent the interruption of treatment for the primary disease.

Keyword(s) : drug-induced hepatic steatosis, antineoplastic drugs, pharmacovigilance, FAERS, adverse events

1585 - 20(s) ginsenoside Rg3 inhibits hepatocellular carcinoma via akkermansia driven regulation of intestinal microbiota and bile acid metabolism

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a leading cause of cancer-related mortality worldwide. The gut-liver axis is increasingly recognized as a critical regulator of HCC pathogenesis. We hypothesized that 20(S)-ginsenoside Rg3, a bioactive component of ginseng, exerts anti-HCC effects by promoting Akkermansia-driven modulation of intestinal microbiota and bile acid metabolic homeostasis, thereby enhancing natural killer (NK) cell function.

Materials and Methods:

HCC models were established via Hepa1-6 cell orthotopic inoculation in C57BL/6 mice, which were treated with 20(S)-ginsenoside Rg3 or PBS by gavage. Germ-free (GF) mice, fecal microbiota transplantation (FMT), and antibiotic depletion experiments validated gut microbiota dependence. 16S rRNA sequencing, targeted metabolomics, RNA sequencing, Western blotting, and flow cytometry were used to analyze microbiota composition, bile acid profiles, and NK cell function.

Results:

20(S)-ginsenoside Rg3 significantly reduced HCC tumor burden without altering mouse weight or directly affecting HCC cell viability. It localized primarily in the intestinal lumen, and its anti-HCC effects were abrogated in GF and antibiotic-treated mice but restored by FMT from Rg3-treated mice. Rg3 dose-dependently promoted Akkermansia growth, which synergistically enhanced anti-HCC efficacy. Moreover, Rg3 increased intestinal and serum ursodeoxycholic acid levels and activated the PI3K/AKT/mTOR pathway to enhance NK cell function.

Conclusions:

20(S)-ginsenoside Rg3 inhibits HCC through the gut microbiota-Akkermansia-bile acid axis, which activates the TGR5/PI3K/AKT pathway to enhance NK cell-mediated anti-tumor immunity. This study provides a promising therapeutic strategy for HCC.

Keyword(s) : 20(S)-ginsenoside Rg3; Hepatocellular carcinoma; Gut microbiota; Akkermansia; Bile acid metabolism; NK cell

1586 - Construction and validation of a nomogram for predicting survival in hepatocellular carcinoma patients with hyperbilirubinemia receiving targeted immunotherapy

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Background / Hypothesis:

To construct a nomogram prediction model for predicting the 3-, 6-, and 12-month survival rates of patients with hepatocellular carcinoma (HCC) accompanied by hyperbilirubinemia undergoing targeted immunotherapy and to evaluate its predictive efficacy.

Materials and Methods:

A retrospective analysis was conducted on 203 patients with HCC accompanied by hyperbilirubinemia who received targeted immunotherapy at The Fifth Medical Center of Chinese PLA General Hospital from June 2020 to September 2024. Overall survival (OS) was calculated based on post-discharge follow-up results, and clinical data were compared between the death and survival groups. Univariate and multivariate Cox regression analyses were used to identify significant variables. A nomogram prediction model for survival prognosis was constructed using R software. The predictive efficacy of the nomogram was comprehensively evaluated and validated by calculating the concordance index (C-index), area under the receiver operating characteristic (ROC) curve (AUC), calibration curves, decision curve analysis (DCA), and risk stratification analysis.

Results: Univariate regression analysis showed that baseline alpha-fetoprotein (AFP) level, baseline total bilirubin (TBil) level, TBil decline within one week, baseline aspartate aminotransferase (AST) level, and Child-Pugh classification were associated with patient survival time. Multivariate Cox regression analysis revealed that baseline AFP level and Child-Pugh classification were independent risk factors for survival time. Based on considerations of clinical utility and comprehensiveness of the predictive model, the study adopted a variable selection strategy grounded in clinical importance. Five variables were included in the final model: baseline AFP level, baseline TBil level, TBil decline within one week, baseline AST level, and Child-Pugh classification. The nomogram demonstrated a C-index of 0.725 (95% CI: 0.674–0.776). The AUC values for predicting 3-, 6-, and 12-month OS were 0.786, 0.786, and 0.821, respectively. Calibration curves and DCA indicated good consistency and strong clinical utility. Risk stratification based on the total model score showed significant differences in OS among low-,

medium-, and high-risk patients ($P < 0.0001$).

Conclusions: The nomogram prediction model, incorporating baseline AFP level, baseline TBil level, TBil decline within one week, baseline AST level, and Child-Pugh classification, has good predictive value for survival in patients with HCC accompanied by hyperbilirubinemia and can serve as a reference for prognostic assessment.

Keyword(s) : Hepatocellular carcinoma; Hyperbilirubinemia; Nomogram; Prognosis

1590 - Decoding the Genetic Overlap A Pathway to Early Diagnosis of AKI in Cirrhotic Patients

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Background / Hypothesis: Acute kidney injury (AKI) is a common and serious complication in patients with cirrhosis, characterized by high morbidity and mortality. The causes of AKI in cirrhotic patients include renal hypoperfusion due to volume depletion, acute tubular necrosis, and hepatorenal syndrome. Clinically, renal injury in these patients is often overlooked, and the onset of hepatorenal syndrome typically indicates progression to an end-stage condition. Therefore, early identification and increased attention to acute kidney injury in cirrhotic patients are of great importance. This study aims to identify shared genetic signatures between cirrhosis and AKI, which may contribute to the early detection of renal impairment in this patient population.

Materials and Methods: Based on transcriptomic data analysis, common differentially expressed genes (DEGs) between cirrhosis and AKI were identified. Subsequent analyses included gene correlation analysis, functional enrichment analysis, and receiver operating characteristic (ROC) analysis of the candidate genes. Finally, the subgroup analysis of cirrhosis patient samples was performed based on the candidate genes.

Results: Compared with the control group, 115 DEGs were identified in cirrhosis patients, of which 98 genes were up-regulated and 17 genes were down-regulated. Meanwhile, 55 DEGs were obtained from AKI patients, including 32 up-regulated and 23 down-regulated

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genes. Ultimately, a total of 9 common DEGs between cirrhosis and AKI were identified (COLEC11, HAO2, ADAMTS1, RGS2, S100A8, CNDP1, NQO1, LCN2, AKR1B10). Functional analysis of these candidate genes indicated significant enrichment in metabolic pathways. ROC analysis demonstrated that the candidate genes exhibited high predictive performance in both cirrhosis and AKI cohorts, suggesting their potential as shared diagnostic biomarkers for cirrhosis and AKI. In addition, two distinct subgroups (C1 and C2) were identified in cirrhosis patients. Comparing the two subgroups, 215 DEGs were acquired, including 174 up-regulated and 41 down-regulated genes.

Conclusions: This study successfully identified the shared gene signatures between liver cirrhosis and AKI, which contributed to the early identification of AKI in cirrhotic patients.

Keyword(s) : liver cirrhosis, acute kidney injury, differentially expressed genes, biomarker, diagnose

1591 - Circulating Hypermethylated RASSF1A as a Possible Marker of Hepatocellular Carcinoma in Chronic HCV patients

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a global health problem and the fourth leading cause of cancer-related deaths worldwide. The carcinogenic role of methylation, a critical epigenetic modification, is well known in HCC. Thus, in this study, we aimed to assess the role of plasma level of methylated RASSF1A in post hepatitis C virus (HCV) cirrhotic patients as a non-invasive diagnostic marker for HCC.

Materials and Methods:

The levels of methylated RASSF1A were determined in plasma of 120 participants who were classified to three groups (40 participants each); HCC patients on top of

chronic HCV infection, post hepatitis C cirrhotic patients without HCC in addition to healthy matched age and sex control group. Methylation-sensitive restriction enzyme digestion and real-time quantitative polymerase chain reaction method was employed to measure the expression level of RASSF1A.

Results:

Patients with HCC exhibited significantly higher levels of circulating hypermethylated RASSF1A than post hepatitis C patients and control groups. In addition, a statistically significant correlation between RASSF1A and the different clinico-pathological parameters was observed. The receiver operating characteristic curves plotted showed that plasma RASSF1A helped in significant differentiation between HCC and cirrhotic patients.

Conclusions:

Circulating hypermethylated RASSF1A could be a non-invasive diagnostic marker for discriminating HCC among post HCV cirrhotic patients and characterizing their progression.

Keyword(s) : circulating DNA; hypermethylation; RASSF1A; liquid biopsy; hepatocellular carcinoma

1593 - Geniposide alleviates ALD by reducing CXCR2+neutrophil infiltration through inhibiting FKBP5 CXCL1 signaling pathway

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Background / Hypothesis: Alcohol-associated liver disease (ALD) is a major cause of liver transplantation and death worldwide, contributing significantly to liver cirrhosis and hepatocellular carcinoma, with limited effective treatment options. Geniposide (GEN) is one of the important active ingredients from Zhizi Dahuang Decoction (ZZDHD), a classic traditional Chinese medicine formula for the treatment of ALD. This study aimed to investigate the effects of GEN treating ALD, and to explore its underlying mechanisms in ALD.

Materials and Methods: NIAAA model was built to evaluate effects of GEN treating ALD. Anti-Ly6G antibody was used to deplete neutrophils in NIAAA

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model. Adeno-associated virus (AAV)-mediated overexpression model of CXCL1 was generated. Single-cell transcriptomics and transcriptome sequencing were conducted to screen the regulatory mechanism. Oil-Red O staining was employed to evaluate lipid deposition. A series of in vitro assays, including cell viability, transwell migration, tunnel were employed. Protein expression levels were analyzed by western blotting and immunofluorescence.

Results: ZSDHD administration and GEN reduced hepatic damage, lipid accumulation and oxidative stress in NIAAA model mice. ZSDHD and GEN reduced neutrophil infiltration in the liver. Analytic results from single-cell transcriptomics showed that CXCR2+neutrophils were recruited in livers of NIAAA model, and the expression levels of CXCL1 and FKBP5 in hepatic cells were increased in NIAAA model. Ethanol treatment promoted the secretion of CXCL1 in AML12 cells, facilitating neutrophil migration, which is partially restored by GEN administration. In addition, hepatocytic apoptosis was reduced by GEN. Overexpression of CXCL1 contributed to the migration of neutrophils and hepatocytic apoptosis, and eliminated the effective effects of GEN treating ALD. FKBP5 and the downstream signaling molecules were inhibited by GEN, which was correlated with CXCL1 expression.

Conclusions: GEN alleviated ALD by reducing hepatocytic apoptosis via inhibiting FKBP5/CXCL1 mediated CXCR2+neutrophil recruitment. These results highlight the potential of GEN as a novel therapeutic agent for the treatment of ALD.

Funding: This work was supported by the National Natural Science Foundation of China (No.82574840).

Keyword(s) : Geniposide; Neutrophils; Alcohol-related liver disease; CXCL1

1594 - A machine learning model for predicting hepatocellular carcinoma in patients with MASLD

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Background/Hypothesis: The risk of hepatocellular carcinoma (HCC) is significantly elevated in patients with metabolic dysfunction-associated steatotic liver disease (MASLD). This study aimed to develop and

validate a machine learning model for predicting HCC in a large, multicenter MASLD cohort.

Materials and Methods: A total of 18,484 MASLD patients from six Korean hospitals (2001–2022) were included after data preprocessing. Patients from Severance Hospital were divided into a training cohort (n=3,721) and an internal validation cohort (n=4,370), while patients from the remaining institutions comprised the external validation cohort (n=10,393). To account for substantial differences in HCC incidence among hospitals, two additional external validation subsets were created: external set 2 excluding institutions with markedly high incidence, and external set 3 including only those institutions. Four machine learning algorithms—gradient boosting machine (GBM), random survival forest (RSF), deep neural network (DNN), and survival support vector machine (surv-SVM)—were developed using 12 variables selected from prior evidence and multivariable Cox regression results. Model performance was compared using the C-index, AUROC, AUPRC, sensitivity, specificity, and F1 score across all cohorts.

Results: During a median follow-up of 51.6 months, 357 HCC events occurred (incidence: 0.45 per 100 person-years). GBM demonstrated the strongest performance in the training cohort (C-index 0.95, AUROC 0.98, AUPRC 0.36), with well-balanced sensitivity (0.93) and specificity (0.97). Internal validation confirmed stable performance (C-index 0.90, AUROC 0.89, AUPRC 0.19), outperforming RSF and the Cox-based scoring model. External validation sets 1–3 consistently supported the robustness of GBM, with AUROCs ranging from 0.83 to 0.89, C-indices from 0.82 to 0.88, and AUPRCs from 0.19 to 0.25, indicating superior discrimination in imbalanced data. SHAP analysis identified liver cirrhosis, age, diabetes, LDL-cholesterol, and antihypertensive medication use as major contributors to increased HCC risk, while higher platelet count and albumin were associated with decreased risk.

Conclusions: This multicenter study developed and validated a GBM-based machine learning model using 12 readily available clinical variables for predicting HCC in MASLD patients. The model demonstrated consistently strong performance across internal and multiple external validation cohorts, exceeding that of traditional Cox-based approaches. Although external validation in non-Korean populations and prospective evaluation of clinical utility remain necessary, the GBM model provides a promising foundation for future individualized HCC surveillance strategies in MASLD.

Keyword(s) : metabolic dysfunction-associated

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steatotic liver diseases, hepatocellular carcinoma, machine learning

1595 - Development and validation of a non invasive model for predicting advanced liver fibrosis in patients with metabolic dysfunction associated fatty liver disease and dysglycemia

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Background / Hypothesis:

The confluence of metabolic dysfunction-associated fatty liver disease (MAFLD) and dysglycemia delineates a high-risk subgroup characterized by a substantially increased likelihood of progressive liver fibrosis. This study aimed to develop and validate a novel, parsimonious non-invasive model for the accurate prediction of advanced fibrosis (stage F3–F4) in this at-risk population.

Materials and Methods:

A retrospective analysis was conducted in 606 consecutive adults with biopsy-proven MAFLD and dysglycemia from Beijing Ditan Hospital (2010–2025). The cohort was randomly split into training (n=424) and validation (n=182) cohorts. Variable selection was performed via multivariable logistic regression. Model performance was rigorously evaluated for discrimination (AUC), calibration, and clinical utility (DCA), including a head-to-head comparison with FIB-4 and APRI.

Results:

Three variables were identified as independent predictors of advanced fibrosis: body mass index (BMI) (adjusted odds ratio [aOR] 1.40 per 1 kg/m² increase, 95% CI 1.30–1.51; P < 0.001), alanine aminotransferase (ALT) (aOR 1.01 per 1 U/L increase, 95% CI 1.01–1.01; P < 0.001), and white blood cell count (WBC) (aOR 1.15 per 1 × 10⁹/L increase, 95% CI 1.01–1.31; P = 0.035).

The resulting model demonstrated exceptional discrimination, with an area under the curve (AUC) of 0.87 (95% CI 0.84–0.91) in the training cohort and 0.91 (95% CI 0.87–0.95) in the validation cohort. It significantly outperformed both FIB-4 and APRI across all performance metrics, exhibited excellent calibration, and provided superior net clinical benefit across a wide range of risk thresholds. The model demonstrated

robust generalizability across subgroups stratified by sex, BMI, and other metabolic factors, with consistently high AUCs (all >0.85).

Conclusions:

We developed a highly accurate and validated non-invasive model based on BMI, ALT, and WBC for predicting advanced fibrosis in patients with MAFLD and dysglycemia. Its simplicity and reliance on readily available parameters render it ideally suited for deployment in primary care and diabetology settings, where it could refine risk stratification and guide specialist referrals.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease(MAFLD); Liver Fibrosis; Prediction Model;Non-invasive; Dysglycemia; Biomarkers.

1597 - Glycyrrhizin Preparations in Liver Diseases A Systematic Review of Mechanisms and Therapeutic Potential

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Glycyrrhizin preparations, derived from the traditional Chinese medicine licorice, are important hepatoprotective agents in modern liver disease treatment. Their main active components include 18 α - and 18 β -glycyrrhetic acid, with representative drugs such as diammonium glycyrrhizinate, compound glycyrrhizin, and magnesium isoglycyrrhizinate. This review systematically elaborates on the pharmacokinetic characteristics of glycyrrhizin preparations and their multi-target pharmacological mechanisms, including anti-inflammatory, membrane-stabilizing, antioxidant, anti-apoptotic, and immunomodulatory effects. It also comprehensively reviews clinical evidence and efficacy in various liver diseases, such as viral hepatitis, drug-induced liver injury, alcoholic liver disease, non-alcoholic fatty liver disease, and autoimmune liver disease. Additionally, the safety profile, particularly the management of pseudoaldosteronism and drug interactions, is discussed. Finally, future research directions are outlined to provide a theoretical basis and practical reference for rational clinical use.

Keyword(s) : Glycyrrhizin; Glycyrrhizin preparations; Liver disease; Pharmacological mechanisms; Clinical applications; Safety

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1599 - Budd Chiari Syndrome and Predictive Factors for Recanalization

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Budd-Chiari Syndrome and Predictive Factors for Recanalization

Auteurs : Souili, borahma, chabib, lagdali, ajana, kadiri

Introduction:

Budd-Chiari syndrome (BCS) is a rare but severe vascular liver disorder caused by obstruction of the hepatic veins and/or inferior vena cava, leading to potentially life-threatening portal hypertension complications. Diagnosis relies primarily on imaging, and management follows a stepwise approach from medical therapy to interventional procedures. Some patients experience spontaneous recanalization with medical treatment alone. This study aimed to analyze the clinical, biological, and radiological characteristics of BCS patients in our department and identify factors predictive of recanalization.

Materials and Methods:

We conducted a retrospective, monocentric, descriptive, and analytical study at the Gastroenterology Department in ibn sina hospital, Rabat, including patients diagnosed with BCS between 1995 and 2025. Data were collected from medical records and analyzed using Jamovi software.

Results:

A total of 38 patients were included (mean age 32 years; F/M ratio 4.5:1). Hypertension was the most frequent comorbidity (18%). Hepatic risk factors were identified in 29% of patients. The most common presenting symptoms were abdominal distension (50%), asthenia (45%), jaundice (32%), and upper gastrointestinal bleeding (26%). On examination, ascites was present in 61%, collateral venous circulation in 66%, splenomegaly in 42%, and liver abnormalities in 53%. Imaging revealed hepatic vein obstruction in 84%, inferior vena cava obstruction in 34%, and chronic liver disease features in 63%. Liver tests showed cholestasis in 68%, cytolysis in 42%, and hepatocellular insufficiency in 42%.

Primary etiologies predominated: thrombophilia (29%), Behçet's disease (13%), myeloproliferative neoplasms (11%), and celiac disease (5%). Secondary causes included hepatocellular carcinoma and sarcoidosis (each 2.6%). Complications included ascites (71%),

upper gastrointestinal bleeding (32%), and hepatic encephalopathy (5%). All patients received etiological treatment and anticoagulation when indicated. Recanalization occurred in 29% of cases. Univariate analysis identified two predictive factors: diagnostic delay <6 months ($p = 0.021$) and anticoagulation combined with treatment of the underlying cause ($p = 0.037$).

Conclusion:

Early diagnosis and prompt management of BCS significantly increase the likelihood of spontaneous recanalization. Patients diagnosed within six months of symptom onset and receiving anticoagulation alongside targeted therapy for the underlying cause demonstrate the best outcomes.

Keyword(s) : budd chiari syndrom, recanalization, anti coagulation treatment,

1600 - Liver Fibrosis Activity on MRI (cT1) Predicts Incident Neurological Events Independent of Brain MRI Metrics in the UK Biobank

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Background / Hypothesis:

Chronic liver disease is increasingly recognised as a systemic inflammatory state with multisystem effects. Hepatic fibro-inflammation promotes cytokine release, metabolic dysfunction, endothelial injury, and immune activation—processes that mirror mechanisms driving cerebral small-vessel disease, neuroinflammation, and neurodegeneration.¹⁻³ These shared pathways suggest a potential liver-brain axis, but the contribution of hepatic fibro-inflammatory activity to neurological outcomes is not well defined. Corrected T1 (cT1), a quantitative MRI biomarker of liver fibro-inflammation, enables non-invasive assessment of this biology. We examined whether cT1 predicts incident neurological events independent of brain MRI burden.

Materials and Methods:

We analysed 29,344 UK Biobank participants with liver MRI (cT1, Proton Density Fat Fraction [PDFF]) and brain MRI (white matter hyperintensity [WMH] volume).

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Neurological events were defined using hospital records coded by ICD-10, including cerebrovascular disease, dementia, and late-life depression. cT1 was categorised as <800 ms, 800–875 ms, and ≥875 ms. High WMH burden was defined as WMH volume above the age-adjusted 80th percentile. Outcomes included (1) neurological events and (2) neurological-related hospitalisation. Prevalent events that occurred prior to the date of imaging were excluded. Cox models were adjusted for age, sex, diabetes, hypertension, dyslipidaemia, smoking, alcohol, education level, and Townsend deprivation index.

Results:

530 incident neurological events occurred during follow-up. Higher cT1 independently predicted neurological events (per 100 ms: HR 1.36, 95% CI 1.14–1.62, $p < 0.001$) and was significant for the ≥875 ms group (HR 2.32, 95% CI 1.14–4.72, $p = 0.021$). WMH >80th percentile (age-adjusted) was also associated with neurological events (HR 1.39, 95% CI 1.10–1.75, $p = 0.006$). Combining cT1 ≥800 ms with WMH >80th percentile produced the highest risk (HR 2.13, 95% CI 1.15–3.96, $p = 0.017$). Liver fat content as measured by PDFF was not associated with increased risk of neurological events.

Conclusions:

Higher liver fibro-inflammatory activity measured by cT1 is independently associated with neurological outcomes, and risk is amplified when combined with high age-adjusted WMH burden. Liver MRI biomarkers identify individuals at heightened risk of brain disease, highlighting a potential liver–brain axis in ageing.

Keyword(s) : liver-brain axis, cT1, PDFF, WMH

1604 - Liver MRI Biomarkers of Fibro inflammation and Steatosis Predict Long term Liver Outcomes and Mortality in the General Population

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Background / Hypothesis:

Liver fibro-inflammation and steatosis contribute to multisystem metabolic dysfunction through chronic

inflammatory signalling, immune activation, and hepatokine-mediated metabolic crosstalk. These pathways may predispose to adverse systemic outcomes, including progressive liver disease and mortality. 1-3 Quantitative liver MRI biomarkers—corrected T1 (cT1), reflecting fibro-inflammation, and proton density fat fraction (PDFF), reflecting steatosis—enable non-invasive assessment of hepatic pathobiology. We examined whether cT1 and PDFF predict incident liver outcomes in a large population cohort.

Materials and Methods:

Participants from the UK Biobank with complete liver MRI (cT1 and PDFF) were included. cT1 was categorised as <800 ms, 800–<875 ms, and ≥875 ms; PDFF as <5%, 5–<10%, and ≥10%. Outcomes included (i) liver events (ICD-10 codes representing chronic liver disease, cirrhosis, portal hypertension, hepatic decompensation, hepatocellular carcinoma, and liver-related procedures), (ii) liver-related hospitalisation, and (iii) all-cause mortality. Prevalent liver events occurring on or before the imaging date were excluded. Cox proportional hazards models adjusted for age, sex, diabetes, hypertension, dyslipidaemia, smoking, and alcohol exposure.

Results:

Among 33,565 individuals (median age 65 years), 1,509 (4.5%) had cT1 800–<875 ms and 263 (0.8%) had cT1 ≥875 ms. Higher cT1 strongly predicted liver events (per 100 ms HR 2.17 [1.75–2.69], $p < 0.001$; cT1 800–<875 ms HR 3.01 [2.04–4.44], $p < 0.001$; cT1 ≥875 ms HR 5.27 [2.81–9.85], $p < 0.001$) (Table 1). PDFF also independently predicted liver events (per 1% HR 1.08 [1.06–1.10], $p < 0.001$; PDFF ≥10% HR 3.15 [2.20–4.50], $p < 0.001$). cT1 ≥875 ms was associated with significantly increased risk of all-cause mortality (HR 2.09 [1.10–3.97], $p = 0.025$).

Conclusions:

cT1 and PDFF are robust, independent predictors of incident liver events in the general population, with cT1 also associated with mortality. Liver MRI biomarkers may enable early risk stratification and preventive intervention.

Keyword(s) : Quantitative liver MRI biomarkers

1605 - Clinical Characteristics and Epidemiological Features of Hepatitis E Virus Infection Among



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People Living with HIV in Shanghai China

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Background / Hypothesis:

Hepatitis E virus (HEV) poses a significant public health concern, particularly among immunocompromised populations. This study aimed to investigate HEV seroprevalence, clinical characteristics, and associated risk factors in people living with HIV (PLWH) in Shanghai, China.

Materials and Methods:

In this retrospective cohort study, 670 adult PLWH(> 18 years) underwent anti-HEV testing at Shanghai Public Health Clinical Center (2018-2024). These included epidemiological characteristics such as age, gender, ethnicity, and marital status. Hepatic profiles were also collected, comprising albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and total bilirubin (TBIL). HIV-specific markers were documented, including CD4 count, antiretroviral therapy (ART) regimen, and WHO clinical stage. Serum anti-HEV IgM and IgG antibodies were detected using Enzyme-Linked Immunosorbent Assay (ELISA) kits. The CD4 count was quantified in peripheral blood specimens from PLWH using flow cytometric analysis.

Results:

A retrospective analysis was conducted on serum IgG and IgM antibodies specific to HEV in 670 PLWH and 464 HIV-negative controls. The overall anti-HEV seropositivity rate among PLWH was 30.15% (202/670, 95% CI 26.68–33.62), with an IgG positivity rate of 30.00% (201/670, 95% CI 26.53–33.47). IgM positivity was observed in 1.19% (8/670, 95% CI 0.59–2.39), and dual IgM/IgG positivity in 1.04% (7/670, 95% CI 0.50–2.16). The seropositivity rate of anti-HEV IgG in the HIV-negative control group was 17.67% (82/464, 95% confidence interval: 14.20–21.14), with no IgM positivity, which was significantly lower than that in PLWH ($\chi^2 = 22.84$, $P < 0.001$). Univariate and multivariate analyses identified advanced World Health Organization (WHO) HIV stage (III/IV) as an independent risk factor for HEV co-infection ($P < 0.05$). Notably, no significant associations were observed with

age, gender, CD4 count, or liver function parameters.

Conclusions:

In summary, this study demonstrates that PLWH in Shanghai exhibit an increased risk of HEV infection, with advanced HIV disease stage (WHO III/IV) identified as a key associated factor. This underscores the necessity of incorporating targeted HEV screening awareness and prevention strategies into HIV care programs, particularly for patients with advanced disease. These findings provide a foundation for future research and potential refinements in clinical practice aiming to enhance care and prognosis for affected individuals.

Keyword(s) : Hepatitis E Virus, HIV, Seroprevalence, Risk Factors

1606 - Marked HBsAb elevation in anti HBc positive cancer patients receiving PD 1 inhibitors two case reports and literature review

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Background / Hypothesis:

PD-1 inhibitors have achieved major advances in cancer treatment and are increasingly studied for their immunological effects in chronic HBV infection. Chronic HBV remains a global health burden, and current therapies rarely achieve complete viral clearance. Persistent antigen exposure leads to sustained PD-1 overexpression and impaired immune control, making PD-1/PD-L1 blockade a potential therapeutic strategy. While previous studies have focused on changes in HBV DNA and HBsAg during PD-1 therapy, little is known about its influence on HBsAb production. This report describes two anti-HBc-positive cancer patients who developed elevated HBsAb after PD-1 inhibitor treatment, along with associated liver enzyme abnormalities.

Materials and Methods:

We retrospectively evaluated two male cancer patients with isolated anti-HBc positivity and negative HBsAg who received PD-1 inhibitors (pembrolizumab or sintilimab). Both developed immune-related liver injury after receiving PD-1 inhibitors, with significant elevations in ALT, AST, bilirubin, ALP, and GGT. Clinical manifestations, liver biochemical markers, HBV serology, and HBV DNA were collected before and after PD-1 inhibitor therapy.

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Results:

Each patient demonstrated a pronounced increase in HBsAb titers, rising from <2.00 and 4.91 mIU/mL at baseline to 303.65 mIU/mL and 405.06 mIU/mL, respectively. Throughout the clinical course and follow-up, HBsAg and HBV DNA remained negative. Liver function progressively improved after immunosuppressive therapy and IVIG administration. No virological rebound or new-onset HBV markers were detected during follow-up.

Conclusions:

PD-1 inhibition was associated with significant HBsAb elevation in two anti-HBc-positive cancer patients. This finding suggests that PD-1 blockade may partially restore exhausted HBV-specific B cells and T cells, reactivating immune memory and enhancing antiviral humoral responses. This immunologic effect provides early clinical observations regarding the impact of checkpoint inhibitors on HBV-related immune responses. The accompanying immune-related hepatic injury highlights the importance of careful monitoring. Larger studies are needed to clarify the clinical significance of this serologic change.

Keyword(s) : PD-1 inhibitor treatment, Hepatitis B virus, HBsAb increase, anti-HBc-positive patients

1610 - STING Aggravates Liver Fibrosis in Chronic hepatitis B concurrent with Metabolic Dysfunction Associated Steatotic Liver Disease by Modulating Gut Microbiota and Inducing Macrophage Derived Lysozyme Expression

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Background / Hypothesis:

The coexistence of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) in individuals with Chronic Hepatitis B (CHB) has been increasing annually. In the early stage of MASLD, high-fat diet-induced impairment of the intestinal epithelial barrier may activate the cyclic GMP-AMP synthase (cGAS)-stimulator of interferon genes (STING) pathway. Interactions along the gut-liver axis are likely to contribute to adverse outcomes in this comorbidity.

We hypothesize that STING activation during the early stage of the concurrent presence of CHB and MASLD exacerbates liver disease progression by altering the gut microenvironment.

Materials and Methods:

A mouse model of CHB concurrent with MASLD was established by hydrodynamic injection of pAAV/HBV1.2 combined with feeding a high-fat diet (HFD). The study utilized STING knockout mice (Sting^{-/-}), alongside their littermate controls. In vitro, THP-1 cells were administered a STING agonist, whereas LX-2 cells were subjected to lysozyme stimulation and appropriate controls were established for all treatments. The evaluation of STING and lysozyme-associated markers was conducted using Western blotting, immunofluorescence and enzyme-linked immunosorbent assay (ELISA).

Results:

At 8 weeks, CHB concurrent with MASLD mice exhibited moderate hepatosteatosis and lobular inflammatory infiltration compared to those in the CHB group. Compared to mice with CHB or MASLD alone, the comorbidity group exhibited more severe gut barrier disruption and altered microbiota composition. Specifically, the comorbid mice showed increased abundances of *Blautia*, *Erysipelatoclostridium*, and *Pseudomonas*, and decreased abundances of *Alloprevotella* and *Lachnospiraceae_UCG_001* relative to the CHB group. STING expression and activation were elevated in intestinal tissue. STING knockout restored gut barrier integrity and microbiota balance. Fecal microbiota transplantation from Sting^{-/-} mice alleviated steatosis and reduced ALT, AST, and free fatty acid levels. STING was highly enriched in intestinal macrophages, and its activation promoted lysozyme expression. Elevated serum lysozyme and increased CD64/lysozyme co-localization were observed in the comorbidity model. In vitro, lysozyme stimulated LX-2 activation, upregulating collagen I and fibronectin.

Conclusions:

Our study demonstrates that intestinal macrophage STING activation modulates gut microbiota and promotes liver fibrosis via lysozyme-induced hepatic stellate cell activation in the early stage of the concurrent CHB and MASLD model. Consequently, lysozyme may represent a potential therapeutic target for mitigating fibrosis in CHB patients concurrent with MASLD.



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Keyword(s) : CHB, MASLD, Gut microenvironment, STING, Lysozyme

1611 - Irreversible Electroporation (NanoKnife) in the Management of Refractory Recurrent Hepatocellular Carcinoma A Systematic Review of Diagnostic and Therapeutic Advances

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Background / Hypothesis:

This review focuses on recent advances and provides a comprehensive overview of the multifaceted research progress of nanoknife technology in the treatment of refractory recurrent hepatocellular carcinoma. Firstly, the pathological mechanisms of refractory recurrent hepatocellular carcinoma, including the risk factors for different types of recurrence, are described. Then, the relevant principles of nanoknife technology, such as the molecular basis of irreversible electroporation mechanism and the development history of this technology, are introduced. Subsequently, its current status in minimally invasive diagnosis and treatment, comparison with other therapeutic strategies, and evaluation of efficacy are discussed from the perspective of clinical application.

Materials and Methods:

We define recurrent refractory hepatocellular carcinoma as the presence of multiple recurrent liver lesions post-surgery (≥ 3 lesions), involvement of major hepatic vessels by recurrent lesions (e.g. invasion of hepatic veins or portal veins, or formation of intravascular tumour thrombi), extrahepatic spread and distant metastases (e.g. lung, bone or brain metastases), multiple intraperitoneal lymph node metastases, invasion of adjacent liver tissues or organs, and peritoneal seeding metastasis. By retrieving and screening relevant medical reports from PubMed (<https://pubmed.ncbi.nlm.nih.gov>) over the past decade, regardless of content, we provide this paper with theoretical and practical support based on the data therein.

Results:

It also covers technological advances and innovations, epidemiological studies, controversies and challenges, and looks forward to potential future breakthroughs in the treatment of this disease, the direction of research on the mechanism of irreversible electroporation, and the prospects for personalized treatment applications, aiming to provide a comprehensive reference for further research and application of nanoknife technology in the treatment of refractory recurrent liver cancer.

Conclusions:

In summary, nanoknife as an innovative non-thermal ablation technology has demonstrated broad application prospects in the diagnosis and treatment of refractory recurrent liver cancer. Its advantages in treating tumors located in complex anatomical positions, along with enhanced efficacy when combined with immunotherapy, gene therapy, and personalized treatment, make it a promising therapeutic option. Future researches should continue to explore these combined strategies to enhance the clinical efficacy of nanoknife technology, and, further large-scale clinical studies remain necessary to verify its long-term efficacy and safety, as well as to optimize clinical application strategies for liver cancer patients.

Keyword(s) : irreversible electroporation, nanoknife, minimally invasive diagnostic and therapeutic techniques, hepatocellular carcinoma, tumor microenvironment

1612 - Kuan Sin Yin potentiates PD1 blockade in hepatocellular carcinoma by enhancing B cell derived Granzyme B mediated apoptosis

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Background / Hypothesis: The Chinese herbal formula Kuan-Sin-Yin (KSY) has unexplored potential in hepatocellular carcinoma (HCC) therapy. Given its lack of direct anti-proliferative effect on HCC cells in vitro, we hypothesized that KSY exerts its anti-tumor activity by modulating the tumor immune microenvironment, particularly in combination with immune checkpoint inhibitors.

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Materials and Methods:HCC cell viability was assessed using the CCK-8 assay. An immune cell-HCC organoid co-culture system and a subcutaneous HCC model in C57BL/6 mice were established to evaluate the anti-tumor efficacy of KSY alone and in combination with an anti-PD1 antibody. Tumor-infiltrating immune cells were analyzed by flow cytometry. UPLC-HRMS was employed to characterize KSY components, and a multi-database analysis (SwissTargetPrediction, TCGA, Genecards, OMIM) was conducted to identify potential targets. GO and KEGG pathway enrichment analyses were performed. Underlying mechanisms were validated using TUNEL staining, Western blotting, immunofluorescence, and neutralizing antibody experiments.

Results:Medium/high doses of KSY significantly inhibited HCC growth in both the immune-co-culture organoid model and in vivo, and synergistically enhanced the efficacy of anti-PD1 therapy. Flow cytometry revealed that KSY promoted the infiltration of various immune cells, including macrophages, B cells, NK cells, and CD8+ T cells, into tumors. Integrated target gene sets were significantly enriched in apoptosis and the PI3K-AKT signaling pathway. Consistently, TUNEL staining and Western blot confirmed that KSY induced tumor cell apoptosis. While KSY broadly modulated the immune landscape, its combination with anti-PD1 specifically and further enhanced B cell infiltration. The supernatant from KSY-primed B cells induced apoptosis and suppressed AKT phosphorylation in HCC cells. This pro-apoptotic effect was specifically abolished by a Granzyme B neutralizing antibody, but not by antibodies against TNF- α or TRAIL. Immunofluorescence further demonstrated increased B cell infiltration accompanied by elevated Granzyme B levels in tumors from the combination therapy group.

Conclusions:This study demonstrates that KSY augments the anti-tumor efficacy of PD1 blockade in HCC by remodeling the tumor immune microenvironment. A key mechanism involves the activation of B cells, which secrete Granzyme B to induce tumor cell apoptosis via inhibition of the PI3K-AKT pathway. These findings provide a compelling rationale for developing KSY as an adjunctive therapy to immune checkpoint blockade in HCC.

Keyword(s) : Hepatocellular carcinoma, Kuan-Sin-Yin, PD-1 blockade, B cells, Granzyme B, Tumor microenvironment, Apoptosis

1613 - History of Abdominal Surgery and of Treatment for Esophagogastric Varices Are Risk Factors for Portal Vein Thrombosis in Patients with Cirrhosis

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Background / Hypothesis:To analyze the factors influencing the development of portal vein thrombosis in patients with cirrhosis.

Materials and Methods:A retrospective study was conducted to collect clinical data of 319 hospitalized patients with cirrhosis admitted to Xinjiang Uygur Autonomous Region Hospital of Traditional Chinese Medicine from January 2021 to December 2024. Demographic information, serological indicators, abdominal ultrasound, portal vein ultrasound, and other relevant clinical data were collected for all patients. CTP scores were calculated for each patient. Patients were then divided into two groups: the portal vein thrombosis group and the non-portal vein thrombosis group. Multivariate logistic regression analysis was further performed to identify factors influencing the development of PVT in patients with cirrhosis.

Results:A total of 61 patients (19.1%) had liver cirrhosis complicated with portal vein thrombosis. Stratified by CTP score: 8 cases (7.3%) were in CTP class A, 36 cases (25.7%) in CTP class B, and 17 cases (24.2%) in CTP class C; the distribution of etiologies (chronic hepatitis B, autoimmune liver disease, and other causes) showed statistically significant differences ($P < 0.05$). Between the two groups, significant differences were observed in spleen thickness, splenic length, portal vein flow velocity, history of abdominal surgery, history of esophagogastric variceal treatment (including endoscopic ligation and sclerotherapy), and severe gastric varices (all $P < 0.05$). For serological parameters, hemoglobin levels, albumin levels, and international normalized ratio (INR) differed significantly between the two groups (all $P < 0.05$). Multivariate logistic regression analysis identified three independent risk factors for PVT in patients with liver cirrhosis: History of

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abdominal surgery (OR=11.986, 95% CI: 1.088-132.010, $P<0.001$), History of esophagogastric variceal treatment (OR=4.229, 95% CI: 2.085-8.577, $P<0.001$), Spleen thickness (OR=1.048, 95% CI: 1.011-1.086, $P=0.011$) were independent risk factors for portal vein thrombosis in patients with cirrhosis.

Conclusions:History of abdominal surgery, history of esophagogastric variceal treatment, and spleen thickness are risk factors for portal vein thrombosis in patients with liver cirrhosis.

Keyword(s) :

1614 - Autoimmune hemolytic anemia complicated by portal vein thrombosis in a pediatric liver transplant recipient

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Background / Hypothesis:Autoimmune hemolytic anemia (AIHA) is a rare but serious complication following pediatric liver transplantation, with reported incidence of 1-3%. Immune dysregulation under chronic immunosuppression is the proposed mechanism. Thrombotic complications associated with post-transplant AIHA, including portal vein thrombosis (PVT), are infrequently reported but represent life-threatening events requiring urgent intervention. This report presents a case demonstrating this severe complication and its successful management.

Materials and Methods:Among 165 pediatric liver transplant recipients at Acibadem Atakent Hospital (2016-2025), three developed AIHA post-transplant (incidence 1.8%). This case report presents the index case complicated by portal vein thrombosis. Analysis included clinical course, immunosuppression protocols, laboratory parameters including hemolytic markers and coagulation studies, imaging findings, therapeutic interventions, and outcomes.

Results:A 10-month-old girl underwent living donor liver transplantation for biliary atresia. Post-operatively,

the patient was maintained on tacrolimus and mycophenolate mofetil following initial steroid taper. Five months post-transplant, she presented with fever and jaundice. Laboratory evaluation revealed severe hemolytic anemia (hemoglobin 7.5 g/dL, hematocrit 6.9%), marked elevation of lactate dehydrogenase (1751 IU/L), hyperbilirubinemia (total bilirubin 4.92 mg/dL), and thrombocytopenia (platelets $75 \times 10^3/\mu\text{L}$). Direct antiglobulin test was positive, confirming AIHA. Coagulation studies demonstrated significant abnormalities with low protein S activity (48.1%) and elevated D-dimer (3.78 mg/L), indicating hypercoagulable state. Doppler ultrasonography identified partial portal vein thrombosis, a rare and serious complication. Treatment consisted of high-dose methylprednisolone (4 mg/kg) and rituximab for AIHA management. Concurrent intravenous heparin anticoagulation was initiated for PVT. The thrombosis resolved completely within five days. Hemolytic parameters gradually normalized, with eventual negative direct antiglobulin test confirming AIHA remission.

Conclusions:Post-transplant AIHA can precipitate life-threatening thrombotic complications including portal vein thrombosis through mechanisms involving severe hemolysis, thrombocytopenia, and acquired hypercoagulability. This case demonstrates the critical importance of comprehensive evaluation including coagulation studies and vascular imaging in AIHA patients. Successful management requires aggressive multimodal therapy combining immunosuppression with prompt anticoagulation. Clinicians managing pediatric liver transplant recipients should maintain vigilance for AIHA and systematically assess for thrombotic complications in this vulnerable population.

Keyword(s) : autoimmune hemolytic anemia, liver transplantation, pediatric, portal vein thrombosis, rituximab

1616 - SNX10 Promotes Lipid Accumulation in Hepatocytes by Inhibiting Autophagic Flux

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Background / Hypothesis:

Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) is a highly prevalent condition with limited therapeutic options. Autophagy, a cellular catabolic process that clears excess lipids, is a critical protective mechanism against hepatic steatosis. Sorting Nexin 10 (SNX10), a member of the endosome-associated SNX protein family, is implicated in regulating the autophagy-lysosome pathway. However, its specific role in the pathogenesis of MAFLD is unknown. This study aimed to elucidate the function of SNX10 in hepatic lipid metabolism and its underlying autophagic mechanism.

Materials and Methods:

An in vitro model of hepatic steatosis was established by treating human LO2 hepatocytes with free fatty acids (FFAs). Gain- and loss-of-function studies were performed using lentiviral-mediated overexpression and siRNA-mediated knockdown of SNX10, respectively. Lipid accumulation was quantified by Oil Red O staining. The expression of SNX10 and key autophagy-related genes (e.g., Atg5, Atg7) and proteins (e.g., LC3, SQSTM1/p62) was analyzed by qPCR and Western blotting. A protein-protein interaction network was generated using the STRING database to explore potential mechanistic links.

Results:

FFA treatment of LO2 cells induced a steatotic phenotype, characterized by increased lipid deposition and a corresponding upregulation of SNX10 at both the mRNA and protein levels. Lentiviral-mediated overexpression of SNX10 was sufficient to significantly increase intracellular lipid deposition. Mechanistically, SNX10 overexpression led to a marked accumulation of the autophagosome marker LC3-II, indicating an impairment of autophagic flux, without significantly altering levels of the autophagy substrate SQSTM1/p62. Conversely, siRNA-mediated knockdown of SNX10 significantly ameliorated lipid accumulation both at baseline and under FFA-induced stress. This protective effect was associated with restored autophagic activity, as evidenced by a significant upregulation of Atg5 expression and a reduction in LC3-II levels. STRING analysis further supported a functional interaction between SNX10 and the core autophagy machinery, including Atg5.

Conclusions:

Our findings demonstrate that SNX10 is a negative regulator of autophagy in hepatocytes. Elevated SNX10 expression, as observed in a cellular model of MAFLD,

promotes lipid droplet accumulation by inhibiting autophagic flux. This study identifies SNX10 as a novel contributor to hepatic steatosis and suggests that targeting SNX10 could be a potential therapeutic strategy to restore autophagy and alleviate lipid burden in MAFLD.

Keyword(s) : MAFLD, SNX10, Autophagy, lipid deposition

1617 - Impact of post Kasai steroid based protocol in Biliary Atresia Real World Data From India

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Background / Hypothesis:

This study aims to assess whether steroid-based regimen enhances the clearance of jaundice and improves native liver survival (NLS).

Materials and Methods:

We included all infants with biliary atresia (BA) undergoing Kasai portoenterostomy (KPE) at our centre. The algorithm-initiated prednisolone was initiated if stools remained acholic.

Results:

Of 127 infants, 70 formed the Steroid Group (SG) and 57 the Historical Cohort (HC). Jaundice clearance at 6 months was superior with steroids (61.4% vs 38.6%; OR 2.53, 95% CI 1.23-5.19, P=0.013). Kaplan-Meier analysis demonstrated improved 24-month NLS (log-rank P=0.044) in SG. Multivariate analysis identified younger age at KPE (aOR 1.021 per day, 95% CI 1.004-1.038, P=0.016), lower pre-operative bilirubin (aOR 1.163, 95% CI 1.024-1.320, P=0.020) and steroid use (aOR 2.13, 95% CI 1.001-4.53, P=0.050) as independent predictors of success.

Conclusions:

Moderate-dose prednisolone after KPE enhances early bile drainage and prolongs native-liver survival. Optimal benefit is achieved when surgery is performed

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at ≤ 70 days of age.

Keyword(s) : Cholangitis; Prednisolone; Jaundice clearance; Native liver survival; Portal hypertension

1618 - Optimizing Nucleos(t)ide Analogs Combined Peg IFN α Regimen for CHB with Low Level Viremia Efficacy and Predictors

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Background / Hypothesis:

The optimization of therapy in chronic hepatitis B with low-level viremia represents a current research hotspot. To compare the efficacy of nucleos(t)ide analogs (NAs) combined with pegylated interferon- α (Peg-IFN α) versus NAs alone in optimizing treatment for NAs-experienced chronic hepatitis B (CHB) patients with low-level viremia, and to analyze influencing factors.

Materials and Methods:

A prospective real-world multicenter study enrolled 362 CHB patients with low-level viremia at Beijing Ditan Hospital Liver Disease Center, Beijing YouAn Hospital and the Fifth Medical Center of the Chinese PLA General Hospital from January 2022 to December 2024. Patients were divided into two groups based on preference: the NAs group and the NAs+PEG-IFN group. Primary endpoints included complete virological response (CVR), HBsAg clearance rate, and HBeAg seroconversion rate at 48 weeks. Secondary endpoints were reductions in HBV DNA and HBsAg levels from baseline.

ResAt 48 weeks, the NAs+PEG-IFN group showed significantly higher CVR and HBsAg clearance rates compared to the NAs group (26.84% vs. 15.56%, $\chi^2=4.72$, $P=0.030$; 15.44% vs. 2.22%, $\chi^2=8.35$, $P=0.004$). HBeAg seroconversion was higher in the NAs+PEG-IFN group (69.177% vs. 55.56%), but the difference lacked statistical significance ($\chi^2=3.48$, $P=0.062$). The NAs+PEG-IFN group achieved greater HBsAg reduction ($-0.65 \log_{10}$ IU/mL vs. $-0.28 \log_{10}$ IU/mL, $Z=-2.45$, $P<0.05$) and HBV DNA reduction ($-0.70 \log_{10}$

IU/mL vs. $-0.53 \log_{10}$ IU/mL, $Z=-2.15$, $P<0.05$). Baseline low HBsAg and rapid HBsAg decline at 12 weeks were independent predictors of HBsAg clearance (OR=0.52 and OR=2.50, $P<0.05$). ults:

Conclusions:

Compared to NAs optimization, the NAs+PEG-IFN regimen significantly improved CVR and HBsAg clearance rates, and reduced HBsAg and HBV DNA levels in NAs-experienced CHB patients with low-level viremia.

Keyword(s) : Chronic hepatitis B, Low-level viremia, Pegylated interferon- α , Nucleos(t)ide analogs, Complete virological response, HBsAg clearance

1620 - Precision Treatment and Frontier Advances of Glycyrrhizin Preparations in Drug Induced Liver Injury

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Background / Hypothesis:Drug-induced liver injury (DILI) poses significant challenges in clinical practice. Glycyrrhizin preparations, derived from licorice (*Glycyrrhiza uralensis*), exhibit multi-target effects and are recommended as a core therapy in various guidelines. This study aims to summarize the evidence-based data, precision treatment strategies, and recent advances of glycyrrhizin preparations for DILI. The central hypothesis is that these preparations exert their therapeutic effects on DILI through multi-target mechanisms, including newly discovered pathways such as gut-liver axis modulation and epigenetic regulation.

Materials and Methods:We conducted a systematic review to comprehensively examine the pharmacological mechanisms, clinical evidence, and guideline recommendations for glycyrrhizin preparations. Furthermore, we specifically investigated precision use strategies based on DILI type, severity, and patient subgroups, and explored novel mechanisms of action, such as regulation of the gut-liver axis.

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Results:Glycyrrhizin preparations demonstrate proven efficacy in treating DILI and are widely endorsed by various guidelines. Their mechanisms of action involve multiple aspects, including the modulation of the gut-liver axis and epigenetic regulation. Management strategies for potential adverse reactions, such as pseudoaldosteronism, were also summarized.

Conclusions:Glycyrrhizin preparations represent a key therapeutic option for DILI. Further research is warranted to validate these novel mechanisms and to optimize their precise clinical application.

Keyword(s) : Glycyrrhizin preparations, Drug-induced liver injury(DILI), Precision treatment, Evidence-based medicine, Gut-liver axis

1621 - Establishment and assessment of a nomogram for predicting hepatic parasites disease

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Background / Hypothesis:

Hepatic parasite disease (HPD) is a widely endemic helminthic disease. Diagnosis involves serum serologic testing for antibodies against hydatid antigens, preferably with imaging by ultrasound or computed tomography (CT) and magnetic resonance imaging (MRI). Although the sensitivity of ultrasound diagnosis reaches 89% and CT yields positive results in 93% of cases, some people still have an unclear imaging diagnosis. Pathology is also an important way of diagnosis. However, inappropriate needle aspiration or open biopsy could lead to spillage of the cyst contents and catastrophic dissemination of the parasites. Therefore, there is an urgent need for an efficient and non-invasive diagnostic method in the clinic. The aim of this study was to combine multiple clinical features into a nomogram for predicting the individual risk of hepatic parasites disease.

Materials and Methods:

216 patients with hepatic space-occupying disease were collected. Patient characteristics and 8 serum markers were subjected to univariate and multivariate analysis, the results of which were used to construct

the nomogram.

Results:

Eosinophil count, typical clinical symptoms, age, and alpha-fetoprotein were significantly significant between hepatic hemangioma and the other hepatic space-occupying diseases. The area under the receiver operating characteristic curve was 0.842 (95% CI: 0.770~0.915) for the training set, and the area under the receiver operating characteristic curve was 0.756 (95% CI: 0.613~0.889) for the validation set, which shows that the model has good discrimination. The use of the nomogram was better than that of a single marker. Furthermore, the nomogram showed better discriminative ability than the biomarker alone. Notably, the validation indicated high stability and reproducibility.

Conclusions:Use of the nomogram is superior to that of a serum marker alone and holds promise in clinical application.

Keyword(s) : Hepatic parasitic disease, Eosinophil count, Typical symptoms, Age, Alpha-fetoprotein, Nomogram

1622 - Impact of Tuberculosis Co-infection on All-Cause Mortality in Patients with Viral Hepatitis A Propensity Score-Matched Retrospective Study

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Background / Hypothesis:

Viral hepatitis and tuberculosis (TB) frequently coexist and may interact to worsen clinical outcomes. We examined the association between TB co-infection and short-term and long-term all-cause mortality among patients with viral hepatitis using a large intensive-care cohort.

Materials and Methods:

Retrospective cohort study using the MIMIC-IV database. A total of 11,777 patients with viral hepatitis were identified (TB group = 1,228; Non-TB group = 10,549). Propensity score matching (PSM) at a 1:3 ratio yielded 4,253 patients (TB group = 1,193; Non-TB group = 3,060). Outcomes were all-cause mortality at 30, 90, 180 and 360 days. Survival was assessed by Kaplan-Meier analysis and adjusted using three Cox proportional hazards models.

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Results:

Before and after PSM, mortality was significantly higher in the TB group at 180 days and 360 days ($p < 0.01$), whereas no significant differences were observed at 30 or 90 days. Kaplan–Meier curves confirmed lower survival in the TB group at 180- and 360-day intervals ($p < 0.05$). After PSM, the adjusted hazard ratios (95% CI) for 360-days mortality in the TB group were 1.385 (1.190–1.612), 1.375 (1.181–1.600) and 1.579 (1.348–1.850) across Models I–III, respectively (all $p < 0.05$). For 180-day mortality, adjusted HRs were 1.323 (1.107–1.580), 1.313 (1.099–1.569) and 1.556 (1.292–1.857) in Models I–III (all $p < 0.05$). Subgroup analyses showed an overall increased mortality associated with TB co-infection (HR 1.38; 95% CI 1.19–1.61; $p < 0.001$), with significant interactions for chronic kidney disease (p for interaction = 0.002), COPD ($p < 0.001$), pneumonia ($p = 0.028$) and diabetes ($p = 0.015$), indicating effect modification by these comorbidities.

Conclusions: In this large matched cohort of patients with viral hepatitis, TB co-infection was associated with significantly increased long-term (180- and 360-day) all-cause mortality but not with short-term (30- or 90-day) mortality. The association was modified by several comorbid conditions, underscoring the need for targeted management and further investigation into mechanisms and therapeutic strategies for high-risk subgroups.

Keyword(s) : Viral hepatitis; Tuberculosis; Co-infection; Mortality; Propensity score matching

1623 - Molecular Association of Circadian Rhythm and Tryptophan Metabolism Pathway in the Pathogenesis of Chronic Hepatitis B

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Background / Hypothesis: Chronic Hepatitis B (CHB) remains a significant global health burden, yet the intricate molecular mechanisms driving its progression are not fully elucidated. This study aimed to investigate the underlying molecular connections between circadian rhythm signaling and metabolic pathways in the pathogenesis of CHB.

Materials and Methods: We retrieved transcriptome data from peripheral blood mononuclear cells (PBMCs) of CHB patients from the GEO public database. Differential Expression Genes (DEGs) were identified by integrating the data with established circadian rhythm

gene sets. Subsequently, a robust pipeline combining three machine learning algorithms—LASSO regression, Support Vector Machine (SVM), and XGBoost—was employed to confidently screen for core key genes associated with CHB. Gene Set Enrichment Analysis (GSEA) was performed to dissect the functional pathways enriched by these key genes. Furthermore, we utilized publicly available microbiome data from CHB patients to conduct functional enrichment analysis on differential gut microbiota, establishing a potential host-microbiota-metabolism axis.

Results: The machine learning screening strategy successfully identified four core genes—HTR7, TH, PPP1CB, and SMARCD3—strongly associated with CHB. GSEA results indicated that the expression of HTR7 was significantly enriched in pathways related to Hepatitis B, circadian rhythm regulation, and tryptophan metabolism. Analysis of the gut microbiota in CHB patients further revealed that differentially abundant bacteria, including *Pseudomonas*, *Clostridium*, and *Bacteroides*, were also highly enriched in the tryptophan metabolic pathway, specifically participating in the Kynurenine (Kyn) pathway.

Conclusions: This study provides evidence revealing a critical molecular link between circadian rhythm dysregulation and the tryptophan-kynurenine metabolic axis in CHB patients. The core genes HTR7, TH, PPP1CB, and SMARCD3 may function as host regulatory factors, driving CHB progression by modulating these pathways. These findings offer new insights into the potential pathophysiological mechanisms of CHB and highlight the potential of developing novel diagnostic biomarkers and therapeutic strategies targeting the circadian rhythm-metabolic axis.

Keyword(s) : Chronic Hepatitis B (CHB), Circadian Rhythm, Tryptophan Metabolism, Kynurenine Pathway, HTR7

1626 - Baseline Gut Microbiota Signatures Associated with Interferon Response and Functional Cure in Chronic Hepatitis B

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Background / Hypothesis: Chronic hepatitis B (CHB) remains a major public health challenge, with

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functional cure (hepatitis B surface antigen [HBsAg] loss) rarely achieved by current therapies. The gut–liver axis has been implicated in antiviral immunity, but it remains unclear whether baseline gut microbiota composition influences interferon (IFN) treatment response or functional cure outcomes. We aimed to identify baseline gut microbiota signatures associated with IFN response and HBsAg loss in CHB.

Materials and Methods: We enrolled adult CHB patients initiating pegylated IFN α -2b therapy (n=102), along with healthy controls (HC, n=28). Baseline stool samples underwent shotgun metagenomic sequencing. We first compared alpha- and beta-diversity between CHB and HC. Among IFN-treated patients, we then performed two subgroup analyses: (i) a virological response analysis comparing responders (R, n=42) and non-responders (NR, n=60); and (ii) a functional cure analysis comparing patients who achieved HBsAg loss (FC, n=12) and those who did not (non-FC, n=90). Differentially abundant genera were identified using Wilcoxon rank-sum tests and LEfSe. Additionally, trends were analyzed along a four-level “clinical remission axis” (NR $\rightarrow$ R without FC $\rightarrow$ FC $\rightarrow$ HC).

Results: Baseline overall diversity did not differ significantly between CHB and HC groups, although CHB showed a modest enrichment of certain bile acid-related and potentially pro-inflammatory genera (e.g., *Escherichia*). Similarly, within IFN-treated patients, alpha- and beta-diversity were comparable between responders and non-responders. However, several short-chain fatty acid (SCFA)-producing genera (e.g., *Roseburia*, *Lacrimispora*) were more abundant in R, whereas the dysbiosis-associated genus *Megamonas* was enriched in NR. In the functional cure analysis, FC patients had higher baseline abundances of SCFA-producing genera, while non-FC patients were enriched in genera associated with dysbiosis or inflammation. Along the clinical remission axis (NR $\rightarrow$ R without FC $\rightarrow$ FC $\rightarrow$ HC), SCFA-producing commensals increased stepwise in relative abundance, whereas potentially pathogenic or dysbiosis-related taxa (e.g., *Escherichia/Shigella*, *Megamonas*) progressively decreased.

Conclusions: Baseline gut microbiota diversity alone was not strongly associated with IFN response, but specific compositional and functional profiles correlated with treatment outcomes. An SCFA-rich, low-inflammatory microbiota — characterized by enrichment of *Roseburia*, *Lacrimispora* and related genera and depletion of *Megamonas* and other

opportunistic taxa — was linked to better IFN response, a higher likelihood of functional cure, and a microbiota profile resembling healthy controls. These findings highlight the gut microbiota as a potentially modifiable determinant of treatment outcomes in CHB and support microbiota-targeted strategies to improve IFN therapy outcomes.

Keyword(s) : chronic hepatitis B, pegylated interferon, gut–liver axis, gut microbiota, functional cure, HBsAg loss

1627 - Clinical and Serological Predictors of Virological Response to Nucleos(t)ide Analogue Therapy in Chronic Hepatitis B A 7 Year Cohort Study

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Background / Hypothesis:

Chronic hepatitis B remains a major global health concern, associated with increased risk of progression to cirrhosis and hepatocellular carcinoma (HCC). Antiviral therapy with nucleos(t)ide analogues aims to suppress viral replication, prevent disease progression, and improve survival. Identifying factors that predict favorable virological response could help optimize patient management. This study aimed to evaluate clinical, virological, and biological characteristics associated with treatment response in patients with chronic hepatitis B receiving nucleos(t)ide analogue therapy.

Materials and Methods:

We conducted a retrospective, descriptive, and analytical study over a 7-year period (2018–2025), including all patients with chronic hepatitis B treated with nucleos(t)ide analogues. Patients under surveillance only or lost to follow-up were excluded. Collected data included demographic characteristics (age, sex), comorbidities (diabetes, hepatic steatosis, immunosuppression), viral coinfections (HCV, HDV), family history of cirrhosis or HCC, serological markers (HBsAg, anti-HBs, HBeAg, anti-HBe), baseline viral load, ALT levels, and fibrosis stage (F0–F2 vs. F3–F4). The primary outcome was sustained virological response (SVR), defined as viral load <2,000 IU/mL after treatment. Secondary outcome was complete virological response (CVR), defined as SVR with HBsAg

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loss. Statistical analyses included univariate analysis (Chi-square for qualitative variables, Mann–Whitney for quantitative variables) and multivariate logistic regression.

Results:

Eighty-one patients were included (45 men, 36 women; male-to-female ratio 1.25). Mean age was 47.5 years (range: 23–82). Comorbidities included diabetes in 12.3% (n=10), hepatic steatosis in 6.2% (n=5), HCV coinfection in 3.7% (n=3), HDV coinfection in 2.5% (n=2), and immunosuppression in 13.6% (n=11). Family history of cirrhosis or HCC was reported in 12.3% (n=10).

Treatment responses:

- SVR was achieved in 92.6% (n=75).
- CVR occurred in 7.4% (n=6).

Univariate analysis identified elevated ALT ($p=0.0029$), anti-HBe positivity ($p=0.0029$), HBeAg positivity ($p=0.0030$), and anti-HBs positivity ($p=0.0041$) as significantly associated with SVR. Age, diabetes, hepatic steatosis, fibrosis stage, and coinfections were not significantly associated with SVR. Analyses of CVR and serological response showed no statistically significant associations.

Multivariate analysis did not identify independent predictors of SVR, likely due to collinearity and the limited number of non-SVR events.

Conclusions:

Serological markers (anti-HBe, HBeAg, anti-HBs) and elevated ALT were associated with virological response, while metabolic and viral comorbidities had no impact. Complete virological response was rare. These findings support the value of baseline serological and biochemical markers in predicting antiviral response in chronic hepatitis B.

Keyword(s) : Chronic hepatitis B – Nucleos(t)ide analogues – Virological response – Predictive factors

1628 - A Prospective Study on a Group Mindfulness Based Intervention for Patients with Metabolic Dysfunction Associated Fatty Liver Disease Effects on Liver Steatosis and Psychological Symptoms

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Background / Hypothesis:

The prevalence of Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) is a growing global health concern. MAFLD is frequently comorbid with psychological distress; for instance, our previous research utilizing the UK Biobank cohort demonstrated a significant incidence of new-onset major depressive disorder among MAFLD patients. Mindfulness-Based Cognitive Therapy (MBCT), a structured group psychological intervention, is effective for treating depression and anxiety. However, its therapeutic potential in the MAFLD population, whose treatment has traditionally focused on pharmacologic and lifestyle interventions, remains unexplored. This study aimed to investigate the effects of a group MBCT intervention on liver steatosis and psychological symptoms in patients with MAFLD.

Materials and Methods:

This prospective, single-arm study recruited 34 patients diagnosed with MAFLD (Controlled Attenuation Parameter [CAP] > 238 dB/m, with comorbid obesity or dyslipidemia) from a hepatology outpatient clinic. Participants underwent an 8-week group MBCT program while continuing standard medical care. Liver steatosis (CAP value via transient elastography), depression (HAMD-17), anxiety (HAMA), sleep quality (PSQI), life satisfaction (SWLS), and mindfulness (MAAS) were assessed at baseline and post-intervention. Paired-sample t-tests were used to compare pre- and post-intervention outcomes. (Ethics Approval No: ZHSYKY2025042706; Clinical Trial Registration No: MR-44-25-077097).

Results:

At baseline, a high prevalence of psychological distress was observed, with 70.6% of participants reporting depressive symptoms and 64.7% reporting anxiety symptoms. Following the 8-week intervention, participants showed a statistically significant reduction in mean scores for depression (HAMD-17, $P = 0.048$) and anxiety (HAMA, $P = 0.014$). Positive, though not statistically significant, trends were observed for improvements in liver steatosis (mean CAP), Body Mass Index (BMI), sleep quality (PSQI), life satisfaction (SWLS), and mindfulness levels (MAAS) (all $P > 0.05$). Details are presented in Table 1 and Figure 1.

Conclusions:



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This study, the first of its kind in China, demonstrates that an 8-week group MBCT intervention can significantly alleviate depressive and anxiety symptoms in patients with MAFLD. While improvements in liver steatosis and other metabolic markers did not reach statistical significance, likely due to the small sample size and short study duration, the positive trends are encouraging. These findings provide a strong rationale and preliminary evidence for conducting larger, randomized controlled trials to validate the benefits of MBCT on both psychological and physiological outcomes in the MAFLD population. This research pioneers the integration of psychological care into the holistic management of MAFLD, suggesting a promising new adjunctive therapeutic avenue.

Keyword(s) : MAFLD MBCT

1630 - The effect of hepatic steatosis on the efficacy of immune based integrative treatment for viral HCC

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Background / Hypothesis:

Studies have shown that immunotherapy combined with other therapies have positive clinical efficacy. However, there are also findings showing that HCC caused by NASH may not respond well to immunotherapy. Most HCC patients in Asian countries are caused by hepatitis virus infection, but with the improvement of life quality, more patients are also combined with varying degrees of hepatic steatosis. So it is significant to find out how patients with viral-HCC as well as liver steatosis respond to immunotherapy. Since immunotherapy alone is rarely used in the clinical treatment of advanced liver cancer, this study explored the effect of hepatic steatosis on the efficacy of immunotherapy-based comprehensive treatment for HCC caused by hepatitis virus, hoping to provide reference for individualized treatment of HCC.

Materials and Methods:

Clinical data, treatment strategies and follow-up data of 213 viral-HCC patients who were not suitable for direct surgical treatment from 2019.01 to 2023.12 in Nanjing Drum Tower Hospital were collected. Their efficacy was evaluated by mRECIST criteria. Patients were divided into four groups according to liver steatosis and whether they were treated with immunotherapy. The efficacy of each group was

compared. The independent risk factors affecting the efficacy were analyzed by univariate and multivariate Logistic regression. For follow-up data, Kaplan-Meier method was used to analyze the difference in survival rate. Cox regression analysis was performed to analyze the independent risk factors affecting the survival of patients.

Results:

1. For patients receiving immune combined therapy, the ORR(11.54%) and DCR(30.77%) in patients with hepatic steatosis were significantly lower than the ORR(41.18%) and DCR(69.93%) in patients without hepatic steatosis($P<0.05$). Hepatic steatosis, portal vein thrombosis and multiple tumors were independent risk factors for poor prognosis; 2. There was no significant difference between the efficacy of non-immune comprehensive treatment for liver steatosis patients and non-steatosis patients; 3. In patients with liver steatosis, the ORR(11.54%) and DCR(30.77%) in samples receiving immune combined therapy were significantly lower than the ORR(77.78%) and DCR(88.89%) in samples receiving non-immune therapy($P<0.05$), but Cox regression showed that the usage of immunotherapy was not an independent risk factor for patients' survival; 4. In patients without liver steatosis, there was no significant difference between the efficacy of immune combined therapy and non-immune combined therapy.

Conclusions: The efficacy of immune combined therapy in hepatic steatosis patients is worse than that in non-steatosis patients.

Keyword(s) : Hepatocellular carcinoma; Comprehensive treatment; Immunotherapy; Hepatic steatosis; Objective response rate; Disease control rate; Overall survival.

1631 - Noninvasive preoperative prediction of major pathological response in unresectable hepatocellular carcinoma treated with conversion therapy using a clinical CT radiomic model a multicenter retrospective study

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Background / Hypothesis: Accurate identification of major pathological response (MPR) following conversion therapy is critical for determining the optimal surgical strategy and guiding the extent of resection in hepatocellular carcinoma (HCC). However, a non-invasive method for reliably predicting MPR remains unavailable. This study aims to develop ensemble learning models that integrated clinical and computed tomography (CT) radiomic features to predict MPR in HCC patients.

Materials and Methods: This retrospective study included 266 confirmed HCC patients from 11 Chinese centers who underwent conversion therapy followed by hepatectomy. The patients were randomly allocated into a training set (n = 186) and a validation set (n = 80). Patient demographics were analyzed to construct a clinical model. Radiomic features were extracted from preoperative contrast-enhanced CT images (venous phase) for each patient. Feature selection was performed using analysis of variance and the least absolute shrinkage and selection operator algorithm. A radiomics score was computed based on a radiomic signature formula. Subsequently, a combined clinical-radiomic model was developed. The model's performance was assessed using the receiver operating characteristic (ROC) curve, calibration curve, and decision curve analysis (DCA).

Results: A total of 1130 radiomic features were

extracted from the CT images of each patient, among which 7 features were selected based on their nonzero coefficients associated with MPR. Three clinically relevant factors—downstaging after conversion therapy, α -fetoprotein response, and Barcelona Clinic Liver Cancer stage—were significantly correlated with MPR. Among the evaluated algorithms, LightGBM achieved the best performance as assessed by five-fold cross-validation on the training set. After integrating the optimal algorithm with clinical factors, the combined radiomic model yielded areas under the ROC curve of 0.900 (95% CI: 0.855–0.938) for the training set and 0.791 (95% CI: 0.689–0.886) for the validation set. DCA further confirmed the clinical utility of the integrated model.

Conclusions: This study successfully developed an integrated clinical-CT radiomic model for predicting MPR, which may assist in guiding surgical decision-making for HCC patients after conversion therapy.

Keyword(s) : hepatocellular carcinoma, conversion therapy, major pathological response, radiomic

1632 - Predictive Value of INR and DBIL for Short Term Prognosis after the First Artificial Liver Support System Treatment in Patients with Acute on Chronic Liver Failure

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Background / Hypothesis:

Acute-on-Chronic Liver Failure (ACLF) carries a poor prognosis, and the Artificial Liver Support System (ALSS) is a critical therapeutic modality. However, early identification of individuals at high risk for poor clinical outcomes following their first ALSS treatment remains a significant clinical challenge. This study aimed to investigate the predictive value of International Normalized Ratio (INR) and Direct Bilirubin (DBIL) for 28-day prognosis in ACLF patients undergoing initial ALSS therapy.

Materials and Methods:

This retrospective study enrolled 150 ACLF patients meeting inclusion criteria who received ALSS treatment at Lanzhou Second People's Hospital, Gansu Province,

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China, between 2017 and 2025. Patients were stratified into a good prognosis group (n=99) and a poor prognosis group (n=51) based on their 28-day outcomes. Baseline characteristics and laboratory parameters were collected and compared between the groups. Univariate and multivariate logistic regression analyses were employed to identify independent prognostic risk factors. The predictive performance of INR and DBIL was evaluated using Receiver Operating Characteristic (ROC) curve analysis.

Results:

- 1、Baseline Characteristics: The poor prognosis group exhibited a significantly higher incidence of infection compared to the good prognosis group (49.0% vs. 31.3%, $P=0.034$). For other baseline comparisons where no significant differences were found (Age, Sex, Etiology, Cirrhosis, Ascites).
- 2、Laboratory Parameters: Levels of DBIL, INR, and Neutrophil-to-Lymphocyte Ratio (NLR) were significantly higher in the poor prognosis group, while Lymphocyte count (LYM) was significantly lower (all $P<0.05$).
- 3、Multivariate Analysis: Multivariate logistic regression analysis confirmed that both INR (OR = 4.318, 95% CI: 1.891-9.858, $P=0.001$) and DBIL (OR = 1.005, 95% CI: 1.001-1.010, $P=0.016$) were independent risk factors for poor 28-day prognosis.
- 4、ROC Curve Analysis: ROC curve analysis demonstrated that the predictive performance of INR (AUC = 0.669, 95% CI: 0.577-0.761) was superior to that of DBIL (AUC = 0.604, 95% CI: 0.508-0.699). At its optimal cut-off value of 1.775, INR predicted poor prognosis with a sensitivity and specificity of 74.5% and 56.6%, respectively.

Conclusions:

INR and DBIL serve as independent risk factors predicting the 28-day prognosis following the first ALSS treatment in ACLF patients. Notably, INR exhibits superior predictive value. In clinical practice, monitoring pre-treatment INR levels, particularly focusing on the critical threshold of >1.775 , holds significant practical value for risk assessment and facilitating individualized treatment strategies.

Keyword(s) : Acute-on-Chronic Liver Failure; INR; DBIL; Prognosis

1633 - MVPS and Thrombophilic Abnormalities as Leading Causes of Non Tumoral Portal Vein Thrombosis Epidemiological and Evolutionary Insights from a Cohort of 83 Patients

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Background / Hypothesis:

Non-tumoral portal vein thrombosis (PVT) results from diverse mechanisms, in which porto-sinusoidal vascular disorders (PSVD/MVPS) and thrombophilic abnormalities play central roles. Their relative contributions and natural course remain incompletely characterized. This study aimed to describe the etiological distribution of non-tumoral PVT in a cohort of 83 patients, with a focus on MVPS and thrombophilic forms, and to analyze their epidemiological, radiological, and evolutionary characteristics.

Materials and Methods:

We retrospectively included 83 patients with imaging-confirmed non-tumoral PVT. Collected data included age, sex, etiology (MVPS, thrombophilia, cirrhosis, idiopathic), type and extension of thrombosis, and clinical evolution. Descriptive analyses were performed, with global comparisons across etiological groups.

Results:

Among 83 patients, main etiologies were MVPS (33; 39.8%), thrombophilia (32; 38.6%), idiopathic (15; 18.1%), and cirrhosis (3; 3.6%). Median age was 45 years, and 77% were women. PVT was total in 78% and partial in 20%. Extension involved the portal vein alone in 49.4%, splanchnic veins in 42.2%, and portal branches in 8.4%. Epidemiological and thrombosis profiles were largely similar between etiologies, although MVPS patients were slightly younger and showed somewhat more splanchnic extension. Clinical course was generally favorable: stable thrombosis in ~70%, cavernoma in 6%, progressive extension in 10%, regression/recanalization in 5%, and disease-related death in 10%. Overall prognosis was comparable across etiologies, though MVPS tended toward more cavernomas and extensions, and mortality was slightly higher in the thrombophilia group.

Conclusions:

In this cohort, MVPS and thrombophilic abnormalities were the leading causes of non-tumoral PVT. While

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clinical and radiological profiles were broadly similar across groups, MVPS and thrombophilic forms tended to exhibit more extensive thrombosis and complications. These findings highlight the importance of a thorough and targeted etiological assessment to guide management and follow-up.

Keyword(s) :

1635 - Predictors of Incomplete Biochemical Response to Ursodeoxycholic Acid in Primary Biliary Cholangitis Insights from a Moroccan Cohort

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Background / Hypothesis:

Ursodeoxycholic acid (UDCA) remains the cornerstone therapy for primary biliary cholangitis (PBC), but a substantial proportion of patients fails to achieve adequate biochemical response, which is associated with worse long-term outcomes. Identifying early predictors of incomplete response is essential for timely therapeutic optimization. This study aimed to determine the prevalence and clinical predictors of UDCA non-response in a Moroccan cohort, after excluding potential confounding factors.

Materials and Methods:

This retrospective analytical study included 101 patients diagnosed with PBC between 2016 and 2025 at a tertiary Moroccan center. Diagnosis was based on the 2017 EASL criteria. All patients received UDCA at standard doses (13–15 mg/kg/day), and biochemical response was assessed at 12 months according to the Paris II criteria. Patients with active autoimmune or endocrine disorders that could bias biochemical evaluation were excluded. Clinical, biochemical, and histological data were systematically analyzed. Comparisons between responders and non-responders were performed using univariate analysis to identify predictors of incomplete UDCA response.

Results:

The mean age at diagnosis was 49.8 years (range 18–78), with marked female predominance (sex ratio M/F = 0.10). Incomplete biochemical response to UDCA was observed in 50.5% of patients. Advanced fibrosis (F3–

F4) was strongly correlated with non-response ($p < 0.001$). Severe cholestasis, reflected by alkaline phosphatase $\geq 2 \times$ ULN ($p = 0.043$) and gamma-glutamyl transferase (GGT) $\geq 5 \times$ ULN ($p = 0.005$), as well as the presence of clinical portal hypertension ($p = 0.027$), were also significantly associated with poorer therapeutic outcomes. Conversely, age, sex, pruritus, anti-mitochondrial antibody positivity, and associated autoimmune diseases showed no significant correlation with UDCA response.

Conclusions:

This study provides additional insight into determinants of biochemical response to UDCA in PBC. The high proportion of incomplete responders highlights the heterogeneous therapeutic profile of the disease and underscores the need for early evaluation of disease severity. Advanced fibrosis and marked cholestasis emerged as the strongest predictors of treatment failure, reinforcing the relevance of baseline risk stratification. These findings support the development of refined treatment algorithms integrating early identification of high-risk patients and timely therapeutic intensification to improve long-term outcomes.

Keyword(s) : Primary biliary cholangitis, ursodeoxycholic acid, incomplete response, advanced fibrosis, predictors

1636 - Metabolic Dysfunction–Associated Steatotic Liver Disease in Normal Weight Individuals Clinical Biological Fibrosis and Autoimmune Profiles in a Moroccan Cohort

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Background / Hypothesis:

Metabolic dysfunction–associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is usually linked to overweight and obesity. However, a subset of patients develops hepatic steatosis despite having a normal body mass index (BMI). This phenotype may involve distinct pathophysiological mechanisms and can progress to severe liver disease despite the absence of obesity. The aim of this study was to describe the prevalence, clinical and biological features, elastographic findings, associated metabolic and

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autoimmune conditions, and evolutionary profile of MASLD in normal-weight individuals in our population, and to raise awareness of this phenotype to guide tailored diagnostic and surveillance strategies.

Materials and Methods:

This retrospective, descriptive study included 68 patients with hepatic steatosis and BMI between 18 and 25 kg/m², selected from a cohort of 170 patients followed in a hepatology and gastroenterology department. Demographic, clinical, laboratory, metabolic, elastography data, and associated autoimmune conditions were collected. Liver fibrosis was assessed by elastography when available.

Results:

The mean age was 47 years (range 23–62), with a male-to-female ratio of 0.79. Most patients were asymptomatic; hepatic steatosis was discovered incidentally in 77%, while 16% presented with biliary colic and 7% with atypical epigastric pain. Liver function tests showed cytotoxicity in 31%, cholestasis in 11%, both in 10%, and normal profiles in 48%. Metabolic assessment was performed in 81% of patients: diabetes was present in 38%, dyslipidemia in 14%, hypertension in 17%, while 25% had no detectable metabolic disorder. Elastography showed F0–F1 fibrosis in 67.6% and F2 in 7.35%; no advanced fibrosis (F3–F4) was observed.

Associated autoimmune and other conditions were frequent: celiac disease in 22%, Crohn's disease in 14%, ulcerative colitis in 1.4%, and hepatitis C infection in 10%.

Conclusions:

MASLD in normal-weight individuals represents a distinct phenotype from obesity-related hepatic steatosis. A substantial proportion of normal-BMI patients develop steatosis, likely linked to metabolic abnormalities such as insulin resistance, dyslipidemia, or gut microbiota alterations, with a notable frequency of associated autoimmune conditions. These findings support recognition of normal-weight MASLD as a specific clinical entity, warranting tailored diagnostic evaluation and surveillance that considers both metabolic and autoimmune risk factors. Further larger studies are needed to confirm these results and to develop locally-adapted diagnostic and management algorithms.

Keyword(s) : Metabolic Dysfunction–Associated Steatotic Liver Disease, normal body mass index, Clinical, Biological, Fibrosis, and Autoimmune Profiles.

1637 - The impact of serum lipids metabolism on the prognosis of hepatitis B virus related acute on chronic liver failure

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Background / Hypothesis:

At present, the relationship between lipids metabolism and the prognosis of liver failure is a hot spot. The purpose of this study was to explore the relationship between serum lipids parameters and the prognosis of hepatitis B virus related acute-on-chronic liver failure (HBV-ACLF).

Materials and Methods:

For patients with HBV-ACLF, to compare dynamic changes of serum lipids parameters in dead group with that of the group of survived more than 12 weeks. The diagnostic criteria were in accord with American Association for the study of Liver Diseases practice guidance on acute-on-chronic liver failure and the management of critically ill patients with cirrhosis in 2023. Differences between groups were made by the t-test. And $P < 0.05$ is considered statistically significant. Differences between groups were made by the t-test. And $P < 0.05$ is considered statistically significant.

Results:

From January 1, 2020 to July 1, 2025, 71 patients with HBV-ACLF had enrolled in Third Affiliated Hospital of Sun Yat-sen University, including 30 cases died within 5 weeks and 41 patients hand survived more than 12 weeks. Their ages were 43.73 ± 11.56 years and 46.38 ± 13.27 years respectively, and no significant difference was between the two groups ($P = 0.377$). The parameters values and the comparisons were shown in Table 1 and 2.

Except for HDL-C and Lipoprotein- α , the other parameters of the four weeks before death were significantly lower than those of survival group (all $P < 0.05$). After three weeks treatment, in survival group, lipids parameters had recovered. However, CHOL, HDL-C, LDL-C and ApoA-I values in one week before death

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were still significantly lower than that of survival group.

Conclusions:

Lipids metabolism disorder may worsen the prognosis of HBV-ACLF, especially hypolipidemia (CHOL, LDL-C and Lipoprotein- α). More treatment and related mechanism research need to be paid to improve serum lipid in HBV-ACLF.

Keyword(s) : Lipids metabolism; Hepatitis B virus related acute-on-chronic liver failure (HBV-ACLF); Prognosis

1638 - Indole 3 carbaldehyde mitigates fibrosis progression in MASH with T2DM via the SIRT1 TGF β SMAD signaling pathway

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Background / Hypothesis:

Hyperglycemia is an independent risk factor for the accelerated progression of metabolic dysfunction-associated steatohepatitis (MASH) to liver fibrosis, with the underlying mechanisms not yet fully elucidated. Microbial metabolites have emerged as critical components that mediate the metabolic effects in various diseases. However, the role of gut-derived metabolites in the development of liver fibrosis in MASH with type 2 diabetes mellitus (T2DM) remains unclear.

Materials and Methods:

A diabetic metabolic dysfunction-associated steatohepatitis (T2DM-MASH) mouse model was successfully established by high-fat diet (HFD) feeding combined with intraperitoneal injections of streptozotocin (STZ) and carbon tetrachloride (CCl₄). Metabolomics screening was conducted to identify characteristic metabolites associated with T2DM-MASH. Indole-3-carbaldehyde (3-Ald) was orally administered at two doses (25 and 50 mg/kg) for 6 weeks. The protective effect of Indole-3-carbaldehyde (3-Ald) on T2DM-MASH was assessed through in vivo experiment.

Results:

The induction of T2DM-MASH model was validated by metabolic disturbances (hyperglycemia, dyslipidemia), hepatic injury, and histopathological evidence of steatohepatitis (steatosis, inflammation, fibrosis). Fecal

3-Ald levels were significantly reduced in T2DM-MASH and inversely correlated with fasting blood glucose, histopathological fibrosis stage and lobular inflammation score. 3-Ald supplementation significantly ameliorated the biochemical results (glucose, cholesterol, triglycerides, alanine transaminase (ALT), aspartate transaminase (AST), $p < 0.05$) and pathological features of T2DM-MASH. Moreover, ultrastructural intestinal damage was observed in T2DM-MASH mice, while administration of 3-Ald restored gut barrier function by upregulating tight junction proteins (ZO-1, occludin; $p < 0.05$), leading to a significant reduction in plasma LPS ($p < 0.05$). Mechanistically, 3-Ald supplementation reduced TNF- α levels ($p < 0.05$), increased MMP-1 and SIRT1 levels ($p < 0.05$), and reduced α -SMA, TIMP-1, TGF- β 1, p-Smad2 and p-Smad3 levels ($p < 0.05$) in T2DM-MASH mice compared to vehicle control, leading to an alleviation of inflammation and fibrosis. The hepatoprotective and antifibrotic effects of 3-Ald were mostly dose-dependent.

Conclusions: Our findings, for the first time, underscore the potential of the microbial metabolite 3-Ald to attenuate fibrosis progression in T2DM-MASH by regulating the SIRT1/TGF- β /SMAD pathway, offering novel insights and promising therapeutic strategies for its management.

Keyword(s) : Metabolic dysfunction-associated steatohepatitis; Type 2 diabetes mellitus; Indole-3-carbaldehyde; Liver fibrosis; SIRT1/TGF- β /SMAD pathway

1639 - TRPV4 Cooperates with HBx to Promote HBV Replication Through a Calcium Mediated Positive Feedback Loop

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Background / Hypothesis: Hepatitis B virus (HBV) infection continues to impose a significant worldwide health burden. A primary obstacle to cure is the persistence of covalently closed circular DNA (cccDNA), which current therapies cannot eradicate. Therefore, further decoding of the HBV life cycle is imperative for developing novel antiviral strategies. We have previously demonstrated that the host factor TMEM203, which modulates calcium signaling, acts as a suppressor of HBV replication. This discovery offers compelling evidence for the critical role of intracellular

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calcium signaling in facilitating the viral replication process.

Materials and Methods:In this study, utilizing two HBV stable cell models (HepAD38 and HepG2.2.15 cells), we performed both knockdown and overexpression of the TRPV4 gene. The levels of hepatitis B surface antigen (HBsAg) and e antigen (HBeAg) in the cell culture supernatant were quantified by enzyme-linked immunosorbent assay (ELISA). HBV DNA in the supernatant, intracellular HBV DNA, and covalently closed circular DNA (cccDNA) were measured by quantitative real-time PCR (qPCR). Intracellular HBV core protein expression was detected by western blot analysis, while HBV RNA levels were assessed using reverse transcription qPCR (RT-qPCR). The effect of TRPV4 on HBx protein stability was evaluated using a protein half-life assay. Intracellular calcium levels were monitored by immunofluorescence. The physical interaction between TRPV4 and HBx was examined by co-immunoprecipitation (Co-IP). Furthermore, the regulatory role of TRPV4 in HBV nucleocapsid assembly was investigated using native agarose gel electrophoresis and a DNA release assay.

Results:In this study, we found that transient receptor potential vanilloid 4 (TRPV4) notably enhances HBV reproduction by investigating the effects of several calcium ion-related molecules on HBV replication. The in-depth study showed a positive feedback loop is established between TRPV4 and HBx: TRPV4 elevates HBx levels by augmenting its transcription and stabilizing the protein, while HBx upregulates TRPV4 expression via TBP, leading to increased cytosolic calcium. This reciprocal regulation ultimately activates the Pyk2/Src pathway to enhance HBV replication.

Conclusions:In conclusion, TRPV4 could interact with HBV core protein and regulate HBV during transcription and replication. These data suggest that TRPV4 exerts multifaceted HBV-related synergistic factors and may serve as a therapeutic target for CHB.

Keyword(s) : TRPV4, HBV, cccDNA, HBx, Calcium signaling

1643 - PREDICTIVE SCORING MODEL FOR HEPATOCELLULAR CARCINOMA AMONG FILIPINO PATIENTS A 10 YEAR RETROSPECTIVE STUDY

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a leading

cause of cancer-related mortality globally. Early risk stratification models integrating clinical and biochemical markers are essential to improve detection and management, particularly among Filipino patients who bear a significant disease burden. This study aimed to develop and validate a predictive scoring model for HCC based on demographic, clinical, and laboratory data.

Materials and Methods:

A retrospective review of 400 patient records, comprising 200 HCC cases and 200 controls without HCC, was conducted. Variables analyzed included age, sex, body mass index, comorbidities, liver function tests, hematological parameters, and tumor marker alpha-fetoprotein (AFP). Binomial logistic regression identified independent predictors of HCC. Model performance metrics such as accuracy, sensitivity, specificity, and area under the receiver operating characteristic curve (AUC) were evaluated.

Results:

Significant predictors of HCC included AFP levels ($p < 0.001$), prothrombin time (INR) ($p = 0.012$), platelet count ($p < 0.001$), and smoking status ($p = 0.008$). Age, ALT, albumin, and platelet-to-lymphocyte ratio were not independently significant. The model attained an accuracy of 73.5%, sensitivity of 78.0%, specificity of 69.3%, and an AUC of 0.834, demonstrating robust discrimination between HCC and non-HCC patients.

Conclusions:

The developed predictive scoring model effectively identifies Filipino patients at elevated risk for HCC, supporting targeted screening and early intervention. Implementation of this model in clinical practice is recommended to improve disease prognosis. Further prospective validation is warranted to optimize clinical utility.

Keyword(s) : Hepatocellular carcinoma, predictive model, alpha-fetoprotein, prothrombin time, platelet count

1644 - Epidemiological Clinical and Therapeutic Profile of Chronic Hepatitis B in a North African Cohort Determinants of Advanced Fibrosis

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Background:

Chronic hepatitis B virus (HBV) infection remains a major global health challenge and a leading cause of cirrhosis and hepatocellular carcinoma (HCC). Despite therapeutic advances, functional cure is rarely achieved, especially in endemic regions. This study aimed to describe the epidemiological, clinical, virological, and therapeutic profiles of chronic HBV infection and to identify factors associated with advanced fibrosis (F3–F4) in a cohort followed over two decades in a North African tertiary center.

Materials and methods:

This retrospective analytical study included 124 patients diagnosed with chronic HBV between 2005 and 2025. Demographic, clinical, biochemical, serological, and histological data were collected, including HBV markers, fibrosis assessment by elastography or biopsy, and viral co-infections. Therapeutic outcomes assessed were biochemical and virological response, HBeAg seroconversion, and HBsAg loss. Logistic regression analysis identified independent predictors of advanced fibrosis.

Results:

The mean age 46.8 years, with a sex ratio M/F of 0.9. Most patients were asymptomatic at diagnosis (75.8%). Abdominal ultrasound revealed signs of chronic liver disease in 16.9%, hepatic steatosis in 16.1%, and features of portal hypertension in 12%. Comorbidities included alcohol use (10.5%), diabetes mellitus (11.3%), and smoking (11.3%). HBsAg was positive in 96.8% of patients, and 7.2% were HBeAg-positive, with a median HBV DNA level of 9.2×10^6 IU/mL. Fibrosis assessment showed F0–F1 in 67.3%, F2 in 18.5%, and advanced fibrosis (F3–F4) in 9.7%.

Antiviral therapy was mainly tenofovir disoproxil fumarate (56.5%), followed by entecavir (3.2%), tenofovir alafenamide (1.6%), pegylated interferon (1.6%), and lamivudine (0.9%), while 37.9% were managed with clinical surveillance. During follow-up, HBV DNA decreased to a median of 10 IU/mL in treated patients. Biochemical response was achieved in 89.5% of treated cases, HBeAg seroconversion occurred in 88.8% of initially positive patients, and HBsAg loss was documented in 5.6%.

Advanced fibrosis was significantly associated with alcohol use, diabetes, age >50 years, and HCV co-infection ($p < 0.05$). Viral load strongly correlated with fibrosis severity ($r = 0.72$, $p < 0.001$), although some discordant cases were observed. In multivariate analysis, male sex, family history of cirrhosis, and

presence of portal hypertension were independent predictors of advanced fibrosis (all $p < 0.05$).

Conclusion:

Chronic HBV infection in this cohort was often silent and detected at early stages. Despite effective viral suppression, HBsAg loss remained rare. The role of alcohol, metabolic factors, and age in fibrosis progression underscores the need for early detection, individualized management, and improved access to antiviral therapy to support HBV elimination goals.

Keyword(s) : Chronic hepatitis B, antiviral therapy, fibrosis, predictors, epidemiology

1645 - The dynamic of APRI and FIB 4 scores over a 10 year period in HCV cirrhotic patients after virological cure with direct acting antivirals therapy

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Background / Hypothesis: Direct Acting Antivirals (DAA) have started being used in Romania since 2015 according to National Protocols for treatment. In the first years, the evaluation of liver fibrosis was made using FibroMax, but presently, the National Protocol for HCV treatment has added the APRI and FIB-4 scores along with Fibromax for fibrosis staging.

Materials and Methods: This prospective study was conducted between 2015 and 2025 in "Matei Balș" National Institute for Infectious Disease. It evaluates the dynamic of other liver tests besides Fibromax to identify easier methods to evaluate the fibrosis for patients treated with DAA and who achieved SVR. The patients were evaluated using FibroTest, AST to Platelet Ratio Index (APRI) and Fibrosis-4 score (FIB-4) at baseline, at 6 months post-end of treatment (post-EOT), at 1-year post-EOT and yearly afterwards.

Results: In this study, 169 patients with HCV compensated cirrhosis were evaluated at baseline. At

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treatment initiation, the median value of Fibrotest was 0.84, the median APRI score was: 1.15 and the median FIB-4 score was: 3.45. APRI and FIB-4 were correlated with the liver fibrosis – graded by Fibrotest ($p=0.03$ for APRI and $p<0.001$ for FIB-4). At 6 months post EOT, 107 patients were analyzed using APRI and FIB-4. The median value for APRI at the 6 months evaluation was 0.45 and the median value for FIB-4 was 2.12. Regarding the correlation between the two fibrosis scores and FibroTest, both are statistically significant correlated ($p=0.03$ for APRI and $p<0.001$ for FIB-4). At 1-year post-EOT, for 71 patients evaluated, the median APRI score was 0.42 and the median FIB-4 score was 1.96. For this evaluation, only FIB-4 correlated statistically with FibroTest ($p=0.019$). At 2 years post-EOT, 44 patients were evaluated and APRI had a median value of 0.41 and FIB-4 had a median value of 2.03 but no longer correlate with the values of FibroTest. At the last evaluation, at 7 years post-EOT, 27 patients had a median value for APRI of 0.38 and for FIB-4 of 2.02, without any correlation with FibroTest. The difference in the number of patients between baseline and the end of follow-up is due to the pandemic when most patients were lost from follow-up.

Conclusions: APRI and FIB-4 prove to be easier non-invasive markers of liver stiffness, highly correlated at baseline and at 6 months post-EOT with Fibrotest, accurate for the diagnosis of cirrhosis, and FIB-4 can be a useful score for monitoring the liver fibrosis post-EOT.

Keyword(s) : APRI, FIB-4, fibrosis, DAA

1648 - Predictive factors of complications and treatment response in patients with autoimmune hepatitis A 8 Year Single Center Study

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Background:

Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease that can lead to severe complications. Identifying predictors of complications and treatment response is crucial for risk stratification and optimizing management. This study aimed to determine factors associated with complications and response to

immunosuppressive therapy in AIH patients.

Materials and Methods:

We conducted an 8-year retrospective single-center study (2017–2025) including 48 AIH patients diagnosed according to simplified IAHG criteria. Demographic, clinical, biochemical, and histological data were collected. Complications included ascites, gastrointestinal bleeding, and hepatic encephalopathy. Therapeutic response was classified as complete or partial based on biochemical normalization. Statistical analysis used Chi-square/Fisher's tests for categorical variables and Student's t-test for continuous variables; significance was set at $p<0.05$.

Results:

The cohort included 48 patients (median age 38 years; male/female ratio 0.2). Autoimmune comorbidities were frequent (75%), mainly primary biliary cholangitis (55.6%) and autoimmune thyroiditis (52.8%). Clinically, jaundice (70.2%), hepatomegaly (60.9%), and splenomegaly (43.5%) were common. Cytolysis occurred in 87.5%, cholestasis in 85.4%, and hepatocellular insufficiency in 41.7%. Histology revealed lymphoplasmacytic infiltrate (86.8%), interface hepatitis (71.7%) and advanced fibrosis F3–F4 (63.5%). Portal hypertension was present in 54.2%. Complications occurred in 39.6% of patients, primarily ascites, GI bleeding, and hepatic encephalopathy. Portal hypertension (OR 12.1, $p<0.001$) and F4 cirrhosis (OR 11.0, $p<0.001$) were independent predictors. Patients already decompensated at diagnosis (initial complications) and those with portal hypertension were more likely to show partial response to immunosuppressive therapy (OR 2.9, $p\approx 0.04$). Age, sex, autoimmune comorbidities, hepatocellular insufficiency, autoantibody profile, and histology did not significantly influence treatment response.

Conclusion:

Portal hypertension and cirrhosis strongly predict complications in AIH. Presence of initial decompensation and portal hypertension at diagnosis is associated with partial response to therapy. Early identification and monitoring of high-risk patients are essential to optimize outcomes.

Keyword(s) : Autoimmune hepatitis, Complications, Portal hypertension, Cirrhosis, Therapeutic response, Predictive factors

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1650 - A Pilot Study on Hepatitis B Core Related Antigen (HBcrAg) as a Novel Marker for Monitoring Antiviral Therapy in Chronic Hepatitis B Patients

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Background / Hypothesis:

Chronic Hepatitis B (CHB) affects over 400 million people worldwide and remains a major cause of cirrhosis and hepatocellular carcinoma. Although nucleos(t)ide analogue (NA) therapy effectively suppresses viral replication, optimal criteria for safe treatment discontinuation remain unclear. Covalently closed circular DNA (cccDNA), the key reservoir sustaining HBV persistence, cannot be evaluated routinely. Hepatitis B core-related antigen (HBcrAg), which correlates with intrahepatic cccDNA and HBV DNA levels, has emerged as a promising surrogate biomarker. This pilot study hypothesized that combined negativity of HBV DNA and HBcrAg could predict sustained remission following NA cessation in CHB patients, and that HBcrAg may serve as a reliable marker to guide treatment discontinuation.

Materials and Methods:

This pilot observational study included 59 adult CHB patients attending the Liver Clinic, Max Super Speciality Hospital, Vaishali, who had received at least four years of NA therapy (entecavir/tenofovir) and achieved HBV DNA negativity at baseline. Patients with cirrhosis, hepatitis C co-infection, autoimmune hepatitis, alcohol-related liver disease, or HCC were excluded. HBcrAg was quantified using a chemiluminescent enzyme immunoassay with a lower detection threshold of 3 log U/mL. Patients who achieved both HBV DNA and HBcrAg negativity discontinued NA therapy and were followed for one year, with assessments at 6-monthly intervals including LFTs, HBV DNA and HBcrAg. Relapse was defined as HBV DNA >2000 IU/mL, reappearance of HBcrAg, or ALT/AST elevation >1.5 × ULN.

Results:

Of 59 patients (mean age 35.9 ± 11.4 years; 76.3% male), 83.1% were HBeAg-negative. All achieved HBV DNA negativity at baseline. After NA cessation, 56/59 patients (94.9%) remained HBV DNA-negative at 6 months, while 3 patients (5.1%) developed virological

relapse. Patients who relapsed demonstrated reappearance of both HBV DNA and HBcrAg. Those who maintained remission had persistently negative HBcrAg throughout follow-up, supporting its value as a surrogate of minimal residual cccDNA activity. No significant biochemical flares or ALT elevations were observed among sustained responders. HBcrAg negativity at cessation strongly correlated with relapse-free status on short-term follow-up.

Conclusions:

In this pilot Indian cohort, combined HBV DNA and HBcrAg negativity at the time of NA cessation was associated with a high rate (94.9%) of sustained viral suppression at 6-month follow-up. HBcrAg proved to be a useful non-invasive biomarker for monitoring antiviral therapy and predicting early relapse after treatment withdrawal. Although long-term follow-up is required, these findings support the incorporation of HBcrAg into treatment-cessation algorithms to improve individualized management of CHB.

Keyword(s) : Chronic Hepatitis B; HBcrAg; HBV DNA; cccDNA surrogate marker; Nucleos(t)ide analogue cessation; Virological relapse; Antiviral therapy monitoring

1651 - The dynamic of alpha fetoprotein in patients with hepatitis C cirrhosis after direct acting antivirals therapy over a 10 year period

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Background / Hypothesis: For patients with hepatitis C virus (HCV) infection, after the virological cure, the main concern is monitoring the dynamic of liver

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fibrosis and the occurrence of hepatic complications (liver decompensation or hepatocellular carcinoma (HCC)).

Materials and Methods:The objective of this study is to evaluate the dynamics of AFP for cirrhotic patients who achieved SVR after treatment with DAA. We conducted a prospective study on HCV compensated cirrhosis patients, monitored in "Matei Bals" National Institute for Infectious Diseases between 2015 - 2025. The patients were evaluated at baseline, at 6 months post end of treatment (EOT), 1 year after EOT, and once a year afterwards. The monitoring included the evaluation of FibroTest and AFP at each visit.

Results:Since November 2015, 169 patients with HCV cirrhosis were enrolled in the study. Before starting the DAA treatment, the median value of AFP was 9.71 ng/ml, with a minimum 0.1 ng/ml and a maximum 253.8 ng/ml. At the 6 months post-EOT visit, 73 patients were evaluated, with a median AFP of 4.9 ng/ml, with a statistically significant decrease from baseline ($p < 0.005$). At 1 year post-EOT, 45 patients were evaluated, with the median AFP of 4.1 ng/ml, without any statistically significant difference from the second visit ($p = 0.3$). At 2 years post-EOT, 28 patients were evaluated, with an AFP median value of 3.3 ng/ml, without any significant difference from the previous evaluation ($p = 0.89$). At 3 years post-EOT, 30 patients were evaluated, with a median value of AFP of 0.85 ng/ml, with a statistically significant decrease from the previous year ($p = 0.009$). At 7 years post-EOT, 19 patients were evaluated, with a median AFP of 3.9 ng/ml, without any significant difference from the evaluation at 3 years post-EOT, but the difference from baseline until the end of monitoring remains statistically significant ($p = 0.006$). None of the patients who were evaluated at 7 years post-EOT developed HCC. The difference in the number of patients between baseline and the end of follow-up is due to the pandemic when most patients were lost from follow-up.

Conclusions:Although the difference of AFP between the 3 years post-EOT visit and 7 years post-EOT visit is not statistically significant, there seems to be an increase in value, which means that cirrhotic HCV

patients need to be closely monitored at least once a year to be able to diagnose the early stages of liver complications predicted by the increase of the value of AFP.

Keyword(s) : fibrosis, DAA, AFP, cirrhosis

1654 - Long Term Prognosis of Acute HBV Cases

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Background / Hypothesis:The study aimed to investigate the long-term prognosis of cases diagnosed with acute viral hepatitis B virus (HBV) infection.

Materials and Methods:Following ethical committee approval, the characteristics and long-term prognosis of acute viral hepatitis B cases hospitalized and monitored at İzmir Bozyaka Training and Research Hospital and İzmir City Hospital were evaluated both through the hospital automation system and by face-to-face interviews with patients who came for follow-up, and by checking their current results. Acute exacerbations of chronic hepatitis and autoimmune hepatitis were excluded.

Results:Data was obtained from a total of 104 patients, 70 of whom were male (67.3%), born between 1991 and 1941. Forty-three point three percent of the cases were born between 1985 and 1990, 27.9% between 1974 and 1984, and 12.5% between 1973 and 1963. The years with the highest number of cases were 2010 (14 cases), 2025 (11 cases), 2009 (9 cases), 2012-2014-2017-2024 (8 cases), 2008 (7 cases), 2011-2013-2016 (8 cases), 2018-2023 (3 cases), 2022 (2 cases), and 2019 (1 case). A significant increase in cases was observed between 2024 and 2025. Approximately 90% of the cases were found to be sexually transmitted. When the long-term follow-up results of the cases were evaluated, as of November 2025, anti-HBs was detected in half of the 104 patients whose data was available (52 individuals); 13 individuals (12.5%) remained HBsAg positive and were undergoing follow-up/treatment; 10 patients (9.6%) had isolated anti-HBcIgG positivity; 1 patient underwent liver transplantation due to fulminant course, 1 patient was referred to an organ transplant center due to high INR, and 1 patient died due to fulminant course and liver failure. Twenty-four patients (23%) did not attend follow-up appointments, and no recent visits were found in the automated records; therefore, no

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information could be obtained about the outcome of these patients. Two patients have not yet completed the 6-month follow-up period, and their HBsAg positivity continues.

Conclusions: Thanks to the national HBV vaccination program implemented in our country since 1998, acute HBV cases have decreased significantly in children and adolescents. However, acute HBV cases continue to be seen in the adult age group, predominantly transmitted through sexual contact. Therefore, we believe that screening adults, especially those in their sexually active years, for HBV and vaccinating seronegative individuals will make a significant contribution, in line with the strategies emphasized in the 2025-2030 version of the Viral Hepatitis Prevention and Control Program in our country.

Keyword(s) : HBV, acute infection, prognosis

1655 - Alcoholic Liver Disease with Sarcopenia Association with Increased Fall Risk and Impaired Activity of Daily Living

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Background / Hypothesis:

To analyze the association of sarcopenia, fall risk, and activity of daily living (ADL) in patients with alcoholic liver disease (ALD)

Materials and Methods:

A cross-sectional study was conducted involving patients hospitalized in the Third Department of Liver Diseases at Beijing Ditan Hospital, Capital Medical University, between January 2021 and December 2024.

Results:

The prevalence of sarcopenia in ALD patients was 34.9%, and the rate of ADL decline was 44.2%. The proportion of patients with ADL decline was significantly higher in the sarcopenia group than in the non-sarcopenia group (24% vs. 20.2%, $\chi^2 = 17.204$, $P < 0.001$). Spearman's correlation analysis indicated that the occurrence of sarcopenia in ALD patients was negatively correlated with ADL scores at admission and discharge, albumin level, hemoglobin level, body mass index (BMI), and blood phosphorus level, and positively correlated with fall risk score (all $P < 0.05$). Binary logistic regression analysis identified the following risk factors for sarcopenia in ALD patients:

age (OR = 1.056, 95% CI: 1.017–1.096, $P = 0.005$), admission ADL score (OR = 0.960, 95% CI: 0.938–0.982, $P < 0.001$), discharge ADL score (OR = 0.941, 95% CI: 0.910–0.974, $P < 0.001$), fall risk score (OR = 1.028, 95% CI: 1.007–1.050, $P = 0.009$), albumin level (OR = 0.853, 95% CI: 0.783–0.930, $P < 0.001$), hemoglobin level (OR = 0.984, 95% CI: 0.970–0.998, $P = 0.022$), blood phosphorus level (OR = 0.127, 95% CI: 0.025–0.630, $P = 0.012$), and BMI (OR = 0.689, 95% CI: 0.588–0.808, $P < 0.001$). Multivariate regression analysis demonstrated that discharge ADL score (OR = 0.940, 95% CI: 0.898–0.985, $P = 0.009$), albumin level (OR = 0.834, 95% CI: 0.737–0.943, $P = 0.004$), and BMI (OR = 0.677, 95% CI: 0.565–0.811, $P < 0.001$) were independent risk factors for sarcopenia in ALD patients.

Conclusion

The prevalence of sarcopenia in ALD patients was 34.9%, and the rate of ADL decline was 44.2%. Sarcopenia was associated with decreased ADL, increased fall risk, hypoalbuminemia, anemia, and low body weight. Low BMI, advanced age, hypoalbuminemia, impaired ADL, and high fall risk were risk factors for sarcopenia, while discharge ADL score, albumin level, and BMI were independent influencing factors for sarcopenia (all $P < 0.05$).

Discussion

It demonstrates that patients with ALD and sarcopenia commonly exhibit reduced ADL and an increased risk of falls. It is therefore essential to develop a systematic nursing care plan focused on fall prevention and ADL improvement for this population.

Keyword(s) : Sarcopenia, Alcoholic Liver Disease, Fall Risk, activity of daily living

1657 - Research on gut microbiota characteristics of PBC patients at different ALBI grades based on machine learning

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Background / Hypothesis:

The Albumin-Bilirubin (ALBI) score and grade are widely used to stratify patients with primary biliary cholangitis (PBC) into different disease statuses and risk levels. Recent studies have increasingly highlighted the role of gut microbiota in autoimmune liver diseases. This study aimed to investigate the differences in gut microbiota among PBC patients with varying ALBI grades.

Materials and Methods:



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Clinical data and stool samples were collected from outpatient and inpatient PBC patients between 2019 and 2022. Gut microbiota profiles were obtained using 16S rDNA sequencing of stool samples. We analyzed alpha diversity, beta diversity, LEfSe analysis and pathway function prediction. Additionally, various machine learning methods—including random forest (RF), lasso, gradient boosting machine (GBM) and support vector machine (SVM)—were employed to identify key features and to build and validate predictive models using bootstrap techniques.

Results:

Clinical characteristics of ALBI grade 1 patients were comparatively better than those of ALBI grade 2 and 3 patients, including multiple laboratory indices. Gut microbiota analysis revealed that species richness and balance were higher in ALBI grade 1 patients. Both the comparison of the most abundant genera and the linear discriminant analysis (LDA) in LEfSe demonstrated that *Lachnospira* had a higher abundance and better discriminative ability in ALBI grade 1. Pathway function prediction indicated that sulfur metabolism was upregulated in higher ALBI grades. Furthermore, RF identified 10 specific genera, which were then used to build and validate models for discriminating PBC patients according to their ALBI grades. All three models, developed using different machine learning methods, demonstrated good discrimination ability (mean AUC 0.75–0.80).

Conclusions:

This study highlights significant differences in gut microbiota profiles among PBC patients with different ALBI grades. The increased abundance of *Lachnospira* and upregulation of sulfur metabolism pathways are notable in patients with lower ALBI grades. The machine learning models developed based on gut microbiota features offer promising tools for discriminating between PBC patients with varying disease severities, which could enhance the precision of treatment strategies.

Keyword(s) : primary biliary cholangitis, albumin-bilirubin grade, gut microbiota, machine learning

1658 - Exploring Factors Influencing Public Intention to Receive Hepatitis E Vaccination Based on the Theory of Planned Behavior

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Background / Hypothesis:

The study applied the Theory of Planned Behavior (TPB), including attitude, subjective norms, and perceived behavioral control, to identify factors influencing public intention and actual behavior regarding hepatitis E (HEV) vaccination, with the goal of informing strategies to improve vaccine uptake.

Materials and Methods:

From December 2023 to April 2024, an anonymous online questionnaire was used to collect data on HEV vaccination intention and outcomes, TPB-related scores, levels of knowledge and attention regarding the HEV vaccine, demographic characteristics, current health status, past health behaviors, and hepatitis-related social exposure. Chi-square tests and binary logistic regression were used to identify factors associated with vaccination intention and actual vaccination behavior.

Results:

A total of 651 valid responses were obtained. Among them, 158 participants (24.23%) were unwilling to receive the HEV vaccine, while 493 (75.73%) expressed willingness; among those willing, 41 individuals (6.30%) had actually been vaccinated. Significant differences in willingness ($p < 0.05$) were observed across age, education, occupation, hepatitis-related vaccination history within the past 10 years, knowledge and attention toward the HEV vaccine, and TPB dimensions (attitude, subjective norms, and perceived behavioral control). Logistic regression identified attitude ($p < 0.001$, $\text{Exp}(B)=3.515$), subjective norms ($p < 0.001$, $\text{Exp}(B)=3.515$), perceived behavioral control ($p=0.014$, $\text{Exp}(B)=1.880$), and attention ($p=0.043$, $\text{Exp}(B)=1.568$) as independent predictors of vaccination intention. Among those expressing willingness, binary analysis indicated that attention ($p=0.027$, $\text{Exp}(B)=4.148$), knowledge ($p=0.019$, $\text{Exp}(B)=2.424$), perceived behavioral control ($p=0.039$, $\text{Exp}(B)=2.819$), and hepatitis-related vaccination in the past 10 years ($p=0.047$, $\text{Exp}(B)=2.119$) influenced actual vaccination behavior.

Conclusions:

The TPB model effectively evaluates public intention to receive the HEV vaccine. A clear gap exists between intention and actual vaccination. Prior hepatitis-related vaccination, knowledge level, attention toward the HEV vaccine, and perceived behavioral control all affected vaccination behavior. Strengthening public education and communication may improve intention, while

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simplifying procedures and optimizing costs may further support vaccination uptake.

Keyword(s) : Hepatitis E vaccine, Vaccination intention, Theory of Planned Behavior, Perceived behavioral control, Public health behavior

1659 - Efficacy Analysis of Prednisone Tablets Combined with Azathioprine in the Treatment of Autoimmune Hepatitis A Single Center Retrospective Cohort Study

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Background / Hypothesis: To evaluate the biochemical and immunological response rates at 3 and 6 months in patients with autoimmune hepatitis (AIH) treated with prednisone tablets combined with azathioprine (AZA).

Materials and Methods: A total of 84 patients with pathologically confirmed AIH between January 2022 and June 2025 were enrolled in this study. Clinical data, liver biopsy results, and follow-up data at 3 and 6 months of treatment were collected. The treatment regimen consisted of prednisone tablets (0.5 mg/kg) combined with azathioprine (50 mg/day) for a 6-month induction period. The primary endpoints were biochemical response (defined as normalization of ALT and AST levels) and immunological response (defined as normalization of immunoglobulin G levels) at 3 and 6 months of treatment.

Results: 1. Baseline characteristics: Among the 84 patients, 69 (82.14%) were female and 15 (17.86%) were male, with a mean age of 51.61 ± 12.31 years. Of these, 50 patients (59.52%) completed both the 3- and 6-month treatment and follow-up, with complete data available.

2. Efficacy analysis: At 3 months of combination therapy, the biochemical response rate was 64.0% (32/50), and the immunological response rate was

54.0% (27/50). At 6 months, the biochemical response rate increased to 86.0% (43/50), and the immunological response rate reached 82.0% (41/50).

3. The combination therapy was well-tolerated, with common adverse effects including moon face and weight gain.

Conclusions: This study demonstrates that the combination of prednisone tablets and azathioprine achieves significant short-term biochemical and immunological responses in AIH patients during the induction period, with a favorable safety profile.

Keyword(s) : Autoimmune hepatitis; Prednisone tablets; Azathioprine; Treatment response

1660 - Four Year Survival in Giant Hepatocellular Carcinoma A Case Report on Long Term Disease Control Following Multimodal Therapy

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Background: Hepatocellular carcinoma (HCC) is the most common primary liver malignancy and a leading cause of cancer-related mortality worldwide. Targeted therapy, immunotherapy, interventional approaches, and radiotherapy have become essential components in the management of intermediate- and advanced-stage HCC. However, significant challenges persist due to the lack of precise strategies for drug selection and the difficulty in optimizing the balance between treatment efficacy and patient safety.

Method: This report summarizes the clinical data of a patient diagnosed with advanced massive hepatocellular carcinoma. A comprehensive review of the patient's medical history, physical examination findings, laboratory results, and imaging studies was conducted. The management strategy, including the anti-tumor treatment regimen administered during hospitalization, and subsequent follow-up outcomes are also documented.

Results: A 58-year-old male with giant hepatocellular carcinoma (154mm×123mm) was successfully treated with combination therapy of atezolizumab and

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bevacizumab, after 6-month treatment, he achieved partial response (83mm×68mm). To alleviate tumor burden, control newly emerged local lesions, and treat portal vein tumor thrombus in this patient with advanced hepatocellular carcinoma, three additional sessions of TACE and localized radiotherapy were administered. Ultimately, the patient has achieved an overall survival duration of approximately 4 years till now. However, proteinuria was observed during course of therapy. Following dose reduction of bevacizumab, the patient's proteinuria markedly improved, as indicated by a substantial decrease in urinary protein quantification. Notably, the patient's tumor status has remained stable following the reduced dosage of bevacizumab. He is currently alive with good healthy condition. Over the course of nearly four years of treatment, the treatment intervals and dosage of bevacizumab were adjusted according to the patient's condition, which not only reduced adverse reactions but also maintained therapeutic efficacy, achieving individualized treatment.

Conclusions: The multimodal and combination therapy of target therapy, immunotherapy, interventional therapy, and radiotherapy may present as a more favorable treatment alternative for patients with advanced HCC. This case may provide safety and efficacy evidence supporting clinical practice adjustments of bevacizumab dosage in response to treatment-related toxicities. The emergence of new local lesions during the treatment of advanced hepatocellular carcinoma does not necessarily indicate the failure of the systemic treatment regimen. Treatment adjustments should not be made hastily, instead, a comprehensive analysis must be conducted, as the currently available systemic treatment options are very limited.

Keyword(s): Hepatocellular carcinoma, combination therapy, atezolizumab, bevacizumab, proteinuria, TACE, radiotherapy

1663 - Diagnostic prediction model of porto-sinusoidal vascular disease patients with gamma glutamyl transferase elevation

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Background / Hypothesis:

More and more patients diagnosed with porto-sinusoidal vascular disease (PSVD) are experiencing gamma-glutamyl transferase (GGT) elevation. There are

currently no non-invasive diagnostic indicators for PSVD patients. This study aims to establish a diagnostic prediction model of PSVD patients with GGT elevation.

Materials and Methods:

This retrospective study included patients who underwent liver biopsy were diagnosed with PSVD at Beijing Ditan Hospital as shown in Figure 1. Patients were stratified into two groups based on the GGT level: patients with GGT elevation >ULN confirmed as PSVD (PSVD+Abnormal GGT group), and patients with normal GGT level (PSVD+Normal GGT group). Clinical features and risk factors were analyzed to identify features specific to PSVD with persistent GGT elevation. Nomogram model was used for diagnostic prediction of PSVD patients with GGT elevation.

Results:

A total of 290 PSVD patients who underwent liver biopsy were enrolled in this study, including PSVD+Normal GGT group (n=156) and PSVD+Abnormal GGT group (n=134). Among them, the proportion of females was higher than that of males, with an even greater female predominance observed in the PSVD+Abnormal GGT group. Compared to the Normal GGT group, the PSVD+Abnormal GGT group exhibited higher levels of white blood cell count, platelet (PLT) count, alanine aminotransferase (ALT), aspartate aminotransferase (AST), GGT, prothrombin activity (PTA), and liver stiffness measurement (LSM), as shown in Table 1. Liver pathology findings revealed a higher proportion of architectural disturbance in the PSVD+Abnormal GGT group (Table 2). Stratified analysis of the two patient groups demonstrated that the PSVD+Abnormal GGT group had significantly higher proportions of females, concomitant portal hypertension, $PLT \geq 150 \times 10^9/L$, $LSM \geq 10$ kPa, and $FIB-4 > 3.25$ compared to the PSVD+Normal GGT group (Table 3). Univariate and multivariate logistic regression analysis revealed that sex [OR 0.406, 95%CI (0.223-0.738), $p=0.003$], ALT [OR 1.032, 95%CI (1.021-1.230), $p=0.016$], and $LSM < 10$ [OR 3.298, 95%CI (1.664-6.535), $p=0.001$] emerged as independent risk factors for PSVD patients with elevated GGT, as shown in Table 4. Nomogram model revealed that AUC 0.772, 95% CI (0.717-0.827), and The specificity of this model is 0.821.

Conclusions:

Sex, ALT, PTA, and LSM were independent influencing factors for PSVD patients with GGT elevation. Nomogram model can effectively predict the disease

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risk of PSVD patients in the population with elevated GGT.

Keyword(s) : porto-sinusoidal vascular disease, gamma-glutamyl transferase, alanine aminotransferase, liver stiffness measurement, nomogram model

1665 - ADAR2 Deficiency Ameliorates Metabolic Dysfunction Associated Steatohepatitis (MASH) and Fibrosis in Mice

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Background / Hypothesis:

Metabolic dysfunction-associated steatohepatitis (MASH) is a chronic, progressive liver disease characterized by steatosis, inflammation, and fibrosis. ADAR2, a member of the adenosine deaminases acting on RNA (ADARs) family, catalyzes the adenosine-to-inosine editing of double-stranded RNA; however, its specific role in MASH remains unclear.

Materials and Methods:

This study aimed to explore the function of ADAR2 in MASH pathogenesis. We employed a choline-deficient, L-amino acid-defined high-fat diet (CDAHFD) to induce MASH in mice. ADAR2^{+/+}/GluR-BR/R mice (wild type, WT) and ADAR2^{-/-}/GluR-BR/R mice (ADAR2 KO) mice of both sexes were fed either a normal chow diet (ND) or CDAHFD for 4 to 6 weeks.

Results:

The results demonstrated that male ADAR2 KO mice exhibited significantly improved glucose tolerance compared to WT mice following CDAHFD feeding. Furthermore, ADAR2 KO mice fed the CDAHFD showed attenuated hepatic lipid accumulation and upregulated expression of genes related to fatty acid beta-oxidation. In addition, hepatic levels of inflammatory markers—including TNF-beta, CD68, and F4/80—were significantly lower in the ADAR2 KO group. Moreover, ADAR2 deficiency alleviated CDAHFD-induced liver fibrosis.

Conclusions:

These findings suggest that ADAR2 plays a detrimental role in MASH progression and highlight ADAR2 as a promising therapeutic target for the disease.

Keyword(s) : MASH, ADAR2, fibrosis

1666 - Diagnostic Performance of Non Invasive Fibrosis Scores Compared with Liver Biopsy in Chronic Hepatitis B

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Background:

Liver biopsy is considered the gold standard for grading liver fibrosis. However, as it is an invasive and costly procedure with potential for serious complications, interest in non-invasive tests to reduce the need for biopsy has increased.

Materials and Methods:

A total of 553 patients with chronic hepatitis B who underwent liver biopsy at our university hospital between January 1, 2014 and July 1, 2024 were included. Epidemiological, clinical, and laboratory data and Ishak fibrosis scores obtained from liver biopsy were retrospectively retrieved from the hospital information system. Concurrently with biopsy, the aspartate aminotransferase (AST)-alanine aminotransferase (ALT) ratio (AAR), AST-to platelet ratio index (APRI), fibrosis-4 score (FIB-4), and age-platelet index (API) were calculated. The diagnostic performance of these non-invasive tests in assessing Ishak fibrosis scores was evaluated. Diagnostic accuracies of APRI, FIB-4, API, and AAR were compared using ROC curve analysis and the DeLong test. In addition, multivariable logistic regression models were constructed to assess the added diagnostic value of combining non-invasive scores.

Results:

Among the 553 patients, 243 (43.9%) were female and 310 (56.1%) were male. When an Ishak fibrosis score of ≥ 3 was used as the threshold, FIB-4 (AUC = 0.804; 95% CI: 0.768–0.836), APRI (AUC = 0.779; 95% CI: 0.742–0.813), and API (AUC = 0.775; 95% CI: 0.738–0.809) demonstrated significant diagnostic performance. A small but statistically significant difference was observed between FIB-4 and API ($p = 0.029$), whereas no significant difference was found between APRI and FIB-4 or between APRI and API. When an Ishak fibrosis score of ≥ 4 was used as the threshold, FIB-4 (AUC = 0.833), APRI (AUC = 0.791), and API (AUC = 0.803) demonstrated significant discriminative ability, with no statistically significant differences among the three scores. In both analyses, AAR showed poor discriminatory performance for fibrosis (AUC = 0.538; $p = 0.139$ and AUC = 0.505; $p = 0.894$, respectively), and APRI, FIB-4, and API were significantly superior to AAR ($p < 0.001$). To evaluate the contribution of combining non-invasive tests to diagnostic performance, three

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logistic regression models were constructed. Among these, only Model I created for fibrosis score ≥ 3 showed a statistically significant difference compared to FIB-4 alone ($p = 0.047$).

Conclusions:

In our study, FIB-4, APRI, and API demonstrated comparable performance; however, FIB-4 was determined with higher sensitivity for excluding fibrosis. Its accessibility, low cost, and reproducibility suggest it as an option for initial fibrosis evaluation.

Keyword(s) : Hepatitis B, Fibrosis, Non-invasive, FIB-4

1667 - Combination of carvedilol and variceal band ligation is superior to carvedilol alone in primary prophylaxis of variceal bleed in cirrhotics with high risk varices A randomized controlled trial

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Background / Hypothesis: Portal hypertension-related variceal bleeding is a major cause of morbidity and mortality in cirrhosis. Both non-selective beta-blockers (NSBBs) like carvedilol and endoscopic variceal ligation (EVL) are recommended for primary prophylaxis. However, the efficacy of combining EVL with carvedilol in high-risk varices remains uncertain. This study evaluates whether combination therapy is superior to carvedilol alone in preventing first variceal bleed.

Materials and Methods: This prospective, single-center, randomized controlled trial included 80 cirrhotic patients with high-risk varices (large size or red color signs) and no prior history of bleeding. Patients were randomized 1:1 to receive either carvedilol alone or carvedilol plus scheduled EVL sessions. Carvedilol was initiated at 6.25 mg/day and titrated to the maximum tolerated dose. EVL was performed every 3 weeks until variceal eradication. Patients were followed for 12 months. The primary endpoint was incidence of first variceal bleeding. Secondary endpoints included mortality, hospitalization for bleeding, and adverse events.

Results: Seventy-two patients completed the study ($n=36$ in each arm). No significant differences were found in baseline demographic or clinical parameters,

including age, ensuring comparability between the groups. Intention-to-treat analysis showed that the combination group had a significantly lower incidence of first variceal bleed compared to those in the carvedilol-alone group after 1 year (22.2 % vs. 50 %, $p = 0.014$). Overall mortality after 1 year was lower in combination group although without a statistically significant difference (11.1 % vs. 19.4 %, $p = 0.325$). Mortality due to bleeding after 1 year was also lower in combination group but without a statistically significant difference (5.6 % vs. 13.9 %, $p = 0.232$). Variceal bleeding was the most common cause of death in both group (5.6 % vs. 13.9 %, $p = 0.232$). Both groups had similar adverse event profiles.

Conclusions: The combination of EVL and carvedilol is superior to carvedilol alone for primary prophylaxis of variceal bleeding in cirrhosis patients with high-risk esophageal varices and there is no significant difference in adverse events between two groups. This dual-modality approach should be considered in primary prophylaxis protocols for this high-risk group.

Keyword(s) : Cirrhosis, Portal Hypertension, Variceal bleed, carvedilol, Endoscopic variceal ligation, Primary prophylaxis

1668 - Metabolic Steatosis in Chronic Hepatitis B High Prevalence but No Impact on Fibrosis or Antiviral Response in a Moroccan Cohort

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Background / Hypothesis:

The coexistence of chronic hepatitis B virus (HBV) infection and metabolic-associated steatotic liver disease (MASLD) is increasingly recognized. MASLD, defined by hepatic fat accumulation in the presence of metabolic risk factors such as diabetes, dyslipidemia, or hypertension, may theoretically influence liver fibrosis progression and antiviral therapy response. However, its clinical significance in HBV-infected patients remains unclear. This study aimed to determine the prevalence of MASLD in chronic HBV, characterize associated demographic and metabolic features, and evaluate its impact on fibrosis and virological response.

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Materials and Methods:

We conducted a single-center retrospective study including 124 patients followed for chronic HBV infection. MASLD was defined as imaging-confirmed steatosis associated with at least one metabolic risk factor. Patients were classified into two groups: HBV + MASLD (Group A) versus HBV without steatosis (Group B). Demographic data, metabolic comorbidities (hypertension, diabetes, dyslipidemia), virological parameters (HBsAg, HBeAg, HBV DNA), fibrosis stage ($\geq F2$), and antiviral treatment response were collected. Statistical analyses included Chi-square and Mann-Whitney tests, with multivariate logistic regression for independent associations.

Results:

MASLD was identified in 50 patients (40.3%). Patients with MASLD were older (49.3 vs 46.2 years; $p=0.31$) and predominantly female (64% vs 41%; $p=0.023$). Metabolic comorbidities were more common in the MASLD group but did not reach statistical significance. Virological profiles, including HBsAg/HBeAg status and median HBV DNA (1870 vs 2450 IU/mL; $p=0.85$), were similar between groups. The prevalence of significant fibrosis ($\geq F2$) was comparable (28% vs 30%; $p=0.95$). Antiviral response rates did not differ (83% vs 78%; $p=0.75$).

Conclusions:

MASLD occurs in nearly 14% of patients with chronic HBV, more frequently in older individuals and women. In this cohort, MASLD did not aggravate fibrosis nor affect antiviral response, indicating it acts as a background metabolic factor rather than an aggravating cofactor. These findings support the continued use of standard antiviral strategies without adjustment solely based on MASLD presence. Clinicians should continue to monitor metabolic comorbidities to optimize overall liver health. Future prospective studies with larger cohorts are warranted to confirm these findings and clarify long-term outcomes in HBV patients with metabolic steatosis.

Keyword(s) : MASLD , HBV , Fibrosis

1669 - "Serum Prothrombin Induced by Vitamin K Antagonist II (PIVKA II) Biomarker in diagnosis of Hepatocellular Carcinoma"

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Hypothesis: PIVKA-II is a superior to AFP in as diagnostic marker for diagnosis of Hepatocellular carcinoma (HCC).

Materials and Methods: This cross-sectional observational study was conducted in the Department of Hepatology, Dhaka Medical college & Hospital, during the period from February 2024 to February 2025. Total 30 patients having cytological (FNAC proved) evidence of HCC irrespective of aetiologies were included in the study as case and another 30 patients with cirrhosis were included in control group and 28 have healthy group. Serum PIVKA-II and AFP levels were measured by (ELISA) method and compared. Quantitative data were analyzed by mean and standard deviation; and comparison was done between two groups by unpaired t test or Mann-Whitney U test. Qualitative data were analyzed by frequency and percentage; and comparison was done between two groups by Chi-Square (2) test or Fisher's Exact test. To determine the optimal cut-off values for PIVKA-II and AFP to diagnose HCC, ROC curves were created. Youden's index was calculated as an index of sensitivity and specificity

Results: Among 88 patients, 30 patients had HCC, age ranged from 30 to 81 years and 30 patients had cirrhosis without HCC, age ranged from 18 to 69 years and Healthy population 28 individual, age range from 19 to 75. Both serum AFP and PIVKA-II level were significantly higher in HCC patients compared to patients with cirrhosis (p value <0.001). The median PIVKA-II value was 66.8ng/ml (IQ range, 0.76-9.80) and 48.9 (IQ range 1.45-3.20 ng/ml) and 17.6ng/ml (IQ range , 0.50-1.70) in HCC ,cirrhosis and healthy individual groups respectively. The results showed that the AUC increased after the addition of serum AFP to the PIVKA-II level (AUC 0.882 vs 0.0.872 and 0.862 respectively, $p<0.001$). The prediction of HCC by PIVKA-II only the sensitivity was 80% and specificity was 86% (at the cut-off value 6.80 ng/ml) and by AFP alone 66.7% and 81%(at the cut-off value 58.5 ng/ml) respectively. Combination of serum AFP and PIVKA-II level increases the sensitivity to 90% at the cost of specificity to 84%. For combined performance of serum AFP and PIVKA-II in predicting early stage of HCC AUC was 0.882 (90%CI 0.813- 0.994). Youden's index found the best sensitivity and specificity 87% and 65% in correctly differentiating HCC stage from cirrhosis and healthy individual



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Conclusion: The results of the study highlighted the added value of PIVKA-II and AFP combination for HCC detection rather than a single biomarker alone.

Keyword(s) : HCC - Hepatocellular carcinoma, PIVKA-II- Protein Induced by Vitamin K Absence or Antagonist-II, AFP - Alpha-fetoprotein

1670 - Evaluation of anti HBs titers in children who received vaccine + HBIG at birth because their mothers were HBsAg positive in subsequent years

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Background / Hypothesis: The objective was to evaluate anti-HBs titers in children who received vaccine+HBIG at birth in subsequent years.

Materials and Methods: HBV test results obtained in subsequent years from children of female patients with chronic HBV infection who had received vaccine+HBIG at birth were requested. The study was initiated in 2019 following ethical committee approval. Evaluations were performed during routine follow-up visits through face-to-face interviews and by reviewing results available from the mothers or accessed via the automation system.

Results: Data from a total of 323 children were evaluated in the study. The children's birth years ranged from 1990 to 2024. The timing of HBV marker testing for children varied greatly; only 39 children (12%) were tested for HBV markers in the first year of life, while the rate of HBV testing in the first 5 years of life was 44.3% (143/323).

The number of children with negative anti-HBs titers : (≤ 9 IU/mL) was 132 (40.8%), those with low-titer positive titers (10-50 IU/mL) was 104 (32.2%), and those with positive titers (≥ 51 IU/mL) was 87 (26.9%). The distribution of children with negative antiHBs titers by birth year is as follows: 12 between 1990 and 1997; 3 between 1998 and 1999; 31 between 2000 and 2005; 41 between 2006 and 2010; 34 children between 2011 and 2015, and 11 children between 2016 and 2020. The periods after vaccination + HBIG administration during which anti-HBs was tested and negativity was detected were as follows: 45 children after 1-5 years; 36 after 6-10 years; 27 after 11-15 years; 16 after 16-20 years; and 8 children after 21 years or more. No acute or chronic HBV infection developed in any of the children.

Conclusion: A decrease in antibody titers over time, even falling below protective levels, is an expected outcome following HBV vaccination. This decline to below protective levels is more frequently observed in infants who received vaccine + HBIG at birth. In our study data, anti-HBs titers fell below the protective level within a few years in 40.8% of the children included in the study, and low antibody titers were determined in 32.2%. The rate of testing pregnant women's children for HBV was also found to be very low. It would be beneficial for both HBsAg-positive pregnant women and the physicians monitoring them to be thoroughly informed about the procedures to be performed during pregnancy, at delivery, and thereafter.

Keyword(s) :

1671 - "Bitter Remedy Bitter Consequence" Mahogany Seeds Causing Herb Induced Liver Injury

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Background / Hypothesis:

Herbal and traditional remedies are increasingly implicated in herb-induced liver injury (HILI), a subset of drug-induced liver injury. Mahogany (Swietenia spp.) seeds are commonly consumed for glycemic control despite absence of validated safety data. This case highlights mahogany seed-associated HILI and reinforces the necessity of eliciting detailed herbal intake history in patients presenting with unexplained hepatitis.

Materials and Methods: A 58-year-old female with type 2 diabetes mellitus consumed crushed mahogany seeds daily for approximately four weeks as an alternative therapy. She presented with jaundice and malaise. Laboratory evaluation demonstrated hepatocellular injury. Comprehensive investigations including viral hepatitis (A, B, C, E), autoimmune profile, metabolic tests, and abdominal imaging excluded other etiologies. Causality assessment was performed using the Roussel Uclaf Causality Assessment Method (RUCAM). The herbal agent was discontinued and the patient was managed conservatively with serial monitoring.

Results:

The patient exhibited marked transaminase elevation

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(>10× upper limit of normal) along with significant hyperbilirubinemia, consistent with acute hepatocellular injury. Total bilirubin levels were markedly elevated at presentation, correlating clinically with icterus. Following cessation of mahogany seed intake, there was progressive improvement in both serum bilirubin and liver enzymes, with near normalization observed over an 8-week period. The Roussel Uclaf Causality Assessment Method (RUCAM) score indicated a probable causal relationship between mahogany seed consumption and hepatotoxicity.

Conclusions: Mahogany seeds represent a potential hepatotoxic herbal agent capable of inducing clinically significant hyperbilirubinemia and hepatocellular injury. This case emphasizes the importance of recognizing unregulated herbal products as reversible causes of liver dysfunction. Prompt identification and withdrawal of the offending agent leads to favorable outcomes, highlighting the critical role of clinician vigilance and patient awareness in preventing HILI.

Keyword(s) : Mahogany seeds, Herb-induced liver injury, HILI, Drug-induced liver injury, DILI, Hyperbilirubinemia, Hepatocellular injury, Swietenia species, Herbal toxicity, Alternative medicine, Roussel Uclaf Causality Assessment Method, Jaundice, Reversible hepatotoxicity, Diabetes self-medication

1674 - CMV Hepatitis Masquerading as Prolonged Cholestatic Phase of Hepatitis A

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Background / Hypothesis:

Prolonged cholestatic hepatitis A is an uncommon complication that may lead to diagnostic confusion, particularly when accompanied by atypical biochemical abnormalities such as iron overload. Superimposed cytomegalovirus (CMV) infection in immunocompetent individuals further complicates the clinical picture and may mimic chronic liver diseases. This case highlights the importance of systematic evaluation to differentiate reversible hepatic insults from irreversible disorders such as hereditary hemochromatosis, thereby preventing misdiagnosis and overtreatment.

Materials and Methods: A 19-year-old immunocompetent male presented with progressive jaundice, pruritus, dark urine, and weight loss for seven weeks following acute hepatitis A infection. Investigations revealed marked cholestasis with disproportionately elevated bilirubin and mild transaminitis. Comprehensive evaluation included viral serology, CMV PCR, autoimmune and metabolic panels, iron studies, immunodeficiency profile, imaging, and liver biopsy. CMV infection was confirmed by positive CMV DNA PCR and serology. The patient received intravenous ganciclovir alongside supportive management and was monitored closely for clinical and biochemical response.

Results: Initial laboratory findings showed severe hyperbilirubinemia (61.75 mg/dL), elevated alkaline phosphatase, high transferrin saturation (95.7%), and ferritin (565 ng/mL). Liver biopsy demonstrated secondary iron overload without evidence of genetic hemochromatosis. CMV DNA levels were 3.5×10^3 copies/mL. Following antiviral therapy, bilirubin levels gradually decreased, CMV DNA became undetectable within three weeks, and symptoms improved significantly. Transient anemia and reactive thrombocytosis were observed as treatment-related effects. Over follow-up, both cholestasis and iron indices normalized without chelation therapy.

Conclusions: This case illustrates a rare triad of prolonged post-hepatitis A cholestasis, secondary reversible iron overload, and CMV hepatitis in an immunocompetent individual. The findings emphasize the need for detailed histological and virological assessment in persistent cholestasis with atypical biochemical patterns. Early recognition and targeted therapy lead to complete resolution, reinforcing the reversibility of secondary iron overload when the primary hepatic insult is addressed.

Keyword(s) : Prolonged cholestasis, Hepatitis A, CMV hepatitis, Secondary iron overload, Hyperbilirubinemia, Immunocompetent host

1675 - Magnitude of Clostridium Difficile Infection in Hospitalized Egyptian Cirrhotic Patients



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Background / Hypothesis:

Watery diarrhea is the cardinal clinical symptom of Clostridium Difficile infection that causes a spectrum of manifestations ranging from asymptomatic carrier state to severe fulminant disease with toxic megacolon. The basis for symptomatic responses range is related to host and pathogen factors. The aim was to study the prevalence of Clostridium Difficile infection in hospitalized cirrhotic patients and its possible risk factors.

Materials and Methods:

This study was carried out on 200 cirrhotic patients admitted to National Liver Institute hospital divided into 2 groups: 100 asymptomatic patients and 100 patients with diarrhea. Direct stool toxin detection was done using an assay named RIDASCREEN® Clostridium Difficile Toxin A/B test; an enzyme immunoassay (EIA) for the qualitative determination of toxins A and B of Clostridium difficile in stool.

Results:

Among 200 patients with mean age of (51.81±12.94), EIA revealed 16 and 32 positive cases in both groups. In group I; there was statistical significance in ALT, serum creatinine (p<0.05) and bilirubin level (p<0.01) as regard EIA test result. Also, statistical significance was found (p<0.05) as regard duration of antibiotic and PPI intake. Logistic regression analysis revealed that gender and duration of PPIs use were independent factors in group I while the patient's age, duration of antibiotic use and GGT serum level were independent factors in group II for clostridium difficile infection development.

Conclusions:

Clostridium difficile infection is not an uncommon infection in hospitalized cirrhotic patients. Risk factors were antibiotic intake, gastric acid suppression, old age, long hospital stay and/or history of malnutrition.

Keyword(s) : Clostridium Difficile; Cirrhotic; Magnitude

1678 - Identification and validation of cuproptosis related genes for early warning of acute liver failure

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Background / Hypothesis:

Acute liver failure (ALF) is a life-threatening syndrome characterized by rapid onset of liver dysfunction. Cuproptosis, a copper-dependent form of programmed cell death, is increasingly recognized as a potential contributor to liver injury. However, the potential involvement of cuproptosis-related genes (CRGs) in ALF has not been fully investigated. Herein, we aimed to identify and validate CRGs that may play an early warning role in the pathogenesis of ALF.

Materials and Methods:

We analyzed GEO microarray datasets related to ALF (GSE14668, GSE38941, and GSE96851) to identify differentially expressed genes (DEGs). These DEGs were then intersected with key module genes obtained from Weighted Gene Co-Expression Network Analysis (WGCNA) to define ALF-specific DEGs. Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) enrichment analyses were subsequently performed on these ALF-specific DEGs. Further intersection with cuproptosis-related genes (CRGs) yielded ALF-specific cuproptosis-related genes (ALF-CRGs). A nomogram model based on these hub genes was constructed and validated using an independent dataset (GSE62029). The model's early warning performance was assessed by Receiver Operating Characteristic (ROC), calibration curves, and decision curve analysis (DCA). Experimental validation was performed using quantitative real-time PCR in D-Galactosamine/Lipopolysaccharide (D-GalN/LPS)-induced ALF mice and immunohistochemistry (IHC) in clinical liver tissues. To explore the underlying regulatory pathways, we analyzed immune infiltration and its correlations, and predicted miRNA/transcription factor (TF) networks.

Results:

Nine ALF-CRGs were identified, among which four hub genes (FDX1, ATP7B, CYP1A1, and MT1M) were selected through machine learning. The nomogram constructed using these hub genes demonstrated excellent early-warning performance (AUC > 0.90).

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Experimental validation confirmed that FDX1, ATP7B, and MT1M were downregulated in liver tissues from ALF mice and patients, while CYP1A1 expression was confirmed at the transcript level. Immune infiltration analysis suggested that these CRGs may influence immune microenvironment alterations during ALF. The miRNA/TF network prediction further identified potential upstream regulators of these hub genes. Conclusions:

Our findings support a critical role of cuproptosis in ALF and identify several hub genes with potential as early-warning biomarkers. The validated hub genes and their regulatory networks provide new insights into potential therapeutic targets for ALF.

Keyword(s) : cuproptosis, acute liver failure, early warning, bioinformatics, machine learning, validation

1680 - Artificial Intelligence Predicts Sex Specific Risk of Metabolic Dysfunction Associated Steatotic Liver Disease

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is known to exhibit sex differences in epidemiology, metabolic drivers, the progression to fibrosis, and extrahepatic consequences. However, whether artificial intelligence (AI) models learn and perform differently across sexes remains unclear. This project aimed to train sex-specific machine learning (ML) classifiers based upon routinely collected clinical variables to evaluate sex-specific patterns of learning and diagnostic performance for both the presence of MASLD and the severity of MASLD.

Materials and Methods:

In this cross-sectional study, a total of 446 adults undergoing routine evaluation at a cardiology referral center were enrolled. Presence (no vs yes) and severity of MASLD (no steatosis vs mild vs moderate-to-severe steatosis) were assessed by abdominal

ultrasonography. Participants underwent train/test splitting (7:3) into a training set (127 men, 185 women) and an independent test set (55 men, 79 women). Eight ML algorithms were trained using two methods: (1) combined-sex models where the training data was the full population dataset, and (2) sex-specific models with separate training for men and women. Model performance to predict the presence and severity of MASLD was evaluated on the unseen test dataset by calculating the area under the curve (AUC) and F1-score. An additional analysis based upon thresholds for rule-in and rule-out was performed. Sex-specific feature importance rankings were compared to assess differential patterns of learning.

Results:

MASLD prevalence was 63.6%, with 41.2% of patients having mild and 22.4% having moderate-to-severe steatosis. Sex-specific modeling improved diagnostic performance in men but remained suboptimal in women. For MASLD presence, AUC/F1-scores of the top-performing models were 0.769/0.856 in the overall data set, 0.793/0.897 in men, and 0.681/0.794 in women. For MASLD severity, the corresponding scores were 0.761/0.671 overall, 0.723/0.608 in men, and 0.718/0.571 in women. Threshold-based analyses demonstrated acceptable rule-in and rule-out performance in men, whereas both capacities were limited in women. Notably, feature importance profiles differed markedly by sex, showing distinct learning patterns.

Conclusions:

ML classifiers trained on routine clinical data reveal pronounced sex-specific learning patterns in MASLD prediction. These models perform better in men, suggesting that widely used clinical variables are more informative for male MASLD phenotypes, while failing to capture female-specific determinants. The observed divergence underscores the need for sex-stratified model development, incorporation of female-specific metabolic and hormonal factors, and balanced datasets to reduce diagnostic inequities. Integrating sex-specific analytic frameworks may enhance ML-driven MASLD risk stratification and support more equitable, individualized clinical care.

Keyword(s) : MASLD; Sex differences; Machine learning; Artificial intelligence; Deep learning; Predictive



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modeling; Diagnostic performance; Cardiometabolic risk; Precision medicine

1682 - AWARENESS OF HEPATITIS B VIRUS REACTIVATION AMONG PHYSICIANS MANAGING PATIENTS RECEIVING IMMUNOSUPPRESSIVE THERAPY A CROSS SECTIONAL STUDY OF 150 PHYSICIANS

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Background / Hypothesis: Hepatitis B virus (HBV) reactivation is a clinically important issue that can result in severe outcomes among patients receiving immunosuppressive therapy. The risk of reactivation depends on the degree and duration of immunosuppression and the patient's serological status. International guidelines recommend comprehensive HBV screening in all candidates for immunosuppressive therapy and advise antiviral prophylaxis or close monitoring based on individual risk. This study aimed to assess physicians' knowledge, screening habits, and clinical management related to HBV reactivation.

Materials and Methods: This cross-sectional study was conducted using an online questionnaire completed by 150 physicians from various specialties involved in the management of patients undergoing immunosuppressive treatment. The 30-item survey evaluated demographic characteristics, frequency of prescribing immunosuppressive therapy, HBV screening practices, antiviral prophylaxis preferences, management strategies in suspected reactivation, and familiarity with guideline recommendations. Descriptive statistics were used for analysis.

Results: Specialties of the participating physicians included Infectious Diseases (n=41), Internal Medicine (n=29), Dermatology (n=21), Neurology (n=18), Pulmonology (n=14), Oncology (n=12), Hematology (n=8), and Rheumatology (n=6).

A total of 94.7% of physicians reported that immunosuppressive treatments increase the risk of HBV reactivation, and the same proportion acknowledged that reactivation can occur in anti-HBc

(+)/HBsAg (-) individuals. Additionally, 7.9% believed anti-HBs positivity completely eliminates reactivation risk. Routine HBV screening before immunosuppressive therapy was performed by 82.9%, while 13.2% screened only selected patients and 3.9% did not screen at all. Frequently requested tests included HBsAg (90.8%), anti-HBs (78.9%), and anti-HBc (59.2%).

Regarding antiviral prophylaxis duration, 56.6% adjusted duration according to risk, whereas 23.7% preferred six months and 14.5% preferred twelve months of prophylaxis.

Timing preferences for antiviral initiation showed that most physicians started therapy before immunosuppression: 60.3% one month before, 24.7% one week before, and 17.8% on the same day. Only 2.7% initiated antiviral therapy after immunosuppressive treatment had already begun, a clinically relevant finding due to the potential risk of reactivation. The APASL 2021 guideline recommends starting antiviral prophylaxis prior to immunosuppressive therapy.

For suspected HBV reactivation, 51.3% of physicians ordered HBV DNA and ALT as initial tests, while 25% preferred consultation with a specialist.

Conclusions: Although awareness of HBV reactivation risk was generally high, considerable heterogeneity was observed in HBV serologic screening, anti-HBc test utilization, initiation of prophylaxis, and duration of antiviral therapy. Notably, 40.8% of physicians did not request anti-HBc before initiating immunosuppressive treatment. These findings highlight the need for improved guideline adherence and targeted educational strategies to reduce HBV reactivation risk in immunosuppressed patients.

Keyword(s) : HBV reactivation, Immunosuppressive therapy, Antiviral prophylaxis

1683 - Hepatic Steatosis Beyond Fat A Clinical Anthropometric and Metabolic Exploration of an At Risk Population

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Background / Hypothesis:

Metabolic dysfunction-Associated Steatotic Liver Disease (MASLD) has emerged as the dominant form of liver steatosis, strongly driven by metabolic abnormalities. Identifying clinical and biochemical features associated with progression is essential. This study aimed to characterize the anthropometric, metabolic and fibrosis profiles of patients with hepatic steatosis and to explore factors associated with evolution toward MASH.

Materials and Methods:

A retrospective study was performed over 7 years, including 103 adults with ultrasound-confirmed steatosis. Demographic variables, anthropometric measurements, metabolic risk factors, biochemical markers, lipid profiles, non-invasive fibrosis scores (FIB-4, APRI) and ultrasound findings were collected. Cardiometabolic comorbidities and weight evolution during follow-up were assessed. MASLD was defined as steatosis with at least one cardiometabolic abnormality. Exploratory comparisons were conducted between MASH and non-MASH groups.

Results:

The mean age was 51 ± 11.7 years, with women representing 83.5% of the cohort. Mean BMI was 30.8 ± 4.8 kg/m², with 52.5% obesity and 41.4% overweight. Diabetes was present in 25.2%, hypertension in 24.3%, dyslipidemia in nearly half of patients, and coronary artery disease in 7.8%. MASLD criteria were fulfilled in 68% of cases, and MASH was diagnosed in 10.7%. Biochemical analyses showed only moderate elevations in ALT (30 ± 25.6 IU/L), AST (27 ± 19 IU/L), and GGT (68 ± 83.7 IU/L), while the lipid profile confirmed substantial metabolic impairment. Advanced fibrosis was uncommon, with high-risk FIB-4 found in 2.9% and elevated APRI in 5.7%. Among the 84 fibrosis assessments, F1 predominated (64.3%), while F3–F4 accounted for only 8.4%. Severe steatosis (S3) was observed in 38.2% of patients. During a mean follow-up of 32.2 months, weight loss remained limited (2.1% on average), and only 15.3% achieved $\geq 5\%$ reduction. Type 2 diabetes emerged as the only metabolic feature enriched in MASH patients (45.4% vs 22.8%), whereas obesity (36.3% vs 52.2%) and hypertension (27.3% vs 23.9%) did not differ significantly, suggesting no association with disease progression in this cohort.

Conclusions:

This real-world cohort underscores the heavy

metabolic burden of MASLD. While advanced fibrosis was infrequent, the presence of MASH and the minimal weight reduction achieved over time highlight the need for more intensive, diabetes-focused metabolic interventions to modify disease trajectory.

Keyword(s) : MASLD , DT2 , MASH

1684 - Patient reported health is decreased in high risk MASH defined by noninvasive biomarkers of fibro inflammation in Japanese Adults

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Background / Hypothesis:

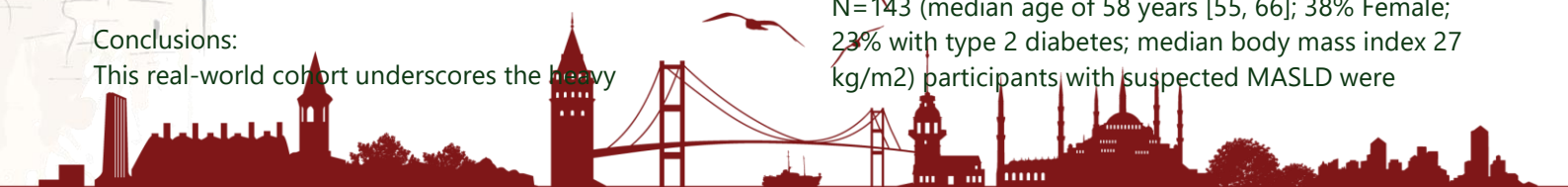
Studies on how individuals with metabolic dysfunction-associated steatotic liver disease (MASLD) or steatohepatitis (MASH) report their health are sparse, particularly in the non-North-American population. Change in patient reported outcomes is an important factor in assessing improvement following interventions and a reliable baseline and its association with biomarkers is needed. We aim to demonstrate how commonly used noninvasive biomarkers in MASLD/MASH associate with patient-reported health.

Materials and Methods:

Health-related quality of life (HRQoL) data was collected through the EQ-5D-5L instrument as part of a single-site prospective observational study at Shin-yurigaoka General Hospital, Japan. The analyses consider the dimensions of health scoring, Japanese specific index values and biomarker metrics. Lower health scores suggest poorer HRQoL. High risk disease states were defined as corrected T1 (cT1) ≥ 875 ms; liver fat content (LFC) $\geq 10\%$; magnetic resonance elastography (MRE) ≥ 3.54 kPa; vibration-controlled transient elastography (VCTE) ≥ 12 kPa; Enhanced liver fibrosis (ELF) ≥ 9.8 and FIB-4 ≥ 1.3 and MASH as cT1 ≥ 800 ms and LFC $\geq 5\%$.

Results:

N=143 (median age of 58 years [55, 66]; 38% Female; 23% with type 2 diabetes; median body mass index 27 kg/m²) participants with suspected MASLD were



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recruited. The domain where patients most commonly reported problems were anxiety and depression (29%), followed by ability to perform usual activities (5%). The mean [SD] EQ-5D-5L health score was 0.88 [10] and decreased slightly with age ($R_s = -0.22$; $p < 0.05$) but was not associated with sex. Correlations were observed between health score and cT1, MRE, VCTE, ELF and FIB-4, (-0.40; -0.53; -0.42; -0.45; -0.40; all $p < 0.001$), while no correlation was observed with liver fat content. The mean health score was significantly lower in those with elevated cT1, MRE, VCTE, ELF and FIB-4 (0.82 [12], 0.81 [12], 0.86 [11], 0.83 [11] and 0.85 [11]; $p < 0.001$, respectively) compared to those without, while elevated LFC did not impact the health score. This can be compared to a mean health index in a Japanese population (40–79 years old) of 0.88–0.95 and in a global MASH trial population of 0.85.

Conclusions:

Reduced patient-reported health was seen for those with elevated noninvasive biomarkers for high-risk MASH or fibrosis, but not for steatosis. These HRQoL results may increase the awareness of disease burden on patients, and longitudinal research is warranted to better understand how changes in liver fibro-inflammation is associated with improved patient reported health outcomes.

Keyword(s) :

1685 - Chronic Portal Vein Thrombosis and Risk of Decompensation Experience of a Moroccan Center

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Background / Hypothesis:

Chronic portal vein thrombosis (PVT) represents a major cause of non-tumoral portal hypertension (PHT) and exposes patients to a substantial risk of clinical decompensation. However, reliable predictors of this outcome remain insufficiently defined. We hypothesized that specific clinical and radiological variables, particularly the degree of liver fibrosis and the anatomical extension of thrombosis, could independently predict PHT decompensation in patients with chronic PVT.

Materials and Methods:

We conducted a retrospective, monocentric study including all patients with chronic PVT managed between 2010 and 2025 in a Moroccan tertiary referral center. Eligible patients were aged ≥ 18 years, had chronic liver disease or cirrhosis, and radiologically confirmed chronic PVT. Exclusion criteria included hepatocellular carcinoma and tumor-related thrombosis. Collected variables comprised demographic data (age, sex), comorbidities, hepatic and thrombophilic etiologies, and detailed radiological characteristics (type of thrombosis, degree and direction of extension, involvement of portal branches, splenic vein, and hepatic fibrosis stage). The primary endpoint was PHT decompensation. Factors associated with decompensation were analyzed using univariate analysis (significance threshold $p < 0.05$), followed by multivariate logistic regression to identify independent predictors.

Results:

Eighty-three patients were included (mean age 44.9 ± 13.7 years; 77% female). PHT decompensation occurred in 31 patients (37.3%). In univariate analysis, advanced fibrosis F3–F4 (OR 4.91; 95%CI 1.36–17.68; $p = 0.015$), hepatitis C etiology (Fisher $p = 0.049$), and extension to the left portal branch (OR 3.06; 95%CI 1.02–9.16; $p = 0.045$) were significantly associated with decompensation. Extension to the splenic vein showed a strong trend without reaching significance (OR 3.14; $p = 0.052$). Other parameters—including thrombosis type, patient comorbidities, age, sex, and thrombophilic etiologies—were not significantly associated. In multivariate analysis, only advanced fibrosis remained an independent predictor of PHT decompensation (adjusted OR 4.19; 95%CI 1.08–16.26; $p = 0.038$).

Conclusions:

In this chronic PVT cohort, PHT decompensation was frequent, affecting more than one-third of patients. Advanced liver fibrosis (F3–F4) emerged as the strongest independent predictor of decompensation, while extension of thrombosis to the left branch or splenic vein demonstrated a contributive but non-significant trend after adjustment. These findings underscore the central role of fibrosis evaluation in risk stratification, management decisions, and long-term follow-up of patients with chronic PVT.

Keyword(s) : PVT, portal hypertension, fibrosis

1686 - Preoperative volume estimation versus actual graft weight in living donor liver



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transplantation a retrospective analysis

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Background / Hypothesis:In living donor liver transplantation (LDLT), accurate estimation of liver graft weight is critical to ensure adequate graft-to-recipient weight ratio (GRWR), ideally greater than or equal to 1% of recipient body weight, while minimizing donor risk. Insufficient GRWR is associated with increased complications and mortality. Preoperative radiological volumetric assessments are essential, but discrepancies of 10% or greater between calculated volume and measured weight are occasionally reported. This study compares preoperatively calculated liver volume with measured graft weight during surgery and analyzes factors contributing to significant discrepancies at Acibadem Atakent Hospital.

Materials and Methods:This retrospective study was conducted at the Organ Transplant Unit of Acibadem Atakent Hospital. Six hundred twenty-three donors who underwent hepatectomy between November 2015 and November 2024 were included. Data collected from patient records included donor demographics, preoperative laboratory values, calculated liver volume, and actual excised graft weight from operative notes. Statistical analysis (SPSS) assessed agreement between calculated volume and measured weight. Cases with 10% or greater discrepancy were analyzed to identify risk factors.

Results:The cohort included 623 donors (45.9% female, 54.1% male). Mean calculated estimated liver volume was $1216.30 \pm 139.81 \text{ cm}^3$, and mean excised graft weight was $733.82 \pm 270.73 \text{ grams}$. Statistically significant positive correlation existed between estimated liver volume and total liver volume ($r=0.684$, $p=0.001$). Notably, 298 cases (47.8%) exhibited 10% or greater difference between calculated volume and measured weight. Cases with 10% or greater discrepancy had statistically significantly lower liver Hounsfield unit values ($p=0.011$) and higher hepatic steatosis rates ($p=0.001$) compared to cases with less than 10% difference. Additional findings showed

higher cholesterol ($p=0.003$) and triglyceride levels ($p=0.001$) in the high-discrepancy group.

Conclusions:The substantial discrepancy rate (47.8%) highlights challenges in accurately predicting graft weight using preoperative radiological methods. Significant discordance was strongly associated with lower liver Hounsfield unit values and increased steatosis. Preoperative hepatic steatosis represents a critical factor contributing to volume-weight mismatch in LDLT donors. Enhanced preoperative assessment including steatosis evaluation may improve graft weight prediction and guide donor selection, potentially improving safety for both donors and recipients.

Keyword(s) : living donor liver transplantation, graft volume, graft weight, hepatic steatosis, Hounsfield unit

1687 - Validation of Agile 3+ and Agile 4 for MASLD with advanced fibrosis and cirrhosis in a Canadian cohort

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Background / Hypothesis:

Agile 3+ and Agile 4 are combined non-invasive tests (NIT) based on parameters that include age, AST/ALT, platelets, gender, presence of diabetes and liver stiffness measurement (LSM by VCTE), and proposed to identify patients with advanced fibrosis or cirrhosis in patients with metabolic dysfunction-associated steatotic liver disease (MASLD). There are few external validation studies. Our aim was to assess the diagnostic performance of Agile 3+ and Agile 4 in comparison with other simple serum markers scores, and LSM by VCTE, for the identification of MASLD advanced fibrosis ($\geq F3$) and cirrhosis on biopsy.

Materials and Methods:

This is a retrospective single-center cohort study that included adult patients with biopsy-proven MASLD between 2010 – 2025. Alternate causes of liver disease were excluded, and patients included based on clinical review, available laboratory parameters, VCTE, and histopathology. Diagnostic test performance was

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assessed using validated thresholds for NIT and Agile 3+ <0.451 and ≥ 0.679 to rule-out and rule-in $\geq F3$ using the area under the receiver operating characteristic (AUROC). For Agile 4 thresholds for NIT were <0.251 and ≥ 0.565 to rule-out and rule-in cirrhosis

Results:

Our cohort included 175 patients, with mean age 50 ± 13 years, mostly male (53.3%), mean BMI 31.7 ± 5.7 kg/m² with prevalence of diabetes mellitus 43.8%, F3-4 63.4%, and cirrhosis 33.3%. Using established thresholds, the proportion of patients with indeterminate scores were Agile+3 (0.451-0.679) 15.7%, LSM (8-12 kPa) 29%, FIB-4 (1.3-2.67) 39.3%, NFS (-1.455-0.676) 35%, APRI (0.5-1.5) 43.2%. For our overall cohort, all NITs had high specificity for F3-4, with AUROC > 0.80 for Agile 3+ and VCTE. Agile 4 had an AUROC 0.79 ($p < 0.0001$) with a sensitivity of 33.3% and specificity of 92.9% to rule-in cirrhosis. Proportion of patients with indeterminate scores for Agile 4 (0.251-0.565) 10.6%, FIB-4 (1.67 – 3.48) 30.8% and APRI (1 – 2) 17.1%.

Conclusions:

In our tertiary center cohort, Agile3+ had a good diagnostic performance for advanced fibrosis, with relatively lower proportion of patients classified in the indeterminate zone at specified thresholds. Agile 4 showed good performance to rule in cirrhosis. Further validation of Agile 3+ and Agile 4 in lower prevalence cohorts is required.

Keyword(s) : agile, biopsy, fibrosis, non-invasive

1688 - Evaluation of HBsAg anti HBcIgG anti HBs and anti HCV test results for individuals who visited primary level Family Health Centers (12 years of data covering the province of Izmir and its districts)

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Background / Hypothesis: The aim was to evaluate the

results of HBsAg, anti-HBcIgG, anti-HBs, and anti-HCV tests performed on individuals who visited primary care Family Health Centers in the province of Izmir, located in western Turkey.

Materials and Methods: Blood samples taken from individuals living in Izmir province and its districts at Family Health Centers are processed at the Provincial Public Health Laboratory; results can be retrieved from the automation system. HBsAg, anti-HBcIgG, anti-HBs, and anti-HCV tests performed over the last 12 years (2012-2024) were examined using patient numbers in accordance with the Personal Data Protection Law; after excluding duplicate data, the distributions by age, gender, and year were evaluated. Ethical committee approval and Provincial Health Directorate permits were obtained for the study. Test results from individuals who visited Izmir City Hospital between October 2023 and October 2024 were evaluated using the same method as the control group.

Results: According to records from the specified years (2012-2024), among the total of 346,952 individuals who underwent HBsAg testing at the primary level, HBsAg positivity was 2.28%; anti-HBcIgG was detected in 10.64% of 56,292 individuals; anti-HBs was detected in 42.1% of 301,842 individuals; and anti-HCV was detected in 0.57% of 256,034 individuals. Among individuals who visited the hospital, these rates were 2.56% among 56,260 individuals for HBsAg; 24% in 17,312 individuals tested for anti-HBcIgG; 46.9% in 42,369 individuals tested for anti-HBs; and 0.34% in 45,673 individuals tested for anti-HCV (Table 1).

According to the Family Health center examinations, the age groups with the highest prevalence of HBsAg positivity are those born between 1973 and 1941 (52-84 years old); followed by those born between 1984 and 1974 (aged 41-51) and those born between 1990 and 1985 (aged 35-40). Similar results were found among hospital patients, with seropositivity increasing with age. Anti-HBcIgG positivity also increases significantly with age, reaching 40-50% seropositivity in those over 50 years of age. Anti-HBs positivity is significantly higher in those born after 1998, when the national HBV vaccination program began. Anti-HBs positivity in older age groups is thought to be associated with past HBV infection. Anti-HCV positivity is also quite low in the community, and it is noteworthy that individuals with positivity are mostly aged 60 and above.

Conclusion: In conclusion, the national HBV vaccination program has significantly reduced HBsAg positivity and the rate of infection through exposure to the virus in children and young adults.

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Keyword(s) :

1689 - Rapidly Progressive Acute Liver Failure Caused by Infiltrative Hepatic Amyloidosis A Case Report

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Background: Hepatic amyloidosis is a rare manifestation of systemic amyloidosis. While mild liver enzyme abnormalities are common, presentation as acute liver failure (ALF) is exceedingly rare and carries a high mortality risk.

Case Presentation: We report the case of a 71-year-old man with progressive jaundice and cholestatic liver enzyme abnormalities. Initial suspicion of drug-induced liver injury due to recent statin initiation was excluded. Laboratory evaluation revealed marked hyperbilirubinemia, coagulopathy, and hypoalbuminemia. Liver biopsy demonstrated massive sinusoidal amyloid deposition with Congo red positivity and apple-green birefringence, confirming an infiltrative pathology. Immunohistochemistry was negative for AA amyloid, and no monoclonal gammopathy was detected. Despite supportive therapies including plasmapheresis and hemodialysis, the patient developed multiorgan failure and died.

Conclusion: Massive sinusoidal amyloid deposition can lead to rapidly progressive ALF with high mortality. Early tissue diagnosis is critical to identify infiltrative hepatic disease and to avoid delays in management, including consideration of liver transplantation.

Keyword(s) : Hepatic amyloidosis, Acute liver failure, Sinusoidal amyloid deposition, Cholestasis

1691 - Effects of biological agents on hepatitis B virus reactivation in patients who are negative for hepatitis B surface antigen and positive for antibodies against hepatitis B core antigen

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Background / Hypothesis:

In HBsAg-negative, anti-HBc IgG-positive individuals, the risk of hepatitis B virus reactivation (HBVr) associated with immunosuppressive therapy varies depending on the immunosuppressive potency of the biological agent used. This study aimed to evaluate the development of HBVr in HBsAg-negative, anti-HBc IgG-positive patients receiving medium- or high-risk biological agents.

Materials and Methods:

A total of 172 patients identified as HBsAg-negative and anti-HBc IgG-positive were retrospectively evaluated. Patients were classified into moderate- and high-risk groups according to the HBVr risk associated with the biological agents used. Rituximab and ocrelizumab were categorized as high-risk agents, while anti-TNF- α agents (adalimumab, infliximab, golimumab, certolizumab, etanercept), the IL-6 inhibitor tocilizumab, and the IL-12/23 inhibitor ustekinumab were considered moderate-risk agents. Demographic and clinical characteristics, treatment details, concomitant immunosuppressive medications, and antiviral prophylaxis status were recorded. Virological and serological data obtained during follow-up were reviewed, and the occurrence of HBVr was assessed.

Results:

The mean age of the study population was 52.3 ± 11.0 years. The most common indications for biological agent therapy were ankylosing spondylitis (30.2%) and rheumatoid arthritis (29.7%). While 20.9% (n=36) of patients received high-risk agents, 79.1% (n=136) received moderate-risk agents. The median follow-up duration was 28.5 months (12–60 months) in the high-risk group and 36 months (12–96 months) in the moderate-risk group. All patients in the high-risk group received antiviral prophylaxis at treatment initiation, and no HBVr events were observed. In contrast, only 33.1% (n=45) of patients in the moderate-risk group received prophylaxis, while 66.9% (n=91) were monitored without prophylaxis. Among those not receiving prophylaxis, only one patient experienced virological reactivation without clinical or biochemical hepatitis. This patient had an initial anti-HBs level of ≥ 1000 IU/L, which remained stable during follow-up.

Conclusions:

Antiviral prophylaxis is effective in preventing HBV reactivation in HBsAg-negative, anti-HBc IgG-positive

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individuals receiving moderate- or high-risk biological agents. For patients treated with moderate-risk agents, regular virological monitoring may serve as a safe alternative to universal prophylaxis, allowing timely identification and management of the rare reactivation events that occur.

Keyword(s) : Hepatitis B Virus Reactivation, Biological Agents, Antiviral Prophylaxis

1692 - Fibrosis Regression Patterns in Chronic Hepatitis B Evidence from Longitudinal Non Invasive Biomarkers

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Introduction:

Liver fibrosis in chronic hepatitis B is a dynamic process that may improve with sustained antiviral therapy. Non-invasive biomarkers such as FIB-4 and APRI are increasingly used to monitor fibrosis over time, yet real-world data describing how fibrosis evolves under treatment or surveillance remain limited. This study aimed to assess fibrosis progression, stability, or regression using non-invasive scores in treated and untreated patients.

Material and Methods:

A retrospective, descriptive, and analytical study was conducted over eight years in a gastroenterology department and included 125 chronic hepatitis B patients. FIB-4 and APRI comparisons before and after antiviral therapy were performed using the Wilcoxon signed-rank test. Fibrosis evolution was categorized as regressive, stable, or progressive based on predefined score variations.

Results:

A total of 125 patients were included in the study, with a mean age of 46.9 ± 12.3 years and a sex ratio M/F of 1.15.

Regarding past medical history, 18(14.4%) were active smokers, 10(8%) had diabetes, 8(6.4%) reported alcohol consumption, 7(5.6%) had dyslipidemia.

Clinically, asthenia was the most frequent presenting symptom, reported in 46(36.8%) patients, followed by abdominal pain in 28(22.4%). Signs of portal hypertension were noted in 10(8%) cases while upper

gastrointestinal bleeding (hematemesis or melena) was observed in 3(2.4%) patients.

Baseline imaging revealed hepatic steatosis in 22(17.6%), chronic hepatopathy in 16(12.8%), radiological signs of portal hypertension in 12(9.6%), and hepatocellular carcinoma in 3(2.4%).

Fibrosis assessment methods included FibroScan in 56(44.8%) patients, liver biopsy in 56(44.8%), and combined evaluation in 5(4%). Before treatment, fibrosis stages were distributed as follows: F0 in 14(11.2%), F1 in 76(60.8%), F2 in 22(17.6%), F3 in 2(1.6%), and F4 in 11(8.8%). Median non-invasive fibrosis scores at baseline were FIB-4=1.65[0.81–2.48] and APRI=0.57[0.26–1.11].

Regarding antiviral management, tenofovir disoproxil fumarate was prescribed in 75 (60%) patients, entecavir in 5(4%), whereas 38(30.4%) were managed under surveillance without antiviral therapy.

After 18 months, 91(72.8%) patients had evaluable follow-up. Median FIB-4 decreased to 0.90[0.62–1.40] ($p=0.006$), and APRI declined to 0.25[0.19–0.37] ($p<0.001$). According to biological evolution scoring, Fibrosis regression was observed in 58(64.2%) patients, stability in 17(18.6%), and progression in 16(17.4%). Among patients under surveillance, no significant change in FIB-4 was observed (1.47vs1.30; $p=0.688$), while APRI showed a mild, non-significant decline (0.435vs0.281; $p=0.375$).

Conclusions:

Antiviral therapy significantly improved non-invasive fibrosis scores, reflecting meaningful fibrosis regression in most patients despite varied baseline stages. These findings highlight the antifibrotic benefit of sustained viral suppression and support the routine use of non-invasive biomarkers for long-term fibrosis monitoring.

Keyword(s) : Hepatitis C Fibrosis Non Invasive biomarkers FIB4 APRI

1693 - Evaluation of Artificial Intelligence in Clinical Decision Support Application to Chronic Hepatitis B Cases According to the 2025 EASL Guidelines

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Background / Hypothesis:

Chronic hepatitis B (HBV) encompasses a broad

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spectrum of clinical presentations requiring individualized therapeutic strategies. The 2025 EASL guidelines introduce updated recommendations for antiviral treatment, including treatment initiation criteria, preferred therapeutic options, monitoring of virological response, and criteria for treatment discontinuation.

The objective of this study was to assess the conformity of clinical decisions generated by an artificial intelligence model in a series of chronic hepatitis B clinical scenarios, comparing them with the 2025 EASL guideline recommendations. The evaluation focused on therapeutic indications, the choice of antiviral agents, virological response management, and decisions regarding treatment cessation.

Materials and Methods:

We conducted a retrospective descriptive study based on a comparative analysis of 22 fictitious clinical cases of patients followed for chronic HBV infection. These cases were classified into three categories according to complexity: simple cases, specific cases, and complex cases.

For each scenario, ChatGPT's responses concerning treatment indications, antiviral drug selection, virological response management, and treatment discontinuation decisions were compared with the 2025 EASL guidelines to assess their conformity.

Results:

Therapeutic indications showed full compliance (100%) in simple cases, despite occasional limitations in the treatment options proposed. In complex cases, indications also reached 100% compliance; however, the therapeutic propositions were incomplete in 50% of situations. For example, in pregnant women, ChatGPT mentioned only tenofovir disoproxil fumarate and omitted tenofovir alafenamide, now recommended by EASL 2025.

Among specific cases, compliance with indications and treatment choices remained high (100%), but omissions were frequent (42.85%), particularly regarding specialized treatments such as interferon for hepatitis delta or immunoglobulins in transplant recipients.

Concerning virological response management, treatment strategies were generally appropriate in 66.66% of cases. Non-compliant management was mostly observed in cirrhotic patients lacking virological response.

Regarding treatment discontinuation,

recommendations were correctly applied in most situations (66.66%). Nevertheless, some errors persisted, notably in HBeAg-negative, non-cirrhotic patients in whom management diverged from guideline indications.

Conclusions:

This study demonstrates that ChatGPT generally aligns well with the 2025 EASL guidelines for chronic hepatitis B management but exhibits notable gaps, particularly in the selection of specific therapeutic options. These findings underscore the essential role of clinical expertise and the need for continuous updating of artificial intelligence tools to ensure optimal patient care.

Keyword(s) : Chronic Hepatitis B , Artificial Intelligence , EASL 2025 Guidelines

1695 - CHATGPT FOR PATIENT QUESTIONS ABOUT CIRRHOSIS USEFUL BUT NOT INFALLIBLE

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Background / Hypothesis:

Patients with cirrhosis often lack knowledge about how to manage and prevent complications related to their disease. ChatGPT, a natural language processing model developed by OpenAI, could offer support by providing clear and understandable answers to patients. While it has been used in medical applications, such as test preparation and report writing, few studies have evaluated its ability to accurately answer clinical questions specific to diseases like cirrhosis. This study aims to evaluate the accuracy and completeness of ChatGPT's responses in order to assess its effectiveness as an informational tool for patients.

Materials and Methods:

We analyzed 44 frequently asked questions about cirrhosis posed by patients in Facebook support groups, categorizing them into four

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groups: basic knowledge (19 questions), diagnosis (3 questions), treatment (15 questions), and lifestyle (7 questions). A liver

specialist evaluated the answers on a scale from 1 to 4:

1. Complete
2. Correct but insufficient
3. Contains both correct and incorrect information
4. Completely incorrect

Results:

The results show that for basic knowledge, 47.3% of answers were complete, 31.5% were correct but insufficient, and 21.05% contained incorrect information. Regarding diagnosis, 66.6% of answers were correct but lacked detail, while 33.3% were complete. For treatment, 33.3% of answers were complete, 40 % were correct but insufficient, and 20% contained errors. Finally, for lifestyle, 42.8% of answers were complete, 28.5% were correct but insufficient, and 14.28% contained errors. Notably, no ChatGPT answer was deemed completely incorrect.

The model demonstrated its ability to provide detailed answers to questions related to basic knowledge and lifestyle. It offered comprehensive explanations of the symptoms, etiology, and prognosis of both compensated and decompensated cirrhosis, as well as risk factors and lifestyle changes that may influence outcomes. While the model provided correct answers in areas like diagnosis and treatment, most responses were rated as correct but insufficient.

Conclusions: This study shows that while the model was able to provide complete and useful answers on basic and lifestyle topics, there is still room for improvement, particularly in more technical areas such as diagnosis and treatment of cirrhosis. It is crucial that medical information is regularly updated to ensure its accuracy and relevance. Additionally, this analysis emphasizes the importance of consulting healthcare professionals for personalized medical advice tailored to each individual situation.

Keyword(s) : Cirrhosis ; ChatGPT

1696 - Characterizing Primary Biliary Cholangitis in Men Clinical Biological and Histological Profile

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Background / Hypothesis:

Primary biliary cholangitis (PBC) predominantly affects women, and male presentation remains uncommon and insufficiently characterized. Emerging evidence suggests that men may present with more advanced disease and a distinct clinical course. This study aimed to describe the clinical, biochemical, immunological, and histological features of male PBC patients and to provide an exploratory comparison with female patients in a single-center cohort.

Materials and Methods:

We conducted a retrospective single-center study including all PBC patients followed between 2016 and 2025. Diagnosis was established according to EASL criteria, based on cholestatic liver tests, anti-mitochondrial antibodies (AMA-M2), and/or compatible liver histology. Clinical, biochemical, immunological, and histological data were collected. Descriptive comparisons between men and women were performed; no formal statistical analysis was conducted due to the limited number of male patients.

Results:

Among 106 PBC patients, 10 were men. Mean age at diagnosis was 52.5 years in men versus 49.2 years in women. Associated autoimmune diseases were present in 60% of men and 54.2% of women. Signs of portal hypertension were observed in 50% of men versus 39.6% of women. Bone disease prevalence was similar (70% vs. 68.8%).

Liver biochemistry showed elevated alkaline phosphatase in 90% of men and 77% of women; GGT elevation was noted in 80% versus 87.5%. Marked cytotoxicity was more frequent in men (30% vs. 16.7%). Anti-M2 antibodies were positive in 70% of men and 75% of women; AMA-negative PBC with specific autoantibodies was rare in both sexes. Advanced fibrosis (F3–F4) was identified in 60% of men versus 54.2% of women. Liver biopsy was performed in 40% of men and 34.4% of women. UDCA response (Paris II criteria) was achieved in 50% of men versus 58.3% of women. PBC–autoimmune hepatitis overlap was diagnosed in 30% of men versus 16.7% of women.

Conclusions:

In our cohort, men with PBC tended to present with more advanced fibrosis, higher rates of cytotoxicity,

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slightly lower UDCA response, and increased prevalence of PBC–AIH overlap. These exploratory findings suggest that male PBC may represent a distinct, potentially more severe phenotype, emphasizing the need for clinical vigilance and multicenter studies to further characterize this subgroup.

Keyword(s) : Autoimmune liver disease; Primary biliary cholangitis; Cholestasis

1697 - Combined use of fibrate and statin to reduce the risk of hepatocellular carcinoma a population based cohort study

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Background: This study investigated the reduced risk of hepatocellular carcinoma (HCC) by the use of fibrates as an add-on to statins.

Methods: This population-based cohort study was conducted from the Korean National Health Insurance Service from 2010 to 2019 in Korea. We compared the incidence of HCC between statin users and those taking fibrates and statins for dyslipidemia, matched using age, sex, and hypertriglyceridemia.

Results: We analysed 789,958 individuals, including 394,979 statin users and 394,979 who were matched users of combined fibrate-statin therapy. During the 1,644,037.6 person-years of follow-up, 791 HCC cases were observed in the statin group, whereas 703 HCC cases were observed in the fibrate-statin group during 1,646,939.6 person-years of follow-up. The HCC incidence rates per 1,000 person-years were 0.48 and 0.43 in the statin and fibrate-statin groups, respectively. The risk of HCC was lower in the fibrate-statin group compared to the statin-only group in the unadjusted and adjusted models (hazard ratio [HR] 0.89 with 95% confidence interval [CI]: 0.80–0.98 and adjusted HR [aHR] 0.74 with 95% CI: 0.67–0.83). The reduction of HCC development by fibrate-statin increased as the duration of fibrate administration lengthened (aHR 0.76 with 95% CI: 0.68–0.86 in the use of fibrate and statin for <1 year; and aHR 0.71 with 95% CI: 0.61–0.82 for >1 year).

Conclusions: Fibrates as an add-on to statin were associated with a reduced risk of HCC compared to statins. Further prospective studies are needed to clarify the role of fibrate on the risk of HCC.

Keyword(s) :

1699 - Endoscopic Management of Complications from Ruptured Hepatic Hydatid Cysts into the Bile Ducts

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Background / Hypothesis:

Liver hydatid cyst(LHC)is a parasitic disease caused by the development of the tapeworm Echinococcus granulosus.Fistulization in the bile ducts is one of the most common complications. Endoscopic treatment is essential due to the risks associated with surgery.The objective of our study was to evaluate the contribution of endoscopic retrograde cholangiopancreatography(ERCP)in the diagnostic and,more importantly,therapeutic management of ruptured LHC in the bile ducts.

Materials and Methods:

This was a retrospective, descriptive and analytical study, conducted from January 2002 to August 2025, involving 50 patients with LHC fistulized in the bile ducts.ERCP and endoscopic biliary sphincterotomy were performed in all patients.Overall success was defined as definitive emptiness of the common bile duct.Statistical analysis was performed using SPSS 24.0software

Results:

During the study period,4.6%(n =50)of ERCPs were performed for hepatic hydatid cysts ruptured into the bile ducts.The mean age was 46.1±14.8 years,with 66% male patients(M/F ratio: 1.94).ERCP was performed preoperatively in 52.2%and postoperatively in 47.8%of cases.Main indications included acute cholangitis(44.9%) and persistent external biliary fistula(34%).Sphincterotomy was done in 96% of cases, with hydatid material successfully extracted in 87.8%.Nasobiliary drainage was required in 24%, and biliary stenting in 8%.The overall success rate was 96%, with a complication rate of 8% (hemobilia and post-sphincterotomy bleeding). Jaundice resolved within 5–10 days, and external fistulas closed by day 10 in most cases.



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Conclusions:

Our study confirms that endoscopic treatment of ruptured hydatid cysts in the bile ducts is an effective therapeutic alternative, with a low rate of immediate complications (8%) and good long-term outcomes.

Keyword(s) :

1700 - Artificial intelligence based prediction of ursodeoxycholic acid response in primary biliary cholangitis comparison of two large language models

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Background / Hypothesis:

Primary biliary cholangitis (PBC) is a chronic autoimmune disease characterized by progressive destruction of the intrahepatic bile ducts. Ursodeoxycholic acid (UDCA) is the standard treatment, and therapeutic response is a major prognostic factor. The recent rise of large language models (LLM) paves the way for their use as potential clinical decision support tools.

This study aimed to compare the performance of two general purpose chatbots (A and B) in predicting the 12-month biological response to UDCA, defined according to Paris II criteria, and to determine which of the two could be the most relevant.

Materials and Methods:

The study included 142 patients with PBC, followed between 2010 and 2025 in a tertiary hepatology department. Clinical, biological, immunological, and histological data available at the start of treatment were collected from medical records.

The actual response at 12 months was defined according to the Paris II criteria, which served as the gold standard. The data were entered into two LLM (chatbot A: ChatGPT and chatbot B: Claude) using an identical prompt in order to obtain a prediction of responder or non responder status.

Predictive performances were evaluated using: accuracy, sensitivity, specificity, and ROC-AUC.

Results:

Chatbot A (ChatGPT) showed high predictive performance. It achieved an AUC of 0.84, with 85% accuracy, 82% sensitivity, and 79% specificity, indicating good discriminative ability and satisfactory agreement between predicted and observed outcomes. Chatbot B (Claude), however, showed substantially lower performance. The generated model displayed limited discrimination, with an AUC of 0.50 and an overall accuracy of approximately 53%. Sensitivity at 64.3% and specificity at 60.7% reflected an insufficient ability to distinguish responders from non responders.

Conclusions:

This study shows that Chatbot A demonstrates good discriminative performance in predicting UDCA response in PBC, with satisfactory concordance with outcomes observed at 12 months. In contrast, Chatbot B exhibits limited performance and insufficient discrimination between responders and non responders.

These results suggest that LLM based tools could assist clinical decision-making in hepatology, but should not replace clinical judgment. Their variable performance across models also highlights the need for further validation before clinical implementation.

Keyword(s) : Artificial intelligence, Primary biliary cholangitis, Ursodeoxycholic acid response, Chatbots, Predictive modeling

1701 - Seronegative primary biliary cholangitis clinical biochemical and histological profile with comparison to seropositive disease

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Background / Hypothesis:

Primary biliary cholangitis (PBC) is an autoimmune cholestatic disease characterized by progressive destruction of intrahepatic bile ducts. While most patients present with anti mitochondrial antibodies (AMA) or specific PBC-related autoantibodies (anti-gp210, anti-sp100), a subset remains seronegative, making diagnosis more challenging. This study aimed

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to describe the clinical, biochemical, and histological features of seronegative PBC (snPBC) and compare them with seropositive forms.

Materials and Methods:

We conducted a retrospective descriptive study including all patients diagnosed with snPBC between 2010 and 2025 in a tertiary hepatology department. Diagnosis relied on cholestatic liver tests and compatible liver histology in the absence of AMA and PBC-specific autoantibodies. Clinical, biochemical, immunological, and histological data were collected. Statistical analysis was performed using Jamovi.

Results:

Sixty-seven snPBC patients were included (mean age 49 years; female-to-male ratio of 3:1). Jaundice was reported in 52%, asthenia in 44%, and pruritus in 32%. Signs of portal hypertension were observed in 40% of cases. Immunologically, all patients were negative for AMA, anti-gp210, and anti-sp100. Biochemically, 61% had cytocholestasis and 39% had isolated cholestasis. Histology showed Scheuer stages: 15% stage 1, 43% stage 2, 33% stage 3, and 9% stage 4. Fibrosis staging revealed advanced fibrosis (F3–F4) in 58% of patients. Associated autoimmune conditions included PBC-autoimmune hepatitis overlap (21%), autoimmune thyroiditis (13.5%), and Sjögren's syndrome (6%). According to Paris II criteria, 57% responded to UDCA and 28% were non-responders. Comparison with seropositive PBC revealed no significant differences regarding demographics, fibrosis severity, portal hypertension, autoimmune associations, or UDCA response.

Conclusions:

Seronegative PBC in our cohort predominantly affected middle-aged women and frequently presented with advanced fibrosis and associated autoimmune diseases. Despite its distinct immunological profile, snPBC did not significantly differ from seropositive PBC in clinical severity or treatment response, suggesting a comparable prognosis and similar therapeutic management.

Keyword(s) : Primary biliary cholangitis, Seronegative primary biliary cholangitis, Autoantibodies, Liver

histology, Portal hypertension, Ursodeoxycholic acid response

1702 - Combining Interferon α and Nucleos(t)ide Analogues to Enhance Early Virological Response in Chronic Hepatitis B

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Background / Hypothesis:

This study evaluated the efficacy of different combination strategies of interferon-alpha (IFN α) and nucleos(t)ide analogues (NAs) in patients with chronic hepatitis B (CHB), focusing on the impact of different administration sequences (initial combination vs. sequential add-on) on virological and serological responses.

Materials and Methods:

A retrospective cohort study was conducted, enrolling 145 patients with CHB. Participants were divided into three groups based on their treatment regimen: initial combination (concurrent IFN α and NAs for 48 weeks), initial IFN α (12 or 24 weeks) followed by add-on NAs (total 48 weeks), and initial NAs (12 or 24 weeks) followed by add-on IFN α (total 48 weeks). Virological, serological, and biochemical markers were collected at baseline, 12, 24, and 48 weeks of combination therapy. The primary endpoints were the virological response rate and HBsAg seroconversion rate at week 48.

Results:

Baseline characteristics were comparable among the three groups, except for HBeAb levels. After 48 weeks, the initial combination group showed a significant reduction in HBV DNA and HBeAg ($P < 0.001$), and demonstrated an earlier and sustained decline in HBsAg from week 12 ($P < 0.01$). No significant differences were observed in the HBV DNA negative rate or HBsAg seroconversion rate.

Conclusions:

Initial combination therapy demonstrated stronger advantages in early viral suppression and HBeAg reduction. Although it did not improve seroconversion outcomes, initiating IFN α and NAs simultaneously optimizes virological and serological responses, providing a new theoretical basis for improving clinical cure strategies for CHB.

Keyword(s) : Chronic Hepatitis B, Interferon-alpha,

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Nucleos(t)ide Analogues, Combination Therapy,
Virological Response

1703 - First Case Report of Tenofovir Disoproxil Fumarate Induced Perichondritis A Case Study from the Perspective of Seronegative Autoimmune Processes

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Background: Tenofovir disoproxil fumarate (TDF) is a safe and potent drug treatment of HBV infection. However, due to the activation of the immune system and the increase in inflammatory processes, some side effects may be observed, although not frequently. This article presents the first case report of perichondritis caused by TDF and will investigate the possible relationship between tenofovir and inflammatory processes.

Case Report: A 43-year-old female patient with a 10-year history of chronic HBV infection. She presented with fatigue and right upper quadrant pain. Liver function tests were normal. HBsAg: 5222, Anti-HBe: positive, HBV DNA: 135.014 IU/mL (fluctuating). AFP: 3,27 µg/L, USG were normal. Liver biopsy; modified Knodell HAI: 4/18, Fibrosis: 1 was detected. HBV focal diffuse (+) positive. TDF treatment was started. The patient also requested treatment.

In the second month of treatment, the patient presented with complaints of redness and pain in both auricles, nausea, intense hair loss, and dry mouth. Physical examination revealed bilateral perichondritis. Hemogram and biochemical analyses were normal. Rheumatoid factor (RF): 21 IU/mL was detected as high. Other autoimmune and rheumatological markers and acute phase reactants were normal. The patient discontinued treatment. The perichondritis has regressed. The pain and nausea have resolved. However, hair loss and dry mouth persisted. RF elevation persisted during follow-up. During follow-up visits, the patient had an attack of perichondritis. Patient was referred to a rheumatology.

Discussion: The development of perichondritis, hair loss, and dry mouth following initiation of TDF therapy, along with negative serological tests, regression of perichondritis following discontinuation suggests a systemic seronegative autoimmune process. Despite elevated RF levels, negative anti-CCP and ANA results suggest the possibility of seronegative rheumatoid arthritis or relapsing polychondritis. Symptoms suggestive of exocrine gland involvement, such as dry mouth and foul-smelling sweating, may be significant

for seronegative Sjögren's syndrome, but similar symptoms can also be seen in systemic autoimmune diseases such as relapsing polychondritis. The possibility of an exocrine-involved seronegative autoimmune process has been strengthened. Perichondritis associated with tenofovir use has not been previously reported. However, there are some case reports regarding rare dermatological/immune side effects of TDF.

The incidence of side effects is not common. However, due to immune system activation and increased inflammatory processes, may be observed, although not frequently. In this case, the recurrence of perichondritis, hair loss, and dry mouth during and after TDF treatment suggests that an underlying latent autoimmune disease may have been triggered.

Keyword(s) : Tenofovir disoproxil fumarate; side effect; perichondritis; inflammation; autoimmune disease

1704 - Case Report of Hematopoietic Disorder Complicated by Severe Liver Injury Clinical Features and Mechanistic Insights into the Co occurrence of Hemophagocytic Syndrome and Aplastic Anemia

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Abstract :

The patient is a 33-year-old woman who was admitted with the chief complaints of "fatigue, poor appetite, and yellow urine for 1 month, exacerbated by fever for 4 days." The clinical course was characterized by progressive liver failure, pancytopenia, and severe inflammatory response. Bone marrow cytology confirmed the diagnosis of hemophagocytic lymphohistiocytosis (HLH) combined with pure red cell aplasia (PRCA), forming a rare mixed hematopoietic dysfunction syndrome. By integrating the clinical progression, dynamic laboratory evolution, and therapeutic response, this study focuses on: (1) exploring the bidirectional pathogenic relationship between liver failure and HLH activation, with particular focus on the central role of hepatic dysfunction in promoting immune dysregulation and cytokine storm ; and (2) investigating the pathogenic interplay between aplastic anemia (AA) presenting as PRCA and HLH, specifically highlighting the critical role of immune microenvironment disruption in mediating lineage-specific hematopoietic suppression. This case provides unique insights into the complex interplay between liver failure and hematopoietic injury under systemic

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immune dysregulation, underscoring the clinical challenges in diagnosing and managing HLH-AA spectrum comorbidities.

Keyword(s) : liver injury; hemophagocytic lymphohistiocytosis; aplastic anemia

1705 - Development of a Novel Animal Model for Acute on chronic liver failure (ACLF) in Metabolic dysfunction associated steatotic liver disease Confirmed the progression to ACLF in a non cirrhotic liver disease stage

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) is a high-mortality syndrome triggered by acute insults in patients with chronic liver disease. Coagulopathy in critically ill patients largely precludes tissue sampling, underscoring the need for animal models as an alternative. Metabolic dysfunction-associated steatotic liver disease (MASLD), a growing public health concern, can progress to cirrhosis and subsequently increase MASLD-ACLF prevalence. While animal models exist for MASLD or ACLF separately, none replicates ACLF development on a MASLD background.

Materials and Methods:

Based on previous research, a MASLD model was established using B6-Alms1-del mice fed an ALMN diet (Western diet). Varying the modeling duration induced steatohepatitis in the mice. After LPS/D-GalN acute insult, serum was collected for liver function tests and liver tissues for histopathology and transcriptomics. Multiplex immunofluorescence was utilized to assess intestinal barrier damage and the potential gut-liver axis during ACLF progression.

Results:

Successful generation of MASLD-ACLF models exhibiting steatohepatitis was achieved. Compared to

the MASLD group, the MASLD-ACLF group showed extensive liver necrosis on H&E staining, along with typical histopathological features supporting ACLF diagnosis, including neutrophilic and lymphocytic infiltration, Mallory bodies, cholestasis, and portal inflammation, concurrent with characteristic steatotic manifestations. Hepatic fibrosis was assessed via α -SMA and Sirius red staining. Serum levels of AST, ALT, ALB, TBIL, GLU, CREA, and TBA were altered. Decreased Muc-2 and Occludin indicated gut barrier impairment. Transcriptomics revealed changes in tissue remodeling, phagocytosis, bile acid metabolism, and neutrophil phenotype markers. Hallmark gene set analysis pathways revealed IL6-JAK-STAT3 and Interferon Alpha Response pathways enriched in MASLD-ACLF group.

Conclusions:

This study successfully established and validated the first MASLD-ACLF animal model. Pathological evidence confirmed progression to ACLF at a non-cirrhotic liver disease stage. Given the growing public health challenge posed by MASLD-ACLF, this model provides a platform enabling direct tissue analysis and longitudinal investigation, thereby facilitating the exploration of disease mechanisms and the development of therapeutic strategies.

Keyword(s) :

1706 - Bushen granule plus entecavir improved the effectiveness for HBeAg negative chronic hepatitis B A multicenter randomized double blind placebo controlled clinical trial

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Background / Hypothesis:

Nucleoside analogs are the first-line therapeutic drugs for the treatment of chronic hepatitis B (CHB), but loss of HBsAg is rarely achieved. Bushen granule (BSG), as the experimental traditional Chinese medicine, is widely used to treat CHB in China alongside NAs. However, there are no clinical data about entecavir (ETV) plus BSG treating HBeAg negative CHB patients. In this paper, we aimed to assess the efficacy of BSG in

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promoting the loss of HBsAg in HBeAg negative CHB patients.

Materials and Methods:

A multicenter, double-blind, placebo-controlled randomized clinical trial involving 555 HBeAg negative CHB patients was conducted from April 2019 to August 2022. (Clinical Trials Registration: ChiCTR1900021370). Patients were randomized into the treatment group (administration of ETV 0.5 mg/day plus BSG 36.0g/time, 2 times/day, n=278) and control group (administration of ETV 0.5 mg/day plus placebo 36.0g/time, 2 times/day, n=277). The treatment period was 120-week. The primary outcomes were the rate of HBsAg loss and the decline of HBsAg at week 120. Secondary outcomes included the change of LSM, CAP and biochemical items.

Results:

Two hundreds and twenty eight patients (82.01%) in the treatment group and 227 patients (81.65%) in the control group completed the study. The rate of HBsAg loss at week 120 in the treatment group was markedly higher than that in the control group (4.4% vs. 0.9%, $P=0.020$). Further sub-group analysis showed that in the sub-group with HBsAg < 1500 IU/ml, the HBsAg loss rate in the treatment group was more remarkable than that in the control group (10.1% vs. 1.9%, $P=0.013$). In the sub-group with HBsAg $\geq$ 1500 IU/ml, the HBsAg decline was much greater in the treatment group than that in the control group. BSG was well tolerated without any severe side effects.

Conclusions:

BSG plus ETV combination treatment increased the rate of HBsAg loss in HBeAg-negative CHB patients, particularly in patients with baseline HBsAg < 1500 IU/ml.

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Keyword(s) : Chronic hepatitis B; the rate of HBsAg loss; Bushen Granule; Entecavir

1707 - Analysis of Risk Factors and Fibrosis Severity in Type 2 Diabetic Patients with Metabolic Associated Fatty Liver Disease A Cross Sectional Study

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Background / Hypothesis:

Metabolic associated fatty liver disease(MAFLD) is closely related to type 2 diabetes mellitus (T2DM). However, the exact risk factors, severity of liver injury, and degree of fibrosis in T2DM patients within our country and the Asia-Pacific region require further clarification. This study aimed to investigate the risk factors for MAFLD, the extent of liver injury and fibrosis, and identify independent interrelated risk factors in T2DM patients at our institution.

Materials and Methods:

This cross-sectional study consecutively enrolled 1350 T2DM patients from our digitalized diabetes and liver disease co-management center between June 2023 and June 2025. All patients were diagnosed based on the MAFLD criteria (evidence based on liver transient elastography concurrently with overweight/diabetes or metabolic dysfunction). We collected demographic data, diabetes duration, anthropometric parameters (BMI, waist circumference), biochemical indicators (fasting blood glucose, HbA1c, blood lipids, liver enzymes), non-invasive liver fibrosis indices (Liver Stiffness Measurement, LSM), and medication history. Multivariable logistic regression models were used to analyze independent risk factors for MAFLD.

Results:

Among the 1350 enrolled T2DM patients (mean age 52.4 ± 10.8 years, 54% male), the overall prevalence of MAFLD, diagnosed based on liver transient elastography evidence concurrently with overweight/diabetes or metabolic dysfunction, was 62.5% (844/1350). Multivariable regression analysis, after adjusting for age and sex, revealed that the following factors were independently associated with MAFLD in T2DM patients:

- Obesity (OR = 2.85, 95% CI: 2.32-3.51, $P < 0.001$)
- Central obesity (elevated waist circumference): OR = 2.41, 95% CI: 1.98-2.93, $P < 0.001$)
- Hypertriglyceridemia (OR = 2.12, 95% CI: 1.75-2.57, $P < 0.001$)

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- Lower HDL-C level (HDL-C <1.0 mmol/L [male] / <1.3 mmol/L [female]: OR = 1.68, 95% CI: 1.38-2.05, P<0.001)
- Higher ALT level (OR = 1.92, 95% CI: 1.55-2.38, P<0.001)
- Poorer glycemic control (OR = 1.55, 95% CI: 1.26-1.91, P<0.001)
- Among diabetic patients with MAFLD, the proportion with LSM suggestive of significant liver fibrosis (>7.3 kPa) reached 15.3%, which was significantly higher than in diabetic patients without MAFLD (P<0.001).
- The association between diabetes duration and MAFLD did not reach statistical significance (P=0.06).

Conclusions: This study demonstrates a high prevalence of MAFLD among T2DM patients at our institution, and patients with comorbid MAFLD have a significantly higher risk of liver fibrosis. Obesity, dyslipidemia, elevated liver enzymes, and poor glycemic control are independent risk factors for MAFLD in T2DM patients. Diabetes duration showed no independent association with MAFLD. These findings emphasize the importance of routine MAFLD screening in T2DM patients and active management of these modifiable risk factors to prevent hepatic and extra-hepatic

Keyword(s) : Type 2 Diabetes; Metabolic Associated Fatty Liver Disease; Prevalence; Risk Factors; Metabolic Syndrome

1709 - Neck circumference and the risk of metabolic dysfunction associated steatotic liver disease in non obese individuals without visceral obesity

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Background / Hypothesis: This study aimed to examine the association between neck circumference and metabolic dysfunction-associated steatotic liver disease (MASLD) in non-obese individuals without visceral obesity.

Materials and Methods: A cross-sectional analysis was performed using nationally representative data from the Korea National Health and Nutrition Examination Survey 2019–2020. Non-obesity was defined as body

mass index (BMI) <25 kg/m². Visceral obesity was defined as a waist circumference ≥90 cm for men and ≥85 cm for women. Increased neck circumference was defined as ≥37.7 cm for men and ≥32.8 cm for women. Multivariable logistic regression was performed to assess the association between neck circumference and MASLD in non-obese individuals without visceral obesity, and adjusted odds ratios (aORs) and 95% confidence intervals (CIs) were reported.

Results: This study included 4,065 non-obese adults without visceral obesity (1,521 men and 2,544 women) aged ≥40 years. The mean neck circumference was 36.5 cm in men and 31.9 cm in women, with 25.3% (n=1,027) classified as having an increased neck circumference. The overall prevalence of MASLD was 3.5% (n=141) and was significantly higher among individuals with increased neck circumference compared with those with normal neck circumference (7.6% vs. 2.1%, P < .001). Increased neck circumference was independently associated with MASLD (aOR 1.95, 95% CI: 1.26-2.99, P = .003).

Conclusions: Increased neck circumference is independently associated with MASLD in non-obese individuals without visceral obesity. Neck circumference may serve as a simple and practical anthropometric marker to identify at-risk individuals who may be overlooked by conventional measures such as BMI or waist circumference.

Keyword(s) : neck circumference; visceral obesity; metabolic dysfunction-associated steatotic liver disease; insulin resistance

1710 - Mn Tannic Acid Immunomodulators Potentiate PDGFRβ CAR T Cell Function Through Enhanced proliferation for Synergistic Anti Hepatic Fibrosis Therapy

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Background / Hypothesis: Chimeric antigen receptor (CAR)-T cell therapy has

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obtained considerable success in treating malignant tumors, and is now being increasingly used to treat a number of other chronic immune diseases. However, the therapeutic efficacy of the treatment for hepatic fibrosis remains to be demonstrated due to the complex pathogenic microenvironment in the liver resulting in negative cause-induced inhibition of cell expansion capacity, limited infiltration capacity, and severe cellular exhaustion and senescence.

Materials and Methods:

Here, we creatively developed Mn-tannic acid (MT) nanoparticle immunomodulators with uniform particle size and excellent T1-weighted MRI imaging performance through the coordination reaction of tannic acid and Mn^{2+} . It possessed the property of remodeling the pathophysiological microenvironment of liver fibrosis and improved the phenotype and cytotoxicity of CAR-T cells. In addition, MT nanoparticles formed strong bonds with multidentate ligands of PDGFR β CAR-T cell membrane proteins via hydrophobic interactions and hydrogen bonding, which was designed for targeting activated hepatic stellate cells (aHSCs).

Results:

MT nanoparticles exhibited enhanced cytotoxicity of PDGFR β CAR-T cells against target cells in vitro. In vivo studies demonstrated that PDGFR β CAR-T cell therapy enhanced by MT nanoparticles could synergistically improve anti-fibrotic treatment outcomes with real-time MRI-guided monitoring of therapeutic response. Crucially, MT nanoparticles had been shown to remodel the hepatic fibrotic microenvironment effectively by alleviating the pro-inflammatory response and oxidative stress, which further enhanced the synergistic therapeutic effect of PDGFR β CAR-T cells and modulated the infiltration of endogenous hepatic immune cells while inhibiting stellate cell activation. In addition, we discovered a novel mechanism by which MT nanoparticles enhanced the proliferation and thus the killing ability of PDGFR β CAR-T cells through upregulating the activity of the RAS signaling pathway. MT nanoparticles could impart superior phenotypes to CAR-T cells.

Conclusions:

Overall, our well-designed MT nanoparticle-modified PDGFR β CAR-T cell immunotherapy strategy demonstrated excellent anti-fibrotic effects, providing

a promising therapeutic regimen for hepatic fibrosis. It also expanded the therapeutic approach of the adoptive cellular therapy embellished with a novel biomaterials, backed by solid evidence and guidance.

Keyword(s) : CAR-T cells, PDGFR β , Mn-Tannic Acid, liver fibrosis, activated hepatic stellate cells

1711 - Rougan Formula Alleviates Liver Fibrosis by Suppressing Macrophage Glycolysis via Downregulation of the Tenascin C HIF 1 α PFKFB3 Pathway

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Background / Hypothesis:

Liver fibrosis is a core pathological process in the progression of chronic liver diseases to cirrhosis and hepatocellular carcinoma, with limited effective treatment options. Rougan Formula(RGF) is the experienced formula for treating liver fibrosis. In this study, we aimed to explore the relevant mechanism of RGF treating liver fibrosis.

Materials and Methods:

A CCL₄-induced liver fibrosis mouse model and an LPS-stimulated RAW264.7 macrophage model were established. Transcriptomic and single-cell sequencing, co-culture assays, TNC silencing, and TNC-knockout techniques were applied to analyze the effects of Rougan Formula on macrophage polarization, glycolysis, and HSC activation. Serum pharmacochimistry and mass spectrometry were used to identify bioactive components of Rougan Formula and validate their inhibitory effects on TNC and liver fibrosis.

Results:

Different doses of RGF can alleviate inflammation and fibrosis in CCL₄-induced models. Transcriptome sequencing confirmed that RGF decreased the expression levels of liver TNC and PFKFB3. Single cell sequencing revealed that RGF significantly inhibited the expression of pro-inflammatory macrophage subpopulation, which was associated with glycolysis. Further experiments showed that RGF significantly reduced the expression levels of GLUT1, TLR4, and HIF-1 α in liver tissues, and decreased iNOS, CD86, CD80,

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TNC, TLR4, HIF-1 α , and PFKFB3 in primary liver macrophages. In vitro, RGF-containing serum reduced glycolysis and pro-inflammatory cytokine levels in RAW264.7 cells and inhibited JS1 cell activation in co-culture systems. In addition, liver fibrosis was alleviated in TNC-KO models. TLR4 and PFKFB3 expression levels were attenuated and glycolysis level was reduced after TNC silencing or knockout. In addition, the anti-fibrotic effects of RGF formula was in TNC-KO models.

Conclusions:

Rougan Formula exerts anti-fibrotic effects by downregulating TNC in macrophages, inhibiting TLR4/HIF-1 α /PFKFB3-mediated glycolysis and pro-inflammatory polarization, and subsequently suppressing HSC activation.

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Keyword(s) : Rougan Formula; Hepatic Fibrosis; Glycolysis; Pro-inflammatory Polarization; Formononetin

1712 - Implementation of a Bidirectional Protection Model for Infection Control in a Dedicated Chronic Hepatitis B Outpatient Clinic A Comprehensive Approach to Break the Chain of Bloodborne Transmission

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Background / Hypothesis:

Chronic hepatitis B (CHB) remains a major public health challenge in China, with an estimated 75 million affected individuals. Despite global efforts toward viral hepatitis elimination by 2030, China's diagnosis and treatment rates remain suboptimal. In outpatient settings managing high volumes of CHB patients, frequent blood exposure, invasive procedures, and environmental contamination pose significant infection risks. We developed and implemented a novel "Bidirectional Protection Model" to enhance infection control while ensuring patient-centered care.

Materials and Methods:

Guided by national disinfection standards, our multidisciplinary team at Zhuhai Third People's Hospital integrated spatial design, workflow optimization, staff training, digital health tools, and

environmental hygiene into a closed-loop, full-process infection control system. The model features: (1) Physical zoning—separate clinical, auxiliary, and clean office areas with distinct patient and staff/utility pathways; (2) Personnel management—mandatory HBV vaccination with antibody monitoring and tiered training for all staff, including support personnel; (3) Digital innovation—an online appointment system limiting patient flow and an electronic medical record platform integrating HBV DNA and liver function data for risk-stratified triage; (4) Micro-level environmental controls, including "one patient—one room—one disinfection," color-coded cleaning tools by risk zone, single-use probe covers for FibroScan devices, and traceable medical waste handling.

Results:

The model effectively minimizes cross-transmission risks among HBV-positive patients, protects HBV-negative individuals (including co-visitors and healthcare workers), and reduces environmental contamination. Patient convenience is enhanced through one-stop services (consultation, lab testing, liver stiffness measurement) and dedicated case managers for continuity of care. Community outreach further reduces stigma and improves early detection.

Conclusions: Our Bidirectional Protection Model transforms infection control from policy into practice, achieving dual goals: advancing CHB treatment coverage and ensuring biosafety for all clinic users. This scalable framework offers a practical blueprint for specialized hepatitis clinics across the Asia-Pacific region, particularly in resource-limited or rural settings where telemedicine and blockchain-based waste tracking could further extend its impact.

Keyword(s) : Chronic hepatitis B; infection control; outpatient management; standard precautions; bloodborne pathogens; bidirectional protection

1714 - Development and External Validation of Machine Learning Based Prognostic Model for Liver Transplant Patients with HCC

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Background / Hypothesis:

Liver transplantation (LT) is an effective treatment option for unresectable, early-stage hepatocellular carcinoma (HCC) patients. Several eligibility criteria has been proposed, but there is a controversy which criteria can be an optimal cutoff for transplantation because of heterogeneity in LT recipients. By considering pre-transplant clinical variables applying competing risk analysis, we developed personalized prognostic criteria to predict post-transplant survival outcomes, aiming to enhance individualized prognostication.

Materials and Methods:

The internal (n=1,059) and external cohorts (n=398) who underwent LT for HCC from two tertiary hospitals in Korea were used and competing risk regression analyses for HCC-specific survival were conducted to develop a new prognostic model using Fine-Gray model. To prevent overfitting, five penalty-based methodologies like machine learning based approach were systematically evaluated through backward elimination with stratified 5-fold cross-validation. Prognostic accuracy was compared with Wolbers' C-index and compared with conventional LT criteria.

Results:

The FG model achieved the highest external validation performance (Wolbers' C index: 0.786), while conventional scores demonstrated considerably lower discrimination: Milan criteria (0.604), Up-to-Seven (UTS) criteria (0.567), and Metroticket 2.0 (0.651). In the time-dependent Area under ROC curve (AUROC) and Integrated Brier score (IBS), the FG models demonstrated higher performance, with AUROC values of 0.802 (compared with 0.612 and 0.575 for the Milan criteria and UTS, respectively; P-values <0.001), and IBS values of 0.065 for the FG models (compared with 0.093 and 0.082 for the Milan criteria and UTS, respectively).

Conclusions:

The FG model based on pre-transplant clinical variables showed superior prognostic performance compared with conventional LT criteria. Clinical decision based on this model enables to select the more optimal LT candidates with HCC.

Keyword(s) : liver transplantation, hepatocellular carcinoma, Fine-Gray model

1716 - Hemoperitoneum as Initial Presentation of Hepatocellular Carcinoma in Chronic Hepatitis C A Case Report

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Background: Spontaneous rupture of hepatocellular carcinoma (HCC) resulting in hemoperitoneum is a rare but potentially fatal complication. It often presents as a surgical emergency, typically in patients with underlying cirrhosis, and poses diagnostic and therapeutic challenges due to its abrupt onset and nonspecific symptoms.

Case Presentation: A 63-year-old Filipino male with no known pre-existing liver disease presented to the emergency department with sudden, severe abdominal pain and shock. On initial assessment, he was tachycardic and hypotensive, requiring fluid resuscitation. An urgent axial contrast-enhanced CT scan of the abdomen revealed a large hepatic mass in segment 5 with associated hemoperitoneum, suggestive of a ruptured hepatic tumor. Due to high perioperative risk, the patient was managed non-surgically with transarterial embolization (TAE) of the feeding vessel and ultrasound-guided abdominal pigtail catheter drainage. Further workup revealed previously undiagnosed liver cirrhosis and chronic hepatitis C infection. Direct-acting antiviral therapy with sofosbuvir and velpatasvir was initiated and was given for 3 months. Metastatic workup was negative. Following clinical stabilization, the patient successfully underwent liver segmentectomy and recovered. Patient had documented sustained virologic response (SVR) 12 weeks after the end of treatment.

Conclusion: This case highlights the rare and dramatic presentation of ruptured HCC as spontaneous hemoperitoneum in an initially undiagnosed cirrhotic patient. Prompt recognition and multidisciplinary coordination are critical to improve survival. Given its rarity and high mortality, clinicians should maintain a high index of suspicion in cirrhotic or high-risk patients presenting with unexplained abdominal pain and hemodynamic instability.



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Keyword(s) : Hepatocellular carcinoma, hepatitis C, hemoperitoneum

1718 - Pediatric MASLD prevalence in the NHANES from 2017 to 2020 and the role of aging related factors

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Background / Hypothesis: Evidence suggests that accelerated biological aging is linked to metabolic dysfunction in adults. However, the association with pediatric Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD) remains unclear. This study investigated the relationship between two clinical biomarker-based measures of biological age and MASLD risk in adolescents.

Materials and Methods: We analyzed data from 1,206 adolescents (ages 12–18) in the 2017–2020 National Health and Nutrition Examination Survey (NHANES). Biological age was estimated using the Klemmera-Doubal Method (KDM) and Phenotypic Age (PhenoAge), both calculated from nine clinical biomarkers. MASLD was defined by a controlled attenuation parameter (CAP) > 248 dB/m. Multivariable logistic regression assessed the association between accelerated biological aging (biological age minus chronological age) and MASLD. Differential and stratified analyses were also performed.

Results: The prevalence of MASLD was 23.5% (n=283) among the 1,206 included adolescents. After multivariable adjustment, accelerated biological aging was significantly and positively associated with MASLD risk, whether measured by KDM (OR = 1.11, 95% CI 1.08–1.14) or PhenoAge (OR = 1.17, 95% CI 1.13–1.22). Nonparametric analysis confirmed that participants with metabolic disorders exhibited more pronounced biological age acceleration ($P < 0.001$). Stratified analyses showed these associations were particularly strong in female adolescents ($P < 0.001$) and those who were overweight or obese ($P < 0.001$).

Conclusions: Accelerated biological aging is significantly associated with pediatric MASLD in U.S. adolescents, especially in female and overweight/obese individuals. These findings suggest that aging-related biological mechanisms may be critical contributors to the development and severity of pediatric MASLD.

Keyword(s) : Pediatric; MASLD; Biological aging; Adolescents; NHANES

1719 - Real world Analysis of HBV pgRNA Dynamics and Its Correlation with Conventional Markers in CHB Management

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Background:

Patients with chronic hepatitis B typically undergo long-term treatment with nucleoside analogs and interferon, yet a complete cure is rarely achieved. The occurrence and progression of CHB are primarily associated with HBV cccDNA. HBV pgRNA in peripheral blood originates from cccDNA in liver tissue and can reflect HBV transcriptional activity. This study analyzes the dynamic changes of HBV pgRNA over time and compares pgRNA with HBsAg, HBeAg and HBV DNA in serum of CHB patients in the real world, aiming to evaluate the clinical utility of HBV pgRNA in management of CHB.

Methods:

This was a retrospective study. Fifty-nine patients with CHB who received more than three serum HBV pgRNA tests in a general hospital in Beijing from April 1, 2024 to April 1, 2025 were included. There were 39 males (66.1%) and 20 females (33.9%), with an average age of 43.10 ± 11.97 and 44.5 ± 7.51 years, respectively. Among the cases, serum HBV pgRNA was measured three times in 36 patients (61.02%), four times in 18 patients (30.51%), five times in 4 patients (6.78%), and six times in only 1 patient (1.69%). During the same period, HBsAg, HBeAg, and HBV DNA levels were analyzed in these patients, followed by comparative analysis with HBV pgRNA.

Results:

During the one-year study period, a total of 206 serum HBV pgRNA tests were performed in 59 patients, in which 97 tests showed undetectable levels or values below the detection limit (classified as 'negative'), while 109 tests showed values exceeding 300 copies/mL (classified as 'positive'). In the HBV pgRNA-negative group, the positive rates of HBsAg, HBeAg and HBV DNA were 92.22%, 14.10% and 11.70%, respectively. Conversely, the HBV pgRNA-positive group demonstrated higher positive rates for these markers at 99.00%, 80.61% and 54.45%, respectively. The median HBV pgRNA levels were 4.35 log copies/mL in HBeAg-positive cases and 2.69 log copies/mL in



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HBeAg-negative cases. Serial measurements revealed median HBV pgRNA levels of 4.51, 3.61, 3.68, 3.38, 3.73 and 2.65 log copies/mL at each respective time point during the observation period. Longitudinal analysis showed that 10 of 59 cases (16.9%) transitioned from HBV pgRNA-positive to negative status during the study period.

Conclusions:

HBV pgRNA detection demonstrated a strong correlation with HBeAg positivity and HBV DNA detection rates, while exhibiting only weak association with HBsAg positivity. Notably, HBV pgRNA levels were significantly elevated in HBeAg-positive cases compared to HBeAg-negative patients. Longitudinal analysis revealed a consistent downward trend in HBV RNA levels over time.

Keyword(s) :

1720 - Integrated experimental and transcriptomic analyses reveal the therapeutic mechanisms of the herbal formula WenQingYin against intrahepatic cholangiocarcinoma

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Background / Hypothesis: Intrahepatic cholangiocarcinoma (ICC) represents a highly aggressive and deadly malignancy with rising global incidence and limited effective therapies. The classic formula Wen Qing Yin(WQY) from the Ming Dynasty's Wan Bing Hui Chun has demonstrated therapeutic effects against ICC in clinical practice at the School of Chinese Medicine in the University of Hong Kong. Therefore, this study aims to reveal the effect and mechanism of WQY in the treatment of ICC.

Materials and Methods: In vitro experiments, we utilized HuCCT1 cell lines to perform MTT assays, colony formation analyses, and flow cytometry for Ki67, evaluating the suppressive effects of WQY on proliferation. The inhibitory impacts of WQY on cell migration and invasion were assessed via transwell and wound healing assays. Additionally, bulk RNA sequencing was conducted to detect the mechanisms underlying WQY's intervention in ICC, with verification of key pathway genes performed using RT-PCR. The differently expressed genes (DEGs) between the control and the WQY groups were screened out to perform further enrichment analyses including the Gene Ontology (GO) and the Kyoto Encyclopedia of Genes

and Genomes (KEGG). In vivo experiments, HuCCT1 xenografts model were established to observe the therapeutic efficacy of WQY in treating ICC. All studies used a concentrated WQY decoction, prepared by machine and concentrated via rotary evaporation.

Results: The IC50 values of WQY against Hucct1 cells were determined by MTT assay (Figure A). Our results confirmed that WQY significantly inhibited HuCCT1 cell proliferation, evidenced by reduced colony formation after 24-hour WQY treatment and decreased Ki-67-positive cells via flow cytometry, indicating fewer actively proliferating cells (Figure B-C). WQY suppressed the invasion and migration of Hucct1 cell. Transwell assays revealed dose-dependent inhibition of HuCCT1 cell invasion after 24-hour treatment, while wound healing assays showed similar reductions in migratory capacity (Figure D-E). Animal studies indicated that WQY strongly inhibited tumor growth in xenograft models of ICC, with significant reductions in tumor volume and weight in treated mice compared to the control group (Figure F-G). The enrichment analyses of DEGs showed that WQY may mainly affect KEGG pathways of ferroptosis and DNA replication and GO pathway of cellular response to nuclear DNA replication and cell cycle (Figure H-L). RT-PCR results indicated that the ferroptosis suppressor genes GPX4 and SLC7A11 were downregulated in WQY-treated cells (Figure M).

Conclusions: In conclusion, WQY demonstrated therapeutic efficacy against ICC, and these effects may be mainly achieved by upregulating ferroptosis, downregulating DNA replication.

Keyword(s) : Intrahepatic cholangiocarcinoma, Traditional Chinese Medicine, Wen QingYin, Transcriptomics, ferroptosis, DNA replication

1721 - Longitudinal Changes in Fibrosis Marker Monitoring Fibrosis Progression in People with HIV and Metabolic Dysfunction Associated Steatotic Liver Disease A Longitudinal Cohort Study

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Background / Hypothesis:

Dynamic fibrosis-4 index (FIB-4) monitoring tracks fibrosis and stiffness progression and predicts clinical

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outcomes in metabolic dysfunction-associated steatotic liver disease (MASLD). This study aims to evaluate dynamic changes in FIB-4 and metabolic parameters in PWH during antiretroviral therapy and to identify risk factors for liver fibrosis progression (LFP).

Materials and Methods:

The diagnosis of MASLD necessitates the exclusion of excessive alcohol consumption, as well as other identifiable etiologies of fatty liver disease, and requires the presence of at least one component of metabolic syndrome. Liver fibrosis progression is defined as an increase in FIB-4. FIB-4 was dichotomised into low (<1.3) and high (≥ 1.3). Increases were defined as $\geq 20\%$ rise and to ≥ 1.3 in the low FIB-4 group, and $\geq 20\%$ rise in the high FIB-4 group. Metabolic parameters assessed at the beginning and end of the follow-up period included body weight, blood glucose, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides, and total cholesterol.

Results:

From March 25, 2024, to December 10, 2024, 2,003 PWH were screened. After excluding those with excessive alcohol use or other liver diseases, 683 with MASLD were enrolled. After a median follow-up period of one year, 28 (3.9%) participants were lost to follow-up, 59 (8.3%) exhibited fibrosis progression, and 596 (83.8%) remained stable. In a multivariable logistic regression, both baseline age (OR = 1.10; 95% CI: 1.06–1.14; $P < 0.001$) and weight change (OR = 1.29; 95% CI: 1.21–1.38; $P < 0.001$) were independently associated with LFP. Regarding the association of obesity status transition with LFP, transition to obesity was independently associated with LFP (OR=6.56; 95% CI: 2.95–14.56; $P < 0.001$).

Conclusions: Liver fibrosis progression occurs in a non-negligible proportion of PWH and MASLD, even within a relatively short period of time. Weight gain and transition to obesity are associated with increased risk of LFP in PWH with MASLD.

Keyword(s) : liver fibrosis; people with HIV; metabolic dysfunction-associated steatotic liver disease.

1723 - Impact of empagliflozin on liver fibrosis in adults with type 2 diabetes and nonalcoholic steatohepatitis

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Background / Hypothesis: Nonalcoholic steatohepatitis (NASH) frequently coexists with type 2 diabetes mellitus (T2DM) and can progress to liver fibrosis, cirrhosis, and hepatocellular carcinoma. Sodium-glucose co-transporter-2 (SGLT2) inhibitors improve metabolic and cardiovascular outcomes in T2DM; however, their potential benefit on hepatic fibrosis remains insufficiently defined. This study hypothesized that empagliflozin may reduce hepatic fibrosis and improve biochemical parameters in T2DM patients with magnetic resonance elastography (MRE)-confirmed NASH.

Materials and Methods: A prospective, randomized, open-label study was conducted at BIRDEM General Hospital, Dhaka, from April to October 2022. Ninety adults with T2DM and MRE-confirmed NASH were randomized (1:1) to receive either empagliflozin 10 mg/day plus standard antidiabetic therapy (Group A) or standard therapy alone (Group B) for at least 3 months. Primary outcome was change in liver stiffness measured by MRE. Secondary outcomes included liver enzymes, lipid profile, BMI, glycemic parameters, and fibrosis stage. Data were collected via clinical assessment and laboratory records and analyzed using SPSS v24. Statistical significance was defined as $p < 0.05$.

Results: Baseline demographic and clinical characteristics were comparable between groups ($p > 0.05$). After 3 months, Group A showed significant reductions in weight ($76.91 \pm 6.4 \rightarrow 73.52 \pm 6.8$ kg, $p < 0.001$) and BMI ($22.52 \pm 4.6 \rightarrow 21.47 \pm 4.6$ kg/m², $p < 0.001$). Glycemic control improved significantly, with reductions in fasting blood glucose, postprandial glucose, and HbA1c ($7.65 \pm 1.5 \rightarrow 7.27 \pm 1.47\%$, $p = 0.012$). Liver enzymes (AST, ALT, GGT) and lipid parameters (TC, TG, LDL) declined significantly in the empagliflozin group, whereas the control group showed minimal or non-significant changes. Liver stiffness significantly decreased in Group A ($3.40 \pm 0.40 \rightarrow 3.28 \pm 0.46$ kPa, $p = 0.031$), accompanied by improvement in fibrosis staging, while no meaningful improvement occurred in Group B.

Conclusions: Empagliflozin significantly improved liver

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stiffness, fibrosis stage, metabolic parameters, liver enzymes, and lipid profile in T2DM patients with NASH over a 3-month period. These findings support the therapeutic potential of SGLT2 inhibition for mitigating hepatic fibrosis. Larger, biopsy-based trials with longer follow-up are warranted to validate these results.

Keyword(s) : Empagliflozin, nonalcoholic steatohepatitis, liver fibrosis, magnetic resonance elastography, type 2 diabetes mellitus, SGLT2 inhibitors

1724 - Some biochemical characteristics among viral hepatitis associated HIV positive people in Mongolia

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Background / Hypothesis:

Viral hepatitis is one of the health concerns in Mongolia, the reported prevalence of hepatitis B surface antigen (HBsAg) was 9-10%, anti-hepatitis C virus ranged 11-25% and the prevalence of hepatitis D virus RNA was about 67.5% among HBsAg-positive people. 420 cases of HIV infection have been registered in Mongolia since 1992. Mongolia is a country with a low (less than 0.1%) prevalence of HIV infection in the population; however, the prevalence among men who have sex with men is 8.8%.

Materials and Methods:

A cross-sectional analytical study was conducted using data from 1992 to 2024. Data was collected from the AIDS, STIs Surveillance Department, and analyzed in the SPSS program as a descriptive prevalence survey. The Biochemical tests were performed on Biosystems BA200 used. We used all biochemical parameters that were analyzed at the time and first and fifth years after HIV diagnosis. The improvement or deterioration in the biochemical profiles, as well as the significance of the same, was analyzed using the chi-square test. The mean, standard deviation and distribution of the variables between the groups (HIV plus HBV; HIV plus HCV) were studied using a t-test and a p-value <0.05 was considered significant.

Results:

420 people are registered with HIV and AIDS in Mongolia, 21.0% (88/420) of them have a hepatitis virus infection. Among these individuals living with HIV 41.5% were HBsAg positive (HBV), 51.2% were anti-HCV positive, 7.3% had HBV and HCV co-infection. ART coverage rates exceeding among HIV+HBV, HIV+HCV and HIV+HBV+HCV were 94.1%, 90.5% and 100%. The mean ALT and AST levels of the study participants at diagnosis were 64.1 ± 110.3 and 66.7 ± 103.4 , and 44.3 ± 38.9 and 59.5 ± 60.6 in the first year after HIV diagnosis, 55.1 ± 58.4 and 59.2 ± 50.1 in the fifth year after HIV diagnosis. Both AST and ALT did not show an upward trend over follow-up and were not statistically significant. The mean ALT was high in HCV/HIV infection in comparison to HBV/HIV and it was also significant ($p < 0.05$). The mean ALT and AST levels were statistically significant ($p < 0.05$) depending on whether the antiretroviral therapy coverage. Conclusions: Approximately 21.0% of individuals living with HIV were co-infected with one or more hepatitis viruses. Although substantial differences in biochemical parameters were observed at the time of initial diagnosis, these differences diminished during follow-up. The extent of improvement varied depending on the type of hepatitis virus involved and on whether the patient was receiving antiretroviral therapy.

Keyword(s) : Mongolia, HIV, HBV, HCV, co-infection

1728 - T Lymphocyte Subsets Participate in the Pathogenesis of Primary Biliary Cholangitis

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Primary biliary cholangitis (PBC) is a chronic autoimmune liver disease characterized by non-suppurative destruction of small intrahepatic bile ducts, with immune dysregulation as a core pathogenic driver. This review synthesizes recent advances in understanding the roles of diverse T lymphocyte subsets in PBC initiation and progression. CD4⁺ T cell subsets, including Th1, Th17, Th2, T follicular helper (Tfh), and peripheral helper T (Tph) cells, orchestrate autoimmune activation and promote chronic inflammation, fibrosis, and humoral immunity by secreting pro-inflammatory cytokines (e.g., IFN- γ , IL-17, IL-21) and activating B cells. CD8⁺ tissue-resident memory T (Trm) cells, particularly CD69⁺CD103⁺

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subsets, directly mediate biliary epithelial cell (BEC) damage through cytotoxic molecules like perforin and granzyme B. Follicular regulatory T (Tfr) cells, which suppress excessive B and Tfh cell activity, show impaired function, with a reduced Tfr/Tfh ratio correlating with disease severity. Innate-like T cells, such as mucosal-associated invariant T (MAIT) cells and natural killer T (NKT) cells, accumulate in the liver to amplify inflammation and fibrosis via cytokine secretion and cytotoxicity. $\gamma\delta$ T cells bridge innate and adaptive immunity, while innate lymphoid cells (ILCs) modulate Th cell responses through subset-specific cytokine profiles. Regulatory T cells (Tregs), critical for immune tolerance, exhibit reduced numbers and dysfunction in PBC, and progenitor exhausted T (Tpex) cells sustain chronic immune activation under persistent antigen stimulation. These T cell subset abnormalities—including Treg/Th17 imbalance, pathogenic Trm accumulation, and dysregulated humoral immunity—collectively drive PBC progression. Understanding their sequential and interactive pathogenic mechanisms provides a foundation for targeted immunotherapies, such as Treg modulation, JAK pathway inhibition, and CAR-T therapy, offering new avenues to improve clinical outcomes for PBC patients.

Keyword(s) :

1730 - Histone deacetylase inhibitor givinostat attenuates metabolic dysfunction associated steatotic hepatitis and liver fibrosis

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Background / Hypothesis:

Metabolic Dysfunction-Associated Steatotic Hepatitis (MASH) is a prevalent chronic liver disease with increasing global incidence. Hepatic inflammation is a key driver of disease progression from metabolic dysfunction-associated steatotic liver (MASL) to MASH; however, effective therapeutic options remain limited. This study investigated the therapeutic potential of givinostat, a histone deacetylase (HDAC) inhibitor, for the treatment of MASH.

Materials and Methods:

MASH was induced in mice using either a methionine- and choline-deficient (MCD) diet or a fructose, palmitate, and cholesterol (FPC) diet. The mice were treated with givinostat (10 mg·kg⁻¹·d⁻¹, intraperitoneally) for 8 or 10 weeks. After the treatment period, liver tissues were collected for analysis. In vitro

studies were conducted using human hepatocellular carcinoma cell line HepG2 and human fetal hepatocyte cell line L02 to assess the effect of givinostat on palmitic acid-induced lipid accumulation.

Results:

Givinostat administration significantly alleviated hepatic inflammation and attenuated fibrosis in MCD diet-induced MASH mice. RNA sequencing of liver tissues from these mice showed that givinostat potently suppressed the expression of inflammation-related genes and modulated a wide array of lipid metabolism-related genes. In both HepG2 and L02 cells, givinostat markedly reduced intracellular lipid accumulation induced by palmitic acid. The therapeutic benefits of givinostat were further confirmed in the FPC diet-induced MASH model, where it significantly ameliorated hepatic steatosis, inflammation, and liver injury.

Conclusions: Givinostat demonstrates efficacy in reversing diet-induced MASH in mouse models by improving steatosis, inflammation, and fibrosis. These findings suggest that givinostat may represent a promising therapeutic agent for the treatment of human MASH.

Keyword(s) : Metabolic Dysfunction-Associated Steatotic Hepatitis; inflammation; histone deacetylase inhibitor; givinostat; epigenetics

1733 - The ATP7B c.1543+40G>A Splice Site Variant was Confirmed as Pathogenic through Familial Segregation Analysis in the Genetic Diagnosis of Wilson Disease

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Background / Hypothesis:

A significant challenge in the genetic diagnosis of Wilson's disease (WD) is the interpretation of variants of uncertain significance (VUS).

The ATP7B c.1543+40G>A variant represents one such VUS with conflicting database classifications, impeding definitive diagnosis. This study aimed to determine the pathogenicity of the ATP7B c.1543+40G>A variant through comprehensive familial segregation analysis and in silico predictive modeling, and to assess its role in resolving diagnostic uncertainty.

Materials and Methods:

Genomic DNA was sequenced using next-generation

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sequencing (NGS) for the full-length ATP7B gene, with a tailored analytical pipeline enabling comprehensive variant screening and filtering. Identified variants were subsequently validated and assessed for zygosity via Sanger sequencing using specific primers and standard PCR amplification. Familial co-segregation analysis was conducted using Sanger sequencing of the proband's mother and sister. The potential impact of the identified variants on splicing was evaluated computationally using SpliceAI and MaxEntScan.

Results:

This study present a 30-year-old male proband with a clinical and biochemical profile consistent with WD, confirmed by a Leipzig score of ≥ 4 . Genetic analysis identified compound heterozygosity for two ATP7B variants: the known likely pathogenic c.2447+5G>T and the VUS c.1543+40G>A. Segregation analysis revealed that the proband's mother was a heterozygous carrier for c.1543+40G>A alone, while his asymptomatic sister also carried both variants in compound heterozygote. This confirmed the in trans inheritance of the VUS with a known pathogenic allele, fulfilling the ACMG/AMP criterion for segregation. In silico analysis confirmed the significant splice-disrupting effect of c.2447+5G>T, while predictions for c.1543+40G>A were benign. The co-segregation evidence enabled the reclassification of c.1543+40G>A from VUS to "Likely Pathogenic."

Conclusions:

This study provides definitive evidence for the pathogenicity of the ATP7B c.1543+40G>A variant through familial segregation analysis, underscoring the critical role of family-based genetic studies in resolving VUS and achieving a precise molecular diagnosis for Wilson's disease. The discordant phenotype between the heterozygous siblings further highlights the incomplete penetrance often associated with this disorder.

Keyword(s) :

1734 - Diminished protection of Vitamin E in pediatric MASLD with lipid disorder findings from a national wide survey

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Background / Hypothesis: The growing global prevalence of pediatric metabolic dysfunction-associated fatty liver disease (MASLD) is a significant concern, contributing to progressive liver fibrosis and

long-term hepatic morbidity. While Vitamin E is the most promising therapeutic candidate, its efficacy in randomized controlled trials remains inconsistent. Evidence from adult studies suggests that hyperlipidemia may diminish the protective effect of Vitamin E against MASLD. We investigated if this diminished protective association is present in adolescents with co-occurring lipid disorders by using cholesterol-adjusted serum α -tocopherol concentrations as a proxy for genuine biological protection.

Materials and Methods: We analyzed cross-sectional data from the 2017-2018 National Health and Nutrition Examination Survey (NHANES), including 305 adolescents aged 12 to 19 years. Pediatric MASLD was defined by expert consensus, requiring steatosis assessed by controlled attenuation parameter (CAP) > 236 dB/m plus at least one metabolic risk factor. Serum α -tocopherol was adjusted for total cholesterol using the residual method to correct for confounding by lipid levels. Lipid status was categorized as normal, borderline, or dyslipidemia based on established pediatric cutoffs. We used weighted logistic regression to examine the association between cholesterol-adjusted serum α -tocopherol and MASLD, stratified by lipid status.

Results: MASLD was identified in 98 participants (32.0%). Among adolescents with normal lipid status, higher cholesterol-adjusted serum α -tocopherol concentrations were inversely associated with MASLD across all models: fully adjusted odds ratio (OR) was 0.37 (95% confidence interval (CI), 0.17-0.84; $p=0.026$). Crucially, this significant protective association was abolished in adolescents with borderline and dyslipidemia lipid status, where no statistically significant association was observed. Sensitivity analysis confirmed these findings.

Conclusions: Our results suggest that the protective association between Vitamin E and MASLD is attenuated in the presence of lipid disorders in adolescents. This indicates a limited protective role of Vitamin E in pediatric MASLD when co-existing lipid dysfunction is present, underscoring the necessity for further research into the underlying mechanism.

Keyword(s) : MASLD, NHANES, Vitamin E, α -tocopherol, lipid disorder

1736 - An Increase in Regulatory B Cells and a Reduction in T Helper 17 Cells Play a Crucial Role in

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Achieving Tolerance in Liver Transplantation

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Background / Hypothesis:

This study investigates the dynamics of regulatory B cells (Bregs) during immunosuppression (IS) tapering and their contribution to tolerance in liver transplantation (LT) patients.

Materials and Methods:

We prospectively enrolled 15 LT patients who had normal liver function, no history of rejection, and had undergone LT more than five years ago. Patients were followed up every three months, and IS doses were tapered by 25% every 6–9 months. At each visit, blood samples were collected, and the proportions of T cells and B cells were analyzed by flow cytometry. Confocal microscopy and in vitro analysis were also performed to assess Breg function in LT patients.

Results:

Over a median follow-up of 42 months, eight patients successfully discontinued IS without rejection (tolerance group). One patient experienced rejection (rejection group), while the remaining six patients reduced their IS dose to 25% without rejection (minimization group). During IS tapering, Treg cells and the Treg/Th17 ratio increased in the tolerance group but decreased before rejection in the rejection group (Figure A). Similarly, the tolerance group exhibited an increase in Bregs and the Breg/Th17 ratio, whereas a decrease was observed in the rejection group (Figure A, B). Moreover, a negative correlation between Bregs and Th17 cells and a significant positive association between Bregs and plasmablast B cells were identified. In liver tissue, confocal analysis showed a significant increase in Tregs and Bregs in the tolerance group compared to the rejection group (Figure C). In vitro analysis demonstrated a reduction in Th17 cells following Breg treatment, and vice versa (Figure D). Furthermore, treatment with Faecalibacterium, enriched in the tolerance group, led to an increase in

Bregs and IL-10 levels.

Conclusions:

During IS tapering, Bregs increase and contribute to the achievement of tolerance in LT patients. These findings suggest that Bregs may serve as potential biomarkers and therapeutic targets for promoting tolerance in LT.

Keyword(s) :

1737 - Prognostic Impact of Cirrhosis in Patients with Hepatocellular Carcinoma and Concurrent MASLD Undergoing Surgical Resection

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as one of the major subtypes of liver disease and a growing etiology of hepatocellular carcinoma (HCC), especially in patients without viral hepatitis. Meanwhile, although cirrhosis is traditionally regarded as an important milestone in liver disease progression, the effect of cirrhosis on the outcome of HCC patients has been variable, particularly for those with concurrent MASLD. This study therefore aimed to determine whether cirrhosis influences baseline characteristics, overall survival (OS), or recurrence-free survival (RFS) in HCC patients with concurrent MASLD undergoing surgical resection.

Materials and Methods:

We retrospectively analyzed 460 treatment-naïve HCC patients with concurrent MASLD who received curative hepatic resection at a tertiary medical center between 2015 and 2023. Cirrhosis was determined by an Ishak fibrosis grade greater than 5 in the surgical specimen. Baseline characteristics were compared between the cirrhosis and non-cirrhosis groups. Univariate and multivariate Cox proportional hazards models were applied to identify independent risk factors for poor OS and RFS. OS and RFS were analyzed and compared using Kaplan–Meier survival analysis.



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Results:

After a median follow-up of 51.8 months (IQR, 13.3–72.0 months), 104 patients died, corresponding to a 5-year OS rate of 76.5%. Baseline demographics showed significant differences in tumor staging, tumor size, inflammatory activity, HCV positivity, and serum biochemistry, including platelet count, international normalized ratio (INR), albumin, aspartate transaminase (AST), and creatinine levels, between the cirrhosis and non-cirrhosis groups. The 5-year OS was comparable between the two groups, with 77.1% in patients with cirrhosis and 76.2% in those without cirrhosis ($p = 0.42$). Five-year RFS was likewise comparable between groups (50.6% vs. 53.2%, $p = 0.27$). Multivariate Cox proportional hazards models did not identify cirrhosis as an independent risk factor for poor OS or RFS.

Conclusions:

Despite differences in tumor characteristics, serum biochemistry, and inflammatory activity, cirrhosis did not significantly influence postoperative OS or RFS in HCC patients with concurrent MASLD undergoing surgical resection.

Keyword(s) : MASLD, Hepatocellular carcinoma, Cirrhosis

1741 - Alcohol Use–Related Outcomes Following Fecal Microbiota Transplantation in Steroid Ineligible Severe Alcohol Associated Hepatitis A Secondary Analysis of a Randomized Controlled Trial

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Background: Steroid-ineligible patients with severe alcohol-associated hepatitis (SAH) have high mortality with no established effective alternatives to corticosteroid therapy. Gut dysbiosis plays a significant pathogenic role in SAH. Fecal microbiota transplantation (FMT) by restoring microbial balance and gut permeability, has been shown to improve short-term survival, and reduce craving behavior in AUD-cirrhosis. However, the effect of FMT on alcohol

use behavior in SAH patients remains underexplored.

Methods: This is a secondary analysis from single-center, randomized controlled trial (2021–2023, ILBS, New Delhi; NCT05285592), 46 steroid-ineligible SAH patients (mDF > 120, sepsis, serum creatinine > 2mg/dl, or > 3 organ failure) between 18–70 years, were randomized in a 2:1 ratio, to receive FMT ($n=31$) or standard medical therapy (SMT, $n=15$). FMT was administered via nasoduodenal/jejunal tube once daily for 7 days using 100 mL fresh donor stool suspension using Institutional Protocol. Primary endpoint was 28-day survival, and secondary endpoints were changes in alcohol craving questionnaire-short form (ACQ-SF) index, relapse rates, MELD and mDF at 90 and 180 days. Microbiome profiling using 16S rRNA sequencing was also performed. In this study we present only the secondary end-points related to AUD. During the follow-up all patients received mandatory sessions of Brief Intervention Counseling from the Addiction medicine team.

Results: Among 46 randomized patients, baseline characteristics (mean age ~40 years, 97% male, mean MELD scores 31.7 vs 29.1; $p=0.8$, mDF 85.1 vs 84.1; $p=0.1$) were comparable between two groups. All patients tolerated 7 FMT doses. At 90 days, FMT recipients had significantly lower alcohol craving (ACQ-SF reduced from 3.5 ± 0.69 to 2.8 ± 0.80) compared to SMT (3.3 ± 0.80 to 3.4 ± 0.59 , $p=0.01$). Alcohol relapse was also significantly lower in the FMT group (19.2% vs. 53.8%, $p<0.05$). Lapse rates (any) at 180 days were lower with FMT (3.2% vs 13.3%), corresponding to a 76% relative risk reduction ($RR = 0.24$), although this did not reach statistical significance ($p = 0.24$) due to low event counts. Among these survivors, lapse (any) occurred in only 1/13 (7.7%) in the FMT arm compared with 2/4 (50%) in the SMT arm. Delta MELD at day 180 was significantly better in the FMT than the SMT group (-5.9 vs -0.7 , $p=0.016$). Longitudinal 16S Metagenome Analysis showed a decline in *Veillonella parvula*, *Lactobacillus agilis*, and *Akkermansia muciniphila*, indicating an early shift (Day 7 vs Day 0); which were further established on Day 30 analysis.

Conclusion: FMT could provide dual benefit in targeting hepatic and behavioral dimensions of alcohol-related liver disease

Keyword(s) : Alcoholic hepatitis, FMT, alcohol craving, steroid ineligible, gut-liver-brain axis, dysbiosis.

1742 - Pretreatment neutrophil to Lymphocyte Ratio as a Predictive Marker for Tumor Response to Atezolizumab plus Bevacizumab in Hepatocellular Carcinoma A Multicenter Retrospective Study



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Background / Hypothesis:

Atezolizumab combined with bevacizumab has been used in the treatment of advanced hepatocellular carcinoma (HCC). However, reliable predictive biomarkers for evaluating tumor response to this therapy remain limited. This study aimed to identify predictive factors in patient with HCC undergoing treatment with atezolizumab and bevacizumab.

Materials and Methods:

This multicenter retrospective study included 943 patients with HCC who received atezolizumab plus bevacizumab. Factors associated with tumor response were analyzed.

Results:

According to the Response Evaluation Criteria in Solid Tumors, the rates of complete remission, partial response, stable disease, and progressive disease were 2.9 %, 22.9 %, 48.8 % and 25.5 %, respectively. The objective response rate (ORR) and disease control rate (DCR) were 25.8 % and 74.6 %, respectively.

Progression free survival and overall survival were significantly associated with best tumor response ($P < 0.001$). Multivariate analysis identified BCLC stage (HR = 0.478, 95% CI = 0.278 – 0.821, $P = 0.007$) and neutrophil-to-lymphocyte ratio (NLR) (HR = 0.670, 95% CI = 0.488 – 0.920, $P = 0.013$) as independent predictive factors for objective tumor response. The

ORR and DCR were 31.2 % and 77.2 %, respectively, in the NLR <2 group, and 23.7 % and 73.5 %, respectively, in the NLR ≥ 2 group ($P < 0.001$).

Conclusions:

Pretreatment NLR was correlated with the tumor response, which was subsequently associated with survival outcomes. NLR may serve as a predictor of a lack of response to atezolizumab plus bevacizumab treatment. Therefore, NLR represents a readily measurable biomarker with the potential to support the timely identification of HCC patients who are more likely to benefit from atezolizumab and bevacizumab treatment.

Keyword(s) : hepatocellular carcinoma, neutrophil-to-lymphocyte ratio, atezolizumab, bevacizumab, tumor response

1743 - Area deprivation and hepatitis C infection in Korea A multilevel analysis 2005 2020

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Background / Hypothesis:

Although the prevalence of hepatitis C virus (HCV) in Korea is decreasing, regional disparities remain. This study aimed to examine the impact and temporal changes in individual characteristics and regional health inequalities on HCV infection, to provide a framework for HCV elimination and management.

Materials and Methods:

This nationwide population-based multilevel case-control study was conducted using National Health Insurance Service (NHIS) and population census data from 2005, 2010, 2015, and 2020. The Area Deprivation Index (ADI) was calculated for each area (region/town) using population census data. Sex- and age-standardized HCV prevalences were estimated for each area using NHIS data. The association between area-level HCV and ADI was examined using correlation and linear regression analyses. Individual- and area-level HCV infection risks were estimated using logistic regression and multilevel logistic regression models incorporating sex, age, HCV prevalence, and ADI.

Results:

The HCV prevalence (per 100,000) decreased from 151 to 117 (2005–2020). The maximum inter-area ADI gap

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decreased modestly (34.64 to 32.50). ADI correlated with area HCV prevalence in all years, with stronger associations in high-prevalence areas ($\geq 200/100,000$). In logistic regression, residents of the most deprived areas had elevated risks versus the least deprived (2020 adjusted odds ratio 1.37). Multilevel models showed age (individual level) and area HCV prevalence (area level) were dominant determinants. Deprivation increased risk in ADI-only models, but not significance after adjusting for area HCV prevalence and individual factors.

Conclusions:

Effective HCV elimination requires tailored screening and treatment strategies that prioritize socioeconomically vulnerable areas among the high-prevalence areas.

Keyword(s) : Area Deprivation Index, Hepatitis C Virus, Prevalence, Health Inequality, Multilevel Analysis

1744 - Proteomics Biomarker for Predicting Responders to Atezolizumab and Bevacizumab Therapy in Hepatocellular Carcinoma

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Background / Hypothesis:

Atezolizumab with bevacizumab are now standard first-line treatments for advanced hepatocellular carcinoma (HCC). Despite their clinical success, predicting treatment response remains difficult due to the lack of validated biomarkers. This study aimed to explore circulating serum proteins as potential predictive biomarkers of immunotherapy efficacy in advanced HCC.

Materials and Methods:

HCC patients on atezolizumab and bevacizumab were classified as responders if they achieved stable disease, partial response, or complete response, and as non-responders if their disease progressed after three

therapy cycles. Differences in pre-treatment serum protein expression between these groups were evaluated using mass spectrometry-based proteomics.

Results:

Of 44 patients, non-responders (n = 12) showed elevated C-reactive protein and S100A9, while responders (n = 32) exhibited higher levels of proteins such as transferrin, transthyretin, APOC3, and clusterin. Network analysis identified APOC3 and CLU as central to protein interaction networks associated with treatment outcomes.

Conclusions:

These findings highlight circulating serum proteins as promising predictive biomarkers for immunotherapy response in advanced HCC, offering a potential tool for more personalized treatment selection.

Keyword(s) : HCC, immunotherapy, proteomics

1745 - Flare to Failure Hemoadsorption Therapy for Acute on Chronic Liver Failure in Chronic Hepatitis B Infection Unmasked During Antikoch's Therapy

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Background / Hypothesis:

Chronic Hepatitis B (CHB) is widespread in the Philippines and poses a risk of liver failure in the absence of therapy. This case illustrates CHB flare that occurred during pulmonary tuberculosis (PTB) treatment, which progressed to acute-on-chronic liver failure (ACLF). It highlights hemoadsorption as a potential bridging therapy where liver transplantation is not readily accessible.

Materials and Methods:

A 39-year-old woman with bacteriologically-confirmed PTB presented with jaundice, anorexia, and epigastric pain. Physical examination revealed jaundice but no stigmata of liver cirrhosis. Laboratory tests showed elevated transaminases, coagulopathy, hypoalbuminemia, hyperbilirubinemia, chronic hepatitis B infection, and newly-diagnosed diabetes. Abdominal ultrasound revealed hepatic steatosis. Anti-TB therapy was discontinued. Antiviral and hepatoprotective agents were started. Supportive care included dietary and electrolyte correction,

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management of hepatic encephalopathy, antibiotics, insulin therapy, and ICU admission.

Results:

Double plasma molecular adsorption system (DPMAS) was initiated as bridging therapy, resulting in clinical and biochemical improvements, including a reduction in hepatic encephalopathy from grade 3 to 1 and decrease in bilirubin. However, due to financial limitations, treatment intervals were suboptimal, reducing therapeutic effectiveness. The patient's condition deteriorated due to hospital-acquired infection which led to her demise.

Conclusions:

This case underscores the need for standardized protocols that include early etiological assessment, risk stratification, and the use of hemoadsorption guided by bilirubin levels and SOFA score trends. Simultaneously, government policy reforms are needed to minimize system-related barriers in liver transplantation and to improve access to advanced liver support therapies.

Keyword(s) : Chronic Hepatitis B, Acute-on-Chronic Liver Failure, Hemoadsorption, DPMAS, CHB Flare, Anti-Tuberculosis Therapy

1746 - Decision Tree model using Exosomes to Predict Favorable Outcome of Atezolizumab and Bevacizumab Combination Therapy in Hepatocellular Carcinoma

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Background / Hypothesis:

Atezolizumab combined with bevacizumab represents the current first-line treatment for advanced hepatocellular carcinoma (HCC). However, no biomarker has yet been firmly established to accurately predict patient responses to this regimen. Previous work from our research group suggested that miR-30a-5p could serve as a potential predictive biomarker. This study aimed to validate the clinical significance of this exosomal microRNA in a larger patient cohort.

Materials and Methods:

Patients with advanced HCC undergoing atezolizumab plus bevacizumab therapy were enrolled. After three treatment cycles, patients were classified as responders if they achieved stable disease, partial response, or complete response, and as non-responders if their disease progressed. Serum extracellular vesicles were isolated from blood samples collected prior to the therapy. Exosomal RNAs were extracted, which were profiled using next-generation small RNA sequencing. Decision tree analysis was employed to identify microRNAs capable of distinguishing responders from non-responders.

Results:

Samples from 13 responders and 11 non-responders were analyzed. Sequencing identified 1,076 microRNAs, among which let-7c-5p, miR-30a-5p, and miR-192-5p were significantly upregulated in responders. Consistent with previous findings, miR-30a-5p levels were markedly higher in responders, showing a significant difference ($p = 0.0039$). Decision tree analysis indicated that a miR-30a-5p expression level of 9.67 or greater effectively distinguished responders from non-responders. Similarly, random forest modeling identified miR-30a-5p as the most influential variable in predicting treatment response.

Conclusions:

miR-30a-5p demonstrates potential as a reliable exosomal biomarker for identifying patients with advanced HCC who are most likely to benefit from atezolizumab and bevacizumab therapy.

Keyword(s) :

1748 - Analysis on the differences of clinicopathological characteristics in drug induced liver injury patients with positive or negative autoantibodies and different clinical types

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Background: To investigate the differences in clinical and pathological characteristics among patients with drug-induced liver injury (DILI) by autoantibody status and R value-based clinical types to inform clinical practice.

Methods: This study included 138 patients hospitalized from January 2016 to July 2023 with biopsy-confirmed DILI. Patients were grouped by autoantibody results (positive/negative), liver histopathological changes ($< G2$ and $\geq G2$, $< G3$ and $\geq G3$, $< G4$ and $\geq G4$, $< S2$ and $\geq S2$, $< S3$ and $\geq S3$, $< S4$ and $\geq S4$, mild pathological injury [$< G2$ + $< S2$] and significant pathological injury [$\geq G2$ and/or $\geq S2$]) and R values for DILI classification ($R \geq 5$: hepatocellular injury type; $2 < R < 5$: mixed type; $R \leq 2$: cholestatic type), with clinicopathological characteristics compared.

Results: Of the enrolled patients, 79 were autoantibody-negative and 59 were autoantibody-positive, including 59 cases (100.00%) positive for anti-nuclear antibody and 8 cases (13.56%) positive for anti-mitochondrial antibody. Traditional Chinese Medicine (including single herbs, herbal dietary supplements, or compound preparations) accounted for the highest proportion of medications causing DILI (55/138, 39.86%). Significantly higher serum globulin (GLB) ($t = 2.983$), alkaline phosphatase ($Z = 2.042$), and adenosine deaminase (ADA) ($Z = 2.582$), and a lower albumin (ALB)/GLB ratio ($t = 2.211$), were found in the autoantibody-positive versus negative group (all $P < 0.05$). The autoantibody-positive group had a higher mean Ridit value for liver inflammation activity grade (G) ($u = 2.302$, $P = 0.021$) and a higher proportion of $\geq G3$ and $G4$ ($\chi^2 = 4.471$, 6.478 ; $P = 0.034$, 0.011). The $R \geq 5$, $2 < R < 5$, and $R \leq 2$ groups differed significantly in serum total bilirubin (TBiL) ($H = 11.166$), alanine aminotransferase (ALT) ($H = 109.01$), aspartate aminotransferase (AST) ($H = 75.663$), ALT/AST ratio ($H = 23.096$), adenosine deaminase (ADA) ($H = 34.013$), and gamma-glutamyl transferase (GGT) ($H = 9.831$) (all $P < 0.01$). The $2 < R < 5$ group had the highest mean Ridit value for G and proportion of $\geq G3$ grades, with significant differences among the three groups ($\chi^2 = 7.385$, 7.163 ; all $P < 0.05$). Independent risk factors for G severity were ALB, GGT, and ALT/AST; for advanced fibrosis stage (S) were ALT/AST, platelet count, and ALB; and for combined G/S severity were GGT, ALB

and ALT/AST.

Conclusions: The clinical and pathological features of DILI vary across different disease states. Autoantibody-positive DILI and mixed-type DILI ($2 < R < 5$) are associated with more severe liver inflammation grades.

Keyword(s) : Drug-induced liver injury, Autoantibody, Clinical types, Pathology, Clinical characteristics

1749 - Seroprevalence and Epidemiological Features of Hepatitis E Virus Infection in South Korea Analysis of KNHANES Serum Samples

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Background / Hypothesis: Epidemiological data on hepatitis E virus (HEV) prevalence in South Korea remain limited. This study aimed to comprehensively assess the national seroprevalence of HEV, with analyses stratified by sex, age, and region.

Materials and Methods: A total of 840 blood samples were selected based on sex, age, and regional distribution from participants of the 2020 Korea National Health and Nutrition Examination Survey (KNHANES). All sera were qualitatively stored at the National Biobank of Korea.

Results: Among the 840 samples analyzed, 182 (21.7%) were positive for anti-HEV IgG only, 15 (1.8%) for anti-HEV IgM only, and 13 (1.5%) for both IgG and IgM. The remaining 630 samples (75.0%) were negative for both antibodies. Anti-HEV IgG positivity was higher in males (28.3%) than in females (18.1%) and showed a marked increase with age, rising from 3.0% in those aged 10–39 to 50.6% in those aged 70 and older. Compared to anti-HEV IgG-negative individuals, those who were IgG-positive were significantly older and showed higher liver fibrosis indices based on FIB-4. The anti-

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HEV IgM positivity rate was 3.3% (28/840), with the highest prevalence in the 40–59 age group. Among IgM-positive individuals, 13 (46.4%) were also IgG-positive. No significant differences in age or sex were observed between IgM-positive and IgM-negative individuals. Similarly, APRI and FIB-4 scores did not differ significantly between the two groups. Although elevations in AST and ALT were more frequent among IgM-positive individuals, these differences were not statistically significant. Real-time reverse transcription PCR performed on all 28 IgM-positive samples yielded negative results for HEV RNA.

Conclusions: This nationwide serological analysis revealed a substantial seroprevalence of both anti-HEV IgG and IgM among the Korean population, indicating significant past and recent exposure to HEV. These findings highlight the importance of hepatitis E as a public health concern in South Korea.

Keyword(s) : Viral hepatitis, Hepatitis E, Epidemiology

1752 - Exploring the Associations of Liver PDFF with Mortality and Disease Incidence A Prospective Study in the UK Biobank

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Background / Hypothesis:

Nonalcoholic fatty liver disease (NAFLD) is closely associated with obesity, diabetes, and cardiovascular disease. Clinical assessment often relies on biopsy or limited imaging, and population-level age- and sex-specific trajectories and long-term outcomes remain unclear. Previous studies using proton density fat fraction (PDFF) provided some insights but had limited sample sizes and lacked comprehensive modeling of nonlinear trends and systematic evaluation of mortality and comorbidities. This study leveraged a large UK Biobank cohort to characterize PDFF distributions and their associations with mortality and metabolic and cardiovascular traits.

Materials and Methods:

We analyzed 37,358 UK Biobank participants with liver PDFF data (median age 64 years; median PDFF 4.8%). Age- and sex-specific distributions and nonlinear trends were assessed using bootstrap resampling and generalized additive models (GAMs). Imaging-derived variables were standardized. Associations between PDFF and all-cause or cancer-specific mortality were examined using Cox proportional hazards models with restricted cubic splines (RCS), adjusting for age, sex, and BMI. Phenome wide association studies (PheWAS)

and Pearson correlations were performed to link PDFF with clinical and biomarker traits.

Results:

PDFF exhibited nonlinear age trajectories, with postmenopausal women exceeding men. High PDFF was significantly associated with increased all-cause mortality ($P < 0.001$), while low PDFF conferred protective effects. Tumor-specific mortality showed a similar pattern, with hazard ratios exceeding 1.0 at lower PDFF levels. PheWAS identified positive associations between PDFF and fatty liver (K76.0), type 2 diabetes (E11.9), and diverticular disease without perforation or abscess (K57.3). PDFF correlated positively with BMI, waist circumference, triglycerides, ALT, and GGT, and inversely with HDL, SHBG, and vitamin D; multiple metabolic and cardiovascular polygenic risk scores were also positively associated with PDFF.

Conclusions:

Liver PDFF is a robust, noninvasive marker of metabolic and mortality risk, providing valuable information for population screening and individual risk assessment. Incorporating PDFF into clinical evaluation may improve early detection and guide preventive interventions.

Keyword(s) : Liver, PDFF, UKBB

1755 - Role of IL 16 in autoimmune hepatitis

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Background / Hypothesis: Autoimmune hepatitis (AIH) is a T cell-mediated chronic liver inflammation with unclear pathogenesis, facing challenges like difficult diagnosis and suboptimal therapy, necessitating novel molecular targets. Interleukin-16 (IL-16), a proinflammatory cytokine regulating T cell function, is linked to inflammatory diseases and liver injury, but its role in AIH—including tissue/serum expression differences and correlation with AIH biomarkers (ALT, AST, IgG)—remains undefined.

Materials and Methods: A case-control study design was adopted, with 30 AIH patients selected as the study group and 30 healthy individuals with completely matched gender and age as the control group. IL-16 in the serum of the two groups was detected by ELISA, IHC, and proteomics. In addition, the concentrations of serum autoantibodies, immunoglobulins (with IgG as

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the core indicator), and related cytokines (including IL-18, soluble CD48 (sCD48), follicle-stimulating hormone (FSH), and transforming growth factor- β (TGF- β)) were simultaneously detected in both groups.

Results: There was no statistically significant difference in the serum IL-16 between the AIH patient group and the HC group ($P > 0.05$); however, IL-16 in the liver tissue of AIH patients was significantly higher than that in the healthy control group, and the difference was statistically significant ($P < 0.01$). Further correlation analysis showed that the serum IL-16 in AIH patients had a certain correlation with ALT, AST, and IgG, suggesting that IL-16 may be closely related to the degree of liver inflammatory injury and the state of immune activation in AIH patients.

Conclusions: The results of this study confirm that IL-16 is involved in the pathogenesis of AIH, providing experimental evidence for clarifying the regulatory role of IL-16 in the progression of AIH and the feasibility of using it as a potential therapeutic target for AIH. Meanwhile, it is suggested that although there is no significant difference in serum IL-16 between the two groups, its correlation with key pathological indicators of AIH still supports its potential as a candidate biomarker for auxiliary diagnosis of AIH, which can provide a new reference direction for the early identification of the disease.

Keyword(s) :

1756 - Recombinant human thrombopoietin promotes platelet recovery in hepatocellular carcinoma patients receiving interventional therapy combined with targeted and immunotherapy (SYNERGY TPOACT) a retrospective cohort study

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Background / Hypothesis:

Combination regimens involving interventional therapy (TACE/HAIC), tyrosine kinase inhibitors (TKIs), and

immune checkpoint inhibitors (ICIs) have become the standard of care for advanced hepatocellular carcinoma (HCC). However, cumulative myelosuppression frequently leads to severe thrombocytopenia, necessitating dose reduction or treatment interruption. While recombinant human thrombopoietin (rhTPO) is effective in chemotherapy-induced and immune thrombocytopenia, evidence in this specific population remains limited.

Materials and Methods:

This retrospective study included 60 HCC patients who developed thrombocytopenia (platelets $\leq 80 \times 10^9/L$) after combined interventional, targeted, and immunotherapy at Guizhou Provincial People's Hospital, China (March 2022-March 2025). All patients received subcutaneous rhTPO. The primary endpoint was overall response rate (ORR), defined as complete response (CR: platelets $\geq 100 \times 10^9/L$) or partial response (PR: platelets increase $\geq 25 \times 10^9/L$). Secondary endpoints included platelet counts, platelet increments analyzed by rhTPO treatment durations and across BCLC stages, and changes in liver/renal function and coagulation parameters.

Results:

Baseline clinical characteristics were comparable across subgroups. Most patients were BCLC stage C (50.0%) and Child-Pugh class B (55.0%). The mean baseline platelet count was $51.93 \times 10^9/L$. Following rhTPO administration, the ORR was 68.3% (CR:38.3%, PR:30.0%). Platelets increased by a mean of $47.76 \times 10^9/L$ ($p < 0.0001$, 95% CI: 36.81-58.70). Mean platelet increments at 3, 5, and 7 days were $25.21 \times 10^9/L$, $42.43 \times 10^9/L$, and $64.91 \times 10^9/L$, respectively, peaking at day 7. Efficacy was consistent across BCLC stages. Notably, a treatment duration ≥ 7 days was significantly associated with achieving ORR (OR=7.45, 95%CI: 1.95-28.50, $p=0.003$). No treatment-related hepatorenal toxicity or thromboembolic events were observed.

Conclusions:

rhTPO is effective and safe for improving treatment-related thrombocytopenia in HCC patients receiving interventional therapy combined with targeted and immunotherapy. Our findings suggest that a minimum treatment duration of 7 days yields optimal platelet recovery. Prospective trials are needed to validate these findings and guide clinical practice.

Keyword(s) : rhTPO, platelet, HCC, interventional therapy combined with targeted and immunotherapy

1759 - Value of Preoperative Contrast Enhanced CT based Radiomics for Predicting CK19 Positive

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Hepatocellular Carcinoma

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Background / Hypothesis: Prediction of cytokeratin 19 (CK19) may help determine treatment strategies for hepatocellular carcinoma (HCC).

Materials and Methods: This multi-center retrospective study enrolled patients with pathologically confirmed HCC who underwent preoperative contrast-enhanced CT between February 2012 and November 2023. Based on postoperative immunohistochemical staining results for CK19, patients were categorized into CK19-positive (n=101) and CK19-negative (n=397) groups. Radiomics features were extracted from the tumor region on non-contrast, arterial, and portal venous phase CT images after image registration. In the training cohort, feature dimensionality reduction was performed using Maximum Relevance - Minimum Redundancy (mRMR) and the Least Absolute Shrinkage and Selection Operator (LASSO) regression. A radiomics model was subsequently constructed using logistic regression. The model was validated in internal and external test cohorts using pathology as the gold standard. A hybrid model integrating radiomics features with significant clinical-radiological characteristics was also developed. Model performance for predicting CK19-positive HCC was evaluated using the Area Under the Receiver Operating Characteristic Curve (AUC), and clinical utility was assessed via Decision Curve Analysis (DCA).

Results: A total of 498 patients (median age 57 years; 407 males) were included and allocated into training (n=239), internal test (n=103), and external test (n=156) cohorts. Multivariate logistic regression identified AFP >400 ng/ml (OR=4.05, 95% CI: 2.12–7.74, P<0.01) and peritumoral arterial enhancement (OR=2.15, 95% CI: 1.10–4.23, P=0.026) as independent predictors of CK19-positive HCC. The radiomics model achieved AUCs of 0.68 and 0.77 in the internal and external test cohorts, respectively. The hybrid model demonstrated superior performance, with AUCs of 0.77 and 0.78 in the internal and external test cohorts, respectively.

Conclusions: The hybrid model, integrating preoperative contrast-enhanced CT radiomics features with clinical-radiological indicators, effectively predicts CK19-positive HCC, demonstrating good generalizability and promising clinical utility.

Keyword(s) : CT Radiomics, Hepatocellular Carcinoma, Cytokeratin 19, Combined Mode

1760 - Comparison of effectiveness between phentermine topiramate and liraglutide in obese patients with metabolic dysfunction associated steatotic liver disease

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Background / Hypothesis: Anti-obesity drugs improve hepatic inflammation in patients with metabolic dysfunction-associated steatotic liver disease (MASLD). We aimed to compare the effects of phentermine/topiramate and liraglutide in obese MASLD patients

Materials and Methods: This retrospective study included 438 obese MASLD patients (body mass index >25 and detection of fatty liver on ultrasound) who received phentermine/topiramate (n=320) or liraglutide (n=118) for more than 12 months. Steatosis was assessed using the hepatic steatosis index (HSI) and controlled attenuation parameter (CAP). Fibrosis was assessed using the fibrosis index based on four factors (FIB-4), and liver stiffness (LS). Improvements in steatosis and fibrosis were compared at 12 months after treatment initiation.

Results: After applying propensity score matching, the baseline characteristics of the phentermine/topiramate group (n=108) and the liraglutide group (n=108) were comparable. Both groups showed significant reductions in steatosis (phentermine/topiramate: CAP, 321→299 dB/m; HSI, 40.6→37.0; liraglutide: CAP, 306→288 dB/m; HSI, 40.3→39.3, all p<0.05), but fibrosis improvement was only observed in the liraglutide group (phentermine/topiramate: FIB-4, 0.905→0.881, p=0.416; LS, 6.7→5.6 kPa, p=0.487; liraglutide: FIB-4, 0.838→0.717, p=0.025, LS, 6.0→4.9 kPa, p=0.011) after 12 months of treatment compared with baseline values. The Phentermine/topiramate group showed significantly greater steatosis reduction than the liraglutide group (Δ HSI, -9.32% vs. -1.87%, p<0.001; Δ CAP: -10.15% vs. -5.22%, p<0.001). However, the

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percentage of fibrosis reduction did not differ significantly between phentermine/topiramate and liraglutide groups (Δ FIB-4, 1.58% vs. -3.83%, $p=0.297$; Δ LS: -7.37% vs. -12.50%, $p=0.144$).

Conclusions: In obese patients with MASLD, phentermine/topiramate treatment resulted in better steatosis improvement, but the effect on fibrosis improvement was minimal in both groups.

Keyword(s) : fibrosis, liraglutide, with metabolic dysfunction-associated steatotic liver disease, phentermine/topiramate, steatosis

1761 - Expression of ALOX15 in the Development and Reversal of Liver Fibrosis and Its Effects on Macrophage Inflammatory Cytokine Secretion

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Background / Hypothesis: Liver fibrosis represents a critical step in the progression of chronic liver disease to cirrhosis and hepatocellular carcinoma. It is known that liver fibrosis can be reversed upon removal of the causative factors, with inflammatory mediators such as IL-6 and TNF- α secreted by macrophages regulating this process. ALOX15 participates in inflammation and lipid metabolism, but its role in reversing liver fibrosis and its effects on macrophage inflammatory function remain unclear. This study aims to elucidate the mechanism by which ALOX15 participates in reversing liver fibrosis through regulating macrophage inflammatory factors.

Materials and Methods: Proteomics analysis was performed to examine the hepatic protein expression profile in a mouse model of reversed liver fibrosis. Protein and mRNA expression levels of ALOX15 in liver tissue were determined via Western blotting and real-time quantitative PCR. Liver tissue samples were collected from 60 patients with liver fibrosis. The expression of ALOX15 in these samples was analyzed to assess its correlation with clinical staging of liver fibrosis. Additionally, in the mouse macrophage cell line RAW264.7, ALOX15 expression was knocked down using small interfering RNA (siRNA). Following inflammation induction with 1 μ g/mL LPS, levels of inflammatory cytokines including IL-6 and TNF- α were measured in cell culture supernatants and cells via WB

and qPCR.

Results: Proteomics analysis revealed that ALOX15 protein expression was significantly downregulated in both serum and liver tissues of mice in the liver fibrosis reversal model. Compared with normal mouse liver tissue, ALOX15 expression levels were markedly elevated in the liver fibrosis model group. In the 4-week and 12-week fibrosis reversal groups, ALOX15 expression exhibited a graded decrease as reversal duration increased. Analysis of clinical samples demonstrated that ALOX15 expression levels in liver tissue from patients with liver fibrosis showed a significant positive correlation with the severity of liver fibrosis. Following ALOX15 knockdown in RAW264.7 cells, mRNA expression levels of LPS-induced intracellular IL-6, TNF- α , and IL-1 β were significantly downregulated, and the protein secretion levels of these inflammatory factors in cell culture supernatants were also markedly reduced.

Conclusions: ALOX15 expression is downregulated in the reversal of mouse liver fibrosis and positively correlates with clinical fibrosis severity. Furthermore, ALOX15 participates in fibrosis reversal by regulating macrophage inflammatory cytokine secretion, suggesting its potential as a target for monitoring fibrosis reversal.

Keyword(s) : liver fibrosis; ALOX15; RAW264.7

1762 - From Liver to Spine Spinal Metastatic Compression as the Initial Manifestation of Hepatocellular Carcinoma

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Background:

Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality with a high propensity for metastatic spread, occurring in approximately one-third of patients. Although HCC commonly metastasizes to bone, spinal metastases are a serious but relatively rare (1.5-7.3%) complication with a poor prognosis. The anatomically critical location of the spinal cord necessitates prompt diagnosis and urgent

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therapeutic intervention to avert permanent neurological deterioration and functional loss. Treatment goals prioritize maintaining function, quality of life, and reducing pain.

Case Illustration:

We present a 71-year-old male who presented with progressive pain radiating from the back to the epigastric since 2 months prior to admission, characterized by stabbing, burning, and pressure sensations with numerical rating scale 8-9. A month later, the patient developed bilateral lower limb weakness and reported difficulty ambulating. Upon hospital admission, neurological examination confirmed upper motor neuron (UMN) type paraparesis with sensory level at T9 and bilateral proprioceptive dysfunction. Imaging revealed multiple nodules in the left hepatic lobe suggestive of HCC, with magnetic resonance imaging (MRI) of the thoracic spine demonstrating light compression of the T8 vertebral body corpus with marked post-contrast enhancement, suggesting metastatic pathology. Laboratory investigations showed elevated alpha-fetoprotein (AFP) 1626 ng/mL and carcinoembryonic antigen (CEA) 8.8 ng/mL. Histopathological and immunohistochemical analyses of surgical specimens confirmed metastatic carcinoma likely originating from hepatic tissue. The tumor cells were positive for CAM5.2, Glypican-3, arginase-1, HepPar1, and weakly positive for AFP and BSEP. The patient underwent hemilaminectomy with spinal cord decompression and tumor removal on day 9 of hospitalization, followed by plans to start physiotherapy and immunotherapy with atezolizumab-bevacizumab. Patient's neurological symptoms improved following surgical decompression and tumor removal.

Conclusion:

This case underscores the need for clinicians to consider hepatic malignancy with spinal metastasis as a potential cause of progressive myelopathy. In patients with current or a history of malignancy, any new or unexplained radicular pain, including epigastric pain possibly related to radiculopathy from spinal stenosis, should raise suspicion for spinal metastasis and prompt diagnostic evaluation. Early recognition through imaging and biomarkers, followed by timely surgical decompression and tumor removal, can help alleviate symptoms and prevent further neurological deterioration. Despite intervention, overall survival remains limited, emphasizing the importance of early management in optimizing outcomes for this devastating complication.

Keyword(s) : hepatocellular carcinoma, spinal metastasis, vertebral canal stenosis, myelopathy, hemilaminectomy

1763 - Cirrhosis in Transition Rapid Metabolic and Alcoholic Rise with Declining Viral Burden in an Indian Tertiary Center

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Background / Hypothesis:

The Indian landscape for cirrhosis has been historically dominated by viral hepatitis and alcoholic liver disease. With the national rollout of affordable antivirals, this study assessed the changing aetiology, clinical presentation, and complication profile of cirrhosis at a tertiary- care centre in Mumbai, India (2024–2025).

Materials and Methods:

A cross-sectional analysis of 140 consecutive cirrhosis patients admitted between April 2024 and April 2025. Aetiology, clinical features, and complications were documented and compared with historic Indian data. Statistical analysis used chi-square and ANOVA tests, with $p < 0.05$ considered significant.

Results:

Mean age 54.7 ± 13.3 years; 77.9 % male.

Alcoholic liver disease (ALD) predominated (74.3 %, 95 % CI 66.6–81.0), followed by metabolic-associated steatohepatitis (MASH, 14.3 %), hepatitis B (6.4 %), and hepatitis C (2.9 %).

Viral causes showed a marked decline versus past Indian cohorts (> 20 %).

Common complications included ascites (65.7 %), encephalopathy (26.4 %), varices (24.3 %), spontaneous bacterial peritonitis (6.4 %), and hepatorenal syndrome (2.9 %).

Alcohol-related cases exhibited higher encephalopathy and infection rates ($p < 0.05$).

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Conclusions:

Indian cirrhosis aetiology has pivoted decisively toward metabolic and alcohol-related disease, mirroring Western trends. Declining viral causes underscore the success of antiviral programs, while the rapid rise of MASH highlights the need for early metabolic screening and tighter public-health interventions across urban India.

Keyword(s) : Cirrhosis, Etiological trends, MASH, Viral hepatitis decline, India

1764 - Multidisciplinary management of diffuse intraductal papillary neoplasm of the bile duct in an elderly patient with limited therapeutic options

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Background / Hypothesis:

Intraductal Papillary Neoplasm of the Bile Duct (IPNB) is a rare biliary precursor lesion with high malignant potential. Curative treatment requires complete surgical resection, but advanced age, frailty, and multilevel ductal involvement may limit the feasibility of definitive therapy. This case highlights the hepatobiliary, oncologic, and ethical considerations when both surgery and standard systemic regimens are contraindicated.

Materials and Methods:

A 77-year-old female presented with recurrent obstructive jaundice and cholangitis. Endoscopic Retrograde Cholangiopancreatography (ERCP) revealed diffuse intraductal lesions involving the common bile duct, common hepatic duct, and central intrahepatic ducts, necessitating repeated stent exchanges for biliary decompression. Histopathology confirmed high-grade IPNB. Due to frailty and severe three-vessel coronary artery disease, the patient was deemed unsuitable for surgical resection. Persistent cytopenias prompted bone marrow evaluation prior to systemic therapy consideration.

Results:

The patient's course was dominated by recurrent biliary

obstruction requiring sequential endoscopic palliation. Bone marrow evaluation confirmed a coexisting Myelodysplastic Neoplasm (MDS), explaining her persistent anemia and thrombocytopenia. Although systemic therapy is not routinely indicated for non-invasive IPNB, its diffuse involvement and high-grade dysplasia warranted consideration. However, the established gemcitabine–cisplatin regimen posed unacceptable myelosuppressive risk. Consequently, the multidisciplinary team redirected management toward safer, lower-intensity systemic options while continuing endoscopic drainage to prevent cholangitis.

Conclusions:

For elderly patients with diffuse high-grade IPNB and prohibitive comorbidities, both curative and standard palliative therapies may be untenable. This case demonstrates how structured multidisciplinary evaluation enables a realistic, patient-centered strategy focused on durable biliary drainage and safe therapeutic modification. It underscores the need for individualized hepatobiliary management when conventional treatment pathways are not feasible.

Keyword(s) : intraductal papillary mucinous neoplasm, bile duct, therapeutic limitations, endoscopic management, multidisciplinary care, biliary system disorders

1765 - Synergistic Hepatoprotection via SREBP 1c PPAR α FXR CYP7A1 and Insulin Signaling Modulation by Empagliflozin and Cholestyramine in STZ induced diabetic rats

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Background / Hypothesis:

Diabetes mellitus accelerates blood glucose levels and hepatic lipotoxicity through insulin resistance, increased de novo lipogenesis (DNL), mitochondrial dysfunction, and impaired bile-acid recirculation. Empagliflozin (SGLT2 inhibitor) ameliorates glucotoxicity and reduces hepatic fat via enhanced β -oxidation and improved adipose liver function. In contrast, Cholestyramine, a bile-acid sequestrant (BAS), interrupts enterohepatic circulation, activating hepatic cholesterol to bile acid conversion and reducing hepatic triglyceride stores through FXR, FGF15/19 modulation. The combined hepatic effects of these complementary pathways have not been well



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characterized.

Materials and Methods:

Diabetic STZ rats were treated with Empagliflozin (10mg/Kg), Cholestyramine [5% w/w], and their combination for 04 weeks, followed by assessment of hepatic enzymes, lipid profile, and oxidative markers. Liver histology was evaluated to determine hepatoprotective effects.

Results:

Empagliflozin lowered glucose and improved insulin sensitivity while reducing steatosis. Cholestyramine reduced hepatic triglycerides. Combined therapy showed maximal benefit, with approximately 60% TG reduction, improved oxidative stress (decreased MDA, increased SOD & CAT). Synergy reflected simultaneous correction of glucotoxicity and bile acid dysregulation, leading to suppressed DNL and enhanced hepatic metabolic balance.

Conclusions:

Empagliflozin and Cholestyramine synergistically improve glucose, lipid, and bile acid metabolism, providing strong hepatoprotection. Their combined therapy may prevent hepatic deterioration and support recovery in acute liver failure, offering a promising adjunctive metabolic liver intervention.

Keyword(s) : Diabetes; Liver Failure; Empagliflozin; Cholestyramine; STZ

1766 - Association Between Baseline Hepatic Nodules and Their 3 Month Dynamic Changes with Hepatocellular Carcinoma Risk in HBV Cirrhosis Patients on Antiviral Therapy

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Background / Hypothesis:

The impact of hepatic nodules detected by ultrasound on hepatocellular carcinoma (HCC) risk in patients with HBV-related cirrhosis receiving nucleoside analogues (NAs) remains unclear. We investigated the association between baseline hepatic nodule status, 3-month dynamic changes, and HCC risk.

Materials and Methods:

The real-world cohort included 1,209 treatment-naïve patients with HBV-related cirrhosis who received NAs antiviral therapy for more than 1 year. Patients were

stratified by baseline hepatic nodule presence. Nodule characteristics were categorized by size (≥ 10 mm vs. < 10 mm), echogenicity (hyperechoic vs. non-hyperechoic), shape (regular vs. irregular), and number (single vs. multiple). Patients were further classified based on 3-month nodule dynamics: no nodules, stable nodules, or progressive nodules. Cox proportional hazards models assessed the association between hepatic nodule status and HCC risk. Cumulative HCC incidence was analyzed using Kaplan-Meier curves and log-rank tests.

Results:

Among 1,209 patients, 806 had no baseline hepatic nodules and 403 had nodules. Of a subset of 354 patients (29.3%) completing 3-month ultrasound follow-up, 187 had no nodules, 97 had stable nodules, and 70 had progressive nodules. Baseline hepatic nodules were associated with increased HCC risk (aHR, 1.700; 95% CI, 1.363-2.119, $P < 0.001$). During a median follow-up time of 7.0 years, 323 patients (26.7%) developed HCC. 1-, 3-, and 5-year cumulative incidences of HCC were 8.3%, 17.1%, 21.5%, respectively. The Kaplan-Meier curves showed that patients with hepatic nodules had a significantly higher cumulative incidence of HCC than those without hepatic nodules (Log-rank $P < 0.001$). We further found Larger nodules (≥ 10 mm) had a higher cumulative incidence of HCC compared to smaller nodules (Log-rank test $P = 0.002$). No significant differences were observed by echogenicity, shape, or number. The Kaplan-Meier curves showed that the risk of developing HCC was significantly different between 3-month hepatic nodules growth, with the lowest cumulative incidence in patients without hepatic nodules and highest in those with progressive hepatic nodules (Log-rank test $P = 0.020$).

Conclusions:

Baseline hepatic nodules and their 3-month progression are significantly associated with HCC in patients with HBV-related cirrhosis patients receiving NAs therapy, highlighting personalized surveillance strategies.

Keyword(s) : Hepatocellular carcinoma, Hepatic nodules, HBV-related cirrhosis, Antiviral therapy, Ultrasound

1767 - Comparison of Treatment Efficacy and Safety Profile of Atezolizumab Plus Bevacizumab in BCLC B and C Patients with Hepatocellular Carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) imposes a considerable global burden, and rigorous evidence to guide therapeutic stratification across BCLC B and C remains limited. Atezolizumab plus bevacizumab (ATE/BEV) is established as first line therapy for advanced HCC, yet effectiveness and safety between BCLC B and C in practice are undefined. Real world BCLC B cohorts are heterogeneous and include patients who are TACE non eligible, such as those with TACE refractoriness, huge HCC, multinodular HCC, or hypovascular HCC. We conducted a multicenter real world analysis comparing ATE/BEV across BCLC B and C, assessing overall and progression free survival, objective response, disease control, and treatment related adverse events.

Aim :

Investigate the real-world tumor response and survival outcome between BCLC-B and BCLC-C after ATE/BEV treatment

Investigate clinical characteristics of BCLC-B and BCLC-C undergoing ATE/BEV treatment

Materials and Methods:

Multi-institutional retrospective cohort study (10 academic hospitals in Korea)

Inclusion criteria

≥ 18 years old

Patients diagnosed as unresectable HCC who underwent atezolizumab plus bevacizumab 2020.07. – 2024.11.

Exclusion criteria

Insufficient clinical records

Patients with duration of ATE/BEV treatment < 3 months

Results:

Baseline characteristics were largely balanced between BCLC B and BCLC C, including mean age (68.34 vs 66.07 years, $p=0.040$), gender distribution (85.7% vs

87.1%, $p=0.709$), and maximum tumor diameter (6.57 vs 6.87 cm, $p=0.604$). Notably, BCLC B had a higher mean tumor count (2.99 vs 2.18, $p<0.001$). Disease control rates were comparable (75.9% vs 68.3%, $p=0.090$), whereas the objective response rate was numerically higher in BCLC C but did not reach statistical significance (15.7% vs 25.6%, $p=0.056$). Kaplan–Meier estimates showed no significant difference in overall survival between groups (median 11.13 vs 10.82 months; $p=0.799$).

Conclusions:

ATE/BEV demonstrated comparable effectiveness in BCLC B and BCLC C in real world practice. BCLC B patients, particularly those unsuitable for TACE, achieved meaningful disease control and survival. Our findings support expanding the role of ATE/BEV as a viable first line option in intermediate stage HCC when TACE is limited or not appropriate. Further studies are needed to refine patient selection for ATE/BEV in BCLC B HCC.

Keyword(s) : Atezolizumab-Bevacizumab, Hepatoceular carcinoma, BCLC, Tumor response, Real-world data

1770 - Serum HBV Markers Reflect Intrahepatic cccDNA and Transcriptional Activity in a Phase Dependent Manner in Chronic HBV Infection

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Background / Hypothesis: cccDNA maintains HBV persistence through its transcriptional activity. We hypothesized that host immunity, reflected by the CHB natural phase, influences HBV transcription and serum markers, thereby altering their correlation with cccDNA activity. We aimed to identify serum markers that differentially reflect intrahepatic cccDNA and its activity.

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Materials and Methods: A total of 406 liver tissue samples from CHB patients were analyzed according to natural phase. Serum markers (HBsAg, HBcrAg, HBV RNA) and intrahepatic markers (iHBVDNA, cccDNA, pgRNA) were quantified using commercial assays and qPCR. Viral genome diversity was assessed by Shannon entropy and nucleotide diversity.

Results: Intrahepatic and serum HBV markers were significantly higher in HBeAg-positive than in HBeAg-negative patients. Most of all HBV markers showed a decreasing trend with increasing age. In HBeAg-positive phases, serum HBV RNA and HBcrAg correlated well with intrahepatic cccDNA, particularly in the immune-tolerant (IT) phase ($r=0.439$ and 0.503 , respectively) and immune-active (IA) phase ($r=0.500$ and 0.443), whereas their correlations were weak in HBeAg-negative hepatitis (ENH) and absent in inactive-carrier (IC). For intrahepatic pgRNA, strong correlations with serum HBV RNA and HBcrAg (all $r>0.400$) were observed in IT and IA, decreasing in ENH ($r=0.369$, $r=0.326$). The best serum markers for cccDNA transcriptional activity (pgRNA/cccDNA) were HBV RNA (IA: $r = 0.586$; ENH: $r=0.524$) and HBcrAg (IA: $r=0.576$; ENH: $r=0.399$). Notably, no serum markers reflected transcriptional activity in the IT and IC phase. Shannon entropy and nucleotide diversity were lowest in IT phase and highest in IC phase, indicating genetic homogeneity in IT and high viral heterogeneity in IC.

Conclusions: Serum HBcrAg and HBV RNA serve as indicators of intrahepatic cccDNA and its activity, though their performance varies by CHB phase. The limited reliability of these markers in IT and IC phases likely reflects a restricted dynamic range, due to saturation and upper-range clustering of cccDNA-derived markers in IT and viral heterogeneity in IC phase. These findings support their phase-specific utility for noninvasive monitoring, treatment decisions, and the design of future cure-focus.

Keyword(s) : Hepatitis B virus, cccDNA, pgRNA, HBV phase

1772 - HLA Variants and Oral Microbiome Signatures Associated with Antibody Mediated Rejection Post Living Donor Liver Transplantation

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Background / Hypothesis:

Antibody-mediated rejection (ABMR) remains a key complication after living donor liver transplantation and may be influenced by host immunogenetics like HLA. We hypothesized that specific HLA alleles may harbor specific oral microbiome profiles and that these combined factors may influence the risk of ABMR.

Materials and Methods:

In this prospective study, 180 donor–recipient pairs undergoing living donor liver transplantation were evaluated. Pre-transplant saliva and blood samples were subjected to 16S rRNA sequencing and high-resolution HLA typing using NGS platforms. Microbial diversity and taxonomic abundance were analyzed using DADA2, QIIME2, GLM-based association models, and LEfSe. HLA amino-acid mismatches were quantified with HLA-EMMA. Clinical follow-up extended to one year for ABMR diagnosis based on Banff 2016 criteria.

Results:

ABMR occurred in 15/180 recipients (8.3%). A significantly higher HLA Class II mismatch burden was observed in those who developed ABMR compared with those with stable graft function, particularly at DPB1, DQB1, and DR loci (median mismatch DPB1: 2 vs 0; DQB1: 2 vs 0; DR: 2 vs 1). At the phylum level, HLA-DRB1*08 correlated with Actinobacteriota, HLA-DPB1*575 with Campylobacterota, and HLA-C*05 with Fusobacteriota. LEfSe identified >4-LDA enrichment of Veillonella, Streptococcus, and Enterobacter in HLA-DQB1*06 carriers who developed ABMR, while Lautropia and Burkholderiaceae were enriched in HLA-DQB1*04 as shown in Figure 1. These taxa have established associations with mucosal inflammation and systemic immune activation. Alpha diversity did not differ by zygosity, suggesting microbial structure rather than richness differs with HLA background.

Conclusions:

HLA polymorphisms and class II mismatch burden influence oral microbial composition in liver transplant recipients. Combined host-genetic and microbiome profiles may help identify patients at increased risk for antibody-mediated rejection. As microbiota are modifiable, these findings support future exploration of oral-microbiome-based interventions to complement immunologic risk assessment and potentially reduce ABMR incidence.

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Keyword(s) : Antibody-mediated rejection, Living donor liver transplantation, HLA class II mismatch, 16S rRNA sequencing, Microbial signatures

1773 - Siwu Decoction Inhibits Hepatocellular Carcinoma via Tumor Necroptosis Induced Suppression of Myeloid Derived Suppressor Cells

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a leading cause of cancer-related mortality worldwide. While sorafenib, as a first-line systemic treatment for advanced HCC, provides survival benefits, its therapeutic efficacy is substantially limited by acquired resistance and the immunosuppressive tumor microenvironment (TME). Myeloid-derived suppressor cells (MDSCs) are major contributors to this immunosuppression, facilitating tumor progression and therapy failure. Siwu Decoction (SWD), a classic traditional Chinese medicine formula known for nourishing the liver and regulating blood circulation, has shown potential in cancer treatment. We hypothesized that SWD could suppress HCC by modulating the TME through induction of tumor necroptosis and subsequent inhibition of MDSCs, thereby offering a complementary approach to overcome the limitations of current therapies like sorafenib.

Materials and Methods:

SWD was prepared from four herbs and its chemical profile was characterized by HPLC and LC-MS/MS. Its anti-HCC effects were evaluated in vitro on human HCC cell lines and in an orthotopic HCC mouse model. Tumor growth and metastasis were monitored. The role of MDSCs was investigated using adoptive transfer experiments and flow cytometry. Necroptosis was inhibited using Necrostatin-1 (Nec-1). The combination effect of SWD with sorafenib and the activity of its component, Albiflorin, were also assessed.

Results:

SWD significantly inhibited HCC growth and lung metastasis in mice without toxicity. It induced necroptosis in HCC cells, evidenced by upregulation of necroptosis-related proteins. SWD reduced intratumoral MDSCs but did not affect circulating MDSCs or directly induce MDSC apoptosis. Instead, supernatant from SWD-treated HCC cells triggered

MDSC apoptosis. Adoptive transfer of MDSCs or Nec-1 treatment reversed SWD's anti-tumor effects.

Combining SWD with sorafenib enhanced efficacy without additional toxicity. Albiflorin, a key component of SWD, mirrored its effects by inducing necroptosis and suppressing MDSCs.

Conclusions:

SWD inhibits HCC by inducing tumor cell necroptosis, which indirectly leads to MDSC apoptosis and alleviates immunosuppression in the TME. Albiflorin is identified as a primary active compound responsible for these effects. These findings support SWD as a promising adjuvant therapy for HCC, particularly in combination with sorafenib, by targeting both tumor cells and the immunosuppressive microenvironment.

Keyword(s) : Myeloid-derived suppressor cells, Necroptosis, Hepatocellular carcinoma, Tumor microenvironment, Siwu decoction

1774 - An Innovative Integrated "Screen to Treat" Model with a Three Tier Network Significantly Enhances Viral Hepatitis Screening Efficiency and Linkage to Care in Zhuhai China A Comparative Study

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Background / Hypothesis:

China bears a disproportionate burden of global liver cancer, with 92% of cases attributable to chronic hepatitis B (HBV). To achieve the WHO's 2030 elimination goal, innovative and scalable screening strategies are essential for identifying asymptomatic adults who missed neonatal vaccination programs. This study aimed to develop and evaluate a novel, integrated model designed to improve screening efficiency, coverage, and linkage to care.

Materials and Methods:

We conducted a comparative study in Zhuhai among permanent residents aged 20-70. Phase one (Jan-July

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2025) utilized a conventional hospital-led screening model. Phase two (July-Nov 2025) implemented our innovative strategy, founded on a three-tier collaborative network integrating government for oversight, a tertiary hospital for technical support, and community health centers for frontline implementation. This structure enabled a comprehensive "screen-to-treat" pathway with diversified delivery through fixed community sites and mobile on-site services. The workflow incorporated digital tools (electronic consent, mobile app for results) and rapid 15-minute point-of-care testing. This system facilitated immediate linkage to care, including on-site health education, HBV vaccination for susceptible individuals, and a "green channel" referral with subsidized follow-up services for seropositive individuals, all managed by a precise follow-up system. A multi-faceted public health campaign amplified the initiative to enhance public awareness.

Results:

The innovative model demonstrated markedly superior efficiency, screening 40,026 individuals in four months compared to the traditional model's 5,086 in seven. In total, 45,112 individuals were screened (median age 37; 58.96% male). The overall prevalence of HBsAg was 5.28%, with a significantly higher rate in males than females (5.95% vs. 4.31%, $P < 0.001$), peaking at 6.73% in the 40-49 age group. The prevalence of anti-HCV was 0.299%, and seven cases of HBV-HCV co-infection were identified. Critically, the model successfully linked 2,512 seropositive individuals to care and facilitated HBV vaccination for 2,218 susceptible persons.

Conclusions: Our innovative screening model, which integrates a three-tier network, a full-chain service pathway, and public health outreach, is significantly more effective than traditional hospital-centric approaches in expanding screening coverage and ensuring linkage to care. This integrated system offers a robust, replicable, and scalable blueprint for accelerating progress towards viral hepatitis elimination. Its success in a region with a large mobile population makes it highly applicable to other urban centers across Asia facing similar public health challenges.

Keyword(s) :

1777 - Metabolic Dysfunction Particularly Diabetes Accelerates Liver Disease Progression in Chronic Hepatitis B

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Background / Hypothesis: The impact of metabolic dysfunction on adverse outcomes in chronic hepatitis B (CHB) patients remains controversial. Understanding the relationship between metabolic risk factors and hepatic outcomes is crucial for improving patient management.

Aims: To evaluate the impact of concomitant metabolic risk factors and fatty liver conditions on adverse hepatic outcomes in CHB patients.

Materials and Methods: We analyzed data from an outpatient CHB cohort (2009–2025), categorizing 7,817 patients into four groups: CHB alone ($n=3,804$), CHB with metabolic dysfunction-associated fatty liver disease (MAFLD, $n=1,154$), CHB with simple fatty liver ($n=600$), and CHB with metabolic risk factors only (MET, $n=2,259$). Multivariate Cox regression models were used to assess risks of hepatocellular carcinoma (HCC) and decompensated liver cirrhosis (DLC), adjusting for age, sex, antiviral treatment, and fibrosis stage. Patients with baseline decompensated cirrhosis were excluded.

Results: During a maximum of 16-year follow-up, only MET was independently associated with increased risk of DLC (adjusted HR [aHR] = 2.55; 95% CI: 1.77-3.68), but not HCC (adjusted HR [aHR] = 1.30; 95% CI: 0.94-1.80). Stratification revealed that diabetes mellitus (DM) conferred significantly higher risks of both HCC (aHR = 1.63; 95% CI: 1.13-2.34) and DLC (aHR = 2.65; 95% CI: 1.83-3.83) in the CHB+MET group. Hypertension (HP) was most strongly linked to DLC (aHR = 1.65; 95% CI: 1.13-2.41). In the CHB+MAFLD group, DM was significantly associated with HCC (aHR = 6.15; 95% CI: 1.74-21.77). No significant associations were observed for simple fatty liver.

Conclusions: Metabolic dysfunction, particularly diabetes mellitus, significantly increases the risk of adverse hepatic outcomes in CHB patients. DM is a key driver of both HCC and DLC, highlighting the importance of comprehensive metabolic assessment for risk stratification and management optimization in CHB.

Keyword(s) : Metabolic Dysfunction, Diabetes, Chronic Hepatitis B

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1780 - NHSL3 Knockout Enhances Sorafenib Sensitivity by Promoting G1 Phase Accumulation in Huh 7 Hepatocellular Carcinoma Cells

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Background / Hypothesis: Liver cancer is the sixth most commonly diagnosed cancer worldwide and the third leading cause of cancer-related mortality.

Hepatocellular carcinoma (HCC) accounts for nearly 85% of all liver cancers. One major reason for the poor survival of HCC patients is the high resistance of HCC cells to current chemotherapeutic agents. Sorafenib is the first and most widely used systemic therapy for advanced HCC. Therefore, identifying molecular regulators that influence the treatment response of HCC cells to sorafenib is essential. NHS Like 3 (NHSL3) is upregulated in HCC and correlates with poor clinical outcomes; however, its cellular functions remain unknown. This study aimed to elucidate the roles of NHSL3 in cell cycle regulation in sorafenib-treated and untreated Huh-7 cells.

Materials and Methods: NHSL3 knockout (KO) Huh-7 cells were generated using the CRISPR/Cas9n method. Cell viability was measured using the WST-1 assay, and half-maximal inhibitory concentration 50 (IC50) values were calculated. Cell cycle phase distribution was assessed via propidium iodide (PI) staining. Levels of key cell cycle regulatory proteins were analyzed by Western blotting.

Results: Following 24 hours of sorafenib treatment, IC50 values were 13.39 μ M for control Huh-7 cells and 5.81 μ M for NHSL3 KO cells, indicating a 2.3-fold increase in sorafenib sensitivity in NHSL3 KO cells. Treatment with 5 μ M sorafenib for 24 hours increased the G1 phase population from 55% to 74% in control cells, and from 64% to 84% in NHSL3 KO cells ($p < 0.0001$ for all). Sorafenib also reduced the S phase population from 38% to 18% in control cells and from 28% to 10% in NHSL3 KO cells ($p < 0.0001$ for all). Western blot analyses revealed that sorafenib-treated NHSL3 KO cells exhibited significant upregulation of phosphorylated (p)-CHK2 (Thr68) and p-WEE1 (Ser642) ($p < 0.0001$ for both). In contrast, levels of p-CDK1 (Tyr15) ($p = 0.02$), Cyclin E2 ($p = 0.0002$), Cyclin A2 ($p < 0.0001$), and Cyclin B1 ($p < 0.0001$) were markedly reduced in sorafenib-treated NHSL3 KO cells relative to control cells.

Conclusions: These findings suggest that NHSL3 functions as a potential oncogenic regulator in HCC by contributing to sorafenib resistance. Loss of NHSL3 enhances G1 phase accumulation through downregulation of Cyclin E2/A2/B1 and activation of the CHK2-WEE1-CDK1 regulatory axis, supporting the concept that targeting NHSL3-driven signaling may improve therapeutic sensitivity and provide a basis for developing new treatment strategies in HCC.

Keyword(s) : Cell cycle, CRISPR/Cas9, Hepatocellular carcinoma, NHSL3, Sorafenib

1784 - Whole Exome Sequencing Identifies PNPLA3 and Rare ABCC8 Variants as Genetic Risk Factors for Metabolic Dysfunction-Associated Steatotic Liver Disease in South Asians

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD), recently redefined as metabolic dysfunction-associated steatotic liver disease (MASLD), is reported to have a prevalence of 30.05%. It is a multifactorial disease where genetic predisposition plays a significant role in individual susceptibility. In this study, we aim to identify pathogenic variants contributing to MASLD in a Pakistani South Asian cohort, followed by their validation in an expanded dataset.

Materials and Methods:

A total of 200 individuals with NAFLD were meticulously selected. The study involved a comprehensive clinical evaluation, including detailed medical history, ultrasound examinations, laboratory investigations, and transient elastography. Furthermore, a case-control analysis was carried out, with six cases selected from the NAFLD cohort and six controls identified from the general population. Whole blood from all the participants ($n=12$) was collected for DNA extraction, and whole-exome sequencing (WES) was employed. Bioinformatics analysis involved alignment of sequences with the human reference genome GRCh38 (hg38) using the Burrow-Wheeler aligner software, Genome Analysis Toolkit, and the

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SnEff tool. Variant interpretation and classification adhered to the American College of Medical Genetics guidelines. Furthermore, various prediction tools were applied, and candidate variants were identified. This was followed by a validation phase where the identified variants were subsequently validated in 52 MASLD patients by Sanger sequencing and compared with 96 ethnically matched controls from the Punjabi Pakistani (PJL) cohort of the 1000 Genomes Project. Statistical analyses were conducted using SPSS version 28.

Results:

ClinVar analysis showed the following common variants: SUMO4 (rs237025), PRKCQ (rs2236379) & MBL2 (rs1800450). Further, two variants in the validation cohort showed a significant association with MASLD

PNPLA3 rs738409: The G allele had a minor allele frequency (MAF) of 0.47 in MASLD cases compared to 0.20 in the PJL cohort ($p=0.001$). The GG genotype was present in 34.6% of cases and 7.3% of the PJL cohort, showing a strong association with disease risk. GC heterozygotes were similarly distributed in both groups.

- ABCC8 rs146378237: The T allele had a MAF of 0.36 in MASLD cases but was absent in all PJL cohort (MAF=0). The TT genotype was seen in 19.2% of cases, and TC in 34.6%, indicating a potentially rare but high-penetrance association.

Conclusions: This study reaffirms the role of PNPLA3 and highlights a novel potential association with ABCC8 rs146378237 in MASLD. The marked enrichment of these variants in cases compared to reference controls underscores the importance of integrating common and rare variants in understanding MASLD susceptibility in Pakistani South

Keyword(s) : MASLD, PNPLA3, ABCC8, whole exome sequencing

1786 - Environmental Risk Factors and Comorbidity Profiles Associated With Autoimmune Hepatitis in Korea A Nationwide Case-Control Study

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Background/hypothesis: Autoimmune hepatitis (AIH) is a chronic immune-mediated liver disease with poorly understood environmental and clinical triggers. This

study aimed to evaluate infectious exposures, medication-related risks, and comorbidity patterns associated with AIH using a nationwide Korean database.

Materials and Methods: In this population-based case-control study, 10,081 patients with incident AIH were matched to 201,620 controls. We analyzed (1) infectious diseases within 1 year before AIH diagnosis, (2) medication exposures within 6 months, and (3) lifetime comorbidities. Differences were assessed using proportions, p-values, and Cohen's h effect sizes.

Results: AIH patients had significantly higher rates of recent infections, including acute hepatitis A (1.32% vs. 0.02%), hepatitis B (0.31% vs. 0.04%), and hepatitis C (0.30% vs. 0.01%). COVID-19 and influenza were also modestly more frequent. Toxic/drug-induced hepatitis (DILI) was markedly more common (16.97% vs. 0.31%; $h=0.74$). Several medications were more commonly used in AIH patients, including statins, amoxicillin-clavulanate, TNF- α inhibitors, interferons, fimasartan, and IV methylprednisolone. AIH cases exhibited a substantially higher burden of autoimmune comorbidities: hypothyroidism (25.95% vs. 15.42%), systemic lupus erythematosus (4.58% vs. 0.12%), sicca syndrome (8.32% vs. 1.23%), rheumatoid arthritis, systemic sclerosis, and inflammatory bowel disease. Strong associations were seen with primary biliary cholangitis (10.26% vs. 0.03%; $h=0.62$) and primary sclerosing cholangitis. Metabolic diseases such as diabetes (39.71% vs. 32.22%) were also more prevalent.

Conclusions: AIH in Korea is associated with increased rates of preceding infections, greater exposure to hepatotoxic medications, and a broad spectrum of autoimmune comorbidities. These findings provide insight into potential environmental and clinical factors related to AIH and may help refine future research on its pathogenesis.

Keyword(s) : autoimmune hepatitis, risk factors, comorbidity

1787 - Metagenomic Study on Adult Cases of Hepatitis of Unknown Etiology No Consistent Novel Pathogen Identified Amidst Highly Heterogeneous Viral Communities

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Background / Hypothesis: To conduct unbiased

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pathogen screening and microbial community characterization in adult patients with hepatitis of unknown etiology using metagenomic approaches, aiming to identify novel or underexplored pathogenic agents.

Materials and Methods: This study enrolled 49 patients with rigorously excluded known etiologies. Fifty-nine samples (44 plasma, 15 stool) were subjected to ultra-multiplex targeted RNA sequencing (covering 139 RNA viruses) and metagenomic sequencing. Pathogen profiles and viral community structures were systematically analyzed through a bioinformatics pipeline.

Results: Targeted sequencing detected hepatitis C virus in one sample, while none of the remaining samples revealed predefined RNA viruses. Metagenomic analysis identified no novel or rare pathogenic virus consistently enriched across patients. Goose circovirus and influenza A virus were sporadically detected, with the former showing high homology to avian-derived strains but no significant case enrichment, and the latter presenting only as a low-titer singleton. Both exhibited ambiguous causal relationships with hepatitis. Viral community profiling demonstrated significant divergence between plasma and stool samples, revealing niche-specific patterns.

Conclusions: Our study failed to identify a unifying pathogenic agent in this adult hepatitis cohort, suggesting a heterogeneous etiological landscape potentially involving unknown pathogens, host immune responses, and environmental interactions.

Keyword(s) : Hepatitis of unknown etiology, Metagenomics, Viruses, Goose circovirus, Influenza A virus

1788 - Yinchenhao Decoction Suppresses Metabolic Associated Fatty Liver Disease Inflammatory Injury by Regulating Monocyte Superoxide Metabolism

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Background / Hypothesis: Yinchenhao Decoction (YCHT), a classic traditional Chinese medicine formula for damp-heat (DH) syndrome, was investigated for its regulatory effects on peripheral monocytes in metabolic-associated fatty liver disease (MAFLD) patients with DH syndrome.

Materials and Methods: We retrospectively analyzed clinical data from 367 MAFLD patients. Single-cell RNA sequencing of peripheral blood mononuclear cells (PBMCs) from DH syndrome patients revealed distinct monocyte metabolic patterns. In vitro, THP-1 cells stimulated with LPS and IFN- γ were treated with YCHT to assess its effects on inflammation and mitochondrial oxidative stress. A high-fat high-sugar (HFHS) diet-induced MASH mouse model was used to evaluate YCHT's therapeutic effects, with monocyte-derived macrophages (MoMFs) detected by flow cytometry.

Results: MAFLD patients with DH syndrome showed significantly higher serum ALT levels. Their monocytes were enriched in inflammatory response pathways and exhibited active ROS production. In vitro, YCHT dose-dependently suppressed the elevation of IL-1 β , CCL2, IL-6, and TNF- α in THP-1 cells, corrected TLRs-MYD88 pathway activation, reduced ATP and ROS levels, and increased SOD activity. In HFHS-fed mice, YCHT treatment reduced body weight, liver NAS scores, hepatic TG content, and serum ALT levels. It also decreased hepatic expression of IL-1 β , IL-6, IFN- γ , and Ccl-2, accompanied by reduced recruitment of F4/80+ macrophages and MoMFs.

Conclusions: YCHT ameliorates MAFLD inflammatory injury in DH syndrome by correcting peripheral monocyte hyper-oxidative metabolism, inhibiting monocyte migration to the liver, and reducing subsequent liver damage.

Keyword(s) :

1789 - Correlation Between Liver Stiffness and Cognition in MASLD Patients Based on MRE and Neurovascular Coupling Assessment

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver

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disease (MASLD) has emerged as the leading cause of chronic liver disease in younger adults. Alarming, MASLD may disrupt brain function even without cirrhosis. Magnetic resonance elastography (MRE), a non-invasive imaging technique that quantifies liver stiffness with high precision, has revolutionized hepatic fibrosis assessment. However, the neurological implications of liver stiffness measured by MRE in non-cirrhotic young MASLD patients remain unclear. This study aimed to characterize the impact of non-cirrhotic MASLD with elevated liver stiffness on brain health using multimodal neuroimaging techniques.

Materials and Methods:

This was a prospective cohort involving 78 MASLD outpatients without cirrhosis [median age 26 years, (18-33)]. Using the Mayo criteria defining 2.5 kPa as the threshold for normal liver or inflammation, patients were stratified by MRE into low liver stiffness (LLS, $MRE < 2.5$ kPa, $n=38$) and high liver stiffness (HLS, $MRE \geq 2.5$ kPa, $n=40$) groups using a cutoff of 2.5 kPa in MRE. Participants underwent neuropsychological assessments, hepatic MRE, and multimodal brain magnetic resonance imaging (MRI), including resting-state fMRI and arterial spin labeling (ASL) for cerebral blood flow (CBF) and regional homogeneity (ReHo) analyses. Neuroimaging data were processed using SPM12, with voxel-wise comparisons and global CBF-ReHo coupling analyses. Statistical analyses included t-tests and nonparametric methods, adjusted for age and education.

Results:

Forty MASLD patients with high liver stiffness (HLS; $MRE \geq 2.5$ kPa) and 38 age-, gender-, and education-matched patients with low liver stiffness (LLS; $MRE < 2.5$ kPa) underwent resting-state fMRI. Global neurovascular coupling was evaluated by examining across-voxel correlations between CBF and ReHo, whereas regional coupling was assessed using the CBF/ReHo ratio. Compared to LLS, HLS patients exhibited significantly reduced global CBF-ReHo coupling ($p=0.044$) and regionally elevated CBF/ReHo ratios in the right temporal pole: superior temporal gyrus (TPOsup). Crucially, these abnormalities were undetectable by unimodal CBF or ReHo analyses. In HLS patients, higher CBF/ReHo ratios were significantly associated with more pronounced clinical deficits and accelerated cognitive decline. Furthermore, HLS

patients showed decreased functional connectivity between the left superior temporal gyrus and right TPOsup.

Conclusions:

These findings reveal novel neurovascular coupling disruptions in non-cirrhotic MASLD patients with elevated liver stiffness (≥ 2.5 kPa), providing insights into associated neuropathological mechanisms.

Keyword(s) : Metabolic dysfunction-associated steatotic liver disease, resting-state functional magnetic resonance imaging, neurovascular coupling, magnetic resonance elastography

1790 - Prognostic utility of the Ammonia to ULN ratio (AMM ULN) for predicting 28 day mortality and liver related complications in hospitalized patients with decompensated cirrhosis.

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Background / Hypothesis: Short-term risk stratification in hospitalized patients with decompensated cirrhosis remains difficult, and routinely available prognostic scores do not fully capture metabolic contributors such as hyperammonemia. Although ammonia plays a key role in the pathophysiology of hepatic decompensation, its clinical prognostic use is not standardized. This study was designed to evaluate the predictive performance of the ammonia-to-upper-limit-of-normal ratio (AMM-ULN) for 28-day mortality and major liver-related complications, and compared it with MELD-Na and Child-Turcotte-Pugh (CTP).

Materials and Methods: A prospective observational study was conducted on 96 adults hospitalized with decompensated cirrhosis. Baseline venous ammonia was standardized using the institutional ULN (118 $\mu\text{mol/L}$) to derive the AMM-ULN ratio. Outcomes assessed over 28 days included mortality and key complications—hepatic encephalopathy (HE), variceal bleeding (VB), ascites, coagulopathy, and spontaneous bacterial peritonitis (SBP). Optimal cut-off for mortality prediction was identified using the ROC-Youden index. Associations were evaluated using Mann-Whitney U tests and logistic regression (univariable and adjusted for age, MELD-Na, and CTP). Discriminative performance was assessed using ROC-AUC.

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Results: Among 96 included patients, the 28-day mortality rate was 16.7%. Median AMM-ULN was 1.47 (IQR 1.06–2.07). Non-survivors had significantly higher AMM-ULN values compared with survivors (1.64 vs. 1.39). ROC analysis identified an optimal AMM-ULN cut-off of 1.48 ($\approx 175 \mu\text{mol/L}$), yielding 75% sensitivity and 56% specificity. AMM-ULN demonstrated an AUC of 0.72, outperforming CTP (0.63) and showing slightly better discrimination than MELD-Na (0.69). Each 0.5-unit rise in AMM-ULN increased mortality odds (OR 2.46; 95% CI 1.38–4.38; $p=0.002$), remaining independently predictive after adjustment (adjusted OR 2.12; 95% CI 1.07–4.19; $p=0.032$). Higher AMM-ULN values were significantly associated with HE ($p<0.01$), VB, ascites, coagulopathy (all $p<0.05$), and SBP on univariable analysis, with independent associations confirmed where complete-case analysis permitted.

Conclusions: AMM-ULN is a simple, inexpensive biomarker that independently predicts short-term mortality and major complications in decompensated cirrhosis. A cut-off of 1.48 effectively identifies high-risk patients. Its prognostic performance is comparable to MELD-Na and superior to CTP, supporting its integration into early inpatient triage and risk-stratification pathways. Overall, AMM-ULN consistently predicted mortality and major complications, highlighting its value as a simple and effective biomarker.

Keyword(s) : cirrhosis, mortality, Ammonia-to-ULN ratio, liver-related complications

1791 - Formulation of the second viral hepatitis action plan in Hong Kong carrying forward the achievements for final push towards elimination

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Background / Hypothesis:

In Hong Kong, about 5.6% of the population, amounting to 410000 people, have hepatitis B, which is associated with about 80% of the local liver cancer cases. Among people with hepatitis B, 40% remained undiagnosed and 70% did not have any medical follow-up. Since the launch of the inaugural Hong Kong Viral Hepatitis Action Plan 2020-2024, a comprehensive set of initiatives has been implemented to strengthen the prevention and control of viral hepatitis and lay a solid foundation towards

eliminating its public health threat. To carry forward this momentum, the second action plan will be promulgated in 2025, with a view to further lowering viral hepatitis-related morbidity and mortality in Hong Kong, especially liver cancer.

Materials and Methods:

Since the establishment of the Steering Committee on Prevention and Control of Viral Hepatitis (SCVH) in 2018, regular meetings have been held to advise the Government on the overall policy, targeted strategies and effective resource allocation related to the prevention and control of viral hepatitis. Membership comprised representatives of academia, experts in different medical specialties, public healthcare providers and the Government. With valuable advice from the SCVH, the second Hong Kong Viral Hepatitis Action Plan 2025-2030 is formulated.

Results:

In the Action Plan 2025-2030, priority actions have been developed along four strategic axes, namely heightening awareness, tracking health sector response, preventing new infections and expanding access to screening, care and treatment. The most prominent initiative would be the roll-out of a new programme to subsidise population-based hepatitis B screening for groups with higher risk of infection, coupled with an enhancement facilitating management of adult patients with chronic hepatitis B (CHB) in primary care. Public awareness campaigns and professional training for healthcare workers would be launched to support the screening and management of hepatitis B in primary care level. For prevention, sustaining and maintaining high coverage of preventive measures against mother-to-child transmission of HBV would be the key to realise a hepatitis B-free generation.

Conclusions:

In view of the substantial size of population affected by hepatitis B in Hong Kong, empowering the community and primary care for screening and management of stable CHB patients would be critical to address the capacity constraints of specialist care and avoid system overload. The new programme would enable early identification of people with hepatitis B in the community and early treatment for CHB to reduce the risk of complications, thereby progressing towards the global targets of diagnosis and treatment coverage.

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Keyword(s) : viral hepatitis, action plan, elimination

1792 - Undiagnosed Metastatic Hepatocellular Carcinoma presenting as Breast Mass in an Adult Male A Case Report

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Background: Breast cancer among males is rare and accounts for only ~1% of all cases. Metastasis to the breast is rarer even in both sexes especially those arising from extramammary tissues. This case presents a first-ever report of hepatocellular carcinoma (HCC) metastatic to the breast in a male patient in the Philippines.

Case Presentation: A 68-year-old hypertensive male, heavy smoker and chronic alcohol drinker, consulted for a two-month history of an enlarging left breast mass. Physical examination showed a non-movable, hard left breast mass (~5x4 cm), icterisia, abdominal distention with positive fluid wave test, and jaundice. Breast ultrasound revealed an irregular, non-movable mass with macrocalcifications. A core-needle biopsy and histopathology confirmed results consistent with malignancy. Immunohistochemical staining was positive for Glypican-3 and HepPar1, congruent with metastatic carcinoma of hepatic origin. Further work-up disclosed elevated AFP levels, negative viral hepatitis serology, and ascitic fluid analysis compatible with portal hypertension from liver cirrhosis. Palliative treatment was initiated, however, worsening infection led to the patient's deterioration and subsequent demise.

Discussion: Hepatocellular carcinoma, often linked to chronic liver disease and cirrhosis, commonly metastasizes to the lungs, lymph nodes, and bones. Metastatic breast cancer from HCC is extraordinarily unusual with less than ten reported cases worldwide in both sexes, two (2) of which are male. Management of breast metastasis from HCC is often confounded by undiagnosed chronic liver disease. Thorough history and physical examination, balanced with appropriate interpretation of laboratory tests are important resources. Treatment options are limited which include systemic chemotherapy, radiotherapy, supportive and symptomatic treatment. Prognosis is poor with majority of patients dying within a year of diagnosis.

Conclusion: This case emphasizes the critical

importance of vigilance for rare malignancies in atypical patient populations. It highlights the need for prompt and comprehensive diagnostic evaluations for early interventions to improve patient outcomes in metastatic HCC.

Keyword(s) : Hepatocellular carcinoma (HCC), breast metastasis, breast mass

1793 - Effectiveness and Safety of 96 Week Tenofovir Amibufenamide in Patients with Hepatitis B Virus Related Hepatocellular Carcinoma A Retrospective Real World Study

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Background / Hypothesis:

Currently, there is a lack of data on the effectiveness and safety of tenofovir amibufenamide (TMF) for patients with hepatitis B virus (HBV)-related hepatocellular carcinoma (HCC) in real-world settings. The aim of this study was to evaluate the 96-week real-life effectiveness and safety profiles of TMF in patients with HBV-related HCC.

Materials and Methods:

This retrospective study enrolled HBV-related HCC patients who underwent antiviral treatment with TMF (25 mg/day) or entecavir (ETV) (0.5 mg/day) following HCC surgery at four hospitals in Henan Province, China, from August 2018 to November 2025. The primary endpoint was HBV DNA undetectable (less than 10 IU/mL) rate at week 96. The secondary endpoints included the rate of alanine aminotransferase (ALT) normalization, changes in lipid profile dynamics at 96 weeks, and short-term survival rate, with comparisons between the two groups.

Results:

A total of 80 patients were enrolled, with 40 patients in the TMF group and 40 in the ETV group. Baseline characteristics were as follows: the mean age was 56.8 ± 12.6 years in the TMF group and 54.6 ± 8.7 years in the ETV group ($P = 0.357$); baseline HBV DNA levels were $1.65 \pm 1.66 \log_{10}$ IU/mL vs. $0.08 \pm 0.35 \log_{10}$ IU/mL ($P = 0.164$); and ALT levels were 32.60 (21.40, 44.25) U/L vs. 29.50 (18.50, 43.75) U/L ($P = 0.613$). At 96 weeks of treatment, the HBV DNA undetectable rates were 85.0% (95% confidence interval [CI], 74.0% to 96.1%) in the TMF group versus 95.0% (95% CI, 88.25% to 100%) in the ETV group ($P = 0.136$), the ALT normalization rate was significantly higher in the TMF group than that in the ETV group (92.5% [95% CI,

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84.3% to 100%] vs. 90.0% [95% CI, 80.7% to 99.29%], $P = 0.692$). In terms of lipid profile, The intra-group comparison showed that the high-density lipoproteins level in the ETV group decreased compared to baseline (-6.14 [-6.14, 0.23], $P = 0.07$), with no statistically significant difference from baseline in the TMF group. Additionally, during the 96-week follow-up of ETV or TMF treatment, there were 2 deaths in the TMF group and 5 deaths in the ETV group ($P = 0.235$).

Conclusions:

This study demonstrate that TMF exhibits favorable effectiveness and safety in the treatment of HBV-related HCC patients; notably, TMF showed a better lipid profile compared to ETV. Further large-scale studies are needed to confirm these findings.

Keyword(s) : Antiviral treatment; Effectiveness; Entecavir; Hepatitis B virus; Hepatocellular carcinoma; Safety; Tenofovir amibufenamide

1794 - Ferulic acid loaded liposomes mitigate Hepatic Ischemia Reperfusion Injury in a rat model associated with Liver Transplantation via altering apoptosis mitochondrial function and TLR 4 NF B NLRP3 inflammatory pathway

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Background / Hypothesis: Hepatic ischemia-reperfusion (I/R) injury is a significant challenge in liver transplantation, leading to severe oxidative stress, inflammation, and hepatocellular damage. Effective therapeutic interventions are necessary to improve transplant success and reduce post-transplant complications. In this study, we investigated the protective effects of ferulic acid-loaded liposomes (FA-LPs) against hepatic I/R injury in a rat model that is associated with liver transplantation. FA, a natural phenolic compound with potent antioxidant and anti-inflammatory properties, was encapsulated in liposomes to enhance its bioavailability and targeted delivery to liver tissues.

Materials and Methods:

Ferulic acid-loaded liposomes were fabricated and characterized using sophisticated instrument. RAW 264.7 cells under the hypoxia/reoxygenation model were used and treated with FA-LPs at 1, 10, and 20 M. All the rats were split into five groups and given FA-LPs (3, 6, 9 mg/kg b.w.) for 14 days and then underwent a liver transplant. Using liver enzymes, cytokine status, hepatocyte death level, TUNEL, neutrophil and pro-

inflammatory cytokines protein, and mRNA expression, the potential of FA-LPs against hepatic ischemia-reperfusion injury were estimated.

Results:

Our findings demonstrated that FA-LPs effectively attenuated liver injury by modulating inflammatory pathways and reducing oxidative damage. Histopathological analysis revealed significant reductions in hepatocellular necrosis, inflammatory cell infiltration, and tissue damage in FA-LP-treated rats compared to untreated controls. Biochemical assessments showed that FA-LPs significantly lowered serum levels of liver injury markers, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST) while enhancing antioxidant enzyme activity. Additionally, FA-LPs suppressed the expression of pro-inflammatory cytokines such as TNF- α , IL-6, and IL-1 β , while upregulating anti-inflammatory mediators. Mechanistically, FA-LPs exerted their protective effects by inhibiting NF- κ B signaling and reducing oxidative stress through the activation of the Nrf2/HO-1 pathway. These molecular interactions contributed to reduced hepatocyte apoptosis and improved mitochondrial function, thereby preserving liver architecture and function post-transplantation. It also reduced TLR-4, p-IB, p-IKK, p-IKK, NLRP3, p-P65MyD88, TNF- α , cleaved caspase-1, IL-1, IL-6 and IL-18.

Conclusions: Overall, our study highlights FA-LPs as a promising nanotherapeutic strategy for mitigating hepatic I/R injury in liver transplantation. By leveraging their anti-inflammatory and antioxidant properties, FA-LPs offer a potential approach to improve liver graft survival and patient outcomes. Further studies and clinical evaluations are warranted to explore their translational potential in liver transplant medicine.

Keyword(s) : Ferulic acid-loaded liposomes, Hepatic Ischemia-Reperfusion Injury, Liver Transplantation, apoptosis, mitochondrial function, TLR-4/NF-B/NLRP3 inflammatory pathway

1795 - Turning the Tide Successful Management of a Complicated Case of Acute on Chronic Liver Failure with Double Plasma Molecular Adsorption System

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Background: Acute-on-chronic liver failure (ACLF) is a distinct syndrome among patients with chronic liver disease (CLD) who had a sudden hepatic decompensation involving one or more organs. Liver support systems such as the double plasma molecular adsorption system (DPMAS) are emerging treatment modalities for patients with ACLF while awaiting liver transplantation. We present a case of alcoholic hepatitis that triggered ACLF complicated by infections and variceal bleeding in a young adult patient.

Case Presentation: A 39-year-old Canadian male, heavy alcohol drinker with a three-year history of cirrhosis, presented with progressive jaundice. Physical examination revealed ictericia, ascites, and jaundice. Laboratory work-up showed hyperbilirubinemia, coagulopathy, an AST:ALT ratio of 4, and a MELD-Na score of 33—findings consistent with severe alcoholic hepatitis. Steroid therapy was initiated. Further evaluation revealed spontaneous bacterial peritonitis (SBP) on ascitic fluid analysis and amoebiasis on fecalysis; appropriate antibiotics were started. The patient subsequently developed variceal bleeding, for which endoscopic variceal ligation was performed. Despite these interventions, his clinical condition deteriorated with development of encephalopathy West-Haven Grade 2-3, acute kidney injury, and further increase in INR and bilirubin trends. Hence, the diagnosis of ACLF was entertained. He underwent two sessions of DPMAS, resulting in significant clinical and biochemical improvement and eventual discharge.

Conclusions: This case underscores the critical significance of a multidisciplinary approach in the diagnosis and management of ACLF and highlights the potential role of DPMAS. It emphasizes the need for prompt and comprehensive diagnostic evaluation and timely interventions to improve outcomes in patients with ACLF.

Keyword(s) : case report, Acute-on-chronic liver failure, DPMAS, hepatic failure

1796 - A novel flavonoid Galangin modulates hepatic ischemia–reperfusion injury and regulates TLR 4 NF κ B NLRP3 inflammatory pathway following orthotopic liver transplants in rat models.

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Background / Hypothesis: Hepatic ischemia can result in

metabolic issues and an inflammatory milieu inside the liver, which may produce early graft malfunction, hepatic pathological damage, apoptosis, or rejection of the graft. In this study, we used orthotopic liver transplant rat models to examine the possibility of flavonoid Galangin in alleviating hepatic IRI and its underlying causes.

Materials and Methods:

We performed orthotopic liver transplantation using a novel magnetic anastomosis approach. Following liver transplantation, hepatic IRI rat models were treated with intraperitoneally 10–20 and 30 mg b.w. of Galangin. Western blotting, qRT-PCR, terminal deoxynucleotidyl transferase dUTP nick-end labeling staining, hematoxylin–eosin staining, immunosorbent assay, qRT-PCR, and hepatic and non-hepatic parameters were used to analyze the impact of Galangin on hepatic IRI. Oxidative stress profile and proinflammatory cytokines were also evaluated. **Results:** Galangin significantly reduces liver function levels (AST, ALP, ALT, and AFP) and serum levels of proinflammatory cytokines (IL-1, IL-18, and TNF- α) in rats following liver transplantation. It also attenuates hepatocyte apoptosis and reduces histopathological changes (degree of coagulation necrosis, architectural anomalies, and hepatocyte vacuolization), according to our in vivo assays. Furthermore, Galangin attenuates the inflammatory response and neutrophil infiltration (IL-1 β , IL-18, IL-6, TNF- α , Ly6G and CD11b, and macrophage M2 polarization) caused by hepatic IRI and decreased the expression of TLR-4/NF- κ B/NLRP3 inflammatory pathway related proteins in rats following liver transplantation (TLR-4, MyD88, p-IKK α , p-IKK β , p-IKK, p-IkB α , p-P65, NLRP3, ASC, Cleaved caspase-1, IL-1 β , IL-18, TNF- α and IL-6). **Conclusions:** Our research demonstrated that Galangin suppresses hepatic inflammatory response after liver transplantation via modifying the TLR-4/NF- κ B/NLRP3 inflammatory pathway.

Keyword(s) : Galangin, hepatic ischemia–reperfusion injury, TLR-4/NF- κ B/NLRP3 inflammatory pathway, orthotopic liver transplants

1797 - Stellate Cell-Derived Complement Component 7 as a Determinant of Fibrosis Progression and Liver Related Events in MASLD An Integrated Multi Omics Study

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Background / Hypothesis:

Risk assessment in metabolic dysfunction-associated steatotic liver disease (MASLD) still relies on surrogate markers that do not adequately predict clinically meaningful outcomes, particularly liver-related events (LREs). We aimed to discover a mechanistically relevant biomarker that reflects both fibrogenic activity and future clinical risk through an integrative multi-omics approach.

Materials and Methods:

Transcriptomic and plasma proteomic screens were jointly analyzed in biopsy-proven MASLD to identify fibrosis-associated candidate markers. Findings were examined in an independent vibration-controlled transient elastography (VCTE) cohort. Spatial transcriptomics, single-nucleus RNA sequencing, and RNAscope in situ hybridization were employed to determine hepatic cellular origins. Functional significance was evaluated through siRNA-mediated knockdown in LX-2 hepatic stellate cells (HSCs). LRE risk was assessed using Cox regression and Kaplan-Meier analysis.

Results:

Complement component 7 (C7) emerged as one of the most strongly upregulated molecules across omics layers. Spatial mapping and snRNA-seq localized C7 expression predominantly to activated HSCs within fibrotic niches, whereas endothelial-cell expression was reduced in MASLD. Serum C7 levels rose proportionally with fibrosis stage and liver stiffness across cohorts. A serum threshold of ≥ 140 $\mu\text{g/mL}$ identified individuals at markedly higher risk for LREs. This threshold remained independently predictive after adjustment (HR 4.5). C7 demonstrated superior diagnostic ability for LREs (AUROC 0.90), outperforming FIB-4, autotaxin, APRI, and AAR. In vitro C7 silencing attenuated expression of key extracellular matrix genes (LAMA2, LAMB1, COL6A3), supporting its direct contribution to fibrogenic remodeling.

Conclusions:

C7 represents a mechanistically validated biomarker that captures both fibrotic burden and LRE risk in MASLD. Its stellate cell-specific induction and functional involvement in extracellular matrix regulation suggest utility for clinical stratification and potential therapeutic targeting.

Keyword(s) :

1801 - Fibrotic Burden in Metabolic Dysfunction associated Steatotic Liver Disease Identifies Cardiovascular and Mortality Risk in Type 2 Diabetes

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) frequently coexists with type 2 diabetes, but its added impact on cardiovascular disease (CVD) and mortality, and whether fibrosis assessed by the fibrosis-4 (FIB-4) index improves risk prediction, remain unclear. We evaluated the associations of MASLD and fibrosis severity with incident CVD and all-cause mortality in adults with type 2 diabetes.

Materials and Methods: We analyzed 30,330 UK Biobank participants with type 2 diabetes without prevalent CVD. MASLD was defined by fatty liver index ≥ 60 . The FIB-4 index was categorized as low (<1.3), indeterminate ($1.3-2.67$), or high (>2.67).

Results: Over a median follow-up of 13.6 years, 6,402 incident CVD events and 4,369 deaths occurred. MASLD demonstrated higher crude rates of both CVD and mortality than non-MASLD. After comprehensive adjustment for demographic and metabolic factors, MASLD remained independently associated with all-cause mortality (hazard ratio [HR] 1.16; 95% CI, 1.07–1.26) but not with incident CVD (HR 1.04; 95% CI, 0.98–1.12). Among participants with MASLD, only the high FIB-4 group showed significantly increased CVD risk compared with non-MASLD participants after multivariable adjustment (HR 1.24; 95% CI, 1.04–1.48), while elevated mortality risk persisted across all FIB-4 strata relative to non-MASLD.

Conclusions: Among individuals with type 2 diabetes, MASLD was associated with higher cardiovascular and mortality risk, and fibrosis severity further refined this risk. Incorporating simple fibrosis assessment, such as the FIB-4 index, into diabetes care may enhance clinical risk stratification.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease, liver fibrosis, cardiovascular disease, mortality, FIB-4

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1807 - Increased risk of decompensation in patients with clinically significant portal hypertension awaiting endoscopic variceal screening a metropolitan Australian study

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Background / Hypothesis: The Baveno VII guidelines recommend non-selective β -blockers (NSBBs) for those with chronic liver disease (CLD) and clinically significant portal hypertension (CSPH). In Australia, many centres still perform endoscopy in all patients with cirrhosis. Little data exist on the potential negative impact of time on the waitlist. We investigated the clinical outcomes of patients with cirrhosis awaiting endoscopic variceal surveillance, between those with and without CSPH.

Materials and Methods: Retrospective cohort study of patients with CLD awaiting endoscopic variceal screening at a large tertiary Australian centre, between July 2020 and March 2024. Primary outcomes were hepatic decompensation, liver-related hospitalisation, and liver-related mortality while on the endoscopy waitlist. CSPH was determined noninvasively by ultrasound or elastography prior to waitlisting. Kaplan-Meier curves were constructed, using the log-rank test for significance with two-tailed P values < 0.05. Hazard ratios (HR) with 95% confidence intervals (CI) were also reported using the Mantel-Haenszel method.

Results: Two hundred and thirty-eight patients (170 (71.4%) with CSPH; 68 (28.6%) without CSPH) were included. Median wait time for endoscopy was 18 (IQR 7–44) weeks. CSPH was associated with a higher rate of hepatic decompensation (42.4% versus 8.8%, $P < 0.001$ - primarily due to ascites (HR 3.03, 95% CI 1.73–5.33, $P < 0.001$), hepatic encephalopathy (HR 2.55, 95% CI 1.13–5.76, $P = 0.025$), and variceal bleeding (HR 3.21, 95% CI 1.01–10.21, $P = 0.048$)), liver-related hospitalisation (HR 2.68, 95% CI 1.46–4.91, $P = 0.001$) and liver-related mortality (HR 3.23, 95% CI 1.12–9.31, $P = 0.03$). Of those with CSPH, 33.8% (23 patients) maintained NSBB use during follow-up, with a 63% reduction in liver-related admissions compared to 133 who did not receive NSBBs (HR 0.37, 95% CI 0.015–

0.88, $P = 0.025$). The remaining 14 received NSBB for <30 days. Overall, 167 (70.2%) underwent endoscopy and 46% had oesophageal varices.

Conclusions: Patients with CLD and CSPH had worse liver-related outcomes while awaiting endoscopy compared to those without CSPH. These data support early identification of CSPH and management guided by Baveno VII guidelines, rather than endoscopy for all, with scope for quality improvement projects of the use of NSBB to improve liver outcomes.

Keyword(s) : Varices, Clinically Significant Portal Hypertension, Baveno VII, Decompensation, Endoscopy

1808 - Overshadowed by Immunotherapy Unmasking Chemotherapy Related Hepatic Vascular Injury (SOS Pattern) and Managing Catastrophic Hemorrhage

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Background:

The integration of immune checkpoint inhibitors (ICIs) with conventional chemotherapy and novel targeted agents treats advanced malignancies, but complicates the diagnosis of drug-induced liver injury (DILI). The primary clinical dilemma involves distinguishing immune-related adverse events (irAEs) from non-immune-mediated hepatotoxicity, specifically vascular pathologies like Sinusoidal Obstruction Syndrome (SOS). In the context of multi-agent regimens, cumulative drug toxicity often results in atypical clinical and radiological phenotypes, complicating diagnosis and management. Furthermore, this multifactorial injury induces severe hepatic parenchymal fragility, which significantly increases the risk of life-threatening hemorrhage during invasive diagnostic procedures. This report highlights the diagnostic conundrum of discordant pathological and radiological findings and underscores the critical role of Transarterial Embolization (TAE) in managing catastrophic hemorrhagic complications.

Case Description:

A 65-year-old male with refractory metastatic squamous cell carcinoma of the head and neck underwent extensive treatment with complex regimens, including Albumin-bound Paclitaxel, Carboplatin, and Pembrolizumab. Following disease progression, he was enrolled in a clinical trial for an investigational targeted

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agent (HMPL-453). Shortly after the first cycle, he developed severe DILI, characterized by an ALT surge to 800–1000 U/L and total bilirubin peaking at 98 μ mol/L. Serological testing revealed positive HBsAb, HBeAb, and HBcAb, but HBV DNA remained undetectable, effectively ruling out viral reactivation. Despite this, DILI attribution remained challenging. A percutaneous liver biopsy was performed to clarify the etiology but was complicated by immediate intra-operative hemorrhage. Pathology indicated that SOS could not be excluded. Conversely, subsequent imaging revealed diffuse dilation of Grade III hepatic arteries with increased flow, a finding inconsistent with classic SOS features. Post-discharge, the patient sustained blunt abdominal trauma from a fall, causing liver rupture and catastrophic intraperitoneal hemorrhage. Emergency TAE was successfully employed to arrest the bleeding and stabilize the patient.

Conclusion:

This case exemplifies the extreme challenge of DILI attribution in patients receiving complex, combined therapeutic modalities. The stark discordance between pathology and imaging—where histology suggested SOS-pattern injury while radiology demonstrated compensatory arterial dilation—confirms that multi-drug interactions can yield "chameleon-like" phenotypes. This uncertainty mandates extreme caution against the reflexive diagnosis of irAEs. Moreover, the cumulative chemical insult renders the liver critically vulnerable to mechanical trauma and invasive procedures. Consequently, transjugular liver biopsy (TJLB) should be strongly preferred over the percutaneous route in such high-risk profiles to avoid hemorrhagic events. Finally, this case validates TAE as an indispensable, life-saving rescue intervention for catastrophic hepatic hemorrhage in the oncologic setting.

Keyword(s) : drug-induced liver injury (DILI), chemo-immunotherapy, Sinusoidal Obstruction Syndrome (SOS) -Pattern, Transarterial Embolization (TAE)

1809 - Transcriptomic Deconvolution Characterizes Intrahepatic Neutrophils in On treatment Chronic Hepatitis B Patients with Liver Fibrosis

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Background / Hypothesis:

the dynamics and functional heterogeneity of intrahepatic neutrophils in chronic hepatitis B (CHB) liver fibrosis during antiviral therapy (AVT) remain poorly characterized.

Materials and Methods:

transcriptomic data from 54 liver biopsies collected from 28 patients at baseline, 78 weeks, and 260 weeks of AVT were analyzed, together with single-cell RNA sequencing (scRNA-seq) data from five CHB liver fibrosis patients (GSE186343). Deconvolution approaches were applied to assess temporal changes and phenotypic heterogeneity of intrahepatic neutrophils throughout AVT.

Results:

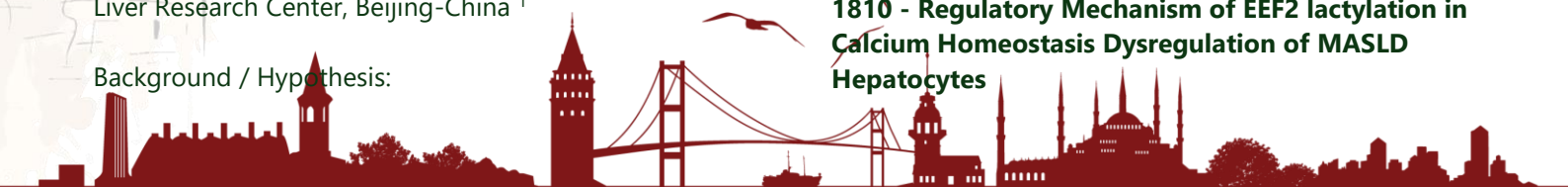
The intrahepatic neutrophil fraction increased at week 78 of AVT ($P = 0.252$) and reached statistical significance by week 260 ($P < 0.001$), accompanied by persistent decreases in effector CD8⁺T cells, B cells, and natural killer cells. Based on median neutrophil fractions, patients were categorized into Neu-low and Neu-high groups. The Neu-low group exhibited elevated expression of liver fibrosis-specific matrisome gene. Gene set enrichment analysis indicated enrichment of extracellular matrix remodeling, immune response, and cell cycle pathways in the Neu-low group, whereas metabolic pathways were downregulated. Re-analysis of scRNA-seq data from the GSE186343 dataset identified two pro-inflammatory intrahepatic neutrophil subclusters (NEU-C1 and NEU-C3) and one immunosuppressive subcluster (NEU-C2). Notably, the proportion of NEU-C2 was lower in treatment-naïve Neu-low patients but progressively increased during AVT.

Conclusions:

our study reveals that distinct neutrophil subsets undergo dynamic remodeling during long-term AVT, with the expansion of the immunosuppressive NEU-C2 subset potentially restoring intrahepatic adaptive immunity and contributing to immune homeostasis. These findings provide insight into neutrophil-mediated regulation of fibrosis and reveal potential therapeutic targets for CHB patients receiving AVT.

Keyword(s) : Chronic hepatitis B; Liver fibrosis; Antiviral therapy; Neutrophils, Bulk RNA sequencing, Single-cell RNA sequencing; Deconvolution analysis

1810 - Regulatory Mechanism of EEF2 lactylation in Calcium Homeostasis Dysregulation of MASLD Hepatocytes



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Background / Hypothesis:Metabolic dysfunction-associated steatohepatitis (MASLD) ranks among the most prevalent chronic liver diseases of the 21st century. During MASLD progression, disrupted lactate metabolism leads to hepatic lactate accumulation. Our prior research confirmed that lactate promotes hepatic lipid deposition and lipid peroxidation, yet its precise mechanisms in MASLD pathogenesis remain unclear. Furthermore, lipid accumulation within steatotic hepatocytes disrupts intracellular calcium homeostasis, thereby accelerating the progression of metabolic dysfunction-associated steatohepatitis (MASH) and hepatocellular carcinoma. Protein modification omics results indicate that eEF2 undergoes lactylation. Simultaneously, the core calcium homeostasis regulatory axis ($\text{Ca}^{2+}/\text{CaM}$)-eEF2K-eEF2-Thr56 phosphorylation plays a crucial role in maintaining protein translation elongation. However, whether this process reciprocally influences intracellular calcium signaling dynamics remains unclear. Our study demonstrates that eEF2 undergoes both phosphorylation and lactylation modifications during MASLD progression. Therefore, the roles of eEF2 phosphorylation and lactylation in MASLD progression warrant further investigation.

Materials and Methods:Using a palmitic acid (PA)-induced in vivo MASLD mouse model and in vitro cell model to evaluate changes in lactate levels and fatty liver indicators. Simultaneously, intracellular Ca^{2+} levels were measured by flow cytometry. Lactic acid-modified eEF2 protein was identified using immunoprecipitation-mass spectrometry (IP-MS) and immunoprecipitation techniques. Lactic acid modification sites were localized by constructing eEF2 mutant plasmids, and changes in relevant indicators following site mutations were analyzed to validate in vivo and in vitro experimental results.

Results:In PA-induced in vitro MASLD cell models and MASLD mice, lactate levels were significantly elevated. Lactic acid levels positively correlated with fatty liver markers, and lactate production was markedly enhanced in both MASLD models. During MASLD progression, eEF2 underwent simultaneous phosphorylation and lactate production. Treatment with a targeted mutant eEF2 plasmid revealed that site-specific mutations improved hepatic steatosis, lipid peroxidation, and macrophage infiltration by regulating calcium homeostasis.

Conclusions:Taken together, dysregulated lactate metabolism impairs lactate modification of eEF2, thereby disrupting calcium homeostasis remodeling and promoting MASLD progression. The pathological feedback loop "elevated calcium → crosstalk between eEF2-Thr56 phosphorylation and lactylation → insufficient calcium pump translation → worsening calcium homeostasis" reveals a novel "translation-metabolism" cross-talk mechanism in MASLD, providing theoretical support for targeting eEF2 as a therapeutic intervention for MASLD.

Keyword(s) : MASLD; Lactate; eEF2; Lactylation; phosphorylation; Ca^{2+} homeostasis

1811 - Combination with glucocorticoids during artificial liver support system treatment does not improve the survival rate of Acute on chronic liver failure patients A retrospective cohort study

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Background / Hypothesis: To investigate retrospectively the efficacy of glucocorticoids in acute-on-chronic liver failure (ACLF) patients receiving artificial liver support system (ALSS) therapy.

Materials and Methods: ACLF patients treated with ALSS were categorized into glucocorticoids-treated group and control group based on whether they receive glucocorticoids treatment. Propensity score matching (PSM) was utilized in a 1:1 ratio to balance baseline characteristics. Subgroups were stratified by the Model for End-Stage Liver Disease (MELD) scores and Chinese Group on the Study of Severe Hepatitis B ACLF 2.0 (COSSH-ACLF 2.0)

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scores. Survival outcomes were analyzed by using Kaplan-Meier curves and log-rank tests. Univariate and multivariate analyses identified predictors of 90-day transplant-free survival (TFS).

Results:

From 2014 to 2025, a total of 124 ACLF patients treated with ALSS were included and 100 patients (control: n=50; glucocorticoids: n=50) were matched by PSM. The glucocorticoids group received dexamethasone at an average dose of 5 mg/d before each ALSS procedure. At 90 days, overall survival was comparable between control and glucocorticoids groups (72.0% vs. 67.8%, $p = 0.6636$), with TFS rates of 58.1% versus 67.8% ($p = 0.3553$). Patients with MELD scores > 24 or COSSH-ACLF 2.0 scores > 7.3 exhibited significantly lower 90-day TFS (both $p < 0.0001$). Glucocorticoids treatment failed to show a survival benefit in any subgroup. Overall infections show no statistically significant between control group and glucocorticoids group (36% vs 30%, $p = 0.3912$). In the univariate analysis, neutrophil count, serum creatinine, blood urea nitrogen (BUN), INR, PT, APTT, ALSS procedures, baseline MELD scores > 24 and COSSH-ACLF 2.0 scores > 7.3 were 90-day mortality predictors. Multivariate analysis confirmed that BUN (HR = 1.19, 95% CI = 1.04–1.37, $p = 0.013$), ammonia (HR = 1.02, 95% CI = 1.01–1.03, $p = 0.005$), ALSS procedures (HR = 0.51, 95% CI = 0.32–0.80, $p = 0.003$), and new infections (HR = 3.99, 95% CI = 1.16–13.74, $p = 0.028$) as independent 90-day mortality determinants.

Conclusions:

Glucocorticoids therapy did not improve transplant-free survival in ACLF patients undergoing ALSS. Key prognostic factors included BUN, ammonia levels, and new infections.

Keyword(s) : acute-on-chronic liver failure, artificial liver support system, glucocorticoids, survival

1814 - Weight Reduction and Its Association With CAP and Liver Stiffness in MASLD

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Background / Hypothesis: The Controlled attenuation parameter (CAP) and the liver stiffness (LS) of transient elastography (TE) are widely used to measure the degree of fatty liver and liver fibrosis. We investigated whether there were significant changes in CAP and LS according to the amount of weight loss in patients with

metabolic dysfunction-associated liver disease (MASLD).

Materials and Methods: The patients with MASLD at Jeju National University Hospital in Korea who had TE tests more than once while losing body weight were analyzed. CAP and LS were compared between the time of the first test and the time of the weight was most lost during the follow-up.

Results: A total of 410 patients were analyzed and 74.6% of patients lost body weight during the follow-up period. The mean age was 51.97 years (standard deviation (SD): 15.10), the mean body mass index (BMI) was 28.71 (SD: 4.21). Patients who lost weight were divided into 5 groups according to the amount of weight loss (WL), and all groups showed significant CAP change (-25.62 in the 0 to 3% WL group, -21.52 in the 3 to 5% WL group, -41.92 in the 5 to 7% WL group, -32.79 in the 7 to 10% WL group, -74.16 in the more than 10% WL group, all p -values < 0.05). The mean LS change by group was +0.75 in the 0 to 3% WL group, -0.30 in the 3 to 5% WL group, +0.40 in the 5 to 7% WL group, -0.40 in the 7 to 10% WL group, -1.31 in the more than 10% WL group, and was significant only in the more than 10% WL group ($p=0.002$). In multivariable analysis, weight change percentage, baseline BMI, and weight loss duration were significantly associated with CAP change, and weight change percentage, baseline BMI, and weight loss duration were significantly associated with LS change.

Conclusions: In patients with MASLD, there was a significant correlation between weight loss and CAP, and LS decreases significantly when weight loss is greater than 10% of baseline body weight.

Keyword(s) : MASLD; weight reduction; controlled attenuation parameters; liver stiffness

1817 - Comparison between Plasma Exchange and Dual plasma molecular adsorption system combined with Plasma Exchange on Acute on Chronic Liver Failure A retrospective cohort study

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Background / Hypothesis:

To compare the efficacy and safety of dual plasma molecular adsorption system (DPMAS) combined with plasma exchange (PE) versus PE alone in patients with

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acute-on-chronic liver failure (ACLF).

Materials and Methods:

A retrospective cohort study included ACLF patients receiving DPMAS + PE (n=51) or PE alone (n=57) from 2014 to 2025. Propensity score matching (PSM) was performed in a 1:1 ratio to balance baseline characteristics. Survival analyses of two groups and subgroups classified by the model for end-stage liver disease (MELD) and Chinese Group on the Study of Severe Hepatitis B ACLF 2.0 (COSSH-ACLF 2.0) score were performed. Cox regression analyses identified factors for 90-day transplant-free survival (TFS).

Results:

After PSM, 102 patients (DPMAS + PE: n=51; PE: n=51) were analyzed. The DPMAS + PE and PE groups showed comparable 90-day overall survival (66.5% vs. 64.7%, $P=0.848$) and TFS (66.5% vs. 57.9%, $P=0.406$). MELD scores = 23 and COSSH-ACLF 2.0 scores = 7.0 were optimal mortality prediction thresholds. Patients with MELD scores > 23 and COSSH-ACLF 2.0 scores > 7 exhibited significantly lower 90-day TFS (both $P<0.05$). Subgroup analyses revealed a non-significant survival trend favoring DPMAS + PE in high-risk subgroups (MELD scores > 23, $P=0.271$; COSSH-ACLF 2.0 scores > 7, $P=0.128$). New infections show no statistically significant between DPMAS + PE and PE group (39.2% vs 29.4%, $P=0.297$). Univariate analysis identified hepatic encephalopathy, blood urea nitrogen (BUN), INR, PT, APTT, baseline MELD scores > 23 and COSSH-ACLF2.0 scores > 7 as significant predictors of 90-day TFS. Multivariate analysis confirmed hepatic encephalopathy (HR = 3.03, 95% CI: 1.10–8.36, $P = 0.032$), BUN (HR = 1.20, 95% CI: 1.02–1.40, $P = 0.024$), and INR (HR = 2.70, 95% CI: 1.14–6.36, $P = 0.024$) as independent prognostic factors of 90-day TFS.

Conclusions:

DPMAS combined with PE showed comparable 90-day survival and safety to PE alone in ACLF, with a potential survival trend in high-risk subgroups.

Keyword(s) : artificial liver support system, dual plasma molecular adsorption system, plasma exchange, acute-on-chronic liver failure, survival

1823 - Study on Risk Factors for Hepatocellular Carcinoma in Chronic Hepatitis B Patients Treated with Interferon

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Department Of Hepatology Division 2, Beijing-China¹

Background / Hypothesis:

To investigate the risk factors for hepatocellular carcinoma (HCC) in chronic hepatitis B (CHB) patients treated with interferon.

Materials and Methods:

A retrospective study was conducted, enrolling 2,034 CHB patients who received interferon antiviral therapy at the affiliated hospital of Capital Medical University between September 2008 and January 2019. Baseline data, including demographic characteristics, virological markers, serological indicators, clinical biochemistry, imaging data, and antiviral treatment information, were collected. The Kaplan-Meier method was used to assess the cumulative incidence and risk of HCC, and multivariate Cox regression analysis was employed to identify independent risk factors for HCC development in these patients.

Results:

Among the 2,034 interferon-treated patients, the median follow-up time was 4.64 (2.46–7.02) years, and 28 patients developed HCC. The HCC group (n=28) had significantly higher baseline age (43 vs. 32 years, $P < 0.001$), AFP level (9.40 vs. 4.20 ng/ml, $P < 0.001$), proportion of baseline cirrhosis (67.9% vs. 9.7%, $\chi^2=98.56$, $P < 0.001$), and male gender proportion (96.4% vs. 70.1%, $P=0.001$) compared to the non-HCC group. Baseline PLT level was significantly lower in the HCC group (110.40 vs. $167.00 \times 10^9/L$, $P=0.002$). However, there were no significant differences in baseline HBV DNA load (4.74 vs. 5.52 log₁₀ IU/mL, $P > 0.05$), ALT (55.60 vs. 76.75 U/L, $P > 0.05$), TBIL (14.80 vs. 14.20 $\mu\text{mol/L}$, $P > 0.05$), ALB (45.60 vs. 45.80 g/L, $P > 0.05$), proportion of baseline HBeAg positivity (67.7% vs. 67.5%, $P > 0.05$), or proportion of fatty liver (36.6% vs. 42.9%, $P > 0.05$) between the two groups. Kaplan-Meier analysis showed that baseline age, gender, presence of cirrhosis, HBV DNA level, PLT level, and AFP level were statistically significant for the cumulative incidence of HCC in interferon-treated patients. The presence of fatty liver during follow-up, baseline HBeAg status, AST level, TBIL level, and ALB level showed no statistical significance (all $P > 0.05$). Multivariate Cox regression analysis of age, gender, baseline cirrhosis, HBV DNA level, PLT level, and AFP level revealed that the presence of baseline cirrhosis was an independent risk factor for HCC in interferon-treated CHB patients ($P < 0.001$, HR=15.097, 95%

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CI=3.447-66.323). Age, gender, baseline HBV DNA level, PLT level, and AFP level were not independent risk factors (all $P > 0.05$)

Conclusions:

Liver cirrhosis is an independent risk factor for HCC development in interferon-treated patients. Preventing or delaying the onset of cirrhosis is key to reducing HCC incidence in CHB patients receiving interferon therapy.

Keyword(s) : Chronic hepatitis B; Antiviral therapy; Interferon; Hepatocellular carcinoma; Risk factors

1826 - Primary biliary cholangitis with concomitant fatty liver disease a reappraisal of clinical features UDCA treatment response and prognostic implications

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Background / Hypothesis: Concomitant fatty liver disease (FLD) is not uncommon in patients with primary biliary cholangitis (PBC), yet its associations with clinical features, treatment response, and prognosis remain unclear. This study aimed to characterize the clinical phenotype and non-invasive fibrosis profiles of treatment-naïve PBC patients with FLD, and to evaluate the impact of FLD on biochemical response and 1-year GLOBE scores following ursodeoxycholic acid (UDCA) therapy. Subgroup analyses were further performed in patients with varying degrees of cirrhosis to clarify the clinical significance of FLD under different backgrounds of hepatic injury.

Materials and Methods: A total of 504 treatment-naïve PBC patients were retrospectively enrolled and categorized according to the presence of FLD on ultrasonography. Liver stiffness status was further classified as definite cirrhosis, suspected cirrhosis, or non-cirrhosis. Baseline biochemistry, immunoglobulins, white blood cell and platelet counts, ALBI score, and FIB-4 index were recorded. Biochemical response at 1 year was defined by Paris I/II criteria, and deep response by complete normalization of ALP and TBIL. The 1-year GLOBE score was calculated to estimate transplant-free survival. Group differences were assessed using the Mann-Whitney U test and χ^2 /Fisher's exact test. Multivariable logistic regression was used to evaluate whether FLD independently predicted treatment response or prognosis, with

additional stratification by cirrhosis status.

Results: 47 patients (9.3%) had FLD, of whom 93.6% exhibited ultrasonographic cirrhosis. Compared with non-FLD patients, the FLD group showed significantly lower baseline ALP, GGT, TBIL, IgA, and IgG, higher albumin and platelet counts, and more favorable ALBI, FIB-4, and GLOBE scores (all $P < 0.05$). These differences persisted within the cirrhosis subgroup. Among 229 patients with 1-year follow-up, FLD patients demonstrated a higher overall biochemical response rate (72.4% vs. 53.0%), but this difference disappeared after stratification by cirrhosis status. Similarly, the lower 1-year GLOBE scores observed in the FLD group were no longer significant after stratification. Multivariable analyses showed that FLD was not independently associated with biochemical response or deep response (all $P > 0.05$).

Conclusions: Although PBC patients with FLD exhibit milder cholestasis, fibrosis burden, and immunologic activity, along with a trend toward better 1-year biochemical response and GLOBE scores, these advantages largely dissipate after adjusting for cirrhosis status and baseline disease severity. FLD does not independently influence treatment response or prognosis, and likely represents an accompanying phenotype linked to disease stage rather than a causal modifier of PBC progression or UDCA responsiveness.

Keyword(s) : Biochemical response, Fatty liver disease, Primary biliary cholangitis;

1827 - Identification of Ferroptosis Related Biomarkers and Hub Genes in Non Alcoholic Fatty Liver; an In Silico Transcriptomic Analysis

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is a widespread metabolic condition, with non-alcoholic fatty liver (NAFL) reflecting an initial yet clinically significant stage. Ferroptosis, an iron-dependent lipid peroxidation-driven form of cell death, has appeared as a principle determinant to hepatic injury. However, ferroptosis-associated biomarkers in NAFL persist as inadequately characterized.

Materials and Methods:

This study designed to identify potential ferroptosis-related biomarkers using an in silico transcriptomic analysis. RNA-seq dataset GSE126848 was retrieved

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from the GEO database. Out of 57 samples, 15 NAFLD and 14 normal-weight samples were selected.

Differential expression analysis was carried out using DESeq2 with cutoffs of adjusted $p < 0.05$ and $|\log_2FC| \geq 1$, resulting in 1247 significant DEGs, including 633 upregulated and 614 downregulated genes.

Ferroptosis-related driver, suppressor, and marker genes were downloaded from FerrDb V3, duplicates were eliminated, and intersection with DEGs using Venny 2.1.0 detected 126 ferroptosis-related genes. STRING was used to build PPI networks, and visualization was done using Cytoscape V3.10.4. Hub genes were identified based on combined in-degree and out-degree scores.

Results:

Among the 126 ferroptosis-related DEGs, 52 genes were classified as drivers, 86 genes as suppressors, and 3 genes as markers. Using a minimum required interaction score of 0.4, STRING network analysis yielded 121 nodes, 331 edges, and significant PPI enrichment $p\text{-value} < 1e-16$. Cytoscape degree analysis underscored top ten hub genes—TGFB1, KDR, NOTCH1, EZH2, SERPINE1, SNAI2, BIRC5, IGF1R, PPARGC1A, and FOXM1—as key regulators of ferroptosis in NAFL. KEGG enrichment analysis highlighted substantial involvement of the HIF-1 ($FDR = 6.26 \times 10^{-5}$), PI3K-Akt ($FDR = 2.2 \times 10^{-4}$), and PPAR ($FDR = 5.5 \times 10^{-3}$) signaling pathways, emphasizing dysregulated metabolic and oxidative stress pathways associated with NAFL pathogenesis.

Conclusions:

This study systematically identified ferroptosis-related biomarkers and hub genes in NAFL, offering novel insights into disease mechanism. These candidate genes may function as potential diagnostic biomarkers or therapeutic targets, necessitating subsequent experimental and clinical validation.

Keyword(s) :

1828 - Association of CORE Score with Parenchymal Liver Disease in HBsAg Positive Patients

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Background / Hypothesis: Chronic liver diseases due to hepatitis B infection may progress to serious

complications such as cirrhosis and hepatocellular carcinoma. Various non-invasive, serum-based scores (e.g., FIB-4, APRI, AST/ALT ratio) have been widely used in clinical practice to estimate disease progression in these patients. Recently, the CORE (Cirrhosis Outcome Risk Estimator) score has emerged as a novel model that uses routine biochemical parameters (age, sex, AST, ALT, GGT) to predict liver-related adverse outcomes with high accuracy (AUC 88%) (1). In this study, we aimed to investigate the association between the CORE score and parenchymal liver disease in HBsAg-positive patients.

Materials and Methods: HBsAg-positive patients followed up as outpatients in our hospital between January 1, 2023, and November 1, 2025 were included in this retrospective cross-sectional study. Laboratory data obtained from the electronic medical record system included age, sex, ALT, AST, GGT, ALP, platelet count, albumin, and AFP levels. Based on these parameters, APRI, FIB-4, AST/ALT ratio, and CORE risk scores were calculated. The CORE score was calculated individually using the online algorithm (<https://rickstra.shinyapps.io/CORE/>). The presence of parenchymal disease was defined by ultrasonographic findings such as fine/coarse granular or heterogeneous parenchyma. Patients were divided into two groups. Comparative analyses were conducted, and a $p\text{-value} < 0.05$ was considered statistically significant.

Results: Of the 208 patients included in the study, 133 were male. The mean age was 53.46 ± 13.46 years (range: 21–86). HBeAg was positive in 22 patients, and anti-HBe was positive in 191. Parenchymal liver disease was detected in 105 patients. Biochemical tests and scoring systems were compared according to the presence of parenchymal liver disease (see Table 1). Among the biochemical parameters, ALT, ALP, GGT, AFP, albumin, and platelet counts were significantly different between the two groups. Similarly, the AST/ALT ratio, APRI, FIB-4, and particularly the CORE risk score were significantly elevated in patients with parenchymal liver disease.

Conclusions: Our study showed that in patients with parenchymal liver disease, laboratory markers such as ALT, ALP, GGT, AFP, albumin, and platelet counts—as well as scoring systems like APRI, FIB-4, AST/ALT ratio, and notably the CORE risk score—were significantly higher. The CORE score stands out as a valuable predictive tool, as it can be calculated using only routine biochemical parameters and provides long-term risk estimation for liver-related events such as cirrhosis. These findings suggest that the CORE score may serve as an effective non-invasive marker for assessing the risk of chronic liver disease in HBsAg-

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positive individuals.

Keyword(s) :

1829 - Clinical Challenges in Diagnosing Primary Testicular Lymphoma Report of Two Cases in Elderly Patients

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Background / Hypothesis: Primary testicular lymphoma (PTL) is a rare but highly aggressive subtype of non-Hodgkin lymphoma that originates in the testis. It predominantly affects elderly men and is characterized by frequent bilateral involvement and a high propensity for extranodal dissemination, resulting in poor prognosis. The disease often presents with nonspecific symptoms such as scrotal swelling and pain, making it easily confused with infectious or inflammatory conditions and leading to delayed diagnosis.

Materials and Methods: We describe two cases of PTL in men over 60 years of age who presented with fever and testicular pain. Case 1: A 66-year-old man experienced persistent fever for over three months. Pelvic contrast-enhanced MRI revealed bilateral epididymal lesions, suggesting the possibility of infection or tuberculosis (figure 1). Despite antibiotic and antituberculous therapy, symptoms persisted. PET/CT indicated a hematologic malignancy, prompting left epididymal–testicular excisional biopsy. Histopathology confirmed intravascular large B-cell lymphoma, establishing the diagnosis of PTL. Case 2: A 68-year-old man with a history of urologic malignancy presented with fever and left testicular swelling. Elevated inflammatory markers and poor response to antimicrobial therapy raised suspicion for a neoplastic process. Pelvic MRI demonstrated a left testicular mass (figure 2), and PET/CT suggested a malignant lesion involving the testis and epididymis with retroperitoneal lymph node metastases. Left orchiectomy was performed, and pathology indicated diffuse large B-cell lymphoma, confirming PTL.

Results: Both patients presented with nonspecific symptoms mimicking infectious diseases, resulting in diagnostic delay. Imaging examinations (ultrasound, MRI, PET/CT) provided important but non-definitive clues. Testicular biopsy is rarely performed due to

procedural risks and diagnostic limitations. In both cases, definitive diagnosis relied on postoperative histopathology combined with immunohistochemistry. These cases highlight that timely imaging and pathological examinations can significantly improve the diagnostic yield.

Conclusions: PTL should be considered in elderly males presenting with unexplained testicular swelling accompanied by systemic symptoms. Early integration of multimodal imaging and timely surgical intervention are essential to avoid diagnostic delay. Given its aggressive nature and high recurrence rate, prompt diagnosis and multidisciplinary management are crucial for improving clinical outcomes.

Keyword(s) : Primary testicular lymphoma, diffuse large B-cell lymphoma, case report, diagnosis, orchiectomy

1830 - Integrative Analysis of Differentially Expressed Ferroptosis Related Genes in Cell Cycle Regulation of Hepatocellular Carcinoma

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Background / Hypothesis:

Globally, Hepatocellular carcinoma (HCC) is the primary cause of liver-related deaths, and investigating its molecular pathways is essential for advancing clinical diagnosis and therapy. Ferroptosis, an iron-dependent programmed cell death pathway, has currently been implicated in tumor development, but its contribution to HCC is still not well understood.

Materials and Methods:

In this research work, we examined the microarray dataset GSE101685 (Homo sapiens, platform GPL570) using GEO2R to detect differentially expressed genes (DEGs) between 8 normal liver and 24 HCC tissues. Volcano plot and box plot were used to identify significant target genes. Ferroptosis-related genes (drivers, suppressors, and markers) were extracted from FerrDb V3, and overlapping genes were assessed through intersection analysis. A protein–protein interaction (PPI) network was generated by uploading shared genes to the STRING database, and hub genes were identified using the Analyze Network tool in Cytoscape based on node degree value. Gene Ontology term enrichment was determined for the selected biomarker candidates and included an analysis of biological processes and cellular

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components.

Results:

49 significant DEGs were obtained after removing duplicates from 54,675 probes, consisting of 19 upregulated and 30 downregulated genes. 12 ferroptosis-related genes were obtained from the intersection using the Venn tool, including 4 drivers and 9 suppressors. PPI network analysis revealed significantly higher connectivity than expected. No significant Reactome or KEGG pathway enrichment was observed, indicating that functional interpretation was mainly based on GO annotations. GO-BP enrichment demonstrated that these genes are markedly associated with mitotic processes and cell cycle regulation, and GO-CC highlighted their localization to nuclear structures, including the midbody, spindle, centromere, and kinetochore, emphasizing their involvement in chromosome segregation and mitotic regulation. Hub genes such as ECT2, KIF14, TOP2A, and BUB1 were extracted from the PPI network using the network analysis tool in Cytoscape, based on degree value, indicating their role in modulating the cell cycle.

Conclusions:

These findings reveal key ferroptosis-related genes differentially expressed in HCC that contribute to cell-cycle-driven tumor proliferation, providing potential targets and offering new perspectives into ferroptosis-mediated mechanisms in hepatocellular carcinoma.

Keyword(s) : HCC, Ferroptosis, DEGs, PPI, String Db, Hub Genes

1831 - Effect of epigallocatechin gallate on ochratoxin induced liver damage

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Background / Hypothesis: Ochratoxin A (OTA), a potent mycotoxin produced by *Aspergillus* and *Penicillium* species, poses a significant public health concern due to its hepatotoxic effects(1). As one of the most

common food contaminants, OTA can lead to multiple organ toxicities, particularly affecting liver(2). The hepatotoxic effects of OTA are known to be associated with several mechanisms, including oxidative stress, inflammation, and apoptosis(3, 4). OTA-induced ER stress is known to activate the unfolded protein response (UPR), in which GRP78 and IRE1 α play central regulatory roles(5). Epigallocatechin Gallate (EGCG), a primary green tea polyphenol, has potent antioxidant and anti-inflammatory effects(6). OTA-induced liver toxicity and the protective effects of antioxidant compounds have been previously reported in several studies(7-9). However, studies on EGCG's protective effect against OTA-induced liver injury are limited. Therefore, this study aims to demonstrate the hepatoprotective potential of EGCG against OTA-induced liver toxicity.

Materials and Methods: In this study, Albino Wistar rats(n=30) were divided into five groups and administered OTA (0.5 mg/kg/day) for 14 days to induce a liver toxicity model. Concurrently, two different doses of EGCG (50 and 100 mg/kg/day) were administered orally to evaluate its potential protective effects against OTA-induced liver injury. At the end of the experiment, hepatic mRNA expression levels of GRP78, IRE1 α and TNF α were analyzed using the RT-PCR method.

Results: GRP78 and IRE1 α mRNA expression, markers of ER stress, increased in the toxicity group compared to the healthy group, and GRP78 mRNA expression also increased dose-dependently in the treatment groups. IRE1 α mRNA expression decreased dose-dependently in the treatment groups. TNF α mRNA expression also increased in the toxicity group and decreased dose-dependently in the treatment groups.

Conclusions: The increase in GRP78 in the toxicity and treatment groups may indicate a protective adaptive response against ER stress due to its central role in the UPR. Studies have shown that GRP78 levels increase to restore ER homeostasis. The increase in IRE1 α in the OTA toxicity group indicates increased ER stress. The decrease in IRE1 α in the EGCG groups suggests that increased GRP78 increases cell survival and reduces the UPR. The increase in TNF α in the toxicity group

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indicates activation of inflammatory pathways, and EGCG treatment suppressed inflammation in a dose-dependent manner. These results demonstrate that EGCG exerts a hepatoprotective effect against OTA-induced liver injury by modulating ER stress and reducing inflammation. This study suggests EGCG as a new potential target for xenobiotic induced chronic liver diseases.

Keyword(s) : Epigallocatechin gallate, Endoplasmic Reticulum Stress, Ochratoxin A, GRP78

1833 - Multifunctional Dialysate for Renal Hepatic and Pulmonary Support

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Background / Hypothesis:

By addition of albumin to dialysate solution both kidney and liver can be extracorporeally supported over one single dialyzer membrane. Here we present the addition of oxygenated hemoglobin to dialysate solution to further support lung function by removal of CO² and introduction of O² over the same hydrophilic membrane (Patent:US2022033933A1). This is a substantial improvement in biocompatibility compared to lipophilic membranes used in ECMO circuits and reduces system complexity.

Materials and Methods:

For the in-vitro experiments hemoglobin was added to albumin infused dialysate solution and recirculated against human blood intoxicated with kidney and liver failure toxins as well as CO². An EMIC² (Fresenius) hydrophilic dialysis membrane was used for therapy. Dialysate solution was regenerated by oxygen introduction. Control experiment was low flow ECCO²R (Fresenius).

Results:

Both systems show comparable capacity for removal CO² and introduction of O² while maintaining the capacity to remove water soluble toxins and albumin bound toxins. Creatinine was reduced from 715,3 to 113,1 µM and bile acids were reduced from 37,1 to 25,4 µM.

Conclusions:

The addition of hemoglobin to dialysate solution allows for additional lung support options in extracorporeal dialysis circuits. This avoids the need for bio-incompatible lipophilic ECMO membranes and reduces system complexity. Flow rates and hemoglobin concentration can be optimized for further mass transfer increase. The system is under current investigation.

Keyword(s) : Albumin dialysis; renal dialysis, dialysis, ECMO, membrane oxygenation, extracorporeal treatments, emerging technologies

1835 - Meta Analysis of Ferroptosis Related Differentially Expressed Genes in Hepatocellular Carcinoma

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Background / Hypothesis:

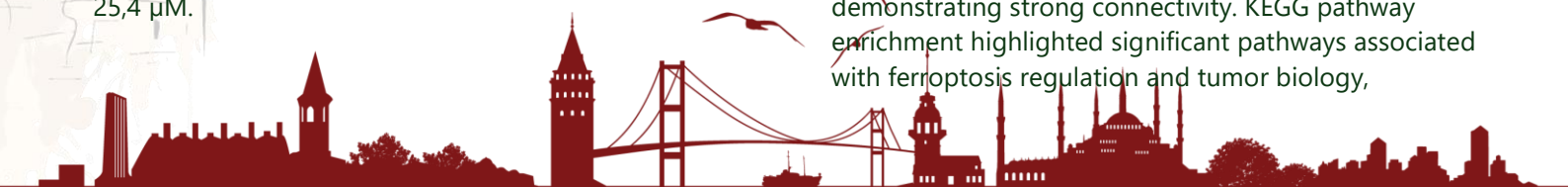
Hepatocellular carcinoma (HCC) remains a clinically aggressive malignancy, and its ferroptosis-related biomarkers are not yet fully defined.

Materials and Methods:

To uncover human ferroptotic-regulatory patterns in HCC, we analyzed the microarray dataset GSE41804 (GPL570), consisting of 40 human liver samples (20 resected non-tumor liver tissues and 20 resected liver tumors). The data was analyzed using GEO2R analysis, FerrDb, PPI analysis through STRING db, KEGG pathway, and Cytoscape analysis.

Results:

GEO2R analysis using adjusted p-value < 0.05 and |log2FC| ≥ 1 yielded 2338 differentially expressed genes (DEGs), narrowed to 1571 unique DEGs, including 951 upregulated and 623 downregulated. These were intersected with human ferroptosis-related genes from FerrDb, resulting in 152 overlapping genes. Within this set, 68 were drivers (39 upregulated, 29 downregulated), 103 suppressors (53 upregulated, 51 downregulated), and 4 markers (2 upregulated, 2 downregulated), with some genes classified in multiple categories. STRING analysis of these 152 genes generated a protein-protein interaction network containing 148 nodes and 572 edges (p < 1.0e-16) with a medium-confidence interaction score of 0.4, demonstrating strong connectivity. KEGG pathway enrichment highlighted significant pathways associated with ferroptosis regulation and tumor biology,



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including the p53 signaling pathway, cytokine–cytokine receptor interaction and the PI3K–Akt signaling pathway, all linked to oxidative stress, immune modulation, and cell survival. Furthermore, hub genes such as ESR1, CDK1, FOXM1, CDKN2A, AURKA, TOP2A, ERBB2, BIRC5, CDH1, and AURKB, were prioritized from the PPI network using the network analyzer tool in Cytoscape, based on degree centrality, emphasizing underscoring their central roles in the ferroptosis network.

Conclusions:

Taken together, this human-centric integrative analysis highlights key ferroptosis-related biomarkers and pathways in HCC, defining a foundation for downstream studies for future mechanistic and therapeutic studies.

Keyword(s) :

1838 - Predictive Value of the aMAP Score for Hepatocellular Carcinoma Risk in Chronic Hepatitis B Patients Treated with Interferon

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Background / Hypothesis:

To evaluate the predictive value of hepatocellular carcinoma (HCC) risk prediction models in chronic hepatitis B (CHB) patients receiving interferon therapy.

Materials and Methods:

This study enrolled CHB patients who received interferon antiviral therapy in the Second Department of Liver Diseases at Beijing Ditan Hospital between September 2008 and January 2019. All patients received at least 12 weeks of interferon therapy. Baseline data, including demographic, virological, serological, clinical biochemical, and imaging parameters, as well as antiviral treatment information, were collected. The cumulative incidence of HCC was plotted using the Kaplan-Meier method. Non-imaging-based HCC risk prediction models were applied to stratify patients according to baseline HCC risk, and the accuracy of the risk scores was analyzed using the area under the receiver operating characteristic curve (AUC).

Results:

A total of 668 patients were enrolled, with a median age of 33 (27–41) years and a median follow-up of 6.47 (3.57–8.44) years. During follow-up, 12 patients developed HCC. The cumulative incidence rates of HCC

at 1, 3, 5, 8, and 10 years were 0.2%, 0.2%, 0.4%, 0.9%, and 1.2%, respectively. The AUCs for the PAGE-B, mPAGE-B, aMAP, and APA-B models at 1 year were 0.53 (95% CI: 0.50–0.57, $P > 0.50$), 0.74 (95% CI: 0.67–0.81, $P > 0.50$), 0.76 (95% CI: 0.73–0.79, $P > 0.50$), and 0.94 (95% CI: 0.92–0.97, $P > 0.50$), respectively. At 3 and 5 years, the AUCs for these models remained suboptimal. However, at 8 years, the AUCs improved to 0.75 (95% CI: 0.64–0.86, $P = 0.011$), 0.78 (95% CI: 0.64–0.92, $P = 0.004$), 0.82 (95% CI: 0.71–0.93, $P = 0.001$), and 0.78 (95% CI: 0.63–0.93, $P = 0.004$), respectively. At 10 years, the AUCs were 0.77 (95% CI: 0.68–0.86, $P = 0.001$), 0.80 (95% CI: 0.68–0.91, $P < 0.001$), 0.82 (95% CI: 0.72–0.92, $P < 0.001$), and 0.78 (95% CI: 0.66–0.91, $P = 0.001$), respectively. Among the four models, aMAP demonstrated the highest predictive ability.

Conclusions: CHB patients treated with interferon have a low risk of developing HCC. The aMAP score shows high accuracy in predicting long-term HCC risk in these patients.

Keyword(s) : Antiviral therapy; Interferon; Chronic hepatitis B; Hepatocellular carcinoma; Prediction model

1839 - Efficacy of long term albumin therapy in the treatment of decompensated cirrhosis.

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Background / Hypothesis: Decompensated liver cirrhosis has a poor prognosis, with a median overall survival of two to four years, which is worse than for many oncological disorders. These patients are highly susceptible to infections due to increased systemic inflammation leading to kidney failure and death. The aim was to study the efficacy of albumin in reducing episodes of decompensation, preventing bacterial infection, kidney dysfunction and mortality.

Materials and Methods: Study involved patients with Child B or C cirrhosis with an albumin level below 3.0 g/dL, who were administered 20% human albumin weekly with standard medical treatment (SMT) for three months or till serum albumin levels were 4.0 g/dL (whichever is earlier) and compared with age and sex-matched controls who received only SMT. The primary end-point was six-month mortality and the secondary end-points were reduction in infections, kidney dysfunction, ascites recurrence, hepatic encephalopathy (HE), gastrointestinal (GI) bleed and

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complications of cirrhosis.

Results: From September 2021 to January 2023, 88 cases and 86 controls were taken and followed up for six months. Overall, six-month survival was not statistically significant between groups (95.1% vs. 91.9%; $p = 0.330$). The incidence of recurrence of ascites (34.09% vs. 59.3%, $p < 0.001$), kidney dysfunction (6.8% vs. 24.4%, $p < 0.001$), HE (15.9% vs. 37.2%, $p = 0.015$), spontaneous bacterial peritonitis (SBP) (3.4% vs 17.4%, $p = 0.002$) and non-SBP infections (7.9% vs. 18.6%, $p = 0.038$) were significantly less in cases as compared with controls; however, GI bleed (14.8% vs. 17.4%, $p = 0.632$) was not statistically significant.

Conclusions: Long-term human albumin acts as a disease-modifying treatment in patients with decompensated cirrhosis

Keyword(s) : Albumin; Cirrhosis; Decompensated cirrhosis; Long-term albumin; Weekly albumin.

1840 - Severe Hepatic Steatosis Attenuates Response to PEG IFN alpha 2b in HBsAg Clearance Favorable Chronic Hepatitis B Patients A Retrospective Cohort Study

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Background / Hypothesis: Hepatic steatosis (HS) is a common comorbidity in chronic hepatitis B (CHB). This study aimed to investigate the impact of varying degrees of HS on the treatment response to pegylated interferon alpha (PEG-IFN α) in CHB patients who are considered favorable for HBsAg clearance.

Materials and Methods: In this multi-center, cohort study, we enrolled 499 HBeAg-negative CHB patients with HBsAg <1500 IU/mL, a population with favorable prospects for HBsAg clearance, who received 48 weeks of PEG-IFN α therapy. Using controlled attenuation parameter (CAP) measurements, patients were classified into two groups: CHB without HS (CHB) and CHB with HS (CHB+HS). The CHB+HS group was further subdivided into mild ($n = 90$), moderate ($n = 41$), and severe ($n = 118$) steatosis subgroups. The primary endpoint was the HBsAg clearance rate at week 48.

Results: An overall HBsAg clearance rate of 39.1%

(195/499) was achieved among the study patients, who were selected for their favorable cure profile and completed the 48-week PEG-IFN α therapy. Although no overall difference was observed between CHB and CHB+HS groups (41.2% vs 37.0%, $P > 0.05$), severe HS was associated with significantly reduced clearance rates (24.6%) compared to both the CHB group (41.2%, $P < 0.01$) and mild or moderate HS subgroups (45.6% and 53.7%, respectively). After adjusting for baseline characteristics using inverse probability of treatment weighting (IPTW), Cox regression analysis identified severe HS as an independent negative predictor of HBsAg clearance (HR = 0.582, 95% CI 0.364–0.931, $p = 0.02$). The significance remained after additional adjustment for age, sex, baseline HBsAg, ALT, AST, and fibrosis stage (adjusted HR = 0.570, 95% CI 0.341–0.954, $p = 0.03$).

Conclusions: In the HBsAg clearance-favorable population, severe hepatic steatosis is significantly associated with a reduced response to PEG-IFN α -2b therapy.

Keyword(s) : chronic hepatitis B; hepatic steatosis; pegylated interferon; HBsAg clearance; treatment response; controlled attenuation parameter; severity

1841 - Timing of endoscopic hemostasis does not affect the short term outcomes in acute variceal bleeding in liver cirrhosis patients.

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Background / Hypothesis: Endoscopic treatment are necessary for hemostasis in acute variceal bleeding, with most guidelines recommending that emergency endoscopy be performed within 12 h of hospital arrival. But, the evidence to support the timing of endoscopy is still few, so we want to double check the short-term clinical outcomes of the acute variceal bleeding in cirrhotic patients according to the timing of starting endoscopy from the arrival at emergency room.

Materials and Methods: The window of the patient enrollment started from Jan 1st 2015 to Jan 21st 2021. This retrospective study included 64 patients admitted with acute esophageal or stomach variceal bleeding of one tertiary hospital in Cheongju, South-Korea. The primary outcome was the mortality and rebleeding in 6 weeks from endoscopic treatment. We adjusted

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confounding factors using the propensity score matching (1:1) method. For these matched patients, we performed between-group comparisons of mortality and rebleeding using Fisher's exact test and Cox proportional hazards model.

Results: When dividing patients with two groups according to EGD timing (urgent group 1 < 12 hrs and non-urgent group 2 $\geq$ 12 hrs), only EGD time (251.6 ± 187.3 vs. 1316.9 ± 962 min, $p=0.001$) and BUN (36.6 ± 22.0 vs. 24.1 ± 13.8 mg/dL, $p=0.01$) out of thirty three baseline characteristics were different between the two groups. The 6 weeks mortality was 12.5% vs. 9.4% ($p=0.701$), the 6 week rebleeding was 12.5 vs. 9.4% ($p=0.395$). Also, the hospital mortality was 9.4 vs. 9.4% ($p=1.0$), hospital rebleeding rate was 3.1 vs. 3.1%, ($p=1.0$)

Conclusions: The timing of endoscopy was not associated with 6 week mortality or 6 week rebleeding rate. The more important factor on patient survival is not early endoscopic intervention, but the early and proper cardiopulmonary resuscitation and intensive care regarding to each patient's condition. A prospective study will be needed for establishing a more accurate optimal timing of endoscopy.

Keyword(s) : Cirrhosis; Endoscopy; Gastroesophageal varices; Timing

1842 - T MSCs Alleviate Autoimmune Hepatitis in a ConA Mouse Model by Inhibiting IL 21-Driven Inflammatory Responses

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Background / Hypothesis: Autoimmune hepatitis (AIH) is characterized by T cell hyperactivation and an imbalance of inflammatory cytokines, potentially leading to acute and severe autoimmune-mediated liver injury. T-MSCs (Trophoblast-derived mesenchymal stem cells) exhibit considerable immunomodulatory potential; however, their protective effects and the underlying mechanisms in AIH remain to be elucidated.

Materials and Methods: A ConA-induced acute liver injury model was established by intravenous injection of ConA (15 mg/kg). T-MSCs (1×10^6 cells/mouse) were administered intravenously 3 hours after ConA injection. Mice were euthanized 24 hours after T-MSC administration, and serum and liver tissues were collected. The hepatoprotective effects of T-MSCs were systematically evaluated by measuring serum biochemical markers (ALT, AST, LDH), performing

hematoxylin and eosin (HE) staining, quantifying inflammatory and necrotic areas, scoring liver pathology using the Ishak Histological Activity Index (HAI), and analyzing IL-21 expression through immunofluorescence.

Results: In the ConA-induced acute immune-mediated liver injury model, T-MSC treatment exerted significant protective effects across both biochemical and histopathological indicators. Serum analyses revealed that ALT and AST levels were markedly elevated following ConA administration but were significantly reduced after T-MSC infusion, with ALT ($p = 0.0022$) and AST ($p = 0.0003$) showing clear improvement. Additionally, LDH levels were significantly lower than those in the ConA group ($p = 0.0067$). Histological evaluation confirmed extensive hepatic necrosis and inflammatory infiltration in the ConA group, while T-MSC treatment significantly reduced the necroinflammatory area ($p < 0.0001$) and substantially lowered the Ishak HAI score ($p < 0.0001$). Immunofluorescence analysis demonstrated that IL-21 expression was markedly upregulated in the ConA group, whereas T-MSC administration led to a significant decrease in IL-21 signal intensity ($p = 0.0001$); quantification of IL-21 per tissue area also showed a notable reduction ($p = 0.0149$).

Conclusions: T-MSCs demonstrated pronounced hepatoprotective effects in the ConA-induced acute immune-mediated liver injury model by significantly reducing serum transaminase levels, alleviating tissue necrosis, improving Ishak inflammation scores, and suppressing IL-21 overexpression.

Keyword(s) :

1844 - Yunpilishi Formula attenuates high fat diet induced obesity and fatty liver via inhibiting tPA

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is the leading cause of chronic liver disease worldwide. Yunpilishi Formula (YPLS) is a traditional Chinese medicine that has been used in the clinical treatment of MAFLD. However, its underlying mechanism for treating MAFLD remains unclear. Recent studies showing that the interaction between tPA, PAI-1, and apoB regulates VLDL secretion, thereby influencing the level of lipid deposition in hepatocytes. We aim to explore whether YPLS treats MAFLD by regulating the tPA/PAI-1/apoB pathway.

Materials and Methods:

Ultra-high performance liquid chromatography-mass spectrometry (UPLC-MS) was used to identify the main components of YPLS. A mouse model of nonalcoholic fatty liver disease was constructed by feeding the mice a high-fat diet for 16 weeks. YPLS was administered for an additional 8 weeks, and Polyene Phosphatidylcholine was used as a positive control. Liver sections were subjected to histopathological assessment. The NovaSeq™ 6000 platform (Illumina) was used to analyze differentially expressed genes (DEGs). Candidate proteins and pathways were validated through qRT-PCR and western blot analysis.

Results:

Twenty-four ingredients were identified in YPLS extracts, which were considered to be key compounds in the treatment of MAFLD. YPLS administration attenuated obesity and fatty liver by reducing the body weight and liver weight in HFD-fed mice. More importantly, hepatic steatosis was significantly attenuated by YPLS administration both in vivo and in vitro. Transcriptome sequencing (RNA-seq) of mouse liver tissues identified 1023 DEGs associated with YPLS treatment. Bioinformatic analysis of DEGs revealed that regulating the lipid and atherosclerosis pathway was the main role of YPLS in MAFLD treatment. This finding also indicated that tPA might be the potential target of YPLS, which was subsequently verified both in vitro and in vivo. The results demonstrated that YPLS enhanced tPA activity, thereby inhibiting PAI-1 expression and promoting apoB lipidation. ApoB lipidation promotes the secretion of VLDL from hepatocytes, leading to reduced lipid accumulation in hepatocytes.

Conclusions:

Overall, our findings confirmed the protective effect of YPLS against MAFLD and revealed the underlying

molecular mechanism of regulating lipid metabolism.

Keyword(s) : tPA; PAI-1; Lipid secretion; MAFLD; Yunpilishi formula.

1845 - Inositol Hexaphosphate Prevent WD CCl₄ Induced Metabolic Dysfunction Associated Steatohepatitis in Mice

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Background / Hypothesis:

Metabolic dysfunction-associated steatohepatitis (MASH) is a significant global public health issue. Since inositol hexakisphosphate (IP6) mainly extracted from plants, have anti-oxidative and anti-tumor effects. This study aimed to explore the preventive effects and mechanisms of IP6 on MASH.

Materials and Methods:

A western diet combined with low doses of CCl₄ (WD/CCl₄) was used to induce NASH in mice and evaluate the protective effects of IP6. Phenotypic assessments included metabolic profiles, histopathology (H&E, Oil Red O, Masson's trichrome), and serum biochemistry. Assessments of transcriptomics, metabolomics, and lipidomics analyses were conducted to explore potential molecular mechanisms. Additionally, the effects of IP6 and INS in vitro using free fatty acid (FFA)-treated HepG2 cells and activated hepatic stellate cells were investigated. In vitro studies were conducted to test whether knocking down PEMT in HepG2 and LX-2 cells would abolish the beneficial effects of IP6 on MASH-related phenotypes.

Results:

The results showed that pretreatment with IP6 significantly reduced obesity, fibrosis and lipid accumulation, alleviated insulin resistance, and prevented hyperglycemia in MASH mice. IP6 also alleviated free fatty acid (FFA)- induced lipid accumulation in HepG2 cells. Transcriptomics analysis of the liver revealed that IP6 reversed MASH-related transcriptomic changes, suppressing the expression of pro-fibrotic, pro-inflammatory, and lipid-metabolic genes. Integrated metabolomics and lipidomics revealed that the preventive effect of IP6 against MASH was mediated through the rectification of disordered glycerophospholipid metabolism. Notably, knockdown

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of PEMT expression abolished the inhibitory effects of IP6 on lipid accumulation, as well as α -SMA and COL-1 expression, in in vitro assays.

Conclusions:

These findings provide evidence that IP6 can prevent the development of MASH by improving disorders in glycerophospholipid metabolism, at least partially through the upregulation of PEMT.

Keyword(s) : Inositol Hexaphosphate (IP6); Inositol (INS); Metabolic dysfunction-associated steatohepatitis; Glycerophospholipid metabolism.

1847 - The Clinical Significance and Optimization Strategies of HBeAg Seroclearance in Chronic Hepatitis B Treatment

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HBeAg seroclearance represents a critical milestone in the treatment of HBeAg-positive chronic hepatitis B (CHB), which is closely associated with disease remission, functional cure, and reduced hepatocellular carcinoma risk. This review comprehensively explores the immunological mechanisms underlying HBeAg seroclearance, thoroughly examines its impact on the natural course of CHB and systematically analyzes the regulatory roles of antiviral drug efficacy, host immune status, and viral genotypes in HBeAg clearance. Integrating updated guidelines and evidence-based research, we propose optimized strategies for managing HBeAg-negative CHB, including goal setting, treatment cessation criteria, and long-term monitoring protocols. Our findings emphasize that HBeAg clearance not only signifies virological suppression but also reflects the dynamic equilibrium of host immune responses, thereby providing both theoretical foundation and practical support for achieving functional cure and reducing HCC risk in CHB patients.

Keyword(s) : Chronic hepatitis B, HBeAg, immune, Treatment, Functional cure

1850 - Curative Outcome in Hepatocellular Carcinoma A Case Report on Complete Pathologic Response After Multimodal Therapy

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Introduction:

In recent years, despite the emergence of therapeutic advances for advanced hepatocellular carcinoma (HCC), complete pathological response following systemic therapy remains uncommon. Complete remission becomes even more rare when considering the entire spectrum of disease stages, particularly in patients with underlying cirrhosis and preserved liver function who may benefit most from surgical resection. In this report, we present a case of initially unresectable HCC in a patient with liver cirrhosis that was successfully downstaged through sequential locoregional therapy and systemic immunotherapy. This multimodal treatment approach eventually enabled curative surgical resection and resulted in complete pathological response, demonstrating the potential of contemporary combination therapies to achieve exceptional outcomes even in advanced disease.

Case Description:

This is a case of a 63-year-old male patient, diagnosed with HCC Barcelona Clinic Liver Cancer (BCLC) B, Albumin-Bilirubin (ALBI) Grade 2 initially presenting with right upper abdominal pain. Triphasic CT scan showed a lobulated, heterogeneous, 10 × 11 × 10.5 cm predominantly hypodense mass located in the right hepatic lobe (segments 5 and 6) exhibiting typical enhancement on arterial phase with subsequent washout during the portovenous phase. An additional smaller lesion with similar enhancement pattern was identified in segment 7, measuring 3.9 × 4.7 × 4.7 cm. Serologic tests supported the diagnosis of HCC with elevated AFP at 7,170 mg/mL and PIVKA-II at 3,359 mAU/mL. The patient then underwent SIRT using Yttrium-90 radioembolization targeted to the right hepatic lobe. In the same interventional session, a small arterially-enhancing lesion identified in segment 3 was treated with TACE using doxorubicin and etoposide. The patient was subsequently initiated on combination immunotherapy - atezolizumab and bevacizumab. After significant tumor downsizing, patient proceeded with bisegmentectomy of segments 5 and 6 with wedge resection of the segment 7 lesion. Final histopathological examination of the resected specimens revealed complete pathologic response with no residual viable tumor and with negative margins. All quarterly surveillance imaging studies on follow-up demonstrated no evidence of new hepatic lesions or extrahepatic metastatic disease, indicating sustained disease-free status.

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Conclusion:

This case report highlights the role of integrating systemic therapy with locoregional treatments in enhancing disease control and potentially improving outcomes in patients with intermediate to advanced disease.

Keyword(s) : Hepatocellular carcinoma, HCC, locoregional therapy, systemic therapy, multimodal therapy, surgical resection, complete pathologic response

1852 - Navigating Vascular Complexity Successful Locoregional Treatment of HCC with a Hepatic Arteriportal Fistula

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BACKGROUND:

Hepatic arteriportal fistulas (HAPFs) are rare but clinically significant vascular abnormalities involving direct communication between the hepatic arterial and portal venous systems. In patients with hepatocellular carcinoma (HCC), their presence may alter hepatic perfusion dynamics, reduce the efficacy of intra-arterial therapies, and increase the risk of portal hypertension or hepatic infarction. Managing HCC in the setting of HAPF requires a nuanced approach that balances tumor control with preservation of hepatic function.

CASE PRESENTATION:

We present a case of a 77-year-old female with an incidentally detected hepatic mass, who presented with mild right upper quadrant abdominal pain. Imaging revealed a 3.0 cm arterial enhancing mass with venous washout in segment II and a 1.2 cm enhancing lesion in segment VII. Serum alpha-fetoprotein was elevated, and ultrasound-guided liver biopsy confirmed well-differentiated HCC.

MANAGEMENT AND OUTCOME

She was referred for transarterial chemoembolization (TACE) as initial therapy. Selective hepatic angiography identified tumoral blush from the middle hepatic and A2 arterial branches. Notably, early portal vein opacification suggested the presence of an arteriportal fistula. Superselective TACE was performed using doxorubicin, mitomycin, Lipiodol, alcohol, and EG-Gel until near-complete vascular stasis

was achieved. Given persistent residual enhancement, immediate adjunct microwave ablation (MWA) was performed under ultrasound and CT guidance.

Post-ablation imaging showed expected hypodense changes with air locules, consistent with coagulative necrosis. Follow-up MRI demonstrated absence of viable tumor with peripheral granulation and blood product deposition—indicative of effective tumor eradication.

CONCLUSION

This case underscores the critical role of multimodal locoregional strategies in managing complex HCC with vascular shunting. Identification of HAPF during angiography can guide therapy, as shunting may impair TACE effectiveness. The combination of superselective embolization with MWA enabled curative intent treatment while minimizing hepatic injury. For hepatologists, collaboration with interventional radiology is essential in tailoring therapy for patients with vascular variants or altered perfusion. In this setting, thermal ablation is a valuable adjunct to overcome the limitations of embolotherapy alone. This case adds to the growing evidence supporting individualized, anatomy-based strategies in advanced HCC management, particularly when complicated by arteriportal communications.

Keyword(s) : Hepatocellular carcinoma; Hepatic arteriportal fistula; Transarterial chemoembolization; Microwave ablation; Locoregional therapy

1853 - Surgical Site Infections Following Hepatectomy and Associated Risk Factors in a Tertiary Liver Surgery Center

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Background / Hypothesis: Surgical site infection (SSI) is a significant postoperative complication following hepatectomy, contributing to prolonged hospitalization, increased healthcare costs, and higher postoperative morbidity. This study aimed to evaluate the incidence of SSI after hepatectomy and identify modifiable perioperative risk factors.

Materials and Methods:

A retrospective study was conducted on 372 adult patients who underwent hepatectomy at a tertiary



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hepatobiliary center in Vietnam during 2024. Patients were categorized into laparoscopic hepatectomy and open hepatectomy groups. Demographics, comorbidities, operative data, drain use, and postoperative transfusion were collected. SSI was defined using Centers for Disease Control 2021 criteria. Statistical analyses included Chi-square testing and logistic regression.

Results:The SSI incidence was 3.5 percent (13 of 372). Laparoscopic hepatectomy showed a lower SSI rate compared with open hepatectomy (1.9 percent versus 5.7 percent). Independent predictors of SSI included open surgery, postoperative transfusion, and drain placement. Patients with SSI had longer hospitalization (18.9 vs. 7.6 days). Most SSIs were superficial, with fewer deep or organ-space infections.

Conclusions:Laparoscopic hepatectomy reduces SSI incidence and improves recovery compared with open hepatectomy. Drain placement and transfusion were significant modifiable risk factors. Enhanced perioperative strategies focusing on selective drain usage and transfusion stewardship are recommended.

Keyword(s) : hepatectomy, surgical site infection, minimally invasive surgery, postoperative outcomes, liver surgery

1854 - Cross Border Collaboration in Primary Sclerosing Cholangitis A Landmark Living Donor Liver Transplant by Makati Medical Center (Philippines) and Asan Medical Center (Korea)
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Introduction: Primary sclerosing cholangitis (PSC) remains one of the leading indications for liver transplantation. This case report features the first case of living donor liver transplantation at Makati Medical Center (MMC), performed in partnership with Asan Medical Center (AMC), highlighting the vital role of international collaboration and physician mentorship, enabling knowledge and skill transfer in complex medical and surgical procedures. The primary aim of this report is to illustrate how cross-border institutional collaboration and capacity-building can facilitate safe

implementation of high-complexity living-donor liver transplantation (LDLT) for PSC in resource-limited settings.

Case Presentation: We present a successful right-lobe LDLT in a 23-year-old male PSC and recurrent bacterial cholangitis, with his biological mother serving as the donor. Recovery for the donor was uncomplicated. On the other hand, recovery for the recipient was complicated by sepsis. After antibiotic treatment, he was able to recover well and at nearly six months post-transplant, he demonstrated preserved graft function and progressive return to baseline activities. The partnership between AMC and MMC began in 2023 with the signing of a memorandum of understanding for medical collaboration. It provides structured and comprehensive training program to enable the latter to perform LDLT independently.

Conclusion: Despite the high burden of end-stage liver disease in the Philippines, LDLT remains challenging due to limited institutional experience. Regional collaborations with neighboring countries are thus critical to augment capacity-building, cultivate technical and clinical proficiency to propel the country towards a new era of transplant medicine in the country.

Keyword(s) : Primary sclerosing cholangitis, living-donor liver transplantation, medical collaboration, transplant program development

1855 - Genotype Phenotype Correlation of ATP7B Mutations in Wilson Disease
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Background / Hypothesis:

Wilson disease (WD) is an autosomal recessive disorder of copper metabolism caused by mutations in the ATP7B gene. Patients exhibit significant variability in age of onset, affected organs, and disease severity. Clarifying the relationship between genotype and phenotype is essential for understanding WD heterogeneity.

Materials and Methods:
We performed ATP7B gene sequencing in 289 WD patients recruited from two centers in China, analyzed

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mutation types and homozygous/heterozygous distributions, and selected the five most frequent mutation combinations for further analysis. Skewed continuous variables were compared using the Kruskal-Wallis test, and categorical data were analyzed with the chi-square or Fisher's exact test. Clinical features—including age at diagnosis, serum ceruloplasmin (CER), 24-hour urinary copper, Kayser-Fleischer (K-F) ring presence, and clinical subtype—were compared among genotype groups. Regression models were used to assess interactions between genotype and age at diagnosis on the risk of cirrhosis and liver failure. Survival curves were employed to evaluate the prognostic differences of loss-of-function (LOF) mutations across distinct age subgroups

Results:

The independent effect of genotype on cirrhosis risk approached statistical significance (LOF vs Missense mutation, $p=0.051$). Among diagnosis age < 12 years old, patients carrying two 2-LOF mutations had the shortest median survival time (2.0 years), which was significantly worse than the 0-LOF group (7.0 years, $p=0.010$). However, in patients ≥ 12 years old, no significant differences in survival rates were observed among groups, suggesting that the negative impact of LOF mutations is more pronounced in early-onset patients. Patients carrying the p.T1106M/p.R778L combination showed significantly later age at diagnosis, higher CER levels, and slightly lower urinary copper, suggesting a milder clinical phenotype. K-F ring positivity and clinical subtype differed among genotype groups. Interaction analysis revealed that the rate of age-related cirrhosis progression in the p.T1106M/p.R778L group was slower than in the p.R778L/p.R778L reference group, whereas interactions with liver failure risk were not significant.

Conclusions:

The interaction between genotype and age at diagnosis has a regulatory effect on cirrhosis risk, but shows limited impact on liver failure. Loss-of-function mutations significantly negatively affect prognosis in early-onset patients (< 12 years), though this effect is attenuated in late-onset patients. The p.T1106M/p.R778L combination can serve as a "mild-type" genetic marker, characterized by delayed disease onset, higher CER levels, and a slower progression to cirrhosis risk.

Keyword(s) :

1856 - THE VALUE OF ALBI AND ALBI PLT SCORE IN PREDICTING ESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS

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Background: Liver cirrhosis is a debilitating disease that often results in fatal complications, with esophageal variceal bleeding (EVB) being the most common. The issue of early detection and treatment of esophageal varices should be addressed. Recent studies have shown the role of ALBI and ALBI-PLT score in predicting esophageal varices. Objective: The aim of this study was to describe the clinical and laboratory characteristics of liver cirrhosis patients with esophageal varices and to evaluate the accuracy of the ALBI and ALBI-PLT score in predicting the presence of esophageal varices in these patients.

Materials and Methods: We conducted a cross-sectional study that enrolled 152 cirrhotic patients from November 2024 to May 2025 at the Gastroenterology Department of Cho Ray Hospital.

Results: The study population consisted of 152 cirrhosis patients, with 100 males and 52 females, ranging in age from 28 to 87 years (mean age of 56 years). The prevalence of portal hypertension in cirrhotic patients is 56%. The ALBI score has moderate predictive ability for the risk of having portal hypertension (AUC = 0.78), as well as for the risk of high-grade portal hypertension (AUC = 0.79). The ALBI-PLT score also demonstrates moderate predictive ability for the risk of having portal hypertension (AUC = 0.76) and high-grade portal hypertension (AUC = 0.77). The cutoff threshold for the ALBI score in predicting portal hypertension in cirrhotic patients is -2.76, with sensitivities and specificities in predicting portal hypertension being 96.5% and 90.3%, respectively. The negative predictive value is 20.4%, and the rate of missing high-grade portal hypertension is 9.7%. For the ALBI- PLT score, the cutoff threshold in predicting portal hypertension in cirrhotic patients is 2, with sensitivities and specificities in predicting portal hypertension being 96.5% and 87%, respectively. The negative predictive value is 15.8%, and the rate of missing high-grade portal hypertension is 4.8%.

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Conclusion: The results of our study demonstrate that the ALBI and ALBI-PLT score is a reliable predictor of esophageal varices in patients with liver cirrhosis.

Keyword(s) : Liver cirrhosis, esophageal varices, ALBI score, ALBI-PLT score

1858 - Analysis of risk factors for portal hypertension in early histological primary biliary cholangitis

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Background / Hypothesis:

Primary biliary cholangitis (PBC) is a chronic progressive intrahepatic cholestatic disease. Its pathogenesis has not been fully elucidated and may be related to immune disorders caused by genetic factors and their interaction with environmental factors.

Materials and Methods:

A retrospective study was conducted on 110 patients diagnosed with histological early (stage I , II) PBC who underwent liver biopsy at Beijing Ditan Hospital from January 2020 to December 2023. Patients were divided into the portal hypertension group (n=27) and the control group (n=83) based on the presence of portal hypertension. Clinical data were compared between the two groups, and logistic regression analysis was performed to identify independent risk factors for portal hypertension in histological early PBC.

Results:

The most common symptom in both groups was fatigue. Abdominal distension and poor appetite were more common in the portal hypertension group (22.3% vs 8.4%) while itching was more common in the control group (18.1% vs 11.1%), but the differences were not statistically significant. The portal hypertension group had a higher proportion of patients with drug allergies and concomitant extrahepatic autoimmune diseases compared to the control group (29.6% vs 8.4%, $P<0.05$; 29.6% vs 13.3%, $P>0.05$, respectively), with no statistical significance in the latter. More patients in the portal hypertension group were histologically classified as stage II (63%) compared to the control group (39.8%), with a statistically significant difference. White blood cell count, hemoglobin, and platelet count were

lower in the portal hypertension group ($P<0.05$). Both groups showed elevated levels of gamma-glutamyl transferase, alkaline phosphatase, and total bile acid. Total bilirubin, alkaline phosphatase, and total bile acid were higher, while albumin, cholinesterase, and low-density lipoprotein were lower in the portal hypertension group, with statistical significance. The portal vein main trunk width and liver stiffness measurement were greater in the portal hypertension group ($P<0.05$). Drug allergy history and portal vein main trunk width were identified as independent risk factors for portal hypertension in histological early PBC.

Conclusions:

Portal hypertension occurs more frequently in patients with histological stage II PBC, with poorer liver function (higher TBIL, ALP, TBA, lower albumin, cholinesterase, and LDL) compared to the control group. Drug allergy history and portal vein main trunk width are independent risk factors for portal hypertension in histological early PBC, and there is no association between autoimmune antibodies and portal hypertension.

Keyword(s) : Primary biliary cholangitis; Early histological stage ; Portal hypertension

1859 - Unmasking Hepatic Tuberculosis A Case Series on Its Hepatobiliary Mimicry Diagnostic Hurdles and Management Challenges

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BACKGROUND:

Hepatic tuberculosis (HTB) is a rare extrapulmonary manifestation of Mycobacterium tuberculosis, often misdiagnosed due to its nonspecific presentation and overlapping features with hepatic malignancy, cirrhosis, or cholestatic disease. Despite being endemic in countries like the Philippines, HTB continues to pose diagnostic and therapeutic dilemmas in clinical hepatology.

CASE SUMMARIES:

We present three distinct cases of hepatic TB illustrating the protean nature of the disease and the importance of histologic confirmation.

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Case 1: A 57-year-old male presented with progressive icterus, ascites, and peripheral edema. Imaging revealed a heterogenous hepatic mass with coarse calcifications. Liver biopsy confirmed *Mycobacterium tuberculosis* via GeneXpert and culture. Anti-TB treatment was withheld due to underlying cardiac dysfunction and renal compromise. Despite supportive care and empiric antibiotics for concurrent pneumonia, the patient expired from septic shock.

Case 2: A 47-year-old female, previously diagnosed with HTB, presented with fever and signs of cholangitis. She had developed severe adverse reactions to both first- and second-line anti-TB regimens and discontinued treatment after three months. Abdominal ultrasound revealed liver cirrhosis with coarse calcifications and hypoechoic nodules. She was managed with intravenous ertapenem for ESBL *E. coli* bacteremia, supportive hepatoprotective therapy, and was referred to the Tropical Disease Foundation (TDF) for further evaluation and individualized anti-TB management. Liver enzymes normalized after three weeks of inpatient care.

Case 3: A 32-year-old male experienced recurrent episodes of fever, jaundice, and tea-colored urine for three years. Imaging showed intrahepatic bile duct dilatation with narrowing of the common hepatic duct. Autoimmune and neoplastic workup was negative. Liver biopsy demonstrated granulomatous inflammation with necrosis and calcifications. QuantiFERON-TB Gold was positive. The patient was started on a 12-month anti-TB regimen, resulting in complete symptom resolution and normalization of liver function tests.

CONCLUSION

These cases emphasize that hepatic TB should be a key differential diagnosis in patients presenting with atypical liver lesions, biliary strictures, or cryptogenic cirrhosis—especially in endemic areas. Imaging alone is insufficient, and biopsy remains critical for diagnosis. Clinical vigilance, multidisciplinary input, and individualized therapy—particularly in drug-intolerant or systemically ill patients—are essential to improving outcomes and avoiding misdiagnosis or delayed treatment.

Keyword(s) : Hepatic tuberculosis, Extrapulmonary tuberculosis, Granulomatous hepatitis, Drug-induced liver injury, Anti-TB therapy

1860 - The Great Imitator in the Liver A Case of Syphilitic Hepatitis Presenting as Cholestatic Jaundice

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Background / Hypothesis:

Syphilitic hepatitis is an uncommon hepatic manifestation of *Treponema pallidum* infection, occurring in approximately 0.2–3% of patients with secondary syphilis. It is characterized by mild cholestatic liver injury but can become fulminant in very rare cases. The condition remains under-recognized due to its rarity and variable presentation.

Materials and Methods:

A 37-year-old male presented with painless jaundice. He denied fever, abdominal pain, pruritus, and weight loss. He smoked vape but denied consumption of any medication, drugs, herbal supplements or alcohol overuse. He had no significant personal or family history, besides history of unprotected sexual relations. Physical exam revealed generalized jaundice, pedal edema, and a maculopapular rash over the cervical area, palms and lower extremities. Liver edge was non-palpable and nontender on palpation. No other stigmata of hepatic disease were present. Laboratory tests showed elevation of liver enzymes [aspartate aminotransferase (AST) 41 U/L, alkaline phosphatase (ALP) 860 U/L, and gamma-glutamyl transferase (GGT) 453 U/L] and hyperbilirubinemia (total bilirubin 11.6 mg/dL, direct bilirubin 10.3 mg/dL). Viral hepatitis panel, leptospirosis, dengue serologies and autoimmune hepatitis workup were all negative. Rapid plasma reagin test was reactive. Magnetic resonance cholangiopancreatography (MRCP) demonstrated cholelithiasis with acute calculous cholecystitis but no biliary obstruction. The constellation of cholestatic jaundice, systemic rash, and positive syphilis serology led to a diagnosis of syphilitic hepatitis in secondary syphilis. The patient was treated with a single dose of intramuscular benzathine penicillin G 2.4 million units. Repeat laboratory tests showed improvement of liver enzymes and hyperbilirubinemia.

Results:

Syphilitic hepatitis, also known as luetic jaundice, is a rare manifestation caused by spirochete dissemination to the liver, thereby inducing pericholangiolar inflammation and granulomatous cholestasis. Liver dysfunction does not usually lead to jaundice but it is common to find a disproportional increase in ALP and

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transaminases. The proposed diagnostic criteria include: (1) abnormal liver enzymes with cholestatic predominance, (2) serologic evidence of syphilis, (3) exclusion of other etiologies, and (4) improvement in liver enzyme after appropriate therapy. This case highlights the diagnostic challenge, as it can mimic biliary obstruction or autoimmune cholestatic disease.

Conclusions:

Syphilis is a venereal disease with multi-organ involvement hence described as 'The Great Imitator'. Clinicians should consider syphilitic hepatitis in cases of unexplained cholestatic jaundice, particularly when accompanied by systemic rash and unsafe sexual exposure. Early recognition is essential, as antibiotic therapy is simple, effective, and curative, preventing progression of systemic syphilis.

Keyword(s) : Syphilitic hepatitis; Secondary syphilis; Cholestatic jaundice; Treponema pallidum

1862 - Radiomics Based Skeletal Muscle Model at L3 for Predicting 90 Day Mortality in Acute on Chronic Liver Failure Superior to MELD Score

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) is a severe disease with high mortality, and accurate prognostic assessment is essential. Sarcopenia is associated with poor outcomes, and computed tomography (CT) at the third lumbar vertebra (L3) can quantify skeletal muscle mass. Radiomics allows extraction of quantitative features from these images, enabling deeper analysis of muscle-related prognostic factors.

Objective: This study aimed to develop a Radiomics Prognostic Model (RPM) combining L3 skeletal muscle radiomic features with clinical factors to predict 90-day mortality in ACLF patients.

Materials and Methods:

A total of 380 ACLF patients were enrolled and divided

into a training cohort (n=229) and a testing cohort (n=151). Radiomic features from L3-level skeletal muscle CT images were extracted, and a radiomics score was constructed using LASSO regression. Multivariate Cox regression combined the radiomics score with clinical risk factors to build a nomogram-based predictive model. Model performance and clinical utility were evaluated with calibration curves, receiver operating characteristic (ROC) analysis, and decision curve analysis.

Results:

The RPM included the radiomics score, total bilirubin, hepatic encephalopathy, INR, and creatinine as independent predictors. The model achieved AUCs of 0.80 (95% CI: 0.74–0.86) in the training cohort and 0.81 (95% CI: 0.74–0.88) in the testing cohort, significantly outperforming MELD and MELD-Na scores ($P < 0.05$). Decision curve analysis demonstrated that the RPM provided superior clinical net benefit.

Conclusions:

The RPM effectively predicts 90-day mortality in ACLF patients, integrating skeletal muscle radiomics with clinical factors, and offers a practical tool for individualized prognostic assessment.

Keyword(s) : Acute-on-chronic liver failure, Prognosis, Sarcopenia, Radiomics, Nomogram, Predictive model

1863 - Differentiating acute onset autoimmune hepatitis from drug induced autoimmune like hepatitis a multicenter study and score development

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Background: The incidence of acute-onset autoimmune hepatitis (A-AIH) is rising, but its diagnosis remains challenging, particularly in patients with a history of

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hepatotoxic drug exposure. The clinical presentations of A-AIH and drug-induced autoimmune-like hepatitis (DI-ALH) at onset are often indistinguishable, complicating their timely diagnosis.

Methods: This three-center retrospective study analyzed 238 cases of acute liver injury with autoimmune features following potential hepatotoxic drug exposure. Among them, 94 cases were diagnosed as A-AIH and 144 as DI-ALH. A comprehensive comparative analysis was conducted, focusing on clinical presentations, immunological characteristics, and histological features. The performance of diagnostic scoring systems for AIH and drug-induced liver injury in distinguishing the two conditions was evaluated. A multivariable logistic regression model was developed and independently validated to improve differentiation between A-AIH and DI-ALH.

Results: Compared to DI-ALH, A-AIH was characterized by higher immunoglobulin G (IgG) levels, reduced platelet counts, and elevated autoantibody titers. Histologically, A-AIH showed more severe features, including portal inflammation, interface hepatitis, fibrosis, and rosette formation. The 2022 AIH histological criteria demonstrated higher sensitivity but lower specificity than the 2008 criteria, with limited capacity to differentiate A-AIH from DI-ALH (AUC 0.62 vs. 0.59, $p = 0.616$). A novel diagnostic model, the Dx-AID score, which incorporates platelet count, IgG, autoantibodies, and histological features, showed high diagnostic accuracy in both the derivation cohort (AUC 0.84, 95% CI 0.77-0.91) and the validation cohort (AUC 0.83, 95% CI 0.72-0.94).

Conclusions: A-AIH and DI-ALH share many clinical similarities, but they differ in immunological and histological profiles. The Dx-AID score effectively integrates these key differences, providing a clinically actionable tool for early differentiation and guiding personalized treatment decisions.

Keyword(s) : drug-induced autoimmune-like hepatitis, autoimmune hepatitis, drug-induced liver injury, acute-onset

1864 - HONEYCOMBED LIVER HEPATIC MELIOIDOSIS A RARE MIMICKER OF PYOGENIC ABSCESS IN A NON EXPOSED DIABETIC PATIENT

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BACKGROUND

Melioidosis is a potentially life-threatening infection caused by *Burkholderia pseudomallei*, a Gram-negative bacterium endemic to Southeast Asia and northern Australia. Hepatic involvement is rare and often misdiagnosed as pyogenic liver abscess, particularly in regions like the Philippines where awareness remains limited. The infection is typically associated with risk factors such as diabetes mellitus and environmental or occupational exposure to contaminated soil or water.

CASE PRESENTATION

We report a case of hepatic melioidosis in a 44-year-old Filipino male with newly diagnosed type 2 diabetes mellitus and no known environmental or occupational exposure. He presented with two weeks of low-grade fever and dry cough but no abdominal symptoms. Laboratory evaluation showed anemia (Hb 9.4 g/dL), leukocytosis ($15.1 \times 10^3/\mu\text{L}$), elevated alkaline phosphatase, gamma-glutamyl transferase, and total bilirubin. Contrast-enhanced abdominal CT revealed multiple hepatic abscesses, the largest measuring 5.8 cm.

MANAGEMENT AND OUTCOME

Empiric antimicrobial therapy was initiated with metronidazole and ertapenem, and image-guided percutaneous drainage was performed. Intraoperatively, thick, grayish, blood-tinged pus was aspirated from honeycomb-like, multiseptated abscesses. Culture of the drained fluid yielded *Burkholderia pseudomallei*, prompting a shift to meropenem, followed by intravenous ceftazidime for two weeks. The patient was discharged on oral cotrimoxazole for 12 weeks.

Serial liver ultrasound studies performed on November 5, November 18, 2024, and February 8, 2025, showed progressive resolution of the hepatic abscesses, with eventual transformation into small, avascular hypoechoic foci. The patient completed the antibiotic regimen without complications and recovered clinically and biochemically, with normalization of liver enzymes and inflammatory markers.

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CONCLUSION:

This case underscores the importance of considering melioidosis in the differential diagnosis of hepatic abscesses, particularly in diabetic patients—even in the absence of classical exposure risks. A high index of suspicion, culture confirmation, and timely initiation of targeted antimicrobial therapy are critical for favorable outcomes. The presence of solid, multiseptated liver abscesses should raise suspicion for melioidosis over common pyogenic etiologies. Prolonged antibiotic therapy, combined with percutaneous drainage and close radiologic monitoring, remains the cornerstone of treatment. Awareness of this rare manifestation is essential for early diagnosis and prevention of complications in endemic settings.

Keyword(s) : Hepatic melioidosis Burkholderia pseudomallei Liver abscess Diabetes mellitus

1865 - Biliary Mass Mimicking Cholangiocarcinoma as a presentation of Hepatobiliary Tuberculosis in a Young and Immunocompetent patient.

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Biliary involvement in tuberculosis is uncommon and, like its other manifestations, it may closely resemble malignant processes, posing significant diagnostic challenges for physicians. Obstructive jaundice from tuberculosis usually results from extrinsic compression by enlarged lymph nodes or strictures caused by infection of the biliary epithelium. In rare cases, it can appear as mass-like lesions.

We present the case of a 30-year-old male who developed obstructive jaundice and was initially misdiagnosed with cholangiocarcinoma based on a triphasic liver CT scan showing intrahepatic ductal ectasia with a heterogeneously enhancing mass in the right posterior ductal branch. A multidisciplinary team of hepatobiliary surgeons, hepatologists, and radiologists met to determine the best management strategy. To confirm tuberculosis, the patient underwent several diagnostic tests, including a bile duct biopsy and PCR testing; however, neither test confirmed tuberculosis, so the patient was empirically treated. Given the clinical presentation and imaging findings suggestive of tuberculosis, the patient began a 9-month course of antituberculous therapy. Following antituberculous therapy, the patient demonstrated clinical recovery, and imaging showed

regression of the pseudotumor. This case highlights the necessity of including tuberculosis in the differential diagnosis of obstructive jaundice, especially in regions where tuberculosis is endemic.

Keyword(s) : Hepatobiliary Tuberculosis, Biliary Tract Mass, Obstructive Jaundice, Anti-tuberculous therapy

1866 - Real world evidence on albumin use in the management of patients with cirrhosis and ascites during paracentesis in Taiwan

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Background / Hypothesis:

Administration of human albumin (HA) at a dose of 6–8 g per liter of ascitic fluid removed is recommended following paracentesis by the APASL international clinical guidelines. Moreover, in cases of large-volume paracentesis (LVP), HA is strongly advised to prevent paracentesis-induced circulatory dysfunction (PICD). This study aims to examine current treatment practices for patients with cirrhosis and ascites undergoing paracentesis in Taiwan.

Materials and Methods:

Adult patients with a catastrophic illness certificate for liver cirrhosis between January 2018 and August 2023, with at least one valid paracentesis record were identified from the Chang Gung Research Database (CGRD). A valid record was defined as one containing volume data, and the first paracentesis event was defined as the index date. Patients with one of the following coexisting diseases during the 1-year baseline were excluded for the analysis: hepatocellular carcinoma, liver transplantation and heart failure. Patient demographics, clinical characteristics and patterns of albumin utilization were analyzed.

Results:



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Among 1,492 adult patients with catastrophic illness certificate for liver cirrhosis, 188 patients were excluded due to the following exclusion criteria: N=148 due to hepatocellular carcinoma, N=1 due to liver transplantation and N=43 due to heart failure. A total of 184 patients with a total of 535 valid paracentesis events were identified among 1,304 eligible cirrhosis patients. 75.5% of patients were male, with a median age of 57.7 years. Mean and median time from cirrhosis diagnosis to first paracentesis was 11.0 and 4.5 months respectively (Table 1). Mean and median serum albumin levels were lower than 3 g/dL in both the albumin and non-albumin cohorts (Table 2). Overall, HA was not administered in most paracentesis procedures (Table 3). Notably, in LVP involving removal of more than 5 liters of ascitic fluid, most patients did not receive albumin, despite strong recommendations for its use in clinical guidelines.

Conclusions:

HA was significantly underutilized in Taiwan, even in guideline-recommended indications such as paracentesis exceeding 5 liters.

Keyword(s) : cirrhosis, RWE, LVP, albumin, paracentesis, human albumin, Taiwan

1868 - Phenobarbital Induced DRESS with Acute Liver Injury A Hepatology Focused Approach Using Early Immunosuppression and Hepatoprotective Support

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BACKGROUND:

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a rare but potentially life-threatening, drug-induced hypersensitivity reaction characterized by rash, fever, eosinophilia, and multiorgan involvement. Among the affected systems, hepatic injury is most common—seen in up to 80% of cases—and significantly influences clinical outcomes. Liver damage may range from asymptomatic transaminitis to fulminant hepatic failure. Early recognition and timely intervention are critical in preventing progression and ensuring full recovery.

CASE PRESENTATION:

We report a case of a 21-year-old Filipino male with

seizure disorder who developed DRESS syndrome two weeks after initiating phenobarbital. He initially presented with fever, morbilliform rash, leukocytosis, and marked eosinophilia (5278/uL). Liver enzymes were normal on admission, but on hospital day 12, he developed acute liver injury with abrupt elevations in AST (1458 U/L), ALT (1111 U/L), and total bilirubin (3.71 mg/dL), with a mixed hepatocellular-cholestatic pattern. Extensive workup ruled out viral hepatitis, CMV, EBV, and HHV. Despite absence of hepatic encephalopathy or coagulopathy, the rapid biochemical deterioration prompted early immunosuppressive therapy.

MANAGEMENT AND OUTCOME:

The patient was initiated on intravenous methylprednisolone (60 mg/day for 4 days) and cyclosporine (150 mg every 12 hours). Glycyrrhizic acid, S-adenosylmethionine, and ursodeoxycholic acid were concurrently administered as hepatoprotective agents. Empiric antibiotics (aztreonam and clindamycin) were given due to elevated procalcitonin and pulmonary symptoms, selected for their minimal hepatic metabolism. Within 48 hours, eosinophilia resolved and liver enzymes began to decline. By day 4, transaminases were halved, and bilirubin stabilized. Corticosteroids were tapered, and cyclosporine was continued for two weeks without adverse effects. The patient achieved complete biochemical recovery with no recurrence on follow-up.

CONCLUSION

This case highlights the pivotal role of liver-focused, timely intervention in DRESS-related hepatotoxicity. Early immunosuppression with short-course corticosteroids and cyclosporine—augmented by hepatoprotective therapies—facilitated rapid reversal of liver injury. Cyclosporine showed excellent tolerability and may serve as a valuable steroid-sparing agent. A multidisciplinary approach grounded in hepatology principles is essential to optimize outcomes and prevent long-term hepatic sequelae in DRESS syndrome.

Keyword(s) : DRESS syndrome; Drug-induced liver injury; Cyclosporine therapy; Immunosuppressive Therapy

1869 - Societal impact of cholestatic pruritus in patients diagnosed with primary biliary cholangitis A cross sectional survey in Japan

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Background / Hypothesis:

Cholestatic pruritus has been described as a deep, intense itch with a profound impact on quality of life. It affects 55–89% of people with primary biliary cholangitis (PBC) during the course of their disease and up to 53% report clinically significant pruritus. Furthermore, 51% of patients with PBC from Japan reported experiencing pruritus. There is a lack of evidence describing the broader societal and economic burden of pruritus in PBC, particularly regarding work productivity. This study aimed to address these gaps by assessing the societal impact of pruritus in patients with PBC and their partners/close family members in Japan.

Materials and Methods:

This study is a non-interventional, cross-sectional online survey in Japan of patients experiencing pruritus as a symptom of PBC and their partners/family members. Patients were ≥ 18 years of age and had experienced itch due to PBC in the week before completing the survey. A separate survey captured insights from partners/close family members (≥ 18 years of age) of patients with PBC pruritus.

Results:

In total, 70 patients were enrolled in the study (female: 66%, n=46; median age: 55 years), most of whom received prescription medication for pruritus (69%, n=48). Among all respondents, 66% (n=46) were employed full- or part-time. The majority of patients reported moderate-to-severe sleep disturbance (56%, n=39) and financial worries due to PBC-related itch (86%, n=60); 37% (n=26) reported a moderate-to-severe impact of itch on their ability to focus at work and on everyday activities.

Overall mean work impairment (reported on the Work Productivity and Activity Index) was 37% in the total population and 47% in patients with severe itch. Patients with moderate-to-severe sleep disturbance due to itch reported 47% work impairment. In addition, 29 partners/close family members were enrolled (female: 28%, n=8; median age: 55 years). The majority worked full time (79%, n=23) and 90% (n=26) reported

providing help due to the patient's itching. Based on responses from 24 partners/close family members, an average of 34% overall work impairment due to problems associated with their partner's/family member's itching was reported.

Conclusions:

In patients with PBC, itch and sleep disturbance had a broad impact on work/employment and everyday activities and carried a financial burden. Itch experienced by patients also negatively impacted the work of their partners/close family. Improving management of pruritus in PBC, including more effective treatment options, would reduce the burden for patients and their partners/family members, and subsequently, the broader societal impact of pruritus in Japan.

Funding: GSK (221129).

Keyword(s) : Societal impact, economic burden, cholestatic pruritus, primary biliary cholangitis, Japan

1870 - SILENT STONES SERIOUS INFECTION THE ROLE OF ASYMPTOMATIC CHOLELITHIASIS IN PYOGENIC LIVER ABSCESS

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Background:

A liver abscess is defined as a pus-filled mass in the liver that can develop from injury to the liver or an intraabdominal infection disseminated from the portal circulation. The majority of these abscesses are categorized into pyogenic or amoebic, although a minority is caused by parasites and fungi. *Klebsiella pneumoniae* is a prominent etiology of pyogenic abscess in Southeast Asia and presents as more severe than other forms of bacterial abscesses, possibly because of enhanced virulence factors of the bacteria. The usual pathophysiology for pyogenic liver abscesses is bowel content leakage and peritonitis where the bacteria travels to the liver via the portal vein and resides there. Infection can also originate in the biliary system due to the proximity of the liver to the gall bladder. Notably, 50–60% of hepatic abscesses are of biliary origin, commonly associated with cholecystitis or choledocholithiasis. However, there is limited literature documenting pyogenic liver abscess arising from isolated asymptomatic cholelithiasis without

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evidence of inflammation or biliary obstruction, making such presentations particularly uncommon.

CASE:

This is the case of a 37-year-old male with no known comorbidities who presented with a 5-day history of fever associated with watery stools and abdominal cramps. Routine fecalysis and urinalysis were unremarkable, with no ova or parasites detected. Laboratory evaluation showed leukocytosis. Whole abdominal ultrasound revealed a 7 × 7 cm liver abscess in the left lobe, along with multiple gallstones (largest measuring 1 cm) without evidence of cholecystitis or CBD stones. Ultrasound-guided drainage of the hepatic abscess was performed, yielding purulent material. Culture grew *Klebsiella pneumoniae*, confirming a pyogenic liver abscess. Tests for amoebic, tuberculous, and fungal etiologies were negative. The patient was started on intravenous Ceftriaxone, with defervescence on third hospital day. Repeat ultrasound showed significant interval reduction in abscess size. He was subsequently discharged in improved condition with oral antibiotics for completion at home. Patient decided to have elective Cholecystectomy on separate admission.

Conclusion:

This case highlights that pyogenic liver abscess may rarely arise from asymptomatic cholelithiasis in the absence of biliary inflammation or obstruction. Early imaging, prompt drainage, and appropriate antibiotic therapy remain essential for favorable outcomes. Clinicians should consider biliary pathology in atypical presentations to facilitate early intervention and optimize patient outcomes.

Keyword(s) :

1871 - ORAL SEMAGLUTIDE FOR WEIGHT MANAGEMENT AND GASTROINTESTINAL SAFETY IN METABOLIC DISEASE; A META ANALYSIS AND NARRATIVE LIVER OUTCOME SYNTHESIS

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BACKGROUND:

Oral semaglutide is the first and only oral glucagon-like peptide-1 receptor agonist (GLP-1 RA), offering a non-injectable therapeutic option for type 2 diabetes (T2D), obesity, and metabolic dysfunction associated

steatotic liver disease (MASLD). Although individual trials have demonstrated its efficacy and safety, existing reviews have combined oral and subcutaneous (SC) formulations, limiting formulation-specific insights. To date, no meta-analysis has focused exclusively on oral semaglutide. A dedicated synthesis is needed to clarify its weight-loss efficacy, gastrointestinal (GI) tolerability, and potential hepatic benefits, particularly as oral GLP-1 therapies gain prominence in metabolic care.

OBJECTIVE:

To evaluate the efficacy of oral semaglutide for achieving clinically meaningful weight reduction and to assess its GI safety profile through meta-analysis, complemented by a narrative review of liver-related outcomes.

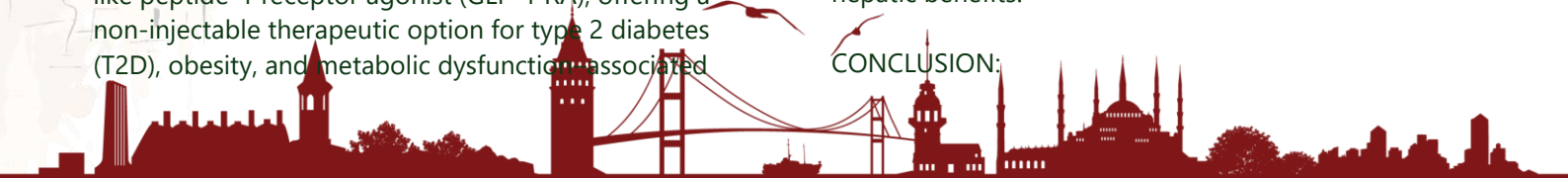
METHODS:

Following PRISMA 2020 guidelines, a comprehensive search of PubMed, Embase, Cochrane CENTRAL, Web of Science, Scopus, ClinicalTrials.gov, and WHO ICTRP was conducted up to 20 August 2025. Eligible studies included randomized controlled trials (RCTs) and prospective interventional studies in adults (≥18 years) with T2D, obesity, or MASLD who received oral semaglutide 14–50 mg daily for at least 12 weeks. The primary endpoint was achievement of ≥5% weight loss. Secondary endpoints included GI adverse events and treatment discontinuation. Hepatic outcomes—changes in aminotransferases, controlled attenuation parameter (CAP), fibrosis scores, and elastography—were narratively synthesized. Random-effects models generated pooled risk ratios (RRs) with corresponding 95% confidence intervals (CIs).

RESULTS:

Five studies met inclusion criteria (four RCTs and one pilot study), enrolling over 2,000 participants. Meta-analysis of OASIS-1, OASIS-2, and PIONEER-1 demonstrated that oral semaglutide markedly increased the likelihood of achieving ≥5% weight loss (RR 5.46, 95% CI 4.37–6.83; $I^2 = 0\%$). However, GI adverse events were significantly more frequent with oral semaglutide (RR 1.95, 95% CI 1.66–2.28), and treatment discontinuations were higher (RR 3.00, 95% CI 1.59–5.67), though overall rates remained modest. The MASLD pilot trial showed improvements in ALT, AST, CAP, and FIB-4, but no meaningful reduction in liver stiffness, suggesting potential but still inconclusive hepatic benefits.

CONCLUSION:



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Oral semaglutide produces substantial and clinically meaningful weight loss, accompanied by increased but generally manageable GI adverse events. Its discontinuation rate remains acceptable within the context of GLP-1 RA therapy. Preliminary evidence suggests metabolic and hepatic improvements, but larger, dedicated RCTs are needed to validate liver-specific outcomes. As the first meta-analysis focused exclusively on oral semaglutide, this study provides timely and formulation-specific evidence to guide clinical decisions in metabolic disease management.

Keyword(s) : Oral semaglutide, Weight reduction, Gastrointestinal adverse events, GLP-1 receptor agonist, Metabolic disease, MASLD

1872 - Circulating C1Q+ monocytes associate with lower HBsAg level in chronic HBV infection

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Background / Hypothesis:

Peripheral myeloid cells, including monocytes, dendritic cells, and macrophages, are key innate immune effectors in viral infections, migrating to infected tissues to phagocytose viruses and secrete pro-inflammatory cytokines like TNF- α . However, they can also serve as viral reservoirs, promoting immune evasion or chronic persistence in chronic HBV infection. Low serum HBsAg levels predict functional cure, but the association with circulating monocyte subsets remains unknown.

Materials and Methods:

We analyzed single-cell transcriptomics from PBMC of 20 chronic HBV patients (9 high HBsAg [920-12447 IU/mL], 11 low HBsAg [1-100 IU/mL]) and paired liver FNA from 8. Data (GSE247322) were integrated with Seurat3, clustered using Louvain (resolution 0.5, top 2000 HVGs, 30 PCs), and annotated manually. DEGs were identified by Wilcoxon test (FDR <0.001, |log2FC| >1). CellPhoneDB and SCIPAC assessed communication and phenotype association. Bulk RNA-seq (GSE173897) validated findings using Fisher's exact test (reference 20,000 genes).

Results:

To elucidate the transcriptional signatures linked to HBsAg-mediated immune modulation in chronic HBV infection, we first quantified upregulated and downregulated differentially expressed genes (up-DEGs) in peripheral blood mononuclear cells and liver

fine-needle aspiration biopsies, contrasting high versus low serum HBsAg levels. Blood up-DEGs (2935, high vs low HBsAg) and liver up-DEGs (849) overlapped in 513 genes ($p = 6.64 \times 10^{-213}$, OR 9.94, 59% liver overlap); down-DEGs overlapped in 137 ($p = 2.02 \times 10^{-34}$, OR 4.22, 32% liver overlap). The overlapping up-DEGs were predominantly enriched in CD33+ myeloid subsets, including IL-1 β + monocytes, SLC8A1+ monocytes, ISG+ monocytes (IFI27, IFI30, SIGLEC1), C1Q+ monocytes (C1QA, VMO1), CD302+ monocytes, cDCs (CD1c, FCER1A), and pDCs (IL3RA). AUC score of overlapping genes was highest in C1Q+ monocytes (mean AUC 0.177; high HBsAg group 0.186, low 0.168). SCIPAC analysis on GSE173897 (97 patients) showed SLC8A1+ monocytes correlated with viral inactive phenotype (HBV DNA <2,000 IU/mL; $p = 6.98 \times 10^{-4}$, $\lambda = -2.19$). CellPhoneDB analysis revealed C1Q+ monocytes interact with ISG+ monocytes via HLA-E-VSIR (enhanced in low HBsAg group), cDCs, CD302+ monocytes, and other C1Q+ monocytes via CD44-TYROBP, CD52-SIGLEC10, CD55-ADGRE5 interaction pairs. Among the 51 DEGs in C1Q+ monocytes, 7 complement-linked (FCN1, CLEC7A, NAIP, CASP3, CD36, NFKBIA, MYH9) genes were significantly upregulated in low HBsAg group.

Conclusions:

Circulating C1Q+ monocytes associate with lower HBsAg in chronic HBV infection, potentially mediated by upregulated membrane-bound complement regulators CD55 and CD97, the exact mechanism of which requires further study.

Keyword(s) : monocytes, HBsAg, HBV

1873 - The Clinical Epidemiology and Treatment of Acute Hepatitis C Caused by Unsafe Clinical Practices

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Background / Hypothesis:

Compared to chronic hepatitis C, acute hepatitis C (AHC) is more difficult to detect and therefore less likely to be treated in a timely manner. This study reports the clinical epidemiological characteristics, treatment, and outcomes of AHC caused by unsafe clinical practices.

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Materials and Methods:

From 2023 to 2024, an outbreak of hepatitis C caused by unsafe clinical practices occurred at a clinic in Xuchang City, Henan Province, China. The local government and Center for Disease Control and Prevention screened 630 individuals. Through comprehensive epidemiological and virological assessments, 72 adolescents and adults aged 5 years and older were identified as having AHC. Among them, 47 AHC patients agreed to receive a 12-week course of direct-acting antiviral (DAA) therapy. The criterion for evaluating treatment efficacy was sustained virological response (SVR) 12, defined as undetectable hepatitis C virus (HCV) RNA 12 weeks after completion of therapy, meanwhile, the safety profiles were closely monitored. Results:

Among the 72 patients with detectable HCV RNA, 38 (52.8%) patients were male, 71 (98.6%) patients tested positive for HCV antibodies. The mean age was 62.5 (54.0, 72.0) years, with 4 (5.6%) patients aged 5-12 years, 4 (5.6%) patients aged 13-18 years, and 64 (88.8%) patients aged over 18 years. The mean HCV RNA level was $5.60 \pm 1.64 \log_{10}$ IU/mL, genotype 1b accounted for 87.5% (63/72). Among the 37 patients with alanine aminotransferase (ALT) results, the mean ALT level was 143.0 (61.8, 378.7) U/L. 58 (80.6%) patients reported history of Injection or dental treatment within the last 6 months (Table 1). Among the 47 patients who received DAA therapy, the regimens used were as follows: Sofosbuvir/Velpatasvir in 24 patients (33.3%), Emitasvir/Sofosbuvir in 20 patients (27.8%), Ledipasvir/Sofosbuvir in 2 patients (2.8%), and Cobloipasvir/Sofosbuvir in 1 patient (1.4%). Notably, 100% (47/47) of DAA-treated patients achieved SVR 12. For the 25 untreated patients, reasons for non-treatment included financial constraints, advanced age with impaired mobility, and unspecified refusal. Within one year, 9 patients were successfully recalled. Among them, 3 patients achieved spontaneous clearance, 2 patients reached SVR12 after antiviral therapy, and 4 patients are currently undergoing antiviral treatment. The remaining patients are undergoing continued follow-up.

Conclusions:

This outbreak highlights the risks of unsafe clinical practices and the importance of early detection and treatment of AHC. DAA therapy was highly effective, with all treated patients achieving SVR12. Strengthened infection control and timely intervention are crucial to preventing similar events.

Keyword(s) : Acute hepatitis C; Outbreak; Unsafe

clinical practices ; Sustained Virological Response (SVR12)

1874 - Research on the Mechanism of LSEC Functional Abnormalities Induced by TMAO in porto sinusoidal vascular disease

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Background / Hypothesis:

Porto-sinusoidal vascular disease (PSVD) is a hepatic vascular disorder characterized by non-cirrhotic portal hypertension. The underlying pathophysiological mechanism primarily involves dysfunction of liver sinusoidal endothelial cells (LSECs) and disruption of the hepatic sinusoidal microenvironmental homeostasis. To date, the precise etiology of PSVD remains poorly understood. In our clinical cohort study on PSVD, trimethylamine N-oxide (TMAO) was identified as having significant clinical relevance. Multi-omics sequencing analyses combined with in vitro experimental validation demonstrated that TMAO induces the expression of serine hydroxymethyltransferase 1 (SHMT1), thereby contributing to LSEC injury. Notably, such an association has not been previously reported. This study aims to investigate the role and molecular mechanisms through which TMAO-induced SHMT1 expression contributes to the pathogenesis of PSVD.

Materials and Methods:

ELISA was used to confirm the elevated level of TMAO in PSVD patients. Subsequently, transcriptome and metabolome sequencing analysis identified SHMT1 as a key biomarker for TMAO-induced LSEC injury. In vitro validation was conducted using techniques such as PCR, WB, IHC, and LC-MS/MS. Functional experiments included migration, invasion, and angiogenesis assays. To explore the mechanism, SHMT1 gene silencing technology was employed to verify that TMAO induces LSEC injury by inducing SHMT1 and altering the one-carbon metabolic flux (SAM/SAH ratio increases, serine consumption rises).

Results:

1. Under electron microscopy of liver tissue in patients with PSVD, damage to the hepatic sinusoidal microenvironment can be observed: the window pores of hepatic sinusoidal endothelial cells disappear and fuse. Hepatic stellate cells are activated and collagen

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fibers increase in the Dlls space.

2. In vitro experiments have verified that TMAO can lead to LSEC damage accompanied by functional impairment. Multi-omics joint analysis (Transcriptome and Metabolome) has confirmed that SHMT1 is a key biomarker for the function of TMAO.

3. When TMAO continuously stimulated for 24 hours, the expression of SHMT1 reached its peak. Silencing SHMT1 could lead to a decrease in the SAM/SAH ratio and a weakening of folate one-carbon metabolic flux, and SHMT1 can be replenished after stimulation with TMAO.

Conclusions:

This study reveals that TMAO is involved in the injury and dysfunction of LSEC in PSVD, and verifies that this process is caused by inducing the expression of SHMT1 to activate the one-carbon metabolic pathway by folate, thereby having a certain promoting effect on the liver sinusoidal vascular injury in PSVD.

Keyword(s) : Porto-sinusoidal vascular disease (PSVD); liver sinusoidal endothelial cells (LSECs); trimethylamine N-oxide (TMAO); injury; hydroxymethyltransferase 1 (SHMT1)

1875 - Circulating NCAN as a protective biomarker and causal modifier of multistage liver disease

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Background / Hypothesis: Multistage liver diseases, from steatosis to cirrhosis and hepatocellular carcinoma (HCC), require biomarkers that capture progression and enable risk stratification. This study systematically evaluated circulating proteins to identify causal drivers across liver disease stages, with a focus on Neurocan (NCAN) as a potential cross-stage protective factor.

Materials and Methods: We integrated cis-pQTLs from eight large proteomic datasets with genome-wide association data from 13 cohorts covering alcoholic liver disease (ALD), non-alcoholic fatty liver disease (NAFLD), cirrhosis, and HCC. Mendelian randomization (MR) assessed causal effects, and cross-validation identified proteins consistently associated across stages. Bulk and single-cell RNA-seq characterized tissue distribution. NCAN dynamics and pathology

correlations were examined in a NASH-HCC mouse model. Clinical relevance was evaluated using 51,966 UK Biobank participants with proteomic profiling and a multi-center Chinese cohort (n = 202). Predictive performance was assessed alone and combined with established scores (FIB-4, LiverRisk, aMAP, mPAGE-B).

Results: MR identified 20 robust protein-disease associations, highlighting 17 circulating proteins. NCAN consistently emerged as a protective protein across NAFLD, cirrhosis, and HCC, with no evidence of reverse causation or pleiotropy. Transcriptomic analyses showed negligible hepatic expression and predominant extrahepatic origin, supporting a circulating rather than liver-derived role. In the NASH-HCC model, NCAN levels progressively decreased from steatosis to carcinoma and were strongly inversely correlated with steatosis grade, lobular inflammation, and fibrosis severity. In UK Biobank, higher circulating NCAN was associated with lower liver disease burden, reduced incident liver disease (HR = 0.82, 95% CI, 0.74-0.91, P = 2.76×10⁻⁴), and improved liver-related survival after multivariable adjustment. Addition of NCAN improved long-term prediction for both liver disease onset and mortality compared with standard clinical models. In the Chinese cohort, NCAN levels declined stepwise from healthy controls to steatotic liver disease, cirrhosis, and HCC. NCAN independently predicted significant fibrosis and HCC (AUC = 0.92 and 0.89, respectively), and integration with FIB-4 or LiverRisk further improved diagnostic performance.

Conclusions: This comprehensive proteogenomic evaluation identifies NCAN as a consistent, cross-stage protective biomarker in liver disease progression. Evidence from genetic analyses, transcriptomics, animal models, and cross-ethnic human cohorts demonstrates that circulating NCAN reflects key pathological processes and enhances prediction of fibrosis, liver disease incidence, and clinical outcomes. Incorporating NCAN into existing clinical scores substantially improves diagnostic and prognostic accuracy, underscoring its potential utility in personalized liver disease surveillance and risk assessment.

Keyword(s) : Liver disease progression; Circulating proteins; Hepatocellular carcinoma; Risk model; Mendelian randomization

1876 - Beyond the Usual Course Prolonged Cholestatic Hepatitis A in a 36 Year Old Female

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Hepatitis A virus (HAV) infection typically manifests as a self-limited, acute viral hepatitis with resolution within weeks. However, a subset of patients may present with a prolonged cholestatic course, posing diagnostic and therapeutic challenges. We report the case of a 36-year-old female with no prior comorbidities, who presented with fever, epigastric pain, postprandial vomiting, malaise, and jaundice. Laboratory evaluation revealed markedly elevated transaminases and progressive hyperbilirubinemia with cholestatic predominance. Serology confirmed acute HAV infection, while autoimmune and viral hepatitis panels were negative. Imaging studies demonstrated hepatomegaly with fatty infiltration, splenomegaly, reactive lymphadenopathy, and findings consistent with acalculous cholecystitis. Despite initial conservative management, her cholestatic symptoms and bilirubin levels persisted, necessitating corticosteroid therapy. Gradual tapering of prednisone led to clinical and biochemical improvement.

This case highlights the uncommon presentation of prolonged cholestatic hepatitis A, which may mimic other hepatobiliary disorders and complicate management, particularly when associated with reactive acalculous cholecystitis. Key diagnostic considerations include exclusion of autoimmune hepatitis, biliary obstruction, and other viral hepatitis. Early recognition of this variant can guide appropriate supportive care, timely use of ursodeoxycholic acid, and judicious corticosteroid therapy in selected patients. Clinicians should maintain vigilance for prolonged cholestasis in adults with HAV, as delayed resolution may impact quality of life and prolong hospitalization. This report underscores the importance of a comprehensive, multidisciplinary approach in managing atypical HAV presentations and contributes to the limited literature on prolonged cholestatic hepatitis A in adults.

Keyword(s) : Prolonged cholestatic hepatitis A; Acalculous cholecystitis; Corticosteroid therapy

1877 - Dynamic Increase in HBsAg during Long Term Antiviral Therapy The Role of HBV Integration and Clonal Hepatocyte Expansion

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Background / Hypothesis:The longitudinal association between dynamic changes in hepatitis B virus (HBV) integration expression and serum hepatitis B surface antigen (HBsAg) during long-term antiviral therapy remain poorly characterized.

Materials and Methods:Patients with liver biopsy-proven HBV-related fibrosis/cirrhosis who had at least one liver biopsy at baseline, week 78, or week 260 were enrolled from a multicenter cohort study. The HBsAg increase was defined by either an increase in HBsAg level ($\Delta > 0$ IU/mL) from baseline to year 4 or at least two increases between consecutive annual visits. Integrated clinical data, RNA sequencing, and histopathological profiling of a subgroup of ten patients with three serial biopsies elucidated the mechanisms driving HBsAg increase. Transcriptionally active HBV integration was identified using ChimericSeq and verified by BLAST, while intrahepatic HBsAg expression was assessed by immunohistochemistry.

Results:Overall, 37.0% (115/311) of patients met the criteria for HBsAg increase, the other were classified into HBsAg stable/decrease group. Among the patients with three serial liver biopsies, overall increasing trend in transcriptionally active integration was observed at week 260 compared with week 78. Patients in the HBsAg increase group (4/10, 40.0%) exhibited a rise in transcriptionally active HBV integration by week 260 relative to week 78, while no such increase was observed in the stable/decrease group.

Correspondingly, intrahepatic HBsAg staining displayed marginal positivity with a clustered distribution pattern that emerged at treatment baseline and persisted during anti-viral treatment. Transcriptomic analyses revealed an attenuated antiviral immune response in the increase group. **Conclusions:**HBsAg increase is linked to rising transcriptionally active integration and clustered, marginally positive HBsAg hepatocytes, suggesting that long-term treatment and immune pressure may preserve hepatocyte clones with integrated HBV.

Keyword(s) : HBV, CHB, HBV integration, HBsAg, antiviral therapy

1878 - Association between the alkaline phosphatase to albumin ratio and all cause mortality in critically ill patients with cirrhosis and



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sepsis a retrospective study

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Background / Hypothesis: Mortality rates among critically ill patients with cirrhosis are high, particularly among those with sepsis. The alkaline phosphatase-to-albumin ratio (APAR) is an emerging biomarker associated with prognostic risk across multiple diseases. We aimed to evaluate the predictive value of APAR for mortality risk in patients with cirrhosis and sepsis.

Materials and Methods: The MIMIC-IV (version 3.0) database used in this study included adult patients (≥ 18 years) with cirrhosis who were admitted to the intensive care unit. The primary endpoint was 365-day all-cause mortality, and the secondary endpoints were 90-day and 28-day mortality after admission. The hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between APAR and mortality outcomes were estimated using Cox proportional hazards models. Different predictive models were applied to assess the association between APAR and mortality in patients with cirrhosis and sepsis. In addition, subgroup analyses were conducted to evaluate the predictive value of the APAR index across different cirrhosis subpopulations.

Results: A total of 2,186 patients with cirrhosis were included from the MIMIC-IV database. These patients were divided into four groups based on their APAR values. When APAR was analyzed as a continuous variable, higher APAR levels were associated with increased 28-day (HR 1.307, 95% CI 1.226–1.393, $p < 0.001$), 90-day (HR 1.377, 95% CI 1.265–1.414, $p < 0.001$), and 365-day mortality (HR 1.355, 95% CI 1.289–1.424, $p < 0.001$). When assessed as a categorical variable, patients in the highest APAR quartile (Q4) had significantly higher 28-day (HR 2.197, 95% CI 1.796–2.687), 90-day (HR 2.368, 95% CI 1.988–2.820), and 365-day mortality (HR 2.527, 95% CI 2.155–2.963) compared with those in the lowest quartile (Q1). APAR demonstrated a linear association with all-cause mortality at 28, 90, and 365 days after admission (nonlinearity $p = 0.296, 0.740$, and 0.077 , respectively). Subgroup analyses showed that among patients with cirrhosis and comorbid hypertension, portal hypertension, malignancy, or hepatorenal syndrome, higher APAR levels were significantly associated with increased short-term mortality. Furthermore, among those with cirrhosis who had malignancy, hepatorenal syndrome, or higher SOFA and MELD scores, elevated APAR levels were significantly associated with

increased long-term mortality.

Conclusions: A higher APAR index was significantly associated with an increased risk of all-cause mortality in patients with cirrhosis. APAR may serve as a potential biomarker for assessing both short-term and long-term prognoses in critically ill patients with cirrhosis.

Keyword(s): Alkaline phosphatase to albumin ratio, Cirrhosis, All-cause mortality

1879 - PRIMARY MYELOFIBROSIS PRESENTING WITH VARICEAL BLEEDING A RARE CAUSE OF NON CIRRHOTIC PORTAL HYPERTENSION

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Background:

Primary myelofibrosis (PMF) is a chronic myeloproliferative neoplasm characterized by bone marrow fibrosis, splenomegaly, and anemia with nucleated and teardrop-shaped red blood cells. Portal hypertension is a rare but significant complication of myelofibrosis, which is characterized by increased pressure in the portal vein system. In primary myelofibrosis, portal hypertension can arise from increased portal flow due to splenomegaly and intrahepatic obstruction resulting from thrombotic obliteration of small portal veins. The development of portal hypertension can lead to the formation of varices (swollen veins) in the esophagus and stomach, which can rupture and cause bleeding.

CASE:

This is a case of a 57-year old male with known primary myelofibrosis who presented with melena. Esophagogastroduodenoscopy revealed esophageal varices Grade III. Rubber band ligation (x5) were done for the noted esophageal varices. Liver blood tests revealed alanine aminotransferase 28.46 U/L, aspartate aminotransferase 35.93 U/L, alkaline phosphatase 155 U/L, bilirubin 0.65 mg/dL, albumin 2.93g/dL, and international normalized ratio (INR) 1.37. Work-up for chronic liver disease were negative for Hepatitis serologies for past and present infection as well as for presence of hemochromatosis. CT scan of the abdomen revealed splenomegaly with prominent splenic vessels but with smooth liver margins. Abdominal ultrasound noted moderate ascites. The portal vein (1.83cm) and splenic vein (1.8cm) are both

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prominent in size on Ultrasound with color doppler with no evidence of portal or hepatic vein thrombus with noted hepatopetal portal flow. Paracentesis done revealed serum to ascites albumin gradient of 2.57 and total protein of 1g/dL. He was started on Ruxolitinib and was eventually discharged

Conclusions:

Non-cirrhotic portal hypertension is a rare but severe complication of advanced primary myelofibrosis. Its pathogenesis may involve increased splanchnic inflow and intrahepatic vascular obstruction, but the mechanisms remain poorly defined. This case highlights the need for heightened clinical awareness and further prospective studies to better characterize the prevalence, screening strategies, and management of portal hypertension in patients with myelofibrosis.

Keyword(s) :

1880 - The predictive value of different attenuation assessment tools for clinical adverse outcomes in patients with cirrhosis

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Background / Hypothesis:

We compared the predictive power of multiple frailty assessment tools in predicting adverse clinical outcomes in cirrhosis patients within one year, and provided reference for clinical prognosis assessment.

Materials and Methods:

This retrospective study analyzed 260 cirrhosis patients admitted to the Hepatology Department of Xinjiang Autonomous Region Hospital of Traditional Chinese Medicine between October 2023 and October 2024. Four frailty assessment tools—Fried Frailty Phenotype (FFP), FRAIL, Liver Frailty Index (LFI), and Clinical Frailty Scale (CFS)—were used for baseline frailty evaluation.

Patients underwent 12-month prospective follow-up to document adverse clinical outcomes. The predictive value of these scales, CTP score, and MELD score for hepatic composite adverse events was assessed using the area under the receiver operating characteristic (ROC) curve.

Results:

(1) This study enrolled 231 patients with liver cirrhosis, including 126 males (54.55%) with an average age of 56.29 ± 11.82 years and 105 females (45.45%) with an average age of 63.86 ± 11.53 years. The prevalence rates of frailty on the Fried, Frail, LFI, and CFS scales were 33.34%, 33.2%, 25.97%, and 23.2%, respectively. During the 12-month follow-up, 29 patients (12.55%) died from liver-related causes, and 94 patients (29.83%) experienced decompensation events.

(2) The Area Under the Curve (AUC) values for mortality prediction by the four scales—LFI, CFS, Frail, and Fried—were 0.771 (95% CI: 0.711, 0.832), 0.739 (95% CI: 0.675, 0.802), 0.729 (95% CI: 0.664, 0.793), and 0.704 (95% CI: 0.636, 0.772), respectively. The Delong test revealed statistically significant differences between the Fried frailty phenotype and LFI (AUC difference: 0.058; 95% CI: 0.012, 0.105; $Z=2.428$; $P=0.0152$), while no significant differences were found among the other scales (all $P>0.05$). ROC curves were plotted for the CTP score and MELD score to predict adverse clinical outcomes, with areas under the curve of 0.781 and 0.708, respectively. The CTP score demonstrated superior predictive performance due to its higher AUC value.

(3) The combined prediction performance of CTP score and LFI was the best (AUC=0.825).

Conclusions:

All four scales can predict the risk of death in 12 months in patients with cirrhosis, and LFI scale has the best predictive power.

Keyword(s) : cirrhosis; frailty assessment tools; correlation;

1881 - Serum free T3 free T4 ratio as a predisposing factor for the development of metabolic dysfunction associated steatotic liver disease a large scaled cohort study of euthyroid state

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Background / Hypothesis:

Thyroid hormones are key regulators of hepatic lipid metabolism, and recent evidence highlights the potential role of thyroid hormone sensitivity, particularly the free triiodothyronine to free thyroxine (FT3/FT4) ratio, in the pathogenesis of metabolic dysfunction-associated steatotic liver disease (MASLD) among euthyroid individuals. However, most prior studies have been cross-sectional, limiting insight into temporal or causal associations.

Materials and Methods:

A cohort of 209,729 apparently healthy men and women who were free from ultrasound-based MASLD at baseline were followed for a median of 5.7 years annually or biennially, from January 1, 2011 to December 31, 2021. Serum FT3, FT4 and thyroid stimulating hormone (TSH) were measured and FT3/FT4 ratios were calculated. Incident MASLD was defined by liver ultrasound findings and metabolic dysfunction criteria. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for MASLD incidence by quintiles of the FT3/FT4 ratio, adjusting for potential confounders.

Results:

Mean age of 209,729 participants was 35.7 years. During a mean follow-up of 5.98 years, Over 1,254,033.5 person-years of follow-up, 51,205 incident MASLD cases were identified. Higher FT3/FT4 ratios were significantly associated with increased MASLD risk in both men and women, with stronger associations in time-dependent models. In time-dependent models, multivariable-adjusted HRs (95% CIs) for MASLD incidence across increasing FT3/FT4 quintiles (Q2-Q5 vs. Q1) were 1.10 (1.06-1.15), 1.17 (1.13-1.22), 1.24 (1.19-1.28) and 1.29 (1.25-1.34), respectively, among men and 1.08 (1.02-1.14), 1.20 (1.14-1.26), 1.30 (1.24-1.37) and 1.44 (1.37-1.51) among women.

Conclusions:

In a large cohort of euthyroid men and women, higher FT3/FT4 ratios within the normal thyroid function range are independently associated with increased risk of

developing MASLD in a dose-dependent manner. The FT3/FT4 ratio may serve as a valuable biomarker for identifying individuals at higher risk of MASLD development, suggesting that thyroid hormone metabolism plays an important role in hepatic steatosis pathogenesis even in euthyroid populations.

Keyword(s) : free triiodothyronine, free thyroxine, metabolic dysfunction-associated steatotic liver disease

1882 - Safety and Effectiveness of Peginterferon Alfa 2b Combined with Tenofovir Amibufenamide in Patients with Chronic Hepatitis B

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Background / Hypothesis: Data on the combination of Peg-IFN α -2b and tenofovir amibufenamide (TMF) remain limited. This study aims to evaluate the safety and effectiveness of Peg-IFN α -2b combined with TMF in patients with CHB.

Materials and Methods: Patients treated with TMF (25mg/day) and Peg-IFN α -2b (180 μ g/week) from January 2022 to May 2025 were retrospectively enrolled from three hospitals in Henan, China. This study included two groups: the switch combination group, consisting of patients who transitioned from nucleos(t)ide analog (NA) monotherapy to a combination of TMF and Peg-IFN α -2b; and the treatment-naïve combination group, comprising patients who received TMF plus Peg-IFN α -2b as initial therapy. The primary endpoint of this study was the rate of hepatitis B surface antigen (HBsAg) loss at 48 weeks of treatment, the secondary endpoints were the rate HBV DNA undetectable (<25 IU/mL) and safety profiles during treatment.

Results: A total of 180 patients were enrolled, with 120 patients in the switch group and 60 patients in the treatment-naïve group. The treatment period of NA monotherapy in switch group was 67.45 $\pm$ 35.36 months. At baseline, the age, HBV DNA, hepatitis B e antigen (HBeAg), HBsAg, alanine aminotransferase (ALT) levels of the patients in the switch group and treatment-naïve group were 38.48 $\pm$ 0.89 vs. 37.20 $\pm$ 1.28 years ($p=0.413$), 4.1 (2.6, 6.5) vs. 4.9 (3.1, 7.8) log₁₀

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IU/mL ($p=0.169$), 255.0 (11.0, 406.6) vs. 304.9 (12.1, 459.7) PEIU/mL ($p=0.536$), 2.63 ± 1.14 vs. 2.79 ± 1.18 log₁₀ IU/mL ($p=0.370$), 34.10 ± 4.31 vs. 50.14 ± 6.62 U/L ($p=0.037$), respectively; At 48 weeks of treatment (Figure 1), the HBV DNA undetectable rates were 99.17% vs. 96.67% ($p=0.537$), the HBeAg clearance rates were 76.67% vs. 42.86% ($p=0.026$), the HBsAg loss rates were 44.16% vs 28.33% ($p=0.040$), in the switch group and treatment-naïve group, respectively. Regarding laboratory abnormalities, the main adverse events during treatment were the ALT elevation (57.50% vs. 33.33%, $p=0.002$), leukopenia (46.67% vs. 31.67%, $p=0.054$), neutropenia (45.83% vs. 28.33%, $p=0.024$), thrombocytopenia (65.83% vs. 40.00%, $p=0.001$), in the switch combination group and treatment-naïve combination group, respectively. Only one patient presented with elevated total bilirubin (39.2 μ mol/L). No serious adverse events occurred. Conclusions: At week 48 of treatment, the switch combination group exhibited significantly higher rates of HBsAg loss and HBeAg seroconversion compared to the treatment-naïve combination group. Both groups maintained a favorable overall safety profile throughout the treatment period. These findings suggest that initiating therapy with nucleos(t)ide analogs followed by combination with Peg-IFN α may be a reasonable and promising strategy to promote HBsAg loss.

Keyword(s) : Combined treatment; Chronic hepatitis B; Effectiveness; Safety; Tenofovir amibufenamide; Peg-IFN α -2b

1884 - Variceal bleeding in toddlers Is the endoscopic outcome in extrahepatic portal hypertension worse than chronic liver disease

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Background / Hypothesis:

Toddler with extrahepatic portal venous obstruction (EHPVO) may exhibit pronounced degree of portal hypertension because of abrupt and complete interruption of portal venous flow causing early and marked development of portosystemic collaterals and large varices. In contrast, chronic liver disease causes portal hypertension more gradually, in parallel with progressive fibrosis and intrahepatic resistance. Hence we aimed to assess the endoscopic outcome of bleeding toddlers with extrahepatic portovenous

obstruction (EHPVO) and Chronic liver disease (CLD).

Materials and Methods:

Endoscopic outcome of toddlers with extrahepatic portal venous obstruction (EHPVO) were compared with chronic liver disease (CLD). As a standard protocol, all bleeders underwent endoscopic sclerotherapy at 2-3 weekly sessions till primary eradication of esophageal varices (EV) followed by 3-6 monthly sessions for endoscopic surveillance. Outcome was noted at follow-up.

Results:

Of the 152 patients, 79 (n=59 EHPVO, n=20 CLD) underwent secondary prophylaxis. Etiology of CLD were operated biliary atresia (n=15), sclerosing cholangitis (n=2) and autoimmune hepatitis (n=3). Figure 1 shows the study flow diagram and comparison of endoscopic outcomes between two groups. As compared to CLD, EHPVO had earlier onset of bleeding [8(4-36) months vs. 20(8-30) months, $p=0.01$], more hypersplenism [61% vs. 25%, $p=0.007$], endoscopic sessions [9(4-15) vs. 5(4-8), $p=0.03$], total volume of sclerosant [17.5(8-53) vs. 10(4-63), $p=0.03$], recurrence of EV [34% vs. 15%, $p=0.04$] and portal hypertensive gastropathy (PHG). 6% had sclerotherapy complications (bleeding ulcers and stricture). Duration of follow-up was 36(12-120) months. 22% EHPVO required secondary eradication of EV. Overall interval bleeding from varices during primary eradication occurred in 20% within 11(7-16) days from last endoscopic session. None of the patients developed bleed from gastric varices or PHG. Using multivariate analysis, the independent predictors of recurrence of EV and secondary eradication were age of onset <9mo (OR:4.8, 95% CI:2.6-13.3, $p<0.01$), hypersplenism (OR:3.6, 95% CI:1.6-15.9, $p<0.01$) and primary gastric varices (OR:5.4, 95% CI:4.5-8.8, $p=0.03$). Non-favorable vascular anatomy for portosystemic shunting was observed (100% for Meso-Rex bypass; 64% for non-selective shunts) in EHPVO. 38% BA were referred for liver transplantation.

Conclusions:

Toddlers with EHPVO have larger varices, recurrent bleeding, and greater degree of portal hypertension, as compared to CLD. Endoscopic therapy is the mainstay since majority may not have favorable anatomy for portosystemic shunting.

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Keyword(s) : Extrahepatic portal hypertension, Chronic liver disease, Endoscopic outcome

1885 - The sweet protein brazzein in ice cream suppresses postprandial glucose elevation in patients with metabolic dysfunction associated steatotic liver disease (MASLD) and diabetes mellitus (DM).

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Introduction: MASLD and DM remain highly prevalent worldwide, and excess intake of added sucrose is a key modifiable contributor to postprandial hyperglycaemia. Rapid glycaemic excursions following sucrose-rich foods intake exacerbate metabolic stress and accelerate progression of comorbid conditions, including MASLD. Objective: To evaluate the differences in postprandial glycemia after consumption of ice cream "a"(containing brazzein, maltitol, oligofructose), ice cream "b" (containing brazzein, maltitol, erythritol, inulin) and ice cream "c" (containing sucrose) in patients with MASLD and DM.

Materials and methods: This double-blind, randomized, crossover clinical study was conducted under a registered protocol (ClinicalTrials.gov: NCT06724913). Interstitial glucose was measured using a continuous glucose monitoring system at 15-min intervals over 150 min following ingestion. Glycaemic trajectories were analysed with linear mixed-effects models including patient-specific random intercepts and adjustment for baseline glucose (time 0). Time was modelled using a cubic B-spline (df = 5) with spline(time)×ice-cream×group interactions; fixed effects included ice-cream type, patient group, period and sequence. Model-derived curves with 95% confidence intervals (CI) were generated, and pairwise time-point contrasts were FDR-corrected using the Benjamini–Hochberg procedure.

Results: A total of 101 participants were analysed (MASLD, n=57; MASLD+DM, n=44). In both groups, sucrose ice cream (type "c") produced marked postprandial glucose rises, peaking at approximately 45–65 min in the MASLD group and 80–95 min in the MASLD+DM group, followed by a decline toward

baseline by 120–140 min. By contrast, both brazzein-based products elicited substantially flatter profiles, with minimal elevation and a gradual decrease over time. In the MASLD group, glucose levels after ice cream "c" were significantly higher than after types "a" and "b" across nearly the entire 20–120-min interval, whereas glucose levels after intakes of ice cream "a" and "b" did not differ significantly. In the MASLD+DM group, differences between ice creams "c" and "a" remained significant from 45 to 145 min, and between "c" and "b" from 60 to 150 min. A modest but significant mid-phase separation between "b" and "a" (30–85 min) suggested a slightly higher blood glucose response to "b", while both remained clearly superior to ice cream "c".

Conclusion: substituting sucrose with brazzein-based sweetening systems in ice cream yields a significantly more favourable postprandial glucose profile in patients with MASLD, including those with concomitant diabetes. These findings support sweet proteins as a potential dietary strategy to reduce postprandial glycaemic load in DM with MASLD comorbidity.

Keyword(s) : brazzein, metabolic dysfunction-associated steatotic liver disease, MASLD, protein sweetener, sucrose

1887 - Dynamic changes of serum lipids metabolism in hepatitis B virus related acute on chronic liver failure

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Background / Hypothesis:

At present, the relationship between lipids metabolism and the prognosis of liver failure is a hotspot. The purpose of this study was to explore the dynamic changes of serum lipids parameters in hepatitis B virus related acute-on-chronic liver failure (HBV-ACLF).

Materials and Methods:

For patients with HBV-ACLF, to describe the dynamic changes in serum lipids parameters pretreatment and after four weeks of treatment in survival group, and that of four weeks before death. The diagnostic criteria were in accord with American Association for the study of Liver Diseases practice guidance on acute-on-chronic liver failure and the management of critically ill patients with cirrhosis in 2023. Differences between groups were made by the t-test. And $P < 0.05$ is considered statistically significant.

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Results:

From June 1, 2020 to March 1, 2025, 70 patients with HBV ACLF had enrolled in Third Affiliated Hospital of Sun Yat-sen University, including 40 patients had survived more than 12 weeks and 30 cases died within 5 weeks. Their ages were 43.38 ± 11.69 years and 46.38 ± 13.23 years respectively, and no significant difference was between the two groups ($t = -0.914$ $P = 0.364$). The parameters values and the comparisons were shown in Table 1 and 2.

HBV-ACLF could cause fluctuations and decreases in serum lipids. After 4 weeks of treatment, lipid parameters gradually recovered in survival groups. Furthermore, except for HDL-C and Lipoprotein- α , the other parameters of four weeks before death were significantly lower than those of survival group (all $P < 0.05$) (table 2).

Conclusions:

Hypolipidemia may worsen HBV-ACLF. To improve it could contribute to the recovery and prognosis of HBV-ACLF.

Keyword(s) : Lipids metabolism; Hepatitis B virus related acute-on-chronic liver failure (HBV-ACLF); dynamic changes

1888 - An Optimized In Hospital Management Model Enhances Linkage to Care for Non Hepatology HBV Infected Patients Interim Analysis of Hot Wave Project 2.0

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Background / Hypothesis:

A significant proportion of HBV-infected individuals in China remain undiagnosed or untreated. We aimed to evaluate whether an optimized in-hospital management model, emphasizing multi-disciplinary collaboration and patient education, improves linkage-to-care (LTC) and antiviral treatment (AVT) rates for HBsAg-positive patients identified in Non-hepatology departments.

Materials and Methods:

The "Hot Wave Project," initiated in 2022 at the Third

Affiliated Hospital of Sun Yat-sen University (SYSU-3AH). Key interventions included collaboration between the Department of Hepatology and various clinical and administrative departments. The measures implemented were automated lab report alerts, establishment of dedicated HBV health clinics, varied patient education programs, and big data monitoring to facilitate professional consultation and treatment for HBV-infected patients. In April 2025, the model expanded to the Fifth Affiliated Hospital of SYSU (SYSU-5AH) and the Sixth Affiliated Hospital of SYSU (SYSU-6AH) as "Hot Wave Project 2.0", featuring enhanced education plans, and call-back mechanisms. We compared data of HBsAg-positive patients from Non-hepatology departments between 2024 and January–September 2025 across these three centers.

Results:

At SYSU-3AH, 15,201 HBsAg-positive patients were identified in 2024, with 9,065 receiving AVT (59.63%) and 2,716 referred to Hepatology (17.88%). From Jan–Sep 2025, among 13,686 identified patients, the AVT rate increased to 61.38% (8,140/13,686, $p = 0.002$), and the referral rate rose significantly to 27.72% (3,797/13,686, $p < 0.0001$). At SYSU-5AH, 6,044 and 4,311 patients were screened in 2024 and Jan–Sep 2025, respectively. The AVT rate improved from 20.14% (1,217/6,044) to 22.67% (978/4,311, $p = 0.002$), while the referral rate remained stable at 16.78% (1,014/6,044) versus 17.26% (744/4,311) ($p = 0.52$). At SYSU-6AH, the AVT rate increased significantly from 21.97% (1,121/5,103) to 35.40% (1,944/5,492) ($p < 0.0001$), and the referral rate improved from 2.12% (108/5,103) to 7.28% (400/5,492) ($p < 0.0001$).

Conclusions:

The implementation of the Hot Wave Project 2.0 was associated with improved antiviral treatment uptake and referral rates for HBV patients in non-hepatology settings across multiple centers. The optimized referral system and education strategies demonstrate potential feasibility for expanding in-hospital HBV management coverage.

Keyword(s) : Hepatitis B Virus, Linkage to Care, Antiviral Treatment, In-hospital Management

1891 - Research progress on the association between metabolic dysfunction associated fatty liver disease and gallstones

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Background:

Metabolic dysfunction-associated fatty liver disease (MAFLD) and gallstones diseases (GSD) are common liver and gallbladder diseases, with highly overlapping epidemiological features and risk factors, confirming a certain correlation between the two, but with different conclusions. With the update of diagnostic criteria for MAFLD, the research dimensions of the correlation between the two continue to expand. This review integrates recent clinical evidence and mechanism research, providing reference for clinical diagnosis and future research.

Materials and Methods:

Search Pubmed and CNKI databases to include research papers on the association between MAFLD and gallstones. Exclude review literature.

Results:

Firstly, the summarized research results indicate that compared with non MAFLD patients, the incidence of GD in NAFLD patients is almost twice that of non MAFLD patients (incidence rates of 47% and 26%, respectively, $p < 0.0001$); Compared with patients without GSD, 56% of patients with gallstones had MAFLD ($p < 0.0001$). Secondly, the research mechanism suggests that the two share three core pathways: lipid metabolism disorders, insulin resistance, and chronic inflammation. The accumulation of free fatty acids leading to bile cholesterol supersaturation is a key link. Insulin resistance serves as a crucial bridge between GSD and MAFLD. The relationship between abnormal glucose metabolism and fatty liver disease is highly recognized, and fasting blood glucose levels are fully correlated with the presence of GSD in MAFLD patients. Finally, in terms of influencing factors, age increase, sex, diabetes, ALP increase, HDL decrease and FBS increase are the only predictors of biliary node in MAFLD patients. Lifestyle interventions can reduce risks. Other studies have shown that asymptomatic patients with MAFLD and non obese individuals also exhibit an increased risk of gallstones. Therefore, in clinical practice, caution should be exercised in treatment and comprehensive screening should be conducted to prevent the occurrence of the disease.

Conclusions:

There is a clear epidemiological association and common pathophysiological basis between MAFLD and gallstones. Clinical attention should be paid to

screening for gallstones in MAFLD patients, especially in high-risk subgroups. Future research should focus more on the molecular mechanisms associated with the two and explore targeted intervention measures, providing favorable references for clinical diagnosis and treatment management.

Keyword(s) : gallstone disease,nonalcoholic fatty liver disease,insulin resistance

1892 - Reactive Pericarditis Secondary to Cholangitic Abscess from a Neglected Biliary Stent A Case Report

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Endoscopic biliary stent insertion is a life-saving procedure for severe cholangitis, often due to choledocholithiasis. In resource-limited settings, high costs and limited follow-up can lead to neglected stents and increased health risks. Patients may present months or years later with severe infections, such as hepatic or cholangitic abscesses from stent failure, or with perforation from stent migration, both of which are life-threatening. Pericarditis is a rare and often fatal complication of cholangitis, typically resulting from direct penetration or abscess extension. We present a rare case of subdiaphragmatic cholangitic abscess from a neglected stent causing reactive pericarditis in an immunocompetent man.

A 45-year-old male with no comorbidities, who had previously undergone endoscopic retrograde cholangiopancreatography (ERCP) with stent insertion for large choledocholithiasis two years prior, presented with right upper quadrant pain, fever, and jaundice. Ultrasound demonstrated left intrahepatic bile duct ectasia. Clinical assessment on admission indicated cholangitis secondary to stent failure. Urgent ERCP confirmed the neglected biliary stent, prompting stent removal and reinsertion, as well as initiation of empiric broad-spectrum antibiotics. Cholangiography revealed a markedly dilated common bile duct with multiple ovoid filling defects. Bile cultures grew *Escherichia coli*. Despite these interventions and one week of antibiotic therapy, the patient developed progressive dyspnea and chest pain. Electrocardiogram and echocardiography confirmed pericarditis. Magnetic resonance cholangiopancreatography (MRCP) identified a subdiaphragmatic abscess adjacent to the

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pericardium. Percutaneous drainage of the abscess and targeted antibiotic therapy resulted in resolution of pericarditis. The patient achieved full recovery and was discharged after six weeks.

This case highlights the need for patient education and consistent post-procedural follow-up to ensure timely stent removal. Prompt source control is essential for optimal outcomes. Clinicians should remain alert to extra-biliary complications of cholangitis, such as pericarditis, particularly in patients with cardiopulmonary symptoms.

Keyword(s) : Pericarditis, Cholangitis, Neglected Biliary Stent

1894 - AI Driven Pathomics Transforming Precision Medicine Across the Chronic Liver Disease Hepatocellular Carcinoma Spectrum

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality worldwide, often arising from a progressive chronic liver disease (CLD) continuum that includes inflammation, fibrosis, and malignant transformation. Conventional histopathology, while the diagnostic gold standard, is limited by subjective interpretation, inter-observer variability, and an inability to quantitatively analyze dynamic tissue changes. The emergence of digital pathology and artificial intelligence (AI) has given rise to pathomics—a field that integrates whole-slide imaging (WSI) with deep learning to decode subvisual morphological, spatial, and molecular patterns(Fig.1).

Materials and Methods:This study systematically investigated the core methodologies and applications of AI-driven liver pathomics through a comprehensive literature review based on the PubMed database.

Results:AI-powered pathomics has demonstrated significant breakthroughs in both CLD and HCC management. In CLD, it enables automated quantitative assessment of hepatic fibrosis inflammatory activity grading in non-alcoholic steatohepatitis, and accurate identification of premalignant lesions. In HCC, AI models assist in differential diagnosis, molecular subtyping, microvascular invasion prediction , and recurrence risk stratification post-resection or transplantation. Multimodal integration further improves prognostic

accuracy and immunotherapy response prediction.

Conclusions: Despite persistent challenges related to data standardization, model transparency, and clinical translation, emerging technologies—such as spatial transcriptomics, quantum-accelerated computing, and federated learning—offer promising pathways forward. Through continued interdisciplinary collaboration and advances in explainable AI, multimodal fusion, and global data governance, AI pathomics is poised to enable proactive risk prediction, early interception, and precision therapy throughout the liver disease continuum.

Keyword(s) : artificial intelligence, pathomics, chronic liver disease, hepatocellular carcinoma, digital pathology

1896 - Xinjiang Uygur Autonomous Region Traditional Chinese Medicine Hospital

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Background / Hypothesis:

To evaluate the correlation between the severity of esophageal varices (EV) and ocular fundus indicators in patients with liver cirrhosis based on fundus images, aiming to provide a non-invasive and convenient clinical method to reduce reliance on invasive examinations.

Materials and Methods:

A total of 86 inpatients with liver cirrhosis from the Department of Liver Diseases, Traditional Chinese Medicine Hospital of Xinjiang Uygur Autonomous Region (December 2024 to September 2025) were

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enrolled. Based on endoscopic findings, patients were divided into three groups: no EV group (n=19), mild EV group (n=24), and moderate-to-severe EV group (n=43). Patients with no or mild EV were defined as the low-risk group (n=43), and those with moderate or severe EV as the high-risk group (n=43). Scanning laser ophthalmoscopy (SLO) fundus images were collected and analyzed using the EvisionAI intelligent fundus image analysis system to extract indicators (e.g., area of fundus hemorrhage, number of hemorrhages). Pearson and Spearman correlation analyses were used to assess the correlation between EV severity/risk level and fundus image indicators.

Results:

- (1) The grading of EV severity showed negative correlations with total leopard spot density, leopard spot density in the optic disc area, average vessel tortuosity in the inferior nasal region within the 6mm-9mm annular zone, the number of peripheral microaneurysms, and leopard spot density within the 10mm-15mm annular zone of the macula.
- (2) Spearman correlation analysis revealed that the high-risk EV group was positively correlated with the AVR ($p=0.21$, $P<0.05$), and negatively correlated with the average vessel tortuosity in the inferior nasal region within the 6mm-9mm annular zone ($p=-0.33$, $P<0.05$), average vessel diameter in the inferior temporal region within the 3-photoreceptor-disc-diameter (3PD) circular zone ($p=-0.22$, $P<0.05$), vessel density in the inferior nasal region within the 6mm-9mm annular zone ($p=-0.23$, $P<0.05$), number of vessels within 1cm of the optic disc ($p=-0.22$, $P<0.05$), leopard spot density within the 10mm-15mm annular zone of the macula ($p=-0.25$, $P<0.05$), and leopard spot density in the superior sector of the 10mm-15mm annular radius circle of the macula ($p=-0.28$, $P<0.05$).

Conclusions:

The high-risk group for esophageal varices in patients with liver cirrhosis is positively correlated with the arteriole-to-venule diameter ratio, suggesting that fundus images can, to some extent, reflect the risk level of esophageal varices in these patients.

Keyword(s) : Liver cirrhosis; Esophageal varices; Fundus images; Correlation ;

1898 - Successful Management of Hepatitis B Virus Reactivation Induced Acute on Chronic Liver Failure with Integrated Traditional Chinese and Western

Medicine A Case Report

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Background: Self-discontinuation of nucleos(t)ide analogue therapy is a critical risk factor for HBV reactivation and progression to acute-on-chronic liver failure (ACLF) in chronic hepatitis B patients. Despite standard supportive care, ACLF mortality remains high. This report demonstrates successful management through integrated Traditional Chinese and Western medicine.

Case Presentation: A 43-year-old male with chronic hepatitis B presented with worsening jaundice 75 days after discontinuing entecavir. Examination showed profound icterus. Laboratories revealed total bilirubin 295.2 $\mu\text{mol/L}$, PTA 17.5%, INR 2.75. Imaging demonstrated cirrhosis with ascites. Diagnosed as ACLF Grade 2 (COSSH criteria) with AARC score 7 (ACLF Grade 1), indicating poor prognosis.

Therapeutic Intervention: Western treatment included tenofovir reinstatement, comprehensive liver protection, infection control, and coagulation support. Traditional Chinese Medicine was initiated in the second month using syndrome differentiation. The treatment strategy followed the disease progression: initial stage employed modified Yinchenhao Tang (with 100g *Artemisia capillaris*) to clear dampness-heat; middle stage utilized Xijiao Dihuang Tang (with water buffalo horn) to resolve heat-toxins; advanced stage incorporated cultured cattle gallstone and bear bile powder to enhance blood-cooling and detoxification effects. This phased approach addressed the evolving pathogenesis patterns throughout the disease course. Outcome: After three months, symptoms resolved completely. Laboratories showed total bilirubin 69.7 $\mu\text{mol/L}$, PTA 42.6%, INR 1.65. The patient no longer met ACLF criteria (COSSH), with AARC score improving from 7 to 4 (non-ACLF). Notably, a significant clinical turning point occurred following the introduction of TCM decoctions, with accelerated improvement in biochemical markers and symptom resolution. This temporal association suggests that the therapeutic effect of TCM intervention extended beyond mere adjunctive support, pointing to a synergistic "1+1>2" interaction with conventional Western treatment.

Conclusion: This case highlights the crucial importance of antiviral adherence and demonstrates the substantial benefits of integrated medicine for HBV-ACLF. The sequential TCM application, meticulously guided by syndrome differentiation, provided targeted

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therapy at different disease stages. This integrative approach likely contributed to effective inflammation control and enhanced hepatic recovery through multi-targeted mechanisms, demonstrating a synergistic effect that warrants further clinical validation and protocol standardization.

Keyword(s) : Acute-on-chronic liver failure; Integrated Chinese-Western medicine; Syndrome differentiation; Case report

1900 - Prevalence of Hepatitis B and C among Adolescents and Young Adults in Sindh Pakistan A Substudy of a Provincial Population Based Survey

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Background / Hypothesis:

Globally, hepatitis B and C virus infections are recognized as the leading cause of liver-related morbidities and mortalities. Sustainable Development Goal 2030s has targeted the elimination of hepatitis B and C. Understanding early-age prevalence and coexisting metabolic risk factors is essential for designing integrated prevention and control strategies to mitigate the rising prevalence of hepatitis B and C. This analysis represents a substudy of a larger provincial population-based survey aimed to determine the prevalence of hepatitis B and C among adolescents and young adults in Sindh, Pakistan.

Materials and Methods:

This provincial cross-sectional, multistage household survey was conducted across all districts of Sindh, Pakistan. Participants included adolescents (10–19 years) and young adults (20–24 years) were enrolled. Data were collected on sociodemographic factors, diet, and physical activity, alongside anthropometric and blood pressure measurements. Each participant underwent laboratory testing for complete blood count, liver function, lipid profile, fasting glucose, and hepatitis B surface antigen (HBsAg) and hepatitis C antibody (anti-HCV). Hepatic steatosis was assessed utilizing FibroTouch elastography.

Results:

A total of 2,136 participants were surveyed, comprising 1,651 adolescents and 485 young adults from both urban and rural areas of Sindh. The sero-prevalence of hepatitis B was 1.4%, while hepatitis C was 0%. Co-

occurrence with elevated liver enzymes and metabolic abnormalities was observed, suggesting an overlapping burden of infectious and metabolic liver disease in this young population.

Conclusions:

Findings of this study highlight a measurable burden of viral hepatitis B and reassuringly low prevalence of Hep C in adolescents and young adults. The coexistence of metabolic disorders exposes new dual threats. Strengthening integrated screening, immunization, and early linkage-to-care programs is critical to maintaining progress toward national and global hepatitis eradication targets.

Keyword(s) : Hepatitis; Liver Disease; Adolescents; Young Adults

1901 - A Modular Assembly of Urine Derived Stem Cells and Hydrogel into a Therapeutic Ectopic Mini Liver for Wilson's Disease

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Background / Hypothesis: Wilson's disease (WD) is a disorder of copper metabolism caused by ATP7B gene mutations. Urine-derived stem cells (USCs) offer a novel cell source for therapy, but their therapeutic potential is limited by poor survival after transplantation. This study explores a strategy using a hydrogel system to form an ectopic mini-liver, leveraging the hydrogel's 3D microenvironment to boost USC viability and efficacy for the treatment of WD.

Materials and Methods: USCs were isolated from a WD patient with a homozygous ATP7B R778L mutation and a healthy donor using repeated centrifugation. Cells were cultured in an optimized medium and characterized by flow cytometry for surface markers (CD44, CD73, CD90, CD105 positive; CD19, CD34, CD45, CD11b, HLA-DR negative) and immunofluorescence for ATP7B. For the therapeutic experiment, ATP7B knockout WD model mice were

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randomized into three groups: normal control, disease model, and USC-treated group. The treatment group received four intraperitoneal injections of WD-USCs (2×10^6 cells per injection in 200 μ L volume, pre-mixed with hydrogel at a 1:1 ratio) over 14 days.

Comprehensive endpoint assessments included serum copper and ceruloplasmin levels, liver function and antioxidant assays, histopathological evaluation (H&E, Oil Red O, Masson, Sirius Red), transmission electron microscopy (TEM), and integrated transcriptomic and targeted metabolomic analyses.

Results: WD-USCs exhibited enhanced proliferative capacity and higher expression of mesenchymal markers (CD44, CD73, CD90, CD105) compared to healthy USCs. Transplantation of hydrogel-encapsulated WD-USCs significantly ameliorated systemic copper metabolism, evidenced by decreased serum copper and increased ceruloplasmin activity. Liver function tests and antioxidant capacity were markedly improved. Histologically, the treatment led to a substantial reduction in hepatic steatosis, inflammatory infiltration, and collagen deposition. Ultrastructural analysis by TEM confirmed the mitigation of critical cellular injuries, including mitochondrial swelling, nuclear fragmentation, and endoplasmic reticulum stress. Integrated transcriptomic and targeted metabolomic analyses further identified that these therapeutic benefits were linked to the regulation of key pathways related to mitochondrial function and metabolism.

Conclusions: This study provides compelling proof-of-concept for a novel regenerative strategy against WD, wherein a bioengineered ectopic liver created from patient-specific USCs and hydrogel corrects core metabolic defects and mitigates hepatotoxicity. Integrated multi-omics analysis reveals that the restoration of mitochondrial homeostasis serves as a fundamental mechanism mediating these therapeutic benefits, positioning the USC-hydrogel platform as a promising and clinically translatable therapeutic avenue for WD.

Keyword(s) : Wilson disease, Urine-derived stem cells, Hydrogel, Mitochondrial homeostasis, Cell therapy

Indeterminate Phase Chronic Hepatitis B with Normal ALT

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Background:

In chronic hepatitis B (CHB), an indeterminate phase exists outside the typical predefined phases. Among patients with CHB in the indeterminate phase, approximately 40% are treatment-naïve. Clinical outcomes and the effect of antiviral therapy on the indeterminate phase remain unclear. Our study investigates the efficacy and clinical benefits of antiviral therapy in indeterminate-phase CHB (IHC) patients with normal alanine aminotransferase (ALT) levels.

Materials and Methods:

In this prospective study, we identified all treatment-naïve IHC patients with normal ALT. Patients were assigned to receive TAF monotherapy or Peg-IFN plus TAF therapy based on the patients' own discretion. They were monitored closely for 52 weeks with liver function tests, complete blood counts, virological markers, and imaging examinations.

Results:

To date, a total of 129 patients have completed 52 weeks of antiviral therapy, including 106 in Peg-IFN- α plus TAF group and 23 in TAF monotherapy group. During the 52-week therapy, hepatitis B virus (HBV) DNA decreased over time in both groups ($P < 0.05$). At week 52, 87.6% (113/129) patients achieved undetectable HBV DNA, with 86.8% (92/106) in the combination group and 91.3% (21/23) in the monotherapy group reaching this outcome. Baseline HBsAg level [2.7 (1.9 - 3.1) vs. 3.3 (3.1 - 3.5) log₁₀ IU/mL, $P < 0.001$], HBV RNA [2.2 (2.0 - 3.0) vs. 3.1 (2.7 - 3.7) log₁₀ copies/mL, $P = 0.001$] and HBcrAg [2.8 (2.0 - 3.3) vs. 4.2 (3.2 - 5.3) log₁₀ U/mL, $P < 0.001$] were significantly lower in the HBV DNA-negative group compared to the HBV DNA-positive group at week 52. The combination group demonstrated significantly greater reductions from baseline at each point compared to the monotherapy group ($P < 0.001$). The HBsAg seroclearance rate in the combination group was 14.7% (18/129) at week 52, and no patient in the monotherapy group achieved HBsAg seroclearance at week 52. Baseline HBsAg level [1.4 (0.7 - 2.1) vs. 2.9 (2.2 - 3.3) log₁₀ IU/mL, $P < 0.001$], HBV RNA [2.0 (2.0 - 2.0) vs. 2.4 (2.0 - 3.2) log₁₀ copies/mL, $P = 0.002$] and HBcrAg [2.4 (2.0 - 3.0) vs. 3.0 (2.5 - 3.6) log₁₀ U/mL, $P = 0.006$] were significantly lower in the HBsAg-seroclearance group compared to the Non-seroclearance group at week 52.

1902 - Antiviral Therapy Leads to Virological Response and Functional Cure in Treatment Naïve

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Conclusions: This study demonstrates that treatment-naïve IHC patients with normal ALT exhibited a favorable response to antiviral therapy, with Peg-IFN- α combination therapy is effective in achieving clinical cure.

Keyword(s) : Chronic hepatitis B, HBV DNA, Indeterminate phase, antiviral therapy,

1904 - High prevalence of Hepatic Steatosis in adolescence and Young Adults A crosssectional survey from Sindh Pakistan

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Background / Hypothesis:

Hepatic Steatosis and its Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) are emerging as a global concern and have a major impact on health. The overall aim of the study was to estimate the prevalence of Hepatic Steatosis and MASLD in adolescents and young adults in the province of Sindh, Pakistan and report associated metabolic factors including obesity, central obesity, dyslipidemia and Prediabetes/Diabetes with Hepatic Steatosis

Materials and Methods:

It was a cross-sectional study using a multistage complex household sampling strategy conducted in all (31) districts of Sindh, Pakistan, in 2023-2024.

Adolescents (10-19 years) and young adults (20-24 years) were included and those who were hepatitis B or C positive were excluded. The primary outcome 1 was Hepatic Steatosis and Primary outcome 2 was MASLD. Hepatic Steatosis was defined as ultrasound attenuation parameter UAP > 244 dB/m using a Fibro touch: Shear-wave Elastography System. MASLD was defined as Hepatic Steatosis and any one of the cardiometabolic criteria (Obesity, hypertension, triglyceride > 150 mg/dl, HDL < 40 mg/dl, Fasting blood sugar > 100 mg/dl,)

Results:

A total of 2,200 participants were invited to participate, of whom 2,136 (97.1%) agreed. Among these, 1,651 (77.3%) were adolescents aged 10–19 years and 485 (22.7%) were young adults aged 20–24 years, with a mean (SD) age of 15.8 ± 4.0 years. Overall, 997 (43.7%) participants were from rural, 1,053 (47.6%) were males. Of the total 456 (21.1%) were underweight, 162 (8.6%) were overweight, and 87 (4.3%) were obese, 519 (26.0%) had impaired fasting glucose, and 93 (8.8%) had diabetes.

Overall, 1,053 (53.3%) participants had hepatic steatosis, including 802 (52.1%) adolescents and 251 (57.6%) young adults. Among adolescents with Hepatic steatosis 444 (55.08%) and 358 (44.92%) were in early (10–14) and Mid-Late Adolescence (15–19). On multivariate regression, overweight or Obesity (OR 2.52 (95 % CI 1.19-5.31), central obesity (OR 3.37 (95 % CI 1.38-8.26) was associated with hepatic steatosis, while being in middle (OR 0.80 (0.59-1.08) or rich (OR 0.87 (0.58-1.30) socio economic index was protective for Hepatic steatosis. The prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD) was 717 (36.3%) overall, comprising 525 (34.9%) adolescents and 192 (41.4%) young adults

Conclusions:

High prevalence of hepatic steatosis and MASLD in adolescents and adults from Sindh, Pakistan indicating that the occurrence happens much early in life. Further studies are required to look at mechanisms that lead to hepatic Steatosis early

Keyword(s) :

1905 - Partial Splenic Embolization in a Cirrhotic Patient with Refractory Variceal Bleeding and Portal Venous Thrombosis

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Significance: Variceal bleeding remains a major cause of mortality among cirrhotic patients. Compared to esophageal varices, gastric varices are less common but more challenging to manage. This case report highlights the use of partial splenic embolization (PSE) as an alternative option in a cirrhotic patient with portal vein thrombus (PVT).



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Case Presentation: A 68-year-old male with cirrhosis from chronic hepatitis B and hepatocellular carcinoma was admitted due to hematemesis. Initial management carried out with antibiotics, octreotide, and supportive blood transfusions. Esophagogastroduodenoscopy showed gastroesophageal varices GOV-1. Beta blockers started, with intent to do rubber band ligation late once the varices are smaller. Dynamic CT of the liver demonstrated tumor thrombi in the proximal main portal vein, immediately after the splenic and superior mesenteric veins. On the 4th hospital day, patient had hematemesis hence on repeat gastroscopy, histoacryl glue was injected into the GOV-1. PSE was planned but family consented only on the 7th hospital day. He underwent PSE using polyvinyl alcohol particles and 500-700 micron microspheres, showing devascularization of 70% of the spleen. After two days, the patient again had hematemesis, hence he opted for palliative care and expired.

Recommendation: In our patient, PSE was done to reduce splenic venous inflow into the portal vein. If done early, PSE might have significantly reduced portal pressure, as demonstrated in other case reports. However, given our patient's overall poor prognosis, PSE was used as a salvage therapy to control his refractory variceal bleeding.

Keyword(s) : Refractory Variceal Bleeding, Cirrhosis, Partial Splenic Embolization

1907 - Delayed diagnosis of intrahepatic cholestasis of pregnancy in North India Uncovering health system gaps through a mixed methods approach

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Background/Hypothesis:

Intrahepatic cholestasis of pregnancy (ICP) is linked to preterm birth, stillbirth and neonatal morbidity, with risks increasing at higher bile acid levels. Evidence on health-system contributors to delayed recognition in rural South Asia is scarce. We quantified diagnostic delays among women with ICP presenting to a rural secondary-care public hospital in North India and examined multi-level barriers to timely diagnosis and management.

Materials and Methods:

We conducted a convergent mixed-methods study in a government hospital serving a predominantly rural population. The quantitative component was a retrospective cohort of pregnant women with ICP. Data were extracted on timing of pruritus onset, first facility contact, bile acid testing, diagnosis and delivery, and fetomaternal outcomes. "Health-system diagnostic delay" was defined a priori as ≥ 7 days from first documented presentation for pruritus to confirmed diagnosis.

The qualitative component included 22 in-depth interviews with women experiencing delay, frontline providers and ASHA workers. Data were analysed thematically using the three-delays framework.

Results:

We included 214 women (mean age 26.9 ± 4.3 years; 61.7% multigravida). Median gestational age at pruritus onset was 30 weeks (IQR 28–32). The median interval from first facility contact to confirmed ICP diagnosis was 9 days (IQR 5–16); 58.4% ($n=125$) met criteria for health-system diagnostic delay. Delay was more frequent among women first seen at sub-centres/primary health centres versus those presenting directly to the hospital ANC clinic (adjusted odds ratio [aOR] 2.11, 95% CI 1.19–3.74) and among those without jaundice (aOR 1.86, 95% CI 1.03–3.37). After adjustment for bile acid levels, gestational age at diagnosis and ICP severity, diagnostic delay was independently associated with the composite adverse perinatal outcome (aOR 1.72, 95% CI 1.01–2.94). The odds of preterm birth (<37 weeks) were also higher in the delayed group (aOR 1.94, 95% CI 1.06–3.55). Qualitative analysis identified four themes: (1) pruritus widely perceived as normal in pregnancy, delaying attention; (2) fragmented referral pathways and limited access to bile acid testing; (3) low prioritisation of liver disease within routine ANC; and (4) structural barriers including laboratory constraints, costs and after-hours access. Theme saturation was reached by the 20th interview.

Conclusion:

More than half of women with ICP experienced health-system diagnostic delay, which remained associated with adverse perinatal outcomes. Improving recognition of pruritus, strengthening referral pathways and ensuring timely bile acid testing could reduce preventable complications in similar rural settings.

Keyword(s) : intrahepatic cholestasis of pregnancy;



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antenatal care; diagnostic delay; maternal health services; India

1910 - A Bibliometric Analysis of the Diagnosis and Treatment of Portal Hypertension Based on the Web of Science Database

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Background / Hypothesis: Portal hypertension refers to a clinical syndrome characterized by abnormally elevated pressure within the portal venous system due to various causes. It imposes a significant global disease burden, with severe and potentially fatal complications. With deepening understanding of the pathophysiological mechanisms underlying portal hypertension, diagnostic and therapeutic techniques have made substantial progress in recent years. Diagnostic and therapeutic strategies for portal hypertension have evolved significantly, yet numerous challenges persist. Therefore, timely and accurate diagnosis coupled with effective treatment are crucial for improving patient outcomes and reducing mortality rates.

Materials and Methods: A retrospective collection of studies related to the diagnosis and treatment of portal hypertension from the Web of Science Core Collection (WOSCC) database was conducted. Bibliometric methods were employed to analyze the number of publications, countries, journals, authors, keywords, and other relevant factors.

Results: A total of 1,200 English-language publications were included in the analysis, showing an overall upward trend in annual output. China ranked highest in co-occurrence frequency (341 instances), demonstrating absolute dominance in research output within this field. The United States exhibited the highest centrality (0.16), occupying a core hub position within the research network. The University of Barcelona (Univ Barcelona) led with 55 co-occurrence frequencies and the highest centrality (0.14),

establishing itself as a core research hub. The most frequently co-occurring author was Thomas Reiberger from the United States (32 times), a key driving force in the current field. Research hotspots in portal hypertension demonstrate vertical deepening from therapeutic techniques toward mechanism analysis and risk assessment, while simultaneously expanding horizontally into specialized studies of specific subtypes and complications.

Conclusions: Research on portal hypertension exhibits thematic differentiation and convergence over time, with technological breakthroughs and clinical needs serving as dual core drivers of its evolution. Future studies should prioritize precision-integrated therapies, prognostic assessment, and molecular target validation, while strengthening the integration of mechanism elucidation with clinical practice.

Keyword(s) : portal hypertension, diagnosis , treatment

1913 - Impact of of air pollution to liver diseases

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Background / Hypothesis:

Liver disease accounts for approximately 2 million deaths per year worldwide and is responsible for 4% of all deaths. Mongolia is one of the most polluted countries in the world. Exposure to PM2.5 is linked to the development of inflammation, which may be a major risk factor in the advancement of non-alcoholic fatty liver disease. Many epidemiological and ecological studies were done in Mongolia on air pollution and health. However, it is still not clear how much ambient air pollution can cause cirrhosis and non-alcoholic fatty liver disease incidence in Mongolia.

Materials and Methods:

This study employed a cross-sectional study design using data from the nationwide health screening program to assess the prevalence and healthy Mongolian population. The nationwide health screening program was conducted by the Ministry of

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Health in Mongolia between 2022 to 2024. The health screen program included 209,055 adults aged over 18 years. Participants with missing data, outliers in key variables, and those had any kind of hepatitis virus infection who live in rural area were excluded, resulting in a final sample of 173,762 adults. Because of only Ulaanbaatar city air pollution data is available, we used only Ulaanbaatar city resident's screening data. We used 16 air quality monitoring stations to measure air pollutant including PM_{2.5} µg/m³, and PM₁₀ mg/m³.

Results:

The study found that the mean age of respondents was 44.01±15.35 years, with females being slightly older on average. Liver enzyme levels were higher among males for both ALT (2.42±12.32) and AST (1.97±9.04). Weight emerged as the strongest predictor of ALT, with age, PM_{2.5}, and PM₁₀ contributing smaller yet statistically significant effects; similar patterns were observed for AST. The combined model of age, weight, and particulate matter (PM_{2.5} and PM₁₀) explained 10.3% of the variance in blood ALT levels and 3.0% of the variance in AST levels. Overall, both liver enzymes showed only weak but direct correlations with particulate air pollution.

Conclusions:

This large population-based analysis indicates that ambient particulate air pollution has only a weak but direct association with liver enzyme levels among healthy adults in Ulaanbaatar.

Keyword(s) : liver enzyme, air pollution, Ulaanbaatar, Mongolia

1914 - Research progress on the association between metabolic dysfunction associated fatty liver disease and gallstones

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Background:

Metabolic dysfunction-associated fatty liver disease (MAFLD) and gallstones diseases (GSD) are common liver and gallbladder diseases, with highly overlapping

epidemiological features and risk factors, confirming a certain correlation between the two, but with different conclusions. With the update of diagnostic criteria for MAFLD, the research dimensions of the correlation between the two continue to expand. This review integrates recent clinical evidence and mechanism research, providing reference for clinical diagnosis and future research.

Materials and Methods:

Search Pubmed and CNKI databases to include research papers on the association between MAFLD and gallstones. Exclude review literature.

Results:

Firstly, the summarized research results indicate that compared with non MAFLD patients, the incidence of GD in NAFLD patients is almost twice that of non MAFLD patients (incidence rates of 47% and 26%, respectively, $p < 0.0001$); Compared with patients without GSD, 56% of patients with gallstones had MAFLD ($p < 0.0001$). Secondly, the research mechanism suggests that the two share three core pathways: lipid metabolism disorders, insulin resistance, and chronic inflammation. The accumulation of free fatty acids leading to bile cholesterol supersaturation is a key link. Insulin resistance serves as a crucial bridge between GSD and MAFLD. The relationship between abnormal glucose metabolism and fatty liver disease is highly recognized, and fasting blood glucose levels are fully correlated with the presence of GSD in MAFLD patients. Finally, in terms of influencing factors, age increase, sex, diabetes, ALP increase, HDL decrease and FBS increase are the only predictors of biliary node in MAFLD patients. Lifestyle interventions can reduce risks. Other studies have shown that asymptomatic patients with MAFLD and non obese individuals also exhibit an increased risk of gallstones. Therefore, in clinical practice, caution should be exercised in treatment and comprehensive screening should be conducted to prevent the occurrence of the disease.

Conclusions:

There is a clear epidemiological association and common pathophysiological basis between MAFLD and gallstones. Clinical attention should be paid to screening for gallstones in MAFLD patients, especially in high-risk subgroups. Future research should focus more on the molecular mechanisms associated with the two and explore targeted intervention measures, providing favorable references for clinical diagnosis and treatment management.

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Keyword(s) : gallstone disease, nonalcoholic fatty liver disease, insulin resistance

1915 - Ibrutinib Associated HBV Reactivation in Hematologic Malignancies Clinical Experience From Our Center

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Background / Hypothesis: Ibrutinib is widely used for chronic lymphocytic leukemia (CLL) and other hematologic malignancies, but its immunosuppressive effects raise concerns about hepatitis B virus (HBV) reactivation in previously exposed patients. This study evaluated HBV serologic status, the frequency and timing of reactivation, and related clinical outcomes in patients receiving ibrutinib at our center.

Materials and Methods: A retrospective analysis was conducted on 29 patients with hematologic malignancies who received ibrutinib therapy between 2013 and 2023. Demographic characteristics, concomitant immunosuppressive treatment, HBV serology, use of antiviral prophylaxis, and laboratory data during treatment were reviewed. HBV reactivation was defined as newly detected HBV DNA positivity in patients previously HBV DNA-negative, a ≥ 1 log increase in HBV DNA level, or HBsAg seroreversion in HBsAg-negative/anti-HBc IgG-positive individuals. Clinical course, prophylaxis, and treatment approaches for patients who experienced reactivation were documented.

Results: Of the 29 patients, 23 received ibrutinib for CLL and 6 for lymphoma. Evidence of past natural HBV infection was present in 18 patients (62%). HBV reactivation occurred in 8 of these 18 patients (44.4%) during ibrutinib therapy. Two of the affected patients were also receiving rituximab. In 6 patients (33.3%), reactivation was attributed solely to ibrutinib. All reactivated patients had baseline resolved immunity (anti-HBc IgG-positive, anti-HBs-positive). One patient experienced reactivation 7 months after discontinuation of ibrutinib, while the remaining five reactivated during therapy at months 26, 20, 12, 7, and 3, respectively. Reactivation manifested as HBV DNA increase in three patients, newly detectable HBV DNA in two patients, and isolated HBsAg seroreversion in one patient. HBV reactivation was more common in patients with anti-HBs < 100 IU/L, although this was not statistically significant ($p = 0.553$). The risk of reactivation was nearly four times higher in this group. All patients were treated with entecavir or tenofovir, resulting in complete virological suppression; no liver

failure or HBV-related mortality occurred.

Conclusions: In our series, patients with hematologic malignancies receiving ibrutinib who had prior HBV exposure exhibited a notably high reactivation rate. Since reactivation may occur at various stages of treatment—even after therapy discontinuation—close virological monitoring is essential in this population. Although ibrutinib is classified as an uncertain-risk agent in some guidelines, our findings indicate that greater vigilance is warranted during prolonged oral ibrutinib therapy. Considering baseline HBV serologic profile and individual risk factors, prophylactic antiviral therapy may be appropriate in selected patients. Larger, prospective studies are needed to determine the optimal preventive and management strategies.

Keyword(s) : HBV reactivation, immunosuppression, hematologic malignancy, ibrutinib

1917 - Association between the platelet high density lipoprotein cholesterol ratio and metabolic dysfunction associated steatotic liver disease A corhot study

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Background / Hypothesis:

The platelet/high-density lipoprotein cholesterol ratio (PHR) is a novel marker of inflammation and hypercoagulability, representing the severity of metabolic syndrome. Hepatic metabolic syndrome manifests as metabolic dysfunction-associated steatotic liver disease (MASLD), which is associated with inflammation and hypercoagulability. This study aims to determine the relationship between PHR and MASLD diagnosed by liver biopsy.

Materials and Methods:

This retrospective study included patients who underwent liver biopsy were diagnosed with MASLD at Beijing Ditan Hospital as shown in Figure 1. The PHR was calculated as the ratio of platelets to high-density lipoprotein cholesterol. According to PHR quartiles, to compare the differences in various clinical indicators at the Q1-Q4 levels, and the relationships between PHR and clinical parameters. The differences in CAP and LSM among the quartiles of PHR, and the differences between MASLD and hepatic fibrosis according to the quartiles of PHR were analyzed.

Results:



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A total of 527 patients diagnosed with MASLD by liver biopsy were enrolled. After adjusting for other clinical variables, patients in the highest PHR quartile had 3.02 times higher odds and 95% CI (1.89-3.78) of having NAFLD compared to those in the lowest quartile. This result was statistically significant ($P=0.003$). While increased PHR was initially associated with fibrosis, this association became not statistically significant ($P=0.093$) after adjusting for other variables.

Conclusions:

PHR is an effective and promising marker for identifying MASLD. The study suggests that interventions aimed at improving a patient's PHR could help reduce the incidence of MASLD.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease, platelet/high-density lipoprotein cholesterol ratio, liver biopsy, Liver stiffness measurement, hepatic fibrosis

1919 - Trends of Chronic Liver Disease and Cirrhosis Mortality in Medically Underserved Areas of Taiwan 2015–2022

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Background / Hypothesis:

The World Health Organization (WHO) aims to eliminate viral hepatitis by 2030, and Taiwan is on track to achieve hepatitis C (HCV) elimination as early as 2025 following nationwide reimbursement of direct-acting antivirals (DAAs) beginning in 2017. Despite these advances, national mortality databases do not document viral etiologies for deaths from chronic liver disease and liver cirrhosis (CLD/LC). Sentinel hospital data are therefore essential to provide HBV/HCV-specific mortality estimates and to evaluate progress among high-risk subpopulations, particularly medically underserved areas (MUAs), where healthcare access and screening remain limited.

Materials and Methods:

This analysis included individuals who died from CLD/LC (ICD-10 K70, K73–K74) between 2015 and 2022 across two major Taiwanese healthcare systems (Chang Gung and Tzu Chi). Deaths due to primary liver cancer were excluded. In addition to HBV and HCV data, the analysis also linked administrative, laboratory, and imaging datasets. MUAs were defined based on the National Health Insurance Administration's 2024 classification. Viral-specific mortality proportions change was calculated.

Results:

Among 6,619 decedents with complete viral testing, overall HCV-related mortality declined markedly from 22.4% in 2015 to 12.8% in 2022. In contrast, HBV-related mortality showed minimal change, and Non-BC mortality increased steadily, suggesting a rising burden of metabolic or lifestyle-related liver disease. Within MUAs, the decline in HCV mortality was even more pronounced, decreasing from 28.1% to 11.6%, while HBV mortality declined modestly from 17.7% to 14.3%. Women experienced a larger reduction in HCV-related deaths (–75%) than men (–49%). MUAs also exhibited disproportionately high smoking and alcohol use among men, coupled with unexpectedly low detection of diabetes and fatty liver disease, indicating under-screening and healthcare access gaps.

Conclusions:

Taiwan has achieved substantial reductions in HCV-related CLD/LC mortality both nationally and within MUAs, reflecting the broad impact of DAA therapy. However, HBV-related mortality remains largely unchanged, and Non-BC mortality continues to rise, highlighting increasing contributions from metabolic and lifestyle-associated liver disease. MUAs represent a

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vulnerable subpopulation characterized by high behavioral risks, limited diagnostic detection, and unequal healthcare access. Strengthening HBV management, improving screening coverage, and enhancing access to antiviral therapy in MUAs will be essential to ensure equitable progress toward Taiwan's 2025 HCV elimination milestone and WHO's 2030 viral hepatitis elimination targets.

Keyword(s) :

1921 - Clinical spectrum and long term adverse outcome of Infantile onset Extrahepatic Portal Vein Obstruction

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Background / Hypothesis:

Extrahepatic portal vein obstruction (EHPVO) is one of the leading causes of portal hypertension in children. Apart from variceal bleeding, significant morbidity occurs due to massive splenomegaly, hypersplenism, portal biliopathy and growth retardation. This study aims to assess the clinical features and outcomes of infantile onset EHPVO and to compare with onset in older children providing insights into how age at presentation influences disease progression, complications and management.

Materials and Methods:

Data from a prospectively maintained database of 1,050 children with EHPVO, spanning from 1993 to 2024, were analysed. Patients were categorized by age at onset: infancy, 1–5 years, 6–10 years, and 11–18 years, and these groups were compared with respect to growth, hypersplenism, and the development of complications such as portal biliopathy and portal colopathy.

Results:

Total 1050 children were diagnosed with EHPVO during the study period. Mean age of onset of symptoms were 6(2.75-9)years and mean age at diagnosis were 9(6-13 years). Presentation in infancy (n=88), between 1-5years (n=268), between 6-10 years (n=430) and between 11-18 years (n=213) with slight male predominance across all groups. Presentation as

variceal bleeding was significantly higher in infancy as compared to older children (89.7% vs. 86.07%, $p=0.043$). Median follow-up of these children were available for 96 (36-144) months. Symptomatic portal biliopathy was seen in significantly higher proportion in infantile onset group (7.95%) as compared to all other groups. Portal colopathy was significantly higher in infantile onset group (17%) compared to children with onset after 6 years of age. Growth failure was significantly higher in children with onset 1-5 years compared to infantile onset and 6-10 years group. Spleen size was significantly large and hemoglobin was significantly low in infantile onset group. No difference was seen with respect to presence of hypersplenism, liver function tests, platelet count and total leukocyte count. Porto-systemic shunt surgery was done in 307 (29.23%) children. Incidence of shunt block was seen in significantly higher proportion in infantile onset group (11.5%) as compared to other groups.

Conclusions:

This large study on EHPVO demonstrates that complications are significantly more severe in patients with disease onset during infancy. This heightened severity is likely due to the prolonged duration of disease, which allows for the progressive consequences of portal hypertension to become more pronounced over time. These findings underscore the critical importance of early detection and proactive management in at-risk populations to minimize long-term complications and improve clinical outcomes.

Keyword(s) : Extrahepatic portal vein obstruction, Portal biliopathy, Portal colopathy

1922 - Clinical and therapeutical characteristics of patients infected with hepatitis B and D viruses in a tertiary center from Bucharest Romania

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Department, Bucharest-Romania ³

Background / Hypothesis:

Hepatitis B and D viruses (HBV and HDV) contribute to high liver disease burden, despite effective vaccination available for almost 30 years. Recent evaluation of HBV prevalence among European countries sets Romania the second highest (6.2%), after Cyprus. Regarding HDV global epidemiology, the assessed prevalence was 4.5% in people with chronic HBV infection, yet insufficient data for an accurate estimation in Romania. In 2024, Bulevirtide has been approved in Romania, preliminary results showing positive effects on slowing disease progression and increasing survival.

Objective: Analysis of the clinical characteristics, complications and treatment of HBV and HDV infection

Materials and Methods:

We conducted a retrospective study on HBV and HDV infection cases among adults evaluated in our department of National Institute for Infectious Diseases "Prof. Dr. Matei Balș" between September 2010 and November 2025. Clinical, biological and treatment-related data were collected and analyzed using SPSS Statistics (version 26).

Results:

We included 110 patients, the median age was 51.25 years (IQR: 48.45-54, age range 21-79 years old) and sex ratio M:F = 1:1. Positive HDV load was found in 67 of 88 patients tested (76.13%), with a median value of 19.876 UI/mL. Cirrhosis was identified in 44 patients and hepatocellular carcinoma in 6 cases. HIV co-infection was diagnosed in 8 patients, with a lower median age than the seronegative group (33.1 years, $p < 0.001$). Median Charlson comorbidity index was 3.65 (IQR: 3.20-4.11). Cirrhosis was associated with higher monocyte-to-lymphocyte ratio than non-cirrhotic patients (mean 0.25 versus 0.41, $p = 0.037$). HBV and HDV reactivation in patients with hematological malignancies receiving chemotherapy occurred in 4 cases. Between 2003 and 2024, Interferon therapy was administered to 46 patients, maximum number of cycles was 4, without successful suppression of HDV. Bulevirtide 2 mg monotherapy or co-administered with nucleoside/nucleotide analogues has been initiated in 21 patients since January 2025, 5 of them with history of IFN treatment, with a decrease in the alanine aminotransferase (ALT) levels (mean 123.85 U/L before treatment to 42.14 U/L after 6 months of treatment) and without adverse events.

Conclusions:

Bulevirtide currently represents the only therapeutic option available for chronic HBV and HDV hepatitis, improving the liver function after 6 months of administration. More eligible patients need to be enrolled in treatment with Bulevirtide and further studies must assess the long-term evolution.

Keyword(s) : hepatitis B virus, hepatitis D virus, bulevirtide

1924 - Safety and feasibility of next generation extracorporeal albumin detoxification dialysis for long term bridging to liver transplantation in secondary sclerosing cholangitis

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Background / Hypothesis:

Secondary sclerosing cholangitis in critically ill patients (SSC-CIP) is a highly lethal condition, with approximately 50% mortality within the first year. We report the course of a 35-year-old male who developed severe necrotizing bacterial pneumonia following RSV pneumonia. His only known comorbidity was IgA nephropathy treated with antihypertensives; he was not immunosuppressed. Right lower-lobe lobectomy was followed by septic shock, hemorrhagic complications, vasopressor-dependent circulatory failure, and acute kidney injury requiring continuous renal replacement therapy (CRRT). Progressive cholestasis and liver injury evolved into critical-illness cholangiopathy, confirmed by ERCP and liver biopsy, with bilirubin exceeding 15 mg/dL.

Materials and Methods:

As bilirubin and bile acids continued to rise—accompanied by severe pruritus—we escalated CRRT to extracorporeal albumin dialysis (ECAD) using the MaxiCycler system, which has been shown to provide enhanced bile-acid clearance compared with MARS, CytoSorb, plasma perfusion over BR350, or plasma exchange (Stange et al., NDT 2024).

Results:

Over the first month of ECAD therapy, multiorgan failure resolved sufficiently for transfer out of the ICU and subsequently home. Because bile excretory function did not recover over more than six months, outpatient ECAD was continued three times weekly. Serial imaging and ERCP confirmed persistent impairment of biliary excretion, although the patient



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exhibited no clinical signs of cirrhosis after one year and 170 ECAD sessions. Each session consistently removed >540 mg of bile acids. The patient maintained weight, remained free of pruritus, and had stable functional status. Liver biopsy later showed progressive fibrosis; the patient was subsequently listed for transplantation and received a liver graft 18 months after initiation of ECAD, following >200 sessions.

Conclusions:

MARS-based ECAD has been shown to ameliorate hepatic encephalopathy, hemodynamic instability, and renal dysfunction in controlled studies, though effects on overall survival remain under investigation. Limitations of earlier systems included restricted adsorption capacity and complex workflows. This case illustrates that next-generation ECAD platforms provide higher detoxification efficiency and are more compatible with routine critical care practice. To our knowledge, this represents the longest continuous outpatient ECAD treatment reported to date.

Keyword(s) : Extracorporeal Albumin Detoxification/-dialysis; ECAD; MARS, Secondary Sclerosing Cholangitis; Bridge to Transplant; cholestatic Pruritus

1925 - Burden of metabolic dysfunction associated steatotic liver disease in Saudi Arabia Evidence from the first nationwide population based cohort study

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a leading cause of chronic liver disease and is strongly linked to obesity and diabetes, all of which are increasing globally and in Saudi Arabia. However, robust national-level data on MASLD prevalence and associated risk factors are still needed. We aimed to estimate MASLD prevalence among Saudi adults using clinical, biochemical, and imaging assessments.

Materials and Methods:

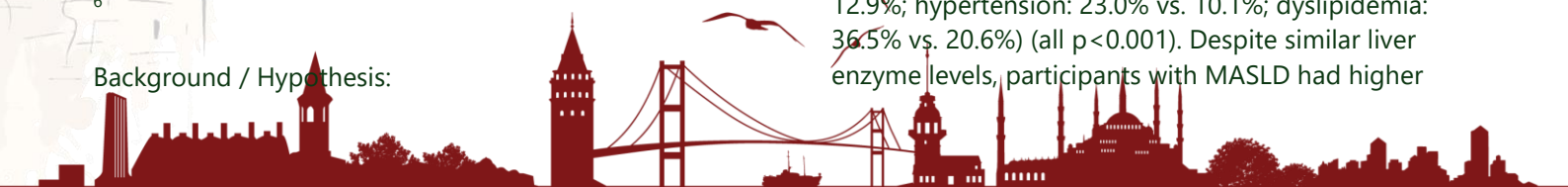
GENESIS is the first large-scale, prospective, population-based cohort study designed to assess the prevalence and severity of MASLD in Saudi Arabia. Adults without prior liver disease, except MASLD, were recruited from primary healthcare centers across three major regions between March 2024 and June 2025. All participants underwent standardized clinical evaluation, laboratory testing, and vibration-controlled transient elastography. MASLD was defined as a controlled attenuation parameter (CAP) ≥ 268 dB/m together with ≥ 1 cardiometabolic risk factor (CMRF) (e.g., BMI ≥ 25 kg/m², elevated HbA1c, hypertension, or dyslipidemia). Sensitivity analyses used alternative definitions (CAP ≥ 220 dB/m and MASLD liver fat score [MASLD-LFS]). Significant fibrosis (F2–F4) was defined as a liver stiffness ≥ 7.1 kPa.

Results:

Among 2,983 participants, the MASLD prevalence was 37.2% (age-adjusted: 34.1%), with higher prevalence in males (43.6% vs. 30.9%). Hepatic steatosis was present in 68.8% of participants, and 20.7% had severe steatosis (CAP ≥ 300 dB/m). Using alternative definitions, steatosis prevalence was 66.1% with CAP ≥ 220 dB/m and 58.6% with MASLD-LFS. Significant fibrosis was observed in 2.9% of participants.

Participants with MASLD were more often male (58.4% vs. 44.8%) and older (mean age: 46.1 vs. 39.5 years) (all $p < 0.001$). No differences were observed in smoking or coffee consumption between MASLD and non-MASLD groups.

Participants with MASLD had higher BMI (median: 31.6 vs. 27.0 kg/m²), higher waist-to-hip ratio (mean: 0.91 vs. 0.86), and greater CMRF burden (diabetes: 33.4% vs. 12.9%; hypertension: 23.0% vs. 10.1%; dyslipidemia: 36.5% vs. 20.6%) (all $p < 0.001$). Despite similar liver enzyme levels, participants with MASLD had higher



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median insulin (13.0 vs. 8.1 μ U/mL) and C-reactive protein (3.5 vs. 2.1 mg/L) (all $p < 0.001$).

Conclusions:

MASLD was present in 37.2% of this nationwide cohort, with hepatic steatosis in two-thirds of participants. Individuals with MASLD were more often male, older, and more likely to have greater CMRF burden. These findings reflect a substantial and growing public health burden and highlight the need for targeted prevention and management strategies. The findings can support future national initiatives for MASLD and broader noncommunicable disease control in Saudi Arabia.

Keyword(s) : MASLD, NAFLD, Steatosis, Fibrosis, Transient Elastography, Epidemiology, Prevalence, Saudi Arabia

1927 - The severity of diabetes mellitus in metabolic dysfunction associated steatotic liver disease in the Saudi population

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Background / Hypothesis:

Diabetes mellitus (DM) is a major metabolic disorder increasingly linked to liver disease progression. However, the full phenotypic burden of DM on liver health and cardiometabolic risk in the Middle East region insufficiently characterized. We hypothesized that DM is associated with more pronounced metabolic liver disease and multidimensional comorbidities.

Materials and Methods:

We analyzed data from 2,983 adults enrolled in the Genomics and Environmental Noninvasive Evaluation for Saudi Intrahepatic Steatosis (GENESIS), a large-scale, population-based, liver health screening project in Saudi Arabia. Between March 2024 and June 2025, adults from the general population without pre-existing liver disease other than metabolic-associated steatotic liver disease (MASLD) were recruited through primary healthcare centers across three major regions to assess the prevalence of MASLD and its associated morbidities. These participants were categorized based on their DM status, which was determined by self-reported DM or current use of antidiabetic medications. Collected data included sociodemographic characteristics, comorbidities, and transient elastography findings (controlled attenuation parameter [CAP] and liver stiffness measurement [LSM]). MASLD was defined as moderate-to-severe hepatic steatosis ($\text{CAP} \geq 268$ dB/m) in the presence of at least one cardiometabolic risk factor. Significant fibrosis (F2–F4) was defined as $\text{LSM} \geq 7.1$ kPa.

Results:

The cohort included 630 participants with DM (21.1%) and 2,353 without DM (78.9%). Participants with DM were older (mean age: 53.7 vs 38.8 years, $P < 0.001$), while sex distribution was similar between groups (males: 50.2% vs 49.8%, $P = 0.91$). DM was associated with a substantially higher prevalence of metabolic syndrome traits, including dyslipidemia (66.5% vs 15.8%), hypertension (41.9% vs 7.6%), and higher BMI (median: 31.0 vs 28.2 kg/m^2) (all $P < 0.001$).

Liver disease severity was significantly elevated among participants with DM, with MASLD prevalence of 60.6% vs 31.0%, severe steatosis ($\text{CAP} \geq 300$ dB/m) at 38.4% vs 15.9%, and advanced fibrosis at 8.1% vs 1.5% (all $P < 0.001$).

Conclusions:

In one of the largest Middle Eastern screening cohorts, DM was strongly associated with higher MASLD prevalence, more severe hepatic steatosis, and increased rates of advanced fibrosis, alongside a markedly higher cardiometabolic burden. These findings highlight the urgent need for integrated MASLD care pathways and support prioritizing individuals with DM for early screening, lifestyle

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interventions, and fibrosis assessment to prevent progression to advanced liver disease and improve long-term outcomes.

Keyword(s) : Diabetes mellitus, metabolic liver disease, MASLD, hepatic steatosis, liver fibrosis, cardiometabolic risk, Middle East, Saudi Arabia

1928 - Beyond the usual suspects" uncovering autoimmune hepatitis overlap with Wilson disease

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Background / Hypothesis:

Autoimmune hepatitis with Wilson disease is a rare overlap and potentially life threatening condition if presents with ALF. We report 3 cases to highlight diagnostic challenges and treatment outcomes in such overlap.

This series aim to describe autoimmune hepatitis and Wilson disease and there clinical presentations and management outcomes.

Case series :

Three patients (2 female & 1 male) aged between 10 to 30 years.

Case 1 & case 2- Two female patients (13 years & 15 years) presented with ALF , (jaundice& hepatic encephalopathy) , both patient had transaminitis , elevated INR, normal ceruloplasmin levels, urine cu 24 hours was more than 100mcg/24hrs, autoimmune hepatitis serology positive ,liver biopsy suggestive of AIH with copper deposits and genetic testing revealed presence of ATP7B gene mutation . Both patient according to AIH simplified & revised international score suggestive of definite AIH. Patients were started on steroids ,chelation and plasma exchange in view of ALF and blood parameters improved with improvement of hepatic encephalopathy, then patients were referred for liver donor liver transplant .

Case 3- male (28 years) patient with alcohol use disorder , presented with jaundice and ascites .transaminitis revealed of ethanol pattern with reduced platelets and reduced ALP initially.then ceruloplasmin normal with elevated urine 24 hrs and liver biopsy suggestive of ?Wilson disease? Cholestatic diseases, gene studies revealed ATP 7B gene mutation

,autoimmune hepatitis serology is positive and AIH simplified & revised international score suggestive of definite AIH.CECT - suggestive of cld with PHTN and ascites.

Diagnosis - ACLF(APASL) , patients was started on steroids and chelation and now patient on waiting list for liver transplant.

Conclusions: case series underscores the importance for early diagnosis of autoimmune hepatitis with wilson disease, for effective management of these overlaps.

In case of ALF in these overlaps plasma exchange is a bridging therapy before liver transplant.

Keyword(s) :

1929 - hUC MSC Derived Exosomal miR 22 3p Attenuates Liver Fibrosis via Inhibition of the NLRP3 Inflammasome

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Background / Hypothesis:

Liver fibrosis features excessive extracellular matrix deposition and tissue remodeling in response to chronic injury and can progress to cirrhosis or hepatocellular carcinoma. Effective antifibrotic drugs are lacking, partly due to poor translation from animal models. Mesenchymal stem cells (MSCs) show immunomodulatory and regenerative potential, but direct transplantation is limited by low survival, immunogenicity and tumorigenicity. Exosomes from MSCs provide a safer, cell-free alternative that delivers bioactive cargos such as miRNAs. This study investigated the therapeutic effect and mechanism of human umbilical cord-derived MSC (hUC-MSC) exosomes on liver fibrosis, focusing on miR-22-3p-mediated regulation of the NLRP3 inflammasome.

Materials and Methods:

hUC-MSCs were isolated and identified by morphology, surface markers and tri-lineage differentiation. Exosomes were obtained by ultracentrifugation and characterized by NTA, TEM and Western blot (CD63, CD81, TSG101). Uptake and antifibrotic effects of PKH67-labeled exosomes were evaluated in LX2 hepatic stellate cells and a carbon tetrachloride (CCl₄)-induced mouse model. LX2 cells were activated by LPS/ATP and treated with hUC-MSC exosomes. Fibrosis markers (Col1A1, α -SMA) and

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NLRP3 inflammasome components (NLRP3, Caspase-1, ASC, GSDMD, IL-1 β , IL-18) were assessed by qPCR, Western blot, IHC and ELISA. Public datasets GSE151098 and GSE153752, bioinformatic analysis and dual-luciferase assays were used to identify miR-22-3p targeting NLRP3. Exosomes enriched or depleted in miR-22-3p via agomir/antagomir transfection were used to verify its function in vitro and in vivo.

Results:

hUC-MSC exosomes were successfully characterized and efficiently internalized by LX2 cells. Exosome treatment reduced Col1A1 and α -SMA expression in activated LX2 cells, preferentially accumulated in the liver, and alleviated CCl₄-induced fibrosis, as shown by improved histology and decreased serum ALT, AST, HA and Hyp. Exosomes inhibited NLRP3 inflammasome activation and lowered IL-1 β and IL-18 production. miRNA profiling and functional validation identified miR-22-3p as a key exosomal miRNA directly targeting NLRP3. Overexpression of miR-22-3p strengthened, whereas its inhibition weakened, the antifibrotic and anti-inflammatory effects of exosomes.

Conclusions:

hUC-MSC-derived exosomes exert potent antifibrotic effects by targeting hepatic stellate cells and suppressing NLRP3 inflammasome activation. miR-22-3p is a critical exosomal cargo mediating these actions, supporting hUC-MSC exosomal miR-22-3p as a promising cell-free strategy for liver fibrosis therapy.

Keyword(s) : Liver fibrosis; hUC-MSC; Exosome; NLRP3; miR-22-3p

1930 - Outcomes of Percutaneous Interventions in Liver Abscess in children 15 year experience from a tertiary care center

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Background / Hypothesis:

Adult studies have demonstrated the safety and effectiveness of percutaneous interventions in liver abscess. However, literature is scarce in children. The aim of our study was to compare the effectiveness of percutaneous needle aspiration (PNA) and

percutaneous catheter drainage (PCD) in the management of liver abscess in children

Materials and Methods:

Prospectively kept database of children (<18 years) diagnosed with liver abscess between 2011 and 2025 were analysed. Diagnosis of liver abscess was based on the clinical symptoms and confirmation by abdominal imaging showing liver abscess. Percutaneous needle aspiration is preferred in cases of solitary abscess, less than 5cm, thin pus, unilocular and non-toxic patients. Percutaneous catheter drainage was preferred in multiloculated abscess, size >5cm, failure of ≥ 1 aspirations, toxic or septic patients. Outcomes assessed were success of intervention, volume of abscess, duration of hospital stay, timing of clinical improvement and radiological resolution and complications related to the procedure performed.

Results:

A total of 145 children with liver abscess were included. Median age was 8 (IQR: 6-14) years with a slight male preponderance (M:F 1.4:1). Most common presentation was fever and abdominal pain (n=108, 74.5%) followed by fever, abdominal pain and breathlessness (n=21, 14.7%), fever, abdominal pain and jaundice (n=3, 1.9% and isolated fever (n=13, 8.8%). The majority of cases had a single abscess (74, 80.4%), with a predilection for the right lobe (70%). Median volume of the abscess was significantly higher in PCD group compared to PNA group; 100 (63.5-200) ml vs. 39 (13.75-67) ml, p=0.008. The success rate in patients treated with PCD were similar to that of PNA (93.8% vs. 91.1%, p=0.256). Time to achieve clinical improvement was similar in both groups [PCD: 18.5 (15.5-22) days vs. PNA 17 (14.5-20) days, p=0.389] and time to achieve radiological resolution of abscess was similar in both groups [PCD: 45 (30-60) days vs. PNA: 45 (40-60) days, p=0.295]. No significant differences were found in the duration of hospitalization [PCD: 21 (16.25-24.75) days vs. PNA: 20.5 (15.5-25) days, p=0.380] or procedure-related complications [PCD: 3.09% vs. PNA: 2.94%, P=0.763].

Conclusions:

Both percutaneous needle aspiration and catheter drainage are safe methods of draining liver abscess in children. Both PCD and PNA has a similar success rate, time to achieve clinical and radiological resolution and hospital stay. Percutaneous needle aspiration can be

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preferred in cases of small (<5cm), unilocular, solitary abscess, in non-toxic patients.

Keyword(s) : Pediatric liver abscess, Percutaneous intervention, Needle aspiration, Catheter drainage

1931 - Hepatitis B Surface Antigen Seroclearance in Chronic Hepatitis B Patients. A Multicenter Study From Türkiye

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Background: In patients with chronic hepatitis B (CHB), hepatitis B surface antigen (HBsAg) seroclearance is the primary target of treatment and is rarely observed. This study aimed to investigate the factors affecting HBsAg loss.

Methods: This multicenter study was conducted in hospitals in eight provinces in different regions of Türkiye. This study was designed to retrospectively evaluate HBsAg status and clinical and laboratory findings in patients with CHB. A total of 8983 chronic hepatitis B (HB) infection patients who were treated or followed between 2014 and 2024 were evaluated retrospectively. 71 CHB patients were examined based on exclusion criteria, and 71 patients without HBsAg loss were matched as controls. A total of 142 patients were included in the study. Patient data and laboratory findings were retrieved from patient files. Statistical analysis was performed using SPSS version 25.0. The chisquare test was used for categorical variables.

Results: The basal mean hepatitis B virus (HBV) DNA level was significantly lower in the HBsAg loss group ($p < 0.001$). There were no significant differences between the sexes. Hepatosteatorsis was detected in 52% of patients in the group with HBsAg loss and in 39% of

patients in the group without HBsAg loss ($p > 0.05$). The prevalence of hyperlipidemia comorbidity ($p = 0.001$) was significantly higher in the HBsAg loss group compared to the non-HBsAg loss group.

Conclusions: In conclusion, our study identified several factors associated with HBsAg seroclearance in CHB patients, including low baseline HBVDNA levels, hyperlipidemia.

Keyword(s) : Chronic hepatitis B, Hepatitis B surface antigen, Seroclearance

1932 - Parkin perturbs Hepatocyte Mitochondria associated Endoplasmic Reticulum Membranes Dynamics

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Background / Hypothesis:

Mitochondrial-associated Endoplasmic Reticulum Membranes (MAM) plays an important intracellular function in facilitating the synergistic interactions and communication between endoplasmic reticulum and mitochondria. The study of MAM contributes to an in-depth understanding of the communication and regulatory mechanisms between intracellular organs, which is of great significance in revealing the pathogenesis and treatment of related diseases.

Materials and Methods:

Animal models were developed in liver-specific HO-1 knockout (HO-1HepKO) and wild-type (WT) mice. Ultrastructure changes in MAM were observed under TEM. Immunofluorescence double labeling was used to determine the co-localization of important proteins on MAM.

Results:

Deletion of HO-1 in mice exacerbated ultrastructural MAM damage as evidenced by the increased MAM length and reduced the number of MAM which were observed under TEM when compared to the WT mice. Deletion of HO-1 up-regulated Parkin and down-regulated MFN2, resulting in a decrease in Mito [Ca²⁺]/Cyto [Ca²⁺] ratio in vitro. However, HO-1 overexpression showed opposite results. Overexpression of Parkin by pcDNA3.1 plasmid in vitro leads to MAM abnormalities similar to those observed in Liver-specific HO-1 knockout mice, and knockdown of Parkin by siRNA plasmid in vitro has the opposite

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results. Moreover, immunofluorescence co-localization showed increased co-localization of Parkin and MFN2 in the MAM region in HO-1HepKO mice, and their interaction may lead to MAM injury.

Conclusions:

We used CRISPR/Cas9 technology to construct a liver-specific knockout HO-1 mice to investigate the role of HO-1 in the structure of r MAM in liver tissue and its regulation of calcium ion transport.

Keyword(s) : Mitochondria-associated Endoplasmic Reticulum Membranes, Parkinson

1933 - Advancing Fibrosis Assessment in Chronic Hepatitis C Through Non Invasive Approaches

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Background

Chronic hepatitis C(CHC) remains a major cause of chronic liver disease worldwide. Early identification of advanced fibrosis is crucial to guide management and prevent complications. Non-invasive fibrosis scores, such as FIB-4 and the Gamma-Glutamyl Transpeptidase-to-Platelet Ratio (GPR), are increasingly used as alternatives to liver biopsy. This study aimed to evaluate the diagnostic performance, sensitivity, specificity, and discriminative ability of FIB-4 and GPR for detecting significant fibrosis in CHC patients.

Methods

We conducted a retrospective descriptive study including CHC patients followed in a gastroenterology department over several years. Clinical, biological, imaging, and fibrosis assessment data were collected from medical records. Fibrosis was evaluated using liver biopsy, FibroScan, or FibroTest. Continuous variables were tested for normality using the Shapiro-Wilk test. The diagnostic performance of FIB-4 and GPR for significant fibrosis (>F3) was assessed using receiver operating characteristic (ROC) curve analysis.

Results

167 patients were included, The mean age was 59.4 ± 14.9 years, with a M/F ratio of 0.8. Comorbidities included hypertension in 47 individuals (28%) and

diabetes in 32 (19.1%), and 21 (12.5%) were tobacco users. CHC was incidentally diagnosed in 75 cases (44.9%). Genotype 1b was the most frequent, found in 97 cases (57.9%). Signs of chronic liver disease were present in 40 (23.7%), and portal hypertension in 31 (18.3%).

Fibrosis assessment showed that 125 (75.1%) had baseline fibrosis: F4 in 43 (26%), F3 in 32 (19%), F2 in 41 (24.6%), and F0-F1 in 49 (29.4%). Median FIB-4 was 3.25 [1.49-4.40], and median GPR was 0.87 [0.46-2.12]. A total of 150 (90%) received direct-acting antivirals, achieving sustained virological response in 136 (81.5%). FIB-4 demonstrated an AUROC of 0.874, with 90.1% sensitivity and 77.6% specificity for detecting significant fibrosis(F3-F4). GPR showed superior performance, with an AUROC of 0.913, 91.4% sensitivity, and 81.1% specificity.

Conclusion

In this cohort of CHC patients, both FIB-4 and GPR demonstrated excellent diagnostic accuracy for predicting significant fibrosis(F3-F4), with GPR outperforming FIB-4 across all metrics. These findings support the use of simple, accessible non-invasive scores in routine clinical practice, particularly in resource-limited settings, to improve early detection and management of advanced fibrosis.

Keyword(s) : fibrosis hepatitis c fib4 gpr biomarkers

1936 - Generalized Anxiety Among the patients of Fatty Liver Disease A Cross Sectional Study

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Background / Hypothesis: Metabolic Dysfunction Associated Steatotic Fatty liver disease (MASLD) is one of the most common morbidities worldwide, and most patients may suffer from anxiety due to uncertainty of future disease progression. This cross-sectional study aimed to investigate the prevalence and correlates of anxiety among patients with MASLD, examining the relationship between demographic factors, disease severity, and anxiety levels.

Materials and Methods: This cross-sectional study was conducted in the National Gastroliver Institute and

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Hospital, Dhaka, from January 2025 to December 2025. A total of 128 patients diagnosed with MASLD based on ultrasound were evaluated for Generalized anxiety by the validated Bengali Generalized Anxiety Disorder-7 (GAD-7) questionnaire. Anxiety levels were assessed using standardized measures and categorized into minimal, mild, moderate, and severe categories. Fatty liver severity was graded using ultrasonography (Grade I-III).

Results: The prevalence of anxiety among fatty liver disease patients was substantial, with 59.4% experiencing some degree of anxiety: minimal anxiety (40.6%), mild anxiety (37.5%), moderate anxiety (12.5%), and severe anxiety (9.4%). The mean anxiety score was 6.25 ± 4.896 (range: 0-18). Age emerged as the most significant predictor of anxiety levels, showing a strong positive correlation ($r = 0.377$, $p = 0.002$) and remaining statistically significant in multivariate regression analysis ($p = 0.001$). The regression model explained 24.7% of the variance in anxiety levels. A trend toward increased anxiety was observed with higher grades of fatty liver severity, but this relationship did not reach statistical significance ($p = 0.074$). Other demographic factors, including sex, occupation, residential location, and monthly family income, showed no significant associations with anxiety levels.

Conclusions: This study reveals that anxiety is highly prevalent among patients with MASLD, affecting nearly 60% of the study population. Age represents the primary demographic risk factor for increased anxiety, with older patients experiencing significantly higher anxiety levels. Healthcare providers should consider routine anxiety screening, particularly for older patients with MASLD, and implement appropriate mental health interventions as part of comprehensive disease management.

Keyword(s) : Metabolic Dysfunction Associated Steatotic Fatty liver disease , Anxiety, Mental Health

1937 - Evaluating Baveno VII Recompensation in Children with Autoimmune Decompensated Cirrhosis Clinical Predictors and Immune Axis Modulation

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Background / Hypothesis:

With immunosuppression, cirrhosis reversal ("recompensation") is increasingly observed in autoimmune hepatitis (AIH), but pediatric insights are limited. We characterized incidence, predictors, cytokine patterns, and clinical outcomes of Baveno-VII-defined recompensation in children with AIH-related decompensated cirrhosis.

Materials and Methods:

Children <18 years with AIH and decompensated cirrhosis (2014–2024) with ≥ 1 -year follow-up were assessed. Recompensation required disease control, ≥ 12 months' absence of decompensating events without supportive therapies, and normalized bilirubin/albumin/INR. Longitudinal liver health, survival, re-decompensation and predictive factors were analyzed. A subset underwent stool-plasma cytokine studies.

Results:

Seventy-eight children (median age 11 years) were included. 69.2% achieved recompensation, with stepwise resolution: INR/HE $\rightarrow$ ascites $\rightarrow$ jaundice $\rightarrow$ albumin, reflecting synthetic recovery lag. Re-decompensation occurred in 12.9%, linked to flare or adherence issues.

Recompensation conferred significant benefit: higher native liver survival (96.6% vs 70.1%, $p=0.003$) and markedly reduced transplantation (1.8% vs 16.6%). On multivariable analysis, spleen size independently predicted recompensation (AOR 1.36 per cm). Smaller spleen signaled lesser portal hypertension and higher reversibility potential.

Cytokine profiling ($n=20$) revealed higher stool IL-10 and IL-1 β alongside lower plasma IL-6 in recompensators. Stool cytokines suggest local immune repair, improved epithelial integrity, and microbiota-mediated tolerance restoration — key to resolving immune-driven hepatic injury. Conversely, lower systemic IL-6 reflects reduced chronic inflammatory burden, a state favoring hepatic regeneration rather than ongoing necroinflammation. These findings collectively support a dual-compartment immune recovery model underpinning recompensation.

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Conclusions:

Recompensation occurs in nearly 70% of children with decompensated AIH, with major gains in survival and reduced transplant requirement. Spleen size is a strong, readily measurable predictor. Cytokine signatures highlight the importance of gut-immune recalibration as a mechanism of liver repair. Future multi-center, biomarker-driven studies should refine prediction and therapeutic targeting

Keyword(s) : Autoimmune Hepatitis; Pediatric Decompensation; Recompensation; Spleen Size; Transplant-Free Survival; Stool-Plasma Cytokines

1939 - Transient elastography versus the fibrosis 4 index for fibrosis detection in metabolic dysfunction associated steatotic liver disease and metabolic dysfunction associated steatohepatitis

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Background / Hypothesis:

The prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH) is increasing in Saudi Arabia. Although fibrosis-4 index (FIB-4) is included among first-line non-invasive tests (NITs) for MASLD risk stratification, its diagnostic accuracy compared with transient elastography (TE)

liver stiffness measurement (LSM) in this population is unclear. We aimed to evaluate performance of FIB-4 overall and across age- and diabetes-defined subgroups.

Materials and Methods:

We analyzed 622 adults with MASLD/MASH from the Saudi Arabia Global Liver Registry who had TE data. Participants were stratified by LSM ≥ 8 kPa (n=53) versus < 8 kPa (n=569). Data on demographics, body mass index (BMI), comorbidities, and liver enzyme levels were collected. FIB-4 was calculated for 525 participants with complete aspartate aminotransferase (AST), alanine aminotransferase (ALT), platelet count, and age data; a cut-off of ≥ 1.30 defined patients at risk. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated overall and stratified by age category and diabetes status.

Results:

Participants with significant fibrosis (LSM ≥ 8 kPa) were older (median 51 vs 47 y) and had higher BMI (32.0 vs 30.5 kg/m²) compared with those with LSM < 8 kPa. Type 2 diabetes (54.7% vs 39.2%), hypertension (41.5% vs 26.7%), and hyperlipidemia (66.0% vs 52.2%) were more frequent in the elevated LSM group. Median AST and ALT levels were higher (28.0 vs 21.0 U/L; 34.0 vs 23.0 U/L), and median FIB-4 was 0.9 versus 0.7, with FIB-4 ≥ 1.30 observed in 20.4% versus 9.7%. Overall, FIB-4 ≥ 1.30 had 20.4% sensitivity, 90.3% specificity, 17.9% PPV, and 91.7% NPV for detecting significant fibrosis.

Age-stratified analysis showed increasing sensitivity with age: < 35 y had 14.3% sensitivity, 96.3% specificity, 25.0% PPV, and 92.8% NPV; 35–64 y had 17.1%, 91.2%, 15.8%, and 91.9%; while ≥ 65 y had 42.9%, 66.7%, 21.4%, and 84.6%. Diabetes-stratified performance was similarly low. Participants with diabetes had 19.2% sensitivity, 89.5% specificity, 19.2% PPV, and 89.5% NPV, whereas participants without diabetes had 21.7%, 90.9%, 16.7%, and 93.3%.

Conclusions:

FIB-4 ≥ 1.30 demonstrates low sensitivity but high specificity for detecting significant fibrosis in MASLD/MASH, with sensitivity increasing and specificity decreasing with age. Low sensitivity across diabetes-defined subgroups indicates that many

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individuals with elevated LSM are missed by FIB-4, suggesting that FIB-4 underestimates the at-risk population, particularly younger adults, and highlight the need for TE-based imaging or biomarker-based approaches to improve fibrosis detection.

Keyword(s) : MASLD, MASH, NAFLD, NASH, TE, FIB-4, Liver Fibrosis, Saudi Arabia, Diabetes, Cardiometabolic risk

1942 - Long term outcomes of chronic hepatitis B Effect of metabolic risk factors on liver stiffness measurement by vibration controlled transient elastography

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Background / Hypothesis: The coexistence of chronic hepatitis B(CHB)and metabolic dysfunction-associated steatotic liver disease (MASLD)is common in clinical practice.This condition,referred to as "dual- etiology liver disease" leads to higher complication rates and increased management complexity. This relationship suggests a model called the "two-hit hypothesis". According to this hypothesis, MASLD-associated insulin resistance,oxidative stress and chronic inflammation may accelerate fibrosis progression by triggering HBV-induced liver injury. Vibration-Controlled Transient Elastography (VCTE)is a non-invasive, rapid and reproducible method that measures liver steatosis (Controlled Attenuation Parameter-CAP) and stiffness (fibrosis, kPa value). The aim of this study was to determine the effects of metabolic risk factors and hepatic steatosis detected by VCTE on liver fibrosis in patients with CHB.

Materials and Methods:The study included 103 CHB patients who were followed at our clinic. Patients with HDV, HCV, or HIV co-infection, those with daily alcohol consumption, and those with missing data were excluded.

Patients were divided into two groups: Metabolic risk factors (MRF) group (patients with a diagnosis of DM, HT, or HL, or those with a BMI $\geq$ 25 kg/m²) and control group (patients who did not have any of these risk factors). Additionally, patients were compared based on their VCTE steatosis grades (Significant steatosis:

S2-S3 vs. Minimal/No steatosis: S0-S1). Liver stiffness measured by VCTE(kPa, rates of advanced fibrosis ($\geq$ F3)

Results:The mean age of the 103 patients was 51.2 $\pm$ 13.4 years, and 65% were male, 42% had a metabolic risk factor and 38% had significant steatosis (S2-S3. The mean VCTE kPa value was significantly higher in patients with MRF (7.9 $\pm$ 4.1) compared with the control group (5.2 $\pm$ 2.3). The rate of advanced fibrosis was also significantly higher in the MRF group (25.4%) compared with the control group (8.9%)(Table1). Similarly, in patients with significant steatosis (S2-S3), the mean kPa value (9.1 $\pm$ 4.5) was higher than in those with minimal steatosis (5.8 $\pm$ 2.7). A significant positive correlation was found between body mass index (BMI) and VCTE kPa value as well as between waist circumference and VCTE kPa value (Table 2).

Conclusions: In conclusion, this study demonstrates that the coexistence of metabolic syndrome and MASLD in patients with HBV is a significant and modifiable risk factor for the progression of liver fibrosis. This suggests that metabolic status should be central to clinical follow-up. Therefore, to improve the long-term health of patients, the clinical approach should focus on optimizing the patient's metabolic profiles in addition to achieving viral suppression.

Keyword(s) : Vibration-Controlled Transient Elastography, Liver stiffness, Metabolic risk factors

1944 - Baveno VII-Defined Hepatic Recompensation in Children with Decompensated Cirrhosis Frequency and Determinants

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Background / Hypothesis:

Decompensated cirrhosis has long been considered irreversible phenomenon. With effective etiological treatment, however, some may achieve "recompensation"—sustained reversal of decompensation with functional recovery. Currently, literature in pediatrics is limited. We aimed to assess

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incidence, predictors, and outcomes of recompensation in children as per Baveno-VII definition.

Materials and Methods:

An ambispective cohort study was conducted in children <18 years with decompensated Wilson disease and autoimmune hepatitis (AIH). Recompensation was defined using modified Baveno VII criteria: aetiological disease control, sustained remission of decompensating events with restored liver function.

Results:

Of 79 Wilson disease and 78 AIH decompensated children with ≥ 1 -year follow-up, recompensation occurred in 88.6% and 69.2%, respectively. In both cohorts, recompensation conferred superior native liver survival ($p < 0.05$). Smaller spleen size, higher platelet/total leukocyte counts, lower alanine aminotransferase levels and lower liver stiffness predicted recompensation in Wilson disease; spleen size was independently predictive of recompensation in AIH. Re-decompensation was infrequent (≈ 11 –13%), largely due to non-adherence. Recovery typically followed the trend: ascites $\rightarrow$ jaundice $\rightarrow$ international normalised ratio $\rightarrow$ serum albumin normalization. Baseline exploratory cytokine profiling suggested higher levels of plasma IL-10 and higher stool IL-6 in Wilson cohort and elevated stool IL-1 β /IL-10 and lower plasma IL-6 in AIH cohort recompensators.

Conclusions:

Recompensation is achievable and durable in pediatric Wilson disease and AIH with appropriate aetiology-directed therapy. Non-invasive markers of portal hypertension (spleen size, platelets, and liver stiffness) may stratify likelihood of reversal and also track recovery. Paired stool–plasma cytokines further merit validation as adjunctive biomarkers.

Keyword(s) : recompensation; Wilson disease; autoimmune hepatitis; pediatric cirrhosis; portal hypertension; elastography

1946 - Comprehensive Assessment of HBV Reactivation in Immunosuppressed Hematology Patients Serological and Clinical Perspectives

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Background / Hypothesis: The aim of this study is to determine the risk of hepatitis B virus (HBV) reactivation in hematology patients receiving immunosuppressive therapy, to evaluate the clinical outcomes of patients who developed reactivation, and to assess the effectiveness of preemptive and prophylactic treatment practices

Materials and Methods: Patients ≥ 18 years receiving immunosuppressive therapy at a tertiary center in İzmir (2013–2023). Data on demographics, diagnosis, treatments, labs (HBsAg, anti-HBc, anti-HBs, HBV DNA, anti-HBe, HBeAg, ALT), antiviral prophylaxis/treatment, HBV reactivation, and outcomes were obtained from hospital records.

Results: A total of 804 patients (mean age 57.14 ± 15.34 years; 53.4% male) were included. HBV exposure was identified in 282 patients (35%): 9 HBsAg positive, 5 isolated anti-HBc IgG positive, and 9 with past immunity (HBsAg negative, anti-HBc IgG and anti-HBs positive). HBV reactivation occurred in 23 patients, 8 during and 15 after immunosuppressive therapy. The timing of post-treatment reactivation was observed in the 3rd, 4th, 5th, 6th, 7th ($n=4$), 10th, 14th, 22nd, 23rd, 24th, 36th, and 60th months after cessation of therapy. Six of these 15 patients had not received antiviral therapy. Four patients were under antiviral treatment; however, one of them did not regularly use tenofovir disoproxil fumarate (TDF), while the others were on lamivudine therapy. In five patients, antiviral therapy was initiated but discontinued before the completion of immunosuppressive therapy or not continued for an adequate period after treatment cessation.

Reactivation was most frequent with rituximab (14/23, 60.8%) and ibrutinib (5/23, 21.7%), followed by doxorubicin (2/23, 8.6%), high-dose corticosteroids (1/23, 4.3%), and ruxolitinib (1/23, 4.3%). Eighteen patients showed increased HBV DNA, four developed HBsAg seroreversion, and one was diagnosed via liver biopsy.

Among those with past immunity, six patients had anti-HBs titers between 10–99 IU/mL, two between 100–299 IU/mL, and only one patient had a titer above 300 IU/mL. During follow-up, 10 of the 23 patients (43.5%) who developed HBV reactivation died; three deaths (13.0%) were associated with hepatic failure and fulminant hepatitis.

Conclusions: The prevention of hepatitis B virus reactivation in hematology patients receiving immunosuppressive therapy relies on regular monitoring, appropriate antiviral prophylaxis, and a multidisciplinary approach. In our study, no reactivation occurred in patients who received

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tenofovir or entecavir correctly for the appropriate duration, highlighting the effectiveness of proper antiviral management. These findings emphasize the importance of collaboration for patient safety and treatment success, and are expected to guide future large-scale, prospective research and strengthen standardized clinical practices.

Keyword(s) : HBV

reactivation, Immunosuppression, Hematology, Rituximab

1947 - Hepatocellular Carcinoma A Seven Year Cohort Revealing Large Tumor Size and Advanced Fibrosis Despite Early BCLC Staging

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a major cause of cancer-related mortality, mainly occurring in patients with advanced fibrosis or cirrhosis. In our region, viral hepatitis continues to be the predominant etiology. This study aimed to describe the demographic, hepatic, and tumor characteristics of patients diagnosed with HCC in our center and to analyze associations between fibrosis severity and tumor/clinical presentation.

Materials and Methods:

A retrospective descriptive study was conducted including all patients diagnosed with HCC between 2019 and 2025. Collected data included demographics, risk factors, fibrosis stage, Child-Pugh and MELD scores, clinical presentation, AFP, MRI characteristics (LI-RADS), number and size of nodules, and BCLC classification. Comparative analysis between severe fibrosis (F3-F4) and mild/no fibrosis (F0-F2) was performed.

Results:

Twenty-three patients were included (mean age 64 years; M/F ratio = 1.3). At least one toxic risk factor (smoking or alcohol) was present in 48% of cases. Severe fibrosis (F3-F4) was observed in 60.7% of patients. Viral hepatitis accounted for 69.6%

of etiologies (HCV 52.2%, HBV 17.4%).

Portal hypertension signs were present in 60% of patients. Among those with F3-F4 fibrosis, 64.3% were Child-Pugh B-C, compared to none in F0-F2. The median MELD score was 11.

AFP levels were less than 20 ng/mL in 34.8%, 20-200 in 30.4%, and > 200 in 17.4%.

MRI identified LI-RADS 5 lesions in 95.4% of evaluated patients. A single nodule was observed in 69.6% of cases, yet tumors were large (median size 58 mm [15-100]), with 82% exceeding 20 mm.

Segment VIII was the most common localization.

Multifocal involvement was present in 34.8%.

According to BCLC staging, stage A was the most prevalent.

Comparative analysis showed:

- Nodules <20 mm were more frequent in F3-F4 (91%) than F0-F2 (67%).
- Child-Pugh B-C occurred exclusively in F3-F4 (64.3% vs. 0%).
- AFP >200 ng/mL was more common in F3-F4 (22% vs. 11%).

Conclusions:

HCC in our cohort was predominantly related to chronic viral hepatitis and frequently associated with severe fibrosis at diagnosis. Patients with advanced fibrosis presented significantly larger tumors and more impaired liver function. Despite large tumor size, many patients remained within early BCLC stages. These findings highlight gaps in surveillance programs and emphasize the need for strengthened screening strategies in high-risk populations to promote earlier detection and improve therapeutic outcomes.

Keyword(s) :

1948 - Genetic and metabolic characteristics of metabolic dysfunction-associated steatotic liver disease in a pilot subset of a population based cohort study in Saudi Arabia

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is highly prevalent across the Middle East, yet the genetic determinants of susceptibility in this population remain poorly characterized. Although several MASLD-associated variants have been established in Western populations, their relevance in Middle Eastern cohorts is unclear. We hypothesized that a pilot subset of the Genomics and Environmental Noninvasive Evaluation for Saudi Intrahepatic Steatosis (GENESIS) cohort would demonstrate genotype-phenotype patterns consistent with established MASLD biology

Materials and Methods:

GENESIS is a nationwide population-based cohort of 2,983 Saudi adults evaluating MASLD prevalence and determinants. We analyzed a pilot subset of 91 participants with available transient elastography (FibroScan®) and targeted genotyping data. MASLD was defined as controlled attenuation parameter ≥ 268 dB/m plus at least one cardiometabolic risk factor. Clinical, biochemical, and genetic characteristics were compared between MASLD (n=46) and non-MASLD (n=45) participants. Variants assessed included PNPLA3 p.I148M, TM6SF2 p.E167K, GCKR p.Leu446Pro, HSD17B13 rs72613567, and MARC1 p.A165T.

Results:

Participants in the MASLD group were older than those without MASLD (mean age: 48.5 vs 41.0 years, $p=0.005$), and gender distribution was comparable (male: 47.8% vs 31.1%, $p=0.134$). The MASLD group had a higher mean body mass index (32.6 vs 27.9 kg/m², $p<0.001$), higher prevalence of dyslipidemia (43.5% vs 11.1%, $p<0.001$) and diabetes (37.0% vs 8.9%, $p=0.001$), higher median triglycerides (1.9 vs 1.0 mmol/L, $p<0.001$) and higher median insulin levels (18.1 vs 9.9 μ U/mL, $p<0.001$).

Genotype distributions showed observed differences between groups. For PNPLA3 p.I148M, wild-type genotypes were more common in MASLD (23.9% vs 11.1%), whereas heterozygous (32.6% vs 35.6%) and homozygous variants (43.5% vs 53.3%) were more frequent in non-MASLD ($p=0.268$). For GCKR p.Leu446Pro, homozygous variant carriers were more prevalent in MASLD (30.4% vs 13.3%), while heterozygous carriers were more common in non-MASLD (44.4% vs 32.6%) ($p=0.134$). TM6SF2 p.E167K heterozygosity was similar between groups (15.2% vs 11.1%, $p=0.758$). The protective HSD17B13 variant remained predominantly homozygous in both groups (71.7% vs 73.3%, $p=0.561$). For MARC1 p.A165T, Wild-type carriers were more common in MASLD (50.0% vs 31.1%), heterozygous variants were more frequent in non-MASLD (60.0% vs 37.0%), and homozygous variants were slightly higher in MASLD (13.0% vs 8.9%) ($p=0.088$).

Conclusions:

This pilot analysis demonstrates distinct metabolic and genetic patterns associated with MASLD within the Saudi population. While limited by sample size, the distribution of risk and protective alleles aligns with established biological pathways and supports expanding genomic and multi-omics analyses across the full GENESIS cohort

Keyword(s) : MASLD, PNPLA3, TM6SF2, GCKR, HSD17B13, MARC1, steatosis, CAP, genetics, Saudi Arabia, multi-omics

1951 - Clinical Laboratory and Microbiological Profile of Post-ERCP Infections A Retrospective Study from a Single Tertiary Referral Center

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Background / Hypothesis:

Endoscopic retrograde cholangiopancreatography(ERCP) is an invasive

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procedure widely used for the diagnosis and treatment of biliary and pancreatic diseases. Infectious complications following ERCP can lead to significant morbidity and mortality. In this study, we compared the clinical, laboratory, and microbiological characteristics of patients who developed post-ERCP infections with mild versus severe clinical courses.

Materials and Methods:

A total of 1092 patients who underwent ERCP at Bezmialem Vakıf University Hospital between January 1, 2023, and October 1, 2025, were retrospectively reviewed. Sixty-two patients (5.67%) who developed ERCP-associated infections were included in the study. Patients were categorized into two groups based on clinical course: Mild and severe infections. Severe infection was defined as the development of sepsis, the need for intensive care, or infection-related mortality. Demographic data, comorbidities, ERCP indications, procedural characteristics, laboratory values, microbiological findings, and clinical outcomes were retrospectively collected from patient records.

Results:

Of the 62 patients who developed post-ERCP infection, 75.8% were female (n=47) and 24.2% were male (n=15). The mean age was 63.63 ± 16.04 years (27–94), and the mean BMI was 25.54 ± 3.75 kg/m². The most common ERCP indication was choledocholithiasis (33.9%; n=21), followed by malignant biliary stricture (22.6%; n=14), benign biliary stricture (17.7%; n=11), pancreatic causes (8.1%; n=5), and other indications (17.7%; n=11). Post-ERCP complications included sepsis in 51.6% (n=32), bacteremia in 48.4% (n=30), pancreatitis in 16.1% (n=10), perforation in 12.9% (n=8), and cholangitis in 9.7% (n=6). Blood cultures were obtained from 96.8% of patients (n=60), with growth in 48.3% (n=30). Among positive cultures, *Escherichia coli* (E.coli) accounted for 46.7% (n=14), *Klebsiella pneumoniae* for 20.0% (n=6), *Pseudomonas aeruginosa* for 16.7% (n=5), and other organisms for 16.7%. Extended spectrum beta-lactamase (ESBL) positivity was 85.7% in E. coli isolates (n=12). In *Klebsiella pneumoniae* ESBL positivity was 16.7% (n=1), multidrug-resistance (MDR) 33.3% (n=2), and extensively drug-resistant 16.7% (n=1). The 30-day mortality rate was 6.5% (n=4). There were no significant differences between mild and severe infection groups regarding ASA score, Charlson comorbidity index, number of procedures, or hospital stay (all p>0.05). Immunosuppressive therapy, higher GGT, elevated procalcitonin, and positive blood cultures were significantly more common in severe infections (p<0.05) while post-ERCP pancreatitis was

more frequent in mild cases (p<0.05).

Conclusions: In post-ERCP infections, severe clinical course is more closely linked to host-related factors, systemic inflammatory burden, and resistant pathogens rather than procedural characteristics. High antimicrobial resistance rates highlight the need for individualized empirical antibiotic selection based on local resistance patterns and patient risk.

Keyword(s) : Endoscopic retrograde cholangiopancreatography (ERCP), pancreatitis, sepsis, cholangitis

1952 - Performance of anti HCV screening and identification of false positive results in a real life cohort

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Background / Hypothesis: The prevalence of hepatitis C virus (HCV) infection is determined by the rate of positivity of anti-HCV antibody. Anti-HCV is a widely used first-line screening test; however, false-positive reactivity remains a major diagnostic challenge, particularly in low-prevalence settings. Differentiating true infections, previously treated infections, and false-positive serology is essential to avoid unnecessary follow-up and antiviral therapy. We hypothesized that the proportion of false-positive anti-HCV results is substantial in real-life practice and that anti-HCV signal-to-cut-off ratio (s/co) levels may aid in distinguishing confirmed infection from false positivity.

Materials and Methods: This retrospective study included 1222 anti-HCV-positive adults tested between January 2020 and December 2023 at a tertiary care university hospital. During the study period, a total of 117,059 anti-HCV tests were performed, of which 1,222 were positive, yielding an overall seropositivity prevalence of 1.04%. HCV RNA results and antiviral treatment information were obtained from the hospital's electronic database. Anti-HCV testing was performed using a chemiluminescent microparticle immunoassay (CMIA) on the Abbott Alinity i platform. Quantitative HCV RNA testing was conducted using a real-time PCR-based assay on the Abbott m2000 system (m2000sp/m2000rt). False-positive anti-HCV was defined as anti-HCV-positive, HCV RNA-negative

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individuals with no history of antiviral therapy. Among the 1,222 anti-HCV-positive patients, 694 (56.8%) were female and 528 (43.2%) were male. Anti-HCV S/CO distributions, categorical S/CO bands, and ROC analysis (AUC and optimal threshold using the Youden index) were performed.

Results: Among 1222 anti-HCV-positive patients, HCV RNA, was not performed in 394 (32.2%), while 828 (67.8%) underwent RNA testing. Active HCV infection was confirmed in 110 patients (13.3%); the remaining 718 (86.7%) were RNA negative. A total of 536 RNA-negative patients without treatment history met criteria for false-positive results, representing 74.6% of all RNA-negative cases and 43.9% of all anti-HCV-positive patients. Median s/co was 17.4 in RNA-positive cases, compared with 3.1 in RNA-negative and 2.0 in false-positive cases. Among false-positive patients, 65.1% had s/co <3 and 92.9% had s/co <10. ROC analysis demonstrated an AUC of 0.86, with the optimal s/co cut-off at 7.34 (sensitivity 96.3%, specificity 69.8%).

Conclusions: In this real-life cohort from 2020–2023, nearly half of all anti-HCV-positive results were false positive. Anti-HCV s/co values showed strong discriminative capacity, and low s/co levels—particularly <3 and <10—were highly indicative of false positivity. Incorporating s/co interpretation into routine screening workflows may reduce unnecessary RNA testing and prevent misclassification, improving diagnostic accuracy and resource utilization.

Keyword(s) : Anti-HCV, Hepatitis C, False-positive, Seroprevalence

1953 - Chronic Dexamethasone Drives Selective SNAT Remodeling to Fuel HNF4 Alpha Mediated Gluconeogenesis

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Background / Hypothesis: Following their synthetic development, glucocorticoids have become life-saving agents used across a wide range of clinical indications. However, despite their broad therapeutic utility, they are associated with numerous adverse effects that require careful consideration. Among these, one of the most significant is hyperglycemia resulting from increased hepatic gluconeogenesis. Glucocorticoids are

master regulators of hepatic gluconeogenesis, a critical survival pathway largely orchestrated by the transcription factor Hepatocyte Nuclear Factor 4 alpha (HNF4- α). To sustain the high rate of glucose production induced by glucocorticoids, the liver requires a continuous supply of key gluconeogenic precursors, specifically alanine and glutamine. This uptake is mediated by Sodium-coupled Neutral Amino Acid Transporters (SNATs). However, it remains unclear whether chronic glucocorticoid exposure actively remodels the hepatic transporter profile to meet this metabolic demand. We hypothesized that chronic glucocorticoid exposure establishes a coordinated transcriptional axis where the upregulation of HNF4- α drives the activation of specific SNAT subtypes, thereby ensuring the necessary amino acid influx to fuel enhanced hepatic gluconeogenesis.

Materials and Methods: Male rats were divided into four groups (n=7): one control and three receiving dexamethasone (1, 2, or 4 mg/kg/day, intraperitoneal, 21 days). Liver tissues were collected, and mRNA expression of HNF4- α and SNAT2, SNAT3, SNAT4, SNAT7, SNAT9, and SNAT10 was quantified by Real-Time PCR. Treated groups were statistically compared with the control.

Results: Dexamethasone produced marked, dose-dependent transcriptional remodeling. HNF4- α expression increased progressively, indicating activation of the gluconeogenic program. In parallel, SNAT2, SNAT3, and SNAT4—key transporters of alanine and glutamine—were significantly upregulated in a dose-dependent manner, peaking at 4 mg/kg (p<0.05). Conversely, SNAT7 and SNAT9 were significantly suppressed with increasing doses, while SNAT10 showed no change across groups.

Conclusions: The study demonstrates that chronic dexamethasone exposure orchestrates a selective metabolic remodeling in the liver. The upregulation of SNAT2, 3, and 4 parallels the rise in HNF4- α , suggesting that the liver adaptively increases transport capacity to ensure sufficient alanine and glutamine availability for enhanced gluconeogenesis. Conversely, the concurrent downregulation of SNAT7 and 9 likely reflects a strategic metabolic trade-off, where the liver suppresses non-essential transport pathways to conserve energy and prioritize gluconeogenic flux. Therefore, therapeutic interventions targeting these upregulated transporters could offer a potential strategy to mitigate the metabolic side effects, particularly hyperglycemia, associated with glucocorticoid therapy.

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Keyword(s) : Dexamethasone, HNF4- α , SNATs, Gluconeogenesis, Liver, Amino Acid Transport

1955 - The overlooked Threat on the Path to Hepatitis B Eradication Delta Hepatitis

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Background / Hypothesis:

Globally, the prevalence of Hepatitis B has decreased significantly as a result of the elimination programs conducted by the World Health Organization (WHO). Delta co-infection tends to be overlooked in Hepatitis B infections and is anticipated to give a considerable health burden due to probable consequences in the forthcoming years. Therefore, it is critical for those diagnosed with Hepatitis D infection to receive a diagnosis promptly, and our study aims to evaluate the level and rate of screening for Delta co-infection in individuals infected with Hepatitis B.

Materials and Methods:

This retrospective analysis evaluated all individuals who underwent HBsAg testing at our hospital from May 27, 2020, to September 30, 2025. Duplicate tests were omitted, and each participant was evaluated solely once. No age limitations were imposed. Results of the Anti-HDV test were evaluated in HBsAg-positive patients, and HDV RNA testing was conducted in those identified as anti-HDV positive. Results for HBsAg, anti-HDV, and HDV RNA were retrieved from the laboratory information management system.

Results:

A total of 885,150 individuals underwent HBsAg testing, with a positive rate of 1.78%. Among the HBsAg-positive individuals, 9,139 (57.87%) were male and 6,652 (42.13%) were female ($p < 0.001$). The positivity rate for HBsAg tests was 2.4% in individuals aged 28 and older, compared to 0.4% in those aged 27 and younger ($p < 0.001$). Anti-HDV testing was conducted in 46.1% of HBsAg-positive patients, with an anti-HDV positivity rate of 4.33% (315 cases). HDV RNA positivity was identified in 91 (29%) of the 315 patients who tested positive for anti-HDV. Of the HDV RNA-positive patients, 58 were male and 33 were female, and this gender difference was statistically significant ($p < 0.01$).

Conclusions:

Our analysis indicates that about 1% of Turkey's population of 85 million has been tested for HBsAg, signifying the attainment of extensive community-wide screening coverage. The study's findings indicate that HBsAg positivity rates among individuals aged 27 and younger are statistically significantly low. This scenario is believed to demonstrate the enduring protective impact of the national hepatitis B vaccination program established in 1998.

Although annual anti-HDV screening is recommended in current guidelines for HBsAg-positive patients, the absence of delta antibody level measurements in over half of the patients indicates that delta infection is being neglected in clinical practice. We believe that this issue could be resolved with the integration of "double reflex test" (automated testing for anti-HDV and, if warranted, HDV RNA in individuals identified as HBsAg-positive).

Keyword(s) : Delta Hepatitis, hepatitis B

1956 - Comparative Evaluation of Serological Virological and Biochemical Outcomes in Chronic Hepatitis B Patients Receiving Tenofovir or Entecavir

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Background / Hypothesis: This study aimed to evaluate changes in HBeAg loss, HBV-DNA suppression, ALT normalization, and other biochemical parameters in chronic hepatitis B (CHB) patients receiving long-term potent antiviral therapy with tenofovir or entecavir.

Materials and Methods: This retrospective study was conducted in tertiary care hospital located in Izmir, Turkey and included patients diagnosed with CHB who initiated treatment between January 2008 and January 2018. Patients who were received potent antiviral treatment (either tenofovir or entecavir (ETV) for a

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minimum of five years were enrolled and grouped. Long-term treatment responses—including HBeAg loss, HBV DNA suppression, ALT normalization, and biochemical parameter changes were compared between the two treatment groups.

Results: A total of 218 non-sirotic CHB patients who underwent treatment (tenofovir group (n=90) and ETV group (n=128)) for a minimum of five years were included. Long-term potent antiviral therapy resulted in significant reductions in HBV DNA levels and improvements in ALT and AST values. There were no significant differences between tenofovir and entecavir groups regarding HBeAg loss or anti-HBe seroconversion. Virological and biochemical response rates were similar in both treatment groups (Table).

Conclusions: Long-term potent antiviral therapy yields favorable virological, biochemical, and serological outcomes in patients with CHB. The findings indicate that tenofovir and ETV demonstrate comparable efficacy in long-term treatment.

Keyword(s) : Chronic hepatitis B; Tenofovir; Entecavir; Long-term potent antiviral therapy

1557 - HBx Upregulates the Interferon Negative Regulator IFITM2 via Activating the JAK STAT Pathway and Impairing Lysosomal Function

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Background / Hypothesis:

Interferon-induced transmembrane protein 2 (IFITM2) is a key negative regulator in Peg-IFN α therapy. HBV has been shown to increase IFITM2 expression and therefore suppress endogenous IFN production. This study aimed to elucidate the mechanisms by which HBV regulates IFITM2, thereby providing insights for optimizing Peg-IFN α efficacy.

Materials and Methods:

Plasmid containing 1.3 copies of the full-length HBV genome or encoding HBx, HBc, or HBs were transfected into Huh7 cells. The STAT1 inhibitor

Fludarabine was applied to block JAK-STAT pathway signaling. IFITM2 expression and JAK-STAT pathway components were assessed by RT-qPCR and western blotting. Nuclear expression and translocation of interferon-stimulated gene factor 3 (ISGF3) were assessed by immunofluorescence and quantified using the Opera Phenix Plus High-Content Screening System. IFITM2 protein stability was evaluated using cycloheximide (CHX) chase assays. Colocalization of IFITM2 with the endo-lysosomal system was analyzed via confocal microscopy. Lysosomal acidity and membrane stability were evaluated using pH-dependent lysosomal probes and acridine orange (AO) staining, respectively.

Results:

HBV and HBx markedly increased IFITM2 transcription and translation. HBV and HBx also induced STAT1, STAT2, IRF9, and major ISGs (ISG15, USP18, MxA), along with increased phosphorylation of STAT1 (S727, Y701) and STAT2 (Y690). Fludarabine suppressed HBV/HBx-induced STAT1 activation, and IFITM2 expression changed in parallel with STAT1 phosphorylation. HBV and HBx enhanced nuclear translocation of pSTAT1, while pSTAT2 and IRF9 showed no significant differences. IFITM2 protein exhibited a half-life of 11.04h, and its degradation after 24 h of CHX treatment was limited to 10.8% and 13.3% in the HBV or HBx expressing groups, respectively. Both proteasome and lysosome inhibition prevented IFITM2 degradation, with lysosomal suppression exerting the predominant effect. IFITM2 protein colocalized with early endosomal marker Rab5, late endosomal marker Rab7 and lysosomes. HBV and HBx impaired lysosomal proteolysis by reducing lysosomal accumulation and acidification, and destabilizing lysosomal membranes, as shown by reductions in AO red fluorescence of $39.38 \pm 5.59\%$ (control), $54.37 \pm 3.46\%$ (HBV), and $49.88 \pm 6.96\%$ (HBx) at 140 min.

Conclusions:

HBx upregulated IFITM2 by activating the JAK-STAT pathway and inhibiting lysosomal degradation. HBx promotes ISGF3 nuclear expression and pSTAT1 nuclear translocation, while also prolonging IFITM2 protein half-life through impaired lysosomal acidification and membrane stability. These findings identify dual mechanisms by which HBx sustains IFITM2 elevation and suggest potential strategies to enhance Peg-IFN α therapeutic responses.

Keyword(s) : Interferon-induced transmembrane

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protein 2, HBV encoding proteins, JAK-STAT pathway, protein degradation, endosome-lysosome system

1958 - Optimizing operator competency in transient elastography for metabolic dysfunction associated steatotic liver disease screening in a multicenter study

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Background / Hypothesis:

Accurate non-invasive assessment of liver steatosis and fibrosis is essential for effective screening of metabolic dysfunction-associated steatotic liver disease (MASLD). Transient elastography (TE) is widely adopted, yet its diagnostic reliability highly depends on operator competency. This study aimed to develop and validate a structured, reproducible TE training protocol for non-expert healthcare workers across multiple centers.

Materials and Methods:

A prospective multicenter study was conducted from March 2024 to May 2025 across six centers in three major regions in Saudi Arabia. A hepatology-led expert committee developed a standardized TE training protocol using FibroScan®. Nine healthcare workers with no prior TE experience (research coordinators) underwent structured, hands-on training delivered by nine certified supervisors. Training included 100 supervised and 10 unsupervised TE examinations with

real-time feedback. Supervisors, blinded to unsupervised trainee results, performed parallel assessments on the same individuals during the validation phase. Measurements included controlled attenuation parameter (CAP), liver stiffness measurements (LSM), and the interquartile range (IQR). LSM reliability was defined as an IQR-to-median ratio <30%. Inter-operator reproducibility was assessed using intraclass correlation coefficients (ICCs), with >0.80 indicating strong reliability.

Results:

Trainees (55.6% female) had a median age of 28 years (range: 23–38) and four years of clinical experience (range: 0–13). Their professional backgrounds included nursing (44.4%), radiology (22.2%), genomics (22.2%), and pharmacy (11.1%). Supervisors (88.9% female) were from diverse professional backgrounds: pharmacists (33.3%), hepatologist/gastroenterologists (22.2%), ultrasound technologist (11.1%), biochemist (11.1%), and genomics specialist (11.1%). The median training duration was 20 days (range: 14–22), with a median of six hours/day (range: 4–8). Each trainee was supervised by a median of two supervisors (range: 1–3). Seven trainees (77.8%) achieved competency on first attempt. Inter-operator agreement was excellent, with a median ICC for CAP was 0.95 (IQR: 0.92–0.98) and 0.96 (0.82–0.99) for LSM. Median IQR-to-median ratios were comparable between trainees (13.5%) and supervisors (14.0%), indicating high measurement reliability.

Conclusions:

A structured, short-duration TE training protocol involving 100 supervised TE assessments over 20 days can effectively train non-expert operators to achieve expert-level proficiency. This standardized protocol enhances reliability of TE measurements and provides a scalable model for MASLD screening programs in diverse healthcare settings.

Keyword(s) : MASLD, MASH, Screening, Transient Elastography, Controlled Attenuation Parameter, CAP, Liver Stiffness Measurement, LSM

1959 - Investigation of the Effects of Nintedanib in Liver Damage Induced by CCl4 in Rats

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Background / Hypothesis: This study aims to evaluate the potential effect of administering Nintedanib, a multiple tyrosine kinase inhibitor with drug-induced liver side effects, at three different times in a carbon tetrachloride (CCl₄)-induced liver damage model.

Materials and Methods: Seventy-two rats were randomly assigned to six groups, each consisting of 12 animals. The healthy group received 1 ml of olive oil intraperitoneally, while the Nintedanib group received 100 mg/kg nintedanib orally. A liver injury model was created in the other four groups using 1 mg/kg CCl₄. Three of these groups received a single dose of 100 mg/kg nintedanib 12 hours before, 30 minutes before, and 12 hours after CCl₄ administration. Half of the rats in each group (n=6) were sacrificed 24 hours after CCl₄ administration, and the other half (n=6) were sacrificed 1 week later. Blood and liver samples were collected after sacrifice. Serum ALT and AST levels were measured; TNF- α and IL-6 were measured in liver tissue by ELISA, and TGF- β and α -SMA gene expression was measured by RT-PCR. In addition, histopathological and immunohistochemical examinations were performed, and TGF- β staining was carried out.

Results: On day 1, in the CCl₄ group where liver injury was induced in sacrificed rats, serum ALT and AST levels, tissue IL-6 and TNF- α levels, and TGF- β and α -SMA gene expression were increased compared to the healthy group. In the group administered Nintedanib 12 hours prior to CCl₄ administration, these parameters increased compared to both the healthy group and the CCl₄ group. These findings are also supported by histopathological evaluation. In rats sacrificed one week after liver damage was induced, these parameters were measured at values similar to those in the healthy group.

Conclusions: Liver injury in rats sacrificed on day 1 was increased in the group given nintedanib 12 hours prior to CCl₄ administration compared to the group treated with CCl₄ alone. The increased injury was resolved in the rats sacrificed at the end of the first week.

Nintedanib should be used with caution when administered with hepatotoxic agents due to the risk of drug-induced liver injury, or in individuals with underlying liver disease.

Keyword(s) : Nintedanib, Drug Induced Liver Injury, CCl₄, TGF- β , inflammation

1960 - High risk of obstructive sleep apnea among adults with metabolic dysfunction associated steatotic liver disease based on findings from a national population study

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is strongly linked with obesity, diabetes, and metabolic syndrome. Sleep disturbances, particularly obstructive sleep apnea (OSA), are increasingly recognized as contributing to the pathogenesis of metabolic diseases, including MASLD. This study aimed to evaluate the prevalence of high OSA risk among adults with and without MASLD in a large, population-based Saudi cohort using the validated Berlin Questionnaire.

Materials and Methods:

Data were drawn from the GENESIS study, a prospective, population-based cohort conducted across three major regions of Saudi Arabia. MASLD was defined using transient elastography with a controlled attenuation parameter (CAP) ≥ 268 dB/m plus ≥ 1 cardiometabolic risk factor. Between March 2024 and

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June 2025, participants underwent clinical assessment for MASLD and completed the Berlin Questionnaire, which categorizes OSA risk into three domains: snoring/apnea (Category 1), fatigue/sleepiness (Category 2), and body mass index (BMI) $>30 \text{ kg/m}^2$ or hypertension (Category 3). BMI was measured through direct clinical measurement; all other questionnaire items were self-reported. High OSA risk was defined as positive scores in ≥ 2 categories.

Results:

Among 2,983 adults included, 1,111 (37.2%) had MASLD and 1,872 (62.8%) did not. High OSA risk was significantly more prevalent in the MASLD group (37.0% vs. 17.0%, $p<0.001$). Among Berlin Questionnaire categories, participants with MASLD showed higher rates in Category 1 (snoring/apnea: 29.8% vs. 16.0%; $p<0.001$) and Category 3 (BMI/hypertension: 71.6% vs. 36.9%; $p<0.001$), while Category 2 (fatigue/sleepiness) did not differ significantly (24.9% vs. 23.3%; $p=0.35$). Self-reported OSA diagnosis was low and comparable between groups (3.6% vs. 3.0%, $p=0.42$).

Conclusions:

Adults with MASLD have a substantially higher Berlin Questionnaire-defined OSA risk, mainly driven by elevated BMI/hypertension and snoring/apnea. The low rate of self-reported OSA highlights its under-recognition in this population. These results support incorporating sleep disorder screening into MASLD management, enabling early identification and intervention to potentially prevent disease progression.

Keyword(s) : MASLD, NAFLD, OSA, Berlin Questionnaire, Sleep Disorders

1961 - HBV Reactivation in Rheumatology Patients With Resolved Hepatitis B Infection Receiving DMARD Therapy

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Background / Hypothesis: Hepatitis B virus reactivation (HBVr) remains an important clinical concern in patients with rheumatologic diseases receiving

immunosuppressive therapy. The risk of HBVr varies depending on the potency and duration of immunosuppression, the underlying rheumatologic condition, and the patient's baseline virological profile. This study aimed to evaluate the risk of HBVr in rheumatology patients treated with disease-modifying antirheumatic drugs (DMARDs).

Materials and Methods: A retrospective review was conducted at Etlik City Hospital between October 2022 and 2025. Patients receiving any form of DMARD therapy who had resolved HBV infection (HBcAb-positive, HBsAg-negative) were included. HBV reactivation was defined as either an increase in HBV DNA levels or HBsAg seroreversion. Risk stratification was performed according to current international guideline recommendations.

Results: A total of 1094 patients with documented rheumatologic diseases, treatments, and baseline HBV status were screened. HBsAg was positive in 2.6% ($n = 29$) of patients, while 23 % ($n = 253$) had anti-HBc IgG positivity. Among the 253 patients 224 patients with resolved infection were included in the study. The mean age was 57 years and 135 (60%) of them were female. The number of patients with anti HBs positivity was 186 (83%). No detectable HBV DNA was observed in patients with resolved HBV infection. Antiviral prophylaxis was administered to 92 of the patients (41%) and 132 (59%) of them were monitored without prophylaxis. No cases of HBVr were observed during the immunosuppressive therapy in any group (mean follow-up: 16 months, min 6-max36). Among the therapies administered, bDMARDs included TNF inhibitors ($n=135$), cytokine/integrin inhibitors ($n=13$), anti-IL-1 agents ($n=6$), a T-cell costimulation inhibitor ($n=6$), and an anti-IL-6 agent ($n=5$); tsDMARDs consisted of Janus kinase inhibitors ($n=10$); csDMARDs encompassed conventional synthetic agents ($n=36$); and B-cell-depleting therapy with rituximab ($n=13$) was also utilized. HBV DNA testing at month 3 and month 6 was performed in 57% (75/132) and 54% (71/132) of these patients, respectively.

Conclusions: Immunosuppressive regimens used in rheumatologic diseases continue to pose a clinically relevant risk for HBVr. No reactivation occurred in resolved HBV patients regardless of prophylaxis status during the study period. The present data encompass a 16-month observation period, and it was observed that a portion of the patients who were not receiving

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prophylaxis did not attend regular follow-up visits. For such patients, prophylaxis may represent a more appropriate management option.

Keyword(s) : HBV reactivation, immunosuppressive therapy, rheumatology, DMARDs, antiviral prophylaxis

1962 - Native liver survival in pediatric hyperacute liver failure Is standard medical therapy effective

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Background / Hypothesis:

There is a paucity of outcome data on Hyperacute liver failure (HALF), a unique subset of pediatric fulminant hepatic failure. We aimed to study the effect of standard medical therapy and risk factors related to outcome in HALF

Materials and Methods:

Clinical data (January 2017 – November 2022) were retrieved from a prospectively maintained database.. HALF,) Acute liver failure (ALF) and subacute liver failure (SALF) were classified as per Pediatric Acute Liver Failure Study Group (PALFSG) criteria (jaundice and coagulopathy of ≤ 7 , 8–28 and ≥ 29 days of presentation). Pediatric End Stage Liver Disease (PELD) score and King's College Hospital Criteria (KCHC) were compared between the subgroups. Outcomes were survival with native liver (SNL) or need for liver transplantation or death.

Results:

Of the 119 patients with paediatric fulminant hepatic failure (Table 1), SNL was seen in 53 (44.5 %) with a complete resolution of liver functions over a median follow up 9 (IQR 5 – 12) months. In HALF group (n=20), univariate regression analysis showed presence of shrunken liver span, hypoalbuminemia and acute kidney injury were predictive of poor outcome. A PELD score of >27 in HALF predicted poor outcome (sensitivity 80 %; specificity 74.6 % [p=0.023, AUC=0.847]) (fig 1). PELD score did not predict prognosis in ALF and SALF subgroups. KCHC had a 90% sensitivity 71.6 % specificity, 80% positive predictive value and 14% negative predictive value overall in determining poor outcome in the HALF group.

Conclusions:

Three-fourth of HALF will survive with native liver on

standard medical therapy. Indicators for poor outcome are shrunken liver span, acute kidney injury and hypoalbuminemia. A PELD score >27 can be used to optimize the listing for liver transplantation.

Keyword(s) : Pediatric Acute liver failure , Native liver survival, Viral hepatitis

1963 - Hepatitis B Screening Prior to Immunosuppressive Therapy in Patients with Multiple Myeloma

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Background / Hypothesis:

Hepatitis B infection remains an important public health concern worldwide, including in Türkiye, which is considered a region of intermediate endemicity for Hepatitis B virus (HBV). Multiple myeloma (MM) is a B-cell malignancy characterized by the clonal proliferation of plasma cells within the bone marrow and accounts for approximately 13% of all hematologic cancers. The use of proteasome inhibitors, immunomodulatory drugs (IMiDs), and monoclonal antibodies in MM therapy has been associated with an increased risk of HBV reactivation. Therefore, as recommended in current clinical guidelines, patients should be evaluated for prior exposure to HBV before initiating treatment. In this study, we examined whether patients diagnosed with MM in the Hematology Department of Etlik City Hospital were appropriately screened for HBV infection.

Materials and Methods:

Newly diagnosed MM patients were retrospectively evaluated between October 2022 and August 2025. Results of HBsAg, anti-HBs, and anti-HBc IgG tests were obtained from the hospital electronic medical record system.

Results:

A total of 193 patients aged 18 years or older who had been diagnosed with MM were included in the study. The mean age was 63.9 ± 11.97 years, and 117 patients (60.6%) were male. Among these patients, HBsAg testing had been performed in 184 individuals, of whom 11 (5.9%) were positive. Anti-HBc IgG was tested in 144 patients, and 44 (30.5%) were positive. Anti-HBs testing was performed in 159 patients, with 40 (25%) testing positive. Eighty-four patients (43.5%) were



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screened with both HBsAg and anti-HBc IgG. The proportion of patients who underwent all three recommended tests (HBsAg, anti-HBc IgG, and anti-HBs) was 41.4%.

Conclusions:

According to the findings of this study, approximately one-third of patients diagnosed with MM had evidence of prior HBV exposure, and 5.9% had chronic HBV infection. The rate of prior HBV exposure was comparable to that reported in other studies conducted in Türkiye. The fact that fewer than half of the patients underwent the complete recommended screening panel highlights the need to increase awareness and adherence to HBV screening guidelines in this population.

Keyword(s) :

1964 - Nephropathy Following Portosystemic Shunt Surgery in Children with Extrahepatic Portal Venous Obstruction A hitherto undescribed entity

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Background / Hypothesis:

Porto-systemic shunt (PSS) surgery is well established for managing portal hypertension and associated complications of extrahepatic portal venous obstruction (EHPVO) in children. However, the long-term renal effects have not been systematically studied. We aimed to evaluate the prevalence, clinical spectrum, histopathology, treatment response, and outcomes of nephropathy developing after PSS in children with EHPVO.

Materials and Methods:

We conducted an ambispective observational cohort study of 315 EHPVO children undergoing non-selective PSS (August 1993-December 2024). Patients were followed with periodic renal monitoring (serum creatinine, blood urea nitrogen, urine protein:creatinine ratio), liver function tests, and Doppler ultrasonography for shunt patency. Renal biopsy was performed if proteinuria was persistent. The clinical, biochemical, and histologic findings, treatment, and outcomes were systematically recorded

Results:

Twelve of 315 children (4%) developed post-shunt nephropathy in a span of 36 months (IQR: 18.5–24) from PSS. Renal biopsies were suggestive of membranoproliferative glomerulonephritis (n=6), IgA nephropathy (n=4), focal segmental glomerulosclerosis (n=1) and diffuse proliferative (n=1) Treatment included angiotensin receptor blockers (n=11) immunosuppressive (n=7), and renal replacement therapy (n=1). Partial remission occurred in 7; two children progressed to end-stage renal disease and died. One patient experienced recurrence of nephropathy in the renal graft.

Conclusions: Post-shunt nephropathy is an underrecognized but serious complication following PSS in EHPVO. Renal surveillance, early biopsy, and timely therapy are critical.

Keyword(s) : Extrahepatic portal vein obstruction , Pediatric hepatology , Shunt surgery , Nephropathy

1966 - Investigating hepatitis B virus recurrence in post liver transplant patients a single center retrospective cohort study

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Background / Hypothesis:

Hepatitis B virus (HBV) infection is a primary indication for liver transplantation globally and in Türkiye, despite effective post-transplant prophylaxis (Hepatitis B immunoglobulin (HBIG) and nucleos(t)ide analogues (NUCs)). HBV recurrence still affects approximately 10% of recipients. High pre-transplant viral load, HBeAg positivity, and poor adherence to immunosuppression are known recurrence determinants. A comprehensive assessment of recurrence risk factors is essential to optimize post-transplant prophylaxis and improve long-term outcomes in high-risk patients. The present

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study aims to investigate the incidence and associated risk factors of HBV recurrence among Turkish patients who underwent living donor liver transplantation (LDLT) for HBV-related disease at Acibadem Atakent Hospital in Türkiye.

Materials and Methods:

This retrospective, single-center cohort study includes records of all adult Turkish patients who underwent LDLT for HBV-related disease at Acibadem Atakent Hospital between 2012 and 2025. Collected data include demographic, clinical, biochemical, virological, and pre-transplant tumor parameters, alongside specific HBV prophylactic and immunosuppressive protocols. Patient characteristics and HBV recurrence incidence will be summarized using descriptive statistics. Univariate and multivariate Cox regression analyses are planned to identify independent risk factors associated with HBV recurrence. Results will be presented with a 95% confidence interval (CI), and a p-value of <0.05 will be considered statistically significant.

Results:

This study encompasses a single-center cohort of Turkish LDLT recipients with HBV-related disease, including patients through 2025. Anticipated results include a detailed incidence rate of HBV recurrence and the identification of independent risk factors, such as high pre-transplant HBV DNA levels and specific immunosuppressive drug use, which will be strongly correlated with recurrence risk. The findings are expected to highlight the critical role of standardized viral and immunological monitoring in preventing late recurrence, which has not been comprehensively addressed in this specific population.

Conclusions:

The successful completion of this study will provide crucial, high-impact data on the long-term management of HBV-related LDLT in a significant regional cohort. By pinpointing key pre- and post-transplant risk factors through robust multivariate analysis, our findings will offer actionable, evidence-based recommendations for optimizing prophylactic regimens and improving surveillance protocols. The derived risk stratification model will serve as an essential tool for clinicians in Türkiye and other regions with similar demographics, thereby leading to reduced HBV recurrence rates and significantly enhanced graft and patient survival in high-risk transplant recipients.

Keyword(s) : hepatitis B virus recurrence, living donor liver transplantation, hepatocellular carcinoma, hepatitis B immunoglobulin

1969 - Patients with Hepatocellular Carcinoma Undergoing Transarterial Therapies Have Higher Risk of Hepatic Decompensation With Transarterial Radioembolization and Are Linked to Worse Prognosis

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Background: Transarterial radioembolization (TARE) with Yttrium-90 resin microspheres and transarterial chemoembolization (TACE) are widely recognized locoregional treatments (LRTs) for patients with unresectable hepatocellular carcinoma (uHCC). In this study, we aimed to identify the predictors of hepatic decompensation (HD) and evaluate its impact on overall survival (OS) in patients with uHCC undergoing TACE or TARE.

Methods: We retrospectively analyzed consecutive patients with uHCC treated with TARE (n=135, 43.3%) or TACE (n=177, 56.7%) across four academic centers. Exclusion criteria included extrahepatic metastases, Child-Pugh (CP) class C disease, and bland embolization. HD was defined as the occurrence of ascites, variceal bleeding, or portosystemic encephalopathy at baseline or ≤6 months post LRT. Median OS (mOS) and objective response (per mRECIST) were assessed.

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Results: The cohort comprised 312 patients, predominantly male (73.9%), with a mean age of 66.8 ± 10.5 years. Most patients had intermediate-stage disease (BCLC B, 54.5%), followed by early-stage (BCLC 0/A, 34.0%) and locally advanced-stage disease (BCLC C, 11.5%). HD was present at baseline in 36 patients (11.5%) and occurred after LRT in 48 patients (15.4%). Patients with post-LRT HD showed lower complete response rates (10.9%) than those without HD (31.4%; $P=0.002$). The mOS in patients without HD (41.0 months [95% CI: 33.0-46.0]) was significantly longer than in those with HD at presentation (15.0 months, 95% CI: 12.0-22.0; HR 2.35, 95% CI: 1.37-4.01) or after LRT (14.0 months, 95% CI: 12.0-21.0; HR 2.66, 95% CI: 1.68-4.26). Multivariable analyses revealed hepatitis C etiology (OR 0.35, 95% CI: 0.15-0.78), CP score (OR 2.28, 95% CI: 1.12-4.65), ALBI score (OR 1.69, 95% CI: 1.14-2.14), and intervention with TARE (OR 2.32; 95% CI: 1.15-4.76) as independent predictors of post-LRT HD.

Conclusions: Post-LRT HD is associated with significantly reduced OS in patients with uHCC. Baseline hepatic reserve and intervention with TARE are key predictors of post-intervention HD. These results highlight the need for individualized, biology-driven treatment planning in uHCC.

Keyword(s) :

1970 - Impact of metabolic dysfunction associated steatotic liver disease on health related quality of life among adults in a national Saudi population study

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a major contributor to chronic liver disease globally. Despite growing research interest, population-level data on MASLD and its impact on health-related quality of life (HRQoL) in Saudi Arabia remain limited. The Genomics and Environmental Noninvasive Evaluation for Saudi Intrahepatic Steatosis (GENESIS) study, a prospective, population-based cohort, was designed to provide comprehensive national data on MASLD prevalence and outcomes. This analysis aimed to assess HRQoL differences between adults with and without MASLD using the RAND 36-Item Health Survey (SF-36).

Materials and Methods:

Adults aged ≥ 18 years were recruited between March 2024 and June 2025 from three major regions in Saudi Arabia using a multistage stratified sampling approach. MASLD was defined using transient elastography with a controlled attenuation parameter (CAP) ≥ 268 dB/m plus ≥ 1 cardiometabolic risk factor. HRQoL was measured using SF-36, covering eight domains: physical functioning, role limitations due to physical health (role-physical), bodily pain, general health, vitality/energy-fatigue, social functioning, role limitations due to emotional problems (role-emotional), and mental health/emotional well-being. Composite scores included physical component summary (PCS), mental component summary (MCS), and overall SF-36 score.

Results:

Of 2,983 participants evaluated, 1,111 (37.2%) had MASLD. Those with MASLD were older (mean age: 46.1 vs. 39.5 years), more often male (58.4% vs. 44.8%), and had higher body mass index (median: 31.6 vs. 27.0 kg/m^2) (all $p < 0.001$). MASLD participants had lower mean scores in physical functioning (82.0 vs. 88.1), role-physical (83.0 vs. 86.7), bodily pain (82.2 vs. 85.7), and general health (71.4 vs. 74.4) (all $p < 0.001$). No significant differences were observed in vitality/energy-fatigue, social functioning, role-emotional, or mental health/emotional well-being. PCS scores were lower in the MASLD group (79.7 vs. 83.8, $p < 0.001$), while MCS scores were similar (79.1 vs. 79.7, $p = 0.37$). Overall SF-36 score was modestly lower in the MASLD group (79.4 vs. 81.7, $p < 0.001$).

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Conclusions:

In this nationally representative cohort, MASLD was linked to lower HRQoL, primarily affecting physical and general health domains. These findings emphasize the need for integrated, patient-centered care strategies to address MASLD-related HRQoL impacts and align with Saudi Vision 2030 priorities for improving population health.

Keyword(s) : MASLD, HRQoL, SF-36, RAND 36-Item Health Survey, Saudi Arabia, Chronic Liver Disease, Liver Steatosis

1971 - Evaluation of HBV screening status and test results for mothers fathers siblings spouses and children of individuals who are HBsAg positive

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Background / Hypothesis: It is crucial to screen the mothers, fathers, siblings, spouses, and children of HBsAg-positive individuals for HBV markers; to vaccinate seronegative individuals; and to follow up on those found to be seropositive. However, worldwide, the rate of people knowing whether they are infected with HBV is quite low, and individuals' awareness of this issue is around 5-10%. Our study aimed to evaluate the exposure to HBV among family members of HBsAg-positive individuals.

Materials and Methods: Study data collection began in 2021 following ethical committee approval. Face-to-face interviews were conducted with patients attending routine follow-up appointments to inquire about HBV testing and/or vaccination status among family members. Responses, along with existing test results for patients and data obtained from the automation system, were entered into an Excel spreadsheet for analysis.

Results: A total of 1083 HBsAg-positive individuals, 683 of whom were women, aged 18-67, participated in the study. The rate of HBV screening among the mothers of index cases was 41.7% (452/1083); the rate of HBV screening among fathers was 23.8% (239/1005); the rate of HBV testing among siblings was 31.2% (1524/4890); the rate of HBV testing among spouses was 65.9% (581/882); the rate of HBV testing among children was 51.3% (1245/2424) (Table 1).

97.3% of mothers are infected with HBV, and 77.6% are HBsAg positive; 94.6% of fathers are infected with HBV, and 69.3% are HBsAg positive; 97.8% of siblings are

infected with HBV, and 94.5% are HBsAg positive. The rate of children infected with HBV is 27.8%, the rate of HBsAg positive is 22.3% (Table 2). As can be seen, vertical transmission is quite significant in our country, and due to transmission from mother to child, there is a high rate of HBsAg positivity in both the mothers and siblings of index cases. In our country, since HBsAg testing has been performed before marriage since 2007, the rates of HBV testing among spouses have been found to be higher. Similarly, the implementation of HBsAg testing during marriage and pregnancy has also ensured higher rates of HBV testing among children. These rates are still insufficient.

Conclusions: In conclusion, although mother-to-child transmission has decreased significantly since the start of the national HBV vaccination program in 1998, there are still many people who were infected in previous years and are currently unaware of their status. Therefore, all index cases should be informed about HBV testing for their family members.

Keyword(s) :

1972 - Comparative Inpatient Outcomes and Comorbidity Profiles in Patients with Alcohol Associated Liver Disease With and Without Metabolic Syndrome Insights from a Nationally Representative US Database

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Uhs,lahore, Medicine, Lahore-Pakistan³

Background / Hypothesis:

The intersection of metabolic syndrome and alcohol-associated liver disease (ALD) is increasingly recognized as a unique phenotype termed MetALD. However, data comparing clinical characteristics and inpatient outcomes between ALD patients with and without metabolic syndrome remain limited.

Materials and Methods:

We identified adult patients (≥18 years) hospitalized with a principal diagnosis of ALD between 2016 and 2021 using the National Inpatient Sample (NIS) and ICD-10 codes. Patients were stratified into two groups based on the presence or absence of metabolic syndrome, defined by ≥ 3 components out of obesity (BMI ≥30), diabetes, hypertension, and hyperlipidemia.

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Weighted analyses were performed to account for the complex survey design. Baseline characteristics were compared using Rao-Scott chi-square and Student's t-tests. Multivariate regression models were developed to assess the impact of Metabolic Syndrome associated ALD (MetS +ALD) on in-hospital outcomes, adjusting for variables with $p < 0.1$ in univariate analysis

Results:

A total of 936,645 weighted hospitalizations were included (ALD: 759,680 [81.1%]; MetS + ALD: 6,965 [18.9%]). Met S+ ALD patients were older (57.8 vs. 52.2 years, $p < 0.001$) and more likely male (80.1% vs. 67.4%, $p < 0.001$). MetS + ALD was significantly associated with higher prevalence of diabetes (91.7% vs. 17.9%), hypertension (96.1% vs. 44.6%), hyperlipidemia (91.7% vs. 12.2%), and chronic kidney disease (25.0% vs. 10.0%; all $p < 0.001$). Despite a higher burden of comorbidities, MetALD patients had lower in-hospital mortality (3.0% vs. 4.9%, $p = 0.0024$) and reduced need for mechanical ventilation (4.2% vs. 5.8%, $p = 0.0125$). No significant differences were noted in length of stay or total hospital charges.

Conclusions:

Patients with MetS + ALD represent a distinct clinical subgroup of ALD with greater metabolic comorbidity burden but paradoxically lower inpatient mortality and respiratory support needs. These findings support further investigation into the natural history and tailored management strategies for MetS + ALD.

Keyword(s) : ALD, MET-ALD, METABOLIC SYNDROME

1974 - Investigation of SNAT Gene Expression in Liver Tissue in an Experimental Diabetes Model

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Background / Hypothesis:

Diabetes is a complex metabolic disorder that causes

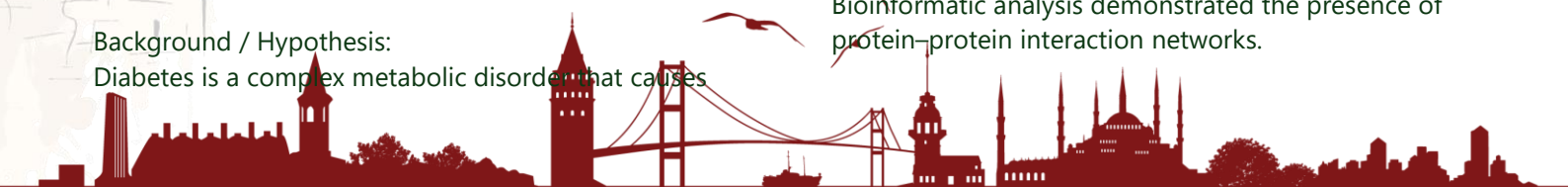
profound impairments in hepatic energy metabolism, insulin signaling, and amino acid utilization. Sodium-dependent neutral amino acid transporters (SNAT; SLC38 family), which play key roles in these processes, are essential regulators of amino acid uptake and metabolic adaptation in hepatocytes. Diabetes-associated metabolic stress may lead to marked alterations in the expression of SNAT transporters. This study aims to provide new insights into the pathophysiology of the diabetic liver by evaluating SNAT gene and protein levels in an experimental diabetes model. Furthermore, the experimental findings were supported by bioinformatic analyses, through which potential molecular mechanisms were examined in greater detail.

Materials and Methods:

Male Wistar rats obtained from Atatürk University were used in this study. The animals were divided into two groups: a control group and a diabetes group induced with streptozotocin (STZ, 200 mg/kg, IP). To reduce the risk of hypoglycemia after STZ administration, 5% glucose was added to the drinking water for 24 hours, and fluid support was maintained until 60 hours. Rats with fasting glucose levels ≥ 300 mg/dL on day 3 were considered diabetic. At the end of the experiment, liver tissues were preserved under appropriate conditions for subsequent analyses. mRNA levels of the SLC38 family transporters (SNAT1–SNAT10) were assessed by RT-qPCR, and their tissue localization was evaluated through immunohistochemistry. Additionally, bioinformatic analyses were performed based on gene expression and pharmacology databases, including differential gene expression analysis, weighted gene co-expression network analysis, and protein–protein interaction assessment through molecular docking.

Results:

In the diabetic group, notable expression alterations were observed in several members of the SNAT transporter family compared with the control group. RT-qPCR results indicated that diabetes induced adaptive responses across SNAT1–SNAT11 subtypes. Immunohistochemical analyses revealed shifts in the cellular localization and regional density of SNAT transporters in diabetic hepatocytes. These alterations are considered to be associated with diabetes-related metabolic stress, impaired insulin signaling, and adaptive regulation of amino acid utilization. Bioinformatic analysis demonstrated the presence of protein–protein interaction networks.



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Conclusions:

This study demonstrates that diabetes induces notable molecular alterations in hepatic amino acid transport systems, particularly those involving SNAT transporters. The adaptive responses observed within the SNAT family may influence amino acid utilization, cellular energy balance, and metabolic stress regulation in the diabetic liver. The findings shed light on the mechanistic significance of the SLC38 family in the pathophysiology of diabetes.

Keyword(s) : SNAT transporters, SLC38 family, diabetes, liver, hepatic metabolic adaptation

1975 - NTCP S267F (rs2296651) in HBV Infection Among Asian Populations A Systematic Review and Meta analysis

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Background:

Chronic hepatitis B virus (HBV) infection poses a significant health burden in Asian populations. The sodium taurocholate co-transporting polypeptide (NTCP) encoded by the SLC10A1 gene is the primary receptor mediating HBV entry into hepatocytes. The S267F variant (rs2296651) produces a loss of function effect that reduces bile acid transport and limits viral entry into hepatocytes, contributing to reduced susceptibility to chronic hepatitis B (CHB). This systematic review and meta-analysis aimed to quantitatively evaluate the association between NTCP S267F polymorphism and CHB risk in Asian populations.

Methods:

A systematic review and meta-analysis were performed in databases including Pubmed, Scopus, Cochrane, Proquest, ScienceDirect, and manual searches following PRISMA guidelines. Eligible studies included case-controls evaluating the S267F genotype in CHB. Study quality was assessed using the Newcastle-Ottawa Scale. RevMan 5.4 was used for analysis under various genetic models. Pooled odds ratios (ORs) were generated using fixed or random effect models based on heterogeneity assessed by I² statistics.

Result:

Sixteen studies were included, consisting of 28,028 individuals with CHB and 111,704 controls across East

and Southeast Asian populations. The dominant model (GG vs GA+AA) showed significantly reduced susceptibility of chronic hepatitis B among variant carriers (OR 0.57; 95% CI 0.44–0.73; $p < 0.0001$). The heterozygous model (GG vs GA) also showed reduced susceptibility (OR 0.58; 95% CI 0.45–0.75; $p < 0.0001$). Similarly, the allelic model (G vs A) demonstrated a significant reduced risk of CHB (OR 0.54; 95% CI 0.40–0.72; $p < 0.0001$). Meanwhile, the homozygous model (AA vs GG) (OR 0.26; 95% CI 0.05–1.44; $p = 0.12$) and the recessive model (AA vs GA+GG), reduced susceptibility remained non-significant (OR 0.27; 95% CI 0.04–1.74; $p = 0.17$). Limitations include overrepresentation of Chinese cohorts, potentially restricting external generalizability, and minimal AA genotype frequency, requiring larger studies to confirm homozygous and recessive model effects.

Conclusion:

The SLC10A1 S267F loss of function variant was significantly associated with reduced susceptibility to CHB among Asian populations, supporting the relevance of host receptor genetic factors in CHB pathogenesis. These findings support clinical implications of NTCP genotyping for risk stratification, screening and NTCP targeting entry inhibitors as potential therapeutic strategies in endemic regions. However, overrepresentation of Chinese cohorts and the rare AA genotype limit generalizability, highlighting the need for larger, multiethnic studies to clarify these findings.

Keyword(s) : NTCP, polymorphism, S267F, rs2296651, hepatitis B infection, chronic hepatitis B, meta-analysis

1976 - Advanced liver fibrosis in patients with metabolic dysfunction associated steatotic liver disease (MASLD) impairs sensory perception of ice cream with the protein sweetener brazzein

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Introduction: In individuals with MASLD, the progression of hepatic fibrosis may be accompanied by alterations in sensory perception, including changes in taste responsiveness. Such effects may have important implications for the acceptability of sugar-reduced or

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sugar-free dietary products. The objective of this study was to evaluate the association between fibrosis severity and the sensory perception of ice cream sweetened with brazzein, a natural sweet protein, and sucrose.

Materials and methods: This clinical investigation was conducted under a protocol registered at ClinicalTrials.gov (NCT06724913). The study employed a double-blind, randomized, two-period crossover design with two treatment sequences (AB and BA), where treatment A consisted of brazzein-sweetened ice cream and treatment B consisted of sucrose-sweetened ice cream. Fibrosis staging was determined using liver stiffness measurement (LSM) by vibration-controlled transient elastography. Sensory perception was assessed using a standardized 5-point hedonic scale evaluating overall impression, flavour, texture, aroma, appearance, and color. The study was supported by the Russian Science Foundation (Project No. 19-76-30014-P).

Results: A total of 103 participants with MASLD were enrolled. Fibrosis staging was as follows: F0–F1 (71.8%), F2 (15.5%), F3 (8.7%), F4 (3.9%). Texture ratings demonstrated a clear, fibrosis-dependent divergence between the two ice creams. Among participants with F0–F1 fibrosis, texture ratings for brazzein- and sucrose-sweetened samples were nearly identical (difference = -0.04 points). In F2–F3 stages, brazzein elicited moderately lower texture ratings (-0.25 и -0.11 points), whereas in F4 the decline exceeded 1 point relative to sucrose, representing a clinically meaningful difference. Sensitivity analyses across various LSM thresholds confirmed the robustness of the sweetener type×fibrosis interaction: no significant interaction was observed at 8 kPa, whereas thresholds ≥ 9 kPa produced statistically significant effects, which strengthened at LSM values of 10–12 kPa. Effect size analyses (Cohen's d) further supported a fibrosis-dependent pattern. In the LSM < 9.6 kPa subgroup, effect sizes were uniformly small (d -0.05 to -0.25), indicating practical equivalence between sweeteners. In contrast, participants with LSM ≥ 9.6 kPa demonstrated medium to large effect sizes (texture d = -0.69; taste d = -0.84; overall impression d = -0.99), indicating substantial degradation of sensory perception specific to brazzein.

Conclusion: Advancing hepatic fibrosis in MASLD is associated with significant impairment in the sensory perception of brazzein-sweetened ice cream, while perception of sucrose-sweetened ice cream remains largely preserved. These findings carry clinical relevance: individuals with advanced fibrosis are among those who most require dietary sugar

reduction, yet diminished acceptability of sweetener-based products may hinder adherence to sugar-restriction strategies.

Keyword(s) : brazzein, metabolic dysfunction-associated steatotic liver disease, MASLD, protein sweetener, sucrose

1977 - PREVALENCE OF LIVER FIBROSIS AMONG A COHORT OF PATIENTS WITH METABOLICALLY ASSOCIATED STEATOTIC LIVER DISEASE (MASLD) FOLLEWED AT AN URBAN SETTING IMPACT OF UTILIZATION OF FIBROSCAN.

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Background / Hypothesis:

Metabolically Associated Steatotic Liver disease is widely recognized as a major cause of progressive liver disease. MASLD can lead to advanced fibrosis and cirrhosis, particularly in patients with more than one component of the metabolic syndrome. Transient elastography (FibroScan®) non-invasively quantifies liver stiffness kPa (fibrosis) and steatosis (CAP™). This study aims to describe the distribution of liver fibrosis and steatosis severity in a large, real-world cohort of patients with MASLD.

Materials and Methods:

We conducted a retrospective analysis of 11,214 patients who underwent FibroScan® at the Toronto Liver Centre between 2020 and 2025. Among them, 8,149 patients (72.7%) were identified with MASLD and formed our study cohort. Liver stiffness (kPa) and CAP (dB/m) values were used to stage fibrosis (F0-F4) and grade steatosis (S0-S3).

Results:

Statistical analysis was performed. The median liver stiffness was 6.6 kPa, and the median CAP was 287 dB/m. Fibrosis was distributed as: F0: n=2705(33%) F0-

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F1: n= 2977(37%), , F1:(n=501, 6.2%), F1-F2: n=907(11%), F2: n=303,(3.7%), F2-F3: n =242(3%), F3:n=113 (1.3%), F3-F4:n=133(1.6%),and F4 (cirrhosis): n=267(3.2%).Steatosis was distributed as: S0:n=1285(15%),S0-S1:n=221(2.7%), S1:n=1521(18.6%), S1-S2:n=115 (1.4%), S2:n=1618(19.9%), S2-3:n=1209(14.8%), S3:n=2176 (26.7%). The majority of scans (70.4%) were performed using the M probe, and 29.6% were performed using the XL probe.

Conclusions:

In one of the largest reported MASLD cohorts assessed by transient elastography, severe steatosis was present in 45%, Mild liver fibrosis was present in about 70% while moderate to advanced liver Fibrosis and Cirrhosis was present in 25%. Fibrocan's widespread use is essential to identify the minority of patients with advanced fibrosis who are at high risk for progression to decompensated liver disease, enabling targeted intervention and resource allocation. This study highlights the importance of Fibroscan as a screening tool for stratification of liver steatosis and fibrosis.

Keyword(s) : MASLD

1978 - Clinical characteristics and one year survival of patients with hepatocellular carcinoma linked to metabolic dysfunction associated steatotic liver disease findings from a comprehensive institutional registry

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Background: Hepatocellular carcinoma (HCC) is the most common type of primary liver cancer globally

and a leading cause of cancer-related mortality in Saudi Arabia. With the control of viral hepatitis, there is a worldwide trend in the etiology of HCC shifting towards MASLD. In Saudi Arabia, research on the clinical characteristics and outcomes of HCC linked to metabolic dysfunction-associated steatotic liver disease (MASLD) is still evolving. We compare patient characteristics, disease presentation, and clinical outcomes between individuals with HCC related to MASLD and those with HCC from other etiologies (non-MASLD-related HCC).

Methods: We conducted a retrospective analysis of a prospectively maintained registry for HCC at King Faisal Specialist Hospital and Research Center in Riyadh, which comprised patients diagnosed with HCC (either through histological or radiological methods) from August 2020 to May 2025. We then compared demographic, clinical, and tumor characteristics, as well as treatment modalities, between patients with MASLD-related and non-MASLD-related HCC. One-year survival was also estimated using Kaplan-Meier analysis, with censoring at the date of last follow-up for patients who did not die within the first year. All patients, including those with less than one year of follow-up, were included in the analysis.

Results: Among the 427 patients diagnosed with HCC, 152 cases (35.6%) were associated with MASLD. Most of the patients were male, accounting for 69.5%, and there was no significant difference in gender distribution between those with MASLD-related HCC and those without ($p=0.971$). Patients with MASLD were slightly older at diagnosis (median age 66 vs. 63 years, $p<0.001$) and more likely to have diabetes (67.8% vs. 46.9%, $p<0.001$). There were no significant differences between the groups in ECOG performance status, BCLC stage, Milan criteria, or Child-Pugh class at the time of diagnosis. Locoregional therapies were the most common first-line treatments in both groups (53.3% in MASLD vs. 57.1% in others, $p=0.913$). One-year survival was similar across groups (log-rank $p=0.44$), with comparable Kaplan-Meier survival estimates.

Conclusions: MASLD has become the predominant etiology of HCC in Saudi Arabia, accounting for over one-third of the cases. Despite presenting at an older age and having a higher diabetes prevalence, patients with MASLD-related HCC had similar tumor burden, treatment approaches, and one-year survival outcomes compared to non-MASLD-related HCC. The findings underscore the need for increased surveillance efforts



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and earlier risk stratification for MASLD. Future research should focus on long-term outcomes and tailoring therapies to the distinct metabolic profile of MASLD-related HCC.

Keyword(s) : Hepatocellular carcinoma, metabolic dysfunction-associated steatotic liver disease, HCC registry, Saudi Arabia

1979 - Squamous cell carcinoma of the gallbladder A case report

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Squamous cell carcinoma of the gallbladder is a rare malignant tumor, representing less than 3% of gallbladder cancers.

We report the case of a 70 year old man with a history of focal segmental glomerulosclerosis for which he was on ace inhibitors, a history of tobacco and cannabis use and risk factors for hepatotoxicity such as traditional cauterization and unsafe dental procedures. One month prior his admission, he presented epigastric pain with cholestatic jaundice that resolved spontaneously, associated with fever, asthenia, anorexia and a 7.6% weight loss in one month. On clinical examination he was vitally stable with a moderate epigastric tenderness and 14 cm hepatomegaly. Laboratory findings revealed cholestasis and an inflammatory syndrome.

The patient was first evaluated by abdominal ultrasound which revealed a hyperechoic mass in hepatic segment V in close contact with the gallbladder fundus, showing focal wall thickening and microlithiasis. Then a computed tomography scan of the chest, abdomen and pelvis was done which revealed a gallbladder mass measuring 55 × 57 × 65 mm infiltrating segments V and VI of the liver, extending to the gallbladder bed, in close contact with the second portion of the duodenum. Deep lymph nodes were present (coeliac, mesenteric and retroperitoneal). The mass was radiologically staged T3 pN2 Mx.

An ultrasound guided biopsy was performed for histological confirmation which identified a well differentiated, keratinizing, infiltrating squamous cell carcinoma of the gallbladder.

The patient was discharged in good condition and referred to the medical oncology department, where he was started on adjuvant chemotherapy.

Gallbladder squamous cell carcinoma is a rare, aggressive tumor often discovered late due to nonspecific symptoms. Diagnosis relies on histopathology, as imaging is frequently inconclusive. Clinicians should keep this entity in mind when evaluating gallbladder or biliary lesions, as each case adds valuable insight into this uncommon malignancy

Keyword(s) : Gallbladder carcinoma, Biliary tract malignancy, Ultrasound-guided biopsy

1980 - SPLEEN STIFFNESS MEASUREMENT AS A FAVORABLE PREDICTOR OF PORTAL HYPERTENSION IN A COHORT OF PATIENTS WITH CHRONIC LIVER DISEASE FOLLOWED AT AN URBAN LIVER CENTRE IN TORONTO (TLC).

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Background / Hypothesis:

Portal hypertension, the primary driver of complications in advanced liver disease, is best measured by the invasive hepatic venous pressure gradient (HVPG). While liver stiffness (LSM by VCTE) correlates with HVPG in early disease, its accuracy plateaus in significant portal hypertension (≥ 10 mmHg), limiting its predictive value for critical outcomes like varices.

As the spleen undergoes consistent morphological changes in response to portal hypertension, SSM has emerged as a more reliable, non-invasive surrogate for HVPG, offering an alternative for stratifying the risk of varices and clinically significant portal hypertension.

We sought to evaluate the liver and spleen stiffness in a cohort of patients who underwent Fibroscan and spleen stiffness at TLC. Clinically significant portal hypertension (CSPH) is defined as one or more of the following: ascites, varices, splenomegaly, encephalopathy, recanalized umbilical vein, collaterals, dilated portal vein, or history of GI bleed. Data on 112 patients who met these criteria were collected and analyzed.

Materials and Methods:



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We conducted a retrospective cohort analysis on patients who underwent a spleen stiffness and FibroScan at an urban Toronto Liver Centre. Fibroscan and spleen stiffness measurements, past medical history, ultrasound findings, endoscopic findings and physical examination findings were analyzed.

Results: The cohort comprised 46% females, 54% males, and an age range of 17 to 87 (median age 63.5). The spleen stiffness score ranged from 11.4 to 100 kPa with a median stiffness of 43.5 kPa.

Fibroscan® readings revealed a liver stiffness range of 3.5 to 75 with a median stiffness of 18.5, and 83 % of patients had advanced fibrosis or cirrhosis (defined as a liver stiffness of ≥ 13 kPa). Controlled attenuation parameter (CAP) ranged from 121 to 400, with a median CAP score of 266. Ascites was present (5.4%), esophageal varices (28.6%), splenomegaly (29.5%), dilated portal veins (4.5%), and variceal bleeding (6.25%).

The underlying liver diseases were: MASLD 41.3%, MASH 3.7%, Hepatitis B 12.8%, Hepatitis C 11.9%, ALD 21 %, AIH 5.5%, PBC 1.8%, and elevated liver enzymes NYD 2.7%. BMI range of 18.2- 59.1 (median BMI 28.4) There was a strong correlation between patients with ascites (0.417) and patients with GI bleed (0.466), and spleen stiffness and liver stiffness measurements.

Conclusion: Based on the medians of the spleen stiffness and Fibroscan measurements, a cut-off of 69.8 kPa for spleen stiffness and 24.5 kPa for liver stiffness was predictive for the presence of CSPH. A prospective study is underway in our centre to further elucidate these findings.

Keyword(s) : MASLD, Spleen Stiffness, Fibroscans, Chronic liver disease

1981 - Improving Elimination in Albumin Dialysis Liver Support Different Responses of Water Soluble and Albumin Bound Toxins

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Background / Hypothesis:

Extracorporeal Albumin Detoxification-Dialysis (ECAD) is the leading procedure to support failing livers by detoxification, it has been used in over 150.000 therapies. Past Meta-Analysis concluded dose of treatment would be an important cofactor determining a good outcome. The introduction of a new generation of adsorbents to maintain dialysate albumin's purity

has increased the bile acid removal compared to MARS and Cytosorb. However, since next generation adsorbents may have moved the set points for optimal blood and dialysate flow rates, the aim of this investigation was to elucidate the role of blood- and dialysate flow on the removal of water soluble toxins, albumin bound toxins and improvement of albumin binding.

Materials and Methods:

Eight Albunique treatments were performed using two blood flow rates (100 and 140 mL/min) and stepwise dialysate flow rates (2–4 L/h). Pre- and post-dialyzer blood samples were collected.

Photometric analysis was used to quantify albumin-bound toxins (bilirubin and bile acids) and water-soluble toxins (creatinine and urea). Albumin binding function at Sudlow site II was assessed by Dansylsarcosine titration, yielding K_d values (concentration at which half of the available binding sites are occupied). Instantaneous filter clearance was calculated and compared across the different flow conditions.

Results:

Instantaneous filter clearance during the eight Albunique treatments was analyzed for albumin-bound toxins (bilirubin and bile acids) and water-soluble toxins (creatinine) under two blood flow rates (100 and 130 mL/min) and stepwise increasing dialysate flow rates (2, 3, and 4 L/h).

Significantly higher clearance was observed for water-soluble toxins, particularly creatinine, with increasing dialysate flow. In contrast, no advantage was demonstrated for albumin-bound toxins; a trend toward reduced clearance was noted at higher dialysate and blood flow rates. Furthermore, K_d values at Sudlow site II did not improve with increased flow rates. These findings indicate a differential response of toxin classes to dialysate flow rate adjustments.

Conclusions:

The present data demonstrate that increasing dialysate and blood flow rates significantly enhance the clearance of water-soluble toxins. In contrast, no beneficial effect was observed for albumin-bound toxins; higher flow rates were not associated with a trend toward improved clearance of bilirubin and bile acids. Similarly, albumin binding function did not improve under higher flow conditions. These findings suggest that aggressive flow escalation does not confer an advantage for the removal of albumin-bound toxins. Instead, lower or moderate flow settings may

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provide more favourable conditions for albumin-mediated detoxification.

Keyword(s) : Extracorporeal Albumin Detoxification-Dialysis (ECAD), Albumin Binding Function, Extracorporeal Liver Support, Clearance Rates

1983 - Unmasking Cryptogenic Cirrhosis Extreme Low LDL C as a Critical Clue for Adult Onset Familial Hypobetalipoproteinemia

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Background/Aims: Familial hypobetalipoproteinemia (FHBL) is a rare genetic disorder primarily caused by mutations in the APOB gene, resulting in impaired hepatic secretion of VLDL and LDL. While often diagnosed asymptotically in childhood through incidental low lipid findings, FHBL can lead to progressive hepatic steatosis and, rarely, cirrhosis in adulthood due to intrahepatic triglyceride accumulation. We present a Turkish father and son diagnosed with FHBL-related cirrhosis in adulthood.

Case Presentation: The index case, a 41-year-old male without comorbidities, was evaluated for severe incidental thrombocytopenia. Abdominal ultrasound revealed findings consistent with liver cirrhosis, including a lobulated liver, splenomegaly (210 mm), and dilated portal/splenic veins. Standard etiological screenings for chronic liver disease (viral, autoimmune, metabolic, Wilson's) were negative. Notably, his lipid profile showed profoundly low LDL-C levels (confirmed on repeat: 14 mg/dL and 8 mg/dL). Genetic analysis identified a heterozygous variant in the APOB gene (c.333-2A>T). This diagnosis prompted a re-evaluation of his 68-year-old father, who had undergone liver transplantation 20 years prior for presumed alcoholic cirrhosis and hepatocellular carcinoma. Retrospective analysis of the father's pre-transplant records revealed extremely low LDL-C levels (6-10 mg/dL), which had normalized (80-100 mg/dL) immediately following transplantation. Genetic testing confirmed the identical APOB mutation in the father.

Conclusions: This family cluster highlights FHBL as an important, yet often overlooked, differential diagnosis in adult patients presenting with cryptogenic cirrhosis. The dramatic normalization of lipid profiles following liver transplantation in the father provides strong

clinical evidence supporting the hepatic origin of the metabolic defect. Clinicians should maintain a high index of suspicion for hypolipoproteinemias when evaluating unexplained liver disease, regardless of patient age, as early identification may guide management and family screening.

Keyword(s) : Liver Cirrhosis, Fatty Liver, Apolipoproteins B, Liver Transplantation, Hypobetalipoproteinemia, Familial

1986 - Polymeric Nanoparticle Mediated Delivery of miR 221 as a Novel Therapeutic Approach for Hepatocellular Carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) has a poor prognosis and ranks as the fourth leading cause of cancer-related mortality worldwide. MicroRNA-221 plays a key role in regulating the cell cycle.

This study investigated the potential therapeutic effect of polymeric nanoparticles as a delivery system for miR-221 in an experimental HCC model.

Materials and Methods:

PLGA nanoparticles were synthesized, characterized, and conjugated with a miR-221 mimic. The nanocomposite was evaluated in vitro against HepG2 cells using the MTT assay, and in vivo in a DEN-induced HCC model in Balb/C mice treated either with PLGA nanoparticles alone or with miR-221-loaded nanoparticles.

Results:

The miR-221 nanocomposite exhibited significant cytotoxic activity toward HepG2 cells, with an IC50 value of 97.12. In vivo findings demonstrated marked improvement in the miR-221 nanocomposite-treated mice compared with the pathological control group, as

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evidenced by histopathological recovery and downregulation of caspase-3, caspase-9, cyclin D1, α -SMA, CD44, Bcl-2, and Ki67. Tumor-associated biomarkers including AFP, VEGF, PDGF, and TNF- α were also significantly reduced compared with the DEN group.

Conclusions: MiR-221-loaded polymeric nanoparticles represent a promising and innovative therapeutic strategy for hepatocellular carcinoma.

Keyword(s) : Liver Cancer- MicroRNAs- PLGA

1987 - The Economic and Health Burden of Liver Cancer in Saudi Arabia An Analysis of Costs Health Outcomes and the Advantages of Early Detection

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Background: Liver cancer is emerging as a leading cause of morbidity and mortality, particularly among men, due to late-stage diagnoses and low survival rates. We aimed to quantify the public health and economic burden of liver cancer in Saudi Arabia in 2022 and to assess the potential economic impact for future early detection scenarios.

Methods: A cohort-based static model was developed to estimate the disease burden in terms of years of life lost (YLL) and economic costs, including both medical

expenses and productivity losses. The model inputs were based on local real-world data, including the population-based Saudi Cancer Registry, statistics from the General Authority for Statistics, and liver cancer incidence and mortality estimates from GLOBOCAN 2022. Medical costs were assessed over a 5-year treatment period, with adjustments made to the baseline year of 2022. Productivity losses were estimated for both patients and caregivers during the treatment phase, whereas productivity loss due to premature death was calculated up to the age of 65 using the human capital approach, considering employment rates. A scenario analysis simulated the impact of shifts in the stage distribution compared to the current state.

Results: Liver cancer caused an estimated 13,800 YLL among men and 4,600 YLL among women in 2022. The total economic burden was approximately USD 219 million, comprising USD 126 million in medical costs. Productivity losses accounted for USD 93 million, 88% of which were incurred by men, for whom treatment and productivity costs were nearly equal. In contrast, productivity losses accounted for approximately 25% of total costs for women, primarily due to lower workforce participation. Scenario analyses showed that shifting late-stage diagnoses to earlier stages reduced YLL and productivity losses mainly through decreased mortality. Although annual per-patient medical costs are lower in earlier stages, total medical costs increased due to prolonged survival and continued healthcare utilization.

Conclusions: Liver cancer imposes a substantial health and economic burden in Saudi Arabia, with productivity losses, particularly among men, constituting a major share of the economic impact. Effective early detection strategies are needed to improve survival, reduce YLL, mitigate productivity losses, and potentially offset increased medical costs.

Keyword(s) : Liver cancer, modeling, economic burden, early detection, productivity loss, Saudi Arabia

1988 - Research on Biomarkers of Liver Injury in Patients with Hepatocellular Carcinoma under Combined therapies Based on ICIs

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Background / Hypothesis: Hepatocellular carcinoma is one of the most prevalent

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and fatal cancers. With the application of systemic therapy, especially combination therapies based on immune checkpoint inhibitors (ICIs), significant therapeutic benefits have been achieved in advanced hepatocellular carcinoma. However, Whether chemotherapeutic drugs, targeted drugs, or ICIs, can cause tissue damage, known as treatment-related adverse events (trAEs). Early identification and assessment of adverse events have significant clinical significance. This study aims to characterize the signatures of adverse events by integrating proteomics and metabolomics.

Materials and Methods:

A total of 40 patients were enrolled in the study. Plasma samples were collected after the first five treatments. 27 patients had various types of adverse events, including 7 cases of liver injury. The plasma samples were analyzed by proteomics and metabolomics, and the adverse event-related proteins and metabolites were characterized by differential abundance analysis, Gene Ontology (GO) enrichment, Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway analysis. Candidate biomarkers were screened using a machine learning model, and the discriminant performance was evaluated by receiver operating characteristic (ROC) curve.

Results:

By comparing the liver injury group with the non-injury group, 102 differentially expressed proteins and 57 differentially expressed metabolites were found. Through functional enrichment and network analysis, it was found that the liver injury is mainly related to metabolic reprogramming in vivo, affecting multiple amino acid and bile acid metabolic pathways to form a synergistic injury network. Four candidate markers (COL12A1, CEACAM1, PRG2 and C4BPB) were screened out through machine learning, and the regions under the ROC curves were 0.875, 0.813, 0.75 and 0.843, respectively.

Conclusions: Through the combined analysis of proteomics and metabolomics, we have selected several potential markers related to liver injury in the combined treatment of aHCC, which need to be further verified in large-scale prospective studies in the future

Keyword(s) : ICIs, biomarker, HCC, Liver Injury

1990 - Description of a Cohort of patients with chronic Liver disease of various etiologies characterized by TEE (Fibroscan) Toronto Liver Centre (TLC) Real world experience. HIGH PREVELECE OF MASLD

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Background / Hypothesis:

Transient elastography (FibroScan) is increasingly recognized as a non-invasive diagnostic tool which measures liver stiffness (Fibrosis) and Controlled Attenuation Parameter (CAP) (steatosis). Real-world data etiological-based patterns remain limited in the diverse population of the Greater Toronto Area (GTA). Understanding these patterns is critical for identifying high-risk groups and guiding public health and screening initiatives tailored to the etiologies. To examine fibrosis (kPa) and steatosis (CAP) distribution across patients with chronic liver disease of different etiologies in a cohort of patients who underwent FibroScan examination in the GTA between 2020 and 2025.

Materials and Methods:

A retrospective analysis was performed on more than 11,214 FibroScan examinations conducted across multiple TLC FibroScan sites in the GTA. Variables included age, sex, ethnicity, etiology, liver stiffness (E median, kPa), hepatic steatosis (CAP median, dB/m), and probe type (M vs XL). Subgroup analyses were conducted across etiologies to highlight clinical variability within the cohort.

Results:

Of the 11,214 patients, the median age of the cohort was 56.3 ± 14.2 years. The cohort was equally distributed by gender (51.8 % male, 48.2 % female). The mean liver stiffness was 6.7 kPa. With a mean CAP value of 275 dB/m. MASLD was the predominant etiology (n=8149, 72.8%), demonstrating the highest

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steatosis levels (median CAP 285 dB/m) with modest stiffness (6.6 kPa). Viral hepatitis B (n=1856, 16.6%) showed the lowest stiffness (5.4 kPa) and relatively low CAP values (246.5db/m), while hepatitis C demonstrated slightly higher stiffness (7.1 kPa) and CAP of (251 dB/m). Alcohol-related liver disease (n=180, 1.6%) with a mean stiffness of 12.4 kPa and a median CAP value of 259 db/m. PBC and PSC accounted for (n=35, 0.3%), with a median stiffness of 6.8 kPa and a CAP of 233 dB/m. Autoimmune hepatitis represented (n=99 0.9%), with median liver stiffness of (7.2 kPa) and CAP of (261 dB/m). Patients on Methotrexate (N=32,0.29%), Mean stiffness (5.0 kPa), and CAP of (273 dB/m). Other etiologies comprised (n=622,5.5%) with a mean liver stiffness of (8.5kpa) and a CAP value of (249 dB/m). Overall, fibrosis and steatosis patterns varied across etiologies, with MASLD dominating steatosis and ALD showing the highest stiffness(Fibrosis)values.

Conclusions:The high prevalence of MASLD highlights the growing impact of steatotic liver disease and the urgent need for public-health strategies focused on metabolic liver disease prevention and screening protocols for early identification and prevention of chronic liver disease.

Keyword(s) : Chronic Liver Disease, MASLD, HBV,HCV, ALD

1991 - Advances in early detection of hepatocellular carcinoma a systematic review of novel diagnostic approaches

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Background:

Hepatocellular carcinoma (HCC) remains a major global health burden, with rising incidence and mortality. Novel diagnostic approaches may improve early detection and streamline diagnostic pathways, crucial for improving survival. We aimed to systematically review novel HCC detection approaches reporting diagnostic accuracy measures

Methods:

A systematic review was conducted using PRISMA guidelines by searching PubMed, EMBASE, Scopus, and the Cochrane Library on Dec 11, 2024. Eligible studies were cohort or randomized controlled trials in design, in English or Arabic, reported a novel diagnostic approach for HCC in any clinical setting, enrolled ≥ 300 participants, and reported diagnostic accuracy measures. Studies identifying incident HCC were included. Two reviewers independently screened studies and assessed risk of bias using the QUADAS-2 tool. Due to heterogeneity in index tests, populations, and outcome measures, meta-analysis was not performed.

Results:

Four studies (n = 50,363) met the inclusion criteria, each evaluating a distinct approach . Three studies focused on patients with existing liver conditions (hepatitis B and cirrhosis); two studies in hepatitis B cohorts reported predictive algorithms aimed at improving HCC detection above usual care. The methylation, mutation, and protein-HCC (M2P-HCC) clinical prediction model for patients with hepatitis B had an AUC of 0.88 (95% CI 0.81-0.95), outperforming standard care (0.76 [0.69-0.84]), with low risk of bias. Another model for patients with hepatitis B that included circulating tumor DNA had an AUC of 0.93 (0.90-0.97); however, CIs overlapped with those of standard care, limiting the strength of the comparative claim. The ALARM model, based on radiomics and deep learning techniques, achieved an AUC of 0.92 (0.90-0.96) in a cohort of patients with cirrhosis. The fourth study identified novel hepatitis B-specific AFP cut-off for screening in a high-prevalence area, reporting an AUC of 0.999 at a pre-specified cut-off of ≥ 8.1 ng/ml, though concerns remain about overfitting and generalizability.

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Conclusions:

This review highlights four novel approaches to HCC diagnosis, encouraging diagnostic performance, particularly in the M2P-HCC and ALARM models. Prospective validation in broader populations is crucial to assess clinical and health economic impacts, alongside regulatory, interpretability, and implementation challenges. The limited number of eligible studies highlights the need for further high-quality, real-world evaluations.

Keyword(s) : Early detection , HCC , Hepatocellular Carcinoma

1992 - Sustained Virologic Response After Only One Week of Glecaprevir Pibrentasvir A Rare Ultra Short Treatment Success

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Background / Hypothesis:

Direct-acting antivirals (DAAs) have revolutionized chronic hepatitis C (CHC) treatment, achieving >95% cure rates with short-duration regimens. Glecaprevir/pibrentasvir (G/P) is a pangenotypic, highly effective regimen with standard treatment durations of 8–12 weeks. Data on ultra-short G/P therapy (≤ 2 weeks) remain extremely limited. We report a rare case in which only one week of G/P therapy resulted in a sustained virologic response (SVR) in a kidney transplant recipient.

Materials and Methods:

A retrospective evaluation was conducted of a kidney-transplanted, treatment-naïve, non-cirrhotic patient diagnosed with genotype 1b CHC. Clinical, laboratory, and viral kinetics data from 2017 to 2025 were reviewed. Therapy duration was confirmed through direct pill count and patient interview.

Results:

A female patient with a history of living-donor kidney transplantation (2008), diabetes mellitus, hypertension, and chronic renal failure presented in 2021. Although diagnosed with CHC in 2017, she remained untreated until presentation. The 2021 work-up revealed an HCV RNA load of 1,020,000 IU/mL (Genotype 1b). Laboratory results showed ALT 140 U/L, AST 121 U/L, and platelets 141,000/ μ L; bilirubin and albumin levels were within normal limits. The patient initiated G/P

therapy but self-discontinued after seven days and was lost to follow-up. Upon reassessment two years later, HCV RNA was undetectable. Medication reconciliation confirmed the ingestion of only one week's dosage. Serial virologic monitoring demonstrated persistent aviremia throughout 2023, 2024, and 2025, confirming durable SVR.

Conclusions:

This case demonstrates a notable achievement of SVR after only one week of glecaprevir/pibrentasvir in a complex post-transplant patient with multiple comorbidities. To date, only two similar cases of success with such ultra-short G/P exposure have been reported. These findings underscore the rapid potency of G/P, suggesting that viral clearance may occur significantly earlier than established treatment guidelines in specific individuals.

Keyword(s) : Hepatitis C; glecaprevir/pibrentasvir; direct-acting antivirals; sustained virologic response.

1994 - Effects of biological agents on hepatitis B virus reactivation in patients who are negative for hepatitis B surface antigen and positive for antibodies against hepatitis B core antigen

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Background / Hypothesis:

In HBsAg-negative, anti-HBc IgG-positive individuals, the risk of hepatitis B virus reactivation (HBVr) associated with immunosuppressive therapy varies depending on the immunosuppressive potency of the biological agent used. This study aimed to evaluate the development of HBVr in HBsAg-negative, anti-HBc IgG-positive patients receiving medium- or high-risk biological agents.

Materials and Methods:

A total of 172 patients identified as HBsAg-negative and anti-HBc IgG-positive were retrospectively evaluated. Patients were classified into moderate- and high-risk groups according to the HBVr risk associated with the biological agents used. Rituximab and ocrelizumab were categorized as high-risk agents, while anti-TNF- α agents (adalimumab, infliximab, golimumab, certolizumab, etanercept), the IL-6 inhibitor tocilizumab, and the IL-12/23 inhibitor

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ustekinumab were considered moderate-risk agents. Demographic and clinical characteristics, treatment details, concomitant immunosuppressive medications, and antiviral prophylaxis status were recorded. Virological and serological data obtained during follow-up were reviewed, and the occurrence of HBVr was assessed.

Results:

The mean age of the study population was 52.3 ± 11.0 years. The most common indications for biological agent therapy were ankylosing spondylitis (30.2%) and rheumatoid arthritis (29.7%). While 20.9% (n=36) of patients received high-risk agents, 79.1% (n=136) received moderate-risk agents. The median follow-up duration was 28.5 months (12–60 months) in the high-risk group and 36 months (12–96 months) in the moderate-risk group. All patients in the high-risk group received antiviral prophylaxis at treatment initiation, and no HBVr events were observed. In contrast, only 33.1% (n=45) of patients in the moderate-risk group received prophylaxis, while 66.9% (n=91) were monitored without prophylaxis. Among those not receiving prophylaxis, only one patient experienced virological reactivation without clinical or biochemical hepatitis. This patient had an initial anti-HBs level of ≥ 1000 IU/L, which remained stable during follow-up.

Conclusions:

Antiviral prophylaxis is effective in preventing HBV reactivation in HBsAg-negative, anti-HBc IgG-positive individuals receiving moderate- or high-risk biological agents. For patients treated with moderate-risk agents, regular virological monitoring may serve as a safe alternative to universal prophylaxis, allowing timely identification and management of the rare reactivation events that occur.

Keyword(s) : Hepatitis B Virus Reactivation, Biological Agents, Antiviral Prophylaxis

1995 - Ethnic Variation in FibroScan Detected Fibrosis and Steatosis in a Large Multi Ethnic Canadian Cohort Implications for Targeted MASLD Screening

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Background / Hypothesis:

FibroScan enables non-invasive assessment of fibrosis and steatosis through liver stiffness measurement (LSM) and controlled attenuation parameter (CAP). It is established that genetic factors play a role in the pathogenesis of MASLD. While international Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) guidelines acknowledge that metabolic risk factors and disease burden vary across ethnic groups, there is limited real-world Canadian evidence describing these ethnic-specific patterns.

To evaluate the distribution of FibroScan-derived fibrosis and steatosis stages across ethnic groups in a large, diverse Canadian cohort.

Materials and Methods:

A retrospective analysis was conducted on adult patients with chronic liver disease who underwent FibroScan examination at participating Greater Toronto Area liver clinics between January 2020 and May 2025. Variables include ethnicity, and fibrosis and steatosis stages, which were classified using validated cut-offs. Statistics were stratified by ethnicity.

Results:

Ethnic variation was observed in the distribution of advanced fibrosis (F3-F4), cirrhosis (F4), and advanced steatosis (S3). The highest proportions of advanced fibrosis within ethnic groups were Northern European (7.4%), Jewish (5.2%), and Southern European (3.4%) populations. Cirrhosis was most prevalent among Caribbean (8.6%), Southern European (6.3%), and Western European (6.3%) groups.

Advanced Steatosis (S3) was greatest in Northern European (37.0%), Caribbean (29.3%), Iberian (27.5%), and Southern European (27.1%) populations. Elevated S3 levels were also observed in other European sub-groups, as well as Hispanic, South Asian, and MENA groups, reflecting broader metabolic susceptibility.

Conclusion:

In this large, diverse Canadian cohort, FibroScan detected fibrosis and steatosis patterns that varied significantly by ethnicity. These real-world findings highlight ethnic variability in liver disease severity that extends beyond traditional risk factors. Understanding

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ethnicity-related patterns may refine FibroScan® interpretation and optimize MASLD screening for diverse populations. Further prospective studies are underway to elucidate these findings.

Keyword(s) :

1996 - Triple Threat A Rare Case of Drug Induced Liver Injury Triggering Autoimmune Hepatitis and Overlapping Systemic Lupus Erythematosus

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Background / Hypothesis:

Autoimmune Hepatitis (AIH) is an uncommon chronic inflammatory liver disease, with a prevalence of 16–18 per 100,000 individuals in Europe. AIH–Lupus Hepatitis overlap syndrome (AIH-L) presents a diagnostic challenge due to shared clinical, serologic, and immunologic features, and is typically considered when a patient meets both the American College of Rheumatology (ACR) criteria for Systemic Lupus Erythematosus (SLE) and the International Autoimmune Hepatitis Group (IAIHG) scoring system. Meanwhile, anti-tuberculosis (TB) medications are recognized causes of drug-induced liver injury (DILI), sometimes displaying autoimmune features that mimic AIH. Differentiating DILI with autoimmune manifestations from true AIH or overlap syndromes is difficult, especially when patients present with acute liver failure. This case highlights a complex diagnostic and therapeutic dilemma in a patient treated with anti-TB drugs who presented with seropositive autoantibodies and rapidly progressive hepatic dysfunction.

Materials and Methods:

We present a 69-year-old female who developed progressive jaundice, abdominal pain, and hepatic dysfunction six weeks after initiating anti-TB therapy for pulmonary tuberculosis. Magnetic resonance cholangiopancreatography (MRCP), autoimmune serologies, immunoglobulin profiling, and viral hepatitis studies were performed. The clinical trajectory, interpretation of autoimmune markers, and therapeutic decisions—including steroid initiation and subsequent complications—were analyzed in the context of available diagnostic frameworks for AIH, SLE,

and DILI.

Results:

Initial evaluation revealed non-dilated bile ducts and absence of structural obstruction on MRCP, supporting DILI as the principal working diagnosis. However, progressive hepatic decompensation prompted reassessment for underlying chronic liver disease. Autoimmune testing showed high-titer ANA positivity, elevated IgG, positive anti-smooth muscle antibody (SMA), and anti-dsDNA, fulfilling both ACR criteria for SLE and IAIHG scoring parameters suggestive of AIH. These findings raised strong suspicion for AIH–SLE overlap syndrome precipitated or unmasked by anti-TB therapy. Steroid therapy was initiated but complicated by pneumonia and worsening thrombocytopenia, which precluded liver biopsy. The patient subsequently developed acute liver failure with superimposed sepsis and succumbed to multiorgan dysfunction despite supportive care.

Conclusions:

AIH–SLE overlap is a rare entity with significant diagnostic complexity, especially when concurrent DILI from anti-TB therapy is present. Overlapping serologies and clinical features make differentiation difficult, and biopsy—often essential—may be unsafe in acute liver failure. Early recognition, prompt cessation of offending drugs, initiation of corticosteroids, and aggressive monitoring for infectious and hematologic complications are critical. This case underscores the importance of high clinical suspicion and multidisciplinary management to optimize outcomes in rapidly deteriorating autoimmune and drug-induced hepatic injury.

Keyword(s) : Autoimmune Hepatitis, Systemic Lupus Erythematosus, Drug Induced Liver Injury, Hepatic Failure, Tuberculosis

1997 - Hepatic resection versus stereotactic body radiation therapy as first line treatment for BCLC stage 0 hepatocellular carcinoma a propensity score weighted analysis

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Background / Hypothesis:

Stereotactic body radiation therapy (SBRT) has emerged as a promising curative treatment for

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hepatocellular carcinoma (HCC); however, robust comparative data with surgical resection remain limited. This study aimed to evaluate long-term outcomes of surgical resection versus SBRT in patients with Barcelona Clinic Liver Cancer (BCLC) stage 0 HCC.

Materials and Methods:

In this single-institution retrospective study, 537 patients with treatment-naïve, single HCC ≤ 2 cm who underwent surgical resection (n=450) or SBRT (n=87) between 2015 and 2023 were analyzed. Inverse probability of treatment weighting (IPTW) was applied to balance baseline characteristics. Overall survival (OS), disease-free survival (DFS), recurrence patterns, and treatment-related toxicities were compared between groups.

Results:

Over a median follow-up of 55.8 months (range, 8.3–117.0), unweighted 5-year OS rates were 95.6% for resection and 83.9% for SBRT (hazard ratio [HR] 4.14; 95% confidence interval [CI], 1.96–8.71; $P < 0.001$). The 5-year DFS rates were 68.1% and 58.6%, respectively (HR 1.76; 95% CI, 1.17–2.64; $P = 0.006$). However, IPTW-weighted analysis showed comparable survival outcomes, with 5-year OS rates of 93.8% versus 96.0% (HR 1.16; 95% CI, 0.44–3.02; $P = 0.766$) and 5-year DFS rates of 64.7% versus 75.2% (HR 1.18; 95% CI, 0.67–2.08; $P = 0.562$) in the resection and SBRT groups, respectively. Grade ≥ 3 complications occurred in 31 (6.9%) patients after resection, whereas no severe toxicities were reported after SBRT.

Conclusions:

After adjusting for baseline imbalances, SBRT demonstrated comparable survival outcomes to surgical resection. These findings support SBRT as a viable alternative for patients with BCLC stage 0 HCC who are suboptimal surgical candidates. Further prospective studies are warranted to optimize treatment strategies in this patient population.

Keyword(s) : very early stage; treatment-naïve patients; overall survival; disease-free survival; adverse events; inverse probability of treatment weighting.

1998 - The Efficacy of Atezolizumab and Bevacizumab Combination in real world for Advanced Hepatocellular Carcinoma

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Background / Hypothesis:

Atezolizumab-bevacizumab combination therapy has been approved for first-line systemic chemotherapy in advanced hepatocellular carcinoma. Real-world retrospective analysis was performed to evaluate the effectiveness and safety of the chemotherapy.

Materials and Methods:

A single-center, retrospective cohort study was performed. Between January 2020 and July 2023, patients with advanced hepatocellular carcinoma who received atezolizumab-bevacizumab systemic therapy were enrolled. Response to the therapy was assessed with mRECIST criteria. Evaluation of first radiologic response, best overall response, progression-free survival, and overall survival was done. Safety data were collected. Also, prognostic factors for first radiologic response, progression-free survival, and overall survival were also assessed.

Results:

A total of 70 patients were investigated. Baseline characteristics showed a median age of 63 years, male dominance (89%), and viral etiology (84%). Four patients achieved complete response and 19 reached partial response when evaluated by best overall response. Response duration was 4.7 months (range 1.5–25.5). Median progression-free survival was 4.1 months (95% CI, 3.7–6.7), and median overall survival was 24.6 months (95% CI, 11.6–). Age and tumor extent of over 50% of the liver was predictive of progression-free survival ($p = 0.009$ and $p = 0.007$, respectively). Tumor extent of over 50% of the liver and ALBI score were predictive of overall survival ($p = 0.025$ and $p = 0.004$, respectively).

Conclusions: Atezolizumab-bevacizumab therapy showed valuable outcomes and tolerable safety profiles in real-world advanced HCC patients. The outcome and safety results were comparable to previous studies.

Keyword(s) :

1999 - Malnutrition and its association with mortality in liver cirrhosis a prospective nutritional assessment in northern Iran

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Background / Hypothesis:

Today chronic liver disease is developing and liver cirrhosis mortality is increasing in Iran. Malnutrition is a complication of liver cirrhosis and is associated with poor outcomes. This study aimed to determine the association between malnutrition and mortality in patients with liver cirrhosis in northern Iran.

Materials and Methods:

This prospective cross-sectional study was conducted on individuals with liver cirrhosis who lived in northern Iran. Patient information, including demographics (age, gender, etiology of cirrhosis, alcohol and smoking history), as well as triceps skinfold (TSF), mid-arm circumference (MAC), Child-Pugh score, and Model for End-Stage Liver Disease (MELD) score, was recorded for all patients. Patients were classified A, B, and C based on the Subjective Global Assessment (SGA) questionnaire.

Results:

A total 984 participants with the mean age 51.38 were recruited in this study. Viral infections were the most common etiology and 61.7% of participants were in the classification A of Child-Pugh Score. TSF ($P < 0.01$) and MAC ($P < 0.001$) were significantly lower in SGA class C. The survival rate in SGA class C was significantly lower than other groups ($P < 0.01$; log-rank test). The risk of mortality in patients with SGA class A and B was 87% (HR value: 0.21; 95% CI: 0.04-0.78; $P < 0.05$) and 72% (HR value: 0.31; 95% CI: 0.07-0.79; $P < 0.02$) lower than those with SGA class C.

Conclusions:

Our findings showed that SGA measure is easy and accessible methods for assessing malnutrition and mortality in individuals with liver cirrhosis.

Keyword(s) :

2001 - Retrospective Analysis of Clinical and Genetic Characteristics in 68 Adult Patients with Gilbert Syndrome from a Tertiary Hospital in Southwest China

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Background / Hypothesis:

Gilbert syndrome (GS) is a benign hereditary condition caused by reduced UGT1A1 activity, leading to intermittent unconjugated hyperbilirubinemia. Data on

adult GS patients in China remain limited, especially in the Southwest region. This study aimed to investigate the clinical features and UGT1A1 variant spectrum in adult GS patients from a tertiary hospital in Southwest China.

Materials and Methods:

This retrospective study included 68 adult patients diagnosed with GS between 2019 and 2022. Demographic characteristics, symptoms, bilirubin profiles, liver biochemical tests, and UGT1A1 sequencing results were analyzed.

Results:

Among the 68 patients, the median age was 30 years (IQR 21.0–42.5), and 41 (60.3%) were male. All patients exhibited elevated bilirubin, and 18 (26.5%) presented with jaundice. The mean total bilirubin was 86.9 ± 45.2 $\mu\text{mol/L}$, with a median indirect bilirubin of 70.0 $\mu\text{mol/L}$ (IQR 50.43–98.73).

UGT1A1 analysis revealed that 56 patients carried a single variant, 11 carried two variants, and 1 carried three variants. The most frequent variants were UGT1A1*28 (41 cases) and UGT1A1*6 (21 cases). Seven patients harbored rare variants, including five PBREM enhancer region variants, one exon 1 missense mutation (c.668G>A), and one UGT1A1*7 mutation (c.1456T>G).

Conclusions:

UGT1A1*28 and UGT1A1*6 were the predominant variants among adult GS patients in Southwest China. Rare promoter, enhancer, and exonic mutations also contributed to the genetic heterogeneity of GS. Genetic testing is valuable for adults presenting with isolated indirect hyperbilirubinemia and may reduce unnecessary diagnostic investigations.

Keyword(s) : Gilbert syndrome, UGT1A1 variants, Unconjugated hyperbilirubinemia

2003 - Mechanism Based Efficacy and Advantage of Traditional Chinese Medicine in Treating Anemia Associated with HBV Related Hepatocellular Carcinoma from an Inflammatory Perspective

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Beijing-China ⁴

Background / Hypothesis:

This study aimed to identify independent prognostic factors for patients with concurrent hepatitis B virus (HBV)-related hepatocellular carcinoma (HCC) and cancer-related anemia (CRA), and to investigate the impact of traditional Chinese medicine (TCM) on patient outcomes. Furthermore, the objective was to determine the patient subset most likely to benefit from TCM intervention for guiding clinical practice.

Materials and Methods:

A total of 532 patients diagnosed with coexisting HBV-related hepatocellular carcinoma and cancer-related anemia at Beijing Ditan Hospital between June 2008 and June 2017 were included. The cohort comprised 318 patients who received TCM treatment and 214 patients who underwent exclusive modern medical therapy. To evaluate the association between TCM intervention and patient survival, multivariable Cox regression analysis was employed to estimate hazard ratios (HRs) while adjusting for potential confounding factors. Kaplan-Meier survival curves were generated to visualize survival distributions, and dendrograms were utilized to explore hierarchical relationships among prognostic factors and stratify mortality risks in this patient population. Building upon the observed synergistic interaction between C-reactive protein (CRP) and alpha-fetoprotein (AFP) in predicting clinical outcomes, this study further developed a decision tree model.

Results:

Patients who received TCM treatment for more than six months demonstrated a significantly higher survival rate compared to those treated with TCM for less than six months. AFP and CRP were identified as independent risk factors for 1-year survival within the TCM treatment group. The subgroup characterized by low levels of both AFP (≤ 350 ng/mL) and CRP (≤ 5 mg/L)—designated as the LALC subgroup—exhibited the most favorable survival outcomes. A decision tree model constructed based on CRP, AFP, platelet count (PLT), and hemoglobin (HGB) provided an intuitive tool for prognostic prediction. Among these variables, CRP level demonstrated a particularly strong association with patient outcomes: patients with CRP ≤ 5 mg/L achieved a 1-year survival rate of 87%, constituting 47% of the study population. Additional indicators positively influencing prognosis in patients with HCC

complicated by anemia included AFP ≤ 350 ng/mL, PLT $\leq 100 \times 10^9$ /L, and HGB < 90 g/L. Patients meeting all these criteria collectively achieved a 1-year survival rate of 60%, representing 11% of the study cohort.

Conclusion: TCM demonstrated protective effects in patients with HBV-related HCC and CRA, and survival was positively associated with the duration of TCM use. Patients with low CRP and AFP levels represent the optimal population for TCM treatment to enhance prognosis. Observing the levels of CRP, AFP, platelets, and hemoglobin can help predict the prognosis.

Keyword(s) : traditional Chinese medicine; hepatitis B virus-related hepatocellular carcinoma; cancer-related anemia; efficacy analysis; superior-effect population

2005 - The positive impact of the national HBV vaccination program implemented in Turkey on the prevalence of HBV among health school students

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Background / Hypothesis: This study aimed to determine hepatitis B virus (HBV) exposure among students enrolled in health-related schools who were attending internships at a training and research hospital in Izmir.

Material and methods: After receiving ethics committee approval, students' HBsAg, anti-HBcIgG, and anti-HBs results were obtained either from the hospital automation system or from the students' personal reports. Türkiye's national HBV vaccination program began in 1998, followed by a catch-up program in schools between 2005 and 2009; thus, individuals born between 1991 and 1997 were also theoretically included in the vaccination cohort. Study data were evaluated based on birth years corresponding to these vaccination periods.

Results: A total of 1,174 students born between 1991 and 2007 were included; 898 were female and 276 were male. HBsAg positivity was detected in five students: two females (born in 1997 and 2003) and three males (born in 1991, 1992, and 2006). Of the nine students who had natural immunity due to past HBV exposure, three were born in 1997, two in 1991, and one each in 1992, 2003, 2005, and 2006. Among the 1,160 students who were not exposed to HBV and were anti-HBcIgG negative, 81% had protective anti-HBs levels ≥ 10 IU/mL.

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A decline in anti-HBs titers over time after infant vaccination is expected. In this study, students born in 2000 or later—who received only the three-dose infant series without subsequent boosters—had significantly higher rates of non-protective antibody titers. To identify when titers began to decline, students with low anti-HBs levels (10–50 IU/mL) were analyzed. Low titers were found in 29.3% of students born between 2000 and 2002, 23.2% of those born in 2003–2004, and 13.6% of those born between 2003 and 2007. All students with negative titers showed an anamnestic response following a booster dose, confirming preserved immune memory.

Conclusion: Overall, the national HBV vaccination program has been highly effective for students preparing for healthcare professions. Among those born after the vaccination program began, 98.8% (1160/1174) were protected through vaccination. Of the five students found to be HBsAg positive, three were born before the vaccination program and two after; in all cases, their mothers were HBsAg positive, suggesting perinatal transmission. The findings highlight both the success of the vaccination program and the importance of monitoring anti-HBs levels in individuals vaccinated only in infancy, as their antibody titers may fall below protective levels over time despite preserved immune memory.

Keyword(s) : HBV, health school student, vaccination

2007 - A Survey on Awareness of Antiviral Therapy Among CHB Patients

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Background / Hypothesis: The overall treatment rate for CHB in China is currently only 27%. This study aimed to evaluate, from the patient perspective, the reasons for not initiating antiviral therapy and the level of awareness regarding antiviral treatment.

Materials and Methods: From August 2025 to November 2025, a self-administered electronic questionnaire was used to collect data from 502 CHB patients attending the Department of Infectious

Diseases in our hospital. The questionnaire contained 16 items covering three domains: awareness of antiviral therapy, reasons for not receiving treatment, and clinical care needs.

Results: Among the 502 surveyed CHB patients, 336 were currently receiving antiviral therapy, yielding an overall treatment rate of 67%; the proportion of patients who had not initiated antiviral therapy remained as high as 33%. A total of 449 patients (89.4%) were over 30 years of age, of whom 132 (29.4%) had not started antiviral therapy (Baseline characteristics shown in Table 1). The results indicated that age, educational level, occupation, medical insurance coverage, economic status, time since CHB diagnosis, and frequency of medical visits were significantly associated with treatment uptake ($P < 0.05$). Sex was not significantly associated with antiviral treatment ($P > 0.05$). Analysis of reasons for not initiating antiviral therapy showed that: 74.7% were unwilling to take long-term medication, 62% were concerned about drug side effects, and 54.8% reported that their physicians had not recommended treatment (Figure 1). Regarding awareness of antiviral therapy, the Likert 5-point scale revealed high scores for “I clearly understand that antiviral therapy reduces the risk of hepatocellular carcinoma” and “I trust my physician’s treatment recommendations,” with scores of 4.39 ± 0.72 and 4.39 ± 0.73 , respectively (one-sample t tests, $P < 0.05$), indicating positive perceptions. In contrast, “I worry that treatment will affect my work or daily life” scored 2.41 ± 1.03 ($P < 0.05$), reflecting negative perceptions that require enhanced patient education. Additionally, 62.5% of patients reported that receiving clear explanations from physicians regarding long-term medication use, drug safety, and other treatment-related concerns would encourage them to initiate therapy. Moreover, 80.3% expressed a desire for a dedicated communication platform for physician–patient interaction

Conclusions: The treatment rate for CHB requires further improvement. Patients who have not initiated antiviral therapy show notable gaps and misconceptions regarding treatment. Targeted and standardized management strategies in clinical practice are needed to promote early antiviral treatment

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Keyword(s) : hepatitis B; antiviral therapy; questionnaire survey

2009 - A research on gastroesophageal varices in patients with liver cirrhosis of different etiologies

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Background / Hypothesis: To explore the differences in the manifestations of gastroesophageal varices in patients with liver cirrhosis of different etiologies.

Materials and Methods: A retrospective analysis was conducted on 101 patients with viral hepatitis, alcoholic hepatitis, and cirrhosis after autoimmune liver disease who were randomly admitted to the medical institution where the researcher worked. The bleeding, degree, location, maximum diameter, red sign, and risk factors of gastroesophageal varices were observed and compared.

Results: In terms of the degree of varicose veins, the proportion of mild varicose veins in the viral hepatitis group (3.03%) was lower than that in alcoholic liver disease (22.73%), and the difference was statistically significant ($P < 0.05$). At the same time, the proportion of mild varicose veins in the viral hepatitis group was lower than that in the autoimmune liver disease group (23.08%), and the difference was statistically significant ($P < 0.05$); The positive rate of RC in the viral hepatitis group (59.09%) was higher than that in the alcoholic liver disease group (27.27%), and the difference was statistically significant ($P < 0.05$); In terms of the maximum inner diameter (mm) of the portal vein in patients with viral hepatitis, it was 15.00 (14.00, 16.00) higher than that in the alcoholic liver disease group, which was 13.50 (11.00, 14.75), and the difference was statistically significant ($P < 0.05$). At the same time, the portal vein inner diameter in the viral hepatitis group was larger than that in the autoimmune liver disease group, which was 12.00 (11.00, 14.00), and the difference was statistically significant ($P < 0.05$).

Conclusions: The maximum diameter of the portal vein and the condition of gastroesophageal varices vary in patients with different etiologies of liver cirrhosis. Patients with viral hepatitis and liver cirrhosis have thicker portal veins, a higher proportion of mild to severe gastroesophageal varices, and a higher positive rate of red signs. Therefore, it is even more important to be vigilant about the rupture and bleeding of

gastroesophageal varices. Active antiviral treatment should be given to patients with viral hepatitis who have indications for antiviral therapy to reduce the likelihood of developing cirrhosis and complications such as esophagogastric variceal bleeding.

Keyword(s) :

2011 - Integrated Multi omics Liquid Biopsy Advancing Early Detection and Precision Management of HCC

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality worldwide, often diagnosed at late stages due to the limited sensitivity and specificity of current surveillance methods such as ultrasound and alpha-fetoprotein (AFP) testing. Liquid biopsy, which non-invasively analyzes circulating biomarkers, offers a promising alternative (Fig 1). However, single-omics approaches often lack the robustness required for early detection. Multi-omics integration presents a transformative strategy to enhance diagnostic accuracy and enable precision oncology in HCC.

Materials and Methods:

This review synthesizes recent advances in multi-omics liquid biopsy for HCC, incorporating genomic (e.g., TERT, TP53 mutations), epigenomic (e.g., RASSF1A methylation), proteomic/glycomic (e.g., AFP-L3, GP73, exosomal glycans), fragmentomic (cfDNA fragmentation patterns), and cellular biomarkers (CTCs, EVs) (Fig 2). Cross-omics integration strategies—such as cSMART, HIFI, and ASDAm-H1—were evaluated alongside emerging technologies including nanopore sequencing, SERS-based single-exosome analysis, and machine learning models (e.g., XAI, GNNs, Bayesian Networks) (Fig 3).

Results:

The integration of multi-omics biomarkers has demonstrated transformative diagnostic performance for early HCC (Table 1). Cross-omics models consistently outperformed single-modality approaches:

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combined genomic-epigenomic assays achieved sensitivities of 80–90% for early-stage detection, while the fragmentomic-epigenomic HIFI model distinguished HCC from cirrhosis with exceptional accuracy (AUC 0.996, sensitivity 95.42%, specificity 97.83%). Integrated protein-epigenetic panels, such as the ASDAm-H1 index, reached an AUC of 0.96, and longitudinal application of multi-parametric scores like GALAD significantly improved sensitivity to 69.2% for early HCC compared to single measurements (53.8%). Fragmentomics-based models enabled early screening with sensitivities exceeding 96%, and metabolomic panels detected preclinical HCC up to one year before clinical diagnosis with 80% sensitivity. Beyond detection, multi-omics liquid biopsy proved vital for precision management: ctDNA mutation profiling enabled dynamic monitoring of targeted therapy response, PD-L1⁺ CTCs and cfDNA copy number variations predicted outcomes to immune checkpoint inhibitors, and machine learning-driven deconvolution of single-exosome SERS fingerprints or cfDNA methylation data provided unprecedented molecular resolution. These approaches not only identified AFP-negative HCC cases but also facilitated early recurrence prediction and molecular subtyping, effectively transitioning liquid biopsy from a passive detection tool to an active component in the dynamic, personalized management of HCC.

Conclusions:

Multi-omics liquid biopsy represents a paradigm shift in HCC management, enabling high-accuracy early detection and dynamic monitoring of disease progression and treatment response. Despite challenges in data integration, standardization, and clinical validation, future directions—including explainable AI, single-cell multi-omics, and closed-loop precision oncology systems—hold great promise (Fig 4). With continued interdisciplinary collaboration and standardization efforts, multi-omics liquid biopsy is poised to become the cornerstone of next-generation HCC care.

Keyword(s) : Hepatocellular carcinoma, Liquid biopsy, Multi-omics integration, Early diagnosis, Clinical translation

2013 - Projected health and economic impact of physical activity on metabolic dysfunction associated steatotic liver disease in Saudi Arabia A

Markov modeling study

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Background:

Inadequate physical activity (PA) remains a global health concern, linked to various chronic conditions, including metabolic dysfunction-associated steatotic liver disease (MASLD). In Saudi Arabia, MASLD affects over 30% of the population. Increasing PA has been shown to reduce hepatic fat and improve liver outcomes. This study aimed to project the future health and economic burden of MASLD under varying PA scenarios in Saudi Arabia.

Methods:

A Markov model was used to simulate disease progression from 2025 to 2045, stratified by age and gender. The model incorporated MASLD prevalence, natural history, and mortality, and was validated using national datasets from the Ministry of Health and General Authority for Statistics, including data on hepatocellular carcinoma (HCC). Three intervention scenarios were modeled to assess the impact of PA on MASLD, assuming 10%, 20%, and 30% of individuals with MASLD achieved a 30% relative reduction in hepatic fat, based on clinical trial evidence associated with histological improvement. Health and economic impacts were analyzed using U.S.-based cost data, adjusted for medical inflation, and expressed in disability-adjusted life years (DALYs). Economic inputs were held constant across scenarios to ensure consistency and isolate the effect of PA on the economic burden of MASLD.

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Results:

In scenarios where 10%, 20%, and 30% of individuals with MASLD achieved a 30% hepatic fat reduction, cumulative liver-related deaths declined by 4.0%, 7.9%, and 11.6%, respectively. Cumulative HCC cases were reduced by 4.7%, 9.3%, and 13.5%. Decompensated cirrhosis cases dropped by up to 13.3%, and total MASLD prevalence declined by approximately 4.6% in the highest scenario. A 30% PA scenario could prevent over 27,800 liver-related deaths and 5,300 HCC cases by 2045. Direct healthcare costs averted by 3.8%, 7.5%, and 11.1% across the scenarios, and DALYs averted ranged from 8.3% to 23.6%.

Conclusions:

Inadequate PA contributes to the increasing burden of MASLD in Saudi Arabia. Promoting PA can significantly reduce the health and economic impacts. The impact of MASLD should be included in national PA promotion campaigns and non-communicable disease advocacy efforts. These findings support the implementation of national PA promotion strategies under Saudi Vision 2030. While the model is based on Saudi data, the framework and findings may be applicable to other countries with high MASLD prevalence and inadequate PA.

Keyword(s) : MASLD, Physical Activity, Metabolic Dysfunction-associated Steatotic Liver Disease, Saudi Arabia, Economic Impact

2014 - Longitudinal Changes in Non Invasive Fibrosis Markers and Risk Factors for Fibrosis Progression in People with HIV and Metabolic Dysfunction Associated Steatotic Liver Disease

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Background / Hypothesis:

Longitudinal monitoring with the fibrosis-4 index (FIB-4) is valuable for tracking fibrosis progression and predicting outcomes in metabolic dysfunction-associated steatotic liver disease (MASLD). This study aimed to evaluate dynamic changes in FIB-4 and metabolic parameters in PWH during antiretroviral therapy and to identify risk factors for liver fibrosis progression (LFP).

Materials and Methods:

MASLD was diagnosed after excluding excessive alcohol consumption and other known causes of liver disease, based on the presence of hepatic steatosis along with at least one cardiometabolic risk factor. LFP was defined based on serial FIB-4 measurements. Participants were stratified by baseline FIB-4: those with a low level (<1.3) were considered to have progression if their FIB-4 increased by $\geq 20\%$ and reached ≥ 1.3 ; those with a high level (≥ 1.3) were considered to have progression with a $\geq 20\%$ increase. Metabolic parameters, including body weight, blood glucose, and lipid profiles, were assessed at baseline and the end of follow-up.

Results:

From March 25, 2024, to December 10, 2024, 2,003 people with HIV (PWH) were screened. After exclusions, 683 with MASLD were enrolled. After a median follow-up of one year, 28 (4.1%) participants were lost to follow-up. Among the remaining 655 participants, 59 (9.0%) exhibited fibrosis progression, and 596 (91.0%) remained stable. In multivariable logistic regression, both baseline age (OR = 1.10; 95% CI: 1.06–1.14; $P < 0.001$) and weight gain (OR = 1.29; 95% CI: 1.21–1.38; $P < 0.001$) were independently associated with LFP. Transition to obesity was also independently associated with LFP (OR = 6.56; 95% CI: 2.95–14.56; $P < 0.001$).

Conclusions: Liver fibrosis progression occurs in a non-negligible proportion of PWH with MASLD, even within a relatively short period. Weight gain and transition to obesity are significant risk factors for LFP in this population.

Keyword(s) : liver fibrosis; people with HIV; metabolic dysfunction-associated steatotic liver disease; FIB-4; obesity

2015 - A research on gastroesophageal varices in patients with liver cirrhosis of different etiologies

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Background / Hypothesis: To explore the differences in the manifestations of gastroesophageal varices in patients with liver cirrhosis of different etiologies.



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Materials and Methods: A retrospective analysis was conducted on 101 patients with viral hepatitis, alcoholic hepatitis, and cirrhosis after autoimmune liver disease who were randomly admitted to the medical institution where the researcher worked. The bleeding, degree, location, maximum diameter, red sign, and risk factors of gastroesophageal varices were observed and compared.

Results: In terms of the degree of varicose veins, the proportion of mild varicose veins in the viral hepatitis group (3.03%) was lower than that in alcoholic liver disease (22.73%), and the difference was statistically significant ($P < 0.05$). At the same time, the proportion of mild varicose veins in the viral hepatitis group was lower than that in the autoimmune liver disease group (23.08%), and the difference was statistically significant ($P < 0.05$); The positive rate of RC in the viral hepatitis group (59.09%) was higher than that in the alcoholic liver disease group (27.27%), and the difference was statistically significant ($P < 0.05$); In terms of the maximum inner diameter (mm) of the portal vein in patients with viral hepatitis, it was 15.00 (14.00, 16.00) higher than that in the alcoholic liver disease group, which was 13.50 (11.00, 14.75), and the difference was statistically significant ($P < 0.05$). At the same time, the portal vein inner diameter in the viral hepatitis group was larger than that in the autoimmune liver disease group, which was 12.00 (11.00, 14.00), and the difference was statistically significant ($P < 0.05$).

Conclusions: The maximum diameter of the portal vein and the condition of gastroesophageal varices vary in patients with different etiologies of liver cirrhosis. Patients with viral hepatitis and liver cirrhosis have thicker portal veins, a higher proportion of mild to severe gastroesophageal varices, and a higher positive rate of red signs. Therefore, it is even more important to be vigilant about the rupture and bleeding of gastroesophageal varices. Active antiviral treatment should be given to patients with viral hepatitis who have indications for antiviral therapy to reduce the likelihood of developing cirrhosis and complications such as esophagogastric variceal bleeding.

Keyword(s) : gastroesophageal varices; esophagogastric variceal bleeding; portal hypertension; liver cirrhosis

2016 - Comparing Protein Induced by Vitamin K Absence II and Alpha Fetoprotein in Hepatocellular Carcinoma Clinical and Tumor Related Correlations in a Filipino Cohort

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Background / Hypothesis:

AFP is the most widely used serum biomarker for HCC with increased levels linked to early recurrence, advanced stage, and unfavorable prognosis. PIVKA-II, a newer biomarker, has been shown to be more sensitive in detecting HCC. Some studies show that PIVKA-II levels also correlate with tumor size and Barcelona Clinic Liver Cancer (BCLC) Stage. This study aims to determine how these biomarkers correlate with the clinicopathologic features of HCC.

Materials and Methods:

This cross-sectional study reviewed records of adult HCC patients in a tertiary hospital. PIVKA-II and AFP levels, demographics, liver function tests, tumor characteristics, presence of portal vein thrombosis and metastases were recorded. Comparative analyses used the Kruskal-Wallis test and Dunn's post hoc comparisons. Receiver Operating Characteristic (ROC) analyses were conducted to assess the diagnostic performance of AFP and PIVKA-II as markers for portal vein thrombosis (PVT), lymphatic, and distant metastasis.

Results:

Among the 92 patients in this study, viral hepatitis-related HCC had significantly higher AFP ($p = 0.010$), not observed with PIVKA-II ($p = 0.071$). Increased levels of both biomarkers correlated with increased tumor size ($p = 0.009$ for AFP, $p = 0.002$ for PIVKA-II) and increased tumor number ($p = 0.010$ for AFP, $p < 0.001$ for PIVKA-II). Both biomarkers rose with advancing BCLC stages ($p = 0.010$ for AFP and $p = 0.002$ for PIVKA-II). The areas under the ROC curve (AUC) of lymph node metastasis in HCC were 0.711 (95% CI, 0.596-0.826, $p < 0.001$) for PIVKA-II, and 0.622 (95% CI 0.622-0.066, $p = 0.063$) for AFP, underscoring PIVKA-II as a superior predictor for lymph node metastasis. At a threshold of 2,960.50 mAU/mL, PIVKA-II showed 72% sensitivity for detecting lymph node metastasis, with a negative predictive value of 86% at lower levels. Neither biomarker showed strong predictive power for PVT or distant metastasis.

Conclusions:

AFP and PIVKA-II correlate with tumor burden and advanced HCC stages. Elevated PIVKA-II levels showed moderate predictive power for lymph node metastasis

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in HCC.

Keyword(s) : hepatocellular cancer (HCC), alpha-fetoprotein (AFP), protein induced by vitamin K absence-II (PIVKA-II), biomarkers, tumor markers

2017 - The Cost Changes in Hepatitis C Patients Before and After the Introduction of Direct Acting Antivirals in Korea

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Background / Hypothesis:

This study evaluates the economic impact of this treatment paradigm shift by analyzing changes in patient population and healthcare costs before and after Direct-Acting Antivirals (DAAs) introduction in Korea.

Materials and Methods:

Total costs were calculated by summing direct and indirect costs. Direct costs included medical costs and non-medical costs (time, transportation, and caregiving expenses). Indirect costs included productivity losses due to premature mortality from HCV-related liver disease, estimated using data from the Korean Statistical Information Service. Direct costs for acute and chronic HCV patients were analyzed using National Health Insurance Service data from 2012 to 2023. The prevalence method was employed for cost calculation. All costs were converted to US dollars (USD, \$1=1,388 KRW).

Results:

Over the 12-year period, 167,534 HCV patients were identified. Total costs rose from \$146 million (2012) to a peak of \$224 million (2016) after DAA introduction, then decreased by 51.8% to \$108 million by 2023. Per-patient costs peaked at \$4,425 in 2016 and declined to \$3,401 by 2020, but increased again to \$3,649 by 2023. Healthcare costs surged from \$31 million to \$120 million in 2016 with DAA introduction, but declined dramatically to \$35 million by 2023, while premature mortality costs decreased consistently from \$96 million to \$66 million over the study period.

Medication costs dominated healthcare expenditure, peaking at \$102.3 million in 2016 with DAA introduction but declining to \$26.8 million by 2023. Injection costs plummeted by 98% from \$12.0 million (2012) to \$0.2 million (2023), reflecting the shift from

interferon-based to DAA therapy. The introduction of pan-genotypic DAAs in 2018 further reduced diagnostic burden, with HCV RNA testing proportion dropping from 27.8% (2012) to 7.6% (2018) of laboratory costs.

During the 12-year period, 64,926 patients received antiviral treatment, with total medication costs of \$494.04 million. Treatment paradigm shifted dramatically from peginterferon-based therapy (\$12.69 million in 2012) to DAA (\$97.49 million in 2016), with peginterferon use virtually eliminated by 2018. DAA costs peaked in 2016 and subsequently declined by 75% to \$24.75 million by 2023.

Conclusions:

Over the 12-year study period spanning pre- and post-DAA eras, Korea experienced initial cost increases with DAA introduction in 2016, followed by continuous declines in both HCV patient numbers and healthcare costs. This likely stems from reduced drug prices, pan-genotypic treatment availability, and improved treatment efficiency. These findings suggest innovative treatments can reduce long-term disease burden, providing evidence for future infectious disease policy development.

Keyword(s) : HEPATITIS C VIRUS, Direct costs, Indirect costs, BIG DATA

2020 - FOXK1 aggravates acute liver failure through HIF 1 α mediated histone lactylation at H4K16

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Background / Hypothesis: Acute liver failure (ALF) is a severe clinical syndrome characterized by rapid onset, rapid progression, and poor prognosis. Recent studies have highlighted the role of epigenetic modifications in the pathogenesis of ALF, particularly histone lactylation, a novel post-translational modification involved in inflammation and metabolic disorders. FOXK1, a transcription factor implicated in hepatic metabolism and stress response, has not been fully explored in the context of histone lactylation and ALF. This study aimed to investigate whether FOXK1 aggravates ALF by promoting H4K16 histone lactylation through the HIF-1 α signaling pathway.

Materials and Methods: An LPS/D-GalN-induced mouse model of ALF was established. FOXK1 expression was knocked down using adenoviral vectors. Serum

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biochemical indices (ALT, AST) and lactate levels were measured. Liver pathology was assessed by H&E staining. Immunohistochemistry and Western blot were used to detect HIF-1 α and H4K16la expression. Chromatin immunoprecipitation (ChIP) was performed to analyze the interaction between FOXK1 and HIF-1 α . Results: In ALF mice, FOXK1 expression was significantly upregulated, accompanied by elevated levels of HIF-1 α and H4K16la. Knockdown of FOXK1 markedly attenuated liver injury, reduced serum ALT/AST and lactate levels. Mechanistic studies revealed that FOXK1 enhanced the stability and transcriptional activity of HIF-1 α , thereby promoting the expression of enzymes involved in lactate metabolism and driving H4K16 histone lactylation. Conclusions: This study reveals for the first time that FOXK1 aggravates acute liver failure by activating the HIF-1 α pathway and inducing H4K16 histone lactylation. The FOXK1-HIF-1 α -H4K16la axis may represent a potential therapeutic target for ALF intervention.

Keyword(s) : Acute liver failure; Histone lactylation; Epigenetics

2021 - Sex related differences in clinical and immunologic phenotype of male primary biliary cholangitis a retrospective cohort study

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Background / Hypothesis:

Primary biliary cholangitis is a chronic autoimmune cholestatic liver disease that predominantly affects women. However, the specific clinical and immunologic features of male disease and potential sex-related differences in phenotype remain incompletely defined.

Materials and Methods:

We conducted a retrospective cohort study of adults with primary biliary cholangitis at Beijing Youan Hospital, Capital Medical University. Clinical data, liver biochemistry, immunoglobulins, autoantibodies, and indices of disease severity were collected at diagnosis. The albumin-bilirubin score and imaging-based assessment were used to evaluate cirrhosis and portal hypertension. Patients were stratified by sex, and between-group comparisons were performed using independent-samples t tests or chi-square tests.

Results:

A total of 964 patients were included, of whom 114 were men and 850 were women. Men accounted for 11.8% of all cases and were older at diagnosis than women (59.55 vs 56.06 years, $P < 0.01$). Men had higher gamma-glutamyltransferase levels (402.07 vs 238.49 units per liter, $P < 0.001$), whereas other liver tests, immunoglobulins, blood counts, and prothrombin time were similar between sexes. Male patients showed a distinct autoantibody pattern, with a lower prevalence of anticentromere antibodies (14.0% vs 27.5%, $P = 0.002$) but a higher prevalence of anti-gp210 antibodies (46.5% vs 33.3%, $P = 0.005$); antimitochondrial and anti-sp100 antibody positivity rates were comparable (95.6% vs 92.4%, $P = 0.208$; 13.2% vs 10.7%, $P = 0.432$, respectively). The prevalence of cirrhosis, portal hypertension, and esophageal varices did not differ between men and women.

Conclusions:

In this large real-world cohort, male primary biliary cholangitis was characterized by later age at diagnosis, more pronounced cholestasis, and an autoantibody profile with higher anti-gp210 and lower anticentromere antibody positivity, while advanced disease manifestations were similar between sexes. These sex-related differences may reflect divergent disease biology and support consideration of male-specific serologic and cholestatic features when tailoring risk assessment and clinical management.

Keyword(s) : Primary biliary cholangitis, Sex differences, Male patients, Autoantibodies, Anti-gp210, Anticentromere antibody, Cholestasis

2022 - Xiaozhi Huaxian Formula Ameliorates Type 2 Diabetes Associated Metabolic Dysfunction Associated Steatohepatitis via the Gut Derived Acetate-Hepatic GPR43 Axis

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Background/Hypothesis:

The global increase in the incidence of type 2 diabetes

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mellitus-associated metabolic dysfunction-associated steatohepatitis (T2DM-associated MASH) poses a significant clinical challenge, as affected patients are at heightened risk for progressive liver injury, including fibrosis, cirrhosis, and hepatocellular carcinoma. The pathogenesis of T2DM-associated MASH is characterized by a complex interplay of metabolic dysregulation and aberrant immune activation. Xiaozhi Huaxian Formula (XHF), a patented Chinese herbal formulation based on a modified classical prescription (Patent No. ZL202310099104.1), has shown promising efficacy in alleviating T2DM-associated MASH in clinical observations. However, the precise molecular mechanisms underlying its hepatoprotective effects remain incompletely understood. Growing evidence underscores the critical role of the gut microbial metabolites in the pathogenesis of metabolic disorders. Therefore, this study aims to elucidate the therapeutic mechanism of XHF from the perspective of gut microbiota-immune system interactions.

Methods:

Male C57BL/6J pups at two days of age received a single subcutaneous injection of streptozotocin (200 µg/20 µL per mouse). Following weaning at 4 weeks, the mice were maintained on a high-fat diet until the end of week 9 to establish a T2DM-associated MASH model. For the in vivo arm of the study, mice were administered the XHF decoction via oral gavage daily for four consecutive weeks, beginning at week 5. Concurrently, hepatocytes were isolated from mice to analyze the mRNA expressions of GPR43, and liver tissue homogenate were prepared to examine pro-inflammatory cytokines levels. Following, hepatocytes were isolated from T2DM-associated MASH mice and subjected to in vitro treatment with 10% XHF-containing serum derived from XHF-fed healthy male C57BL/6J mice for subsequent cellular-level analyses.

Results:

Relative to normal controls, T2DM associated MASH mice exhibited a significant reduction in plasma acetate levels within the portal vein and in hepatocyte GPR43 mRNA expression, alongside a marked elevation of IL 6 and TNF α levels in liver tissue homogenates. XHF treatment dose-dependently ameliorated both gut microbial dysmetabolism and hepatic immune dysregulation in T2DM-associated MASH mice (all $p < 0.005$, respectively). In vitro, treatment with XHF containing serum significantly up regulated GPR43 mRNA expression in primary

hepatocytes from T2DM associated MASH mice compared with control serum, and concurrently suppressed acetate induced secretion of IL 6 and TNF α (all $p < 0.001$, respectively).

Conclusions:

XHF conferred protection against T2DM-associated MASH by enhancing the production of the anti-inflammatory gut microbial metabolite acetate and upregulating hepatic GPR43 signaling, thereby suppressing the production of pro-inflammatory cytokines in the liver.

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Keyword(s) : Xiaozhi Huaxian formula, T2DM, MASH, Acetate, GPR43, gut microbiota-immune system

2024 - Coronary heart disease is an independent risk factor for liver fibrosis progression in patients with MAFLD

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Background / Hypothesis: Coronary Heart Disease (CHD) and metabolic dysfunction-associated fatty liver disease (MAFLD) share lots of overlapping pathogenic mechanisms, and the population with both MAFLD and CHD is quite large. However, there are few clinical data on whether CHD is an independent risk factor for the progression of liver fibrosis in patients with MAFLD.

Materials and Methods: We retrospectively analyzed 270 MAFLD patients with CHD diagnosed by coronary angiography and 854 MAFLD patients undergoing health check-up in Beijing Friendship Hospital, Capital Medical University (Beijing, China). Propensity score matching (PSM) were employed to mitigate confounding effects, including age, gender, BMI and diabetes. Multivariate logistic regression analyses were used to examine the association between CHD and the

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progression of liver fibrosis (defined as FIB-4 increased by $\geq 30\%$) after a 2-year follow-up. Sensitivity analysis were conducted to test the robustness of the results.

Results:After PSM, among the 348 included patients with MAFLD, 174 cases (50%) were complicated with CHD, who had a median age of 53 years, a median BMI of 26.9 kg/m², and 86.2% were male. The number of cases with progression of liver fibrosis after a 2-year follow-up was 56 (32.2%) and 19 (10.9%) in the MAFLD with CHD group and the MAFLD group respectively. Multivariate logistic regression analysis revealed that after adjusting for age, gender, BMI, hypertension, diabetes, TG, HDL, AST and baseline FIB-4, CHD was a significant risk factor for the progression of liver fibrosis (OR = 3.43, 95% CI: 1.66 - 7.09). In sensitivity analysis, the endpoint was defined as FIB-4 increasing by $\geq$ one grade (FIB-4 was classified into three grades based on 1.3 and 2.67) after 2-year follow-up and the association remained significant after adjusting for confounding factors (OR = 2.42, 95% CI: 1.06 - 5.54).

Conclusions:In a retrospective cohort study of patients with MAFLD, CHD was a strong independent risk factor for the progression of liver fibrosis, indicating that prevention and treatment of CHD may reduce the risk of liver fibrosis.

Keyword(s) : MAFLD, CHD, liver fibrosis

2025 - Targeting the Gut Liver Axis Novel Strategies for Liver Cirrhosis Intervention

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Background / Hypothesis:Liver cirrhosis is a chronic disease characterized by fibrosis and structural damage, primarily caused by hepatitis B and alcohol in China. Current treatments are limited to symptom management. Recent gut-liver axis research offers new therapeutic insights.

Materials and Methods: This research involved systematically searching and reviewing relevant literature from databases including Web of Science, PubMed, and Google Scholar, integrating theoretical analysis with cutting-edge empirical studies. It first elucidates the core components (including gut microbiota, intestinal barrier, bile acid metabolism; and

immune crosstalk) and functions of the gut-liver axis. It then focuses on discussing how gut-liver axis disruption drives the inflammatory and fibrotic processes in cirrhosis through core pathways such as "gut microbiota dysbiosis - intestinal barrier damage - endotoxemia." Finally, it systematically summarizes the efficacy and molecular mechanisms of intervention strategies targeting the gut-liver axis (e.g., probiotics, fecal microbiota transplantation, bile acid receptor agonists) as demonstrated in preclinical and clinical studies.

Results:Analysis indicates that the gut-liver axis is a highly promising new target for the prevention and treatment of liver cirrhosis. Specifically:**Mechanistic Level:** Patients with liver cirrhosis exhibit significant gut microbiota dysbiosis and increased intestinal permeability. This leads to the translocation of gut bacteria and their metabolites to the liver via the portal vein, activating hepatic immune cells and triggering/sustaining chronic inflammation and hepatic stellate cell activation through signaling pathways such as TLR4, thereby exacerbating liver fibrosis.

Intervention Level: Strategies targeting the regulation of the gut-liver axis have been proven effective in ameliorating the progression of cirrhosis.

Conclusion : Targeting the gut-liver axis presents a promising therapeutic strategy for cirrhosis, warranting further mechanistic and clinical investigation.

Keyword(s) : Gut-liver axis; Liver cirrhosis; Gut microbiota; Microbiota-host interactions; Intestinal barrier; Pathogenesis.

2026 - A Bibliometric Analysis of Medicinal Food in Chronic Liver Disease Research

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Background / Hypothesis:Chronic liver disease (CLD) has a high global prevalence, and existing treatment options have limitations. Medicinal food, a key concept in traditional Chinese medicine (TCM), has demonstrated great potential in the prevention and management of CLD due to its safety profile and multi-target advantages. Consequently, related research is rapidly expanding. However, the current research landscape in this field is complex and fragmented, lacking a systematic review from a macro perspective. Therefore, this study employs bibliometric methods to visually analyze the research hotspots and development trends in this field, aiming to provide

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direction for future investigations.

Materials and Methods:Based on bibliometric methods, we systematically retrieved relevant research literature on medicinal food and CLD from the Web of Science Core Collection database (covering the period from January 1, 2005, to November 29, 2025). Using visualization software such as CiteSpace and VOSviewer, we conducted quantitative statistics and visual mapping analysis on annual publication volume, country/institutional collaboration networks, author co-occurrence, keyword co-occurrence, keyword clustering, and burst detection.

Results:A total of 832 publications were included. The analysis results show:The annual publication volume in this field shows a rapid growth trend, particularly over the past decade, indicating strong research vitality.China holds an absolute dominant position in this field, but international collaboration remains limited. Several core research teams have formed, yet inter-institutional collaboration networks need strengthening.Keyword cluster analysis revealed four core knowledge groups: 1) pharmacological mechanism research on specific herbs (e.g., Bupleurum, Salvia miltiorrhiza, Glycyrrhiza) and their active components (e.g., glycyrrhizic acid, curcumin); 2) the prevention and treatment of non-alcoholic fatty liver disease (NAFLD) as the current absolute focus; 3) molecular mechanisms such as "oxidative stress, insulin resistance, apoptosis" being core pathways for in-depth exploration; and 4) "hepatoprotective effects, safety, clinical efficacy" as common themes spanning basic to clinical research.Burst detection of keywords indicates that the research frontier is dynamically shifting from traditional "anti-hepatic fibrosis" towards emerging interdisciplinary fields such as "gut microbiota," "immunomodulation," "network pharmacology," and "multi-omics integration."

Conclusions: This study visually elucidates the research panorama of medicinal food in the CLD field through knowledge mapping. Current research hotspots focus on the modern pharmacological interpretation of core herbs, while future trends will place greater emphasis on holistic mechanism exploration based on systems biology concepts and the translation of high-quality clinical evidence. This field holds broad prospects for development.

Keyword(s) : Medicinal food; Chronic liver disease; Bibliometrics; Knowledge map; Research front; Visual analysis.

2030 - Irritabl Bağırsak Sendromu Olan Reprodüktif Dönemdeki Kadınlarda Yaşam Kalitesinin Değerlendirilmesi

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Background / Hypothesis:

İrritabl bağırsak sendromu (İBS) toplumda sık görülen bir hastalıktır. Bu çalışmada reprodüktif dönemdeki kadın İBS hastalarının yaşam kalitesi, İBS olmayan sağlıklı kontrollerle karşılaştırılması amaçlanmıştır.

Materials and Methods:

Çalışmaya Roma IV kriterlerine göre İBS tanısı alan 18-55 yaş arası ardışık 50 kadın hasta ve benzer demografik özelliklere sahip malign ve kronik bir hastalığı, İBS'si olmayan 50 sağlıklı kontrol alınmıştır. Her iki grubun demografik verileri, sağlık anksiyete ölçeği, BECK anksiyete ölçeği, yaşam kalite ölçeği anketleri doldurulmuş ve gruplar karşılaştırılmıştır.

Results:

İBS tanısı alan 50 hastanın yaş ortalaması 34.84 ± 11.44 (yaş dağılımı 18-54), kontrol grubun yaş ortalaması 28.18 ± 9.39 (dağılım18-53) idi. Her iki grubun demografik özellikleri benzer idi. İBS hastalarının 9'unda (%18) psikiyatrik hastalık vardı, kontrol grubunda 4 (%8) ($p<0.05$). İBS hastalarının 11'i (%22) sigara içiyordu, kontrol grubunda 6 (%12) ($p<0.05$). İBS hastalarında kontrol grubuna göre sağlık anksiyete ortalaması benzerken, BECK anksiyete ölçeği ve İBS yaşam kalite ölçeği kontrol grubuna göre anlamlı yüksek bulunmuştur (35.44 ± 8.8 vs 33.36 ± 8.8 $p>0.05$, 25.06 ± 9.4 vs 13.26 ± 10.8 $p<0.05$, 98.3 ± 22.3 vs 37.8 ± 6.6 $p<0.05$).

Conclusions:

Reprodüktif dönemdeki İBS hastalarında benzer yaş ve

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demografik sağlıklı kontrol grubuna göre sağlık anksiyete ölçeği benzerken, BECK puanı yaklaşık iki kat, İBS yaşam kalite ölçeği puanı yaklaşık 3 kat yüksek çıkmıştır.

Keyword(s) : irritable barsak sendromu, kadın, yaşam kalitesi

2031 - Global Burden and Trends of Primary Liver Cancer Attributable to Comorbid Type 2 Diabetes Mellitus Among People Living with NASH An Observational Trend Study from 1990 to 2021

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Background / Hypothesis:The synergistic interaction between non-alcoholic steatohepatitis (NASH) and type 2 diabetes mellitus (T2DM) is an emerging driver of hepatocellular carcinoma (HCC). However, a comprehensive global assessment of the liver cancer burden specifically attributable to this comorbidity is lacking. This study aimed to quantify the global burden and temporal trends of primary liver cancer due to NASH-T2DM comorbidity from 1990 to 2021.

Materials and Methods:Using Global Burden of Disease (GBD) 2021 data from 204 countries (1990-2021), we estimated liver cancer burden attributable to NASH-T2DM comorbidity. Population attributable fractions (PAFs) were calculated using Levin's formula. We analyzed age-standardized prevalence, disability-adjusted life years (DALYs), and mortality rates, stratified by region, sociodemographic index (SDI), sex, and age. Temporal trends were assessed using estimated annual percentage change (EAPC).

Results:In 2021, the global age-standardized prevalence and DALY rates were 13.6 (95% Uncertainty Interval [UI]: 2.2–36.4) and 257.1 (95% UI: 42.9–688.6) per 10,000,000 individuals, respectively. The highest burdens were observed in high-SDI regions, particularly High-income Asia Pacific and North America. The disease burden was approximately five-fold higher in individuals aged ≥60 years, and males exhibited a higher burden across most age groups. While EAPC point estimates were positive across all

regions, wide uncertainty intervals precluded statistical significance, indicating substantial uncertainty in temporal trends.

Conclusions:The comorbidity of NASH and T2DM represents a substantial and growing driver of the global liver cancer burden, disproportionately affecting high-income nations and older adults. The observed statistical uncertainty highlights critical gaps in global surveillance and diagnosis of metabolic-associated liver cancer. These findings highlight the need for integrated metabolic health strategies, region-specific prevention, and targeted surveillance for high-risk populations.

Keyword(s) : Non-alcoholic steatohepatitis, type 2 diabetes mellitus, liver cancer, Global Burden of Disease, prevalence, Disability-adjusted life years

2032 - The Therapeutic Potential of Traditional Chinese Medicine in the Management of Chronic Hepatitis B

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Background / Hypothesis:The pathogenesis of chronic hepatitis B (CHB) involves complex mechanisms, with hepatitis B virus-induced immune responses playing a central role in hepatocyte injury and inflammatory necrosis. This condition is not only a driver of liver pathology but also demonstrates significant associations with various extrahepatic manifestations, including depression, cognitive dysfunction, diabetes mellitus, insomnia, non-alcoholic fatty liver disease, and thrombocytopenia. Conventional antiviral therapy faces limitations such as low hepatitis B surface antigen clearance rates and frequent adverse drug reactions.

Materials and Methods:By reviewing and analyzing relevant studies on representative TCM formulations (e.g., Yinchenhao Decoction, Xiaochaihu Decoction, and Fuzheng Huayu Tablet), their modes of action were summarized. These involve multi-ingredient, multi-target approaches to modulate immune pathways and intervene in key stages of the HBV lifecycle. **Results:**TCM achieves a tripartite therapeutic synergy in CHB management, encompassing immune reconstitution, viral suppression, and hepatic repair. It demonstrates distinct immunomodulatory effects and

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therapeutic advantages.

Conclusions: Traditional Chinese Medicine holds significant value in the comprehensive management of chronic hepatitis B. Future research should employ modern technologies to further elucidate its mechanisms, facilitating the development of standardized and optimized treatment protocols with considerable potential for clinical translation.

Keyword(s) : Chronic Hepatitis B; Traditional Chinese Medicine; Immunomodulation; Multi-target Therapy; Integrated Chinese and Western Medicine

2033 - Risk stratification of chronic kidney disease using noninvasive tests in metabolic dysfunction associated steatotic liver disease

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Background / Hypothesis: The association between metabolic dysfunction-associated steatotic liver disease (MASLD) and chronic kidney disease (CKD) is well known, yet data identifying high risk patients remain scarce. We investigated whether the European Association for the Study of the Liver (EASL)/ Korean Association for the Study of the Liver (KASL) and the American Gastroenterological Association (AGA) algorithms can stratify CKD risk in patients with MASLD.

Materials and Methods: This retrospective cohort study included 3,240 MASLD patients without pre-existing CKD who underwent vibration-controlled transient elastography between April 2006 and October 2018. The primary outcome was incident CKD, defined as estimated glomerular filtration rate (eGFR) <60 mL/min/1.73m² or proteinuria (≥1+ on dipstick) on two consecutive measurements. Secondary outcome included a 25% eGFR decline on two consecutive visits and 3- and 5-year CKD risk.

Results: The mean age was 51.7 years and 1,899

(58.6%) patients were male. During a mean follow-up of 3.3 years, 187 (5.8%) patients developed incident CKD. When stratified by EASL/KASL algorithms, multivariable Cox models revealed a 2.40-fold (95% confidence interval [CI], 1.57-3.66) higher risk of incident CKD in the high risk group compared to the low risk group. The high risk group also had a 2.16-fold higher risk (95% CI, 1.63-2.87) of a 25% eGFR decline (mean follow-up 4.3 years) and a 2.24-fold (95% CI, 1.29-3.91) higher 5-year CKD risk. Similar findings were observed for the AGA algorithm.

Conclusions: The two-step algorithms proposed by the EASL/KASL and AGA can effectively stratify CKD risk in patients with MASLD.

Keyword(s) : Metabolic dysfunction-associated steatotic liver disease; chronic kidney disease; liver fibrosis; fibrosis-4 index; vibration-controlled transient elastography.

2034 - Pathomorphological characteristics of the marginal gingiva in patients with viral hepatitis B

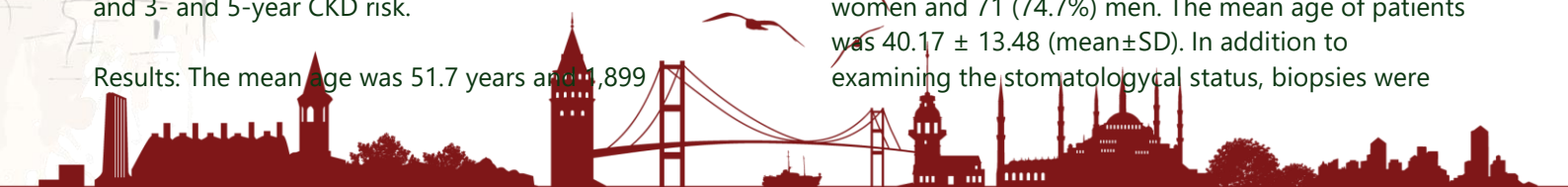
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Background / Hypothesis:

Oral cavity examinations in chronic diffuse liver diseases are of great interest to dentistry, since pathological processes developing in the liver usually lead to organic and functional disorders in the oral cavity. Every year, 30,000 healthcare workers worldwide become infected with HBV in their professional activities, one of whom dies. Stomatologists are a high-risk group for hepatitis B virus infection. The aim of this study was to describe the marginal gingiva in patients with HBV.

Materials and Methods:

The study included 95 patients with HBV, 24 (25.3%) women and 71 (74.7%) men. The mean age of patients was 40.17 ± 13.48 (mean±SD). In addition to examining the stomatological status, biopsies were



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taken from the marginal gingiva for pathological examination. The tissue samples were fixed in 10% neutral formalin, dehydrated, and embedded in paraffin according to standard histological procedures. A series of 5- μ m-thick sections were made from the blocks and stained with hematoxylin and eosin and picrofuchsin according to Van Gieson for a general assessment of the condition of the tissues being studied. The slides were examined using a Primostar Zeiss trinocular microscope at 100x and 400x, (immersion) magnification.

Results:

In the oral cavity, the primary site of pathological process was the alveolar gingiva—in 100% of patients. Inflammatory infiltration was lymphoplasmacytic in 95% (90) of patients ($p < 0.001$). Neutrophil admixture was observed in 40% (38) of patients ($p < 0.005$). Circulatory disorders were manifested by edema, hemorrhage. Changes in the squamous epithelium were in the form of acanthosis, parakeratosis and thickening (Fig. 1,2). The inflammatory infiltrate was primarily composed of lymphocytes, plasma cells, and segmented leukocytes ($p < 0.001$). This is a manifestation of both severe chronic inflammation and the involvement of immune mechanisms in the development of the pathological process.

Conclusions: Thus, numerous morphological signs in patients with HBV can be conditionally divided into those that are indicators of the severity and activity of inflammation and those associated with a long-term chronic course of inflammation of the alveolar part of the gum.

Keyword(s) : HBV, marginal gingiva, oral cavity, biopsies

2036 - Targeting T Cell Exhaustion in Chronic Hepatitis B Therapeutic Potential and Mechanisms of Traditional Chinese Medicine and natural products

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Background / Hypothesis: Hypothesis Chronic hepatitis B (CHB) remains a global health challenge, with functional cure rarely achieved by nucleos(t)ide analogues (NAs) due to the profound exhaustion of HBV-specific T cells. Unlike in oncology, single-target checkpoint blockade in CHB often fails due to the

compensatory redundancy of inhibitory networks and deep-seated epigenetic and metabolic barriers. We hypothesized that Traditional Chinese Medicine (TCM) and natural products, characterized by their multi-component and holistic regulatory properties, could offer a systems-level therapeutic strategy to synergistically dismantle these multifaceted exhaustion mechanisms and restore durable antiviral immunity.

Materials and Methods: This review systematically synthesizes mechanistic insights, preclinical experimental evidence, and clinical data to evaluate the therapeutic potential of TCM in ameliorating HBV-specific T cell exhaustion. We categorized and analyzed distinct bioactive phytochemicals (flavonoids, terpenoids, alkaloids, and polysaccharides) and compound formulas, focusing on their specific molecular interventions in antigen persistence, inhibitory receptor signaling, metabolic dysfunction (e.g., mitochondrial depolarization), and the immunosuppressive hepatic microenvironment.

Results: Pharmacological evidence demonstrates that active TCM components exert a "dual-action" effect: they directly suppress viral replication to lower the antigen load driving exhaustion and simultaneously remodel the intracellular metabolic and transcriptional landscapes of T cells to reverse functional paralysis. Specifically, agents like Astragalus polysaccharides and various Ginsenosides were found to enhance T-cell bioenergetics and downregulate inhibitory checkpoints (PD-1, TIM-3). Clinically, meta-analyses and randomized controlled trials indicate that integrating TCM with NAs significantly improves HBsAg clearance rates, promotes HBeAg seroconversion, and reduces off-therapy virological relapse compared to NA monotherapy, with a favorable safety profile.

Conclusions: TCM represents a vital mechanistic partner to conventional antivirals, capable of breaching the barriers of T-cell exhaustion that current therapies cannot address alone. To advance this from an alternative therapy to precision immunotherapy, future research must leverage multi-omics and deep phenotyping to rigorously map molecular targets and standardize "minimal effective sets" for global clinical

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application.

Keyword(s) : Chronic Hepatitis B; T Cell Exhaustion;
Traditional Chinese Medicine; Immunomodulation;
Phytochemicals

2037 - Assessment of hemoglobin A1c in hemodialysis patients with diabetes before and after erythropoietin treatment

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Background / Hypothesis:

Hemoglobin A1c (HbA1c) remains the most widely utilized laboratory marker for assessing long-term glycemic control. However, its reliability as a metabolic indicator in diabetic patients (DM) undergoing hemodialysis (HD) and receiving erythropoietin (EPO) therapy continues to be a subject of ongoing debate, primarily due to altered erythrocyte turnover characteristic of this patient population. Objectives were to assess EPO treatment effect on HbA1c levels and its reliability as a glycemic marker in diabetic patients undergoing regular HD.

Materials and Methods:

This prospective cohort study was carried out on 80 patients with renal disease undergoing regular HD, both prior to and following a three-month course of EPO therapy. Participants were stratified into two groups: Group I (n = 40) comprised EPO-naïve diabetic patients on regular HD, while Group II (n = 40) consisted of EPO-naïve nondiabetic patients receiving same treatment. All patients underwent clinical evaluation, comprehensive laboratory testing, and HbA1c assessment at baseline and after three months of EPO administration.

Results:

At baseline, Group I showed significantly higher HbA1c ($7.5 \pm 0.61\%$ vs. $4.4 \pm 0.68\%$, $P < 0.001$), fasting insulin, and HOMA-IR relative to Group II. After EPO therapy, HbA1c significantly declined in Group I (to $5.2 \pm 0.61\%$, $P < 0.001$) without significant changes in FBG. HbA1c demonstrated a significant moderate negative

correlation with Hb levels post-EPO in both groups ($r = -0.585$, $P < 0.001$, Group II).

Conclusions:

HbA1c proved to be an unreliable indicator of glycemic control in this patient population, as EPO therapy in diabetic individuals undergoing regular HD was associated with a notable reduction in HbA1c levels, despite the absence of a corresponding change in FBG and a lack of direct correlation between HbA1c and FBS.

Keyword(s) : HbA1c, EPO, Hemodialysis, Chronic kidney disease

2038 - Normal FIB 4 values do not exclude advanced fibrosis in Egyptian MASLD patients

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the leading cause of chronic liver disease worldwide. Early identification of individuals at risk of advanced fibrosis is essential to ensure timely referral to specialist care. Current guidelines recommend initial risk stratification using non-invasive fibrosis scores such as FIB-4 and/or the NAFLD Fibrosis Score, followed by transient elastography when indicated. This study aimed to evaluate the performance of low FIB-4 values in patients with MASLD compared with transient elastography and histology at baseline, and to document their long-term clinical outcomes.

Materials and Methods:

Consecutive patients diagnosed with MASLD between 2023 and 2025 were retrospectively reviewed. Clinical, anthropometric, and biochemical data, as well as outcomes at the end of follow-up, were collected. Clinical outcomes included mortality, cardiovascular events, extrahepatic malignancies, and liver-related outcomes (new cirrhosis, hepatocellular carcinoma, or hepatic decompensation defined as encephalopathy, variceal bleeding, or ascites). Baseline FIB-4 values were compared with liver stiffness measurements (LSM)

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obtained by FibroScan®, fibrosis stage on liver biopsy, and subsequent clinical events. Statistical analyses were performed using SPSS.

Results:

A total of 263 patients were included; 66% were male, the median age was 51 years, and the median BMI was 29.6 kg/m². One hundred forty-two patients (54%) had both a low FIB-4 score (<1.3) and normal LSM (<8 kPa), representing true negatives. Fifty-two patients (9%) had a low FIB-4 but elevated LSM (>8 kPa), constituting the false-negative group. Thirty patients with FIB-4 <1.3 underwent biopsy; notably, 15% had advanced fibrosis (stage ≥3). Among the false-negative cohort, 4 patients (7.7%) developed cirrhosis (median time 8 years), 4 (7.7%) experienced cardiovascular events (median 6.5 years), and 1 developed extrahepatic cancer. Hypertension was independently associated with false-negative FIB-4 results in multivariate analysis, regardless of whether LSM or biopsy fibrosis stage was used as the reference standard.

Conclusions: A substantial proportion of MASLD patients with low FIB-4 scores had undetected advanced fibrosis or elevated liver stiffness, highlighting a clinically vulnerable group that would be overlooked under current guideline-recommended pathways. These findings underscore the need to refine referral strategies and validate enhanced risk-stratification approaches in prospective studies.

Keyword(s) : MASLD; FIB-4, Fibrosis

2040 - Analysis of Clinical Characteristics in Patients with Early Stage Primary Biliary Cholangitis Complicated by Portal Hypertension

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Background / Hypothesis:

Analyze the clinical characteristics of patients with primary biliary cholangitis complicated by portal hypertension in the early histological stage, and compare them with those of patients with primary biliary cholangitis complicated by portal hypertension in histological stage IV (cirrhosis stage).

Materials and Methods:

A retrospective study was conducted on patients diagnosed with PBC via liver biopsy at Beijing Ditan

Hospital, Capital Medical University, between April 1, 2020, and December 1, 2023. The study included 21 cases of histologically early-stage (stage I/II) PBC with clinical manifestations of portal hypertension and 14 cases of histologically stage IV PBC with portal hypertension. Clinical characteristics of the early-stage group were analyzed and compared with those of the stage IV group.

Results:

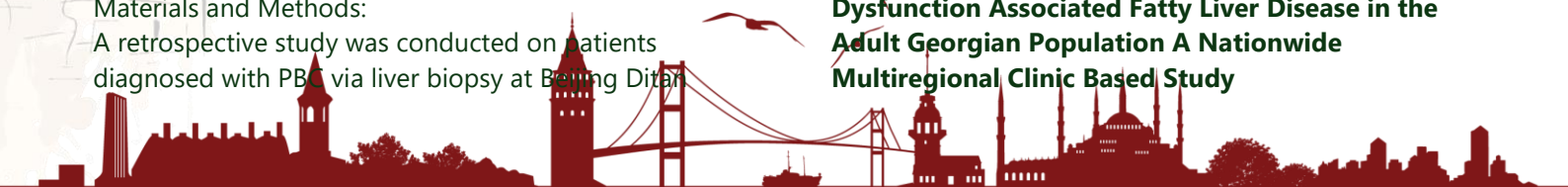
Liver elasticity measurement showed that 81% of patients had an LSM value <20 kPa, mainly concentrated in the range of 10 - 15 kPa (47.6%). In the group of patients with histological stage IV combined with portal hypertension, the disease course was longer (48 (18.5, 120) months vs 24 (10, 60) months), and the proportion of asymptomatic patients was lower (14.3% vs 38.1%), but the differences were not statistically significant. Compared to the early-stage group, stage IV patients had a longer disease duration (48 [18.5, 120] vs. 24 [10, 60] months) and fewer asymptomatic cases (14.3% vs. 38.1%), though these differences were not statistically significant. Stage IV patients were older (59.29 ± 10.33 vs. 49.62 ± 11.19 years, P < 0.05) and had lower white blood cell counts (P < 0.05). ALT, AST, TBIL, GGT, ALP, and TBA were higher in the stage IV group, while ALB, albumin/globulin ratio, and CHE were lower, though only AST and albumin/globulin ratio showed statistically significant differences. Lipid profiles and serum creatinine did not differ significantly between groups. Anti-gp210 antibody positivity was higher in the early-stage group (52.4% vs. 14.3%, P < 0.05). LSM values were significantly higher in the stage IV group (27.65 vs. 12, P < 0.05). Splenomegaly was more pronounced in the early-stage group, while varices and ascites were more common in the stage IV group, though these differences were not statistically significant.

Conclusion:

Diagnosing PBC with portal hypertension at an early histological stage remains challenging, and liver biopsy remains essential. However, age at onset, anti-gp210 positivity, LSM, AST levels, and albumin/globulin ratio may assist in distinguishing early-stage PBC with portal hypertension from cirrhotic PBC.

Keyword(s) : Primary biliary cholangitis; Histological staging; Portal hypertension

2042 - Prevalence and Risk Factors of Metabolic Dysfunction Associated Fatty Liver Disease in the Adult Georgian Population A Nationwide Multiregional Clinic Based Study



E-POSTER ABSTRACTS

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Background / Hypothesis: In Georgia, the prevalence of MAFLD remains poorly defined, as evidence largely stems from small-scale hospital studies. This research used a nationwide multiregional clinic-based sample to estimate MAFLD prevalence and its associated factors.

Materials and Methods: A cross-sectional, clinic-based study was conducted on 11,269 adults attending check-up clinics across Georgia from 2021 to 2025. A stratified random sampling approach considering age, sex, BMI, comorbidities and region was used to derive a representative analytic sample (n = 5,602). MAFLD was diagnosed using ultrasound and international consensus positive diagnostic criteria.

Results: MAFLD affected 51.6% of Georgian adults, with higher prevalence in older age, men and individuals with obesity or metabolic comorbidities. Prevalence varied significantly across regions, with the highest in Tbilisi.

Conclusions: MAFLD is highly prevalent in Georgia. Due to the clinic-based nature of the sample, results should be interpreted with consideration of potential selection bias.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease, MAFLD, prevalence, Georgia, obesity, diabetes, metabolic syndrome.

2043 - Effects of immunosuppressants on HAV replication in human hepatoma cell lines

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Jichi Medical University School Of Medicine, Division Of Virology, Department Of Infection And Immunity, Shimotsuke-shi-Japan³ Niigata University School Of Medicine,, Department Of General Medicine, Niigata-Japan⁴

Background / Hypothesis: Although hepatitis A breakthrough infections after hepatitis A vaccination are rare, breakthrough HAV infections may look more common in immunocompromised people. It is important for patients treated with immunosuppressants to develop anti-HAV. The effects of several immunosuppressants on HAV replication are investigated.

Materials and Methods: We examined the effects of azathioprine, cyclosporin or mycophenolic acid on HAV and HAV replicon replications in human hepatoma cell lines Huh7 and HuhT7, respectively. HAV HA11-1299 genotype IIIA or HAV HM175/18f genotype IB competent replicon and incompetent replicon were used in Huh7 and HuhT7 cells, respectively. HAV RNA levels were measured by real-time RT-PCR. HAV replicon replication was evaluated by reporter assays. HAV internal ribosomal entry site (IRES) activity was measured by reporter assay. Cytotoxicity was evaluated using MTS assay.

Results: (1) In Huh7 cells, 50 ng/mL and 250 ng/mL cyclosporin or 1 µg/mL and 10 µg/mL mycophenolic acid had no inhibitory effects on HAV replication without cytotoxicity. (2) In Huh7 cells, azathioprine had inhibitory effects on HAV replication significantly (0.6-FC (p < 0.05) and 0.4-FC (p < 0.05) at 0.5 µmol/mL and 1.0 µmol/mL, respectively). These concentrations of azathioprine had no cytotoxicity to Huh7 cells for 48 hrs. (3) In HuhT7 cells, 0.5 µmol/mL and 1.0 µmol/mL azathioprine, respectively, also had inhibitory effects on HAV competent replicon (68% (p < 0.01)). However, azathioprine had no effects on incompetent replicon replication. (4) Azathioprine had no inhibitory effects on HAV IRES activity at same concentrations.

Conclusion: Among immunocompromised hosts, severe or prolonged hepatitis A is occasionally observed. Azathioprine could inhibit HAV replication in vitro.

Keyword(s) : HAV; Immunosuppressants; Anti-virals; ALF; ACLF

2044 - Spatiotemporal Changes in the Burden of Viral Hepatitis and the Influence of COVID 19 in China A 20 Year Analysis

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E-POSTER ABSTRACTS

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Background / Hypothesis: Viral hepatitis remains a critical public health challenge in China, characterized by complex spatiotemporal heterogeneity. The COVID-19 pandemic and associated non-pharmaceutical interventions (NPIs) significantly disrupted infectious disease transmission and reporting systems. This study aims to characterize the 20-year spatiotemporal trends of viral hepatitis in China and quantify the impact of the COVID-19 pandemic.

Materials and Methods: Data on the incidence and mortality of viral hepatitis (types A, B, C, and E) from 2004 to 2023 were sourced from the China Information System for Disease Control and Prevention. We employed Joinpoint regression to calculate Average Annual Percent Changes (AAPC). To quantify the pandemic's impact, we constructed a counterfactual inference framework. ARIMA and hybrid ARIMA-LSTM models were trained on pre-pandemic data (2004–2017) and validated using 2018–2019 data. The optimal model for each hepatitis subtype was selected based on Root Mean Square Error (RMSE) minimization to generate "expected" incidence rates for 2020–2023, which were then compared with observed surveillance data to assess relative and absolute rate differences.

Results: From 2004 to 2023, 25,527,158 cases and 17,261 deaths of viral hepatitis were reported. Geographically, the disease burden exhibited a persistent "West-to-East" gradient. High-incidence clusters of Hepatitis B and C were predominantly concentrated in the northwestern provinces of Xinjiang and Qinghai, as well as the southern region. Temporally, Hepatitis A declined significantly (AAPC = -10.5%). Conversely, Hepatitis C showed a concerning upward trend (AAPC = 7.5%), while Hepatitis B maintained a high-level stable epidemic (AAPC = -0.1%). The pandemic impact was heterogeneous. Enteric Hepatitis E experienced a sharp but temporary decline in 2020, with an incidence 32.88% lower than expected. Blood-borne Hepatitis C remained consistently suppressed throughout the pandemic (2020 RRD: -16.07%). Uniquely, Hepatitis B displayed a volatile trajectory: following an initial decline, it exhibited a significant post-pandemic rebound in 2023,

where the observed incidence (74.69 per 100,000) substantially exceeded the counterfactual expectation (70.41 per 100,000).

Conclusions: Although the burden of viral hepatitis has decreased, blood-borne hepatitis remains a severe challenge, particularly in western China. The COVID-19 pandemic exerted a complex inhibitory effect; however, the significant rebound of Hepatitis B in 2023 suggests a compensatory rise in case detection and the rapid recovery of healthcare services. Post-pandemic control strategies must prioritize resource allocation to high-risk western regions to mitigate the rising burden.

Keyword(s) : viral hepatitis; COVID-19; Spatiotemporal analysis

2046 - Clinical Characteristics and Outcomes for Hepatocellular Carcinoma with Extrahepatic Metastases

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a significant health concern in Taiwan, ranking fifth in incidence and second in cancer-related deaths. HCC with extrahepatic metastases (EHM) impacted overall survival and systemic therapies were recommended by several guidelines. The role of aggressive locoregional control for advanced-stage HCC with EHM remains controversial.

Materials and Methods:

An analysis of 4,161 HCC patients admitted to Kaohsiung Veterans General Hospital between 2005 and 2022 were performed and 552 patients with EHM at initial diagnosis were identified. Clinical characteristics and outcomes were analyzed.

Results: EHM was detected in 552 (13.3%) of patients. Metastases to lymph nodes in 266 (48.2%), lungs in 182 (33%), bones in 61 (11%), adrenal glands in 34 (6.2%), and other sites in 8 (1.4%) patients were found.

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Various treatments were administered, including locoregional, systemic, and combined locoregional and systemic treatments and palliative treatment. Multivariate analyses revealed alcohol use, tumor numbers more than 2, tumor size greater than 5 cm, and portal vein invasion as significant risk factors for developing EHM. Patients without EHM had a significant longer median survival than those with EHM (24 Vs. 3 months, $p < 0.001$). In HCC patients with EHM, tumor numbers more than 2, tumor size greater than 5 cm, and portal vein invasion were independent risk factors for survival. In patients with EHM who received systemic treatments had longer survival (6 Vs. 2 months, $P = 0.016$). In EHM patients who received systemic therapy, combined locoregional therapy demonstrated a significant survival benefit (8 Vs. 3 months, $p < 0.001$).

Conclusions:

This study disclosed the clinical features and prognostic factors of HCC with EHM. In EHM patients who received systemic therapy, combined locoregional therapy demonstrated a significant survival benefit

Keyword(s) : HEPATOCELLULAR CARCINOMA, METASTASIS, EXTRAHEPATIC

2048 - INVESTIGATION OF THE RELATIONSHIP BETWEEN SUBCUTANEOUS AND VISCERAL FAT TISSUE MEASUREMENTS AND NON INVASIVE FIBROSIS SCORES (FIB 4 APRI AAR) IN THE EVALUATION OF LIVER DAMAGE ASSOCIATED WITH CELIAC DISEASE

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Background / Hypothesis: Celiac disease (CD) is a chronic autoimmune enteropathy that occurs in genetically predisposed individuals due to gluten consumption. CD can affect multiple extraintestinal systems, including the liver. Although elevated liver enzymes are common in CD, the diagnostic value of non-invasive fibrosis scores (FIB-4, APRI, AAR) in assessing liver damage in these patients is unclear. In this study, we aimed to more comprehensively evaluate CD-related liver involvement by examining the relationship between adipose tissue distribution (visceral and subcutaneous fat) and non-invasive fibrosis markers.

Materials and Methods: A retrospective analysis was conducted on 103 celiac patients and 51 healthy controls followed at Firat University Hospital. Celiac diagnosis was confirmed by serological testing and duodenal biopsy. Biochemical parameters, the presence of hepatosteatosis using abdominal CT and ultrasonography, and visceral and subcutaneous fat tissue measurements were examined in all subjects. Additionally, FIB-4, APRI, and AAR scores were calculated using standard formulas and their relationship to liver damage was analyzed. ROC analyses were performed to determine the severity of hepatosteatosis.

Results: AST and ALT levels were significantly higher in the CD group compared to the control group ($p < 0.001$). Glucose, triglyceride, LDL, HDL, vitamin D, and HbA1c values were significantly lower in the CD group ($p < 0.001-0.009$). FIB-4, APRI, and AAR showed inadequate diagnostic performance in determining the severity of hepatosteatosis in both groups (AUC not significant; $p > 0.05$). No significant correlation was found between VAD/SAD measurements and non-invasive fibrosis scores.

Conclusions: Our study demonstrates that elevated liver enzymes are frequently observed in celiac patients, independent of hepatosteatosis, and may be associated with immune-mediated celiac hepatitis. Low glucose, lipid, and vitamin D levels, along with decreased subcutaneous fat, were found to be consistent with active malabsorption. However, FIB-4, APRI, and AAR scores were insufficient to predict the severity of hepatosteatosis. These findings emphasize that non-invasive scores alone are not sufficient to assess liver involvement in celiac patients

Keyword(s) : Celiac disease, Hepatosteatosis, Visceral adipose tissue, Non-invasive fibrosis scores

2049 - Zinc acetate enhances glucose regulated protein 78 and anti HAV activity of ribavirin in vitro

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Background / Hypothesis: Hepatitis A virus (HAV) infection occasionally leads to acute liver failure or acute-on-chronic liver failure, which is often fatal, especially in patients with diabetes mellitus or elderly individuals. However, effective drugs for hepatitis A are currently unavailable. Glucose-regulated protein 78 (GRP78/BiP/HSPA5) is an antiviral agent that has been reported to prevent HAV replication. Zinc sulfate inhibits HAV replicon replication in the accompany with the enhancement of GRP78 expression. Although ribavirin has anti-HAV activity, its use is limited due to multiple adverse events. In this study, we investigated the safety and efficacy of zinc acetate for hepatitis A in vitro.

Materials and Methods:

Effects of zinc acetate on HAV HA11–1299 genotype IIIA or HAV HM175/18f genotype IB replication and changes in GRP78 expression levels in human hepatocytes with or without HAV infection were examined by real-time RT-PCR and ELISA, respectively. The cytotoxicity of zinc acetate and ribavirin was evaluated by MTS assay. We also examined the enhancement of zinc acetate on the effects of anti-HAV activity of ribavirin.

Results: Zinc acetate inhibited HAV replication in both Huh7 and GL37 cells. Inhibition of HAV replication by zinc acetate was similar those of zinc sulfate or zinc chloride. Zinc acetate also inhibited HAV replication in both low- and high-glucose media. The IC₅₀ value of zinc acetate was 13.6 μ M for the suppression of HAV RNA. We did not observe any acute hepato-cellular injuries by zinc acetate. Zinc acetate increased the expression of GRP78 in response to HAV infection. The combination of zinc acetate with ribavirin led to the suppression of both HAV HA11-1299 genotype IIIA and HAV HM175/18f genotype IB replication in Huh7 cells than that of ribavirin alone.

Conclusions: Zinc acetate inhibits HAV replication in accompany with the elevation of GRP78 expression without causing cellular toxicity. We also demonstrated that zinc acetate enhances the anti-HAV activities of ribavirin. Thus, combination therapies could help to decrease the ribavirin dose and its adverse events in patients infected with HAV.

Keyword(s) : HAV; Zinc; GRP78; Ribavirin

2050 - Long term Outcomes of Tenofovir Therapy in Vietnamese Chronic Hepatitis B Patients A 5 Year Retrospective Cohort Study

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Background / Hypothesis:

Chronic hepatitis B remains a major cause of cirrhosis and hepatocellular carcinoma in Vietnam. Long-term real-world data on Tenofovir disoproxil fumarate (TDF) in Vietnamese patients are still limited. We aimed to evaluate 5-year virological, biochemical, and fibrosis outcomes in chronic HBV patients treated with TDF in routine clinical practice.

Materials and Methods:

We conducted a retrospective cohort study including 145 chronic HBV patients treated with Tenofovir at the Department of Infectious Diseases, UMPSM. Clinical data, HBV DNA, ALT, qHBsAg, APRI, and ARFI measurements were collected at baseline and at multiple time points up to 60 months. Virological response was classified as complete, partial, or non-response based on HBV DNA decline patterns.

Results:

At baseline, 62,8% were male and median age was 46 years. Virological suppression was rapid: at month 12, 92,4% achieved complete response and at month 24, 100% had undetectable HBV DNA. ALT normalization reached 86–90% after 24 months and remained stable thereafter. Fibrosis indices improved significantly: median APRI decreased from 0,58 to 0,30 at month 24, and ARFI from 1,42 m/s to 1,28 m/s, indicating regression from F2 to F1–F0. qHBsAg declined slowly ($\approx 0,40 \log_{10}$ after 60 months). Clinical outcomes were favorable: only 1 case (0,7%) developed HCC and 2 cases (1,4%) had new-onset decompensation during 5-year follow-up.

Conclusions:

Long-term Tenofovir therapy resulted in excellent virological suppression, high ALT normalization rates,



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and meaningful improvement in non-invasive fibrosis markers. Real-world outcomes in Vietnamese patients are comparable to international data, supporting early initiation and long-term adherence to TDF therapy.

Keyword(s) : Hepatitis B; Tenofovir; Fibrosis; Virological response; Real-world cohort

2051 - PILOT STUDY ON LONG TERM EFFECTIVENESS OF THE KARLOVY VARY THERMAL SPRING DRINKING CURE FOR MASLD

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Objectives

The aim of this pilot study was to provide scientifically substantiated evidence on the long-term effects of the Karlovy Vary mineral spring drinking cure on liver steatosis in individuals with metabolic dysfunction-associated steatotic liver disease (MASLD). The primary outcomes were changes in hepatic steatosis measured by the controlled attenuation parameter (CAP) and steatosis grade after a 6-month period. Liver stiffness measurements (LSM) and selected biochemical markers were also assessed.

Methods

The drinking treatment lasted 21 days, mirroring the duration of the traditional comprehensive spa treatment, and involved thrice-daily intake of prescribed volumes (200–300 ml) of specific mineral springs. The study included 50 patients.

Laboratory parameters were assessed at baseline and post-treatment (3 weeks) in all participants. FibroScan CAP and LSM were performed at baseline, post-treatment, and at 6-month follow-up; complete longitudinal data were available for 37 patients.

Statistical analysis included paired t-tests or Wilcoxon signed-rank tests depending on normality, and the Stuart-Maxwell test for categorical variables. All analyses were performed in R version 4.4.2.

Results

A statistically significant reduction in CAP was observed both immediately after the drinking cure (mean change -25.3 dB/m; $p < 0.001$) and at the 6-month follow-up (-17.2 dB/m; $p = 0.002$). Steatosis grade improved significantly post-treatment ($p = 0.001$) and remained

improved at follow-up ($p = 0.007$). No statistically significant changes were observed in LSM or fibrosis stage. Biochemical parameters showed minimal change; the limited effect on laboratory values was likely due to most parameters being within the normal ranges at baseline.

Conclusion

This study suggests that the Karlovy Vary mineral spring drinking cure has the potential to reduce hepatic steatosis in individuals with MASLD, with effects persisting at 6 months. While fibrosis parameters remained unchanged, the improvement in steatosis grade and CAP supports the potential of mineral water therapy as a non-pharmacological intervention. Further controlled studies are needed to confirm these findings and to better understand the mechanisms underlying the observed effects.

This work was co-developed within the project Spa Research Center – CZ.10.01.01/00/22_001/0000261 supported by the European Just Transition Fund.

Keyword(s) : Balneology, Spa, Mineral Springs, MASLD, FibroScan

2052 - A Young Patient Presenting With Hepatocellular Carcinoma and Portal Vein Thrombosis Developing on A Background of Non Cirrhotic Hepatitis B A Case Report

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is a primary malignant liver tumor that develops in the setting of chronic liver disease, particularly in patients with cirrhosis or chronic hepatitis B virus (HBV) infection. Approximately 75% of primary liver tumors are HCC.

More than 50% of the 770,000 HCC cases seen worldwide are associated with chronic HBV. Patients with chronic HBV infection are at risk of developing HCC even in the absence of cirrhosis.

Here, we present a young patient with HCC that developed on a non-cirrhotic background and presented with portal vein thrombosis.

Case: A 23-year-old male patient presented to the outpatient clinic with complaints of fatigue, tiredness, and epigastric pain. Physical examination revealed a 3 cm hepatomegaly and a 2 cm splenomegaly. Laboratory findings were as follows: platelets $162,000/\text{mm}^3$, AST 160 IU, ALT 35 IU, total bilirubin 0.7

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mg/dl, direct bilirubin 0.2 mg/dl, albumin 3.9 g/dl, prothrombin time 14.5 seconds, HBsAg (+), HBeAg (-), Anti-HCV (-), and AFP >1000 ng/ml. Abdominal Doppler ultrasonography (USG) showed a liver of normal size with a heterogeneous parenchymal appearance, and the spleen measured 15 cm. The main portal vein and its branches were observed to be thrombosed, with no detectable flow. Upper abdominal contrast-enhanced magnetic resonance imaging (MRI) revealed a lesion consistent with HCC, measuring 17 × 13 × 15 cm at its widest point, filling the right lobe of the liver, demonstrating arterial phase enhancement, venous phase washout, and diffusion restriction. The portal vein and its branches appeared occluded due to tumor thrombus.

The patient was diagnosed with diffuse HCC and portal vein thrombosis developing on the background of chronic Hepatitis B. As the patient was not considered suitable for liver transplantation, follow-up with medical oncology and outpatient clinic visits was recommended.

Conclusions: Hepatitis B is an important public health issue in our country. Every patient with HBV, even if young, should be screened for HCC.

Keyword(s) : HBV, HCC, portal vein thrombosis

2054 - Association of SGLT2 Inhibitors with Reduced Hepatocellular Carcinoma Risk in Patients with Type 2 Diabetes

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Background / Hypothesis: Sodium-glucose cotransporter-2 inhibitors (SGLT2is) are widely prescribed for type 2 diabetes mellitus (T2DM) and may confer hepatoprotective effects. This study investigated their association with hepatocellular carcinoma (HCC) risk compared with other antidiabetic medications.

Materials and Methods: We conducted a retrospective cohort study using the electronic health records from

Mass General Brigham (Boston, USA) and Asan Medical Center (Seoul, Korea) from 2013 to 2024. Adults with T2DM newly initiating SGLT2i, dipeptidyl peptidase-4 inhibitor (DPP4i), glucagon-like peptide-1 receptor agonist (GLP-1RA), or sulfonylureas were included. A 6-month landmark analysis was employed to minimize early event bias. Inverse probability weighting combined with Fine-Gray competing risk models estimated subdistribution hazard ratios (SHRs) for incident HCC, accounting for transplant and death as competing events.

Results: After weighting, data from 6,733 SGLT2i, 4,495 GLP-1RA, 23,229 DPP4i, and 17,034 sulfonylurea initiators were analyzed. Over a median follow-up of 3.9 years (277,155 person-years), 623 HCC cases occurred. SGLT2i use was associated with significantly lower HCC risk versus sulfonylureas (SHR 0.44, 95% CI: 0.25–0.79) and DPP4i (SHR 0.53, 95% CI: 0.30–0.93). The comparison with GLP-1RA showed comparable risk (SHR 0.87, 95% CI: 0.40–1.91). Subgroup analyses demonstrated consistent protective associations of SGLT2i in patients ≤65 years, males, and those with chronic liver disease. Sensitivity analyses, including 12-month landmark analysis and adjustments for additional confounders, confirmed robustness of findings.

Conclusions: SGLT2 inhibitor therapy was associated with reduced risk of HCC compared with DPP4 inhibitors and sulfonylureas, supporting their potential chemopreventive role in patients with T2DM.

Keyword(s) :

2055 - Cost Effectiveness Evaluation of Elmetenovir in the Treatment of Chronic Hepatitis B

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Background / HypoCost-Effectiveness Evaluation of Elmetenovir in the Treatment of Chronic Hepatitis Bthesis:

Materials and Methods:

Results:



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Conclusions:

Keyword(s) :

2057 - Application Value of HBV RNA Quantitative Detection in Antiviral Treatment Monitoring for Chronic Hepatitis B Patients

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Background To explore the application value of HBV RNA quantitative detection in monitoring antiviral treatment for patients with chronic hepatitis B (CHB)./
Hypothesis:

Materials and Methods:

Results:

Conclusions:

Keyword(s) : Application Value of HBV RNA Quantitative Detection in Antiviral Treatment Monitoring for Chronic Hepatitis B Patients

2058 - Clinical Application Value of HBV RNA Detection in HBV DNA Negative Chronic Hepatitis B Patients

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Background / Hypothesis:
Clinical Application Value of HBV RNA Detection in HBV DNA-Negative Chronic Hepatitis B Patients

Materials and Methods:

Results:

Conclusions:

Keyword(s) : Clinical Application Value of HBV RNA Detection in HBV DNA-Negative Chronic Hepatitis B Patients

2059 - Application Value of HBV RNA Detection in Assessing the Risk of Relapse After Discontinuation of Antiviral Treatment in Patients with Chronic Hepatitis B

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Background / Hypothesis:

Application Value of HBV RNA Detection in Assessing the Risk of Relapse After Discontinuation of Antiviral Treatment in Patients with Chronic Hepatitis B

Materials and Methods:

Results:

Conclusions:

Keyword(s) : Application Value of HBV RNA Detection in Assessing the Risk of Relapse After Discontinuation of Antiviral Treatment in Patients with Chronic Hepatitis B

2060 - Efficacy and Safety of Tenofovir Amibufenamide in the Treatment of Elderly Patients with Chronic Hepatitis B and Liver Cirrhosis

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Background / Hypothesis:
Efficacy and Safety of Tenofovir Amibufenamide in the Treatment of Elderly Patients with Chronic Hepatitis B and Liver Cirrhosis

Materials and Methods:



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Results:

Conclusions:

Keyword(s) : Efficacy and Safety of Tenofovir Amibufenamide in the Treatment of Elderly Patients with Chronic Hepatitis B and Liver Cirrhosis

2061 - Real World Study of Tenofovir Amibufenamide in the Treatment of Hepatitis B Related Liver Cirrhosis

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Background / Hypothesis:

Real-World Study of Tenofovir Amibufenamide in the Treatment of Hepatitis B-Related Liver Cirrhosis

Materials and Methods:

Results:

Conclusions:

Keyword(s) : Real-World Study of Tenofovir Amibufenamide in the Treatment of Hepatitis B-Related Liver Cirrhosis

2062 - Clinical Significance of Prognostic Nutritional Index for Prognostication of Decompensated Liver Cirrhosis A Retrospective Cohort Study

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Background/Hypothesis Liver cirrhosis represents a significant global health burden, with decompensation markedly increasing mortality risk. While the Model for End-Stage Liver Disease (MELD) variants are the standard for prognostication, their reliance on multiple

laboratory parameters can limit accessibility in resource-constrained settings. The Prognostic Nutritional Index (PNI), derived solely from serum albumin and lymphocyte count, offers a simple marker of immunonutritional status. This study hypothesized that PNI could serve as a viable prognostic tool for decompensated liver cirrhosis (DLC). The objective was to evaluate the predictive accuracy of PNI for short- and long-term mortality in comparison to MELD-Na and MELD 3.0.

Materials and Methods We conducted a single-center, retrospective cohort study utilizing the Liver Banks Data Registry at St. Luke's Medical Center, Quezon City. The study included 260 adult patients diagnosed with DLC excluding patients with non-HCC malignancies, autoimmune diseases, or prior transplants. PNI, MELD-Na, and MELD 3.0 scores were calculated at admission. The primary outcome was all-cause mortality at 3, 6, and 12 months. Diagnostic accuracy was assessed using AUC-ROC curves, and optimal cut-offs were determined using the Youden Index. Survival curves were generated using the Kaplan-Meier method.

Results The cohort had a median age of 66 years and was predominantly male (64.6%), with Hepatitis B (40.8%) and NASH/MAFLD (31.5%) as the leading etiologies. Non-survivors presented with significantly lower serum albumin (2.87 vs 3.38 g/dL, $p < 0.001$) and lymphocyte percentage (13.0% vs 19.2%, $p < 0.001$) compared to survivors. An optimal PNI cut-off of 32.67 was identified. Patients with a High PNI (>32.67) demonstrated significantly better overall survival compared to the Low PNI group ($p < 0.001$). However, PNI displayed limited discriminative ability with an AUC of ~ 0.64 across all time points. In contrast, MELD-Na and MELD 3.0 demonstrated superior accuracy, achieving AUCs ranging from 0.72 — 0.74 and 0.74 0.76, respectively. PNI sensitivity was notably low (~ 55 58%), likely due to its inability to capture renal dysfunction.

Conclusions While the Prognostic Nutritional Index is an independent predictor of mortality in decompensated liver cirrhosis, identifying a high-risk cohort below a cut-off of 32.67, its performance as a standalone prognostic tool is inferior to MELD-Na and MELD 3.0. The exclusion of renal markers in PNI limits its sensitivity in predicting short-term mortality driven by hepatorenal physiology. Nevertheless, PNI remains a valuable, cost-effective adjunctive tool for identifying nutritional frailty, particularly in resource-limited settings where comprehensive scoring is unavailable.

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Keyword(s) : Decompensated Liver Cirrhosis, Prognostic Nutritional Index, MELD-Na, MELD 3.0, Mortality, Risk Stratification, Malnutrition

2063 - Research Trends and Hotspots in Autoimmune Hepatitis A Bibliometric Analysis

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Background / Hypothesis:

Autoimmune hepatitis (AIH) is a chronic, relapsing inflammatory disease of the liver that is characterized by hepatocellular injury, which can progress to cirrhosis and liver failure. The diagnostic and treatment regimen for AIH is based on steroid induction therapy. However, long-term use of corticosteroids can cause significant side effects in patients. Meanwhile, a comprehensive bibliometric analysis of the field of AIH has not been published. The purpose of this paper is to describe the current status and trends of AIH research worldwide using data from the Web of Science (WOS) core database.

Materials and Methods:

The analysis was conducted using data from WOS's core database, to examine articles related to the field of AIH that were published from 2014 to 2024. In addition, the CiteSpace and VOSviewer software were utilized to conduct a comprehensive analysis of publications based on various criteria, including issue, author, journal, country, institution, keyword, co-citation, and reference.

Results:

A total of 2,426 articles were included in this study. The United States has the most publications in this field, followed by China. The University Medical Center Hamburg-Eppendorf (Germany) is the leading institution in AIH, with Lohse from Germany having made significant achievements in this area. We considered the Journal of Hepatology to be a high-impact journal in this field. The keywords were then subjected to a cluster analysis, which resulted in the identification of four primary clusters: (1) primary sclerosing cholangitis, (2) concanavalin A, (3) hepatitis C virus, and (4) drug-induced liver injury.

Conclusions:

This study systematically summarized research findings in the field of AIH from 2014 to 2024. We identified AIH-related research centered on disease diagnosis, treatment, management, efficacy assessment, and the

enterohepatic axis. We also identified four core research directions in the field of AIH and the research trends they reflect (primary sclerosing cholangitis, concanavalin A, multiple sclerosis, and drug-induced liver injury).

Keyword(s) : Autoimmune Hepatitis; Bibliometrics analysis; Research trends and hotspots; Treatment

2064 - Early Antibiotic Exposure Shortens Progression Free Survival in Unresectable Intermediate Advanced HCC Treated With ICI Plus TKI and TACE A Propensity Score Matched Study

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Background / Hypothesis:

To evaluate whether early antibiotic (ATB) exposure adversely affects long-term outcomes in patients with unresectable, intermediate-to-advanced hepatocellular carcinoma (HCC) receiving immune-checkpoint inhibitors (ICIs) plus tyrosine-kinase inhibitors (TKIs) and trans-arterial chemo-embolization (TACE).

Materials and Methods:

In this single-centre, retrospective cohort study we consecutively enrolled 224 BCLC stage B/C HCC patients (Child-Pugh A/B) who received at least 3 cycles of ICI+TKI+TACE at the Affiliated Hospital of Xuzhou Medical University between January 2020 and December 2024. (Retrospective analysis approved by the Institutional Review Board of the Affiliated Hospital of Xuzhou Medical University; requirement for informed consent was waived.) Patients who received ATB within 30 days before or after the first ICI dose (n = 25) were compared with ATB-free controls. After 1:1 propensity-score matching (PSM), 20 pairs were analysed. Primary end-points were progression-free survival (PFS) and overall survival (OS); adverse events (AEs) were secondary. Survival distributions were estimated with Kaplan-Meier methods and compared by log-rank test; independent prognostic factors were identified using Cox regression.

Results:

Baseline characteristics were well balanced after PSM. Median OS was 18.2 vs 24.0 months (P = 0.692, not significant). Median PFS was significantly shorter in the ATB group (7.6 vs 12.3 months; HR = 2.76, 95% CI 1.31-5.83, P = 0.006). Multivariate analysis identified ATB exposure, Child-Pugh B status and HBV DNA ≥

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2,000 IU/mL as independent predictors of poorer PFS. Subgroup analyses revealed more pronounced PFS loss in patients aged >60 years, Child-Pugh B, BCLC stage C, HBV DNA 20–2,000 IU/mL or AFP $\geq$ 400 ng/mL. Overall and grade 3–4 AE rates did not differ between groups.

Conclusions:

Early ATB exposure did not affect OS but independently shortened PFS in unresectable, intermediate-to-advanced HCC treated with ICI+TKI+TACE. Routine prophylactic antibiotics should be used cautiously, especially in patients with limited hepatic reserve, high tumour burden or intermediate HBV replication, to avoid compromising oncological outcomes.

Keyword(s) : hepatocellular carcinoma, immune-checkpoint inhibitors, TACE, PFS, antibiotic

2069 - Effects of Angelicae Sinensis Radix Combined with Paeoniae Radix Alba on Enterogenous Endotoxin and Hepatic Toll like Receptor 4 Signaling Pathway in Mice with High Fat Diet induced Metabolic Dysfunction Associated Steatohepatitis (MASH)

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Background / Hypothesis:

MASH represents a major global health burden due to its progressive nature, which can lead to advanced fibrosis, cirrhosis, HCC. The intricate and incompletely elucidated pathogenesis of MASH has posed substantial challenges for therapeutic development, with Resmetirom remaining the sole approved pharmacotherapy to date. In light of the multi-target potential inherent to traditional Chinese medicine for complex multifactorial diseases, combined with evidence from our prior investigations, this study was designed to further elucidate the potential mechanism by which the herb pairs, that is Angelicae Sinensis Radix combined with Paeoniae Radix Alba, improve liver injury and promote hepatocytes regeneration in

MASH through regulating enterogenous endotoxin (LPS) and toll-like receptor 4 (TLR4) signaling pathway.

Materials and Methods:

After decoction, filtration, and concentration using the traditional method, an aqueous extract of Angelicae Sinensis Radix (9g) and Paeoniae Radix Alba (18g) was prepared. Male C57BL/6J mice were randomly assigned to either a normal chow-fed control group (NG) or underwent induction of the MASH model using a high-fat diet. Upon successful model establishment, mice were further divided into the following groups (n=15 per group): model group (MG), and low-, mid-, and high-dose herb pairs intervention groups (LIG, MIG, and HIG, respectively). Corresponding treatments were administered for four consecutive weeks. Portal vein plasma was collected for LPS quantification. Primary hepatocytes were isolated from mice to analyze the mRNA expression levels of key components in the TLR4 signaling pathway. Additionally, the secretions of tumor necrosis factor-alpha (TNF- α) and interleukin-6 (IL-6) from LPS-stimulated primary hepatocytes were measured.

Results:

LPS levels were elevated in the MG compared to the NG ($p < 0.005$). All group of herb pairs treatment dose-dependently reduced LPS levels compared with the MG (all $p < 0.005$). mRNA expressions of TLR4 (~5.11-fold), myeloid differentiation primary response 88 (~6.19-fold), tumor necrosis factor receptor-associated factor 6 (~4.68-fold), and nuclear factor kappa-B (~4.11-fold) were upregulated in the MG relative to the NG (all $p < 0.005$). The herb pairs intervention downregulated these transcripts compared to the MG (all $p < 0.005$), exhibiting a clear dose-dependent suppression across the treatment groups ($p < 0.005$). In vitro, LPS stimulation enhanced IL-6 and TNF- α secretion from primary hepatocytes in the MG higher than the NG ($p < 0.005$). The herb pairs potently inhibited LPS-induced cytokine release in a dose-dependent manner (all $p < 0.005$).

Conclusion:

The therapeutic mechanism of Angelicae Sinensis Radix combined with Paeoniae Radix Alba in MASH involves a dose-dependent reduction in enterogenous endotoxin level and the downregulation of hepatic TLR4 signaling pathway expression and activity.

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Keyword(s) : Angelicae Sinensis Radix, Paeoniae Radix Alba, Herb pairs, MASH, Enterogenous endotoxin, TLR4, Signaling pathway

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2070 - THE USE OF A MODIFIED GREAT SAPHENOUS VEIN FOR HEPATIC VEIN RECONSTRUCTION IN RIGHT LOBE LIVING DONOR LIVER TRANSPLANTATION

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Background / Hypothesis: Reconstruction of hepatic veins is the most technically challenging stage of living donor liver transplantation (LDLT). Impaired venous outflow from the graft is one of the main causes of postoperative liver failure. During right lobe liver transplantation, it is crucial to consider reconstruction of significantly large veins of segments 5 and 8 (greater than 5 mm in diameter) in order to prevent congestion in the anterior sector of the graft. To evaluate the outcomes of complex reconstruction of the hepatic veins during transplantation of the right liver lobe at our clinic.

Materials and Methods: From December 2011 to November 2025, 347 LT in adults and children were conducted in our center. The following types of grafts were used: RL – 240 (69.3%), LL – 31 (9%), posterior lateral sector – 1 (0.3%), dual graft – 1 (0.3%), LLS – 41 (11.8%), whole liver – 32 (9%), and split transplantation – 1 (0.3%). Reconstruction of hepatic veins of segment 5 and 8 during transplantation of the RL of the liver was performed in 112 (32.4%) cases. Of these, reconstruction of hepatic veins of the 5 and 8 segments of the liver using the recipient's portal vein in 80 (71.4%) cases, vascular prosthesis in 11 (9.8%) cases, with a modified great saphenous vein (GSV) in 15 (13.4%) cases and vessels of a deceased donor in 6 (5.4%) cases. To assess the functioning of the reconstructed veins, patients underwent a control CT scan after 7 days, 14 days, 1 month, 3 months.

Results: The patency of the neo-MHV using the recipient's portal vein was 90% at 1 week and 86.7% at 2 weeks. In the group with reconstruction using a vascular prosthesis, patency was 81.9% at 1 week and 72.8% at 2 weeks. In the group with reconstruction using a modified great saphenous vein (GSV), patency was 100% at 1 week and 93% at 2 weeks. In the group

with reconstruction using vessels from deceased donors, patency was 66.7% at 1 week and 66.7% at 2 weeks. No hepatic vein-related complications were observed in patients who underwent reconstruction of segments 5 and 8 hepatic veins.

Conclusions: Reconstruction of the hepatic veins of segments 5 and 8 using a modified great saphenous vein provides good outcomes and also reduces the duration of warm and cold ischemia as well as the anhepatic phase, which subsequently has a positive effect on postoperative recovery.

Keyword(s) : a modified great saphenous vein, LDLT, Reconstruction of hepatic veins

2071 - PORTAL FLOW MODULATION IN LIVING DONOR LIVER TRANSPLANTATION

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Background / Hypothesis:

Living donor liver transplantation (LDLT) is one of the primary strategies to address the growing imbalance between the demand for liver transplantation and the limited availability of deceased donor organs. Moreover, in many countries of the Middle East and Asia, access to deceased donor liver transplantation is restricted due to cultural and religious factors, making LDLT the only realistic option for patients with end-stage liver disease. Small-for-size syndrome (SFSS) is a widely recognized complication after liver transplantation, occurring in up to 20% of LDLT procedures. Portal inflow modulation aimed at maintaining a portal vein pressure below 15 mmHg or a portal vein flow rate of less than 250 mL/min/100 g of graft weight has been shown to improve clinical outcomes.

Materials and Methods:

From December 2011 to November 2025, a total of 347 liver transplantations in adults and children were performed in our center. The following graft types

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were used: right lobe (RL) — 240 (69.2%), left lobe (LL) — 31 (8.9%), posterior lateral sector — 1 (0.3%), dual graft — 1 (0.3%), left lateral section (LLS) — 41 (11.8%), whole liver — 32 (9.2%), and split transplantation — 1 (0.3%). Portal inflow modulation during LDLT was performed in 34 cases (9.8%). Among these patients, intraoperative splenectomy was performed in 9 cases (26.5%), postoperative splenectomy in 4 cases (11.8%), intraoperative splenic artery ligation — in 19 cases (55.9%), and postoperative splenic artery embolization — in 2 cases (5.8%). In patients requiring inflow modulation, the graft-to-recipient weight ratio (GRWR) ranged from 0.5 to 1.1.

Results:

The mean operative time with portal inflow modulation was 615 minutes. Mean intraoperative blood loss was 3000 mL. Warm ischemia time was 27.8 minutes, and cold ischemia time was 33.1 minutes. Postoperative complications were observed in 7 patients (28%). Biliary complications occurred in 3 cases (42.8%), pulmonary edema — in 1 case (14.3%), steal syndrome — in 1 case (14.3%), portal anastomotic thrombosis — in 1 case (14.3%), and portal vein thrombosis — in 1 case (14.3%), which required reoperation. No hospital mortality was reported.

Conclusions:

Excessive portal inflow is a key determinant of SFSS. Intraoperative inflow modulation allows achieving optimal hemodynamics and enhances graft function and patient outcomes in LDLT. This personalized approach should be considered a standard preventive measure to reduce the risk of SFSS and improve long-term survival.

Keyword(s) : Living Donor Liver Transplantation (LDLT), Portal flow modulation, small for size syndrome

2072 - Role of TRIM37 IKK γ axis in hepatic ischemia reperfusion injury

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Background / Hypothesis:

Hepatic ischemia/reperfusion (I/R) injury is one of the major pathological processes associated with various liver surgeries. However, there is still a lack of strategies to protect against hepatic I/R injury because of the unknown underlying mechanism. The present study aimed to identify a potential strategy and

provide a fundamental experimental basis for treating hepatic I/R injury.

Materials and Methods:

A classic 70% ischemia/reperfusion injury was established. Immunoprecipitation was used to identify direct interactions between proteins. The expression of proteins from different subcellular localizations was detected by Western blotting. Cell translocation was directly observed by immunofluorescence. HE, TUNEL and ELISA were performed for function tests.

Results:

We report that tripartite motif containing 37 (TRIM37) aggravates hepatic I/R injury through the reinforcement of IKK-induced inflammation following dual patterns. Mechanistically, TRIM37 directly interacts with tumor necrosis factor receptor-associated factor 6 (TRAF6), inducing K63 ubiquitination and eventually leading to the phosphorylation of IKK β . TRIM37 enhances the translocation of IKK γ , a regulatory subunit of the IKK complex, from the nucleus to the cytoplasm, thereby stabilizing the cytoplasmic IKK complex and prolonging the duration of inflammation. Inhibition of IKK rescued the function of TRIM37 in vivo and in vitro.

Conclusions:

Collectively, the present study discloses some potential function of TRIM37 in hepatic I/R injury. Targeting TRIM37 might be potential for treatment against hepatic I/R injury. Targeting TRIM37 might be a potential treatment strategy against hepatic I/R injury.

Keyword(s) : liver transplantation, liver ischemia reperfusion, interactions between proteins

2073 - FAM49B inhibits TRAF6 ubiquitination and alleviates liver ischemia reperfusion injury

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Background / Hypothesis:

Hepatic ischemia-reperfusion occurs following liver transplantation and various liver surgeries. The ubiquitin chain inflammatory response mediated by TRAF6 is a crucial molecular step. How to target TRAF6 in order to inhibit the phosphorylation of the IKK inflammatory complex has been our long-term focus of research.

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Materials and Methods:

We established a classic mouse liver IRI model and used a time point of 1/6 hour. Immunoprecipitation was used to identify direct interactions between proteins. The expression of proteins from different subcellular localizations was detected by Western blotting. Cell translocation was directly observed by immunofluorescence. HE, TUNEL and ELISA were performed for function tests.

Results:

FAM49B was significantly downregulated in the liver IRI model. Overexpression of FAM49B could significantly reduce the phosphorylation of nuclear factor kappa-B kinase inhibitors (IKK) and downstream pro-inflammatory proteins. Mechanistically, FAM49B directly interacts with TRAF6, resulting in a decrease in the ubiquitination level of TRAF6. The use of IKK activators can significantly reverse the function of FAM49B.

Conclusions:

We initially evaluated the function of FAM49B in liver ischemia-reperfusion injury (IRI) and demonstrated that its interaction with TRAF6 has a crucial impact on the IKK-mediated inflammatory cascade reaction.

Keyword(s) : liver transplantation, liver ischemia reperfusion, TRAF

2074 - Prediction of Non Alcoholic Fatty Liver Disease by Machine Learning Algorithms

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Background / Hypothesis: Closely associated with metabolic disorders, non-alcoholic fatty liver disease (NAFLD) has been a major disorder of liver disease and substantially increases the risk of hepatocellular carcinoma. The integration of artificial intelligence (AI) into medical research has facilitated the extensive application of machine learning (ML) in predicting liver disease risk and treatment outcomes. Under the hypothesis that ML might be significantly useful in clinical application of prediction of NAFLD in Taiwanese patients, the present study aimed to apply

ML algorithms to a community-based cohort in southern Taiwan to identify key risk factors for NAFLD.

Materials and Methods: We have collected data of 7,700 participants (39.2% male; mean age 50.9 ± 14.9 years) derived from community health examinations, and clinical and demographic features in southern Taiwan. NAFLD is diagnosed through imaging or histological confirmation of significant hepatic steatosis. The five ML algorithms were evaluated, including logistic regression (LR), random forest (RF), K-nearest neighbors (KNN), adaptive boosting (AdaBoost), and extreme gradient boosting (XGBoost) for analyzing the data. Model performance was assessed using accuracy, precision, recall, F1 score, and the area under the receiver operating characteristic curve (AUROC).

Results: The 7,700 cases were randomly partitioned into a training dataset (6,160) for model development and a testing dataset (1,540) for internal validation, with no significant differences observed between groups. Feature importance analysis identified LDL-C, body mass index (BMI), triglycerides (TG), age, and waist circumference as the most influential predictors of NAFLD. The performance metrics for LR, RF, KNN, AdaBoost, and XGBoost on the testing dataset for accuracy, precision, recall, F1 score, and AUROC were evaluated. Among all models, XGBoost achieved the highest performance, with an accuracy of 91.7%, precision of 91.8%, recall of 91.5, F1 score of 91.7%, and AUROC of 96.9%.

Conclusions: ML algorithms, particularly XGBoost, demonstrated high accuracy in predicting NAFLD and effectively identified key clinical predictors.

Keyword(s) : Prediction, Non-Alcoholic Fatty Liver Disease, Machine Learning

2075 - Efficacy and safety of transarterial chemoembolization plus dual immune and anti angiogenic blockers for unresectable hepatocellular carcinoma a systematic review and meta analysis

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Background / Hypothesis:

Transarterial chemoembolization (TACE) remains a standard treatment for intermediate-stage hepatocellular carcinoma (HCC), but its efficacy is limited by post-procedural angiogenesis and immune escape. Combining TACE with dual systemic therapy—

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immune checkpoint inhibition and anti-angiogenic blockade—offers complementary mechanisms that may enhance tumor control. This systematic review and meta-analysis evaluated the efficacy and safety of this combination compared with TACE alone.

Materials and Methods:

A comprehensive search of PubMed, Scopus, Web of Science, and Google Scholar was performed through October 2025. Eligible studies compared TACE combined with immune checkpoint inhibitors plus anti-angiogenic agents versus TACE monotherapy in unresectable or intermediate-stage HCC. Pooled hazard ratios (HRs) for progression-free survival (PFS) and overall survival (OS), and odds ratios (ORs) for grade 3–5 adverse events (AEs) were calculated using fixed-effects models in the absence of heterogeneity or random-effects models otherwise. Heterogeneity was assessed and resolved by sensitivity analysis. Study quality was evaluated using the Cochrane Risk-of-Bias tool for randomized trials and the Newcastle–Ottawa Scale (NOS) for observational studies.

Results:

Four studies including 1,361 patients (three phase III randomized trials and one multicenter cohort) were analyzed. Combination therapy significantly improved PFS (HR = 0.70, 95% CI 0.61–0.80; $p < 0.00001$; $I^2 = 0\%$), showing a consistent benefit. OS showed a favorable but non-significant trend (HR = 0.59, 95% CI 0.28–1.23; $p = 0.16$; $I^2 = 70\%$). Grade 3–5 AEs were more frequent with combination therapy (OR = 2.42, 95% CI 1.83–3.19; $p < 0.00001$; $I^2 = 0\%$), with low heterogeneity after sensitivity testing using the leave-one-out test. All randomized trials were rated low risk of bias, and the observational study scored 8/9 on the NOS, indicating high methodological quality.

Conclusions:

TACE combined with dual immune and anti-angiogenic blockade significantly improves HCC control compared with TACE alone, supporting synergistic local and systemic effects. Although high-grade AEs are more frequent, their incidence is consistent and manageable. This integrated approach represents a promising advancement toward optimized multimodal therapy for intermediate-stage HCC.

Keyword(s) : Hepatocellular carcinoma, transarterial chemoembolization, immunotherapy, anti-angiogenic therapy, immune checkpoint inhibitors, progression-free survival

2076 - Xiaozhi Huaxian Formula ameliorates type 2 diabetes mellitus associated metabolic dysfunction associated steatohepatitis through modulating hepatic interleukin 23 interleukin 17 Axis

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Background / Hypothesis:

Type 2 diabetes mellitus (T2DM) can cause metabolic dysfunction-associated steatotic liver disease (MASLD) and promote it rapid progression to metabolic dysfunction-associated steatohepatitis (MASH), hepatic fibrosis, cirrhosis even to hepatocellular carcinoma. The improvement of the complex diseases needs the rationally designed multi-target, multi-level and multi-angle therapeutic strategy. Xiaozhi Huaxian Formula (XHF), a patented Chinese herbal formulation derived from a modified classical prescription (Patent NO. ZL202310099104.1), has demonstrated therapeutic efficacy in ameliorating T2DM-associated MASH. Nevertheless, the precise molecular mechanisms responsible for its hepatoprotective effects remain to be fully elucidated.

Materials and Methods:

A mouse model of T2DM-associated MASH was established by administering streptozotocin to 2-day-old male C57BL/6J pups, followed by post-weaning feeding with a high-fat diet. After model confirmation, mice received daily oral XHF or vehicle for four weeks. Metabolic parameters and liver function were assessed from serum, and interleukin-23 (IL-23), IL-17A, augmenter of liver regeneration (ALR) and albumin (ALB) were measured in supernatant of liver tissue homogenate. Statistical correlation analyses were performed to evaluate the relationships between IL-23/IL-17A levels and the other measured indicators.

Results:

XHF administration dose-dependently ameliorated insulin resistance, glucose and lipid dysmetabolism (fasting blood glucose, triglyceride, insulin, homeostasis model assessment of insulin resistance index), hepatic steatosis, inflammation, and serum liver enzyme elevations (alanine aminotransferase, aspartic transaminase, alkaline phosphatase, and glutamyl transpeptidase), leading to a significant reduction in the NAFLD activity score (all $P < 0.001$, respectively). Concurrently, XHF treatment downregulated hepatic IL-23 and IL-17A secretions in T2DM-associated MASH mice, exhibiting a clear dose-response relationship (all

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$P < 0.001$, respectively). Moreover, XHF significantly promoted hepatocyte regeneration, as evidenced by increased synthesis and secretion of ALR and ALB in the blood and liver (all $P < 0.005$, respectively). Correlation analysis revealed a significant positive association between IL-23 and IL-17A levels. Furthermore, both cytokines demonstrated significant inverse correlations with parameters of glucose and lipid metabolism, liver function, and hepatocellular regeneration (all $P < 0.005$, respectively).

Conclusions:

Our study elucidates that XHF alleviates T2DM-associated MASH by inhibition of the IL-23/IL-17A axis. These findings uncover a novel mechanism of action for XHF and highlight its potential as a therapeutic agent for MASH and related metabolic diseases.

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Keyword(s) : Xiaozhi Huaxian formula, T2DM, MASH, IL-23, IL-17

2077 - Xiaozhi Huaxian Formula ameliorates type 2 diabetes mellitus associated metabolic dysfunction associated steatohepatitis through modulating hepatic interleukin 23 interleukin 17 Axis

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Background / Hypothesis:

Type 2 diabetes mellitus (T2DM) can cause metabolic dysfunction-associated steatotic liver disease (MASLD) and promote its rapid progression to metabolic dysfunction-associated steatohepatitis (MASH), hepatic fibrosis, cirrhosis even to hepatocellular carcinoma. The improvement of the complex diseases needs the rationally designed multi-target, multi-level and multi-angle therapeutic strategy. Xiaozhi Huaxian Formula (XHF), a patented Chinese herbal formulation derived from a modified classical prescription (Patent NO. ZL202310099104.1), has demonstrated therapeutic efficacy in ameliorating T2DM-associated MASH. Nevertheless, the precise molecular mechanisms responsible for its hepatoprotective effects remain to

be fully elucidated.

Materials and Methods:

A mouse model of T2DM-associated MASH was established by administering streptozotocin to 2-day-old male C57BL/6J pups, followed by post-weaning feeding with a high-fat diet. After model confirmation, mice received daily oral XHF or vehicle for four weeks. Metabolic parameters and liver function were assessed from serum, and interleukin-23 (IL-23), IL-17A, augmentor of liver regeneration (ALR) and albumin (ALB) were measured in supernatant of liver tissue homogenate. Statistical correlation analyses were performed to evaluate the relationships between IL-23/IL-17A levels and the other measured indicators.

Results:

XHF administration dose-dependently ameliorated insulin resistance (homeostasis model assessment of insulin resistance index, homeostasis model assessment for beta-cell function index), glucose and lipid dysmetabolism (fasting blood glucose, serum and liver triglyceride, insulin), hepatic steatosis and inflammation, serum liver enzyme elevations (alanine aminotransferase, aspartic transaminase, alkaline phosphatase, and glutamyl transpeptidase), and the NAFLD activity score (all $P < 0.001$, respectively). Concurrently, XHF treatment downregulated hepatic IL-23 and IL-17A secretions in T2DM-associated MASH mice, exhibiting a clear dose-response relationship (all $P < 0.001$, respectively). Moreover, XHF significantly promoted hepatocyte regeneration, as evidenced by increased synthesis and secretion of ALR and ALB in the blood and liver (all $P < 0.005$, respectively). Correlation analysis revealed a significant positive association between IL-23 and IL-17A levels. Furthermore, both cytokines demonstrated significant inverse correlations with the improvement of parameters of the NAFLD activity score, insulin resistance, glucose and lipid metabolism, liver function, and hepatocellular regeneration (all $P < 0.005$, respectively).

Conclusions:

Our study elucidates that XHF alleviates T2DM-associated MASH by inhibition of the IL-23/IL-17A axis. These findings uncover a novel mechanism of action for XHF and highlight its potential as a therapeutic agent for MASH and related metabolic diseases.

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Keyword(s) : Xiaozhi Huaxian formula, T2DM, MASH, IL-23, IL-17

2078 - Highly Discriminative Risk Stratification for Long Term Liver Related Mortality in MASLD Patients

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Background / Hypothesis:

Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is increasingly prevalent and linked to obesity, diabetes, dyslipidemia, and other cardiometabolic risk factors (CMRFs). Advanced MASLD can lead to cirrhosis, hepatocellular carcinoma, and liver-related death (LRD). However, robust long-term risk stratification tools for LRD remain limited. Therefore, we aimed to quantify long-term liver-related mortality and to develop a clinically applicable risk score for LRD among individuals with MASLD.

Materials and Methods:

Using the MJ cohort (1996-2022), we constructed Cox models and evaluated discrimination via time-dependent AUCs and calibration. An integer-based cumulative risk score was derived from the optimal model using weighted regression coefficients.

Results:

A total of 140,247 naïve MASLD participants without baseline major events from the MJ health-check cohort were enrolled. Over a median follow-up of 17 years, 813 LRDs occurred (0.58%), yielding a mortality rate of 3.5 deaths per 10,000 person-years. Predictive models incorporating cardiometabolic risk factors (CMRFs), age, sex, and the FIB-4 index demonstrated superior discrimination compared to models relying only on CMRFs or age/sex, with AUCs 0.93-0.88 over 5-20 years. A Risk Score was derived from: Age (6–12 points), obesity (BMI ≥ 30 , 1 point), diabetes (up to 3 points), HDL-C (up to 2 points), hypertriglyceridemia (-2 points), and FIB-4 (>3.25 assigned 18 points). Compared to the reference group (Score ≤ 5 ; 0.3 per 10,000 PY), the highest risk group (Score ≥ 30) had 219.4 per 10,000 PY and HR 664.06 (95% CI: 407.65-

1081.23, $P < 0.001$).

Conclusions:

The proposed risk score, combining readily available CMRFs, age, and FIB-4, provides a simple, highly discriminative tool for long-term LRD risk stratification in MASLD, who may benefit from intensive monitoring and early intervention.

Keyword(s) : Metabolic Dysfunction-Associated Steatotic Liver Disease; Liver-related mortality; Risk score; Cardiometabolic risk factors

2079 - Clinical characteristics of patients with primary biliary cholangitis across various disease stages

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Background / Hypothesis:

Currently, the gold standard for definitive diagnosis of primary biliary cholangitis (PBC) remains percutaneous liver biopsy with histological assessment. This study aims to characterized the clinical profile of patients with PBC and compared features between early and late disease stages.

Materials and Methods:

This retrospective study included patients who underwent liver biopsy were diagnosed with PBC at Beijing Ditan Hospital. Patients were stratified into two groups based on the pathological staging: patients in I/II stage (Early stage group), and patients in III/IV stage (Late stage group). Clinical features were analyzed.

Results:

A total of 218 PBC patients were included, predominantly female (86.70%), with a mean age of 49.92 years. The cohort was stratified into early-stage (n=154) and late-stage (n=64) groups for comparative analysis. Key differences in laboratory parameters were observed. Late-stage patients exhibited significantly higher medians of total bilirubin (21.90 vs. 13.20 $\mu\text{mol/L}$), direct bilirubin (11.40 vs. 4.70 $\mu\text{mol/L}$), alkaline phosphatase (ALP) (229.10 vs. 127.65 U/L), gamma-glutamyl transferase (GGT) (223.95 vs. 123.85 U/L), total bile acids (41.55 vs. 10.35 $\mu\text{mol/L}$), and immunoglobulin M (IgM) (2.67 vs. 2.15 g/L) compared to early-stage patients (all $p < 0.05$). Conversely, late-stage patients had significantly lower levels of hemoglobin (120.08 vs. 126.70 g/L), platelet count

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(148.00 vs. 214.50 x10⁹/L), and albumin (38.77 vs. 41.01 g/L) (all p<0.05). Serologically, the prevalence of anti-Ro-52 antibody was significantly higher in the late-stage group (59.38% vs. 30.52%, p<0.001). The distribution of lymphocyte count, GGT, ALP, and IgM further illustrated the divergence between groups (Figure 1).

Conclusions:

Significant differences in biochemical, hematological, and immunological markers exist between early and late-stage PBC, reflecting progressive cholestasis, synthetic dysfunction, and potentially distinct immune activity with disease advancement.

Keyword(s) : primary biliary cholangitis, gamma-glutamyl transferase, alkaline phosphatase, Anti mitochondrial antibody, early stage

2080 - Comparing Care Settings for Sofosbuvir Ravidasvir Delivery in Hepatitis C Nested Cohort Findings from the EASE Trial

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Background / Hypothesis: The EASE trial led by Malaysia's Ministry of Health demonstrated that an 8-week sofosbuvir/ ravidasvir regimen is non-inferior to the conventional 12-week course for individuals with non-cirrhotic hepatitis C virus (HCV) infection. The trial was deployed in two different public healthcare environments: specialist-run hospital clinics and primary care facilities supervised by family physicians. How these differing service models, staffing structures, and levels of research experience influence treatment delivery has not been clearly characterized. This study aimed to evaluate whether treatment adherence and sustained virological response at 12 weeks after therapy (SVR12) differ between participants treated in hospital settings versus those managed in primary care.

Materials and Methods: We conducted a nested cohort

analysis within the EASE trial involving 322 participants (hospital: n=102; primary care: n=220). Adherence was assessed using pill counts and patient-reported diaries. SVR12 was defined as HCV RNA <15 IU/mL. We compared demographic and clinical variables between groups and examined the relationship between facility type and SVR12 using unadjusted and adjusted logistic regression models. Covariates included sociodemographic factors, treatment duration, adherence, and adverse events.

Results: Baseline characteristics were largely comparable across settings, with the exception of age (hospital: 46.6 ± 8.8 years; primary care: 43.5 ± 10.4 years; p=0.009). Most participants were male and had attained secondary-level education. Use of the 8-week regimen was similar between groups. Adherence rates were high in both settings (hospital: 99.0%; primary care: 97.3%) with no significant difference observed. SVR12 rates were also equivalent (93.9% vs. 93.2%). In adjusted models, treatment setting was not associated with SVR12 (aOR 0.75; 95% CI 0.26–2.38; p=0.608).

Conclusions: HCV treatment delivered through primary care centers performed on par with hospital-based services in both adherence and virological outcomes. These findings reinforce the feasibility and effectiveness of decentralizing HCV care and support the use of task-shifting approaches to expand treatment access. They also highlight the potential for primary care networks to contribute meaningfully to future clinical research across a broader spectrum of diseases.

Keyword(s) : hepatitis C, ravidasvir, primary care, HCV, viral load, suppression, Malaysia

2082 - Time to Viral Load Suppression with 8 versus 12 Week Ravidasvir Sofosbuvir Regimens among Chronic Hepatitis C Patients in Malaysia

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Background / Hypothesis: Hepatitis C virus (HCV) infection remains a major global health burden, contributing to chronic liver disease and significant mortality. Malaysia has implemented direct-acting antiviral (DAA) therapy to support the World Health Organization's goal of eliminating HCV by 2030. Ravidasvir/sofosbuvir (RDV/SOF) is a potent, pan-genotypic DAA regimen that has demonstrated high efficacy, yet evidence comparing the time to viral load suppression between shorter and standard treatment durations is limited. This study aimed to determine the time to HCV RNA viral load suppression for the 8-week and 12-week RDV/SOF regimens among chronic HCV patients in Malaysia and to identify factors associated with suppression time.

Materials and Methods: This secondary analysis utilized data from an open-label, multicentre, parallel-group clinical trial (2021–2023) involving 322 adult patients with chronic HCV infection. Data was analysed using R statistical package (4.5.1). Kaplan–Meier survival analysis and Cox proportional hazards regression were used to evaluate time to viral suppression (HCV RNA <15 IU/mL) and associated predictors. The final model was stratified by baseline HCV RNA load, concomitant medication, and imprisonment risk factor to satisfy proportional hazards assumptions.

Results: The median time to viral load suppression was 28 days for both treatment groups, with no significant difference in Kaplan–Meier analysis ($p = 0.076$). In multivariable Cox regression, patients receiving the 8-week regimen were more likely to achieve viral suppression earlier than those on the 12-week regimen (adjusted hazard ratio [aHR] = 1.29, 95% CI: 1.01–1.63, $p = 0.038$). Higher baseline HCV RNA load was significantly associated with delayed suppression ($p < 0.001$). Other demographic and clinical factors were not significant.

Conclusions: Both 8- and 12-week RDV/SOF regimens achieved rapid viral suppression, with comparable median times. The 8-week regimen demonstrated a

higher likelihood of earlier suppression, suggesting its potential as an effective and cost-efficient alternative to the standard 12-week course in Malaysia's HCV elimination program.

Keyword(s) : survival analysis, HCV, RNA, viral load, suppression, Malaysia

2084 - Workflow Optimization to Reduce Chemotherapy Drug Waiting Time in Inpatient Settings

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Background / Hypothesis:

In recent years, the increasing number of cancer patients has led to a growing demand for inpatient chemotherapy. Patients undergoing cancer treatment already endure significant physical and emotional burdens; prolonged waiting times during hospitalization further exacerbate their discomfort and dissatisfaction. This project aimed to analyze the inpatient chemotherapy workflow, identify sources of delay, and reduce the average waiting time for chemotherapy drug preparation. Ultimately, the goal was to improve medical service quality, decrease patient waiting time, and enhance patient and family satisfaction.

Materials and Methods:

Chemotherapy waiting time was defined as the duration from the patient's arrival in the ward to the initiation of chemotherapy administration by nursing staff. The project reviewed and analyzed the workflow of the inpatient chemotherapy unit, focusing on key process bottlenecks, including chemotherapy order entry, physician verification, message return, and the scheduled delivery of chemotherapy drugs to the ward. Two major interventions were implemented: (1) establishing a workflow for physicians to enter chemotherapy orders prior to patient arrival, and (2) optimizing and standardizing the delivery schedule of chemotherapy drugs.

Results:

Overall waiting time was successfully reduced from 278 minutes to 162 minutes, achieving a total reduction of 116 minutes. Additional benefits included:

- Bed transfer rate decreased from 19.2% to 11.3%.
- Bed occupancy rate increased from 82.64% to 89.4%.
- Patient satisfaction improved significantly from 73.4%

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to 100%.

Conclusions:

Implementing pre-arrival chemotherapy order entry effectively simplified the workflow and reduced waiting times for chemotherapy preparation. However, system limitations—such as the inability to synchronize treatment dates—created operational inconvenience. Additionally, delayed drug delivery time to 12:30 PM impacted pharmacy staff lunch breaks. These areas require further improvement, and continued collaboration with relevant departments is recommended to establish a more efficient and sustainable workflow.

Keyword(s) : chemotherapy, workflow optimization, inpatient process improvement, waiting time reduction, patient satisfaction, oncology nursing

2085 - Inhibition of Aurora B Induces Senescence and Potentiates Immunotherapy in Hepatocellular Carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a global health challenge, with limited treatment options for advanced-stage patients. Although recent approvals of targeted therapies and immune checkpoint inhibitors (ICIs) have expanded the therapeutic landscape, their clinical benefits are often constrained by modest response rates and acquired resistance. Despite ongoing efforts to identify new therapeutic targets, only few inhibitors have progressed to clinical trials, benefiting a small subset of patients. These limitations underscore the need for more effective therapeutic strategies.

Materials and Methods:

We integrated data from DepMap and TCGA to identify HCC-specific vulnerabilities. A kinome-wide CRISPR screen validated essential targets. Pharmacological inhibition (AZD1152) and genetic knockout of Aurora B (AURKB) were employed for functional validation. Senescence was assessed via SA- β -gal staining, transcriptomics, and SASP analysis. Immune interactions were evaluated using co-culture assays with T cells and flow cytometry for MHC-I and immune markers. In vivo efficacy was tested in immunocompetent murine HCC models.

Results:

Aurora B was identified as a top candidate essential for HCC survival, overexpressed in tumors, and correlated with poor prognosis. A kinome-wide CRISPR screen further confirmed its essential role in HCC cell survival. Pharmacologic inhibition or genetic knockout of Aurora B markedly suppressed proliferation of HCC cells and induced robust cellular senescence, characterized by cell-cycle arrest, DNA damage, and SASP. These senescent cells exhibited heightened susceptibility to T-cell-mediated cytotoxicity, associated with increased immunoproteasome activity and upregulated HLA expression. Co-culture assays revealed that senescent tumor cells promoted interferon- γ production and enhanced cytotoxicity in CD8⁺ T cells as well as PD-1 expression. In both in vitro and in vivo models, combining Aurora B inhibition with anti-PD-1 therapy markedly suppressed tumor growth.

Conclusions:

This study identifies Aurora B as a therapeutically actionable vulnerability in HCC, whose inhibition induces senescence and enhances tumor immunogenicity. The combination of Aurora B inhibitor with anti-PD-1 therapy yields synergistic anti-tumor effects, supporting its potential as a promising strategy to improve immunotherapy outcomes in HCC.

Keyword(s) : Hepatocellular carcinoma, Senescence, Immunotherapy.

2088 - The Effect of Semaglutide on Liver Histology in Metabolic Dysfunction Associated Steatohepatitis A Systematic Review and Meta Analysis of Randomized Controlled Trials

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common chronic liver disease, affecting over 30% globally. Defined by hepatic fat accumulation within the context of metabolic syndrome, it poses a major public health challenge and is a leading cause of liver-related illness and death. A significant subset of MASLD progresses to metabolic dysfunction-associated steatohepatitis (MASH) and the worst outcome is Liver Cirrhosis. Histologically, MASH involves hepatocyte injury and lobular inflammation, which primarily drives hepatic fibrogenesis. Liver fibrosis progression is the single most important

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prognostic factor in MASLD, directly correlating with the risk of cirrhosis, hepatic failure, hepatocellular carcinoma, the need for liver transplantation, and all-cause mortality.

Materials and Methods: The study conducted in accordance with PRISMA guidelines. We performed a systematic search of PubMed/MEDLINE, Embase, Cochrane Central Register of Controlled Trials (CENTRAL), and ClinicalTrials.gov for RCTs published in the last 20 years. Meta-analysis was performed using RevMan 5.4 tool.

Results: The search identified three large-scale RCTs that met the strict inclusion criteria, enrolling a total of 1,233 patients between the selected studies. The pooled analysis of all three trials found statistical significance in histological improvement in steatohepatitis without worsening fibrosis odds ratio of 3.43 (95% CI 2.59-4.53, $p < 0.001$) and improvement in fibrosis without worsening steatohepatitis odds ratio 1.73 (95% CI 1.29, 2.33, $p < 0.0003$).

Conclusions: In conclusion, the meta-analysis demonstrates that semaglutide has beneficial histologic effects: both in Steatohepatitis and Fibrosis in patients with NAFLD with F2-F3 Fibrosis, with a well-tolerated safety profile. However, the results are limited by the small number of included studies and clinical heterogeneity, which restricts the generalizability of these findings across the spectrum of MAFLD.

Keyword(s) : MAFLD, MASH, NAFLD, NASH, Steatohepatitis, Fibrosis, Semaglutide, Ozempic, Wegovy, Rybelsus

2089 - Secondary Biliary Cirrhosis Following Iatrogenic Bile Duct Injury

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Abstract: Secondary biliary cirrhosis is a chronic liver disease caused by prolonged biliary obstruction. One of the most common iatrogenic causes is biliary stricture following laparoscopic cholecystectomy. We present the case of a 39-year-old woman who developed progressive jaundice and secondary biliary cirrhosis several years after cholecystectomy, attributed to an undiagnosed biliary stricture. Early identification and intervention are key to preventing irreversible

hepatic damage.

Introduction: Laparoscopic cholecystectomy is the gold standard for the treatment of symptomatic gallstones. However, bile duct injuries (BDIs) are a known complication, with an incidence of 0.3–0.7%. If unrecognized or inadequately managed, BDIs can lead to chronic biliary obstruction, cholangitis, and secondary biliary cirrhosis (SBC).

Case Presentation: A 39-year-old woman presented with progressive jaundice, fatigue, pruritus, and abdominal distention for 5 months.

History: She underwent laparoscopic cholecystectomy 13 years earlier for symptomatic cholelithiasis. The surgery was reported to be uneventful, though detailed operative records were unavailable. Over the past 5 years, she experienced intermittent episodes of jaundice and abdominal pain, managed symptomatically.

Examination: Icterus, ascites and splenomegaly were present

Investigations

Liver Function Tests:

- o Bilirubin: 18.4 mg/dL
- o AST: 110 IU/L, ALT: 95 IU/L
- o ALP: 980 IU/L, GGT: 420 IU/L
- o INR: 1.6
- o Albumin: 2.1 g/dL

Mild anemia (Hb 9.8 g/dL), thrombocytopenia (platelets 80,000/mm³)

Ultrasound Abdomen: Coarse hepatic parenchyma, dilated intrahepatic bile ducts, splenomegaly and ascites were present

- MRCP: A stricture at the level of CHD with significant upstream IHB dilatation. Liver parenchyma showed features suggestive of cirrhosis.

- Endoscopy: Oesophageal varices II-III

Diagnosis

Secondary biliary cirrhosis due to chronic biliary obstruction from post-cholecystectomy biliary stricture.

Management

ERCP with plastic stent placement was performed. The patient was referred for surgical consultation for evaluation of definitive biliary reconstruction.

Discussion

Bile duct injury is a feared complication of laparoscopic cholecystectomy and may go unrecognized, especially if minor. Chronic biliary obstruction can silently progress to secondary biliary cirrhosis, a potentially irreversible condition if not identified early.

MRCP remains the imaging modality of choice, and ERCP plays both diagnostic and therapeutic roles.

Long-standing strictures often require surgical correction to prevent recurrence.

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Conclusion

This case highlights the long-term consequence of unrecognized post-cholecystectomy biliary stricture leading to secondary biliary cirrhosis. High clinical suspicion, timely imaging, and endoscopic intervention can prevent progression to cirrhosis.

Keyword(s) :

2090 - Integration of Microbial Signatures into Machine Learning Models Significantly Improves Prediction of Adverse Outcomes in Patients with Hepatitis B Related Cirrhotic Ascites

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Background: Traditional clinical assessment methods have limitations in early identification of poor prognosis in hepatitis B-related cirrhotic ascites. This study aimed to evaluate the value of a novel machine learning model that integrates microbial signatures with conventional clinical features for predicting 90-day end-stage liver disease (ESLD) events in patients with hepatitis B-related cirrhotic ascites.

Methods: A total of 306 patients with cirrhotic ascites who underwent ascitic fluid culture between 2019 and 2024 were retrospectively enrolled. Among them, 206 patients from 2019–2022 were assigned as the training set, and 100 patients from 2022–2024 served as an independent test set. The primary outcome was ESLD events occurring within 90 days after ascitic fluid sampling, including liver failure-related death, liver transplantation, hepatorenal syndrome, or grade III–IV hepatic encephalopathy. Clinical and microbial features were analyzed. Three models were constructed: a baseline MELD-Na model, a logistic regression model, and an eXtreme Gradient Boosting (XGBoost) model integrating all features. Model performance was evaluated on the independent test set. For the XGBoost model, SHapley Additive exPlanations (SHAP) values were used to rank feature importance.

Results: The overall 90-day endpoint event rate was 35%. Compared to the non-ESLD group, patients with ESLD exhibited significantly worse microbial profiles (higher rates of positive ascitic fluid culture, polymicrobial infection, carbapenem resistance, and availability of fewer than two effective antibiotics) and clinical parameters (MELD-Na score, ascitic fluid polymorphonuclear leukocyte count, procalcitonin, and serum creatinine). The XGBoost model demonstrated

significantly superior predictive performance, with an AUC of 0.85 on the test set, outperforming both the MELD-Na model (AUC = 0.72) and the logistic regression model (AUC = 0.78) ($P < 0.05$). SHAP analysis further confirmed that carbapenem resistance was the most influential microbial predictor, while MELD-Na score, procalcitonin, and availability of fewer than two effective antibiotics were also important features.

Conclusion: Integrating microbial signatures, with a focus on antimicrobial resistance, substantially improves the prediction of short-term adverse outcomes in patients with cirrhotic ascites. This model may facilitate early identification of high-risk patients and guide precise clinical intervention.

Keyword(s) : Hepatitis B-related cirrhotic ascites; Prognostic prediction; Carbapenem resistance; XGBoost model; Machine learning

2091 - Treatment outcome of interferon based therapy versus sofosbuvir ribavirin therapy in chronic hepatitis C genotype 3 and compensated liver cirrhosis

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Background / Hypothesis: Chronic hepatitis C remains a major global cause of progressive liver disease. Genotype 3 is associated with rapid fibrosis progression and lower treatment response compared with several other viral genotypes. Although directly acting oral antivirals have transformed hepatitis C management, interferon-based regimens are still used in many regions where access to newer agents is limited. This study hypothesized that a twelve-week regimen combining pegylated interferon alfa two a with sofosbuvir and ribavirin would provide similar virological efficacy to a twenty-four week all oral regimen of sofosbuvir and ribavirin in treatment naive genotype 3 chronic hepatitis C and compensated cirrhosis.

Materials and Methods: This randomized open label clinical study was conducted in the Department of Hepatology at a tertiary center from January two thousand sixteen to August two thousand seventeen.

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Thirty treatment naive adults with genotype 3 chronic hepatitis C or compensated cirrhosis were enrolled and randomly assigned into two treatment groups. Group one received twelve weeks of once weekly pegylated interferon alfa two a plus daily sofosbuvir and ribavirin. Group two received twenty-four weeks of daily sofosbuvir and ribavirin. Viral load testing was performed at baseline, end of treatment, and twelve weeks after completion to determine sustained virological response. Patients were monitored throughout therapy for adverse events and treatment tolerability.

Results: All thirty participants completed therapy without treatment discontinuation or loss to follow up. Both treatment groups demonstrated rapid virological response, end of treatment response, and sustained virological response at twelve weeks in one hundred percent of patients. No virological breakthrough or relapse occurred in either group. Reported adverse effects were mostly mild and included fatigue, insomnia, hair loss, and nausea in both groups. Flulike symptoms were observed only in patients receiving pegylated interferon. No serious adverse events or hepatic decompensation occurred.

Conclusions: Both regimens produced excellent virological outcomes in genotype 3 chronic hepatitis C and compensated cirrhosis, achieving one hundred percent sustained virological response at twelve weeks. The combination of sofosbuvir and ribavirin, with or without pegylated interferon, remains an effective therapeutic option where access to newer antiviral agents is limited. The interferon containing twelve-week regimen offers a shorter but equally effective alternative to the twenty-four-week oral regimen.

Keyword(s) : Hepatitis C, genotype 3, interferon alfa, sofosbuvir, ribavirin, cirrhosis, antiviral therapy

2093 - Effects of Extracellular Vesicles Secreted by Gut Microbiota on the Function of Peripheral CD8⁺ T Cells and Regulatory T Cells in Patients with Hepatocellular Carcinoma

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Background / Hypothesis:

The gut microbiota can modulate anti-tumor immune responses in patients with hepatocellular carcinoma (HCC). CD8⁺ T cells and CD4⁺ T regulatory cells (Tregs) play crucial roles in anti-tumor immunity, but the specific molecular mechanisms by which the gut microbiota regulates their function remain unclear. This study aimed to explore the effects of extracellular vesicles (EVs) derived from different gut microbiota sources on the function of peripheral CD8⁺ T cells and Tregs in HCC patients, as well as the underlying mechanisms

Materials and Methods:

We isolated EVs from different gut microbiota and co-cultured them with peripheral blood mononuclear cells (PBMCs) obtained from HCC patients. The functions of CD8⁺T cells and Tregs were assessed by flow cytometry, and cytokine levels in culture supernatants were detected using enzyme-linked immunosorbent assay (ELISA). To identify which component in the EVs regulate T cell function, the EV preparations were pretreated with DNase or protease. An IRF3 inhibitor was used to verify whether EVs enhance CD8⁺ T cell function via the cGAS-STING pathway. Finally, 16S rRNA gene sequencing was performed to analyze the bacterial DNA within EVs

Results:

EVs derived from healthy donors and patients who responded to immune checkpoint inhibitors (ICIs) can downregulate the frequency of Tregs in PBMCs from patients with HCC and enhanced function of CD8⁺ T cells. This effect is associated with the activation of the cGAS-STING pathway by exogenous bacterial DNA harbored in EVs, accompanied by significant changes in the levels of various cytokine. 16S rRNA sequencing analysis revealed that the bacterial DNA profiles within EVs exhibited significant differences among three groups: ICI non-responders, ICI responders, and healthy controls

Conclusions:Healthy individuals and HCC patients who respond to ICIs treatment have distinct gut microbiota profiles, and their microbiota-derived EVs can enhance peripheral anti-tumor immune responses in HCC patients

Keyword(s) : HCC; extracellular vesicles; CD8⁺ T cells; regulatory T cells; antitumor immunity

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2095 - Preliminary Evaluation of Real World Efficacy and Safety of Tenofovir Prafulvate Tablets in the Treatment of 16 Patients with Chronic HBV Infection

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Background / Hypothesis:Chronic Hepatitis B (CHB) is a global public health concern. The treatment rate of chronic HBV infection in China is far below WHO's 2030 targets, and existing nucleoside/nucleotide analogs have limitations in efficacy or safety. Although Pradefovir (approved in October 2024) possess the advantage of liver targeting, real-world data on this drug remains scarce.

Materials and Methods:This retrospective study enrolled 16 chronic HBV-infected patients treated with Pradefovir at the Infectious Diseases Department of Yan'an University Affiliated Hospital from January to July 2025. Changes in HBV DNA, HBsAg, HBeAg, liver function indicators, liver ultrasound, and LSM values before and after treatment, while documenting adverse reactions during therapy.

Results:After 12 weeks of treatment with Pradefovir, 16 chronic HBV-infected patients showed significant improvements in virological and serological indicators: HBV DNA levels decreased in 80% of patients (12/15 cases); HBsAg and HBeAg seroconversion rates were both 93.75% (15/16 cases), with 1 case each in the treatment-naïve and treatment-experienced groups achieving HBeAg seroconversion (16.67%, 10.00%); liver function indicators remained stable throughout treatment; Hepatological histology parameters improved: Among 11 evaluable patients, the LSM normalization rate increased from baseline 36.36% (4/11) to 63.64% (7/11); portal vein width narrowed or remained normal in 81.82% (9/11) of patients; and spleen thickness decreased in 45.45% (5/11) of patients. No significant adverse reactions were observed, and all patients reported no special discomfort during treatment.

Conclusions:Pradefovir demonstrate potential for rapidly suppressing viral replication, promoting antigen

clearance, and improving liver histology in the short-term treatment of chronic HBV infection.

Keyword(s) : Pradefovir; Chronic hepatitis B; Curative effect; Safety

2096 - Case Report of Decreased Cholinesterase Levels During Treatment with Prifosfamide Mesylate in a Patient with Chronic HBV Infection

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Background / Hypothesis:If chronic hepatitis B virus (HBV) infection is not well controlled, it can progress to liver fibrosis, liver cirrhosis, and even hepatic carcinoma, and antiviral therapy is the core treatment modality. Pradefovir is a novel nucleoside (nucleotide) analog (NAs), which exerts its effect by inhibiting HBV reverse transcriptase. Serum cholinesterase (CHE) is a sensitive indicator reflecting hepatic synthetic function; however, its association with pradefovir mesylate treatment has not been reported yet, and its mechanism and clinical significance remain unclear.

Materials and Methods:Eighteen patients with chronic HBV infection receiving pefenavir mesylate treatment at Yan'an University Affiliated Hospital were enrolled. Changes in serum CHE levels were observed before and after 12 weeks of treatment.

Results:All patients exhibited varying degrees of serum CHE reduction post-treatment, with a significantly lower rate of normal CHE levels compared to baseline. Regarding antiviral efficacy, most patients achieved HBV DNA levels below the detection limit, while only a minority maintained low viral loads. No statistically significant difference was observed in ALT levels before and after treatment. No significant adverse reactions occurred throughout the treatment period, with all patients presenting "asymptomatic CHE reduction."

Conclusions:Twelve weeks of pefarnavir mesylate treatment in chronic HBV-infected individuals induces a widespread and significant decrease in serum CHE levels. However, this change does not currently affect the drug's antiviral efficacy, and short-term treatment

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demonstrates good safety.

Keyword(s) : Pradefovir; chronic hepatitis B; Cholinesterase;

2097 - Improving Elimination in Albumin Dialysis Liver Support Different Responses of Water Soluble and Albumin Bound Toxins

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Background / Hypothesis:

Extracorporeal Albumin Detoxification-Dialysis (ECAD) is the leading procedure to support failing livers by detoxification, it has been used in over 150.000 therapies. Past Meta-Analysis concluded dose of treatment would be an important cofactor determining a good outcome. The introduction of a new generation of adsorbents to maintain dialysate albumin's purity has increased the bile acid removal compared to MARS and Cytosorb. However, since next generation adsorbents may have moved the set points for optimal blood and dialysate flow rates, the aim of this investigation was to elucidate the role of blood- and dialysate flow on the removal of water soluble toxins, albumin bound toxins and improvement of albumin binding.

Materials and Methods:

Eight Albunique treatments were performed using two blood flow rates (100 and 130 mL/min) and stepwise increasing dialysate flow rates (2, 3 and 4 L/h). Pre- and post-dialyzer blood samples were collected. Photometric analysis was used to quantify albumin-bound toxins (bilirubin and bile acids) and water-soluble toxins (creatinine and urea). Albumin binding function at Sudlow site II was assessed by Dansylsarcosine titration, yielding K_d values (concentration at which half of the available binding sites are occupied). Instantaneous filter clearance was calculated and compared across the different flow conditions.

Results:

Instantaneous filter clearance during the eight Albunique treatments was analyzed for albumin-bound toxins (bilirubin and bile acids) and water-soluble toxins (creatinine) under two blood flow rates and stepwise increasing dialysate flow rates. Significantly higher clearance was observed for water-soluble toxins, particularly creatinine, with increasing dialysate flow. In contrast, no advantage was

demonstrated for albumin-bound toxins; a trend toward reduced clearance was noted at higher dialysate and blood flow rates. Furthermore, K_d values at Sudlow site II did not improve with increased flow rates. These findings indicate a differential response of toxin classes to dialysate flow rate adjustments.

Conclusions:

The present data demonstrate that increasing dialysate and blood flow rates significantly enhance the clearance of water-soluble toxins. In contrast, no beneficial effect was observed for albumin-bound toxins; higher flow rates were not associated with a trend toward improved clearance of bilirubin and bile acids. Similarly, albumin binding function did not improve under higher flow conditions. These findings suggest that aggressive flow escalation does not confer an advantage for the removal of albumin-bound toxins. Instead, lower or moderate flow settings may provide more favourable conditions for albumin-mediated detoxification.

Keyword(s) : Extracorporeal Albumin Detoxification-Dialysis (ECAD), Albumin Binding Function, Extracorporeal Liver Support, Clearance Rates

2098 - An Observational Study on the Early Intervention Treatment of Fuzheng Huayu Capsule Combined with Antiviral Drugs in High Risk Populations of HBV Related Hepatocellular Carcinoma

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Background / Hypothesis:

To investigate the effects of Fuzheng Huayu Capsule combined with nucleos(t)ide analogues (NAs) on liver fibrosis, liver function and the incidence of long-term endpoint events in high-risk populations of hepatitis B virus (HBV)-related hepatocellular carcinoma (HCC), and to provide evidence-based basis for clinically reducing the risk of end-stage liver disease in such populations.

Materials and Methods:

A total of HBV-related HCC high-risk patients who visited the Department of Infectious Diseases of Yan'an University Affiliated Hospital from January 2015 to July 2025 were selected as research subjects. They were

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divided into monotherapy group (treated with first-line NAs alone) and combination group (treated with NAs combined with Fuzheng Huayu Capsule) according to the treatment regimen. The changes of liver function, liver fibrosis-related indexes, liver stiffness measurement (LSM) and anteroposterior diameter of main portal vein at baseline and 144 weeks after treatment were compared between the two groups. The incidence of clinical endpoint events such as HCC and decompensated liver cirrhosis within 3 years was counted, and COX regression model was used to analyze the risk factors of endpoint events.

Results:

After treatment, liver function indexes (ALT, AST) in both groups were improved compared with baseline, but there was no significant difference between the two groups ($P > 0.05$). The improvement amplitudes of liver fibrosis indexes (HA, LN, PCIII, IV-C), LSM and anteroposterior diameter of main portal vein in the combination group were significantly better than those in the monotherapy group, with statistically significant differences ($P < 0.05$). Follow-up of endpoint events showed that the cumulative incidence of clinical endpoint events in the combination group was significantly lower than that in the monotherapy group ($P < 0.05$). COX multivariate regression analysis suggested that high platelet level was a protective factor for HCC in such populations (HR=0.977, 95% CI: 0.962-0.994, $P=0.006$).

Conclusions:

Fuzheng Huayu Capsule combined with antiviral drugs can more effectively improve the degree of liver fibrosis and liver stiffness in high-risk populations of HBV-related HCC, and reduce the incidence of endpoint events such as decompensated liver cirrhosis, which has important clinical promotion value.

Keyword(s) : Fuzheng Huayu Capsule; Nucleos(t)ide Analogues; Hepatitis B Virus; High-risk Population of Hepatocellular Carcinoma; Liver Fibrosis; Decompensated Liver Cirrhosis

2099 - Clinical Analysis of Early Renal Injury in HBV Infected Patients Treated with Nucleos(t)ide Analogues

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Background / Hypothesis: This study aimed to

investigate the incidence, risk factors, and intervention effects of early renal injury in hepatitis B virus (HBV)-infected patients treated with nucleos(t)ide analogues (NAs), so as to provide a theoretical basis for clinical diagnosis and treatment.

Materials and Methods: A total of 447 eligible HBV-infected patients from the Department of Infectious Diseases, Yan'an University Affiliated Hospital, between January 2023 and June 2025 were enrolled. All patients had received NAs for at least 1 year with complete clinical data, and those with concurrent other viral infections, primary kidney diseases, hypertension, diabetes mellitus, etc., were excluded. Multiple renal function indicators including serum creatinine (Scr), estimated glomerular filtration rate (eGFR), and urinary $\beta 2$ -microglobulin (urinary $\beta 2$ -MG) were detected to calculate the abnormal detection rate, analyze risk factors, and compare indicator changes between patients with adjusted treatment regimens (renal protection therapy or drug replacement) and those with maintained original regimens.

Results: The results showed that 68.90% of the patients had at least one abnormal renal function indicator. The abnormal rate of urinary $\beta 2$ -MG was the highest (45.41%), and the abnormal rate of urinary microalbumin/creatinine (mAlb/Ucr) was 21.03%. Compared with the monotherapy group, the combined therapy group had a higher abnormal rate of urinary $\beta 2$ -MG (61.11% vs 43.26%, $P=0.013$). Among the monotherapy group, patients treated with tenofovir disoproxil fumarate (TDF) had significantly higher abnormal rates of urinary $\beta 2$ -MG and serum phosphorus than those treated with entecavir (ETV), tenofovir alafenamide fumarate (TAF), and tenofovir lamivudine (TMF) ($P < 0.05$). Logistic regression analysis indicated that age (OR=1.041) and monotherapy with TDF (OR=2.085) were independent risk factors for early renal injury ($P < 0.05$). Regarding the intervention effect, the levels of urinary $\beta 2$ -MG, mAlb, and mAlb/Ucr in the regimen adjustment group decreased significantly after treatment, with normal rates reaching 56.0%, 69.57%, and 51.61% respectively, and the decline rates all exceeding 80%, which were significantly different from those in the maintenance treatment group ($P < 0.05$).

Conclusions: In conclusion, the incidence of early renal injury in HBV-infected patients treated with NAs is relatively high, and advanced age and monotherapy with TDF are the main risk factors. Urinary $\beta 2$ -MG, mAlb, and mAlb/Ucr can be used as sensitive indicators for early screening. Timely adjustment of the treatment

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regimen can effectively improve the indicators related to renal injury. Clinically, attention should be paid to the monitoring of renal function and individualized intervention in such patients to improve the safety of treatment and the quality of life of patients.

Keyword(s) : HBV-infected individual, Urinary β 2-microglobulin, Early renal injury

2101 - A Non invasive Diagnostic Prediction Model for AMA Negative Patients with Primary Biliary Cholangitis

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Background / Hypothesis:

Approximately 5-10% of patients with primary biliary cholangitis (PBC) are anti-mitochondrial antibody (AMA)-negative, posing a diagnostic challenge. This study aimed to develop a non-invasive diagnostic prediction model for this subgroup.

Materials and Methods:

This retrospective study included patients who underwent liver biopsy were diagnosed with PBC at Beijing Ditan Hospital as shown in Figure 1. Patients were stratified into two groups: AMA-negative and AMA-positive groups. Clinical characteristics were compared, and logistic regression was used to identify independent predictors for AMA-negative status. A nomogram model was used for diagnostic prediction of AMA-negative patients with PBC.

Results:

A total of 218 PSVD patients undergoing liver biopsy were enrolled, including AMA-negative (n=66) and AMA-positive (n=152) groups. Significant differences were observed between the groups. The AMA-negative group demonstrated a notably higher median gamma-glutamyl transferase (GGT) level (167.55 U/L vs. 127.60 U/L, $p=0.044$) but lower levels of Immunoglobulin G (IgG) (13.75 g/L vs. 15.35 g/L, $p=0.008$) and IgM (2.08 g/L vs. 2.33 g/L, $p=0.049$). Serologically, AMA-negative patients had a higher antinuclear antibody (ANA) positive rate (78.79% vs. 64.47%, $p=0.036$) and a higher prevalence of anti-SP100 antibodies (30.30% vs. 17.76%, $p=0.039$), while anti-gp210 antibodies were more common in the AMA-positive group (46.05% vs. 30.30%, $p=0.030$). Multivariate analysis identified four independent predictors for AMA-negative PBC: elevated GGT (OR=0.999, 95% CI: 0.997-1.000, $p=0.027$), lower IgG (OR=1.132, 95% CI: 1.037-1.235,

$p=0.006$), ANA positivity (OR=0.419, 95% CI: 0.205-0.858, $p=0.017$), and higher prothrombin activity (PTA) (OR=1.040, 95% CI: 1.014-1.065, $p=0.002$). The nomogram model incorporating these factors showed good diagnostic performance.

Conclusions:

A practical, non-invasive prediction model based on four routine clinical parameters (PTA, GGT, IgG, and ANA) was developed to aid in diagnosing AMA-negative PBC, potentially improving diagnostic accuracy for this challenging subgroup.

Keyword(s) : primary biliary cholangitis, anti-mitochondrial antibody, gamma-glutamyl transferase, Immunoglobulin G, nomogram model

2103 - Association of MR elastography parameters with prognostic indices neutrophil to lymphocyte ratio hepatocyte function and cholestasis parameters in chronic liver disease

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Background / Hypothesis: Magnetic Resonance Elastography (MRE) is a key non-invasive tool for staging fibrosis, yet its relationship with specific biochemical pathways may differ by etiology of chronic liver disease (1). Neutrophil-to-lymphocyte ratio (NLR) is a biomarker associated with systemic inflammation and could be used as a prognostic factor in chronic liver disease (2). We hypothesized that the determinants of liver stiffness and the utility of prognostic indices (MELD, NLR) diverge between hepatocellular (chronic viral hepatitis, CH) and cholestatic (primary biliary cholangitis, PBC) liver diseases, even when fibrosis burden is comparable.

Materials and Methods: This retrospective study included 80 patients: 54 with CH and 26 with PBC. The baseline characteristics are shown in Table-1. MRE stiffness values were categorized into fibrosis stages (F0-F4) using standard cut-offs. We assessed the correlation of stiffness with hepatocyte function, cholestasis parameters, and inflammatory markers (NLR). Multivariate linear regression and Receiver Operating Characteristic (ROC) analyses were performed to identify independent determinants of stiffness and predictors of high-risk disease (MELD >9).

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Results: There was no significant difference in mean liver stiffness (3.9 vs. 3.6 kPa; $p=0.29$) and the prevalence of advanced fibrosis ($\geq F3$) between CH (62.3%) and PBC (54.2%; $p=0.48$). As expected, the PBC group had significantly higher ALP levels ($p < 0.001$) and a higher proportion of female patients ($p = 0.009$). PBC patients exhibited higher total cholesterol ($p=0.006$) and LDL ($p=0.045$). Notably, a significant correlation was observed between liver stiffness and NLR in the CH group ($r=0.35$, $p=0.009$), whereas no such correlation existed in the PBC group ($p=0.63$). In multivariate analysis, stiffness in CH was independently associated with age ($p=0.003$) and AST ($p<0.001$), whereas in PBC, stiffness was predicted solely by AST ($p<0.001$) (Table 2). MELD 3.0 showed the strongest correlation with stiffness in the CH cohort ($r=0.51$). ROC analysis demonstrated that platelet count (AUC=0.84) and MRE (AUC=0.72) were the strongest independent predictors of a MELD score >9 .

Conclusions: While the stage of fibrosis is similarly distributed across etiologies, the drivers of liver stiffness differ fundamentally. In CH, stiffness is linked to systemic inflammation (NLR) and reflects both chronicity and active injury. In contrast, PBC stiffness is driven solely by enzymatic activity, with no link to systemic inflammatory markers.

Keyword(s) : Magnetic resonance elastography, liver stiffness, neutrophil-to-lymphocyte ratio, chronic viral hepatitis, primary biliary cholangitis

2106 - Disease burden of chronic hepatitis B among pregnant women increased dramatically with age – a territory wide study

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Background / Hypothesis:

An integrated model nurse clinic (IMNC) was established in Hong Kong as a pivotal part of the Hong Kong Hepatitis Action Plan 2020-2024 to achieve hepatitis elimination by 2030. IMNC has led to a major increase in HBsAg and HBV DNA testing in women of child-bearing age. This study evaluated the secular trends in diagnostic rates of chronic hepatitis B virus (HBV) infection in pregnant women who were born

before and after 1988, the start of the universal HBV vaccination to newborns in Hong Kong.

Materials and Methods:

Individuals with CHB were identified from a territory-wide database from year 2006 to 2024. We performed a subgroup analysis for pregnant women of age <30 or ≥ 30 .

Results:

A total of 840,281 pregnant women were identified; 61,107 individuals were diagnosed with chronic HBV infection. Newly diagnosed HBV rates increased over time in both age groups, with a much more dramatic increase observed in pregnant women born before the era of universal HBV vaccination. In contrast, the newly diagnosed HBV rates among pregnant women born in the era of universal HBV vaccination remained steadily low over time. The rates only showed a modest increase over time from 0.51% in 2018 to 1.03% in 2024 (age ≥ 30), and from 0.24% in 2006 to 0.14% in 2018 (younger than 30 years old) (Figure).

Conclusions: Newly diagnosed HBV rates showed substantial increases in pregnant women born before the era of universal HBV vaccination, while the rates remained steadily low in those born in the era of universal HBV vaccination. These findings provide important data for the allocation of resources in IMNC, with more targeted screening, education and pre-emptive antiviral therapy for pregnant women born before the era of universal HBV vaccination in order to minimise MTCT of HBV.

Founding: This work was supported by the Investigator Sponsored Research of Gilead Sciences (Reference: IN-HK-987-7181).

Keyword(s) : HBV, MTCT

2108 - Post-Liver Transplant Outcomes and Donor Safety in Bangladesh A Nationwide Questionnaire Based Observational Study

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Background / Hypothesis: Liver transplantation (LT) is increasingly accessible for Bangladeshi patients, though most undergo surgery abroad. Data on post-transplant outcomes and donor safety in Bangladesh remain limited. This study aimed to evaluate recipient outcomes, donor recovery, follow-up adherence, complications, and overall quality of life among post-LT patients across the country.

Materials and Methods: A cross-sectional observational study was conducted among post-liver transplant recipients in Bangladesh. Data were collected nationwide through a structured Google Form. Demographic details, transplant indications, donor profile, complications, adherence, and quality-of-life, outcomes were analyzed descriptively.

Results: Among the respondents, 97% underwent transplantation in India. The cost of LT ranged from 20,00,000 to 80,00,000 Bangladeshi taka. Most of the respondents (52.94%) were in the pediatric age group. Mothers were the most common donors (32.4%), followed by close relatives (20.6%) and fathers (17.6%). All donors survived after donation and maintained good health; most (70.6%) recovered within 2 months, and the rest (29.4%) recovered within 6 months.

The leading indications for transplantation were biliary atresia (47.1%) and HBV-related cirrhosis (23.5%). After transplant, 64.7% of recipients showed complete adherence to follow-up, while 2.9% were completely lost to follow-up. Regarding access to care, 17.6% reported no post-transplant problems, whereas others faced challenges obtaining timely treatment when ill (18%) or accessing immunosuppressive medications and tacrolimus levels (20%).

Overall, 97% reported a good quality of life after transplant. Post-transplant complications included no complications in 47.1%, infections in 23.5%, and biliary leakage in 11.8%. New-onset medical issues following immunosuppression included hypertension (8.8%), diabetes mellitus (5.9%), and chronic kidney disease (2.9%). All patients were alive at follow-up except one (mortality 2.9%).

Conclusions: This nationwide survey demonstrates excellent post-transplant survival, high donor safety, and good quality of life among liver transplant recipients in Bangladesh. However, notable challenges remain in follow-up adherence and access to immunosuppressive medication and monitoring. Strengthening local transplant support systems could further improve outcome.

Keyword(s) : LT – Liver Transplant, HBV – Hepatitis B Virus

2109 - Soluble CD14 Predicts Prognosis Diuretic Response and Acute Kidney Injury in Decompensated Cirrhosis with Refractory Water Retention

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Background / Hypothesis: In decompensated liver cirrhosis, refractory water retention is frequently accompanied by acute kidney injury (AKI) and poor prognosis, in which bacterial translocation-driven systemic inflammation is thought to play a central role. Soluble CD14 (sCD14) is a circulating marker of bacterial translocation, but its clinical utility in this setting remains unclear. This study aimed to clarify the prognostic value of sCD14 in patients with decompensated cirrhosis and water retention, and to determine its association with diuretic responsiveness, AKI development, liver-related mortality, and spontaneous bacterial peritonitis (SBP).

Materials and Methods: A total of 134 patients with decompensated cirrhosis and refractory water retention treated with tolvaptan were retrospectively enrolled. Baseline serum sCD14 levels were measured, and their relationships with short- and long-term diuretic response, AKI development, and liver-related mortality were evaluated. In a subset analysis excluding patients with advanced hepatocellular carcinoma, the prognostic performance of sCD14 was compared with other bacterial translocation markers (soluble CD163 and mannose receptor). The cumulative incidence of SBP was also assessed according to sCD14 levels using competing-risk analysis.

Results: Lower sCD14 levels were significantly associated with better short- and long-term diuretic responses and a lower incidence of AKI. Patients with sCD14 ≤ 2340 pg/mL had significantly longer liver-related survival than those with higher levels (median



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survival: 24.5 vs. 4.8 months, $p < 0.01$). On multivariate analysis, sCD14 > 2340 pg/mL independently predicted liver-related mortality (hazard ratio 2.71, $p < 0.01$). sCD14 showed superior prognostic value compared with soluble CD163 and mannose receptor. Concomitant rifaximin use was significantly associated with lower sCD14 levels. The cumulative incidence of SBP was numerically higher in patients with high sCD14 than in those with low sCD14; however, this difference did not reach statistical significance ($p = 0.18$). Conclusions: sCD14 is a promising biomarker for predicting prognosis, diuretic response, and AKI development in patients with decompensated cirrhosis and water retention, outperforming other bacterial translocation markers. The association between rifaximin use and lower sCD14 levels suggests a potential suppressive effect of rifaximin on bacterial translocation.

Keyword(s) : Decompensated cirrhosis ,Soluble CD14 ,Diuretic response ,Acute kidney injury

2111 - Prevalence of Liver Enzyme Abnormalities in PCR Confirmed Respiratory Viral Infections A 321 Patient Cohort

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Background / Hypothesis:

Respiratory viral infections may extend beyond the respiratory tract and lead to transient hepatic involvement, manifested by elevated liver enzymes. The aim of this study was to evaluate the prevalence and patterns of liver function test (LFT) abnormalities in a large single-center cohort of patients with PCR-confirmed respiratory viral infections during 2025.

Materials and Methods:

We conducted a retrospective review of all patients with a positive multiplex respiratory viral PCR between January 1 and December 1, 2025 at our tertiary center. A total of 321 patients had simultaneous ALT and AST measurements and were included. LFT abnormality was defined as ALT or AST $> 1.5 \times$ upper limit of normal (ULN), to avoid overestimating mild physiological fluctuations and to ensure clinically meaningful elevations were captured. Viral etiologies included SARS-CoV-2; Influenza A and B (with subtypes H3, H1N1); Adenovirus; Rhinovirus; RSV A/V; Parainfluenza 1-4; seasonal human coronaviruses (229E, OC43, NL63, HKU1); Bocavirus; and Metapneumovirus. For each virus, the proportion of patients with LFT elevation was

calculated.

Results:

A total of 321 patients with PCR-confirmed respiratory viral infections were included. Overall, 84 patients (26.1%) showed elevated ALT and/or AST levels. Median ALT was 41 IU/L, and median AST was 38 IU/L. Severe elevation ($> 5 \times$ ULN) occurred in 4 patients (1.2%). The highest proportions of LFT abnormalities were observed in Adenovirus (32%) and SARS-CoV-2 (28%) infections. RSV and Influenza B were associated with LFT elevation in 25% and 18% of cases, respectively. Lower rates were found in Rhinovirus (14%), seasonal coronaviruses (12%), Parainfluenza viruses (12%), and Bocavirus (6%). Differences between high-risk viruses (Adenovirus, SARS-CoV-2) and lower-risk viruses (Rhinovirus, Parainfluenza) were statistically significant ($p < 0.05$).

Conclusions:

Mild to moderate LFT abnormalities were found in approximately one quarter of patients with PCR-confirmed respiratory viral infections in 2025. Adenovirus and SARS-CoV-2 infections exhibited the highest rates of hepatic involvement, while other viruses showed lower but non-negligible rates. Awareness of this pattern may help clinicians interpret transient LFT elevations in the context of recent respiratory viral infections and potentially avoid unnecessary extensive hepatic work-ups.

Keyword(s) : Respiratory viral infections, liver function tests, transaminase elevation, adenovirus, SARS-CoV-2

2112 - Core genes with opposite expression changes and their inflammation related regulatory network during the progression and regression of NASH liver fibrosis

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Background and aims

Liver fibrosis associated with nonalcoholic steatohepatitis (NASH) is a major cause of end-stage liver disease and liver transplantation. Although fibrosis is reversible in a subset of patients, its molecular basis remains poorly understood. We compared transcriptional profiles of progressive and regressive NASH liver fibrosis to identify genes with opposite expression changes between these states and to

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characterize their functional pathways, upstream regulation and intercellular communication features.

Methods

We performed differential expression analyses on a NASH fibrosis progression dataset (GSE135251) and a regression dataset (GSE83452). An intersection strategy was used to screen profibrotic genes upregulated during progression but downregulated during regression, and antifibrotic genes showing the opposite pattern. Functional enrichment, transcription factor prediction and single-cell transcriptomic analyses were integrated to assess pathways and changes in cell-cell communication mediated by key secreted proteins.

Results

We identified differentially expressed genes in both cohorts and, by cross-cohort analysis, defined 13 core profibrotic genes (ACSL4, ANXA2, CCL2, CXCL10, CXCL8, DUSP8, FAT1, GDF15, IL32, PRAMEF10, SAA1, SPP1 and THBS1) that were consistently upregulated in progression but downregulated in regression, as well as one antifibrotic lncRNA, JAKMIP2-AS1, with the opposite pattern. Expression of this 13-gene set increased stepwise with advancing fibrosis stage in the progression cohort but was globally suppressed in the regression cohort, indicating a close association with disease severity and reversibility. Functional enrichment showed inflammatory cell chemotaxis and migration, cytokine/chemokine signaling, PDGF-related pathways and ATF4/PERK-mediated endoplasmic reticulum stress, suggesting a profibrotic signaling module driven by inflammation and stress. Transcription factor analysis indicated that CEBPA/CEBPB, IRF3, XBP1 and ATF4 regulate this gene set and co-target chemokines and profibrotic factors, linking innate immune activation and endoplasmic reticulum stress to inflammatory cell recruitment and fibrosis progression. Single-cell transcriptomic analysis showed that, from healthy liver through metabolic dysfunction-associated steatotic liver disease and steatohepatitis to cirrhosis and end-stage liver disease, expression of this core gene set gradually increased across major hepatic cell populations. Cell-cell communication analysis focusing on secreted proteins revealed progressive strengthening of profibrotic ligand-receptor signaling, such as THBS1-CD47, SPP1-CD44 and CXCL8-CXCR2.

Conclusions

We identified a core profibrotic gene module that participates in both progression and regression of

NASH liver fibrosis, involving inflammatory cell chemotaxis, cytokine signaling, fibroblast activation and endoplasmic reticulum stress. These genes and their associated intercellular communication signals reflect disease severity and may influence fibrosis reversibility, providing insights into mechanisms of NASH fibrosis regression and potential antifibrotic targets.

Keyword(s) :

2114 - Targeted Activation of Lipophagy Attenuates MASH Progression in a Murine Model

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Background / Hypothesis:

Background

Metabolic dysfunction-associated steatohepatitis (MASH), used to be called nonalcoholic steatohepatitis (NASH), is a progressive liver disease that affects adults worldwide. MASH is a major cause of chronic liver disease, characterized by excessive lipid accumulation, inflammation, and cellular stress that progressively impair liver function. MASH has become increasingly prevalent due to the global rise in obesity and metabolic syndrome.

Hypothesis

Targeted activation of lipophagy—selective autophagic degradation of lipid droplets—may alleviate these pathological processes by reducing lipid burden and improving cellular homeostasis.

Materials and Methods:

RNA sequencing dataset GSE185911 were downloaded from NCBI database and reanalyzed to compare gene expression between lipophagy-induced (LIR-treated) and control groups through R packages. The differentially expressed genes (DEGs) were identified by FDR (false discovery rate) <0.05 and |log₂ fold change| > 0.25. Gene Ontology (GO) enrichment and pathway analyses were performed to classify DEGs into biological processes, cellular components, molecular functions, and metabolic pathways relevant to MASH pathology.



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Results:

Lipophagy activation produced marked gene expression shifts in murine model. Upregulated DEGs included chaperone proteins Hspa5 and Dnajb9, which facilitate ER stress reduction, and metabolic regulators Manf, Lpin1, and Ass1, which enhance lipid processing and restore metabolic balance. Pro-inflammatory genes such as Socs3 and Anxa2 were downregulated, indicating a potential anti-inflammatory effect. Pathway analysis demonstrated that lipophagy significantly influences ER stress responses, lipid metabolic process and regulation, response to peptide hormone and cellular stress adaptation.

Conclusions:

This study reveals that targeted lipophagy activation modulates critical molecular pathways involved in MASH progression. The observed improvements in ER stress regulation, lipid metabolism, and inflammatory signaling support lipophagy as a promising therapeutic strategy. These findings provide a foundation for future investigations into the clinical translation of lipophagy-based interventions for MASH.

Keyword(s) : MASH, Lipid, Autophagy

2115 - Determinants of severe liver fibrosis in patients with MASLD a five year retrospective cross sectional study

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease globally, with prevalence rising over recent decades. Advanced fibrosis ($\geq F3$) is strongly linked to liver-related complications and mortality. While obesity, poor glycemic control, dyslipidemia, and elevated liver enzymes are established predictors, data on additional factors such as comorbidities and smoking history remain limited. This study aimed to identify independent risk factors for severe fibrosis and cirrhosis in MASLD patients.

Materials and Methods: In this retrospective cross-sectional study, 2,082 transient elastography (TE) results were reviewed. Among 1,112 patients with fatty liver on imaging, 718 with MASLD were included.

Demographic data, metabolic parameters, comorbidities, biochemical tests, and noninvasive fibrosis scores were extracted. Logistic regression was used to identify independent predictors of severe fibrosis (F3) and cirrhosis (F4) using F0-F1 as the reference.

Results: Of the 738 MASLD patients, 56 (7.6%) had severe fibrosis (F3) and 180 (24.4%) had cirrhosis (F4). Multivariate analysis revealed that independent predictors of F3 were BMI $>23 \text{ kg/m}^2$ (adjusted OR 5.16, 95% CI 2.25–11.85), HBA1c $\geq 7\%$ (adjusted OR 6.65, 95% CI 2.23–19.80), ALT $\geq 2 \times$ ULN (adjusted OR 5.55, 95% CI 2.05–15.04), HDL $<40 \text{ mg/dL}$ (adjusted OR 3.55, 95% CI 1.56–8.11), and AST/ALT ratio ≥ 1 (adjusted OR 2.12, 95% CI 1.05–4.29). For cirrhosis (F4), BMI $>23 \text{ kg/m}^2$ (adjusted OR 2.75, 95% CI 1.40–5.40), HBA1c $\geq 7\%$ (adjusted OR 1.65, 95% CI 1.05–2.60), HDL $<40 \text{ mg/dL}$ (adjusted OR 2.00, 95% CI 1.15–3.50), and AST/ALT ratio ≥ 1 (adjusted OR 2.50, 95% CI 1.10–5.70) remained independent risk factors. Other variables, including age, sex, FBS, LDL, triglycerides, comorbidities, DM duration, and smoking, were not independently associated with advanced fibrosis. Notably, a proportion of patients with F3 and F4 had BMI $<23 \text{ kg/m}^2$, showing the occurrence of lean MASLD with clinically significant fibrosis.

Conclusions: In MASLD patients, higher BMI, poor glycemic control, low HDL, and elevated AST/ALT ratio are independently associated with severe fibrosis and cirrhosis. Importantly, advanced fibrosis also occurs in lean individuals, showing the clinical significance of lean MASLD and the need to screen for fibrosis regardless of BMI. These findings support risk assessment beyond obesity and highlight the utility of transient elastography for early identification of high-risk MASLD patients.

Keyword(s) : MASLD, NAFLD, fatty liver, transient elastography, fibroscan, liver fibrosis, cirrhosis

2116 - Acute liver failure after transarterial embolization of hemorrhaging hepatocellular adenoma

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Significance: Hepatocellular adenoma (HCA) is typically managed with surgical resection for medium to large lesions. Recent guidelines recommend transarterial embolization (TAE) as the first-line intervention for

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bleeding HCAs presenting with hemodynamic instability. Although TAE is generally associated with low morbidity, serious complications, including hepatic failure, may still occur.

Clinical Presentation: We report the case of a 67-year-old diabetic male with biopsy-proven HCA who presented to the emergency room with sudden, severe right upper quadrant pain. On arrival, he was hypotensive (80/60 mmHg), tachycardic, and anemic (hemoglobin 9.8 mg/dL). Physical examination revealed peritonitis and a firm right upper quadrant mass.

Management: The patient underwent aggressive resuscitation with intravenous fluids and blood transfusion. CT imaging demonstrated multiple hepatic masses, including an 11.5 × 12.8 cm lesion in the right lobe and a 4.7 × 4.1 cm lesion in the left lobe, accompanied by hemoperitoneum. A diagnosis of hypovolemic shock secondary to ruptured HCA was made. Emergent angiography identified a hypervascular mass supplied by branches of the inferior pancreaticoduodenal artery, which were selectively catheterized and embolized. Several hours post-embolization, the patient developed worsening mental status, dyspnea, and hypotension. Chest radiography suggested acute respiratory distress syndrome, and liver enzymes were markedly elevated (SGPT 49,153.6 U/L; SGOT 33,480.9 U/L). Despite maximal resuscitative efforts, the patient died.

Recommendation: TAE is a minimally invasive and guideline-recommended treatment for ruptured HCAs, particularly among patients who are hemodynamically unstable. However, timely recognition of cases requiring more aggressive or adjunctive interventions is critical. Larger adenoma size, bilateral involvement, and signs of impending hepatic failure should prompt early reconsideration of surgical resection. Hepatic failure remains a serious but underrecognized complication of TAE. Existing criteria for predicting post-TAE hepatic failure are derived from limited data and lack prospective validation. Further studies are needed to better define risk factors and improve decision-making algorithms for managing ruptured HCAs.

Keyword(s) : hepatic adenoma, hepatocellular adenoma, ruptured hepatic adenoma, transarterial embolization, liver failure

2118 - Hepatitis B and C Prevalence Among Transgender Individuals and Men Who Have Sex

with Men in Pakistan

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Background / Hypothesis:

In Pakistan, Transgenders (TGs) and Men who Have Sex with Men (MSM) are marginalized communities at high risk of sexually and parenterally transmitted infectious diseases. The prevalence of hepatitis B virus (HBV) and hepatitis C virus (HCV) infection in these populations has not been well described in Pakistan, and access to care and treatment is lacking.

Materials and Methods:

From 2020-2022, community health workers tested 2241 transgenders (TGs) and men who have sex with men (MSM) for hepatitis B and C. Those with hepatitis B were referred to provincial programs, while those with hepatitis C were linked to "One Stop Shop" community-based organizations for same-day testing, treatment, and care, including HCV RNA testing and 12-24 weeks of Sofosbuvir and Daclatasvir treatment based on APRI scores.

Results:

A total of 201/2241 (9%) tested positive for anti-HCV, and 69/2241 (3.1%) tested positive for HBsAg. All participants who tested HBsAg+ were referred to the provincial hepatitis control program for additional care. The 201 who tested anti-HCV+ were referred to CBOs for additional testing and care under One Stop Shop model. Of those, 161 (80%) were tested for HCV RNA, of which 99 (62%) tested positive for HCV RNA. Of these 87 (88%) initiated treatment and 77/87 (89%) completed treatment; all were tested for cure (sustained virologic response; SVR), of which 72 (94%) were HCV RNA negative (cured).

Conclusions:

Outreach to marginalized populations with same day testing and referral for HBV and HCV is feasible and effective in Pakistan. Key populations were willing to be screened, tested and treated. Partnering with CBOs was key to the success in this pilot project in reaching "key" populations with limited access to health services and achieving high rates of linkage to care and adherence



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with treatment for HCV.

Keyword(s) : Key Populations, Transgender Individuals, Men who have sex with men, One Stop Shop Model, Community based Organizations, Hepatitis B, Hepatitis C, Evidence based Public Health Policy Formulation, Pakistan, Replicable and Sustainable Project Model in similar settings, Community Outreach, Community Mobilization

2119 - Introducing Hepatitis B Birth Dose Vaccination Within 24 Hours of Birth A Pilot Project in Pakistan

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Background / Hypothesis:

Pakistan has 5 million Hepatitis B cases (2.5% prevalence), mostly acquired at birth or early childhood. Chronic infection can lead to liver disease, cirrhosis, and cancer. Co-infection with Hepatitis Delta Virus worsens outcomes. Despite Hepatitis B vaccine introduction in 2002, nationwide birth dose implementation is lacking.

Materials and Methods:

This pilot project was undertaken at two tertiary care public sector birthing units of Islamabad. Each hospital was given the target of vaccinating all consecutive deliveries daily 24/7 till they reached a figure of 1000. The delivery staff was trained to fill the vaccination card and give the vaccine. The data about mother's demography was retrieved from the delivery register along with the time and date of delivery and administration of hepatitis B vaccine. The field supervisor collected the records and entered it in the electronic data file which was shared with the project director and any gaps found were addressed on daily basis.

Results:

A total of 2008 deliveries were recorded over 3 months in 2 Islamabad hospitals. 2000 newborns received Hepatitis B birth dose within 1 hour of delivery, with 8 initially missed due to staff misconceptions. No side effects or extra costs were reported. Delivery time and vaccination time showed strong correlation ($r=0.954$, $p<0.01$).

Conclusions:

The hepatitis B birth dose can be easily implemented throughout the country with little additional funding for its procurement and transportation while negligible funding is required for its implementation. Central procurement by the Federal Directorate of Immunization is the best way to procure a cheap and effective vaccine and improve not only the vaccination coverage but also prevent a large quantum of chronic hepatitis B and D and hepatocellular carcinoma and their associated high morbidity and mortality.

Keyword(s) : Hepatitis B, Birth Dose Vaccine, Evidence based results for Public Health Policy formulation, Implementation mechanism

2120 - GLP 1 receptor agonists associated with reduced risk of digestive cancers in patients with diabetes global real world evidence

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Background / Hypothesis:

The global incidence of digestive cancers is rising, with a disproportionately high prevalence found in individuals with type 2 diabetes mellitus (T2DM). We aimed to evaluate whether the use of glucagon-like peptide-1 receptor agonists (GLP-1 RAs) is associated with a reduced risk of major digestive cancers compared with other commonly used antidiabetic agents.

Materials and Methods:

We conducted a retrospective cohort study using the global TriNetX research network, including treatment-naïve patients with T2DM who initiated metformin and subsequently received a second-line antidiabetic agent

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between January 2005 and October 2024. Patients with any prior digestive malignancy were excluded. The index date was defined as the first prescription of a GLP-1 RA or other diabetic drugs, which included insulin, sulfonylureas, dipeptidyl peptidase-4 inhibitors (DPP-4 inhibitors), Sodium-Glucose Cotransporter 2 inhibitors (SGLT2 inhibitors), and thiazolidinediones (TZD), after at least 3 months of continuous metformin use. A 1-year washout period was applied. For each drug pair, 1:1 propensity score matching was based on more than 60 baseline covariates. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (Cis) for any digestive malignancy and site-specific cancers, with prespecified subgroup and sensitivity analyses.

Results:

A total of 668,409 eligible patients with T2DM were identified. After matching, 92,509 GLP-1 RA users were compared with insulin users, 75,005 with sulfonylurea users, 43,044 with DPP-4 inhibitor users, 47,742 with SGLT2 inhibitor users, and 9,428 with TZD users. GLP-1 RA therapy was associated with a significantly lower risk of digestive malignancy versus insulin (HR=0.25, 95% CI: 0.21–0.29), sulfonylureas (HR=0.59, 95% CI: 0.53–0.66), DPP-4 inhibitors (HR=0.68, 95% CI: 0.54–0.85), SGLT2 inhibitors (HR=0.60, 95% CI: 0.51–0.70), and TZD (HR=0.56, 95% CI: 0.40–0.78). These protective associations were especially observed for hepatocellular carcinoma (HCC), colorectal cancer (CRC), and pancreatic cancer (PA) across most comparisons. The reduced digestive cancer risk was found consistently across sex, age, BMI, and HbA1c strata, and robust to sensitivity analysis with alternative lag periods, intention-to-treat analyses, and stricter exposure definitions.

Conclusions:

Compared with other diabetic medications, GLP-1 RA use is significantly associated with lower risk of digestive cancer, particularly HCC, CRC and PA. More studies are needed to explore the chemopreventive effect of GLP-1 RA in digestive cancers.

Keyword(s) : Hepatocellular carcinoma, Diabetes, GLP-1 RA, DPP4i, SGLT2i, TZD, Insulin

2122 - Effect of a Porous Glass Membrane Emulsification Method on Radiological Response in Epirubicin-Lipiodol cTACE for HCC

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Background / Hypothesis: The therapeutic performance of conventional transarterial chemoembolization (cTACE) is influenced by the physical properties of the epirubicin-lipiodol emulsion, which vary substantially with manual mixing. A porous glass-membrane emulsification device was introduced to improve consistency of water-in-oil emulsions. This study evaluated its impact on radiological response and clinical outcomes in initial cTACE for hepatocellular carcinoma (HCC).

Materials and Methods: This single-centre retrospective cohort included consecutive patients undergoing first cTACE using epirubicin-lipiodol emulsions between 2017 and 2024. Emulsions were prepared either by a three-way stopcock (TS) or a glass-membrane device (GMD). Tumor response was assessed using mRECIST. Logistic regression and Cox models adjusted for operator, baseline liver function, and tumor distribution. Adverse events (AEs) were graded by CTCAE v5.0.

Results: Sixty-one eligible patients were analyzed (TS, n=29; GMD, n=32). GMD was associated with significantly higher target-lesion objective response (adjusted OR 3.66, p=0.033). Overall ORR and 6-month best overall response also favored GMD. For time-to-progression (mRECIST-defined), recurrence-free survival, and overall survival, GMD showed numerically favorable but non-significant trends (e.g., TTP HR 0.78). Early liver function change demonstrated a small mean worsening in the GMD group (adjusted Δ ALBI +0.13, p=0.035), and grade ≥ 3 hepatic enzyme elevation occurred more frequently in GMD (25.0% vs 10.3%), although without statistical significance (p=0.19). Overall AE profiles were otherwise comparable between groups.

Conclusions: Use of a glass-membrane emulsification device was associated with improved radiological tumor response in initial cTACE for HCC. While survival outcomes were similar between groups, early liver function decline and higher rates of grade ≥ 3 hepatic enzyme elevation were observed numerically in the

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GMD group, warranting careful interpretation. These findings suggest potential benefits and trade-offs associated with GMD-assisted emulsification, supporting further evaluation in larger or prospective studies.

Keyword(s) : hepatocellular carcinoma, transarterial chemoembolization, Porous Glass-Membrane Emulsification device

2123 - Efficacy and safety of glucocorticoids in multi etiology liver injury a single center real world retrospective cohort study

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Background / Hypothesis: The use of glucocorticoids (GCs) in liver injury is supported by limited real-world, comparative data across different etiologies, and how efficacy and safety vary by etiology and disease severity remains unclear.

Materials and Methods: In this single-center retrospective cohort, we included 450 inpatients with liver injury who received systemic GCs between 2020 and 2022. Patients were classified by primary etiology into drug-induced liver injury (DILI), viral hepatitis-related liver injury (VH), autoimmune hepatitis (AIH), and other causes, and further stratified by the presence of liver failure. Short-term (1-week) biochemical responses in alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin (TBIL), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) were assessed, along with long-term complete biochemical response (CBR; normalization of ALT, AST, and TBIL), all-cause mortality, and adverse events. Results: The median age was 51 years, 44.4% were male, and 141 patients (31.3%) had liver failure at baseline. DILI was the most frequent etiology (n = 197, 43.8%), comprising 164 cases of cholestatic DILI (83.2% of DILI) and 33 cases (16.8%) of drug-induced autoimmune-like hepatitis; VH accounted for 82 cases (18.2%), AIH for 58 (12.9%), and 113 patients (25.1%) were classified as other causes. At 1 week, a graded biochemical improvement pattern (AST > ALT > TBIL > ALP > GGT) was observed, with DILI—particularly drug-induced autoimmune-like hepatitis—showing the largest reductions in aminotransferases and TBIL. Over a median follow-up of 8 months, the overall CBR rate

was 37.33%, without significant differences among etiologic groups. Patients without liver failure achieved higher CBR rates than those with liver failure (40.45% vs 30.50%). Adverse events occurred in 9.8% of patients, were more frequent in the liver failure subgroup (22.0% vs 4.2%), and nearly all deaths and liver transplantations occurred among patients with liver failure. The initial GC dose showed no clear linear association with the magnitude of improvement in most biochemical indices.

Conclusions: In this real-world, multi-etiology inpatient cohort, systemic GCs produced clear short-term biochemical improvement, with approximately one-third of patients achieving CBR during follow-up. Efficacy and safety were strongly modified by liver failure status, underscoring the need to individualize GC therapy according to hepatic functional reserve and overall disease severity.

Keyword(s) : Glucocorticoids; Liver injury; Drug-induced liver injury; Viral hepatitis-related liver injury; Autoimmune hepatitis; Liver failure; Real-world study

2126 - Pemphigus vulgaris in immunotherapy of hepatocellular carcinoma and response to treatment

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Background / Hypothesis:

Hepatocellular carcinoma is the sixth most common tumor worldwide, with increased prevalence in Egypt due to the high incidence HCV infection. Bevacizumab is a monoclonal antibody that target vascular endothelial growth factor that inhibit angiogenesis and tumor growth and Atezolizumab is programmed cell death protein 1 (PD-1)/programmed cell death ligand 1 (PD-L1) inhibitors (immune checkpoint inhibitors) and they are treatment option for patients with advanced HCC.

Materials and Methods:

Among 267 patients received atezolizumab and bevacizumab in national liver institute, Egypt, male patient 63 years old with HCV infection treated with direct acting antiviral drugs in 2019.

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He was diabetic and was on oral hypoglycemic drugs for 10 years then shifted to insulin 3 years ago and hypertensive on amlodipine 10 mg per day presented to us with hepatocellular carcinoma infiltrative focal lesion seg VII about 4.6*6.9 cm with bi-lobar satellites and PVT diagnosed by contrast enhanced imaging.

Alpha feto protein was 3.03 ng/ml.

Immunotherapy (atezolizumab/bevacizumab) was started and after 8 cycles there was a complete resolution of hepatic focal lesions and tumoral thrombus of portal vein.

After the 11th cycle the patient suffered from rash all over the body with bullous formation.

Dermatologists prescribed antibiotics and antihistaminic, but the condition worsened after the next cycle. so, the dermatologist decided to take skin biopsy from the lesion which is diagnosed as bullous pemphigoid, oral corticosteroids 60 mg per day and then immunosuppressive drugs in form of azathioprine 50 mg twice daily were taken and the lesions almost disappeared completely after 2 weeks and immunotherapy was stopped since the corticosteroids added.

The patient is on follow up now from 6 months after stopping immunotherapy and two sequential contrast imaging showed sustained complete response with no enhanced hepatic focal lesions.

Results:

Bullous pemphigoid constitutes a rare dermatological immune-related adverse event of PD-1/PDL-I inhibitors. A delayed onset of bullous pemphigoid after cumulative doses of immunotherapy as well as refractory pruritic eruption prior to blister development were observed.

So, this patient has complete response to immune check point inhibitors and response was sustained 6 months till now after stopping the drug.

Conclusions: However, bullous pemphigoids can suspect good response to treatment or not as well as hand and foot syndrome in tyrosine kinase inhibitors, but we need more studies.

Keyword(s) : bullous pemphigoid, HCC, immunotherapy, PD-1/PDL-I inhibitors.

2127 - Association of copper exposure with fatty liver progression and mediation by perturbation of metabolic pathways

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Background / Hypothesis:

Exposure to copper has been associated with fatty liver development and progression, but the detailed molecular mechanisms remain largely unknown.

Materials and Methods:

Based on the Kailuan Cohort, copper was measured in whole blood of 241 participants during follow-up from 2016 to 2017. Untargeted metabolomics profiling was conducted in serum. The degree of fatty liver was evaluated using ultrasonography. Differential metabolites were selected by OPLS-DA VIP>1 and T test p value<0.05 for high blood copper vs. low blood copper and fatty liver progression vs. no progression. A meet-in-the-middle strategy was to identify the overlapping differential metabolites that potentially bridged the association between copper exposure and fatty liver progression.

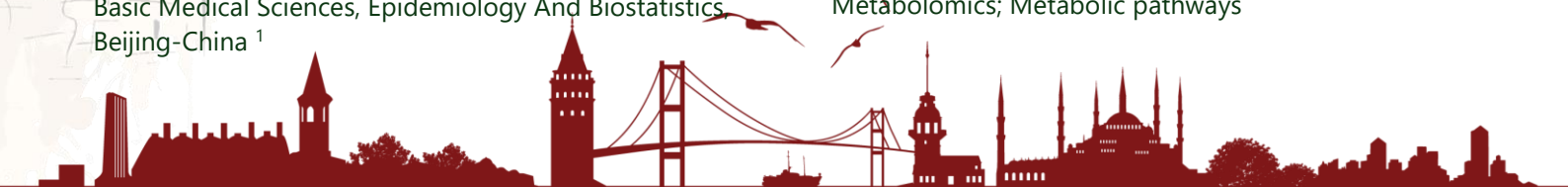
Results:

For high blood copper vs. low blood copper, 39 differential metabolites were found, including 34 lipids and lipid-like molecules, one organic oxygen compound, three organo-heterocyclic compounds, and one benzenoid. The above 39 differential metabolites were significantly enriched on six metabolic pathways. For fatty liver progression vs. no progression, 26 differential metabolites were revealed, including 9 lipids and lipid-like molecules, one organic oxygen compound, two organo-heterocyclic compounds, one organic nitrogen compounds, ten organic acids and derivatives, one nucleoside, nucleotide, and analogue, and two benzenoids. The above 26 differential metabolites were significantly enriched on five metabolic pathways. The overlapping differential metabolites were tauroursodeoxycholate (lower in high blood copper and lower in fatty liver progression) and 17-epi-17-J4-NeuroP (higher in high blood copper and higher in fatty liver progression).

Conclusions:

Exposure to copper may cause perturbation of metabolic pathways, including fatty acids and bile acids, which may lead to development and progression of fatty liver.

Keyword(s) : Copper; Fatty liver; Progression; Metabolomics; Metabolic pathways



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2128 - Triglycerides and high density lipoprotein cholesterol are primary lipid determinants of steatosis severity in metabolic dysfunction associated fatty liver disease

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Background / Hypothesis: Metabolic dysfunction-associated fatty liver disease (MAFLD) is closely linked to dyslipidemia, yet the individual and synergistic impacts of specific lipid abnormalities on hepatic steatosis severity remain unclear. We hypothesized that triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) are key determinants of steatosis severity, with potential threshold effects and metabolic context-dependency.

Materials and Methods: In this cross-sectional study of 6,810 participants (2,177 with MAFLD), hepatic steatosis was quantified via transient elastography (CAP). Multivariable ordinal logistic regression was used to assess independent and synergistic associations of TG, HDL-C, and LDL-C with steatosis severity. Nonlinear relationships were examined using restricted cubic splines, and interactions with metabolic factors were evaluated.

Results: Elevated TG (adjusted OR: 1.77, 95% CI: 1.48–2.11) and reduced HDL-C (OR: 1.35, 95% CI: 1.11–1.65) were independently associated with advanced steatosis, with the strongest association observed for their combination (OR: 2.40, 95% CI: 1.87–3.07). A nonlinear threshold effect for TG was identified at 1.51 mmol/L, with a steeper increase in steatosis below this level. The association between low HDL-C and steatosis was confined to males, younger adults, and metabolically healthier subgroups.

Conclusions: TG and HDL-C are primary lipid drivers of hepatic steatosis severity in MAFLD, with synergistic effects and metabolic interactions further stratifying risk. These findings support targeted lipid management for precision prevention and treatment of MAFLD.

Keyword(s) : Triglycerides, HDL cholesterol, hepatic steatosis, MAFLD, dyslipidemia

2129 - Bile Duct Reduction Without Ductular Reaction A Key Pathological Feature of Alagille Syndrome

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Background / Hypothesis:

Alagille syndrome (ALGS; OMIM 118450) is an autosomal dominant genetic disorder caused by JAG1 or NOTCH2 mutations, affecting multiple organs and systems, including the liver, heart, facial features, skeleton, and eyes. Liver manifestations often include neonatal liver disease, direct hyperbilirubinemia, chronic cholestasis, and bile duct abnormalities. This study aimed to investigate the pathological relationship between bile duct reduction and the absence of ductular reaction in four cases of ALGS.

Materials and Methods:

Four patients diagnosed with ALGS were included at the Second Hospital of Nanjing between January 2018 and December 2022. The demographic information, laboratory results, and histopathological findings. The diagnosis was confirmed based on clinical features and JAG1 genetic mutations.

Results:

Four patients (three males and one female, aged 12–35 years) presented with chronic cholestasis. Among the four cases studied, 25% (1/4) of the patients developed cirrhosis, characterized by pseudolobular nodules of varying sizes. Another 25% (1/4) showed signs of early cirrhosis and non-idiopathic portal hypertension, including minor dilation of portal veins in the portal areas. Additionally, 25% (1/4) exhibited intralobular hepatocellular edema and necrosis. Interface

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inflammation was observed in 50% (2/4) of the cases, while 75% (3/4) presented with portal area enlargement and bile duct defects. Histological analysis revealed a significant reduction in bile ducts, with fewer than 50% of portal tracts containing bile ducts. Despite cholestasis and liver injury, no ductular reaction was observed, suggesting impaired bile duct regeneration due to JAG1 mutation-associated NOTCH signaling dysfunction. Histopathological findings also demonstrated fibrosis and other compensatory changes.

Conclusions: Bile duct reduction and the absence of ductular reaction are important pathological features in ALGS. These findings highlight the critical role of JAG1 mutations in NOTCH signaling dysfunction and bile duct developmental defects. Recognizing these features is essential for accurate diagnosis and effective management of ALGS.

Keyword(s) : Alagille Syndrome; Ductular Reaction

2131 - PROGNOSTIC VALUE OF SERUM AMMONIA FOR SHORT TERM MORTALITY IN CIRRHOSIS A SYSTEMATIC REVIEW

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OBJECTIVE/BACKGROUND

The role of serum ammonia in assessing the progression of hepatic encephalopathy (HE) in liver disease patients has been a subject of ongoing debate. However, its prognostic utility for short-term mortality in adult cirrhotic patients remains inconclusive. Given that acute-on-chronic liver failure (ACLF), intensive care unit (ICU) admissions, and outpatient decompensation all occur within the context of cirrhosis, this review aimed to evaluate whether baseline or serial serum ammonia levels predict short-term mortality in cirrhotic adults across diverse clinical settings.

METHOD

A systematic search for studies published between January 2020 and December 2024 was conducted. Eligible studies included adults with confirmed cirrhosis (including those meeting criteria for ACLF), reported serum ammonia measurements (arterial or venous), and documented short-term mortality outcomes such

as 28-day, 30-day, or in-hospital mortality. Seven studies met the inclusion criteria for qualitative synthesis. Effect sizes were extracted as hazard ratios (HR) or odds ratios (OR). Studies with complete confidence intervals (CI) were included in a pooled random-effects meta-analysis. Heterogeneity was assessed using the I^2 statistic.

RESULT

Seven studies (n=3,716 cirrhotic patients) were included in the analysis. Across all individual studies, elevated serum ammonia levels consistently correlated with increased short-term mortality. A pooled analysis of three studies with complete HR data indicated a trend toward increased mortality risk with rising ammonia levels (pooled HR 1.08; 95% CI 0.99–1.18; $I^2=89\%$). Two studies with OR data demonstrated a directionally similar association (pooled OR 2.88; 95% CI 0.42–19.88; $I^2=97\%$). The precision of these findings was limited by variations in study design, ammonia measurement methodologies, and patient populations (ACLF, ICU, and outpatient cirrhosis). Two additional high-quality ACLF studies reported independent associations between ammonia and 28-day mortality but were excluded from quantitative pooling due to missing CI.

CONCLUSION

Across ACLF, ICU, and outpatient cohorts, all comprising adults with cirrhosis, higher serum ammonia levels were associated with poorer short-term outcomes. While heterogeneity and incomplete reporting constrained statistical precision, the consistent direction of the observed effect suggests that serum ammonia may serve as a clinically significant prognostic biomarker for short-term mortality in cirrhosis. Harmonized ammonia measurement techniques and standardized reporting are essential to enhance prognostic modeling in this area.

Keyword(s) : Ammonia Cirrhosis Acute-on-chronic liver failure Mortality Prognosis

2133 - Hepatitis (B C & D) and HIV Seroprevalence Survey in Sindh Province Pakistan

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² Who Country Office, Public Health, Islamabad-Pakistan ³ Phrc, Data Analysis, Karachi-Pakistan ⁴ Sindh Health Department, Cdc, Sindh, Karachi-Pakistan ⁵

Background / Hypothesis:

Pakistan is facing with one of the highest hepatitis C disease burden globally, necessitating urgent public health action. The first national hepatitis survey was carried out in 2008 followed by a provincial (second) survey in Punjab in 2018. In 2019, the third survey was carried out by World Health Organization and Department of Health Sindh to assess the latest Hepatitis B, C, D and HIV seroprevalence in Sindh province.

The survey aimed to find the latest prevalence of hepatitis B C D and HIV in Sindh province that should be statistically representative of the whole province.

Materials and Methods:

A stratified two-stage sample design was used to collect blood samples from 6,672 individuals across all the 29 districts of Sindh Province. Samples were tested at the hepatitis control program laboratory, and data were analysed using Epi Info and SPSS version 19.0.

Results:

A total of 1153 households from 29 districts of Sindh province were visited. There were 7133 household members with 6769 (94.9%) individuals present at the time of survey (average 5.8 persons/house). Refusal was seen in 97 people (1.4%). There were 3214 (47.5%) males, 3547 (52.4%) females and 08 transgenders (0.1%). HCV positivity was 6.1%, HBV 1.05%, HDV 32.8% and HIV 0.03%. Comparing the 2008 data with the 2019 data shows that the HCV disease prevalence has increased from previous figure of 5% to 6.1% while HBV prevalence has reduced from 2.5% to 1%.

Conclusions:

The survey findings highlighted the need for mass testing and treatment through a multisectoral, integrated approach to control hepatitis and HIV spread and prevent the disease progression. Recommendations include integration of healthcare services to save the resources, scaling up testing and treatment services, promoting awareness, and implementing targeted interventions. This survey provided valuable insights for policymakers and healthcare professionals working towards eliminating

hepatitis as a major public health threat in Pakistan.

Keyword(s) : HIV, Hepatitis B, Hepatitis C, Hepatitis D, Hepatitis A, Hepatitis E, Monitoring and Evaluation, Data Collection, Reporting, Monitoring and Evaluation, Prevalence, Sindh, Pakistan

2135 - Assessing HCV Re infection Rates and Linkage to Care among Transgenders and MSM in Pakistan A Follow up Study

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²

Background / Hypothesis:

Pakistan faces a significant hepatitis C epidemic, with limited data on prevalence among key populations like transgenders (TGs) and men who have sex with men (MSM). This study aimed to reassess HCV re-infection rates among these populations and provide linkage to care, building on a previous study conducted in 2022.

1. To estimate the re-infection rates of HCV in TGs and MSM in Pakistan
2. To provide linkage to care to the reinfected cases
3. To capture the data on World Health Organizations prescribed cascade of care

Materials and Methods:

A total of 123 TGs and MSM from the 2022 study were re-recruited in 2024. They were tested for anti-HCV, and positive cases underwent further testing. A One-Stop Shop model was established, providing screening, testing, and treatment services under one roof. Community-Based Organizations (CBOs) played a crucial role in recruiting participants and delivering services.

Results:

Out of 123 participants, 17 (13.8%) tested anti-HCV positive, and all were HCV RNA positive. Among these, 3 (17.6%) were previously not cured in the 2022 study, while 14 (82.4%) were newly detected. All 17 individuals initiated treatment, and 100% completed it. The One-Stop Shop model facilitated same-day testing, treatment initiation, and linkage to care.

Conclusions:

This study demonstrates the feasibility and

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effectiveness of outreach programs for marginalized populations in Pakistan. Partnering with CBOs was instrumental in accessing vulnerable communities and achieving high rates of linkage to care and treatment adherence. The One-Stop Shop model shows promise for scaling up HCV care and treatment services for key populations in Pakistan.

Community-based approaches, same-day testing, and treatment initiation can improve HCV care outcomes among marginalized populations. CBOs play a vital role in reaching and engaging these communities, highlighting the importance of collaborative efforts in public health initiatives.

Keyword(s) : Key Populations, Transgender Individuals, Men who have Sex with Men, Hepatitis, Prevalence, Pakistan

2136 - Implementing a Micro Elimination Program for Hepatitis C and Hepatitis B Diagnosis and Referral in Khaipur District Sindh Province Pakistan A Pilot Project for National HCV Elimination Initiative

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Background / Hypothesis:

Pakistan has a high burden of hepatitis C, with approximately 10 million people living with the infection. Two union councils (UCs) in Khaipur district, Sindh Province, Pakistan, with high disease rates, were selected for this pilot project aimed at implementing a micro-elimination program for hepatitis C and diagnosis and referral of hepatitis B.

Objectives:

The project objectives were to increase awareness, testing, diagnosis, and linkage to care and treatment for hepatitis B and C, and to pilot an Electronic Medical Record (EMR) system to track the hepatitis B referral and hepatitis C care cascade.

Materials and Methods:

The project targeted a population of 62,500 individuals, with 45,349 eligible for screening (aged 12+ years). Community healthcare workers implemented awareness campaigns, while trained Lady Health Workers (LHWs) screened individuals for hepatitis B

and C using rapid tests and collected demographic data using an EMR system.

Results:

As of June 2025, a total of 25,203 individuals were screened, with 541 (2.1%) HBsAg positive and 2,873 (11.4%) anti-HCV positive. Of the anti-HCV positive patients, 1,760 (61.3%) were tested for HCV RNA, and 1,075 (61.1%) were HCV RNA positive. All HCV RNA positive patients were initiated on treatment.

Conclusions:

This pilot project demonstrated the feasibility of implementing a hepatitis C elimination program in Pakistan, despite challenges such as internet connectivity issues and limited LHW coverage. The project successfully piloted an EMR system, which proved to be a valuable tool for tracking the hepatitis care cascade. The project findings helped understand on-ground challenges and revive the national HCV elimination strategy. The lessons learned from this project can inform province-wide and nationwide hepatitis elimination programs, contributing to Pakistan's goal of eliminating hepatitis C by 2030

Keyword(s) : Hepatitis B, Hepatitis C, Community Outreach, Community Mobilization, Awareness and Advocacy, Public Private Partnership, Micro elimination, Linkage to Care

2138 - Evaluation of the Elecsys HCV Duo Assay for Screening and Testing of Hepatitis C Virus in Pakistan

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Background / Hypothesis:

Pakistan has a high hepatitis C prevalence, with around 10 million people living with the infection, many unaware of their disease. Key populations like people who inject drugs (62% HCV prevalence) and homosexual persons (28.4% HCV prevalence) are disproportionately affected. Current screening involves two-step testing including anti-HCV and HCV RNA, but WHO recommends reflex testing to reduce patient loss.

Materials and Methods:



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From December 2023-November 2024, venous blood samples were collected from a total of 3612 persons that included 1532 cases from key populations including Transgenders (TGIs), People who Inject Drugs (PWIDs) and Men who Have Sex with Men (MSM) and 2080 from the general population seeking testing and treatment of hepatitis C at gastroenterology clinic (750 cases) and Sindh hepatitis control program treatment site (1330 cases). Blood was collected in two plasma preparation tubes, one for Duo assay (anti HCV and HCVcAg) and other for HCVRNA. Both tests were run on Roche instruments and results were compared. HCVRNA results were taken as final. Rapid anti HCV test was available prior to the Duo testing in patients from GI clinic and the hepatitis program /community but were not available in the high-risk populations.

Results:

Comparison of the known anti-HCV reactive rapid test results with the HCV Duo anti-HCV test results in 2080 patients showed 96% sensitivity. HCV Duo HCVcAg positivity compared with HCV RNA positivity revealed that HCVcAg was detected in only about 50% of cases across all three groups. Alternatively, if HCVcAg is reactive, there is a high chance that the virus is present, but if it is negative and anti-HCV is positive, the person might still be viraemic. The sensitivity of HCVcAg was low (38.6%-50.6%), but its specificity was high (>90%) in all groups. When an HCVcAg COI value of 10 and above is used as a cutoff to determine positivity, it detects almost 99% of cases with confirmed HCV RNA.

Conclusions:

The Duo anti-HCV test showed a sensitivity of 96% compared to rapid anti-HCV testing. However, the HCV core antigen (HCVcAg) test had a lower sensitivity of 50% compared to HCV RNA testing across all groups. It's worth noting that false positives from the rapid diagnostic test (RDT) could also be a factor. The HCV Duo test provides a workflow that requires PCR confirmation for samples with discordant results (Ab+/Ag-), making it suitable for the intended use case.

Keyword(s) : Hepatitis C, Linkage to Care, Duo Assay, Key Populations, High Risk Population, Cascade of Care

2139 - Multi Omics Biomarkers for Early Detection of Metabolic Dysfunction-Associated Fatty Liver Disease

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¹, Liujie Zhu¹, Chunrun Li¹, Shuo Huang¹, Jiawei Ran¹
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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a major cause of chronic liver disease worldwide. Traditional diagnostic tools lack sensitivity for early-stage disease. We hypothesized that circulating multi-omics biomarkers could improve early detection and risk stratification of MAFLD beyond conventional liver enzymes.

Materials and Methods:

We conducted a cross-sectional study enrolling individuals with metabolic risk factors. Clinical data, biochemical parameters, and plasma samples were collected. Targeted metabolomics and proteomics analyses were performed to identify candidate biomarkers associated with hepatic steatosis and metabolic dysfunction. Diagnostic performance was evaluated using receiver operating characteristic (ROC) analysis.

Results:

Several metabolites involved in lipid and amino acid metabolism, including branched-chain amino acids and bile acid derivatives, were significantly altered in MAFLD patients compared with controls. Proteomic analysis identified elevated levels of FGF21 and cytokeratin-18 fragments. A combined biomarker panel demonstrated superior diagnostic accuracy compared with ALT alone (AUC 0.87 vs. 0.63).

Conclusions:

Multi-omics-derived biomarkers offer a promising non-invasive strategy for early MAFLD detection. Integrative panels may facilitate timely intervention and personalized management in metabolically at-risk populations.

Keyword(s) : MAFLD; Biomarkers; Metabolomics; Proteomics; Early Diagnosis; Metabolic Dysfunction

2140 - A cost effective analysis of transportation of blood for HCVRNA using the plasma preparation tube vs the plasma separation card

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Background / Hypothesis:

Detection of viral RNA is crucial for hepatitis C diagnosis. Traditional plasma preparation tubes (PPT) require centrifugation and cold chain transportation, posing challenges in resource-limited settings. The Plasma Separation Card (PSC) offers a promising alternative, eliminating the need for centrifugation and cold chain.

Objectives:

To compare the cost of preparation and transportation of PSC vs PPT for HCV RNA detection and to compare viral load in both methods

Materials and Methods:

A study was conducted in Khairpur district, Sindh, Pakistan, involving 100 rapid diagnostic test-reactive anti-HCV individuals. Venous blood samples were collected in PPT and PSC, and costs were calculated for transportation and handling.

Results:

The overall cost of PSC was approximately 50% lower than PPT, mainly due to reduced costs for centrifugation, refrigeration, and transportation. The viral detection rate was excellent in both PPT and PSC, with no significant difference in HCV viral load. Direct spotting of venous blood onto PSC showed good correlation with plasma results.

Conclusions:

PSC is a cost-effective and efficient method for HCV RNA detection, particularly in resource-limited settings. The elimination of centrifugation and cold chain requirements makes it an attractive alternative to traditional PPT. The study's findings suggest that PSC can be a valuable tool for scaling up HCV diagnosis and treatment programs, especially in low-middle-income countries like Pakistan.

This study highlights the potential of PSC to improve access to HCV diagnosis and treatment in resource-constrained settings, contributing to global efforts to eliminate hepatitis C.

Keyword(s) : Hepatitis C, Linkage to Care, Plasma Separation Card (PSC), Key Populations, High Risk Population, Cascade of Care

2141 - Environmental Toxicity-Related Metabolic Signatures and Disease Severity in MAFLD

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Background / Hypothesis:

Toxic exposures and metabolic dysfunction synergistically contribute to MAFLD progression. We hypothesized that environmental toxin-related metabolic signatures are associated with disease severity in MAFLD patients.

Materials and Methods:

Adult MAFLD patients were stratified according to disease severity. Serum levels of environmental toxins and metabolic intermediates were measured using mass spectrometry. Associations with fibrosis indices were analyzed using multivariate regression models.

Results:

Higher levels of selected environmental toxins correlated with altered lipidomic profiles and increased fibrosis scores. Patients with advanced disease exhibited distinct metabolic signatures characterized by impaired fatty acid oxidation and mitochondrial dysfunction.

Conclusions:

Environmental toxicity may modulate metabolic pathways involved in MAFLD progression. Metabolic signatures reflecting toxin exposure could serve as novel indicators of disease severity.

Keyword(s) : MAFLD; Environmental Toxicity; Lipidomics; Fibrosis; Metabolic Signature

2142 - Artificial Intelligence-Assisted Ultrasound Imaging for Non Invasive Assessment of Liver Fibrosis

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Background / Hypothesis:

Artificial intelligence (AI) has emerged as a powerful tool in medical imaging. We hypothesized that AI-based models could improve non-invasive identification of liver fibrosis in chronic liver disease.

Materials and Methods:

Ultrasound images and clinical data from patients with

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chronic liver disease were retrospectively analyzed. A deep learning model was trained to predict fibrosis stages, with histology or elastography as reference standards.

Results:

The AI model demonstrated high accuracy in detecting significant fibrosis (AUC 0.89). Integration of clinical variables further improved model performance. The algorithm outperformed conventional scoring systems.

Conclusions:

AI-assisted imaging analysis represents a reliable and accessible approach for fibrosis assessment, with potential to enhance early diagnosis and clinical decision-making.

Keyword(s) : Artificial Intelligence; Liver Fibrosis; Ultrasound; Deep Learning; Non-Invasive Diagnosis

2144 - Beyond complete response of advanced hepatocellular carcinoma to immunotherapy what can we do

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Background / Hypothesis:

Tremelimumab is a monoclonal antibody that blocks cytotoxic T lymphocyte-associated antigen-4 (CTLA-4) and Durvalumab also a monoclonal antibody that blocks programmed cell-death ligand 1.

A single, priming dose of intravenous tremelimumab is used in combination with durvalumab, in a regimen known as STRIDE (Single Tremelimumab Regular Interval Durvalumab).

STRIDE is approved for the first-line treatment of adults with advanced/unresectable HCC in Europe and in the phase III HIMALAYA trial, STRIDE significantly improved overall survival compared with sorafenib in unresectable HCC and no prior systemic therapy, with manageable safety profile.

Materials and Methods:

Among 42 patients two male patients with advanced stage HCC received tremelimumab once and repeated doses of durvalumab every 28 days and achieved complete radiological response of HCC.

Male patient 58 years old diabetic on insulin from 3 years with HCV treated by direct acting antiviral drugs in 2019 presented to us by advanced stage HCC, Child-Pugh Score: A5, BCLC: B and performance statuses 0. contrast enhanced CT revealed infiltrative right lobe focal lesion about 10*12 cm with patent portal vein and alpha feto protein was 2800 ng/ml.

STRIDE regimen was started and only after three doses alpha feto protein dropped to 3.1 ng/ml and contrast enhanced CT showed complete resolution of hepatic focal lesion with no arterial enhancement.

The other male patient 71 years old hypertensive, HCV, not treated. Child-Pugh Score: A5, BCLC: C and performance statuses 0.

Contrast enhanced imaging revealed multiple right lobe focal lesions largest 3.8*3.4 cm with enlarged porto-caval lymph node 2*2.3 cm.

After six doses of STRIDE regimen contrast enhanced CT showed complete resolution of hepatic focal lesions and lymph node with no arterial enhancement.

Results:

In many trials and multicenter analysis of patients treated with immunotherapy, complete responders demonstrated prolonged overall and progression free survival even after discontinuation of therapy, But the duration of treatment and when to stop is still a question.

Also, further evaluation of immune cell PD-L1 expression and circulating tumor DNA as potential biomarkers is under investigations.

Despite using immunotherapy before a liver transplant can make transplant feasible for more advanced cases, but requires careful management due to risks like graft rejection, necessitating a washout period of at least 50 days (ideally 3 months) between the last treatment and transplant to significantly reduce rejection risk.

Conclusions:

After complete response to immunotherapy the background of cirrhosis in most cases with its possible complications and decompensation. still an obstacle stands against complete cure.

Keyword(s) : HCC, immunotherapy, PD-1/PDL-1 inhibitors, STRIDE regimen, complete response.

2146 - Inhibition of TGF β Signaling Attenuates Liver Fibrogenesis in an Experimental Cirrhosis Model

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Background / Hypothesis:



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Chronic liver injury leads to progressive fibrogenesis and ultimately cirrhosis, driven largely by activated hepatic stellate cells (HSCs) and profibrotic cytokines like transforming growth factor-beta (TGF- β). Currently, there are no approved antifibrotic drugs for cirrhosis, highlighting a critical need for targeted therapies. We hypothesized that pharmacological inhibition of the TGF- β signaling pathway would dampen HSC activation and collagen deposition, thereby attenuating liver fibrosis progression.

Materials and Methods:

A preclinical study was conducted using a carbon tetrachloride (CCl₄)-induced cirrhosis model in mice. A selective TGF- β receptor 1 kinase inhibitor (designated Compound XYZ) was administered intraperitoneally to treatment group mice (N=15) for 6 weeks, while control mice (N=15) received vehicle. Liver fibrosis was assessed by histological staining for collagen (Sirius Red quantification) and hydroxyproline content. HSC activation markers (α -SMA, collagen I mRNA) and TGF- β pathway activity (phospho-Smad3 levels) were measured via qRT-PCR and Western blot. Liver function tests and inflammatory cytokine levels were also monitored.

Results:

Compound XYZ was well tolerated and led to a marked reduction in fibrosis compared to controls. Treated mice showed a 45% decrease in Sirius Red-positive area and significantly lower hepatic hydroxyproline content ($p < 0.01$). Expression of α -SMA and collagen I in the liver was reduced by 50–60% in the treatment group, indicating suppressed HSC activation. Additionally, phospho-Smad3 levels were markedly diminished, confirming effective TGF- β pathway blockade. Treated mice exhibited improved liver function (ALT levels 30% lower than controls, $p < 0.05$) and reduced pro-inflammatory cytokine expression, suggesting overall amelioration of liver injury.

Conclusions:

Targeted inhibition of TGF- β signaling significantly attenuated experimental liver fibrosis, supporting TGF- β pathway blockers as promising antifibrotic agents. The results demonstrate that Compound XYZ effectively reduces HSC activation and collagen deposition, improving liver function in a cirrhosis model. These findings provide a strong rationale for further development of TGF- β inhibitors as potential therapies for patients with advanced fibrosis and cirrhosis.

Keyword(s) : fibrogenesis; cirrhosis; TGF- β ; hepatic stellate cells; antifibrotic therapy

2147 - Single Cell Transcriptomics Reveals Distinct Hepatic Stellate Cell Subpopulations Driving Liver Fibrogenesis

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Background / Hypothesis:

Liver fibrogenesis in cirrhosis arises from complex interactions among various hepatic cell types, with hepatic stellate cells (HSCs) as key collagen-producing cells. Emerging evidence suggests that HSCs are heterogeneous, but the specific subpopulations contributing to fibrosis progression remain poorly characterized. We hypothesized that single-cell transcriptomic profiling of fibrotic liver tissue would identify distinct HSC subpopulations, including a subset with a pronounced pro-fibrogenic gene expression profile that drives scar formation.

Materials and Methods:

Liver tissue samples from patients with advanced fibrosis/cirrhosis (Ishak fibrosis score ≥ 5 , $n=5$) and non-fibrotic control liver tissue ($n=3$) were subjected to single-cell RNA sequencing (scRNA-seq). Cell clustering and differential gene expression analysis were performed to define cell types and subpopulations, with a focus on HSCs. Key marker genes for each HSC cluster were identified, and immunofluorescence staining on liver sections was used to validate the spatial distribution of identified HSC subsets in situ.

Results:

scRNA-seq identified multiple discrete HSC subpopulations in cirrhotic livers. Notably, one HSC subset (Cluster A) was uniquely expanded in cirrhosis and exhibited high expression of canonical fibrogenic markers, including COL1A1, ACTA2 (α -SMA), and TGFB1, indicating a myofibroblastic phenotype. Another subset (Cluster B) expressed markers related to extracellular matrix remodeling (MMP2) and immune signaling (chemokines CXCL12/CXCL14), suggesting a regulatory role. This pro-fibrogenic Cluster A also expressed a novel surface marker (CDxx) not prominent in quiescent HSCs from healthy liver, providing a potential therapeutic target. Immunofluorescence confirmed accumulation of Cluster A-like cells around fibrotic septa in patient biopsies.



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Conclusions:

Single-cell analysis reveals significant heterogeneity among HSCs in human cirrhosis, uncovering a distinct subpopulation of highly activated, collagen-producing HSCs that drive fibrosis. Identification of a novel marker for pro-fibrogenic HSCs offers new insights into fibrogenesis and suggests potential targeted antifibrotic strategies. These findings underscore the value of high-resolution single-cell approaches in deciphering liver disease mechanisms.

Keyword(s) : liver fibrosis; single-cell RNA sequencing; hepatic stellate cells; cellular heterogeneity; fibrogenesis

2149 - Hepatitis E Vaccination in Women of Childbearing Age outcome and Implementation Challenges.

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Background / Hypothesis:

Hepatitis E virus (HEV) is prevalence in 3.3 million individuals worldwide and in Pakistan the risk is higher due to poor hygiene and sanitation. Hepatitis E virus (HEV) infection poses severe risks during pregnancy, including maternal mortality (10–50%), fetal loss, and acute liver failure, particularly in endemic regions. The HEV vaccine proven to be safe in high risk group but still, many women in Pakistan refuse for HEV vaccine due to illiteracy, fear of side effect and misconception about the vaccine. This study aim to evaluate the outcomes and implementation challenges of Hepatitis E vaccination among women of childbearing age.

Materials and Methods:

A prospective observational study was conducted at Gastroenterology ward, Jinnah Postgraduate Medical Centre (JPMC), Karachi and Atia General Hospital, Karachi to evaluate the safety, adherence, and outcomes of the HEV vaccine among women. Non-pregnant adult females of child bearing age were included after verbal consent, from September 2023 and patients were followed till September 2025. Data were collected on demographics (age group, marital status, province), vaccine uptake (number of doses), and post-vaccination side effects. Side effects were documented after each dose, and adherence was assessed based on completion of all three doses. Data

were analyzed using descriptive statistics. Associations between variables were tested using Chi-square and binary logistic regression, with significance at $p < 0.05$.

Results:

A total of 600 women were offered the Hepatitis E vaccine, of whom 225 (37.5%) declined, while 375 (62.5%) accepted and received at least one dose. Among the vaccinated, 75 (20%) consented only after detailed counseling, emphasizing the health care provider rule. Among the 375, 317 (84.5%) completed the full three-dose schedule. The most represented age group was 15–24 years, comprising 238 participants (63.5%). Regarding dose adherence, 16 individuals (4.2%) missed the second dose and 59 (15.7%) missed the third. A total of 40 participants (10.6%) reported symptoms after the first dose, and an additional 9 (2.4%) reported symptoms on second or third follow-up, including 4 participants who experienced symptoms after each of all three doses. The most common adverse effects reported were flu-like symptoms 24 (6.4%), fever 14 (3.7%), and headache 8 (2.1%). All symptoms were mild and self-limiting, with no serious adverse events

Conclusion:

This study reveals key challenges in Hepatitis E vaccine uptake among women in Pakistan, with over one-third refusing or not completing vaccination and counselling improved initial acceptance. The vaccine was well-tolerated, with only mild side effects and no significant links to demographic factors.

Keyword(s) : Hepatitis E, Vaccine, liver disease

2151 - HBV infection accelerates hepatocyte senescence and induces T cell exhaustion via SASP secretion pathway

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Background/Hypothesis: In chronic liver disease, ongoing cellular damage leads to the persistent generation of senescent cells, ultimately exacerbating tissue dysfunction. Senescent cells secrete a plethora of chemokines, cytokines, growth factors, and proteases, collectively known as the senescence-associated secretory phenotype (SASP). Chronic HBV infection often manifests as diminished and absent virus-specific T cell responses, described as an "exhausted" state. However, whether cellular senescence contributes to T

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cell exhaustion remains unclear. In this study, we aim to elucidate the relationship between HBV-mediated cellular senescence and T cell exhaustion, alongside potential underlying mechanisms.

Materials and Methods: We assessed cellular senescence in mouse models (AAV-HBV1.3 at 2 months vs. 15 months of age) and in cellular models (HepG2-hNTCP cell line) through in vitro and in vivo experiments. We examined the functional characteristics of T cells in the liver using flow cytometry and transcriptomics. Isolated liver immune cells from mice were evaluated in vitro to assess the effects of SASP on T cell exhaustion. Additionally, we treated mice with IFN α alone or in combination with the senolytic agents ABT263, to evaluate liver SASP levels and the phenotype and functional status of liver T cells.

Results: HBV infection significantly increased the mRNA levels of CDKN1A in liver tissue, and multiple immunofluorescence staining revealed a marked increase in senescent cells in the liver following HBV infection. From day 7 post-infection in the HepG2-hNTCP cell line, serum DNA damage increased, antioxidant levels decreased, and markers of hepatocyte senescence (β -gal, γ -H2AX, p21) significantly elevated. Transcriptomic sequencing of the liver demonstrated a decline in T cell function in 15-month-old HBV-infected mice, whereas 15-month-old uninfected mice did not display altered T cell functionality compared to 2-month-old mice. Flow cytometry showed increased indicators of T cell exhaustion (LAG-3, CTLA-4, TIM-3) in the livers of 15-month-old HBV-infected mice. GSVA analysis revealed significant enrichment of SASP-related pathways in 15-month-old HBV-infected mice. Luminex multiplex assay identified elevations of CCL11, SCF, IL-1RA and β -NGF in 15-month-old HBV-infected mice, with notable increases observed following HBV infection. In both mouse liver immune cells and human peripheral blood mononuclear cells (PBMCs), these four SASP factors were validated to induce T cell exhaustion. Treatment with ABT263 reduced SASP levels and reversed T cell exhaustion in 15-month-old HBV mice.

Conclusions: HBV infection can induce cellular senescence, which in turn may drive T cell exhaustion through the upregulation of the SASP. Our findings suggest that targeting the senescence-associated pathway could be a novel therapeutic strategy against HBV infection.

Keyword(s) : HBV, Hepatitis, Cell senescence, SASP, T cell exhaustion

2153 - When the Heart Fails the Liver Breaks Congestive Cholestasis Presenting as ACLF A Case Report and Literature Review

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Background / Hypothesis:

Cardio-hepatic interactions are increasingly recognized, yet ACLF precipitated primarily by acute cardiac decompensation remains underreported. Cardiac dysfunction—ranging from cardiogenic shock to pericardial tamponade—can trigger abrupt hepatic congestion, ischemia, cholestasis, coagulopathy, and multiorgan injury (Kawut et al., 2020). Case literature demonstrates that severe cardiac events may present as acute liver failure even in the absence of chronic liver disease or viral hepatitis (Boumitri et al., 2016; Mokrzycki et al., 2020). We present an elderly patient who developed high-grade congestive cholestasis with ACLF physiology, triggered by acute cardiac deterioration.

Materials and Methods:

We present a case of a 72-year-old male presenting with sudden cardiac and hepatic decompensation marked by massive pericardial effusion, biventricular congestion, and rapidly progressive jaundice. Laboratory and imaging data were serially reviewed, including bilirubin kinetics, INR progression, thrombocytopenia, creatinine trends, and pleural/ascitic fluid accumulation. The patient was evaluated against APASL and EASL-CLIF ACLF frameworks, while accounting for known cardiac hepatopathy patterns. Literature review of cardiogenic liver injury and cardiac-triggered ACLF was performed to contextualize the case.

Results:

The patient developed hyperbilirubinemia (TB 51 μ mol/L), INR 2.19, worsening thrombocytopenia, acute kidney injury, and progressive pleural/ascitic fluid accumulation. Imaging revealed coarsened hepatic parenchyma with significant portal congestion, but no evidence of viral hepatitis (HBsAg–, Anti-HCV–) or alcohol misuse. Clinical deterioration corresponded temporally with cardiac failure and pericardial tamponade physiology. These findings aligned with reported cases of cardiogenic liver failure (Boumitri et al., 2016; Mokrzycki et al., 2020) and the



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pathophysiology of hypoxic and congestive hepatopathy (Kawut et al., 2020), supporting the interpretation of Type 2 Cardio-Hepatic Syndrome presenting with ACLF-like physiology.

Conclusions:

This case highlights an underrecognized phenotype of ACLF triggered by acute cardiac collapse, reinforcing the need for heightened clinical suspicion of cardiac etiologies when cholestasis and coagulopathy are disproportionate to baseline liver disease. Recognition of cardiac-induced ACLF is essential for timely intervention, prognostication, and multidisciplinary management.

Keyword(s) : ACLF (Acute-on-Chronic Liver Failure), Cardio-hepatic syndrome, Congestive hepatopathy, Heart failure-induced liver injury, Pericardial effusion

2154 - "Saroglitazar in MASLD Translating Clinical Trials into Practice Through Liver Metrics

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is an escalating global health concern, often progressing to advanced fibrosis and cirrhosis if untreated. Its burden is amplified by comorbidities such as diabetes mellitus, hypertension, and obesity, which accelerate liver damage and complicate management. Saroglitazar, a dual PPAR α/γ agonist, has shown promise in clinical trials, but its real-world impact remains underreported. This 18-month hospital-based study evaluates the efficacy of Saroglitazar in MASLD using Fibro Scan and liver function tests (LFTs), offering translational insights that bridge controlled research and routine clinical practice.

Materials and Methods: This single-Centre, open-label observational study was conducted over 18 months to evaluate the real-world efficacy of Saroglitazar in MASLD. A total of 150 patients were screened using FibroScan, liver function tests (LFTs), clinical evaluation, and medical history. Fifty patients met diagnostic criteria for MASLD based on elevated Controlled Attenuation Parameter (CAP) and Liver Stiffness Measurement (LSM) values. All enrolled patients received Saroglitazar (Bilypsa) 4 mg once daily. Hepatic changes were monitored at baseline, 6 months, and 12 months using FibroScan and LFTs, while metabolic

parameters such as weight and BMI were tracked. Patients with comorbidities, hypertension, and obesity were maintained on standard therapies including antidiabetics, antihypertensives, and statins. Statistical analysis was performed using paired t-tests, with $p < 0.05$ considered significant.

Results: Out of 150 patients screened, 50 were diagnosed with MASLD and enrolled (mean age: 58.16 ± 10.93 years; 30 males, 20 females). Comorbidities included diabetes (n=38), hypertension (n=16), obesity (n=21), and concurrent alcoholic liver disease (n=16). Baseline Fibro Scan revealed predominantly Grade II steatosis, with elevated CAP (273.60 ± 40 dB/m) and LSM (12.08 ± 5.32 kPa). After 12 months of Saroglitazar 4 mg daily, CAP decreased by 17.4% to 226.10 ± 40.55 dB/m ($p = 0.013$), LSM declined by 25.2% to 9.04 ± 1.53 kPa, AST dropped by 40.2% (from 53.52 to 32.00 U/L), and ALT by 23.0% (from 59.54 to 45.80 U/L). The drug was well tolerated with no reported adverse events.

Conclusions: Saroglitazar 4 mg once daily demonstrated significant real-world efficacy in MASLD over 18 months, with marked reductions in hepatic steatosis (CAP), fibrosis (LSM), and liver enzymes (AST, ALT). Improvements were consistent across patients with metabolic comorbidities, and the drug was well tolerated with no adverse events, supporting its translational value in routine clinical practice.

Keyword(s) : MASLD, Bilypsa, Saroglitazar, rwe

2155 - Gut Microbiota Dysbiosis Correlates with Disease Severity in Autoimmune Hepatitis

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Background / Hypothesis:

The gut-liver axis plays a pivotal role in liver immunity, and disruptions in gut microbiota composition (dysbiosis) have been implicated in various liver diseases. In autoimmune hepatitis (AIH), an immune-mediated liver inflammation, the contribution of gut microbiota is not well defined. We hypothesized that AIH patients exhibit a distinct gut microbiome profile compared to healthy individuals and that the degree of microbiota dysbiosis correlates with AIH disease severity, potentially influencing immune regulation in the liver.



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Materials and Methods:

A cross-sectional study was performed involving 20 patients with established AIH (mean Ishak histological activity index = 9) and 20 age-/sex-matched healthy controls. Fecal samples were collected and analyzed by 16S rRNA gene sequencing to characterize microbiota composition and diversity. Alpha diversity (Shannon index) and beta diversity metrics were calculated. Differential abundance analysis identified taxa with significantly different relative abundance between AIH and controls. Clinical data (ALT, AST, IgG levels) and histological severity scores were correlated with microbial diversity and specific taxa abundance. Additionally, peripheral blood T cell subsets (Th17 and Treg frequencies by flow cytometry) were assessed in AIH patients to explore immunological correlations.

Results:

AIH patients exhibited marked gut microbiota dysbiosis, with a ~25% reduction in microbial diversity (Shannon index) compared to controls ($p < 0.01$). Compositionally, AIH microbiomes were enriched in pro-inflammatory genera such as Enterobacteriaceae and Streptococcus, and depleted in short-chain fatty acid-producing bacteria like Faecalibacterium and Lachnospiraceae ($p < 0.05$ for multiple taxa). Notably, the relative abundance of Faecalibacterium negatively correlated with serum ALT levels ($r = -0.65$, $p < 0.01$), while an overgrowth of Enterobacteriaceae was associated with higher IgG and histological inflammation scores. AIH patients with the most pronounced dysbiosis showed a decreased peripheral Treg/Th17 ratio, suggesting a link between gut microbial imbalance and systemic immune dysregulation.

Conclusions:

Patients with autoimmune hepatitis have a distinct gut microbiota profile characterized by reduced beneficial microbes and increased potentially pathogenic taxa, and the extent of this dysbiosis correlates with disease severity and immune imbalance. These findings implicate the gut microbiome in AIH pathogenesis and raise the prospect of using microbiota-based biomarkers or therapeutics (such as probiotics or fecal microbiota transplantation) as adjunctive strategies to modulate disease activity.

Keyword(s) : autoimmune hepatitis; gut microbiota; dysbiosis; immune regulation; liver inflammation

Advanced Hepatocellular Carcinoma

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Background / Hypothesis:

Advanced hepatocellular carcinoma (HCC) carries a poor prognosis, and traditional monotherapies like tyrosine kinase inhibitors (e.g., sorafenib) offer modest survival benefits. Immune checkpoint inhibitors (ICIs) and antiangiogenic agents have individually shown efficacy in HCC by enhancing anti-tumor immunity and inhibiting tumor vascularization, respectively. Preclinical studies suggest synergistic effects when combining these modalities. We hypothesized that concurrent PD-1/PD-L1 immune checkpoint blockade and VEGF pathway inhibition would significantly improve clinical outcomes in patients with unresectable HCC compared to standard therapy.

Materials and Methods:

We performed a multi-center Phase II randomized trial in 120 patients with untreated, advanced HCC. Patients were randomized 1:1 to receive combination immunotherapy plus antiangiogenic therapy (nivolumab, an anti-PD-1 antibody, 240 mg IV every 2 weeks, plus bevacizumab, an anti-VEGF antibody, 15 mg/kg IV every 3 weeks; $n=60$) or standard-of-care sorafenib (400 mg orally twice daily; $n=60$). The primary endpoint was overall survival (OS); secondary endpoints included progression-free survival (PFS), objective response rate (ORR by RECIST v1.1), and safety/tolerability. Tumor assessments were conducted every 8 weeks and adverse events were graded per CTCAE v5.0.

Results:

The combination therapy demonstrated superior efficacy over sorafenib. At a median follow-up of 18 months, median OS was extended in the combination arm (20.1 months) compared to the sorafenib arm (13.2 months; hazard ratio 0.60, 95% CI 0.45–0.80, $p < 0.001$). One-year survival rate was 67% with combination treatment versus 45% with sorafenib. ORR was significantly higher in the combination group (35% vs 12%, $p=0.002$), whereas disease control rates were 80% vs 52%. Median PFS improved to 9.7 months with combination therapy versus 5.5 months with sorafenib ($p < 0.001$). The safety profile of the combination was manageable: expected immune-related events (e.g., hepatitis, dermatologic reactions in 15% of patients) and antiangiogenic effects (grade 3 hypertension in

2156 - Combination Immune Checkpoint Inhibition and Antiangiogenic Therapy Improves Survival in

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10%, minor bleeding in 8%) were observed, but no unexpected toxicities occurred.

Conclusions:

Combined PD-1 immune checkpoint inhibition and VEGF-targeted therapy yielded significantly improved survival and response rates in advanced HCC compared to sorafenib. The results support the efficacy of this combination approach, suggesting a new standard of care for first-line management of advanced HCC. Further phase III trials and longer follow-up are warranted to confirm these benefits and monitor long-term safety.

Keyword(s) : hepatocellular carcinoma; immunotherapy; antiangiogenic therapy; combination treatment; clinical trial

2157 - A Rare etiology of Liver mass in a patient of chronic Hepatitis C

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Background / Hypothesis:

A Rare etiology of Liver mass in a patient of chronic Hepatitis C

ABSTRACT:

Follicular Dendritic Cell sarcoma (FDCC) of the liver is rare¹. It mostly presents in the lymph nodes; extra nodal presentation is rare. We present the case of a 45-year-old male of successfully treated for chronic hepatitis C (CHC), who complained of pain in the right hypochondrium, abdominal distension, and weight loss for the last 2 months. A triphasic computed tomography (CT) abdominal scan showed a large irregular mass of approximately 17.7 × 15.7×15.9 cm in the right lobe of the liver, with a thrombus in the Portal vein, splenomegaly, and mild abdominal ascites representing atypical Hepatoma. A liver biopsy revealed FDCC. The patient started on Gemcitabine and Doxorubicin. After completion of chemotherapy, the patient died of myocardial infarction. Early diagnosis is crucial, and surgery plays a key role in treatment, along with adjuvant chemotherapy and radiotherapy

Keywords: Follicular dendritic cell sarcoma, Chronic hepatitis C, Atypical hepatoma

Materials and Methods:

Results:

Conclusions:

Keyword(s) :

2158 - "Evaluating the Early Biochemical Impact of Saroglitazar on Liver Function Tests in MASLD Patients

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Mar Sleeva Medicity, Pala, Kottayam, Medical Gastroenterologist-senior Consultant And Hod, Kottayam-India¹

Background / Hypothesis:

Saroglitazar, a dual PPAR α/γ agonist, has emerged as a promising therapeutic candidate in the management of metabolic dysfunction-associated steatotic liver disease (MASLD). Early biochemical responses, particularly liver function test (LFT) alterations, may serve as surrogate markers for hepatic improvement and treatment monitoring. This study investigates the impact of Saroglitazar therapy on key LFT parameters in MASLD patients, aiming to characterize trends in transaminase levels as indicators of biochemical efficacy and hepatic safety

Materials and Methods: A total of 100 patients with suspected MASLD were screened over 12 months using clinical evaluation and liver function tests (LFTs) as primary assessment tools. Of these, 16 patients met the diagnostic criteria for MASLD based on elevated transaminases and clinical features. All 16 diagnosed patients underwent baseline LFTs and lipid profiling. They were initiated on Saroglitazar 4 mg once daily. Follow-up biochemical monitoring, including repeat LFTs and lipid panels, was conducted every 6 months for up to one year to evaluate therapeutic response and hepatic safety.

Results: Of the 16 MASLD patients (10 males, 6 females; mean age $\pm$ SD: 60.12 $\pm$ 13.1 years; weight:

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72.4 ± 13.2 kg), baseline AST, ALT, and total cholesterol were 27.29 IU, 29.78 IU, and 250 mg/dL, respectively. Following 6 months of Saroglitazar therapy, these declined to 20.29 IU (↓25.7%), 21.62 IU (↓27.4%), and 180 mg/dL (↓28%), respectively. These reductions indicate early biochemical response in hepatic transaminases and lipid profiles.

Conclusions: Saroglitazar 4 mg showed early biochemical efficacy in MASLD, reducing AST, ALT, and total cholesterol by 25.7%, 27.4%, and 28% over 6 months. These improvements reflect its dual PPAR α/γ activity targeting hepatic and metabolic pathways

Keyword(s) : MASLD,Bilypsa,Saroglitazar,RWE,LFTs

2159 - A rare lymphoproliferative disorder in teenagers with spontaneous resolution on PET scan.

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Background / Hypothesis:

Materials and Methods:

Results:

Rosai Dorfman Disease (RDD), also called Sinus Histocytes, is a rare disease of enlarged lymph nodes with no known etiologies. It mainly involves the cervical lymph nodes. Involvement of the isolated mesenteric lymph nodes has not been reported previously. Herein, we report a case of mesenteric lymph node involvement by RDD, which occurred in a 16-year-old boy. He presented with intermittent right lower quadrant pain, fever, altered bowel habits, and weight loss for the last 2 months. The clinical and radiological findings were consistent with lymphoma with ileoileal intussusception. The patient underwent a laparoscopic biopsy of the lymph node. Histopathology and immunochemistry studies of the lymph node confirmed the diagnosis of RDD. The clinical symptoms and lymph nodes resolved spontaneously after 6 months of keen observation, confirmed on a Positron emission tomography (PET) scan.

Keywords: Rosai Dorfman Disease, sinus histocytes, mesenteric lymph nodes, spontaneous resolution,

Conclusions:

Keyword(s) :

2160 - Finite versus indefinite nucleos(t)ide analogue therapy in HBeAg-negative chronic hepatitis B Meta-analysis of Randomized Trials.

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Background:

Hepatitis B e antigen (HBeAg)-negative chronic infection is now the predominant global phenotype. Long-term nucleos(t)ide analogue (NA) therapy reliably suppresses HBV replication but seldom achieves loss HBsAg or functional cure, motivating interest in finite-duration NA strategies.

Objective:

To compare the efficacy and safety of finite versus continuous NA therapy in adults with HBeAg-negative chronic hepatitis B, using evidence restricted to randomized controlled trials (RCTs).

Methods:

We identified parallel-group RCTs enrolling non-cirrhotic, virally suppressed HBeAg-negative adults randomized to protocol-defined NA cessation versus continued NA therapy. Dichotomous outcomes were pooled as risk ratios (RRs) with 95% confidence intervals (CIs) using random-effects models. Risk of bias was assessed with RoB 2, and certainty of evidence was graded using GRADE.

Results:

Four RCTs (n≈357) met eligibility criteria. Compared with continued therapy, finite NA cessation increased HBsAg loss (RR≈5.0, 95% CI≈1.2–21.7; moderate certainty), corresponding to approximately 5–6 additional HBsAg losses per 100 treated patients over 2–3 years. Finite therapy resulted in substantially higher risks of virologic relapse (RR≈16) and retreatment (RR≈19), and a lower probability of sustained off-therapy virologic response (RR≈0.45; moderate certainty). ALT flares were more common following NA discontinuation but were generally self-limited or resolved promptly with retreatment. Across trials, no hepatic decompensation or treatment-related serious adverse events were observed.

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Conclusions:

Among carefully selected non-cirrhotic, HBeAg-negative adults managed under structured monitoring, finite NA therapy increases the likelihood of HBsAg loss at the expense of predictable virologic relapse and retreatment, without evidence of excess severe liver-related harm in RCT settings. These findings support the selective, patient-centered use of finite NA therapy in expert clinical programs aiming to promote functional cure.

Keyword(s) :

2162 - A rare cause of Gastric malignancy with liver metastasis in a young Patient.

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Background / Hypothesis:

Materials and Methods:

Gastrointestinal stromal tumors (GISTs) are rare tumors accounting for 0.1%–3% of all gastrointestinal (GI) cancers¹. GISTs mostly involve the stomach (60%) and 20%–25% are malignant, with metastases involving the liver or peritoneum². Gastric GISTs with liver metastases in teenagers are extremely rare. In the literature, only three such cases have been reported so far. Here, we present a rare case of a 19-year-old male with no known comorbid presented with complaints of black stools on and off for 11 months.

Esophagogastroduodenoscopy (EGD) showed large polypoidal growth in the fundus of the stomach; multiple biopsies were taken. Histopathology confirmed the diagnosis of GIST. Computed Tomography (CT) scan showed a gastric mass of 16 cm with metastasis involving both lobes of the liver. The patient was started on Imatinib. He has been under continuous follow-up for the last 5 months.

Keywords: Gastrointestinal stromal tumors, teenagers, Stomach, Liver Metastasis.

Results:

Conclusions:

Keyword(s) :

2163 - A rare etiology of Liver mass in a patient of chronic Hepatitis C

Ghulam Mohiuddin¹, Nazish Butt¹

Jinnah Postgraduate Medical Complex, Kara, Gastroenterology And Hepatology, Karachi.-Pakistan¹

Follicular Dendritic Cell sarcoma (FDCS) of the liver is a rare clinical entity. It mostly presents in the lymph nodes; extra lymph nodal presentation is rare. We present the case of a 45-year-old male with a case of successfully treated chronic hepatitis C (CHC), who complained of pain in the right hypochondrium, abdominal distension, and weight loss for the last 2 months. Triphasic computed tomography (CT) abdominal scan showed a large irregular mass of approximately 17.7 × 15.7 × 15.9 cm in the right lobe of the liver with thrombus in the Portal vein, splenomegaly and mild abdominal ascites representing atypical Hepatoma. A liver biopsy performed revealed FDCS. The patient started on Gemcitabine and Doxorubicin. After completion of chemotherapy, the patient died of myocardial infarction.

Keyword(s) : Follicular dendritic cell sarcoma, Chronic hepatitis C, Atypical hepatoma.

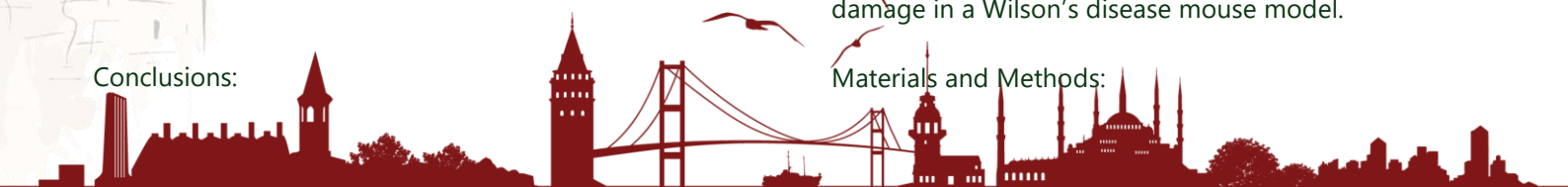
2164 - AAV Mediated ATP7B Gene Therapy Corrects Copper Metabolism in a Wilson's Disease Mouse Model

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Beijing University Of Chinese Medicine, School Of Traditional Chinese Medicine, Beijing-China¹

Background / Hypothesis:

Wilson's disease is an inherited disorder of copper metabolism caused by loss-of-function mutations in the ATP7B gene, leading to toxic copper accumulation in the liver and other organs. Current treatments (chelating agents and zinc) reduce copper levels but do not address the underlying genetic defect and can have significant side effects. Gene therapy offers a potential cure by restoring functional ATP7B. We hypothesized that delivering a healthy ATP7B gene via an adeno-associated virus (AAV) vector to the liver would restore copper homeostasis and prevent liver damage in a Wilson's disease mouse model.

Materials and Methods:



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Using the ATP7B-knockout “toxic milk” mouse model of Wilson’s disease, we administered a single intravenous dose of an AAV8 vector carrying the human ATP7B gene to Wilson’s disease mice (n=10). Control knockout mice (n=10) received saline. Liver copper content was measured 8 weeks post-treatment. Serum ceruloplasmin levels (an indicator of copper transport function) were also measured. Liver injury was evaluated via serum transaminases (ALT, AST) and histopathology (hemosiderin and copper staining, inflammation, fibrosis scoring). Survival was monitored over 6 months.

Results:

AAV8-ATP7B treated mice showed dramatic improvements in copper metabolism and liver health compared to untreated controls. Hepatic copper concentration decreased by ~70% in treated mice ($p < 0.001$), approaching levels seen in wild-type mice. Serum ceruloplasmin increased from virtually undetectable in untreated knockout mice to 85% of normal levels in gene-treated mice, indicating restoration of copper transport activity. Treated mice had near-normal ALT/AST levels and significantly less liver histopathology: inflammatory lesions and fibrotic changes were minimal, whereas untreated mice exhibited extensive hepatitis and early cirrhosis. All AAV-treated mice survived the 6-month study period, while 40% of control mice died or required euthanasia due to liver failure. No adverse events or off-target organ damage were observed in treated animals.

Conclusions:

Liver-directed AAV-mediated gene therapy effectively corrected the metabolic defect in a Wilson’s disease mouse model, resulting in normalization of copper handling and prevention of liver damage. These findings demonstrate the promise of ATP7B gene replacement as a disease-modifying therapy for Wilson’s disease. Further studies are warranted to evaluate long-term durability, optimal dosing, and safety in larger animal models, paving the way for eventual clinical trials in human patients.

Keyword(s) : Wilson’s disease; gene therapy; AAV vector; copper metabolism; ATP7B

2165 - A rare lymphoproliferative disorder in teenagers with spontaneous resolution on PET scan.

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Rosai Dorfman Disease (RDD), also called Sinus Histocytes, is a rare disease of enlarged lymph nodes with no known etiologies. It mainly involves the cervical lymph nodes. Involvement of the isolated mesenteric lymph nodes has not been reported previously. Herein, we report a case of mesenteric lymph node involvement by RDD, which occurred in a 16-year-old boy. He presented with intermittent right lower quadrant pain, fever, altered bowel habits, and weight loss for the last 2 months. The clinical and radiological findings were consistent with lymphoma with ileoileal intussusception. The patient underwent a laparoscopic biopsy of the lymph node. Histopathology and immunochemistry studies of the lymph node confirmed the diagnosis of RDD. The clinical symptoms and lymph nodes resolved spontaneously after 6 months of keen observation, confirmed on a Positron emission tomography (PET) scan.

Keyword(s) : Rosai Dorfman Disease, sinus histocytes, mesenteric lymph nodes, spontaneous resolution,

2166 - Whole Genome Sequencing Identifies Novel Genetic Mutations in Unexplained Pediatric Cholestatic Liver Disease

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Background / Hypothesis:

A substantial fraction of infants and children with chronic cholestatic liver disease remain etiologically undiagnosed after standard clinical workup, which typically includes tests for infectious, metabolic, and known genetic causes. This diagnostic gap leads to delayed or suboptimal treatment and may obscure novel disease entities. Whole-genome sequencing (WGS) offers a comprehensive approach to identify both known and new genetic mutations. We hypothesized that WGS would increase the diagnostic yield in pediatric cholestasis cases that were previously unexplained and uncover novel genetic mutations associated with cholestatic liver disease.

Materials and Methods:

Twenty-five pediatric patients (age <5 years) with idiopathic cholestatic liver disease were enrolled. All had unrevealing prior evaluations. Trio WGS (child and both parents) was performed to facilitate variant

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interpretation. Bioinformatic analysis focused on rare, predicted deleterious variants consistent with Mendelian inheritance patterns. Variants in known cholestasis-related genes were evaluated for pathogenicity. Novel candidate genes not previously linked to cholestasis were further analyzed for functional relevance.

Results:

WGS provided a genetic diagnosis in 15 of 25 patients (60%). Thirteen patients harbored pathogenic or likely pathogenic variants in known cholestasis genes that had been missed by prior targeted testing – including mutations in genes associated with PFIC and Alagille syndrome – which expanded the mutational spectrum of known conditions. Importantly, two patients harbored biallelic mutations in a gene not previously associated with cholestasis, suggesting a novel genetic cause; preliminary functional assays supported this gene's role in bile acid transport. In the remaining unsolved cases, WGS highlighted a few candidate variants for further investigation. Based on WGS results, clinical management was modified in several patients (e.g., listing for transplant expedited in those with progressive genetic cholestasis, and avoidance of triggers in a patient diagnosed with a metabolic cholestatic condition).

Conclusions:

Whole-genome sequencing substantially improved the diagnostic rate for unexplained pediatric cholestasis and uncovered novel genetic causes in a subset of patients. These findings underscore the utility of WGS as a diagnostic tool in pediatric cholestatic disorders, enabling precision medicine and expanding the understanding of genetic liver diseases. Integrating WGS into routine evaluation of idiopathic cholestasis will accelerate diagnosis and inform management in challenging cases.

Keyword(s) : cholestatic liver disease; genetics; whole-genome sequencing; pediatric; novel mutation

2169 - Multiple Cutaneous and Gastrointestinal Hemangiomas Presenting with Iron Deficiency Anemia A Rare Case Report of Blue Rubber Bleb Nevus Syndrome.

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Blue Rubber Bleb Nevus Syndrome (BRBNS) is a rare venous malformation disorder characterized by multiple hemangiomas of the skin and gastrointestinal (GI) tract. Because GI lesions can lead to microcytic anemia, early recognition and management are critical.

Case presentation: An eleven-year-old female patient presented with symptomatic iron-deficiency anemia. On examination, multiple bluish, dome-shaped, compressible nodules were noted on the arms and dorsum of the feet. Colonoscopy and upper endoscopy revealed numerous submucosal blue-tinged lesions in the colon and stomach, consistent with cavernous hemangiomas. Cutaneous biopsy confirmed venous malformation. The patient was managed conservatively with blood transfusion and by iron replacement and showed resolution of Anemia.

Conclusion: BRBNS should be considered in patients with unexplained Microcytic Anemia and characteristic cutaneous lesions. Endoscopic therapy can effectively diagnose and improve the quality of life.

Keyword(s) : Blue Rubber Bleb Nevus Syndrome, venous malformation, hemangioma, Iron deficiency Anemia

2170 - PROBABLE ASHWAGANDHA INDUCED HEPATITIS IN A HEALTHY YOUNG ADULT A CASE REPORT

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Background / Hypothesis:

Ashwagandha (*Withania asomnifera*) is a popular adaptogenic herb in Ayurvedic medicine, Often regraded as safe. However, recent reports indicate potential hepatotoxicity, including cholestatic liver injury. We present a biopsy-confirmed case of probable Ashwagandha-induced cholestatic hepatitis in a healthy young adult, highlighting the diagnostic challenges associated with herbal hepatotoxicity and the necessity of comprehensive supplement history-taking in cases of unexplained liver injury.

Materials and Methods:



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A 24-year-old male with no significant medical history presented with a two-month history of right upper quadrant pain and progressive jaundice. He began Ashwagandha supplementation 2–3 weeks prior to symptom onset.

Laboratory tests revealed a cholestatic pattern of liver injury: total bilirubin 14.2 mg/dL, alkaline phosphatase (ALP) 380 U/L, aspartate aminotransferase (AST) 165 U/L, and alanine aminotransferase (ALT) 142 U/L.

Extensive workup, including viral hepatitis serology, autoimmune markers, metabolic studies, abdominal ultrasound, and Magnetic resonance cholangiopancreatography, were normal. Liver biopsy revealed marked canalicular cholestasis with feathery degeneration, lobular inflammation, and mild bile duct injury, consistent with drug-induced cholestatic hepatitis. The patient received a tapering course of prednisone and ursodeoxycholic acid with discontinuation of Ashwagandha and that led to gradual normalization of liver tests and complete resolution of symptoms.

Conclusions:

This case underscores the potential for Ashwagandha to induce cholestatic hepatitis in young adults, reinforcing concerns regarding its hepatotoxicity. Diagnosis was supported by exclusion of other etiologies, biopsy findings, and a RUCAM score of 7. The injury is likely idiosyncratic, possibly associated with withanolide toxicity, CYP450 enzyme inhibition, or contaminants. While steroids were administered despite the absence of autoimmune features, their efficacy remains uncertain. This case highlights the importance of routine screening for herbal supplement use, improved regulation of dietary supplements, and further research into risk factors and treatment options for herb-induced liver injury.

Keyword(s) : Ashwagandha, induced cholestatic hepatitis, herbs induced hepatitis

2171 - HBV RNA as a Predictor of Hepatocellular Carcinoma in Antiviral suppressed Chronic Hepatitis B A Systematic Review and Meta analysis

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Background: Serum hepatitis B virus RNA (HBV RNA), representing transcriptionally active covalently closed circular DNA (cccDNA), has emerged as a potential biomarker of residual viral activity among nucleoside analogue (NA)–suppressed patients. While HBV DNA becomes undetectable with long-term therapy, persistent HBV RNA production may reflect ongoing oncogenic risk. We hypothesized that detectable or higher serum HBV RNA levels are independently associated with increased incidence of hepatocellular carcinoma (HCC), and that pooling available observational evidence would clarify the strength and consistency of this association.

Materials and Methods: A systematic search of PubMed, Embase, and clinical trial registries was conducted for studies published between 2020 and 2025 evaluating serum HBV RNA or pregenomic RNA (pgRNA) in relation to HCC outcomes. Cohort studies reporting adjusted hazard ratios (HR) for incident HCC were included in the analysis. Two reviewers independently extracted study characteristics, assay methods, follow-up, and effect estimates. Risk of bias was assessed using ROBINS-I. Random-effects meta-analysis was performed, with Hartung–Knapp adjustment for confidence intervals given the small number of studies. Leave-one-out analyses evaluated robustness.

Results: Three independent cohorts (N ≈ 3389) met the inclusion criteria for incident HCC analysis. All studies utilized adjustments for major clinical confounders. ROBINS-I indicated moderate overall risk of bias, mainly due to residual confounding and variable adjustment sets. Pooled random-effects analysis demonstrated that detectable or quantifiable HBV RNA was significantly associated with incident HCC: pooled HR 2.57 (95% CI 1.62–4.09). Hartung–Knapp adjustment yielded similar findings (HR 2.57; 95% CI 2.15–3.08). Heterogeneity was low ($I^2 = 0\%$). Leave-one-out analyses produced consistent HRs (range 2.41–3.70), indicating stability of the association. Supporting studies on recurrence after resection and diagnostic case–control cohorts showed concordant trends, with higher HBV RNA levels observed in HCC patients.

Conclusion: Detectable serum HBV RNA is consistently associated with a substantially increased risk of incident HCC among NA-treated chronic hepatitis B patients. Although current evidence is limited to a small number of moderate-quality observational

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cohorts, the association is robust across sensitivity analyses and biologically plausible. Larger prospective studies and standardized HBV RNA assays are needed to validate its role as a prognostic biomarker and to inform HCC surveillance strategies.

Keyword(s) : HBV RNA, chronic hepatitis B, HBV DNA, hepatocellular carcinoma

2173 - Dietary Recommendations for Patients with Chronic Liver Diseases The Need for Increased Awareness Among Non Hepatologist Physicians

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Background / Hypothesis:

Nutritional awareness is a significant challenge for patients with chronic liver diseases (CLD), especially for those with conditions such as ascites, hepatic encephalopathy, and fatty liver disease. This survey aims to assess nutritional awareness regarding liver disease among healthcare providers (hepatologists and non-hepatologists).

Materials and Methods:

A structured online questionnaire was created based on clinical knowledge and established guidelines related to nutrition in liver disease. The survey consisted of 13 questions addressing nutritional considerations for liver disease patients.

Results:

A total of 124 out of 362 survey recipients responded. Among them, 60.5% were hepatologists, while the remaining respondents included general practitioners, endocrinologists, and cardiologists. Hepatologists were more likely than non-hepatologists to recognize the significance of nutritional guidance for their patients, with 74.7% of hepatologists supporting this view compared to only 40.8% of non-hepatologists ($p < 0.001$). For patients with compensated cirrhosis, 82.7% of hepatologists preferred no dietary restrictions, whereas only 46.9% of non-hepatologists agreed ($p < 0.001$). Both groups favored protein restriction in cases of hepatic encephalopathy, ($p = 0.2$). Regarding patients with ascites, both hepatologists and non-hepatologists recommended salt restriction, with hepatologists showing a stronger inclination. When it

comes to alcohol consumption, 80% of hepatologists and 61.2% of non-hepatologists favored restricting alcohol intake in patients with chronic liver disease ($p = 0.03$).

Conclusions:

There are notable differences in nutritional priorities and recommendations between hepatologists and non-hepatologists. There is a pressing need to raise awareness among non-hepatologist physicians concerning dietary recommendations for patients with chronic liver disease.

Keyword(s) : chronic liver disease, dietary recommendations, awareness

2174 - MAFLD aggravates Colorectal Cancer Progression A quantitative MRI PDFF based study

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) significantly implicated in cancer risk, yet its effects on colorectal cancer (CRC) progression are poorly understood. This study investigated MAFLD's influence on CRC outcomes.

Materials and Methods:

We conducted a retrospective observational study on 246 CRC patients. MAFLD presence was determined by MRI-based proton density fat fraction (PDFF) for hepatic steatosis and diagnostic criteria for metabolic dysfunction. Multivariate logistic regression identified risk factors for advanced CRC. Additionally, bioinformatic analysis, including shared differentially expressed genes (DEGs), enriched pathways, and survival validation of candidate genes, was performed using public datasets.

Results:

Among 246 CRC patients, 73 (29.7%) fulfilled MAFLD criteria. CRC-MAFLD patients showed significantly higher rates of lymph node involvement (87.7% vs. 35.8%, $p < 0.001$), distant metastasis (31.5% vs. 13.9%, $p = 0.002$), and mucinous/signet ring histology. In multivariate analysis, MAFLD has emerged as an independent predictor of lymph node involvement

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(OR, 11.7; 95% CI, 4.5–30.6; $p < 0.001$) and distant metastasis (OR, 3.4; 95% CI, 1.4–8.7; $p = 0.009$) in CRC patients. Bioinformatic analysis revealed shared DEGs, with upregulated genomic integrity pathways and downregulated metabolism/detoxification. Notably, APPL2 and GLTP were highly expressed in severe MAFLD (MASH) and linked to poorer CRC survival.

Conclusions:

MAFLD is an independent risk factor for severe CRC phenotypes, highlighting its systemic metabolic impact on cancer outcomes that warrants consideration in management.

Keyword(s) : Colorectal cancer, MAFLD, MRI-PDFF, Steatosis

2175 - High Prevalence of Unrecognized Advanced Fibrosis in Patients with MAFLD

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Background / Hypothesis:

Metabolic dysfunction associated fatty liver disease (MAFLD) is the leading cause of chronic liver disease worldwide. Although liver Fibrosis is the main determinant of hepatic and cardiometabolic outcomes, screening for clinically significant fibrosis remains inconsistently applied in practice. This study aims to evaluate the coexisting gap of liver fibrosis awareness among patients referred for reassessment within the vibration-controlled transient elastography (VCTE) workflow.

Materials and Methods:

149 patients fulfilled the diagnostic criteria of MAFLD according to CAP ≥ 264 dB/m and metabolic criteria. Fibrosis was assessed using the VCTE liver stiffness measurement (LSM). Significant fibrosis was defined as LSM ≥ 8 kPa. Multivariable logistic regression identified risk factors of fibrosis.

Results:

Among the MAFLD cohort, 15.4% had significant fibrosis and 10.1% had advanced fibrosis. Patients with significant fibrosis ($F \geq 2$) exhibited a higher metabolic burden and biochemical risk profile, including higher BMI, waist circumference, HbA1c, elevated transaminases, and lower platelet counts after

adjustment, HbA1c (OR 1.4, 95% CI: 1.1–1.8; $p = 0.01$) and platelet count (OR 0.99, 95% CI: 0.98–0.99; $p = 0.03$) remained independent predictors.

Conclusions:

Among patients undergoing initial evaluation for MAFLD, a substantial proportion harbored clinically significant fibrosis without prior suspicion. These findings highlight the gap in awareness in current practice and support the adoption of structured, risk-based screening pathways, serum fibrosis scores, and VCTE within metabolic and primary care practices to achieve earlier identification and timely intervention.

Keyword(s) : MAFLD, Liver Fibrosis, awareness, VCTE, liver stiffness.

2176 - Metabolic predictors of Liver fibrosis across MAFLD phenotypes A cross sectional study using Transient Elastography

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Background / Hypothesis:

Liver fibrosis serves as a key risk factor that influences severe liver outcomes, including cirrhosis, and the need for transplants in Metabolic-associated fatty liver disease (MAFLD). Yet, data on metabolic risk predictors for significant fibrosis in those patients remains limited. Aim: This study aims to identify metabolic risk factors for significant fibrosis across MAFLD phenotypes.

Materials and Methods:

We conducted a cross-sectional survey of 390 patients diagnosed with MAFLD using imaging (Transient Elastography) and metabolic evaluations. Participants were classified into three groups: Group 1, with a BMI ≥ 25 ; Group 2, with T2DM; and Group 3, with a BMI < 25 and $\geq$ two metabolic risk factors. We defined significant fibrosis ($F \geq 2$) with a liver stiffness measurement cut-off ≥ 8.2 kPa.

Results: 24.6% of the cohort exhibited significant fibrosis, with the highest prevalence observed in Groups 1 (68.7%) and 2 (55.2%). Group 3 had the lowest prevalence (31.2%). Patients in Group 1 had an odds ratio of 3.8 (p -value 0.003) for significant fibrosis, while those in Group 2 had an OR of 2.8 (p -value 0.01).

Conclusions:

TE reveals significant fibrosis varies among MAFLD patients based on metabolic syndrome. Recognizing

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predictive factors like the Fib-4 index, degree of steatosis, and metabolic risks, particularly BMI $\geq$ 25 and T2DM, is crucial for improving patient management and outcomes

Keyword(s) : Significant fibrosis, Metabolic risk factors, Transient elastography, MAFLD

2177 - MAFLD CKM Modified score better predicts long term all cause mortality

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Background / Hypothesis: The American Heart Association (AHA) developed the cardiovascular-kidney-metabolic (CKM) model, which reframes cardiovascular, renal, and metabolic risk as a marker for the worst adverse events but leaves out hepatic dysfunction. Cardiorenal mortality is closely associated with metabolic-associated fatty liver disease (MAFLD), which is very common throughout the world. This suggests an underappreciated gap in current CKM staging. The purpose of this study is to assess the additional risk of MAFLD in both specific-cause and all-cause mortality across the CKM criterion.

Methods: In this cross-section study we used data of 9,309 adults aged 20–74 years from NHANES III (1988–1994) with mortality follow-up through 2019. The AHA framework was used to classify the CKM stage. Hepatic steatosis and typical metabolic dysfunction were the hallmarks of MAFLD. We evaluated all-cause and cause-specific mortality using Cox regression adjusted for age, sex, race, and smoking. We proposed a modified (M-CKM) score that integrated MAFLD status into CKM staging. For those modified models, a Kaplan-Meier curve was plotted. Results: Over a median 23-year follow-up, 38.9% mortality rose stepwise across CKM stages: 20% no CKM, 35.9% early CKM, and 66.5% late CKM ($p < 0.001$). MAFLD was present in 41.2% and 52.1% of early and late CKM. The M-CKM score demonstrated a better prognostic value than discrete strata, predicting increased risks of all-cause mortality (HR 2.51, 95% CI 1.42–4.44) and specific mortality from diabetes, cardiovascular, and cerebrovascular causes. M-CKM had significantly lower survival across all outcomes, according to Kaplan-Meier analyses ($p < 0.001$)

Conclusion: The Risk prediction for long-term mortality is significantly improved by incorporating MAFLD into

the CKM framework. The suggested M-CKM encourages a paradigm change in favour of an all-encompassing cardio-renal-hepatic-metabolic model that is utilised for roadmap evaluation of potential adverse events.

Keyword(s) :

2178 - Predictors and gaps in hepatocellular carcinoma staging and management A multicenter prospective study in Bangladesh

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Background / Hypothesis: Primary liver cancer is the sixth most common cancer globally. Current guidelines recommend routine surveillance for high-risk patients to facilitate early detection. However, global adherence remains low worldwide. Effective etiological management and structured follow-up are essential to prevent hepatocellular carcinoma (HCC) or detect it at an early stage. This study aimed to assess the stage at diagnosis, related clinical outcomes, and adherence to guideline-recommended evaluation and treatment in patients with hepatocellular carcinoma.

Materials and Methods: This multicenter, prospective, cross-sectional analytical study was conducted across five tertiary care centers in Bangladesh from December 2024 to November 2025. Data were collected prospectively from the consecutive patients diagnosed with HCC using a standardized questionnaire.

Results: A total of 245 patients diagnosed with HCC were included. The median age was 50 years (IQR: 41–60), and 85.3% (209/245) were male. The most common underlying etiology was hepatitis B virus (HBV) infection, 69.8%; followed by hepatitis C virus (HCV) infection, 11.4%; and metabolic dysfunction-associated fatty liver disease (MAFLD) at 6.5%. HBV–HCV coinfection was identified in 0.8% of patients (Table 1). According to the BCLC staging system, only 6.9% patients presented with BCLC 0–A, 24.1% with BCLC-B, 39.6% with BCLC-C, and 29.4% with BCLC-D. Physician adherence to AASLD guidelines was 72.2%.

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Imaging-based diagnosis and biopsy with histopathology were used in 75.9% and 21.2% of cases, respectively. During evaluation, 26.9% of patients have portal vein tumoral thrombosis (PVTT). In the multivariate analysis, a higher CTP class was associated with advanced-stage HCC (for CTP-C, OR 6.65, $p = 0.003$). Performance status (ECOG) was a strong predictor of advanced BCLC stage (ORs 8.99 to 69.70; $p < 0.001$). PVTT was strongly associated with advanced-stage disease (OR 5.63, $p < 0.001$) (Table 2). Systemic chemotherapy was the most commonly recommended treatment (48.2%). However, only 28.6% received the treatment. Notably, 48.2% of patients had no documented treatment received due to loss to follow-up (Table 3).

Conclusions: A substantial proportion of patients with hepatocellular carcinoma presented at advanced stages. This reflects gaps in surveillance adherence and late recognition of chronic liver disease. Disease severity (CTP class), poor performance status, and portal vein tumor thrombosis were strong predictors of advanced-stage presentation. In addition, there are major gaps between guideline-recommended therapy and actual treatment delivery in routine clinical practice. These findings highlight the need to strengthen HCC surveillance practices, improve early detection strategies, and enhance access to appropriate treatment in resource-limited settings.

Keyword(s): HCC, Hepatocellular carcinoma, CTP, BCLC, HBV

2179 - Correlation analysis of HBVRNA and hepatitis B virological and biochemical indicators in treated chronic HBV infected patients

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Background / Hypothesis: HBV infection is a global issue, with approximately 296 million chronic infected individuals, causing 820,000 deaths each year. Chronic hepatitis B is the main cause of liver cirrhosis and hepatocellular carcinoma

Materials and Methods: Chronic HBV-infected individuals who visited the outpatient and inpatient departments of the Affiliated Hospital of Yan 'an University from August 2024 to June 2025 were selected. According to the inclusion and exclusion

criteria, a total of 133 patients were finally enrolled and all received antiviral treatment, including 101 patients with positive HBV RNA and 32 patients with negative HBV RNA. By detecting the quantification of serum HBV RNA, the changes in the natural course of different diseases were observed, and its relationship with HBV was analyzed. The correlation between DNA, hepatitis B surface antigen (HBsAg), HBeAg, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and glutamyl transpeptidase (GGT).

Results: A total of 133 patients with chronic hepatitis B were included in this study, among which: 101 cases (75.94%) were in the RNA positive group and 32 cases (24.06%) were in the RNA negative group. The comparison between the two groups showed that there were significant difference indicators ($P < 0.05$): HBsAg (quantitative), HBeAg (quantitative), RNA qualitative results, HBsAg qualitative results, HBeAg qualitative results. No significant difference indicators ($P > 0.05$): HBV DNA (quantitative), ALT, AST, GGT, ALP (quantitative and qualitative results);

In the RNA-positive group, 101 cases (75.94%) of patients indicated that the virus had transcriptional activity, while in the RNA-negative group, 32 cases (24.06%) of patients indicated that the virus was transcriptional quistatic. Among the virological indicators HBsAg (quantitative + qualitative), the HBsAg level in the RNA-positive group was significantly higher, suggesting high expression of viral protein. In HBeAg (quantitative + qualitative), the level of HBeAg was higher in the RNA-positive group, reflecting the high infectivity of the virus. The RNA qualitative results (self-grouping variables) showed significant differences in the positive rate of RNA between groups, suggesting heterogeneity in transcriptional activity. In terms of viral load, the RNA status in HBV DNA (quantitative + qualitative) does not affect the viral replication amount (DNA level), while the RNA status in biochemical indicators such as liver function ALT/AST/GGT (quantitative + qualitative) is not related to the degree of liver injury.

Conclusions:

Keyword(s): Hepatitis B HBVRNA; HBVDNA; Hepatitis B surface antigen Clinical cure

2180 - The value of serum HBV RNA in the diagnosis treatment and prognosis of chronic

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hepatitis B

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Background / Hypothesis: HBV (hepatitis B virus) infection is a global public health issue, among which chronic hepatitis B (CHB) is the main cause of liver cirrhosis and hepatocellular carcinoma (HCC). In recent years, with the in-depth study of the life cycle of HBV, HBV pgRNA (pregenomic RNA), as a key molecule in the replication and transcription process of HBV, has gradually attracted the attention of researchers. This article will explore in detail the structural characteristics of HBV-pgRNA, its role in the course of CHB, its relationship with disease progression, and its potential applications in diagnosis and treatment.

Materials and Methods: The basic characteristics of HBV-RNA: HBV-RNA is an RNA molecule transcribed from cccDNA, including pregenomic RNA (pgRNA) and subgenomic RNA. pgRNA is the direct transcription product of cccDNA and has the functions of reverse transcription template and viral capsid assembly signal. Subgenomic RNA includes RNA encoding the large protein, medium/small protein and HBx protein of HBsAg. Splicing variants and truncated RNAs are other types of HBV RNA. HBV pgRNA has specific structural features, such as the 5' -end ϵ stem ring structure, Poly(A) tail and internal ribosome entry site (IRES), which are crucial for viral DNA synthesis, RNA stability and translation efficiency. The core functions of pgRNA include reverse transcription templates, capsid assembly signals, and cccDNA circulation hubs. Splicing variants and 3' truncated Pgrnas are associated with the occurrence of liver cancer and antiviral therapy. The generation of HBV-RNA involves the HBV replication cycle, including viral entry, rcDNA nuclear entry, transcription and translation, capsid assembly and reverse transcription, as well as viral particle release. The transcriptional regulation of cccDNA involves epigenetic modifications and viral protein feedback. The release pathways of HBV-RNA include virus-like granular and non-granular forms

The basic characteristics of HBV-RNA: HBV-RNA is an RNA molecule transcribed from cccDNA, including pregenomic RNA (pgRNA) and subgenomic RNA. pgRNA is the direct transcription product of cccDNA and has the functions of reverse transcription template

and viral capsid assembly signal. Subgenomic RNA includes RNA encoding the large protein, medium/small protein and HBx protein of HBsAg. Splicing variants and truncated RNAs are other types of HBV RNA. HBV pgRNA has specific structural features, such as the 5' -end ϵ stem ring structure, Poly(A) tail and internal ribosome entry site (IRES), which are crucial for viral DNA synthesis, RNA stability and translation efficiency.

Keyword(s) : HBV pgRNA; cccDNA; Hepatitis B virus

2181 - Analysis of the clinical efficacy of sequential combined Peg IFN treatment after NAs treatment in CHB patients

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Background / Hypothesis:
Preliminary analysis of the clinical efficacy of sequential combination of Peg-IFN after NAs treatment in CHB patients will provide real data for more CHB patients to pursue clinical cure.

Materials and Methods: From January 2018 to March 2020, CHB patients who were treated at Yan'an University Affiliated Hospital and treated with NAs for ≥ 1 year were collected. According to the inclusion and exclusion criteria, 60 patients were finally enrolled. When enrolling 30 patients in the sequential combination group, 30 NAs monotherapy patients were enrolled in a 1:1 ratio as a control. The sequential combination group continued the NAs treatment while sequentially combined with Peg-IFN α -2b; The NAs single-drug group continued to take NAs. Among them, NAs (ETV/TDF) was taken orally, once a day; Peg-IFN α -2b (Pegabine) 180ug, subcutaneously injected, once a week, observed for 48 weeks. The demographic and baseline characteristics of the two groups were collected and rechecked every 12 weeks. Follow up and record the HsHBV DNA, HBV serological indicators, liver function and four items of liver fiber, LSM of the two groups of patients, the occurrence of adverse reactions, and the corresponding measures for adverse reactions. SPSS22.0 was used for data analysis to observe the HBsAg level and the

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negative conversion rate, the negative status of HBeAg and HBV DNA, and the adverse reactions of the NAs treated CHB patients with sequential combined Peg-IFN treatment for 48 weeks

Results: 1. After 48 weeks of treatment, the HBsAg clearance rate, HBeAg conversion rate, and HBV DNA conversion rate in the sequential combination group were higher than those in the NAs single-agent group. Comparing the HBsAg levels of the two groups after 48 weeks of treatment, the sequential combination group had a high degree of HBsAg decline. Finally, 7 patients in the sequential combination group achieved HBsAg clearance, and the NAs single-drug group did not show HBsAg clearance. The HBsAg clearance rates of the two groups were respectively 23.3%, 0%, the former is significantly higher ($P=0.016$); There were more HBeAg negative patients in the sequential combination group, accounting for 93.3%, while the NAs single-drug group only accounted for 53.3%, ($P<0.001$);

Conclusion

1. For NAs treated CHB patients with sequential combination of Peg-IFN treatment, the HBsAg decline level and the rate of negative conversion, the rate of negative conversion of HBeAg, and the rate of HBV DNA conversion are better than those of continuing NAs monotherapy

Keyword(s) : Nucleoside analogues; Pegylated interferon; Sequential combination; Chronic hepatitis B; Clinical cure

2182 - Comparison of multiple drugs for metabolic dysfunction associated fatty liver disease A systematic review and network meta analysis

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Background / Hypothesis: The most prevalent chronic liver disease worldwide is metabolic dysfunction-associated fatty liver disease (MAFLD). The purpose of

this meta-analysis was to compare the efficacy of multiple drugs in treating MAFLD patients.

Materials and Methods: We searched PubMed, Embase, Web of Science and Cochrane Library databases to identify randomized controlled trials. Hepatic fat fraction (HFF) and liver stiffness assessed non-invasively by magnetic resonance imaging proton density fat fraction and elastography, serum liver enzymes, lipid profile and glucose metabolism were evaluated. Mean differences and risk ratios with 95% confidence intervals were pooled using a random-effect model.

Results: For HFF reduction, GLP-1-based poly-agonists showed significant improvement (mean difference: -42.86, 95% confidence interval [CI]: -67.97 to -22.30) compared with placebo. For liver stiffness, FGF-21 analogues were the most effective drugs (mean difference: -7.78, 95% confidence interval [CI]: -18.95 to -0.33) compared with placebo. In terms of serum liver enzymes, GLP-1-based poly-agonists appeared to be the most effective drugs compared with placebo. Regarding lipid profile, GLP-1-based poly-agonists demonstrated greater improvement compared with placebo. For glucose metabolism, GLP-1-based poly-agonists showed superior efficacy compared with placebo.

Conclusions: The present study suggests the superiority of GLP-1-based poly-agonists in improving liver steatosis, liver injury and glucose metabolism, whereas FGF-21 analogues are effective at improving liver fibrosis.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease; Glucagon-like peptide-1; Fibroblast growth factor 21; Meta-analysis;

2183 - Predictors of HBsAg Clearance and Development of a Nomogram in Chronic Hepatitis B Patients with Low Level Viremia Treated with Pegylated Interferon α 2b

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Background / Hypothesis: To develop a predictive model for hepatitis B surface antigen (HBsAg) clearance at week 48 in chronic hepatitis B patients with low-level viremia treated with pegylated interferon α -2b, based on serological and clinical parameters using the least absolute shrinkage and selection operator (LASSO) regression, and to evaluate the predictive performance of the model.

Materials and Methods: This is a prospective cohort study. A total of 303 patients with chronic hepatitis B and low-level viremia who received pegylated interferon α -2b therapy and completed 48 weeks of follow-up were included. Demographic and clinical data, including virological, biochemical, and hematological parameters at baseline and week 12, were collected. HBsAg clearance at week 48 was defined as the study outcome. Group comparisons were performed using the t test or Wilcoxon rank-sum test for continuous variables and the χ^2 test for categorical variables. Variable selection was conducted using univariate analysis and least absolute shrinkage and selection operator (LASSO) regression. Independent predictors were identified by multivariate logistic regression, and a nomogram was constructed. Model performance was assessed using the area under the receiver operating characteristic curve, bootstrap validation with 1,000 resamples, calibration curves, decision curve analysis, and clinical impact curves.

Results: Among the 303 patients with chronic hepatitis B and low-level viremia, 46 achieved HBsAg clearance at week 48. Based on univariate logistic regression ($P < 0.20$), eight variables were entered into LASSO regression. After LASSO selection, baseline albumin level (ALB.0W), HBsAg decline from baseline to week 12 (HBsAg_drop12W), and HBeAg status at week 12 (HBeAg.12W) were included in multivariate logistic regression analysis. Multivariate analysis identified HBeAg.12W (OR = 0.29, 95% CI: 0.11–0.68) and HBsAg_drop12W (OR = 0.31, 95% CI: 0.20–0.47) as independent predictors of HBsAg clearance at week 48. A nomogram based on these variables yielded an area

under the receiver operating characteristic curve of 0.879. Bootstrap internal validation with 1,000 resamples demonstrated good discrimination and stability. Calibration curves showed good agreement between predicted and observed probabilities, and decision curve analysis indicated favorable clinical net benefit across a wide range of threshold probabilities.

Conclusions: Based on baseline clinical characteristics and serological parameters, a nomogram incorporating HBeAg status at week 12 and HBsAg decline from baseline to week 12 was developed using LASSO regression. This model can predict the probability of HBsAg clearance in patients with chronic hepatitis B with low-level viremia treated with pegylated interferon α -2b, and may provide useful support for individualized clinical decision-making.

Keyword(s) : Chronic hepatitis B; Low-level viremia; Pegylated interferon α -2b; Nomogram

2184 - Related factors of fever after endoscopic treatment of variceal bleeding in patients with liver Cirrhosis complicated with esophageal variceal bleeding A retrospective study

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Background / Hypothesis:

To explore the current status of postoperative fever following endoscopic treatment for esophagogastric variceal bleeding and its impact on the prognosis of patients with liver disease.

Materials and Methods:

We retrospectively analyzed patients with decompensated cirrhosis treated endoscopically for esophagogastric variceal bleeding from December 2018 to June 2024, comparing clinical characteristics and prognosis between those with and without post-procedure fever.

Results:

A total of 251 patients treated with precise endoscopic variceal devascularization combined with endoscopic variceal sclerotherapy were included, comprising 181 males (72.1%) and 70 females (27.9%). Postoperative

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fever occurred in 94 patients (37.5%), including 62 males (24.7%) and 32 females (12.7%). The median age of the 251 patients was 56.00 (49.0, 64.0) years. Compared to the non-febrile group, the febrile group showed no statistically significant differences in age or degree of esophagogastric varices ($P > 0.05$). However, the febrile group had significantly more severe ascites and a higher proportion of Child-Pugh class C ($P < 0.001$).

Further analysis of clinical data from febrile patients revealed that the rate of postoperative bacterial infection-related fever was lower in those who received preoperative prophylactic antibiotics ($n=9$, 3.9%) compared to those who did not ($n=15$, 6.5%), a difference that was statistically significant ($P=0.001$). Alpha-fetoprotein level was associated with in-hospital bacterial infection ($r=0.184$, $P=0.049$).

Spearman correlation analysis revealed that the Model for End-Stage Liver Disease (MELD) score was positively correlated with gender, fever, infection-related fever, total bilirubin, direct bilirubin, International Normalized Ratio (INR), urea nitrogen (BUN), creatinine ($r=0.446$, $P<0.001$), Child-Pugh class, and bacterial infection. Conversely, the MELD score was negatively correlated with albumin, cholinesterase, platelet count, red blood cell count ($r=-0.280$, $P=0.002$), and hemoglobin.

Postoperative fever (OR=2.598, 95% CI 1.205–5.542, $P=0.015$), albumin (OR=0.865, 95% CI 0.789–0.939, $P<0.001$), platelet count (OR=0.990, 95% CI 0.982–0.998, $P=0.018$), coagulation time (OR=2.423, 95% CI 1.711–3.431, $P<0.001$), prothrombin activity (OR=0.879, 95% CI 0.836–0.924, $P<0.001$), and Child-Pugh class (OR=5.644, 95% CI 2.658–11.983, $P<0.001$) were identified as risk factors for a high MELD score. Multivariate regression analysis identified prothrombin time (OR=2.423, 95% CI 1.711–3.431, $P<0.001$) as an independent predictor of a high MELD score.

Conclusions:

In patients with liver cirrhosis and esophagogastric variceal bleeding undergoing endoscopic hemostasis, efforts to improve liver function, coagulation profile, and renal function, along with prophylactic anti-infection measures, may reduce postoperative complications and improve patient prognosis.

Keyword(s) : liver cirrhosis, esophagogastric variceal hemorrhage, endoscopic treatment, fever, prognosis

2186 - Demographic Characteristics and Treatment Status of Primary Biliary Cholangitis A Single Center Study from Xinjiang China.

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Background / Hypothesis:

Primary biliary cholangitis (PBC) is a chronic autoimmune intrahepatic cholestatic liver disease that predominantly affects middle-aged women. Fatigue and pruritus are the most common clinical manifestations. In recent years, the incidence of PBC has been increasing worldwide as well as in China. Understanding the demographic characteristics and current treatment status of PBC patients is essential for optimizing disease management. However, large-scale data on PBC patients in Xinjiang region of China are still limited. This study is to investigate the disease composition, demographic characteristics, and treatment status of PBC patients in a single center in Xinjiang, China.

Materials and Methods:

This retrospective study included patients diagnosed with PBC and treated at the Infectious Liver Disease Center of the First Affiliated Hospital of Xinjiang Medical University between January 2020 and October 2025. Demographic data, clinical features, immunological profiles, treatment regimens, and outcomes were collected and analyzed.

Results:

A total of 1,321 PBC patients were enrolled, accounting for 2.5% (1,321/52,640) of all liver disease patients during the same period. Among them, 76 patients (5.75%) had concomitant chronic hepatitis B, and 127 patients (9.61%) had PBC–autoimmune hepatitis overlap syndrome. Females predominated, with a male-to-female ratio of approximately 1:4 (251:1,069). The median age was 59 years (IQR 51–68). The major ethnic groups were Han (59.90%), Uyghur (22.80%), and Kazakh (9.62%). Liver cirrhosis was present in 48.83% of patients (645/1,321), and 27.33% presented with jaundice (361/1,321). Anti-mitochondrial antibody was positive in 60.33% of patients, while 78.80% were antinuclear antibody positive. Ursodeoxycholic acid (UDCA) monotherapy was administered in 84.48% of patients (1,116/1,321), with a biochemical response rate of 60.48% (799/1,321) according to Paris criteria. Notably, 7.34% of patients (97/1,321) received no treatment. During follow-up, 4.16% ($n=55$) developed hepatocellular carcinoma, 5.15% ($n=68$) progressed to liver failure, 1.81% ($n=24$) underwent liver transplantation, and 1.97% ($n=26$) died.

Conclusions:

This single-center study from Xinjiang demonstrates that PBC is not uncommon among liver disease

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patients, with a considerable proportion presenting with advanced disease and concomitant liver conditions at diagnosis. A subset of patients did not receive standardized diagnostic evaluation or treatment, highlighting the importance of early recognition and timely management. Furthermore, the suboptimal response to first-line UDCA therapy underscores the urgent need for effective second-line treatments. In addition, patient-reported outcomes such as pruritus and fatigue were not systematically assessed, suggesting insufficient attention to quality-of-life issues in routine clinical practice, education is needed.

Keyword(s) : Primary Biliary Cholangitis

2187 - Budd Chiari Syndrome Secondary to Combined Protein C Protein S and Antithrombin III Deficiencies in a Young Filipino Woman A Rare Case Report

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Background / Hypothesis:

Budd-Chiari Syndrome (BCS) is a rare hepatic venous outflow obstruction, most often linked to acquired hypercoagulable states such as myeloproliferative neoplasms. Inherited thrombophilias, particularly combined deficiencies of Protein C, Protein S, and Antithrombin III, are exceedingly uncommon causes, with limited reports worldwide. In high-tuberculosis-burden regions like the Philippines, nonspecific symptoms (ascites, jaundice, abdominal pain) often lead to diagnostic delays due to overlap with infectious etiologies. We hypothesized that comprehensive thrombophilia screening and advanced imaging are essential for identifying rare inherited causes in young patients with unexplained portal hypertension, enabling targeted management and improved outcomes.

Materials and Methods:

This case report describes a 19-year-old Filipino female presenting with progressive abdominal distension, jaundice, and ascites. Data were collected via retrospective review of clinical records, laboratory investigations (including thrombophilia panel), ascitic fluid analysis, and multimodal imaging (ultrasound, contrast-enhanced CT, and MRI with hepatobiliary contrast). Management involved multidisciplinary input from gastroenterology, hematology, infectious

diseases, and radiology. Treatment included targeted antibiotics for confirmed spontaneous bacterial peritonitis (SBP) and anticoagulation with warfarin. Ethical standards for case reporting were followed.

Results:

The patient was initially treated elsewhere with empirical anti-tubercular therapy and antibiotics, with recurrent ascites requiring drainage. Upon referral, imaging demonstrated hepatomegaly, hepatic vein obstruction, intrahepatic inferior vena cava narrowing, and patchy parenchymal enhancement consistent with BCS. Thrombophilia testing confirmed combined deficiencies of Protein C, Protein S, and Antithrombin III, establishing a rare inherited hypercoagulable state as the underlying cause. Ascitic fluid culture grew multidrug-resistant *Acinetobacter baumannii*, confirming SBP complicating BCS. After 21 days of targeted antibiotics and initiation of warfarin, the patient improved clinically with ascites resolution, jaundice regression, and stabilization of liver function. No interventional procedures were required; long-term anticoagulation and close follow-up were planned.

Conclusions:

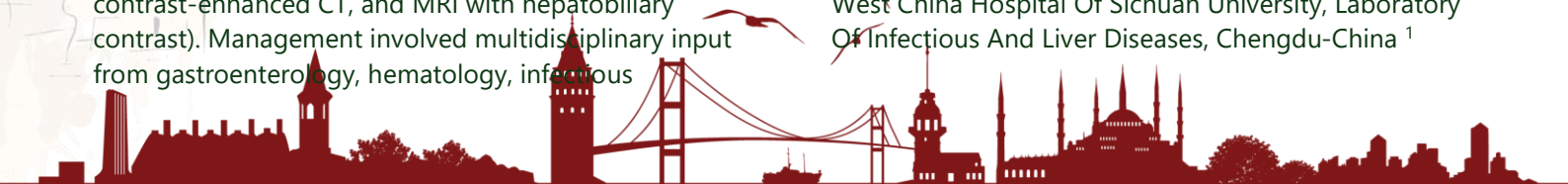
This case highlights the need for early suspicion, advanced imaging (CT/MRI), and comprehensive thrombophilia screening to diagnose Budd-Chiari Syndrome, especially in rare inherited states such as combined Protein C, Protein S, and Antithrombin III deficiencies. In TB-endemic settings, overlapping symptoms can delay recognition. Multidisciplinary care with prompt infection control and early anticoagulation led to favorable short-term outcomes and may prevent progression, adding to limited literature on triple inherited thrombophilias causing BCS.

Keyword(s) : Budd-Chiari Syndrome, inherited thrombophilia, Protein C deficiency, Protein S deficiency, Antithrombin III deficiency, hepatic venous outflow obstruction, young adult, Philippines

2190 - A novel intratumoral microbial risk score predicts survival and characterizes the immunosuppressive microenvironment in hepatocellular carcinoma

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Background / Hypothesis:

Intratumoral microbiota dysbiosis may promote hepatocellular carcinoma (HCC). We hypothesized that a genus-level intratumoral microbial risk score independently predicts prognosis and reflects key pathways and an immunosuppressive tumor microenvironment.

Materials and Methods:

We integrated intratumoral bacterial genus abundances, transcriptomic, mutational, and clinical data from the TCGA-LIHC cohort. Diversity and community composition were compared between tumor and adjacent tissues, identifying differentially abundant genera via statistical analysis of metagenomic profiles (STAMP). Cox regression analyses were performed for overall survival (OS) and disease-specific survival (DSS) to identify intersection genera significant in both. These were incorporated into a multivariate Cox model to construct a microbial risk score using a weighted linear combination of regression coefficients, stratifying patients into high- and low-risk groups. We compared KEGG pathway enrichment, immune cell infiltration, ESTIMATE scores, and tumor mutation burden (TMB) between groups. To evaluate the potential of the microbial risk score in predicting OS, ten machine learning models were compared to identify the optimal prognostic model, followed by SHAP analysis to interpret 13 key features. Finally, a nomogram integrating the risk score with clinicopathological factors was constructed.

Results:

Compared with adjacent tissues, HCC samples showed lower α diversity, separated β diversity and a reshaped community with *Paenibacillus* enrichment and depletion of *Bacillus* and other dominant genera. *Paenibacillus* and *Bacillus*, significant in both OS and DSS analyses, were used to construct the microbial risk score, which robustly separated patients into high- and low-risk groups with shorter OS in the high-risk group. High-risk tumors were enriched in extracellular matrix organization, focal adhesion and PI3K-AKT pathways and showed suppressed oxidative phosphorylation. They also exhibited higher myeloid-derived suppressor cells, regulatory T cells, plasmacytoid dendritic cells and type 2 T helper cells, together with increased stromal and ESTIMATE scores and reduced tumor purity, indicating a high-stroma, low-purity, immunosuppressive microenvironment. Driver mutation spectra and TMB differed between groups, and combining the microbial risk score with TMB further improved prognostic discrimination. Among

ten machine-learning algorithms used to assess the microbial risk score for OS prediction, extreme gradient boosting (XGBoost) performed best (AUC = 0.996). SHAP analysis of the XGBoost model showed that the microbial risk score had the highest mean absolute SHAP value and was the most important prognostic feature. The nomogram demonstrated good discrimination and calibration for predicting 1-, 3- and 5-year OS.

Conclusions:

The intratumoral microbial risk score is a powerful prognostic indicator for HCC, reflects an immunosuppressive tumor microenvironment and may facilitate individualized clinical assessment.

Keyword(s) : hepatocellular carcinoma, intratumoral microbiota, prognostic risk score, tumor microenvironment, tumor mutation burden, machine learning

2191 - Evolution of hepatocellular carcinoma staging within Egypt's national surveillance system a success story (2019–2024)

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Background / Hypothesis: In 2019, following the "100 Million Seha" initiative, which aimed at hepatitis C virus (HCV) elimination, Egypt initiated a national surveillance system for hepatocellular carcinoma (HCC). This study evaluates the impact of the national program by analyzing staging trends from 2019 through 2024.

Materials and Methods: We conducted a retrospective analysis of 25,689 patients diagnosed with HCC. The cohort was divided into two periods: 2019–2021 (n=10,882), and 2022–2024 (n=14,807). We analyzed the annual change in patient staging according to the Barcelona Clinic Liver Cancer (BCLC) system (0/A, B, C, D) to assess stage migration. Statistical significance was evaluated using Chi-square trend tests and Z-tests of independent proportions, comparing the baseline average with the 2024 dataset.

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Results: A distinct stage migration was observed following the program's implementation. In the 2019–2021 baseline, stage B accounted for only 26.0% (n=2,829) of cases. By 2024, this proportion had increased to 45.1% (n=1,746; $p<0.001$), a 1.7-fold increase. Conversely, stage D dropped significantly from 25.0% (n=2,721) at baseline to 8.9% (n=345) in 2024 ($p<0.001$). Stage C initially peaked at 41.4% (n=2,284) in 2022, but significantly declined to 30.9% (n=1,196) by 2024 ($p<0.001$ vs baseline). Stage 0/A showed a modest but statistically significant improvement, rising from 13.2% (n=1,436) to 15.2% (n=588) ($p<0.01$).

Conclusions: The transition from the 2019–2021 baseline to 2024 demonstrates a successful "shift to the left," characterized by a massive migration of patients from terminal/advanced stages into the intermediate stage (stage B). This down-staging creates a larger window for locoregional therapies, validating the national surveillance system's impact on intercepting disease progression.

Keyword(s) : Hepatocellular carcinoma, Surveillance, Staging

2193 - Serum sIL 2R Predicts Significant Histological Disease in Indeterminate Phase Chronic Hepatitis B

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Background / Hypothesis:

Many chronic hepatitis B (CHB) patients do not fit the established natural history stages and are classified as indeterminate (IM) phase. This study examines the liver histology of IM patients to inform antiviral treatment decisions.

Materials and Methods:

From 2018 to 2020, 598 treatment-naïve CHB patients were recruited from 33 Chinese medical centers. Serum levels of nine cytokines were measured using chemiluminescence immunoassay. The natural history stages were determined according to the EASL 2025 Hepatitis B Guidelines, with further stratification of IM patients based on corresponding HBeAg status and ALT levels. We analyzed liver histopathology distribution and identified independent factors for significant histological disease in the IM patients. A

nomogram was developed to evaluate significant histological disease. Liver tissue transcriptomics revealed immune and functional changes in patients with high IL2RA expression.

Results:

Of 211(35.4%) patients in IM phase (61.6% male and median age 46 years), with 74.9% showing significant histological disease. HBeAg (+) IM patients (IM-A, IM-B) had a higher rate of significant histological disease than HBeAg (-) patients (IM-C, IM-D) (90.5% vs. 68.2%, $p = 0.0012$). IM-C (57.3%) was the predominant category, followed by IM-A (26.5%). IM-B patients had the highest proportion of significant histological disease (100.0%), followed by IM-A (84.0%). Serum sIL-2R was an independent risk factor for significant histological disease in IM patients. Elevated sIL-2R levels strongly predicted significant histological injury in IM patients (middle tertile: OR = 2.29, 95% CI: 1.08–4.99, $p = 0.0337$; highest tertile: OR = 3.69, 95% CI: 1.58–9.21, $p = 0.0035$). The nomogram combining sIL-2R, AST, and ALB showed good discrimination in both training (AUC = 0.754) and validation sets (AUC = 0.746). High hepatic IL2RA expression correlated with immune activation and impaired liver function.

Conclusions:

sIL-2R is an effective biomarker for significant histological disease in IM patients. The sIL-2R-based nomogram has good performance in stratification of liver damage in IM patients.

Keyword(s) : Chronic hepatitis B, Cytokines, sIL-2R, Indeterminate Phase, Grey zone, Liver biopsy, Significant histological disease

2194 - A case of hepatocellular carcinoma developed after antiviral treatment cessation due to HBsAg seroclerance.

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Background / Hypothesis:

During the natural course of CHB, HBsAg loss is a very rare transition that indicates potential cure of HBV infection. Complete cure, or "sterilizing cure," of HBV infection implies seroclearance of HBsAg and HBV DNA as well as complete clearance of intrahepatic covalently closed circular DNA (cccDNA) and/or integrated HBV

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DNA. I report a case of hepatocellular carcinoma developed after antiviral treatment cessation due to HBsAg seroclerance.

Case :

A 74-years-old male patient admitted due to right upper quadrant pain and discomfort.

Liver CT and Liver MRI shows large heterogeneous hypodense mass with some irregular vasularities involving S8 of the liver. Liver biopsy shows hepatocellular carcinoma (HCC), Edmonson steiner grade II.

Patient's laboratory finding shows AFP 105.0ng/ml, HBsAg/Ab (-/+), IgG anti-HBc(+), HBeAg/Ab (-/-), HBV DNA : 837 international unit/ml.

Patient's past medical history began antiviral treatment for CHB 15years ago with Entecavir, and HBeAg seroclerance became six years after antiviral treatment, and HBsAg seroclerance became after 11years of antiviral treatment.

HCC occurred three years after the interruption of antiviral treatment and follow up.

Conclusions:

HBsAg loss is the state of functional cure and is associated with a reduced risk of cirrhosis. However, significant risk of HCC development remains even after HBsAg loss in male patients and in settings where HBsAg loss has been achieved late (presence of cirrhosis or age ≥ 50 years). Therefore, even if there is no cirrhosis, it is believed that patients who are elderly and have a long prevalence period will need regular follow-up tests.

Keyword(s) : HBs Ag loss, HCC

2196 - Early Risk Prediction of Hepatocellular Carcinoma in Cirrhosis Patients Based on Non Contrast CT

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Background / Hypothesis:

Although the annual incidence of hepatocellular carcinoma (HCC) in cirrhotic patients ranges from 2% to 8%, guideline-recommended surveillance combining biannual ultrasound with serum alpha-fetoprotein (AFP) misses nearly 40% of tumors ≤ 2 cm, resulting in the majority of cases being diagnosed at stages no longer amenable to curative therapies. We therefore developed and externally validated a CNN-Transformer

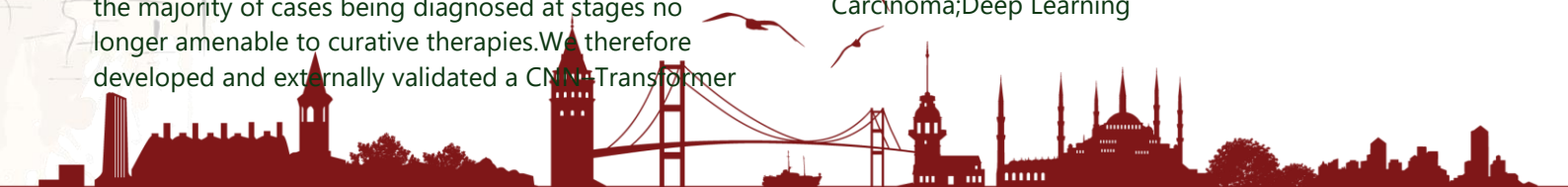
deep-learning model that uses routine non-contrast abdominal CT to predict individual HCC risk within the next 3–36 months, converting an existing imaging episode into an early, actionable warning.

Materials and Methods: This was a multicenter, retrospective-prospective cohort study designed to develop and validate a deep learning model based on non-contrast CT for predicting the risk of HCC in patients with cirrhosis. The training cohort, comprising 251 patients consecutively enrolled from two hospitals (Center A and B), was used for model development and internal validation via 5-fold cross-validation. An independent external testing cohort consisted of 1254 patients prospectively enrolled primarily from PreCar cohort (NCT03588442). For patients who developed HCC, non-contrast CT images obtained 3-36 months prior to diagnosis were collected. For those without HCC, images from their last follow-up were used. A hybrid deep learning model integrating Convolutional Neural Networks (CNN) and Transformer architectures was established to analyze the imaging data. The optimal model selected from internal validation was locked and its performance was rigorously evaluated on the independent prospective external testing cohort.

Results: The resulting model, named LCHP (Liver-Cirrhosis-to-HCC Predictor), leverages attention-based feature aggregation on a fine-tuned DenseNet121 backbone to estimate the 3-year HCC risk from a single non-contrast CT examination. In the internal training set, five-fold cross-validation yielded an AUC of 0.818 (95% CI 0.795–0.892). The internal validation set AUC was 0.829 (95% CI 0.735–0.914), and the independent prospective external test set AUC was 0.758 (95% CI 0.646–0.861), confirming robust generalisability.

Conclusions: The LCHP model quantifies 3-year HCC risk from a single non-contrast CT in patients with cirrhosis. This risk stratification allows clinicians to intensify surveillance in high-risk compensated cases and to initiate transplant evaluation earlier for those with decompensated disease, potentially shortening wait-list time and improving long-term outcomes.

Keyword(s) : Liver Cirrhosis;Hepatocellular Carcinoma;Deep Learning



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2197 - A survey of the current status of research on the diagnosis and treatment of hepatitis C virus antibody positive pregnant women

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Background / Hypothesis: To analyze the screening, diagnosis, epidemiology, pregnancy outcomes and treatment status in hepatitis C virus antibody-positive pregnant women, in order to provide clinical evidence for further improving prevention and control of maternal and infant safety.

Materials and Methods: Data of 246 HCV antibody-positive pregnant women admitted to the Second Hospital of Nanjing from January 2014 to December 2019 were analyzed by epidemiological survey research method. Statistical analysis was performed according to different data using t-test, χ^2 test, corrected χ^2 test or Fisher's exact test.

Results: 80 of 246 HCV antibody-positive women had confirmed infection before pregnancy. Of these, 85% were HCV RNA positive, and 16 became pregnant after antiviral therapy. Prenatal examination during pregnancy found that 166 cases were HCV RNA positive, and the HCV RNA positive rate was 81.93%. In the relationship between infection route and birth cohort in HCV antibody-positive pregnant women, there was a statistically significant differences in the proportions of transmission route among birth cohort ($\chi^2=115.6$, $P<0.001$). With the delay of birth cohort, the proportion of infection through drug use was decreased ($P<0.001$), while the proportion of acupuncture-associated infection ($P=0.043$) and infant hospitalization history were increased ($P=0.005$). Among pregnancy complications, HCV antibody-positive pregnant women in HCV RNA $<5.0 \text{ E}+02 \text{ IU/ml}$ and $\geq 5.0 \text{ E}+02 \text{ IU/ml}$ groups had intrahepatic cholestasis of pregnancy ($\chi^2=4.73$, $P=0.030$) and gestational hypertension ($\chi^2=8.65$, $P=0.003$), and the difference in incidence was statistically significant. Postpartum treatment strategy data analysis showed that the treatment rate was highest in the first year of postpartum, and then showed an upward trend

year by year, with a statistically significant difference ($\chi^2=17.26$, $P=0.004$).

Conclusions: Anti-HCV screening rates are lower among pregnant and reproductive age women. HCV screening should be used as an important supplementary means to strengthen maternal safety and health education management during pregnancy. Active postpartum antiviral therapy, with particularly emphasis on management within the first year after delivery, can significantly improve the treatment rate among women of child bearing age.

Keyword(s) : Chronic hepatitis C; Concurrent disease; Pregnancy

2198 - "When Natural Becomes Toxic Herbal Medicine-Induced Acute Liver Failure Treated With Double Plasma Molecular Adsorption System"

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Background / Hypothesis:

Acute liver failure (ALF) is a life-threatening condition characterized by rapid deterioration of hepatic function, coagulopathy, and hepatic encephalopathy in individuals without pre-existing liver disease. Drug-induced liver injury remains one of the leading causes of ALF, with herbal medicine-related hepatotoxicity increasingly reported, particularly in the Asia-Pacific region where traditional remedies are widely used. The double plasma molecular adsorption system (DPMAS) is an extracorporeal artificial liver support modality designed to selectively remove bilirubin, ammonia, and inflammatory mediators, potentially serving as a bridge to hepatic recovery.

Case Presentation:

We report a case of a 34-year-old previously healthy female who presented with acute alteration in sensorium and jaundice. On arrival, she was lethargic and slow to respond. Her vital signs were stable and there were no noted focal neurologic deficit. Initial evaluation revealed markedly elevated liver transaminases, hyperbilirubinemia, and coagulopathy, fulfilling diagnostic criteria for acute liver failure. The patient was admitted and managed in a multidisciplinary setting. Comprehensive diagnostic workup, including viral, autoimmune, metabolic, and radiologic investigations, failed to identify a

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conventional etiology. Detailed history subsequently revealed recent ingestion of herbal medicine, which was determined to be the most likely precipitating factor for ALF.

Management and Intervention:

The patient received standard supportive management for acute liver failure, including close neurologic monitoring and correction of metabolic derangements. IV administration of N-acetylcysteine (NAC) was initiated early, with the aim of limiting ongoing hepatocellular injury and enhancing the potential for hepatic recovery. However, in view of persistent hepatic dysfunction despite initial medical therapy, DPMAS therapy was initiated as an adjunctive liver support strategy. The procedure utilized the HA-330 II adsorption column for ammonia removal and the BS-330 adsorption column for selective bilirubin removal. DPMAS was well tolerated, with no procedure-related complications.

Results:

Following DPMAS therapy, the patient demonstrated significant clinical improvement, with resolution of altered sensorium and stabilization of coagulation parameters. Serial laboratory monitoring showed progressive improvement in liver enzymes and bilirubin levels. The patient did not require emergency liver transplantation and was discharged in stable condition with continued hepatology follow-up.

Conclusions:

This case highlights herbal medicine as an important and potentially preventable cause of acute liver failure. It also demonstrates the potential utility of DPMAS as an effective supportive therapy in ALF, facilitating toxin clearance and serving as a bridge to hepatic recovery. Early recognition of etiology and timely initiation of artificial liver support may improve clinical outcomes.

Keyword(s) : ACUTE LIVER FAILURE, HERBAL INDUCED LIVER INJURY (HILI), Double Plasma Molecular Adsorption System (DPMAS)

2199 - Bridging numerical scores and lived experience in acute and chronic liver failure

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Background / Hypothesis:

Acute and chronic liver failure present with rapid clinical deterioration, but existing prognostic scores often fail to capture fluctuating symptoms, extrahepatic complications, and traditional holistic assessments used in daily practice. Emerging studies suggest that biologic markers and clinical narratives describe different dimensions of disease burden, yet it is unclear how these perspectives intersect at the bedside. We aimed to describe this gap and to generate hypotheses for more integrative evaluation strategies.

Materials and Methods:

We performed an exploratory mixed methods study in adults with acute or chronic liver failure managed at a tertiary liver unit. Prospective clinical assessments included standard prognostic scores, serial laboratory testing, and structured symptom diaries. In parallel, we collected narrative descriptions from physicians and patients, including traditional syndrome patterns and perceived changes in vitality, appetite, and mental clarity. Qualitative and quantitative findings were compared using thematic analysis and simple pattern recognition techniques.

Results:

Across diverse etiologies and stages of liver failure, we observed frequent discordance between numerical risk estimates and patient reported experiences, especially regarding fatigue, sleep disturbance, cognitive slowing, and anxiety. Clinicians consistently described dimensions of instability and “tipping points” that were not reflected in established scores. Holistic descriptors, including pattern based terminology and contextual information on diet and stress, often anticipated abrupt deterioration or recovery before laboratory markers changed. These observations revealed recurring combinations of symptoms and contextual factors that may constitute distinct clinical phenotypes.

Conclusions:

Our exploratory work highlights a systematic mismatch between traditional prognostic scores and the lived experience of acute and chronic liver failure. Qualitative patterns and holistic assessments appear to capture early transitions in disease state that are invisible to current numerical tools. These findings support the development of hybrid prognostic approaches that combine standard scores, patient reported outcomes, and structured narrative elements, and they suggest concrete targets for future prospective validation and



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digital implementation.

Keyword(s) :

2200 - Acute Liver and Gastric Injury Complicated by Acute Kidney Injury Following Massive Ferrous Sulfate Overdose A Case Report

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Background / Hypothesis:

Acute liver injury resulting from iron toxicity is uncommon but potentially life-threatening. Massive ferrous sulfate ingestion can induce hepatocellular injury via oxidative stress, mitochondrial dysfunction, and direct iron-mediated toxicity. Iron overload may also precipitate acute kidney injury (AKI) through hemoglobinuria, tubular injury, and direct nephrotoxicity. Gastrointestinal mucosal damage is another recognized complication. Early recognition and prompt interventions, including iron chelation and extracorporeal blood purification, are critical to prevent multi-organ failure.

Case Presentation:

A 23-year-old man with major depressive disorder, noncompliant with Fluoxetine, ingested approximately 200 tablets of ferrous sulfate (13 g elemental iron, ~185 mg/kg) in a suicide attempt. He presented with coffee-ground emesis, hematuria, and oliguria. On examination, he was alert, oriented, and hemodynamically stable, without evidence of hepatic encephalopathy.

Laboratory evaluation revealed markedly elevated transaminases, mild hyperbilirubinemia, and rising serum creatinine and blood urea nitrogen, consistent with acute liver and kidney injury. Upper endoscopy demonstrated superficial ulcerations extending from the proximal gastric body to the first portion of the duodenum (D1). Workup excluded viral, autoimmune, metabolic, and structural causes of hepatocellular injury.

Management:

Supportive care included intravenous fluids, correction of metabolic derangements, and close monitoring. Deferoxamine therapy was initiated to reduce systemic iron burden. Due to persistent hepatocellular injury

and renal involvement, hemoperfusion was performed to accelerate clearance of circulating iron and toxins, followed by hemodialysis for AKI management and prevention of uremic complications. Both interventions were well tolerated.

Results:

The patient demonstrated progressive improvement in liver enzymes and bilirubin, with recovery of renal function, evidenced by normalization of urine output and resolution of hematuria. Gastrointestinal symptoms resolved. He remained free of encephalopathy and coagulopathy and was discharged in stable condition with hepatology, nephrology, and psychiatric follow-up.

Conclusions:

This case illustrates massive ferrous sulfate overdose (~185 mg/kg) as a rare cause of acute liver and gastric mucosal injury complicated by AKI without progression to liver failure. It underscores the importance of early recognition, prompt iron chelation, and the use of hemoperfusion and hemodialysis in severe multi-organ iron toxicity. Timely, multimodal intervention can prevent progression to organ failure and improve clinical outcomes.

Keyword(s) : Drug-Induced Liver Injury (DILI), Ferrous Sulfate Overdose, Acute Liver Injury

2202 - Bridging numerical scores and lived experience in acute and chronic liver failure

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Background / Hypothesis:

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Our exploratory work highlights a systematic mismatch between traditional prognostic scores and the lived experience of acute and chronic liver failure. Qualitative patterns and holistic assessments appear to capture early transitions in disease state that are invisible to current numerical tools. These findings support the development of hybrid prognostic approaches that combine standard scores, patient reported outcomes, and structured narrative elements, and they suggest concrete targets for future prospective validation and digital implementation.

Keyword(s) : acute liver failure, chronic liver failure, prognostic scores, patient reported outcomes, holistic assessment, clinical phenotypes

2203 - Why definitions of acute on chronic liver failure matter for trials and everyday decisions

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Background / Hypothesis:

Definitions of acute on chronic liver failure vary substantially across regions and societies, leading to inconsistent inclusion criteria, outcome measures, and therapeutic targets. Clinicians may therefore encounter apparently conflicting evidence when treating similar patients. We hypothesized that these definitional differences have concrete consequences for who is studied, how results are interpreted, and how recommendations are applied in routine care.

Materials and Methods:

We conducted a scoping review of contemporary clinical studies that used the term acute on chronic liver failure or closely related concepts. From each study, we extracted how investigators defined underlying chronic liver disease, acute insults, extrahepatic organ failures, and primary endpoints. We then held structured multidisciplinary discussions with hepatologists, intensivists, and methodologists using hypothetical case scenarios to explore how shifting a few definitional elements would change eligibility and perceived treatment benefit for individual patients.

Results:

We observed wide variation in the required severity of underlying liver dysfunction, in what counted as an acute precipitating event, and in the thresholds chosen to define organ failure. Some definitions focused on very short term mortality, whereas others emphasized longer term decompensation, producing overlapping but clinically distinct populations. In case based discussions, modest changes to criteria were sufficient to reclassify patients from “ineligible” to “ideal candidates” for hypothetical interventions or early transplantation. Participants also noted that reviewers and guideline authors seldom make these definitional choices explicit, leaving front line clinicians to reconcile differences on their own.

Conclusions:

Our synthesis suggests that inconsistent definitions of acute on chronic liver failure are a major hidden source of heterogeneity in the evidence base. Recognizing this problem is essential for designing future studies that

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are comparable across settings and for developing bedside tools that allow clinicians to understand which definition was used when interpreting trial results. More transparent reporting of inclusion concepts and simple crosswalks between definitions may help translate research into coherent guidance at the patient level.

Keyword(s) : acute on chronic liver failure, definitions, study design, clinical trials, guideline development, risk stratification

2204 - Fragmented collaboration between liver and addiction services in alcohol related liver disease

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Background / Hypothesis:

Alcohol related liver disease is driven by sustained harmful drinking, yet care pathways frequently prioritise management of portal hypertension and other complications over long term support for behaviour change. Reports from different systems suggest fragmented collaboration between liver and addiction services, but its concrete manifestations in daily practice are not well described. We aimed to characterise how this fragmentation is experienced by patients and clinicians and to identify leverage points for more integrated models of care.

Materials and Methods:

We undertook a qualitative study using semi structured interviews and focused group discussions with adults living with alcohol related liver disease, hepatology clinicians, nurses, and addiction specialists at a tertiary centre. Interviews explored perceptions of responsibility for alcohol counselling, barriers to referral, and experiences with shared care. Audio recordings were transcribed and analysed thematically by a multidisciplinary team, and preliminary themes were refined through feedback sessions with participants to ensure credibility and relevance to practice.

Results:

Patients frequently reported receiving mixed messages about the relative importance of abstinence compared

with control of complications, and many were unsure which part of the system “owned” their alcohol use disorder. Hepatology clinicians described time pressure, limited training in structured counselling, and concern about jeopardising rapport by repeatedly emphasising abstinence. Addiction specialists perceived referrals as late and sporadic, often triggered only after multiple decompensations. Across groups, participants highlighted simple organisational barriers, such as separate clinic locations and incompatible schedules, that reinforced the sense of two parallel rather than integrated services.

Conclusions:

Our findings reveal a pervasive disconnect between liver focused care and addiction focused care in alcohol related liver disease. This gap is sustained not only by resource constraints but also by uncertainty about roles, language, and ownership of long term behaviour change. Rather than creating entirely new services, interventions that clarify shared responsibilities, co locate key elements of care, and embed brief structured counselling tools into hepatology clinics may offer a feasible route towards more integrated and patient centred pathways.

Keyword(s) : alcohol related liver disease, addiction services, care pathways, qualitative research, multidisciplinary collaboration, patient centred care

2205 - Misaligned clinical and patient defined recovery in alcohol related liver disease

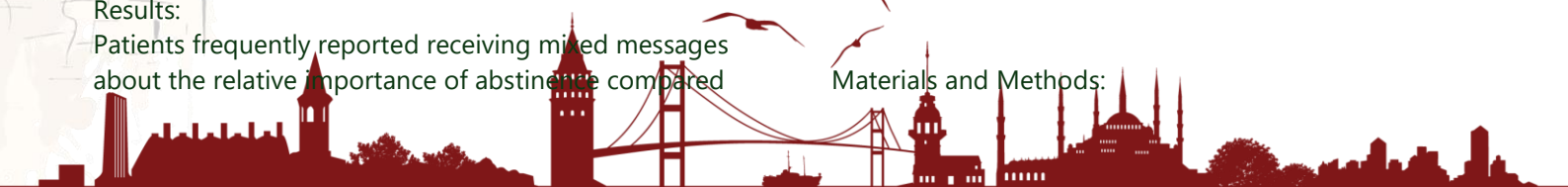
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Background / Hypothesis:

Clinical conversations about alcohol related liver disease often centre on biochemical improvement and imaging changes, while patients describe recovery in terms of energy, cognition, relationships, and control over cravings. This misalignment may weaken motivation and adherence, yet it has rarely been examined systematically. We aimed to explore how clinician defined recovery and patient defined recovery differ and what this means for planning follow up care.

Materials and Methods:



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We conducted a small exploratory cohort study of adults with alcohol related liver disease who had recently achieved sustained abstinence. Over a six month period, participants attended combined hepatology and psychosocial follow up visits. At each visit, routine liver tests and global clinical impressions of stability were recorded. In parallel, participants completed brief questionnaires on fatigue, mood, cognition, sleep, and social participation, and provided short free text accounts of what "feeling recovered" meant to them at that moment.

Results:

Clinicians tended to regard recovery as beginning once liver tests had stabilised and admissions for decompensation had ceased, whereas many patients described this as only the first step. Participants frequently reported that concentration, sleep, and confidence in social situations improved more slowly than blood results, and some experienced new anxiety when visits became less frequent. The language used by patients to describe recovery emphasised regaining meaningful roles, trust within families, and the ability to manage cravings, with little reference to numerical thresholds. These narratives suggested that clinic discharge criteria may not fully reflect patient priorities.

Conclusions:

Our exploratory work illustrates a persistent gap between biochemical recovery and lived recovery in alcohol related liver disease. Relying solely on laboratory and imaging improvement may lead clinicians to underestimate ongoing cognitive and psychosocial burdens. Integrating brief patient reported measures and explicit discussions of recovery goals into routine follow up may help align expectations, support sustained abstinence, and guide decisions about the timing of step down or continued multidisciplinary care.

Keyword(s) : alcohol related liver disease, recovery, patient perspective, follow up care, patient reported outcomes, psychosocial factors

2206 - Silent degradation of artificial intelligence tools for liver disease in routine care

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Background / Hypothesis:

Artificial intelligence tools for liver disease increasingly report expert level accuracy, yet most are developed within single datasets and then quietly deployed elsewhere. When models encounter new scanners, documentation styles, or patient populations, performance may deteriorate in ways that are hard to detect. We hypothesised that clinicians experience this "silent" degradation as a specific kind of risk and that describing their experiences could guide more realistic monitoring strategies.

Materials and Methods:

We carried out a qualitative multi stakeholder study at centres that had piloted artificial intelligence assisted imaging tools for liver disease. Semi structured interviews and workshops were conducted with hepatologists, radiologists, sonographers, and information technology staff. Participants were invited to recount examples of unexpected model behaviour, local responses to perceived errors, and informal practices for monitoring performance over time. Transcripts were analysed using inductive thematic analysis, with specific attention to perceptions of risk, trust, and acceptable thresholds for intervention.

Results:

Participants described an early phase of enthusiasm followed by growing uncertainty as they encountered cases in which artificial intelligence suggestions conflicted with clinical judgement or later outcomes. Most centres lacked formal dashboards or predefined rules for recalibration, relying instead on informal feedback from a few "super users." Clinicians worried that gradually worsening performance might remain unnoticed until a serious error occurred, particularly in busy services where manual audits were unrealistic. At the same time, they expressed reluctance to rely on opaque aggregate metrics that did not clearly translate into risk for individual patients.

Conclusions:

Our study highlights performance drift and silent degradation as under recognised challenges when artificial intelligence tools are moved from development environments into routine liver care. Clinicians articulated a need for simple and interpretable safeguards, such as periodic review of

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discordant cases, real time discrepancy flags, and clear procedures for temporarily suspending or retraining models. These user driven insights can inform monitoring frameworks that respect clinical workflows while providing a pragmatic layer of protection for patient safety.

Keyword(s) : artificial intelligence, liver disease, model drift, patient safety, implementation, qualitative study

2207 - Making artificial intelligence in hepatology more holistic and aligned with clinical reasoning

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Background / Hypothesis:

Most artificial intelligence research in hepatology focuses on imaging data and routine laboratory values, while patient reported outcomes and holistic descriptors used in integrative medicine are rarely captured. This narrow view of the patient may limit the relevance and acceptability of artificial intelligence tools. We hypothesised that an audit of current documentation practices could reveal simple opportunities to make future models more reflective of real world clinical reasoning.

Materials and Methods:

We performed an observational documentation audit in hepatology clinics at an academic centre where both conventional and traditional medical frameworks are applied. For consecutive patients with chronic liver disease, we recorded which elements of the consultation were stored in structured fields, which were present only in free text, and which were routinely discussed but not documented at all. We then convened workshops with clinicians, data scientists, and record specialists to prototype alternative templates that could capture additional holistic information in a concise, machine readable format.

Results:

Laboratory values and imaging summaries were consistently structured, whereas symptom patterns, lifestyle context, and traditional syndrome labels were scattered across narrative notes or left undocumented.

Clinicians reported that these under recorded elements played a major role in decisions about follow up timing, suitability for lifestyle or herbal interventions, and perceptions of resilience, yet they were effectively invisible to current artificial intelligence approaches. Workshop participants identified a small set of brief check boxes and scales that could capture much of this information without lengthening visits, while still preserving space for nuanced narrative descriptions.

Conclusions:

Our exploratory work reveals a striking mismatch between the information that shapes everyday hepatology decisions and the data typically available to artificial intelligence models. By modestly redesigning documentation to include concise structured fields for patient experience and holistic assessments, it may be possible to build artificial intelligence systems that are better aligned with clinician reasoning and with integrative models of care, thereby improving both usefulness and trust at the point of care.

Keyword(s) : hepatology, artificial intelligence, documentation, holistic assessment, integrative medicine, clinical reasoning

2208 - How endocrine information is missing from contemporary chronic liver disease research

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Background / Hypothesis:

Endocrine pathways involving thyroid hormones, sex steroids, and adrenal function are increasingly recognised as modulators of liver injury and repair. Nevertheless, major studies in chronic liver disease rarely include detailed endocrine profiling, making it difficult to translate mechanistic insights into clinical tools. We hypothesised that mapping how endocrine variables are currently represented in hepatology research would reveal specific gaps and opportunities for a more integrated endocrine hepatology agenda.

Materials and Methods:

We performed a methodological review of recent clinical studies in chronic liver disease that reported on natural history, prognostic scores, or treatment

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outcomes. For each study, we recorded whether thyroid, gonadal, or adrenal measurements were obtained, how these measurements were categorised or analysed, and whether they were discussed as potential modifiers of liver related outcomes. Informal interviews with selected investigators were used to explore practical reasons for including or excluding endocrine variables during protocol development.

Results:

Across a broad range of study designs and disease etiologies, endocrine measurements were rarely treated as core data elements. When hormonal values were collected, they were often reduced to simple binary categories or used as exclusion criteria, rather than explored as mechanistic contributors to variation in liver outcomes. Investigators cited limited funding, competing priorities for laboratory testing, and uncertainty about which assays would be most informative as reasons for omission. Several also noted that dialogue between liver specialists and endocrinologists during protocol design was either brief or absent, leading to missed opportunities for targeted substudies.

Conclusions:

Our review underscores a systematic under representation of endocrine information in contemporary chronic liver disease research. This imbalance limits the ability to test endocrine based hypotheses and may slow the translation of endocrine hepatology concepts into practice. Prioritising a small set of hormonal markers, embedding them into standard data dictionaries, and fostering closer collaboration between hepatology and endocrinology teams may help bridge the current divide and support more mechanistically informed clinical studies.

Keyword(s) : endocrine hepatology, chronic liver disease, thyroid hormones, sex steroids, study design, data elements

2209 - Overlapping metabolic reproductive and hepatic disturbance in women with fatty liver disease

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Background / Hypothesis:

Women with fatty liver disease frequently report irregular menses, features of androgen excess, and central adiposity, yet these disturbances are seldom viewed through an integrated endocrine hepatology lens. Clinical experience suggests that such constellations may mark a subgroup with distinct risks and concerns, especially regarding fertility and long term metabolic health. We aimed to describe how this overlapping pattern presents in routine practice and how hepatologists currently respond to it.

Materials and Methods:

We undertook a descriptive case series of women attending a fatty liver clinic who reported menstrual irregularity or clinical signs of androgen excess during routine assessment. For each case, we summarised liver disease severity, metabolic risk profile, reproductive history, and any available endocrine investigations. Brief interviews with treating hepatologists were used to explore referral patterns to gynecology or endocrinology, perceived links between hormonal disturbance and liver outcomes, and practical challenges in coordinating cross specialty care.

Results:

Across cases, we observed recurring clusters of central adiposity, insulin resistance, menstrual disturbance, and clinical hyperandrogenism. However, documentation of formal endocrine diagnoses and targeted hormone testing was inconsistent, and many patients had never been offered a structured discussion of reproductive planning or endocrine risks. Hepatologists often suspected underlying syndromes such as polycystic ovary syndrome but expressed uncertainty about when to initiate referral and which aspects of management should take precedence. Several described liver focused visits as poorly suited to address questions about fertility, contraception, or long term endocrine follow up despite obvious patient interest.

Conclusions:

Our descriptive work points to a recognisable but under addressed pattern of overlapping metabolic, reproductive, and hepatic disturbance in women with fatty liver disease. The current separation between liver and endocrine services leaves many patients navigating fragmented advice and uncoordinated follow up.

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Developing shared assessment pathways and joint clinics that explicitly integrate endocrine and hepatology perspectives may better meet patient needs and create a practical platform for future mechanistic and interventional studies in endocrine hepatology.

Keyword(s) : endocrine hepatology, fatty liver disease, women health, reproductive disturbance, metabolic risk, integrated care

2210 - "Demographic Characteristics of MAFLD with Emphasis on Young Onset Disease in Pakistan

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly being diagnosed at a younger age worldwide. In Pakistan, data regarding the burden of MAFLD among young individuals is limited. We hypothesized that a major proportion of MAFLD patients in Pakistan present at a young age, indicating an emerging public health concern.

Materials and Methods:

This observational study included 211 patients diagnosed with MAFLD. Demographic data including age, sex, and area of residence were recorded. Patients were categorized into two age groups: ≤ 45 years and > 45 years. Descriptive statistical analysis was performed to evaluate the proportion of MAFLD in the younger population and to describe demographic characteristics.

Results:

The mean age of the study population was 44 years, with the youngest patient aged 16 years. Ninety-five patients (45.0%) were aged 45 years or younger, indicating a high proportion of young individuals affected by MAFLD. The cohort consisted predominantly of females ($n=158$), followed by males ($n=52$) and one transgender individual. A majority of patients belonged to urban areas ($n=173$), while 38 patients were from rural regions.

Conclusions: Almost half of the MAFLD patients in this cohort were aged 45 years or younger, exhibiting an early age of presentation in the Pakistani population.

The findings suggest a rising burden of MAFLD in younger individuals, particularly in urban settings. Early identification, lifestyle interventions, and targeted screening strategies are warranted to alleviate long-term liver-related and cardiometabolic complications.

Keyword(s) :

2211 - Frequency of metabolic dysfunction associated steatotic liver disease (MASLD) amongst patients with inflammatory bowel disease (IBD)

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Background / Hypothesis:

There is paucity of data regarding the frequency of metabolic dysfunction associated steatotic liver disease (MAFLD) amongst patients with inflammatory bowel disease (IBD) in south Asia. This cross-sectional comparative study was conducted to find the frequency of metabolic dysfunction associated steatotic liver disease (MASLD) amongst patients with inflammatory bowel disease (IBD).

Materials and Methods:

This cross-sectional comparative study was conducted at the IBD department of the Asian Institute of Gastroenterology Hospitals, Hyderabad, India from June to December in 2022.

Results:

IBD was 199 patients while Non IBD was 119 patients. One third of the IBD patients had MAFLD. BMI was significantly less among the IBD patients compared to the controls ($p < 0.002$). Significant differences existed regarding smoking habits among the cases and controls where IBD patients had more non smokers and ex smokers whereas as control groups had more current smokers ($p < 0.002$). Liver enzymes namely SGOT and SGPT were significantly higher amongst the control groups ($p < 0.001$). IBD patients had significantly lower FBS compared to the controls ($p = 0.44$). IBD patients had significantly higher Triglycerides level in contrast to the controls ($p < 0.020$). MASLD occurred in comparatively older age in contrast to the controls ($p < 0.001$). Patients having both IBD and MASLD had higher BMI ($p < 0.001$), greater waist circumference ($p < 0.001$), and a higher incidence of Diabetes Mellitus ($p < 0.039$), suggesting that this cohort could be at elevated risk for metabolic complications.

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Conclusions:

Metabolic dysfunction associated fatty liver disease (MASLD) needs to be addressed while treating inflammatory bowel disease patients.

Keyword(s) :

2212 - Regorafenib as a second line therapy for Egyptian patients with an advanced stage HCC real life data from a single center

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Background / Hypothesis:

Regorafenib has been approved as one of the treatment options for patients with advanced stage colorectal cancer (CRC), hepatocellular carcinoma (HCC), and gastrointestinal stromal tumors (GIST). Aim: To assess the efficacy and safety of regorafenib in Egyptian patients with HCC who progressed on sorafenib treatment, Assessment of drug adverse effects and Assessment of tumor response according to modified Response evaluation criteria in solid tumors (mRECIST).

Materials and Methods:

We conducted a prospective study on 248 patients with advanced HCC who were given sorafenib as first line therapy, 55 patients shifted directly to regorafenib after failure to respond to sorafenib therapy according to (mRECIST) criteria, at National Liver Institute (NLI), Menofia, Egypt from January 2022 to June 2024. Data collected include aetiology, patient demographics, performance status, duration of previous line of treatment, number of treatment cycles, side effects, best-tolerated dose, laboratory, and radiological results and treatment discontinuation due to intolerance. The outcomes included overall survival (OS) and progression-free survival (PFS).

Results:

A total of 55 from 248 patients received regorafenib as a 2nd-line treatment after sorafenib progression. Regarding the BCLC staging system, (61.7%) were in BCLC stage B and (38.3%) were in BCLC stage C. 24 of patients (43.6%) had progressive course of the disease with mean disease free progression period 14.44 [95% confidence interval (CI): 11.29-17.59] months, 36 of

patients (65.45%) died. The mean survival time was 16.78 (95% CI, 13.832 -19.742) months. The most common toxicities were liver decompensation (32.6%), hand-foot skin reaction (13.5%), diarrhea (9.8%). Regarding sorafenib, the mean disease free progression period 18.24 [95% confidence interval (CI): 15.29-21.19] months, 93 of patients (48.18%) died. The mean survival time was 22.253 (95% CI, 19.734 -24.266) months.

Conclusions:

Regorafenib appears to be safe and clinically effective in patients with advanced HCC who progressed on first-line sorafenib. Regorafenib could effectively improve the prognosis of patients after first-line treatment failure.

Keyword(s) : HCC, Regorafenib

2213 - Pain Management Gaps in Hepatocellular Carcinoma A Cross Sectional Survey Study of Patient Perceptions Misconceptions and Unmet Needs

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Background / Hypothesis: Adequate cancer pain control is crucial for quality of life and anti-cancer treatment compliance. However, patients with hepatocellular carcinoma (HCC) with underlying liver cirrhosis often experience inadequate pain management due to misconceptions about analgesics and liver toxicity concerns. This study investigated pain perception and management practices among patients with HCC.

Materials and Methods: A structured 30-item questionnaire was administered to 200 patients with HCC (aged 19-79 years) at Asan Medical Center, Seoul, from January to November 2025. The survey assessed pain presence and intensity (12 items), perceptions of cancer pain (5 items), attitudes toward analgesics including opioids (8 items), and needs for improved pain management (5 items). Statistical analyses included Mann-Whitney U test and Spearman correlation.

Results: Among 200 respondents (mean age 63.3±8.7 years; 87% male), 55 (27.5%) reported current pain with mean intensity of 3.7/10 (maximum pain 5.1/10, minimum pain 1.7/10). Of 55 patients with pain, only 31 (56.4%) were taking analgesics while 24 (43.6%)

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remained untreated despite significant pain. Patients on analgesics experienced higher pain intensity (mean 4.0 vs 3.1, maximum pain 6.2 vs 3.8, $p=0.0007$), suggesting both inadequate pain control in treated patients and undertreatment in non-users. Paradoxically, while 54% acknowledged pain should not be inevitable, 69.9% worried about liver damage from analgesics. Opioid-related misconceptions were significantly higher among older patients ($r=0.189$, $p=0.009$) and married/divorced individuals (53.8-56.3%) compared to unmarried patients (15.0%, $p=0.004$). Over 80% requested dedicated pain management nurses and comprehensive analgesic education.

Conclusions: Patients with HCC demonstrate critical gaps in pain management with substantial undertreatment and misconceptions about analgesic safety, particularly regarding liver toxicity and opioid use. Age- and marital status-targeted educational interventions with dedicated pain counseling services are urgently needed to improve pain control in this vulnerable population.

Keyword(s) :

2216 - Gut derived microbial nucleic acids modulate hepatic innate immune responses in steatotic liver disease

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Background: Steatotic liver disease (SLD), including metabolic dysfunction-associated steatotic liver disease (MASLD) and alcohol-related liver disease (ALD), is increasingly recognized as a condition driven by dysregulation of the gut-liver axis. Disruption of the intestinal barrier allows translocation of gut-derived microbial components, including nucleic acid-derived pathogen-associated molecular patterns (PAMPs), into the portal circulation. These microbial molecules can activate hepatic innate immune responses through pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs), RIG-I-like receptors (RLRs), and NOD-like receptors (NLRs). However, direct human evidence linking portal microbial translocation with intrahepatic PRR expression remains limited. This study aimed to clarify the association between portal microbial nucleic acids and hepatic PRR expression in patients with SLD with particular focus on sex- and etiology-related differences. **Methods:** Liver biopsy specimens from 62

patients with SLD (MASLD, $n = 44$; ALD, $n = 18$) were analyzed to assess intrahepatic expression of PRRs, including TLR2, TLR3, TLR4, TLR5, TLR9, RIG-I, MDA5, NOD1, and NOD2. Portal venous blood samples were additionally obtained from eight patients undergoing endoscopic variceal treatment (MASLD, $n = 3$; ALD, $n = 3$; other etiologies, $n = 2$). Stool, portal blood, and peripheral blood samples were analyzed using 16S rRNA gene sequencing to characterize microbial nucleic acid profiles and to evaluate microbial translocation into the portal and systemic circulation. **Results:** Female patients exhibited significantly higher hepatic expression of RIG-I and TLR3 than male patients, with a trend toward increased MDA5 expression. In contrast, no significant sex differences were observed for TLR2, TLR4, TLR5, TLR9, NOD1, or NOD2. Disease-based analyses demonstrated significantly higher hepatic expression of RIG-I and TLR4 in patients with ALD compared with those with MASLD. Microbial taxa shared between stool and portal blood were detected in seven of eight patients, including Enterobacteriaceae, Clostridia, Bacteroidia, and Veillonella, indicating frequent portal microbial nucleic acid translocation. Translocation of microbial signatures into peripheral blood was observed more frequently in ALD than in MASLD, suggesting enhanced systemic exposure in ALD. **Conclusion:** Gut-derived microbial components enter the portal circulation and may modulate hepatic innate immune responses in SLD. Variations in PRR expression according to sex and disease etiology likely reflect differences in the amount and characteristics of microbial molecules translocating from the gut to the liver.

Keyword(s) :

2217 - The Impact of Prophylactic Antibiotics on the Prognosis of Patients with Cirrhosis and Upper Gastrointestinal Bleeding

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Background / Hypothesis:

The optimal duration of prophylactic antibiotic therapy and the most suitable patient population for its use in patients with cirrhosis and upper gastrointestinal bleeding remain unclear. This study aimed to investigate the effects of different durations of

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prophylactic antibiotics on clinical outcomes in patients with varying risk levels.

Materials and Methods:

A retrospective analysis was conducted on the medical records of 199 patients with cirrhosis and upper gastrointestinal bleeding admitted to the Affiliated Hospital of Xuzhou Medical University from January to December 2023. Based on the use of prophylactic antibiotics, patients were divided into an antibiotic group (89 cases) and a non-antibiotic group (110 cases). Clinical outcomes including rebleeding, readmission, mortality, and infection rates were compared between the two groups. The impact of different Child-Pugh classifications and antibiotic treatment durations on prognosis was also analyzed.

Results:

Within 60 days after discharge, the incidence of rebleeding in the antibiotic group (29.2%) was significantly lower than that in the non-antibiotic group (50.0%), and the difference was statistically significant ($P=0.04$). There were no statistically significant differences between the two groups in readmission rates (33.7% vs. 36.4%), infection rates (25.8% vs. 16.4%), or mortality rates (19.1% vs. 30.0%) (all $P>0.05$). Among patients with Child-Pugh class C cirrhosis, the survival prognosis in the antibiotic group was significantly better than that in the non-antibiotic group (Log Rank $P=0.018$). However, among patients with Child-Pugh class A/B cirrhosis, there was no statistically significant difference in 60-day post-discharge survival prognosis between the two groups (Log Rank $P=0.188$). The mortality rate in the group with an antibiotic course > 6 days (28.2%) was higher than that in the group with a 1~3 day course (8.0%), and the difference was statistically significant ($P=0.049$).

Conclusions: Prophylactic antibiotics can reduce the risk of rebleeding in patients with cirrhosis and upper gastrointestinal bleeding and improve the survival prognosis of patients with Child-Pugh class C cirrhosis. A short-course prophylactic regimen (1~3 days) may be more advantageous, and caution is advised when considering its use in low-risk patients.

Keyword(s) :

2219 - Higher matrix stiffness accelerates liver fibrosis by modulating fatty acid oxidation via PPAR α CPT1A signaling axis in hepatic stellate cells

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Background / Hypothesis:

Matrix stiffness is a key mechanical feature during hepatic fibrosis progression. Fatty acid oxidation (FAO) is a crucial biological process regulating hepatic stellate cell (HSC) activation, which drives fibrosis. This study aimed to investigate the effect of matrix stiffness on FAO in HSCs and its underlying regulatory mechanism during hepatic fibrogenesis.

Materials and Methods:

An in vitro cell culture platform was established to mimic liver tissue stiffness at different pathological stages (1, 8, 16, 32 kPa). The human HSC line LX2 was cultured on these substrates. Activation markers (α -SMA, Collagen I, TIMP1) and key FAO enzymes (CPT1, ACOX1) were detected by Western blot and qRT-PCR. Transcriptome sequencing was used to identify mechanosensitive targets. PPAR α function was validated using agonist treatment and overexpression experiments. A CCl₄-induced mouse model of hepatic fibrosis was established. Histopathological staining assessed fibrosis, and immunohistochemistry verified key molecule expression.

Results:

Increased matrix stiffness significantly upregulated the expression of α -SMA, Col1a1, and TIMP1 in LX2 cells, accompanied by decreased expression of FAO-related genes such as CPT1 and ACOX1. Transcriptome analysis and validation showed that high matrix stiffness markedly downregulated PPAR α expression and inhibited its nuclear translocation. Treatment with the PPAR α agonist WY14643 significantly increased CPT1 expression and FAO levels, thereby inhibiting HSC activation induced by high stiffness. In vivo, the PPAR α agonist significantly attenuated the degree of liver fibrosis in model mice.

Conclusions:

High matrix stiffness promotes HSC activation and drives hepatic fibrosis progression by suppressing the PPAR α -CPT1 signaling axis and subsequently inhibiting fatty acid oxidation in HSCs.

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Keyword(s) : matrix stiffness, liver fibrosis, fatty acid oxidation, LX2

2220 - Prospective Study on Prognostic Improvement and Suppression of Hepatocellular Carcinoma Development after Zinc Supplementation in Patients with Liver Cirrhosis

Background and Aim

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Background / Hypothesis:

Serum zinc levels decrease with the progression of hepatic fibrosis in patients with chronic liver disease, and zinc deficiency is a known risk factor for the development of hepatocellular carcinoma (HCC). In our previous retrospective study, zinc supplementation was associated with reduced mortality, and decreased HCC development. This prospective study aimed to evaluate whether long-term zinc supplementation improves prognosis, suppresses HCC development, and prevents deterioration of liver function in patients with cirrhosis treated with zinc acetate (Novelzin) in routine clinical practice.

Materials and Methods:

Among 1,067 consecutive patients diagnosed with liver cirrhosis between May 2018 and October 2023, 563 patients were enrolled after excluding those with a history of HCC, an observation period of less than three months, or lack of informed consent. Patients were classified into a zinc-taking group (n=130) and a zinc-non-taking group (n=433). Changes in laboratory parameters, overall survival, and incidence of cancer development were compared between the two groups.

Results:

The baseline characteristics of the cohort were as follows: mean age 70.4±12.3 years; male/female ratio 343/220; etiology alcohol/HBV/HCV/NASH 231/53/123/105; total bilirubin 1.8±2.5 mg/dL; albumin 3.23±0.73 g/dL; prothrombin time (PT) 70.9±20.1%; serum zinc 56.3±20.3 µg/dL; and mean observation period 17.7±18.5 months. In the zinc-taking group, serum zinc levels, albumin, and PT significantly improved at six months after treatment compared with baseline (51.8→87.6 µg/dL, 3.03→3.35 g/dL, and 66.3→71.7%, respectively; all p<0.01). The zinc-taking group showed significantly better outcomes than the zinc-non-taking group, with a higher 3-year survival rate (69.0% vs. 56.2%, p=0.035) and a lower 3-year

incidence of cancer development (1.5% vs. 8.1%, p=0.043).

Conclusions:

Consistent with our retrospective findings, prospective zinc supplementation significantly improved liver function, reduced HCC incidence, and enhanced survival in patients with cirrhosis. Zinc supplementation may represent an effective therapeutic strategy for cirrhosis regardless of underlying etiology.

Keyword(s) : zinc, cirrhosis, HCC, prognostic improvement

2221 - FABP4 as a Prognostic Biomarker and Its Clinical Relevance in Drug Induced Cholestasis

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Background / Hypothesis:

Drug-induced cholestasis (DIC) is a relatively severe and prognostically challenging subtype of drug-induced liver injury (DILI). It is often characterized by a protracted clinical course, a high risk of chronicity, and prolonged hospitalization, imposing a significant burden on patients and healthcare systems. Currently, there is a notable lack of reliable biomarkers for predicting disease progression and outcomes in DIC. Fatty acid-binding protein 4 (FABP4), a key mediator in metabolic inflammation, has not been well characterized in this clinical context. This study aimed to investigate the association between FABP4 and disease severity/prognosis in patients with DIC.

Materials and Methods:

Eighty DILI patients (subtyped as hepatocellular [H-DILI, n=40] and cholestatic [C-DILI, n=40]) and 35 healthy controls (HC) were enrolled. Hepatic FABP4 expression was assessed using immunohistochemistry (IHC) and Western blot, while plasma FABP4 levels were measured via enzyme-linked immunosorbent assay (ELISA). Correlations between FABP4 levels, clinical indicators (total bilirubin [TBil]), and outcomes were analyzed. Complementary mouse models of ANIT (cholestatic) and acetaminophen (APAP, hepatocellular) liver injury were used to validate FABP4 expression patterns.

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Results:

C-DILI patients and H-DILI patients exhibited significantly elevated FABP4 expression in liver tissue and plasma compared to HC (Figure A-D, $P < 0.05$). Serum FABP4 levels showed strong positive correlations with liver injury grade ($r = 0.4299$, $P < 0.0001$) and TBil ($r = 0.4016$, $P = 0.0002$) and disease severity, reaching peak values in patients requiring liver transplantation or nonsurvival (Figure D-E, $P < 0.05$). Conversely, FABP4 exhibited significant negative correlations with serum cholesterol ($r = -0.4352$, $P = 0.0003$), LDL ($r = -0.5249$, $P < 0.0001$), and HDL ($r = -0.4948$, $P < 0.0001$) (Figure F, H). Plasma FABP4 demonstrated significant prognostic value for C-DILI outcomes (Figure G, $P < 0.05$). In murine models, both ANIT and APAP-induced liver injury triggered substantial FABP4 upregulation (Figure I-M, $P < 0.05$), with spatial expression patterns highly colocalized with inflammatory infiltrates (F4/80+ macrophages, MPO+ neutrophils) and necrotic areas.

Conclusions:

FABP4 serves as a novel biomarker for disease severity and prognosis in DIC patients. Its expression levels enable clinical stratification and outcome prediction, providing a new tool for precise diagnosis and management of DIC.

Keyword(s) : Drug-induced Cholestasis, FABP4, Prognosis, Biomarker

2222 - The correlation between autoantibody spectrum and clinical pathological phenotype of Primary Biliary Cholangitis Patients

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Background / Hypothesis:

Primary biliary cholangitis (PBC) is characterized by a broad spectrum of autoantibodies, but their correlations with clinical phenotypes remain to be fully elucidated.

Materials and Methods:

This retrospective study included patients who underwent liver biopsy were diagnosed with PBC at Beijing Ditan Hospital. The prevalence of various autoantibodies—including anti-mitochondrial antibody

(AMA), antinuclear antibody (ANA), anti-centromere antibody (ACA), anti-Ro-52, anti-SP100, and anti-gp210—was analyzed. Patients were stratified based on autoantibody status (e.g., anti-SP100 positive/negative, anti-gp210 positive/negative), and their demographic, biochemical, immunological, and histopathological features were compared using appropriate statistical tests. Correlation analysis was performed between AMA titer and key liver biochemical indicators.

Results:

In the cohort, A total of 218 PSVD patients undergoing liver biopsy were enrolled, and 69.72% were AMA-positive and 68.81% were ANA-positive. The positive rates for anti-gp210, anti-Ro-52, anti-SP100, and ACA were 39.91%, 37.16%, 20.64%, and 17.43%, respectively. Comparative analysis revealed that anti-SP100-positive patients had a significantly higher ANA-positive rate (93.33% vs. 62.43%, $p = 0.007$). Anti-gp210-positive patients exhibited higher levels of total bile acid (TBA) (median 19.90 vs. 11.70 $\mu\text{mol/L}$, $p = 0.038$), total cholesterol (TC) (5.21 vs. 4.75 mmol/L , $p = 0.044$), and IgM (2.84 vs. 2.20 g/L , $p = 0.016$), as well as a higher ANA-positive rate (85.06% vs. 58.02%, $p < 0.001$) and AMA-positive rate (74.71% vs. 61.83%, $p = 0.024$). AMA titer showed significant positive correlations with ALT, AST, GGT, ALP, and IgM levels (all $p < 0.05$). No significant differences were found in the prevalence of advanced fibrosis between the different antibody subgroups.

Conclusions:

The autoantibody profile in PBC patients is heterogeneous. Anti-gp210 positivity is associated with distinct biochemical features (elevated TBA, TC, and IgM), while anti-SP100 positivity is strongly linked to ANA seropositivity. AMA titer correlates with disease activity markers. These findings highlight the clinical relevance of the autoantibody spectrum in stratifying PBC phenotypes.

Keyword(s) : primary biliary cholangitis, anti-mitochondrial antibody, anti-SP100 antibody, anti-gp210 antibody, pathological stage

2223 - Hepatic IGFBP3 Attenuates Osteoblastic Differentiation in Cirrhotic Liver-Bone Axis via NF- κ B Signaling

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Background / Hypothesis: With the rising prevalence of aging and metabolic disorders, secondary osteoporosis is increasingly common, particularly among patients with liver cirrhosis. Cirrhosis contributes to abnormal bone metabolism, representing a significant risk factor for osteoporosis, though the underlying mechanisms remain unclear. Insulin-like growth factor binding protein 3 (IGFBP3), primarily synthesized in the liver, serves as the major binding protein for insulin-like growth factor 1 (IGF-1) and plays a key role in bone development and remodeling by modulating IGF-1 bioavailability. However, the specific function of IGFBP3 in cirrhosis-associated osteoporosis still requires further clarification.

Materials and Methods: We established a mouse model of cirrhosis-osteoporosis comorbidity by combining ovariectomy with carbon tetrachloride (CCl₄) administration and intervened with IGFBP3. Liver pathology was assessed by histology and serum liver function tests. Bone microarchitecture and morphology were evaluated via micro-CT and histochemical staining. Hepatic and bone IGFBP3 levels, along with osteogenic markers, were determined by Western blotting and immunohistochemistry. Weighted gene co-expression network analysis (WGCNA) was applied to a proteomic dataset from cirrhosis and osteoporosis to identify core differentially expressed genes, which were then subjected to KEGG pathway enrichment analysis and validation. For in vitro studies, an AML12 hepatocytes/MC3T3-E1 osteoblasts co-culture system was established. Following LPS induced liver impairment, IGFBP3 was administered, and Western blotting and RT-qPCR were used to examine osteogenic markers (ALP, OCN, and Runx2) and changes in the NF- κ B signaling pathway.

Results: In vivo, mice with cirrhosis alone or cirrhosis combined with osteoporosis demonstrated exacerbated hepatic inflammation, fibrosis progression, and impaired liver function compared with controls and osteoporosis-only mice. The cirrhosis and cirrhosis-osteoporosis group also showed reduced bone mass and fewer trabeculae relative to mice

without cirrhosis. WGCNA of proteomic data from cirrhosis and osteoporosis models identified core common differentially expressed genes, including significantly downregulated IGFBP3. KEGG analysis showed significant enrichment of these genes in the NF- κ B signaling pathway. In cirrhosis mice, IGFBP3 was decreased in both liver and bone. Exogenous IGFBP3 increased bone mass and osteogenic markers in vivo. In vitro, LPS-stimulated co-culture of AML12 hepatocytes and MC3T3-E1 osteogenic cells impaired osteogenic differentiation, which was restored by IGFBP3 addition along with suppression of NF- κ B signaling.

Conclusions: Cirrhosis exacerbates bone loss and osteoporosis progression, primarily mediated by systemic inflammation. Hepatically-produced IGFBP3 promotes osteoblastic differentiation via suppression of NF- κ B signaling pathway, thereby delaying osteoporosis onset. These findings identify IGFBP3 as a key molecular mechanism linking cirrhosis and osteoporosis, highlighting its therapeutic potential.

Keyword(s) : cirrhosis, osteoporosis, IGFBP3, inflammation

2224 - Hepatitis D related liver disease as a major indication for liver transplantation experience from a new liver transplant program in pakistan

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Background / Hypothesis:

Hepatitis D virus infection is the most severe form of chronic viral hepatitis and can rapidly progress to cirrhosis and liver failure. In endemic regions such as Pakistan, it contributes substantially to advanced liver disease, yet its representation among liver transplant indications is not well described. We assessed the burden and clinical characteristics of hepatitis D-related liver disease as an indication for liver transplantation in the initial experience of our center.

Materials and Methods:

We retrospectively reviewed the first 42 liver transplants performed for chronic liver disease at our program in Pakistan. Patients were categorized into hepatitis D virus-related disease (n=16) and non-hepatitis D virus etiologies (n=26). Demographics,

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transplant indication, Child–Pugh score, Model for End-Stage Liver Disease sodium score, and decompensation status (ascites and/or hepatic encephalopathy) were recorded. Group comparisons were performed using t-tests and chi-square analyses.

Results:

Among 42 transplants, 37 (88.1%) recipients were male, with a mean age of 46.2 ± 10.5 years. Hepatitis D virus–related chronic liver disease was the leading transplant indication (38.1%, n=16), followed by hepatitis C (21.4%, n=9), alcoholic liver disease (19.0%, n=8), hepatitis B (11.9%, n=5), metabolic dysfunction–associated fatty liver disease (4.8%, n=2), primary biliary cholangitis (2.4%, n=1), and autoimmune hepatitis (2.4%, n=1). Hepatitis D virus recipients were significantly younger than non–hepatitis D virus recipients (40.5 ± 11.6 vs 48.9 ± 7.2 years; $p=0.05$). Child–Pugh score (9.7 ± 3.0 vs 8.8 ± 2.1 ; $p=0.276$), Model for End-Stage Liver Disease sodium score (17.3 ± 5.1 vs 14.6 ± 5.2 ; $p=0.108$), and decompensation rates (93.8% vs 76.9%; $p=0.320$) were not statistically different. Hepatocellular carcinoma was present in 25% (n=4) of hepatitis D virus cases.

Conclusions:

In our early liver transplant experience, hepatitis D virus–related liver disease emerged as the most common indication for transplantation and affected significantly younger patients, highlighting its aggressive course in endemic settings. Strengthening screening, early diagnosis, and timely transplant referral pathways for hepatitis D virus infection may help reduce the burden of advanced disease.

Keyword(s) : Hepatitis D virus, liver transplantation, end-stage liver disease, transplant indications, hepatocellular carcinoma

2227 - Unilateral Sialadenosis as Initial Presentation of Alcoholic Hepatitis A Case Report

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Background: Sialadenosis is a non-inflammatory, non-neoplastic, bilateral, painless swelling of the parotid glands or less frequently, the submandibular glands. It is often linked to chronic alcoholism and those with diabetes and malnourishment. We present a first-ever reported case of unilateral Sialadenosis of the right submandibular gland in an alcoholic male patient with alcoholic hepatitis.

Case Presentation: A 51-year-old diabetic and hypertensive, Filipino male, heavy alcohol drinker sought consultation due to right submandibular swelling and jaundice. The swelling in the right submandibular area started four weeks before seeking medical consultation which was subsequently followed by jaundice and generalized body weakness. Pertinent physical examination revealed icterisia, pale conjunctivae, nontender, non-mobile right submandibular mass ~6x5 cm, slightly distended abdomen, and jaundice. Laboratory work-up showed macrocytic anemia, hyperbilirubinemia, hypoalbuminemia, an AST:ALT ratio of 1.88, a MELD-Na score of 29 and Maddrey's Discriminant Function (MDF) 33.2 --- findings consistent with severe alcoholic hepatitis. Neck ultrasound revealed enlarged right submandibular gland, with normal left submandibular and parotid glands. Fine needle aspiration (FNA) biopsy of the right submandibular gland was nondiagnostic with note of few lymphocytes and neutrophils with abundant red blood cells. Further examinations revealed previous hepatitis B, fatty liver changes, minimal ascites, and no concomitant infection. Steroid therapy, along with alcohol cessation, was initiated. Significant clinical and biochemical improvement were observed after a week. Treatment was completed for four (4) weeks with resolution of the submandibular enlargement and jaundice.

Conclusion: This case shows that unilateral sialadenosis can be an initial presentation of alcoholic hepatitis and resolves with appropriate management. It stresses the importance of thorough history-taking and physical examination, along with comprehensive diagnostic tests and apt interventions to ensure better outcomes in patients with alcoholic hepatitis.

Keyword(s) : unilateral sialadenosis, alcoholic hepatitis, case report

2228 - HBsAg Levels as Predictors of Fibrosis in Chronic Hepatitis B A Systematic Review and Meta analysis

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Background / Hypothesis: Chronic hepatitis B virus (HBV) infection remains a major global cause of liver-

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related morbidity and mortality. Accurate assessment of liver fibrosis is essential for prognostication and treatment decisions. Quantitative hepatitis B surface antigen (HBsAg) levels have emerged as a potential non-invasive marker reflecting viral activity and immune control; however, their role in predicting liver fibrosis remains controversial across studies.

Aim:To evaluate the association between serum HBsAg levels and liver fibrosis severity in patients with chronic hepatitis B.

Materials and Methods:

A systematic literature search was performed using PubMed, Scopus, and Web of Science to identify studies assessing the relationship between quantitative HBsAg levels and liver fibrosis in adult patients with chronic hepatitis B. Studies using histology, elastography, or validated non-invasive fibrosis assessments were included. Data were synthesized using meta-analytic methods, and study quality was assessed using standardized tools.

Results:Across included studies, HBsAg levels demonstrated a significant association with fibrosis severity in chronic hepatitis B patients. Lower HBsAg levels were generally associated with more advanced fibrosis, particularly in HBeAg-negative disease. This relationship was observed across different populations and fibrosis assessment methods, suggesting a potential role for HBsAg quantification in fibrosis risk stratification.

Conclusions:HBsAg levels may serve as a useful non-invasive biomarker for predicting liver fibrosis in patients with chronic hepatitis B. Incorporating quantitative HBsAg assessment into routine clinical evaluation may aid in identifying patients at higher risk of advanced fibrosis and guide monitoring and management strategies.

Keyword(s) : Keywords: Chronic hepatitis B, HBsAg, Liver fibrosis, Non-invasive biomarkers, Meta-analysis

2230 - HBsAg Levels as Predictors of Fibrosis in Chronic Hepatitis B A Systematic Review and Meta analysis

Javaria Younas Muhammad Younas¹, Mahnoor Washdil Washdil²

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Background / Hypothesis:

Chronic hepatitis B virus (HBV) infection remains a major global cause of liver-related morbidity and mortality. Accurate assessment of liver fibrosis is essential for prognostication and treatment decisions.

Quantitative hepatitis B surface antigen (HBsAg) levels have emerged as a potential non-invasive marker reflecting viral activity and immune control; however, their role in predicting liver fibrosis remains controversial across studies. **Aim:**To evaluate the association between serum HBsAg levels and liver fibrosis severity in patients with chronic hepatitis B.

Materials and Methods:

A systematic literature search was performed using PubMed, Scopus, and Web of Science to identify studies assessing the relationship between quantitative HBsAg levels and liver fibrosis in adult patients with chronic hepatitis B. Studies using histology, elastography, or validated non-invasive fibrosis assessments were included. Data were synthesized using meta-analytic methods, and study quality was assessed using standardized tools.

Results:

Across included studies, HBsAg levels demonstrated a significant association with fibrosis severity in chronic hepatitis B patients. Lower HBsAg levels were generally associated with more advanced fibrosis, particularly in HBeAg-negative disease. This relationship was observed across different populations and fibrosis assessment methods, suggesting a potential role for HBsAg quantification in fibrosis risk stratification.

Conclusions: HBsAg levels may serve as a useful non-invasive biomarker for predicting liver fibrosis in patients with chronic hepatitis B. Incorporating quantitative HBsAg assessment into routine clinical evaluation may aid in identifying patients at higher risk of advanced fibrosis and guide monitoring and management strategies.

Keyword(s) : Keywords: Chronic hepatitis B, HBsAg, Liver fibrosis, Non-invasive biomarkers, Meta-analysis

2231 - Hepatocellular Carcinoma Surveillance among Chronic Hepatitis C patients after successful SVR Systematic Review and Meta analysis of Economic Evaluation Studies

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Background / Hypothesis: Although sustained virologic

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response (SVR) significantly reduces the risk of hepatocellular carcinoma (HCC) in chronic hepatitis C (CHC) patients, the residual risk, especially among those with advanced fibrosis or cirrhosis, necessitates consideration of ongoing surveillance. Current guidelines offer inconsistent recommendations. This systematic review and meta-analysis evaluates the cost-effectiveness of HCC surveillance strategies post-SVR, to inform evidence-based clinical and policy decisions.

Materials and Methods: A systematic search was performed in PubMed, Embase, Scopus, Google Scholar, and the Cochrane Library to identify economic evaluations of HCC surveillance strategies in CHC patients post-SVR. Studies reporting cost-effectiveness outcomes. Incremental net monetary benefit (INMB) values were calculated for meta-analysis. Study quality was assessed using the ECOBIAS checklist. Subgroup analyses were conducted based on surveillance modality, interval, and willingness-to-pay (WTP) thresholds.

Results: Of 157 identified records, seven studies were included in the meta-analysis. All studies were conducted in American regions. Four focused exclusively on patients with cirrhosis, while three included both advanced fibrosis (F3) and cirrhosis. Most applied Markov models from a healthcare payer perspective. At a WTP threshold of USD 100,000, pooled INMB was significantly positive (USD 1,743; 95%CI: 468–3,019; $p=0.007$), particularly among cirrhosis-only populations. No significant cost-effectiveness was observed at WTP thresholds of USD 50,000 or 150,000. Subgroup analyses at a WTP threshold of USD 50,000 showed no surveillance strategy yielded statistically significant INMB. Six-monthly ultrasound with alpha fetoprotein (AFP) had the highest pooled INMB (USD –265), though not significant. Across modeling domains, results remained non-significant regardless of surveillance interval, HCC incidence, time horizon, SVR population, or model perspective. Studies assuming lower HCC incidence ($<1.5\%$ /year) tended to report lower INMBs. Overall, substantial consistency across subgroups was observed, with negligible heterogeneity ($I^2=0\%$), suggesting limited cost-effectiveness of post-SVR surveillance under lower WTP thresholds.

Conclusions: Current international guidelines show divergence in post-SVR HCC surveillance recommendations for CHC patients with advanced fibrosis. While AASLD and EASL do not recommend

surveillance in this group, APASL advises semiannual ultrasound with or without AFP up to two years post-SVR, and annually thereafter. Japan and Korea further support routine surveillance regardless of fibrosis stage in these patients population. However, existing cost-effectiveness evidence is based solely on Western settings. HCC surveillance post-SVR may be cost-effective at higher WTP thresholds, especially in cirrhotic patients. Given the economic and epidemiological differences in Asia-Pacific countries, region-specific economic evaluations are urgently needed to guide appropriate surveillance policies and resource allocation in post-SVR populations across diverse healthcare systems.

Keyword(s) : HCC surveillance, Chronic Hepatitis C, Post-SVR, Cost-effectiveness

2232 - Diagnostic Accuracy of FIB 4 Score for Detecting Advanced Fibrosis in NAFLD A Systematic Review and Meta analysis

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Background / Hypothesis:

Advanced fibrosis is the key determinant of prognosis in non-alcoholic fatty liver disease (NAFLD). Early identification of patients with advanced fibrosis is crucial to prevent liver-related complications. The FIB-4 score, a non-invasive and cost-effective biomarker-based tool, is commonly used to detect significant fibrosis, but its diagnostic accuracy shows variability across different studies and populations. Aim: To systematically evaluate the performance of the FIB-4 score in diagnosing advanced fibrosis among NAFLD patients.

Materials and Methods:

A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science for studies assessing FIB-4 against liver biopsy or validated non-invasive fibrosis reference standards. Adult NAFLD populations were included. Data on sensitivity, specificity, positive and negative predictive values, and area under the curve (AUC) were extracted. Meta-analytic techniques were applied to pool diagnostic accuracy estimates.

Results:

Analysis of the available studies indicates that FIB-4 demonstrates good discriminative ability for advanced

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fibrosis in NAFLD. The score shows reliable sensitivity and specificity across multiple cohorts and remains consistent regardless of study design or geographical region. FIB-4 effectively identifies patients at high risk for advanced fibrosis, allowing clinicians to prioritize further assessment and management.

Conclusions: The FIB-4 score is a practical, non-invasive tool for detecting advanced fibrosis in NAFLD. Its implementation in routine clinical practice can reduce unnecessary liver biopsies and facilitate timely intervention for high-risk patients. These findings underscore the value of FIB-4 as part of risk-stratification strategies in NAFLD management.

Keyword(s) : Keywords: NAFLD, FIB-4, Advanced fibrosis, Diagnostic performance, Non-invasive assessment

2233 - Real World Two Year Outcomes of Atezolizumab and Bevacizumab in Unresectable Hepatocellular Carcinoma Single Center Experience

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Background / Hypothesis

Hepatocellular carcinoma (HCC) ranks as the sixth most prevalent cancer globally and the fourth in Egypt. The combination of atezolizumab (an anti-PD-L1 agent) and bevacizumab (an anti-VEGF therapy) is established as the standard first-line treatment for unresectable HCC, leading to notable improvements in patient survival rates. This study aims to evaluate tumor response, overall survival, progression-free survival, and any potential adverse effects associated with the treatment.

Materials and Methods

Between November 2023 and December 2025, a group

of 169 patients diagnosed with unresectable hepatocellular carcinoma (HCC) who had not undergone systemic therapy prior to this study received a combination of atezolizumab and bevacizumab. Treatment continued until either intolerable adverse effects emerged or there was a decline in clinical efficacy. The main primary outcomes were overall survival and progression-free survival within the intention-to-treat cohort, evaluated using the Response Evaluation Criteria in Solid Tumors, specifically the modified RECIST (mRECIST) guidelines and assessment of AFP level.

Results

The overall survival for patients was 14.32 months (95% CI, 11.364 to 17.285), while the median progression-free survival was reported at 15.21 months (95% CI, 13.05 to 17.36). A complete response was observed in 2 patients (1.2%), while 51 patients (30.2%) demonstrated a partial response. Stable disease was noted in 33 patients (19.5%), and progressive disease was identified in 56 patients (33.1%). Treatment discontinuation occurred for 27 patients (16%), with 6 individuals ceasing treatment due to adverse drug effects and the remaining 21 due to either drug-related issues or high tumor burden.

Adverse effects reported included infusion-related reactions at a rate of 14.8%, hypertension also at 14.8%, and pneumonia affecting 17.1%. Additional side effects encompassed elevated ALT and AST levels in 11.8% of cases, increased bilirubin levels in 13%, bleeding occurring in 8.9%, thyroid disorders impacting 2.9% of patients, and colitis seen in 5.9%. Furthermore, embolization—either arterial or venous—was documented in 4 cases (2.4%). Grade 4 adverse events were reported in 5.3% of our patients, whereas Grade 5 events occurred in 2.9%. Dose adjustments or treatment delays were implemented for 60% of the study cohort.

Conclusions

In patients with unresectable hepatocellular carcinoma, atezolizumab combined with bevacizumab resulted in better overall and progression-free survival outcomes with tolerable adverse effects.

Keyword(s) : Immunotherapy, atezolizumab combined bevacizumab, hepatocellular carcinoma



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2234 - Preliminary results of STRIDE regimen in Egyptian HCC patients A Single Center Real World Experience

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Background / Hypothesis

On October 21, 2022, the FDA approved tremelimumab (Imjudo) in combination with durvalumab for adult patients with unresectable hepatocellular carcinoma (HCC). This study aimed to assess the response of tumors and identify any possible side effects related to the treatment.

Materials and Methods

Between January 2025 and November 2025, a group of 52 patients diagnosed with unresectable HCC who had not undergone systemic therapy prior to this study received a combination of durvalumab and tremelimumab. Treatment continued until either intolerable adverse effects emerged or there was a decline in clinical efficacy. All patients evaluated using the Response Evaluation Criteria in Solid Tumors, specifically the modified RECIST (mRECIST) guidelines and assessment of AFP level.

Results

As for the tumor response, one patient (1.9%) experienced a complete response, while 18 patients (26.4%) were classified as partial responders. Additionally, stable disease was observed in 14 patients (26.4%), and 10 patients (18.9%) exhibited progressive disease. Treatment was discontinued for 9 patients (17.3%), with 3 of these cases attributed to adverse effects such as immune-mediated thrombocytopenia, immune-mediated pancreatitis, and severe renal impairment; the remaining 6 discontinued treatment

due to decompensated liver disease. The overall response rate was 36.53%, whereas the disease control rate was 63.46%. Dose modifications or delays in treatment occurred in 30% of the participants in the study cohort. The most frequently reported adverse effects included pruritus at 23%, rash at 11.5%, elevated liver enzymes (ALT, AST) at 23%, increased bilirubin level at 17.3%, and both colitis and thyroid disorders affecting 7.7% of participants each. G3/4 toxicity was noted with a 19% occurrence of rash and a 17% incidence of pruritus. Additionally, G5 toxicity was associated with liver injury, reported at a rate of 2.1%.

Conclusions

Utilizing a combination of tremelimumab and durvalumab in Egyptian patients with unresectable hepatocellular carcinoma led to tumor responses similar to those observed in real-world studies, accompanied by tolerable side effects.

Keyword(s) : Immunotherapy, tremelimumab and durvalumab, hepatocellular carcinoma

2235 - Association Between HbA1c Levels and Risk of Liver Related Events in NAFLD Patients A Systematic Review and Meta analysis

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is closely linked with metabolic disorders, particularly diabetes. Glycated hemoglobin (HbA1c) reflects long-term glycemic control and may influence the risk of liver-related complications. However, the relationship between HbA1c levels and liver-related events in NAFLD patients remains unclear.

Aim :To evaluate the association between HbA1c levels and the risk of liver-related events in patients with NAFLD.

Materials and Methods: A systematic literature search was performed in PubMed, Scopus, and Web of Science for studies reporting HbA1c and liver-related outcomes, including cirrhosis, liver decompensation, hepatocellular carcinoma, or liver-related mortality in NAFLD patients. Data on study design, sample size, HbA1c categories, and liver-related event rates were extracted and synthesized qualitatively and quantitatively.

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Results: Evidence across multiple studies suggests that higher HbA1c levels are associated with an increased risk of liver-related events in NAFLD patients. This association was observed in both cohort and cross-sectional studies, independent of age, sex, and other metabolic risk factors. Findings indicate that poor glycemic control significantly elevates the risk of progressive liver disease in this population.

Conclusion: Elevated HbA1c is a significant predictor of liver-related events in NAFLD patients. Monitoring and optimizing glycemic control may help reduce the risk of adverse liver outcomes. These findings support integrating HbA1c assessment into risk stratification and management strategies for NAFLD patients.

Keyword(s) : Keywords: NAFLD, HbA1c, Liver-related events, Glycemic control, Meta-analysis

2236 - Cimicifugae Rhizoma Suppresses Hepatoblastoma Proliferation by Downregulating G6PD Expression

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Background / Hypothesis: Hepatoblastoma (HB) is the most common malignant pediatric liver tumor, and treatment options remain limited. Glucose-6-phosphate dehydrogenase (G6PD), a key rate-limiting enzyme in the pentose phosphate pathway, has been shown to promote HB progression. Cimicifugae Rhizoma (CR), a traditional Chinese medicinal herb, possesses antitumor properties, but its effect on HB development and prognosis remains unclear.

Materials and Methods: We first evaluated G6PD expression across cancers using the TIMER database and specifically in HB using three GEO datasets. The diagnostic value of G6PD for HB was assessed via ROC curve analysis. Due to the lack of HB patient prognosis data, its prognostic significance was explored in hepatocellular carcinoma (HCC) using GEPIA 2. Molecular docking analysis using CB-Dock2 were performed to analyze interactions between CR and G6PD. Finally, the effects of three principal active components of CR on HepG2 cells were investigated. Cell proliferation was measured via CCK-8 assay, and G6PD mRNA expression was detected by qRT-PCR.

Results: G6PD was highly expressed in most

malignancies, including HB, and demonstrated strong diagnostic value for HB. Survival analysis linked high G6PD expression with poor prognosis in HCC patients. Molecular docking indicated that the CR components Asiaticoside B, cimifugin, and Prim-O-glucosylcimifugin (POG) exhibited favorable binding affinity for G6PD. Experimental validation confirmed that CR inhibits HepG2 cell proliferation and downregulates G6PD mRNA expression.

Conclusions: This study is the first to demonstrate that Cimicifugae Rhizoma inhibits hepatoblastoma proliferation by modulating G6PD, a key gene in the pentose phosphate pathway, thereby providing novel insights and a potential therapeutic strategy for HB treatment.

Keyword(s) : Cimicifugae Rhizoma, Hepatoblastoma, G6PD, Proliferation

2237 - RSRC1 Promotes Hepatocellular Carcinoma Progression by Mediating Homologous Recombination Repair and Regulates Sensitivity to PARP Inhibitors

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Background / Hypothesis:

The dysregulation of DNA damage repair is a critical factor in the development and progression of hepatocellular carcinoma (HCC), yet its core regulatory mechanisms remain unclear. Despite the significant success of PARP inhibitors (PARPi) in cancer therapy, their limited response rate, particularly in HCC, remains a bottleneck for clinical application. Herein, we investigated the molecular basis of the RNA-binding protein RSRC1 in regulating PARPi sensitivity and explored a potential therapeutic strategy for enhancing PARPi efficacy.

Materials and Methods:

The expression level of RSRC1 and its correlation with prognosis were evaluated by analyzing clinical HCC sample databases. In vitro cell proliferation and invasion assays, along with in vivo mouse xenograft tumor models, were employed to validate the biological functions of RSRC1. The interaction between RSRC1 and DNA repair proteins, as well as its recruitment to DNA damage sites, was investigated using techniques such as co-immunoprecipitation and laser microirradiation-induced DNA damage. The

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impact of RSRC1 on PARP inhibitor sensitivity was explored using gene knockdown and pharmacological inhibition methods.

Results:

Multi-omics analysis revealed that RSRC1 was significantly overexpressed in HCC tissues, and this high expression was closely associated with poor patient prognosis. Functional experiments confirmed that RSRC1 promoted hepatocellular carcinoma cell proliferation, invasion, and tumor growth. Mechanistic studies demonstrated that under DNA damage stress, RSRC1 was specifically recruited to DNA double-strand break sites, where it directly interacted with RPA2, a key homologous recombination repair protein. This interaction regulated the phosphorylation of RPA2, thereby promoting homologous recombination repair. Importantly, inhibition of RSRC1 expression significantly enhanced the sensitivity of HCC cells to PARP inhibitors. Combined intervention exhibited notable synergistic anti-tumor effects both in vitro and in vivo.

Conclusions:

RSRC1 played a crucial role in promoting HCC progression by activating the DNA repair response. Our findings reveal a novel therapeutic strategy of co-targeting RSRC1 and PARP Inhibitor as a potential treatment for HCC.

Keyword(s) : RSRC1; Hepatocellular Carcinoma; Combination Therapy; DNA Damage; Homologous Recombination

2238 - UNCOVERING THE ONCOGENIC ROLE OF PKMYT1 IN HEPATOCELLULAR CARCINOMA THROUGH INTEGRATED BIOINFORMATICS

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Background / Hypothesis:

PKMYT1 (Protein Kinase, Membrane Associated Tyrosine/Threonine 1), a member of the WEE family, plays a crucial role in regulating cell cycle transitions and has recently emerged as a promising therapeutic target in various cancers, including hepatocellular carcinoma (HCC). However, the precise mechanisms governing PKMYT1 expression in HCC remain largely unexplored. This study aims to elucidate the mechanisms of PKMYT1 expression in HCC through comprehensive bioinformatic analyses.

Materials and Methods:

PKMYT1 expression and its prognostic significance across various cancers were assessed using SangerBox. In HCC, we analyzed associations with prognosis, immune infiltration, and mutations utilizing UALCAN, BoxKaplan-Meier Plotter, GEPIA, Human Protein Atlas, TIMER 2.0, and SangerBox. Co-expressed genes were identified using STRING, and miRNA-gene interactions were explored through the DIANA-MicroT Webserver.

Results:

PKMYT1 expression was significantly elevated in a range of cancers, including GBM, LGG, UCEC, BRCA, LUAD, ESCA, STES, KIRP, KIPAN, COAD, PRAD, STAD, HNSC, KIRC, LUSC, LIHC, SKCM, BLCA, THCA, READ, OV, PAAD, UCS, LAML, PCPG, ACC, KICH, and CHOL. In HCC, high levels of PKMYT1 were correlated with poorer survival outcomes and associated with increased immune cell infiltration, particularly CD4 T cells, neutrophils, macrophages, and dendritic cells. Mechanistic analysis identified co-expressed genes, including CCNB1, CCNA2, CCNB2, CKS2, CDK1, PLK1, CDC25A, RPS6KA1, and STEAP3. Correlation analysis indicated that most of these genes exhibited positive correlations with PKMYT1, with the exception of STEAP3, which showed a negative correlation. Additionally, hsa-miR-608 was identified as a potential regulator of PKMYT1 expression.

Conclusions:

Our findings underscore the oncogenic role of PKMYT1 and its prognostic significance across various cancers. In HCC, elevated PKMYT1 expression influences progression, prognosis, and immune regulation, suggesting its potential as a diagnostic biomarker and therapeutic target.

Keyword(s) : PKMYT1; HCC

2239 - The ratio of GGT to PLT indices is a predictive factor for the progression of liver fibrosis in NA treated chronic hepatitis B patients with advanced fibrosis plus PEG IFN α 2a therapy

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Background / Hypothesis:



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Combination therapy with pegylated interferon α -2a (PEG-IFN α -2a) and nucleoside analogue (NA) has been shown to improve liver histology in patients with chronic hepatitis B (CHB) and hepatic fibrosis. PEG-IFN α -2a effectiveness was assessed in NA-treated CHB patients with advanced fibrosis/compensated cirrhosis.

Materials and Methods:

This retrospective multicenter study included NA-treated CHB patients with advanced fibrosis/compensated cirrhosis. Patients received 48-week PEG-IFN α -2a plus 24-week follow-up. The primary endpoint was liver fibrosis status 24 weeks post-treatment.

Results:

A total of 102 patients were enrolled in the study, including 35 and 67 baseline HBeAg-positive and HBeAg-negative CHB patients, respectively. At the end of PEG-IFN α -2a treatment, a total of 18 (17.6%) patients achieved HBsAg loss, and 23 (65.7%) achieved HBeAg loss. Sixty-nine patients completed the comparison of liver fibrosis and cirrhosis before and after treatment, of whom 16 (23.2%) experienced progression of liver fibrosis or cirrhosis, 32 (46.4%) experienced liver fibrosis reversal, and 21 (30.4%) remained stable. Penalized logistic regression analysis demonstrated that GPR (Intercept = -1.200; Coefficients = 0.102) and GGT levels (Intercept = -1.198; Coefficients = 0.048) at 24 weeks of PEG-IFN α -2a treatment were independent predictors of fibrosis progression. Among these, GPR at 24 weeks exhibited the highest predictive value (Corrected AUC = 0.805).

Conclusions:

Approximately one-fifth of the NA-treated CHB patients with advanced liver fibrosis and compensated cirrhosis experienced progression of liver fibrosis after PEG-IFN α -2a treatment. The GPR at 24 weeks of treatment was an independent predictive factor for the progression of liver fibrosis in this population.

Keyword(s) : Chronic hepatitis B, advanced liver fibrosis, compensated cirrhosis, pegylated interferon α -2a.

2242 - Dynamic Survival Prediction and Treatment Adjustment System for Advanced Biliary Tract Cancer

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Background / Hypothesis: First-line treatment for advanced biliary tract cancer (BTC) involves immunotherapy plus chemotherapy (immuno-chemotherapy). However, treatment response varies considerably among patients, requiring ongoing monitoring and treatment adaptations. We aimed to develop an AI system that would dynamically update survival predictions for patients with advanced BTC using evolving clinical data, enabling real-time prognostic stratification and treatment adjustment.

Materials and Methods: This study included patients with advanced BTC who were receiving immuno-chemotherapy across eight centres. Patients were divided into development, internal validation, and external validation cohorts (two retrospective, one prospective). We developed a Bayesian joint model, the Individualized Dynamic Risk Estimation Model for BTC (iDREM-BTC), that integrated baseline clinical and imaging data with serial measurements of C-reactive protein (CRP), carbohydrate antigen 19-9 (CA19-9), and total bilirubin (TBil). An enhanced model (iDREM(Pro)-BTC) also incorporated baseline immunohistochemistry and genetic data. Performance was evaluated via dynamic area under the curve (AUC) and calibration analyses versus baseline Cox models. The models' interpretability was evaluated through ablation study (ClinicalTrials.gov: NCT06849193).

Results: Data from 2314 patients were analysed (development cohort, n=841; internal validation cohort, n=360; external validation cohorts, n=1113). Machine learning methods identified age, ECOG performance status, tumour burden, tumour stage, CRP level, CA19-9 level, and TBil level as predictors of mortality. iDREM-BTC demonstrated robust performance in the development cohort (overall dynamic AUC 0.730 [95% CI 0.689–0.794]), with AUCs progressively improving from 0.688 to 0.798; internal validation cohort (0.718 [95% CI 0.670–0.778], AUCs improving from 0.668 to 0.778), external validation cohort 1 (0.755 [0.707–0.808], AUCs improving from 0.705 to 0.810), external validation cohort 2 (0.705 [0.639–0.773], AUCs improving from 0.633 to 0.779), and prospective external validation cohort (0.745 [0.691–0.802], AUCs improving from 0.690 to 0.804). iDREM(Pro)-BTC achieved better discrimination in the development

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cohort (AUC 0.807 [95% CI 0.781–0.839], AUCs improving from 0.780 to 0.839), and external validation confirmed these findings (0.718 [0.669–0.787], AUCs improving from 0.666 to 0.791). The model enables dynamic risk stratification, showing that high-risk patients receive little benefit from later-line immuno-chemotherapy. This suggests early discontinuation or a shift to palliative care.

Conclusions: iDREM-BTC and iDREM(Pro)-BTC offer dynamic, real-time prognostic prediction for patients with advanced BTC undergoing immuno-chemotherapy. The two models suit different clinical scenarios, providing individualized prognostic stratification and treatment adjustment.

Keyword(s) : biliary tract cancer, immuno-chemotherapy, dynamic prediction.

2243 - Qutan huoxue decoction alleviates MASH induced fibrosis by regulating LC3 mediated macrophage mitophagy

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Background / Hypothesis:

MASH has become a prevalent chronic liver disease that progresses to fibrosis in nearly 40% of patients, and considering the current lack of effective therapies, understanding disease mechanisms is crucial. Macrophage mitophagy plays a central role in the progression of MASH-induced liver fibrosis. Based on evidence that Qutan huoxue decoction (QTHX) alleviates MASH, this study investigates its underlying antifibrotic mechanisms.

Materials and Methods:

In this study, we investigated the therapeutic effects of QTHX using liver fibrosis induced by both HFD and CDAHFD diets, and BMDM mitophagy defect model in vitro. Network pharmacology, 4D-DIA proteomic, Single-cell transcriptomics were used to explore the mechanism underlying the effects of QTHX against MAS-induced liver fibrosis.

Results:

QTHX significantly ameliorated MASH-induced liver fibrosis in both HFD and CDAHFD models, as evidenced by improved serum biomarkers, attenuated histopathological injury, and reduced hepatic levels of

Hyp and α -SMA. Single-cell transcriptomics revealed that an association between QTHX and the modulation of the Subsequent in vivo and in vitro experiments confirmed that QTHX enhanced macrophage mitophagy, as evidenced by increased LC3B protein levels. This led to a reduction in cytosolic mtDNA, which in turn suppressed the cGAS–STING pathway and subsequently decreased the production of proinflammatory cytokines, including TNF- α and IL-1 β .
Conclusions:

This study demonstrated that QTHX alleviates MASH-induced fibrosis by promoting LC3-mediated mitophagy in macrophages, thereby inhibiting the cGAS–STING pathway.

Keyword(s) : MASH; Liver fibrosis; Qutan huoxue; Macrophage mitophagy; cGAS–STING.

2246 - Sulforaphane Inhibits N Nitrosodimethylamine induced Hepatocellular Carcinoma Stem Cell Properties via ZNF652 Sonic Hedgehog axis

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Background / Hypothesis: N-Nitrosodimethylamine (NDMA) is a potent carcinogen implicated in hepatocellular carcinoma (HCC) pathogenesis and cancer stem cell (CSC) emergence. Zinc finger protein ZNF652 exhibits tumor-suppressive properties, while Sonic Hedgehog signaling promotes CSC characteristics. Sulforaphane (SFN), a natural compound, shows anticancer potential, but its regulatory role in NDMA-induced liver CSCs remains unclear.

Materials and Methods: Bioinformatics analyses evaluated ZNF652's prognostic significance and Sonic Hedgehog pathway correlation. In vitro experiments induced LO2 cell malignant transformation via chronic NDMA exposure, performed sphere formation assays, and validated ZNF652/GLI1 interactions through overexpression, downregulation, and dual-luciferase assays. In vivo subcutaneous xenograft models treated with SFN (12.5/25 mg/kg) demonstrated dose-dependent tumor suppression, reduced CD133/GLI1 expression, and restored ZNF652 levels.

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Results:Chronic NDMA exposure (1 µg/mL) transformed LO2 hepatocytes into malignant TLO2 cells with CSC traits, including elevated HCC, CSC and EMT markers. ZNF652 expression decreased in metastatic HCC tissues and NDMA-induced CSCs. Overexpressing ZNF652 reduced sphere formation, suppressed CSC markers (CD133, SOX2, OCT4, EPCAM), and inhibited Sonic Hedgehog pathway activity by directly targeting GLI1 via promoter binding. SFN treatment diminished CSC self-renewal, downregulated GLI1 expression, and reversed ZNF652 knockdown-induced CSC properties in vitro. In nude mouse xenografts, SFN dose-dependently reduced tumor volume/weight, lowered CD133/GLI1 levels, and restored ZNF652 expression compared to NDMA-exposed group.

Conclusions:SFN inhibits NDMA-induced liver cancer stemness via the ZNF652/Sonic Hedgehog axis, offering a novel therapeutic strategy for hepatocellular carcinoma.

Keyword(s) : N-nitrosodimethylamine, hepatocellular carcinoma, ZNF652, Sonic hedgehog pathway, sulforaphane, cancer stem cell

2247 - ASSESSMENT OF HEPATOCELLULAR CARCINOMA RISK USING COMBINED SERUM FERRITIN AFP AND PIVKA II BIOMARKERS

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a leading cause of cancer-related mortality worldwide, largely due to late-stage diagnosis. Alpha-fetoprotein (AFP) is widely used for surveillance but lacks sufficient sensitivity when used alone. Protein induced by vitamin K absence or antagonist-II (PIVKA-II) provides improved diagnostic performance, particularly in AFP-negative cases. Serum ferritin, a marker of hepatic inflammation and iron overload, has been suggested

to be associated with increased HCC risk. We hypothesized that elevated ferritin levels are associated with concomitant increases in AFP and PIVKA-II, and that a combined biomarker approach may improve HCC risk stratification.

Materials and Methods:

A cross-sectional study was conducted including male and female participants. Subjects were stratified into normal and elevated ferritin groups. Serum ferritin, AFP, and PIVKA-II levels, along with age, were analyzed and expressed as mean ± standard deviation. HCC risk categories were defined using internationally accepted cutoff values: AFP >10 and >400 ng/mL; PIVKA-II 50–149 and ≥150 mAU/mL; ferritin >400 and >1000 ng/mL. Sex- and age-related differences in biomarker profiles were evaluated. A p-value <0.05 was considered statistically significant.

Results:

Elevated ferritin levels were associated with increased AFP and PIVKA-II levels, particularly among male participants. In males, ferritin increased 2.45-fold, accompanied by a 2.14-fold increase in AFP and a 3-fold increase in PIVKA-II. High HCC risk categories were observed in 72.8% of individuals with ferritin levels of 400–1000 ng/mL and in 22.8% with levels >1000 ng/mL. In female participants, ferritin elevation was more strongly associated with advancing age, while PIVKA-II levels largely remained within normal ranges. Combined elevation of biomarkers consistently corresponded to higher HCC risk categories.

Conclusions:

Ferritin is not a tumor-specific marker; however, when combined with AFP and PIVKA-II, it enhances hepatocellular carcinoma risk stratification. A multi-biomarker approach may improve early identification of high-risk individuals and strengthen surveillance strategies.

Keyword(s) : hepatocellular carcinoma, ferritin, alpha-fetoprotein, PIVKA-II, risk stratification, biomarkers

2248 - ERα FGF13 PROX1 cascade regulates fatty acid β oxidation to mediate sexual dimorphism in MAFLD

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Background / Hypothesis:

MAFLD exhibits marked sexual dimorphism in both prevalence and progression, with males generally experiencing more severe outcomes, including faster progression to metabolic dysfunction-associated steatohepatitis (MASH), liver fibrosis, and hepatocellular carcinoma (HCC). The protective role of estrogen in premenopausal females and the increased risk after menopause highlight the influence of sex hormones in MAFLD pathophysiology. Fibroblast growth factor 13 (FGF13), an X-linked non-secretory FGF homologous factor, has recently been implicated in energy metabolism and obesity, but its role in MAFLD and its underlying mechanisms remain unexplored.

Materials and Methods:

MAFLD was induced in wild-type and liver-specific Fgf13 knockout (cKO) mice by high-fat diet (HFD) feeding for 16 weeks. Liver tissues were collected for histological, biochemical, and molecular analyses. Human MAFLD gene expression datasets (GEO) were analyzed to identify sexually dimorphic genes. Co-immunoprecipitation coupled with mass spectrometry (Co-IP/MS) was used to identify FGF13-interacting proteins. Chromatin immunoprecipitation sequencing (ChIP-seq) data were analyzed to identify PROX1 target genes. Luciferase reporter assays and ER α agonist treatments were performed to validate transcriptional regulation.

Results:

1. MAFLD male mice exhibited more severe hepatic steatosis than females. Bioinformatic analysis of human MAFLD datasets identified FGF13 as a key sexually dimorphic gene.

2. Hepatic FGF13 expression was higher in females than in males under HFD conditions, and liver-specific deletion of Fgf13 exacerbated steatosis, injury, inflammation, and fibrosis in both sexes, abolishing the sex-based differences.

3. Co-IP/MS revealed PROX1 as a major FGF13-interacting protein. FGF13 stabilized PROX1 by inhibiting its ubiquitin-mediated degradation.

4. PROX1 ChIP-seq analysis identified binding to promoters of fatty acid β -oxidation genes (CPT-1A, ACSL3, PGC-1 α). HFD reduced PROX1 binding in both sexes, with greater retention in females; Fgf13 cKO eliminated this sexual difference and downregulated

β -oxidation gene expression.

5. ER α was predicted and validated as a transcriptional regulator of FGF13. Estrogen and ER α agonist treatments upregulated FGF13 expression in vitro and in vivo.

Conclusions:

Our findings demonstrate that ER α -driven FGF13 expression stabilizes PROX1, enhances fatty acid β -oxidation, and protects against hepatic lipid accumulation, thereby contributing to the sexual dimorphism in MAFLD. This newly identified ER α -FGF13-PROX1 regulatory axis provides a mechanistic basis for sex-specific differences in MAFLD progression and highlights potential targets for sex-stratified therapeutic strategies.

Keyword(s) : MAFLD, FGF13, PROX1, fatty acid β -oxidation, sexual dimorphism

2249 - Maternal High Fat Diet Induces Lifelong Epigenetic Priming and Trained Immunity Driving Offspring Susceptibility to MASLD

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Background / Hypothesis: Maternal high-fat diet (HFD) is known to increase the risk of metabolic dysfunction in offspring, but the mechanisms driving susceptibility to MASLD remain unclear. Emerging evidence suggests that early-life epigenetic changes and innate immune training may contribute to this vulnerability. We hypothesized that maternal HFD induces epigenetic priming and a trained immunity-like phenotype in offspring Kupffer cells, predisposing them to heightened hepatic inflammation and MASLD progression, especially under secondary dietary challenges.

Materials and Methods: A maternal-offspring mouse model was established by feeding female C57BL/6J mice either an HFD or chow prior to and during pregnancy. Offspring were analyzed at baseline and after postweaning dietary challenges. Liver tissues were evaluated using histology (H&E, Oil Red O, Masson's

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trichrome), immunofluorescence (MARCO, IBA1, CD31, H3K14ac), and flow cytometry to assess macrophage phenotypes. Epigenetic profiling included ChIP assays for H3K14ac and H3K27ac and bisulfite sequencing for global DNA methylation. Cytokine levels and inflammatory gene expression were measured via qPCR, Western blotting, and ELISA.

Results: Offspring of HFD-fed dams exhibited markedly increased hepatic steatosis, enhanced M1-polarized macrophage infiltration, and elevated IL-6 and TNF- α levels, despite minimal changes in body weight. Kupffer cells showed strong periportal/pericentral clustering and increased expression of MARCO and IBA1. Epigenetic analyses revealed significant enrichment of H3K14ac and H3K27ac at promoters of inflammatory cytokine genes, along with global DNA hypomethylation in metabolic pathways, suggesting persistent transcriptional activation. Postweaning HFD exposure synergistically exacerbated lipid accumulation and inflammatory activation, consistent with a trained immunity-like phenotype amplifying responses to secondary metabolic stress.

Conclusions: Maternal HFD establishes durable epigenetic priming and trained immunity in offspring hepatic macrophages, increasing susceptibility to MASLD progression. These findings highlight maternal nutrition as a key determinant of intergenerational liver health and suggest that targeting epigenetic regulators or innate immune memory may offer new avenues for MASLD prevention and therapy.

Keyword(s) : maternal high-fat diet, trained immunity, epigenetic programming, Kupffer cells, MASLD, intergenerational metabolic disease

2250 - Piezo1 mediates matrix stiffness induced hepatic stellate cell activation by inhibiting mitophagy in liver fibrosis

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Background / Hypothesis:

Liver fibrosis is characterized by excessive extracellular matrix deposition and increased tissue stiffness.

Hepatic stellate cells (HSCs) are the primary effector cells driving fibrosis, and their activation is mechanosensitive. The mechanosensitive ion channel Piezo1 senses matrix stiffness, but its role in HSCs during fibrosis is not fully understood. Mitophagy, the

selective autophagy of damaged mitochondria, is crucial for cellular homeostasis. We hypothesized that increased matrix stiffness promotes HSC activation through Piezo1-mediated impairment of mitophagy.

Materials and Methods:

A carbon tetrachloride (CCl₄)-induced liver fibrosis model was established in mice. Adjustable-stiffness 2D and 3D in vitro platforms (soft: 1 kPa, medium: 8 kPa, hard: 16 kPa) were constructed to mimic the mechanical microenvironment of normal and fibrotic livers. Piezo1 expression was assessed in vivo by Western blot and immunohistochemistry, and in vitro by Western blot, quantitative PCR, and immunofluorescence. By using specific antagonist GsMTx4 or siRNA-mediated knockdown of Piezo1, HSC activation markers (α -SMA and collagen I) were assessed by western blot, quantitative PCR, and immunofluorescence. Mitophagy activity was evaluated by monitoring mitochondrial morphology (MitoTracker), autophagic flux, and the colocalization of mitochondria with lysosomes. The involvement of the PINK1-Parkin pathway.

Results:

Piezo1 expression was significantly upregulated in fibrotic liver tissues and in LX2 cells cultured on stiff substrates. Stiff matrix promoted LX2 activation, concomitant with impaired mitophagy, evidenced by accumulated damaged mitochondria and decreased mitochondrial-lysosome colocalization. Pharmacological inhibition or genetic knockdown of Piezo1 attenuated stiffness-induced HSC activation and restored mitophagic flux in vitro. Furthermore, in vivo inhibition of Piezo1 alleviated CCl₄-induced liver fibrosis. Mechanistically, stiff matrix downregulated the PINK1-Parkin signaling pathway, a key mediator of mitophagy, and this suppression was reversed upon Piezo1 inhibition.

Conclusions:

Our findings demonstrate that increased matrix stiffness upregulates Piezo1 in HSCs, which in turn suppresses the PINK1-Parkin-mediated mitophagy, leading to HSC activation and exacerbation of liver fibrosis. Targeting the Piezo1-mitophagy axis may represent a novel therapeutic strategy for treating liver fibrosis.

Keyword(s) : Piezo1, liver fibrosis, matrix stiffness, mitophagy

2251 - Multisystem Involvement Of Salmonella Infection Liver Abscess With Secondary Pleural Effusion In Diabetes Mellitus

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Background / Hypothesis:

Salmonella liver abscess is an infrequent condition and its occurrence alongside pleural effusion is reported only on rare occasions. Individuals with diabetes mellitus have an elevated susceptibility to severe and unusual infections because of compromised immune defenses.

Materials and Methods:

This case report outlined a 48 years old male patient who developed a hepatic abscess and accompanying pleural effusion as complications of a Salmonella infection in the setting of diabetes mellitus. Clinical information was gathered from the patient's medical history, physical examination, laboratory evaluations, abscess culture, and imaging modalities such as CT scanning and chest X-ray. The therapeutic management and clinical progression were monitored and recorded throughout the hospital stay and follow-up period.

Results:

The patient arrived at the emergency room with complaints of abdominal discomfort and fever a week prior and respiratory manifestations during inpatient care. Laboratory evaluations indicated raised inflammatory markers (CRP: 227.9 mg/L, reference value 0.0-3.0 mg/L) along with elevated blood glucose levels (POCT random blood glucose: 189 mg/dL, reference value <140 mg/dL). Imaging revealed approximately 12.2 × 11 × 15 cm in size of hepatic abscess from MSCT Scan accompanied by a right-sided pleural effusion from chest X-ray. Microbiological analysis of the abscess identified Salmonella species. The patient was treated with targeted intravenous antibiotics and percutaneous drainage of the abscess, leading to progressive clinical improvement. The pleural effusion subsided with continued medical therapy and supportive management. By the time of discharge, the patient had shown marked recovery.

Conclusions:

This report underscores the multisystem manifestations of Salmonella infection in a diabetic patient, evidenced by a hepatic abscess accompanied by secondary pleural effusion. Identifying unusual organ involvement is crucial when managing systemic infections in individuals with diabetes. Prompt imaging,

microbiological confirmation, and timely treatment, including appropriate antibiotics and abscess drainage, are key to avoiding complications and achieving favorable clinical outcomes.

Keyword(s) : Salmonella, Pyogenic Liver Abscess, Pleural Effusion, Diabetes Mellitus

2253 - Discrepancies in Treatment Eligibility Across Different International Guidelines for Chronic Hepatitis B

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Background / Hypothesis:

Simplifying treatment and expanding indications are key future directions for chronic hepatitis B (CHB) management. Recently released international guidelines (EASL 2025, AASLD/IDSA 2025, WHO 2024) for the management of CHB have updated treatment recommendations. However, inconsistencies remain, and their impact on real-world treatment eligibility warrants evaluation.

Materials and Methods:

This multicenter, retrospective, cross-sectional study analyzed data from a real-world cohort of 15,763 treatment-naïve CHB patients from five hospitals in China between 2015 and 2025.

Results:



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The proportions of patients eligible for antiviral therapy according to the AASLD/IDSA 2025, EASL 2025, and WHO 2024 guidelines were 32.2%, 37.3%, and 70.5%, respectively. The WHO 2024 guidelines identified substantially more patients as eligible for treatment, followed by the EASL 2025 and the AASLD/IDSA 2025 guidelines. Patients with HBeAg-positive had significantly higher treatment eligibility rates compared to HBeAg-negative patients across all guidelines (all $P < 0.001$). Notably, the treatment eligibility rate (92.5%) was substantially higher under the WHO 2024 guidelines than that under the EASL 2025 (74.6%, $P < 0.001$) and AASLD/IDSA 2025 (64.4%, $P < 0.001$) guidelines for HBeAg-positive patients. Similar findings were observed in HBeAg-negative patients. Additionally, treatment eligibility rates gradually increased with age, regardless of the guidelines applied. However, the WHO 2024 guidelines consistently yielded the highest eligibility rates across age categories, compared with the AASLD/IDSA 2025 and EASL 2025 guidelines.

Conclusions:

Substantial differences in CHB treatment eligibility were observed across updated international guidelines, with the highest eligibility proportion (70.5%) observed under the WHO 2024 guideline. Additional studies are warranted to explore optimal treatment criteria for CHB.

Keyword(s) : chronic hepatitis B, treatment eligibility, international guidelines, antiviral therapy

2257 - Construction and Validation of a 90 Day Prognostic Prediction Model for Patients with Acute on Chronic Liver Failure After Artificial Liver Support Therapy

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) progresses rapidly and has a high short-term mortality rate. The artificial liver support system (ALSS) promotes hepatocyte regeneration by removing toxins and inflammatory mediators. This study aims to develop an early prediction model to identify ACLF patients with poor prognosis following ALSS, thereby supporting clinical decision-making.

Materials and Methods:

This study included 182 patients with confirmed ACLF who underwent at least two sessions of artificial liver therapy at the Third Hospital of Hebei Medical University from February 2021 to May 2025. Independent predictors were identified through univariate and forward stepwise multivariate logistic regression analyses, which were subsequently used to construct a prediction model. The area under the curve (AUC) were used to assess the model's accuracy. The calibration curve and decision curve analysis (DCA) were employed to assess the discriminability and clinical effectiveness, respectively. Statistical analyses were performed using Zstats software and R version 4.3.3.

Results:

The baseline characteristics of the included patients and the training cohort are presented in Table 1 and Table 2, respectively. Multivariate analysis identified the MELD score (OR = 1.175, 95% CI: 1.084–1.274) and the number of complications (OR = 1.656, 95% CI: 1.055–2.598) as independent prognostic factors. The model demonstrated good fit (Hosmer-Lemeshow test, $P > 0.05$) and showed no significant multicollinearity, with variance inflation factors (VIF) for all predictors below 5. The prediction model was derived as: $\text{logit (90 day outcome)} = -5.446 + 0.161 \times \text{MELD score} + 0.504 \times \text{Number of complications}$. The AUC were 0.754 for the MELD score, 0.674 for the number of complications, and 0.782 for their combination (Figure 2). The calibration curve confirmed good agreement between prediction and the ideal curve (Figure 3). DCA showed that the model provided a higher net benefit than the treat-all and treat-none strategies across a threshold probability range of 19.35% to 87.70%, supporting its clinical utility (Figure 4). A nomogram was developed to visually present the combined prediction model (Figure 5). In the validation cohort, the combined model demonstrated an AUC of 0.783 (95% CI: 0.658–0.907) (Figure 6). Both the calibration and decision curve analyses indicated good predictive performance and clinical utility (Figures 7, 8).

Conclusions:

This study developed and validated a prediction model for early identification of ACLF patients at risk of poor 90-day prognosis after artificial liver therapy,

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demonstrates superior predictive performance compared to MELD score, showing potential to aid clinical decision making.

Keyword(s) : Acute-on-chronic liver failure, Artificial liver therapy, prediction model, MELD score

2258 - DNA Methylation Profiles Predict MASLD Progression

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Epigenetic modifications represent a critical interface between environmental factors and disease pathogenesis in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). We hypothesized that specific DNA methylation patterns in liver tissue and peripheral blood are associated with disease severity and can serve as predictive biomarkers for fibrosis progression.

We conducted an integrated translational study combining preclinical and clinical investigations. In a diet-induced murine MASLD model (n=40), we performed genome-wide DNA methylation sequencing (Whole Genome Bisulfite Sequencing) of liver tissues at different disease stages. In a prospective human cohort of 320 biopsy-proven MASLD patients, we validated findings using targeted bisulfite sequencing of candidate loci in both liver biopsies and paired peripheral blood mononuclear cells (PBMCs). Methylation profiles were correlated with histological features and clinical outcomes over 3-year follow-up. In the murine model, we identified 2,147 differentially methylated regions (DMRs) during disease progression, with hypermethylation in fibrosis-related genes showing the strongest association. In human samples, a 5-gene methylation signature (including PPARA, TGFB1, and COL1A1 regulatory regions) demonstrated high concordance between liver tissue and PBMCs. Patients with the "hypermethylated profile" had 3.2-fold increased risk of fibrosis progression (95% CI: 1.8-5.7, p<0.001) and showed more rapid decline in liver function. The methylation score outperformed conventional biomarkers (FIB-4, NFS) in predicting 3-year clinical outcomes (AUC: 0.89 vs 0.71-0.75).

Mechanistically, the identified DMRs were enriched in pathways regulating hepatic stellate cell activation and extracellular matrix organization.

This study establishes disease-specific DNA methylation signatures as novel, accessible biomarkers for MASLD progression. The high concordance between liver and blood methylation patterns enables non-invasive monitoring, addressing a critical need in

public health screening. These findings position epigenetic profiling as a powerful tool for risk stratification and early intervention, potentially transforming MASLD management through precision prevention strategies.

Keyword(s) : DNA Methylation, Epigenetics, MASLD, Biomarkers, Precision Prevention, Risk Stratification

2259 - Managing Portal Hypertension Complications in Hepatic Arterioportal Fistula The Role of Transcatheter Arterial Embolization

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Background / Hypothesis:

Hepatic arterioportal fistula (HAPF) is a potential cause of refractory symptomatic portal hypertension, frequently manifesting as variceal hemorrhage and intractable ascites. This article evaluates the short- and long-term clinical outcomes of transcatheter arterial embolization (TAE) for managing symptomatic portal hypertension caused by HAPF.

Materials and Methods:

We conducted a retrospective study of 9 consecutive patients with HAPF-related portal hypertension (manifesting as variceal bleeding/ascites) who underwent TAE at our institution (Jan 2016-Jun 2025). All patients, after the exclusion of hepatocellular carcinoma, were diagnosed by digital subtraction angiography (DSA). The fistulas were embolized using coils and cyanoacrylate glue. Baseline characteristics and follow-up data were analyzed to evaluate procedural efficacy and safety. The primary outcomes were technical success, procedure-related complications, clinical efficacy in controlling variceal bleeding or ascites, long-term rebleeding rate, and survival.

Results:

Technical success was achieved in all patients (9/9, 100%). Intraoperative DSA confirmed complete fistula occlusion in six patients (66.7%) during the initial procedure; the remaining three (33.3%) underwent successful planned repeat embolization. Immediate hemostasis was attained in all five patients (100%) presenting with active variceal bleeding. Refractory ascites significantly resolved or reduced to mild,

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controllable levels within four weeks in 83.3% (5/6) of cases. Minor post-embolization syndrome (transient fever or abdominal pain) occurred in four patients (4/9, 44.4%) and resolved with supportive care; no instances of ectopic embolism, hepatic infarction, or other major complications were observed. Liver function and coagulation parameters remained stable throughout follow-up, with significant post-procedural improvements in hemoglobin and albumin levels ($p < 0.05$). Over a median follow-up of 48 months (IQR: 22–50), endoscopic surveillance demonstrated variceal improvement in all five bleeding patients, and complete ascites resolution was radiologically confirmed in all six affected patients. Rebleeding occurred in one patient (20%) at 24 months. The overall survival rate was 100%.

Conclusions:

For portal hypertensive complications arising from HAPF, TAE offers a safe and feasible approach with marked efficacy. Its capability to occlude the fistula and promptly address critical complications like bleeding and ascites translates into meaningful clinical benefits, including improved quality of life and survival.

Keyword(s) : Transcatheter arterial embolization; Hepatic arterioportal fistula (HAPF); Portal hypertension.

2260 - Comparative Analysis of MAFLD in Diabetic vs. Non Diabetic Patients Metabolic and Fibrotic Profiles

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Background / Hypothesis:

Type 2 diabetes mellitus (T2DM) is a major metabolic comorbidity frequently associated with metabolic-associated fatty liver disease (MAFLD). The interplay between diabetes and MAFLD progression, particularly fibrosis severity and metabolic dysfunction, requires further investigation

Materials and Methods:

Aim

To compare metabolic parameters and liver fibrosis severity between diabetic and non-diabetic patients

diagnosed with MAFLD.

Method

This study included 150 MAFLD patients, comprising 75 with T2DM and 75 without diabetes, enrolled between January 2024 and March 2025. Diagnosis of MAFLD was based on ultrasound and metabolic criteria. Data collected included BMI, liver enzymes (ALT, AST), lipid profile, fasting glucose, HbA1c, and FibroScan liver stiffness measurements. Statistical analysis compared the two groups with significance set at $p < 0.05$.

Result:

Diabetic patients had higher BMI, ALT, triglycerides, fasting glucose, HbA1c, and liver stiffness than non-diabetic patients.

Non-diabetic patients showed milder metabolic disturbances and lower fibrosis scores.

AST difference was not statistically significant.

Conclusions:

Comparison of Diabetic vs Non-Diabetic MAFLD Patients

MAFLD patients with T2DM exhibit more severe metabolic derangements and greater liver fibrosis compared to non-diabetic counterparts. This underscores the importance of rigorous metabolic control and fibrosis monitoring in diabetic MAFLD patients to prevent progression and complications.

Keyword(s) :

2261 - UNVEILING THE ONCOGENIC AND PROGNOSTIC ROLE OF KRT17 IN HEPATOCELLULAR CARCINOMA

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Background / Hypothesis:

KRT17 (Keratin 17) has been implicated in the development of hepatocellular carcinoma (HCC). However, the precise regulatory mechanisms governing HCC progression remain poorly understood. This study aims to conduct a comprehensive analysis of the role of KRT17 in HCC.

Materials and Methods:

Data for this study were sourced from multiple

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bioinformatics databases, including GEO (Gene Expression Omnibus), The Cancer Genome Atlas (TCGA), Genotype-Tissue Expression (GTEx), and the Clinical Proteomic Tumor Analysis Consortium (CPTAC). We utilized the R programming language and various online tools to analyze KRT17 expression across different cancer types, particularly in HCC. We also examined its associations with tumor molecular subtypes, immune subtypes, diagnosis, prognosis, clinical characteristics, methylation status, and the immune microenvironment. Mechanistic analyses were performed to identify co-expressed proteins and regulatory microRNAs (miRNAs).

Results:

Compared to normal samples, KRT17 expression was significantly elevated in various cancers, including LIHC. High levels of KRT17 were associated with poorer survival outcomes in HCC and correlated with increased infiltration of immune cells, particularly neutrophils, macrophages, and dendritic cells. Mechanistic analysis revealed co-expressed genes such as KRT5, KRT6A, KRT6B, KRT14, KRT16, KRT18, EGFR, CCHCR1, SFN, and TXNDC9. Similar Genes Detection analysis indicated that TMEM130 exhibited a similar expression pattern and a positive correlation with KRT17. Additionally, hsa-miR-5701 was identified as a potential regulator of KRT17 expression.

Conclusions:

The findings of this study offer important insights into the oncogenic role of KRT17 and its prognostic significance in HCC, highlighting its potential as both a diagnostic biomarker and a therapeutic target. These studies underscore the need for further molecular assays to elucidate the role of KRT17 in tumorigenesis and to establish a foundation for developing novel therapeutic strategies.

Keyword(s) : KRT17; HCC

2262 - Circadian Clock Disruption Drives MAFLD Through Bile Acid Dysregulation

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The hepatic circadian clock coordinates metabolic homeostasis, yet its disruption in modern 24/7 lifestyles remains an unquantified public health risk for Metabolic Dysfunction-Associated Fatty Liver Disease

(MAFLD). We hypothesized that genetic or environmental disruption of the core clock gene BMAL1 dysregulates the temporal orchestration of bile acid metabolism, creating a sustained lipotoxic and pro-inflammatory hepatic environment that accelerates MAFLD progression.

We utilized hepatocyte-specific BMAL1 knockout (*LS-Bmal1* KO) mice and wild-type (WT) mice subjected to chronic jet lag (CJL) to model genetic and environmental circadian disruption, respectively. Mice were fed a chow or high-fat diet (HFD) for 16 weeks. Hepatic transcriptomics, serum and hepatic bile acid profiles (by UPLC-MS/MS), and metabolic phenotyping were performed. Human relevance was tested in a cohort of 50 shift workers and 50 day workers matched for age and BMI, who underwent VCTE and serum metabolomic profiling.

Both *LS-Bmal1* KO and CJL-WT mice on HFD developed markedly exacerbated steatohepatitis and fibrosis compared to controls ($p < 0.01$). Transcriptomics revealed a loss of rhythmic expression in key bile acid synthesis (Cyp7a1, Cyp8b1) and transport (Bsep) genes in knockout mice. This led to a pathological accumulation of cytotoxic deoxycholic acid (DCA) and a reduction in protective ursodeoxycholic acid (UDCA) during the rest phase. Restoring bile acid rhythm via timed supplementation of a bile acid sequestrant in CJL mice significantly attenuated liver injury ($p < 0.05$). In humans, shift workers exhibited significantly higher liver stiffness ($p < 0.05$) and a similarly dysregulated, non-rhythmic serum bile acid profile enriched in DCA compared to day workers.

Our findings establish circadian disruption as a direct pathogenic driver of MAFLD, mediated through the loss of temporal control over bile acid metabolism. This links an emerging environmental risk factor (shift work/chronodisruption) to a concrete molecular mechanism. From a public health perspective, this advocates for the integration of circadian health measures into MAFLD prevention guidelines and suggests chrono-nutrition or timed pharmacological interventions as novel, mechanism-based strategies for at-risk populations.

Keyword(s) : Circadian Rhythm, MAFLD, Bile Acids, Chronodisruption, Shift Work, Precision Prevention

2263 - Social Jetlag Disrupts Pituitary Hormone Rhythm to Drive MASLD

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Social jetlag (SJL), the misalignment between social and

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biological clocks, affects over 40% of the global population and is a growing public health concern. Beyond the well-known disruption of core clock genes, its impact on endocrine rhythms and subsequent metabolic disease remains unclear. We hypothesized that SJL specifically disrupts the circadian secretion of pituitary prolactin (PRL)—a hormone with emerging metabolic functions—thereby promoting the development of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD).

We employed a multi-level approach. First, transcriptomic data from a human liver biopsy cohort were analyzed using the Zeitzeiger algorithm to assess SJL's impact on hepatic rhythmic pathways. Second, a PRL global knockout mouse model combined with a simulated SJL protocol (chronic 6-hour light cycle advances) was used to investigate causality. Serum PRL rhythms, liver clock gene expression (ROR α), and key lipogenic enzymes were measured. Finally, a therapeutic proof-of-concept was tested using a novel biomimetic photothermal microneedle system designed for circadian-timed PRL release.

In humans, SJL was associated with significant disruption in the hepatic circadian transcriptome, particularly in PRL receptor signaling and fatty acid metabolism pathways. In mice, SJL attenuated the binding of clock gene ROR α to the PRL promoter, leading to abolished serum PRL rhythm. This resulted in suppressed hepatic MAPK/CCND1 signaling, upregulation of lipogenic enzymes (FAS, ACC), and accelerated liver steatosis. PRL knockout mice confirmed the essential protective role of PRL. Crucially, restoring the PRL rhythm via timed supplementation or the circadian-release microneedle system significantly reduced hepatic lipid deposition, outperforming constant-dose treatment.

Our study identifies the disruption of pituitary-derived PRL rhythm as a novel and critical mechanistic link between the common lifestyle stressor SJL and MASLD pathogenesis. This moves the paradigm beyond hepatocyte-autonomous clock defects to a systemic endocrine axis. The development of a chrono-release therapeutic device underscores the translational potential. From a public health perspective, these findings argue for the assessment of circadian hormonal health in high-risk individuals and pave the way for "chrono-therapeutic" strategies against the escalating MASLD epidemic.

Keyword(s) : Social Jetlag, Circadian Rhythm, Prolactin, MASLD, Chronotherapy, Endocrine Metabolism

2264 - Impact of Updated International Guidelines on Treatment Eligibility for Chronic Hepatitis B

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Background / Hypothesis:

Antiviral therapy substantially reduces the complications of chronic hepatitis B (CHB) and improve patient outcomes. The AASLD and EASL updated their guidelines for the management of CHB in 2025, with modified recommendations for antiviral treatment. This study evaluated the impact of updated international guidelines on CHB treatment eligibility by comparing eligibility under the updated guidelines with their respective previous versions.

Materials and Methods:

This study retrospectively included 14,269 treatment-naïve CHB patients from five hospitals in China between 2017 and 2025. The proportion of patients eligible for antiviral therapy was assessed and compared according to the AASLD 2025 and EASL 2025 guidelines, alongside their prior versions (AASLD 2018 and EASL 2017).

Results:

Expanded eligibility for antiviral treatment compared with their respective previous versions (AASLD 2025 vs. AASLD 2018: 31.8% vs. 26.9%, $P < 0.001$; EASL 2025 vs. EASL 2017: 36.7% vs. 31.1%, $P < 0.001$) was observed. The EASL 2025 treatment criteria resulted in a slightly

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higher overall treatment eligibility rate compared to AASLD 2025 ($P < 0.001$). This trend was consistent across subgroups stratified by sex and age, as well as among patients with HBeAg negativity. Among HBeAg-positive patients, the treatment eligibility rate based on the updated AASLD 2025 guidelines remained higher than that based on AASLD 2018 (64.7% vs 58.3%; $P < 0.001$), whereas the rates were comparable between the EASL 2025 and EASL 2017 guidelines (74.1% vs 73.4%; $P = 0.529$).

Conclusions:

The updated AASLD and EASL guidelines expanded CHB treatment eligibility, resulting in approximately a 5% increase in the proportion of patients eligible for antiviral therapy under both guidelines. More studies are needed to evaluate real-world treatment uptake despite expanded CHB treatment eligibility under current guidelines.

Keyword(s) : chronic hepatitis B, treatment eligibility, antiviral therapy, clinical practice guidelines

2265 - Construction and Validation of a 90 Day Prognostic Prediction Model for Patients with Acute on Chronic Liver Failure After Artificial Liver Support Therapy

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) progresses rapidly and has a high short-term mortality rate. The artificial liver support system (ALSS) promotes hepatocyte regeneration by removing toxins and inflammatory mediators. This study aims to develop an early prediction model to identify ACLF patients with poor prognosis following ALSS, thereby supporting clinical decision-making.

Materials and Methods:

This study included 182 patients with confirmed ACLF who underwent at least two sessions of artificial liver therapy at the Third Hospital of Hebei Medical University from February 2021 to May 2025. Independent predictors were identified through univariate and forward stepwise multivariate logistic regression analyses, which were subsequently used to

construct a prediction model. The area under the curve (AUC) were used to assess the model's accuracy. The calibration curve and decision curve analysis (DCA) were employed to assess the discriminability and clinical effectiveness, respectively. Statistical analyses were performed using Zstats software and R version 4.3.3.

Results:

The baseline characteristics of the included patients and the training cohort are presented in Table 1 and Table 2, respectively. Multivariate analysis identified the MELD score (OR = 1.175, 95% CI: 1.084–1.274) and the number of complications (OR = 1.656, 95% CI: 1.055–2.598) as independent prognostic factors. The model demonstrated good fit (Hosmer-Lemeshow test, $P > 0.05$) and showed no significant multicollinearity, with variance inflation factors (VIF) for all predictors below 5. The prediction model was derived as: $\text{logit (90 day outcome)} = -5.446 + 0.161 \times \text{MELD score} + 0.504 \times \text{Number of complications}$. The AUC were 0.754 for the MELD score, 0.674 for the number of complications, and 0.782 for their combination (Figure 2). The calibration curve confirmed good agreement between prediction and the ideal curve (Figure 3). DCA showed that the model provided a higher net benefit than the treat-all and treat-none strategies across a threshold probability range of 19.35% to 87.70%, supporting its clinical utility (Figure 4). A nomogram was developed to visually present the combined prediction model (Figure 5). In the validation cohort, the combined model demonstrated an AUC of 0.783 (95% CI: 0.658–0.907) (Figure 6). Both the calibration and decision curve analyses indicated good predictive performance and clinical utility (Figures 7, 8).

Conclusions:

This study developed and validated a prediction model for early identification of ACLF patients at risk of poor 90-day prognosis after artificial liver therapy, demonstrates superior predictive performance compared to MELD score, showing potential to aid clinical decision making.

Keyword(s) : Acute-on-chronic liver failure, Artificial liver therapy, prediction model, MELD score

2266 - Yang Deficiency Constitution and Altered Bile Acid Metabolism in MAFLD

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Background / Hypothesis:

While the Phlegm-Dampness constitution is widely recognized as a risk factor for Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), the metabolic impact of the Yang-Deficiency constitution remains understudied. Individuals with Yang-Deficiency often exhibit cold intolerance and fatigue, traits suggestive of systemic energy metabolism dysregulation. We hypothesized that the Yang-Deficiency constitution is associated with a distinct alteration in host bile acid metabolism, which disrupts hepatic lipid homeostasis and contributes to MAFLD development, offering a novel metabolic explanation for this TCM constitutional type.

Materials and Methods:

We conducted a cross-sectional study involving 180 participants classified into Yang-Deficiency (n=90) and Balanced (Gentle, n=90) constitution groups using the standardized Constitution in Chinese Medicine Questionnaire. All participants underwent abdominal ultrasonography for MAFLD diagnosis and Liver Stiffness Measurement (LSM) via VCTE. Serum levels of primary (cholic acid, CA; chenodeoxycholic acid, CDCA) and secondary bile acids (deoxycholic acid, DCA; lithocholic acid, LCA) were quantified using UPLC-MS/MS. Hepatic gene expression of key bile acid synthesis enzymes (CYP7A1, CYP8B1) and receptors (FXR, TGR5) was analyzed in a subset of 40 patients who underwent liver biopsy for clinical indications.

Results:

The prevalence of moderate-to-severe MAFLD (LSM ≥ 8.0 kPa) was significantly higher in the Yang-Deficiency group (38.9% vs. 16.7%, $p < 0.01$). Serum metabolomic profiling revealed a distinctive signature: Yang-Deficiency individuals exhibited significantly elevated levels of total and secondary bile acids (especially DCA), alongside a reduced CA/CDCA ratio compared to the Balanced group ($p < 0.001$). This profile correlated with increased hepatic steatosis and fibrosis scores ($r = 0.52$ and $r = 0.47$, respectively). Liver tissue analysis confirmed the molecular basis, showing downregulated CYP8B1 expression and a resultant shift in bile acid composition. Furthermore, Yang-Deficiency subjects with this specific bile acid profile had a 3.5-fold increased risk of MAFLD progression over a 2-year follow-up period (OR=3.5, 95% CI: 1.8-6.8).

Conclusions:

This study is the first to link the TCM Yang-Deficiency constitution with a specific, clinically significant dysregulation of bile acid metabolism that drives MAFLD pathogenesis. It moves beyond descriptive association to reveal a tangible metabolic

mechanism—impaired bile acid synthesis and an altered bile acid pool. From a public health and precision medicine perspective, serum bile acid profiling could serve as a novel biomarker for identifying high-risk Yang-Deficiency individuals, enabling early, constitution-targeted dietary or pharmacological interventions (e.g., bile acid sequestrants or FXR agonists) to prevent liver disease progression.

Keyword(s) : Yang-Deficiency Constitution, Bile Acid Metabolism, MAFLD, Precision Medicine, TCM Biomarker, Metabolic Dysregulation

2267 - Qi Stagnation Constitution Predisposes to Hepatic Microcirculatory Dysfunction in MAFLD

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Background / Hypothesis:

The Qi-Stagnation constitution, characterized by emotional constraint and inhibited Qi dynamics, is traditionally linked to systemic “stagnation” pathologies. We hypothesized that this constitution manifests at the hepatic microvascular level, predisposing individuals to early microcirculatory dysfunction—a critical yet overlooked precursor to inflammation and fibrosis in Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD).

Materials and Methods:

A prospective cohort of 300 treatment-naïve MAFLD patients was rigorously phenotyped using the Constitution in Chinese Medicine Questionnaire (CCMQ). Participants underwent paired assessments: (1) Hepatic Venous Pressure Gradient (HVPG) measurement for indirect portal pressure, and (2) Contrast-Enhanced Ultrasonography (CEUS) with time-intensity curve analysis for direct, real-time quantification of liver perfusion parameters (peak intensity, time-to-peak, mean transit time). Serum metabolomics and autonomic nervous system tone (via heart rate variability) were analyzed. A 12-week mind-body intervention trial (guided music therapy + diaphragmatic breathing) was conducted in a Qi-Stagnation subgroup (n=40) to assess microcirculatory reversibility.

Results:

Patients with Qi-Stagnation constitution (n=102) exhibited a distinct “functional ischemia” profile despite similar steatosis grades. Compared to Balanced-constitution controls, they had significantly prolonged hepatic mean transit time (+38%, $p < 0.001$) and reduced peak intensity (-25%, $p < 0.01$) on CEUS,

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indicating impaired microvascular perfusion. Importantly, 85% of Qi-Stagnation patients with abnormal perfusion had normal HVPG (<5 mmHg), confirming isolated microcirculatory impairment preceding macrovascular portal hypertension. Metabolomics revealed elevated kynurenine pathway metabolites, linking emotional stress physiology to local vascular tone dysregulation. The mind-body intervention significantly improved both subjective stress scores and objective CEUS perfusion parameters ($p < 0.05$), demonstrating functional reversibility.

Conclusions:

This study is the first to mechanistically link the TCM Qi-Stagnation constitution to objective, quantifiable hepatic microcirculatory dysfunction in MAFLD. It identifies a novel, "pre-fibrotic" high-risk phenotype where emotional stress and autonomic imbalance may directly compromise liver perfusion. Crucially, we demonstrate this dysfunction is detectable before conventional signs of portal hypertension and is modifiable through non-pharmacological intervention. This bridges TCM theory with vascular pathophysiology, positioning Qi-Stagnation as a key target for early public health strategies focused on stress management and microvascular health to prevent MAFLD progression.

Keyword(s) : Qi-Stagnation Constitution, Hepatic Microcirculation, MAFLD, Contrast-Enhanced Ultrasonography, Mind-Body Intervention, Preventive Medicine

2269 - Hepatoduodenal fistula as a complication of Hepatobiliary tuberculosis A case report

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Background / Hypothesis:

Tuberculosis has a high prevalence in developing countries like the Philippines. Gastrointestinal tuberculosis is the third most common form of extrapulmonary tuberculosis, of which hepatobiliary tuberculosis (HBTB) is the most common, according to a local study. HBTB presents with nonspecific symptoms. A significant minority of cases that affect the bile duct present with jaundice, usually due to hilar obstruction resembling cholangiocarcinoma. Complications such as hepatoduodenal fistula and duodenal mass are very rare occurrences and have very limited data. Our objective for this report is to add this case to the very limited existing literature of hepatobiliary tuberculosis presenting with a hilar mass

extending to duodenum with fistulizing complication (hepatoduodenal fistula), and to describe the successful response to combined treatment that includes establishing good biliary drainage through stent placement on the fistula and compliance with antitubercular therapy.

Materials and Methods:

We present a case of a 46-year-old female who presented with symptoms of cholangitis. CT imaging revealed dilated hepatic ducts with suspicious narrowing of common hepatic duct. Endoscopic retrograde cholangiopancreatography (ERCP) showed a proximal duodenal mass. Cannulation of the native papilla was unsuccessful. Further evaluation of the mass through gastroscopy showed a friable semicircumferential mass in the proximal duodenum along with presumed fistula that intermittently egresses bile. Biopsies were taken for histopathology. Further evaluation through MRCP revealed hilar mass and hepatic nodules, considering central cholangiocarcinoma.

Results:

Biopsy of the duodenal mass revealed chronic granulomatous process; hence, Antikoch's treatment was initiated. After noncompliance with anti-tubercular medications due to adverse effects, the cholangitic symptoms recurred after several months. On ERCP, the native papilla was again identified with no egress of bile. Successful cannulation of the fistulous opening showed its connection to the intrahepatic ducts on cholangiogram. Good biliary drainage was established following successful stent placement through this fistulous opening. A month later, she came again due to jaundice and abdominal pain due to distally migrated stent. A repeat ERCP was done, and ductal clearance yielded stones and sludge. A double pigtail stent was successfully placed. The re-initiation of anti-Koch's led to the resolution of hilar and liver lesions on subsequent follow-up MRCP.

Conclusions:

This case emphasizes the significance of including tuberculosis as differential diagnosis for biliary obstructive diseases and hilar lesions. The development of fistula, though a complication, can help in establishing biliary drainage. However, assessment for adequacy of bile drainage through ductal clearance and stent placement is essential for effective and safer anti-tubercular treatment.

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Keyword(s) : Hepatoduodenal fistula; hepatobiliary tuberculosis; cholangiocarcinoma; hilar lesion

2270 - TweakR promotes hepatic fibrosis via NF- κ B signaling in metabolic dysfunction associated fatty liver disease and intervention by obacunone

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Background / Hypothesis: Metabolic dysfunction-associated fatty liver disease (MAFLD) is a prevalent chronic liver condition where fibrosis represents a critical pathological stage. Current anti-fibrotic therapies have limited efficacy, highlighting the need for novel drug development. TWEAK signaling is implicated in MAFLD-related fibrosis, yet its precise mechanism is unclear. Based on our preliminary findings, we hypothesized that TweakR promotes MAFLD-associated liver fibrosis by activating hepatocyte NF- κ B, and that obacunone—a key active component of FZHY—binds TweakR with high affinity to block TWEAK pathway, thereby inhibiting NF- κ B signaling and reversing fibrosis.

Materials and Methods: Public database analysis revealed progressive upregulation of hepatic TweakR expression with advancing MAFLD-related fibrosis. Single-cell RNA sequencing of MAFLD patient livers showed TweakR specifically highly expressed in hepatocytes, where NF- κ B signaling pathways were notably activated. Multi-omics data from high-fat diet-induced MAFLD fibrotic mice demonstrated elevated NF- κ B signaling pathway activity at transcriptional, protein, and phosphoprotein levels. TweakR knockout in hepatocyte models significantly reduced expression of the NF- κ B signaling driver p52. Using TweakR-targeted molecular fishing, we identified obacunone from FZHY formula as a high-affinity ligand binding TweakR, confirmed by high-resolution mass spectrometry, molecular docking, and dynamics simulations.

Results: TweakR expression was specifically elevated in hepatocytes and increased with MAFLD fibrosis progression, closely associated with NF- κ B activation. In MAFLD fibrotic mice, NF- κ B pathways were broadly

activated across multi-omics levels, while TweakR knockout downregulated p52. FZHY demonstrated significant anti-fibrotic effects in vivo, reducing TweakR and NF- κ B pathway markers expression. Molecular fishing confirmed obacunone as the primary TweakR-binding ligand in the formula, with docking and dynamics simulations verifying strong binding stability.

Conclusions: TweakR promotes MAFLD-related liver fibrosis by driving NF- κ B signaling pathway. Obacunone, a key constituent of FZHY formula, binds TweakR with high affinity and blocks TWEAK signaling, thereby suppressing NF- κ B and offering a potential therapeutic strategy for MAFLD fibrosis.

Keyword(s) : MAFLD, liver fibrosis, TweakR, obacunone, FZHY

2271 - Machine Learning Predicts MASLD Fibrosis Risk via TCM Constitution Clusters

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Background / Hypothesis:

The combined predictive value of Traditional Chinese Medicine (TCM) constitutions for Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) progression is unknown. We hypothesized that patients form natural "TCM Constitution Clusters," and that these clusters enhance machine learning models for predicting liver fibrosis risk.

Materials and Methods:

In a multi-center cohort of 1,500 MASLD patients, TCM constitution was assessed using a validated scale. Liver fibrosis was evaluated via VCTE at baseline and 24 months. Unsupervised learning (k-means) identified natural TCM clusters. A Gradient Boosting Machine model predicting 24-month fibrosis progression was trained using these clusters and clinical features (e.g., BMI, ALT), and tested against a clinical-only model.

Results:

Four TCM clusters were identified. The "Complex Dampness-Heat & Blood Stasis" cluster had the highest baseline fibrosis prevalence (32.1%) and progression rate (18.7%). The integrated (TCM + clinical) model significantly outperformed the clinical-only model, with an AUC of 0.89 vs. 0.78 ($P < 0.01$) for predicting progression. It successfully re-stratified 28% of patients deemed "indeterminate risk" by conventional scores.

Conclusions:



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TCM constitution clusters provide unique information that significantly improves machine learning-based prediction of MASLD fibrosis progression. This approach enables precise, early identification of high-risk patients for tailored interventions.

Keyword(s) : TCM Constitution, Machine Learning, MASLD, Risk Prediction, Precision Medicine, Fibrosis Progression

2272 - Interventional Embolization of Spontaneous Portosystemic Shunts Improves Survival in Decompensated Cirrhosis A Retrospective Matched Analysis

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Background / Hypothesis:

Traditionally viewed as compensatory, spontaneous portosystemic shunts (SPSS) may conversely induce a "portal steal phenomenon," sustaining hepatic hypoperfusion and accelerating liver failure. While embolization (PSSE) is established for refractory hepatic encephalopathy (HE), its potential to restore hepatopetal flow and improve survival in decompensated cirrhosis remains unclear. This propensity score-matched (PSM) study evaluated the impact of PSSE on long-term outcomes.

Materials and Methods:

We retrospectively analyzed patients with decompensated cirrhosis and SPSS (2016–2023). A 1:1 PSM model based on age, sex, and Child-Turcotte-Pugh (CTP) score generated a cohort of 70 patients (35 embolization, 35 standard therapy). Primary endpoints were 24-month overall survival and further decompensation events. Inverse probability of censoring weighting (IPCW) was used to analyze longitudinal trajectories of liver volume, portal vein diameter, and functional reserve.

Results:

Embolization significantly reduced 24-month all-cause mortality compared to controls (11.4% vs. 34.3%, $P=0.023$) and decreased further decompensation events ($P=0.014$). IPCW-adjusted analysis showed the embolization group achieved significant hepatic remodeling, with mean liver volume increasing by 148.67 cm^3 ($P<0.001$) and portal vein diameter widening. Functionally, albumin levels rose and CTP

scores decreased. Conversely, controls exhibited progressive liver volume reduction ($P=0.042$) and INR worsening ($P=0.002$). Subgroup analysis revealed survival benefits were concentrated in patients with CTP <10 ($P=0.004$) and those presenting with HE ($P=0.035$). No survival benefit was observed in patients with CTP ≥ 10 or those presenting with variceal bleeding.

Conclusions:

For decompensated cirrhosis patients with preserved reserve (CTP <10), embolization serves as a disease-modifying strategy rather than merely symptom control. By occluding "portal steal" and restoring hepatopetal flow, it induces hepatic remodeling and functional recovery, translating into long-term survival benefits. Management strategies should balance portal pressure reduction with the maintenance of effective hepatic perfusion.

Keyword(s) : Spontaneous Portosystemic Shunt; Decompensated Cirrhosis; Portosystemic Shunt Embolization

2273 - Yin Deficiency Constitution Predisposes to HCC via Gut Immune Dysregulation

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Background / Hypothesis:

The Yin-Deficiency (YD) constitution in Traditional Chinese Medicine is characterized by a "hyperactive" metabolic state. We hypothesize that YD creates a permissive gut-liver-immune microenvironment in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), impairing immunosurveillance and elevating the risk of hepatocellular carcinoma (HCC).

Materials and Methods:

We conducted a nested case-control study within a prospective MASLD cohort ($n=3,200$). YD was determined using the validated CCMQ. A YD-subgroup and matched controls provided stool and blood for metagenomic sequencing and immunophenotyping. Mice with YD traits (induced by chronic heat-stress diet) were used to model HCC susceptibility and investigate gut barrier integrity, bacterial translocation, and hepatic immune landscape via scRNA-seq.

Results:

In the human cohort, YD constitution was an independent risk factor for HCC development over 5 years (HR=2.95, 95% CI: 1.88-4.62). YD individuals

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exhibited a distinct gut microbiome with reduced butyrate producers and enriched LPS-bearing pathobionts. This correlated with increased serum intestinal fatty-acid binding protein (I-FABP, gut leakage marker), elevated systemic LPS, and a functionally exhausted peripheral and intrahepatic CD8⁺ T cell profile. In the murine model, YD mice showed accelerated hepatocarcinogenesis. scRNA-seq revealed a depleted resident Kupffer cell population and an expansion of immunosuppressive macrophages in pre-neoplastic livers, a shift traced to increased translocation of gut-derived metabolites.

Conclusions:

This study mechanistically links the TCM YD constitution to HCC risk through a gut-derived "leakage-immunosuppression" axis. It identifies YD as a novel, biologically defined high-risk phenotype for liver cancer. From a public health perspective, screening for YD constitution and its associated gut-immune signature could enable ultra-early, targeted surveillance and preventive interventions (e.g., microbiome modulation) in the MASLD population.

Keyword(s) : Yin-Deficiency Constitution, Hepatocellular Carcinoma, Gut-Liver Axis, Immunosurveillance, MASLD, Precision Prevention

2275 - Dampness Heat Constitution Impairs Adipose Browning in MASLD

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Background / Hypothesis:

The Dampness-Heat (DH) constitution is prevalent in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) populations. Beyond its metabolic associations, we hypothesize that DH constitution is characterized by intrinsic dysfunction in adipose tissue, specifically an impaired ability of subcutaneous white adipose tissue (scWAT) to undergo "browning" (beiging). This failure in adaptive thermogenesis exacerbates hepatic lipotoxicity and inflammation.

Materials and Methods:

A cohort of 120 patients with biopsy-proven MASLD was assessed for DH constitution using the CCMQ and a quantified tongue coating index. Abdominal subcutaneous adipose tissue biopsies were obtained. Gene and protein expression of browning markers (UCP1, PRDM16, CIDEA) and mitochondrial content in scWAT were analyzed. Systemic inflammatory markers and hepatic transcriptomic profiles were correlated with browning capacity. A diet-induced obese mouse

model receiving a high-fat, high-sugar diet mimicking "DH" etiological factors was used to validate the causal role of impaired browning in liver injury.

Results:

Patients with DH constitution exhibited a significant reduction in scWAT browning capacity, with 60% lower UCP1 protein expression compared to Balanced constitution controls ($p < 0.001$). This "browning deficiency" strongly correlated with increased hepatic saturated diacylglycerol content ($r = -0.68$), higher NAFLD Activity Score ($r = -0.61$), and elevated plasma IL-6 levels ($r = -0.55$). Hepatic transcriptomics revealed upregulation of pathways involved in saturated fatty acid metabolism and chemokine signaling in the DH group. In the mouse model, animals on the DH-mimicking diet developed pronounced impairment in scWAT browning prior to severe steatohepatitis. Pharmacological induction of browning in these mice attenuated liver triglyceride accumulation and lobular inflammation.

Conclusions:

This study identifies impaired adipose tissue browning as a novel, functional metabolic defect underlying the Dampness-Heat constitution in MASLD. It provides a mechanistic link between this TCM phenotype and a worse hepatic phenotype through the lens of inter-organ energy metabolism. Targeting adipose tissue plasticity and browning represents a promising, constitution-specific therapeutic strategy for a significant subset of MASLD patients.

Keyword(s) : Dampness-Heat Constitution, Adipose Tissue Browning, MASLD, Thermogenesis, Inter-organ Crosstalk, Precision Therapy

2277 - Balanced Constitution Maintains HPA Axis and Hepatic Gluconeogenesis

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Background / Hypothesis:

The Balanced (Gentle) constitution in Traditional Chinese Medicine represents an optimal state of health and resilience. We hypothesize that this constitution is underpinned by a robust and stable Hypothalamic-Pituitary-Adrenal (HPA) axis. This neuroendocrine stability confers metabolic protection in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) by maintaining precise hepatic gluconeogenic flux, preventing both excessive fasting glucose production and lipid overaccumulation.

Materials and Methods:

A multi-center cohort of 400 MASLD patients was

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prospectively followed. Participants were classified into Balanced (n=200) or Non-Balanced (n=200, a composite of various unbalanced constitutions) groups using the CCMQ. Diurnal salivary cortisol profiles (4 samples/day) were measured to assess HPA axis rhythm. All participants underwent a 16-hour fasting metabolic assessment with stable isotope tracers ([6,6-²H₂]glucose) to quantify hepatic gluconeogenesis. Liver biopsies were analyzed for expression of gluconeogenic enzymes (PEPCK, G6Pase) and markers of endoplasmic reticulum (ER) stress.

Results:

Individuals with a Balanced constitution exhibited a significantly more robust diurnal cortisol rhythm, characterized by a steeper morning decline and lower evening levels ($p < 0.01$), indicating superior HPA axis regulation. During fasting, the Balanced group demonstrated a 22% more efficient hepatic gluconeogenic rate, meeting systemic glucose demand without overproduction ($p < 0.05$). This "metabolic precision" correlated with lower intrahepatic triglyceride content and reduced expression of ER stress markers (GRP78, CHOP) compared to the Non-Balanced group. In the Non-Balanced group, a blunted cortisol rhythm predicted both excessive gluconeogenesis and more severe hepatic steatosis ($r = 0.58$, $p < 0.001$).

Conclusions:

This study provides the first evidence linking the TCM Balanced constitution to superior neuroendocrine regulation, specifically a resilient HPA axis. This stability translates to precise control of a key hepatic metabolic pathway—gluconeogenesis—offering a novel mechanism for its documented protective role against metabolic disease progression. It shifts the paradigm from studying "risk constitutions" to defining the "protective physiology" of health. This insight paves the way for interventions aimed at restoring HPA axis rhythm and metabolic precision as a therapeutic strategy for MASLD patients with unbalanced constitutions.

Keyword(s) : Balanced Constitution, HPA Axis, Hepatic Gluconeogenesis, Metabolic Precision, MASLD, Neuroendocrinology

2278 - Spatiotemporal Transcriptomics Decodes Constitution Specific Liver Zonation

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Background / Hypothesis:

Traditional Chinese Medicine (TCM) constitution theory

posits that individuals have inherent, whole-body physiological predispositions. We hypothesize that these constitutional types are not merely clinical phenotypes but are fundamentally encoded in the spatial architecture and temporal dynamics of the liver itself. Specifically, we propose that different TCM constitutions exhibit unique, spatially resolved transcriptional programs across liver lobules, and that this "constitutional zonation" dictates heterogeneous responses to metabolic stress and progression of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD).

Materials and Methods:

We performed a high-resolution, multi-modality analysis on ultrasound-guided liver biopsies from a well-phenotyped cohort of 60 individuals, representing Balanced, Qi-Deficiency, and Dampness-Heat constitutions (n=20 each), all with early-stage MASLD. Using spatial transcriptomics (10x Visium) and single-nucleus RNA sequencing (snRNA-seq) on adjacent tissue sections, we mapped the complete transcriptome across the porto-central axis of the liver lobule. A novel computational pipeline was developed to deconvolute spatial data using snRNA-seq references and to quantify zonation gradients. Participants underwent a standardized mixed-meal challenge, with serial blood sampling for metabolomics to assess dynamic metabolic flexibility.

Results:

Spatial transcriptomics revealed constitution-specific distortion of metabolic zonation. The Dampness-Heat constitution exhibited a pronounced expansion of a high-inflammatory, lipogenic transcriptional zone from the periportal region, compressing the normal oxidative metabolic zone. In contrast, the Qi-Deficiency constitution showed a blunted gradient, with loss of zonation sharpness and decreased expression of genes involved in energy metabolism and stress response in the pericentral area. The Balanced constitution maintained the most preserved and harmonious zonation pattern. These spatial maps were corroborated by snRNA-seq, identifying distinct hepatocyte subpopulations enriched in each constitution. Furthermore, the spatial dysregulation score correlated strongly with postprandial metabolic inflexibility ($r = 0.78$, $p < 0.001$) and early fibrosis markers.

Conclusions:

This study pioneers the use of spatiotemporal omics to visualize and quantify the biological basis of TCM constitutions directly within the liver's architectural blueprint. We demonstrate that constitutions correspond to distinct, spatially organized hepatic "ecosystems," providing a novel mechanistic

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framework for understanding individualized disease susceptibility and progression. This moves TCM diagnostics from syndromic categorization to a molecularly defined, spatially-aware precision medicine paradigm. Identifying these high-risk spatial architectures early could enable targeted, location-specific interventions (e.g., zonation-aware nutraceuticals or gene therapies) to restore metabolic harmony and prevent MASLD advancement.

Keyword(s) : Spatial Transcriptomics, Liver Zonation, TCM Constitution, Metabolic Heterogeneity, Precision Hepatology, MASLD

2279 - Constitution Specific Gut Flora Directs Liver Epigenome in MAFLD

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Background / Hypothesis:

Emerging evidence highlights the gut-liver axis and epigenetic regulation in Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD). We hypothesize that distinct Traditional Chinese Medicine (TCM) constitutions harbor unique gut microbiota profiles, which produce specific metabolite signatures. These microbial metabolites, in turn, function as epigenetic modulators, directly shaping the hepatic DNA methylome and histone landscape, thereby predisposing individuals to divergent MAFLD phenotypes.

Materials and Methods:

In a discovery cohort of 150 biopsy-proven MAFLD patients (balanced, dampness-heat, and qi-deficiency constitutions), we performed multi-omics profiling. Metagenomic sequencing characterized fecal microbiota. Serum and fecal short-chain fatty acids (SCFAs) and bile acids were quantified. Genome-wide DNA methylation (Infinium EPIC array) and H3K27ac histone modification (ChIP-seq) were analyzed in matched liver tissues. Validation was performed in a humanized gnotobiotic mouse model colonized with donor microbiota from different constitutional types.

Results:

We identified constitution-specific microbial consortia and metabolite patterns. The Dampness-Heat constitution was enriched in pro-inflammatory *Escherichia/Shigella* and associated with elevated deoxycholic acid. Its liver tissue displayed significant hypomethylation at promoters of lipogenic (SREBP1) and chemokine (CCL2) genes. Conversely, the Qi-Deficiency constitution showed reduced butyrate-producing flora (*Faecalibacterium*), correlating with

hypermethylation and suppressed expression of mitochondrial β -oxidation genes (PPARA, CPT1A). Mice colonized with Dampness-Heat flora developed more severe steatohepatitis and recapitulated the human epigenetic alterations, which were ameliorated by butyrate supplementation.

Conclusions:

This study establishes a novel "Gut Flora-Metabolite-Epigenome" axis that mechanistically links TCM constitutions to MAFLD pathogenesis. It provides a functional genomic basis for constitutional taxonomy, moving beyond correlation to causality. Our findings position gut microbiota-derived metabolites as both prognostic biomarkers and potential therapeutic targets (e.g., epigenetic diet) for personalized MAFLD management based on TCM constitution.

Keyword(s) : TCM Constitution, Gut Microbiota, DNA Methylation, Epigenetics, MAFLD, Personalized Medicine

2280 - Atopic Constitution Primes Hepatic ILC2 Macrophage Axis in MASLD

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Background / Hypothesis:

The Atopic (Te Bing) constitution, a subtype in TCM theory, predisposes individuals to allergic and immune-dysregulated states. Its role in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), a condition of sterile inflammation, is unknown. We hypothesize that the Atopic constitution establishes a liver-resident type 2 immune microenvironment, characterized by expanded group 2 innate lymphoid cells (ILC2s), which drives alternatively activated macrophage polarization, thereby exacerbating hepatic steatosis and fibrosis.

Materials and Methods:

We enrolled 180 biopsy-proven MASLD patients and classified them via the CCMQ, with a focus on identifying Atopic constitution (n=45) versus Balanced controls (n=45). Liver tissues were analyzed by multicolor flow cytometry and spatial transcriptomics (Visium) to map immune landscapes. Serum levels of type 2 cytokines (IL-4, IL-5, IL-13, IL-33) and periostin were measured. A murine model of MASLD was subjected to a protocol inducing allergic airway inflammation to mimic the Atopic state, followed by analysis of liver ILC2 dynamics and intervention with an IL-33 receptor blocker.

Results:

Patients with Atopic constitution exhibited a 3.2-fold increase in hepatic ILC2 frequency compared to

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controls ($p < 0.001$). Spatial transcriptomics revealed these ILC2s were clustered in portal areas adjacent to IL-33-expressing stromal cells and CD206⁺ M2 macrophages. This spatial niche correlated with higher histological steatosis and ballooning scores ($p < 0.01$). Serum IL-33 and periostin were elevated and served as predictive biomarkers for faster fibrosis progression ($HR = 2.4$). In the murine model, induction of atopy led to hepatic ILC2 expansion and accelerated steatohepatitis, which was mitigated by IL-33/ST2 pathway blockade.

Conclusions:

This study uncovers a novel immune phenotype linking TCM Atopic constitution to MASLD severity via an ILC2-driven type 2 hepatic axis. It positions allergic predisposition as a previously unrecognized risk modulator for liver disease progression. The identified IL-33-periostin axis offers both a mechanistic target for therapy and a serum biomarker for risk stratification, paving the way for immune-centric precision medicine in MASLD.

Keyword(s) : Atopic Constitution, Group 2 Innate Lymphoid Cells (ILC2s), Type 2 Immunity, MASLD, IL-33, Spatial Immunology

2281 - Reversing Qi Deficiency Slows Hepatocyte Senescence via AI

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Background / Hypothesis:

The Qi-Deficiency constitution in TCM, associated with aging and energy depletion, may accelerate cellular senescence. We hypothesize that this constitution correlates with a specific hepatic senescence-associated secretory phenotype (SASP) and that a machine learning model can predict the efficacy of constitution-targeted herbal therapy in reversing this process in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD).

Materials and Methods:

A 12-month prospective cohort of 300 MASLD patients with Qi-Deficiency was enrolled. Baseline assessments included CCMQ, serum SASP factors (IL-6, PAI-1, MMPs), and liver stiffness measurement (LSM). A 6-month tailored herbal regimen was administered. Single-nucleus RNA sequencing was performed on paired liver biopsies from a subset ($n = 20$) pre- and post-intervention to map senescence signatures. An XGBoost model was trained on multi-omics data to predict LSM response.

Results:

At baseline, Qi-Deficiency strongly correlated with a high hepatic SASP burden. The herbal intervention significantly reduced serum SASP factors and LSM in 68% of patients. snRNA-seq confirmed a marked reduction in senescence programs within hepatocyte subclusters post-treatment. The XGBoost model, incorporating baseline SASP profile and gut microbiota features, predicted treatment responders with 86% accuracy ($AUC = 0.89$). Non-responders were enriched in a distinct, resistant senescent cell population.

Conclusions:

This study mechanistically links Qi-Deficiency to accelerated liver aging via senescence. It pioneers an AI-guided framework to personalize constitution-modifying therapy, demonstrating that reversing a TCM constitution can biologically decelerate a core aging mechanism in MASLD.

Keyword(s) : Qi-Deficiency, Cellular Senescence, SASP, Artificial Intelligence, Herbal Therapy, Precision Medicine

2283 - Phlegm Dampness Constitution Disturbs Hepatic Circadian Rhythms in MASLD

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Background / Hypothesis:

The liver's circadian clock orchestrates daily metabolic cycles. The Phlegm-Dampness (PD) constitution, central to metabolic disorders, may disrupt this temporal order. We hypothesize that PD constitution is associated with dampened circadian oscillations of core clock genes in the liver, leading to a phase shift in lipid metabolism programs that exacerbates Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD).

Materials and Methods:

A cohort of 80 patients with biopsy-proven MASLD (40 PD, 40 Balanced) was recruited. PD diagnosis was based on the CCMQ and a standardized tongue coating score. Liver biopsies were obtained at two circadian times (8:00 AM, 8:00 PM). Rhythmic expression of core clock genes (BMAL1, CLOCK, PER2, CRY1) and key metabolic enzymes (SREBP1, PPAR α) was analyzed using RT-qPCR and RNA-seq. Circadian parameters (amplitude, phase, period) were computed. Plasma melatonin and cortisol rhythms were measured. Circadian locomotor activity was monitored via actigraphy.

Results:

The PD group exhibited significantly dampened amplitude of hepatic BMAL1 (55% reduction, $p < 0.001$)

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and PER2 oscillations. Their hepatic transcriptome showed a phase advance of lipogenic genes (SREBP1) and a phase delay of fatty acid oxidation genes (PPAR α), creating a metabolic “mismatch.” This internal desynchrony correlated strongly with higher hepatic triglyceride content ($r = 0.71$) and NAFLD activity score ($r = 0.65$). The PD group also showed blunted plasma melatonin rhythms and disrupted rest-activity cycles. A machine learning classifier using four core clock gene expression levels at a single time point could identify PD constitution with 82% accuracy (AUC=0.86).

Conclusions:

This study establishes for the first time that the TCM Phlegm-Dampness constitution is characterized by a distinct, measurable disruption of the hepatic circadian clock, creating a metabolic state prone to steatosis. It provides a chronobiological mechanism for PD constitution. This insight opens a new avenue for chrono-targeted interventions (e.g., timed diet or light therapy) tailored to reset the circadian phase in PD individuals for MASLD management.

Keyword(s) : Phlegm-Dampness Constitution, Circadian Clock, MASLD, Chronobiology, Phase Shift, Metabolic Rhythm

2284 - Stomach Heat Constitution Impairs Postprandial Hepatic Metabolic Flexibility

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Background / Hypothesis:

The Stomach-Heat (SH) constitution, characterized by excessive “heat” and rapid digestion, is a common but understudied TCM phenotype in metabolic disorders. We hypothesize that this constitution is defined not by static parameters, but by a pathological hyper-response to nutrient influx, leading to impaired hepatic metabolic flexibility. Specifically, we propose that SH individuals exhibit an exaggerated but inefficient switch from fasting to fed state, causing a transient “metabolic traffic jam” that promotes lipogenesis and inflammation in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD).

Materials and Methods:

A dynamic metabolic challenge study was conducted in 100 individuals with early MASLD, stratified into SH and Balanced constitutions using the CCMQ and a quantitative infrared thermography of the epigastrium. Participants underwent a standardized mixed-meal tolerance test. Hepatic metabolic fluxes were quantified in real-time using a combination of [1-¹³C]acetate and [6,²H₂]glucose stable isotope tracers,

coupled with continuous breath ¹³CO₂ analysis. Blood was sampled for metabolomics and inflammatory cytokines at multiple timepoints. A machine learning model integrated baseline multi-omics data (gut metagenomics, serum metabolomics) to predict the dynamic metabolic response.

Results:

Following the meal challenge, the SH group exhibited a 40% greater initial spike in hepatic de novo lipogenesis (DNL) flux compared to the Balanced group ($p < 0.001$). However, this was followed by a significantly delayed and blunted suppression of endogenous glucose production, indicating defective pathway switching. This “metabolic inflexibility” score strongly correlated with postprandial spikes in serum IL-1 β and liver stiffness measurement ($r = 0.69$). The SH gut microbiome was enriched in bacterial genes for rapid carbohydrate fermentation. The machine learning model accurately predicted an individual’s postprandial DNL response from baseline data (AUC=0.84), with the gut microbial signature being the top predictive feature.

Conclusions:

This study redefines the TCM Stomach-Heat constitution through the lens of dynamic systems biology, identifying it as a state of impaired hepatic metabolic flexibility in response to feeding. The trans-omic AI model bridges baseline constitution features with real-time metabolic pathophysiology. This novel framework allows for the early identification of high-risk individuals and suggests personalized, meal-timed interventions (e.g., prebiotics or pharmacological modulators of nutrient sensing) to restore metabolic plasticity and prevent MASLD progression.

Keyword(s) : Stomach-Heat Constitution, Metabolic Flexibility, Stable Isotope Tracers, Postprandial Metabolism, MASLD, Systems Biology

2285 - Blood Stasis EVs Reprogram Hepatic Stellate Cells via YAP

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Background / Hypothesis:

The Blood-Stasis (BS) constitution, a core concept in Traditional Chinese Medicine (TCM), is clinically linked to fibrotic progression in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Beyond static circulatory impairment, we hypothesize that BS dynamically communicates pro-fibrotic signals through liver sinusoidal endothelial cell (LSEC)-derived extracellular vesicles (EVs). These “BS-EVs” exhibit a

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unique proteomic cargo that aberrantly activates the mechanosensitive YAP/TAZ pathway in hepatic stellate cells (HSCs), driving a persistent fibrogenic phenotype.

Materials and Methods:

A discovery cohort of 80 MASLD patients (40 BS, 40 Balanced) was phenotyped using the CCMQ and nailfold capillaroscopy. LSEC-EVs were isolated from peripheral blood using CD146-immunoaffinity capture and characterized by nanoparticle tracking analysis and proteomics. Functional validation was performed by treating primary human HSCs with purified EVs.

Mechanistic interrogation involved YAP/TAZ inhibition, phosphoproteomics, and a murine model of carbon tetrachloride-induced fibrosis with adoptive transfer of human BS-EVs.

Results:

Patients with BS constitution harbored a significantly higher concentration of circulating LSEC-EVs with a distinct surface phosphatidylserine exposure. Proteomic analysis revealed BS-EVs were enriched in fibronectin and mechanotransduction-related proteins (e.g., ANXA2). Treatment of quiescent HSCs with BS-EVs, but not control EVs, triggered rapid YAP nuclear translocation, actin cytoskeleton remodeling, and a pro-fibrotic gene signature (α -SMA, COL1A1). This effect was abolished by a YAP inhibitor. In vivo, mice receiving BS-EVs developed exacerbated liver fibrosis, characterized by expanded α -SMA+ areas. A diagnostic model combining serum EV concentration and YAP activity signature predicted significant fibrosis ($F \geq 2$) with an AUC of 0.87.

Conclusions:

This study pioneers the concept of "constitution-specific EVs," identifying BS-EVs as novel, circulating mediators that translate a TCM constitutional state into a concrete pro-fibrotic molecular program via YAP/TAZ activation. It redefines blood stasis from a hemodynamic phenomenon to an active, vesicle-based signaling disorder. Targeting the BS-EV-YAP axis offers a promising, constitution-precise therapeutic strategy to halt fibrosis in MASLD.

Keyword(s) : Blood-Stasis Constitution, Extracellular Vesicles, Liver Sinusoidal Endothelial Cells, YAP/TAZ Pathway, Hepatic Stellate Cells, MASLD Fibrosis

2286 - Atopic Constitution Primes Liver Metastasis via Gut EV Immunomodulation

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Background / Hypothesis:

The Atopic constitution, predisposed to allergic and

immune-dysregulated states, may create a permissive microenvironment for hepatic complications beyond simple steatosis. We hypothesize that in Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), the Atopic phenotype drives increased intestinal permeability, leading to systemic release of gut-derived, immune-modulatory extracellular vesicles (EVs). These EVs preferentially home to the liver, where they reprogram hepatic macrophages toward an immunosuppressive phenotype, thereby priming a fertile soil for hepatocellular carcinoma (HCC) development and metastasis.

Materials and Methods:

A nested case-control study was conducted within a prospective MAFLD cohort (n=2,500). Atopic constitution was identified using a validated TCM questionnaire combined with serum IgE and T-helper 2 (Th2) cytokine profiling. Plasma EVs were isolated from Atopic and non-Atopic subjects, characterized by nanoparticle tracking analysis and proteomics, with gut origin confirmed by intestinal fatty acid-binding protein (I-FABP) and microbial markers. Their effects on primary human monocyte-derived macrophages and hepatic macrophage cell lines were assessed. Liver biopsies were analyzed by multiplex immunofluorescence for macrophage polarization markers (CD206, iNOS) and pre-metastatic niche factors. Findings were validated in a diet-induced MAFLD mouse model with ovalbumin-induced atopy.

Results:

Patients with Atopic constitution had significantly elevated levels of plasma EVs carrying a distinct cargo rich in heat shock proteins (e.g., HSP90) and Th2-skewing cytokines. These EVs were positively correlated with gut permeability markers (serum I-FABP, $r=0.62$) and hepatic steatosis-fibrosis scores. In vitro, Atopic-derived EVs potently induced an M2-like, immunosuppressive phenotype in macrophages, characterized by increased CD206 and IL-10 expression. This was associated with suppressed T cell activation capacity. In liver tissues, Atopic MAFLD patients showed a significant expansion of CD206+ macrophages within steatotic areas compared to controls. The murine model confirmed that atopic sensitization accelerated high-fat diet-induced liver injury and increased the number and immunosuppressive function of hepatic macrophages, which was transferable via adoptive transfer of donor plasma EVs.

Conclusions:

This study reveals a novel "gut-liver-immune" axis where the TCM Atopic constitution exacerbates MAFLD progression towards a protumorigenic state. The

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mechanism involves gut barrier dysfunction and the release of specialized EVs that systemically educate hepatic macrophages towards immunosuppression. This positions the Atopic constitution as a novel, biologically defined high-risk phenotype for HCC in MAFLD and identifies gut-derived EVs and their cargo (e.g., HSP90) as potential therapeutic targets and early risk stratification biomarkers.

Keyword(s) : Atopic Constitution, Extracellular Vesicles (EVs), Macrophage Polarization, MAFLD, Tumor Microenvironment, Gut-Liver Axis

2287 - Bioactive Folate Supplementation Mitigates Valproic Acid-Induced Liver Injury and Steatosis in a Rat Model

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Background and Aim: Drug-induced liver injury is a major cause of acute and chronic liver disease. Of pharmacotherapies, antiepileptic drugs-notably, valproic acid-are well-documented causes of hepatic steatosis and mitochondrial dysfunction. VPA-induced fatty liver is a unique form of DILI that exacerbates the progression of NAFLD by interfering with folate metabolism, increasing oxidative stress, and activating inflammatory signaling pathways. The present study aimed to evaluate the impact of bioactive folates, folic acid and 5-methyltetrahydrofolate, on sodium valproate-induced nonalcoholic fatty liver disease in rats. Specifically, this study assessed whether folate supplementation would prevent hepatic steatosis and inflammation by mitigating valproic acid induced disruption of folate metabolism and restoring one-carbon cycle functionality.

Methods:Adult female Sprague Dawley rats were divided into three groups: control group, diseased group treated with valproic acid, and diseased + treated group treated with valproic acid and mixture of bioactive folate. Comprehensive analysis including liver function test, histopathological examination, gene expression analysis and molecular docking was carried out.

Results:Liver function tests indicated that the increase in alanine aminotransferase, alkaline phosphatase, and total bilirubin levels was highly significant in the diseased group, indicative of hepatic injury. Histopathological examination demonstrated severe steatosis in VPA-treated animals, which was

significantly improved in the folate-treated group. The NAFLD Activity Score, a mild NAFLD was observed in the diseased group with a total score of 2. Gene expression studies showed high upregulation of HO-1 and pro-inflammatory cytokines TNF- α and IL-6 in the diseased group, whereas downregulation was observed in folate-treated rats. Furthermore, molecular docking studies indicated that folic acid and 5mTHF showed high affinities for binding to KEAP1 protein, suggestive of activation of the Nrf2-KEAP1 antioxidant pathway. Conclusion:Collectively, our data provide significant evidence for the therapeutic approach of bioactive folate supplementation in the amelioration of the VPA-induced liver injury through modulation of oxidative stress, inflammation, and lipid accumulation.

Keyword(s) : DILI, NAFLD, valproic acid, bioactive folates, KEAP1, liver histology, oxidative stress, Nrf2.

2288 - Adverse reaction report of Peg IFN α 2b in 14 cases of compensated hepatitis B liver cirrhosis patients treated with NAs

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Background / Hypothesis:

Chronic hepatitis B (CHB) is an important cause of liver cirrhosis and hepatocellular carcinoma (HCC). For CHB patients treated with nucleoside analogues (NAs), combining pegylated interferon α -2b (Peg-IFN- α -2b) can increase the possibility of clinical cure. However, the risk of adverse reactions in patients with compensated liver cirrhosis is relatively high, and the safety is still controversial, so close monitoring is required.

Materials and Methods:

A retrospective analysis was conducted on the data of 14 patients with compensated hepatitis B cirrhosis who had been treated with nucleos(t)ide analogs (NAs) and visited the Affiliated Hospital of Yan'an University from January 2019 to June 2025. On the basis of continuing NAs treatment, all patients were combined with Peg - IFN - α - 2b (180 μ g/week, subcutaneous injection). The adverse reactions that occurred during the treatment were recorded, and their composition and outcomes were analyzed.

Results:



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During the treatment process, the incidence of adverse reactions was relatively high, mainly manifested as follows:

Influenza-like symptoms (fever, headache, myalgia, etc.): 11 cases (78.57%);

Manifestations of bone marrow suppression: bleeding of the gums or skin and mucous membranes in 5 cases (35.71%), decreased white blood cell count in 4 cases (28.57%);

Endocrine and skin reactions: hyperthyroidism in 3 cases (21.43%), hair loss in 5 cases (35.71%), skin rash in 3 cases (21.43%);

Others: 1 case each of hypertension, anxiety, and erectile dysfunction (7.14% each). All adverse reactions improved after symptomatic treatment or drug withdrawal.

Conclusions:

Combined treatment with Peg - IFN - α - 2b in treatment - experienced compensated hepatitis B liver cirrhosis patients with NAs can achieve partial clinical cure, but adverse reactions are relatively common, especially influenza - like symptoms and manifestations related to bone marrow suppression. In clinical practice, blood routine, thyroid function, liver function and neuropsychiatric status should be closely monitored, and timely intervention should be carried out to ensure the safety of treatment.

Keyword(s) : Compensated hepatitis B cirrhosis; Pegylated interferon; Adverse reactions; Clinical cure

2289 - Tumor burden score combined with alpha fetoprotein stratification Evaluating efficacy of triple conversion therapy in unresectable hepatocellular carcinoma – A multicenter retrospective study

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Background / Hypothesis:

Hepatocellular carcinoma is a common malignant tumor worldwide. With the development of immunotherapeutic agents, triple conversion therapy (Transarterial Chemoembolization+Tyrosine Kinase Inhibitor+Immune Checkpoint Inhibitors) has emerged as an effective option for patients with unresectable hepatocellular carcinoma. This study aims to combine the tumor burden score and alpha-fetoprotein to stratify patients, identify the population with the optimal benefit, and provide a decision-making basis for clinical diagnosis and treatment.

Materials and Methods:

This multicenter retrospective study included 359 patients with unresectable hepatocellular carcinoma. A novel scoring index was developed by integrating the tumor burden score and alpha-fetoprotein, which was then used to stratify the patients. The Cox proportional hazards model was applied to assess the association of this new index with patient survival and tumor progression. Kaplan-Meier curves were generated, and the log-rank test was conducted to compare differences in overall survival and progression-free survival among different stratified subgroups, with the aim of identifying the population that obtains the optimal benefit from triple therapy.

Results:

In the Cox regression analysis, TBA score value ($p=0.048$; high vs. middle hazard ratio [HR]: 2.775; 95% confidence interval [CI]: 1.231-6.258; middle vs. low HR: 1.943; 95% CI: 0.995-3.794) and undergoing hepatectomy after triple therapy were identified as independent risk factors for patients' overall survival (OS). For progression-free survival (PFS), the independent risk factors included tumor number, TBA score value ($p=0.008$; high vs. middle HR: 2.338; 95% CI: 1.322-4.135; middle vs. low HR: 1.817; 95% CI: 1.189-2.778), and undergoing hepatectomy after triple therapy. In the Kaplan-Meier curve and log-rank test, the low TBA group exhibited the best prognosis: the median progression-free survival (PFS) was 24.3 months, and the median OS was not reached. This outcome was significantly superior to that of the middle TBA group and high TBA group. Notably, the lower the TBA score value, the better the efficacy of conversion therapy in patients.

Conclusions:

The TBA score value, which integrates tumor burden

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score and alpha-fetoprotein, can effectively stratify the prognosis of unresectable hepatocellular carcinoma patients receiving triple conversion therapy. Further validation of its broad clinical applicability is required in larger sample sizes and different systemic treatment regimens.

Keyword(s) : TBA score value, unresectable hepatocellular carcinoma, triple conversion therapy, prognosis stratification

2293 - Rhabdomyolysis triggered acute on chronic liver failure in alcohol related cirrhosis a rapidly fatal multiorgan failure case

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) is characterised by acute decompensation of cirrhosis with extrahepatic organ failures and high short-term mortality. While infection is the most common precipitant, non-infectious inflammatory insults such as rhabdomyolysis are increasingly recognised but remain under-reported. Rhabdomyolysis, caused by muscle breakdown with release of myoglobin, creatine kinase (CK) and pro-inflammatory mediators, can exacerbate renal injury, metabolic acidosis and systemic inflammation, thereby accelerating hepatic decompensation. We hypothesised that severe rhabdomyolysis may act as a rare but clinically significant trigger of ACLF in alcohol-related cirrhosis.

Materials and Methods:

We describe a 74-year-old man with recently diagnosed alcohol-related liver disease who presented after a fall and prolonged immobilisation. Clinical data, laboratory results, imaging and CLIF-C ACLF scoring were reviewed to assess disease progression and prognosis. Supportive interventions included intensive care admission, continuous renal replacement therapy (CRRT), rhythm control for atrial fibrillation, and broad monitoring for infection and systemic inflammation

Results:

On admission, the patient had severe rhabdomyolysis (CK 40,721 U/L), acute kidney injury stage 3, metabolic acidosis, coagulopathy (INR 2.31) and hepatic encephalopathy. His CLIF-C ACLF score was 61, predicting high short-term mortality. Despite CRRT,

electrolyte correction and maximal supportive care, multiorgan failure progressed. Inflammatory markers remained persistently elevated (CRP 122–150 mg/L) without a clear infective source. He developed new-onset atrial fibrillation, managed with amiodarone and digoxin. Muscle injury showed biochemical improvement (CK falling to 765 U/L), yet hepatic and renal dysfunction worsened. His CLIF-C ACLF score rose to 75, consistent with extremely poor prognosis (<20% 28-day survival). Following multidisciplinary discussions, treatment was deemed futile and care transitioned to palliation. He died four days after presentation.

Conclusions:

This case highlights rhabdomyolysis as an uncommon but important precipitant of ACLF in alcohol-related cirrhosis. Prolonged immobilisation led to extensive muscle necrosis, with myoglobin-induced renal injury and systemic inflammation driving hyperacute multiorgan failure. Despite aggressive organ support, prognosis remained dismal, as reflected by CLIF-C ACLF scoring. Recognition of rhabdomyolysis as a potential ACLF trigger is essential, and objective scoring systems provide valuable guidance for prognosis and goals-of-care decisions.

Keyword(s) : Acute-on-chronic liver failure (ACLF), Rhabdomyolysis, CLIF-C ACLF score, Alcohol-related cirrhosis

2295 - Acute methyl ethyl ketone peroxide poisoning presenting with polycythemia

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Background / Hypothesis: Methyl ethyl ketone peroxide (MEKP) is widely used as a hardener in the production of synthetic rubbers in industrial applications. Ingestion of MEKP causes proximal airway edema and obstruction, corrosive burns leading to stenosis, inhalation pneumonia or acute respiratory distress syndrome, acidosis, liver failure, and kidney failure. MEKP is a colorless liquid indistinguishable from drinking water in appearance and is easily ingested accidentally in daily life. We present this case of someone who accidentally drank about 100 cc from a water bottle filled with MEKP.

Case: A 29-year-old male patient accidentally ingested approximately 100 cc of a 33% methyl ethyl ketone



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peroxide (MEKP) solution from a clear plastic bottle while working at his workplace. Upon admission to the emergency department, his blood pressure was normotensive (120/70 mmHg), temperature was normal (36.8 °C), and oxygen saturation was normal (99%), but he had tachycardia (120/min) and tachypnea (25/min). Other physical examination findings were normal. Blood tests performed in the emergency department showed a white blood cell count (WBC): $33730 \times 10^9/L$, HGB: 21.3 gr/dL, and PLT: $351000 \times 10^9/L$. The patient was prescribed intravenous hydration, high-dose pantoprazol, antiemetic therapy, ampicillin-sulbactam, and N-acetylcysteine. Approximately 12 hours after admission, the patient underwent esophagogastroduodenoscopy, which revealed grade 2 corrosive damage in the esophagus. During follow-up, the patient's WBC count rose to $40250 \times 10^9/L$ on day 1 of hospitalization, and the HGB count increased to 22.9 g/dL on day 1. These levels gradually regressed, returning to normal values on day 9. The patient's creatinine level, however, progressed during follow-up, rising to 2.4 mg/dL. The patient, who also had acidosis in blood gas analysis, was referred to nephrology, and hydration and monitoring were recommended, but dialysis was not considered. Creatinine levels began to regress from day 3 of hospitalization and returned to normal values on day 9. Liver enzymes, which were three times higher than normal upon admission, increased up to eight times during follow-up and then progressively regressed.

Conclusion:MEKP is a potentially deadly chemical. Ingestion can cause both corrosive damage and acute organ failure in organs such as the liver and kidneys. However, unlike previous cases, this particular case developed severe polycythemia and recovered without any lasting damage thanks to supportive treatment. Raising public awareness and educating the public is crucial to prevent such toxicities.

Keyword(s) :

2298 - Divergent Oncologic and Survival Implications of Stiffness Regression After SVR A Decade Long Longitudinal FibroScan Study in Advanced Fibrosis

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Background

Although liver stiffness regression after sustained virologic response (SVR) is widely interpreted as a marker of fibrosis reversal, its clinical implications across fibrosis stages remain unclear. We aimed to characterize long-term stiffness trajectories and their associations with hepatocellular carcinoma (HCC) incidence and overall survival (OS).

Methods

We analyzed 657 SVR patients with ≥ 2 FibroScan assessments over 10 years. Annual stiffness change (slope, kPa/year) was estimated using mixed-effects models. Stiffness regression was defined according to QIBA criteria. Outcomes included HCC incidence and OS, stratified by fibrosis group (F0–2, F3, F4).

Results

Two distinct post-SVR stiffness trajectories emerged.

Regression patients showed a sustained decline in stiffness (-0.51 kPa/year), whereas non-regression patients remained stable around 9–10 kPa.

Importantly, the clinical implications of regression differed sharply by fibrosis stage.

HCC incidence: In F3 disease, stiffness regression tended to reduce HCC incidence, although not statistically significant.

In contrast, in F4 disease, regression did not suppress HCC risk, and cumulative incidence curves for regression and non-regression groups were nearly identical, indicating that oncogenic risk persists despite stiffness improvement.

Overall survival: In F3, OS was similarly excellent regardless of regression status.

However, in F4, regression was strongly associated with improved OS, whereas non-regression patients had significantly poorer survival. Thus, stiffness regression is oncologically uncoupled from HCC reduction but prognostically coupled with survival benefit in cirrhosis.

Conclusions

Stiffness regression after SVR has fundamentally different implications across fibrosis stages. In cirrhosis, stiffness improvements do not mitigate HCC risk but remain a powerful determinant of survival.

Keyword(s) : Hepatitis C, SVR, Fibrosis, Regression, OS

2299 - Shifting hepatitis A virus seroprevalence among healthcare workers and the general population



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Background / Hypothesis

Hepatitis A virus (HAV) epidemiology has changed markedly in recent years, both globally and in Türkiye. Improved sanitation and routine childhood vaccination have substantially reduced early-life exposure. Within the national immunization program, children born after March 2011 have been vaccinated against HAV, leading to decreased susceptibility during childhood. In parallel, seronegative healthcare workers are recognized as a risk group and vaccinated when identified. As a result, HAV susceptibility has shifted toward adolescents and young adults. This study aimed to evaluate age-specific HAV seroprevalence patterns among healthcare workers and the general population.

Materials and Methods

This retrospective observational study evaluated anti-hepatitis A virus (HAV) immunoglobulin G (IgG) test results obtained from a tertiary care center. The study was conducted at İzmir City Hospital, a newly established institution with a high number of newly recruited healthcare workers. Anti-HAV IgG results of healthcare workers and individuals from the general population who attended the hospital between October 23, 2023, and September 30, 2025 were retrieved from electronic medical records and the Occupational Health Unit database. Seroprevalence was analyzed according to predefined age groups. Data were summarized as numbers and proportions, and comparisons were performed using the chi-square test, with $p < 0.05$ considered statistically significant.

Results

A total of 3,363 healthcare workers aged 19–68 years and 6,230 individuals from the general population aged 17–98 years were included. Among healthcare workers, HAV seronegativity was highest in the youngest age groups and declined progressively with age. Higher seronegativity was observed up to 35 years, with a marked decrease thereafter, although seronegative individuals remained present in the 40–45-year age group. A similar age-related trend was observed in the general population, characterized by higher seronegativity in younger individuals and increasing seropositivity with advancing age. Overall, seronegativity rates were lower among healthcare

workers, particularly among those born in 2000 or later, likely reflecting vaccination practices.

Conclusions

HAV seronegativity remains significantly higher among young adults under 35 years of age in both healthcare workers and the general population, while seropositivity increases with age, indicating a favorable shift in HAV epidemiology. Despite lower seronegativity among younger healthcare workers due to vaccination, expanding serological screening and vaccination remains essential. In the general population, HAV serology should be assessed regardless of age in the presence of risk factors such as chronic hepatitis (HBV or HCV), immunosuppression, or advanced age, and seronegative individuals should be vaccinated.

Keyword(s) : hepatitis A virus, seroprevalence, young adults, healthcare workers, epidemiologic shift

2301 - Gastric Variceal Rupture due to Sinusoidal Obstruction Syndrome after Oxaliplatin Chemotherapy

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Background / Hypothesis: While sinusoidal obstruction syndrome (SOS) is a known side effect of oxaliplatin, the presentation of gastric variceal rupture is rare.

Materials and Methods: A male patient in his 50s was transported to our hospital by emergency services complaining of hematemesis, 1 year and 7 months after completing 12 cycles of Modified Fluorouracil + Folinic Acid + Oxaliplatin (mFOLFOX) chemotherapy following surgical resection of a hepatic metastasis and primary sigmoid colon cancer. A CT scan revealed marked hepatosplenomegaly and the development of extrahepatic collateral circulation, which had not been observed before the chemotherapy. Upper gastrointestinal endoscopy confirmed a ruptured gastric varix. The rupture was attributed to gastric varices secondary to oxaliplatin-induced SOS.

Results: Since temporary hemostasis had been achieved, emergency Balloon-Occluded Retrograde Transvenous Obliteration (BRTO) was performed. The patient experienced no further recurrence of symptoms thereafter.

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Conclusions: Patients who have undergone oxaliplatin-based chemotherapy require careful long-term follow-up, considering the risk of esophagogastric variceal rupture.

Keyword(s) : oxaliplatin, sinusoidal obstruction syndrome, ruptured gastric varix

2303 - Predictive Value of Peripheral Blood T Cell Counts for HBsAg Clearance in Pediatric Patients with Chronic Hepatitis B Receiving Antiviral Therapy

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Background / Hypothesis:

Response to antiviral therapy varies among pediatric patients with chronic hepatitis B (CHB). This study aims to investigate the predictive value of peripheral blood T cell counts for HBsAg clearance in this population.

Materials and Methods:

A total of 288 HBsAg-positive children with chronic CHB aged 1–18 years old were randomly enrolled. All subjects received 48 weeks of standardized antiviral treatment and subsequent follow-up. The serological indicators were measured through routine laboratory operating procedures. According to the HBsAg clearance status after treatment, the children were divided into the HBsAg clearance group and the non-clearance group. Statistical analysis was used to clarify the correlation between T cell counts and treatment efficacy, and the area under the receiver operating characteristic curve (AUROC) was used to evaluate its predictive value.

Results:

HBsAg clearance was achieved in 26.04% of the enrolled children. The HBsAg clearance group exhibited significantly higher baseline total T cell counts, 12-week CD4+ T cell counts, and 12-week CD8+ T cell counts compared to the non-clearance group; these metrics were negatively correlated with 48-week HBsAg levels ($P < 0.05$). The combined use of these three indicators for predicting HBsAg clearance yielded an AUROC of 0.8078 ($P < 0.0001$), with a sensitivity of 73.3% and specificity of 80.2%.

Conclusions:

The baseline total count of peripheral blood T lymphocytes, the 12-week CD4+ T cell count, and the 12-week CD8+ T cell count are effective predictive indicators for evaluating HBsAg clearance in children with CHB. The combined application of the three indicators can improve the accuracy of prediction and provide valuable references for clinical efficacy evaluation.

Keyword(s) : Chronic hepatitis B; T lymphocyte; Hepatitis B surface antigen clearance; Antiviral therapy

2304 - Hepatocellular carcinoma detection using pre transplant triphasic computed tomography and correlation with explant pathology

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Background / Hypothesis:

Triphasic CT is widely used to stage hepatocellular carcinoma (HCC) before liver transplantation, yet radiology can underestimate tumor burden relative to explant pathology—especially for multifocal or early lesions. Meta-analyses and guideline frameworks (e.g., Liver Imaging Reporting and Data System) show that CT's sensitivity for HCC is lower than MRI and drops further for early-stage disease, while explant may reveal more tumors than imaging suggests. We aimed to find out the diagnostic yield of triphasic CT scan for HCC in correlation with explant findings.

Materials and Methods:

We retrospectively reviewed 46 consecutive living-donor liver transplants at our new liver transplant program. Pre-transplant triphasic CT scans were compared with explant pathology.

Results:

Of the 46 patients, 41 (89%) were male, with a mean age of 45.2 ± 10.3 years. On computed tomography (CT), 17 patients (36.9%) were diagnosed with hepatocellular carcinoma (HCC). Explant pathology confirmed HCC in 18 patients (39.1%). The additional patient had a dysplastic nodule on CT imaging, accompanied by elevated PIVKA-II levels but normal alpha-fetoprotein (AFP) levels. PIVKA-II was elevated in

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all patients with HCC, whereas AFP was elevated in only 8 of 18 patients (44.4%). Multicentric HCC was detected on CT in 12 patients; however, histopathological examination confirmed multicentric disease in 10 of 18 patients (55.5%).

Conclusions:

In this single-center liver transplant cohort, pre-transplant triphasic computed tomography proved to be a reliable modality for the detection of hepatocellular carcinoma, identifying a proportion of cases comparable to final histopathology. Triphasic CT effectively characterized tumor morphology, including multicentric disease, supporting its continued use as a primary imaging tool in the pre-transplant evaluation of HCC. Additionally, elevated tumor markers—particularly PIVKA-II—served as valuable adjuncts in cases with indeterminate or dysplastic lesions on imaging, enhancing diagnostic confidence and aiding clinical decision-making.

Keyword(s) : hepatocellular carcinoma, triphasic computed tomography, liver transplantation, explant pathology, alpha-fetoprotein, PIVKA-II

2305 - Transcriptomic Characteristics of Peripheral Blood T Cells in Pediatric Chronic Hepatitis B with Different Antiviral Therapy Outcomes

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Background / Hypothesis:

Currently, antiviral therapy is the main method for clinical cure in children with chronic hepatitis B (CHB). However, there are significant interindividual differences in treatment efficacy during antiviral therapy in children. The underlying mechanisms of such differences have not been fully elucidated so far. At present, variations in clinical outcomes of children with CHB after antiviral therapy have become a key bottleneck restricting their functional cure. The purpose of this study is to analyze the transcriptomic characteristics of peripheral blood T cells in children with different clinical outcomes, clarify the key immune cell subsets and target genes associated with HBsAg clearance so as to provide experimental evidence for improving the clinical cure rate of antiviral therapy in children.

Materials and Methods:

A total of 8 pediatric patients with CHB receiving antiviral therapy were enrolled. 10× Genomics single-cell RNA sequencing was performed on peripheral blood T lymphocytes from CHB children with different treatment responses. After data quality control, Seurat software was used for cell clustering and subset annotation to screen differential cell subsets by inter-group comparison. Differential gene expression analysis was conducted on these subsets, combined with GO and KEGG enrichment analyses to clarify key genes mechanism in antiviral response.

Results:

Single-cell RNA sequencing identified multiple peripheral blood T cell subsets. Significant proportional differences of cell subsets existed between groups, with specific subsets enriched in the response group. The proportion of CD4-*ISG*-Treg was significantly higher in the functional cure group ($p = 0.036$) and was negatively correlated with HBsAg levels ($r = -0.8$, $p = 0.017$). *ISG15* was the most significantly upregulated gene in CD4-*ISG*-Treg, and the baseline plasma *ISG15* level was significantly higher in the HBsAg clearance group than in the non-clearance group ($p = 0.0002$). The area under the receiver operating characteristic curve (AUROC) of *ISG15* for predicting HBsAg clearance was 0.749 (95% confidence interval [CI] = 0.633 – 0.865, $p < 0.0001$), with an optimal cutoff value of 176.87 pg/mL, a sensitivity of 83.8%, and a specificity of 55.2%. The "*ISG15* antiviral mechanism" pathway was highly enriched in CD4-*ISG*-Treg; *ISG15* promoted the development of this subset.

Conclusions:

The CD4-*ISG*-Treg subset plays a pivotal role in HBsAg clearance. *ISG15* maybe a potential therapeutic target in pediatric patients with CHB, which may help improve the functional cure rate.

Keyword(s) : Chronic hepatitis B; Antiviral therapy; T lymphocytes; Transcriptomic characteristics

2307 - The Thirteen Year Shadow Delayed Biliary Cirrhosis Following Gallbladder Tuberculosis in a Young Filipino Female

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Background

Gallbladder tuberculosis (GBTB) is an extremely rare form of extrapulmonary TB, even in areas where TB is common with a global prevalence of 1% of all abdominal TB cases. Similar to global data, specific large-scale prevalence data for isolated Gallbladder TB in the Philippines is limited and primarily documented through case reports. Its nonspecific clinical presentation frequently leads to misdiagnosis as more common hepatobiliary conditions, with definitive diagnosis often established only through histopathological examination following surgery. Chronic inflammation from hepatobiliary TB may result in biliary obstruction or strictures, leading to prolonged cholestasis, progressive hepatic injury, and eventual secondary biliary cirrhosis.

CASE PRESENTATION:

We report a case of a 32-year old Filipino female who presented with progressive abdominal distention, jaundice and bloatedness. Thirteen years, the patient was diagnosed with gallbladder tuberculosis after presenting with Acute Cholecystitis for which she underwent Laparoscopic Cholecystectomy. Histopathology report revealed GBTB and she completed a full course of anti-tuberculous therapy with apparent clinical resolution. Despite successful treatment, she developed a delayed sequela: an extrahepatic biliary stricture resulting in chronic cholestasis and progressive liver damage.

Her current presentation reflected decompensated liver cirrhosis with evidence of severe portal hypertension, including moderate ascites, hypersplenism, splenomegaly, and the formation of portosystemic collaterals.

Esophagogastroduodenoscopy (EGD) performed revealed one column of small esophageal varices and small gastric varices, without portal hypertensive gastropathy.

Laboratory studies showed markedly elevated bilirubin levels and severe cholestasis. Abdominal MRI confirmed features of advanced cirrhosis, demonstrated pronounced hepatic duct dilatation at the confluence, and characterized the high-grade extrahepatic biliary stricture as the likely driver of her deteriorating hepatic function.

Given her young age and lack of other significant comorbidities, liver transplant was offered as the definitive treatment option; however, the patient refused the procedure. The patient was managed medically for her portal hypertension with the non-selective beta-blocker Carvedilol and IV albumin infusion.

Conclusions:

This case highlights the importance of long-term surveillance following localized hepatobiliary tuberculosis, even after successful primary medical treatment. Fibrotic and structuring process initiated by the initial Mycobacterium tuberculosis infection can progress insidiously over years, culminating in secondary biliary cirrhosis and life-threatening portal hypertension. Managing these complex late complications requires addressing both the ongoing effects of biliary obstruction and the consequences of portal hypertension. Increased awareness of these rare but severe late sequelae is essential, particularly in TB-endemic regions.

Keyword(s) :

2308 - Application of Anti HBc in Clinical diagnosis for Pediatric Hepatitis B

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Background / Hypothesis:

Chronic hepatitis B (CHB) is often accompanied by liver inflammation, and invasive liver biopsy remains the gold standard for assessing inflammatory severity. Serum quantitative hepatitis B core antibody (qanti-HBc) has emerged as a potential non-invasive biomarker for liver disease activity in adult CHB patients, but its diagnostic value in pediatric populations remains unclear. This study aimed to investigate the role of qanti-HBc in diagnosing significant liver inflammation in treatment-naïve HBeAg-positive children.

Materials and Methods:

A total of 60 HBeAg-positive children with CHB were enrolled in this study. Baseline serum qanti-HBc levels were quantified using a double-antigen

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immunosandwich assay, and liver biopsy specimens were evaluated for histological inflammation (G0–4) and fibrosis (S0–4) stages by experienced pathologists. Patients were divided into non-significant (G0–1) and significant (G2–4) liver inflammation groups. Pearson correlation, multifactorial logistic regression, and receiver operating characteristic (ROC) curve analyses were performed to assess the diagnostic performance of qanti-HBc.

Results:

Baseline qanti-HBc levels showed a significant positive correlation with liver tissue inflammation grade ($r=0.60$, $p<0.001$) and fibrosis stage ($r=0.29$, $p=0.019$). Multifactorial regression analysis identified baseline qanti-HBc (odds ratio [OR]=10.910, $p=0.013$) as the only independent predictor of significant liver inflammation in pediatric CHB. The area under the ROC curve (AUROC) of qanti-HBc for diagnosing significant liver inflammation was 0.815 (95% confidence interval [CI]: 0.708–0.921), which was superior to ALT (AUROC=0.624, 95%CI: 0.484–0.763) and AST (AUROC=0.710, 95%CI: 0.484–0.763). A cutoff value of qanti-HBc $>4 \log_{10}$ IU/mL yielded a sensitivity of 67.0%, specificity of 88.0% for significant liver inflammation.

Conclusions:

Serum qanti-HBc maybe a new non-invasive diagnostic indicator for significant liver inflammation in treatment-naïve HBeAg-positive children.

Keyword(s) : Children; Chronic hepatitis B; Liver inflammation; Quantitative hepatitis B core antibody

2312 - A Rare Case Report of Primary Hepatic Lymphoma Complicated by Hemophagocytic Lymphohistiocytosis

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Background / Hypothesis:

Primary hepatic lymphoma (PHL) is a rare disease characterized by non-specific clinical manifestations, laboratory findings, and imaging features, which may lead to misdiagnosis as hepatocellular carcinoma or hepatic infectious lesions. However, whether PHL can secondarily develop hemophagocytic lymphohistiocytosis (HLH) like other hematological lymphomas has not been reported.

Materials and Methods:

We present a diagnostically challenging case initially mimicking a pyogenic liver abscess. A 74-year-old woman with no history of past illness was admitted with persistent high fever accompanied by fatigue and loss of appetite for three weeks. Her vital signs were normal on physical examination. Computed tomography, magnetic resonance imaging, and 18F-FDG PET/CT showed a liver infectious lesion as a hepatic abscess. A subsequent laparoscopic exploration and liver biopsy confirmed the diagnosis of germinal center B-cell-like diffuse large B-cell lymphoma. Concurrently, with the progressive thrombocytopenia and hemoglobin reduction alongside persistent fever, the life-threatening complication of hemophagocytic lymphohistiocytosis (HLH) secondary to PHL was first reported by the further workup per the HLH-2004 criteria.

Results:

The modified R-ECHOP chemotherapy regimen was initiated to address both conditions. While during subsequent follow-up evaluations, significant myelosuppression was identified in the patient. The second-cycle chemotherapy regimen was modified to the R-miniCHOP protocol. After four cycles of chemotherapy, the follow-up PET-CT scan indicated complete response of the liver tumor lesions.

Conclusions:

PHL is rare and frequently clinically mistaken for a liver abscess. Histopathological examination remains the gold standard for definitive diagnosis. Similar to other hematological lymphomas, PHL may also secondarily develop HLH. Early recognition, diagnosis, and intervention are critical to improving prognosis.

Keyword(s) : hepatic abscess, diffuse large B-cell lymphoma, primary hepatic lymphoma, hemophagocytic lymphohistiocytosis, case report

2313 - The role of hepatic CD36 in metabolic dysfunction associated steatotic liver disease

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Background/Hypothesis: CD36, a type B scavenger receptor, exhibits multifunctionality and conserved primary structure across various mammalian and vertebrate cell types. It facilitates fatty acid binding and

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transport, with increased expression observed in hepatocytes and circulating systems of metabolic dysfunction-associated steatotic liver disease (MASLD) patients and animal models, suggesting a potential role in MASLD pathophysiology. However, conflicting evidence exists regarding whether CD36 expression exacerbates hepatic injury. This study aimed to analyze changes in CD36 expression induced by MASLD and examines its impact on hepatocyte injury and lipid profiles.

Materials and Methods: We developed CD36 genetically-engineered zebrafish lines and hepatocyte lines. MASLD mouse model and zebrafish model were constructed by high fat, high cholesterol diet maintenance. Histological and biochemical examinations were conducted to observe liver injury and regeneration. High-resolution fluorescence imaging was used to observe the dynamic subcellular distribution of CD36. Liquid chromatography mass spectrometry (LC-MS) and QToF desorption electrospray ionization mass spectrometry imaging (MSI)-based lipidomics analyses were performed to identify the lipids difference between liver samples from CD36-engineered models.

Results: CD36 does not significantly influence liver development or regeneration following partial hepatectomy in zebrafish. During liver regeneration, CD36 expression peaks in the acute phase and returns to baseline within seven days. In MASLD models, CD36 levels remain elevated compared to controls, with increased localization to hepatocyte membranes correlating with lipid accumulation. Compared with the wild-type controls, both systemic knockdown and hepatic overexpression of CD36 result in higher hepatic cholesterol and triglyceride levels in the liver, alongside increased expression of inflammatory cytokines (il1b, tnf, il6) but no significant difference in cellular apoptosis. In vitro studies with primary hepatocytes treated with fatty acids reveal that CD36 knockdown cells accumulate more lipids and exhibit elevated ALT and AST levels, whereas CD36 overexpression reduces lipid droplets and enzyme levels, compared with the wild-type controls. LC-MS and MSI analyses consistently demonstrated that CD36 overexpression increases triglycerides while decreasing diacylglycerol levels compared to knockdown models. **Conclusions:** Our data suggest that CD36 has increased expression during acute injury and MASLD. Under persistent metabolic overload, hepatic CD36 appears to help attenuate the accumulation of toxic lipids, and its absence cannot attenuate the steatosis injury.

Keyword(s) : CD36, metabolic dysfunction-associated steatotic liver disease, hepatocytes, lipid accumulation, liver injury.

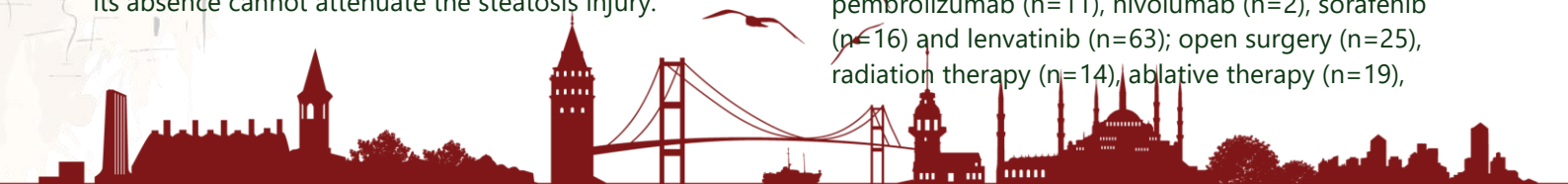
2315 - Epidemiological and clinical characteristics of hepatocellular carcinoma a 10 year registry from tertiary private cancer centre hospital

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Background: Hepatocellular carcinoma (HCC) is the sixth most prevalent cancer and the third leading cause of cancer-related mortality worldwide. HCC accounts for approximately 85–90% of primary liver cancers, followed by intrahepatic cholangiocarcinoma. This study aimed to describe the epidemiological profile and clinical characteristics of HCC patients registered at MRCCC Siloam Hospital Semanggi, Indonesia, over a ten-year period (2013–2022).

Materials and Methods: We conducted a retrospective registry-based analysis of 326 patients diagnosed with HCC between 2013 and 2022. Data collected included demographics (age, sex), performance status (ECOG), etiology of liver disease, imaging findings (tumor size, multifocality, vascular invasion), tumor stage (BCLC, Child–Pugh), type of treatment received and patient follow-up status. Missing or unavailable data were recorded as “unknown.” Descriptive statistics summarized patient characteristics, with categorical variables presented as counts. Trends over the decade were explored for etiology and treatment allocation.

Results: Among 326 patients, most were male (n=248), and the most prevalent age group was ≥65 years. The majority presented with ECOG performance status 3, although some data were missing. HBV was the predominant etiology (n=100), followed by HCV (n=30), NAFLD (n=15), others (n=15), alcohol (n=4) and autoimmune (n=1); a significant proportion of etiology data was unknown. Child–Pugh class A was observed in 33 patients, with remaining data missing. BCLC stages C and D were equally represented (n=28), followed by BCLC stage B (n=19). From imaging finding tumor size >5 cm was observed in 127 patients; 97 patients had multifocal lesions, while 93 had no vascular invasion. Treatments included systemic therapy; bevacizumab (n=11), atezolizumab (n=10), pembrolizumab (n=11), nivolumab (n=2), sorafenib (n=16) and lenvatinib (n=63); open surgery (n=25), radiation therapy (n=14), ablative therapy (n=19),



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Transarterial Chemoembolization (TACE)(n=80) and liver transplantation (n=2). Most patients were lost to follow-up due to multifactorial reasons.

Conclusions: This ten-year registry provides real-world insights into the epidemiology and clinical characteristics of HCC patients in Indonesia. HCC was more common in males, with HBV remaining the predominant etiology. Treatment patterns reflected tumor characteristics and patient condition, while trends over the decade highlight evolving etiological profiles and therapeutic approaches. These findings offer valuable regional data to inform HCC management strategies and future outcome studies.

Keyword(s) : Hepatocellular carcinoma, HCC, HBV, HCV

2316 - Precision ablation with nanosecond pulsed electric field (nsPEF) in early hepatocellular carcinoma a phase I trial integrating multi omics analysis

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Background / Hypothesis:

Nanosecond pulsed electric field (nsPEF) is a promising non-thermal ablation for hepatocellular carcinoma (HCC) in high-risk locations, yet its long-term efficacy and systemic effects remain unclear.

Materials and Methods:

In this pre-specified sub-analysis of a multicenter Phase I clinical trial, we recruited 61 early-stage HCC patients from our center. Extended follow-up and longitudinal multi-omics were performed to assess survival benefits and systemic responses post-ablation.

Results:

Results showed 100% technical success and an initial (1-month) complete ablation response rate of 88.14%. The 1-, 2-, and 3-year actuarial overall survival (OS) rates were 98.18%, 88.89%, and 76.47%, respectively, and the actuarial progression-free survival (PFS) rates were 84.78%, 73.91%, and 62.86%. Most adverse events were grade 1 (97.12%). Multi-omics analysis revealed preoperative enrichment of opportunistic pathogens (e.g., *Massilia* and *Brevundimonas*) in oral and gut microbiota, with postoperative increases in beneficial SCFA-producing bacteria (e.g., *Bacteroides* and *Blautia*). Metabolomics analysis further revealed

significant improvements in the pathways related to energy, amino acid and lipid metabolisms after surgery. Importantly, we found that patients' pre-operative (baseline) microbial and metabolic profiles were associated with treatment response, as those with more favorable profiles achieved significantly higher rates of complete ablation (CR). Building on these findings, we developed a Cox model integrating microbiome, metabolome, and clinical data, with the fecal microbiome-based model performing best (AUCs of 0.802 and 0.822 at 6 and 12 months, respectively).

Conclusions:

This study establishes for the first time that local nsPEF ablation induces dynamic, systemic remodeling of the gut microbiome and plasma metabolome, and pre-ablation multi-omics features are potent predictors of treatment outcome, providing a crucial rationale for personalizing nsPEF therapy and exploring novel microbiome-targeting adjuvant interventions.

Keyword(s) : Nanosecond pulsed electric field (nsPEF), Nonthermal ablation, Hepatocellular carcinoma, Multi-omics, Microbiome, Metabolomics, Prognostic prediction model

2317 - The identification of a novel compound suppressing the early stages of Hepatitis B Virus (HBV) infection

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Background / Hypothesis:

Hepatitis B virus (HBV), an enveloped DNA virus from the Hepadnaviridae family, constitutes a global health problem. As per World Health Organization estimates, HBV has infected nearly 2 billion individuals worldwide, with approximately 257 million suffering from chronic hepatitis B (CHB). Although current antiviral therapies, including nucleos(t)ide analogues (NAs) and pegylated interferon-alpha (PEG-IFN- α), can effectively suppress viral replication, they rarely achieve a functional cure due to the persistence of covalently closed circular DNA (cccDNA) and integrated DNA (iDNA) in infected hepatocytes. Hence, the urgent need for more effective therapeutic options.

Materials and Methods:

Walking in this direction, we conducted a high-throughput screening of 948 featured novel bioactive

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compounds to assess their antiviral activity against HBV. Only one compound (X) meets the selection criteria (more than 5 times suppression of HBV-DNA (extracellular and Intracellular), without affecting the cellular viability.

Results:

Compound X treatment of HBV-infected cells showed a potent decrease in the viral proteins (HBeAg and HBsAg).

Conclusions:

This compound is a promising candidate as novel anti-HBV compound. Further mechanistic and functional investigation are undergoing in vitro/in vivo and will be presented.

Keyword(s) : Hepatitis B virus, HBV, compound screening

2319 - The Cancer Cell Hepatocyte Interface in Colorectal Cancer Liver Metastasis Mechanisms and Clinical Translation

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Background / Hypothesis:

Colorectal cancer liver metastasis is a leading cause of death in patients with colorectal cancer. Growing evidence indicates that the cancer cell–hepatocyte interface constitutes a critical niche that integrates multiple functions, including growth patterns, vascular supply, metabolic processes, and immune evasion. We hypothesize that molecular events occurring at this interface—including transforming growth factor beta (TGF β) driven hepatocyte remodeling and hepatocyte derived exosomes—may explain clinical heterogeneity and reveal novel therapeutic targets.

Materials and Methods:

We conducted a narrative review of experimental and clinical studies retrieved from PubMed and Web of Science. We focused on histopathological growth patterns, molecular pathways regulating hepatocyte injury and remodeling, metabolic and exosomal crosstalk between hepatocytes and tumors, and specifically highlighted studies of clinically annotated

colorectal cancer liver metastasis cohorts that link interface biology to treatment response or prognosis.

Results:

Desmoplastic and replacement growth patterns define two distinct cancer cell–hepatocyte interfaces, which are strongly associated with patient outcomes and sensitivity to anti angiogenic therapy. The replacement growth pattern involves vascular co option, TGF β dependent hepatocyte apoptosis or partial epithelial mesenchymal transition, and profound remodeling of glucose and amino acid metabolism. Hepatocyte derived exosomes, particularly those regulated by Golgi phosphoprotein 73 (GOLPH3) in metabolic liver disease, enhance the migration, invasion, and hepatic seeding capacity of colorectal cancer cells, and shape an immunosuppressive pre metastatic niche conducive to liver dissemination and outgrowth.

Conclusions:

The cancer cell–hepatocyte interface is a central determinant of vascular strategy, metabolic adaptation, and immune evasion in colorectal cancer liver metastasis. Standardized reporting of growth patterns, combined with interface focused molecular biomarkers, may improve risk stratification and guide integrated therapeutic strategies that target both tumor cells and hepatocytes through combined anti angiogenic, metabolic, and immunomodulatory approaches.

Keyword(s) : Colorectal cancer; Liver metastasis; Cancer cell–hepatocyte interface; Vascular co option; Transforming growth factor beta (TGF β); Golgi phosphoprotein 73 (GOLPH3); Exosomes; Pre metastatic niche

2321 - A novel prognostic model to predict mortality in patients with drug induced liver injury in intensive care unit a study from Pakistan

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Background&Aim:

DILI is one of the causes of acute liver injury leading to increased mortality rates&usually requires intensive care unit(ICU)admission,however it is uncommon&often challenging to diagnose DILI.Proper prognosis for these patients is crucial for early referral

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for liver transplantation. In light of paucity of data on DILI especially from South East Asia, this study aims to evaluate the clinical spectrum and predictors of mortality in ICU admitted patients with suspected DILI by building & validating a novel scoring system

Methods:

531 cases were identified and categorized on the basis of COIMS/RUCAM score¹ and exclusion of other liver diseases from 2010-2024. Ratio (R value) between ALT (Alanine transferase) & AP (alkaline phosphatase) expressed as

$R > 5$; hepatocellular, $R < 2$; cholestatic, & $R > 2 < 5$; mixed. Clinical & laboratory parameters were analyzed to identify the predictors of hospital mortality. The prognostic values for mortality were assessed by ROC analysis. A novel model was developed and internally validated using fivefold cross-validation.

Results:

Out of 531 patients, there were 333 (62.7%) males with the mean age being 50.83 years (range, 20-94 years). DILI was classified as definite or highly probable in 31.1%, probable in 62.5%, and possible in 7.4% of cases. Pattern of liver injury was hepatocellular in 25.1%, cholestatic in 56.17% and mixed in 18.72% of patients. Mean total bilirubin levels, ALT and AP levels were 5.37 mg/dl, 358.65 IU/L and 168.68 IU/L respectively.

Antituberculous (ATT) drugs were found to be the most common cause (63.9%) followed by homeopathic or herbal meds (9.5%), antiarrhythmic drugs (8.7%), statins (7.3%), antibiotics (6.1%), antifungals (5.6%), chemotherapeutic agents (4.4%) and antiepileptics (1.7%). Altered mental status was present in 142 (26.7%) of patients while rest presented with abdominal pain, vomiting, jaundice & pruritus; 57.1%, 57.1%, 54.1%, and 42.3% respectively.

In-hospital mortality was 26.5% and prolonged hospital stay was observed in 35.93% of patients. On multivariate analysis, mortality was significantly greater in patients with altered mental status, male gender, increased age, hepatocellular pattern of DILI, increased INR (> 1.5), increased bilirubin levels (> 3 mg/dl) & arterial gradient (A-a gradient). The newly developed prognostic model, incorporating age, gender, pattern of liver injury, alveolar-arterial gradient (A-a gradient), INR, total bilirubin level, INR, and HE grades, exhibited significantly improved performance in predicting 1-month and 3-month mortality with AUROC of 0.863 0.829, respectively. Likewise, prolonged hospital stay was associated with female gender, increased ALT, AST aspartate aminotransferase levels, and mixed pattern of DILI

Conclusion:

DILI leads to increased mortality, & use of this prognostic model predicting mortality in patients with drug-induced liver injury in intensive care unit can be useful. However, further rigorous validation is required in other liver-related ICUs.

Keyword(s) : DILI

2322 - Gut Microbiota Modulation on Non Alcoholic Fatty Liver Disease Progression A Systematic Review and Meta Analysis from Human Trials

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is a growing global health concern with limited treatment options. Gut microbiota dysbiosis has been implicated in NAFLD pathogenesis. This systematic review and meta-analysis aimed to evaluate the effects of gut microbiota modulation strategies on NAFLD progression.

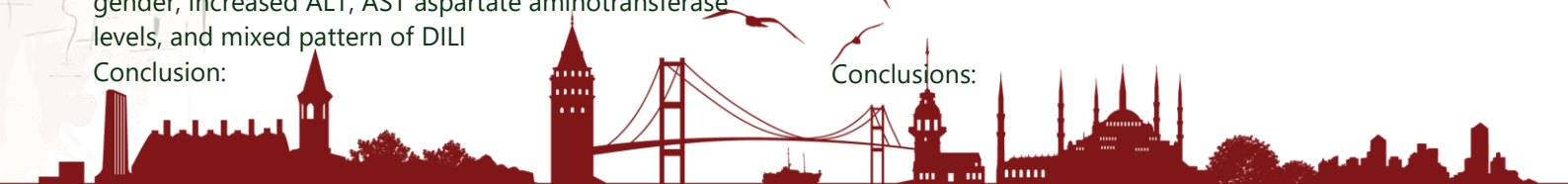
Materials and Methods:

We included randomized controlled trials and prospective cohort studies involving adults and children with NAFLD/NASH receiving probiotics, prebiotics, synbiotics, antibiotics, fecal microbiota transplantation (FMT), or farnesoid X receptor (FXR) agonists compared to placebo or standard care. PubMed, EMBASE, CENTRAL, and ClinicalTrials.gov were searched until March 15, 2025. Risk of bias was assessed using the Cochrane RoB 2.0 tool. A random-effects model synthesized continuous outcomes as mean differences (MD) with 95% confidence intervals (CI).

Results:

Forty studies involving 3,415 participants were included. Probiotics/prebiotics/synbiotics, antibiotics, FMT, and FXR agonists significantly improved liver enzymes (ALT MD: -11.49 U/L, 95% CI: -14.69 to -8.30; AST MD: -5.95 U/L, 95% CI: -6.52 to -5.38). Gut microbial diversity also improved. Heterogeneity was moderate to high across outcomes. Risk of bias was low to some concerns for most studies. Adverse events were mild and infrequent across interventions.

Conclusions:



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Gut microbiota modulation offers a promising therapeutic avenue for improving liver biochemistry and metabolic parameters in NAFLD, although heterogeneity and moderate certainty of evidence warrant cautious interpretation. Larger, standardized trials are needed.

Keyword(s) :

2323 - RNAi Therapies for HBsAg Reduction in Chronic Hepatitis B A Systematic Review and Meta Analysis of Early Phase Trials

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Background / Hypothesis:

Chronic hepatitis B (CHB) remains a major global health burden, with current nucleos(t)ide analogue (NA) therapy achieving functional cure in less than 5% of patients. RNA interference (RNAi)-based therapeutics have emerged as a promising strategy by directly silencing hepatitis B surface antigen (HBsAg) expression. This systematic review and meta-analysis evaluated the efficacy, safety, and early antiviral responses associated with RNAi therapies across Phase I-II human trials.

Materials and Methods:

We included Phase I-II clinical trials evaluating RNAi or antisense oligonucleotide agents (e.g., ARC-520, ARB-1467, VIR-2218, JNJ-3989, RG6346, ALG-125755) in adults with chronic hepatitis B. Studies were eligible if they reported quantitative HBsAg outcomes, responder proportions, adverse events, ALT flares, or post-treatment follow-up. Searches of PubMed, EMBASE, CENTRAL, ClinicalTrials.gov, WHO ICTRP, EudraCT, Web of Science, and conference abstracts (AASLD, EASL, APASL) were conducted up to February 2025. Risk of bias was assessed using RoB 2 and ROBINS-I. A random-effects model generated pooled mean differences (MD) and proportions with 95% confidence intervals (CI).

Results:

Ten studies met the inclusion criteria. RNAi therapy produced a significant reduction in HBsAg, with a pooled mean change of $-1.47 \log_{10}$ IU/mL (95% CI: -1.58 to -1.36). The pooled proportion of participants achieving $\geq 1 \log_{10}$ HBsAg reduction was 0.79 (95% CI: 0.63–0.90). ALT flares occurred in 0.66 of patients (95%

CI: 0.48–0.81), predominantly among VIR-2218 combination cohorts. Treatment-emergent adverse events were common but mostly mild, with a pooled incidence of 0.57 (95% CI: 0.46–0.67). Serious adverse events were rare (0.022, 95% CI: 0.006–0.078). None of the included studies provided extractable data on durability of HBsAg suppression, limiting long-term assessment. Additional early-phase studies identified in the systematic review demonstrated antigen declines but lacked complete numerical data for meta-analysis. Overall risk of bias ranged from low to some concerns.

Conclusions:

RNAi therapies demonstrate potent short-term antiviral activity in CHB, achieving meaningful reductions in HBsAg and high responder rates across multiple agents. Although ALT flares were frequent, they were generally self-limited and aligned with immune-reactivation profiles. Safety signals were acceptable across all studies. However, the absence of durability data prevents conclusions regarding sustained off-treatment responses. Standardized reporting and longer follow-up in Phase II–III trials are urgently needed to confirm whether RNAi-mediated antigen suppression can progress toward functional cure.

Keyword(s) :

2324 - Association of Serum Amylase and Lipase with Clinical Outcomes of Leptospirosis Patients at a Philippine Tertiary Government Hospital A Retrospective Cohort Study

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Background / Hypothesis:

Leptospirosis is a major public health concern, especially in the Philippines. Nonetheless, local studies on this disease remain limited. Moreover, very few studies examined the prognostic role of pancreatic enzymes.

Materials and Methods:

This retrospective cohort study included all 492 adult patients with confirmed diagnosis of leptospirosis admitted from 2021 to 2024, and with available amylase and lipase values. Firth's bias-reduced logistic regression analysis was performed to determine the association of amylase and lipase with in-hospital mortality.

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Results:

The median age was 34 years, and most were males. All were febrile, and the median duration of symptoms was 5 days. Elevated amylase and lipase were recorded in 60% and 26% of patients. Most patients had severe leptospirosis (71%), underwent renal replacement (59%), and had prolonged length of hospital stay (65%). The incidence of in-hospital mortality was 17.28% (95% CI: 14.04-20.91%). Elevated amylase level was significantly associated with all clinical outcomes except in-hospital mortality and myocarditis (all $p \leq 0.05$). Meanwhile, elevated lipase was only associated with severe leptospirosis, acute pancreatitis, renal replacement, and prolonged length of hospital stay (all $p \leq 0.05$). Multivariable analysis revealed that elevated amylase ($aOR=0.18; p=0.132$) and lipase ($aOR=2.40; p=0.345$) were not associated with in-hospital mortality.

Conclusions:

Increased amylase and lipase levels were observed in most admitted leptospirosis patients and were associated with most clinical outcomes related to disease severity. However, pancreatic enzymes were not associated with mortality.

Keyword(s) :

2325 - Rabdosis Rubescens Inhibits Toll Like Receptor 4 Dependent Helper T Lymphocytes Infiltration in the Liver of Type 2 Diabetes Associated Metabolic Dysfunction Associated Steatohepatitis Mice

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Background/Hypothesis:

The global prevalence of T2DM-associated MASH is rising, constituting a major public health challenge. A central immunopathological hallmark of T2DM-associated MASH is the intrahepatic infiltration and activation of Th17, driving disease progression. *Rabdosis rubescens* has demonstrated potential in alleviating hepatic steatosis and improving liver function in MASH models, yet its precise mechanism of

action remains elusive. This study was therefore designed to test the hypothesis that the hepatoprotective effect of *Rabdosis rubescens* against T2DM-associated MASH is mediated, at least in part, by suppression of TLR4-dependent pathogenic Th17 infiltration into the liver.

Materials and Methods:

A T2DM-associated MASH model was established in two-day-old male C57BL/6J mice via a streptozotocin (STZ)-high-fat diet (HFD) protocol. Briefly, neonatal mice received a subcutaneous injection of STZ. At 4 weeks of age, they were switched to an HFD for 6 weeks. Starting from week 5, mice were randomly allocated into four groups for the 4-week intervention period: (1) Normal control (NC; normal diet+vehicle), (2) model group (MG; HFD+vehicle), (3) *Rabdosis rubescens* group (RG; HFD+*Rabdosis rubescens* decoction via oral gavage), (4) lipopolysaccharide (LPS) group (LG; HFD+vehicle+weekly intraperitoneal injections of LPS), and (5) *Rabdosis rubescens*+LPS group (RLG; HFD+*Rabdosis rubescens*+weekly intraperitoneal injections of LPS). Mice in the other groups received weekly normal saline injections.

Results:

Compared with NG, the MG exhibited characteristic histopathological alterations, including hepatocellular vacuolar degeneration and inflammatory cell infiltration, accompanied by a significantly higher NAFLD Activity Score. Serum levels of ALT, AST, GGT, TG, and hepatic TG, TNF- α , and IL-17 were all markedly elevated in the MG (all $p < 0.005$). As expected, lipopolysaccharide (LPS) challenge in the LG exacerbated these injury markers compared to the MG ($p < 0.005$). *Rabdosis rubescens* effectively attenuated the hepatic injury in the RG, as evidenced by significant improvements in all aforementioned biochemical and inflammatory indices versus the MG ($p < 0.001$). Notably, in mice of the RLG, the herbal treatment significantly counteracted LPS-induced exacerbation of liver injury ($p < 0.005$ vs. LG). Consistent with the functional improvement, immunohistochemical analysis demonstrated a significant reduction in the intrahepatic proportion of Th17 cells in the RLG compared to the LG. This reduction was further supported by a significant decrease in IL-17A levels within liver tissue homogenates ($p < 0.001$).

Conclusions:



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Our findings demonstrate that the hepatoprotective effect of *Rabdosia rubescens* against T2DM-associated MASH is mediated through the suppression of TLR4-dependent intrahepatic Th17 infiltration and IL-17A production at least in part.

*Corresponding Author: Xin Wang.

This study was supported by the Higher Education Innovation Foundation Project of Gansu Provincial Department of Education (2021B-173).

Keyword(s) : *Rabdosia Rubescens*, TLR4, Th17, T2DM, MASH

2327 - Impact of Metabolic Dysfunction–Associated Fatty Liver Disease on Severity and Recovery of Drug Induced Liver Injury Experience from a Pakistani Cohort

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Background / Hypothesis:

Drug-induced liver injury (DILI) is a leading cause of acute liver dysfunction worldwide. South Asia has a high and rapidly rising burden of metabolic dysfunction–associated fatty liver disease (MAFLD), which may influence the severity and clinical course of DILI. However, data evaluating the interaction between MAFLD and DILI outcomes are limited from the region. To evaluate the effect of underlying MAFLD on disease severity, clinical course, and recovery outcomes in patients presenting with DILI in Pakistan.

Materials and Methods:

We conducted a retrospective/prospective observational study of adult patients diagnosed with DILI at a tertiary care center in Pakistan between 2015–2025. DILI was defined using international consensus criteria and causality was assessed by RUCAM/COIMS. MAFLD was diagnosed based on imaging evidence of fatty liver and non-invasive workup, including FIB4 and APRI with metabolic risk factors. Patients were categorized into MAFLD and non-MAFLD groups. Primary outcomes included DILI severity (peak ALT, bilirubin, INR, Hy's law) and time to biochemical recovery. Secondary outcomes included hospitalization duration, development of acute liver failure, and mortality.

Results:

Among 533 patients with confirmed DILI 321 patients had underlying MAFLD. Patients with MAFLD

presented with significantly higher peak ALT (median 1200 vs 780 IU/L; $p < 0.05$) and bilirubin levels (median 23 vs 12 mg/dL; $p < 0.01$) compared to non-MAFLD patients. Severe DILI occurred more frequently in the MAFLD group (88% vs 52%, $p < 0.01$), with a higher proportion meeting Hy's law criteria. Time to normalization of liver enzymes was significantly longer in MAFLD patients (median 15 vs 5 days, $p < 0.01$). On multivariable analysis, MAFLD independently predicted severe DILI (adjusted OR 1.8, 95% CI 1.2–2.7), $p = 0.004$

Conclusions:

Underlying MAFLD was associated with increased severity and delayed recovery in patients with DILI among south asian patients. Given the high burden of metabolic disease in the region, recognition of MAFLD as a risk modifier for DILI is crucial. Early identification and closer monitoring of MAFLD patients exposed to potentially hepatotoxic drugs may improve clinical outcomes.

Keyword(s) : DILI, MAFLD

2330 - Dietary omega 3 and omega 6 Polyunsaturated Fatty Acid Intake and Mortality Among Individuals with Metabolic Dysfunction Associated Steatotic Liver Disease A Prospective Cohort Study

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Background / Hypothesis: Evidence from large cohort studies on the impact of dietary polyunsaturated fatty acids (PUFAs) on mortality among patients with metabolic dysfunction-associated steatotic liver disease (MASLD) is limited. We aimed to investigate the associations of omega-3 and omega-6 PUFA intake with all-cause and cardiovascular disease (CVD) mortality in MASLD, and to identify potential dietary thresholds for mortality reduction.

Materials and Methods: A total of 5,694 participants with MASLD at baseline [weighted 57.3% male; mean (SD) age 50.5 (0.35) years] were included from the National Health and Nutrition Examination Survey (1999–2018). Hepatic steatosis was defined by the United States Fatty Liver Index. Dietary intake was assessed via 24-hour dietary recalls. Mortality was ascertained through linkage to the National Death Index through December 31, 2019. Associations of

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dietary omega-3 PUFA and omega-6 PUFAs with all-cause and CVD mortality were evaluated using Kaplan-Meier curves, weighted Cox proportional hazards models, and restricted cubic splines.

Results: During a median follow-up of 9.2 years (IQR 4.8-13.5), 981 deaths occurred, including 340 due to CVD. Kaplan-Meier analysis showed that higher intakes of total omega-3 and omega-6 PUFAs were associated with lower all-cause and CVD mortality (log-rank $P < 0.05$). After adjustments for demographics, lifestyle factors, hypertension, diabetes, body mass index and total energy intake, higher intake of alpha-linolenic acid (ALA), eicosapentaenoic acid (EPA) and docosapentaenoic acid (DPA) was associated with 20% (HR 0.80, 95% CI 0.67-0.95), 17% (HR 0.83, 95% CI 0.71-0.98), and 19% (HR 0.81, 95% CI 0.68-0.96) lower risk of all-cause mortality, respectively (all $P < 0.05$), whereas associations with CVD mortality were not statistically significant. Total omega-6 PUFA and linoleic acid showed linear associations with all-cause mortality (P for linear < 0.05). ALA demonstrated both linear and modest non-linear associations (both $P < 0.05$), indicating a generally consistent protective effect across intake levels. Notably, EPA and DPA exhibited non-linear relationships, reaching maximal protective effects at intake thresholds of 0.125 g/day and 0.021 g/day, respectively (P for non-linearity < 0.05).

Conclusions: In this prospective cohort of MASLD patients, higher dietary intake of omega-3 PUFAs, particularly ALA, EPA, and DPA, was associated with reduced all-cause mortality. ALA demonstrated a generally linear protective effect, while EPA and DPA showed threshold-dependent maximal benefits, highlighting potential dietary targets for mortality reduction in this high-risk population. These findings provide actionable insights for nutritional strategies and future interventional studies in MASLD.

Keyword(s) : MASLD, Polyunsaturated fatty acid, Mortality, Prospective cohort study, ALA, EPA, DPA

2331 - Prognosis of ursodeoxycholic acid treated patients with primary biliary cholangitis A single center cohort study from China

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Background / Hypothesis: Primary biliary cholangitis (PBC) is a rare disease characterized by intrahepatic

cholestasis. This study aims to characterize the baseline profile, treatment response to ursodeoxycholic acid (UDCA), and long-term outcomes of patients with primary biliary cholangitis (PBC) in a Chinese population.

Materials and Methods: We retrospectively reviewed medical records of PBC patients and conducted regular follow-up assessments. The endpoints were defined as liver-related death and/or liver transplantation (LT). Hepatic events including decompensation and hepatocellular carcinoma were recorded. Deep response was defined as normalization of alkaline phosphatase (ALP) after 1 year treatment of UDCA.
Results: A total of 1,326 PBC patients with complete baseline information and at least one year of follow-up were included in this study. Unsurprisingly, compared with non-cirrhotic PBC patients ($n=1026$), cirrhotic PBC patients ($n=300$) exhibited more severe disease at baseline (Table 1). Overall, the biochemical response rates according to the POISE, Paris I, Paris II, and deep response criteria were 83.2%, 60.3%, 52.8%, and 50.9%, respectively. The biochemical response rates in non-cirrhotic patients at baseline were significantly higher than that in cirrhotic patients, and this finding was independent of the response criteria applied. During a median follow-up of 4.83 years, 139 individuals experienced LT or liver-related death. Out of 1192 PBC patients without decompensation at baseline, 198 experienced hepatic events. Overall, the 5-year, 10-year, and 15-year transplant-free survival rates were 93.1%, 82.4%, and 70.1%, respectively. Responders, identified by either the POISE criteria (Fig. 1A, B) or the deep response criteria (Fig. 1C, D), showed significantly higher transplant-free survival and a lower incidence of hepatic events compared to non-responders.

Conclusions: This large-scale cohort delineates the characteristics and prognosis of the Chinese PBC population. Cirrhotic patients exhibited a significantly lower biochemical response rate compared to non-cirrhotic patients. Patients achieving a favorable response showed markedly better survival outcomes than those with a poor response.

Keyword(s) : primary biliary cholangitis, cirrhosis, baseline characteristics, prognosis, survival

2332 - Hepatocellular carcinoma without cirrhosis in Australasia an under represented but high risk population

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) occurring in the absence of cirrhosis represents a clinically important proportion of liver cancer globally. However, contemporary reviews of non-cirrhotic HCC epidemiology have largely excluded data from Australasia, limiting representation of regions with distinct demographic profiles and risk factor distributions. Australia and New Zealand include substantial Indigenous (Aboriginal and Torres Strait Islander peoples, Māori, and Pasifika) and migrant populations with a high burden of chronic hepatitis B (CHB) and rapidly increasing metabolic dysfunction-associated steatotic liver disease (MASLD). We aimed to summarise contemporary Australasian epidemiology of non-cirrhotic HCC and highlight implications for surveillance.

Methods:

We synthesised published epidemiological data from Australian and New Zealand cohort studies, national surveillance reports, and clinical guidelines describing the prevalence, aetiology, and incidence of non-cirrhotic HCC across key aetiologies.

Results:

Non-cirrhotic HCC accounts for approximately 17–43% of all HCC cases in Australia and New Zealand. In a large Australian multicentre cohort (2008–2021), CHB was the leading aetiology of non-cirrhotic HCC (36%), followed by MASLD (28%), unknown aetiology (23%), hepatitis C virus infection (12%), and alcohol-related liver disease (10%). Among individuals with CHB, annual HCC incidence without cirrhosis was 0.4–0.6% in Asian men aged >40 years and 0.3–0.6% in Asian women aged >50 years. For Aboriginal and Torres Strait Islander peoples aged >50 years with CHB, incidence was estimated at 0.36–0.9%. Over 70% of people living with CHB in Australia were born overseas, with 46% born in Asia and 4.3% born in sub-Saharan Africa, though incidence data for non-cirrhotic HCC in African-born populations are lacking. HBV genotype diversity reflects Australia's migrant profile, with genotypes B and C predominating among Asian migrants and genotype D among European and Aboriginal populations. MASLD prevalence is rising rapidly, with 5.7 million affected Australians in 2020 and MASLD-associated HCC projected to increase by 75% by 2030. Recent data indicate that 31% of MASLD-related HCC occurs in non-cirrhotic livers. Data on non-cirrhotic HCC among Pasifika peoples remain

limited and are largely derived from migrant cohorts.

Conclusions:

Australasian populations contribute important and distinct epidemiological insights into non-cirrhotic HCC risk. Omission of this region from global syntheses perpetuates geographic bias and risks under-informing surveillance strategies. Addressing this deficit is essential for a truly global understanding of HCC and equitable guideline development across the Asia Pacific region.

Keyword(s) : non-cirrhotic HCC, Australasia, Pacific

2333 - Evaluation of Liver Fibrosis Before and After Treatment of Chronic Hepatitis B

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Background / Hypothesis:

Chronic hepatitis B (CHB) is a viral infection affecting nearly 296 million people worldwide and remains a major cause of morbidity and mortality. Without treatment, the disease may progress silently to fibrosis, cirrhosis, and hepatocellular carcinoma. Fibrosis staging is a key determinant of prognosis and is essential for therapeutic decision-making. Current antiviral therapies not only suppress viral replication but also slow, and in some cases reverse, the progression of fibrosis. This study aimed to evaluate the impact of antiviral therapy on liver fibrosis before and after treatment initiation in patients with CHB.

Materials and Methods:

This retrospective, analytical, monocentric study was conducted in the Department of Hepato-Gastroenterology at Ibn Sina University Hospital over a five-year period from August 2020 to August 2025. We included all patients with chronic hepatitis B who were started on antiviral therapy, had regular follow-up, and presented with fibrosis stage $\geq$ F2. Patients without treatment indication, those lost to follow-up, and those with F0–F1 fibrosis or decompensated cirrhosis were excluded. Fibrosis assessment before and during treatment was performed using liver biopsy or FibroScan. Paired comparisons of fibrosis staging before versus after treatment were analyzed using the Wilcoxon test. Statistical analysis was performed using

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SPSS, with significance set at $p < 0.05$.

Results:

Eighty-two patients with chronic hepatitis B were included, with a mean age of 55.9 ± 12.4 years; 42.6% were men and 57.3% were women. Most patients were treated with Tenofovir (79.3%), while 20.7% received Entecavir. Before treatment, fibrosis assessment was performed using FibroScan in 75.6% of cases and liver biopsy in 24.4%. After treatment, FibroScan was used in 95.1% of cases and biopsy in 4.9%. At baseline, fibrosis stages were distributed as follows: F2 in 47.5%, F3 in 21.9%, and F4 in 30.5% of patients. Following treatment, there was an overall improvement, with an increase in F0–F1 stages (24.4%), a decrease in F2 (30.5%) and F3 (14.6%), while F4 remained unchanged at 30.5%. Statistical analysis showed a significant reduction in F2 after treatment ($p = 0.037$), a non-significant decrease in F3 ($p = 0.313$), and no change in F4 ($p = 1.000$). Overall, the Wilcoxon test demonstrated a significant improvement in fibrosis staging after therapy ($p < 0.001$), with the median fibrosis stage decreasing from F2 to F1.

Conclusions:

This study demonstrates a significant improvement in liver fibrosis among chronic hepatitis B patients receiving antiviral therapy. Tenofovir and Entecavir confirmed their therapeutic efficacy, particularly in the regression of intermediate

Keyword(s) : fibrosis, hepatitis B, evaluation

2334 - US guided liver biopsy Fine needle aspiration versus semi automatic cutting needle

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Background / Hypothesis:

Despite advances in Endoscopic UltraSound and transjugular approaches, percutaneous liver biopsy remains essential and continues to be the gold standard for the diagnosis and prognostic assessment of liver diseases. Several sampling devices are available, particularly the aspiration needle and the cutting needle, which allows mechanical coring of hepatic tissue. This study aims to compare the diagnostic yield and tolerability of these two techniques.

Materials and Methods:

We conducted a prospective descriptive study including all percutaneous liver biopsies performed under ultrasound guidance between January 2025 and November 2025 in the Department of Gastroenterology and Proctology, Medicine B. Biopsy quality was defined by the presence of ≥ 7 portal tracts and a specimen length of ≥ 2 cm. Two groups were compared using independent samples t-test: Group A, using an aspiration needle (Menghini type), and Group B, using a semi-automatic cutting needle, both 18G. A p-value < 0.05 was considered statistically significant.

Results:

A total of 38 patients were included, 23 (71.9%) were women and 9 (28.1%) men, with a mean age of 49.5 ± 14.35 years. The aspiration needle was used in 14 patients (43.8%), and the cutting needle was used in 18 patients (56.8%).

The indications were as follows: unexplained liver test abnormalities in 25%, fibrosis assessment in 8.3%, suspicion of autoimmune hepatitis (AIH) in 8.3%, and assessment of AIH activity in 4.2%.

The mean biopsy length was 2.55 ± 1.62 cm in Group A and 2.37 ± 1.31 cm in Group B.

The mean number of portal tracts was 9 ± 3.95 in Group A and 7.07 ± 5 in Group B.

Comparisons between these means showed no statistically significant difference ($p = 0.7$, $p = 0.3$ respectively).

No difference in complication rates was observed between the two groups.

Conclusions:

In our experience, the aspiration needle tended to yield specimens richer in portal tracts than the cutting needle, although the difference was not statistically significant.

Percutaneous liver biopsy proved to be a generally well-tolerated procedure, with a low and transient rate of complications.

Keyword(s) : Biopsy, US, comparison, needles, quality

2337 - Diagnostic challenges at the interface of drug induced autoimmune hepatitis and classical autoimmune hepatitis a case report



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Background:

Distinguishing drug-induced autoimmune hepatitis (DI-AIH) from classical autoimmune hepatitis (AIH) remains challenging, even for experienced clinicians, as no single serological or histological feature reliably differentiates the two at presentation. This distinction is clinically important, as misclassification may result in unnecessary long-term immunosuppression or, conversely, delayed treatment. We report a diagnostically challenging case initially managed as drug-induced liver injury (DILI) and DI-AIH, in which longitudinal follow-up ultimately supported a diagnosis of classical (idiopathic) AIH.

Case:

A 46-year-old woman with no significant past medical history presented with a one-week history of abdominal bloating and jaundice. She had travelled to Vietnam two months earlier but remained afebrile with no gastrointestinal symptoms. Admission laboratory tests revealed bilirubin 21 $\mu\text{mol/L}$, AST 693 U/L, ALT 675 U/L, ALP 164 U/L and GGT 564 U/L. Viral hepatitis serologies and autoimmune markers, including IgG, ANA, anti-smooth muscle antibody (SMA), anti-mitochondrial M2, anti-LKM-1, anti-LC-1, and anti-SLA/LP antibodies, were negative. Magnetic resonance cholangiopancreatography (MRCP) demonstrated gallstones without biliary dilatation and a normal liver parenchyma.

Further history revealed recent use of cosmetic whitening supplements, some containing turmeric, which were discontinued on admission. Given fulfilment of Hy's law and concern for severe DILI, an urgent liver biopsy was performed. Histology showed mild to moderate portal inflammation with lymphocytes and plasma cells, lobular inflammation with hepatocyte ballooning, and absence of interface hepatitis, consistent with acute hepatitis with hepatocellular injury.

Unfortunately, despite withdrawal of the suspected agents, liver biochemistry worsened (bilirubin 83 $\mu\text{mol/L}$, AST 946 U/L, ALT 1322 U/L, ALP 236 U/L, GGT 339 U/L), prompting initiation of corticosteroid therapy (prednisolone 30 mg daily) for presumed DI-AIH. This led to marked biochemical improvement with near normalization of liver enzymes. Steroids were

subsequently tapered due to side effects, in keeping with the working diagnosis of DI-AIH. However, upon reduction to prednisolone 10 mg daily, the patient developed an abrupt biochemical relapse. Re-induction with corticosteroids and the addition of azathioprine 50 mg daily resulted in complete and sustained normalization of liver function tests.

Conclusion:

The steroid-dependent, relapsing course strongly supports a diagnosis of idiopathic AIH rather than DI-AIH, indicating the need for long-term immunosuppression. This case highlights the persistent diagnostic ambiguity at the intersection of DILI, DI-AIH, and AIH, where initial serology and histology may be non-specific. In such cases, relapse following steroid withdrawal is a pragmatic discriminator favouring classical AIH and should prompt reclassification and initiation of maintenance therapy.

Keyword(s) : AIH, DI-AIH

2338 - Association between Glycemic Control (HbA1c) and Liver Transaminase Levels (ALT and AST) in Mongolian patients

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Background / Hypothesis:

This cross-sectional study aimed to assess the prevalence of impaired glycemic control (measured by HbA1c) and elevated liver transaminases (ALT and AST) in a cohort of N = 71 individuals. Secondary aims included determining the correlation between long-term glycemic control and transaminase elevation and identifying potential gender differences.

Materials and Methods:

Data for 71 individuals (46 males, 25 females; mean age 49.5 years) were collected from January to August 2024 and analyzed using descriptive and correlational statistics. Since this was a single-point-in-time assessment, the analysis relied on Pearson correlation coefficients (r) to assess the strength of associations between metabolic and hepatic markers

Results:



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The cohort exhibited evidence of significant metabolic and hepatic impairment, with a high mean HbA1c of 7.22%. Both mean ALT (80.62 U/L) and mean AST (50.54 U/L) were substantially elevated above typical reference ranges. A strong positive correlation was found between ALT and AST ($r = 0.762$). Crucially, the correlation between HbA1c and ALT ($r = 0.026$) or AST ($r = 0.142$) was statistically very weak. Gender analysis revealed that males demonstrated higher mean values across all three markers, including HbA1c (7.38% vs 6.93%) and ALT (87.21 U/L vs 68.50 U/L).

Conclusions:

The findings indicate a high co-prevalence of sub-optimal glycemic control and hepatic enzyme elevation in this study population. The lack of a strong association between HbA1c and transaminases suggests that factors independent of hyperglycemia, such as Non-Alcoholic Fatty Liver Disease (NAFLD), may be the primary driver of liver injury. These gender disparities underscore the need for targeted screening and gender-specific management strategies in clinical practice in Mongolia.

Keyword(s) : DM, ALT, AST

2339 - The metagenomics derived gut mycobiome biomarker for non invasive diagnosis of early hepatocellular carcinoma

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Background / Hypothesis:

The early diagnosis of hepatocellular carcinoma (HCC) remains clinically challenging, and the role of gut fungi in hepatocarcinogenesis is poorly understood.

Materials and Methods:

This prospective multi-center study performed metagenomic sequencing on 514 fecal samples. In the training set (HCC vs. healthy controls [HCs]), 117 classification models were developed based on fungal features using nine machine learning algorithms, subsequently validated in cross-regional and cross-disease cohorts. We further assessed clinical applicability using a large public dataset ($n=3487$) comprising multiple diseases and HCs.

Results:

Early HCC patients exhibited reduced gut fungal diversity and altered mycobiota structure with 46 differentially abundant fungal species identified.

Schizosaccharomyces-dominant enterotype was strongly associated with HCC risk (36-fold increase compared to Aspergillus-dominant enterotype). Functionally, HCC showed enriched KEGG pathways such as lipopolysaccharide biosynthesis and shifted carbohydrate-active enzyme profiles. Clinically, a history of chronic hepatitis B (CHB) was the strongest driver of community variation at both taxonomic and functional levels ($R^2 = 0.093$ and 0.060 , respectively). The optimal model, constructed with glm+Ridge algorithm using 11 fungal species, demonstrated strong discriminatory performance across all test sets (average area under curve [AUC] = 0.87) and maintained low false-positive rates in public cohorts. SHAP analysis identified *Pyricularia_ grisea* and *Schizosaccharomyces_pombe* as the top predictive features.

Conclusions:

This study characterizes gut mycobiota dysbiosis in early HCC and provides novel insights into the fungal gut-liver axis. The developed non-invasive fungal model exhibits robust cross-regional validity and high disease specificity, highlighting its translational potential for early and differential diagnosis of HCC.

Keyword(s) : Hepatocellular carcinoma, Metagenomics, Gut Mycobiome, Biomarkers, Non-invasive Diagnosis, Correlation Analysis

2341 - Correlation Between tumor markers (AFP CA19 9 CEA)

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Background / Hypothesis:

Serum biomarkers such as AFP (alpha-fetoprotein), CA19-9 (cancer antigen 19-9), and CEA (carcinoembryonic antigen) might serve as non-invasive, objective, and repeatable tools for early detection, diagnosis, and monitoring of HCC.

Materials and Methods:

A retrospective cross-sectional study was conducted including $n=150$ outpatients who visited Happy Veritas Liver Center and underwent laboratory tests between March and August 2025. Participants were selected

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using random sampling. Inclusion criteria were age ≥ 18 years, complete values for AFP, CEA, and CA19-9, and a diagnosis of primary hepatocellular carcinoma (PHC) or liver cirrhosis. Exclusion criteria included other serious comorbid diseases, patients receiving chemotherapy/radiation therapy, pregnant or breastfeeding women, and incomplete or inconsistent test data. Clinical data collected included demographic information and patients' laboratory test results. All statistical analyses were performed using SPSS 26 software, with statistical significance set at 95% ($p < .05$). Normality of data distribution was assessed using the Shapiro–Wilk test; since the distribution was non-normal, Spearman's rank correlation was used for correlation analysis. The study was approved by the Ethics Committee of Happy Veritas Clinic (Protocol No. HV-2024-02). Personal information such as names, codes, and registration numbers of participants was fully anonymized and used solely for statistical purposes.

Results:

- The correlation between AFP and CA19-9 was very weak positive ($r = 0.03$), at a level with no statistical or clinical significance.
- Between AFP and CEA, an almost negligible weak negative correlation was observed ($r = -0.02$), indicating no linear relationship between these variables.
- Between CA19-9 and CEA, a weak positive correlation ($r = 0.087$) was observed, but the magnitude was small, with no established clinical significance.

Based on the values of Pearson's coefficients, only nominal, clinically insignificant weak associations were observed between all three markers. Within the scope of the study data, these tumor biochemical markers appear to change independently of one another.

Conclusions: The statistical correlation between tumor markers AFP, CA19-9, and CEA is very low and clinically insignificant.

Keyword(s) : Tumor markers

2342 - Performance of Non invasive Baveno Seven Criteria for Predicting Clinically Significant Portal Hypertension in Burned out Metabolic Dysfunction associated Fatty Liver Disease

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Background / Hypothesis:

Burned-out metabolic dysfunction-associated fatty liver disease (absent steatosis despite significant fibrosis) may represent a high-risk phenotype for portal hypertension, yet evidence on non-invasive stratification is limited. We hypothesized that burned-out metabolic dysfunction-associated fatty liver disease has a high prevalence of non-invasive clinically significant portal hypertension and that Baveno Seven would place most patients in rule-in or grey-zone categories.

Materials and Methods:

A retrospective single-center pilot study. Baseline liver stiffness measurement, controlled attenuation parameter, platelet count, and Child–Pugh class were collected. Burned-out metabolic dysfunction-associated fatty liver disease was defined as controlled attenuation parameter < 248 dB/m (S0) with liver stiffness measurement ≥ 8 kPa. Clinically significant portal hypertension was defined by a non-invasive surrogate of liver stiffness measurement ≥ 25 kPa. Baveno Seven categories were rule-out (liver stiffness measurement < 15 kPa and platelets $> 150 \times 10^9/L$), grey zone, and rule-in (liver stiffness measurement ≥ 25 kPa). Discrimination was assessed using area under the receiver operating characteristic curve.

Results:

Among 44 patients (median age 59.5 years; 61% male), 16 (36.4%) met criteria for burned-out metabolic dysfunction-associated fatty liver disease. Non-invasive clinically significant portal hypertension was present in 50% overall and was higher in burned-out vs non-burned-out metabolic dysfunction-associated fatty liver disease (62.5% vs 42.9%). In burned-out metabolic dysfunction-associated fatty liver disease, Baveno Seven categorized 62.5% as rule-in, 31.2% as grey zone, and only 6.2% as rule-out. Clinically significant portal hypertension occurred in 55.6% of Child–Pugh A, 80.0% of Child–Pugh B, and 50.0% of Child–Pugh C burned-out patients. Platelet count and Child–Pugh class showed poor discrimination (area under the curve ≈ 0.50 and 0.59).

Conclusions:

Burned-out metabolic dysfunction-associated fatty liver disease is enriched for non-invasive clinically significant portal hypertension, with most patients classified as Baveno Seven rule-in and very few

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meeting rule-out thresholds. This suggests limited endoscopy-sparing capacity of current non-invasive criteria in the burned-out phenotype and supports the need for phenotype-specific risk stratification validated in prospective cohorts.

Keyword(s) : Burned-out metabolic dysfunction-associated fatty liver disease; clinically significant portal hypertension; Baveno Seven; non-invasive assessment; liver stiffness measurement

2343 - Gut Microbiome Dysbiosis in Metabolic Associated Fatty Liver Disease A Systematic Review and Meta Analysis Pakistan

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Background / Hypothesis:

Metabolic associated fatty liver disease (MAFLD) is a leading cause of chronic liver disease worldwide. Recent studies suggest that gut microbiome dysbiosis contributes to the pathogenesis of MAFLD by modulating hepatic inflammation, lipid metabolism, and insulin resistance. However, the specific microbial signatures associated with MAFLD remain heterogeneous across studies.

Aim:

To systematically review and meta-analyze existing evidence on gut microbiome alterations in patients with MAFLD compared to healthy controls, focusing on microbial diversity, composition, and functional implications.

Materials and Methods:

A comprehensive literature search was conducted in PubMed, Embase, and Cochrane Library up to December 2025. Studies reporting gut microbiome profiles in adult patients with MAFLD were included. Data on microbial diversity (alpha and beta diversity), relative abundance of bacterial taxa, and functional pathways were extracted. Meta-analysis was performed using random-effects models for consistent microbial changes. Risk of bias was assessed using the Newcastle-Ottawa Scale.

Results:

A total of 42 studies including 3,560 MAFLD patients and 2,940 healthy controls were analyzed. Patients with MAFLD consistently exhibited reduced gut microbial diversity (pooled standardized mean difference = -0.42, 95% CI -0.55 to -0.29, $p < 0.001$). At the phylum level, an increased Firmicutes/Bacteroidetes ratio and enrichment of Proteobacteria were observed. Genera such as *Escherichia*, *Lactobacillus*, and *Prevotella* were

significantly elevated, while *Faecalibacterium* and *Roseburia* were depleted. Functional analyses suggested enhanced lipopolysaccharide biosynthesis, bile acid metabolism alterations, and short-chain fatty acid dysregulation in MAFLD. Heterogeneity was moderate, primarily driven by geographic and sequencing methodology differences.

Conclusions:

Gut microbiome dysbiosis is a consistent feature of MAFLD, characterized by reduced microbial diversity, pro-inflammatory microbial enrichment, and altered metabolic pathways. These findings underscore the role of the gut-liver axis in MAFLD pathogenesis and highlight potential microbiota-targeted therapeutic strategies. Future longitudinal and interventional studies are warranted to establish causality and clinical utility.

Keyword(s) :

2344 - Is Pneumococcal Vaccination Safe in Patients with Liver Cirrhosis

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Background / Hypothesis:

Patients with liver cirrhosis may become susceptible to infectious diseases due to immune system dysfunction. Therefore, vaccination against vaccine-preventable diseases is essential in this population. Pneumococcal infections can cause significant morbidity and mortality in patients with cirrhosis.

The aim of this study was to retrospectively evaluate the pneumococcal vaccination status of patients with liver cirrhosis followed in the hepatology and adult vaccination clinics of our university.

Materials and Methods:

Patients aged 18 years and older with a diagnosis of liver cirrhosis who were followed at the Adult Vaccination Clinic of the Department of Infectious Diseases, Ege University Faculty of Medicine Hospital, between October 2015 and December 2025 were included in the study. Patient data were obtained from the Hospital Information Management System and the Ministry of Health Vaccination Tracking System.

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Collected data were analyzed using the SPSS statistical software.

Results:

A total of 533 patients with liver cirrhosis who were referred to the adult vaccination clinic received pneumococcal vaccination. No serious adverse events were observed during follow-up.

Conclusions:

The risk of invasive pneumococcal disease is 2 to 13 times higher in patients with chronic liver disease compared to the general population. Vaccination is the most effective and reliable strategy to prevent pneumococcal infections. Our findings indicate that pneumococcal vaccination is safe in patients with liver cirrhosis in terms of adverse effects.

Keyword(s) : Adult immunization, Liver cirrhosis, Pneumococcal vaccination

2345 - Gut Microbiota Alterations in Patients with Chronic Hepatitis B and Metabolic Associated Fatty Liver Disease Implications for Fibrosis Progression

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Background / Hypothesis:

Background:

Metabolic associated fatty liver disease (MAFLD) is increasingly recognized in patients with chronic hepatitis B (CHB) and may influence disease progression. Emerging evidence suggests that gut microbiota modulates hepatic inflammation, fibrosis, and antiviral immunity. The interplay between MAFLD, HBV, and gut microbiota remains largely unexplored.

Aim:

To characterize the gut microbiota in patients with CHB and MAFLD and evaluate its association with liver fibrosis and HBV activity.

Methods:

A cross-sectional study was conducted including 120 CHB patients, 60 with MAFLD and 60 without. Stool samples were analyzed using 16S rRNA sequencing to assess microbial diversity and composition. Clinical data including HBV DNA, ALT, HBeAg status, and fibrosis (via transient elastography) were collected. Correlations between microbial taxa, metabolic parameters, fibrosis, and viral activity were evaluated.

Results:

Patients with CHB and MAFLD exhibited reduced gut microbial diversity compared to CHB-only patients (Shannon index: 3.2 ± 0.5 vs. 3.8 ± 0.4 , $p < 0.01$). Specific bacterial genera, including Firmicutes and Bacteroides, were significantly enriched in MAFLD patients. Dysbiosis was associated with higher fibrosis scores independent of HBV viral load (OR 2.5, 95% CI 1.3–4.8, $p = 0.005$). Certain microbial metabolites correlated with ALT elevation and insulin resistance, suggesting a link between gut-liver axis alterations and liver injury.

Conclusion:

CHB patients with MAFLD show distinct gut microbiota alterations that are associated with fibrosis progression and metabolic dysfunction. These findings highlight the potential role of the gut-liver axis in modulating liver injury in HBV-infected individuals and may inform microbiota-targeted interventions.

Keyword(s) :

2346 - Correlation Between Liver Biomarkers and Hemoglobin Levels in Mongolian Patients

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Background / Hypothesis:

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are key biochemical markers widely used to evaluate hepatocellular injury. Elevations of these enzymes reflect hepatocyte membrane damage and intracellular enzyme leakage, often associated with viral hepatitis, alcohol-related liver injury, metabolic syndrome, and non-alcoholic fatty liver disease. Hemoglobin (Hb), the primary determinant of oxygen-carrying capacity, can influence tissue oxygenation, redox balance, and mitochondrial function. Alterations in Hb levels may contribute to hepatocellular injury through impaired oxygen delivery and oxidative stress mechanisms, as reported in several studies. However, no published studies to date have assessed the relationship between hemoglobin levels and liver enzymes among the Mongolian population.

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Therefore, investigation using real-world clinical data is warranted.

Materials and Methods:

A retrospective cross-sectional study was conducted among $n = 137$ patients who underwent laboratory testing at the Happy Veritas Liver Center between January and August 2025. Participants were selected using simple random sampling. Inclusion criteria included age ≥ 18 years and availability of complete laboratory results for hemoglobin, ALT, and AST collected from the same blood sample on the same day. Normality of the data was assessed using the Shapiro–Wilk test, and because the variables were non-normally distributed, Spearman's rank correlation was applied. The anticipated effect size was $r = 0.25$. Using 80% statistical power and 5% alpha, the required minimum sample size was 123; thus, the collected sample of 137 participants was adequate. The study protocol was approved by the Institutional Review Board of Happy Veritas Hospital (Protocol No. HV-2024-02). All personal identifiers were removed to ensure confidentiality.

Results:

A total of 137 participants were included. The mean age was 41.6 ± 12.8 years (range: 18–72). Of these, 79.6% were male and 20.4% were female. Mean laboratory values were as follows: ALT 53.5 ± 66.0 U/L, AST 38.0 ± 36.2 U/L, and hemoglobin 14.7 ± 1.5 g/dL. Spearman's correlation analysis demonstrated a strong positive correlation between ALT and AST ($r = 0.933$, $p < 0.001$). Hemoglobin showed a moderate positive correlation with both ALT ($r = 0.418$, $p < 0.001$) and AST ($r = 0.434$, $p < 0.001$). Simple linear regression indicated that ALT ($\beta = 0.004$, $p < 0.001$).

Conclusion: Higher hemoglobin levels were moderately associated with increased liver enzyme activity. These findings suggest that elevated hemoglobin may be linked to hepatocellular stress or injury; however, further prospective and mechanistic studies are needed to clarify the causal pathways.

Keyword(s) : Liver enzymes, hemoglobin, correlation, ALT, AST

2348 - Observational study comparing Hepatocellular carcinoma patients on HCC surveillance with those not on surveillance at diagnosis.

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Background / Hypothesis:

Surveillance for Hepatocellular carcinoma (HCC) in chronic liver disease to improve disease outcomes.

Materials and Methods:

This observational study included consecutive patients of Hepatocellular carcinoma and were categorized by surveillance status. Group A included patients not on HCC surveillance ($n=71$) and Group B on regular surveillance ($n=60$). Baseline comorbidities, etiologies (NASH, ethanol, viral hepatitis), liver dysfunction severity (MELD, CTP), tumor characteristics (BCLC stage, lesion size/number, AFP, Portal vein thrombosis), radiological response and outcomes were evaluated.

Results:

Comorbidities, etiologies, gender, MELD/CTP, lesion number, AFP and presence of portal vein thrombosis (PVT) in both groups showed no significant differences (all $p > 0.05$). Patients on surveillance showed shift towards early BCLC stages (0/A/B, $p=0.019$) and smaller tumor size (<3 cm: 28 vs 17, $p=0.032$). Higher proportion of patients on surveillance underwent locoregional therapy (48.3% in group B vs 29.5% in group A.) Radiological complete response was higher in group B (48.2% vs 33.3%). Mean time to death was 9.9 months (group A) vs 12.11 months (group B).

Conclusions:

Surveillance enables earlier HCC detection (lower BCLC stage, smaller tumors), enabling access to curative options, which can reduce future mortality and morbidity. This study underscores need of regular screening for HCC in liver disease.

Keyword(s) : Hepatocellular carcinoma, HCC, cirrhosis, AFP, BCLC, PVT, locoregional therapy

2352 - The Economic Burden of Cirrhosis Financial Classification and Disease Severity as Key Predictors of Direct Hospital Costs in the Philippines a Retrospective Analysis

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Background: Liver cirrhosis represents end-stage liver



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disease and is one of the leading causes of morbidity and mortality globally, with significant associated healthcare costs in the developed world. Despite this, cost-of-illness data remain severely limited in developing nations including the Philippines, hindering efforts to formulate cost-containment and policy strategies. This lack of data is especially crucial in the Philippine context where a regular employee is only expected to earn approximately PHP 30,000.00 per month, where a significant hospitalization cost can represent a devastating financial burden. This study aims to quantify direct hospitalization costs and to identify key predictors of higher expenditure among cirrhotic patients.

Materials and Methods: A retrospective, cross-sectional analytical study was conducted. Demographics, clinical profiles, total hospitalization costs and reimbursement data were collected based on available data from January 2023 to December 2024. Data analysis was performed using Stata 17. Normality of continuous variables was evaluated using the Shapiro–Wilk test. Multivariate analysis via Generalized Linear Model (GLM), Gamma distribution, identity-link were used to identify statistical differences and predictors of increased costs. A p-value <0.05 was considered statistically significant.

Results: 282 patients were included in the analysis. The median age was 62 years and 41.13% (n = 116) were female. The predominant etiology of cirrhosis was Hepatitis B (48.23%) with most patients classified as Child-Pugh Class B (n = 143, 50.71%). The average hospitalization cost was PHP 231,242.30 per admission (median PHP 156,077.77). Majority of costs were driven by diagnostic and laboratory fees (33.20%) and professional fees (23.61%). PhilHealth reimbursements accounted for only a minimal fraction of the total costs (median PHP 12,771.76) covering <0.01% of the average total, with patients shouldering 59.59% of expenses out-of-pocket (median PHP 151,850.30). In multivariate analysis, only Child-Pugh Class of B (P = 0.045) and a service financial classification (P = 0.001) remained significant predictors of increased direct healthcare costs.

Conclusions: Hospitalization for liver cirrhosis imposes a substantial and inequitable financial burden, driven predominantly by diagnostic and laboratory costs, with negligible PhilHealth coverage. Financial classification and moderate disease severity (Child-Pugh Class B) were the only significant predictors of higher costs. These findings urgently highlight the need for

expanded insurance coverage and targeted cost-containment strategies focused on diagnostic services to alleviate patient economic distress.

Keyword(s) : Liver cirrhosis, Child-Pugh score, cost of illness, hospital costs, Generalized Linear Model (GLM)

2354 - COMPARATIVE STUDY OF THE ETIOLOGY OF LIVER CANCER AND THE MANIFESTATION OF HEPATIC ENCEPHALOPATHY

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Background / Hypothesis:

Primary liver cancer typically develops on the background of chronic liver disease, and hepatic encephalopathy—one of the severe manifestations of liver dysfunction—significantly impacts patients' quality of life and mortality. The objective of this study was to classify liver cancer by etiology, compare the frequency of hepatic encephalopathy manifestation, and determine in which etiological group hepatic encephalopathy is more predominant. The study included 216 cases of liver cancer, divided into HBV-related, HCV-related, and non-viral etiological groups for analysis. Results showed that hepatic encephalopathy occurred most frequently in HCV-related liver cancer and least frequently in non-viral liver cancer. This indicates that the etiological classification of liver cancer is an important factor in determining the risk of hepatic encephalopathy.

Materials and Methods:

The study was conducted in a retrospective, observational manner. Clinical records of a total of 216 patients with primary liver cancer were included. Patients were classified into the following three groups based on the etiology of liver cancer:

- HBV-related liver cancer (n = 79)
- HCV-related liver cancer (n = 106)

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· Non-viral liver cancer (n = 31)

The diagnosis of hepatic encephalopathy was confirmed based on clinical signs and symptoms, laboratory findings, imaging diagnostics, and physician notes. Study data were compiled quantitatively, and the frequency of hepatic encephalopathy manifestation was compared and analyzed between groups.

Results:

Among the total 216 liver cancer patients included in the study, the manifestation of hepatic encephalopathy varied depending on etiological classification. The highest number of cases was recorded in the HCV-related liver cancer group, with hepatic encephalopathy observed in 106 patients. This was followed by the HBV-related liver cancer group with 79 cases, while the non-viral liver cancer group had the lowest number of hepatic encephalopathy cases, at 31. These results suggest that the prolonged inflammation, liver fibrosis, and cirrhosis caused by chronic HCV infection may reach deeper levels. In contrast, in the non-viral liver cancer group, relatively preserved liver reserve function in many cases may account for the lower frequency of hepatic encephalopathy.

Conclusions:

The results of this study show that the manifestation of hepatic encephalopathy among liver cancer patients is partially dependent on the etiological classification of the cancer. Hepatic encephalopathy occurred with the highest frequency in HCV-related liver cancer, at a moderate level in HBV-related cancer, and at the lowest level in non-viral cancer. Therefore, when developing clinical monitoring and treatment plans for liver cancer, it is essential to consider not only the cancer stage but also the etiology

Keyword(s) : Encephalopathy, HCC, HCC etiology

2355 - A Machine Learning Based Screening Tool for Population Level Screening of MASLD to identify outlier patients current tools miss

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Background / Hypothesis: Metabolic associated steatotic liver disease (MASLD) is the most common cause of liver disease, 25% of the population have

MASLD, of concern is 1 in 5 (20%) of these patients have silent steatohepatitis, of whom 20% develop cirrhosis. Body Mass Index (BMI) has a 0.75 AUROC in predicting MASLD, with limitations related to high muscle/high BMI and low muscle/low BMI patients. We hypothesised that a machine learning (ML) based screening tool requiring anthropometric measurements and age can achieve comparable predictive performance, enabling a low-cost, easily accessible and truly non-invasive screening tool for population-level screening.

Materials and Methods: An observational study of 1912 healthy volunteers aged 18-90 years was conducted NCT04873258. The data collected consists of demographics, anthropometrics, bioimpedance vector analysis (BIVA) and laboratory bloods. FibroScan was performed as a pragmatic gold standard for diagnosing MASLD with thresholds of ≥ 290 dB/m or ≥ 7.6 kPa defining fatty liver or early but significant fibrosis. We developed the models to predict MASLD cases using LASSO logistic regression (LR), elastic net LR, Random Forest (RF) and XGBoost. The initial models were trained using all available features, followed by model re-training using anthropometric variables and age alone. The predictive performance was evaluated and SHAP analysis was carried out to determine feature importance.

Results: All models were able to achieve > 0.87 AUROC. Utilising all variables, the RF model performed best, achieving an AUROC of 0.892 [95% CI 0.859-0.917] and sensitivity of 0.862 [95% CI 0.790-0.906]. The top 10 features in this model were waist-to-height ratio, waist circumference, BMI, weight, age, GGT, ALT, body fat percentage, visceral fat type II and AST. When feature set was simplified to anthropometric variables and age, it surpassed the predictive value of BMI alone for MASLD (AUROC 0.75) with a AUROC of 0.864 [95% CI 0.828-0.892]; sensitivity = 0.899 [95% CI 0.826-0.935]).

Conclusions: We present a non-invasive and low-cost ML screening tool to identify patients with MASLD with a performance comparable to established non-invasive MASLD screening methods. This low-cost model offers a significant potential for large-scale population screening in the community without the need for blood tests and may better identify outlier patients which are missed by current tools. Once identified, patients can undergo screening to determine their risk

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of, or the presence of, liver fibrosis and underlying steatohepatitis – the target for most new drug treatments.

Keyword(s) : MASLD, Population Screening, Machine Learning, Early Detection

2356 - Mechanism research of α Amyrin targeting Sirt1 PGC 1 α PPAR α axis to regulate fatty acid metabolism reprogramming against liver fibrosis

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Background / Hypothesis:

Liver fibrosis remains one of the serious public health problems worldwide at present. Unfortunately, there is no first-line small molecule drug recommended by clinical guidelines for liver fibrosis currently. Therefore, the development of innovative targeted small molecule drugs is urgent. Previous studies have shown that the natural small molecule α -Amyrin has significant anti-inflammatory effects. However, whether it has anti-cirrhotic effects is unclear. In this study, the anti-fibrosis effect of α -Amyrin was evaluated for the first time at the basic research level, and both its mechanism and active compounds were researched.

Materials and Methods:

In this study, we use an in vitro model of fibrosis in human hepatic stellate LX-2 cells induced with TGF- β 1 and an in vivo mouse model with CCl₄ to evaluate the anti-fibrosis potential of α -Amyrin using multiple approaches. Employing multi-omics and molecular biology techniques to investigate the mechanism of α -Amyrin counteracting hepatic fibrosis. The interaction targets of α -Amyrin were examined through molecular docking, molecular dynamics simulations and surface

plasmon resonance. The anti-hepatic fibrosis mechanism of α -Amyrin was further validated using both in vitro and in vivo PPAR α knockout or inhibition models.

Results:

In vivo, α -Amyrin significantly inhibited the formation of hepatic Pseudolobules and suppressed hepatic inflammation compared with the model control group. In vitro, α -Amyrin significantly down-regulated the expression levels of well-established markers of liver fibrosis. The results of transcriptomics and metabolomics provided clues to the mechanisms of the PPAR α pathway and fatty acid metabolism. Further experimental validation of these mechanisms was conducted. In vitro and in vivo, α -Amyrin significantly promoted fatty acid oxidation levels which is consistent with up-regulation of PPAR α pathway involved in this process, including Sirt1, PPAR α , PGC-1 α , CPT1A, ACOX1, and its downstream targets. Moreover, α -Amyrin markedly enhanced PPAR α nuclear translocation and facilitated the Sirt1-PGC-1 α interaction. Molecular docking, dynamics simulations, and surface plasmon resonance confirmed direct binding of α -Amyrin to PPAR α . Critically, the anti-hepatic fibrosis effect of α -Amyrin was significantly compromised in both the in vivo PPAR α inhibitor model and the in vitro PPAR α knockout model.

Conclusions:

The possible mechanism of α -Amyrin's anti-fibrosis effect is to target the PPAR α axis to promote the reprogramming of fatty acid oxidation metabolism, which in turn exerts an anti-inflammatory effect and reverses the inflammation-fibrosis pathological process. The above results are expected to provide an innovative strategy for the development of small molecule drugs targeting liver fibrosis.

Keyword(s) :

2357 - Pediatric Seronegative Autoimmune Hepatitis has a comparable clinical and laboratory profile to Seropositive Disease

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Background / Hypothesis: Seronegative autoimmune

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hepatitis (SN-AIH) represent a distinct subset of autoimmune liver disease in which children present with typical clinical and histological features of AIH but lack conventional autoantibodies. Limited pediatric data exist regarding their histological spectrum and long-term outcomes.

Materials and Methods: Retrospective analysis of prospectively collected data with a diagnosis of AIH presenting between January 2011 to July 2025 was done. Diagnosis of SN-AIH was based on characteristic clinical features, raised aminotransferases, compatible histology, elevated IgG, and exclusion of other causes of liver disease. For children enrolled before 2017, seronegative status was defined by the absence of traditional antibodies (ANA, SMA, LKM), while those enrolled during or after 2017, this was defined additionally by the absence of extended antibody testing (SLA/LP, LC1 and p-ANCA) at presentation or at follow-up. SN-AIH was compared with seropositive AIH (SP-AIH) for clinical, laboratory and histological profile, and outcome in terms of difficult-to-treat AIH and survival with native liver.

Results: Out of 232 patients, 66 (28.4%) were labelled as seronegative AIH. SN-AIH was comparable to SP-AIH in terms of gender, age at diagnosis and presentation. All the laboratory parameters were comparable except for higher bilirubin in the SN-AIH cohort (mean bilirubin: 11.5 ± 9.8 versus 8.7 ± 8.5 mg/dL, $p=0.030$). Mean simplified score in the SN-AIH cohort was lower than the SP-AIH (mean 5.5 ± 0.7 versus 7.4 ± 1.0 , $p<0.001$). Histologically, both the SN-AIH and SP-AIH were comparable with the most commonly demonstrated findings being lymphoplasmacytic infiltrates, interface hepatitis, lobular inflammation and pseudo-rosetting. Cholestasis, ductular reaction and duct damage are also comparable. Mean fibrosis score was higher in the SP-AIH cohort (3.0 ± 2.0 versus 2.2 ± 2.0 , $p=0.005$), however the mean liver stiffness measurements were comparable between the 2 cohorts (Table 1). At the end of 12 months, 30 patients lost to follow-up. Of the 202 children, 9 required liver transplantation and 28 died. 30 patients had difficult to treat AIH – 5 belonged to SN-AIH group. Both the survival with native liver and difficult-to-treat AIH were comparable between 2 groups ($p=NS$).

Conclusions: Seronegative AIH in children, though lacking conventional autoantibodies, demonstrates classical histological features and favorable response to standard immunosuppressive therapy. Histology plays

a crucial role in diagnosis, and early recognition is essential to prevent disease progression. Long-term outcomes are similar to seropositive AIH.

Keyword(s) : Seronegative AIH, Sn-AIH, children, Histology

2358 - Serum HBV RNA as a Marker of Occult HBV Infection in Patients with Hepatocellular Carcinoma

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Background / Hypothesis: In recent years, particular attention has been paid to the phenomenon of occult HBV infection, which is characterized by the absence of HBsAg in the blood serum despite the presence of HBV DNA in liver tissue. Biopsy-based determination of HBV DNA is informative but an invasive method. In this regard, for non-invasive assessment of viral activity, the serum marker HBV RNA is increasingly used, which allows determination of the etiological structure of HCC and detection of occult forms of infection, influencing prognosis and therapeutic strategy.

Materials and Methods: We examined 75 patients with a confirmed diagnosis of HCC, in whom serological and molecular markers of HBV (HBsAg and HBV DNA) and HCV (anti-HCV and HCV RNA) were determined. As a result of the analysis, two main groups of patients were identified. The first group included 21 patients with the presence of anti-HCV and HCV RNA and negative HBsAg. The second group included 54 patients who had neither HBsAg nor anti-HCV. Detection of HBV RNA was performed using the "ROSSamed HBV-RNA" kit (LLC ROSSA, Uzbekistan).

Results: Among patients with anti-HCV and HCV RNA and negative HBsAg, HBV replication confirmed by the presence of HBV RNA was detected in 2 (9.5%) individuals. This finding demonstrates the presence of HBV replication in patients with HCV infection. Based on these data, it can be assumed that these patients may have an occult form of HBV infection - a condition in which viral activity persists despite negative serological HBsAg results. These results indicate latent HBV replication in patients with HCV infection. In 3 (5.6%) patients without serological markers of HBV and

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HCV (negative HBsAg and anti-HCV), HBV RNA was detected, indicating the presence of occult HBV infection - a diagnostically challenging condition in which the virus persists without obvious signs in standard serological screening.

Conclusions: The data obtained indicate that the determination of HBV RNA in serum can serve as an informative non-invasive marker of occult HBV infection in patients with HCC. This approach allows detection of hidden forms of HBV infection without the need for liver biopsy, which is particularly important for patients who do not consent to invasive procedures. Comprehensive use of serological and molecular methods, including HBV RNA, may contribute

Keyword(s) : HCC, HBV RNA, HBsAg, anti-HCV, HCV, occult HBV infection

2362 - Gallstone Disease in Pediatric Wilson Disease Prevalence Risk Factors and Outcome

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Background / Hypothesis: Gallstone disease (GSD), including gallstones and biliary sludge, is increasingly recognized in children with Wilson disease (WD), but pediatric data on prevalence, risk factors, and early evolution are limited. This study aimed to quantify the burden of GSD at diagnosis and during follow-up in a pediatric WD cohort, identify independent correlates, and evaluate early course and management outcomes.

Materials and Methods: We performed a retrospective analysis of prospectively collected data from 152 children with WD managed at ILBS between January 2011 and July 2025. WD diagnosis was based on a Leipzig score ≥ 4 and/or detection of one homozygous or ≥ 2 compound heterozygous pathogenic ATP7B mutations. GSD was defined ultrasonographically as gallstones (echogenic foci with acoustic shadowing) or biliary sludge (low-amplitude homogeneous echogenicity layering along the posterior gallbladder wall without acoustic shadowing, with or without mobility). The primary objective was to determine the prevalence of GSD at diagnosis. Secondary objectives

included assessing the natural history of GSD, incidence of new gallstones or sludge among those stone-free at baseline, and outcomes of symptomatic and asymptomatic GSD during follow-up. Statistical analysis was performed using SPSS version 26, with $p < 0.05$ considered significant.

Results: Of the 152 children with WD (mean age 8.5 years), 51 (33.6%) had GSD at diagnosis - 33 (65%) had sludge and 13 (25%) had stone. In the univariate analysis, younger age ($p=0.04$), higher bilirubin ($p=0.012$), lower SAP ($p=0.025$), lower Hb ($p<0.001$) and higher reticulocyte count ($p<0.001$), and presence of hemolysis ($p<0.001$) were associated with GSD. On multivariate analysis, hemolysis was independently associated with baseline GSD (adjusted odds ratio [aOR] 2.42; 95% CI 1.00–5.84; $p=0.049$), while older age was inversely associated (aOR 0.85 per year; 95% CI 0.73–0.98; $p=0.028$). Total bilirubin (log-transformed) showed a positive, non-significant trend (aOR 1.33; 95% CI 0.94–1.89; $p=0.105$). Over a median follow-up of 8 (6–24) months, 64.5% sludge persisted, 22.6% progressed to stone formation 12.9% resolved. Baseline stones showed increase in number and size in 30.8%, resolution in 15.4%, persistence in 53.8%. 5 children developed incident stones over a mean follow-up of 90 months. Of the 20 children with gallstones, 1 patient developed Mirizzi syndrome requiring cholecystectomy, while remaining 19 remained asymptomatic.

Conclusions: One-third of children with Wilson disease have gallstone disease at diagnosis, associated with younger age and hemolysis. Routine baseline and follow-up ultrasonography is warranted, particularly in hemolytic phenotype. Most of gallstones and biliary sludge remained asymptomatic.

Keyword(s) : Gall stone, Wilson disease, sludge

2363 - An Innovative Digital Oncology Platform for Enhanced Precision Management of Hepatocellular Carcinoma in China

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Background and Aims:

Hepatocellular carcinoma (HCC) imposes a severe burden in China, ranking as the fourth most common malignant tumor and the second leading cause of cancer-related death, characterized by late diagnosis, complex comorbidities, and highly heterogeneous treatment responses. Multi-Disciplinary Treatment (MDT) and real-world data utilization are critical for improving outcomes, yet traditional workflows are limited by fragmented data and inefficient analysis. We hypothesized that a comprehensive digital oncology platform could enhance multidisciplinary consultation efficiency, enable prognosis-oriented clinical data mining, and support personalized treatment strategies for patients with HCC.

Methods:

A digital oncology platform (Oncology Hub, OH) was designed and implemented by both Roche and Zhongshan hospital to integrate clinical data across the entire care pathway of HCC, including diagnosis, treatment, and follow-up. The clinical data was derived from 2,465 HCC patients who sought medical care between 2020 and 2022 and underwent corresponding follow-up. Advanced analytics and large language model-based digital tools were applied to mine longitudinal real-world data, identify prognostic patterns aligned with clinical needs, and retrieve historical cohorts of clinically similar patients to inform treatment planning and efficacy prediction. System performance was evaluated in terms of consultation efficiency, data processing speed, and clinical decision support capability.

Results:

The Oncology Hub (OH) significantly boosted MDT efficiency in complex HCC cases by accelerating data aggregation and preparation up to 12-fold and enhancing expert collaboration, leading to an expected >500% increase in MDT discussions productivity. The data analysis could be done from OH platform, based on different treatment strategy achieved relatively disease-free survival(DFS) across all stages including I.II.III stages (CNLC). Prognosis-oriented data mining allowed clinicians to clarify the evolution of treatment decision-making, with decision focus shifting from "patient-based" (considering comorbidities in stage I) to "tumor-based" (assessing risk levels in stages II/III). Adjuvant therapy equalized survival for high-risk patients to the standard level in stages III although this

benefit waned in stage III. By leveraging a digital large language model, the system identified historically similar patients from the database to enable evidence-based and stage-specific treatment strategy selection. This improved clinical efficiency, decision confidence, and personalized care for HCC patients across all stages.

Conclusion:

The Oncology Hub (OH) which integrates automated data capture, advanced analytics, and large language model, demonstrates significant potential in providing enhanced clinical benefits in HCC management.

Keyword(s) : Digital oncology platform, Oncology Hub, hepatocellular carcinoma, Multi-Disciplinary Treatment, prognosis analysis, personalized treatment

2365 - Cholangioscopy Guided Diagnosis of Acquired Biliary Web in a Pediatric Patient A Case Report

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Background / Hypothesis:

Biliary webs are one of the rare causes of biliary obstruction in the pediatric population and are usually congenital. Choledocholithiasis contributes to web formation via prolonged biliary stasis and chronic inflammation. Endoscopic retrograde cholangiopancreatography (ERCP) with biliary dilation and cholangioscopy offers a minimally invasive therapeutic option for biliary web formation. We report a case of obstructive jaundice from acquired biliary webs from large common bile duct stone diagnosed with cholangioscopy and successfully managed with ERCP and biliary dilation

Materials and Methods:

A 13-year-old male, previously healthy, presented with right upper quadrant abdominal pain and generalized jaundice. MRCP was done showing intraluminal filling defect in distal common bile duct measuring 1.7 x 1.2 x 1.3 cm with noted gallbladder hydrops and gallbladder sludge. ERCP was performed with cholangiogram that showed markedly dilated left and right intrahepatic ducts (16mm), common hepatic duct (30mm) and common bile duct (18 mm), with a circumferential band-like constriction in the distal CBD, located 15mm from the ampulla. Spyglass cholangioscope was used to

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directly visualize the constriction, which appeared to be a circumferential stenosis with smooth mucosa and without obvious mass. Saline irrigation and aspiration ensured adequate visualization. Using a 12-15mm CRE balloon, the sphincter of oddi and the distal CBD constriction were dilated to 12mm (3atm) for 3 minutes, until the balloon waists were obliterated. Spontaneous passage of black bile, sludge, and stone fragments was noted. Both IHDs were cannulated and balloon sweeps were done extracting some biliary sludge. Occlusion cholangiogram no longer showed any filling defects in the biliary tree. The patient was monitored clinically and biochemically post-procedure.

Results:

ERCP confirmed the presence of a distal biliary web, with upstream bile duct dilatation, likely acquired from a large obstructing choledocholithiasis. Balloon dilation resulted in disruption of the biliary web with direct visualization using cholangioscopy, allowing biliary drainage and improved ductal patency. The patient showed clinical improvement after the ERCP, with relief of obstruction and abdominal pain. Follow-up evaluation showed resolution of jaundice and decrease of liver enzymes and bilirubin levels to normal range.

Conclusions:

This case presents acquired biliary web as a rare but important cause of biliary obstruction in pediatric patients with large choledocholithiasis. Cholangioscopy is crucial in identifying a benign circumferential biliary web. ERCP with biliary dilation is a minimally invasive approach for both diagnosis and treatment, potentially avoiding surgical

Keyword(s) : biliary web, obstructive jaundice, choledocholithiasis, cholangioscopy

2366 - Sex Disparities in Hepatocellular Carcinoma in a Korean Nationwide Cohort

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Background

Hepatocellular carcinoma (HCC) is a malignancy with a well-established male predominance, with incidence rates two to three times higher in men than in women. However, sex-related disparities in HCC have not been

well characterized in Asian populations

Methods

This study included patients newly diagnosed with HCC between 2008 and 2020 from the Korea Central Cancer Registry. Exclusion criteria were age <18 years, missing data, and combined HCC and cholangiocarcinoma. Tumor stage was classified according to the modified Union for International Cancer Control (mUICC) staging system.

Results

A total of 19,729 patients with HCC were included, of whom 15,595 (79.0%) were men. HCC in men peaked in their 50s and declined thereafter, whereas in women, the number of cases increased with age. The male-to-female (M:F) ratio was highest in patients aged 40–59 years (6.0:1), followed by 60s (3.7:1) and ≥70 years (2.4:1), indicating a decreasing male predominance with age.

By etiology, the M:F ratio was 2.7 for HBV, 1.4 for HCV, 22.0 for alcohol-related HCC, and 2.2 for other causes, with the most pronounced male predominance in alcohol-related cases. From 2008 to 2020, the proportion of women with alcohol-related HCC increased from 3.6% to 5.6% ($P < 0.05$), while no significant sex-based changes were observed in other etiologies ($P > 0.05$).

At diagnosis, the proportion of women decreased with advancing stage: 28.2% in stage I, 23.2% in stage II, 18.2% in stage III, and 14.9% in stage IV ($P < 0.001$), suggesting later-stage diagnosis in men.

In stage I, treatment patterns did not differ by sex.

However, in stage II, men were more likely to undergo surgical resection (35.2% vs. 28.2%). In stage IVa, systemic therapy was more common in men (17.4% vs. 12.6%), whereas more women remained untreated (43.2% vs. 36.1%). In stage IVb, men more frequently received transarterial therapy (24.4% vs. 17.7%) and systemic therapy (26.4% vs. 22.6%), while untreated rates were higher among women (50.9% vs. 41.0%).

These findings indicate that men were more likely to receive active treatment, especially in advanced stages. In total patients, women had better survival ($P < 0.001$); however, no difference was observed when analyzed by stage.

Conclusion

The incidence of HCC differs by sex across age groups and underlying etiologies, with the most pronounced disparity observed in alcohol-related liver disease. Notably, the proportion of women developing alcohol-related HCC has been gradually increasing. Following diagnosis, men tend to receive more active treatment compared to women. Recognizing these sex-based

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differences in HCC is essential to guide strategies aimed at improving outcomes

Keyword(s) : Hepatocellular carcinoma, Sex, stage, treatment

2367 - Hepatic arterial infusion chemotherapy sequential transarterial embolization combined with lenvatinib and tislelizumab in patients with high risk unresectable hepatocellular carcinoma an update analysis of a phase 2 trial

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Background / Hypothesis: Several studies have confirmed the efficacy of hepatic arterial infusion chemotherapy (HAIC) combined with lenvatinib and anti-PD-1 therapy in unresectable hepatocellular carcinoma (HCC). This phase 2 trial aims to evaluate the safety and efficacy of HAIC followed by transarterial embolization (TAE) combined with lenvatinib and tislelizumab (an anti-PD-1) as a first-line treatment in patients with high-risk unresectable HCC.

Materials and Methods: This is a single-arm, open-label, phase 2 trial (NCT05532319). Patients with high-risk unresectable HCC (CNLC stage Ib, II, and III) were enrolled and received at least one cycle of HAIC followed by TAE (every 4 weeks) combined with lenvatinib and tislelizumab (200 mg intravenously every 3 weeks). The primary endpoint was the progression-free survival (PFS) rate at six months per RECIST 1.1. Secondary endpoints included objective response rate (ORR), the proportion of patients who underwent curative treatments after partial response (including hepatectomy, ablation, radiotherapy, and liver transplantation), significant tumor necrosis on pathological examination (defined as >70% necrosis of the resected tumor), recurrence-free survival, overall survival and safety. Tumor necrosis was analyzed in patients who received completed hepatectomy.

Results: A total of 64 patients were enrolled and received the study treatment between November 2022 to September 2025. Follow-up was updated as of September 2025. The six-month and twelve-month progression-free survival rates were 84.8% and 55.0%, respectively, with a median progression-free survival of

13.0 months. The objective response rate was 65.6% (42/64) per RECIST 1.1 and 84.4% (54/64) per mRECIST. Following partial response, 31 patients (48.4%) underwent curative treatment, including 20 (31.3%) with R0 hepatic resection, 8 (12.5%) with radiotherapy, 2 (3.1%) with radiofrequency ablation, and 1 (1.6%) with liver transplantation. The median recurrence-free survival was not reached. Other 3 (4.7%) patients received palliative radiotherapy. Based on postoperative histopathological assessment, all patients who underwent complete hepatectomy achieved a major pathologic response ($\geq 70\%$) in their tumors, with 50.0% (10/20) attaining a pathologic complete response. Although the median overall survival was not reached, 19 patients (29.7%) died due to tumor progression (n=15), persistent duodenal ulcer perforation (n=1), continuous hematemesis caused by liver cirrhosis (n=1), liver failure resulting from multiple treatments (n=1), or cerebral hemorrhage of unknown etiology (n=1). Most treatment-related adverse events were grade 1 or 2, including epigastric pain, low-grade fever, transient liver function impairment, and thrombocytopenia.

Conclusions: HAIC sequential TAE combined with lenvatinib and tislelizumab was well tolerated and demonstrated promising survival efficacy in patients with high-risk unresectable HCC.

Keyword(s) :

2370 - Comparison of surgical resection percutaneous ablation and transarterial chemoembolization for hepatocellular carcinoma in patients with recompensated cirrhosis a multicenter retrospective cohort study

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Background / Hypothesis: The concept of recompensation cirrhosis was introduced by the Baveno VII consensus in 2021 and was later recognized in the European Association for the Study of Liver (EASL) guidelines in 2023. However, few studies have focused on the treatment strategies for hepatocellular carcinoma (HCC) in patients with recompensated cirrhosis. This study aims to compare the clinical efficacy and safety of curative treatments, including surgical resection and percutaneous ablation, versus palliative treatment (transarterial chemoembolization, TACE) in HCC patients with recompensated cirrhosis.

Materials and Methods: This multicenter retrospective cohort study including 135 patients from three hospitals: 102 in the curative treatment (58 with surgical resection and 44 with percutaneous ablation) group and 33 in the palliative treatment (TACE) group. Overall survival (OS), progression-free survival (PFS), and treatment-related complications were evaluated and compared. Inverse probability of treatment weighting (IPTW) and subgroup analyses were performed.

Results: Patients in the radical treatment group were more frequently with hepatitis B-related HCC, solitary tumor nodule, smaller in diameter, and earlier tumor stage. After adjustment using IPTW, the baseline characteristics between the two groups were effectively balanced. The 5-year OS rates were 68.3% in the curative treatment group and 60.1% in the palliative treatment group, while the 5-year PFS rates were 51.0% and 58.1%, respectively. No statistically significant differences in OS or PFS were observed between the two groups ($p=0.39$, $p=0.22$). Subgroup analysis demonstrated that the better PFS of curative treatment was more pronounced in patients with lower tumor burden (tumor diameter <3 cm, BCLC stage 0/A, single lesion), better hepatic functional reserve (favorable Child-Pugh score), and stable recompensation status (duration ≥ 5 years). Within the curative treatment group, a comparison between surgical resection and percutaneous ablation therapy showed comparable OS ($p=0.83$), while PFS was significantly better in the surgical resection group than percutaneous ablation group ($p=0.002$). The curative treatment group had a significantly higher incidence of multiple adverse reactions, including liver dysfunction, electrolyte disturbances, and hypoproteinemia, compared to the palliative treatment group.

Conclusions: For HCC patients with recompensated

cirrhosis, the OS were comparable between curative treatments (surgery/ablation) and palliative treatment (TACE). Patients with low tumor burden, well-preserved liver function, and stable recompensation status may achieve better PFS after curative treatments.

Keyword(s) : hepatocellular carcinoma, recompensated cirrhosis, surgical resection, ablation, transarterial chemoembolization

2371 - Prevalence and Significance of Portal Tract Histological Changes in Metabolic Dysfunction-Associated Steatohepatitis

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Background / Hypothesis:

Metabolic dysfunction associated steatohepatitis (MASH) is a leading cause of advanced liver fibrosis globally. The diagnosis of MASH relies on the assessment of histological features of steatosis, ballooning, lobular inflammation, and fibrosis. The NASH Clinical Research Network histological scoring system, the most widely used scoring, is an unweighted composite score of individual grades for steatosis, lobular inflammation, and ballooning. Despite evidence that portal inflammation correlates with fibrosis progression, it is not included in the score, and the prevalence and significance of portal-based features in MASH remain unclear.

To study the prevalence of portal tract changes, including portal inflammation, interface hepatitis, ductular reaction, bile duct damage, plasma cell infiltration, and their correlation with fibrosis in liver biopsies from patients with MASH.

Materials and Methods:

This retrospective cross-sectional study analyzed 505 liver biopsies (>15 mm) from patients with clinically and histopathologically confirmed MASH. Histological scoring was performed using NAS-CRN. Fibrosis stage 3 and 4 were further sub-staged as 3A, 3B, 3C and 4A, 4B, 4C (Laennec cirrhosis substaging), respectively. Portal inflammation, ductular reaction (CK7 immunohistochemistry), interface hepatitis, and plasma cells were semi-quantitatively graded. Fibrosis in Sirius red-stained, whole-slide scanned digitized tissue

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sections was quantified using image analysis. Hepatic progenitor cells were quantified using ImageJ software on CK7-immunostained digitized slides.

Results:

The mean patient age was 45.1 ± 12.5 years, and 65% of the patients were male. Cirrhosis was present in 28.7% of cases, and fibrosis F2–F4 were observed in 69.1%. Portal inflammation was present in 69.3%, of which 53.7% showed mild inflammation. Moderate-severe portal inflammation ($p=0.000$), plasma cells ($p=0.000$) and interface activity ($p=0.002$), significant ductular reaction ($p=0.000$), duct damage ($p=0.000$) were significantly associated with advanced fibrosis stages (F3–F4). HPC (≥ 150) showed significant association with advance fibrosis stages ($p<0.005$). Portal inflammation demonstrated significant correlation with ductular reaction ($p<0.000$), interface activity ($p<0.000$), plasma cell infiltration ($p<0.000$), bile duct damage ($p=0.002$), progenitor cells ($p<0.005$), and fibrosis fraction ($p<0.000$).

Conclusions:

This study confirms that portal inflammation and portal-based histological changes are strongly associated with advanced fibrosis in MASH. The significant correlations between portal inflammation and ductular reaction, interface activity, plasma cells, and HPC expansion suggest a multifactorial role of portal-based changes in disease progression. These findings support the need to incorporate portal inflammation into histological scoring for MASH and to routinely document the severity of portal-based features in the histopathology reports.

Keyword(s) : MASH, Histological scoring, portal inflammation, fibrosis, portal features, quantification

2372 - Refractory Cytomegalovirus Colitis Complicating Ulcerative Colitis After Liver Transplantation The Role of Serial Intestinal Ultrasound

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Background / Hypothesis:

Cytomegalovirus (CMV) is a significant opportunistic pathogen in immunocompromised patients and may complicate colitis in individuals with inflammatory

bowel disease (IBD). In transplant recipients, CMV infection is associated with increased morbidity, and timely diagnosis and antiviral therapy are essential to optimize outcomes.

Materials and Methods:

We report a case of a 23-year-old man with extensive ulcerative colitis (UC) who underwent liver transplantation for Child–Pugh B cirrhosis secondary to autoimmune hepatitis and primary sclerosing cholangitis. The donor was CMV-positive, and the recipient was CMV-negative. At transplantation, UC was in remission on subcutaneous adalimumab. Post-transplant immunosuppression included prednisolone 2.5 mg once daily and tacrolimus (Advagraf®) 8 mg once daily, with valganciclovir 900 mg once daily as CMV prophylaxis.

Results:

Colonoscopy demonstrated diffuse colonic bleeding with scattered deep, punched-out ulcers, most prominent in the left colon. Intestinal ultrasound (IUS) showed active left-sided colitis from the proximal to mid-sigmoid colon. Histology revealed Nancy Histological Index (NHI) grade 4 active chronic inflammation, with CMV inclusions (9/HPF). Antiviral therapy was initiated for 3 weeks. IUS performed 2 days after treatment initiation showed improvement in bowel wall thickness.

Despite initial therapy, repeat colonoscopy showed worsening ulceration and inflammation, with a marked increase in CMV inclusions (up to 60/HPF). Intravenous ganciclovir was restarted, while baseline immunosuppression was maintained. Follow-up IUS after 1 week demonstrated worsening pancolitis, left-predominant. Repeat colonoscopy at 3 weeks showed pancolitis with luminal bleeding and ulcers (UCEIS E1V2B2), and histology again confirmed NHI grade 4 disease with persistent CMV (1–4/HPF).

Antiviral therapy was changed to intravenous foscarnet to complete a total of 6 weeks. UC therapy was switched to intravenous ustekinumab induction. Two-week follow-up IUS demonstrated marked improvement in bowel wall thickening. Colonoscopy confirmed mucosal healing with residual small erosions (UCEIS E1V2B0), and histology was negative for CMV. Maintenance subcutaneous ustekinumab was continued.



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Conclusions:

This case highlights the complexity of managing refractory CMV colitis in post-transplant UC. Serial IUS provided an objective, non-invasive modality to monitor inflammatory activity, detect treatment failure, and assess response to antiviral escalation. IUS findings closely correlated with endoscopic and histological outcomes, supporting its role in guiding timely therapeutic decisions in complex immunocompromised patients.

Keyword(s) : Liver Transplant, Ulcerative Colitis, Intestinal ultrasound

2373 - One step in hospital HCV screening using a qualitative dual detection immunoassay for HCV core antigen and antibodies (Elecys HCV Duo) initial implementation and performance

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Background / Hypothesis: Hepatitis C virus (HCV) infection remains a global health concern. Although the World Health Organization (WHO) proposed a strategy to eliminate HCV by 2030, Japan faces challenges owing to limited access and insufficient support for high-risk populations. Previously, HCV diagnoses required a two-step process, delaying results and increasing costs. In 2024, Japan introduced the Elecys HCV Duo immunoassay, which enables the qualitative dual detection of HCV core antigen (HCV Ag) and antibodies against HCV (anti-HCV). This study aimed to evaluate its effectiveness in identifying patients with an active HCV infection.

Materials and Methods: A retrospective analysis was conducted on 13,045 patients who underwent HCV screening using the Elecys HCV Duo immunoassay.

Results: Of these, 244 were positive for HCV Duo. All 14 patients positive for HCV Ag and anti-HCV had active infection, as confirmed by HCV-RNA testing. Five HCV Ag-positive but anti-HCV-negative patients were identified as false positives. In contrast, five anti-HCV-positive but HCV Ag-negative patients had active infections with detectable HCV-RNA. A low viral load and possible antibody interference were identified as potential reasons for false-negative HCV Ag results.

Conclusions: The Elecys HCV Duo immunoassay effectively simplified HCV diagnosis and facilitated early detection and treatment initiation. Although

false-negative HCV Ag results occurred, the assay ensured linkage to care for patients with both HCV Ag and anti-HCV positivity. These advancements may contribute to enhanced HCV care and support national elimination efforts.

Keyword(s) : HCV, screening, HCV Duo, HCV core antigen

2376 - PNPLA3 I148M and E434K Variants drive Lipid Dysregulation in Pre diabetes and Diabetics

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Background: The PNPLA3 gene, encoding adiponutrin, is a key regulator of hepatic lipid turnover and metabolic homeostasis. Two key single nucleotide polymorphisms (SNPs), I148M (rs738409) and E434K (rs2294918), have emerged as important modulators of lipid accumulation and insulin sensitivity. E434K may have a protective effect, whereas I148M is a known risk allele for metabolic disease and hepatic steatosis. The aim of this study is to examine the PNPLA3 I148M and E434K variations affect lipid metabolism in patients with pre-diabetes and type 2 diabetes.

Methods: Patients with prediabetes and type 2 diabetes (n=391) participated in a case-control genetic association study. PNPLA3 variants I148M and E434K were genotyped by direct sequencing and PCR-RFLP. Liver enzymes, insulin resistance indicators, and serum lipid profiles (triglycerides, HDL-C, LDL-C, and total cholesterol) were assessed. The statistical t-test and hardy Weinberg equilibrium were also carried out.

Results: The total patients had hypertriglyceridemia of 46.9% and hypercholesterolemia of 58.6%. We observed that the 'G' carrier was 39.6 and 42.6%, and the genotype frequencies of I148M were CC (48.4 and 36.9%), CG (24 and 40.9%), and GG (27.6 and 22.3%) in Pre- and T2DM, respectively. The genotype frequencies of E434K in the groups were 45 and 58.6%, 48.8 and 30%, and 6.1 and 11.4% for GG, GA, and AA genotypes, respectively. The patients mean levels of HDL, LDL, VLDL, triglycerides, and total cholesterol were 210±42 mg/dl, 194.6±49.8 mg/dl, 59.6±16.2 mg/dl, 122.8±36.4 mg/dl, and 45.8±17.0 mg/dl, respectively. Higher levels of HDL (71.5 vs 40.1 mg/dl, p<0.03), LDL (133.8 vs 68 mg/dl, P<0.005), VLDL (57.3 vs 25.9 mg/dl, P<0.001), triglycerides (224.8 vs 108 mg/dl, p<0.01), and total cholesterol (223.6 vs 138 mg/dl, P<0.01) were all significantly correlated with CG/GG genotype but not

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GA/GG.

Conclusion: The PNPLA3 variants I148M and E434K are strong inducers of lipid dysregulation that may accelerate the onset of diabetes.

Keyword(s) : Diabetes, Dyslipidemia, Variants

2379 - Exploring The Inflammatory Cancer Transformation From MASLD To Hepatocellular Carcinoma Using Multi Omics And Machine Learning

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease has become a leading cause of hepatocellular carcinoma, yet the key mechanisms driving the transition from steatosis/inflammation to cancer remain unclear. This study hypothesized that integrating multi-omics data with machine learning could systematically uncover the driving molecules and signaling pathways during MASLD-related inflammatory-cancer transformation.

Materials and Methods:

We collected tissue and blood samples from patients with metabolic dysfunction-associated steatotic liver disease, metabolic dysfunction-associated steatohepatitis, and hepatocellular carcinoma. Transcriptomic, proteomic, and metabolomic profiling were performed. Machine learning algorithms, including Random Forest and Support Vector Machine, were employed to construct classification models and identify core biomarkers through network analysis.

Results:

Integrated multi-omics analysis identified crucial dynamic changes in genes, proteins, and metabolites during the progression from MASLD to HCC. The machine learning model successfully distinguished different disease stages with a prediction accuracy of 89.5%. Further analysis revealed that immunometabolic reprogramming and specific inflammatory pathways play a central role in the inflammatory-cancer transformation.

Conclusions:

This study systematically elucidates the key molecular

mechanisms underlying the progression from MASLD to hepatocellular carcinoma through multi-omics and machine learning approaches, providing potential biomarkers and a theoretical basis for early warning and targeted intervention.

Keyword(s) : Metabolic dysfunction-associated steatotic liver disease; Hepatocellular carcinoma; Multi-omics; Machine learning; Inflammation-cancer transformation

2380 - The Unexpected Twist Distal Esophageal Obstruction After Rubber Band Ligation in a 51 Year Old Woman with Decompensated Liver Cirrhosis

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Background / Hypothesis:

Rubber band ligation (RBL) is the standard endoscopic therapy for esophageal varices in patients with cirrhosis and is widely regarded as safe and effective. Rarely, complications such as distal esophageal obstruction may occur, leading to acute dysphagia, vomiting, and hematemesis, with resultant morbidity. Early recognition is crucial, particularly in patients with advanced liver disease.

Case Presentation:

We report the case of a 51-year-old woman with decompensated liver cirrhosis who underwent elective RBL for distal esophageal varices. Within 24 hours of the procedure, she developed sudden-onset dysphagia, retrosternal pain, repeated vomiting, hypersalivation, and an episode of hematemesis. Imaging studies excluded esophageal perforation and mediastinal complications. Urgent upper gastrointestinal endoscopy revealed distal esophageal obstruction caused by post-ligation edema and necrotic mucosal tissue, with superficial oozing consistent with recent hematemesis.

Results:

The patient was managed conservatively with intravenous fluids, a proton pump inhibitor, a somatostatin analog, and close clinical monitoring. Her symptoms gradually improved over 4–5 days. Follow-up endoscopy demonstrated sloughing of necrotic

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tissue with complete restoration of distal esophageal patency. She subsequently resumed oral intake and was discharged without recurrence of symptoms or further complications.

Discussion:

Distal esophageal obstruction is an uncommon but potentially serious complication of RBL. Proposed risk factors include advanced cirrhosis, fragile esophageal mucosa, multiple bands applied during a single session, and pre-existing esophagitis. Hematemesis may occur secondary to mucosal necrosis and edema at the banding site. Prompt recognition, urgent endoscopic evaluation, and supportive care are generally sufficient to achieve recovery, thereby avoiding invasive interventions and preventing progression to severe outcomes such as perforation or stricture formation.

Conclusions:

Although RBL remains the cornerstone of management for esophageal varices, clinicians should remain vigilant for rare complications such as distal esophageal obstruction with hematemesis, particularly in patients with decompensated cirrhosis. Timely endoscopic evaluation and supportive care typically result in favorable outcomes, underscoring the importance of careful post-procedure monitoring.

Keyword(s) : Distal esophageal obstruction, rubber band ligation, decompensated cirrhosis, MASLD, hepatitis, esophageal varices

2382 - A Novel VExUS Based Model Integrating Renal Venous Congestion and Mesenteric Hemodynamics for Noninvasive Risk Stratification of High Risk Esophageal Varices in Cirrhosis

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Background / Hypothesis:

Accurate noninvasive identification of high-risk esophageal varices remains a critical challenge in cirrhosis management. This study aimed to develop and validate a predictive model based on venous excess ultrasound (VExUS) parameters that reflect dynamic portal hypertensive changes, specifically integrating renal venous congestion and superior mesenteric artery (SMA) hemodynamics.

Materials and Methods:

In this retrospective study, 161 patients with liver cirrhosis were analyzed. Patients were classified into a high-risk group (n=78, requiring endoscopic band ligation ± pharmacotherapy) or a low-risk group (n=83, managed conservatively) based on endoscopic findings as the reference standard. VExUS parameters (renal venous Doppler patterns and SMA peak systolic velocity [PSV]) and liver function markers were assessed. A multivariate logistic regression model was constructed.

Results:

The combined model (renal venous congestion + SMA-PSV + ALT) demonstrated excellent discriminatory performance, with an area under the receiver operating characteristic curve (AUC) of 0.931 (95% CI: 0.892–0.970). This significantly outperformed any single parameter, including renal venous congestion alone (AUC 0.841, 95% CI: 0.775–0.907; all p-values <0.01).

Conclusions:

This novel VExUS-based model provides a highly accurate and completely noninvasive tool for stratifying the risk of high-risk esophageal varices. It holds strong potential to refine clinical decision-making and reduce reliance on routine screening endoscopy in patients with cirrhosis.

Keyword(s) : Cirrhosis, Esophageal varices, VExUS, Portal hypertension, Hemodynamics, Non-invasive assessment

2383 - Multikingdom and functional gut microbiota markers for cholangiocarcinoma

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Background / Hypothesis:

While the association between gut microbiota and cholangiocarcinoma (CCA) has been explored, existing research has been limited to bacterial components. The alterations in non-bacterial members (fungi, archaea, and viruses) and their functional characteristics in CCA remain unclear. This study aimed to comprehensively characterize the multi-kingdom gut microbiota and functional profiles in CCA patients and develop a non-invasive diagnostic model.

Materials and Methods:

We performed metagenomic sequencing on 700 fecal

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samples collected from three distinct regions in China, comprising 289 CCA patients, 312 healthy controls, and 99 hepatocellular carcinoma (HCC) patients. Multi-kingdom microbial composition and functional profiles were analyzed. We integrated multiple machine learning algorithms to generate 117 distinct modeling schemes for constructing and screening the optimal CCA diagnostic model. The model was validated across three independent cohorts and tested for specificity against HCC.

Results:

Extensive alterations were identified in CCA patients across multi-kingdom and functional layers, including 198 archaeal, 154 bacterial, 78 fungal, and 120 viral species, as well as 278 genes, 99 pathways, and 151 carbohydrate-active enzymes (CAZymes). A panel of 21 multi-kingdom and functional markers demonstrated superior diagnostic accuracy with an average AUC of 91% across three validation cohorts, outperforming any single-kingdom model. The model exhibited high specificity in distinguishing CCA from HCC, achieving an AUC of 99.74% with a false positive rate of 11.11%.

Conclusions:

This study represents the first comprehensive characterization of multi-kingdom gut microbiome and functional markers in cholangiocarcinoma. Gut multi-kingdom and functional microbial signatures represent a powerful and promising tool for the non-invasive diagnosis of cholangiocarcinoma.

Keyword(s) : cholangiocarcinoma, gut microbiota, Multikingdom microbiota, metagenome, diagnostic model, liver cancer

2384 - Key T Cell Characteristics Associated with Suboptimal Treatment Responses in Pediatric Chronic Hepatitis B

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Background / Hypothesis:

Adult studies have demonstrated that T cell-mediated immune dysfunction is a key mechanism for suboptimal antiviral treatment outcomes, however, the underlying immunological mechanisms driving poor treatment responses in pediatric patients with chronic

hepatitis B (CHB) remain largely unclear. This study aimed to systematically characterize the composition of T cell subsets, gene expression profiles, and functional pathway differences between pediatric CHB patients who achieved HBsAg seroconversion and those who did not following antiviral treatment using single-cell RNA sequencing (scRNA-seq).

Materials and Methods:

8 pediatric patients who received IFN- α and NAs combination therapy was enrolled. Patients were stratified into the seroconversion group and non-seroconversion group based on post-treatment HBsAg seroconversion status. Peripheral blood mononuclear cells (PBMCs) were isolated from peripheral blood samples and subjected to scRNA-seq using the 10 $\times$ Genomics platform. Seurat software was utilized for cell clustering, expression matrix filtering, and cell subset annotation. The Cluster Profiler R package was employed for differential gene expression (DGE) analysis and Gene Ontology (GO)/ Kyoto Encyclopedia of Genes and Genomes (KEGG) functional enrichment analysis to compare T cell subset composition and gene expression profiles between the two groups.

Results:

scRNA-seq identified 11 distinct T cell subsets, including CD4⁺ effector memory T cells, CD4⁺ naive T cells, CD4⁺ regulatory T cells (Tregs), CD8⁺ cytotoxic T cells, and CD8⁺ naive T cells. The proportions of CD4⁺ effector memory T cells ($p < 0.01$) and CD8⁺ T cells ($p < 0.05$) were significantly higher in the seroconversion group compared with the non-seroconversion group, accompanied by marked expansion of T cells with memory and effector functions. A total of 14 differentially expressed genes (DEGs) were highly expressed in the seroconversion group. Functional enrichment analysis demonstrated that these DEGs were predominantly enriched in pathways related to viral response, type I interferon signaling, and immune defense responses.

Conclusions:

The variability in antiviral treatment response among pediatric patients is closely associated with differences in T cell subset composition and functional pathway activation. Our study further identifies expansion of memory/effector T cells and high expression of viral response pathway-related genes as pediatric-specific immune correlates of HBsAg seroconversion. These

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findings provide novel molecular and cellular targets for predicting treatment efficacy and optimizing antiviral therapeutic strategies in pediatric CHB.

Keyword(s) : Chronic hepatitis B; Pediatric patients; Single-cell RNA sequencing; T cell subsets; Antiviral treatment response

2387 - A Comprehensive Ultrasound Model Integrating Gastrointestinal and Hemodynamic Parameters for Non Invasive Risk Stratification of Esophageal Varices

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Background / Hypothesis:

To reduce unnecessary endoscopies in cirrhosis, better non-invasive tools are needed. We aimed to develop and validate a comprehensive ultrasound model, incorporating parameters of Acute Gastrointestinal Injury (AGI) and splanchnic hemodynamics, for predicting high-risk esophageal varices (HRV).

Materials and Methods:

In this retrospective study of 155 cirrhotic patients, we performed a standardized ultrasound protocol assessing gastrointestinal metrics (bowel diameter, wall thickness, motility) and hemodynamic parameters (superior mesenteric artery PSV). Using endoscopic findings as the reference standard, a multivariable logistic regression model was built to predict HRV.

Results:

The ultrasound-derived AGI score was significantly elevated in patients with HRV requiring intervention versus those without (3.04 ± 2.06 vs. 0.67 ± 1.16 , $p < 0.001$). The final composite model (AGI score + Child-Pugh + hemodynamic parameter) demonstrated excellent diagnostic accuracy with an AUC of 0.935 (95% CI: 0.898–0.973), outperforming individual components.

Conclusions:

This physiology-based, multimodal ultrasound assessment offers a highly accurate and non-invasive method for HRV prediction. It holds promise as a triage tool to guide more efficient and selective use of screening endoscopy.

Keyword(s) : Cirrhosis, Esophageal varices, AGIUS,

Portal hypertension, Hemodynamics, Non-invasive assessment

2388 - Serum ATX Predicts Posthepatectomy Recurrence in HBV Related HCC

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Background / Hypothesis:

Hepatitis B virus-related hepatocellular carcinoma (HBV-HCC) is a leading cause of cancer-related death in the Asia-Pacific region, and posthepatectomy recurrence remains a major clinical challenge. Autotaxin (ATX), a secreted glycoprotein, contributes to tumor progression, but its predictive value for posthepatectomy recurrence in HBV-HCC patients remains undefined. This study aimed to explore the association between serum ATX levels and posthepatectomy recurrence in HBV-HCC patients, and to evaluate the predictive efficacy of ATX combined with tumor morphological features.

Materials and Methods:

A total of 120 HBV-HCC patients who underwent hepatectomy were enrolled. Based on the final recurrence outcome, patients were stratified into two subgroups: the recurrence group and the non-recurrence group. Serum ATX levels were measured in all patients. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors for posthepatectomy recurrence. Receiver operating characteristic (ROC) curve analysis was used to assess the predictive value of serum ATX alone and the combined biomarker panel.

Results:

Serum ATX levels were significantly higher in the recurrence group than in the non-recurrence group ($p < 0.05$). Univariate analysis showed that maximum tumor diameter, number of tumors, and serum ATX level were correlated with posthepatectomy recurrence. Multivariate logistic regression analysis confirmed that number of tumors, maximum tumor diameter, and serum ATX level were independent predictors of posthepatectomy recurrence. ROC curve analysis indicated that serum ATX alone had an area under the curve (AUC) of 0.680 (95%CI: 0.610–0.714)



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for predicting posthepatectomy recurrence. the combined detection of ATX, maximum tumor diameter, and number of tumors yielded an AUC of 0.881 (95%CI: 0.802–0.936), with significantly enhanced predictive performance.

Conclusions:

Serum ATX level, maximum tumor diameter, and number of tumors are valuable predictors for posthepatectomy recurrence in HBV-HCC patients, and their combined use can achieve superior predictive efficacy.

Keyword(s) :

2391 - Apilarnil Alleviates Potassium Dichromate Induced Hepatotoxicity in Rats by Modulating the Ferroptosis Pathway (GPx4 TFRC)

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Background / Hypothesis: Potassium dichromate ($K_2Cr_2O_7$) is an industrial and environmental toxin that induces potent oxidative stress and hepatocellular injury. Apilarnil is a bee product rich in bioactive components, such as proteins and steroid hormones. The aim of this study was to investigate the hepatoprotective efficacy of Apilarnil against $K_2Cr_2O_7$ -induced acute liver injury and to evaluate its regulatory role on the ferroptotic cell death pathway [Glutathione Peroxidase 4 (GPx4) and Transferrin Receptor (TFRC)].

Materials and Methods: Rats were randomized into four groups. The Control and toxicity ($K_2Cr_2O_7$) groups received saline, while the treatment groups underwent Apilarnil pre-treatment (400 and 800 mg/kg) via oral gavage for 14 days. On day 15, hepatotoxicity was induced by a single intraperitoneal injection of $K_2Cr_2O_7$ (15 mg/kg) in all groups except the control, which received saline. Blood and liver tissues were harvested 48 hours post-injection. Serum AST, ALT, ALP, and total bilirubin levels were measured using an autoanalyzer. Histopathological alterations, including necrosis,

hydropic degeneration, and periportal inflammatory infiltration, were evaluated using Hematoxylin-Eosin (H&E) staining. Additionally, to investigate the role of the ferroptotic pathway in tissue injury, GPx4 and TFRC expressions were assessed via immunofluorescence analysis.

The study protocol was approved by the Local Ethics Committee for Animal Experiments of Sivas Cumhuriyet University (Date: 01.09.2025, Decision No: 65202830-050.04.04-84).

Results: $K_2Cr_2O_7$ administration caused a significant increase in serum AST, ALT, and Total Bilirubin levels compared to the control group, along with extensive hepatocellular necrosis. The absence of significant differences in ALP levels confirmed the hepatocellular rather than cholestatic nature of the injury.

Immunofluorescence analysis of liver tissues revealed that the expression of the anti-ferroptotic enzyme GPx4 was suppressed, while TFRC levels, indicative of iron uptake, were upregulated in the toxic group. Apilarnil pre-treatment significantly attenuated serum enzyme elevations and histopathological damage scores. Notably, Apilarnil regulated the ferroptotic process by preserving GPx4 expression and suppressing the upregulation of TFRC.

Conclusions: To the best of our knowledge, this is the first study to demonstrate the hepatoprotective efficacy of Apilarnil against $K_2Cr_2O_7$ toxicity. The protective mechanism is attributed not only to its general antioxidant effects but also to its modulation of the ferroptosis pathway (GPx4/TFRC axis) in liver tissue. These findings suggest that Apilarnil may serve as a potential therapeutic agent against chemical-induced liver injury and as a promising candidate for ferroptosis-targeted therapies in preclinical studies.

Keyword(s) : Apilarnil, Ferroptosis, Potassium dichromate, Hepatotoxicity, GPx4, TFRC

2392 - Plug Assisted Retrograded Transvenous Obliteration for Gastric Varices Treatment

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Background / Hypothesis:

This study, with longer follow-up period than our previous study, aimed to further investigate the effectiveness and safety of vascular plug-assisted retrograde vein occlusion in the gastric varices.

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Materials and Methods:

A retrospective study was conducted on 81 patients who underwent PARTO for gastric varices from January 2018, to June 2025.

Results:

The patients were 49 men and 32 women, with ages ranging from 35 to 90 years old. The cause of cirrhosis was alcohol in 37 patients, HBV in 19 patients, unknown cause in 14 patients, metabolic dysfunction associated steatotic liver disease in 5 patients and HCV in 4 patients. There was one patient with alcohol and HBV, and one patient with GIST metastasis

Of 81 patients in whom PARTO was attempted, the procedure was unsuccessful in three patients presenting with gastric variceal bleeding because the catheter could not be advanced to the gastric varices. The PARTO procedure was performed on 42 patients who were hospitalized due to gastric variceal bleeding, and 39 patients who did not have gastric variceal bleeding but underwent the procedure for prevention of future bleeding.

Of the 42 patients with gastric variceal bleeding, PARTO was technically successful in 39 patients, whereas clinical hemostasis with PARTO was achieved in 33 patients. Death within 7 days after PARTO occurred in 6 patients due to gastric variceal bleeding.

Prophylactic PARTO was successfully performed in 39 patients at high risk for future gastric variceal bleeding and there was no gastric varicose bleeding during follow-up.

Conclusions: With a longer follow-up period than our previous study, the present study demonstrates that PARTO is a relatively safe and effective option for both primary and prophylactic management of gastric variceal bleeding.

Keyword(s) : Gastric varices, PARTO, cirrhosis

2393 - Lipopolysaccharide Inhibits Autophagy and Promotes Inflammatory Responses via p38 MAPK Induced Proteasomal Degradation of Atg13 in Hepatic Stellate Cells

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Background / Hypothesis:

Inflammation is central to acute-on-chronic liver failure (ACLF). Hepatic stellate cells (HSCs) are now recognized as inflammatory contributors. While autophagy regulates inflammation, the role of the ubiquitin-proteasome system (UPS) in HSC inflammation during ACLF is unknown. We investigated how LPS, a key driver of ACLF, regulates inflammation in HSCs, focusing on the interplay between UPS and autophagy via the essential autophagic protein Atg13.

Materials and Methods:

Clinical data and liver tissues from ACLF patients were analyzed. In vitro, human LX-2 HSCs were stimulated with LPS. Key techniques included qRT-PCR, Western blot, ELISA, co-immunoprecipitation, and siRNA knockdown of Atg13 to assess inflammatory responses, protein interactions, ubiquitination, and signaling pathways (p38 MAPK).

Results:

In ACLF patients, serum LPS levels correlated with disease severity, and p38 MAPK was overexpressed in liver tissue. In vitro, LPS activated p38 MAPK and triggered inflammatory cytokine (e.g., IL-1 β) secretion in LX-2 cells. This was accompanied by inhibited autophagy and decreased Atg13 protein expression. Knockdown of Atg13 mimicked and exacerbated LPS effects, suppressing autophagy and promoting inflammation. Mechanistically, LPS induced p38 MAPK-dependent polyubiquitination and proteasomal degradation of Atg13.

Conclusions:

LPS promotes HSC inflammation via a novel p38 MAPK/UPS axis that targets Atg13 for degradation, thereby inhibiting autophagy. This cross-talk between major degradation pathways positions Atg13 as a critical mediator. Targeting Atg13 stability or proteasome activity in HSCs may represent a novel therapeutic strategy for mitigating inflammation in ACLF.

Keyword(s) :

2396 - Comparison of Bevacizumab Treatment Outcomes by Hepatitis Virus Infection Gender and Age Group

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a highly prevalent disease in Mongolia, most commonly associated with chronic infections from hepatitis B virus (HBV), hepatitis C virus (HCV), and hepatitis D virus (HDV).

Bevacizumab (Avastin), a VEGF inhibitor, is widely used as a targeted therapy for HCC. This study reviewed medical records from 21 patients to compare treatment outcomes with bevacizumab—measured by the number of treatment courses, which reflects long-term tolerance and duration—across different types of viral infections, genders, and age groups. A higher number of courses was interpreted as indicating better treatment outcomes. The data were sourced from HCC patient records at Interferon Alpha Hospital in 2025.

Materials and Methods:

Information on age, gender, viral status (HBV, HCV, HDV), and the number of bevacizumab treatment courses was extracted from PDF records of 21 patients. Age data were missing for some patients, so analyses involving age were limited to 18 patients. Patients were grouped by viral infection as follows: HBV only, HCV only, HBV + HCV, and HBV + HDV.

Statistical Methods

Average number of treatment courses was calculated for each subgroup using the pandas library in Python. A correlation coefficient was computed to evaluate the relationship between age and the number of courses. Subgroups analyzed included viral infection type (four categories), gender (male/female), and age (<50 years, 50–60 years, >60 years).

Results:

A total of 21 patients were included, with an overall average of 6.95 treatment courses (range: 1–19). There were 13 males (average: 7.15 courses) and 8 females (average: 6.62 courses). The average age was 60.3 years (range: 36–75 years).

Males demonstrated a slightly higher average of 7.15 courses (13 patients) compared to females at 6.62 courses (8 patients). The difference was small but favored males.

Patients over 60 years of age had the highest average at 9.43 courses (7 patients). Those aged 50–60 years.

Conclusion: Bevacizumab treatment demonstrated higher efficacy in patients with HCV only infection, in males, and in those over 60 years of age.

research should involve larger-scale studies and incorporate additional analyses of biochemical markers, such as alpha-fetoprotein (AFP), to further validate these findings.

Keyword(s) :

2397 - Immune Profiling Reveals Distinct Cytokine Signatures in Autoimmune Hepatitis Flares

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Background / Hypothesis:

Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease characterized by immune-mediated hepatocyte injury. Despite effective immunosuppressive therapy, disease flares are common and their immunological underpinnings remain poorly defined. We hypothesized that patients experiencing an AIH flare exhibit a distinct cytokine profile compared to patients in remission and healthy individuals, reflecting an exaggerated T helper 17 (Th17) cell-mediated immune response.

Materials and Methods:

We conducted a case-control study involving 30 AIH patients in active flare, 30 AIH patients in remission, and 30 healthy controls. Serum cytokine levels (including interleukin-17 (IL-17), IL-10, interferon-gamma (IFN- γ), and tumor necrosis factor-alpha (TNF- α)) were quantified using multiplex ELISA. Clinical disease activity was assessed by serum alanine aminotransferase (ALT) levels and Ishak histological activity index on liver biopsy. Group comparisons were made using ANOVA and correlations were evaluated with Pearson's coefficient.

Results:

Patients in AIH flare demonstrated a distinct cytokine signature. Mean serum IL-17 levels were significantly elevated during flares (24.5 ± 5.2 pg/mL) compared to remission (8.7 ± 3.1 pg/mL) and controls (5.4 ± 2.0 pg/mL; $P < 0.001$). Conversely, the anti-inflammatory cytokine IL-10 was markedly lower in flares (3.1 ± 1.0 pg/mL) versus remission (9.8 ± 2.5 pg/mL; $P = 0.002$). IFN- γ and TNF- α levels were also higher in flares than in remission ($P < 0.01$ for both). IL-17 levels correlated positively with ALT ($r = 0.62$, $P < 0.001$) and histological activity, whereas IL-10 levels showed an inverse correlation ($r = -0.45$, $P = 0.008$). No significant differences in cytokine levels were observed between remission-phase patients and healthy controls.

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Conclusions:

AIH flares are associated with a heightened pro-inflammatory Th17-dominant cytokine milieu and reduced anti-inflammatory signals. This distinct immunological profile suggests that Th17-associated cytokines (particularly IL-17) play a key role in AIH flares, and highlights IL-17 as a potential biomarker of disease activity and a target for novel therapeutic interventions to prevent or mitigate flares.

Keyword(s) : Autoimmune hepatitis; cytokines; immune response; disease activity

2399 - Trends in Cholangiocarcinoma Mortality and Incidence in Asia 2000–2023 Insights from Joinpoint Analysis

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Background / Hypothesis:

Cholangiocarcinoma (CCA) is a highly lethal cancer across Asia, where early detection remains uncommon and most patients present with advanced disease. This study evaluates long-term trends in CCA mortality, incidence, annual percent change (APC), average annual percent change (AAPC), and mortality-to-incidence ratio (MIR) from 2000–2023 to identify regional gaps in early detection and guide biomarker development priorities.

Materials and Methods:

Age-standardized mortality and incidence rates by sex and region were obtained from the Institute for Health Metrics and Evaluation (IHME). Temporal patterns were assessed using Joinpoint regression (version 5.4.0) to estimate APC and AAPC. MIR trends were used as a proxy for survival relative to underlying incidence.

Results:

In South Asia (SA), CCA mortality and incidence generally increased between 2000 and 2023. Among women, mortality rose during 2000–2007 (APC 1.82%,

95% CI 1.45–2.23) and accelerated during 2020–2023 (APC 2.87%, 95% CI 1.65–4.34). Men experienced a rise from 2000–2010 (APC 2.11%, 95% CI 1.69–2.71), a temporary decline from 2010–2015 (APC –1.56%, 95% CI –3.81 to –0.36), and renewed increases thereafter (2015–2023 APC 2.08%, 95% CI 1.52–3.08). Incidence trends paralleled mortality. Critically, the MIR in SA remained >1 across the study period, indicating that deaths consistently exceeded diagnoses—strong evidence of late-stage detection, limited diagnostic access, and poor survival.

In Southeast Asia, East Asia, and Oceania (SEAEAO), CCA mortality and incidence declined from 2000 to ~2020, followed by modest increases. Mortality declined among women from 2000–2008 (APC –1.66%, 95% CI –1.89 to –1.36) and among men from 2000–2007 (APC –0.92%, 95% CI –1.21 to –0.64), with fluctuating trends thereafter and slight increases during 2020–2023. Incidence trends mirrored mortality. The MIR decreased from ~0.9 to 0.7, reflecting improved survival, earlier detection, or more effective care compared with SA.

Conclusions:

CCA remains a substantial and uneven burden across Asia. The persistently high MIR in South Asia, contrasted with improving MIR in SEAEAO, highlights major disparities in early diagnosis and treatment. These findings underscore an urgent need for robust, affordable early-detection biomarkers, particularly for resource-limited settings. Emerging liquid biopsy approaches, including circulating tumor DNA, methylation signatures, fragmentation patterns, and multi-omics classifier panels, offer promising avenues to detect CCA at earlier, more treatable stages. Integrating such biomarkers into regional screening strategies, coupled with strengthened diagnostic and treatment capacity, is essential to reduce CCA mortality across Asia.

Keyword(s) : Cholangiocarcinoma; Joinpoint Analysis; annual percent change; average annual percent change; mortality-to-incidence ratio

2400 - Rituximab Achieves High Remission Rates in Steroid Refractory Autoimmune Hepatitis A Controlled Pilot Trial

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Background / Hypothesis:

A subset of autoimmune hepatitis (AIH) patients fail to

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achieve remission with standard corticosteroid and azathioprine therapy, highlighting the need for alternative treatments. B-cell driven autoimmunity is implicated in AIH pathogenesis, suggesting that B-cell depletion therapy could induce remission in refractory cases. We hypothesized that adding rituximab (anti-CD20 monoclonal antibody) to the treatment regimen would improve remission rates in patients with steroid-refractory AIH compared to optimized standard immunosuppressive therapy alone.

Materials and Methods:

In a prospective controlled trial, 50 patients with biopsy-proven AIH who remained active (ALT > 3×ULN, interface hepatitis on biopsy) despite ≥6 months of standard therapy were enrolled. Participants were randomized 1:1 to receive rituximab (two 1,000 mg infusions two weeks apart, alongside ongoing low-dose prednisone) or to intensified standard care (optimized doses of corticosteroids plus introduction or dose escalation of mycophenolate mofetil). The primary endpoint was biochemical remission at 6 months (normalization of ALT and IgG levels). Secondary endpoints included histological improvement, steroid dose reduction, and adverse events.

Results:

At 6 months, the rituximab group achieved a significantly higher remission rate compared to standard care (68% vs 32%, $P = 0.01$). ALT levels normalized in 17 of 25 (68%) rituximab-treated patients, versus 8 of 25 (32%) control patients. Mean serum IgG declined by 35% in the rituximab group, indicating reduced immunoglobulin production, compared to a 10% decline in controls ($P = 0.004$). Histological activity improved by ≥2 points (Ishak score) in 12 rituximab patients (48%) versus 5 controls (20%, $P = 0.05$). Furthermore, 60% of patients in the rituximab arm were able to taper off steroids completely by 6 months, compared to 20% in the control arm ($P = 0.02$). No severe infections or infusion reactions were observed; adverse event rates were comparable between groups.

Conclusions:

B-cell depletion with rituximab markedly improves biochemical and histological remission rates in steroid-refractory AIH and facilitates steroid sparing. These findings support rituximab as an effective and well-tolerated second-line therapy in difficult-to-treat AIH, warranting larger randomized trials to confirm long-

term efficacy and safety.

Keyword(s) : Autoimmune hepatitis; refractory; B-cell therapy; rituximab; immunosuppression

2401 - A Model Incorporating Serum N Terminal Propeptide of Type III Collagen and Platelet Count Predicts Advanced Fibrosis in Chronic Hepatitis B

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Background / Hypothesis:

Accurate non-invasive identification of advanced fibrosis (≥S3) is crucial for improving outcomes in chronic hepatitis B (CHB) patients. Serum N-Terminal Propeptide of Type III Collagen (Pro-C3), a marker of collagen synthesis, has shown promise. This study aimed to develop and validate a novel serum-based model incorporating Pro-C3 and routine clinical parameters for diagnosing CHB-related advanced fibrosis (≥S3).

Materials and Methods:

This retrospective study enrolled 324 treatment-naïve CHB patients who underwent liver biopsy from two centers. Patients from center 1 (n=227) formed the training cohort, and those from center 2 (n=97) formed the validation cohort. Baseline clinical characteristics, serum Pro-C3 (ELISA), liver stiffness measurement (LSM), APRI, and FIB-4 were collected. Histological fibrosis staging used the Scheuer system (S0-S4), with ≥S3 defined as advanced fibrosis. Univariate and multivariate logistic regression were performed to identify independent predictors and construct a predictive model. The model's diagnostic performance was evaluated using the area under the receiver operating characteristic curve (AUC), sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), decision curve analysis (DCA), and calibration curves. Its robustness was tested across various patient subgroups.

Results:

In the overall cohort (n=324), 83 patients (25.6%) had ≥S3 fibrosis. In multivariate analysis, Pro-C3 (OR: 1.04, 95% CI: 1.02-1.05, $P < 0.01$) and platelet count (PLT) (OR: 0.99, 95% CI: 0.98-1.00, $P = 0.04$) were independent predictors. The derived model was: $\text{Logit } P = -1.190 + 0.038 \times (\text{Pro-C3}) - 0.007 \times (\text{PLT})$. In the training cohort, this Pro-C3 and PLT model achieved an AUC of 0.80 (95% CI: 0.74-0.85) for diagnosing ≥S3 fibrosis, with a

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PPV of 59.5% and NPV of 88.1%. Its performance was superior to FIB-4 (AUC: 0.67, $P < 0.01$) and APRI (AUC: 0.71, $P = 0.02$), and comparable to Pro-C3 alone (AUC: 0.78, $P = 0.17$). The model was validated in the external cohort with an AUC of 0.84 (95% CI: 0.75-0.91). DCA indicated greater net clinical benefit, and calibration curves showed good agreement between predicted and observed probabilities. The model maintained diagnostic efficacy (AUCs > 0.70) across all tested subgroups, including different genders, ages, ALT levels, HBeAg statuses, and HBV DNA levels.

Conclusions:

The novel serum model combining Pro-C3 and PLT demonstrates good and robust diagnostic performance for identifying CHB-related advanced fibrosis and cirrhosis ($\geq S3$), offering a convenient and accurate non-invasive tool for clinical decision-making.

Keyword(s) : Chronic hepatitis B; Advanced liver fibrosis; N-Terminal propeptide of type III collagen; Platelet.

2402 - Association Between Three Month Average Blood Glucose Levels and Hepatic Steatosis and Liver Fibrosis

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Background / Hypothesis:

Background: Hepatic steatosis is closely associated with metabolic syndrome and is one of the leading causes of chronic liver disease worldwide. Diabetes mellitus and insulin resistance are major risk factors for the development and progression of fatty liver disease. Glycated hemoglobin (HbA1c), reflecting average blood glucose levels over the previous three months, is widely used in clinical practice. Recently, increasing attention has been paid to the association between HbA1c levels and liver steatosis and fibrosis. This study aimed to evaluate the association between HbA1c levels and hepatic steatosis and liver fibrosis.

Methods: This study included 211 adults aged 28–77 years who underwent both HbA1c testing and liver steatosis and fibrosis assessment at Happy Veritas Hospital between 2024 and 2025. HbA1c levels were measured in whole blood using a fully automated COBAS-6000 biochemical analyzer. Hepatic steatosis

and fibrosis were assessed using the FibroTouch liver scan device. Steatosis was classified as normal, mild (S1), moderate (S2), or severe (S3), and fibrosis stages were categorized from F1 to F4. HbA1c levels were defined as normal, prediabetes, or diabetes. Statistical analysis was performed using SPSS version 25.

Results: A total of 211 adults participated in the study, with a mean age of 52.4 ± 10.4 years; 189 (47.7%) were female and 207 (52.3%) were male. The mean HbA1c level was $6.27 \pm 1.15\%$. HbA1c showed a moderate and statistically significant correlation with hepatic steatosis ($r = 0.359$, $p < 0.001$) and a weak but significant correlation with liver fibrosis ($r = 0.187$, $p = 0.007$). Body mass index was strongly correlated with hepatic steatosis ($r = 0.564$, $p < 0.001$), while age was associated with liver fibrosis ($r = 0.230$, $p = 0.001$).

Conclusion: Higher HbA1c levels are significantly associated with hepatic steatosis and liver fibrosis. HbA1c may serve as a useful biomarker for evaluating fatty liver disease. Further studies are required to clarify its clinical significance.

Keyword(s) : HbA1c, hepatic steatosis, liver fibrosis, BMI

2403 - Optimizing Community Based Hepatitis C Screening Strategies Package of Point of care Testing Reflex Testing and Big Data Based Risk Concentration

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Background / Hypothesis: Remarkable efforts have been made in hepatitis C virus (HCV) management in China. The Centers for Disease Control and Prevention (CDC) has established effective screening and linkage-to-care procedures for high-risk populations. However, whether universal HCV screening should be implemented in the general population and how it can be practically carried out and linked to care in large-scale community settings remain controversial. This study aims to evaluate the feasibility of universal HCV screening in community-based settings and explore strategies to optimize screening efficiency.

Materials and Methods: The study was conducted in two phases. Phase 1 was integrated into annual routine

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health examinations in communities across four provinces. All participants underwent rapid anti-HCV testing using either point-of-care testing (POCT) from fingertip blood or residual blood from other laboratory tests. Those who tested positive received HCV RNA testing via either callback or reflex testing using residual samples. All RNA-positive samples were genotyped. Building on the findings of Phase 1, Phase 2 was conducted in Shanghai in collaboration with the local CDC. A big data approach was used to pre-concentrate individuals at higher risk within the community.

Results: In Phase 1, 21598 participants were screened, with 20 (0.093%) testing anti-HCV positive. HCV RNA testing was successfully performed in 11 of these cases (4 from reflex testing, 7 from call-back and sample retake, 9 lost in call-back process), identifying 6 RNA-positive infections (genotypes: 1b in 3, 2a in 2, and 2b in 1). In Phase 2, after big data-based risk concentration, 8280 participants were screened, with 67(0.81%) individuals testing anti-HCV positive. Reflex RNA testing was performed in 61 cases with adequate samples, confirming 6 active HCV infections.

Conclusions: Utilizing residual blood from routine health examinations for POCT-based anti-HCV screening and reflex HCV RNA testing is feasible for large-scale community screening. Using fingertip blood for POCT HCV antibody screening is also economical and can be considered as a home-lab testing in the future, but the rate of linkage-to-care need improvement. A strategy incorporating big data to concentrate the target population can improve screening efficiency and positivity rates. However, further optimization is needed to enhance the precision of risk stratification and to distinguish between active infections and previously treated cases, thereby avoiding redundant resource use.

Keyword(s) : Hepatitis C virus, community-based screening, big data, point-of-care testing, reflex testing

2405 - Predictors of Relapse After Immunosuppressive Therapy Withdrawal in Autoimmune Hepatitis

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Background / Hypothesis:

Although prolonged immunosuppressive therapy can induce clinical remission in autoimmune hepatitis (AIH),

many patients relapse after drug withdrawal. Identifying patients who can maintain long-term remission off therapy remains a clinical challenge. We hypothesized that certain baseline and treatment-related factors predict the risk of relapse upon cessation of therapy, enabling better selection of patients for safe drug withdrawal.

Materials and Methods:

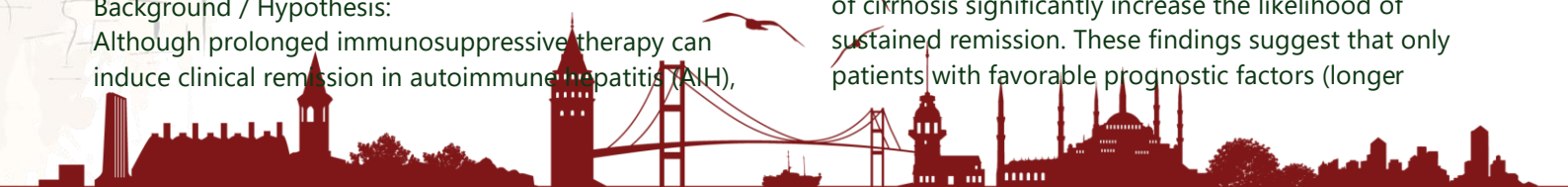
We performed a retrospective cohort study of 120 AIH patients (80% female, mean age 45) who achieved complete remission (normalized liver enzymes and IgG levels, and resolution of interface hepatitis on biopsy) and subsequently discontinued immunosuppressive therapy after a minimum of 2 years of treatment. Patients were followed for a median of 30 months (range 12–60) after withdrawal. Relapse was defined as re-elevation of ALT > 3×ULN or recurrence of histologic AIH requiring treatment resumption. Clinical, biochemical, and histological variables at the time of withdrawal (including age, sex, treatment duration, liver histology, autoantibody titers, IgG level, and presence of cirrhosis) were analyzed. Kaplan-Meier relapse-free survival was calculated, and predictors of relapse were identified using Cox proportional hazards modeling.

Results:

During follow-up, 72 of 120 patients (60%) experienced a relapse after therapy withdrawal, most within the first year (median time to relapse 8 months). In contrast, 48 patients (40%) remained in sustained remission off treatment at last follow-up. Patients who relapsed had a shorter duration of prior therapy (median 30 vs 48 months in non-relapsers, $P = 0.003$) and were more likely to have had cirrhosis at initial diagnosis (35% vs 10%, $P = 0.001$). The presence of residual portal inflammation on the remission biopsy was also associated with relapse (seen in 50% of relapsers vs 15% of non-relapsers, $P = 0.002$). Multivariate Cox analysis identified treatment duration <36 months (HR 2.9, 95%CI 1.5–5.6) and baseline cirrhosis (HR 3.8, 95%CI 1.8–8.0) as independent predictors of relapse ($P < 0.01$). Age, sex, and autoantibody titers were not significant predictors. Patients without these risk factors had a 70% probability of remaining relapse-free at 2 years off therapy.

Conclusions:

A majority of AIH patients relapse after stopping therapy, but extended treatment duration and absence of cirrhosis significantly increase the likelihood of sustained remission. These findings suggest that only patients with favorable prognostic factors (longer



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remission duration and no cirrhosis or residual inflammation) should be considered for therapy withdrawal, and highlight the need for individualized decisions to balance the risks of relapse against long-term medication exposure.

Keyword(s) : Autoimmune hepatitis; remission; relapse; withdrawal; predictors

2407 - Comparison of Bevacizumab Treatment Outcomes (Measured by Number of Courses) with Biopsy Characteristics

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Background / Hypothesis:

The objective of this study was to identify which biopsy indicators—specifically the degree of differentiation, tumor type, and pT stage—show the strongest correlation with the duration of tolerance to Bevacizumab (Avastin) treatment, as measured by the number of treatment courses administered.

Materials and Methods:

Biopsy results from 21 patients were compiled along with the corresponding number of Bevacizumab courses received. Patients were categorized based on degree of differentiation, tumor type (e.g., trabecular, solid, mixed), and tumor stage (T1, T2, T3, T4). The average number of courses was then calculated for each category.

Results:

- Degree of Differentiation:
- Well-differentiated: No patients in the dataset.
- Moderately differentiated: 7 patients, average 10.14 courses, highest 19 courses. This group showed the highest overall results.
- Poorly differentiated/trabecular: 8 patients, average 4.63 courses, highest 13 courses. This group had, on average, 2.2 times fewer courses than the moderately differentiated group.
- Tumor Stage:
- T1: 2 patients, average 4.00 courses, highest 7 and 5 courses. Relatively low number of courses.

- T2: 9 patients, average 9.67 courses, highest including 19, 19, 14, and 11 courses. This stage showed very high results.

- T3: 4 patients, average 4.25 courses, highest 9 and 5 courses. Moderate results.

- T4: 3 patients, average 7.00 courses, highest 13 and 19 courses. High results, though based on few cases.

- Other Characteristics:

- With cholangiocarcinoma component (mixed HCC-CCC with neuroendocrine features): 1 patient, 2 courses. Very low number of courses.

- Neuroendocrine differentiation present: 1 patient, 2 courses. Worst results observed.

Statistical Results (n=21)

- Moderately differentiated: average 10.14 courses (p < 0.01, t-test comparing G2 vs. others).

- Poorly differentiated/trabecular: average 4.63 courses.

- T2 stage: average 9.67 courses (p < 0.05).

- T3–T4 stages combined: average 5.29 courses.

Top 5 Patients with the Highest Number of Courses

1. 19 courses: Patient 01 – HCC pT2NxMx (moderately differentiated).
2. 19 courses: Patient 02 – HCC moderately differentiated pT4NxMx.
3. 14 courses: Patient 03 – HCC pT3N0Mx (moderately differentiated).
4. 13 courses: Patient 04 – Poorly differentiated HCC pT4NxMx.
5. 11 courses: Patient 05 – HCC pT3bNxMx (moderately differentiated).

Conclusions: The best and longest-lasting outcomes for Bevacizumab treatment, as measured by the number of courses tolerated, were seen in patients with the following biopsy characteristics:

1. Moderately differentiated hepatocellular carcinoma (HCC) – average of 10.1 courses.
2. T2 stage HCC – average of 9.7 courses.
3. The combination of moderately differentiated and T2 stage – yielding the highest individual numbers of courses (19, 19, 14, and 11).

Keyword(s) : Bevasizumab

2408 - ASSOCIATION BETWEEN SERUM IRON LEVELS AND HEPATIC STEATOSIS

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Background / Hypothesis: Evidence from multiple studies suggests that excessive hepatic iron accumulation may exert hepatotoxic effects. Therefore, maintaining iron homeostasis in the body may be particularly important for liver health, especially in the context of nonalcoholic fatty liver disease. NAFLD can progress to hepatic fibrosis, cirrhosis, and ultimately hepatocellular carcinoma. The mechanisms underlying the development of nonalcoholic fatty liver disease have not yet been fully elucidated. Therefore, the aim of this study was to examine the association between serum iron levels and the risk of hepatic steatosis.

Materials and Methods: Between January 2024 and 2025, a total of 396 individuals with concurrent measurements of serum iron levels and hepatic steatosis at "Happy Veritas" Hospital were selected and included in this study. Serum iron levels were determined using a Cobas 6000 fully automated biochemical analyzer, and the degree of hepatic steatosis was assessed using a FibroTouch Liver Scan device. Hepatic steatosis measured by FibroTouch liver scan was classified as follows: 0–240 dB/m was considered normal, 240–265 dB/m as S1, 265–295 dB/m as S2, and values above 295 dB/m as S3. Serum iron levels of 11–25 $\mu\text{mol/L}$ were defined as normal, values below 11 $\mu\text{mol/L}$ as low, and values above 25 $\mu\text{mol/L}$ as elevated. Data were analyzed using SPSS version 25.

Results: A total of 396 adults with concurrent measurements of serum iron levels and hepatic steatosis were included in this study. Of these, 189 (47.7%) were female and 207 (52.27%) were male. Among females, the mean age was 50.54 ± 10.84 years (range 27–74 years). Serum iron levels ranged from 2.68 to 49.93 $\mu\text{mol/L}$, with a mean of 16.19 ± 7.53 $\mu\text{mol/L}$. The mean hepatic steatosis value was 250.78 ± 43.39 dB/m (range 163–371 dB/m). Among males, the mean age was 43.27 ± 11.15 years (range 18–81 years). The mean serum iron level was 19.91 ± 8.15 $\mu\text{mol/L}$ (range 6.13–53.00 $\mu\text{mol/L}$). The mean hepatic steatosis value was 275.45 ± 51.4 dB/m (range 181–396 dB/m). The correlation between serum iron and hepatic steatosis was not significant in females ($r = 0.028$, $p = 0.69$) and showed a weak, non-significant correlation in males ($r = 0.08$, $p = 0.31$).

Conclusions: When examining the association between iron status and hepatic steatosis, it is necessary to take into account other disease and dietary factors. In

particular, among women, iron levels may vary substantially due to menstrual status, parity, and pregnancy, and these factors should be carefully considered. In future studies, incorporating additional iron-related parameters such as ferritin, transferrin, and total iron-binding capacity would provide more comprehensive information.

Keyword(s) : nonalcoholic fatty liver disease, hepatic steatosis, serum iron, iron overload

2409 - Deep Learning Assisted Ultrasound Improves Early Hepatocellular Carcinoma Detection in Cirrhosis

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Background / Hypothesis:

Ultrasound surveillance for hepatocellular carcinoma (HCC) in cirrhosis suffers from limited sensitivity, especially for small or early-stage tumors. Missed early HCC can delay curative treatments. Recent advances in artificial intelligence (AI) suggest that deep learning image analysis could enhance tumor detection on ultrasound. We hypothesized that a deep learning-assisted ultrasound system would improve the accuracy of HCC detection in at-risk cirrhotic patients compared to standard radiologist interpretation alone.

Materials and Methods:

We developed a convolutional neural network (CNN) model trained on a dataset of 5,000 ultrasound images (including 1,200 images with pathologically confirmed HCC lesions ≤ 2 cm and 3,800 non-tumor images from cirrhotic livers). The CNN was validated on an independent test set of 200 patients undergoing HCC screening ultrasound (including 50 with small HCC nodules undetectable by prior conventional ultrasound). In a reader study, three blinded radiologists interpreted the ultrasound images with and without AI assistance. Sensitivity, specificity, and area under the ROC curve (AUC) for HCC detection were compared between standard reading and AI-assisted reading. Statistical significance was assessed using McNemar's test for paired proportions and DeLong's test for AUC.

Results:

The deep learning model achieved an AUC of 0.93 in detecting HCC nodules ≤ 2 cm on the test set. AI-assisted interpretation significantly increased sensitivity for small HCC from 68% (radiologists alone) to 85%

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with AI support ($P = 0.01$), with a modest reduction in specificity from 95% to 90% ($P = 0.04$) due to a few false positives. Overall AUC improved from 0.81 (unassisted) to 0.90 (AI-assisted, $P = 0.03$). Notably, 10 out of 15 early HCC lesions missed by all three radiologists on initial review were correctly flagged by the AI system. Integration of the AI tool slightly increased reading time by 10% on average, but radiologists reported improved confidence in their interpretations with AI support.

Conclusions:

Deep learning-assisted ultrasound significantly improves the detection of early-stage HCC in cirrhotic patients, increasing sensitivity for lesions that are often missed by human readers. Implementing AI support in HCC surveillance could lead to earlier tumor detection and better patient outcomes, though careful management of false positives and workflow integration will be important for clinical adoption.

Keyword(s) : hepatocellular carcinoma; ultrasound; deep learning; screening; cirrhosis

2411 - A Machine Learning Model Predicts Hepatic Decompensation Risk in Compensated Cirrhosis

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Background / Hypothesis:

Patients with compensated cirrhosis are at variable risk of progressing to hepatic decompensation (development of ascites, variceal hemorrhage, or encephalopathy), but existing clinical scoring systems (e.g., MELD, Child-Pugh) have limited ability to predict individual risk of decompensation. Machine learning (ML) approaches can analyze complex clinical data to improve risk stratification. We hypothesized that an ML-based model using routine clinical parameters could more accurately predict 1-year decompensation in compensated cirrhosis compared to traditional risk scores.

Materials and Methods:

A retrospective analysis was performed on a cohort of 800 patients with compensated cirrhosis (no prior decompensation) from a tertiary center database. Baseline clinical and laboratory variables (demographics, liver function tests, platelets, liver stiffness by elastography, presence of varices on endoscopy, etc.) were collected. Patients were followed for 12 months for occurrence of first decompensation

event. A random forest ML model was trained on 600 patients and internally validated on 200 patients. Model performance (AUC, sensitivity, specificity) for predicting decompensation at 1 year was compared with MELD and Child-Pugh scores. Feature importance analysis identified key predictors within the ML model.

Results:

Within 1 year, 120 of 800 patients (15%) developed a decompensation event (ascites most commonly). The ML model demonstrated superior predictive accuracy: in the validation set, the random forest achieved an AUC of 0.88 (95% CI 0.82–0.94) for predicting 1-year decompensation, compared to AUC 0.72 for MELD and 0.70 for Child-Pugh scores ($P < 0.01$ for both comparisons). At an optimized risk threshold, the ML model's sensitivity was 80% and specificity 85%, versus 60% and 75% for MELD. Key predictors identified by the model included serum albumin, platelet count, liver stiffness, and INR. Patients stratified as high-risk by the ML model had a 30% observed decompensation rate, versus 5% in low-risk patients (HR 6.5, $P < 0.001$). Calibration analysis showed good agreement between predicted and observed risk.

Conclusions:

A machine learning model using standard clinical data outperformed conventional scores in predicting short-term decompensation risk in compensated cirrhosis. Such an ML-based risk stratification tool could help clinicians identify high-risk patients for closer monitoring or early intervention (e.g., preemptive beta-blockers or transplant evaluation), while sparing low-risk patients from unnecessary invasive surveillance. Prospective validation and integration into clinical workflows are warranted to realize its potential in personalized cirrhosis care.

Keyword(s) : cirrhosis; decompensation; machine learning; risk prediction; prognosis

2412 - Influence of clinical stages of decompensated cirrhosis on optimal portal pressure decrease to control ascites while minimizing hepatic encephalopathy after TIPS

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¹

Background / Hypothesis:

Transjugular intrahepatic portosystemic shunt (TIPS) reduces the portal pressure gradient (PPG), which

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raises two major clinical concerns: how to effectively prevent rebleeding and how to avoid inducing hepatic encephalopathy (HE) due to excessive shunting. For patients with decompensated cirrhosis presenting with both bleeding and ascites, even within the same range of PPG reduction, they may be more susceptible to post-TIPS complications such as HE. This highlights the need for a more nuanced understanding of how an individual's clinical stage of decompensated cirrhosis and baseline liver function influence the outcomes of TIPS procedures.

Materials and Methods:

This retrospective study included 162 patients with cirrhotic variceal bleeding complicated by ascites who were subjected to TIPS at Shandong Provincial Hospital between February 2015 and February 2025. The primary endpoint of this study was all-cause rebleeding. The secondary endpoints were the occurrence of all-cause overt hepatic encephalopathy (OHE), ascites, and portal hypertension complications (PHC).

Results:

To validate different PPG values as targets for hemorrhage prevention, all patients were stratified based on post TIPS PPG. A significant difference in rebleeding was observed between the $<14\text{mmHg}$ and $\geq 14\text{mmHg}$ groups (18.27% vs. 43.10%, $p = 0.012$; sHR = 0.476, 95% CI 0.267–0.847; Gray's test $p = 0.018$). However, no significant differences were found for ascites recurrence, OHE, or PHC, respectively). Meanwhile, a significant difference in OHE was observed between the $<12\text{mmHg}$ group and the $\geq 12\text{mmHg}$ group ($p = 0.037$, sHR = 1.767, 95% CI 1.034–3.019, Gray's test $p = 0.036$), whereas no significant differences were found for rebleeding, ascites recurrence, or PHC. Among patients with Child-Pugh B cirrhosis, a significant difference in the rate of rebleeding was observed between the group with a portal pressure gradient (PPG) $< 14\text{ mmHg}$ and the group with PPG $\geq 14\text{mmHg}$ (18.39% vs. 44.89%, $p = 0.022$, HR = 0.481, 95% CI 0.257–0.900, Gray's test $p = 0.039$). It is noteworthy that when the postprandial glucose increment (PPG) was $< 12\text{mmHg}$, no significant difference was observed in the incidence of OHE between the two groups (37.50% vs 25.00%, $p = 0.140$, sHR = 1.577, 95% CI 0.855–2.908, Gray's test $p = 0.140$). These results remained robust after propensity score matching (PSM) and inverse probability of treatment weighting (IPTW).

Keyword(s) : Transjugular intrahepatic portosystemic

shunt; portal pressure gradient; rebleeding; overt hepatic encephalopathy; ascites; portal hypertension complications

2413 - Breath Volatile Organic Compounds and Machine Learning Enable Non Invasive Detection of Early MAFLD

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD, also known as NAFLD) often goes undiagnosed until advanced stages, as current screening relies on liver enzymes and imaging that can miss early disease. Exhaled breath contains volatile organic compounds (VOCs) reflective of systemic metabolism and oxidative stress, which might serve as non-invasive biomarkers for early liver fat accumulation. We hypothesized that a VOC signature in breath, analyzed with machine learning, can distinguish early MAFLD patients from healthy individuals with high accuracy.

Materials and Methods:

We conducted a cross-sectional study of exhaled breath analysis in 100 subjects: 60 with MRI-confirmed early-stage MAFLD (liver fat fraction 5–15%, normal ALT) and 40 age- and BMI-matched healthy controls without fatty liver. Breath samples were collected using a standardized device and analyzed by gas chromatography–mass spectrometry to identify VOC profiles. Concentrations of key VOCs (e.g., ethane, pentane, isoprene, acetone and others) were quantified. A machine learning classification model (support vector machine with radial kernel) was trained (70% of data) and tested (30% hold-out) to differentiate MAFLD vs control based on VOC patterns. Model performance was evaluated by area under the ROC curve (AUC), sensitivity, and specificity. Important VOC predictors were identified using model feature weights.

Results:

Breath VOC analysis identified a distinct metabolic signature in MAFLD patients. The ML model achieved an AUC of 0.94 (95% CI 0.88–0.99) for detecting MAFLD in the test set. At the optimal probability threshold, sensitivity was 90% and specificity 85% for early MAFLD detection. MAFLD patients showed significantly higher breath levels of ethane and pentane (by 2-fold, $P < 0.001$), consistent with increased lipid peroxidation,

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and lower isoprene (−30%, $P = 0.01$) compared to controls. The top VOC contributors to the model were ethane, acetone, and isoprene. A simplified 3-VOC panel (ethane, acetone, and 2-butanone) still classified MAFLD with AUC 0.88.

Conclusions:

Exhaled breath VOC profiling combined with machine learning provides a promising non-invasive approach for early detection of MAFLD, outperforming traditional liver enzyme screening in sensitivity. The VOC signature of early fatty liver reflects underlying metabolic and oxidative changes, offering a potential breath test for population screening. Further validation in larger cohorts and standardization of breath collection will be important before clinical implementation of this technology for liver disease screening.

Keyword(s) : metabolic fatty liver; MAFLD; biomarkers; breath analysis; machine learning

2414 - COMBINED ASSESSMENT OF FERRITIN AFP AND PIVKA II IN THE DIAGNOSIS OF HEPATOCELLULAR CARCINOMA

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Background: Hepatocellular carcinoma (HCC) remains one of the leading causes of cancer-related mortality worldwide, largely due to diagnosis at advanced stages. Alpha-fetoprotein (AFP) is widely used for surveillance and screening of HCC; however, its sensitivity is insufficient when used alone. Protein induced by vitamin K absence or antagonist-II (PIVKA-II) has been reported to improve diagnostic sensitivity when combined with AFP. Serum ferritin is a marker of hepatic inflammation and iron storage, and several studies have reported elevated ferritin levels in patients with HCC. The combined assessment of ferritin, AFP, and PIVKA-II may therefore represent an important laboratory indicator in the diagnosis of HCC. This study aimed to evaluate and compare these three biomarkers.

Materials and Methods: Among 8,000 adult patients who underwent PIVKA-II and AFP testing at Happy

Veritas Hospital between 2024 and 2025, 194 individuals who also had serum ferritin measured were included. Participants were aged 21–73 years and included 134 males (69.1%) and 60 females (30.9%). Serum ferritin, AFP, and PIVKA-II levels were measured using a fully automated Maglumi immunological analyzer. Participants were classified into normal and elevated ferritin groups. Biomarker levels and age were analyzed and expressed as mean $\pm$ standard deviation. HCC risk categories were defined using internationally accepted cutoff values: AFP >10 and >400 ng/mL; PIVKA-II 50–149 and ≥ 150 mAU/mL; and ferritin >400 and >1000 ng/mL. The influence of age and sex on biomarker patterns was evaluated. A p-value <0.05 was considered statistically significant.

Results: Elevated ferritin levels were associated with increased AFP and PIVKA-II levels, with a stronger association observed in male participants. In males, a 2.45-fold increase in ferritin corresponded to a 2.14-fold increase in AFP and a threefold increase in PIVKA-II. Among participants with ferritin levels of 400–1000 ng/mL, 72.8% were classified as high risk for HCC, while 22.8% of those with ferritin levels >1000 ng/mL were categorized as high risk. In female participants, ferritin elevation was more strongly associated with increasing age, whereas PIVKA-II levels were mostly within the normal range. Combined elevation of biomarkers consistently overlapped with high-risk HCC classifications.

Conclusions: Although ferritin is not a tumor-specific marker, its combined use with AFP and PIVKA-II improves HCC risk assessment. A multi-biomarker approach may enhance early identification of high-risk individuals and strengthen surveillance and screening strategies.

Keyword(s) : Hepatocellular carcinoma, ferritin, alpha-fetoprotein, PIVKA-II, risk assessment, biomarkers

2415 - Fenofibrate Add On Therapy Improves Biochemical Response in Primary Biliary Cholangitis Patients with Incomplete UDCA Response

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Background / Hypothesis:

Up to 30–40% of primary biliary cholangitis (PBC) patients have an incomplete biochemical response to first-line therapy with ursodeoxycholic acid (UDCA),

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leaving them at higher risk for disease progression. Fenofibrate, a peroxisome proliferator-activated receptor- α (PPAR- α) agonist, has shown potential to improve cholestatic markers in PBC. We hypothesized that adding fenofibrate to UDCA in patients with suboptimal response would enhance biochemical response rates and improve cholestatic liver enzymes compared to UDCA alone.

Materials and Methods:

We conducted a 12-month multicenter randomized controlled trial in 80 PBC patients who had an incomplete response to at least 1 year of UDCA (alkaline phosphatase (ALP) $> 1.5\times$ upper limit of normal (ULN) or bilirubin $> 1\times$ ULN). Participants were randomized to receive fenofibrate (160 mg daily) plus continued UDCA (13–15 mg/kg/day) ($n = 40$) or UDCA alone ($n = 40$). The primary endpoint was a biochemical response at 12 months, defined as ALP $< 1.5\times$ ULN and normal bilirubin. Secondary outcomes included absolute change in ALP, gamma-glutamyl transferase (GGT), immunoglobulin M (IgM) levels, liver stiffness by FibroScan, symptom changes (pruritus and fatigue scores), and safety parameters (renal function, transaminases).

Results:

Fenofibrate add-on therapy significantly improved biochemical response rates. At 12 months, 60% of patients in the UDCA+fenofibrate group achieved the primary biochemical response (ALP $< 1.5\times$ ULN, normal bilirubin) compared to 25% in the UDCA-only group ($P = 0.002$). Mean ALP levels decreased by 45% from baseline in the combination group (from 280 to 154 U/L), versus a 20% decrease in the UDCA group (from 290 to 232 U/L, $P < 0.001$ between groups). GGT and IgM levels also declined more in the fenofibrate group (GGT -50% vs -15% ; IgM -30% vs -5% , $P < 0.01$). Liver stiffness improved modestly in the combination arm (median -1.8 kPa) while remaining unchanged with UDCA alone. Symptomatically, 50% of fenofibrate-treated patients reported improvement in pruritus, compared to 20% with UDCA ($P = 0.03$). Fenofibrate was well tolerated; no significant hepatotoxicity was observed, though mild reversible increases in serum creatinine occurred in 10% of patients on fenofibrate.

Conclusions:

In PBC patients with incomplete UDCA response, add-on fenofibrate therapy markedly improves cholestatic biochemical indices, with a higher rate of achieving ALP and bilirubin treatment targets and greater reductions in ALP, GGT, and IgM levels compared to UDCA alone.

These results support the use of fenofibrate as an effective adjunct therapy in UDCA-unresponsive PBC to potentially reduce disease progression.

Keyword(s) : primary biliary cholangitis; fenofibrate; ursodeoxycholic acid; cholestasis; clinical trial

2416 - Oral Vancomycin Therapy in Primary Sclerosing Cholangitis A Pilot Study Demonstrates Biochemical Improvement

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Background / Hypothesis:

Primary sclerosing cholangitis (PSC) is a chronic, immune-mediated cholangiopathy with no established medical therapy proven to alter its course. Mounting evidence suggests a role of the gut microbiota and immune dysregulation in PSC pathogenesis. Oral vancomycin, a minimally absorbed antibiotic, has shown promise in small pediatric studies, potentially by modulating the gut-liver immune axis. We hypothesized that oral vancomycin therapy in adults with PSC would improve cholestatic liver enzyme levels and reduce inflammation compared to placebo.

Materials and Methods:

We conducted a 6-month single-center pilot randomized controlled trial in 40 adults with PSC (median age 38, 70% male). Patients were randomized 1:1 to oral vancomycin (125 mg four times daily, $n = 20$) or placebo ($n = 20$), stratified by presence of inflammatory bowel disease. All patients were on stable ursodiol doses or no UDCA for ≥ 3 months prior to enrollment. Primary outcome was change in serum alkaline phosphatase (ALP) at 6 months. Secondary outcomes included changes in bilirubin, liver aminotransferases, symptom scores (pruritus and fatigue), inflammatory markers (CRP), and adverse events. Between-group differences were assessed using t-tests or Mann-Whitney U as appropriate.

Results:

Vancomycin therapy was associated with significant biochemical improvement. Mean ALP declined by 40% in the vancomycin group (from 320 ± 150 to 190 ± 100 U/L), compared to a 5% decline in the placebo group (310 ± 130 to 295 ± 140 U/L; $P = 0.01$ for between-group ALP change). 30% of vancomycin-treated patients achieved a $\geq 50\%$ ALP reduction vs. 0% of placebo ($P = 0.008$). Total bilirubin remained stable in the vancomycin group but slightly increased in placebo

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(+0.3 mg/dL, $P = 0.15$). There were trends toward improved fatigue scores in the vancomycin arm (median improvement 2 points vs. 0 in placebo, $P = 0.08$) and lower CRP (-2.5 mg/L vs. $+0.5$ mg/L, $P = 0.04$). Pruritus improved in 4 of 10 symptomatic vancomycin patients, versus 1 of 9 in placebo (non-significant). No serious adverse events occurred; 3 patients on vancomycin reported mild nausea, and 2 had transient diarrhea, but no *C. difficile* infections were observed.

Conclusions:

In this pilot trial, oral vancomycin therapy in PSC was associated with significant reductions in ALP and signals of clinical improvement compared to placebo over 6 months. These findings support the concept of microbiota-targeted therapy in PSC.

Keyword(s) : primary sclerosing cholangitis; gut microbiota; vancomycin; cholestatic liver disease; trial

2417 - Clinical Course and Short Term Outcomes of Acute Hepatitis B in a Tertiary Liver Transplant Center

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Background / Hypothesis:

Acute hepatitis B is generally a self-limited infection in adults. However, a subset of patients may develop severe clinical conditions such as acute liver failure and liver transplantation. This study aimed to evaluate the clinical characteristics and short-term follow-up outcomes of patients diagnosed with acute hepatitis B and to identify factors that may be associated with severe disease and mortality.

Materials and Methods:

Between January 2020-December 2024, patients with anti-HBc IgM positivity identified in electronic medical records at a tertiary university hospital were screened. Cases with immunosuppression-related flare and missing follow-up data were excluded. Demographic data, clinical and laboratory findings, and initiation of nucleos(t)ide analogue (NA) therapy were recorded. Virological follow-up included HBsAg and anti-HBs results. Non-normally distributed continuous variables were compared using the Mann-Whitney U test, and

categorical variables were compared using the chi-square test or Fisher's exact test. A p value <0.05 was considered statistically significant.

Results:

A total of 97 patients were included; the median age was 56.5 years [IQR: 39.8–70.3], and 66% were male. The need for liver transplantation was 3.1% ($n=3$). Seven patients (7.2%) had acute exacerbation of chronic hepatitis B (AECHBV) presentation. The distribution of demographic and clinical characteristics is presented in Table 1.

Among hospitalized patients, AST/ALT, baseline/peak total/direct bilirubin, INR/peak INR were higher, whereas albumin was lower; MELD and MELD-Na scores were significantly higher (all $p \leq 0.009$, most $p < 0.001$). In patients admitted to the ICU, platelet counts were lower ($p=0.007$), while bilirubin, INR/peak INR, and MELD/MELD-Na scores were higher (most $p < 0.001$). In addition, encephalopathy and immunosuppression were significantly associated with ICU admission ($p=0.019$ and $p=0.035$, respectively). Patients with HBsAg loss were younger ($p=0.001$) and had higher AST/ALT levels ($p \leq 0.001$); platelet counts were also higher ($p=0.016$). No significant differences were observed between HBsAg loss and bilirubin, INR, or MELD scores ($p > 0.05$). Anti-HBs positivity at months 3, 6, and 12 was strongly associated with HBsAg loss ($p \leq 0.013$) (Tables 2 and 3).

Both patients who died were identified as having AECHBV. Among these patients, 5 (71.2%) required hospitalization, 3 (42.8%) required transplantation, and 1 (14.2%) achieved HBsAg loss.

Conclusions:

Acute hepatitis B in adults is usually benign; however, hospitalization and ICU admission rates were high in our tertiary referral center with a liver transplantation program. These findings identify patients who may require closer monitoring and earlier supportive care, suggest predictors of serological recovery, and underscore earlier recognition and closer follow-up AECHBV cases for organ failure.

Keyword(s) : acute hepatitis b, liver transplantation, hospitalization, intensive care unit

2419 - Generalized Pustular Psoriasis Complicated by Metabolic Dysfunction Associated Fatty Liver Disease and Immunosuppressive Therapy-Associated Transaminitis Highlighting the Skin-Liver Inflammatory Axis

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Background / Hypothesis:

Generalized pustular psoriasis (GPP) is a severe systemic inflammatory disease driven by innate immune dysregulation, including activation of the interleukin-36(IL-36) pathway. Increasing evidence supports a bidirectional interaction between chronic cutaneous inflammation and metabolic liver disease. In patients with metabolic dysfunction-associated fatty liver disease (MAFLD), systemic immunosuppressive therapy may increase susceptibility to transaminitis, even in young and non-obese individuals.

Case Presentation:

A 27-year-old woman with long-standing psoriasis vulgaris complicated by GPP and psoriatic arthritis presented with recurrent flares. She had a prior history of overweight but was normoweight at presentation. After inadequate response to topical therapy and cyclosporine, low-dose methotrexate was initiated with gradual escalation. The patient showed marked clinical improvement with complete resolution of pustulation and a 61% reduction in Psoriasis Area and Severity Index. During treatment under immunosuppressive therapy, serial monitoring demonstrated persistent alanine aminotransferase elevation up to 2.1× the upper limit of normal. Viral hepatitis markers were negative. Abdominal ultrasonography showed fatty liver, and FibroScan confirmed hepatic steatosis (controlled attenuation parameter 281 dB/m) with low liver stiffness (4.5 kPa), consistent with MAFLD without advanced fibrosis. Hepatology evaluation considered the transaminitis multifactorial in the setting of MAFLD and exposure to multiple immunosuppressive agents, and recommended cautious continuation of systemic therapy with close biochemical surveillance.

Discussion:

IL-36, a key cytokine axis in GPP, amplifies neutrophilic inflammation via downstream NF-κB/MAPK signaling and induces broader cytokine cascades, supporting systemic inflammatory spillover beyond the skin. In MAFLD, a pro-inflammatory hepatic milieu and metabolic stress may lower the threshold for hepatocellular injury, increasing vulnerability to drug-associated transaminitis. In this case, liver enzyme elevation occurred despite young age, current normoweight status, and absence of advanced fibrosis, suggesting that host inflammatory and metabolic

susceptibility—potentially influenced by IL-36-linked systemic inflammation may be as important as cumulative drug exposure in determining hepatic tolerance. This phenotype differs from primary autoimmune hepatitis and is more consistent with innate immune-primed liver vulnerability on a MAFLD background.

Conclusions:

Severe GPP may contribute to hepatic vulnerability through an IL-36-linked skin–liver inflammatory axis, while MAFLD may act as a susceptible substrate for immunosuppressive therapy–associated transaminitis in young normoweight patients. Coordinated hepatology follow-up and proactive liver monitoring are essential when prescribing systemic immunosuppression in GPP.

Keyword(s) : Generalized pustular psoriasis; metabolic dysfunction–associated fatty liver disease; transaminitis; immunosuppressive therapy; skin–liver inflammatory axis; interleukin-36.

2420 - Autoimmune Hepatitis–Primary Biliary Cholangitis Overlap Syndrome Clinical Characteristics and Comparative Outcomes

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Background / Hypothesis:

A subset of patients exhibit features of both autoimmune hepatitis (AIH) and primary biliary cholangitis (PBC), known as AIH-PBC overlap syndrome. These patients often require combined immunosuppressive and ursodeoxycholic acid therapy, but their long-term outcomes relative to isolated AIH or PBC remain unclear. We hypothesized that overlap syndrome patients present with more advanced disease but, with appropriate dual therapy, can achieve similar biochemical control and long-term outcomes as single-diagnosis AIH or PBC patients.

Materials and Methods:

We retrospectively studied 50 patients with AIH-PBC overlap (meeting accepted diagnostic criteria, e.g. Paris criteria) from three centers, and compared them with 100 matched AIH-only and 100 PBC-only patients. Baseline features (including presence of cirrhosis) were recorded. All overlap patients received both UDCA and corticosteroid-based immunosuppression. Biochemical remission at 1 and 2 years was evaluated (criteria:

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normalization of ALT and IgG for AIH, ALP $<1.5 \times \text{ULN}$ and normal bilirubin for PBC, both required for overlap). Liver-related outcomes (development of cirrhosis, liver transplant, or liver-related death) were compared over median 8-year follow-up.

Results:

Overlap patients had more aggressive initial disease. At diagnosis, 40% of overlap patients had cirrhosis, vs 15% of AIH-only and 20% of PBC-only ($P < 0.01$). Baseline ALT and ALP were significantly higher in overlap than in AIH or PBC ($P < 0.01$). Despite this, overlap patients achieved biochemical remission rates by 2 years comparable to isolated AIH or PBC: 65% of overlap patients met combined remission criteria, vs 70% of AIH patients (ALT normalization) and 60% of PBC patients (ALP target) ($P = 0.6$). Long-term outcomes were also similar. Ten-year transplant-free survival was ~87% in overlap, 88% in AIH, and 90% in PBC (no significant difference). Rates of liver transplantation (~10% in each cohort) and liver-related mortality (~8–10%) did not differ between groups. Overlap patients did have a higher prevalence of other concomitant autoimmune diseases (30% vs ~10% in AIH, $P = 0.005$).

Conclusions:

AIH-PBC overlap syndrome presents with more advanced liver disease and higher inflammatory/cholestatic markers at baseline, but with appropriate combined immunosuppressive (steroids plus UDCA) therapy, these patients can achieve remission and long-term outcomes comparable to isolated AIH or PBC. Early recognition and aggressive dual treatment of overlap syndrome is critical, as it appears to mitigate the initially severe disease course and leads to outcomes similar to single-diagnosis autoimmune liver conditions.

Keyword(s) : overlap syndrome; autoimmune hepatitis; primary biliary cholangitis; outcomes; cirrhosis

2421 - When Etiologies Intersect Concomitant Hepatitis B Reactivation Hepatic Tuberculosis and Cholelithiasis in a Filipino Male Presenting with Jaundice

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BACKGROUND / HYPOTHESIS:

Hepatic lesions in patients with chronic hepatitis B (CHB) are frequently presumed malignant due to high risk to develop hepatocellular carcinoma, however, infectious etiologies including hepatic tuberculosis (TB) can mimic neoplastic lesions posing diagnostic challenges. We explore a case of a patient presenting with jaundice arising from concomitant hepatitis B reactivation, hepatic TB, and choledocholithiasis further complicating clinical picture, emphasizing the need for thorough assessment and a multidisciplinary approach for targeted treatment.

MATERIALS AND METHODS:

Patient: 58 y/o male with CHB since 1996, not on antiviral treatment, monitored with annual liver function tests; with prior undetectable HBV DNA and fatty liver on ultrasound.

Clinical presentation: 2-month history of jaundice, anorexia, weight loss, RUQ discomfort, and body weakness.

Laboratory: Serum AST, ALT, Bilirubin and ALP, hepatitis serologies, HBV DNA, tumor markers (AFP, CA 19-9), coagulation profile, albumin, and platelet counts.

Imaging: Abdominal ultrasound and triphasic contrast-enhanced CT of the liver.

Procedures: ERCP, percutaneous liver biopsy.

Molecular test: GeneXpert MTB/RIF assay.

Treatment: Antiviral therapy (Tenofovir Disoproxil Fumarate), CBD stone extraction (via ERCP), Anti TB Therapy, Supportive care for liver cirrhosis.

RESULTS:

Laboratory findings: Marked direct hyperbilirubinemia ($10.4 \times \text{ULN}$), elevated AST and ALT ($>3 \times \text{ULN}$), hypoalbuminemia; normal ALP, platelets, INR, AFP and CA19-9; HBsAg(+), HBeAg(-), AntiHBe(+), Anti HBS(-), AntiHBc Total(+) (consistent with HBeAg Negative Chronic Hepatitis B) and HBV DNA: 48,824 IU/ml (from previously undetectable indicating reactivation).

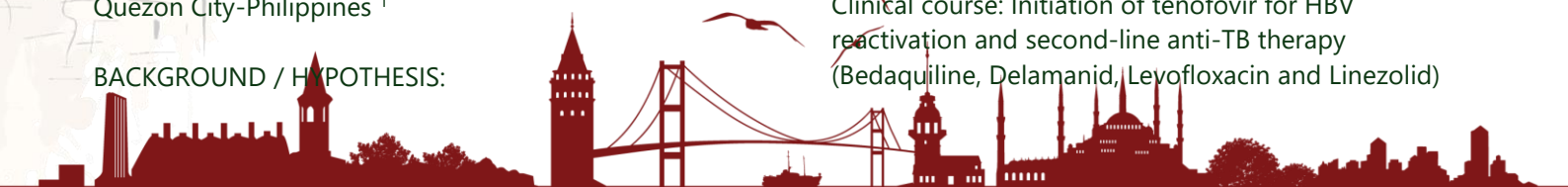
Imaging: Liver cirrhosis with a right lobe hepatic mass (6.2cm $\times$ 4.8cm $\times$ 5.8 cm), intrahepatic bile duct dilatation, and common bile duct stones.

ERCP: Choledocholithiasis confirmed and removed via sphincterotomy and basket destruction and extraction, resulted in partial resolution of jaundice and decrease in AST, ALT, bilirubin trends.

Liver biopsy: Nodular parenchyma with fibrosis and hyalinization; no malignancy or granulomas.

GeneXpert: Positive for Mycobacterium Tuberculosis, rifampicin resistance detected

Clinical course: Initiation of tenofovir for HBV reactivation and second-line anti-TB therapy (Bedaquiline, Delamanid, Levofloxacin and Linezolid)



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and CBD stone extraction via ERCP led to complete resolution of jaundice, improvement of symptoms and laboratory values.

CONCLUSIONS:

This case presents multifactorial jaundice in a patient with hepatitis B reactivation, hepatic tuberculosis, and choledocholithiasis. Accurate diagnosis required a combination of imaging, endoscopic intervention, liver biopsy, and molecular testing. Management targeting all contributing factors - antiviral therapy for HBV, stone removal for biliary obstruction, and anti-TB treatment, resulted in full clinical recovery. Clinicians should maintain a high index of suspicion for coexisting hepatic infections and biliary pathology in patients with CHB presenting with jaundice.

Keyword(s) :

2423 - Snat1 Mediated Glutamine Reprogramming in Macrophages Accelerates MAFLD Associated Colorectal Cancer Liver Metastasis

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Background / Hypothesis: Liver metastasis is the leading cause of death in colorectal cancer (CRC), and metabolic dysfunction-associated fatty liver disease (MAFLD) is a critical driver of CRC liver metastasis. The hepatic microenvironment is intimately linked to metastatic progression.

Materials and Methods: Male C57BL/6J mice were fed a high-fat diet (HFD) for 12 weeks and then 5×10⁵ MC38 cells were injected to the spleen of mice to establish the MAFLD-driven CRLM model. Hepatic metastases, hepatic lipid deposition, and biochemical parameters were assessed. Immunofluorescence assay was used to detect the activation macrophages. Liver transcriptomics and metabolomics were employed to profile gene-expression and metabolic changes. In vitro, palmitic acid and oleic acid (PA) -induced hepatocyte steatosis were incubated with macrophages, immunofluorescence and flow cytometry were used to detect the activation of macrophages. Transwell and wound-healing assays were conducted to measure tumor cell metastasis. And siRNA-mediated Snat1 knockdown and glutamine supplementation were applied to validate the mechanistic role of Snat1.

Results: The number of liver metastases were

significantly increased in MAFLD group, with enhanced hepatic steatosis, higher plasma ALT, TG, TC levels and more pro-tumor macrophages infiltration.

Transcriptomic analysis revealed that differentially expressed genes were highly enriched in the glutamine metabolic pathway, with Snat1 identified as a key hub gene. Metabolomic profiling showed that glutamine conversion to α -Ketoglutarate (α -KG). PA-induced hepatocytes markedly potentiated pro-tumor macrophage activation, an effect abolished by hepatocyte-specific Snat1 knockdown. Transwell and wound-healing assays revealed that macrophages educated by PA-stimulated hepatocytes significantly enhanced colorectal cancer cell invasion and migration, whereas Snat1 silencing in hepatocytes reversed these prometastatic capacities. Mechanistically, PA-challenged hepatocytes elevated Snat1 and H3K27me3 in macrophages; Snat1 knockdown reversed these changes and reduced pro-tumor macrophage activation. Whereas supplementation with an α -KG analogue restored Snat1/H3K27me3 levels and reinstated the activation of pro-tumor macrophages.

Conclusions: Snat1 is an essential driver of MAFLD-associated colorectal cancer liver metastasis via modulation of the α -KG/H3K27me3 axis, offering a novel therapeutic target for the development of clinical treatment therapy.

Keyword(s) : colorectal cancer; sodium-coupled neutral amino acid transporter 1; glutamine metabolism; metabolic dysfunction-associated fatty liver disease

2424 - Sustained virologic response 4 testing as a measure of hepatitis C virus cure in re treated patients following direct acting antiviral failure A sustained virologic response 4 12 concordance evaluation from POLARIS trials

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Background / Hypothesis: Guidance from the American



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Association for the Study of Liver Diseases and the Infectious Diseases Society of America (AASLD/IDSA) and the European Association for the Study of the Liver have endorsed treatment simplification as part of a global call to action for hepatitis C virus (HCV) elimination. Within this simplification strategy, AASLD/IDSA has recently developed a new Hepatitis C Point of Care Test and Treat Algorithm, in which sustained virologic response at week 4 (SVR4) is an acceptable alternative measure of HCV cure for people without cirrhosis or prior direct-acting antiviral (DAA) exposure. However, we hypothesize that SVR4 could also apply to vulnerable patients with prior DAA exposure, to help reduce the risk of patients being lost to follow-up (LTFU). For this reason, we evaluate the SVR4/SVR at week 12 (SVR12) concordance of sofosbuvir/velpatasvir/voxilaprevir (S/V/V) in randomized clinical trials (RCTs). The POLARIS studies included patients who had previously been treated with a DAA and failed.

Materials and Methods: HCV RNA data from patients in the registrational POLARIS-1, -2, -3, and -4 RCTs evaluating treatment with S/V/V for 8 and 12 weeks were evaluated. SVR was defined as patients with HCV RNA less than the lower limit of quantification (15 IU/mL) using the COBAS TaqMan HCV Test v2.0. Only patients with both SVR4 and SVR12 data were included in the concordance analysis.

Results: Overall, 1,056 patients were evaluated for concordance from the POLARIS (1–4) trials: 611 patients were treated with 8 weeks of S/V/V (POLARIS-2, -3) and 445 patients were treated with 12 weeks of S/V/V (POLARIS-1, -4). In the 8-week group, 575 (96%) patients achieved both SVR4 and SVR12 (true positive), giving a 100% sensitivity and 99% positive predictive value (PPV; 5 patients achieved SVR4 but failed SVR12). In the 8-week group, 18 patients failed to achieve both SVR4 and SVR12 (true negative), showing a 78% specificity and 100% negative predictive value (NPV). In the 12-week group, 426 patients achieved both SVR4 and SVR12, showing a 100% sensitivity and 99% PPV (3 patients achieved SVR4 but failed SVR12). In the 12-week group, 5 patients failed both SVR4 and SVR12, showing a 63% specificity and 100% NPV.

Conclusions: HCV treatment with S/V/V can be infrequent in the real world, and achieving SVR4 is highly predictive of HCV cure. This earlier time point to confirm HCV cure may be particularly important in persons at high-risk of being LTFU.

Keyword(s) : Hepatitis C virus (HCV), virology, HCV cure, sustained virologic response, POLARIS

2425 - POST ERCP DUODENAL HEMATOMA AS A CAUSE OF GASTRIC OBSTRUCTION

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Background / Hypothesis:

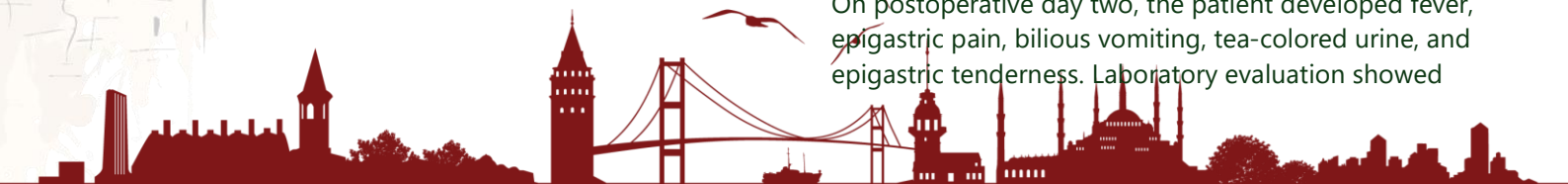
Intramural duodenal hematoma (IDH) is an uncommon but potentially serious complication of endoscopic retrograde cholangiopancreatography (ERCP). Traditionally associated with blunt abdominal trauma, IDH has increasingly been reported following endoscopic interventions, even in young patients without coagulopathy or significant comorbidities. Due to its rarity and nonspecific clinical presentation, diagnosis is often delayed, leading to obstructive symptoms and metabolic derangements. We hypothesize that early recognition of post-ERCP IDH and prompt conservative management can lead to favorable outcomes and avoid unnecessary invasive interventions.

Materials and Methods:

We report a single-center case of a 20-year-old male with no known comorbidities who was admitted for acute calculous cholecystitis with concomitant choledocholithiasis. ERCP with sphincterotomy was performed, during which minimal bleeding occurred and was successfully controlled with local epinephrine injection. The patient subsequently underwent an uncomplicated laparoscopic cholecystectomy. Clinical progression, laboratory investigations, imaging findings, endoscopic evaluation, and management strategies were reviewed. Contrast-enhanced computed tomography (CT) of the abdomen and esophagogastroduodenoscopy (EGD) were utilized for diagnostic confirmation, and follow-up imaging assessed treatment response.

Results:

On postoperative day two, the patient developed fever, epigastric pain, bilious vomiting, tea-colored urine, and epigastric tenderness. Laboratory evaluation showed



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improving transaminases but worsening cholestatic markers, with rising bilirubin and alkaline phosphatase levels, and marked hyperlipasemia (lipase 18× upper limit of normal). Contrast-enhanced CT revealed a large hyperdense intramural lesion extending from the first to third portions of the duodenum, causing luminal narrowing and gastric outlet obstruction. EGD confirmed duodenal obstruction secondary to intramural hematoma. The patient was managed conservatively with bowel rest, nasogastric decompression, intravenous fluids, and close monitoring. Clinical symptoms gradually improved, oral intake was resumed, and the patient was discharged tolerating liquids. A repeat CT scan after two weeks demonstrated significant regression of the hematoma.

Conclusions:

Intramural duodenal hematoma is a rare but important complication following ERCP, even in young patients without predisposing risk factors. Iatrogenic mucosal injury during sphincterotomy may contribute to hematoma formation. A high index of suspicion is essential in patients presenting with post-procedural obstructive symptoms and evolving laboratory abnormalities. Cross-sectional imaging and endoscopy are key to diagnosis. Conservative management remains the cornerstone of treatment and is associated with excellent outcomes, reserving invasive interventions for refractory cases.

Keyword(s) : Jaundice, Intramural Duodenal Hematoma, Gastric Obstruction, ERCP

2428 - Glucocorticoid Therapy Modulates HMGB 1 and K18 Levels in Chronic Drug Induced Liver Injury

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Background / Hypothesis:

Chronic drug-induced liver injury (DILI) affects 8%-20% of DILI patients and may progress to cirrhosis, yet lacks specific therapy. The therapeutic mechanisms of glucocorticoids (GC) in chronic DILI remain unclear.

High Mobility Group Protein 1 (HMGB-1) participates in inflammatory responses, while keratin 18 (K18) and its apoptotic index (ccK18/FL-K18) reflect hepatocellular injury and apoptosis. This study investigated the effects of GC therapy on these biomarkers in chronic DILI patients to optimize treatment strategies.

Materials and Methods:

A retrospective paired study was conducted on 98 chronic DILI patients admitted to the Fifth Medical Center of the Chinese PLA General Hospital from January 2016 to March 2024, all receiving GC therapy. Blood biomarkers (HMGB-1 by ELISA; ccK18 and FL-K18 by chemiluminescence immunoassay) and apoptotic index (ccK18/FL-K18) were measured pre-GC and at 6 months of GC therapy. Wilcoxon signed-rank test was performed to compare biomarker levels and apoptotic index between pre-GC and post-GC therapy.

Results:

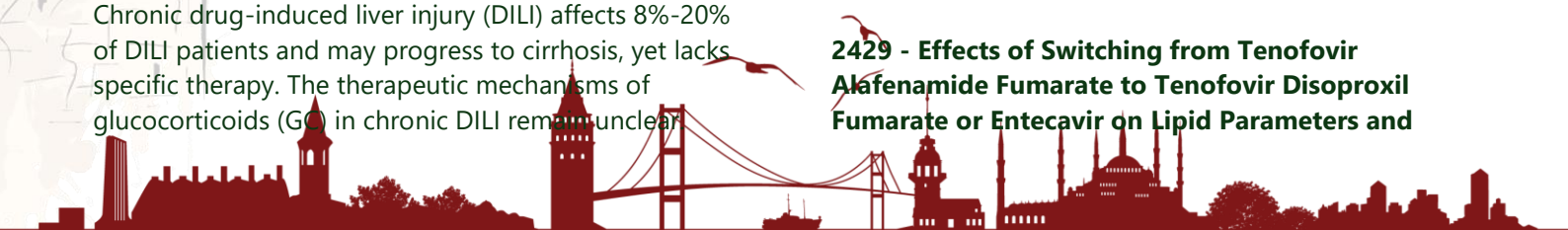
After 6 months of GC therapy, median HMGB-1 increased from 5.25 (2.81-9.56) ng/mL to 16.410 (7.80-34.24) ng/mL, while median ccK18 decreased from 863.50 (297.93-2035.75) U/L to 148.68 (98.20-216.35) U/L and median FL-K18 decreased from 1966.50 (588.80-4607.75) U/L to 296.40 (206.00-402.88) U/L. The apoptotic index increased from 0.45 (0.40-0.53) to 0.48 (0.44-0.57). Wilcoxon signed-rank test demonstrated significant decreases in ccK18 and FL-K18 (both $P < 0.001$), alongside significant increases in HMGB-1 ($P < 0.001$) and apoptotic index ($P = 0.015$).

Conclusions:

After 6 months of GC therapy, median HMGB-1 increased from 5.25 (2.81-9.56) ng/mL to 16.410 (7.80-34.24) ng/mL, while median ccK18 decreased from 863.50 (297.93-2035.75) U/L to 148.68 (98.20-216.35) U/L and median FL-K18 decreased from 1966.50 (588.80-4607.75) U/L to 296.40 (206.00-402.88) U/L. The apoptotic index increased from 0.45 (0.40-0.53) to 0.48 (0.44-0.57). Wilcoxon signed-rank test demonstrated significant decreases in ccK18 and FL-K18 (both $P < 0.001$), alongside significant increases in HMGB-1 ($P < 0.001$) and apoptotic index ($P = 0.015$).

Keyword(s) : Chronic drug-induced liver injury; glucocorticoids; High Mobility Group Protein 1 (HMGB-1); keratin 18 (K18)

2429 - Effects of Switching from Tenofovir Alafenamide Fumarate to Tenofovir Disoproxil Fumarate or Entecavir on Lipid Parameters and



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Atherosclerotic Cardiovascular Disease Risk

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Background / Hypothesis: Given that tenofovir alafenamide fumarate (TAF) is known to adversely affect lipid profiles in patients with chronic hepatitis B, this study aimed to assess the impact of switching from TAF to tenofovir disoproxil fumarate (TDF) or entecavir (ETV) on plasma lipid concentrations and other laboratory parameters.

Materials and Methods: This retrospective cohort study included adult patients (≥ 18 years) with chronic hepatitis B who had received tenofovir alafenamide fumarate (TAF) for at least one year and subsequently switched from TAF to TDF or entecavir for any reason. Patients were required to have no history of lipid-lowering therapy and a baseline low-density lipoprotein cholesterol (LDL-C) level < 190 mg/dL. Lipid parameters, body weight, and body mass index were assessed at baseline and at 3, 6, and 12 months. Demographic characteristics, comorbidities, antiviral and concomitant medications, and relevant laboratory parameters were collected.

Results: A total of 47 patients from three university hospitals were included in the study. Of these, 38 patients switched from TAF to entecavir and 9 to TDF. The mean age of the cohort was 52.7 ± 11.6 years, and 59.6% were male. The clinical and laboratory characteristics of the patients are summarized in Table 1. Following treatment switch, total cholesterol and triglyceride levels showed a transient increase at 3 months, followed by a progressive decline at 6 and 12 months, approaching or falling below baseline values. LDL cholesterol levels remained relatively stable over time, whereas HDL cholesterol demonstrated a temporary decrease with subsequent recovery by 12 months (Figure 1). Renal and hepatic laboratory parameters remained stable during follow-up, and no consistent worsening in metabolic parameters was observed.

Conclusions: These findings indicate that TAF-associated lipid alterations may be reversible and that switching to alternative antiviral agents could have favorable cardiometabolic implications in selected patients with chronic hepatitis B.

Keyword(s) : Dyslipidemia; Chronic hepatitis B; Tenofovir alafenamide; Antiviral therapy switch

2430 - HBsAg Quantification Based Models for Predicting Hepatocellular Carcinoma Risk in Patients with Chronic Hepatitis B The HCC HBsAg Score

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Background / Hypothesis:

Chronic hepatitis B virus (HBV) infection remains a major cause of hepatocellular carcinoma (HCC). Although nucleos(t)ide analogs (NUCs) suppress HBV replication, residual HCC risk requires accurate stratification. Existing prediction models (e.g., PAGE-B, REACH-B) rarely include quantitative hepatitis B surface antigen (HBsAg), limiting their predictive power. We aimed to develop HCC risk-prediction tools for both NUC-naïve and NUC-treated chronic hepatitis B (CHB) patients by incorporating HBsAg levels.

Materials and Methods:

We conducted a retrospective cohort study of CHB patients treated at the Fifth Medical Center of the PLA General Hospital from January 2009 to September 2025. Demographic and clinical data were obtained from electronic records. Patients were classified as NUC-naïve or NUC-treated, and analyses were performed separately. Follow-up extended from HBV diagnosis to HCC development or last contact. Independent predictors of HCC were identified using multivariable Cox proportional hazards models and integrated into nomograms.

Results:

In NUC-treated group (n=1083), 74.2% were male,

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median age was 51 years, and median follow-up was 13 years; 385 patients (35.5%) developed HCC. Independent predictors included age (HR =1.024), male sex (HR =1.992), higher HBsAg levels (500–1500 IU/mL: HR 2.436; 1500–3000 IU/mL: HR 3.417; $\geq$ 3000 IU/mL: HR 3.562), and elevated APRI (0.5–1: HR 2.156; $\geq$ 1: HR 3.251). In NUC-naïve group (n=877), 70.2% were male, median age was 46 years, and median follow-up was 10 years; 212 patients (24.2%) developed HCC. Independent predictors included age (HR 1.029), male sex (HR 1.789), higher HBsAg levels (500–1500 IU/mL: HR 1.787; 1500–3000 IU/mL: HR 2.110; $\geq$ 3000 IU/mL: HR 2.476), HBV DNA (2000– 2×10^7 IU/mL: HR 5.097; <2000 IU/mL: HR 8.798), and elevated APRI (0.5–1: HR 2.445; $\geq$ 1: HR 3.119). Based on these predictors, we developed the HBsAg-HCC score, with separate algorithms for NUC-treated and NUC-naïve patients. (Figures 1A/C). Model performance was superior to existing tools: the area under the curve (AUC) of the model was 0.835 (95% confidence interval (CI): 0.810 - 0.847) vs. PAGE-B 0.826 (95% CI: 0.794 - 0.793, Figure 1B) in NUC-treated patients, and AUC 0.777 (95% CI: 0.732 - 0.809) vs. REACH-B 0.685 (95% CI: 0.615 - 0.734, Figure 1D) in NUC-naïve patients.

Conclusions:

Incorporating quantitative HBsAg significantly enhances HCC risk prediction. The HBsAg-HCC score provides a more accurate tool for individualized risk stratification, early intervention, and improved prognosis in both NA-treated and NA-naïve CHB patients.

Keyword(s) : hepatocellular carcinoma; nucleos(t)ide analogs (NUCs); Quantification HBsAg; Risk prediction

2431 - Metabolomics Reveals Metabolic Reprogramming and Novel Biomarkers in Glucocorticoid Treated Chronic DILI

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Background / Hypothesis:

The mechanisms of glucocorticoid (GC) therapy in

chronic drug-induced liver injury (DILI) remain unclear. This study aimed to analyze metabolite differences between pre-GC and post-GC therapy to explore therapeutic mechanisms and identify treatment-associated metabolic biomarkers.

Materials and Methods:

98 chronic DILI patients receiving GC therapy from our hospital (January 2016-March 2024) were enrolled. Fasting plasma samples were collected at pre-GC and 6 months of GC therapy for untargeted metabolomics analysis using UHPLC-HRMS. Principal component analysis (PCA) and orthogonal partial least squares discriminant analysis (OPLS-DA) were employed to assess metabolic profile alterations. Differential metabolites between pre-GC and post-GC therapy were identified by paired t-test with criteria: $P < 0.05$, $VIP > 1$, and $|\log_2(\text{fold change})| > 1$. KEGG pathway enrichment analysis was performed on these differential metabolites to elucidate metabolic regulatory mechanisms. Subsequently, least absolute shrinkage and selection operator (LASSO) regression was applied to select feature metabolites from the differential metabolites, followed by logistic regression analysis to identify treatment-associated metabolic biomarkers.

Results:

PCA and OPLS-DA analysis revealed significant separation of metabolic profiles between pre-GC and post-GC therapy. Volcano plot showed 743 differential metabolites, including 363 upregulated and 380 downregulated metabolites. KEGG pathway enrichment analysis showed these metabolites were primarily enriched in choline metabolism in cancer, cholesterol metabolism, linoleic acid metabolism, neuroactive ligand-receptor interaction, bile secretion, and glycerophospholipid metabolism. Then LASSO regression selected 30 feature metabolites, logistic regression analysis identified four endogenous metabolites significantly associated with GC therapy: Pe(38:6) (OR=0.141, 95%CI: 0.025-0.804, $P=0.027$), Norvaline (OR=0.272, 95%CI: 0.106-0.698, $P=0.007$), SM(d18:1/22:1(13Z)) (OR=4.896, 95%CI: 1.229-19.508, $P=0.024$), and GPEtn(18:1/16:1) (OR=7.313, 95%CI: 2.375-22.514, $P=0.001$).

Conclusions:

GC therapy induced significant metabolic reprogramming in chronic DILI patients, with lipid metabolism pathways showing the most prominent alterations, suggesting GC exerts therapeutic effects through lipid metabolic remodeling. Four potential

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treatment-associated biomarkers were identified: Pe(38:6), Norvaline, SM(d18:1/22:1(13Z)), and GPEtn(18:1/16:1). These findings provide novel insights into the metabolic mechanisms and biomarker development for GC therapy in chronic DILI.

Keyword(s) : glucocorticoid (GC) therapy; chronic drug-induced liver injury (DILI);

2434 - Clinical HCC Predictors in HBV related Cirrhosis A Real World Study

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Background / Hypothesis:

HBV-related cirrhosis patients who progress to hepatocellular carcinoma(HCC) are still poorly characterised in real-world cohorts.

Materials and Methods:

From January 2024 to January 2025, a total of 234 consecutive patients hospitalized with HBV-related cirrhosis at our center were enrolled in this study. Among them, 114 patients remained free of hepatocellular carcinoma (HCC), whereas 120 were diagnosed with HCC. We compared demographics, virological and biochemical profiles, Child-Pugh class, and C-reactive protein levels between the groups. Independent predictors of HCC were identified through logistic regression and receiver operating characteristic (ROC) analyses.

Results:

The HCC group was significantly older(59.8 ± 9.1 vs. 54.4 ± 11.2 years, $P=0.001$), had more males(80% vs. 65%, $P=0.015$), and a longer median cirrhosis duration [9 (4–19) vs. 5 (0.7–10) years, $P<0.001$]. This group showed lower HBeAg positivity(4.7% vs. 26%, $P<0.001$) and HBV-DNA [1.70 (1.70 – 3.64) vs. 3.08 (1.70 – 6.97) log IU/mL, $P=0.008$], but higher levels of AFP, GGTALP, CRP, platelets, total cholesterol, C3, and C4 (all $P \leq 0.004$). Independent predictors from multivariate modeling were cirrhosis duration ≥ 1.5 y (OR 7.14), HBV-DNA ≤ 4.59 log IU/mL (OR 4.55), and AFP ≥ 32.5 ng/mL (OR 3.22), achieving an AUC of 0.792 (95%CI 0.721–0.864).

Conclusions: In treated HBV-cirrhosis, longer disease duration, suppressed viral load and elevated AFP signal

imminent HCC; combined use of these metrics may enhance early detection strategies.

Keyword(s) :

2435 - Safety and Efficacy Analysis of Argon Helium Cryoablation Combined with Targeted Drugs and Programmed Death Receptor 1 Monoclonal Antibody in Elderly Patients with Unresectable Hepatocellular Carcinoma

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Background / Hypothesis:

The proportion of patients with unresectable hepatocellular carcinoma (uHCC) aged > 60 years is increasing. Cryoablation promotes the release of tumor-associated antigen and initiates systemic anti-tumor immune response, potentially creating a favorable immune micro-environment for subsequent therapies. We aimed to evaluate the efficacy and safety of cryoablation combined with targeted therapy and anti-Programmed Death-1 (PD-1) monoclonal antibody in patients over 60 years of age with uHCC.

Materials and Methods:

A retrospective analysis was conducted on clinical data of HCC patients treated at the Fifth Medical Center of PLA General Hospital between January 2013 and September 2024. 86 patients received cryoablation alone (monotherapy group), 61 patients received cryoablation plus targeted therapy (double combination group), while 57 received cryoablation plus targeted therapy and anti-PD-1 monoclonal antibody (triple combination group). And then efficacy and safety were evaluated. Factors influencing survival prognosis were analyzed using the Cox proportional hazards regression model.

Results:

A total of 204 patients with moderate or advanced uHCC aged > 60 years were included. Median progression-free survival was significantly longer with the triple combination group than with the double combination group and monotherapy group. Both

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median progression-free survival (19.3 months vs.15.3 months vs.4.6 months; $P < 0.001$) and median overall survival showed the same pattern (35.3 months vs.26.5 months vs.14.7 months; $P < 0.001$). Multivariate Cox proportional hazards regression model analysis demonstrated that, relative to monotherapy, the double combination group improved overall survival ($HR=0.39$; $P < 0.001$) and progression-free survival ($HR=0.39$; $P < 0.001$). Compared with the double combination group, the triple combination group was superior in improving both overall survival ($HR=0.53$; $P=0.014$) and progression-free survival ($HR=0.51$; $P=0.001$). There was no difference in the incidence rate of adverse events between the two groups ($P>0.05$).

Conclusions:

This study confirms that the triple combination group is a safe and effective approach for elderly uHCC patients aged >60 years, which provides a clinical basis for personalized treatment in this population.

Keyword(s) : Hepatocellular Carcinoma, Unresectable Hepatocellular Carcinoma, Molecular Targeted Drugs, anti-Programmed Death-1 (PD-1) monoclonal antibody

2436 - Causative Pathogens of Intra abdominal Abscesses Following Whipple Procedure and Comparison with Expected Abdominal Flora

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Background and Aim

Pancreaticoduodenectomy (Whipple procedure) is a complex surgical intervention associated with substantial postoperative morbidity. Intra-abdominal abscesses are among the most common infectious complications and may significantly impact clinical outcomes. Due to extensive surgical manipulation, biliary-pancreatic anastomoses, prolonged hospitalization, and antimicrobial exposure, the causative pathogens may differ from the expected gastrointestinal and biliary flora (FLARA).

This study aimed to identify the microbiological profile of intra-abdominal abscesses developing after Whipple procedure and to compare the isolated pathogens with the expected abdominal flora.

Methods

This retrospective single-center study included patients who underwent Whipple procedure and subsequently developed intra-abdominal abscesses with positive microbiological cultures. Demographic data, time interval between surgery and abscess development, and microbiological culture results obtained from percutaneous or surgical drainage samples were analyzed. Isolated microorganisms were categorized as flora-compatible or flora-discordant based on expected gastrointestinal and biliary flora patterns. Polymicrobial infections were recorded.

Results

Patients who developed intra-abdominal abscesses after Whipple procedure were evaluated. Abscess formation occurred within a median of several days following surgery.

The most frequently isolated pathogens were Enterobacterales, predominantly Escherichia coli, Klebsiella pneumoniae, and Citrobacter freundii. In addition, viridans group streptococci, including Streptococcus sanguinis and Streptococcus parasanguinis, were identified. Polymicrobial infections were observed in a subset of patients.

While a proportion of isolates were compatible with the expected abdominal flora, a considerable number demonstrated partial or complete discordance with FLARA, particularly those associated with oral flora or atypical gastrointestinal colonization.

Conclusion

Intra-abdominal abscesses following Whipple procedure exhibit a heterogeneous microbiological profile with a notable deviation from expected abdominal flora. The presence of flora-discordant and polymicrobial pathogens underscores the importance of culture-guided antimicrobial therapy and may have significant implications for empirical treatment strategies in postoperative pancreatic surgery patients.

Keyword(s) : Whipple procedure; intra-abdominal abscess; postoperative infection; gastrointestinal flora; pancreatic surgery

2437 - Advancing Pediatric Care in Gastroenterology Endoscopic Ultrasound in Pediatrics at a Referral Endoscopy Center in Pakistan

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Background / Hypothesis: Endoscopic ultrasound (EUS) emerged as a revolutionary intervention, serving both diagnostic and therapeutic purposes for four decades among adult patients, however utility in pediatric patients remained limited due to EUS endoscope design e.g. rigidity, diameter, traumatic distal tip, furthermore complicated by need of Monitored Anesthesia Care(MAC) or General Anesthesia(GA) for intervention. Fewer studies with smaller data have been conducted for EUS effectiveness and safety among children. This article aims to describe the utility of EUS for diagnostic and therapeutic purposes among children.

Materials and Methods:: Retrospective analysis of patients ≤ 18 years, underwent EUS at single tertiary referral endoscopy center in Pakistan over 18 years, from 2007 to 2024.

Results: 124 patients ≤ 18 years' age with mean age of 14.75 ± 3.21 years. 54% were male. 90 diagnostic EUS were performed, 17 FNB and 4 FNA. Among Therapeutic EUS, 23 pseudocyst drainage, 2 celiac plexus block for chronic pancreatitis pain, one EUS-guided pancreatic duct cannulation and one EUS-guided antegrade rendezvous' technique-choledocoduodenostomy. No complications were seen.

Conclusions: In this modern era of advanced diagnostic tools and minimally invasive interventions, EUS related procedures are safe and effective in pediatric group of patients too. However, this procedure should be performed at specialized centers with experienced pediatric anesthesia team.

Keyword(s) : EUS , Paediatric

2442 - Comparison of gestational age and birth weights of infants born to HBsAg positive and HBsAg negative pregnant women

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Background / Hypothesis: Some studies have reported that HBsAg positivity during pregnancy may lead to preterm birth or low birth weight. This study evaluated the birth weights and gestational ages of babies born to HBsAg-positive and HBsAg-negative mothers.

Materials and Methods: Data on HBsAg-positive pregnant women were obtained from a multicenter study conducted nationwide, while data on infants born to HBsAg-negative pregnant women in the last two years at Izmir City Hospital were used as the control group. Data were anonymized in accordance with personal data security and transferred to an Excel form, and the necessary analyses were performed.

Results: The incidence of birth weight ≤ 2500 grams was found to be 17.5% in infants of HBsAg-negative mothers and 6.6% in infants of HBsAg-positive mothers. The incidence of birth weight ≥ 2501 grams was found to be 82.5% and 93.4%, respectively ($p < 0.05$). The incidence of birth weight ≤ 36 weeks was 22.3% in infants of HBsAg-negative mothers and 11.2% in infants of HBsAg-positive mothers. The incidence of birth weight ≥ 37 weeks was 77.7% and 88.8%, respectively ($p < 0.05$).

In conclusion, the rate of low birth weight infants

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among HBsAg-negative mothers was found to be higher than that among HBsAg-positive mothers. Similarly, the rate of preterm birth among HBsAg-negative mothers was also found to be higher than that among HBsAg-positive mothers.

Conclusions: When our data was evaluated, it was determined that HBsAg positivity did not pose an additional risk in terms of either giving birth to low birth weight babies or premature birth; in fact, higher rates were observed in the babies of HBsAg-negative mothers.

Keyword(s) : HBsAg, pregnant woman, infants

2444 - Bioinformatics Mapping of Ferroptosis Pathways in NAFLD Progression

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is a progressive liver disorder ranging from simple steatosis (NAFL) to non-alcoholic steatohepatitis (NASH), potentially advancing to fibrosis, cirrhosis, and hepatocellular carcinoma. In recent research, ferroptosis—a type of regulated cell death associated with lipid peroxidation—has emerged as significant in metabolic and inflammatory liver disorders. Compared with microarrays, RNA-seq offers higher sensitivity and accuracy in quantifying gene expression. In the present study, an integrated bioinformatics approach identified ferroptosis-related hub genes involved in NAFLD using RNA-seq datasets, followed by independent validation to improve the robustness of findings.

Methods:

The discovery RNA-seq dataset GSE135251 (GPL18573) included 216 samples (206 NAFLD: NAFL and NASH with fibrosis stages; 10 controls). DEGs were identified using DESeq2 ($|\log_2FC| > 1$, $p < 0.05$). Ferroptosis-related driver, suppressor, and marker genes were obtained from FerrDb V3, duplicates removed, and intersected with DEGs using Venny 2.1.0 to identify shared ferroptosis-associated genes. Shared genes were analyzed through a STRING-based PPI network, visualized in Cytoscape 3.10.4, and key hub genes were selected for downstream analysis using CytoHubba algorithms (MCC, MNC, degree). GO enrichment (BP, CC, MF), KEGG and Reactome pathway analyses were performed using Enrichr. Validation employed RNA-

seq dataset GSE130970 (GPL16791, 78 samples: 72 NAFLD, 6 controls) applying the same differential expression cutoff criteria.

Results:

Differential expression analysis of the discovery dataset identified 2,170 DEGs, including 874 upregulated and 1,296 downregulated genes. Intersection with ferroptosis-related genes yielded 167 shared genes. The PPI network constructed 156 nodes and 700 edges (average node degree 8.97; $p < 1.0 \times 10^{-16}$). Using CytoHubba, eight hub genes were identified: JUN, MYC, IL1B, CCL2, CCND1, MMP9, PTGS2, and ERBB2, with five upregulated and three downregulated in discovery dataset. GO analysis indicated that BP involved neuroinflammatory regulation, unsaturated fatty acid and prostaglandin biosynthesis; GO-CC included AP-1 transcription factor complex and nuclear membranes; GO-MF encompassed cytokine activity, DNA-binding transcription repressor activity, and interleukin-1 receptor binding. KEGG pathway analysis demonstrated enrichment in IL-17, TNF, and bladder cancer pathways while Reactome analysis highlighted interleukin-4, interleukin-13, and cytokine signaling pathways. Validation dataset confirmed significant differential expression of IL1B and MMP9. MMP9 was consistently downregulated across both datasets, suggesting its potential as a robust biomarker. In contrast IL1B showing opposite trends between discovery and validation datasets, reflecting disease heterogeneity or platform variability.

Conclusions:

This study identified key ferroptosis-related hub genes driving NAFLD progression, revealing inflammatory and immune-related pathways and providing novel mechanistic insights and potential targets for future validation.

Keyword(s) : Ferroptosis pathways, NAFLD progression, RNA-seq datasets, Protein-protein interaction network, Biomarker discovery

2445 - Ferroptosis Linked Cell Cycle Hub Genes Reveal New Therapeutic Angels in Sepsis

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²

Background / Hypothesis:

Sepsis, a life-threatening critical emergency, results from an uncontrolled host immune response leading to systemic inflammation and progressive organ

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dysfunction. It is a body response to extreme infections and may result in death if not treated properly and promptly. Rising evidence from literature shows that in most cases, tissue damage, oxidative stress, and multiorgan failure during sepsis are regulated by ferroptosis-related mechanisms. By evaluating the molecular links between sepsis and ferroptosis, viable targets can be detected for disease therapy and biological markers, therefore optimizing patient outcomes.

The literature regarding exact phenomena and principles of how ferroptosis-related genes are linked to sepsis remain limited. However, a detailed transcriptomic analysis is being used to uncover differentially expressed genes (DEGS) correlated with ferroptosis, providing insights into pathophysiology. Materials and Methods:

In this research work, for both discovery (GSE154918) and validation (GSE185263) cohorts were employed. GEO2R was used for conducting differential expression analysis, and then healthy and sepsis patient groups were defined. Thresholds of $|\log_2 \text{fold change}| \geq 1$ and adjusted $p\text{-value} < 0.05$ were adjusted for selecting significant DEGs. Ferroptosis-related genes—including drivers, suppressors, and markers—were obtained from FerrDb v3, filtered for human specificity, and intersected with significant DEGs to identify candidates linked to ferroptosis in sepsis. STRING was used for constructing Protein–protein interaction (PPI) networks, and hub genes were identified using Cytoscape's CytoHubba plugin with MCC ranking. Functional enrichment analyses, including Gene Ontology (GO) and pathway analyses, were performed to determine the biological processes, molecular functions, and pathways most significantly associated with these hub genes using Enrichr.

Results:

After assessment of the discovery dataset, 4,505 significant DEGs, comprising 1,947 upregulated and 2,558 downregulated genes, were revealed. 394 overlapping candidates were yielded after intersecting 2,083 human-specific ferroptosis-related genes. Protein–protein interaction analysis illustrates functional correlation among ferroptosis genes in sepsis, which shows a highly integrated network (383 nodes, 2874 edges; $p\text{-value} < 1.0e-16$). Consistently downregulated hub-genes in both data-sets were CDK1, AURKA, AURKB, CDC20, PLK1, TOP2A, RRM2, BUB1, NEK2, BIRC5. These downregulated hub-genes contribute to cell cycle regulation, mitotic spindle checkpoint, and p53 signaling pathways, which were highlighted by the functional enrichment indicating their promising involvement in modulating ferroptosis-

driven cellular dysfunction throughout the sepsis period.

Conclusion

Comprehensive RNA-Seq-based analysis and database study identify the ferroptosis-associated hub genes that are downregulated consistently and significantly in sepsis. These findings strongly lay a foundation for the development of targeted therapeutic strategies. These evidences also support that ferroptosis is a central regulatory mechanism in sepsis.

Keyword(s) : Cell Cycle Regulation, Ferroptosis, Hub Genes, RNA-Seq, Sepsis

2446 - Shared and stage Specific ferroptosis related biomarkers Across NAFL and NASH An RNA seq Analysis

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) advances from simple steatosis (NAFL) to non-alcoholic steatohepatitis (NASH), a stage characterized by inflammatory response, oxidative stress, and fibrosis risk. Ferroptosis—an iron-dependent lipid peroxidation process—has emerged as a key contributor to NAFLD pathological progression, yet stage-specific ferroptosis-linked molecular signatures remain poorly defined in humans. This study aimed to identify ferroptosis-associated central genes distinguishing NAFL and NASH and to reveal their functional pathways using integrated transcriptomic and network analyses supported by external validation.

Methods:

RNA-seq dataset GSE135251 (Control $n=10$; NAFL $n=51$; NASH $n=155$) was analyzed using GEO2R. Differentially expressed genes (DEGs) were filtered using $p < 0.05$ and $|\log_2 \text{FC}| \geq 1$, yielding 1928 NAFL and 2155 NASH DEGs. Overlap with FerrDB produced 155 NAFL and 177 NASH ferroptosis-related DEGs. STRING-based protein–protein interaction networks were constructed in Cytoscape, and CytoHubba algorithms (MCC, MNC, Degree) identified top hub genes. Consensus hubs were defined by intersecting top genes across algorithms. Functional annotation was performed using the Enrichr tool based on GO ontology, KEGG, and Reactome pathways. External validation was conducted using RNA-seq dataset GSE126848, and both discovery and cross validation

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datasets were sourced from a common platform (GPL18573).

Results:

Nine consensus hubs were identified for NAFL and seven for NASH, of which five were shared hubs (MYC, JUN, IL1B, CCL2, PTGS2), all statistically significant. Shared hubs were upregulated, indicating concurrent activation of oxidative stress, inflammatory signaling, iron dysregulation, and lipid peroxidation. Pathway enrichment demonstrated progressive intensification of ferroptosis-associated pathways from NAFL to NASH, including increased ROS generation, cytokine–chemokine signaling, NF-κB activation, and senescence-linked Reactome pathways. In the independent validation dataset, five of nine NAFL and five of seven NASH consensus hubs were observed. Among the five shared hubs, MYC, JUN, and CCL2 were common to both NAFL and NASH and were supported by the independent validation dataset. FerrDB annotation identified PTGS2 as a ferroptosis marker and highlighted context-dependent regulatory roles for MYC and CCL2.

Conclusions:

This integrative analysis identifies MYC, JUN, and CCL2 as independently validated ferroptosis-associated biomarkers across NAFL and NASH, underscoring ferroptosis as a central mechanism in NAFLD progression and highlighting promising molecular targets for future therapeutic exploration.

Keyword(s) : NASH, NAFLD, RNA Seq, Ferroptosis, Bioinformatics

2447 - Hepatitis B reactivation secondary to rituximab treatment

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Introduction

Hepatitis B virus (HBV) reactivation is a significant complication in patients receiving long-term chemotherapy or immunosuppressive therapy. In this case, we aimed to present a patient who developed acute liver failure due to HBV reactivation after a rituximab-containing chemotherapy protocol.

Case Report

A 79-year-old male patient. The patient was diagnosed with Chronic Lymphocytic Leukemia one year ago. Following diagnosis, he was initiated on a rituximab-containing chemotherapy protocol at an external center. During this period, the patient's liver enzymes,

INR, and bilirubin levels were normal. HBsAg was negative, anti-HBs was positive, and anti-HBc IgG was not tested. Approximately one year after starting treatment, the patient presented to the gastroenterology clinic with complaints of fatigue, yellowing of the entire body, and dark urine. The patient's blood tests revealed alanine aminotransferase (ALT) of 911 U/L, aspart aminotransferase (AST) of 740 U/L, total bilirubin of 1.96 mg/dl, direct bilirubin of 1.49 mg/dl, and an INR of 1.28. A hepatitis panel was ordered and found to be positive for HBsAg, positive for Anti-HBs, positive for Anti-HBc IgG, negative for Anti-HBc IgM, and negative for HBV PCR of 586514 IU/ml. The patient was suspected of developing HBV reactivation due to rituximab treatment, and Tenofovir Disoproxil Fumarate 245 mg was initiated as antiviral therapy. Despite treatment, progressive increases in bilirubin and INR levels were observed during follow-up, and the patient was admitted to the intensive care unit. The patient, who had no chance of liver transplantation during follow-up, died due to acute liver failure and complications related to HBV reactivation.

Conclusion

Use of drugs with a high risk of reactivation, such as rituximab, and positive HBV serologic markers can result in hepatitis B reactivation in these patients. Therefore, prophylactic antiviral therapy against hepatitis B should be initiated before starting chemotherapeutic drugs with a high risk of reactivation.

Keyword(s) : hepatitis b, Rituximab, reactivation

2448 - DNA microarray based identification of ferroptosis related biomarkers in alcoholic hepatitis

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Background:

Alcoholic hepatitis (AH) is a severe inflammatory liver disorder associated with prolonged alcohol consumption, marked by oxidative stress, metabolic dysregulation, and hepatocyte injury. Ferroptosis—an iron-dependent, lipid peroxidation-driven form of cell death—has emerged as a key contributor to alcohol-induced liver damage, but ferroptosis-related transcriptional biomarkers in AH remain poorly defined

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. This study aimed to identify these biomarkers in alcoholic hepatitis in humans.

Methods:

DNA microarray datasets from GSE28619 (7 controls, 15 AH samples; GPL570) were analyzed using GEO2R (adjusted $p < 0.05$, $|\log_2FC| \geq 1$). After probe deduplication, 3151 DEGs retrieved from volcano plot were reduced to 2019 unique genes; intersection with FerrDb yielded 282 ferroptosis-related DEGs. A protein-protein interaction (PPI) network of ferroptosis-related DEGs was constructed using STRING, and six consensus hub genes associated with ferroptosis were identified from MCC, MNC, and Degree analyses via CytoHubba. Functional enrichment, including GO and KEGG pathway analysis, was performed using the Enrichr tool on MCC-identified hub genes to explore their biological relevance. Gene Set Enrichment Analysis (GSEA; Hallmark gene sets) evaluated coordinated pathway alterations.

Results:

The ferroptosis-related PPI network contained 280 nodes and 2109 edges ($P < 1 \times 10^{-16}$), reflecting significant interactions. Enrichment analysis of MCC hub genes revealed involvement in apoptosis, regulatory processes, oxidative stress responses, and mitochondrial stability. Six consensus hub genes (BCL2, EGFR, JUN, ESR1, PTEN, SIRT1), obtained by intersecting top-ranked genes from MCC, MNC, and Degree analyses, highlighted their central role in ferroptosis-related network regulation.. Significance of all genes was confirmed. Differential GSEA showed that upregulated pathways (bile acid, xenobiotic, and fatty acid metabolism; TNF- α /NF- κ B signaling) reflected cholestatic stress, oxidative injury, and lipid remodeling, whereas downregulated pathways (MYC Targets, E2F Targets, protein secretion, unfolded protein response, DNA repair, mTORC1 signaling, oxidative phosphorylation) indicated metabolic suppression, mitochondrial dysfunction, and redox imbalance. This contrast highlights a molecular environment highly permissive to ferroptosis.

Conclusions:

AH demonstrates a ferroptosis-associated molecular signature characterized by oxidative stress, metabolic collapse, and impaired mitochondrial and ER function. Identified hub genes represent potential biomarkers and regulatory components of ferroptosis-driven hepatocellular injury, providing insights for targeted therapeutic strategies in alcoholic hepatitis.

Keyword(s) : Alcoholic hepatitis , Microarray, Ferroptosis, Liver

2449 - Real World Barriers to Post SVR Monitoring in HCV Insights from a Physician Survey

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Background / Hypothesis:

Despite the success achieved with direct-acting antivirals (DAAs) in sustained virologic response (SVR) in hepatitis C virus (HCV), the real-world challenges and barriers to post-treatment follow-up are not well understood. Identifying these obstacles is crucial for improving long-term outcomes. This study aims to evaluate physicians' real-world barriers to (post-SVR) follow-up.

Materials and Methods:

A cross-sectional survey was conducted among 103 physicians using a structured questionnaire designed by an expert hepatologist to capture post-treatment follow-up practices and barriers to adherence. The questionnaire was developed based on current clinical guidelines and expert input to ensure comprehensive coverage. Content validity was confirmed through a pilot study, and reliability was assessed using internal consistency measures.

Results:

Among 103 respondents, 64.1% hepatologists and 35.9% non-hepatologists; most had over 10 years of clinical experience (76.7%). The majority considered liver cirrhosis to be the main influence in post-SVR follow-up (93.2%). Fear of hepatocellular carcinoma (HCC) was the predominant concern (84.5%), with advanced fibrosis/cirrhosis (94.2%) and MAFLD (65%) identified as major risk factors post-SVR. Abdominal ultrasound (92%) and alpha-fetoprotein (AFP) (80%) were the most frequently requested investigations. Reported barriers for post-treatment surveillance included low patient awareness (64.1%), lack of clear national pathways (32%), and limited resources (23.3%).

Conclusions:



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Low patient awareness and the lack of standardized national pathways are the main barriers to consistent post-SVR follow-up. Future strategies should prioritize implementing structured national protocols and improving patient education for surveillance.

Keyword(s) : Barriers, Cirrhosis, Hepatocellular carcinoma, Hepatitis C virus, Sustained virologic response

2451 - Late T cell mediated Rejection (TCMR) in liver allograft biopsies Impact of heterogeneity on the progression to chronic TCMR

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Background / Hypothesis:

Late T-cell mediated Rejection (LAR) is clinically, histopathologically, & mechanistically heterogeneous & under-represented in Banff RAI score. The Banff Liver group has recently stressed that LAR and chronic TCMR (CR) have different subtypes and certain prognostically important features are not assessed currently. There is an urgent need to understand the behavior of different subtypes of LAR, risk of progression to CR and the clinical outcome. This study aims to determine the prevalence of LAR and the impact of its subtypes on the clinical course.

Materials and Methods:

Retrospective cross-sectional study of liver allograft biopsies (n=939) of liver transplant recipients (n=1276) between 2010-2025. Liver biopsies reported as rejection (n=458) obtained from 166 patients were further analysed. Those with histological diagnosis of LAR at ≥ 6 months post-liver transplant (n=116) from equal number of recipients were reviewed. Only patients with multiple biopsies with the diagnosis of TCMR were selected as the study group (n=30). Clinical and histological characteristics were recorded: post-transplant duration of LAR, episodes of rejections, progression to CR, time period to last follow-up and total post-transplant survival, phenotypes of LAR and CR. LAR was histologically characterised as chronic hepatitis (CH), isolated central perivenulitis (ICP), and

PCRR. CR was categorised as ductopenic, CH with fibrosis, or mixed.

Results:

Age ranged between 5-59 years with male: female ratio of 6.5. LAR diagnosed 6 months- 8.5 years post-liver transplant. Histological subtypes were CH (23), ICP (5), PCRR (2). LAR progressed to CR in 22 (73.3%) in 15 days- 8 years. CR subtypes were ductopenia (3), CH with fibrosis (13), ductopenia with fibrosis (6). Histological diagnosis of chronic rejection to last follow-up was 1 month- 7 years and post-transplant survival was 9 months- 14 years, with some on follow-up. CR (ductopenic or mixed ductopenic with fibrosis subtype) had previous LAR with portal duct damage more than zone 3 damage (78%); whereas CR (CH type) had zone 3 changes more than portal changes (69%). Recipients with chronic TCMR with fibrosis showed longer survival than those with ductopenic with/out advanced fibrosis (1 year, 1 year 7 months respectively). Survival determined by the comorbidities and ductopenic rejection.

Conclusions:

This study confirms that late and chronic TCMR are heterogeneous with variable effect on clinical course. LAR with predominant zone 3 changes progresses to fibrotic CR whereas TCMR with duct damage causes ductopenic rejection. Zone 3 microvascular injury is important determinant of fibrosis progression; this along with severity of duct damage and interface activity should also be recorded

Keyword(s) : late TCMR, chronic TCMR, Banff, subtypes, chronic hepatitis, fibrosis, Isolated central perivenulitis, ductopenic.

2452 - Efficacy of Intermittent Fasting on Hepatic Enzymes Fat Content and Fibrosis in Patients with Non Alcoholic Fatty Liver Disease A Systematic Review and Meta Analysis of Randomized Controlled Trials

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is a growing global health concern with limited effective non-

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pharmacological treatment options. Intermittent fasting (IF) has gained attention as a dietary intervention that may improve metabolic and hepatic outcomes. However, evidence from randomized controlled trials (RCTs) remains variable. This systematic review and meta-analysis aimed to evaluate the efficacy of intermittent fasting on hepatic enzymes, liver fat content, and liver fibrosis in patients with NAFLD.

Materials and Methods:

A comprehensive search of PubMed, Embase, Scopus, and the Cochrane Library was conducted up to November 2025. Eight RCTs involving 592 participants with NAFLD were included. Pooled standardized mean differences (SMDs) with 95% confidence intervals (CIs) were calculated using random-effects models. Statistical heterogeneity was assessed using the I^2 statistic. Publication bias was evaluated using Egger's regression test.

Results:

Intermittent fasting was associated with a significant reduction in alanine aminotransferase (ALT) levels (SMD = -0.58; 95% CI: -0.83 to -0.33; I^2 = 0.00%; p < 0.01) and aspartate aminotransferase (AST) levels (SMD = -0.44; 95% CI: -0.81 to -0.07; I^2 = 48.23%; p = 0.02). IF also led to a marked improvement in liver fat content (SMD = -0.86; 95% CI: -1.21 to -0.50; I^2 = 0.00%; p < 0.01). Additionally, a significant reduction in liver fibrosis was observed (SMD = -0.44; 95% CI: -0.80 to -0.07; I^2 = 73.48%; p = 0.02), despite substantial heterogeneity. Egger's regression test indicated no evidence of publication bias.

Conclusions:

This meta-analysis of randomized controlled trials demonstrates that intermittent fasting significantly improves hepatic enzymes, liver fat content, and liver fibrosis in patients with NAFLD. These findings support intermittent fasting as a promising dietary strategy for the management of NAFLD, although further large-scale, long-term RCTs are warranted to confirm its sustainability and clinical applicability.

Keyword(s) : Intermittent fasting, Non-alcoholic fatty liver disease, ALT, AST, Liver fibrosis, Systematic Review, Meta-analysis

2453 - Impact of Prior Drug Allergies on severity of drug induced liver Injury a study from tertiary care hospital Pakistan

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BackDrug-induced liver injury (DILI) remains a significant diagnostic and clinical challenge in hospital settings. It can present as either predictable (dose-dependent) or unpredictable (idiosyncratic) liver toxicity caused by commonly used medications such as anti-tubercular agents, antiepileptics, or even therapeutic doses of drugs like paracetamol. While the mechanisms of many idiosyncratic reactions remain unclear, genetic susceptibility, immune dysregulation, and allergic or hypersensitivity responses are believed to play important roles in increasing vulnerability to hepatotoxicity. In light of encountering a steady increase in hospitalizations related to drug intake in the past few years, this study aims to evaluate the role of prior drug allergies in a population of patients with well-characterized drug-induced liver injury.

ground / Hypothesis:

Materials and Methods:

DILI cases were identified and categorized on the basis of COIMS/RUCAM score and the exclusion of other liver diseases. This study included two arms, one arm included the DILI cases with no prior drug allergy history and the other consisted of all those identified DILI cases who had experienced any prior drug allergies including itching, coughing, hives, periorbital swelling, abdominal pain, vomiting, and diarrhea. Clinical and laboratory parameters were analyzed to identify the predictors of morbidity (prolonged hospital stay > 7 days) and mortality.

Results:

Out of 460 patients, 120 (25.8%) cases of DILI had a prior history of drug allergies and the remaining had no prior history.

DILI was classified as definite or highly probable in 31.1%, probable in 62.5%, and possible in 7.4% of cases. Pattern of liver injury was hepatocellular in 25.1%, cholestatic in 56.17%, and mixed in 18.72% of patients.

The patients with a prior drug allergy were mainly male (78%) and significantly older, with a mean age of 54 versus 45 with no prior drug allergy (P = .009), and they were more likely to have an underlying chronic disease like diabetes and Hypertension (P = .016).

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Clinically, patients with prior drug allergy were more encephalopathic (21.6% vs 10 ;P=0.02) and jaundiced (54.1% vs 38 % p= 0.04) .Those with a prior drug allergy also had significantly hepatocellular pattern of DILI(68% vs 35% ; P= 0.003), lower median values of albumin (2.8 g/L vs 3.8 g/L P= 0.04)and alkaline phosphatase levels versus no drug allergies (1.4 vs 1.6 × ULN; P = .045) and lower platelet counts (189 vs 226 x103/ ml; P = .011), while aspartate aminotransferase (AST) , bilirubin levels and International normalized ratio (INR) showed a trend towards increased levels in patients with prior drug allergy (9.4 vs

Keyword(s) :

2455 - Diabetes association with liver diseases and prognostic risk factors of liver related complications in diabetic patients.

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Background: Emerging evidence highlights a strong bidirectional association between diabetes mellitus and liver diseases. This interplay, mediated by disruptions in glucose and lipid metabolism, contributes to the onset and progression of hepatic disorders from steatosis to cirrhosis. However, data on the prognostic risk factors of liver-related complications among patients with type 2 diabetes mellitus (T2DM) remains limited.

Materials and Methods:

A retrospective study was conducted that identified 8234 adult patients with the T2DM between January 2020 and December 2023. demographic data with clinical characteristics were noted. Patients with T2DM having chronic viral hepatitis, excessive alcohol use, other metabolic and autoimmune liver diseases, malignancies, and liver related events (LRE) before T2DM diagnosis were excluded. The primary endpoint was Liver Related Events, defined as hepatic decompensation as development of ascites , encephalopathy and GI bleed or the development of hepatocellular carcinoma. Uni- and multi-variable Fine-Gray regression model was estimated.

Results:

1,010 patients with T2DM were included (mean age 58.9 years; 41.3% males); the incidence rate of Liver related events was 0.279 (95% CI 1.243-1.315) cases per 500 person-years at risk. Female gender, older age, higher level of HBA1c, hypertension, overweight and noncompliance to medicines were associated with

an increased risk of Liver related events. By multivariable analysis, central obesity and higher body mass index was associated with a higher risk of Liver related events, whereas aspirin, coffee use and insulin use at baseline was associated with a lower risk. Among all covariates, older age explained the most about the incidence of Liver Related Events (36.7%) followed by high body mass index (20.1%).

Conclusions: Older age, female gender, high BMI, high HBA1c levels and non compliance to medicines were risk factors for Liver related events in patients with T2DM. Advanced age was the most important risk factor for Liver related events and high BMI. In conclusion , When your blood sugar is high over a long time, it can damage your liver so it needs special attention to avoid liver related events..

Keyword(s) : Type 2 Diabetes Mellitus , Liver-Related Events , Endo-Hepatology , Prognostic Risk Factors , Metabolic Liver Disease

2456 - Rurality and Neighborhood Socioeconomic Deprivation as Predictors of Hepatocellular Carcinoma Surveillance Adherence Among Adults with Cirrhosis in the United States

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is among the most prevalent and deadliest forms of cancer, and cirrhosis is one of its strongest risk factors. Despite established guidelines recommending semiannual surveillance, adherence rates remain poor. Limited studies have

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evaluated the impacts of neighborhood socioeconomic factors and rurality on surveillance adherence. This study examines the associations between 1) rurality assessed by Rural-Urban Commuting Area (RUCA) codes and 2) neighborhood socioeconomic status measured by the Area Deprivation Index (ADI), with adherence to HCC surveillance among adult patients with cirrhosis.

Materials and Methods:

This retrospective cohort study utilized the Ohio State University PCORnet electronic health record database to identify adults with cirrhosis from 2011 to 2023. Rurality was classified using PCORnet-provided RUCA codes. Neighborhood socioeconomic status was categorized as high deprivation (ADI $\geq$ 75th percentile) or low deprivation (ADI < 75th percentile). HCC surveillance adherence was calculated as the proportion of follow-up time covered (PFTC) by liver imaging (ultrasound, CT, or MRI), with high adherence defined as $\geq 75\%$ coverage. Overlap Propensity Score Weighting (OPSW) was applied to account for confounding. Crude and adjusted modified Poisson regression analyses were fitted using generalized estimating equations (GEEs) with binary outcomes and robust standard errors to account for clustering, and were used to assess the associations between rurality, deprivation, and surveillance adherence.

Results:

Among 11,744 patients with cirrhosis, the mean (STD) age was 55.9 (12.7), 54.5% were male, and 78.9% self-identified as non-Hispanic White. The mean (STD) PFTC was 29.8% (33%) (median 16.2%), and only 15% of patients achieved high adherence. An estimated 31.7% were rural residents, and 44.6% had high neighborhood deprivation.

Rural residence was associated with 26% lower odds of surveillance adherence compared with urban residence (aOR: 0.74; 95% CI: 0.65 to 0.85). Patients living in high-deprivation neighborhoods had 12% lower adjusted odds of adherence than those in low-deprivation neighborhoods (aOR: 0.88; 95% CI: 0.78 to 0.99). Additionally, neighborhood deprivation demonstrated a dose-response relationship with adherence. Each quartile increase in ADI was associated with a 9% lower adjusted odds ratio of adherence (aOR: 0.91; 95% CI: 0.85 to 0.97).

Conclusions: HCC surveillance adherence among adults with cirrhosis remains very poor. Both rurality and high neighborhood deprivation were independently associated with lower adherence, suggesting

socioeconomic and geographic barriers to HCC surveillance. These findings highlight the need for targeted, neighborhood-level interventions to reduce disparities and improve cancer prevention in socioeconomically disadvantaged communities.

Keyword(s) :

2458 - Wilson's Disease in Oman A National Cohort Study of Clinical Spectrum Diagnostic Delay and Long Term Outcomes

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Background / Hypothesis: Wilson's disease (WD) is a rare autosomal recessive disorder of copper metabolism that results in hepatic, neurological, and psychiatric manifestations. Despite being described globally, data from the Middle East remains limited. This study presents the first comprehensive national cohort analysis of WD in Oman, examining clinical features, diagnostic challenges, treatment patterns, and long-term outcomes.

Materials and Methods: A retrospective cohort study was conducted on 36 Omani patients diagnosed with WD between 2013 and 2020 at Sultan Qaboos University Hospital using AASLD diagnostic criteria. Clinical presentation, biochemical parameters, treatment regimens, and progression-free survival were analyzed.

Results: The median age at diagnosis was 14.5 years, with a slight female predominance (55.6%). Clinical presentation varied: 25% had hepatic symptoms, 22.2% had mixed hepatic-neurological features, and 16.7% presented with neurological symptoms alone. Asymptomatic cases identified via family screening accounted for 33.3%. Diagnostic delays were most pronounced among patients presenting with neurological symptoms. A positive family history was reported in 88.9% of cases, suggesting strong familial clustering despite a low rate of consanguinity (5.6%). Regional distribution was concentrated in Ash Sharqiyah North and Muscat. Chelation therapy with trientine or penicillamine, often combined with zinc, was the mainstay of treatment. Treatment adherence was significantly associated with improved

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progression-free survival ($p = 0.012$).

Conclusions: WD in Oman is marked by heterogeneous presentations, frequent diagnostic delays, and strong familial clustering. Early detection through cascade screening and sustained treatment adherence are critical for favorable outcomes. These findings support the need for national screening policies and structured long-term care models for WD in the region.

Keyword(s) :

2459 - Superinfection with Hepatitis Delta Virus Elevates the Risk of Hepatocellular Carcinoma in Individuals with Chronic Hepatitis B Virus Infection

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Background / Hypothesis:

The incidence and mortality of hepatocellular Carcinoma (HCC) continue to rise in the United States, despite declining trends in numerous other cancers. While the hepatitis delta virus (HDV) increases the risk of adverse hepatic manifestations, research on the association between HDV and HCC risk has yielded mixed results. In addition, most research on this topic has focused on chronic Hepatitis B virus (CHBV)/HDV coinfection, with limited studies on HBV-HDV superinfection. We aimed to examine the association between CHBV-HDV superinfection and HCC risk in patients with CHBV.

Materials and Methods:

Using the MarketScan® Commercial Claims databases

from January 1, 2008, to December 31, 2017, we combined claims data from inpatient admissions and outpatient service records to identify patients with CHBV using ICD-9 and 10 codes. HBV-HDV superinfection was defined as a new HDV diagnosis > 90 days after the first date of CHBV diagnosis. HDV status was modeled as a time-varying covariate to account for the effects of the wait time between the date of CHBV diagnosis and the first record of HDV diagnosis. Inverse probability of treatment weighted Cox proportional hazard models were used to assess the adjusted risks of HCC for CHBV mono-infection versus CHBV-HDV superinfection.

Results:

The study included 36,689 adults with CHBV (53.7% males, average age 45 years), of whom 1,633 had CHBV-HDV superinfection. The average time from CHB diagnosis to HBV-HDV superinfection was 26.2 months. During the study period, 201 patients developed HCC. The median follow-up period was 19 months. The risk of HCC was significantly higher in super-infected patients than in mono-infected patients (1.4% vs. 0.5%; $P < 0.001$). The incidence rate of HCC was more than 2.5-fold higher in super-infected patients than in mono-infected patients (6.7 vs. 2.3 per 1000 Person-years %; $P < 0.001$). The adjusted risk of cirrhosis was significantly higher in super-infected patients than in mono-infected patients (hazard ratio (HR), 2.64 (95% CI, 2.11–3.28). In the fully adjusted models, HDV superinfection was associated with a 93% increase in the risk of HCC (HR, 1.93; 95% CI, 1.20–2.97) compared to CHBV mono-infection.

Conclusions:

Our comprehensive analysis of a large commercial claims database suggests a markedly increased risk of HCC in patients with CHBV-HDV superinfection compared to those with CHBV mono-infection. These findings underscore the crucial importance of early diagnosis, surveillance, and targeted intervention strategies in patients with HBV-HDV superinfection.

Keyword(s) :

2462 - Prevalence risk factors and demographic insights of sickle cell hepatopathy in Omani adolescents and adults A retrospective analysis

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Background / Hypothesis:

Sickle cell hepatopathy (SCH) is a significant yet under-researched complication of sickle cell disease (SCD). Despite the substantial prevalence of SCD in Oman and the Gulf region, the burden and characteristics of SCH remain poorly defined. This study aimed to determine the prevalence of SCH and its associated demographic, clinical, and laboratory features among Omani adolescents and adults with SCD.

Materials and Methods:

We conducted a retrospective cross-sectional study at Sultan Qaboos University Hospital. Of 2,257 patients screened, 384 were diagnosed with SCH (prevalence 17%). After applying inclusion and exclusion criteria, 750 patients were analyzed: 384 SCH cases and 366 randomly selected controls without SCH. Demographic, clinical, and laboratory data were reviewed to identify associations.

Results:

SCH was significantly associated with cholelithiasis (56.6% vs. 38.5%, $P = 0.0001$) and exchange transfusions (36.6% vs. 20.8%, $P = 0.0001$). Patients with SCH were more likely to be male (51.1% vs. 37.8%, $P = 0.0001$) and younger (28.9 ± 8.7 vs. 31.0 ± 10.2 years, $P = 0.002$). Markers of hemolysis, including elevated lactate dehydrogenase (522.4 vs. 341.6 U/L, $P < 0.0001$) and higher reticulocyte counts (8.5% vs. 5.4%, $P < 0.0001$), were also associated with SCH. However, no independent predictors were identified in multivariate analysis.

Conclusions:

SCH affects nearly one in six Omani patients with SCD, with associations to younger age, male gender, cholelithiasis, and transfusion history. These findings highlight the importance of proactive screening and personalized management to improve outcomes in this high-risk population.

Keyword(s) : Sickle Cell Hepatopathy (SCH), Sickle Cell Disease (SCD), Prevalence, Risk Factors, Oman

2464 - Beyond Liver Function Albuminuria and a novel Child Turcotte Pugh AlbU score improve mortality prediction in medical intensive care unit patients with hepatic encephalopathy

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Background / Hypothesis:

This study aimed to evaluate whether incorporating albuminuria (Alb) into the Child-Turcotte-Pugh (CTP) score improves the prediction of mortality in patients with hepatic encephalopathy (HE).

Materials and Methods:

This prospective study was conducted between January 2013 and January 2015 at Al-Azhar University, Cairo, Egypt. Patients who were diagnosed with HE at the time of admission to the medical intensive care unit (MICU) were included. Upon admission, patients underwent HE grading, CTP classification, and an albumin-to-creatinine ratio (ACR) assessment. A novel scoring system, CTP-AlbU, was calculated by combining existing CTP scores with the admission degree of ACR. The primary outcome measure was MICU mortality.

Results:

A total of 78 consecutive cirrhotic patients were included in this study with a mean age of 57.65 ± 12.9 years, with equal male and female distribution. HE grades were categorised as 2, 3, and 4 in 16 (20.5%), 44 (56.4%), and 18 (23.1%) patients, respectively. CTP classifications were B and C in 12 (15.4%) and 66 (84.6%) patients, respectively. The overall mortality rate was 32.1%. Statistical analysis revealed a significant association between increased mortality and both elevated ACR and higher CTP-AlbU scores. Furthermore, the CTP-AlbU score emerged as an independent risk factor for mortality.

Conclusions:

This study demonstrates that incorporating albuminuria into the CTP score, resulting in the CTP-AlbU score, enhances the prediction of mortality in HE patients. This novel scoring system displayed superior sensitivity compared to the original CTP score, suggesting its potential as a tool for predicting short-term outcomes in this patient population.

Keyword(s) : Prognosis, Hepatic Encephalopathy, ICU, Albuminuria, Egypt

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2466 - Real World Impact of Cardiovascular Comedications on DDI risk in Patients receiving direct acting antiviral (DAA) Hepatitis C Therapy

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Background / Hypothesis:

The AASLD/IDSA hepatitis C virus (HCV) guidance recommends assessment of comedications prior to initiation of direct-acting antivirals (DAAs). Medication reconciliation, especially for cardiovascular comedications—remains a critical step to mitigate drug-drug interaction (DDI) risks. This study evaluates the DDI risk of cardiovascular comedications in a real-world subset of US patients with HCV, with insights relevant for APAC regions where cardiovascular disease prevalence is rising.

Materials and Methods:

We analyzed the IQVIA PharMetrics Plus claims database (July 2015–June 2023) to assess co-administration of lipid-lowering drugs and antihypertensives with pan-genotypic DAAs (pDAAs; sofosbuvir/velpatasvir [SOF/VEL] and glecaprevir/pibrentasvir [GLE/PIB]). DDI risk was determined using the Liverpool DDI tracker. Propensity score matching (1:1, without replacement) was used to create comparable SOF/VEL and GLE/PIB cohorts.

Results:

Among 11,324 HCV patients on pDAAs, 11.6% received lipid-lowering and 2.5% received antihypertensive comedications. After matching, both cohorts—SOF/VEL and GLE/PIB—each comprised 431 patients receiving lipid-lowering agents and 91 patients receiving antihypertensives, respectively. Among SOF/VEL-treated patients, DDI risk was present in 88.2% of those on lipid-lowering agents and 23.1% of those on antihypertensive comedications. In contrast, DDI risk was present in all GLE/PIB patients with these comedications. For lipid-lowering comedications, no

SOF/VEL patients had contraindicated DDI risk claims, whereas 74.4% of GLE/PIB patients did.

Contraindications with GLE/PIB were predominantly associated with atorvastatin, lovastatin, and simvastatin. For antihypertensives, olmesartan, prazosin, and enalapril were the most frequent comedications posing clinically significant DDI risk.

Conclusions:

Although the number of affected patients is relatively small, DDI risk remains common between DAAs and cardiovascular medications, particularly with GLE/PIB. Careful selection of DAA regimens based on individual DDI profiles is essential for ensuring appropriate HCV treatment.

Keyword(s) : HCV, DDI, DAA

2467 - A case of acute hepatitis C that resolved spontaneously

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Introduction:

Globally, chronic hepatitis C virus infection affects 50 million people, with approximately one million new cases each year. Chronic hepatitis C virus infection becomes chronic in 70% of cases, while acute hepatitis C infection resolves spontaneously within 6 months in 30% of cases.

In chronic hepatitis C virus infection, jaundice occurs in 25% of cases, and gastrointestinal symptoms occur in approximately 20% of cases.

Case:

A 24-year-old female patient presented to our outpatient clinic with complaints of nausea, vomiting, loss of appetite, fatigue, dark urine, and burning sensation during urination. She did not have jaundice. She had no chronic disease. Her history included intravenous drug use in both herself and her husband. Her husband had been diagnosed with hepatitis C virus infection.

The laboratory tests have been compatible with acute hepatitis: AST/ALT 453/855 U/L, GGT 116 U/L, white blood cell count was 9.3/μL, hemoglobin 13.3 g/dL, platelet count was 209×10⁹/L, sedimentation 6 mm/h. The abdominal echography of the abdomen

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showed no biliary obstructions or mass lesions in the liver. There were no findings of liver cirrhosis or fatty liver. The viral serological tests revealed protection for hepatitis A (IgM neg/IgG pos), negative for HVB infection (AgHBs neg, anti-HBc neg), positive antibodies for HCV and positive HCV RNA (4419 UI/ml), HCV genotype 1a.

Since the patient was using medication containing buprenorphine and naloxone for addiction treatment, we monitored her closely without administering additional antiviral drugs.

The patient's fatigue and appetite gradually improved. Liver enzyme levels decreased and HCV RNA became negative without any anti-HCV treatment (eg, administration of interferon or direct-acting antiviral drugs).

She spontaneously cleared HCV infection after 1 month, maintaining negative HCV RNA 3 months after infection. The patient has cured the HCV infection with no need for antiviral treatment.

Discussion

Diagnosing acute hepatitis is difficult. Most cases are asymptomatic. Diagnosis and treatment of acute hepatitis C at an early stage of infection is important for preventing disease progression to the chronic phase. Initiating treatment 4–12 weeks after diagnosis of the HCV infection (ie, during the early stage of infection) is recommended and prevents disease progression. When acute hepatitis C is diagnosed, the patient needs to be closely monitored in the initial period to ensure they do not lose the chance for antiviral treatment.

Keyword(s) :

2468 - Antipsychotic Comedication and Drug Drug Interaction Risk in US Patients Treated with HCV DAAs Focus on PWUD Healthcare Resource Use Impact and AASLD Algorithm Implications

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Background / Hypothesis:

Concomitant use of antipsychotics and direct-acting antivirals (DAAs) in US patients with hepatitis C virus (HCV), including people who use drugs (PWUD) raises concerns about drug-drug interactions (DDIs), healthcare resource use (HCRU), and rapid start of HCV treatment as per the new AASLD/IDSA algorithm.

Materials and Methods:

A retrospective cohort study using IQVIA PharMetrics Plus claims data (2015–2023) identified HCV patients treated with sofosbuvir/velpatasvir (SOF/VEL) or glecaprevir/pibrentasvir (GLE/PIB) and concomitant antipsychotics. A PWUD subgroup and patients receiving opioid substitution therapy (OST) were also analyzed. DDI risk and healthcare resource use was compared between DAA regimens using propensity score matching (1:1, without replacement).

Results:

Overall, 3.7% of HCV patients received concomitant antipsychotic medications; while among PWUD, 42.4% received antipsychotics. 5% of patients received >1 antipsychotic, indicating multi-DDI risk. In the matched cohort, 142 patients were treated with SOF/VEL and 142 with GLE/PIB. DDI risk was present in 13.2% of SOF/VEL patients and 100% of GLE/PIB patients. No significant differences were found in the frequency of HCV-related healthcare visits. Quetiapine was the most frequent concomitant antipsychotic used, followed by aripiprazole and lurasidone.

Conclusions:

Antipsychotic use in US patients with HCV, especially PWUD, is common and linked to significant DDI risk, especially with the use of medications carrying risks of QTc prolongation, such as quetiapine. In this study, DDI risk was present in all GLE/PIB patients with concomitant antipsychotics, whereas fewer SOF/VEL patients had DDI risk present. There were no significant differences in HCV-related HCRU. HCV treatment initiation, including rapid start, requires careful choice of DAA treatment based on individual patient profiles to avoid DDI risk.

Keyword(s) : HCV, DDI, DAA, Antipsychotics

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2469 - Study on Patients Undergoing Triple Therapy for Liver Steatosis in Mongolia

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Abstract

In Mongolia, liver steatosis is highly prevalent, frequently linked to chronic HBV and HCV infections. Triple therapy (pegylated interferon, ribavirin, and protease inhibitor) proves effective. This retrospective study analyzed treatment outcomes, steatosis reduction levels and degrees, viral load changes, correlations with liver cirrhosis, and viral types in 816 patients' records from Happy-Veritas Hospital and the Mongolian National University of Medical Sciences (2025 data). Results revealed 98.6% treatment efficacy in reducing steatosis, viral load reduction in 57.4%, and the highest prevalence of HCV type C (47.3%). The study offers baseline data for assessing triple therapy effectiveness in the Mongolian context.

Objectives

The study aims to retrospectively analyze liver steatosis cases treated with triple therapy in Mongolia and determine:

1. Overall treatment outcomes (steatosis reduced or unchanged).
2. Steatosis reduction levels by groups.
3. Changes in steatosis degree.
4. Viral load status (present or reduced).
5. Correlation between steatosis and liver cirrhosis.
6. Correlation of steatosis with hepatitis virus types (B, C, BD, unknown).
7. Evidence-based recommendations to optimize triple therapy strategies.

Methodology

This retrospective study reviewed medical records of 816 patients with liver steatosis from Happy-Veritas Hospital and the Mongolian National University of Medical Sciences in 2025. Data were extracted from PDF records and classified by treatment outcomes, steatosis reduction levels and degrees, viral load, cirrhosis correlation, and virus types. Actual counts and percentages were calculated. Statistical analysis used Python's pandas library, with graphs generated from real data for visual comparisons.

Results

Data from 816 patients were analyzed and presented graphically.

1. Treatment Outcomes Steatosis decreased in 98.6% of patients.
2. Level of Steatosis Reduction Reduction levels were distributed across groups (0-9, 10-19, etc.), with the majority in the 40-99 group.
3. Degree of Steatosis Reduction The highest reduction degree (3-1) involved 126 patients.
4. Correlation Between Virus and Steatosis HCV type C was most prevalent (47.3%).
5. Correlation Between Cirrhosis and Steatosis Cirrhosis decreased in 77.1% of patients.
6. Correlation Between Liver Steatosis and Hepatitis Virus Steatosis was most associated with HCV type C.

Conclusion

Triple therapy for liver steatosis in Mongolia achieved high outcomes, with 98.6% steatosis reduction. However, viral load persisted at 57.4%, and cirrhosis reduction reached 77.1%. Steatosis predominated in HCV patients, with even reduction levels and degrees indicating treatment efficacy. Limitations include limited sample size and missing data. Future large-scale studies are recommended, emphasizing viral prevention and early detection.

Keyword(s) : NAFLD, Fatty

2470 - Causes of New Hepatitis Virus Infections in Mongolia

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Background / Hypothesis:

Hepatitis B (HBV) and C (HCV) infections are primary drivers of liver cancer and cirrhosis in Mongolia. This study examined 398 newly detected cases (HBV: 154, HCV: 244) to identify transmission causes. Of these, 317 patients (79.6%) identified their infection source,

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while 81 (20.4%) did not. Leading causes included cosmetic procedures (65 cases, 20.5%), tattoos (53, 16.7%), and dental treatment (37, 11.7%), highlighting iatrogenic transmission. HCV cases predominated (244 vs. 154 HBV), with higher susceptibility in cosmetic and tattoo procedures. These 2025 findings underscore the urgent need for enhanced infection control in healthcare settings and increased public awareness to support Mongolia's hepatitis elimination goals.

Introduction

Mongolia has one of the highest global burdens of hepatitis viruses, with HBV prevalence at 5.7–11.1% and HCV at 3.73–8.5%. New infections are predominantly iatrogenic, transmitted via medical, cosmetic, or tattoo procedures, fueling rising liver cancer rates. Identifying transmission routes is critical for meeting the WHO's 2030 elimination targets. This study analyzes infection causes in 398 newly detected cases, including HBV/HCV distribution and cause classification.

Materials and Methods:

Data Source

Data were drawn from 398 newly detected HBV (154) and HCV (244) cases recorded in Sheet1 of "hepatitis virus infection.xls" as of December 2025. Cases were categorized by whether patients knew their infection cause (317 known, 81 unknown), with known causes grouped into 10 categories.

Results:

General Overview of Newly Detected Cases

HCV cases outnumbered HBV by 1.58 times (244 vs. 154), reflecting higher HCV incidence. Notably, 79.6% of patients identified their infection source, indicating effective epidemiological tracing.

Identified Causes of Infection

Iatrogenic routes—cosmetic procedures (20.5%), tattoos (16.7%), and dental treatment (11.7%)—accounted for 49% of known causes. HCV was more associated with cosmetic procedures (39 vs. 26 HBV) and tattoos (32 vs. 21 HBV). Familial transmission was slightly higher in HBV (12% vs. 10.9% in HCV). The majority (79.6%) of new infections had traceable causes, predominantly iatrogenic, linked to inadequate sterilization and weak regulation in cosmetic and tattoo services. Unknown causes comprised 20.4%, suggesting room for improved tracing.

Conclusions:

New hepatitis infections in Mongolia are mainly iatrogenic, driven by cosmetic procedures, tattoos, and dental treatments. HCV predominates over HBV, necessitating stricter oversight in medical and non-medical procedures. Enhanced sterilization, public education, and the "Hepatitis-Free Mongolia" initiative are vital. Limitations include small sample size and absence of demographic data (age, gender). Future research should involve larger cohorts and molecular analyses for deeper insights.

Keyword(s) :

2471 - Comparative Study of Liver Indicators in Patients with and without Diabetes Mellitus

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Background / Hypothesis:

In Mongolia, diabetes mellitus (DM) is potentially linked to liver conditions like steatosis and fibrosis. This retrospective study compared liver indicators (ALT, AST, fibrosis, steatosis) between diabetic (n=14) and non-diabetic (n=14) groups, computing averages and Pearson's correlation coefficients using November 2025 data from Happy-Veritas Hospital. Diabetic group averages: ALT 69.81, AST 54.97, fibrosis 11.05, steatosis 300.19. Non-diabetic: ALT 49.12, AST 63.33, fibrosis 9.04, steatosis 262.80. Correlation analysis revealed a strong ALT-AST link (0.98) in the non-diabetic group. These findings establish baseline evidence for exploring DM's impact on liver health.

Materials and Methods:

Retrospective analysis of 28 patients (14 diabetic, 14 non-diabetic) from Happy-Veritas Hospital's November 2025 Excel registry. Groups were stratified by DM status. Averages were calculated via Python's pandas (.mean()) and numpy libraries; correlations used .corr() for Pearson's coefficients, displayed in tables.

Results:

Analysis of 28 patients yielded the following:

Average Indicators



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Diabetic group (n=14): ALT 69.81, AST 54.97, fibrosis 11.05, steatosis 300.19.

Non-diabetic group (n=14): ALT 49.12, AST 63.33, fibrosis 9.04, steatosis 262.80.

Conclusion: Higher ALT and steatosis in diabetic group; higher AST and fibrosis in non-diabetic.

Correlation Analysis

Diabetic Group (Pearson's coefficients):

ALT-AST: 0.17 (weak positive).

ALT-fibrosis: 0.46 (moderate positive).

ALT-steatosis: 0.06 (very weak positive).

AST-fibrosis: -0.14 (weak negative).

AST-steatosis: -0.41 (moderate negative).

Fibrosis-steatosis: 0.09 (very weak positive).

Conclusion: Moderate ALT-fibrosis positive; moderate

AST-steatosis negative.

Non-Diabetic Group:

ALT-AST: 0.98 (very strong positive).

ALT-fibrosis: 0.57 (moderate positive).

ALT-steatosis: 0.35 (weak-moderate positive).

AST-fibrosis: 0.59 (moderate positive).

AST-steatosis: 0.40 (moderate positive).

Fibrosis-steatosis: 0.17 (weak positive).

Conclusion: Very strong ALT-AST correlation; moderate positives elsewhere.

Conclusions:

Liver indicators in diabetic patients showed elevated ALT and steatosis, while non-diabetics had higher AST.

Correlations were stronger (e.g., ALT-AST 0.98) in non-diabetics, implying diabetes disrupts indicator

interrelations. Limitations: Small sample (n=28), inter-patient variability (age $\pm$ 5 years, gender differences).

Larger studies are essential for deeper DM-liver disease insights.

Keyword(s) :

2472 - Correlation Between the Degree of Liver Steatosis and Liver Enzyme (ALT AST GGT) Levels

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Background / Hypothesis:

Liver steatosis (NAFLD/NASH) has emerged as a major health concern globally and in Mongolia driven by lifestyle changes, obesity, diabetes, and metabolic

syndrome. Early stages are often asymptomatic, with diagnosis relying on costly or limited imaging (ultrasound, CT/MRI). This highlights the need to evaluate affordable blood markers—ALT, AST, and GGT—for early detection and their correlation with steatosis severity. Clinical studies on this relationship in Mongolia are scarce, making this analysis of Happy Veritas Clinic patients a pioneering contribution.

Objective

To investigate the correlation between the degree of liver steatosis and blood levels of ALT, AST, and GGT enzymes.

Materials and Methods:

A retrospective cross-sectional study included 103 outpatients who underwent laboratory tests at Happy Veritas Liver Center in September 2025. Inclusion criteria: age $\geq$ 18 years and complete ALT, AST, GGT, UAP (steatosis), and Stiffness (fibrosis) measurements. All statistical analyses were performed using Excel.

Results:

General Characteristics

Of 103 participants: females 63 (61.2%), males 40 (38.8%). Age range 18–86 years, predominantly 40–60 years.

Liver Enzyme Levels

ALT elevation was more frequent than AST. GGT was markedly higher in severe steatosis cases. Enzyme changes paralleled increases in UAP and Stiffness.

Correlation with Steatosis (UAP)

Higher UAP (dB/m) values were associated with rising ALT (significant trend), milder AST increase, and sharp GGT elevation, indicating enzyme sensitivity to steatosis severity.

Correlation with Fibrosis (Stiffness)

Increased Stiffness (kPa) correlated with greater GGT rise and mild AST/ALT elevations, identifying potential high-risk fibrosis groups.

Age-Related Patterns

Lowest steatosis/enzyme elevation in 18–40 years; peak in 41–60 years; pronounced Stiffness and GGT in >60 years, confirming higher risk after middle age.

Conclusions:

As steatosis degree (UAP) increased, ALT and GGT showed statistically significant elevations, with GGT emerging as a sensitive marker. High-steatosis groups exhibited prominent AST, ALT, and GGT rises. Elevated

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enzymes in high-Stiffness cases signal fibrosis risk. The 40–60 age group faces the highest risk, warranting targeted prevention and monitoring.

Keyword(s) : Liver steatosis, enzymes, liver fibrosis

2473 - Correlation Between Blood Triglyceride and Cholesterol Levels in Individuals with Liver Steatosis

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is linked to modern lifestyles and dyslipidemia. This study examined correlations between triglyceride (TG) and total cholesterol (T-CHO) levels in 130 Mongolian patients (after outlier exclusion). Pearson's correlation between TG and T-CHO was 0.678 ($p < 0.001$), indicating moderately strong positive association. Steatosis index (K-Steatosis) correlated highly with TG (0.858) and T-CHO (0.829). Results suggest that worsening lipid metabolism accompanies steatosis progression, supporting integrated lipid monitoring for NAFLD management in Mongolia.

Liver steatosis involves fat accumulation in $>5\%$ of hepatocytes and is associated with obesity, insulin resistance, and dyslipidemia. In Mongolia, rising obesity drives increasing NAFLD prevalence. Blood TG and T-CHO are key markers, and their interrelationship aids disease prediction. This study analyzes correlations between TG and T-CHO using data from 131 patients (130 after excluding one outlier), including age, gender, TG, T-CHO, liver stiffness (TP-Stiffness), steatosis (K-Steatosis), and BMI.

Materials and Methods:

Data Source

Clinical results from 130 patients were analyzed.

Statistical Analysis

Processed with Python's pandas library. Descriptive statistics (mean, SD, min/max) were calculated.

Pearson's correlation coefficients were computed for TG, T-CHO, steatosis, and other indicators. Correlations >0.5 were deemed strong.

Results:

General Characteristics

Average age: 50.52 years (SD 12.56, range 20–85).
TG: 1.30 mmol/L (SD 0.61, range 0.13–2.56).
T-CHO: 4.89 mmol/L (SD 1.26, range 2.61–10.88).
TP-Stiffness: 70.40 (SD 5.04, range 53.99–78.26).
K-Steatosis: 279.96 (SD 52.03, range 173.69–391.33).
BMI: 28.81 (SD 5.09, range 17.84–44.00).
Gender: 60% male, 40% female. Average BMI 28.8 indicates early obesity.
Correlation Analysis
TG vs. T-CHO: 0.678 (moderately strong positive).
K-Steatosis vs. TG: 0.858 (high).
K-Steatosis vs. T-CHO: 0.829 (high).
Age vs. TG: 0.821; Age vs. T-CHO: 0.806.
BMI vs. TG: 0.825; BMI vs. T-CHO: 0.826.
Higher steatosis was strongly linked to elevated lipids.

Conclusions:

A moderately strong correlation (0.678) exists between TG and T-CHO in liver steatosis patients. Lipid testing should be prioritized in NAFLD management. Future studies require larger samples and standardized steatosis indices.

Keyword(s) :

2474 - Determining Risk by Glucose and Triglyceride Ratio in Individuals with Liver Steatosis

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Background / Hypothesis:

Rationale

Liver steatosis occurs when fat exceeds 5-10% of liver weight, primarily from alcoholic or metabolic causes. Excessive alcohol disrupts liver metabolism, while metabolic factors—diabetes, hypertriglyceridemia, obesity, and sedentary lifestyle—promote fat accumulation. Elevated glucose reduces liver glucose processing, increases fat synthesis, and leads to triglyceride buildup. Hypertriglyceridemia (>1.7 mmol/L) raises steatosis risk 2-3 times (Journal of Hepatology, 2022), often accompanied by decreased HDL and increased LDL. Studies show 70-90% of steatosis patients have glucose issues, with high type 2 diabetes prevalence. As a chronic condition, steatosis

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progresses through inflammation to fibrosis, cirrhosis, and potentially liver cancer, driven mainly by elevated glucose and triglycerides.

Materials and Methods:

From January 2024 to September 2025, among 3736 outpatients tested at Happy Veritas Hospital Liver Center, data from 71 patients with elevated glucose and triglycerides were selected. The dataset included age, gender, and key laboratory indicators.

Demographic and test results were compiled for analysis.

Results:

Average liver steatosis indicators increased consistently with age. Groups aged 56-60, 71-75, and 76-80 exceeded 300, reflecting age-related metabolic slowdown and impaired fat processing.

Among 7 individuals with diabetes but normal triglycerides, average steatosis was 299, indicating high glucose alone poses significant risk (equivalent to grade 3 steatosis).

In 71 individuals with both elevated glucose and triglycerides, average steatosis was 297 (grade 2-3), showing comparable risk when both factors are raised.

Conclusions:

Steatosis levels rise steadily with age, exceeding 300 in those over 56, underscoring the need for strict glucose and triglyceride control in older adults. High glucose maintains elevated steatosis even with normal triglycerides, confirming that hyperglycemia independently drives substantial risk. Thus, both elevated glucose and triglycerides are major contributors to liver steatosis.

Keyword(s) :

2475 - Correlation Between Liver Fat Accumulation Level and Body Mass Index

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Background / Hypothesis:

In recent years, hepatic steatosis has increased globally, affecting not only overweight/obese individuals but also those with normal BMI—a condition known as "lean fatty liver." It carries similar risks, including inflammation, cirrhosis, and metabolic disorders. In Mongolia, lifestyle changes (reduced activity, high-carbohydrate/fat diets, stress, sleep deprivation) promote steatosis even in normal-BMI populations, often with high visceral fat. Misconceptions delay diagnosis and treatment. Studying causes, risks, diagnosis, and lifestyle impacts in normal-BMI individuals is crucial for early detection, prevention, and improved health services.

Materials and Methods:

A retrospective cross-sectional study included 300 randomly selected patients (aged 20–90 years) with normal BMI and diagnosed steatosis, visiting Happy Veritas Hospital Liver Center outpatient department. Data were collected via a standard questionnaire. The study was approved by the hospital's Ethics Committee (Protocol No. H5652); all personal information was anonymized for statistical use only.

Questionnaire items:

Diabetes diagnosis?
Pancreatic pain/issues?
Gallbladder disease?
High blood cholesterol?
Occupation (current/previous)?
Sleep quality?
Flour-based product consumption?
Regular medications?
Daily exercise/physical activity?
Alcohol consumption?

Results:

Of 300 participants with normal BMI: 58% female (173), 42% male (127). Age distribution: 20–29 (9), 30–39 (26), 40–49 (63), 50–59 (90), 60–69 (75), 70–90 (37).

Key findings:

Diabetes: 12% yes (36), 88% no (264).
Pancreatic issues: 40% yes (120), 60% no (180).
Gallbladder disease: 33% yes (99), 67% no (201).
High cholesterol: 29% yes (87), 24% normal (72), 47% unknown (141).
Good sleep: 39% (117), poor: 61% (183).
Regular exercise: 53% yes (159), 47% no (141).
High flour-based consumption: 53% (159), moderate 24% (72), low 23% (69).

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Chemical exposure at work: 6% (18).
Alcohol consumption: 49% yes (147), 51% no (153).
Regular medications: 58% yes (174), 42% no (126).

Conclusions:

Liver fat accumulation occurs in normal-BMI individuals, not just the overweight. Primary risk factors include high flour-based product consumption, elevated blood cholesterol, poor sleep quality, and regular medication use. These disrupt fat metabolism, promoting steatosis. Targeted interventions addressing these factors are essential for prevention and management.

Keyword(s) :

2476 - The Impact of Changes in Liver Enzymes and Body Mass Index on Liver Fibrosis

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Background / Hypothesis:

Rationale

Liver cirrhosis involves gradual replacement of healthy liver cells with connective tissue, leading to structural loss and impaired function. Often developing over years from inflammation or alcohol-related damage, it is largely irreversible but early detection prevents complications. The liver filters toxins, aids digestion, stores nutrients, and manages inflammation. Repeated damage triggers regeneration that forms scar tissue, eventually disrupting function. Obesity and diabetes exacerbate fatty liver disease, a cirrhosis precursor. ALT and AST are key markers of liver cell damage; elevated levels signal injury from viral hepatitis, alcohol, metabolic syndrome, or steatosis. In Mongolia, persistent high alcohol use, metabolic disorders, and viral infections necessitate integrated evaluation of enzyme changes and BMI. This study clarifies their relationship, aiding early cirrhosis detection, risk stratification, prevention, and management. Gender differences are notable: working-age male mortality is 2.8 times higher, linked to health attitudes, alcohol/tobacco use, inactivity, and poor diet.

Materials and Methods:

From 3000 patients tested at Happy Venus Liver

Center (January–September 2025), 212 with repeated AST/ALT measurements and BMI data were selected for comparison.

Results:

Of 212 participants: 84 men (39.6%), 128 women (60.3%). Age predominantly middle/older (40–69 years: ~68%).

By enzyme levels:

Men: Normal enzymes → 5% cirrhosis, 70% fibrosis, 25% healthy; Elevated → 34.1% cirrhosis, 50% fibrosis, 15.9% healthy.

Women: Normal → 4.6% cirrhosis, 61.5% fibrosis, 33.9% healthy; Elevated → 26.9% cirrhosis, 66.6% fibrosis, 6.5% healthy.

Elevated enzymes markedly increase fibrosis/cirrhosis risk in both genders.

By BMI category:

Men: Normal weight → 12.5% cirrhosis; Overweight → 19.5%; Obese → 16.7%.

Women: Normal → 12.7% cirrhosis; Overweight → 9%; Obese → 26.3%.

Higher BMI elevates morphological change risk, especially fibrosis.

Conclusions:

Conclusion

BMI increase impacts women more strongly (obese women: 26.3% cirrhosis vs. 12.5% normal weight; men: 18.7% obese vs. 14.2% normal).

Elevated enzymes (ALT/AST >80 U/L) show strongest correlation, with 30.4% cirrhosis vs. 9.8% normal (3-fold risk, $p < 0.01$).

Men face 1.3 times higher cirrhosis risk with elevated enzymes than women; BMI is a secondary factor.

Keyword(s) :

2478 - Association of MAFLD with joint lifestyle exposures Background & aims

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Background / Hypothesis:



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Metabolically-dysfunction-associated fatty liver disease (MAFLD) is characterized by hepatic steatosis in the setting of overweight/obesity, type 2 diabetes, and/or metabolic dysregulation. MAFLD may co-exist with a broad range of comorbid liver and cardiometabolic conditions. High-calorie consumption and decreased physical exercise are key lifestyle variables that contribute to the occurrence of MAFLD. So far, single lifestyle exposures associated with MAFLD have been studied, like Exercise and nutrition, which have been demonstrated to be helpful lifestyle therapies for MAFLD prevention and treatment when sufficient weight reduction is accomplished. However, the effect of joint lifestyle exposures remains unclear, hindering the development of targeted prevention and control strategies. We aimed to investigate the joint associations between lifestyle exposomes and MAFLD.

Materials and Methods: This study included 1,002 participants from the outpatient clinic of the Gastroenterology department. Lifestyle exposomes, including metabolic equivalent of task (MET), sleep duration, smoking exposure and Alternative Healthy Eating Index (AHEI) score, were identified from questionnaire data. Non-invasive Liver disease Assessment was used to diagnose MAFLD, including Vibration-controlled transient elastography measurements and laboratory data like FIB-4 and APRI score. A logistic regression model and the weighted quantile sum method were used to evaluate the associations of single and joint lifestyle exposomes, respectively, with MAFLD. To assess the population benefits of different intervention strategies, the population attributable fractions (PAFs) were calculated.

Results: Per-quartile increases in sleep duration (OR = 0.783, 95% CI: 0.526–0.944), MET (OR = 0.816, 95% CI: 0.771–0.963), and AHEI score (OR = 0.827, 95% CI: 0.756–0.904) were all significantly associated with reduced risk of MAFLD. When these three factors were considered together, their combined exposure showed a stronger association with MAFLD risk reduction (OR = 0.772, 95% CI: 0.688–0.865), with MET contributing the greatest weight (0.526) among them. Population attributable fraction (PAF) analysis indicated that interventions focused on enhancing MET proved to be the most effective strategy compared to improving sleep and dietary quality (AHEI), which would yield

greater benefits in the population at risk of MAFLD.

Conclusions: This study revealed significant associations between MAFLD and lifestyle exposures to sleep duration, MET, and AHEI, exploring physical activity as the most important

Keyword(s) : Association of MAFLD with joint lifestyle exposures Background & aims

2479 - Epidemiological Clinical and Paraclinical Profile of Decompensated Liver Cirrhosis at Khmer Soviet Friendship Hospital a Tertiary Hospital in Cambodia

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Background / Hypothesis:

Decompensated liver cirrhosis is an advanced stage of chronic liver disease characterized by severe complications such as ascites, variceal hemorrhage, hepatic encephalopathy, and hepatocellular carcinoma (HCC). It is a major cause of morbidity and mortality, particularly in low- and middle-income countries. In Cambodia, hepatitis B virus (HBV), hepatitis C virus (HCV), and chronic alcohol consumption are the principal etiological factors. Despite the significant burden of disease, local data describing the clinical and paraclinical profile of decompensated cirrhosis remain limited. Our objectives are to describe the epidemiological, clinical, and paraclinical characteristics of patients with decompensated liver cirrhosis admitted to the Gastroenterology Department of Khmer-Soviet Friendship Hospital between January 1, 2018, and December 31, 2019.

Materials and Methods:

This retrospective, cross-sectional, single-center, hospital-based descriptive study reviewed medical records of 504 patients diagnosed with decompensated liver cirrhosis over a two-year period. Collected data included demographic characteristics, clinical manifestations, laboratory findings, imaging and endoscopic results, Child-Turcotte-Pugh (CTP) classification, etiologies of cirrhosis, and cirrhosis-related complications.

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Results:

Of the 504 patients, 304 (60.3%) were male and 200 (39.7%) were female, yielding a male-to-female ratio of 1.52:1. The mean age was 56.3 ± 10.9 years (range: 19–93 years), with the highest prevalence in the 50–59-year age group. Most patients (64.6%) originated from rural areas outside Phnom Penh.

The most common presenting symptoms were abdominal pain (39.7%), abdominal distension (29.6%), and upper gastrointestinal bleeding (22.1%). Jaundice was observed in 14.3% of patients, and hepatic encephalopathy in 4%. Laboratory abnormalities included anemia (67.2%), thrombocytopenia (55.1%), elevated AST (83.5%) and ALT (65.6%), hypoalbuminemia (41.5%), hyperbilirubinemia (50.8%), and prolonged prothrombin time (36.7%).

According to the Child-Pugh classification, 40.1% of patients were classified as Child-Pugh B, 31.4% as Child-Pugh C, and 28.5% as Child-Pugh A. Ultrasonography showed a dysmorphic liver in 73.5% and ascites in 65.6%. Endoscopy revealed esophageal varices in 48% and portal hypertensive gastropathy in 32.1%. Alcohol-related liver disease was the leading etiology (41.1%), followed by HBV (34%) and HCV (14.2%). The most frequent complications were ascites (35.8%), upper gastrointestinal bleeding (22.1%), and HCC (19.4%).

Conclusions:

Decompensated liver cirrhosis remains a major clinical and public health challenge in Cambodia, predominantly affecting middle-aged men. Alcohol consumption and HBV infection are the leading causes. The high prevalence of advanced liver dysfunction and serious complications highlights the need for earlier diagnosis, improved access to antiviral therapy, effective alcohol cessation programs, and multidisciplinary management.

Keyword(s) : Decompensated liver cirrhosis; Child-Pugh classification; ascites; hepatocellular carcinoma; upper gastrointestinal bleeding; esophageal varices; hepatitis B virus; alcohol-related liver disease; Cambodia; retrospective study; clinical profile; paraclinical findings; cirrhosis complications.

2482 - Comparative Efficacy and Safety of Second Line and Later Systemic Therapies After First Line Treatment Failure in Hepatocellular Carcinoma A Systematic Review and Meta Analysis

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Introduction: Establishing optimal second-line and later systemic treatment strategies after failure of first-line therapy has become an increasingly important challenge in hepatocellular carcinoma (HCC). However, evidence guiding treatment selection remains limited because randomized controlled trials and direct head-to-head comparisons between agents are scarce.

Methods: We conducted a systematic review and meta-analysis of published studies evaluating second-line or later systemic therapies for HCC, excluding studies limited to sorafenib failure alone. Overall survival (OS), progression-free survival (PFS), objective response rate (ORR), disease control rate (DCR), and grade 3–4 adverse events (AEs) were pooled using meta-analytic methods.

Results: In the meta-analysis of second-line and later systemic therapies after failure of atezolizumab plus bevacizumab, lenvatinib demonstrated the most favorable survival outcomes. The pooled median OS was 12.42 months for lenvatinib (95% CI, 10.02–14.82; $I^2=89.4\%$), compared with 7.83 months for sorafenib (95% CI, 6.29–9.36; $I^2=7.36\%$). Regorafenib, cabozantinib, and ramucirumab showed pooled median OS of 9.19 months (95% CI, 3.60–14.78; $I^2=94.5\%$), 9.59 months (95% CI, 4.91–14.27; $I^2=91.4\%$), and 5.85 months (95% CI, 1.76–9.94; $I^2=20.6\%$), respectively. Compared with sorafenib, lenvatinib significantly reduced the risk of death (risk ratio [RR], 0.64; 95% CI, 0.49–0.84; $I^2=34.2\%$), whereas regorafenib and cabozantinib did not show significant benefit. The pooled median PFS was also longest with lenvatinib at 4.74 months (95% CI, 3.76–5.72; $I^2=96.4\%$), followed by cabozantinib (3.96 months), regorafenib (3.05 months), and sorafenib (2.86 months). Lenvatinib significantly reduced the risk of disease progression compared with sorafenib (RR, 0.58; 95% CI, 0.42–0.80; $I^2=65.3\%$). Reconstructed survival curves confirmed longer median OS (11.87 vs. 8.30 months) and PFS (3.80 vs. 1.93 months) with lenvatinib compared with sorafenib. The pooled ORR was highest with lenvatinib at 13.0% (95% CI, 9.1–18.1; $I^2=51.0\%$), whereas sorafenib showed the lowest ORR (4.7%). DCRs were 62.4% for lenvatinib and

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43.4% for sorafenib. The incidence of grade 3–4 AEs was comparable between lenvatinib (36%) and sorafenib (34%). After lenvatinib failure, atezolizumab plus bevacizumab achieved a pooled median OS of 14.98 months (95% CI, 9.57–20.38; $I^2=42\%$) and PFS of 5.78 months (95% CI, 3.19–8.37; $I^2=66\%$), along with the highest ORR (28.3%) and DCR (69.6%), although with a higher incidence of grade 3–4 AEs (53%).

Conclusion: Lenvatinib demonstrates clinical benefit after failure of atezolizumab plus bevacizumab, while multiple systemic therapies, including atezolizumab plus bevacizumab, may provide meaningful efficacy after lenvatinib failure.

Keyword(s) :

2485 - Hepatic Dysfunction in Critically Ill Children Prognostic Value of PRISM III and PELOD 2 Scores in the Pediatric Intensive Care Unit

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Background / Hypothesis:

Hepatic dysfunction is a common condition in critically ill children and is most often a secondary manifestation of systemic diseases such as sepsis and multiple organ failure. However, its prognostic significance in the pediatric intensive care unit (PICU) has not been fully elucidated. This study aimed to investigate the prevalence of hepatic dysfunction in children admitted to the PICU and to evaluate the association of PRISM III and PELOD-2 scores with prognosis.

Materials and Methods: Patients hospitalized in the PICU who were diagnosed with hepatic dysfunction were included in the study. Demographic, clinical, and laboratory data were retrospectively obtained from hospital records. Patients were classified as having primary or secondary hepatic dysfunction. Organ dysfunction was assessed using the Pediatric Risk of Mortality Score (PRISM III) and the Pediatric Logistic Organ Dysfunction Score (PELOD-2) on both the first day of admission and day 7. Associations with mortality and length of stay (LOS) in the intensive care unit were analyzed.

Results: During the study period, hepatic dysfunction

was identified in 50 (8.8%) of 567 patients admitted to the PICU. The overall mortality rate was 22%. Both PRISM III and PELOD-2 scores were significantly higher in patients who died compared with survivors on day 1 (PRISM III: 19.9 ± 9.6 vs. 7.7 ± 8.8 , $p = 0.001$; PELOD-2: 28.3 ± 17.5 vs. 7.1 ± 8.6 , $p < 0.001$) and on day 7 (PRISM III: 19.5 ± 10.4 vs. 7.7 ± 8.0 , $p = 0.013$; PELOD-2: 29.7 ± 11.6 vs. 9.1 ± 8.9 , $p = 0.001$). Sepsis ($p = 0.042$), mechanical ventilation ($p = 0.004$), and inotropic support ($p < 0.001$) were significantly associated with mortality. Thresholds of ALT $\geq 2 \times$ the upper limit of normal (ULN) and total bilirubin > 2 mg/dL were not associated with mortality ($p > 0.05$). Prolonged length of stay (> 7 days) was associated with higher PRISM III scores, as well as sepsis, mechanical ventilation, inotropic support, blood product transfusion, and antibiotic use.

Conclusions: Hepatic dysfunction is common in critically ill children and is closely associated with adverse outcomes. PRISM III and particularly PELOD-2 scores are reliable predictors of mortality. Sepsis, mechanical ventilation, and inotropic support are major risk factors for mortality, whereas conventional biochemical thresholds such as ALT and bilirubin have limited prognostic value. Early recognition of hepatic dysfunction and regular monitoring using organ dysfunction scores in the PICU may contribute to improved risk stratification and optimization of patient management.

Keyword(s) : Hepatic dysfunction; pediatric intensive care unit; PRISM III; PELOD-2; mortality; sepsis

2486 - Targeting a Cytosolic Nucleotide Pyrophosphatase Suppresses Malignant Phenotypes of Hepatocellular Carcinoma

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) has a poor prognosis due to late diagnosis, high recurrence, and metastasis.

Materials and Methods:

We investigated a cytosolic nucleotide pyrophosphatase that sanitizes the intracellular dNTP pool by hydrolyzing abnormal deoxycytidine triphosphates, preventing their incorporation into DNA.



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Results:

Public immunohistochemistry data and qRT-PCR in 132 paired tissues showed marked overexpression of this enzyme in HCC, which was further validated by Western blot. In HCC cell lines, siRNA-mediated knockdown significantly inhibited proliferation, clonogenicity, migration, and invasion. Mechanistically, enzyme suppression accelerated mRNA decay and downregulated proteins associated with epithelial-mesenchymal transition (EMT; N-cadherin, Snail), stemness (Nanog, Oct-4A, CD133), proliferation (PCNA), and angiogenesis (VEGF, HIF-1 α), while increasing epithelial markers (Occludin, ZO-1, Claudin-1).

Conclusions:

These findings indicate that nucleotide-metabolizing enzyme sustains EMT, stemness, and angiogenesis to promote malignant progression, and that its targeted inhibition effectively attenuates aggressive tumor phenotypes, nominating it as a promising molecular target for HCC therapeutics.

Keyword(s) : hepatocellular carcinoma, EMT, stemness, therapeutic target

2487 - Clinical and Laboratory Profile of Ascites in Decompensated Liver Cirrhosis and Prevalence of Spontaneous Bacterial Peritonitis A Retrospective Study at Khmer Soviet Friendship Hospital

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Background / Hypothesis:

Spontaneous bacterial peritonitis (SBP) is an infection of ascitic fluid (AF) in the absence of an obvious surgically treatable intra-abdominal source. Diagnosis relies on an AF polymorphonuclear leukocyte (PMN) count ≥ 250 cells/mm³ and/or a positive AF culture. Early recognition and prompt management are critical to reduce morbidity and mortality. This study aimed to determine the prevalence of SBP among patients with cirrhotic ascites and to identify the most significant clinical and paraclinical features associated with SBP to facilitate early diagnosis.

Materials and Methods:

A retrospective, analytical, single-center study was conducted at the Gastroenterology and Hepatology Department of Khmer-Soviet Friendship Hospital. Medical records of patients with cirrhotic ascites admitted between January 1, 2019, and December 30,

2021, were reviewed. Data on demographics, cirrhosis etiology, clinical features, laboratory findings, ascitic fluid analysis, and culture results were collected and analyzed.

Results:

Among 315 patients with cirrhotic ascites, 55 cases (17.5%) were diagnosed with SBP. The cohort included 34 males (61.8%) and 21 females (38.2%), with a male-to-female ratio of 1.61:1. The mean age was 54.06 ± 11.69 years. The leading causes of cirrhosis were hepatitis B virus (34.3%), alcohol-related liver disease (32.4%), and hepatitis C virus (18.1%). Clinical and paraclinical factors significantly associated with SBP included fever, abdominal pain, jaundice, advanced liver disease (Child-Pugh class C), low ascitic protein concentration (< 16 g/L), cloudy ascitic fluid, and elevated serum bilirubin (> 4.45 mg/dL). Routine AF analysis and culture were performed in all cases. Among SBP patients, only nine cultures (16.36%) were positive, identifying *Escherichia coli* and *Klebsiella pneumoniae* as causative pathogens.

Conclusions:

SBP remains a significant complication among patients with cirrhotic ascites, predominantly affecting men. Key associated factors include fever, abdominal pain, jaundice, advanced liver disease, and cloudy ascitic fluid. Despite low culture positivity, diagnostic paracentesis with PMN count remains a reliable, simple, and essential test and should be performed immediately upon admission—preferably before initiating empiric antibiotic therapy—for all patients with decompensated cirrhosis and ascites, particularly when associated clinical or laboratory indicators are present.

Keyword(s) : Spontaneous Bacterial Peritonitis, Cirrhotic Ascites, Diagnostic Paracentesis, Liver Cirrhosis, Ascitic Fluid Infection

2489 - HEPATOCELLULAR CANCER RISK IN CHRONIC HEPATITIS D AND CHRONIC HEPATITIS B PATIENTS WITH EFFICIENT TREATMENT A PROPENSITY WEIGHTED ANALYSIS

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Background / Hypothesis: Recent meta-analyses report higher HCC risk in chronic hepatitis D (CHD) vs chronic hepatitis B (CHB). This study aims to compare well-defined CHD and CHB cohorts with respect to HCC development.

Materials and Methods: Patients receiving treatment for CHB and CHD with nucleos(t)ide analogs (NAs) and interferon alfa (IFN), respectively, were extracted from our databases. 124 CHD patients (71% male, mean age 40.3 years, 45 [36.3%] cirrhotic) received IFN and 238 CHB patients (69% male, mean age 48.5, 65 cirrhotic at baseline) received tenofovir [n:163] or entecavir (n:75)]. Forty out of 124 CHD patients had a maintained virological response (MVR), defined as undetectable HDV RNA for 2 years post-treatment. CHB patients were older, had higher platelets, but lower ALT and GGT, compared to CHD ($p < 0.05$ for all comparisons). Median follow-up (FU) was 111 and 115 months for CHB and CHD, respectively. Cumulative HCC development probabilities were estimated by the Kaplan-Meier method and compared with the log-rank test. Propensity score weighting combined with Cox proportional hazards model was used to reduce selection bias risk.

Results: Sixty-five (27%) CHD and 45 CHB (36%) patients had compensated cirrhosis ($p = 0.4$). HCC developed in 21 (8.8%) CHB and 21 (16.9%) CHD patients. Cumulative HCC development probability was higher with CHD compared to CHB ($p = 0.046$). HCC incidence rates were 0.4%, 3.8%; 7.2%, 8.2%, 10.0% and 10.0% and 0.8%, 4.9%, 9.3%, 11.2%, 18% and 23.2% at 12, 36, 60, 84, 120, and 144 months of FU for CHB and CHD patients, respectively. Yearly incidence rates were 1.28% and 1.92% within 60 months and 0.76% and 1.99% after 60 months of FU for CHB and CHD, respectively ($p = 0.04$). CHB, compared to CHD patients with an MVR, did not display a cumulative HCC difference (8.8% vs 10%). After propensity score weighting CHD displayed a strong trend for an increased HCC risk with a HR of 1.86 (95% CI: 0.98-3.52, $p = 0.056$). When CHD patients responding to IFN treatment were compared to CHB patients (responding to NAs), CHD no longer displayed an increased risk

($p = 0.761$).

Conclusions: This study provides evidence that HDV per se may not be an additional risk factor for HCC development, compared to CHB mono-infection. The reported increased risk in CHD may be linked to fibrosis progression and rapidly developing cirrhosis facilitated by the lack of an effective treatment.

Keyword(s): Chronic Hepatitis D, Hepatocellular Carcinoma, Chronic Hepatitis B, Interferon Therapy, Cirrhosis

2490 - A Rare Case of Zieve's Syndrome in a cirrhotic patient Hemolytic Anemia Associated with Alcoholic Liver Disease and Hyperlipidemia

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Background:

Zieve's syndrome is a rarely reported disease resulting from alcohol abuse, characterized by hemolytic anemia, cholestatic jaundice, and transient hyperlipidemia. It is largely under-recognized and under-reported, possibly due to a lack of awareness of the condition among physicians. However, due to the prevalent and persistent excessive alcohol consumption, it is believed that the actual occurrence of this condition might be higher than reported. The exact pathophysiology remains uncertain, with hypotheses ranging from alcohol-induced liver damage to autoimmune processes.

Materials and Methods:

A 60-year-old male with a history of alcoholic liver disease (average consumption of > 60 g of alcohol a day, last used 3 months ago), presented with worsening right upper quadrant pain, jaundice, and fatigue. The symptoms began approximately 2-3 months prior. The patient denied recent use of drugs, toxins or herbal products. Blood analysis showed total bilirubin $173.8 \mu\text{mol/L}$,

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indirect bilirubin 108.2 $\mu\text{mol/L}$, Cr 54 $\mu\text{mol/L}$, ALP 152 U/L, ALB 24 g/L, GOT (AST) 82.1 U/L, GGT 65 U/L, GPT (ALT) 64.4 U/L, Hb 83 g/L, HCT 23.6%, MCV 100 fL, MCH 35.2 pg, RDW-SD 59.5 fL, RDW-CV 16.4%, platelets 77,000; INR 2.02; triglycerides 3.16 mmol/L, R-factor 1.6. Serology for hepatitis B and C viruses was negative. Investigations with an ultrasound showed the liver with hepatic steatosis, signs of cirrhosis and portal hypertension, splenomegaly and minimal ascites. Endoscopic examination excluded gastrointestinal bleeding (esophageal varices I grade, GOV1 Sarin's classification). Autoimmune diseases serological markers: AMA <1:80 (negative), ANA 1:160 (possible antigen association: SM, U1RNP, SS-B (La), SS-A (Ro60)).

Results:

The patient was evaluated by a surgeon, rheumatologist, and hematologist, and was diagnosed with alcohol-associated liver cirrhosis, C class Child-Pugh score, MELD-Na-23, and hemolytic anemia. The patient was monitored by a hematologist, and symptoms resolved in 6–8 weeks with alcohol abstinence and conservative therapy.

Conclusions:

This case underscores the diagnostic challenges associated with Zieve's syndrome due to its rarity and overlapping clinical features with other conditions. This case highlights the importance of a comprehensive evaluation, serial investigations, and multidisciplinary collaboration to reach an accurate diagnosis and provide appropriate management. Recognizing Zieve's syndrome promptly is crucial to prevent unwarranted and potentially detrimental interventions. Early identification can spare patients from ineffective or potentially harmful therapies, focusing instead on the most effective approach.

Keyword(s) :

2492 - Development and validation of an artificial intelligence driven convolutional neural network for automated stool colour classification to facilitate early diagnosis of biliary atresia

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Background / Hypothesis:

Early recognition of acholic (pale) stools in infants with cholestasis is critical. Misclassification of stool colour by parents and frontline community health workers remains common. To address this gap, we developed and validated an AI-driven convolutional neural network (CNN) capable of automated stool colour classification. The long-term goal is to deploy this tool widely so that caregivers can upload a stool image and receive immediate guidance on whether the stool is pale, prompting earlier medical evaluation.

Materials and Methods:

Pale stools from confirmed biliary atresia patients (2023-2025) were included. Pigmented stools were obtained from healthy infants. Images were captured using parent-owned mobile phones with varying resolution, lighting, and clarity. Stool color was confirmed by 2 pediatric hepatologists. After excluding severely blurred images, 74 pale and 110 pigmented images formed the training set. AI model used a DenseNet121 architecture. A validation cohort of 37 images was labelled by a pediatric hepatologist (gold standard) and compared with classifications by a parent, intern, nurse and the AI model.

Results:

AI model achieved perfect agreement with experts, identical to the nurse ($\kappa = 1.00$; Sensitivity 100%; Specificity 100%; AUROC 1.00). This was followed by stool classification by the intern ($\kappa = 0.94$; Sensitivity 100%, Specificity 93%). Parents showed only moderate agreement ($\kappa = 0.62$) with Sensitivity 74% and Specificity 94%, misclassifying 7/37 stools. AUROC values were 1.00 (AI model and nurse), 0.97 (intern), and 0.84 (parent). Fisher's exact testing confirmed strong association with expert classification for AI model and nurse ($p = 1.6 \times 10^{-10}$) and intern ($p = 3.9 \times 10^{-9}$), with weaker association for parents ($p = 1.1 \times 10^{-4}$)

Conclusions:

Parents show suboptimal correlation with expert, underscoring the need for an automated tool. The

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DenseNet121 model performed at expert level and can be deployed through QR codes on immunisation cards, enabling caregivers to upload stool images and receive immediate guidance

Keyword(s) :

2493 - Impact of continuous renal replacement therapy on survival and ammonia clearance in pediatric acute liver failure with advanced hepatic encephalopathy a two centre retrospective cohort analysis

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Background / Hypothesis:

To assess the impact of Continuous Renal Replacement Therapy (CRRT) on survival in children with pediatric acute liver failure (PALF) and high-grade hepatic encephalopathy (HE III–IV)

Materials and Methods:

This two-centre retrospective cohort study included 52 children with PALF and HE grade III/IV admitted between January 2018 and November 2025. Twenty-eight patients (54%) received CRRT following consent, while 24 (46%) who declined CRRT were managed with standard medical therapy (SMT) and served as controls. None of the patients underwent liver transplantation. Clinical, biochemical, and outcome data were analysed, with survival as the primary endpoint

Results:

The predominant etiology was Hepatitis A virus infection (87%), followed by Hepatitis E virus (4%), Dengue virus (2%), and drug-induced liver injury (2%). Baseline demographic and clinical parameters were comparable between groups, except for significantly higher baseline serum ammonia in the CRRT group

(292 µg/dL vs 150 µg/dL, $p=0.001$).

Overall survival did not differ significantly between the CRRT and SMT groups (54% vs 66%, $p=0.337$). Survival at Day 3 ($p=0.594$) and Day 7 ($p=0.091$) was also comparable. CRRT was associated with more effective early ammonia clearance, with $\geq 10\%$ reduction at 24 hours achieved in 48% of patients compared to 25% in the SMT group ($p=0.004$).

On multivariate analysis, mortality was significantly associated with failure to achieve $\geq 30\%$ reduction in ammonia by Day 2 ($p=0.003$), requirement of inotropic support at admission ($p=0.043$), and fulfilment of King's College Hospital (KCH) criteria ($p=0.014$). Cox proportional hazards modelling identified KCH criteria (HR 4.08, $p=0.047$) and elevated ammonia levels (HR 5.0, $p=0.031$) as independent predictors of mortality (Figure1)

Conclusions:

CRRT did not confer a survival advantage over standard medical therapy in children with PALF and HE grade III–IV. Although CRRT significantly enhanced early ammonia reduction. Fulfilment of KCH criteria and failure to achieve $\geq 30\%$ ammonia reduction by Day 2 independently predicted mortality

Keyword(s) : Acute Liver Failure, CRRT, Transplant Free survival

2494 - Clinically significant weight loss through combination of glp 1 agonist and sedation free swallowable intragastric balloon in prader-willi syndrome with severe cardiopulmonary risk

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Background / Hypothesis:

Prader–Willi Syndrome (PWS) is characterized by hyperphagia, morbid obesity, endocrine dysfunction, and multisystem comorbidities. Weight reduction is essential but challenging, particularly in patients with severe obesity and cardiopulmonary compromise who are not candidates for anesthesia. We report the

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successful use of combined GLP-1 agonist therapy and sedation-free swallowable intragastric balloon in a child with PWS and high anesthesia risk.

Materials and Methods:

A multidisciplinary team evaluated a 15 year old male, genetically confirmed PWS patient. He had morbid obesity (BMI 64 kg/m²; weight 148 kg; height 152 cm) and severe comorbidities including obstructive sleep apnea, pulmonary artery hypertension (pulmonary pressure 65 mmHg), chronic hypercapnia (PCO₂ 55 mmHg), type II diabetes mellitus, hypothyroidism, generalized hypotonia, intellectual disability and MASLD. Diazoxide choline was not procurable. Due to severe pulmonary hypertension and respiratory acidosis, anesthesia clearance for bariatric surgery and endoscopic intragastric balloon placement was denied. The team proceeded with GLP-1 agonist therapy (semaglutide) followed by placement of the sedation-free swallowable intragastric balloon.

Results:

Semaglutide dose was rapidly escalated to 2.4 mg/week over nine weeks, resulting in modest weight loss of 3 kg. Given inadequate response, a swallowable intragastric balloon was placed after two days of patient preparatory training to overcome behavioural challenges. The procedure was completed successfully without sedation. Post-placement abdominal pain resolved within 48 hours. Over the subsequent three weeks, while continuing full-dose semaglutide, the patient achieved an additional 10 kg weight loss. Total weight loss was 13 kg (~10% of baseline).

Conclusions:

In high-risk PWS patients, a combination of GLP-1 agonist therapy and a sedation-free swallowable intragastric balloon may offer a viable and effective bridge to weight reduction. This case demonstrates meaningful early weight loss and acceptable tolerability, supporting the potential role of combined therapy in selected PWS patients with extreme obesity and limited therapeutic options.

Keyword(s) : Morbid obesity, Prader Willi Syndrome, GLP 1 agonists, swallowable intragastric balloon

2495 - Non destructive Epstein Bar Virus associated post transplant lymphoproliferative disorder

presenting with gastrointestinal bleeding in a post liver transplant child diagnostic and therapeutic challenges

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Background / Hypothesis:

Gastrointestinal (GI) bleeding in pediatric liver-transplant recipients has a broad differential, including infectious ulcers, portal vein (PV) complications, drug-related mucosal injury, and post-transplant lymphoproliferative disorder (PTLD). Distinguishing inflammatory etiologies from early PTLD is challenging, particularly in children with complex post-transplant courses. We describe the diagnostic evaluation and management of a post-living donor liver transplant (LDLT) child who developed EBV-driven non-destructive PTLD presenting with multifocal GI ulcers and hematochezia

Materials and Methods:

We report a detailed case review of a 22-month-old boy transplanted for biliary atresia at 8 months of age, with prior PV stenosis requiring stenting and one episode of rejection. He presented with haematochezia, fever, cytopenia, and persistently elevated tacrolimus levels. Evaluation included endoscopy, colonoscopy, imaging (triple-phase CT, CT angiography, PET-CT), viral PCRs, lymph node biopsies, and bone marrow studies.

Results:

Endoscopy revealed a polypoidal antral lesion and deep ulcers in the ascending colon and ileocecal region. Histology showed active colitis without dysplasia. CMV and EBV PCRs were positive; however, CMV immunohistochemistry was negative. PET-CT demonstrated FDG-avid cervical and mesenteric lymph nodes and GI mural lesions. Lymph node biopsy showed preserved architecture, CD20-positive follicles, abundant CD138-positive plasma cells, and polyclonality on kappa/lambda ISH. EBER positivity confirmed non-destructive PTLD – plasmacytic hyperplasia subtype. Rising EBV viremia and cytopenias persisted despite withdrawal of tacrolimus. The child responded rapidly to rituximab, with defervescence within 24 hours and resolution of cytopenias and GI blood loss. Subsequent transaminase elevation raised

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suspicion of acute cellular rejection, requiring ongoing immunosuppression adjustment.

Conclusions:

GI bleeding in post-LT children requires early consideration of EBV-associated PTLT, especially in the context of high calcineurin inhibitor levels and lymphadenopathy. Multimodal diagnostics and prompt immunomodulatory therapy, including rituximab, are crucial for favourable outcomes. This case highlights the intricate balance between infection control, PTLT treatment, and rejection risk in pediatric liver-transplant care.

Keyword(s) : Gastrointestinal - post transplant lympho proliferative disorder

2497 - Hepatic Hemodynamics Detected by Ultrasound Localization Microscopy is Improved after Transjugular Intrahepatic Portosystemic Shunt

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Background / Hypothesis:

To investigate the alteration of hepatic hemodynamics after transjugular intrahepatic portosystemic shunt (TIPS) using ultrasound localization microscopy (ULM).

Materials and Methods:

This prospective study enrolled patients scheduled for TIPS from July, 2025 to September, 2025 at two tertiary care centers. ULM was examined two times using Mindray A20 (Mindray, China) within 24 hours before and after TIPS, respectively. Quantitative parameters of liver parenchyma including vessel density, fractal dimension, perfusion index, intensity, velocity, and direction were analyzed. The correlation between above parameters and portal pressure gradient (PPG) were analyzed by Spearman's correlation analysis. The quantitative parameters before and after TIPS was compared using paired t-test analysis.

Results:

A total of 7 patients were included in the final analysis, including 85.71% (6/7) males, with a mean age of 54.86 ± 8.17 years old. The mean PPG pre-operation is 23.86 ± 4.60 mmHg, and the mean PPG post-operation is 12.71 ± 6.13 mmHg. The mean and median velocity post-operation were correlated with PPG post-operation ($p=0.818$, $p=0.024$; $p=0.764$, $p=0.046$). The perfusion index, velocity entropy, mean velocity and median velocity were increased after TIPS compared to pre-operation (9.77 ± 3.34 vs. 14.23 ± 4.12 , $p=0.015$; 0.82 ± 0.10 vs. 0.93 ± 0.14 , $p=0.040$; 22.73 ± 2.85 mm/s vs. 27.21 ± 2.94 mm/s, $p=0.014$; 21.62 ± 3.30 mm/s vs. 26.52 ± 3.73 mm/s, $p=0.023$).

Conclusions:

The improvement of perfusion in liver parenchyma after TIPS could be detected with ULM.

Keyword(s) :

2498 - Liver disease severity and comorbidity in HBV mono infection versus HBV HDV co infection a population based study

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Background / Hypothesis:

Hepatitis B virus (HBV)/hepatitis D virus (HDV) co-infection accelerates progression of liver diseases, including cirrhosis, hepatic decompensation, and hepatocellular carcinoma (HCC). This study aimed to describe the epidemiology and clinical outcomes in patients with HBV mono-infection and HBV/HDV co-infection in South Korea.

Materials and Methods:

This nationwide population-based study analyzed data from the Korean Health Insurance Review and Assessment Service database. Adults (≥ 18 years) with ≥ 1 inpatient/outpatient chronic hepatitis B (CHB)-related claim were identified between 2014–2018.

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HBV/HDV co-infection was defined by ≥ 1 HDV-related and HDV antibody testing claims, and HBV mono-infection by no HDV-related claims. Patient demographics and clinical characteristics were summarized using descriptive statistics.

Results:

Among 590,102 eligible patients, 589,349 (99.87%) presented HBV mono-infection and 753 (0.13%) had HBV/HDV co-infection, with most patients aged 51–60 years. HBV/HDV co-infection group had significantly higher proportion of patients with comorbidities (91.6% vs. 74.0%), Charlson Comorbidity Index (CCI) score ≥ 3 (63.2% vs. 27.7%), hypertension (34.4% vs. 26.8%), diabetes (34.0% vs. 17.7%), liver-related diseases (59.2% vs. 15.2%), and antiviral therapy use for CHB (63.1% vs. 23.1%) compared to HBV mono-infection group (all $P < 0.0001$). HCC incidence among the HBV/HDV co-infection and HBV mono-infection groups was 2.9% and 3.6%, with 5-year cumulative risks of 4.8% (95% CI, 1.7–7.9%) and 3.5% (95% CI, 3.5–3.6%), respectively.

Conclusions:

Patients with HBV/HDV co-infection had a greater burden of comorbidities and liver-related complications than those with HBV mono-infection. Early identification, accurate diagnosis, and proper management of HBV/HDV co-infection among CHB patients are crucial to prevent liver disease progression.

Keyword(s) : hepatitis B; hepatitis D; co-infection; liver-related complication

2500 - Adipokine dysregulation in adipose tissue in cirrhosis

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Background and Rationale

Adipokine expression is known to be dysregulated in obesity, metabolic fatty liver disease, and cirrhosis. However, how adipokine expression within adipose tissue itself changes in cirrhosis remains poorly

understood. For example, circulating adiponectin levels are reduced in obesity and fatty liver disease and decline further in steatohepatitis, yet paradoxically increase in cirrhosis. Whether this rise reflects enhanced adipose tissue expression or reduced plasma clearance is unclear. To address this gap, we investigated the expression of key adipokines and adipose tissue-regulatory genes—including PPARG, SREBF1, SERBP1, PNPLA3, HAPLN2, ACVR1, PAQR9, GYG2, SERPINA12, DLK1, and SELE—in adipose tissue from patients with cirrhosis.

Materials and Methods

Subcutaneous adipose tissue samples were obtained from 15 patients with cirrhosis undergoing living-donor liver transplantation (LDLT) and from their healthy living donors. Total RNA and protein were extracted. Quantitative real-time PCR was performed using gene-specific primers, and relative mRNA expression was calculated using the $\Delta\Delta C_t$ method, normalized to the housekeeping gene GAPDH. Protein expression was assessed by Western blotting.

Results

The mean age was 33 ± 10 vs. 46 ± 8 years for controls and cirrhosis patients, respectively; mean BMI was comparable (25 ± 3 vs. 25 ± 5 kg/m²). Men constituted 87% of controls and 92% of cirrhosis patients. The mean MELD score among cirrhosis patients was 23 ± 6 . Mean fasting blood glucose levels were 79 ± 12 mg/dL in controls and 115 ± 34 mg/dL in patients; 28% of cirrhosis patients had diabetes. All biochemical and imaging parameters were within normal limits in healthy donors.

Adipose tissue from cirrhosis patients demonstrated increased expression of leptin, adiponectin, HAPLN2, PNPLA3, SREBF1, ACVR1, SERBP1, PAQR9, GYG2, SERPINA12, DLK1, and SELE compared with controls. In contrast, adipisin and spexin (SPX) expression was significantly reduced.

Conclusion

Adipose tissue from patients with cirrhosis exhibits upregulated transcription of several adipokines, including leptin and adiponectin. These findings suggest that the elevated circulating levels of these adipokines in cirrhosis are at least partly attributable to increased adipose tissue expression rather than impaired clearance alone. This transcriptional

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upregulation may represent a compensatory or stress-adaptive response in the setting of chronic liver disease and sarcopenia, particularly in populations with lower baseline body mass, such as Indian patients with cirrhosis.

Keyword(s) :

2501 - Evaluation of the Effect of the National Hepatitis B Vaccination Programme on Hepatitis B Seroprevalence (Healthcare worker and community based assessment)

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Background / Hypothesis:The national hepatitis B vaccination programme was introduced in Turkey in 1998, followed by a catch-up programme between 2005 and 2009 targeting individuals born between 1991 and 1997. The Ministry of Health has also provided HBV vaccination free of charge to healthcare workers and other high-risk groups. These interventions have substantially altered HBV seroprevalence patterns, particularly among younger age groups. This study aimed to evaluate HBV-related serological profiles among healthcare workers and the general adult population in Izmir, a western province of Turkey with high vaccination coverage.

Materials and Methods:HBV serological test results (Anti-HBs, HBsAg, anti-HBc IgG) of healthcare workers employed at Izmir City Hospital and adults aged ≥ 18 years from the community were retrospectively retrieved from the hospital automation system between October 2023 and December 2025. Data were anonymized, duplicate records were excluded. Analyses were performed according to age group.

Results:The study included 6,485 healthcare workers (aged 18–73) and 178,823 individuals from the general population (aged 18–102). In both groups, HBsAg was the most frequently requested test, while anti-HBc IgG was the least requested. Anti-HBc IgG seropositivity increased significantly with age, particularly in the general population, reaching approximately 50% among individuals aged over 50. Similarly, HBsAg positivity was higher in the general population, increasing notably after the age of 35. However, HBsAg positivity declined sharply (<1%) among individuals born after 1998 in both groups. Vaccine-related anti-HBs positivity was also higher in age groups covered by the vaccination programme. Since HBV vaccination is administered to

healthcare workers in all age groups, high anti-HBs positivity rates were detected in almost all age groups. In individuals in the community, anti-HBs positivity rates reached 82% among those vaccinated under the national vaccination program and catch-up programmes, while anti-HBs positivity decreased to around 30–40% after the age of 35. This situation was similar to that observed in age groups with increased anti-HBc IgG positivity, suggesting that it may be related to natural transmission of HBV infection.

Conclusions:The findings demonstrate that the national HBV vaccination programme has markedly reduced chronic HBV infection and exposure among younger adult populations. The detection of high anti-HBs titres in healthcare workers of all ages demonstrates the benefit of the Ministry of Health's policy of providing free vaccinations to this occupational group. The high anti-HBc IgG positivity detected in middle-aged and older individuals, along with the parallel anti-HBs positivity in these age groups, indicates that exposure to the virus was higher in previous years and that our country was previously among the regions with high HBV endemicity. However, thanks to the national vaccination programme, the endemicity level has now decreased to low.

Keyword(s) : Hepatitis B, Seroprevalence, Vaccination programme, Healthcare workers, Community

2502 - Turkish Real World Effectiveness and Safety of Bulevirtide in Chronic Hepatitis Delta A Multicentric Experience with Long Term Follow Up

Devrim Muge Ozari Gulnar ¹, Ozgur Aktas ², Cigdem Mermutluoglu ³, Mehmet Akif Yaglı ⁴, Gokhan Kabacam ⁵, Yilmaz Cakaloglu ⁶, Hakan Bozkaya ⁷, Genco Gencdal ¹, Murat Akyildiz ¹, Mujdat Zeybel ¹, Sabahattin Kaymakoglu ⁴, Mustafa Kemal Celen ³, Cihan Yurdaydin ⁸

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Background/Hypothesis: Bulevirtide (BLV), was approved in Europe in 2020 for treating compensated

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CHD. It is also available in Russia and Australia. BLV is not licensed in Turkey, a CHD endemic country. We present the first real-world data on BLV use in CHD in Turkey. This study evaluated daily 2mgBLV therapy in CHD patients for up to 36-months, with emphasis on virologic and biochemical response (VR, BR) and viral breakthrough (VBr).

Methods: 33 patients with CHD who initiated BLV therapy between 2020-2023 were analyzed. All patients had baseline detectable HDVRNA and elevated ALT. Five patients had decompensated cirrhosis, and all other patients had compensated liver disease. Seven patients used the European label Hepcludex, and remaining 26 patients received Myrcludex from Russia. Primary outcomes were VR (defined as undetectability or > 2 log decrease in HDVRNA) and BR (defined as ALT normalization) at end of on-treatment follow-up (FU). VBr was defined as an increase of HDV RNA > 1 log from nadir or HDV RNA becoming detectable after undetectability. Statistical analyses included t and Mann-Whitney U tests for continuous and Fisher's exact and Chi-squared tests for categorical variables.

Results: Mean ($\pm$ SD) baseline ALT was 99.6 ($\pm$ 66.7) U/L and HDVRNA was 4.94 ($\pm$ 1.11 log) IU/mL. At 12 months, VR was achieved in 74.1% (20/27) of evaluable patients, and BR was 70.4% (19/27). Response rates were sustained through 24 months reaching 61.1% (11/18) with VR and 64.7% (11/17) with BR. VBr occurred in 4 of 26 patients on the Russian BLV (15.4%). Patients with VBr had higher baseline INR (1.22 ± 0.03 vs 1.04 ± 0.14 , $p=0.033$) and a trend toward higher baseline HDVRNA (5.82 ± 0.81 vs 4.82 ± 1.10 log. IU/mL, $p=0.091$). Two patients with VBr had no on-treatment FU HDVRNA testing. Remaining 2 patients continued therapy and had persistent viremia throughout FU. Mean time to VBr was 18.5 ± 3.87 months (range: 15-24 months). Following VBr, 3 patients experienced ALT elevations. Seven patients (21%) discontinued BLV treatment (21.2%) at median 19 months (range 12-27). Decompensated patients ($n=5$, 15.2%) demonstrated similar VR rates compared to patients with compensated liver disease, suggesting efficacy across the disease severity spectrum. BLV was well tolerated.

Conclusion: In this real-world Turkish cohort, BLV demonstrated VR in 74% of patients at 12 months on-treatment and, maintained VR in the majority throughout 36 months. An unexpectedly high VBr rate was observed with the Russian BLV. Patients denied non-compliance.

Keyword(s) : Hepatitis D virus, bulevirtide, antiviral therapy, virological breakthrough

2503 - Tumor Lysis Syndrome Following Atezolizumab–Bevacizumab Therapy in Advanced Hepatocellular Carcinoma A Rare and Life Threatening Complication

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Background

Tumor lysis syndrome (TLS) is an oncologic emergency most commonly associated with hematologic malignancies and is rarely observed in solid tumors. With the increasing use of immune checkpoint inhibitors, TLS has been sporadically reported following immunotherapy

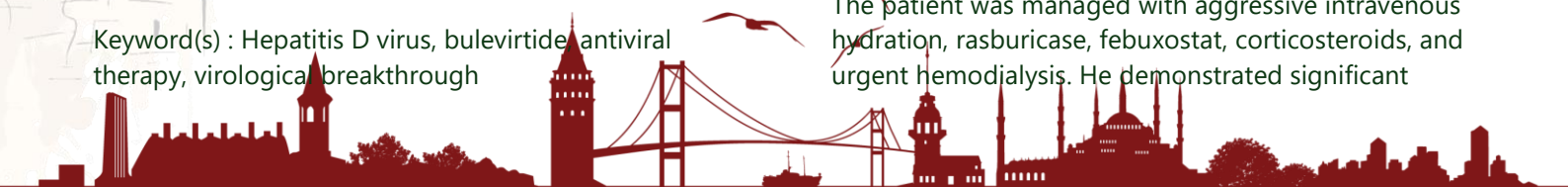
Case report

a 64-year-old male with DM and a history of chronic HCV infection, who achieved sustained virologic response after direct-acting antiviral therapy seven years prior. He presented with advanced hepatocellular carcinoma characterized by a large hepatic focal lesion, malignant lymphadenopathy, and portal vein thrombosis. Liver function was preserved (Child–Pugh A6), and the disease was staged as BCLC stage C. The patient was initiated on first-line atezolizumab plus bevacizumab therapy.

One week after the first treatment cycle, he developed oliguria and acute clinical deterioration. Laboratory evaluation revealed marked hyperuricemia (16 mg/dl), acute kidney injury (serum creatinine 5.6 mg/dl), hyperphosphatemia (10.5 mg/dl), hypocalcemia (8.4 mg/dl), and elevated transaminases, fulfilling Cairo–Bishop criteria for clinical tumor lysis syndrome.

Management and Outcome

The patient was managed with aggressive intravenous hydration, rasburicase, febuxostat, corticosteroids, and urgent hemodialysis. He demonstrated significant



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improvement in renal function, metabolic abnormalities, and overall clinical status.

Conclusion

This case underscores tumor lysis syndrome as a rare but potentially fatal complication of atezolizumab-bevacizumab therapy in advanced HCC. Clinicians should maintain heightened vigilance and closely monitor metabolic parameters, particularly in patients with high tumor burden and vascular invasion

Keyword(s) : Immunotherapy, atezolizumab-bevacizumab, hepatocellular carcinoma

2509 - Feasibility And Limitations Of Living Donor Liver Transplantation In Armenia

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Background / Hypothesis:

Liver transplantation remains the definitive treatment for selected patients with end-stage liver disease. In Armenia, the liver transplantation program is relatively nascent and primarily reliant on living donors due to the current absence of active deceased donor organ procurement systems.

Materials and Methods:

A retrospective review of national data from Armenia's transplant centers and government reports was conducted. Key indicators included the number of liver transplants performed, waiting list characteristics, donor types, and systemic barriers affecting program expansion.

Results:

The first living donor liver transplantation (LDLT) in Armenia was successfully performed in 2019 at Astghik Medical Center, marking a milestone in the country's transplant history. Over recent years, Armenia has implemented LDLT and performed multiple liver transplants at domestic centers, alongside kidney and corneal transplants primarily from living related

donors. According to Ministry of Health data from 2021–2023, approximately ten liver transplants were conducted under the state program. National figures suggest that around 29 patients currently require liver transplantation, with a waiting list reflecting diverse etiologies including viral and autoimmune cirrhosis and cryptogenic disease. Despite legislative progress toward enabling deceased donor transplantation by updating the list of transplantable organs, practical implementation of a deceased donor program remains pending.

Conclusions:

Armenia's liver transplant program has demonstrated feasibility and clinical success within the constraints of a living donor model. However, the limited number of procedures relative to need, lack of an active deceased donor system, and resource constraints underscore the necessity for national strategic initiatives to develop organ donation infrastructure, expand donor registries, and improve equitable access to transplantation. Further research and registry development are essential to monitor long-term outcomes and inform health policy.

Keyword(s) : Living donor liver transplantation, end-stage liver disease, feasibility

2512 - Evaluation of the 24 year prognosis of patients with inactive HBsAg carriage

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Background / Hypothesis: This study aimed to evaluate long-term real-life outcomes and prognosis of patients diagnosed with HBsAg positivity and followed as inactive carriers.

Materials and Methods: Patient records of individuals diagnosed with inactive chronic hepatitis B and followed between January 1994 and March 2018 were retrospectively reviewed using the hospital automation system. Sociodemographic characteristics, baseline and follow-up clinical findings, and laboratory results were recorded. Patients who had not attended follow-up visits for more than one year were contacted by telephone and invited for reassessment. Information regarding follow-up adherence, reasons for non-attendance, liver biopsy status, fibrosis score, cirrhosis,

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and hepatocellular carcinoma (HCC) development was collected through telephone interviews or face-to-face visits.

Results: A total of 1,147 patients were included, with a mean age of 50.17 ± 12 years; 579 (50.5%) were male. Regular follow-up attendance was observed in 448 patients (39%). Of these, 418 could be contacted, and 305 agreed to participate in interviews; however, only 75 attended a follow-up visit at the hospital. Total follow-up duration ranged from 6 months to 23 years.

Regarding follow-up adherence, 370 patients (32%) attended regularly, 116 (10%) rarely missed visits, 272 (24%) attended irregularly, and 389 (34%) had discontinued follow-up for more than one year. Among patients with irregular follow-up, the most common reasons were follow-up at another center (51.5%); forgetting appointments (17.5%); followed by perceiving follow-up as unnecessary; work-related issues; lack of symptoms, caregiving responsibilities; or not being invited by physicians.

Liver biopsy was performed in 63 patients (5.5%), with histological activity index scores ranging from 2 to 9 and fibrosis scores from 0 to 2. Antiviral therapy was initiated in 29 patients (2.5%). At the most recent evaluation, 837 patients (73%) remained inactive carriers, 126 (11%) experienced HBV flare-ups, 156 (13.6%) achieved spontaneous HBsAg loss, and six patients (0.5%) developed cirrhosis or HCC. Regular follow-up was observed in 55% of treated patients and 33% of those with flare-ups. Among patients with spontaneous HBsAg loss, 48% had previously attended follow-up but later discontinued. Of the six patients who developed cirrhosis-HCC, four had a family history of chronic HBV infection.

Conclusions: This study demonstrates that although most inactive HBsAg-positive patients remain stable, significant complications may still occur. The asymptomatic course and absence of treatment often lead patients to underestimate disease severity and discontinue follow-up. Regular monitoring and patient education about long-term risks are essential to prevent loss to follow-up and ensure early detection of complications. (Note: This work is the doctoral thesis of Dr. İrem Altaş).

Keyword(s) : Inactive chronic HBV, prognosis, complications

2516 - Evaluation of the 24 year prognosis of patients with inactive HBsAg carriage

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Background / Hypothesis: This study aims to evaluate the long-term real-life data and prognosis of individuals diagnosed with HBsAg positivity and monitored as inactive carriers.

Materials and Methods: The follow-up files and existing records in the automation system of patients diagnosed with inactive chronic hepatitis B and monitored between January 1994 -March 2018, were individually reviewed. Their sociodemographic data, all findings related to their initial and follow-up visits, and test results were recorded in an Excel file. In addition, patients who had not attended follow-up appointments for more than a year were contacted by telephone and invited to attend a follow-up appointment. These patients were then interviewed either during the telephone call or face-to-face during their outpatient clinic visit. The patients' sociodemographic data, diagnosis date, follow-up period, whether they attended follow-ups regularly, the reason for non-attendance if applicable, whether a liver biopsy was performed during follow-up, and if so, the fibrosis score, cirrhosis, or HCC development status were examined.

Results: The study included a total of 1,147 patients with a mean age of 50.17 ± 12 , 579 (50.5%) of whom were male. It was determined that 448 (39%) of the patients attended follow-up appointments regularly, while 699 (61%) had not attended for over a year. Although non-attending patients were contacted by phone, only 418 could be reached, and 305 of these agreed to participate in a face-to-face survey; only 75 of the patients invited to the hospital attended the follow-up.

The total follow-up periods of the patients since their years of application to our hospital ranged from a minimum of 6 months to a maximum of 23 years. When the patients' attendance at their check-ups was evaluated, it was determined that 370 (32%) attended their check-ups regularly, 116 (10%) rarely missed their check-up intervals, 272 (24%) did not attend regularly, and 389 (34%) had previously attended regularly but had not attended their check-ups for over a year.

Keyword(s) : inactive HBsAg carriage

2517 - Is primary biliary cholangitis excluded in patients negative for PBC specific autoantibodies and without cholestatic features

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Background / Hypothesis:

The diagnosis and therapeutic decision-making for atypical primary biliary cholangitis (PBC) remain controversial. Based on clinical practice, we report for the first time a new pathological subtype of PBC characterized by negativity for PBC-specific autoantibodies and normal alkaline phosphatase (ALP) levels. We systematically analyzed its clinical and pathological features to provide a basis for improving clinical recognition and guiding early treatment decisions.

Materials and Methods:

We retrospectively reviewed and summarized the clinical and pathological data of 10 patients diagnosed with histologically confirmed PBC at the Second Hospital of Nanjing from 2016 to 2025.

Results:

Among the 10 patients, 80% were female, with a median age of 54.5 years. The majority of patients presented with mild elevations of aminotransferases and gamma-glutamyl transferase (GGT) as the main biochemical abnormalities, accompanied by elevated immunoglobulin M (IgM). Liver histology showed chronic non-suppurative destructive cholangitis in all patients, predominantly at Ludwig stage II and Nakanuma stage II. Some patients already had advanced liver fibrosis and/or portal hypertension at diagnosis. Following ursodeoxycholic acid (UDCA) therapy, 88.9% of patients maintained persistently normal ALP and GGT levels. No patient developed hepatic decompensation during follow-up.

Conclusions:

Typical PBC-like pathological changes can exist even in patients with negative PBC-specific autoantibodies and normal ALP levels, suggesting that bile duct injury may precede biochemical abnormalities. Liver biopsy is essential for early identification in suspected cases, and timely UDCA therapy can achieve favorable biochemical response and prognosis. Future multicenter, long-term follow-up studies are still needed to further clarify the natural history and pathogenesis of this subtype.

Keyword(s) : primary biliary cholangitis; PBC-specific

autoantibodies; normal alkaline phosphatase; liver biopsy

2520 - A RARE CASE IN LIVING DONOR LIVER TRANSPLANTATION GRAFT TO GRAFT OF PORTAL VEIN ANASTOMOSIS

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Background / Hypothesis:

In this report, we present a successful liver transplantation in which a living donor graft with anomalous portal venous branching (APVB) was reconstructed on the back table using a homolog portal Y-graft. This reconstructed graft was then anastomosed to a cadaveric interposition graft of the recipient with Yerdel grade 3 portal vein thrombosis, resulting in a graft-to-graft portal vein anastomosis.

Materials and Methods:

A 68-year-old Middle Anatolian female patient presented with clinical features consistent with chronic liver disease. She reported abdominal distension, fatigue, and weakness. Her MELD-Na score was calculated as 16. Preoperative dynamic liver CT demonstrated portal vein thrombosis (PVT) with minimal residual flow. The patient underwent right-lobe living donor liver transplantation for cryptogenic liver failure and Yerdel type 3 PVT. The recipient hepatectomy was performed using standard surgical procedures. Prior to graft implantation, thromboendovenectomy was attempted. However, during the procedure, a bleeding occurred at the portal vein confluence. Hemostasis was achieved by inflating a Foley catheter balloon at the confluence, and a cadaveric vein graft was anastomosed outflow of Superior Mesenteric Vein-Splenic vein for extension. After clamping the vena cava, the right-lobe graft was implanted. The donor graft had anomalous portal venous branching; therefore, a homolog portal Y-graft interposition was performed on the back table. The reconstructed right-lobe portal vein was then anastomosed to the recipient's extension graft. Ensuring continuous and physiological portal flow and successful graft perfusion.

Results:

Our case required both portal venous reconstruction with homolog portal Y-graft on the donor graft and a

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jump graft from the portal confluence due to PVT. This case involves highly complex procedures. In this report, we aim to emphasize that when physiological reconstruction pathways are used, long-term outcomes can be favorable and this case represents an encouraging example.

Conclusions:

In right-lobe living donor liver transplantation with APVB, successful outcomes can be achieved using appropriate surgical techniques. This case demonstrates that in living donor liver transplantation with PVT, a combined portal Y-graft and extension graft, completed with graft-to-graft anastomosis, can be safely used for portal vein reconstruction.

Keyword(s) : Jump graft, Jump graft and Y-graft anastomosis, Liver transplantation, Portal vein reconstruction

2521 - A novel clustered regularly interspaced short palindromic repeats based assay for intrahepatic hepatitis B virus covalently closed circular DNA quantification

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Background: The persistence of covalently closed circular DNA (cccDNA) within infected hepatocytes represents the principal barrier to achieving a functional cure for hepatitis B virus (HBV) infection. Novel drug developments and therapeutic strategies aimed at eliminating cccDNA are essential. However, existing cccDNA quantification methods are limited by suboptimal sensitivity and specificity, frequently resulting in false-positive detection of non-cccDNA intermediates.

Materials and Methods: We developed a clustered regularly interspaced short palindromic repeats (CRISPR)/CRISPR-associated protein 12a (Cas12a)-based assay to accurately quantify intrahepatic cccDNA. CRISPR RNAs (crRNAs) were designed to target regions that do not overlap between relaxed circular DNA (rcDNA) and cccDNA. The crRNAs were conserved across HBV genotypes A–D. Assay parameters, including single-stranded DNA (ssDNA) reporter concentration, fluorescence signal gain, and incubation time, were optimized to enhance Cas12a collateral activity. To overcome limited sensitivity, recombinase polymerase amplification (RPA) was

integrated as a pre-amplification step. Quantitative PCR (qPCR) was used as a reference method to validate the assay.

Results: The combination of RPA and CRISPR/Cas12a achieved a high sensitivity at 10 copies/reaction. Reliable quantification between expected and measured values was observed over a dynamic range of 10^1 – 10^6 copies/reaction, exhibiting a strong correlation ($R^2 = 0.9263$). Comparative analysis with qPCR showed excellent agreement between the two methods ($R^2 = 0.9992$, $p < 0.0001$). Importantly, the assay exhibited high specificity for cccDNA, with specific detection in liver tissue samples, and no detection in serum or plasma samples. Application of the assay to 25 non-cancerous liver tissue samples obtained from HBV-related hepatocellular carcinoma (HCC) patients resulted in successful cccDNA quantification in 11 samples, which strongly correlated with qPCR measurements ($R^2 = 0.9590$).

Conclusion: This newly developed CRISPR-based assay provides a sensitive, specific, and accurate approach for cccDNA quantification, supporting its utility in research and development toward HBV cure.

Keyword(s) : hepatitis B virus (HBV), covalently closed circular DNA (cccDNA), clustered regularly interspaced short palindromic repeats (CRISPR)

2522 - Triple Trouble A Case of Hepatocellular Carcinoma in a Patient with Previous Renal and Colonic Malignancies

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Background / Hypothesis:

Multiple primary tumors (MPTs) are defined as two or more synchronous or metachronous primary malignancies in a single patient. The frequency of MPTs ranges from 2% to 17%, with triple primary cancers being exceedingly rare. We report a case of a patient with three primary malignant tumors: Renal cell carcinoma, colon adenocarcinoma and hepatocellular carcinoma.

Materials and Methods:

We present a 74-year-old male with a history of synchronous renal cell carcinoma and stage IA colon adenocarcinoma diagnosed in 2008. Both were surgically managed in the same year with partial nephrectomy and left hemicolectomy. The patient was lost to follow-up until seventeen years postoperatively,

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when, in 2025, he developed progressive weight loss. Laboratory tests showed elevated liver enzymes [aspartate aminotransferase (AST) 99 U/L, alanine aminotransferase (ALT) 102 U/L], and ultrasound revealed a fairly defined heterogeneous mass measuring 99 × 85 × 94 mm. Carcinoembryonic antigen (CEA) was 6.67 ng/mL, and protein induced by vitamin K absence or antagonist-II (PIVKA-II) was elevated at 441.42 mAU/mL. Other tumor markers, including CA19-9 and α-fetoprotein, were within normal limits. Magnetic resonance imaging of the whole abdomen demonstrated a large right hepatic mass involving segments VIII/VII/V/VI measuring 72 × 61 × 63 mm (AP × T × CC), with arterial enhancement and washout, initially raising suspicion for metastasis from the previous colon adenocarcinoma. Upper endoscopy showed no high-risk stigmata for bleeding, and colonoscopy was negative for disease relapse. Liver biopsy revealed hepatocytes in trabecular arrangements with immunohistochemical staining positive for Arginase and Glypican-3, confirming hepatocellular carcinoma. Thus, a diagnosis of a third primary malignancy of the liver was made rather than metastatic recurrence. The patient subsequently received three cycles of immunotherapy with atezolizumab and bevacizumab. A follow-up positron emission tomography (PET) scan showed a minimal decrease in size of the hepatic mass with significantly reduced arterial enhancement, which may reflect treatment response.

Results:

This case highlights the diagnostic challenge of differentiating hepatic metastasis from de novo HCC in patients with prior malignancies. Current guidelines recommend biopsy confirmation when imaging is inconclusive, particularly in non-cirrhotic liver. Recognition of a new primary malignancy allowed consideration of liver-directed therapy instead of palliative systemic treatment for presumed metastasis.

Conclusions:

The possibility of multiple primary cancers, while rare, underscores the importance of thorough follow-up. Early detection and risk reduction strategies are vital in improving outcomes and guiding appropriate treatment.

Keyword(s) : Hepatocellular carcinoma, Multiple primary tumors, Triple primary cancer, Renal cell carcinoma, Colon adenocarcinoma

2523 - HepPlus Protocol for a multicenter study of a web based platform to support hepatitis B and C management in Thailand

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Background: Hepatitis B and C remain leading causes of liver-related morbidity and mortality in Thailand, with many patients lost to follow-up due to fragmented care and limited interoperability between healthcare facilities. There is an urgent need for digital solutions that enhance care coordination, support clinical decision-making, and enable national disease surveillance. This study describes the protocol for the development and pilot implementation of Hep Plus, a web-based application designed to: (1) serve as a national e-registry for hepatitis B virus (HBV) and hepatitis C virus (HCV); (2) support healthcare providers in treatment decisions through embedded clinical decision support (CDS); and (3) improve care coordination through standardized documentation and secure communication.

Methods: This is a prospective, multicenter, non-randomized observational study piloted in Thailand's Sixth Regional Health Area. In Phase 1, nurse practitioners at primary care clinics will screen patients using rapid HBV/HCV tests and register confirmed cases into the Hep Plus platform. In Phase 2, patients will be managed at district or general hospitals, with participating physicians completing the modified Healthcare Systems Usability Scale (HSUS) at six months and joining focus groups at month nine. In Phase 3, patients with complex conditions referred to tertiary hospitals will be managed by subspecialists using the platform. The providers will complete a modified HSUS at month nine and participate in focus groups at month twelve. The primary outcome is platform usability across patient safety, workflow integration, and user control. Secondary outcomes include patient retention and the proportion of patients receiving appropriate treatment.

Results: Hep Plus system has achieved Technology Readiness Level 9 (TRL9) and is implemented across Thailand's Sixth Regional Health Area. To date, 319 cases have been recorded, comprising 188 positive and 14 negative hepatitis B cases (14 negative) and 104

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positive hepatitis C cases (7 negative). Initial satisfaction assessments indicate that users agree on the system's utility and effectiveness. However, user-friendliness and workflow integration were rated at a moderate level, serving as key areas for future system optimization to better align with clinical workloads.

Conclusions: Hep Plus may strengthen hepatitis B and C care through standardized documentation, clinical decision support, and improved care coordination. Its web-based design supports use in diverse settings and may contribute to real-time surveillance and national digital health efforts. Integration with electronic health record systems remains a key area for future work.

Keyword(s) : Hepatitis B , Hepatitis C , Digital health, Clinical decision support system , Electronic registry , Screening and linkage to care

2524 - Baseline Albumin Structural Defects and Soluble Inflammatory Markers Identify Poor Responders to Albumin Therapy in Cirrhosis

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Background / Hypothesis:

Cirrhotic patients often exhibit functionally impaired albumin associated with immune dysfunction and poor outcomes. It is unclear whether this condition affects the therapeutic efficacy of albumin infusion treatment. We aimed to identify predictors of poor response to albumin therapy in patients with decompensated cirrhosis.

Materials and Methods:

We studied 262 hospitalized cirrhotic patients who received at least weekly albumin infusions at a dose ≥ 0.5 g/kg. Serum samples were collected at baseline for biomarker analysis. Twenty-one inflammatory mediators were measured by high-throughput electrochemiluminescence immunoassay. Serum albumin structural variants were profiled by UPLC-ESI-QTOF mass spectrometry, classifying human serum albumin (HSA) into seven isoforms: native (unmodified) HSA, HSA-DA, HSA-L, glycated HSA, cysteinylated HSA, glycated-cysteinylated HSA, and oxidized HSA (sulfated/sulfonated, HSA-SO₂/SO₃). We defined non-responder after albumin treatment as patients who died by 90-day follow-up and compared baseline markers between responders and non-responders.

Fine-Gray competing risk regression was used to identify predictors of treatment response, treating liver transplantation as a competing event.

Results:

Among all the patients, there were 57 (21.8%) non-responders and 205 (78.2%) responders. Non-responders had more severe baseline disease and an inflammation profile. They presented with higher baseline MELD-Na scores (median 28.33 vs 17.03, $p < 0.001$) and a higher incidence of bacterial infection at admission (63.2% vs 24.4%, $p < 0.001$). Pro-inflammatory markers including IL-6, IL-8, MIP-3 α , MIP-1 β (all $p < 0.05$) were significantly higher in non-responders than those in responders. Baseline serum albumin concentration did not differ significantly between groups (median 25 vs 27 g/L, $p = 0.46$). However, baseline profile of albumin functional parameters were significantly different between non-responders and responders. Non-responders had significantly lower fraction of native (unmodified) HSA (median 9.6% vs 13.3%, $p = 0.001$) and higher levels of oxidatively modified albumin including glycated albumin, and sulfated/sulfonated HSA than in responders. As a result, non-responders had a significantly lower effective albumin (median 3.66 vs 2.37 g/dL, $p = 0.001$) than responders. In competing-risk regression, higher IL-6, MIP-3 α , glycated albumin, and HSA-SO₂ and lower effective albumin were associated with significantly higher risk of non-responsive to treatment (all $p < 0.05$). The combination of these four markers demonstrate the C-index of 0.71 (95%CI, 0.58-0.84) in predicting non-responsiveness, significantly higher than that of MELD-NA (0.60, 0.47-0.74).

Conclusions:

Cirrhotic patients who respond poorly to albumin infusion therapy are characterized by a state of intense systemic inflammation and qualitatively defective albumin which could predict poor albumin therapeutic response.

Keyword(s) : Decompensated cirrhosis, Albumin treatment, outcome, inflammation, albumin function

2526 - Differences between non alcoholic liver disease and alcoholic liver disease related HCC

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Background / Hypothesis: Non-alcoholic liver disease

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and alcoholic liver disease begin as simple steatosis that may progress to steatohepatitis and ensuring liver-related complications such as cirrhosis and hepatocellular carcinoma (HCC). We investigated differences in clinical characteristics between non-alcoholic liver disease (NALD) and alcoholic liver disease (ALD)-related HCC.

Materials and Methods: NALD and ALD patients were identified from our department's prospective HCC database. A total of 54 and 45 patients met predefined inclusion and exclusion criteria for the NALD-HCC and ALD-HCC groups, respectively. Clinical, biochemical and tumor characteristics were studied.

Results: NALD-HCC patients were older compared to ALD-HCC patients (71 ± 9 vs. 65 ± 9 years, $P < 0.001$) and less male predominant (64% vs. 97%, $P < 0.001$). Prevalence of diabetes mellitus (77% vs. 35%, $P < 0.001$) and hypertension (79% vs. 57%, $P < 0.001$) were significantly higher in the NASH-HCC group. There were no differences in alpha-fetoprotein level, lesions found at diagnosis (unifocal/multifocal) or prevalence of portal vein invasion. In both group, almost half of the patients were in TNM stage 4 at the time of diagnosis and more than 50% of patients were not suitable for any therapy. Median survival in the NALD-HCC and ALD-HCC groups were 13 and 7 months respectively ($P = 0.114$).

Conclusions: Despite significant differences in demography of the NALD-HCC and ALD-HCC groups, liver and tumor characteristics were comparable. Most patients were diagnosed late and were not amenable to curative or locoregional therapies. Better characterization of patients with NALD and ALD at risk of HCC is necessary to optimize screening, surveillance, and management strategies.

Keyword(s) : Hepatocellular Carcinoma, Alcohol

2528 - Gut Microbiota Dysbiosis and Its Role in the Pathogenesis of Metabolic Dysfunction Associated Steatotic Liver Disease

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) has become the most prevalent chronic liver disease in the world; an estimated 30–35% of the world's population is affected, and the prevalence of MASLD has been increasing in recent years. An increasing number of studies have indicated a close relationship between dysbiosis and MASLD.

Hence, there is an interest in exploring fatty infiltration as a result of dysbiosis, which includes bacterial composition disturbance, with a profound analysis of preventive and therapeutic factors that impact the occurrence and progression of liver disease.

Materials and Methods:

This study involved 123 subjects divided into two groups (65 MASLD, 18 MASH, and 40 healthy individuals in the control group) with a median age of 49 (23–75) diagnosed with MASH/MASLD based on ultrasound and biochemical tests. The criteria for fatty infiltration included a diffuse increase in the echogenicity of the liver parenchyma, decreased attenuation of the liver, and a ratio between the brightness level of the liver and the right kidney, calculated for the hepato-renal index (HRI) determination. The biochemical evaluation included liver function tests and a lipid profile. Dysbiosis was assessed using a stool bacteriological test and a stool test for dysbiosis.

Results:

he genus *Enterococcus*, like *Streptococcus*, was increased in patients with MASLD/MASH compared with controls. Additionally, uncultured *Clostridiales*, as well as entero-hemolytic *Escherichia coli*, were also increased. In contrast, genus *Bifidobacterium* and *Lactobacillaceae* were decreased in patients with MASLD/MASH. A significant loss of beneficial bacteria, such as *Faecalibacterium*, was observed, affecting intestinal barrier function. The diversity of the microbiota was decreased in patients compared with controls.

Conclusions:

Our study revealed an association between the abundance of *Bacteroides* in the gut and MASLD/MASH. MASLD developed initially in patients with significant dysbiosis. These results demonstrate that gut microbiota analysis provides prognostic information in addition to classical risk factors for MASLD severity, and strongly suggest that the gut microbiota plays a significant role in the pathogenesis of MASLD. Patients with MASLD should be tested for dysbiosis, and vice versa. It also suggests that correcting the gut microbiome may be beneficial for treating patients with MASLD/MASH.

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Keyword(s) :

2529 - Clinical Manifestations of the Oral Mucosa in Patients with Viral Hepatitis C in Armenia 2025 Years

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Background / Hypothesis:

HCV patients including cirrhosis and hepatocellular carcinoma represent a diverse group with various patterns of viral replication and great variations of immune profiles resulting in different hepatic and extrahepatic manifestations. It is well-known, that the basis of the pathogenesis of any inflammatory process is the result of combination of two main factors: the effects on the tissue of any irritant and local tissue reaction. The latter depends on the general state of the body, its local and overall immunity. There are many clinical evidences of pathogenic interactions between diseases of internal organs and oral mucous diseases. The aim of this study was to evaluate the changes of the oral mucous membrane in patients with HCV.

Materials and Methods:

Our study included 250 patients with HCV, with a mean age of 51 years (25 to 77 years). Among these, 173 were men (69.2%). To assess the condition of the oral mucosa, an objective dental examination of the oral cavity and the vermillion border of the lips was performed.

Results:

Oral mucosal discoloration—hyperemia and cyanosis—was observed in all patients ($p < 0.001$). Tongue coating was detected in 47.8% of HCV patients. Hemorrhages in the mucosa, in the form of hemorrhages, were detected in 92.7% of patients, along with liver disease severity ($p < 0.001$). Gum changes included hyperemia (35.8%), swelling (42.3%), bleeding, and tenderness (53.6%), with statistical significance at $p < 0.001$. Purulent discharge from gingival pockets was observed (32.8%), along with necrotic areas (27.6%).

Conclusions: Changes in the oral mucosa in HCV were characterized by various manifestations related to the severity of liver disease. It has been established that HCV treatment leads to improvement in the condition of the oral mucosa and a decrease in the proliferation of pathogenic microorganisms.

Keyword(s) : oral mucosae, hemorrhages, HCV

2530 - Fibroblast and macrophage derived thrombospondin 1 orchestrates the fibroinflammatory niche in metabolic dysfunction associated steatohepatitis induced fibrosis

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Background / Hypothesis: Thrombospondin-1 (THBS1) activates latent TGF- β 1 and contributes to stromal-immune communication. However, how THBS1 derived from fibroblasts and macrophages collectively contributes to fibroinflammatory remodeling in MASH has not been clearly defined.

Materials and Methods: THBS1 expression was examined in liver tissue and serum from patients with MASLD and in two dietary murine MASH models. We integrated single-cell RNA sequencing, immunohistochemistry, confocal microscopy, and flow cytometry with targeted perturbation of fibroblasts and macrophages using nintedanib, the THBS1-blocking peptide LSKL, and siRNA-mediated THBS1 silencing.

Results: THBS1 and the fibroblast activation marker FAP increased in association with fibrosis severity and IL-6 levels. scRNA-seq and spatial imaging demonstrated THBS1 expression in both FAP⁺ fibroblasts and monocyte-derived macrophages, indicating a coordinated fibroinflammatory niche. Nintedanib reduced THBS1 expression in both compartments, decreased CD11b⁺MERTK⁺ macrophages and FAP⁺PD-L1⁺ fibroblasts, and improved histologic fibrosis. Direct THBS1 inhibition produced similar antifibrotic effects. In vitro, THBS1 knockdown or blockade suppressed TGF- β 1-induced fibroblast activation and reduced LPS-stimulated IL-1 β and IL-6 production in macrophages, supporting reciprocal THBS1-dependent stromal-immune

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signaling.

Conclusions: THBS1 functions as a shared signaling mediator between fibroblasts and macrophages in MASH. Therapeutic suppression of THBS1, either through nintedanib or direct inhibition, disrupts this fibroinflammatory circuit and may represent a viable strategy to limit fibrosis progression in MASLD/MASH.

Keyword(s) : thrombospondin-1, nintedanib, fibroblast activation, metabolic dysfunction-associated steatohepatitis, liver fibrosis, immune–stromal crosstalk

2532 - COMPARISON OF ATTENUATION PARAMETER AND LIVER STIFFNESS MEASUREMENT BETWEEN HEPATUS AND FIBROSCAN IN PATIENTS WITH CHRONIC LIVER DISEASE A PROSPECTIVE STUDY

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Background / Hypothesis:

We compared controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) obtained using Hepatus® (HP, Mindray, Shenzhen, China) and Fibroscan (FS, Echosens, Paris, France). Additionally, we evaluated intra- and inter-observer variability and compared examination times using the two devices.

Materials and Methods:

HP and FS examinations were performed on patients with chronic liver disease by two operators from different departments, twice on each patient, at two different time points, independent of each other.

Results:

The data for 158 patients with 1264 examinations was analyzed (mean age 58.8 ± 13.8 years, male 47.5%, mean body mass index 26.6 ± 5.3 kg/m²). There was moderate correlation between HP and FS for CAP (Spearman's rho 0.67, p<0.001) and strong correlation for LSM (Spearman's rho 0.74, p<0.001). The mean difference (95% confidence interval, CI) between CAP obtained by HP and FS was 15 (-69 – 99) dB/m, and for LSM, this was 1.2 (-5.0 – 7.2) kPa. There was substantial agreement between HP and FS when using 10kPa and

15kPa LSM cut-offs, moderate agreement when using 20kPa and 248dB/m and 268dB/m CAP cut-offs, and fair agreement when using 280dB/m. The IQR for CAP and IQR/median for LSM were lower for HP compared with FS (12 vs 33dB/m, p<0.001, and 13 vs 14, p<0.001, respectively). The intra- and inter-observer reliability of CAP and LSM using HP and FS were good to excellent with intraclass correlation coefficients of 0.86–0.97. HP had shorter examination time (43s vs 62s, p<0.001) and less invalid measurements (0 vs 1, p<0.001).

Conclusions:

Measurements obtained with HP and FS are moderate to strongly correlated, but differences in their absolute values, consistency, and examination time were observed. However, the differences do not appear to be clinically significant. Either device can be used by healthcare personnel of different training backgrounds when sufficiently trained.

Keyword(s) : Hepatus, fibroscan, CAP, liver stiffness

2535 - Regulation and Clinical Translation of m⁶A Modification in Intrahepatic Cholangiocarcinoma

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Background / Hypothesis:

Intrahepatic cholangiocarcinoma (ICC) is a highly aggressive liver cancer with limited therapeutic options and poor prognosis. Epigenetic dysregulation, particularly N⁶-methyladenosine (m⁶A) RNA modification, has emerged as a key player in tumorigenesis. We hypothesize that m⁶A modification serves as a central regulatory mechanism in ICC progression, immune evasion, and chemoresistance, and that m⁶A-related molecules hold significant potential as diagnostic biomarkers, prognostic indicators, and therapeutic targets.

Materials and Methods:

This narrative review systematically synthesizes current evidence from published preclinical and clinical studies on m⁶A modification in ICC. Literature was retrieved from PubMed, Web of Science, and Google Scholar using keywords including "m⁶A," "intrahepatic cholangiocarcinoma," "METTL3," "ALKBH5," "YTHDF," "ferroptosis," "immunotherapy," and "chemoresistance." Data on molecular mechanisms, clinical correlations, and therapeutic interventions were extracted and thematically analyzed.

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Results:

Mechanistic Insights: m⁶A regulators (METTL3, ALKBH5, YTHDF family) modulate ICC proliferation, invasion, ferroptosis, and stemness via coding/non-coding RNA targets (e.g., IFIT2, BUB1B, PD-L1, circSLCO1B3).

Tumor Microenvironment: m⁶A modification reshapes the immune landscape by regulating PD-L1 expression, promoting immunosuppressive cell infiltration, and mediating immune escape.

Chemoresistance: The YTHDF2-CDKN1B axis drives cisplatin resistance, while METTL3-mediated metabolic reprogramming further supports chemoresistant phenotypes.

Clinical Translation:

Diagnosis: m⁶A regulators (e.g., METTL3, ALKBH5) show high diagnostic accuracy (AUC 0.78–0.85) in tissue and liquid biopsies.

Prognosis: High expression of ALKBH5, YTHDF2, etc., correlates with poor survival (HR 2.31–3.15).

Therapy: Preclinical studies demonstrate synergistic efficacy of m⁶A-targeted inhibitors (e.g., STM2457, IOX1) with chemotherapy/immunotherapy (tumor inhibition rate 58–72%).

Conclusions:

m⁶A modification is a pivotal epigenetic driver in ICC, influencing tumor biology, immune response, and treatment resistance. Its associated molecules exhibit strong clinical translation potential as non-invasive biomarkers and novel therapeutic targets. Future efforts should focus on validating these findings in large prospective cohorts and developing m⁶A-based combination therapies to improve outcomes in ICC patients.

Keyword(s) : N6-methyladenosine (m⁶A) ; Intrahepatic cholangiocarcinoma ; Tumor microenvironment ; Therapeutic resistance ; METTL3 ; ALKBH5 ; Combination therapy

2536 - ANTIFIBROTIC EFFECTS OF ANGIOTENSIN RECEPTOR BLOCKERS IN ORGANOPHOSPHATE (CHLORPYRIFOS) INDUCED LIVER FIBROSIS

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Background / Hypothesis:

Organophosphates (OPs) are one of the pesticides causing toxicity to humans and animals. OPs toxicity involve many organs such as liver, kidney nervous system and immune system. Liver fibrosis is a wound

healing response in which there is increased production and deposition of extracellular matrix (ECM), due to prolonged injury to liver by viral hepatitis, alcohol consumption, metabolic diseases and toxins (pesticides). Angiotensin receptor blockers (ARBs) are potential therapeutic agents for liver fibrosis.

Objectives: To determine the effects of ARBs on

Chlorpyrifos(CPF) induced liver fibrosis

Materials and Methods:

A Experimental study was conducted at animal house, Faculty of Animal Husbandry and Veterinary Sciences SAU, Tandojam. Laboratory work was carried out in Postgraduate Laboratory, Isra University Hospital, Hyderabad and Molecular Laboratory, SAU Tandojam, from March 2023 to August 2023. Forty adult male albino wistar rats were used in the study. Rats were grouped as control group (A1), positive control group (A2) and experimental groups (B, C and D). Group A2 was treated with carbon tetrachloride (CCL4) to induce liver fibrosis, with 2 ml/kg of 50% solution of CCL4 by oral gavage two times per week for eight weeks, group B was treated with 1ml/bw/day CPF intraperitoneally for eight weeks. Group C was treated with 1ml/bw/day CPF intraperitoneally for eight weeks and then ARBs orally (30mg/kg/day) for 8 weeks. Group D was simultaneously treated with CPF and ARBs for 8 weeks with same doses as for group C. At the end of experiment blood was collected by cardiac puncture for liver function test and rats were sacrificed to collect liver samples. Pathological changes in liver were examined using H&E and Masson's trichrome staining. Real time PCR was used to detect alpha smooth muscle actin (α -SMA), transforming growth factor beta 1(TGF- β 1) and Collagen-I(coll-I) mRNA expression.

Results:

CPF caused liver injury evidenced by raised liver enzymes and bilirubin with histological changes and increased expression of genes. ARBs decreases liver enzymes, CPF induced histological changes and decreases the expression of α -SMA, TGF- β 1 and Coll-I.

Conclusions:

The present study suggests that ARBs prevent fibrogenesis and may serve as a promising therapeutic agent to reduce CPF induced liver fibrosis

Keyword(s) : Liver fibrosis, chlorpyrifos, angiotensin receptor blockers, collagen, alpha smooth muscle actin.

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2537 - Correlation of interleukin 17 serum levels with degree of liver fibrosis in chronic hepatitis B patients at Mohammad Hoesin Hospital Palembang

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Background / Hypothesis:

Chronic hepatitis B (CHB) is a major global cause of progressive liver disease, and immune-mediated hepatic inflammation plays a critical role in driving fibrosis. Interleukin-17 (IL-17), a key cytokine produced by Th17 cells, has been implicated in hepatocellular inflammation, fibrogenesis, and disease progression in chronic liver disorders. However, data on the association between circulating IL-17 levels and liver stiffness in CHB patients remain limited. This study aimed to evaluate the correlation between serum IL-17 levels and the degree of liver fibrosis assessed using transient elastography.

Materials and Methods:

We conducted an analytical observational study with a cross-sectional design among patients with CHB at Dr. Mohammad Hoesin Hospital Palembang. Subjects were enrolled through consecutive non-probability sampling. Serum IL-17 levels were quantified, and liver fibrosis was measured using transient elastography. Continuous variables were summarized as means and ranges. The correlation between IL-17 serum levels and liver stiffness values was analyzed using Spearman's rank correlation coefficient.

Results:

A total of 40 patients were included, with a mean age of 37 years (range 22–60 years). The majority were male (75%). Serum IL-17 levels and liver stiffness values were both treated as numerical variables. Spearman's analysis demonstrated a statistically significant positive correlation between serum IL-17 concentration and liver stiffness ($r = 0.388$; $p = 0.013$).

Conclusions:

Serum IL-17 levels show a significant positive correlation with liver fibrosis severity in patients with chronic hepatitis B. IL-17 may serve as a useful non-invasive biomarker to complement elastography in assessing fibrotic progression, especially in patients in whom liver biopsy is not feasible.

Keyword(s) : interleukin-17, liver fibrosis, chronic hepatitis B, transient elastography.

2538 - Comparison of sensitivity and specificity of vascular endothelial growth factor and alpha fetoprotein levels in detecting hepatocellular carcinoma in patients with hepatitis b related liver cirrhosis at Mohammad Hoesin Hospital Palembang

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is the second leading cause of cancer-related mortality worldwide, largely due to delayed early diagnosis. In Indonesia, chronic hepatitis B-related liver cirrhosis is the most common underlying condition for HCC. Alpha-fetoprotein (AFP) is widely used as a diagnostic biomarker; however, its sensitivity and specificity remain suboptimal. Therefore, this study aimed to evaluate vascular endothelial growth factor (VEGF) as an alternative biomarker and to compare its diagnostic performance with AFP in detecting HCC.

Materials and Methods:

This diagnostic study included 52 patients with hepatitis B-related liver cirrhosis treated at Dr. Mohammad Hoesin General Hospital, Palembang, between August and November 2023. Blood samples were collected to measure serum VEGF and AFP levels. The diagnosis of HCC was confirmed using triphasic abdominal computed tomography based on the 2023 HCC management guidelines. Statistical analyses were performed using SPSS version 29.

Results:

Among the 52 subjects, AFP demonstrated a sensitivity of 62.96% and a specificity of 84% for diagnosing HCC. In comparison, VEGF showed a higher sensitivity of 74.07% but a lower specificity of 68%.

Conclusions:

VEGF exhibits higher sensitivity than AFP in detecting hepatocellular carcinoma among patients with hepatitis B-related liver cirrhosis, although its specificity is lower. VEGF may serve as a

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complementary diagnostic biomarker alongside AFP for HCC detection

Keyword(s) : vascular endothelial growth factor, alpha-fetoprotein, hepatocellular carcinoma, liver cirrhosis, hepatitis B

2539 - Association Between Clinical Predictors And Albumin Billirubin Score In Hepatocellular Carcinoma Patients Receiving Lenvatinib At Dr. Mohammad Hoesin General Hospital Palembang During 2025

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Background / Hypothesis:

Lenvatinib is widely used as a first-line systemic therapy for hepatocellular carcinoma (HCC), but treatment responses vary substantially among patients. Clinical predictors such as hepatic functional reserve and performance status may influence treatment outcomes. This study examined the relationship between Child–Pugh class, ECOG performance status, and Albumin bilirubin (ALBI) score in patients with HCC receiving lenvatinib at a tertiary referral center in Indonesia.

Materials and Methods:

A retrospective review was conducted using complete records of HCC patients treated with lenvatinib in 2025. Data collected included Child–Pugh classification, ECOG performance status, and ALBI score. Descriptive statistics were used to summarize patient characteristics. Pearson correlation analysis was applied to assess associations between clinical predictors and ALBI scores.

Results:

Twenty patients were eligible for analysis. Most patients were classified as Child–Pugh A or B, and ECOG performance status was predominantly 0–1. ALBI scores clustered mainly within grades 1 and 2. Better hepatic reserve (Child–Pugh A) and ECOG 0 were consistently associated with more favorable ALBI grades. Significant correlations were observed between Child–Pugh class and ALBI ($r = 0.79$), with a weaker correlation between ECOG status and ALBI ($r = 0.41$). These results indicate alignment between clinical assessments of hepatic function and biochemical liver

status.

Conclusions:

Child–Pugh classification and ECOG performance status correlate with ALBI score in patients receiving lenvatinib for HCC. Patients with better preserved liver function and performance status demonstrated more favorable ALBI profiles. These findings support the use of ALBI as an objective complement to established clinical indicators when assessing suitability for lenvatinib therapy and planning treatment strategies.

Keyword(s) : Hepatocellular Carcinoma, Clinical Predictors, ALBI score, Lenvatinib

2540 - Fulminant Hepatic Failure Following Antiviral Therapy Discontinuation in Chronic Hepatitis B A Fatal Case

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Background / Hypothesis: Chronic hepatitis B requires long-term antiviral therapy to prevent disease progression and hepatic decompensation. Discontinuation of treatment may lead to severe HBV reactivation with fatal outcomes. We aimed to present a fatal case of antiviral therapy discontinuation and emphasize the importance of treatment adherence.

Materials and Methods: This poster presents a retrospective review of clinical, laboratory, imaging, and treatment data of a patient with chronic hepatitis B. Disease severity was assessed using Child–Pugh classification.

Results: A 44-year-old male with chronic hepatitis B infection, hypertension, and diabetes mellitus presented with fatigue, nausea, and vomiting lasting for two weeks. He had been known to be HBsAg-positive for four years but had no regular follow-up. Family history was notable for hepatitis B infection in his siblings. The patient denied alcohol consumption.

Physical examination revealed jaundice and ascites, without overt hepatic encephalopathy. Laboratory evaluation showed severe hepatic injury (AST 1189 U/L, ALT 1162 U/L), cholestasis (total bilirubin 12.26 mg/dL),

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and coagulopathy (INR 1.58). Platelet count was 147,000/mm³, albumin 2.98 g/dL, and creatinine 0.50 mg/dL. The patient was classified as Child–Pugh class C. Serologic testing revealed HBsAg positivity, anti-HBe positivity, negative HDV serology, and an HBV DNA level of 7.7×10^6 IU/mL.

Hospitalization was recommended due to severe hepatic dysfunction; however, the patient refused admission and opted for outpatient follow-up. Due to impaired coagulation parameters, liver biopsy was contraindicated, and tenofovir disoproxil fumarate (300 mg once daily) was initiated. Abdominal ultrasonography demonstrated hepatomegaly and diffuse ascites. Upper gastrointestinal endoscopy revealed esophageal varices, and non-selective beta-blocker therapy was started.

At the one-month follow-up, marked biochemical and virologic improvement was observed (AST 59 U/L, ALT 59 U/L, INR 1.25, HBV DNA 44 IU/mL). After several months of follow-up, the patient discontinued antiviral therapy and clinic visits.

Approximately one year later, he re-presented with recurrent symptoms. Laboratory findings indicated severe HBV reactivation and acute liver failure (ALT 2160 U/L, AST 2072 U/L, total bilirubin 10.81 mg/dL, INR 1.81). Ascites persisted, hepatic encephalopathy developed, and despite re-initiation of antiviral therapy, the patient progressed to fulminant hepatic failure and died while awaiting liver transplantation.

Conclusions: Discontinuation of antiviral therapy in chronic hepatitis B may cause fatal HBV reactivation and fulminant hepatic failure, even after initial clinical improvement, highlighting the critical importance of lifelong treatment adherence and patient education.

Keyword(s) : Chronic hepatitis B; antiviral therapy; treatment non-adherence; HBV reactivation; acute liver failure; fulminant hepatic failure; patient education

2542 - Construction of an imaging omics machine learning prediction model for post liver transplantation recurrence of liver cancer based on ECV

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Background / Hypothesis:

This study aims to establish an imaging biomarker model related to the prognosis of liver transplant patients based on the extracellular volume (ECV) subtraction images obtained from abdominal enhanced CT scans before liver transplantation. This is intended to further clarify the predictive role of ECV subtraction images in liver cancer patients after liver transplantation.

Materials and Methods:

A total of 145 patients with hepatocellular carcinoma who underwent liver transplantation were retrospectively included and randomly divided into a training set and a test set at a ratio of 7:3. The radiomics features of the tumors and the surrounding tissues of the patients' preoperative enhanced CT plain scan, delayed scan, and ECV subtraction images were extracted. After feature selection, the optimal machine learning algorithm was used to construct the ECV-ML model, and a combined model was established by combining clinical factors. The performance of the model was comprehensively evaluated using C-index, decision curve, calibration curve, and survival analysis.

Results:

In the clinical model, the ratio of monocytes to lymphocytes and microvascular infiltration are independent risk factors for poor prognosis in liver cancer patients after liver transplantation. Using LASSO regression under ten-fold cross-validation, a total of 6 optimal features were selected, including 2 features of peri-tumor subtraction images, 2 features of peri-tumor plain scan images, and 2 features of tumor delayed images. In the ECV-ML model, the algorithm based on XGBoost has the best predictive effect for early recurrence after transplantation. The combined model and the ECV-ML model both performed better than the clinical model in predicting early postoperative recurrence (the C-index of the training set was 0.86, 0.85, and 0.61 respectively, and the C-index of the test set was 0.83, 0.82, and 0.65 respectively), but there was no significant difference between the two models.

Conclusions:

The ECV-ML model based on preoperative ECV parameter graphs and CT images demonstrates effective predictive performance in predicting the early

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recurrence of HCC patients after liver transplantation, and can significantly improve the predictive performance of the simple clinical parameter-based liver cancer liver transplantation recurrence prediction model.

Keyword(s) : Machine learning; Hepatocellular carcinoma; Liver transplantation; Extracellular volume; Microvascular invasion

2544 - Comparison of Predictive Accuracy of Monocyte to Lymphocyte Ratio (MLR) Neutrophil To Lymphocyte Ratio (NLR) and Systemic Immune Inflammation Index (SII) for Colonoscopic and Clinical Outcomes among Patients with Gastrointestinal Tuberculosis (GITB)

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Background / Hypothesis: Gastrointestinal tuberculosis (GITB) has heterogeneous endoscopic manifestations, and reliable predictors of disease severity and treatment response remain limited. Inflammatory biomarkers derived from routine blood tests, including the monocyte-to-lymphocyte ratio (MLR), neutrophil-to-lymphocyte ratio (NLR), and systemic immune-inflammation index (SII), may reflect disease activity.

Materials and Methods:

This prospective cohort study included 103 patients diagnosed with GITB who underwent colonoscopy at a tertiary referral center. Baseline clinical, laboratory, and colonoscopic data were collected. MLR, NLR, and SII were compared across colonoscopic patterns. Logistic regression was performed to assess associations with treatment response, and receiver operating characteristic (ROC) curves were used to evaluate predictive accuracy.

Results: MLR, NLR, and SII differed significantly across colonoscopic patterns ($p < 0.05$), with higher values observed in ulcero-hypertrophic lesions. In univariate analysis, NLR (OR: 1.24, $p = 0.009$) and SII (OR: 1.01, $p = 0.005$) were associated with poor response to treatment; however, these associations were not significant after adjustment for other confounders. ROC analysis showed poor discriminatory performance for all biomarkers, with AUC values of 0.544 for MLR, 0.534 for NLR, and 0.604 for SII.

Conclusions: Although novel inflammatory biomarkers

vary across colonoscopic phenotypes in GITB, their predictive value for treatment response is limited. These markers may serve as adjunctive indicators but should not replace established clinical and endoscopic assessments.

Keyword(s) : Gastrointestinal tuberculosis; inflammatory biomarkers; neutrophil-to-lymphocyte ratio; systemic immune-inflammation index; colonoscopy

2545 - COMPARISON OF CLINICAL OUTCOME AND ENDOSCOPIC FINDINGS BETWEEN ACIDIC AND ALKALI CAUSTIC INJURIES IN A TERTIARY REFERRAL HOSPITAL

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Background / Hypothesis: Caustic injuries present a significant public health concern due to the severe tissue damage they can inflict. This current study evaluated the endoscopic findings and clinical outcomes of patients with acidic and alkali caustic injuries.

Materials and Methods:

In this retrospective cohort study, we performed a posteriori, using information and data from chart review of 241 consecutive patients admitted in a tertiary government hospital due to caustic injuries. We compared patients with acidic and alkali caustic injuries based on Zargar's grading classification of mucosal injury and their clinical outcomes.

Results:

Ingestion of alkali was more prevalent than ingestion of acidic caustic substances (63.9% vs 36.1%). Weak base (61.83%) was the most common caustic substance ingested followed by weak acid (19.09%), strong base (2.07%), and weak base (0.41%). At baseline, alkali and acid groups varied significantly in terms of age ($p = 0.029$), sex ($p = 0.001$), marital status ($p = 0.009$) and initial presentation ($p < 0.001$). Ingestion of acid was associated with significantly higher grades of esophageal ($p < 0.001$), stomach ($p < 0.001$), and duodenal ($p = 0.001$) injuries. A significantly higher proportion of patients in the acid group required NGT insertion ($p < 0.001$), endotracheal intubation ($p = 0.001$) and antibiotic therapy ($p = 0.001$) than the alkali group. Finally, acid ingestion was associated with a higher rate of esophageal stenosis ($p = 0.008$) and longer length of hospital stay ($p = 0.005$).



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Conclusions: While caustic injuries were predominant alkali, acids may lead to severe injury and complications following caustic ingestion. Early endoscopy is crucial for assessing severity, guiding management, and predicting patient outcomes.

Keyword(s) : Caustic injury, alkali, acid, endoscopy, severity, outcomes

2546 - Use of Double Plasma Molecular Adsorption System (DPMAS) in Intrahepatic Cholestasis of Pregnancy Refractory to Medical Management A Case Report

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Background / Hypothesis: Intrahepatic cholestasis of pregnancy (ICP) is a pregnancy-specific liver disorder characterized by pruritus, elevated bile acids, and abnormal liver function tests, typically resolving after delivery². The main treatments for intrahepatic cholestasis of pregnancy (ICP) are medication with ursodeoxycholic acid (UDCA), monitoring of the mother and fetus, and potentially early delivery. This case documents a novel, safe, and effective application of DPMAS in a pregnant patient with severe ICP unresponsive to UDCA, demonstrating rapid bile acid reduction, symptom resolution, and favorable maternal-fetal outcomes.

Materials and Methods: A 36-year-old G3P1 (1011) was admitted at 15 weeks of gestation with pruritis, tea colored urine, and jaundice. On admission, laboratory results showed hyperbilirubinemia at 24.6 mg/dl (Direct Bilirubin: 19.8 mg/dl Indirect Bilirubinemia 4.4), and transaminitis (ALT 340 AST 821). Initial PT, PTT, Alkaline Phosphatase, GGT are within normal limits. Whole abdominal ultrasound, hepatitis serologies, autoimmune markers, and metabolic workup were all unremarkable. She was managed as a case of Early Onset Intrahepatic Cholestasis of Pregnancy. She was started with Ursodeoxycholic acid (UDCA) 15 mg/kg/day, and Glycyrrhizinate drip. Serial bile acid levels and liver enzymes were monitored with regular fetal surveillance. However despite medical management, pruritus persisted with worsening nocturnal symptoms.

Results:

Given her refractory ICP, the multidisciplinary team

(Obstetrics, Hepatology, and Nephrology) decided to initiate Double Plasma Molecular Adsorption System (DPMAS) therapy to rapidly reduce circulating bile acids and improve maternal symptoms. The patient underwent DPMAS for three consecutive sessions. After the third session total bile acids decreased to 8mg/dl. Jaundice and pruritus significantly improved and ALT and AST levels decreased to near-normal ranges. She was maintained on UDCA post-DPMAS, with close fetal monitoring until delivery.

Conclusions: In refractory severe ICP, DPMAS represents a promising adjunctive therapy when UDCA fails. Given the scarcity of such reports, it expands therapeutic possibilities, informs clinical decision-making, and lays groundwork for future studies on extracorporeal liver support in pregnancy.

Keyword(s) : Cholestasis of Pregnancy; DPMAS

2547 - When the Liver Speaks for the Vessels A Diagnostic Odyssey of a rare case of Non HBV Associated Polyarteritis Nodosa Presenting with Jaundice and Transaminitis

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Background / Hypothesis: Polyarteritis nodosa (PAN) is a rare necrotizing vasculitis of medium-sized arteries that classically produces systemic symptoms, multisystem ischemia, and visceral microaneurysms. Presentation is protean and diagnosis requires synthesis of clinical, laboratory, and angiographic data. Hepatic involvement with transaminitis and jaundice is an uncommon initial manifestation and can obscure the underlying vasculitic etiology.

Materials and Methods:

A 22-year-old Filipino male with no significant past medical history presented with six weeks of progressive malaise followed by migratory polyarthralgia/oligoarthritis and jaundice. Initial symptoms included transient knee warmth, severe crampy lower abdominal pain with undocumented fever, progressive proximal joint swelling and weakness, and eventual scleral icterus. Examination revealed icteric sclera, hepatomegaly without focal tenderness, and warmth/swelling of multiple joints. Laboratory evaluation demonstrated leukocytosis (WBC

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25.3 $\times 10^9/L$, anemia (Hgb 10.0 g/dL), markedly elevated ALT (471 U/L), AST elevation, direct hyperbilirubinemia (total 3.95 mg/dL, direct 2.98 mg/dL), hypoalbuminemia, and markedly elevated inflammatory markers (ESR 144 mm/hr, CRP 160 mg/L). Viral hepatitis and autoimmune serologies were negative. Contrast-enhanced CT angiography revealed multiple enhancing nodular lesions and saccular dilatations/microaneurysms in branches of the hepatic and renal arteries, consistent with medium-vessel vasculitis.

Conclusions: This case illustrates an atypical presentation of PAN with prominent hepatic dysfunction and polyarthritis in a young adult. Angiographic identification of visceral microaneurysms was pivotal to diagnosis. Early immunosuppressive therapy led to clinical resolution, emphasizing the importance of considering medium-vessel vasculitis in patients with unexplained transaminitis and systemic inflammatory features.

Keyword(s) : Polyarteritis Nodosa

2548 - Multiverse of Upper Gastrointestinal Tuberculosis A case series of GITB with involvement of Esophagus Gastric and Duodenal TB

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Background / Hypothesis: This case series presents five patients diagnosed with upper gastrointestinal tuberculosis (GI TB), presenting from esophagus to duodenum, comprising four males and a female, aged between 19 and 50 years. Among these patients, 1 is HIV reactive. Each patient sought consultation for a range of gastrointestinal symptoms, including abdominal pain, weight loss, jaundice, hematemesis, and ascites. Common diagnostic tests employed included imaging studies such as CT scans and endoscopic evaluations, which revealed various findings such as abdominal lymphadenopathy and masses in the gastrointestinal tract. Notably, the cases highlighted the atypical presentations of upper GI TB which are less frequently reported in the literature.

Materials and Methods: The case series presented six distinct cases of gastrointestinal tuberculosis, each exhibiting varying manifestations that challenge traditional diagnostic paradigms. Case 1 involved a 32-year-old male with esophageal and duodenal TB, while

Case 2 featured a 32-year-old female with gastric TB presenting as a submucosal mass. Case 3 documented a 50-year-old male with hematemesis, and Case number 4 presented with melena. Case 5 presented with a fistula from esophagus leading to mediastinal mass. These diagnoses were confirmed through a combination of endoscopic biopsy results and imaging studies, which indicated chronic granulomatous inflammation consistent with TB.

Results: Treatment for all patients involved a standard regimen of antitubercular therapy, which included first-line medications such as isoniazid, rifampicin, ethambutol, and pyrazinamide. The outcomes varied, with most patients demonstrating significant clinical improvement and resolution of symptoms over the course of treatment. Notably, both patients presenting with fistula had complicated clinical course leading to demise. The overall response to treatment underscores the importance of early diagnosis and intervention in managing gastrointestinal tuberculosis effectively.

Conclusions: In conclusion, this case series emphasizes the diverse presentations of gastrointestinal tuberculosis, highlighting the necessity for clinicians to maintain a high index of suspicion when faced with unexplained gastrointestinal symptoms. The findings advocate for a comprehensive diagnostic approach, including imaging and endoscopic evaluations, to facilitate timely diagnosis and treatment. Furthermore, the series underscores the value of a multidisciplinary management strategy, integrating gastroenterology and infectious disease expertise to optimize patient outcomes. Future research should focus on larger, multicenter studies to validate these findings and further explore the mechanisms underlying the varied manifestations of GI TB, as well as the impact of drug resistance on treatment efficacy. By addressing these areas, the medical community can enhance understanding and improve management strategies for this complex condition.

Keyword(s) : GITB

2549 - Outcome Of Patients Presenting With Upper Gastrointestinal Bleeding A Single High Volume Center Prospective Study

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Background / Hypothesis:

This study aims to determine the outcome of patients presenting with upper gastrointestinal bleeding: A Single High-Volume Center Prospective

Materials and Methods:

This prospectus on-going hospital-based study conducted at Jinnah Postgraduate Medical Center (JPMC) Karachi, Pakistan, in the Department of Gastroenterology & Hepatology ward 25, on all the patients presenting with UGIB due to any cause, aged >16 years of either gender in one-year duration. A total number of patients were 521 in whom outcome of such patients were evaluated.

Results:

Our study subjects had mean age of 41.49 ± 9.02 years with higher percentage of males (61.03%, n = 318). Urban dwellers were more common than rural dwellers, 57.96% (n = 302) vs. 42.03% (n = 219), respectively. The most common cause of UGIB was peptic ulcer disease (18.80%, n = 98), followed by use of NSAIDs (14.77%, n = 77), and viral hepatitis leading esophageal varices (n = 10.36%, n = 54). In multivariate analysis, UGIB due to oesophageal varices appeared significantly more likely (P = 0.01) to have poor outcome (OR = 13.5, 95% CI: 3.22, 8.35).

Conclusions:

Higher chances of re-bleed and mortality was observed in patients presented with UGIB due to esophageal varices and patients with peptic ulcer disease were responsive to treatment and good overall outcome.

Keyword(s) : Upper GI bleed, Liver disease, Outcome, Southeast Asia

2550 - PROGNOSTIC VALUE OF THE ALBUMIN BILIRUBIN SCORE FOR VARICEAL REBLEEDING AND HEPATIC ENCEPHALOPATHY RELATED HOSPITALIZATION IN CIRRHOTIC PATIENTS WITH CHRONIC HBV INFECTION

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Background / Hypothesis:

Variceal rebleeding and hepatic encephalopathy (HE) are major complications in cirrhotic patients with chronic hepatitis B virus (HBV) infection, increasing morbidity, mortality, and hospitalization. The Albumin-Bilirubin (ALBI) score serves as an objective measure of liver function and may act as a prognostic biomarker for adverse liver outcomes. This investigation assesses the predictive accuracy of the ALBI score for variceal rebleeding and HE-related hospitalization.

Materials and Methods:

A retrospective cohort study was conducted on 162 cirrhotic patients with chronic HBV infection admitted for variceal bleeding between January 2022 and December 2024 at Mohammad Hoesin Hospital Palembang. Baseline ALBI scores were calculated, and patients were followed for 12 months to assess the incidence of variceal rebleeding and HE-related hospitalizations. Prognostic value of the ALBI score was assessed using Kaplan-Meier survival analysis and Cox proportional hazards regression.

Results:

The mean age of the study population was 51.0 ± 9.3 years, with 110 (67.8%) males. Variceal rebleeding occurred in 59 (36.7%) patients, while 42 (26.1%) were admitted to hospital related to HE. Patients who experienced variceal rebleeding had significantly higher ALBI scores at baseline (-1.96 ± 0.42 vs. -2.45 ± 0.39 , p < 0.001) and also hospitalized for HE (-1.89 ± 0.38 vs. -2.41 ± 0.36 , p = 0.002). ROC analysis demonstrated that the ALBI score was a strong predictor of variceal rebleeding (AUC = 0.81, 95% CI: 0.74–0.87, p < 0.001) and HE-related hospitalization (AUC = 0.78, 95% CI: 0.70–0.85, p = 0.003). Multivariate Cox regression confirmed that the ALBI score independently predicted variceal rebleeding (HR = 2.71, 95% CI: 1.87–3.91, p < 0.001) and HE-related hospitalization (HR = 2.38, 95% CI: 1.62–3.48, p < 0.001).

Conclusions:

The ALBI score is a valuable independent predictor of variceal rebleeding and HE-related hospitalization in cirrhotic patients with chronic HBV infection. Its integration into clinical risk stratification may improve patient management and outcomes.

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Keyword(s) : Albumin-Bilirubin score, cirrhosis, chronic hepatitis B, variceal rebleeding, hepatic encephalopathy, prognosis, survival.

2551 - Noninvasive Detection of Metabolic dysfunction associated steatohepatitis and Fibrotic Burden Using Ultrasound Derived LIID and liver stiffness measurement in Metabolic dysfunction associated steatotic liver disease A Multicenter Study.

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Background / Hypothesis:Metabolic dysfunction-associated steatohepatitis (MASH), the progressive form of metabolic dysfunction-associated steatotic liver disease (MASLD), is associated with adverse hepatic and cardiometabolic outcomes. The study aimed to validate a novel quantitative ultrasound (QUS)-derived score, the LIID score, for noninvasive diagnosis of MASH, and to evaluate the performance of liver stiffness measurement (LSM) using the same ultrasound platform for detecting fibrotic burden in MASLD.

Materials and Methods:This multicenter study enrolled 438 participants with MASLD who underwent both iLivTouch examination and liver biopsy between November 2018 and December 2024 from ten hospitals in China. After the exclusion criteria were applied, a total of 410 patients with biopsy-proven MASLD and successful QUS measurements were included to assess the performance of the LIID score. According to radiofrequency (RF) data acquired from iLivTouch, a total of 18 ultrasonographic features were obtained with different time gain compensation and named as P1 to P18 based on the intensity, attenuation and scattering characteristics of the ultrasound signals, including LSM (P3) and UAP (P1).(Figure 1A) P2-P10 are mainly related to intensity, whereas P11-P18 are mainly related to scattering. The LIID score was derived from the RF data, including information on the intensity, frequency, scattering, and attenuation characteristics of the ultrasound signals. The formula of the LIID score was as follows: $LIID = 0.063 \times P7 + 0.019 \times P13$.

Results: The LIID score demonstrated an area under the receiver operating characteristic curve (AUROC) of 0.71 (95% CI: 0.66–0.76) for detecting MASH. A rule-out threshold of 6.0 achieved high sensitivity (89.6%) and negative predictive value (78.2%), while a rule-in threshold of 7.8 provided high specificity (88.4%) and positive predictive value (70.5%). The LIID score increased in parallel with histological disease activity, as assessed by the NAS score ($p < 0.01$, Figure 1B). LSM showed excellent diagnostic performance for advanced fibrosis (AUROC: 0.86; 95% CI: 0.79-0.92), and acceptable accuracy for significant fibrosis (AUROC: 0.75; 95% CI: 0.69-0.79) and cirrhosis (AUROC: 0.78; 95% CI: 0.61-0.95). In addition, LSM values correlated positively with histological fibrosis stage (F0-F4), with a statistically significant trend ($p < 0.01$, Figure 1C).

Conclusions:The LIID score offers a noninvasive, ultrasound-based approach for identifying MASH. Combined with LSM, this strategy enables comprehensive, noninvasive assessment of hepatic inflammation and fibrosis in MASLD and may reduce the need for liver biopsy in clinical practice.

Keyword(s) : MASLD, MASH, noninvasive diagnosis, quantitative ultrasound, fibrosis

2552 - DPMAS Induced Stabilization in a Terminal Patient Short Term Benefit in the Face of Terminal Prognosis

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Background / Hypothesis:

Acute-on-chronic liver failure (ACLF) is a rapidly progressive condition with a high mortality rate ranging from 50% to 90%. Artificial liver support systems, such as the double plasma molecular adsorption system (DPMAS), are used to stabilize patients, support failing organs, and serve as a bridge to recovery or transplantation. This case highlights the transient recovery following DPMAS in a patient with a known terminal condition.

Materials and Methods:

This report features a 56-year-old woman with decompensated liver cirrhosis (Child-Pugh C, MELD-Na 32) due to chronic hepatitis C, complicated by hepatocellular carcinoma, portal hypertension, and portal vein thrombosis. She had previously received multiple treatments including immunotherapy

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(Tremelimumab + Durvalumab) and tyrosine kinase inhibitors (Lenvatinib, Cabozantinib, Sorafenib). She presented with worsening jaundice, hepatic encephalopathy, coagulopathy, ascites, electrolyte imbalance, and multi-organ dysfunction, indicating acute decompensation. Despite multiple lines of therapy, her disease continued to progress, and she remained aggressive in pursuing treatment despite her terminal status.

Results:

Initial labs showed marked hyperbilirubinemia, transaminase elevation ($>25\times$ normal), hypoalbuminemia, pancytopenia, acute kidney injury (creatinine 2.05 mg/dL, elevated BUN), hyponatremia (Na 128 mEq/L), prolonged PT (24.5s), and increased INR (2.17). She underwent four DPMAS sessions. Although she remained lethargic and jaundiced, post-treatment labs showed significant biochemical improvement. However, following the fourth session, her laboratory values began to rise again, and her clinical condition worsened.

Conclusions:

This case illustrates that while DPMAS may not reverse terminal liver disease, it can temporarily improve laboratory markers and stabilize clinical parameters. Its role is palliative and supportive, providing time for further decision-making in patients with ACLF, especially when transplantation is not an option.

Keyword(s) : DPMAS, acute-on-chronic liver failure, bilirubin, artificial liver support system

2555 - A Preclinical study on Protective Effects of Atorvastatin Against Methotrexate Induced Hepato Renal Toxicity Role of Oxidative Stress and Inflammation

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Background / Hypothesis:

Methotrexate (MTX) is widely used as an immunosuppressive and anticancer agent; however, its clinical utility is often limited by dose-dependent hepatotoxicity and nephrotoxicity. Accumulating evidence suggests that oxidative stress and inflammation are central to MTX-induced organ injury. Atorvastatin, a commonly prescribed HMG-CoA reductase inhibitor, exhibits pleiotropic antioxidant and

anti-inflammatory properties beyond lipid lowering. The present study investigated the potential hepatoprotective and nephroprotective effects of atorvastatin in an experimental model of MTX-induced toxicity and compared its efficacy with silymarin, a standard reference drug.

Materials and Methods:

Wistar albino rats were divided into six groups (n=6): normal control, atorvastatin alone (10 mg/kg), MTX control (20 mg/kg, single intraperitoneal dose), MTX plus atorvastatin (3 mg/kg), MTX plus atorvastatin (10 mg/kg), and MTX plus silymarin (300 mg/kg). Atorvastatin and silymarin were administered orally for 7 days prior to and 21 days following MTX administration. Hepatic and renal function was assessed using serum biochemical markers. Oxidative stress parameters (TBARS, GSH, SOD) and inflammatory mediators (nitrite, MPO, TNF- α , PGE₂) were evaluated in liver and kidney tissues. Histopathological examination was performed to assess structural damage.

Results:

Methotrexate administration resulted in significant hepato-renal injury, evidenced by deranged serum biochemical markers, increased oxidative stress, elevated inflammatory mediators, and pronounced histopathological alterations in both liver and kidney tissues. Atorvastatin treatment significantly attenuated MTX-induced inflammatory responses, as demonstrated by reduced tissue levels of TNF- α , MPO, COX-2, and PGE₂ in both organs. However, in the liver, atorvastatin was associated with a paradoxical increase in oxidative stress, accompanied by persistent elevation of liver function parameters and disruption of redox homeostasis. In contrast, renal tissues showed improvement in oxidative stress markers and functional indices following atorvastatin treatment. Histopathological evaluation supported the biochemical findings, revealing partial restoration of tissue architecture, with more pronounced protection observed in the kidney. Collectively, these results indicate that atorvastatin exerts organ-specific effects in MTX-induced toxicity, providing anti-inflammatory benefits while potentially promoting pro-oxidant effects in the liver under conditions of MTX-associated oxidative stress.

Conclusions:

These findings demonstrate that methotrexate induces

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significant hepato-renal toxicity mediated by oxidative stress and inflammation. Atorvastatin exhibits marked anti-inflammatory effects in both liver and kidney; however, its influence on oxidative homeostasis is organ-specific. While clear renoprotection is observed, atorvastatin may exacerbate hepatic oxidative stress and functional impairment under methotrexate-induced conditions. This context-dependent response warrants caution when considering atorvastatin as an adjunct to methotrexate therapy and highlights the importance of organ-specific safety evaluation.

Keyword(s) :

2556 - Clinical Benefit of Perioperative Prophylactic Antibiotics in Hepatocellular Carcinoma (HCC) Patients Undergoing Transcatheter Arterial Chemoembolization (TACE)

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Background / Hypothesis:

Clinical evidence supporting the use of prophylactic antibiotics to prevent infection and improve outcomes in patients undergoing transarterial chemoembolization (TACE) for hepatocellular carcinoma (HCC) remains limited. This study aimed to evaluate the association between perioperative prophylactic antibiotic administration and the risk of post-TACE infection.

Materials and Methods:

This retrospective cohort study included HCC patients who underwent TACE between January 2022 and December 2025. Patients in the prophylactic antibiotic group received 1.5 g of cefuroxime sodium 30 minutes prior to TACE, while those in the control group received no prophylactic antibiotic intervention. The primary outcomes were the incidence of infection within 30 days postoperatively, defined by procalcitonin (PCT) ≥ 0.66 ng/mL or C-reactive protein (CRP) ≥ 28 mg/L. Secondary outcomes included the diagnosis of systemic inflammatory response syndrome (SIRS), total hospitalization cost, and length of hospital stay (LOS). Outcomes were compared between the two groups to assess the necessity of prophylactic antibiotics for TACE.

Results:

A total of 286 eligible patients were enrolled, with 123 receiving prophylactic antibiotics and 163 not. No statistically significant differences were observed between the antibiotic group and the non-antibiotic

group in the incidence of infection (defined by PCT ≥ 0.66 ng/mL: $p=0.142$; defined by CRP ≥ 28 mg/L: $p=0.679$) within 30 days postoperatively. Additionally, there were no significant differences in the rate of SIRS ($p=0.097$), total hospitalization cost ($p=0.475$), or LOS ($p=0.413$) between the two groups.

Conclusions:

Perioperative prophylactic use of antibiotics in patients undergoing TACE is not significantly associated with a reduced risk of postoperative infection, shorter LOS, or lower hospitalization costs. Large-scale prospective studies and randomized controlled trials (RCTs) are warranted to further validate these findings.

Keyword(s) : Prophylactic Antibiotic Therapy; HCC; TACE; infection

2557 - Silybin Attenuates Liver Fibrosis by Suppressing Hepatic Stellate Cell Activation

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Background / Hypothesis: Liver fibrosis is a common pathological consequence of chronic liver injury, primarily driven by the persistent activation of hepatic stellate cells (HSCs). Silybin, a natural flavonolignan derived from *Silybum marianum*, is widely recognized for its hepatoprotective properties; however, its direct effects on hepatic fibrogenesis remain incompletely characterized. This study aimed to investigate the anti-fibrotic efficacy of silybin and its specific impact on HSC activation both in vivo and in vitro.

Materials and Methods: To evaluate in vivo efficacy of silybin, a mouse model of liver fibrosis was induced via bile duct ligation (BDL). Liver injury and fibrosis were assessed via serum aminotransferase levels, Sirius Red staining, hepatic hydroxyproline content, and the expression of fibrosis-related proteins. In vitro, the human HSC line LX-2 was utilized as a model of fibrogenic activation. Cells were stimulated with

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transforming growth factor- β (TGF- β) and treated with graded concentrations of silybin. Cellular contractility, proliferation, and migration were assessed by collagen gel contraction assay, EdU incorporation assay, and Transwell migration assay, respectively.

Results: In vivo, silybin administration significantly attenuated liver injury, as evidenced by reduced serum ALT and AST levels. Furthermore, histological analysis demonstrated a marked decrease in collagen deposition and fibrotic area in silybin-treated mice. Consistently, hepatic hydroxyproline content and α -SMA protein expression were significantly reduced (Figure 1). In vitro, activated HSCs exhibited enhanced contractile activity, proliferation, and migratory capacity. Silybin treatment effectively suppressed collagen gel contraction, reduced the number of EdU-positive cells, and inhibited cell migration (Figure 2).

Conclusions: Silybin exerts potent anti-fibrotic effects by inhibiting the activation, proliferation, and migration of hepatic stellate cells, consequently limiting extracellular matrix accumulation. These findings provide experimental evidence supporting silybin as a potential therapeutic agent for the treatment of liver fibrosis.

Keyword(s) : liver fibrosis, silybin, hepatic stellate cells

2558 - Diagnostic Performance of Baveno VII Criteria in a Real World Multicenter Cohort Across Liver Disease Etiologies

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Background: Accurate non-invasive diagnosis of

compensated advanced chronic liver disease (cACLD) is essential for risk stratification across the spectrum of chronic liver diseases. Although liver stiffness measurement (LSM) by elastography is a cornerstone of assessment, debate persists regarding the optimal diagnostic thresholds for cACLD, particularly across different devices and etiological backgrounds. This study aimed to perform a practical, real-world validation of the widely adopted Baveno VII criteria (10 and 15 kPa) using the iLivTouch elastography system in a large, etiologically diverse patient cohort.

Methods: In a multicenter retrospective study, 1123 patients with various chronic liver diseases (including chronic hepatitis B [41.1%], NAFLD [25.1%], autoimmune liver disease [21.6%], and others) underwent both liver biopsy (histological cACLD defined as $\geq S3$) and iLivTouch liver stiffness measurement. Diagnostic performance was evaluated using receiver operating characteristic (ROC) analysis. Model calibration was assessed with calibration curves, and clinical utility was evaluated via decision curve analysis (DCA) and clinical impact curves (CIC). Optimal dual thresholds were derived based on 90% sensitivity/specificity criteria.

Results: iLivTouch demonstrated superior diagnostic accuracy for cACLD compared to FIB-4 and APRI, with an area under the ROC curve (AUC) of 0.84. Calibration curves indicated excellent agreement between iLivTouch predictions and observed outcomes. DCA and CIC confirmed its greater net benefit and favorable clinical impact across a range of threshold probabilities. The optimal liver stiffness thresholds were 9.75 kPa (90% sensitivity) and 14.95 kPa (90% specificity), closely aligning with the Baveno VII cut-offs (10 kPa and 15 kPa). The 10 kPa threshold provided a sensitivity of 88.8% and a negative predictive value (NPV) of 92.9%, while the 15 kPa threshold achieved a specificity of 90.3%. iLivTouch performed consistently well across diverse etiologies, maintaining an AUC > 0.80 in all subgroups.

Conclusion: This large, multicenter study provides practical validation that the Baveno VII criteria can be effectively applied using the iLivTouch system. iLivTouch offers reliable, etiology-independent performance for diagnosing cACLD, supporting its integration into clinical practice for risk stratification across diverse settings.

Keyword(s) : Compensated advanced chronic liver disease, cACLD, transient elastography, Baveno VII

2561 - Correlation Between Platelet Derived Growth Factor BB As Liver Fibrosis Biomarker and



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Degree Of Liver Fibrosis Based On Fibroscan On Chronic Hepatitis B

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Background / Hypothesis: Liver fibrosis is scar tissue that forms due to excessive accumulation of ECM proteins due to chronic liver injury, mediated by cytokines involving complex cellular interactions, such as Platelet Derived Growth Factor BB (PDGF-BB). In Indonesia, no research has been conducted to determine the correlation between PDGFBB levels and degree of liver fibrosis based on fibroscan. The aim of this study is to assess the correlation between PDGF-BB levels and degree of liver fibrosis based on fibroscan in chronic Hepatitis B.

Materials and Methods: A cross sectional study was conducted on chronic hepatitis B population at Internal Medicine clinic of dr. Moh. Hoesin General Hospital Palembang from January to Agustus 2023. Assessment uses fibroscan to determine the degree of liver fibrosis, measured in kilopascals (kPa). PDGF-BB levels measured by ELISA.

Results: From 40 subjects, the majority of chronic hepatitis B patients was women (57,5%). The lowest of PDGF-BB level was found in chronic hepatitis B patients with fibroscan F0, and the highest PDGF-BB level in fibroscan F4. Fibroscan results are directly proportional to PDGF-BB level.

Conclusions: PDGF-BB have strong positive correlation with the degree of fibroscan in chronic Hepatitis B patients in dr. Moh. Hoesin General Hospital. The higher the range of PDGF-BB levels, the higher the degree of fibroscan in chronic Hepatitis B patients.

Keyword(s) : liver fibrosis, fibroscan, PDGF-BB

2563 - Is Laparoscopic Cholecystectomy Necessary at an Asymptomatic Stage

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Background / Hypothesis: Although prophylactic cholecystectomy in patients with asymptomatic gallstone is currently not recommended, increase in the elderly population and resultant increase in the occurrence of serious complications in the aged

mandates reevaluation on the necessity of this procedure. Therefore, we conducted this study to investigate the feasibility of prophylactic cholecystectomy in the elderly patients with asymptomatic gallstones.

Materials and Methods: A total of 453 patients who underwent laparoscopic cholecystectomy at Korea University Ansan Hospital during the 5 year study period were enrolled in this study. The following data were obtained by retrospectively reviewing the medical records and analyzed: sex, age, underlying diseases (diabetes, hypertension), gallbladder stone perception duration, symptom duration, admission route (emergency department vs. outpatient department), operation type (emergency vs. elective), conversion to open surgery, complications (cholecystitis-related and post-operative), readmission within 6 months after discharge, duration of hospital stay (total and post-operative), mortality, and biochemical parameters.

Results: Elderly patients more frequently were admitted through emergency department ($p=0.013$), underwent emergency operation ($p=0.019$), required conversion to open surgery ($p=0.002$), developed cholecystitis-related ($p=0.017$) and post-operative complications ($p<0.001$), were readmitted within 6 months ($p=0.021$), and succumbed to death. Moreover, the duration of total hospital stay ($\gamma=0.403$, $p<0.001$) and post-operative hospital stay ($\gamma=0.325$, $p<0.001$) showed positive correlation with increase in age.

Conclusions: Although further risk group stratification should be performed in order to justify prophylactic cholecystectomy in patients with asymptomatic gallstones, it could be necessary in the elderly patients due to higher risk of morbidity and mortality related to cholecystitis.

Keyword(s) :

2566 - Serum DCP and GALAD Score Reflect Tumor Burden and Stage Progression More Accurately Than AFP Based Markers in Intermediate and Advanced Hepatocellular Carcinoma

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Background / Hypothesis: In real-world clinical practice across Asia, hepatocellular carcinoma (HCC) is frequently diagnosed at intermediate or advanced stages, where conventional serum biomarkers such as alpha-fetoprotein (AFP) often fail to reflect tumor burden or biological disease progression. This study aimed to evaluate the ability of AFP, AFP-L3, des-gamma-carboxyprothrombin (DCP/PIVKA-II), and the GALAD score to discriminate disease stage and reflect tumor burden across Barcelona Clinic Liver Cancer (BCLC) stages, with a particular focus on intermediate and advanced HCC.

Materials and Methods: We conducted a cross-sectional analysis of 83 patients with imaging-confirmed HCC diagnosed according to national criteria aligned with EASL and AASLD guidelines. Serum AFP, AFP-L3, and DCP levels were measured, and GALAD scores were calculated. Biomarker distributions were compared across BCLC stages. Stage-discriminatory performance in patients with BCLC B or higher was assessed using area under the receiver operating characteristic curve (AUROC) analysis with pairwise comparisons.

Results: Nearly 70% of patients presented with intermediate or advanced disease (BCLC B-C). Median GALAD scores increased progressively with advancing BCLC stage (BCLC 0: 1.1; A: 4.3; B: 6.9; C: 11.0; D: 11.6; $p < 0.001$). In patients with BCLC B or higher, DCP demonstrated the highest stage-discriminatory performance (AUROC 0.964), followed by the GALAD score (AUROC 0.939), both significantly outperforming AFP and AFP-L3 ($p < 0.01$). No significant difference was observed between DCP and GALAD ($p = 0.089$). AFP-based markers showed substantial overlap across BCLC stages and limited ability to reflect disease progression.

Conclusions: In intermediate and advanced hepatocellular carcinoma, serum DCP and the GALAD score more closely reflect tumor burden and biological disease progression across BCLC stages than AFP-based biomarkers. Their superior stage-discriminatory performance supports their clinical utility for stage-informed risk stratification and disease monitoring in settings where late-stage HCC predominates.

Keyword(s) : Hepatocellular Carcinoma, DCP, GALAD score, BCLC stage, Tumor Burden

2567 - Prediabetes as an Early Clinical Indicator of Advanced Liver Fibrosis in Non Diabetic MASLD Patients A Non Invasive Risk Stratification Study

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a leading cause of chronic liver disease worldwide. In routine clinical practice, fibrosis assessment is often initiated only after the development of overt diabetes, potentially missing an earlier window for intervention. Prediabetes represents a highly prevalent intermediate metabolic state, yet its association with advanced liver fibrosis in non-diabetic MASLD remains insufficiently characterized. This study aimed to evaluate the burden of advanced fibrosis among non-diabetic MASLD patients with prediabetes and to explore the role of non-invasive assessment in early risk stratification.

Materials and Methods: This cross-sectional study enrolled 107 non-diabetic MASLD patients at a tertiary referral center between 2023 and 2024. Prediabetes was defined according to standard glycemic criteria. Liver fibrosis was assessed using the fibrosis-4 (FIB-4) index and Acoustic Radiation Force Impulse (ARFI) elastography. The prevalence of significant ($F \geq 2$) and advanced fibrosis ($F \geq 3$) was determined. Factors associated with advanced fibrosis were analyzed using multivariable regression models.

Results: The mean age was 47.3 ± 12.6 years, and 56.1% of patients were male. Significant fibrosis ($F \geq 2$) and advanced fibrosis ($F \geq 3$) were identified in 15.0% and 11.3% of the overall cohort, respectively. Among patients with prediabetes, 41.7% had already developed advanced fibrosis. FIB-4 demonstrated excellent discrimination for advanced fibrosis (AUROC 0.949 for $F \geq 3$). Age and aspartate aminotransferase levels were independently associated with advanced fibrosis.

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Conclusions: Prediabetes is associated with a substantial burden of advanced liver fibrosis in non-diabetic MASLD patients. These findings suggest that deferring fibrosis evaluation until the onset of overt diabetes may delay the identification of high-risk individuals. Incorporating simple non-invasive tools into the assessment of patients with prediabetes may enable earlier risk stratification and timely preventive strategies in clinical practice.

Keyword(s) : MASLD; Prediabetes; Liver fibrosis; FIB-4; ARFI elastography

2568 - Gut Liver Axis Mediated Dysbiosis in the Pathogenesis of Liver Fibrosis and Intervention Strategies

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Background / Hypothesis: Liver fibrosis is a common pathological endpoint of chronic liver diseases, characterized by the activation of hepatic stellate cells (HSCs) leading to excessive deposition of extracellular matrix (ECM). In recent years, the "gut-liver axis" has become a focal point in liver fibrosis research.

Numerous studies indicate that the gut microbiota can remodel the hepatic microenvironment by influencing the intestinal barrier, bile acid signaling, microbial metabolites, and immune responses. Gut dysbiosis can lead to the translocation of pathogen-associated molecular patterns (PAMPs), activating Kupffer cells and HSCs, thereby triggering inflammation and fibrosis. Furthermore, dysregulation of key metabolic pathways, such as the bile acid FXR-FGF19 axis and short-chain fatty acid (SCFA) production, significantly contributes to hepatic fibrogenesis. Based on existing research and integrating the latest evidence from systems biology, metabolomics, and microbiomics, this review comprehensively elaborates on the pathological mechanisms by which gut-liver axis-mediated dysbiosis leads to liver fibrosis. It also summarizes current intervention strategies, aiming to provide references for clinical liver fibrosis treatment and target research.

Materials and Methods: The gut-liver axis refers to the bidirectional relationship between the intestine (including its microbiota) and the liver^{[5]^}. These two organs are interconnected through the portal vein;

biliary tract, systemic circulation, and various systemic mediators. The liver is the first organ exposed via portal blood to gut-derived microbial products and metabolites. A healthy intestinal barrier prevents the translocation of gut-derived endotoxins or metabolites into the liver. Damage to the intestinal barrier and dysbiosis can lead to the translocation of gut microbes, endotoxins, etc., activating the immune system and triggering inflammation or fibrosis. This review primarily focuses on the pathological mechanisms and intervention strategies for liver fibrosis mediated by gut-liver axis dysbiosis.

Results: The role of the gut-liver axis and gut microbiota in liver diseases has become a major research focus in recent years. Extensive clinical and basic research confirms that the gut microbiota plays a crucial role in various liver diseases at different stages. The treatment of liver fibrosis requires a holistic approach, addressing both the liver and the gut. By modulating the gut microbiota and intestinal barrier function, liver function can be restored, disease progression delayed, and transmission of liver

Keyword(s) : Gut-liver axis; Liver fibrosis; Gut microbiota

2569 - Clinical Characteristics and Complication Patterns in Hospitalized Elderly Patients with Liver Cirrhosis A Single Center Retrospective Analysis of 1 736 Admissions

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Background / Hypothesis:

To investigate the clinical characteristics and distribution patterns of complications in elderly patients with liver cirrhosis, and to provide an evidence-based foundation for the clinical management and individualized treatment of the aged population.

Materials and Methods:

A retrospective analysis was conducted on clinical data from 1,736 admissions (representing 1,095 individual patients) diagnosed with liver cirrhosis at the Second Affiliated Hospital of Baotou Medical College between January 1, 2025, and January 1, 2026. Participants were categorized into the elderly group (≥ 65 years, $n = 683$ admissions) and the non-elderly group (< 65 years, $n = 1,053$ admissions). Complication rates, including

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hepatocellular carcinoma (HCC), hepatic encephalopathy (HE), ascites, pleural effusion, gastrointestinal (GI) bleeding, portal vein thrombosis (PVT), infection, and hepatorenal syndrome (HRS), were statistically compared between the two groups.

Results:

Demographic Composition: Among the 1,736 admissions, the 50–60 age group accounted for the highest proportion (29.3%). The elderly group (≥ 65 years) comprised 39.3% ($n = 683$) of the total, with patients over 80 years old representing 12.9% (88/683) of the elderly cohort.

Complication Distribution:

Elderly Group (≥ 65 years): Showed a significantly higher prevalence of infection (37.5%, 256/683), hepatorenal syndrome (HRS) (19.3%, 132/683), ascites (57.1%, 390/683), and pleural effusion (17.7%, 121/683).

Non-elderly Group (< 65 years): Demonstrated a significantly higher incidence of gastrointestinal bleeding (38.2%, 402/1,053) and a HCC complication rate of 12.7% (134/1,053).

Others: The incidence of HE was comparable between groups (17.1% in elderly vs. 15.3% in non-elderly), and PVT remained stable at approximately 10% in both cohorts.

Statistical Analysis: Compared to the non-elderly group, the elderly group exhibited a significant upward trend in the risks of infection, HRS, and effusions ($P < 0.05$), whereas GI bleeding was more prominent in the non-elderly population.

Conclusions:

Clinical presentations of elderly patients with liver cirrhosis exhibit significant heterogeneity. Infection and HRS represent the primary clinical threats for this demographic, necessitating enhanced monitoring of immune status and renal function. Conversely, management of younger patients should focus more on acute portal hypertension-related complications such as GI bleeding. These findings support a multidisciplinary management strategy for elderly cirrhotic patients centered on "infection prevention and renal protection."

Keyword(s) : Liver Cirrhosis; Elderly; Complications; Infection; Hepatorenal Syndrome.

2570 - Improving HBV Screening and Prophylaxis in Rituximab Treated Patients A Quality Improvement

Study

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Background / Hypothesis:

Rituximab (RTX) is associated with a high risk of hepatitis B virus (HBV) reactivation in patients with hematologic malignancies. In our previously published cohort, major gaps in HBV screening and antiviral prophylaxis were identified, resulting in preventable reactivation events. Building on these experiences, a quality-improvement strategy incorporating electronic alerts and a locally developed HBV management algorithm was implemented. We aim to evaluate HBV screening and prophylaxis practices among rituximab-treated patients following implementation of system-level interventions.

Materials and Methods:

We conducted a retrospective quality-improvement study including adult patients with hematologic malignancies who received rituximab between 01 July 2023 and 01 July 2025 at a tertiary care center. HBV screening was evaluated using HBsAg and anti-HBc serology, and HBV DNA (PCR) testing was reviewed when clinically indicated. Screening data up to six months prior to rituximab initiation were assessed. Antiviral prophylaxis practices and HBV DNA-based monitoring strategies were identified from electronic medical records. Clinical management was guided by a locally developed HBV screening and prophylaxis algorithm.

Results:

Among rituximab-treated patients screened during the two-year study period ($N \approx 986$), 913 patients (92.6%) underwent appropriate HBV screening, while 34 patients (3.4%) had incomplete screening and 12 patients (1.3%) were not screened. Overall, 97 patients (9.8%) met criteria for antiviral prophylaxis. Among these, 85 patients (87.6%) received guideline-concordant antiviral prophylaxis, whereas 12 patients (12.3%) were managed with serial HBV DNA (PCR) monitoring without prophylaxis. No HBV reactivation was reported during at least one year of follow-up.

Conclusions:

HBV screening and prophylaxis practices among rituximab-treated patients improved markedly

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following implementation of electronic alerts and a local management algorithm informed by prior institutional experience. These findings support the effectiveness of pragmatic, system-embedded interventions in reducing preventable gaps in HBV care in high-risk hematology populations.

Keyword(s) :

2571 - Predicting key components and mechanisms of silymarin in treating sepsis related liver injury using GraphBAN

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Background / Hypothesis:

Sepsis-related liver injury (SRLI), a common complication of sepsis, exacerbates organ dysfunction and worsens patient prognosis. Traditional Chinese medicine has accumulated substantial experience in liver injury management. This study employs network pharmacology and compound-protein interaction (CPI) analysis to identify key active components and targets in *Silybum marianum* (L.) Gaertn related to SRLI, offering new perspectives for its targeted therapy.

Materials and Methods:

This study obtained SRLI-related data and chemical composition information from milk thistle through public databases. By integrating differential gene expression analysis with CPI prediction technology based on GraphBAN, potential CPI pairs were identified. Further integration of physicochemical properties and toxicological parameters successfully pinpointed key active components and their corresponding targets. Subsequently, this study conducted an in-depth analysis of interactions among key targets and their involvement in biological functions. Molecular docking and molecular dynamics simulations were employed to evaluate binding stability between key bioactive compounds and their targets. Finally, gene set enrichment analysis was performed to explore the biological functions and signaling pathways involved in key genes.

Results:

A total of 1,554 differentially expressed genes and 11 bioactive compounds were identified, with quercetin identified as the key active component and REN and SIGMAR1 as its primary targets. Molecular docking revealed binding energies of -7.1 and -8.3 kcal/mol

between quercetin and the REN and SIGMAR1 targets, respectively, forming stable complexes in kinetic simulations. Enrichment analysis indicated that key targets were primarily involved in multiple signaling pathways, including SARS-CoV-2 infection-related responses, and responses to LPS-induced mechanical ventilation etc. Key genes showed significant correlations with multiple genes within gene sets related to inflammation, mitochondria, and oxidative stress.

Conclusions:

This study employed network pharmacology combined with GraphBAN analysis to predict one key active ingredient and two key targets for treating SRLI, providing crucial evidence for the development of targeted traditional Chinese medicine therapies for SRLI

Keyword(s) : Sepsis-related liver injury; GraphBAN; Network pharmacology; Quercetin

2573 - NRF2 Mediated Gsta3 Transcriptional Activation Protects Against Sepsis Associated Liver Injury

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Background / Hypothesis:

Oxidative stress participates in the pathogenesis of sepsis-associated liver injury (SALI), and the glutathione system plays a pivotal role in cellular antioxidant defense mechanisms. Gsta3 predominantly expressed in the liver, is a key transferase in the glutathione system; however, its specific mechanism of action in SALI remains to be fully elucidated.

Materials and Methods:

This study aimed to evaluate CLP- and LPS/D-GalN-induced SALI models, clarify Gsta3's role and mechanism, and identify causes of its downregulation. Methods: Bioinformatics analyses were performed using transcriptomic and single-cell sequencing data (GSE279167, GSE254497). SALI models were established, with Gsta3 expression detected by ELISA, qPCR and Western blotting. In vitro and in vivo experiments (including gene overexpression/knockdown, ChIP and dual-luciferase assays) were conducted to verify mechanisms.

Results:

Both models successfully induced SALI with hepatocyte pyroptosis; Gsta3 was downregulated in liver tissues (negatively correlated with ALT/AST) but upregulated in septic patients' serum. Gsta3 alleviated hepatocyte

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injury by improving mitochondrial function and inhibiting pyroptosis. NRF2 directly bound to Gsta3 promoter and activated its transcription; LPS/D-GalN inhibited NRF2 nuclear translocation, reducing Gsta3 expression. NRF2-knockout mice had lower Gsta3 and severer liver injury, which was rescued by AAV-mediated Gsta3 overexpression.

Conclusions: Gsta3 antagonizes SALI by maintaining mitochondrial homeostasis and suppressing pyroptosis. LPS/D-GalN downregulates Gsta3 via inhibiting NRF2 nuclear translocation, providing a theoretical and experimental basis for SALI targeted therapy.

Keyword(s) : sepsis, liver injury, Gsta3, NRF2, Pyroptosis

2575 - Acute Pancreatitis Secondary to Tirzepatide Therapy

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Background / Hypothesis: Glucagon-like peptide-1 (GLP-1) receptor agonists and dual-acting GIP/GLP-1 agonists are increasingly used in the treatment of type 2 diabetes and obesity. Tirzepatide is a new generation agent that activates both GIP and GLP-1 receptors. Although the risk of pancreatitis has been reported as low in clinical studies, acute pancreatitis has been reported, albeit rarely, in real-life data. In this case report, we aim to present a patient who developed acute pancreatitis during tirzepatide treatment.

Case: A 36-year-old male patient with type 2 diabetes and obesity was started on tirzepatide treatment (5 mg weekly, subcutaneous) approximately 2 months ago. The patient had no prior history of pancreatitis, alcohol use, recent viral infection, or hypertriglyceridemia. He reportedly lost approximately 15 kg during treatment. He presented to the emergency department with severe epigastric and back pain radiating to the lower back, nausea, and vomiting. Physical examination revealed epigastric tenderness. Laboratory investigations showed amylase: 537 U/L, lipase: 1071 U/L. Liver enzymes, bilirubin levels, and serum calcium levels were normal. Triglyceride level was measured at 117 mg/dL. Abdominal ultrasonography showed no gallstones or common bile duct dilation. Magnetic resonance cholangiopancreatography (MRCP) imaging revealed increased volume in the distal tail section of the pancreatic body and contamination in the

surrounding mesenteric fat tissue, consistent with pancreatitis. The biliary tract and common bile duct were reported as normal. Based on the current clinical, laboratory, and radiological findings, the patient was diagnosed with acute pancreatitis. After ruling out possible etiological causes, it was thought that the pancreatitis developed due to tirzepatide treatment, and the drug was discontinued. The patient was monitored with supportive treatment. Improvement was observed in clinical and laboratory findings, and the patient was discharged without complications after a 5-day hospital stay. No recurrence of pancreatitis was observed during follow-up.

Conclusions: Acute pancreatitis is a rare but potentially serious side effect of tirzepatide treatment. Particular attention should be paid to pancreatitis in patients experiencing rapid weight loss and unexplained epigastric pain. In patients who develop acute pancreatitis, tirzepatide treatment should be discontinued and not restarted. This case highlights the importance of not ignoring the risk of pancreatitis in patients using tirzepatide.

Keyword(s) :

2578 - Integrated Multi omics Liquid Biopsy Advancing Early Detection and Precision Management of HCC

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a leading cause of cancer-related mortality worldwide, often diagnosed at late stages due to the limited sensitivity and specificity of current surveillance methods such as ultrasound and alpha-fetoprotein (AFP) testing. Liquid biopsy, which non-invasively analyzes circulating biomarkers, offers a promising alternative (Fig 1). However, single-omics approaches often lack the robustness required for early detection. Multi-omics integration presents a transformative strategy to enhance diagnostic accuracy and enable precision oncology in HCC.

Materials and Methods:

This review synthesizes recent advances in multi-omics

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liquid biopsy for HCC, incorporating genomic (e.g., TERT, TP53 mutations), epigenomic (e.g., RASSF1A methylation), proteomic/glycomic (e.g., AFP-L3, GP73, exosomal glycans), fragmentomic (cfDNA fragmentation patterns), and cellular biomarkers (CTCs, EVs) (Fig 2) . Cross-omics integration strategies—such as cSMART, HIFI, and ASDAm-H1—were evaluated alongside emerging technologies including nanopore sequencing, SERS-based single-exosome analysis, and machine learning models (e.g., XAI, GNNs, Bayesian Networks) (Fig 3) .

Results:

The integration of multi-omics biomarkers has demonstrated transformative diagnostic performance for early HCC (Table 1) . Cross-omics models consistently outperformed single-modality approaches: combined genomic-epigenomic assays achieved sensitivities of 80–90% for early-stage detection, while the fragmentomic-epigenomic HIFI model distinguished HCC from cirrhosis with exceptional accuracy (AUC 0.996, sensitivity 95.42%, specificity 97.83%). Integrated protein-epigenetic panels, such as the ASDAm-H1 index, reached an AUC of 0.96, and longitudinal application of multi-parametric scores like GALAD significantly improved sensitivity to 69.2% for early HCC compared to single measurements (53.8%). Fragmentomics-based models enabled early screening with sensitivities exceeding 96%, and metabolomic panels detected preclinical HCC up to one year before clinical diagnosis with 80% sensitivity. Beyond detection, multi-omics liquid biopsy proved vital for precision management: ctDNA mutation profiling enabled dynamic monitoring of targeted therapy response, PD-L1⁺ CTCs and cfDNA copy number variations predicted outcomes to immune checkpoint inhibitors, and machine learning-driven deconvolution of single-exosome SERS fingerprints or cfDNA methylation data provided unprecedented molecular resolution. These approaches not only identified AFP-negative HCC cases but also facilitated early recurrence prediction and molecular subtyping, effectively transitioning liquid biopsy from a passive detection tool to an active component in the dynamic, personalized management of HCC.

Conclusions:

Multi-omics liquid biopsy represents a paradigm shift in HCC management, enabling high-accuracy early detection and dynamic monitoring of disease progression and treatment response. Despite challenges in data integration, standardization, and clinical validation, future directions—including

explainable AI, single-cell multi-omics, and closed-loop precision oncology systems—hold great promise (Fig 4) . With continued interdisciplinary collaboration and standardization efforts, multi-omics liquid biopsy is poised to become the cornerstone of next-generation HCC care.

Keyword(s) : Hepatocellular carcinoma, Liquid biopsy, Multi-omics integration, Early diagnosis, Clinical translation

2581 - Association between Acid Suppressant Use and Hepatic Encephalopathy in Patients with Liver Cirrhosis An Inverse Probability of Treatment Weighting Analysis

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Background: Acid suppressants, including proton pump inhibitors (PPIs), are

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frequently prescribed for patients with chronic liver disease. While associations between acid suppressant use and hepatic encephalopathy (HE) have been suggested, causality remains unclear due to indication bias, as these drugs are often prescribed to patients with greater disease severity. This study investigated the association between acid suppressants and liver-related events using a large-scale prospective cohort. Methods: Of 1,206 patients with chronic liver disease registered between

February 2020 and March 2022, 373 patients with liver cirrhosis were included. Patients were categorized into PPI/potassium-competitive acid blocker (P-CAB) user and non-user groups. The primary endpoint was the cumulative incidence of HE, and the secondary endpoint was the cumulative incidence of variceal bleeding. Inverse probability of treatment weighting (IPTW) was performed to adjust for background factors, including ALBI score, history of HE, ascites, varices, and use of non-absorbable disaccharides or rifaximin. Balance was assessed using the standardized mean difference (SMD).

Results: In the unadjusted cohort (non-users: n=170; users: n=197), users had significantly worse liver function and a more frequent history of decompensation or HE treatment, as indicated by the ALBI score (median -2.51 vs. -2.24), history of HE (9.4% vs. 13.7%), ascites (20.0% vs. 34.5%), varices (27.1% vs. 55.8%), and use of non-absorbable disaccharides (5.9% vs. 15.2%) or rifaximin (1.8% vs. 14.2%). After IPTW adjustment (non-users: n=163; users: n=196), background factors were well balanced (all SMDs < 0.1 or close to 0.1). The 1- and 2-year cumulative HE incidence rates were 2.3% vs. 3.8% and 4.8% vs. 6.8% for non-users and users, respectively. Although the rate was higher in users, the difference was not statistically significant (HR 1.74, 95% CI 0.68-4.46, p=0.247). The 1- and 2-year cumulative variceal bleeding rates were 3.9% vs. 2.3% and 3.9% vs. 2.3%, respectively, showing a lower trend in users but no significant difference (HR 0.66, 95% CI 0.19-1.96, p=0.40).

Conclusion: Although a trend toward higher HE incidence was observed in acid suppressant users, no statistically significant difference was found after adjusting for confounding factors. This study did not find clear evidence that acid suppressant use significantly increases the risk of HE in patients with cirrhosis.

Keyword(s) : Cirrhosis, Proton pump inhibitors, Hepatic

encephalopathy, Inverse probability of treatment weighting, Prospective cohort

2582 - Neurological Outcomes and Survival in Hepatic Myelopathy A Multicenter Cohort Study

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Background / Hypothesis:

Hepatic myelopathy (HM) is a disabling neurological complication of advanced liver disease. Data on neurological reversibility and long-term prognosis under different treatment strategies remain limited. We aimed to characterize clinical features, management strategies, neurological outcomes, and survival in a multicenter cohort of patients with HM.

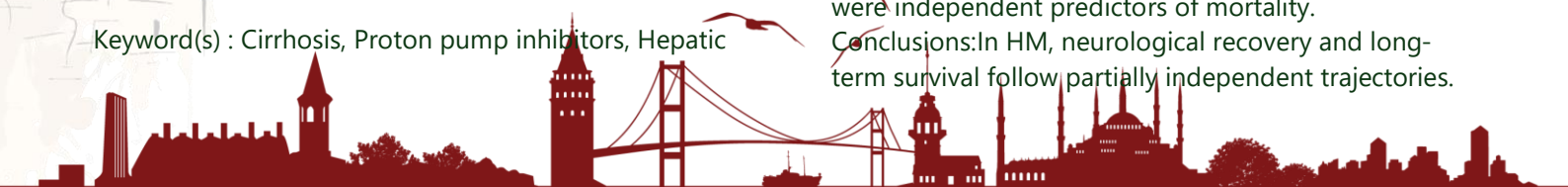
Materials and Methods:

We retrospectively included 184 patients diagnosed with HM. Clinical characteristics, neuroimaging and electrophysiological findings, and initial treatment strategies (conservative management, interventional shunt modulation, or liver transplantation) were collected. Neurological outcome at last follow-up was assessed using functional status and the modified Rankin Scale (mRS). Overall survival was analyzed using Kaplan-Meier methods and multivariable Cox regression.

Results:

Radiologically detectable portosystemic shunts were present in 179/184 patients (97.3%), including 61 with prior transjugular intrahepatic portosystemic shunts and 118 with spontaneous shunts. Brain MRI commonly demonstrated changes consistent with metabolic encephalopathy, while spinal MRI revealed no consistent structural spinal cord lesions. Evoked potentials frequently showed central conduction abnormalities, whereas peripheral nerve involvement on nerve conduction studies or electromyography was less common. Neurological improvement occurred significantly more often after interventional shunt modulation or liver transplantation than after conservative management (P < 0.001). Although unadjusted survival differed across treatment strategies, treatment strategy was not independently associated with survival after adjustment; higher baseline MELD score and hepatocellular carcinoma were independent predictors of mortality.

Conclusions: In HM, neurological recovery and long-term survival follow partially independent trajectories.



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Neurological dysfunction may be modifiable in selected patients, whereas overall prognosis is primarily determined by the severity of underlying liver disease.

Keyword(s) :

2584 - Cytopenia Following Albendazole Use A Real World Observation

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Background / Hypothesis: Albendazole is a commonly used antiparasitic drug. In extremely rare cases, however, it can cause serious adverse reactions, such as bone marrow suppression and neutropenia.

Materials and Methods: This study reports three cases of pancytopenia in patients with liver parasitic diseases who were treated with albendazole. We also analyse cases of cytopenia reported in the FDA Adverse Event Reporting System (FAERS) database following albendazole use.

Results: One case was deemed to be systemic inflammatory response syndrome triggered by the death of a large number of parasites, while the remaining two cases were considered to be adverse reactions to albendazole. Only one patient experienced timely detection of myelosuppression and made a full recovery following treatment; the other two patients died from the condition. From January 2004 to June 2025, the FAERS system recorded 88 instances of reduced blood cell counts associated with albendazole use, including 25 cases of pancytopenia and 35 cases of granulocytopenia. There were 11 deaths.

Conclusions: Although albendazole-induced cytopenia is rare, clinical vigilance remains essential, particularly in individuals with liver disease. Our aim is to raise clinicians' awareness of this potential adverse reaction.

Keyword(s) : Liver parasitic disease; Albendazole; Pancytopenia; Anthelmintic drugs; Adverse drug reactions

2585 - Clinical Characteristics of Non Cirrhotic Portal Hypertension Associated with Oxaliplatin

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Background / Hypothesis:

Non-cirrhotic portal hypertension (NCPH) is a group of diseases characterised by elevated portal vein pressure in the absence of cirrhosis. Several studies have shown that patients undergoing oxaliplatin (OX) chemotherapy can develop NCPH within three to five years

Objective: To summarise the clinical features of NCPH following OX therapy to highlight the relationship between delayed-onset NCPH and OX for the attention of clinicians.

Materials and Methods:

A retrospective analysis was conducted on six patients diagnosed with OX-related NCPH between 2013 and 2025. The study examined the patients' general information, clinical characteristics, laboratory test results, imaging findings and liver histopathology. Treatment regimens were summarised to explore the clinical features of patients who developed NCPH following OX chemotherapy.

Results:

Of the six patients, one developed manifestations of portal hypertension (PH) four months after receiving OX chemotherapy, and magnetic resonance imaging suggested sinusoidal obstruction syndrome. The remaining five patients exhibited PH manifestations between two years and four years post- OX chemotherapy. Pathological examination of liver tissue from four of these patients revealed no cirrhosis, resulting in a diagnosis of NCPH. Liver function tests and liver stiffness measurements were generally

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normal or only slightly abnormal

Conclusions:

In patients with malignant tumours undergoing OX chemotherapy, the presence of manifestations of portal hypertension, such as oesophageal-gastric varices, splenomegaly, thrombocytopenia or ascites, without significant abnormalities in liver size or function, should prompt consideration of OX-induced NCPH. A liver biopsy can provide definitive confirmation of the diagnosis.

Keyword(s) : Oxaliplatin; Non-cirrhotic Portal Hypertension; NCPH ; Idiosyncratic drug-induced liver injury; Gastrointestinal cancer

2586 - The traditional Chinese medicine prescription Danjin Shugan Formula exerts anti fibrotic effects by regulating the mTOR S6K1 4E BP1 signaling pathway and YAP TAZ proteins

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Background / Hypothesis:

We aimed to investigate the anti-hepatic fibrosis efficacy of Danjin Shugan recipe(DJSG) and its potential mechanism for attenuating hepatic fibrosis.

Materials and Methods:

We induced and constructed a hepatic fibrosis model in Wistar rats by intraperitoneal injection of carbon tetrachloride (CCl₄) and used Fuzheng Huayu decoction (FZHY) as a positive control drug. Subsequently, hematoxylin-eosin, Masson staining and immunohistochemical staining were performed on the liver tissues of rats in each group at weeks 4 and 8, and peripheral blood was collected to test liver function indexes. We used Western blotting and qRT-PCR to detect changes in the mTOR-S6K1/4E-BP1 signaling pathway, YAP/TAZ and HNF-4 α factors in each group of rats. In addition, we randomly divided 60 patients into an experimental group (receiving Danjin Shugan Capsules combined with anti-hepatitis B virus treatment) and a control group (receiving Fuzheng Huayu Capsules combined with anti-hepatitis B virus treatment). At baseline and after 3, 6, and 12 months of treatment, liver fibrosis indicators (liver stiffness value, FIB-4, APRI index) were followed up, collected, and compared. At the same time, relevant adverse reactions observed in patients were recorded.

Results:

DJSG significantly reduced liver injury and fibrosis in CCl₄-induced rats. Compared with the CCl₄ group, DJSG, and FZHY significantly reduced the protein expression levels of p-mTOR, p-S6K1, and p-4E-BP1 at week 4 and week 8, and the protein expression levels of p-mTOR and p-4E-BP1 decreased even more significantly in the CCl₄+FZHY group. At the same time point, DJSG and FZHY may ameliorate rat liver fibrosis by inhibiting YAP/TAZ protein expression. After 3, 6, and 12 months of treatment, improvements in liver stiffness values were observed in both groups, with no significant difference compared to the Fuzheng Huayu treatment group (P<0.05) .

Conclusions:

Our study demonstrated that DJSG improved liver function and liver fibrosis levels in CCl₄ rats and may be associated with the YAP/TAZ protein and mTOR-S6K1/4E-BP1 signaling pathway. Danjin Shugan Capsules exhibit a certain anti-fibrotic effect. It has been found that a 12-week treatment course can achieve a certain degree of reversal of liver fibrosis and early-stage cirrhosis.

Keyword(s) : Hepatic fibrosis, Traditional Chinese medicine, Danjin Shugan recipe, mTOR-S6K1/4E-BP1 signaling pathways, YAP/TAZ protein

2587 - Genetically Confirmed Dubin Johnson Syndrome Presenting as Long Standing Isolated Conjugated Hyperbilirubinemia

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Background / Hypothesis: Chronic intermittent jaundice with isolated hyperbilirubinemia is commonly attributed to benign conditions such as Gilbert syndrome. However, when conjugated bilirubin predominates, inherited disorders of hepatocellular bilirubin transport should be considered. Dubin-Johnson syndrome is a rare autosomal recessive condition caused by loss-of-function variants in the *ABCC2* gene, leading to impaired canalicular excretion of conjugated bilirubin.

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Materials and Methods:We report a 30-year-old man with a 10-year history of intermittent jaundice and dark urine without pruritus, abdominal pain, systemic symptoms, alcohol use, or hepatotoxic drug exposure. Comprehensive biochemical testing, abdominal imaging, liver stiffness assessment, and targeted genetic analysis were performed.

Results:The patient demonstrated persistent isolated hyperbilirubinemia with conjugated predominance, while aminotransferases, cholestatic enzymes, and hepatic synthetic function remained normal. Markers of hemolysis, viral hepatitis, and autoimmune liver disease were negative. Abdominal ultrasound and magnetic resonance imaging showed normal liver morphology without biliary obstruction, and shear-wave elastography indicated normal liver stiffness. Targeted genetic testing identified a homozygous truncating loss-of-function variant in the ABCC2 gene (p.Trp1206Ter), confirming the diagnosis of Dubin-Johnson syndrome.

Conclusions:Dubin-Johnson syndrome should be considered in patients presenting with chronic intermittent jaundice and isolated conjugated hyperbilirubinemia with normal liver enzymes and unremarkable imaging. A stepwise diagnostic approach integrating bilirubin fractionation and targeted ABCC2 genetic testing enables definitive diagnosis, avoids unnecessary invasive investigations, and reassures patients regarding the benign and non-progressive nature of the condition.

Keyword(s) : Dubin-Johnson syndrome; ABCC2; conjugated hyperbilirubinemia; genetic liver disease

2588 - Epidemiology of Delta Virus Infection in Turkey Current Seroprevalence

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Background-hypothesis: Hepatitis delta virus (HDV) infection is a significant health and economic problem worldwide, particularly in endemic regions such as the Mediterranean, Southern and Eastern Europe, the Middle East, and Turkey. Approximately 15 million people worldwide suffer from HDV, accounting for 5-10.6% of all HBV infections globally. The prevalence of HDV among HBsAg-positive blood donors, outpatients, and inpatients was found to be 3.37%, 5.05%, and 29.06%, respectively. In our study, we aimed to investigate the seroprevalence of Delta virus infection in HBsAg-positive patients in Turkey.

Method: Patients aged 18 and over who were HBsAg positive and followed up in the Infectious Diseases outpatient clinics of the participating hospitals between January 1, 2015, and December 31, 2024, were included in the study. Demographic data, laboratory results, and treatments received by the patients were retrospectively reviewed from their patient records.

Results: Data were obtained from hospitals in a total of 27 different provinces in Turkey. 31,514 patients were included in the study, 55.4% of whom were male. The mean age was 47.4 ± 14.1 years. Of the patients, 34.2% had HBeAg-negative chronic hepatitis B, 57.2% had HBeAg-negative chronic HBV infection, 4.6% had HBeAg-positive chronic hepatitis B, and 4% had HBeAg-positive chronic HBV infection. 4.6% of HBsAg positive patients had clinically diagnosed cirrhosis. In HBsAg-positive patients, 0.7% were anti-HCV positive and 0.4% were anti-HIV positive. Anti-HDV testing was performed in 29,085 of the participating patients, and the anti-HDV positivity rate was 5.2%. HDV RNA tests were found to be positive in 568 patients. Of the patients for whom current data were available, 299 were waiting for delta treatment. A total of 113 patients had undergone liver transplantation due to HDV. Currently, 20 patients are receiving bulevirtide treatment, and 40 are receiving pegylated interferon treatment. Pegylated interferon treatment was the most common treatment among the patients' previous treatments for delta hepatitis.

Conclusion: Delta hepatitis remains an important disease in our country. In our study, it has been observed that there are still centers where anti-HDV testing is not performed. Performing anti-HDV

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screening in all HBsAg-positive patients would be beneficial in countries where HBV infection is endemic, in terms of earlier diagnosis and treatment of patients with delta hepatitis.

Keyword(s) : delta hepatitis, seroprevalance, Turkey.

2589 - Adult Onset Wilson Disease Without Classical Features Diagnostic Value of ATP7B Genotyping and Leipzig Scoring

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Background / Hypothesis:Wilson disease is an autosomal recessive genetic disorder of copper metabolism caused by pathogenic variants in the ATP7B gene. In adults, the disease may present without classical ophthalmologic or neurological features, leading to diagnostic delay. This study aimed to highlight the diagnostic value of integrating genetic analysis with functional copper assessment and Leipzig scoring in an atypical adult-onset presentation.

Materials and Methods:An adult patient presented with progressive jaundice and cholestatic liver dysfunction. Viral hepatitis, autoimmune liver disease and drug-induced liver injury were excluded. Ophthalmologic examination showed no Kayser-Fleischer rings, and no neurological manifestations were observed. Serum ceruloplasmin, 24-hour urinary copper excretion and liver biochemistry were assessed. ATP7B gene analysis was performed using next-generation sequencing. Clinical and biochemical responses were evaluated after initiation of copper chelation therapy.

Results:Serum ceruloplasmin was within the normal range. Baseline 24-hour urinary copper excretion was markedly elevated at 456 µg/24 h. Genetic testing identified a compound heterozygous state with one pathogenic frameshift variant (c.2304dup, p.Met769HisfsTer26) and one likely pathogenic missense variant (c.2549C>T, p.Thr850Ile) in the ATP7B

gene. Despite the absence of Kayser-Fleischer rings and neurological involvement, the Leipzig score reached 4 points, fulfilling diagnostic criteria for Wilson disease. After three weeks of copper chelation therapy, urinary copper excretion decreased to 378 µg/24 h, accompanied by significant clinical improvement and marked reduction in serum bilirubin levels.

Conclusions:Adult-onset Wilson disease may present without classical clinical features and with normal ceruloplasmin levels. Integration of ATP7B genotyping with functional copper assessment and Leipzig scoring is essential for accurate diagnosis and timely treatment in atypical cases.

Keyword(s) : Wilson disease; ATP7B; Adult-onset; Genetic liver disease; Leipzig score; Diagnostic challenge

2591 - AMA negative Primary Biliary Cholangitis Diagnosed by a Reproducible Dechallenge Rechallenge Response to Ursodeoxycholic Acid

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Background / Hypothesis:Primary biliary cholangitis (PBC) is a chronic autoimmune cholestatic liver disease typically associated with antimitochondrial antibodies (AMA). However, approximately 5-10% of patients are AMA-negative, creating diagnostic challenges, particularly when liver biopsy is unavailable or deferred. This report describes a case of suspected AMA-negative PBC diagnosed based on a reproducible biochemical response to ursodeoxycholic acid (UDCA).

Materials and Methods:We report a 65-year-old man with a two-year history of persistent cholestatic liver enzyme elevation, characterized by markedly elevated gamma-glutamyl transferase (GGT). There was no history of alcohol intake, hepatotoxic drug exposure, or viral hepatitis. Imaging excluded biliary obstruction, and liver stiffness measurement was consistent with moderate fibrosis (F2). Autoimmune serology revealed borderline and subsequently negative AMA titres.

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Serial liver biochemistry was longitudinally analyzed in relation to UDCA exposure.

Results: During periods of UDCA withdrawal, cholestatic enzymes progressively worsened, with GGT increasing from 1023.74 to 1301 U/L and, during a subsequent interruption, from 923.6 to 1449.56 U/L. In contrast, initiation and reintroduction of UDCA were consistently followed by marked biochemical improvement, with GGT decreasing from 1301 to 923.6 U/L and subsequently from 1449.56 to 644.6 U/L. Aminotransferase levels showed parallel improvement, while serum bilirubin remained stable without progression to overt jaundice. This highly reproducible on-off biochemical pattern provided strong functional evidence supporting a diagnosis of AMA-negative PBC.

Conclusions: A reproducible dechallenge-rechallenge biochemical response to UDCA may serve as valuable functional diagnostic evidence in suspected AMA-negative PBC. Careful longitudinal monitoring of liver enzymes may help support diagnosis and reduce the need for invasive liver biopsy in selected patients.

Keyword(s) : Primary biliary cholangitis; AMA-negative; Ursodeoxycholic acid; Cholestatic liver disease; Dechallenge-rechallenge

2592 - Ultra High HBV Viral Load Not Equivalent to the Immune Tolerant Phase.

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Background / Hypothesis:

While an ultra-high viral load often suggests immune tolerance in chronic hepatitis B, it has now been recognized that such cases exhibit significant heterogeneity, including variations in gender, age, virological markers, and biochemical profiles.

Materials and Methods:

This study ultimately enrolled 412 chronic hepatitis B patients with ultra-high viral loads who had not yet received antiviral treatment. All patients had high-sensitivity HBV-DNA levels $>10^7$ IU/ml and were HBeAg-positive. General information, laboratory test

results, and examination data were collected from the patients. The virological characteristics were compared across groups based on factors such as age, gender, and ALT levels, aiming to understand the clinical features of patients with high viral loads.

Results:

Patients with ultra-high viral loads exhibit significant heterogeneity, characterized by a high rate of abnormal ALT levels and a certain degree of significant fibrosis and hepatic steatosis. In these patients, elevated ALT is more closely associated with a decrease in HBsAg levels rather than an increase in steatosis. The rise in liver stiffness measurement (LSM) is considered to correlate with the reduction in hepatitis B viral load. Patients over 30 years of age with ultra-high viral loads demonstrate clear progression in fibrosis, supporting the age of 30 as a critical threshold for initiating antiviral therapy. Additionally, an HBV-DNA level $>10^8$ IU/ml is more indicative of immune-tolerant characteristics.

Conclusions:

Keyword(s) : Ultra-High HBV Viral Load, chronic hepatitis B, HBeAg-positive

2595 - A case of artificial liver support system for Wilson's disease related acute on chronic liver failure complicated with hyperthyroidism

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Background :

Wilson's disease is one of the causes of acute-on-chronic liver failure, characterized by a dangerous condition, rapid progression, and extremely high mortality.

CASE SUMMARY

The patient is a 60-year-old male who presented with jaundice of the skin and sclera for 11 days. He had a history of hyperthyroidism and was previously treated with tapazole. After developing jaundice, he was switched to iodine therapy. He had a history of alcohol consumption but had quit. At the time of admission, the patient's liver function tests showed ALT 197U/L, AST 149U/L, ALP 151 U/L, GGT 41 U/L, TBIL 265.1umol/L, DBIL 235.7 umol/L, INR 1.69, HBsAb, HBeAb, HbcAb (+), HBV-DNA (-), immune tests and respiratory virus tests were all negative, IgG 8.31g/L, AFP 242ng/ml, FT3 2.11pmol/L, FT4 17.4pmol/L, TSH

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<0.005, thyroid peroxidase antibody 158.75IU/ml, and thyroglobulin antibody 56.27 IU/ml.. The abdominal MRI indicates edema and thickening of the gallbladder wall, as well as a small amount of fluid accumulation around the liver and spleen. Upon admission, the patient was considered to have hyperthyroidism combined with subacute liver failure and was treated with medical therapy. However, the patient's liver function deteriorated progressively. Further examinations revealed ceruloplasmin 15 mg/dl and urinary copper 257.6 µg/24 h. An ophthalmological consultation identified K-F rings. The patient was treated with DPMAS plus half-volume plasma exchange therapy for 5 times, combined with comprehensive medical treatment, and continued to receive iodine therapy for hyperthyroidism. Genetic testing revealed a pathogenic variation in the ATP7B gene, with the mutation site being c.2975C>T (p.Pro992Leu). The patient was diagnosed with Wilson's disease, combined with acute-on-chronic liver failure and hyperthyroidism. After artificial liver therapy combined with comprehensive medical treatment, the patient's liver function and coagulation improved, and thyroid function stabilized. The patient is currently taking penicillamine for treatment. Follow-up outpatient examinations showed completely normal liver function and coagulation, as well as normal thyroid function. His daughter also underwent genetic testing and was found to carry the same gene variation.

Conclusions:

Wilson's disease induced acute-on-chronic liver failure is a critical condition, and the combination of hyperthyroidism increases the difficulty of treatment. Liver transplantation is an effective rescue method. In the context of a shortage of liver sources, artificial liver therapy combined with comprehensive medical treatment still achieves very effective treatment results.

Keyword(s) :

2597 - Asthenia Anaemia Liver cirrhosis Brucellosis

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Background : Brucellosis is a zoonotic disease typically presenting with fever, fatigue, and arthralgia. Atypical afebrile cases with multisystem involvement are easily misdiagnosed, especially when complicated with cirrhosis. This study reports such a case to provide clinical references.

Methods: We report a 43-year-old male with alcoholic cirrhosis presented with fatigue for over one month, accompanied by anemia, shoulder arthritis, lower extremity numbness, dizziness, and hyperpigmentation (no fever/abdominal pain). Laboratory findings: severe anemia (Hb 56g/L, unimproved post-transfusion), elevated immunoglobulins, negative hemolysis screening, polyclonal hypergammaglobulinemia, transudative ascites, and *Brucella* spp. isolated from ascitic fluid culture.

Results: Differential diagnoses (cirrhosis-related anemia, POEMS syndrome, hemolytic anemia) were ruled out. Diagnosis was confirmed by SAAG 2.95g/L, 3-day positive ascitic fluid culture, and serum agglutination test (1:200). Piperacillin-tazobactam (4.5g q8h) for 48h rendered ascitic fluid mNGS negative. After 2-month anti-infective therapy (ceftriaxone/moxifloxacin/doxycycline), Hb improved to 71g/L with ascites absorption; the patient continues moxifloxacin 0.4g QD + doxycycline 0.1g q12h.

Conclusion : Brucellosis' "anemia-ascites-shoulder pain" triad is non-specific. Cirrhotic ascites with SAAG >1.1g/L requires culture (transudate ≠ sterile). Early positive culture indicates high bacterial load. Piperacillin-tazobactam may be effective for abdominal brucellosis, mNGS negativity may relate to technique limitations, reduced bacterial load, or treatment efficacy. Cirrhotic patients with anemia need comprehensive etiology screening.

Keyword(s) : Liver cirrhosis, Brucellosis, Asthenia, Anaemia

2598 - Research on Artificial Liver Treatment for Non Hepatocellular Carcinoma Patients with Severe Immune Checkpoints Related Liver Injury

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Background:

As immunotherapy is widely applied in various tumors, immune-related liver injury, as an important part of drug-induced liver injury, has attracted increasing attention. The majority of patients recover liver function after hormone treatment, but patients with poor response to hormone therapy pose a challenge for clinicians.



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Materials and Methods:

This study reviewed 18 patients with liver injury caused by immune checkpoint inhibitors who were admitted to Tianjin Third Central Hospital after October 2020. We have meticulously documented the patients' anti-tumor medication usage, clinical biochemical indicators, and artificial liver conditions, and will continue to follow up until December 31, 2025.

Results:

There were 17 males and 3 females with an average age of 62.20 ± 11.18 years. The tumors included 8 cases of lung cancer, 6 cases of gastric cancer, 1 case of esophageal cancer, 1 case of colon cancer, 1 case of soft tissue malignancy, and 1 case of bile duct cancer. Among them, there were 2 patients with chronic viral hepatitis B and 1 patient with liver metastasis. The immune checkpoint drugs used included Tislelizumab, Indilimumab, Carilimumab, Tremelimumab, and Soolilimumab. Upon admission, the patients' liver function tests showed ALT 487.95 ± 291.49 U/L, GGT 637.35 ± 440.93 U/L, TBIL 221.89 ± 98.84 μ mol/L, total bile acid 265.41 ± 150.95 μ mol/L, BUN 7.20 ± 4.44 mmol/L, Cr 73.79 ± 45.92 μ mol/L, NR 1.13 ± 0.44 , IgG 17.41 ± 6.39 g/L, ANA 1:100 in 5 cases, and ANA 1:320 in 1 case. The patients did not respond well to hormone and drug treatments and were all given artificial liver therapy. The modes of artificial liver therapy included plasma exchange, plasma adsorption, dual plasma molecular adsorption, and dual plasma molecular adsorption combined with plasma exchange. The number of treatments ranged from 1 to 20. Among them, 12 patients died, 8 patients survived, and 3 patients resumed immunotherapy.

Conclusions:

Immune checkpoint-related liver injury is a significant adverse reaction affecting the survival of cancer patients. On the one hand, patients may have underlying chronic liver disease that is not screened before cancer treatment. On the other hand, various combination therapies such as chemotherapy and targeted therapy may exacerbate liver injury. Therefore, hormone therapy is ineffective, and patients may even progress to liver failure, with a high mortality rate. Given that cancer patients often do not have the opportunity for liver transplantation, artificial liver therapy may become an important treatment method for rescuing immune-related liver injury.

Keyword(s) :

2599 - Residual metabolic and steatotic risk in chronic viral hepatitis real world evidence from the

Italian PITER cohort

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Background / Hypothesis:

In chronic viral hepatitis, liver disease progression is mainly driven by viral injury. However, steatosis and metabolic dysfunction features included in the MASLD construct (BMI ≥ 25 kg/m², diabetes, hypertension, dyslipidemia) may represent an additional residual risk in patients with eradicated HCV RNA, controlled HBV infection, and ongoing chronic HDV infection.

Materials and Methods:

Cross-sectional analysis of a nationwide Italian PITER cohort. We described the demographics, cirrhosis and HCC prevalence, and the distribution of steatosis and metabolic dysfunction features in the cohorts for HCV, HBV, and HDV. In HBV and HCV, we compared the distributions of ALT, GGT, and fibrosis stage between patients with and without steatosis and metabolic dysfunction.

Results:

We evaluated 6,804 HCV, 4,203 HBV, and 694 HDV patients. Median ages were 62, 59, and 56 years, and males accounted for 54.5%, 62.1%, and 61.0%, respectively. Cirrhosis prevalence was 50.5% (HCV), 26.5% (HBV) and 64.5% (HDV); HCC prevalence was 4.0%, 3.1% and 9.8%, respectively. Steatosis prevalence was 31.1% (HCV), 28.2% (HBV) and 23.9% (HDV). Overweight/obesity prevalence was 49.9% (HCV), 53.7% (HBV), and 46.4% (HDV); diabetes 14.3 (HCV)%, 10.1% (HBV), and 7.1% (HDV); hypertension 33.1% (HCV), 22.1% (HBV), and 19.2% (HDV); dyslipidemia

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5.3% HCV), 22.0% (HBV), and 13.4% (HDV), respectively. In HBV, compared with patients without steatosis plus metabolic dysfunction features, those with this phenotype were older (63 vs 58 years, $p < 0.001$), more frequently Italian-born (89.7% vs 76.4%, $p < 0.001$), and had higher ALT > 35 (17.9% vs 13.4%, $p = 0.021$), higher GGT > 35 (24.4% vs 13.8%, $p < 0.001$), and higher cirrhosis prevalence (24.5% vs 19.2%, $p < 0.001$).

In HCV, compared with patients without steatosis plus metabolic dysfunction features, those with this phenotype had a significantly different fibrosis stage distribution ($p < 0.001$), with lower F0–F1 (22.6% vs 28.0%) and higher F3 (15.7% vs 11.0%), and F4 (51.3% vs 49.2%).

In multivariable models, steatosis plus metabolic dysfunction features remained independently associated with advanced fibrosis in HBV (adjusted OR 1.36, 95% CI 1.00–1.85) and HCV (adjusted OR 1.28, 95% CI 1.08–1.51), but not in HDV (adjusted OR 0.87, 95% CI 0.52–1.47 for F3–F4/cirrhosis and/or HCC vs F0–F2 and no HCC).

Conclusions:

Steatosis and metabolic dysfunction features are common in viral hepatitis and identify a residual risk phenotype beyond viral injury. This identifies a large, currently under-recognised population that may be eligible for MASH therapies in real-world practice.

Keyword(s) : viral hepatitis, steatosis, metabolic dysfunction, fibrosis, cirrhosis, real world cohort, risk stratification

2603 - Prevalence and Diagnostic Variability in Cirrhotic Cardiomyopathy A Meta analysis

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Background / Hypothesis:

Cirrhotic cardiomyopathy (CCM) is an underrecognized but clinically significant complication in patients with advanced liver disease. Prevalence estimates in the literature vary widely, likely due to inconsistent diagnostic criteria and methodological heterogeneity. In this meta-analysis, we aimed to estimate the pooled prevalence of CCM and evaluate the influence of diagnostic criteria and reported risk factors through subgroup and meta-regression analyses.

Materials and Methods:

A systematic review was conducted to identify studies reporting the prevalence of CCM among adult cirrhotic patients. Seven studies ($n = 1,138$) met the inclusion criteria and were included in the meta-analysis, which was performed using the Jamovi platform with a random-effects model (REML).

Results:

The overall pooled prevalence of CCM was 39.4% (95% CI: 25.4–53.4%). Heterogeneity was low and statistically insignificant ($I^2 = 0\%$, $Q = 3.74$, $p = 0.71$).

Subgroup analysis according to diagnostic criteria revealed notable differences in prevalence: 48.6% in studies using Cirrhotic Cardiomyopathy Criteria 2019 (CCC 2019), 39.6% with the Montreal definition, and 37.2% with classical criteria. This suggests that variability in diagnostic definitions contributes substantially to the reported differences in prevalence. Linear regression using proBNP reporting and age as predictors yielded no statistically significant associations with prevalence ($R^2 = 11.7\%$, $p = 0.78$). QTc interval reporting could not be included in the regression model, as all studies in the dataset reported QTc, leading to insufficient variability for modeling.

Conclusions: In conclusion, the prevalence of CCM remains high at approximately 40%, though diagnostic criteria significantly influence this estimate. Despite low statistical heterogeneity, methodological variability—particularly in diagnostic definitions—limits comparability across studies. There is a clear need for standardized diagnostic criteria in future research to enable more consistent epidemiological characterization and to identify clinically relevant risk factors.

Keyword(s) : cardiomyopathy, cirrhosis, prevalence

2610 - Comparative Efficacy of Combined Targeted and Immunotherapy Regimens in Unresectable Hepatocellular Carcinoma Impact on Survival and Conversion Surgery Outcomes

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Background / Hypothesis:

The combination of targeted therapy and immunotherapy has emerged as a pivotal strategy for unresectable hepatocellular carcinoma (uHCC), with the potential to enable conversion surgery. However, comparative outcomes among different regimens and

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predictors of long-term survival remain inadequately defined.

Materials and Methods:In this retrospective cohort study, 360 patients with uHCC receiving combined targeted and immunotherapy between January 2020 and September 2025 were included. Patients were stratified into four groups based on regimen: bevacizumab plus PD-1 inhibitor, lenvatinib plus PD-1 inhibitor, donafenib plus PD-1 inhibitor, and apatinib plus PD-1 inhibitor. Overall survival (OS) was analyzed using Kaplan–Meier method and Cox regression. Perioperative outcomes were compared between patients undergoing conversion surgery and those treated with direct resection.

Results:Median OS was 30 months in the bevacizumab group, 28 months in the lenvatinib group, 36 months in the donafenib group, and 30 months in the apatinib group, with no statistically significant difference across groups ($P = 0.373$). Objective response (OR) and subsequent conversion surgery were identified as the strongest prognostic factors. Responders showed significantly longer median OS than non responders (37 vs. 24 months, $P < 0.01$). Patients who underwent conversion surgery achieved a median OS of 51 months, superior to 29 months in those without resection ($P = 0.015$). The conversion therapy group exhibited distinct preoperative alterations, including lower lymphocyte counts, coagulopathy, and elevated transaminases, which were associated with longer operative times and greater intraoperative blood loss.

Conclusions:In patients with uHCC treated with combined targeted immunotherapy, the specific regimen had limited impact on overall survival. Achieving an objective response followed by successful conversion surgery constituted the primary determinant of prolonged survival. The distinct preoperative profile of conversion candidates underscores the need for meticulous perioperative management.

Keyword(s) : Hepatocellular carcinoma; Targeted therapy; Immunotherapy; Conversion therapy; Survival analysis; Hepatectomy.

2611 - Pre emptive Transjugular Intrahepatic Portosystemic Shunt Improves Survival in Advanced Cirrhosis Patients with Gastric Fundal Variceal Bleeding A Multicenter Cohort Study

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Background / Hypothesis:

Although preemptive TIPS (p-TIPS) is the standard of care for high-risk esophageal variceal bleeding, its survival benefit for gastric fundal varices remains uncertain. We aimed to compare the survival outcomes of p-TIPS versus standard care in patients with acute gastric fundal variceal bleeding.

Materials and Methods:

We performed a multicenter, retrospective cohort study of patients with cirrhosis treated for gastric fundal variceal bleeding between June 2019 and June 2024. Patients received either p-TIPS (within 5 days of bleeding) or endoscopic cyanoacrylate injection (ECI) plus NSBB. Propensity score matching (PSM) was used to balance baseline characteristics. The primary endpoint was all-cause mortality.

Results:

The propensity-matched cohort included 836 patients with a median follow-up of 29.2 months. Overall, p-TIPS significantly reduced rebleeding (19.95% vs. 48.53%, $p < 0.001$) and further decompensation (58.32% vs. 77.23%, $p < 0.001$) compared to ECI+NSBB, though overall mortality was similar (26.53% vs. 30.55%, $p = 0.105$). Notably, stratification by liver function revealed a significant survival benefit for p-TIPS in patients with Child-Pugh class B or C (mortality: 28.66% vs. 36.31%, $p = 0.028$), alongside reductions in rebleeding and decompensation. In contrast, for Child-Pugh class A patients, p-TIPS offered no survival advantage (21.89% vs. 15.89%, $p = 0.377$).

Conclusions:

While p-TIPS is superior to ECI+NSBB in preventing rebleeding for all patients with gastric fundal varices, its survival benefit is restricted to those with Child-Pugh class B or C cirrhosis. These findings support the use of p-TIPS as the preferred strategy for high-risk patients, whereas it may be unnecessary for patients with preserved liver function (Child-Pugh A).

Keyword(s) : liver cirrhosis; portal hypertension; gastric varices; TIPS; survival analysis

2613 - Development and validation of a time stratified risk model for acute variceal bleeding in hepatocellular carcinoma

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Background / Hypothesis: Acute variceal bleeding (AVB) in patients with hepatocellular carcinoma (HCC) often delays decision-making regarding tumor-directed therapies. To inform precision clinical decision-making for HCC management, this study aimed to develop and validate a prognostic model using risk factors for AVB occurrence, enabling time-dependent risk stratification of AVB in patients with HCC.

Materials and Methods: A two-center retrospective analysis was performed on patients diagnosed with HCC. A competing risk model was constructed in the training cohort and externally validated in the validation cohort. The primary endpoint was the occurrence of variceal bleeding. The secondary endpoint was all-cause mortality.

Results: Among 457 patients (training cohort: 372 patients; validation cohort: 85 patients), multivariate analysis identified the LDRf classification of esophagogastric varices (hazard ratio [HR], 4.910 [95% confidence interval (CI), 3.594–6.709]), portal vein tumor thrombus grade (HR, 1.628 [95% CI, 1.328–1.997]), and ascites grade (HR, 1.372 [95% CI, 1.139–1.653]) as independent predictors of AVB in HCC. The model stratified patients into low-, medium-, and high-risk groups. In the training cohort, the cumulative incidence of bleeding at 3, 6, 12, and 24 months was (1.5%, 18.0%, 57.6%), (5.1%, 24.0%, 64.7%), (7.3%, 36.0%, 75.3%), and (10.2%, 41.0%, 80.0%), respectively (all $P < 0.0001$). The model demonstrated robust performance in the validation cohort.

Conclusions: The time-dependent risk stratification model based on clinical parameters including the LDRf classification of esophagogastric varices, portal vein tumor thrombus grade, and ascites grade effectively stratifies patients with HCC into distinct risk groups for AVB. By integrating readily accessible clinical parameters, this practical tool holds promise for facilitating and optimizing personalized treatment strategies for patients with HCC.

Keyword(s) : hepatocellular carcinoma, variceal bleeding, risk model, Clinical decision-making

2615 - Liver graft rejection after liver transplantation in Kazakhstan a single center experience

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¹

Background / Hypothesis: Liver graft rejection remains a common and clinically relevant complication after liver transplantation (LT). Despite advances in immunosuppressive therapy, rejection rates and outcomes vary across centers. This study aimed to identify predisposing factors and evaluate treatment response after liver transplantation.

Materials and Methods: Clinical data of 127 patients following LT at our center between January 2013 and December 2025 were retrospectively analyzed.

Results: Rejection was observed in 29 LT recipients (45 episodes) - 14 women and 15 men. 34 episodes were acute liver rejection (ACR) and 11 were chronic liver rejection (CR). Living donor liver transplantation was performed in 71.4% of cases, while 28.6% underwent deceased donor liver transplantation.

The mean age at transplantation was 42 years (range 23-63). HBV/HDV co-infection (44.8%) and primary biliary cholangitis (10.3%) were the most common cause of end stage liver disease.

In univariate analysis, subtherapeutic tacrolimus concentrations and non-compliance with immunosuppressive therapy (57.78%), biliary complications (42.22%) were significantly associated with graft rejection.

Late acute rejection (>91 days) predominated among ACR episodes (n = 25). Liver biopsy according to the Banff criteria was performed in 24 cases of ACR, with mild (50%, n = 12), moderate (37.5%, n = 9), and severe rejection (12.5%, n = 3) based on the rejection activity index (RAI). In 10 cases, ACR was diagnosed clinically.

Pulse corticosteroid therapy was administered in 24 cases of ACR, and was successful in 66.7% cases. Among non-responders, antithymocyte globulin therapy was initiated in 4 patients and was effective in 2 of them. 9 ACR cases treated with immunosuppressive escalation, a response was achieved in 78% of them.

Statistically significant changes were detected in liver function tests + ALT, AST, total bilirubin, ALP ($p < 0.05$).

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All 11 cases of CR were managed by adjustment of immunosuppressive therapy, without clinical response. 5 patients required LT.

Overall survival rates at 1, 3, and 5 years after rejection were 54.8% (95% confidence interval [CI]: 38.4–78.1%), 48.7% (95% CI: 31.9–74.3%), and 36.5% (95% CI: 20.4–65.4%), respectively.

Conclusions: In our study, late ACR was more frequently observed among episodes of acute liver graft rejection. The most common predisposing risk factors were biliary complications and non-compliance with immunosuppressive therapy. ACR generally showed a good response to pulse corticosteroid therapy. In contrast, CR was poorly responsive to medical treatment and frequently required liver retransplantation.

Keyword(s) : Acute rejection, Chronic rejection, Liver transplantation, Immunosuppression

2616 - The influence of hepatitis HBV HCV and HDV viruses on the development of sarcopenia in patients with liver cirrhosis

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Background / Hypothesis:

The presence of long-term chronic liver disease contributes to the development and progression of muscle mass and strength disorders. The number of patients with sarcopenia increases with the progression of liver cirrhosis (LC). In our study, we investigated how HBV, HCV, and HDV viruses affected the development of sarcopenia in patients with LC.

Materials and Methods:

During the period from 2021 to 2024, we consistently included into the study patients with verified LC of HBV, HCV, and HDV aetiology. All patients were divided into grades of LC according to the Child-Pugh (Ch-P) scale in agreement with the criteria. Patients underwent assessment of body mass index (BMI); handgrip strength (less than 20 kg in women, less than 30 kg in men); muscle mass assessment using computed tomography (CT) of native images at the middle of the L3 transverse section of the spinal column (less than 55 cm²/m² for men and less than 39 cm²/m² for women).

Results:

Among 73 patients with liver cirrhosis grade A on the

Ch-P scale, there were 26 patients, with grade B – there were 34, and with grade C – there were 14. It was found that sarcopenia was present in 31% of cases with LC. According to the severity of LC, sarcopenia was found in 6 (8%) patients with Ch-P grade A and in 23% of cases with grades B and C. Regardless of the etiology, it was 1.2 times more common in patients who had both HBV and HDV (95% CI: 0.23-6.90; p=1). In the group of patients with sarcopenia, viral hepatitis was diagnosed on a median of 2009 ± 2.3 years, and in patients without sarcopenia, on a median of 2013 ± 2.5 years (p=0.26). Early onset of viral hepatitis, on average 4 years with a high viral load of HBV DNA 6.9 log₄ ± 6.9 log₄ IU/ml, led to the development of sarcopenia in 57% of cases.

Conclusions:

Perhaps the increase in the incidence of sarcopenia is associated with progressive destruction of hepatocytes during active replication of HBV viruses, impaired detoxification function of the liver, high levels of ammonia in the blood, and the presence of portal hypertension. Consequently, patients need to receive effective antiviral therapy against viral hepatitis with monitoring of HBV DNA levels in the blood.

Keyword(s) : liver cirrhosis, sarcopenia, viral hepatitis

2618 - Alistipes putredinis Alleviates Metabolic Associated Fatty Liver Disease via the Acetate GPR43 Axis by Suppressing Neutrophil ROS Generation

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Background / Hypothesis:

Intestinal dysbiosis contributes to the pathogenesis of metabolic-associated fatty liver disease (MAFLD), though precise bacterial targets and mechanisms remain incompletely elucidated. This study investigated whether the depletion of *Alistipes putredinis* (AP) in adolescents obesity-associated MAFLD is causally linked to disease progression and aimed to delineate its downstream signaling pathway.

Materials and Methods:

Fecal samples from 29 obese adolescents with MAFLD and 17 healthy controls in Hainan Province were analyzed by metagenomic sequencing. A high-fat diet-induced MAFLD mouse model was established and

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randomized into control, MAFLD, and AP-gavaged groups (12 weeks). Untargeted metabolomics and targeted short-chain fatty acid profiling were performed on fecal, hepatic, and AP culture supernatant samples. Liver transcriptomics, Western blot, and co-immunoprecipitation were employed to identify and validate involved molecular pathways.

Results:

AP abundance was significantly lower in MAFLD patients compared with controls. AP supplementation ameliorated hepatic lipid accumulation in MAFLD mice. Metabolomic analysis identified acetate as the principal mediator of AP's protective effects. Acetate derived from AP activated the GPR43 receptor on hepatic neutrophils, inhibiting intracellular Ca^{2+} flux and NADPH oxidase 2 (NOX2) activity, thereby reducing reactive oxygen species (ROS) generation. Reduced ROS levels attenuated JNK phosphorylation, decreased LXR α binding to the SREBP-1c promoter, and suppressed proteolytic activation of SREBP-1c, leading to downregulation of key lipogenic enzymes ACC1 and FASN.

Conclusions:

Alistipes putredinis ameliorates MAFLD via acetate-mediated activation of the GPR43 receptor on hepatic neutrophils, which suppresses the Ca^{2+} -NOX2-ROS signaling cascade and inhibits ROS-dependent SREBP-1c activation and lipogenic gene expression. These findings reveal a previously unrecognized gut microbiota-neutrophil signaling axis as a potential therapeutic target in MAFLD.

Keyword(s) : Adolescents; Metabolic-associated fatty liver disease; *Alistipes putredinis*; acetate; GPR43; neutrophil; reactive oxygen species; SREBP-1c

2621 - Construction and Evaluation of a Nobiletin Hepatic Targeted Nanodelivery System for the Treatment of MASLD

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Background:

The disease spectrum of MASLD encompasses a continuous pathological progression from simple hepatocellular steatosis, to MASH accompanied by hepatocyte injury and inflammation, and further to liver fibrosis, cirrhosis, and even hepatocellular carcinoma.

The four key pathological components, namely lipotoxicity, hypoxia, inflammation, and fibrosis, do not advance in a linear manner; instead, they constitute a tightly coupled, self-amplifying vicious network cycle. Interventions targeting any single node can be counteracted by other persistently active driving forces within the cycle, which explains why numerous therapeutic strategies directed at a single target have exhibited limited efficacy in clinical trials. Therefore, the development of novel therapeutic strategies capable of concurrently intervening at multiple nodes and exerting the potential to break this cycle has become an urgent need and a major focus of current research.

Materials and Methods:As a natural polymethoxylated flavonoid extracted from citrus peels, nobiletin has exhibited multifaceted ameliorative effects against MASLD/MASH in numerous preclinical studies. Its pharmacological mechanisms involve multiple processes associated with the aforementioned vicious cycle, including antioxidation, anti-inflammation, regulation of lipid metabolism, and potential anti-fibrotic activity. Nanozyme-based delivery platforms present unique and integrative advantages, which elevate them beyond conventional carriers to act as an integrated "theranostic-delivery" or "therapy-delivery" system. The core value of nanozymes lies in their intrinsic enzyme-mimetic catalytic activity. A mild in situ hydrofluoric acid generation etching method is commonly employed. Using Ti3AlC2 MAX phase as the precursor, a mixed solution of hydrochloric acid and lithium fluoride is utilized as the etchant. LiF reacts with HCl to generate HF in situ, and the Al atomic layers are selectively etched away under controlled conditions, yielding multilayered Ti3C2Tx MXene (where T denotes surface terminal groups, i.e., -OH, -O, and -F). Residual etchant and byproducts are removed via repeated centrifugation and washing. Subsequently, gentle ultrasonication or an intercalation-exfoliation process is performed under inert gas protection to further delaminate the multilayered MXene into few-layered or single-layered nanosheets, so as to achieve a larger specific surface area and more active edge sites. A stably dispersed colloidal solution of few-layered MXene is obtained by centrifugation, or the product is freeze-dried into powder for subsequent use.

Results Conclusions:Nanomaterials can break the pathological vicious cycle mediated by lipotoxicity, play a role in

Keyword(s) : MASLD;Nobiletin;MXene/CeO₂@Chuan

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Chenpi Su" Targeted Nanotherapy
Platform;Nanotherapy

2622 - Itaconic acid improves MASH through regulating ferroptosis by Nrf2 HO 1

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is one of the most prevalent chronic liver diseases worldwide. Its pathogenesis is complex and not yet fully elucidated, and there are currently no widely accepted disease-specific therapeutic agents. In recent years, itaconate has attracted increasing attention because of its anti-inflammatory and antioxidant properties. Accumulating evidence indicates that ferroptosis is involved in the initiation and progression of MAFLD, and that inhibition of ferroptosis can alleviate disease severity. However, whether itaconate mitigates MAFLD through the regulation of ferroptosis remains unclear.

Materials and Methods:

An in vivo MASH model was established by feeding wild-type mice and myeloid-specific ACOD1 knockout mice a high-fat, high-sugar, high-cholesterol diet for 21 weeks, followed by intervention with 4-octyl itaconate (4-OI). In parallel, an in vitro MASH model was generated by incubating AML-12 cells with free fatty acids for 24 hours.

Results:

Compared with control mice, myeloid-specific ACOD1 knockout mice exhibited more pronounced hepatic steatosis, inflammation, and fibrosis, accompanied by enhanced oxidative stress, aggravated mitochondrial damage, and increased ferroptosis, along with marked downregulation of the Nrf2/HO-1 signaling pathway. In contrast, intraperitoneal administration of 4-OI significantly attenuated GAN diet-induced hepatic steatosis and inflammation, and alleviated mitochondrial damage and ferroptosis. In vitro, 4-OI similarly improved steatosis, inflammatory responses, lipid metabolic dysregulation, and ferroptosis in the NASH cell model, whereas silencing Nrf2 partially reversed the protective effects of 4-OI.

Conclusions: Collectively, these findings suggest that itaconate exerts a protective effect against GAN diet-induced hepatotoxicity by activating Nrf2-dependent antioxidant pathways and suppressing ferroptosis, providing new mechanistic insights into potential therapeutic targets for MAFLD.

Keyword(s) : MAFLD, Nrf2, ferroptosis, oxidative stress

2624 - Risk Factors for Liver Fibrosis Severity in Patients with Nonalcoholic Steatohepatitis A Study Based on Liver Pathology

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Background / Hypothesis:

To identify independent risk factors associated with the severity of liver fibrosis in patients with nonalcoholic steatohepatitis (NASH) based on liver pathology results.

Materials and Methods:

A retrospective analysis was conducted on 122 patients diagnosed with NASH via liver biopsy at Tianjin Second People's Hospital between December 2014 and December 2024. Clinical, laboratory, and pathological data were collected. Patients were categorized into F1, F2, and F3 stages based on fibrosis scores. Univariate analysis was used to compare variables across fibrosis and liver pathology stages. We used the Kappa statistic to evaluate the consistency between LSM findings and pathological liver fibrosis staging. Ordinal logistic regression with backward stepwise selection was applied to identify independent risk factors, with odds ratios (ORs) and 95% confidence intervals (CIs) calculated.

Results:

1.The mean age of participants was 41.6 ± 14.2 years, with 66 males (54.1%).According to fibrosis staging, there were 21 cases (17.2%) in F1 stage, 86(70.5%) in F2 stage, and 15(12.3%) in F3 stage. 2. Liver pathology in all groups showed that with the progression of liver fibrosis, the degree of hepatocellular steatosis and interlobular inflammation in all three groups tended to worsen, with statistically significant differences ($P < 0.05$). There was no statistically significant difference in the degree of ballooning degeneration among the three groups.3.Contingency Table Analysis showed that the overall concordance rate between

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LSM measurement results and pathological liver fibrosis staging was 48.4%, with an underestimation rate of 37.7%. Agreement analysis showed poor consistency between LSM and pathological fibrosis staging (Kappa=0.206). 4. ALT and AST levels increased significantly with the progression of hepatocellular steatosis, while only AST levels increased significantly with the progression of interlobular inflammation. These differences were statistically significant ($P < 0.05$). Among patients with different degrees of ballooning degeneration, AST levels showed marginal significance ($P=0.068$). 5. Ordinal logistic regression identified lobular inflammation (OR=5.058, $P<0.001$), hepatic steatosis (OR=2.768, $P=0.003$), diabetes (OR=3.815, $P=0.021$), and elevated LDL levels (OR=1.669, $P=0.025$) as independent risk factors associated with the severity of liver fibrosis in patients with NASH.

Conclusions:

Lobular inflammation, hepatic steatosis, diabetes, and elevated LDL levels are independent risk factors associated with the severity of liver fibrosis in patients with NASH.

Keyword(s) : Nonalcoholic steatohepatitis; liver fibrosis; liver biopsy; risk factors

2628 - Effect of Rifaximin on Platelet Count in Hepatitis C-Related Liver Cirrhosis A Randomized Controlled Clinical Trial

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Background / Hypothesis:

Thrombocytopenia (TP) is the earliest and most common cytopenia in patients with liver cirrhosis (LC), with a reported prevalence of up to 77%. TP may interfere with routine clinical care by delaying or complicating diagnostic and therapeutic procedures. Elevated circulating endotoxins and pro-inflammatory cytokines in LC contribute to increased platelet consumption and the development of TP. Rifaximin is a non-absorbable, broad-spectrum antibiotic targeting aerobic and anaerobic Gram-positive and Gram-

negative gastrointestinal microorganisms, and its administration has been associated with reduced endotoxemia.

The study aimed to assess the effect of intestinal decontamination with rifaximin on thrombocytopenia in patients with hepatitis C-related liver cirrhosis

Materials and Methods:

This randomized controlled clinical trial included 165 cirrhotic patients with thrombocytopenia attending hepatology clinics at two tertiary hospitals in Egypt. Cirrhosis severity was assessed using the Child-Pugh classification. Inclusion criteria were: absence of active bacterial infection; no history of variceal bleeding within two weeks before enrollment; no antibiotic use (including rifaximin) or cytokine-modifying agents within eight weeks before enrollment; absence of neoplasia, including hepatocellular carcinoma; and no red blood cell or plasma transfusion within one month before enrollment. Thrombocytopenia was classified as mild ($>75,000/\text{mm}^3$), moderate ($50,000-75,000/\text{mm}^3$), or severe ($<50,000/\text{mm}^3$).

Patients were randomized into two groups: group A ($n=85$) received rifaximin 550 mg twice daily for four weeks, while group B ($n=80$) served as the control group and did not receive rifaximin. All patients underwent baseline clinical evaluation and laboratory investigations, including complete blood count, liver function tests, and renal function tests. Platelet counts were reassessed after four weeks.

Results:

Mean platelet count increased significantly in the rifaximin group compared with the control group (median increase of 14.5%, $p < 0.001$). A significant improvement in platelet count was observed in patients with Child-Pugh class B and C cirrhosis ($p < 0.001$), whereas no significant change was observed in patients with Child-Pugh class A cirrhosis ($p=0.506$). A greater increase in platelet count was observed in patients with moderate to severe thrombocytopenia (24.6%) than in those with mild thrombocytopenia (8.1%).

Conclusions:

Rifaximin therapy significantly improves thrombocytopenia in patients with advanced hepatitis

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C-related liver cirrhosis

Keyword(s) : Liver cirrhosis; Thrombocytopenia; Rifaximin

2629 - Clinical characteristics and risk factors for early liver function abnormalities in young adults with MAFLD

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Background / Hypothesis: Metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly prevalent among young adults, yet early liver function abnormalities are often overlooked. We aimed to identify risk factors for early liver function abnormalities in young adults with MAFLD.

Materials and Methods: A total of 1008 young adults (18-45 years) with MAFLD detected during health examinations at Fudan University Pudong Medical Center were retrospectively analyzed. Participants were categorized into abnormal (n=506) and normal (n=502) liver function group. Variables significant in univariate analysis were further refined using least absolute shrinkage and selection operator (LASSO) regression and multivariate logistic regression.

Results: The prevalence of MAFLD among young adults was 19.9%, with 33.1% showing early liver function abnormalities—significantly higher than in middle-aged and older adults (33.1% vs 15.6%, $P < 0.05$). The degree of alanine aminotransferase elevation was closely associated with metabolic disturbances, with central obesity, hypercholesterolemia, and hyperuricemia significantly more prevalent in the moderate and severe elevation groups. Independent risk factors included waist circumference ($P < 0.001$, OR = 1.034), hemoglobin ($P < 0.001$, OR = 1.043), uric acid ($P < 0.001$, OR = 1.005), and low-density lipoprotein cholesterol ($P < 0.001$, OR = 2.04) as independent risk factors, while age ($P = 0.008$, OR = 0.967) and high-density lipoprotein cholesterol ($P = 0.001$, OR = 0.267) were protective.

Conclusions: Early liver function abnormalities are common in young adults with MAFLD. Identifying modifiable metabolic risk factors facilitates early stratification and timely intervention to delay or reverse disease progression.

Keyword(s) : Metabolic dysfunction-associated fatty

liver disease; Young adults; Liver function abnormalities; Risk factors; Metabolic disturbances

2631 - High ammonia promotes EHHADH dependent pyrimidine degradation to induce inflammatory cell death in HCC

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Background / Hypothesis

Metabolic reprogramming characterizes Hepatocellular Carcinoma (HCC), where urea cycle disruption leads to ammonia buildup in the liver. Ammonia influences tumor cells differently based on its concentration: low levels encourage tumor growth, while high levels are toxic and can cause cell death. Given that high systemic ammonia correlates with poor patient outcomes, this study seeks to identify targets involved in ammonia-induced HCC cell death to create new treatments.

Materials and Methods:

We developed a new transgenic HCC mouse model on a high-ammonia diet and analyzed the role of the high-ammonia-induced gene EHHADH in cell death through metabolomics, metabolic flux analysis, transcriptomics, proteomics, and modification-omics. Cell viability was measured by LDH and HMGB1 release, with inflammatory cell death visualized via electron microscopy. Dihydropyrimidine Dehydrogenase (DPYD) activity was assessed through protein purification and enzymatic assays. We also explored how EHHADH overexpression affects the HCC immune microenvironment using single-cell RNA sequencing. Lastly, we evaluated the therapeutic potential of combining an EHHADH agonist with an anti-PD-1 monoclonal antibody in vivo.

Results:

High ammonia treatment effectively inhibited HCC tumor growth by inducing inflammatory cell death marked by Cleaved-Caspase-3 and GSDME-N. This process involved the accumulation of polyunsaturated fatty acids (PUFAs), which activated PPARA to increase EHHADH expression. Elevated EHHADH boosted peroxisomal ROS production and disrupted the mitochondrial TCA cycle, leading to cytosolic Acetyl-CoA accumulation. This shift caused DPYD acetylation, enhancing its activity and accelerating pyrimidine catabolism, resulting in nucleotide depletion and

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replication stress, which ultimately triggered GSDME-dependent pyroptosis. Furthermore, targeting EHHADH-mediated inflammatory cell death substantially augmented the infiltration of phagocytes and CD8⁺ T cells into the tumor microenvironment (TME), thereby synergistically enhancing the efficacy of anti-PD-1 immunotherapy.

Conclusions:

This study elucidates a novel "Metabolic-Immune" axis in which elevated ammonia levels exploit the PPAR- α -EHHADH signaling pathway to promote pyrimidine catabolism and replication stress, consequently inducing inflammatory cell death. Targeting EHHADH emerges as a promising therapeutic strategy to inhibit tumor growth and enhance the sensitivity of hepatocellular carcinoma to immune checkpoint blockade therapy.

Keyword(s) : Hepatocellular Carcinoma; EHHADH; Ammonia; Pyrimidine Metabolism; Inflammatory Cell Death

2632 - Study on the Efficacy and Mechanism of the Spleen Strengthening and Lipid Regulating herbal formula in Treating MAFLD An Exploration Based on the PPAR Pathway

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Background / Hypothesis: Metabolic dysfunction-associated fatty liver disease (MAFLD) has become a global health issue due to its high prevalence and widespread health consequences. The spleen-strengthening and lipid-regulating herbal formula (JPTZ), a traditional Chinese preparation, is used to treat hyperlipidemia, obesity, and even MAFLD, but its molecular mechanisms remain unclear.

Materials and Methods: Clinically, data on various indicators were collected, including body weight, waist circumference, plasma lipids, fasting glucose and insulin, HbA1c, liver and renal function tests, complete blood count, and FibroTouch test, both before and after a 3 - month administration of JPTZ in patients with MAFLD, in order to assess its efficacy and safety in the treatment of MAFLD. For basic research, establish a mouse model of MAFLD to evaluate the therapeutic efficacy of JPTZ and determine the key regulatory pathways through which JPTZ improves MAFLD via mRNA sequencing. Subsequently, HPLC-QTOF/MS and ADMET rules were utilized to identify the candidate bioactive constituents in JPTZ, and the corresponding

putative targets were predicted via the TCMIP platform. An interaction network was constructed to link MAFLD-related differentially expressed genes and JPTZ's putative targets, and to screen the key targets using CytoScape, which were verified through a series of in-vivo experiments.

Results: JPTZ significantly improves abnormal liver function and lipid metabolism disorders in MAFLD patients, as well as FibroTouch examination parameters. In animal studies, JPTZ alleviated hepatic steatosis, liver injury, and fibrosis in high-fat diet-induced MAFLD mice, while improving their glucose tolerance and insulin resistance. Chemical profiling identified 178 constituents in JPTZ, 43 of which met the optimal ADMET criteria. The transcriptomic results indicated that the PPAR signaling pathway was one of the key pathways through which JPTZ exerted its effects. The experiments further verified that JPTZ directly bound to PPAR α and PPAR γ , activated downstream signaling, regulated lipid metabolism, and alleviate inflammatory response.

Conclusions: JPTZ is a potential pan-PPAR agonist that improves lipid metabolism and alleviates inflammation by modulating the PPAR pathway, thereby treating MAFLD and offering a novel therapeutic strategy for this condition.

Keyword(s) : Metabolic dysfunction-associated fatty liver disease; the spleen-strengthening and lipid-regulating herbal formula; PPAR signaling pathway; Lipid metabolism

2633 - Chylous Ascites in Liver Cirrhosis Diverse Mechanisms Diagnostic Challenges and Multidisciplinary Management — A Case Series

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Background:

Chylous ascites (CA), defined as triglyceride-rich milky peritoneal fluid (>200 mg/dL), is an uncommon but clinically significant complication of advanced liver disease, accounting for approximately 1 in 20,000 hospital admissions. In cirrhosis, CA may result from portal hypertension-related lymphatic rupture, malignancy-associated obstruction, or iatrogenic lymphatic injury. Because its presentation may mimic

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refractory or infected ascites, delayed recognition can lead to repeated procedures, malnutrition, and poor outcomes. We describe the clinical spectrum, mechanisms, and management challenges of CA in cirrhotic patients.

Methods:

We conducted a descriptive case series of three adults with established cirrhosis who developed CA. Clinical features, imaging, ascitic fluid analysis, etiologies, treatment strategies, and outcomes were reviewed. Diagnosis was confirmed by diagnostic paracentesis demonstrating elevated ascitic triglycerides.

Results:

All patients presented with progressive abdominal distension and turbid or milky ascites. Ascitic triglyceride levels ranged from 248 to 1,356 mg/dL. Etiologies included lymphatic obstruction from non-Hodgkin lymphoma with cirrhosis, portal hypertension with repeated paracentesis-related lymphatic trauma, and metabolic dysfunction-associated steatotic liver disease-related cirrhosis complicated by hepatohydrothorax. Management targeted the underlying cause and included chemotherapy, sodium restriction, diuretics, medium-chain triglyceride-based low-fat diet, albumin, octreotide, and serial therapeutic paracentesis. Resolution or reduction of chylous output was achieved in two patients, while one progressed with cirrhosis-related complications.

Conclusion:

Chylous ascites represents a rare but important manifestation of decompensated cirrhosis with heterogeneous mechanisms. Routine triglyceride analysis of atypical or turbid ascites facilitates early diagnosis. A multidisciplinary, etiology-directed approach combining nutritional, pharmacologic, and disease-specific therapy may reduce morbidity and unnecessary invasive interventions.

Keyword(s) : chylous ascites, cirrhosis, portal hypertension, MASLD, case series

2636 - Macrophage ACAT1 aggravates alcoholic hepatitis by inducing mitochondrial damage and NLRP3 activation

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Background / Hypothesis:

ALD is caused by chronic excessive alcohol consumption, with current clinical treatments limited and ineffective. As a key esterification enzyme in macrophages, ACAT1 mediates cholesterol esterification involved in inflammatory regulation. This study investigated the expression, function and associated pathway enrichment of ACAT1 in ALD, and further verified that ACAT1 modulates alcohol- and LPS-induced macrophage inflammation by inducing mitochondrial damage to activate the NLRP3 inflammasome, so as to explore its potential as a therapeutic target for ALD.

Materials and Methods:

In vitro, macrophages were stimulated with alcohol plus LPS; Western blot and RT-qPCR detected the protein and mRNA levels of ACAT1 and NLRP3 inflammasome-related inflammatory molecules, and immunofluorescence co-localization verified the expression of F4/80 and ACAT1. Flow cytometry assessed ROS, mtROS and MMP, TEM observed mitochondrial morphology, and transcriptome sequencing analyzed ALD-associated signaling pathways. Additionally, ACAT1 inhibition by K-604 and knockdown by siRNA were performed to evaluate its regulatory effects on inflammatory molecules in macrophages. In vivo, an ALD mouse model was established and treated with K-604 inhibitor and clodronate liposomes; liver histopathology was examined by H&E, Oil Red O and ROS immunofluorescence staining, and liver function was evaluated via ALT, AST levels and liver-to-body weight ratio. RT-qPCR and Western blot quantified ACAT1 and inflammatory factor expression in liver tissues, while ELISA detected serum inflammatory factors and triglyceride levels in mice.

Results:

- (1) ACAT1 mRNA and protein were significantly upregulated in the ALD model; in E+L-stimulated macrophages, ACAT1 was synchronously elevated with the macrophage marker F4/80 and colocalized with it.
- (2) E+L significantly upregulated the transcriptional and protein levels of proinflammatory factors and NLRP3 inflammasome-related molecules in macrophages; ACAT1 inhibition/knockdown reversed

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this effect, whereas its overexpression further promoted their expression.

(3) E+L induced increased ROS and mtROS production, mitochondrial damage, and lipid droplet accumulation in macrophages, which were significantly ameliorated by K-604. Transcriptome sequencing identified the NOD-like receptor signaling pathway as a key ACAT1-regulated differential pathway in ALD.

(4) In vivo, hepatic ACAT1 expression was significantly elevated in ALD model mice. Macrophage depletion or K-604 administration significantly reduced hepatic ALT, AST, TG and ROS levels, diminished lipid deposition, and downregulated hepatic proinflammatory factors.

Conclusions:

ACAT1 is highly expressed in macrophages and acts as a key regulator of ALD inflammation. It induces mitochondrial damage to activate the NLRP3 inflammasome, promoting the expression and release of proinflammatory cytokines and ROS, and enhancing lipid deposition to exacerbate macrophage inflammation.

Keyword(s) : ALD, ACAT1, NLRP3, mitochondrial damage

2637 - Predictors of minimal clinically important improvement in cardiorespiratory fitness following unsupervised exercise training in metabolic dysfunction associated steatotic liver disease

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Background: Our previous randomized controlled trial showed that patients with metabolic dysfunction-associated steatotic liver disease (MASLD) following unsupervised high-intensity interval training (HIIT) and moderate-intensity continuous training (MCT) showed varied improvement in cardiorespiratory fitness and exercise performance. This study aimed to identify factors associated with the improvement in cardiorespiratory fitness which reached minimal clinically important difference (MCID) following HIIT or MCT in this patient population.

Materials and Methods: We analyzed data from a randomized controlled trial which recruited 95 patients with MASLD who were randomly assigned to an

unsupervised HIIT or MCT program for 24 weeks. Ninety-four participants with complete data at baseline and at week 24 were included in this study. Participants exhibiting a change in peak oxygen consumption ($\dot{V}O_{2peak}$) of ≥ 1 metabolic equivalent (MET) following exercise training were classified as responders and all others were classified as non-responders. Univariate binary logistic regression analyses were performed to screen for independent variables with a p value < 0.10 . These selected variables were further entered multivariate binary logistic regression model to identify predictors associated with clinically important improvement in $\dot{V}O_{2peak}$. The significance level was set at p < 0.05 .

Results: Thirty-eight patients exhibited clinically important improvement in their $\dot{V}O_{2peak}$ (2.1 ± 0.9 MET) following exercise training and were classified as the responders, with 25 patients (65.8%) from the HIIT group and 13 patients (34.2%) from the MCT group. After adjustment for age, sex, and baseline body mass index, patients undergoing HIIT, compared with MCT, had higher odds of reaching MCID in $\dot{V}O_{2peak}$ following exercise training (adjusted odds ratio [aOR] 6.30, 95% confidence interval [CI] 2.11–18.81, p = 0.001). Higher exercise adherence (aOR 1.04, 95% CI 1.02–1.06, p < 0.001) and baseline step counts < 10000 steps/day (aOR 4.87, 95% CI 1.07–22.26, p = 0.041) were also independently associated with greater odds of reaching MCID in $\dot{V}O_{2peak}$ following exercise training, while higher baseline cardiorespiratory fitness ($\dot{V}O_{2peak}$ expressed in MET unit) lowered the odds of clinically significant improvement in $\dot{V}O_{2peak}$ (aOR 0.65, 95% CI 0.45–0.93, p = 0.020).

Conclusions: These findings suggest that patients with MASLD who underwent HIIT, demonstrated higher adherence to exercise training, and had lower baseline physical activity levels are more likely to reach minimal clinically important improvement in cardiorespiratory fitness after 24 weeks of unsupervised exercise training, while higher baseline cardiorespiratory fitness reduces this likelihood.

Keyword(s) : Metabolic dysfunction-associated steatotic liver disease, high-intensity interval training, moderate-intensity continuous training, cardiorespiratory fitness, minimal clinically important improvement

2638 - Machine learning prediction of functional cure to pegylated interferon alpha therapy in two HBV populations advantaged populations and HBeAg negative patients

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Background / Hypothesis: Achieving a functional cure (sustained HBsAg loss) in chronic hepatitis B (CHB) remains a challenge. We aimed to develop and validate baseline prediction models using routine clinical indicators to identify HBeAg-negative patients, particularly the guideline-defined "advantaged population", who are most likely to achieve functional cure with pegylated interferon-alpha (PEG-IFN α) therapy.

Materials and Methods: This retrospective study included 202 HBeAg-negative CHB patients (114 advantaged) for derivation/internal validation and 183 external patients (117 advantaged) for validation. From 48 baseline variables, key predictors were selected via LASSO regression. Two nomogram models were constructed using multivariable logistic regression: the SHAN model for all HBeAg-negative patients and the FLASH-N model specifically for the advantaged population.

Results: The SHAN model, based on four predictors (log(HBsAg+1), age, neutrophil percentage, sex), achieved areas under the curve (AUCs) of 0.908 (training) and 0.949 (testing). The FLASH-N model, incorporating two additional variables (alpha-fetoprotein, lactate dehydrogenase), showed an AUC of 0.920 (bootstrap-corrected 0.889). In external validation, the SHAN and FLASH-N models maintained robust discriminative ability with AUCs of 0.861 and 0.800, respectively. Both models demonstrated excellent calibration and provided superior clinical net benefit in decision curve analysis.

Conclusions: We developed two practical nomograms using readily available baseline variables. These models effectively predict the probability of functional cure with PEG-IFN α therapy, offering valuable tools for personalized clinical decision-making and refined risk stratification in HBeAg-negative and advantaged CHB patients.

Keyword(s) : Chronic hepatitis B; Functional cure; Prediction model; Pegylated interferon-alpha; Machine learning

2642 - Design development and pilot evaluation of a comprehensive liver disease registry in the Philippines

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Background / Hypothesis: Liver diseases impose a growing public health burden in the Philippines, yet the lack of a standardized national registry limits surveillance, research, and health planning. This study describes the design, development, and pilot evaluation of a comprehensive liver disease registry targeting six priority conditions – Hepatitis B, Hepatitis C, Metabolic Dysfunction-Associated Steatotic Liver Disease, Alcohol-related Liver Disease, Liver Cirrhosis, and Hepatocellular Carcinoma. We focused on feasibility, usability, data quality, and readiness for national scale-up.

Materials and Methods: We conducted a mixed-methods implementation study. Digital health readiness was evaluated using the E-Ready questionnaire. Registry design was informed by a consensus dataset established through a three-round modified Delphi process (reported separately). The architecture enabled manual entry, bulk uploads, and application programming interface (API)-based submissions. Pilot deployment occurred in one public tertiary hospital and one private specialty hospital, enrolling 181 patients. We assessed registry quality and functionality using the CIPROS checklist and usability with the System Usability Scale (SUS). Preliminary data quality assessment was performed on 112 required variables from 47 randomly selected patients.

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Qualitative feedback from end-users was thematically analyzed.

Results: Readiness assessment revealed strong individual competencies and positive organizational attitudes towards registry use, but highlighted perceived structural constraints, including limited time, resources, and trained personnel. The registry was deployed as a secure, FHIR-based web platform with built-in validation, audit trails, and modular user management functions. A total of 181 patients were enrolled, with 77.9% recruited from the public hospital. Mean SUS scores were 70.8 post-beta and 67.1 post-training/deployment, indicating marginal to acceptable usability. Data quality assessment demonstrated 100% structural completeness, 67.1% data completeness, and 77.4% data accuracy, with errors stemming from unstructured documentation, manual encoding, and variability in routine clinical recording practices. CIPROS evaluation demonstrated strengths in software architecture, standards alignment, security and privacy compliance, organizational governance, and training, while highlighting gaps in interfaces and interoperability, analytical functionality, and sustainability mechanisms.

Conclusions: The pilot demonstrates the feasibility of implementing a comprehensive, multi-disease liver registry in the Philippines. While core architecture, governance, and usability are acceptable for early deployment, improvements in interoperability, workflow integration, and analytical capabilities are essential for scale-up and institutionalization. Our findings also reveal critical trade-offs between data comprehensiveness, workflow burden, and data reliability, providing actionable insights to inform system refinement and expansion of disease registries in similar resource-constrained settings.

Keyword(s) : liver disease registry, clinical registries, health information systems, usability evaluation, data quality assessment, interoperability, developing countries, Philippines

2645 - Traditional Medicine Yiqi Huoxue Relieves Nonalcoholic Steatohepatitis by Remodeling Intestinal Microbiota and Metabolites

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Background/Hypothesis: Non-alcoholic steatohepatitis

(NASH) lacks effective therapy. Yiqi Huoxue Formula (YQHX) shows potential, but its mechanisms remain unclear. YQHX alleviates NASH by modulating the gut-liver axis and contains active components that directly inhibit hepatocyte necroptosis.

Materials and Methods: C57BL/6J mice were fed a high-fat diet for 120 days to establish NASH, then treated with YQHX for 14 days. Metabolic parameters, liver pathology, gut microbiota (16S sequencing), serum metabolomics, and hepatic transcriptomics were analyzed. Intestinal barrier integrity and key proteins were examined. YQHX-derived components were identified via UPLC-MS/MS and screened through molecular docking. Cimifugin's effects were validated in FFA-induced AML12 cells using flow cytometry, TEM, and functional assays.

Results: YQHX improved metabolic indices, liver steatosis, and inflammation. It restored gut microbiota composition, enhanced intestinal barrier function, and modulated serum metabolites. Transcriptomics revealed inhibition of necroptosis and inflammatory pathways. YQHX reduced hepatic p-MLKL and macrophage infiltration. Cimifugin (10 μ M) inhibited FFA-induced necroptosis, improved mitochondrial structure and function, and enhanced fatty acid β -oxidation.

Conclusion: YQHX alleviates NASH through gut-liver axis modulation, with Cimifugin directly targeting mitochondrial function and necroptosis. This study provides mechanistic insights supporting YQHX's therapeutic potential.

Keyword(s) : Yiqi Huoxue Formula (YQHX), Non-alcoholic steatohepatitis (NASH), Gut-liver axis, Necroptosis

2646 - Exploring the Predictive Value of the DiabetesLiver Score for Liver Fibrosis in Patients with Type 2 Diabetes Mellitus and MAFLD Based on the Dual Disease Nursing Management Model

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Background / Hypothesis:

To explore the predictive value of the DiabetesLiver Score (DLS) for liver fibrosis in patients with type 2

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diabetes mellitus and metabolic dysfunction-associated steatotic liver disease (MAFLD) based on the dual disease nursing management model.

Materials and Methods:

A total of 120 T2DM patients with MAFLD were enrolled in the present study. All patients were given the interventions of the diabetes liver disease nursing management model and followed up for one year. Changes of weight, waist circumference, serological indicators (complete blood count, blood lipids, blood glucose, glycated hemoglobin, liver function), liver transient elastography (TE) examination, as well as indices such as the Fibrosis-4 Index (FIB-4), DLS, and the Non-alcoholic Fatty Liver Disease Fibrosis Score (NFS) were compared before and after the follow-up period. Furthermore, based on Receiver Operator Characteristic (ROC) curve analysis, the diagnostic performance of the three non-invasive models (FIB-4, DLS, and NFS) for liver fibrosis was compared.

Results:

Compared with the pre-follow-up period, the patients showed an increase in albumin after one year of follow-up, along with significant decreases in cholesterol, low-density lipoprotein, fasting blood glucose, DLS, NFS, as well as the CAP value and LSM measured by TE examination. The differences between the two groups were statistically significant ($P < 0.05$). DLS demonstrated good performance in predicting $\text{LSM} \geq 8 \text{ kPa}$, $\geq 10 \text{ kPa}$, and $\geq 12 \text{ kPa}$, with area under the curve (AUC) values of 0.824, 0.973, and 0.981, respectively. Its predictive performance was superior to that of FIB-4 and NFS, whose AUC values were 0.785, 0.922, 0.927 and 0.748, 0.799, 0.889, respectively.

Conclusions:

The Diabetes Liver Disease Nursing Management Model is an effective comprehensive intervention measure, and the DLS demonstrated good performance in identifying liver fibrosis in T2DM population.

Keyword(s) : Diabetes mellitus; Metabolic fatty liver disease; Dual disease nursing management model; DiabetesLiver score

2648 - Comparative Analysis of WHO 2024 EASL 2025 AASLD IDSA 2025 and APASL 2026 CHB Treatment Guidelines Implications for Treatment Eligibility and Antiviral Management Strategies

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Background / Hypothesis:

Major international guidelines for chronic hepatitis B (CHB) differ substantially in treatment thresholds, fibrosis assessment, and nucleos(t)ide analogue (NUCs) discontinuation strategies. This review compared the WHO 2024, EASL 2025, AASLD/IDSA 2025, APASL 2026 guidelines to identify key divergences.

Materials and Methods:

A structured, domain-based comparison of the guidelines was conducted across three key domains: (1) indications for treatment by alanine aminotransferase (ALT), HBV DNA, and fibrosis thresholds; (2) recommended first-line antiviral regimens; and (3) strategies for NA discontinuation. Recommendations were systematically extracted and compared to highlight differences in clinical approach.

Results:

Who and when to treat: WHO 2024 expanded treatment eligibility using a pragmatic framework incorporating significant fibrosis ($\text{APRI} > 0.5$ or transient elastography $> 7.0 \text{ kPa}$), elevated ALT with HBV DNA $> 2,000 \text{ IU/mL}$, presence of co-infections or comorbidities, or persistently elevated ALT when virologic testing is unavailable. EASL 2025 broadened access via a phase-based algorithm integrating HBV DNA and non-invasive fibrosis assessment, including patients with normal ALT but elevated HCC risk.

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AASLD/IDSA 2025 maintained a conservative, risk-stratified approach with strict numeric thresholds (HBV DNA $\geq 20,000$ IU/mL for HBeAg-positive and $\geq 2,000$ IU/mL for HBeAg-negative disease). APASL 2026 adopted a simplified “treat-all” approach, removing most biomarker restrictions to encourage early initiation. All guidelines recommended treatment for patients with cirrhosis.

How to treat: First-line therapy across guidelines is high-genetic-barrier NUCs (entecavir, tenofovir disoproxil fumarate, or tenofovir alafenamide), with tenofovir preferred during pregnancy. WHO recommends tenofovir plus lamivudine (3TC) or tenofovir plus emtricitabine (FTC) as alternative regimens. APASL 2026 uniquely positions pegylated interferon- α as first-line or add-on option to promote HBsAg loss. EASL 2025 most extensively integrates quantitative biomarkers (HBcrAg, HBV RNA, qHBsAg) to guide finite therapy.

When to stop therapy: NUC discontinuation is generally restricted to patients with sustained HBsAg loss, although EASL 2025 and APASL 2026 provide more permissive stopping criteria in selected non-cirrhotic patients.

Conclusions:

Major HBV guidelines reflect different priorities: APASL 2026 adopts the most inclusive treatment approach, EASL 2025 balances broad eligibility with finite-therapy pathways, AASLD/IDSA 2025 emphasizes shared decision-making with predominantly indefinite therapy, and WHO 2024 prioritizes feasibility in resource-limited settings. These differed priorities have implications for implementation and HBV elimination strategies in the Asia-Pacific region.

Keyword(s) : chronic hepatitis B; Clinical Guidelines; biomarkers; pegylated interferon

2650 - The Evolving Role of Vitamin D in Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) From Pathogenesis to Clinical Practice – An Updated Synthesis for 2026

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Background / Hypothesis: The escalating global burden of MASLD underscores the urgent need to elucidate modifiable determinants of disease onset and progression. Vitamin D—a secosteroid hormone with well-established roles in calcium homeostasis, immune modulation, insulin sensitivity, and hepatic metabolic regulation—has emerged as a biologically plausible candidate. Although numerous observational studies consistently associate vitamin D deficiency with higher MASLD prevalence, severity, and adverse histological outcomes, the nature of this association—causal versus epiphenomenal—remains unresolved. This review critically integrates contemporary evidence to advance understanding beyond mere correlation, focusing on mechanistic pathways linking vitamin D status to MASLD pathogenesis—including genetic susceptibility (e.g., VDR variants), gut-liver axis dysregulation, and downstream cellular and molecular targets.

Materials and Methods: We performed a rigorous, non-systematic narrative synthesis prioritizing high-quality, conceptually influential evidence. Key sources included: (1) prospective longitudinal cohort studies and robust Mendelian randomization (MR) investigations evaluating causal inference; (2) RCTs assessing the efficacy of vitamin D supplementation on imaging- or biopsy-confirmed hepatic steatosis, inflammation, and fibrosis; (3) mechanistic preclinical and translational studies examining VDR-mediated signaling in hepatocytes, hepatic stellate cells, Kupffer cells, and intestinal epithelial cells; (4) emerging research on vitamin D-microbiome-bile acid interactions; and (5) epigenomic analyses exploring vitamin D-dependent regulation of genes involved in lipid metabolism, oxidative stress, and fibrogenesis.

Results: Current evidence reveals a multifaceted and context-dependent relationship. While low circulating 25(OH)D concentrations remain strongly and independently associated with MASLD diagnosis, advanced fibrosis, and cardiometabolic comorbidities in observational cohorts, MR analyses do not support a direct causal role for vitamin D deficiency in MASLD development—instead implicating shared underlying drivers such as obesity, physical inactivity, and systemic inflammation. Similarly, RCTs evaluating vitamin D monotherapy have generally failed to demonstrate clinically meaningful reductions in liver fat content or fibrosis stage. Nevertheless, experimental data increasingly highlight vitamin D's regulatory influence on hepatic autophagy flux, mitochondrial bioenergetics, NLRP3 inflammasome activation, and

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endotoxin tolerance. Critically, vitamin D insufficiency appears to potentiate hepatocellular injury under conditions of "second hits", including gut-derived LPS translocation secondary to dysbiosis and increased intestinal permeability. Preliminary subgroup findings suggest that targeted repletion—particularly in patients with baseline 25(OH)D < 20 ng / mL and concomitant high-risk metabolic phenotypes—may yield modest benefits when integrated with comprehensive lifestyle modification.

Conclusions: Vitamin D is a disease modifier, not a primary driver, in MASLD. Universal supplementation is unsupported, but correcting VD deficiency remains a prudent clinical adjunct. Future work must define responsive subgroups and explore its role in combinatorial, precision-hepatology strategies.

Keyword(s) : Vitamin D; Metabolic Dysfunction-Associated Steatotic Liver Disease; Vitamin D receptor (VDR) ; 25-hydroxyvitamin D [25(OH)D]; Lifestyle Modification; Biomarker

2651 - A Systems Cross omics Framework Uncovers Microbiota Driven Molecular Targets for MAFLD Therapy

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Background / Hypothesis: Metabolic dysfunction-associated fatty liver disease (MAFLD) is strongly influenced by the gut microbiota, yet microbiota-regulated host therapeutic targets remain incompletely defined. We aimed to develop a genetics-informed framework to identify and prioritize actionable targets for microbiota-driven MAFLD intervention.

Materials and Methods: Using GWAS data, we assessed the causal effects of gut microbial traits on MAFLD, mapped causal variants to host genes, and integrated cross-omics evidence across transcriptomic, proteomic, metabolomic, and splicing layers. Liver transcriptomic datasets were used for in silico validation. Druggability annotation, phenome-wide MR across 679 traits for safety profiling, composite evidence scoring for prioritization, and molecular docking for binding affinity estimation were subsequently performed to rank actionable targets.

Results: We identified 15 gut microbial traits causally associated with MAFLD, mapping 215 instrumental

SNPs to 191 candidate host genes. Cross-omics integration prioritized 55 high-confidence targets. A composite evidence-scoring system incorporating multi-omics support, liver expression, pathway enrichment, druggability, and phenome-wide safety stratified candidates into four tiers, highlighting nine Tier-1 genes with CMKLR1 ranked highest. Overall, 21 targets showed favorable safety profiles, and molecular docking supported strong binding between prioritized targets and metabolic agents, with CMKLR1 exhibiting the highest affinity for canagliflozin.

Conclusions: This integrative framework prioritizes host targets linking gut microbiota to MAFLD and identifies CMKLR1 as the leading therapeutic candidate supported by multi-omics and pharmacological evidence, providing a roadmap for microbiota-targeted drug discovery.

Keyword(s) :

2652 - A retrospective study on nirmatrelvir ritonavir and azvudine in treating COVID 19 in patients with cirrhosis

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Background / Hypothesis: While numerous pivotal clinical studies have compared anti-COVID-19 therapies, no study has yet evaluated the efficacy of nirmatrelvir/ritonavir versus azvudine specifically in the cirrhotic population.

Materials and Methods: Data from 156 hospitalized cirrhosis patients with COVID-19 at The Second Affiliated Hospital of Nanchang University between November 1, 2022, and November 30, 2023, were collected. The analysis included 97 patients in the no-treatment group, 29 in the nirmatrelvir/ritonavir group, and 22 in the azvudine group. Clinical outcomes were compared among the three groups, including 30-day all-cause mortality, mechanical ventilation rate, and ICU admission rate.

Results: Among the included patients, regarding mortality, 13 deaths occurred in the no-treatment group (22.68%), 7 in the Paxlovid group (24.14%), and 7 in the azvudine group (31.82%) (P=0.666). From the perspective of mechanical ventilation, 23 cases required ventilation in the no-treatment group

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(23.71%), 4 in the Paxlovid group (13.79%), and 4 in the azvudine group (18.18%) ($P=0.485$). In terms of ICU admission, 22 cases were admitted in the no-treatment group (22.68%), 4 in the Paxlovid group (13.79%), and 5 in the azvudine group (22.73%) ($P=0.573$).

Conclusions: This study indicates that among cirrhosis patients with COVID-19, no significant differences were observed in 30-day mortality, mechanical ventilation, or ICU admission among the no-treatment, Paxlovid, and azvudine groups. These findings may be related to the reduced incidence of severe COVID-19 outcomes following widespread vaccination.

Keyword(s) :

2653 - From Prediction to Decision A Dynamic Multi Modal Causal Machine Learning System for Optimized Clinical Management of Chronic Hepatitis B in a 150 000 Patient Longitudinal Cohort

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Background / Hypothesis:

Achieving functional cure, determining safe cessation of Nucleos(t)ide Analogues (NAs), and precise surveillance of Hepatocellular Carcinoma (HCC) remain the three major challenges in Chronic Hepatitis B (CHB) management. Traditional AI models focus on correlation-based prediction but fail to answer "what-if" interventional questions. We aimed to develop a dynamic artificial intelligence approach utilizing a massive multi-modal longitudinal cohort to estimate Individual Treatment Effects (ITE) and provide actionable clinical decision support.

Materials and Methods:

We constructed a longitudinal cohort of 150,000 CHB patients from a single tertiary center, reconstructing their complete clinical history. The dataset integrated multi-modal real-world data (RWD), including longitudinal laboratory tests, electronic health records, imaging (CT/MRI/US), and liver pathology. We employed deep representation learning combined with counterfactual inference algorithms to construct a holistic decision-support engine. This system quantifies

Individual Treatment Effects (ITE) across the full spectrum of disease progression, enabling precision strategies for the critical trilemma of functional cure attainment, safe nucleos(t)ide analogue cessation, and dynamic hepatocellular carcinoma surveillance.

Results:

We successfully established a massive, high-fidelity longitudinal cohort comprising 158,214 unique CHB patients. The time span ranges from December 2006 to December 2025, the cohort is predominantly male (60.20%), the average age is 48 years old (IQR: 36-58). This dataset is highly multidimensional, containing over 130,000 (83%) abdominal ultrasound records and 41,000 (26.5%) multimodal imaging data of abdominal CT and abdominal MRI. Through rigorous data cleaning, we achieved a high reconstruction rate; All patients possessed complete, granular longitudinal data specifically quality-controlled for causal inference algorithms. The AI engine has been successfully integrated into the hospital's clinical workflow. Preliminary deployment validates the system's computational feasibility to process multi-modal inputs in real-time and generate individual counterfactual treatment trajectories.

Conclusions: Our study showcases a pioneering dynamic Causal AI framework built on a massive multi-modal cohort. By moving beyond conventional prediction to dynamic counterfactual analysis, the system facilitates personalized clinical decision-making for functional cure, safe NA cessation and hepatocellular carcinoma surveillance. This initiative underscores the power of causal inference in transforming large-scale RWD into actionable, dynamic insights for chronic hepatitis B management.

Keyword(s) : Chronic Hepatitis B; Causal Machine Learning; Individual Treatment Effects; Clinical Decision Support System

2655 - Single Cell Trajectory and Communication Profiling Reveals a Metabolic Death Switch Driven by Macrophage Polarization in Hepatocellular Carcinoma Radioresistance

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Background: Radioresistance remains a formidable challenge in the treatment of hepatocellular carcinoma

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(HCC). While intra-tumor heterogeneity is a known driver of treatment failure, the dynamic evolutionary process and the extrinsic microenvironmental triggers that govern the transition from a radiosensitive to a resistant state remain poorly understood.

Methods: We performed single-cell RNA sequencing (scRNA-seq) on HCC clinical samples, categorized into complete response (CR) and non-complete response (non-CR) groups. We utilized Monocle3 for pseudotime trajectory inference and CellChat to characterize the cell-cell communication landscape. A functional scoring system was developed to quantify "Mitoxyperiosis"—a specific mode of oxidative-stress-induced cell death.

Results: Trajectory analysis revealed a clear evolutionary continuum from sensitive to resistant aneuploid epithelial cells. We identified a critical "Metabolic-Death Switch" at the late stage of evolution: as cells progressed toward the resistant pole (LowMitoxy), there was a dramatic down-regulation of the Mitoxyperiosis score, coupled with a sharp induction of FABP1 expression. Notably, FABP1 acted as a metabolic shield, neutralizing oxidative stress and preventing cell death.

Integrated microenvironmental analysis demonstrated that this switch was tightly regulated by macrophage polarization. M1 macrophages were significantly enriched in CR samples and exhibited intense pro-apoptotic signaling toward the HighMitoxy (sensitive) subpopulation. Conversely, M2 macrophages were predominantly clustered in non-CR samples and established a robust communication axis with the LowMitoxy (resistant) subpopulation. Specifically, M2-derived ligands (e.g., TGF- β 1 and SPP1) were identified as key extrinsic inducers that triggered FABP1-mediated metabolic reprogramming in tumor cells.

Conclusion: Our study delineates a novel mechanism of radioresistance where the M2 macrophage-dominant niche drives HCC cells to undergo a metabolic transition, characterized by FABP1 elevation and Mitoxyperiosis evasion. These findings identify the M2-TGF β 1-FABP1 axis as a potential therapeutic target to sensitize HCC to radiotherapy.

Keyword(s) : Mitoxyperiosis, Hepatocellular carcinoma, Single-cell RNA sequencing, Macrophage polarization

2656 - Relationship of liver fibrosis risk score (FIB 4) with echocardiographic findings in myocardial infarction with non obstructive coronary artery disease (MINOCA)

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is associated with cardiac disease including myocardial infarction. Myocardial infarction with non-obstructive coronary artery disease (MINOCA) accounts for 6–8% of myocardial infarction presentations. The FIB-4 index is a non-invasive risk tool for assessing hepatic fibrosis and may predict major adverse cardiovascular events. We hypothesized that MINOCA patients with higher FIB-4 scores would demonstrate left ventricular (LV) systolic and diastolic dysfunction compared with those with lower FIB-4 scores.

Materials and Methods:

This retrospective study identified patients with MINOCA from October 2022 to October 2023 at Westmead Hospital, New South Wales, Australia. Exclusion criteria included obstructive coronary artery disease ($\geq 50\%$ stenosis), MINOCA mimicking disease processes, prosthetic or severe valvular disease, pre-existing liver disease, or unavailable liver function pathology within 3 months of presentation. Contemporaneous transthoracic echocardiograms were analysed offline for LV, right ventricular (RV) and left atrial (LA) structural and functional parameters, diastolic function indices, and speckle tracking derived global longitudinal strain (GLS). FIB-4 scores were calculated using age, liver tests and platelet count.



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Results:

Seventy patients comprised the study cohort, categorized into low, indeterminate and high-risk groups using the FIB-4 score. There were 34 patients in the low-risk FIB-4 group (<1.30), 31 in the indeterminate group ($1.30\text{--}2.67$) and 5 in the high-risk group (>2.67); the indeterminate and high-risk groups were combined for analysis. Overall, 34 patients were female (48.6%) and mean age was 57 ± 18 years. The low vs indeterminate and high FIB-4 groups differed in age (48 vs 66 years, $p<0.001$), with no other significant demographic differences. Echocardiographic parameters significantly different between groups were peak A wave velocity (64 vs 79 cm/s, $p=0.028$), lateral e' (9 vs 8, $p=0.028$), E/e' ratio (9.09 vs 11.41, $p=0.021$), LVEF (55.7% vs 53.7%, $p=0.008$) and LV GLS (-17.1% vs -14.7% , $p<0.001$). On age-adjusted univariate general linear modelling, peak E wave velocity and LV GLS were associated with FIB-4 score ($p<0.2$). When included in multivariate age-adjusted analysis model, including LV GLS and peak E wave velocity, only LV GLS was significantly associated with FIB-4 score ($p=0.010$).

Conclusions:

Among echocardiographic parameters, LV GLS was the only independent marker associated with FIB-4 group after adjusting for age. LV GLS is a sensitive marker of subclinical LV function and is associated with worse cardiovascular outcomes. Higher FIB-4 scores were associated with reduced GLS, indicating impaired myocardial strain could be associated with higher liver fibrosis stage.

Keyword(s) : FIB-4, Echocardiography, MINOCA

2657 - Clinical characteristics and influencing factors for recompensation in patients with first time decompensated hepatitis B cirrhosis with esophageal and gastric variceal rupture and bleeding in the Western Region of

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Background / Hypothesis:

To explore the clinical characteristics and influencing factors of patients with first-time decompensated hepatitis B cirrhosis accompanied by esophageal and gastric fundus variceal rupture and bleeding who have achieved recompensation in the western region of Inner Mongolia

Materials and Methods:

A retrospective analysis was conducted on inpatients with hepatitis B cirrhosis in the western region of Inner Mongolia from January 2015 to January 2024. All patients were followed up with the occurrence of the first recompensation or ≥ 2 decompensations (continuous decompensation) as the end point. The outcomes of the patients were divided into the recompensation group and the continuous decompensation group. Analyze the independent influencing factors of re-compensation. Compare the differences in the cumulative occurrence of liver cancer among patients with different compensatory states. The comparison of measurement data between the two groups was conducted using the Mann-Whitney U test; The comparison of count data between the two groups was conducted using the χ^2 test. The influencing factors of recompensation were analyzed using the multivariate Cox proportional hazards regression model. The survival curve was plotted using the Kaplan-Meier method, and the survival curve was tested by log-rank.

Results:

among 69 patients with decompensated hepatitis B cirrhosis, ultimately, 32 cases achieved re-decompensation and 37 cases were persistent decompensation. Compared with patients with persistent decompensation, Age {compensated group 53.22 (9.42) vs decompensated group 60.49 (10.42)} (HR=0.94, 95%CI 0.90-0.99, $p=0.02$), CTP scores {compensated group 6(5,6.75) vs decompensated group 9 (8,11.5)} (HR=0.63, 95%CI 0.46-0.86, $p<0.05$), Meld scores {compensated group 6.1 (3.29,8.28) vs decompensated group 12.74 (7.16,17.45)} (HR=0.9, 95%CI 0.85-0.96, $P<0.05$) are independent influencing factors of re-compensation; The comparison of the recompensation group and its first decompensation clinical data: there was a significant statistical difference in CTP scores {compensated group 6.12 (1.07) vs first decompensated group 7.91 (1.59)} (OR= 1.79, 95%CI 1.01-3.16, $P=0.05$), ALB {compensated group 38.80 (4.51) vs first decompensated group 30.98 (4.98)} (OR=0.82, 95%CI 0.68-0.97, $P=0.02$), HGB {compensated group 123.28 (28.96) vs first decompensated group 89.69 (23.49)} (OR= 0.96, 95%CI 0.93-0.99) ($P=0.01$); the incidence of tumors in patients with recompensation was significantly lower than that in patients with continuous decompensation



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(HR=0.23, 95%CI 0.37-0.57, P=0.02)

Conclusions:

Compared with the baseline data of patients with first decompensation, there were significant statistical differences in CTP score, ALB and HGB in the re-decompensation group. Compared with the data of the continuous decompensation group, patients with younger age, lower CTP scores and Meld scores were more likely to achieve recompensation, and the cumulative incidence of tumors in recompensated patients was significantly lower than that in continuous

Keyword(s) :

2658 - Real world impact of 48 week tenofovir amibufenamide therapy on lipid profiles in chronic hepatitis B

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Background / Hypothesis:As a first-line treatment for chronic hepatitis B (CHB), the long-term impact of tenofovir amibufenamide (TMF) on lipid metabolism remains to be further clarified in real-world settings. This study aimed to evaluate the effect of 48-week TMF treatment on the lipid profile of CHB patients.

Materials and Methods:This was a single-center retrospective study conducted at Beijing Ditan Hospital, Capital Medical University. A total of 302 CHB patients who received TMF monotherapy for at least 48 weeks were consecutively included. Lipid parameters, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were collected and compared at baseline and after 48 weeks of treatment.

Results:Compared with the baseline level, no significant changes were observed in any lipid parameters after 48 weeks of treatment: TC was 4.52 (IQR: 4.07, 5.09) vs 4.37 (IQR: 3.91, 5.26) mmol/L, P = 0.310; TG was 1.06 (IQR:0.68, 1.48) vs 1.02 (IQR:0.72, 1.57) mmol/L, P = 0.120; HDL-C was 1.23 (IQR:1.04, 1.40) vs 1.18 (IQR:1.01, 1.44) mmol/L, P = 0.469; LDL-C was 2.74 (IQR: 2.32, 3.24) vs 2.69 (IQR:2.16, 3.30) mmol/L, P = 0.150.

Conclusions:This real-world study demonstrates that 48-week TMF treatment has no significant adverse effect on lipid metabolism in patients with CHB,

confirming its favorable lipid safety profile.

Keyword(s) : Tenofovir amibufenamide;Chronic hepatitis B;Lipid profile

2659 - Agenesis of the Dorsal Pancreas Caused by HNF 1 β Gene Mutation Presenting with Abnormal Liver Function as the Initial Manifestation A Case Report

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Background / Hypothesis:

Agenesis of the dorsal pancreas (ADP) is a rare congenital anomaly in which the dorsal pancreatic anlage fails to develop, resulting in partial or complete absence of the pancreatic body and tail. While most cases are sporadic, mutations in developmental genes such as hepatocyte nuclear factor 1 β (HNF-1 β) and GATA binding protein 4 (GATA4) have been implicated. Patients may be asymptomatic or present with diabetes, recurrent pancreatitis, steatorrhea, or non-specific abdominal complaints; hepatic dysfunction is rarely the entry sign. We describe an adolescent boy who initially presented with persistently elevated transaminases and was ultimately diagnosed with HNF-1 β -related ADP.

Materials and Methods:

We describe a 17-year-old male who presented with elevated transaminases and was found to have hyperglycemia. Imaging revealed absence of the pancreatic body and tail and multiple bilateral renal cysts. Liver biopsy suggested non-alcoholic fatty liver disease. Genetic testing identified a heterozygous pathogenic deletion of HNF-1 β exons 1–9, confirming HNF-1 β -related ADP.

Results:

After initiation of insulin and hepatoprotective therapy, liver enzymes and glycemic control improved.

Conclusions:

This case broadens the phenotypic spectrum of ADP and highlights the importance of considering this diagnosis in adolescents with unexplained abnormal liver function and diabetes, prompting timely imaging and genetic evaluation.

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Keyword(s) : Agenesis of the dorsal pancreas; hepatocyte nuclear factor-1 β ; abnormal liver function; diabetes; gene deletion

2661 - Macrophage metabolic reprogramming mediated by PKM2 deacetylation in liver fibrosis

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Liver fibrosis is a common pathological process of a variety of chronic liver diseases, which is characterized by the activation of hepatic astrocytes (HSCs) and excessive deposition of extracellular matrix. In recent years, immunometabolism research has revealed that macrophages play a key role between inflammation and fibrosis, among which metabolic reprogramming is the core mechanism that determines their function. Pyruvate kinase M2 (PKM2) drives the anti-inflammatory effect of M1 macrophages by regulating glycolysis and inflammatory signals; while silent information regulatory factor 3 (SIRT3) regulates the configuration of PKM2 through deacetylation modification, reduces lactic acid production, and promotes the transformation of macrophages to anti-inflammatory phenotypes, thus reducing Fibrosis. Existing research confirms that the SIRT3-PKM2 axis not only affects macrophage metabolism, but also forms crosstalk with HSC through lactic acid and epigenetic modification, forming the core loop of inflammation and fibrosis. This paper reviews the mechanism of PKM2 and its deacetylation in liver fibrosis, emphasizes its value as a potential treatment target, and puts forward a multi-dimensional strategy that should be combined with metabolic regulation, immune intervention and epigenetic regulation in the future to achieve accurate treatment of liver fibrosis.

Keyword(s) : liver fibrosis; macrophage polarization; PKM2; SIRT3; metabolic reprogramming; glycolysis; treatment strategy

2662 - Exploring the Dose Response of Shugan Xiaozhi Formula Granules and MRI Radiomics Based MAFLD Prediction

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Background / Hypothesis:

Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) is the most common chronic liver disease worldwide and is closely related to metabolic syndrome. There is a lack of specific therapeutic drugs. Traditional Chinese medicine (TCM) has shown multi-target advantages in the treatment of MAFLD, but traditional decoctions have limitations. As a modern dosage form, the equivalence and dose-effect relationship of TCM formula granules need to be verified. At the same time, the diagnosis of MAFLD relies on invasive examinations, and radiomics can provide non-invasive predictive tools.

This study aims to evaluate the clinical equivalence, safety, and dose-effect relationship of the Shugan Xiaozhi Decoction and different doses of formula granules in patients with liver depression and spleen deficiency type MAFLD, and to construct a MAFLD prediction model based on MRI radiomics.

Materials and Methods:

The first part is a literature review and a clinical randomized controlled trial, including 120 patients with liver depression and spleen deficiency type MAFLD, randomly divided into a decoction group and full-dose, 2/3-dose, and 1/2-dose granule groups (30 cases each). The intervention lasted for 12 weeks, and changes in MRI-PDFF, CAP values, BMI, blood lipids, liver function, and TCM syndromes were observed. The second part is a retrospective study, including 172 patients (112 with MAFLD and 60 healthy controls). MRI radiomics features were extracted, and the Rad-score was constructed. Serum markers (ALT, AST, TC) were integrated to establish a combined prediction model.

Results:

In the clinical trial, the Shugan Xiaozhi Decoction significantly improved liver steatosis, metabolic indicators, and TCM syndromes ($P < 0.05$). The decoction group was equivalent to the full-dose group ($P > 0.05$), and a dose-dependent relationship was observed (higher dose was better than lower dose). The safety was good, with no serious adverse events. In the radiomics model, the Rad-score was a strong independent predictor ($OR = 1.98$, $P < 0.001$), and the combined model had an AUC of 0.918, an internal validation C-index of 0.896, and excellent calibration and clinical utility.

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Conclusions:

The full-dose Shugan Xiaozhi Decoction granules are equivalent to the decoction and have a dose-dependent relationship. The radiomics combined model provides an efficient tool for non-invasive diagnosis of MAFLD. Future multicenter validation is needed to optimize its application.

Keyword(s) : Metabolic Dysfunction-Associated Fatty Liver Disease; Shugan Xiaozhi Decoction; TCM formula granules; Dose-effect relationship; Radiomics; Prediction model Abstract

2665 - Evaluation of the Egyptian Club of Ascites Diagnostic Algorithm for Etiological Diagnosis of Ascites A Single Center Study"

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Background / Hypothesis:

Ascites is a common clinical problem with diverse etiologies. Accurate differentiation between portal hypertension-related and non-portal hypertension causes is essential for appropriate management and prognosis. The Egyptian Club of Ascites (ECA) proposed a diagnostic algorithm based primarily on serum-ascites albumin gradient (SAAG) and ascitic fluid total protein (AFTP). The study aimed to evaluate the performance of the Egyptian Club of Ascites (ECA) diagnostic algorithm in determining the etiology of ascites.

Materials and Methods:

A cross-sectional study included 100 patients with previously uninvestigated ascites at a single Egyptian tertiary center (April–October 2025). All patients underwent detailed clinical evaluation and diagnostic paracentesis. Ascitic fluid analysis included SAAG and AFTP measurements. Additional laboratory, radiological, and diagnostic procedures were performed as clinically indicated to establish the final etiology of ascites.

Results:

Patients had a mean age of 57.4 ± 10.5 years; 57% were males, and cirrhotic ascites predominated (87%). Ascitic fluid appearance was significantly associated with etiology ($p < 0.001$); transparent yellow ascites

predominated in cirrhotic cases, whereas cloudy, bloody, or milky ascites were more frequent in malignant, tuberculous, inflammatory, and traumatic causes.

High SAAG (≥ 1.1 g/dL) was observed in 87% of patients and identified portal hypertension-related ascites with a sensitivity of 96.7%, specificity of 100%, and AUC of 98.4% ($p < 0.001$). Low SAAG (< 1.1 g/dL) was detected in 13% of patients; four cases (30.7%) were related to portal hypertension, while all malignant ascites, tuberculous peritonitis, SLE serositis, and hemoperitoneum cases had low SAAG values ($p = 0.001$).

Low AFTP (< 2.5 g/dL) predominated in cirrhotic ascites, whereas high AFTP (≥ 2.5 g/dL) was mainly associated with cardiac, malignant, tuberculous, and inflammatory ascites. AFTP demonstrated high diagnostic accuracy for transudative ascites (sensitivity 94.6%, specificity 100%, AUC 97.3%; $p < 0.001$).

The combined ECA algorithm (SAAG + AFTP) demonstrated high diagnostic performance, with sensitivity and specificity of 96.6% and 100% for hepatic ascites, 75% and 100% for cardiac ascites, 100% and 96% for Nephrogenic ascites and protein-losing enteropathy, and 87.5% and 98.9% for tuberculous and malignant ascites (all $p < 0.001$).

Conclusions:

The Egyptian Club of Ascites' diagnostic approach provides a reliable and highly accurate framework for etiological diagnosis of ascites. The combined use of SAAG and AFTP provides excellent diagnostic discrimination between portal hypertension-related and non-portal causes and can be confidently applied in routine clinical practice.

Keyword(s) :

2667 - HBV recurrence in patients with hepatocellular carcinoma and cholangiocarcinoma following liver transplantation

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Background / Hypothesis:

To investigate risk factors for hepatitis B virus (HBV)

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recurrence following liver transplantation in patients with HBV-related hepatocellular carcinoma (HCC) or cholangiocarcinoma.

Materials and Methods:

This study included patients with cirrhosis due to chronic HBV infection who underwent liver transplantation for HCC or cholangiocarcinoma between February 2015 and February 2019 at a tertiary hospital. Participants were stratified based on post-transplant HBV recurrence status. Inter-group comparisons for categorical variables were performed using the chi-square test, while continuous variables were analyzed using Student's t-test or the Mann-Whitney U test, as appropriate. Univariate and multivariate Cox proportional hazards regression analyses were conducted to identify independent risk factors.

Results:

Among 112 enrolled patients, 109 had HCC and 3 had cholangiocarcinoma. Compared to patients without HBV recurrence, those with recurrence had significantly higher rates of detectable HBV DNA pre-transplantation (75.0% vs. 27.0%, $P=0.001$), microvascular invasion (100% vs. 67.8%, $P=0.020$), and more advanced TNM stages (Stage III+IV: 58.3% vs. 16.5%, $P=0.001$). They also exhibited larger maximum tumor diameter (6.3 cm vs. 2.5 cm, $P<0.001$) and were more likely to exceed the "Up-to-seven" criteria (83.3% vs. 22.0%, $P<0.001$). No significant differences were found in age ($P = 0.362$), sex ($P = 0.480$), or MELD score ($P = 0.678$). Post-transplantation, HBV recurrence occurred in 12 patients (10.71%) after a median interval of 15.0 months (IQR: 6.5-21.1). Of these, 11 cases (10.1% of HCC patients) followed HCC recurrence, and 1 case (33.3% of cholangiocarcinoma patients) followed cholangiocarcinoma recurrence. Overall, 33 patients experienced tumor recurrence, and 30 died. Among the 12 with HBV recurrence, 11 died due to tumor progression, while 1 remains alive with HCC under treatment. Multivariate analysis identified exceeding the "Up-to-seven" criteria (OR: 7.99, 95% CI: 1.19-53.49, $P=0.032$) and the presence of cholangiocarcinoma (OR: 116.7, 95% CI: 6.06-2250.5, $P=0.002$) as independent risk factors for HBV recurrence.

Conclusions: HBV recurrence is closely associated with tumor recurrence post-liver transplantation. Patients presenting with larger or multiple tumors or with

cholangiocarcinoma before transplantation, are at significantly higher risk for HBV recurrence. Mortality in this cohort was primarily driven by tumor progression rather than HBV recurrence itself.

Keyword(s) : HBV recurrence; HCC recurrence; liver transplantation; risk factors.

2668 - Efficacy and Safety of Elobixibat in Patients with Chronic Constipation A Meta Analysis of Randomized Controlled Trials

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Background / Hypothesis:

Elobixibat is a first-in-class, minimally absorbed, locally acting inhibitor of the ileal bile acid transporter (IBAT), approved for the treatment of chronic constipation. By interrupting enterohepatic bile acid circulation in the terminal ileum, elobixibat increases colonic bile acid delivery, stimulating fluid secretion and high-amplitude propagated contractions to enhance colonic motility. It's overall clinical efficacy and safety profile remain inconsistent across individual trials. This meta-analysis aimed to synthesize available evidence on the therapeutic efficacy and safety of elobixibat in chronic constipation.

Materials and Methods:

A systematic search of PubMed, EMBASE, and Cochrane was conducted for randomized controlled trials (RCTs) published through January 2026. Adult patients diagnosed with functional constipation according to Rome IV criteria were included. Study quality and risk of bias were assessed in accordance with PRISMA guidelines. Data were pooled using RevMan version 5.4. Efficacy outcomes were expressed as risk ratios (RRs) for categorical variables and least-square mean differences for continuous variables, with statistical significance set at $p < 0.05$.

Results:

Three RCTs comprising 507 patients met inclusion criteria. Elobixibat significantly improved weekly spontaneous bowel movement frequency compared with placebo (RR = 1.45; 95% CI, 1.20-1.75; $p < 0.001$). Abdominal pain, the most commonly reported adverse effect, occurred less frequently in the elobixibat group than in the placebo group [RR = 0.26 (0.08-0.82), $P = .02$]. Most reported adverse events were mild in severity.

Conclusions: Elobixibat is effective in improving bowel

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movement frequency in patients with chronic constipation and demonstrates a favorable safety profile. These findings support its role as a therapeutic option for patients requiring prompt and sustained symptom relief. Larger, multicenter randomized trials are warranted to further define its long-term efficacy and adverse event profile.

Keyword(s) : elobixibat, ileal bile acid transporter inhibitor, IBAT, constipation

2669 - Multicohort metagenomic analysis of cholangiocarcinoma non invasive diagnosis and functional validation of key microbes

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Background / Hypothesis:

Cholangiocarcinoma (CCA) is a highly lethal malignancy with limited early diagnostic options. While emerging evidence implicates the gut microbiome in CCA tumorigenesis, its potential as a non-invasive diagnostic biomarker remains unexplored. This study aimed to characterize CCA-associated gut microbial alterations and evaluate their diagnostic utility across diverse Chinese populations.

Materials and Methods:

Metagenomic sequencing was performed on fecal samples collected from 643 individuals across three geographically distinct regions in China (East, Central, and Northwest). We analyzed microbial community composition and functional pathways to identify CCA-specific dysbiosis patterns. Diagnostic models were constructed using both species-level taxonomic markers and functional pathway-based features. Model performance was evaluated in the discovery cohort and validated in two independent cross-regional cohorts. Additionally, we assessed the model's ability to differentiate CCA from hepatocellular carcinoma (HCC) and examined disease specificity using 6,847 samples from 31 published studies spanning 11 disease conditions.

Results:

CCA patients exhibited distinct gut microbial dysbiosis characterized by significant enrichment of lipopolysaccharide-producing bacteria (*Klebsiella aerogenes*, *Citrobacter koseri*) and depletion of butyrate-synthesizing organisms (*Faecalibacterium*

prausnitzii, *Roseburia intestinalis*). Species-level diagnostic models demonstrated superior discriminatory capacity compared to pathway-based approaches, achieving area under the curve (AUC) values of 98.63% and 99.42% in the East China discovery cohort. Importantly, robust cross-regional validation yielded AUCs of 80.89% in Central China and 80.43% in Northwest China, confirming model generalizability. The model effectively distinguished CCA from HCC (AUC=95.58%), addressing a critical clinical differential diagnosis challenge. Meta-analysis of public datasets revealed moderate disease specificity for the CCA microbiome signature, with performance influenced by biomarker overlap across different pathological conditions. Mechanistically, depleted *Bifidobacterium pseudocatenulatum* suppressed CCA progression through inhibition of the PI3K-AKT-mTOR pathway.

Conclusions:

This study establishes gut microbiome metagenomics as a promising non-invasive diagnostic tool and identifies potential therapeutic targets for cholangiocarcinoma.

Keyword(s) : cholangiocarcinoma, gut microbiome, metagenome, diagnostic model, liver cancer

2674 - Model combining MELD ICG R15 and systemic immune inflammation index predicts 28 day clinical progression in hepatitis B virus related acute on chronic liver failure patients

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Background / Hypothesis:

Hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF) carries high short-term mortality. Existing prognostic scores are often static and may not fully capture dynamic liver reserve and systemic inflammation.

Materials and Methods:

A retrospective study included 66 HBV-ACLF patients admitted between June 2023 and December 2024, categorized by 28-day outcome into improvement (n=49) and progression (n=17) groups. Clinical data, indocyanine green clearance test parameters, and systemic immune-inflammation index (SII) were collected. Logistic regression identified predictors for model construction. Model discrimination was

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evaluated using the receiver operating characteristic curve, with internal validation via bootstrap and assessment of calibration and clinical net benefit.

Results:

Multivariate analysis identified SII/1000 as an independent risk factor (OR=5.77, 95% CI: 1.64-20.37, P=0.006), with MELD-ICG R15 score showing marginal significance (P=0.082). The final model was: $\text{logit}(P) = -6.658 + 0.060 \times (\text{MELD-ICG R15}) + 1.753 \times (\text{SII}/1000)$. The area under the curve was 0.761 (95% CI: 0.627-0.873), with bootstrap-corrected area under the curve of 0.732. At the optimal cutoff of 0.209, sensitivity was 82.4% and specificity 65.3%. The calibration curve showed good agreement, and decision curve analysis indicated positive clinical net benefit.

Conclusions:

The combination of MELD-ICG R15 and SII effectively predicts 28-day clinical progression risk in HBV-ACLF patients, offering a potential tool for early risk stratification and personalized intervention.

Keyword(s) : hepatitis B virus, acute-on-chronic liver failure, indocyanine green clearance test, systemic immune-inflammation index, prognosis, prediction model

2675 - The Mechanism of Necroptosis in Metabolic dysfunction associated Fatty Liver Disease and related targeted therapy

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Metabolic dysfunction-associated fatty liver disease is a common chronic liver disease worldwide, with a continuously rising incidence rate. The traditional "two-hit" theory has gradually been replaced by the "multiple hit" hypothesis, which emphasizes the synergistic effect of multiple pathogenic factors. Necroptosis, as an inflammatory form of death that combines the morphological characteristics of necrosis and the signaling mechanism of apoptosis, its core lies in the RIPK1-RIPK3-MLKL signaling axis. It not only directly leads to the death of hepatocytes, releasing damage-related molecular patterns to trigger inflammatory responses and promote the fibrosis process, but is also closely related to the metabolic reprogramming of the liver. This review aims to

summarize the activation mechanism of necroptosis in metabolic dysfunction-associated fatty liver disease, its effects on liver inflammation, fibrosis and metabolism, and explore its therapeutic strategies and application prospects.

Keyword(s) : Necroptosis, MAFLD, mechanism, treatment

2676 - the Mechanism of Ruangan Formula in Regulating Macrophage Glycolysis for the Prevention and Treatment of Liver Fibrosis via SIRT1 Mediated PKM2 Deacetylation

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Background / Hypothesis:

Liver fibrosis is a critical stage in the progression of chronic liver diseases, and effective drugs for its reversal are currently lacking. Inflammatory polarization of macrophages (M1 phenotype) is a key driver of fibrosis, closely associated with cellular metabolic reprogramming, particularly the activation of glycolysis. Ruangan Formula is an effective traditional Chinese medicinal compound for clinically preventing and treating liver fibrosis, but its mechanism of action remains incompletely understood. This study aims to investigate whether Ruangan Formula exerts its anti-fibrotic effects by regulating macrophage glycolysis and the SIRT1/PKM2 signaling axis.

Materials and Methods:

A CCl₄-induced mouse liver fibrosis model and an LPS-stimulated primary bone marrow-derived macrophage (BMDM) polarization model were established. The effects of Ruangan Formula on liver histopathology, collagen deposition, inflammatory cytokine expression, and glycolytic indicators were evaluated. Network pharmacology was employed to predict core targets, and molecular docking was used to verify the binding affinity of Ruangan Formula's active components with SIRT1. Myeloid-specific PKM2 knockout mice were generated using the Cre-loxP system. Combined with the SIRT1 inhibitor EX527, the necessity of the SIRT1/PKM2 pathway in the action of Ruangan Formula was validated both in vivo and in vitro. Immunoprecipitation, mass spectrometry, and site-directed mutagenesis were used to identify PKM2 acetylation sites.

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Results:

Clinical and animal experiments confirmed that Ruangan Formula significantly improved pathological indicators of liver fibrosis, inhibited M1 macrophage polarization, and reduced the production of lactate, a key glycolytic product. Network pharmacology screening suggested SIRT1 as a core target of Ruangan Formula, and molecular docking showed good binding potential between its active components and SIRT1. Experiments demonstrated that Ruangan Formula upregulated SIRT1 expression, decreased PKM2 acetylation levels, inhibited its nuclear translocation, and suppressed the transcription of downstream glycolytic genes. The anti-fibrotic and metabolic regulatory effects of Ruangan Formula were significantly attenuated following PKM2 knockout or SIRT1 inhibition.

Conclusions:

Ruangan Formula likely upregulates SIRT1, promotes PKM2 deacetylation, inhibits its nuclear translocation and glycolysis reprogramming, thereby modulating macrophage polarization towards the M1 phenotype and ultimately exerting anti-liver fibrosis effects. This research provides a modern molecular mechanism for the traditional Chinese medicine principle of "Strengthening Healthy Qi and Removing Blood Stasis" and offers new scientific evidence for the clinical application of Ruangan Formula.

Keyword(s) : Liver fibrosis; Ruangan Formula; Macrophage polarization; Glycolysis; SIRT1; PKM2; Deacetylation

2677 - Barriers and Facilitators to Chronic Hepatitis B Management in Primary Care A Systematic Review from Patient and Physician Perspectives

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Background / Hypothesis:

Decentralisation of chronic hepatitis B (CHB) management to primary care is increasingly promoted to improve diagnosis, linkage to care, and long-term follow-up. However, successful implementation depends on the preferences, experiences, and perceived barriers of both patients and physicians. This systematic review synthesised patient- and physician-level perspectives on CHB management in primary care settings.

Materials and Methods:

A systematic literature search using subject headings and keywords was conducted in PubMed, Embase, Web of Science, and PsycINFO, yielding 7,087 records. After duplicate removal (n = 6,555), 532 records were screened by title and abstract, and 437 were excluded. Ninety-five articles underwent full-text review, of which 50 met inclusion criteria (22 patient-focused and 28 physician-focused). Data were analysed using thematic synthesis to identify patient- and provider-level barriers and proposed solutions.

Results:

At the patient level, five major barriers to CHB management in primary care were identified: (1) healthcare system barriers (e.g. fragmented care, poor continuity, long waiting times, and perceived low quality of primary care); (2) cultural and social barriers (e.g. language discordance, conflicting health beliefs, family dynamics); (3) stigma and psychosocial barriers (e.g. fear of discrimination and shame); (4) knowledge barriers (e.g. low awareness and misconceptions about CHB); and (5) economic barriers (transportation, time constraints). Proposed solutions included decentralization of care to community settings, culturally and linguistically tailored health education, strengthened reminder systems, and financial support mechanisms.

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At the physician level, barriers included systemic constraints (limited diagnostics, funding, and unclear guidelines), limited knowledge and confidence in CHB management, communication challenges with migrant and high-risk groups and stigma towards affected populations. Proposed solutions included targeted continuing medical education, simplified and standardized guidelines, implementation of clinical tools and recall systems, culturally adapted communication strategies, and shared-care models between primary and specialist services.

Conclusions:

This systematic review demonstrated that multilevel barriers hinder effective CHB management in primary care. Addressing system, cultural, educational, economic-level challenges through integrated, culturally sensitive, and shared-care models is critical to scaling up primary care-led CHB management and achieving goals towards viral hepatitis elimination.

Keyword(s) : Chronic Hepatitis B; Primary health care; Decentralization; Shared-care model

2680 - Evaluation of NLR as a novel Predicting biomarker for Disease Severity and Clinical Outcomes in Acute Hepatitis A and E

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Background / Hypothesis:

Acute hepatitis A (HAV) and E (HEV) infections can progress to severe liver injury or acute liver failure (ALF). Early biomarkers for risk stratification are essential. This study was aimed to evaluate the utility of the neutrophil-to-lymphocyte ratio (NLR) in predicting disease severity and outcomes in acute HAV and HEV.

Materials and Methods:

A cross-sectional study of 200 acute hepatitis patients (100 HAV, 100 HEV) was conducted over 12 months from February 2024 to February 2025 at Department of gastroenterology, Hepatology, Medicine of Rangpur medical college Hospital and five private chamber in Rangpur city. NLR, clinical parameters (bilirubin, ALT, INR, albumin), and outcomes (liver failure, hospitalization duration, mortality) were analyzed.

Severity was classified as mild (no complications), moderate (coagulopathy/or prolonged symptoms), or severe (ALF/death). Statistical analyses included ROC curves, multivariate regression, and correlation testing.

Results:

Elevated NLR (>5.0) strongly correlated with disease severity ($p < 0.001$) in acute hepatitis. HEV patients had higher median NLR (6.2 vs. 4.8; $p = 0.003$) and ALF rates (18% vs. 8%). NLR >5.0 predicted ALF with 84% sensitivity and 76% specificity (AUC: 0.87). Patients with NLR >5.0 had longer hospital stays (14 vs. 7 days; $p < 0.001$) and higher mortality (10% vs. 1.5%; $p = 0.002$). NLR positively correlated with bilirubin ($r = 0.72$) and INR ($r = 0.65$), and negatively with albumin ($r = -0.58$).

Conclusions: NLR is a cost-effective biomarker for predicting severity and poor outcomes in acute HAV and HEV. An NLR >5.0 signals high risk for ALF, prolonged hospitalization, or death, enabling early intervention.

Keyword(s) : Neutrophil-to-lymphocyte ratio (NLR), Acute hepatitis A (HAV), Acute hepatitis E (HEV), Disease severity, Outcomes

2682 - Isolated Pancreatic tuberculosis masquerading as Malignancy in a patient with drug induced liver injury A case report

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A 25-year-old Asian male was admitted to our department with a 3-day yellow urine after took Norfloxacin for diarrhea and vomiting. Laboratory examinations showed elevated liver enzymes and serum bilirubin. Drug-induced liver injury should be considered because the patient had used a liver injury drug. Autoimmune-antibodies showed indicate positive ANA and elevated IgG levels, so Autoimmune Hepatitis should be excluded clinically. However, he was misdiagnosed with pancreatic carcinoma because abdominal enhanced CT, MRI and PET-CT showed a space-occupying lesion in the pancreatic head, but his alpha-fetoprotein, carbohydrate antigen 19-9, and ferroprotein levels were normal. Bilirubin was significantly decreased after anti-infective treatment, so the pancreatic lesion considered may be an infectious lesion. A subsequent T-Spot test result was positive and erythrocyte sedimentation rate increased.

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patient then underwent endoscopic pancreatic fine needle aspiration biopsy and cytology and metagenomic detection of pathogenic microorganism. The histological of the pancreatic mass showed granulomatosis, and mycobacterium tuberculosis was detected by NGS, so the mass diagnosed as a isolated pancreatic tuberculosis. The patient had elevated liver enzymes, so ultrasound-guided liver biopsy was performed. The histologic characteristics include mild inflammation, fibrous and small bile duct proliferation of interlobular portal areas, and normal structure of hepatic lobule, so the pathologic diagnosis might be drug-induced liver injury. The patient's liver function of was improved after liver protection therapy, but active anti-tuberculosis therapy was needed due to the pancreatic tuberculosis. The patient was complicated with severe liver injury, and the anti-tuberculosis treatment started with streptomycin combined with levofloxacin with very low hepatotoxicity. The patient continued to have normal liver function and and the drugs were gradually adjusted to the first-line anti-tuberculosis treatment regimen: Streptomycin, Ethambutol, Isoniazid and Rifampicin. The patient accordingly received anti tuberculosis agents for 12 months, resulting in a significant decrease in the size of the pancreatic mass and recovered well.

Keyword(s) : Drug-Induced Liver Injury; Isolated Pancreatic Tuberculosis; Anti-tuberculosis treatment

2683 - Prior Tyrosine Kinase Inhibitor Therapy Is Associated with an Increased Risk of Hepatitis B Virus Reactivation in HBV Related Hepatocellular Carcinoma Patients with Undetectable HBV DNA Receiving PD 1 Inhibitor-Based Combination Therapy

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Background / Hypothesis:Although prophylactic antiviral therapy is recommended for hepatocellular carcinoma (HCC) patients receiving programmed cell death protein-1 (PD-1) inhibitors combined with tyrosine kinase inhibitors (TKIs), hepatitis B virus reactivation (HBVr) still occurs, even in patients with low baseline viral load. Baseline high HBV DNA levels are a well-established risk factor for HBVr;however, the

incidence and risk factors of HBVr in HCC patients with undetectable HBV DNA prior to treatment remain unclear. This study aimed to evaluate the incidence and identify risk factors for HBVr in this specific population receiving PD-1 inhibitor plus TKI therapy.

Materials and Methods:This retrospective study included 163 patients with HBV-related HCC who received PD-1 inhibitors combined with TKIs at our center between March 2020 and June 2024. Among them, 45 patients with undetectable serum HBV DNA before treatment (defined as <20 IU/mL) were enrolled for analysis. Patients were classified into an HBVr group (n = 14) and a non-HBVr group (n = 31) based on whether HBV DNA became detectable during treatment. Univariate and multivariate Cox proportional hazards models were applied to identify independent predictors of HBV reactivation.

Results:During a median follow-up of 13.8 months (range: 1.2–59.2 months), HBV reactivation occurred in 31.1% (14/45) of patients. In baseline characteristics, larger maximum tumor diameter (HR = 1.21, P = 0.003) and absence of ablation therapy (HR = 0.23, P = 0.024) were significantly associated with an increased risk of HBVr. Multivariate Cox regression analysis identified prior TKI therapy (HR = 5.83, 95% CI: 1.81–18.79, P = 0.003) and higher serum gamma-glutamyl transferase (GGT) levels (HR = 1.01, 95% CI: 1.01–1.01, P = 0.004) as independent risk factors for HBV reactivation.

Conclusions:HBV reactivation can still occur in patients with HBV-related HCC despite prophylactic nucleos(t)ide analogue therapy. HCC patients with undetectable baseline HBV DNA remain at risk of HBVr when treated with PD-1 inhibitor plus TKI combination therapy. Prior exposure to TKI therapy and elevated GGT levels are independent predictors of HBVr. Therefore, close monitoring of HBV DNA during treatment is essential, and treatment sequencing should be carefully considered to minimize the risk of HBV reactivation.

Keyword(s) : Hepatocellular carcinoma, Programmed cell death protein-1 inhibitor, Tyrosine kinase inhibitor, Undetectable hepatitis b virus DNA, Reactivation

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2684 - Hydrogen sulfide donor S Propargyl cysteine remodels the HCC microenvironment and enhances immunotherapy efficacy an integrated metabolomic and molecular analysis

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Background / Hypothesis:

Hepatocellular carcinoma exhibits aberrant angiogenesis and immune suppression that restrict durable benefit from PD 1 blockade. We hypothesized that S propargyl cysteine (SPRC), an endogenous H₂S donor, engages cystathionine gamma lyase (CSE) to suppress NF κB and STAT3 signaling, reduce VEGF activity and tumor cell PD L1 expression, and enhance antitumor T cell function.

Materials and Methods:

SPRC binding to CSE was measured by surface plasmon resonance. In HUVECs, NF κB p65 and STAT3 phosphorylation and VEGFA expression were assessed by immunoblotting, and angiogenic capacity was quantified by Matrigel tube formation with CSE knockdown and rescue. In HepG2 and T cell co culture, tumor cell PD L1 and T cell phenotypes were analyzed by flow cytometry, and cytotoxic activity was evaluated by granzyme B secretion, with STAT3 inhibition as a mechanistic comparator. Promoter associated H3K27ac enrichment at RELA and STAT3 was measured by ChIP qPCR. In an HCC mouse model, anti PD 1 alone or combined with SPRC was evaluated by tumor volume, intratumoral CD8 positive T cells and Tregs by flow cytometry, and vascular density by CD31 immunohistochemistry. Untargeted LC MS metabolomics and oxidative stress indices including MDA and SOD were used to characterize metabolic coordination and redox status.

Results:

SPRC bound recombinant CSE with nanomolar affinity and reduced NF κB p65 and STAT3 phosphorylation in endothelial cells, accompanied by decreased VEGFA and impaired tube formation. These antiangiogenic effects were attenuated by CSE silencing and partially restored by CSE re expression, supporting CSE dependence. In co culture, SPRC reduced PD L1 on HepG2 cells, increased CD8 alpha, decreased FoxP3, and increased granzyme B secretion, consistent with

improved cytotoxic effector function. SPRC increased H3K27ac enrichment at RELA and STAT3 promoters. In vivo, SPRC plus anti PD 1 achieved greater tumor growth inhibition than anti PD 1 alone, increased intratumoral CD8 positive T cells, decreased Tregs, and reduced CD31 indexed vascular density. Metabolomic profiling indicated coordinated pathway level rewiring and was accompanied by reduced MDA and increased SOD activity, consistent with alleviated oxidative stress.

Conclusions:

SPRC engages a CSE linked program that suppresses NF κB and STAT3 to couple antiangiogenic activity with reduced PD L1 mediated immune inhibition and enhanced cytotoxic immunity. SPRC augments PD 1 blockade in HCC with coordinated immune, vascular, metabolic, and redox remodeling, supporting its development as an adjunct strategy to overcome the vascular immune barrier.

Keyword(s) : Hydrogen Sulfide Donor; Tumor Microenvironment; SPRC; LC-MS Metabolomics; HCC

2685 - Early diagnostic and analysis of the high risk factors of covert hepatic encephalopathy

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Background: The present study was designed to analyze the risk factors for Covert Hepatic Encephalopathy (CHE) in cirrhotic patients, to evaluate the diagnostic efficacy of different neuropsychological tests in cirrhotic patients with CHE, and to establish an easy-to-administer screening and diagnostic method with reliable results, so as to provide a new clinical idea for the early diagnosis of CHE.

Methods: Patients who were hospitalized in the Department of Hepatology of Tianjin Second People's Hospital and Tianjin First Central Hospital with clinical and/or pathological diagnosis of cirrhosis from November 2024 to March 2025, as well as subjects who had healthy physical examinations were included as study subjects. The gender, age, and years of education of cirrhotic patients and healthy subjects were recorded, and the laboratory examination data of cirrhotic patients were collected to complete the Psychological Score of HE (PHES), Encephal APP, Animal Naming Test (ANT), and the Chinese version of Montreal Cognitive Assessment-Based (MoCA-BC)

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scale. SPSS 26.0 was applied to process the data, and independent risk factors for the occurrence of CHE in cirrhotic patients were analyzed by binary logistic regression.

Results: Eighty patients with cirrhosis were finally included in this study. The combination of MoCA-BC with other neuropsychological tests yielded that the diagnostic efficacy of the MoCA-BC-NCT-A combination, the MoCA-BC-SDT combination, and the MoCA-BC-LTT combination was superior to the other combinations in the cirrhotic population in the low-years-of-education group for the diagnosis of CHE, with a statistically significant difference ($P < 0.01$), and that the MoCA-BC-Quick Stroop combination was superior to the other combinations in the cirrhotic population in the middle- and high-years-of-education group ($P < 0.01$). MoCA-BC-Quick Stroop combination had better diagnostic efficacy for CHE than other combinations, with statistical differences ($P < 0.01$). Conclusion: The MoCA-BC test has good diagnostic efficacy in diagnosing CHE with a cutoff score of 20 in the low-education group and 26 in the middle-high education group. MoCA-BC-NCT-A combination, MoCA-BC-SDT combination and MoCA-BC-LTT combination can be selected for CHE diagnosis in the cirrhosis population of the low education group; in the cirrhosis population of the middle and high education group, MoCA-BC-Quick Stroop combination can be selected for CHE diagnosis, which has a good diagnostic efficacy.

Keyword(s) : Liver cirrhosis Covert hepatic encephalopathy Cognitive function Early diagnostic Risk factor

2686 - Multisystem Crosstalk in MAFLD An Integrative Perspective from Organ Dialogue to Hepatic Pathology

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MASLD/MAFLD) has evolved from the traditional "second-hit" pathological model, where the liver serves as the initial site of disease onset, into a systemic disorder involving dynamic interactions among multiple organs. The core mechanism lies in dysregulated crosstalk among multiple organ axes, with the gut-liver axis serving as the disease initiation point. Gut barrier disruption and dysbiosis lead to lipopolysaccharide translocation and bile acid

metabolism disorders, subsequently activating hepatic inflammatory responses via the portal vein. Building upon this, the adipose tissue-liver axis exacerbates hepatic lipotoxicity through free fatty acid overflow and adipokine imbalance, while insufficient activation of brown adipose tissue weakens protective lipid clearance mechanisms. Additionally, the brain-liver axis modulates hepatic metabolism and inflammation via neuroendocrine signaling (e.g. brain-derived neurotrophic factor and vagus nerve pathways), while the kidney-liver axis shares insulin resistance and fibrosis pathways, forming bidirectional pathological associations. At the intrahepatic level, intercellular communication networks further amplify systemic signals: hepatic lipid accumulation triggers Kupffer cell polarization toward a proinflammatory phenotype, accelerating stellate cell activation and fibrosis via the senescence-associated secretory phenotype; sinusoidal endothelial capillary formation induces microenvironment hypoxia, synergistically promoting disease progression. These multiscale interactions collectively drive a vicious cycle of chronic inflammation, mitochondrial dysfunction, and metabolic reprogramming.

Based on these mechanisms, therapeutic strategies shift toward multi-target integrated interventions. For instance, FXR agonists and PPAR agonists can simultaneously repair the gut-liver axis and lipid metabolism, while GLP-1 receptor agonists concurrently improve pancreatic function, central appetite regulation, and hepatic inflammation. Cellular senescence-targeting drugs (e.g. senolytics) and fibrosis pathway inhibitors address advanced pathological stages. Future directions emphasize personalized treatment through multi-omics data and patient stratification, overcoming the limitations of single-pathway therapies. This crosstalk perspective not only explains disease heterogeneity but also provides a theoretical basis for combination therapies, marking the entry of MASLD/MAFLD management into the era of systems medicine.

Materials and Methods: none

Results: none

Conclusions: none

Keyword(s) : MAFLD multi-organ crosstalk organ dialogue multi-target therapy systems medicine

2687 - The Mediating Roles of HOMA β and TG HDL C in the Associations of General and Central

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Adiposity with metabolic dysfunction associated fatty liver disease A Cross Sectional Study

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Background / Hypothesis:

Obesity is a major risk factor for metabolic dysfunction-associated fatty liver disease (MAFLD), but its underlying mechanisms have not been fully elucidated. This study aims to quantify and compare the mediating roles of pancreatic islet β -cell function (HOMA- β) and atherogenic lipid profile (TG/HDL-C) in the association between general and central obesity and MAFLD.

Materials and Methods:

This study was a cross-sectional analysis, involving a total of 200 examinees. MAFLD was diagnosed according to the 2023 EASL criteria. General obesity was defined as BMI ≥ 24 kg/m², and central obesity was defined according to the China waist circumference standard. Multivariate logistic regression was used to analyze associations, and restricted cubic spline was applied to test nonlinear relationships. The proportion of mediating effects between HOMA- β and TG/HDL-C was quantified using a Bootstrap-based mediation model, and subgroup analysis was conducted to explore heterogeneity.

Results:

After adjusting for confounding factors, both BMI and waist circumference were significantly positively correlated with the risk of MAFLD (both $P < 0.01$). Mediation analysis showed that HOMA- β mediated 18.23% of the total effect of the BMI-MAFLD association and 12.35% of the waist circumference-MAFLD association (both $P = 0.026$). TG/HDL-C mediated 9.69% and 9.15% of the above associations, respectively (both $P = 0.04$). Subgroup analysis revealed that the association between BMI and MAFLD was stronger in individuals without hypertension (interaction $P = 0.031$), while the association between waist circumference and MAFLD was stronger in individuals without a history of diabetes and without a family history of diabetes (interaction $P = 0.007$ and 0.043 , respectively).

Conclusions:

In the association between obesity and MAFLD, both HOMA- β and TG/HDL-C play significant partial mediating roles, and the mediating effect of the islet β -cell function pathway accounts for a relatively higher proportion. The research results suggest that interventions targeting islet function and lipid metabolism may be of great value in the early pathogenesis of obesity-related MAFLD.

Keyword(s) : MAFLD; BMI; WC; HOMA- β ; TG/HDL-C

2688 - Immediate Improvement in HCV RNA Testing Following Active In Hospital Management An Interrupted Time Series Analysis

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Background / Hypothesis:

The attrition between hepatitis C virus (HCV) antibody screening and confirmatory RNA testing remains a critical barrier to treatment initiation. We evaluated the immediate and sustained impact of a multi-component, active case management strategy implemented within a large hospital on this key step in the care cascade.

Materials and Methods:

A single-center, retrospective observational study was conducted. An interrupted time series analysis (ITS) was used to model the HCV RNA testing rate among anti-HCV-positive individuals before and after the sequential implementation of integrated interventions, including an informatics alert system, patient recall, and dynamic clinical reminders, from 2017 to 2024.

Results:

Linear regression modeling of the ITS data showed: 1) a non-significant pre-intervention annual trend increase of +2.47% ($P=0.056$); 2) a significant immediate level increase of +27.86% ($P=0.028$) upon intervention; and 3) a non-significant change in the post-intervention slope (-1.15% per year, $P=0.627$). This indicates a substantial, abrupt improvement in the RNA testing rate that was subsequently maintained at a higher plateau.

Conclusions:

A proactive, in-hospital management strategy resulted in a significant and immediate "step-up" in HCV RNA

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confirmation testing, effectively strengthening a fragile link in the micro-elimination cascade. The intervention's primary effect was an initial implementation shock that durably elevated performance.

Keyword(s) : Hepatitis C, micro-elimination, care cascade, interrupted time series, in-hospital screening

2692 - Predictors of recompensation in autoimmune hepatitis related decompensated cirrhosis following immunosuppressive therapy in India

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Background / Hypothesis: Data on the predictors of recompensation following immunosuppressive therapy (IST) in autoimmune hepatitis-related decompensated cirrhosis (AIH-DC) are scarce

Materials and Methods: In this retrospective study, data of all patients with AIH-DC who underwent treatment from 2015 to 2024 were retrieved from a prospectively maintained electronic database. Frequency and predictors of recompensation (according to Baveno VII) following IST were assessed in patients with AIH-DC and were compared with those of patients who received standard medical therapy (SMT). Other outcomes include overall survival (OS), flare-free survival, and infection rate in both cohorts.

Results: Of 77 patients, 53 received IST (12 high-dose steroids and 41 low-dose steroids). The other 23 patients received SMT. Median age at presentation was 45 [interquartile range (IQR) 34-51] years, and the majority were females (78%). Median follow-up period was 39 [IQR: 35.5-48] months. Baseline parameters were comparable between the IST and SMT groups. Of 54 patients, 18 (34%) achieved recompensation in the IST group ($p=0.001$). Predictors of recompensation include patients with less than grade 3 ascites [Odds ratio (OR): 95% CI: 34 (3.7-304); $p=0.002$] hepatic encephalopathy < grade 3 [OR 1.66 (95% CI: 1.05-2.78);

$p=0.035$], Child-Turcot Pugh (CTP) score <9 [OR 6.78 (95% CI: 1.49-30.15); $p=0.013$], model for end stage liver disease-sodium score <25 [OR 3.9 (95% CI 1.14-39.04); $p=0.001$], and serum immunoglobulin-G levels <2300 g/dL [OR 3.38 (95% CI 1.0-11.2); $p=0.046$]. On Kaplan Meier analysis IST group had a better OS as compared to the SMT group. No significant differences were noted in liver-related outcomes or infection rate between the high-dose and low-dose steroid groups.

Conclusions: 34% patients achieved recompensation following IST in patients with IST. Lower CTP, MELD-Na score, serum IgG, lesser grade of HE and ascites predicted recompensation following IST in AIH-DC.

Keyword(s) :

2694 - Comparison of Diagnostic Performance of Non Invasive Steatosis and Fibrosis Scores and Ultrasound versus FibroScan in Metabolic Associated Fatty Liver Disease

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Background / Hypothesis:

Metabolically associated fatty liver disease (MAFLD) is an increasing global health burden. Early identification of hepatic steatosis and fibrosis is critical for diagnosis, risk stratification, and appropriate management of MAFLD. Although transient elastography (FibroScan) is widely used, simpler metabolic scores and conventional ultrasound (US) may offer more accessible alternatives, particularly in resource-limited settings.

The study aimed to compare the diagnostic performance of non-invasive serum-based metabolic scores and ultrasound with FibroScan for the evaluation of hepatic steatosis and fibrosis in patients with MAFLD.

Materials and Methods:

This cross-sectional study included 200 adult patients attending the hepatology clinic of a tertiary-care Egyptian hospital for FibroScan evaluation. All participants underwent detailed clinical assessment

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and anthropometric measurements, including body mass index, waist and hip circumferences, and waist-hip ratio. Laboratory investigations comprised complete blood count, liver and renal function tests, lipid profile, viral hepatitis markers, fasting blood glucose, 2-hour postprandial glucose, and HbA1c. Abdominal ultrasound and FibroScan were performed for all patients. Hepatic steatosis was defined using the controlled attenuation parameter (CAP), while liver fibrosis was assessed by liver stiffness measurement (LSM). The diagnostic performance of steatosis indices (HSI, FLI, TyG index, TyG-BMI, TyG-WC, TyG-WHR, TG/HDL ratio, and VAI) and fibrosis scores (FAST, Agile 3+, Agile 4, FIB-4, NAFLD fibrosis score, APRI, and BARD) was evaluated using receiver operating characteristic (ROC) curve analysis.

Results:

Ninety-eight patients (49%) were diagnosed with MAFLD based on CAP values. All evaluated non-invasive indices showed significant diagnostic ability for detecting hepatic steatosis ($p < 0.001$). Ultrasound demonstrated the highest diagnostic accuracy (AUC 0.892), followed by TyG-BMI (0.874), TyG-WC (0.863), FLI (0.846), and HSI (0.808), whereas the TyG index alone showed modest performance (AUC 0.715). Liver fibrosis was detected in 46 patients (23%) by LSM. Agile 3+ and Agile 4 scores showed the best overall performance for fibrosis detection (AUC 0.828 and 0.810, respectively; $p < 0.001$). Agile 3+ exhibited high sensitivity (85.3%) and negative predictive value (94.1%), while Agile 4 achieved the highest specificity (93.3%) and diagnostic accuracy (85.7%). Other scores like FIB-4, NFS, and BARD have moderate diagnostic performance, and APRI was not statistically significant.

Conclusions:

Ultrasound and selected metabolic indices, particularly TyG-BMI, TyG-WC, and FLI, demonstrate excellent diagnostic performance for hepatic steatosis compared with FibroScan. Agile fibrosis scores outperform traditional fibrosis indices, providing reliable, non-invasive tools for MAFLD assessment and supporting their use in routine clinical practice, especially in resource-constrained.

Keyword(s) : Metabolic associated fatty liver disease; FibroScan; Liver Fibrosis; Steatosis; Ultrasound; Non-invasive Scores

2696 - Map of Digestive and Liver Diseases in Egypt
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Background / Hypothesis:

Gastrointestinal (GI) diseases constitute a major public health burden in Egypt. However, nationally

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representative data describing their prevalence, geographic distribution, and associated risk factors remain limited. This study aimed to map the epidemiology of digestive diseases across Egyptian governorates and identify key demographic and lifestyle-related risk factors.

Materials and Methods:

A multicenter cross-sectional study was conducted between March 2023 to December 2024 and involved 2,027 patients presenting with gastrointestinal symptoms or diagnosed GI or liver diseases at hospitals and private clinics across multiple Egyptian governorates. Data collection included demographic characteristics, clinical presentations, comorbidities, lifestyle factors, and place of residence. Diagnoses were established according to ICD-11 criteria. Diseases were analyzed by anatomical category, geographic distribution, gender, and associated risk factors. Statistical analysis included chi-square tests, independent t-tests, and multivariate logistic regression to identify independent predictors.

Results:

Esophageal disorders were the most prevalent GI conditions (36.1%), followed by gastroduodenal (33.8%) and hepatic disorders (33.6%). Gastroesophageal reflux disease (GERD) accounted for 57.3% of esophageal disorders, while chronic gastritis represented 78% of gastroduodenal diseases. Viral hepatitis constituted 17.7% of hepatic cases, with hepatitis C virus (HCV) accounting for 80.5%. Functional gastrointestinal disorders were significantly more common among females ($p < 0.05$), whereas liver diseases predominated in males ($p < 0.001$). Diabetes mellitus showed a strong association with hepatic and malignant GI diseases, smoking was significantly linked to functional GI disorders, and urban residence emerged as an independent risk factor. Geographic variation was evident, with esophageal disorders most prevalent in Monufia (32.4%) and viral hepatitis highest in Asyut (18.8%).

Conclusions:

This study provides the first national-level epidemiological map of gastrointestinal diseases in Egypt, demonstrating marked regional, gender-based, and etiologic variability. GERD, chronic gastritis, and HCV-related liver disease remain dominant contributors to GI morbidity. These findings

underscore the need for region-specific prevention strategies, targeted screening programs, and tailored public health interventions to address Egypt's evolving digestive disease burden.

Keyword(s) : Gastrointestinal diseases; liver Disease; Egypt; Epidemiology; Geographic distribution

2697 - Demographic Profile and Clinical Indications of Patients Undergoing Liver Elastography in a Tertiary Government Hospital A Pilot Study

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Background / Hypothesis: Chronic liver disease (CLD) is a major global health problem, with progressive fibrosis leading to cirrhosis and its complications. Accurate assessment of fibrosis stage is crucial because the degree of liver fibrosis determines prognosis and guides management in chronic liver disease. Our hospital – a tertiary government referral center – recently acquired shear-wave liver elastography capability (since 2024) to enhance care for patients with liver disease. However, local data on the profile of patients undergoing this test and the clinical contexts prompting its use are limited. This pilot study will retrospectively review the initial cases of liver SWE performed at our center. It will characterize the demographic profile of these patients and document the clinical indications for elastography. Ultimately, the findings will shed light on the patterns of elastography utilization in a government hospital setting, and whether they align with global trends. Such data can inform future larger studies and guide optimal use of elastography in our local practice.

Materials and Methods: The target population is all patients who underwent a liver shear-wave elastography scan at the hospital from 2024 to September 2025. The author employ total enumeration sampling. Based on radiology logs, approximately 60 patients have undergone liver elastography since the service's introduction in 2024, which will constitute our sample size.

•Inclusion Criteria:

- Patients of any age or gender who underwent a clinically indicated liver elastography exam at the hospital from 2024 to September 2025.
- Those with available medical records or referral forms detailing the reason for elastography and relevant clinical data.

•Exclusion Criteria:



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-Any elastography exam that was technically unsuccessful or invalid.

-Patients who had liver elastography done outside the study period will not be included in this pilot analysis.

Results: In terms of clinical indications, the majority of patients (65%) were referred for elastography due to cirrhosis or chronic liver disease, underscoring the role of liver elastography as a noninvasive tool for fibrosis assessment in patients with established or suspected advanced liver disease. The substantial proportion of patients with chronic viral hepatitis (11.7%), particularly hepatitis B, highlights the continued public health burden of viral hepatitis in the Philippines and reinforces the importance of elastography in disease staging and monitoring.

Conclusions: This pilot study describes the results of shear-wave liver elastography in a tertiary government hospital, demonstrating its use predominantly among middle-aged patients with suspected or established chronic liver disease.

Keyword(s) : elastography, chronic liver disease, hepatitis

2698 - Model combining MELD ICG R15 and systemic immune inflammation index predicts 28 day clinical progression in hepatitis B virus related acute on chronic liver failure patients

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Background / Hypothesis:

Hepatitis B virus-related acute-on-chronic liver failure (HBV-ACLF) carries high short-term mortality. Existing prognostic scores are often static and may not fully capture dynamic liver reserve and systemic inflammation.

Materials and Methods:

A retrospective study included 66 HBV-ACLF patients admitted between June 2023 and December 2024, categorized by 28-day outcome into improvement (n=49) and progression (n=17) groups. Clinical data, indocyanine green clearance test parameters, and systemic immune-inflammation index (SII) were collected. Logistic regression identified predictors for model construction. Model discrimination was evaluated using the receiver operating characteristic curve, with internal validation via bootstrap and assessment of calibration and clinical net benefit.

Results:

Multivariate analysis identified SII/1000 as an independent risk factor (OR=5.77, 95% CI: 1.64-20.37, P=0.006), with MELD-ICG R15 score showing marginal significance (P=0.082). The final model was: $\text{logit}(P) = -6.658 + 0.060 \times (\text{MELD-ICG R15}) + 1.753 \times (\text{SII}/1000)$. The area under the curve was 0.761 (95% CI: 0.627-0.873), with bootstrap-corrected area under the curve of 0.732. At the optimal cutoff of 0.209, sensitivity was 82.4% and specificity 65.3%. The calibration curve showed good agreement, and decision curve analysis indicated positive clinical net benefit.

Conclusions:

The combination of MELD-ICG R15 and SII effectively predicts 28-day clinical progression risk in HBV-ACLF patients, offering a potential tool for early risk stratification and personalized intervention.

Keyword(s) : hepatitis B virus, acute-on-chronic liver failure, indocyanine green clearance test, systemic immune-inflammation index, prognosis, prediction model

2701 - MSC mediated Modulation of the IL 4 CD39 Axis in Treg cells Restores Immune Tolerance and Improves Treatment Outcomes in Steroid Resistant Autoimmune Hepatitis

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Background / Hypothesis:

Autoimmune hepatitis (AIH) is an immune-mediated liver disease that poses significant challenges in treatment, particularly for patients who are steroid-resistant. Recent studies have highlighted that reduced CD39 expression on Treg cells, leading to impaired immunosuppressive function, is one of the key pathogenic mechanisms in AIH. Mesenchymal stem cells (MSCs), as a potential therapy, have demonstrated promising results in treating immune-related diseases. This study aims to explore the role of MSCs in restoring Treg cell function by modulating the IL-4/CD39 axis, providing a novel therapeutic strategy for steroid-resistant AIH patients.

Materials and Methods:

We assessed the expression of IL-4 in peripheral blood mononuclear cells (PBMCs) from steroid-resistant AIH patients using flow cytometry, and investigated the

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correlation between IL-4 expression and CD39 in Treg cells. Additionally, immunofluorescence staining was performed to examine the distribution of CD39 and IL-4 in liver tissue from these patients. To investigate the causal relationship between IL-4 and AIH susceptibility, we conducted a two-sample Mendelian randomization (MR) analysis. Furthermore, we evaluated the therapeutic effects of MSCs in a ConA-induced AIH mouse model by measuring liver injury biomarkers, immunoglobulin levels, and Treg cell subtypes and function, with a focus on the regulation of the IL-4/CD39 axis.

Results:

In PBMCs from steroid-resistant AIH patients, we observed significantly elevated IL-4 levels ($P < 0.01$), which correlated negatively with CD39 expression on Treg cells ($R = -0.42$, $P = 0.02$). MR analysis confirmed a significant causal relationship between IL-4 and AIH susceptibility. The analysis using the Inverse Variance Weighted method showed statistical significance (OR = 1.459027, 95% CI: 1.006 -2.115, $P = 0.046$) suggesting that IL-4 may increase the risk of AIH. These findings demonstrate a critical link between IL-4, CD39 deficiency, and impaired immune suppression in Treg cells. In the MSC-treated AIH mouse model, MSC treatment significantly reduced ALT, AST, and IgG levels ($P < 0.0001$), indicating effective immunoregulation. MSC treatment also significantly increased the proportion of Treg cells, restored CD39 expression on Treg cells, and decreased IL-4 expression, suggesting that MSCs restore Treg cell function by modulating the IL-4/CD39 axis.

Conclusions:

Our findings demonstrate that MSCs can modulate the IL-4/CD39 axis in Treg cells to restore immune tolerance and offer a potential new therapeutic solution for steroid-resistant AIH patients. This approach represents a novel strategy for treating autoimmune liver diseases.

Keyword(s) : MSC, IL-4, Treg, CD39, steroid-resistant, autoimmune hepatitis, immunoregulation, stem cell therapy

2702 - Modified Leipzig (Leipzig U) score is equally helpful for the early diagnosis of Wilson disease in children presenting with liver disease

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Background and Aim: Leipzig score ≥ 4 confirms the diagnosis of Wilson disease (WD). However, 24-hour urine copper estimation and genetic results delay its application and timely initiation of chelation. Low uric acid (UA) is seen in WD due to proximal tubular dysfunction. We studied the diagnostic utility of serum UA as a simple biomarker for WD with its incorporation into a modified version of Leipzig score.

Methods: This retrospective data of 221 children with chronic liver disease was divided into 2 cohorts (derivation, $n=120$, WD = 58 and validation, $n=101$, WD = 58). UA levels were studied and centiles were derived from the derivation cohort. Based on centiles of UA, a score of 0, 1 and 2 were derived which were added into the Leipzig score without genetic diagnosis and without 24-hour urinary copper results. Area under receiver operating characteristic curves (AUROC) were studied.

Results: Mean UA levels were 2.60 ± 1.27 which were significantly lower in WD than autoimmune liver disease (ALD) (1.90 ± 0.89 vs 3.26 ± 1.22 , $p < 0.001$). Cut-off values at 50th (≤ 2.5 mg/dL) and 40th centiles (≤ 2.2 mg/dL) had a positive likelihood ratio of WD of 3.86 and 4.29. A score of 0, 1 and 2 were given for UA levels ≥ 2.5 , 2.2-2.5 and ≤ 2.2 mg/dL. Removing 24-hour urine copper and genetic diagnosis from the Leipzig score and adding UA score conferred a sensitivity and specificity of 91.4% and 100% (AUROC 0.986, $p < 0.001$) in the derivation cohort, and 94.8% and 97.7% (AUROC 0.965, $p < 0.001$) in the validation cohort, which is comparable to Leipzig score without genetic diagnosis in the whole cohort (sensitivity 91.4%, specificity 100%, AUROC 0.998, $p < 0.001$).

Conclusion: Using serum uric acid levels helps in early diagnosis of WD in the absence of genetic testing. Modified Leipzig (Leipzig-U) score needs validation in an external cohort.

Keyword(s) : wilson disease, leipzig score

2703 - Sequential plasma adsorption and therapeutic plasma exchange for extreme hyperbilirubinemia in Budd-Chiari syndrome with acute on chronic liver failure

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Background / Hypothesis:

Extreme hyperbilirubinemia in advanced liver disease can contribute to multiorgan dysfunction and may

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complicate haemostatic assessment because of “rebalanced” haemostasis, in which the international normalized ratio (INR) does not reliably reflect bleeding risk. We hypothesized that sequential artificial liver support combining plasma adsorption and therapeutic plasma exchange can rapidly debulk bilirubin in Budd–Chiari–related acute-on-chronic liver failure, and that viscoelastic testing can provide clinically more relevant haemostatic information than INR alone. Primary endpoints were to evaluate the safety and efficacy of the therapy and bilirubin kinetics (total and direct). Secondary endpoints included renal markers (creatinine, cystatin C), inflammation (CRP, IL-6), conventional coagulation parameters (INR, fibrinogen, factor V), and viscoelastic haemostasis (thromboelastography with functional fibrinogen assessment).

Materials and Methods:

We report the case of a 47-year-old woman with Budd–Chiari syndrome complicated by acute-on-chronic liver failure and profound jaundice. Five procedures were performed over five consecutive days: two sessions of double plasma molecular adsorption and three sessions of therapeutic plasma exchange. Laboratory sampling was standardized to four time points per day: morning baseline, immediately pre-procedure, immediately post-procedure, and 6–12 h post-procedure (with next-morning values representing 24 h follow-up).

Results:

Initial bilirubin concentration was extreme (total bilirubin 1003 $\mu\text{mol/L}$; direct bilirubin 677 $\mu\text{mol/L}$). Each procedure achieved a marked immediate reduction in total bilirubin (pre→post): 1003.0→740.4 (–26.2%), 813.5→500.7 (–38.5%), 729.1→472.0 (–35.3%), 601.7→407.2 (–32.3%), and 538.0→366.6 $\mu\text{mol/L}$ (–31.9%). A rebound pattern was observed 6–12 h post-procedure (range +2.3% to +24.6%). Across the 5-day sequence, total bilirubin decreased from 1003.0 to 366.6 $\mu\text{mol/L}$.

Conventional coagulation parameters changed discordantly depending on treatment modality: adsorption sessions were followed by INR worsening (2.08→4.13; 1.68→2.84), whereas plasma exchange improved INR (1.99→1.39; 3.15→1.40; 1.52→1.42) and increased factor V activity (30→61%; 35→59%; 46→73%) and fibrinogen levels (e.g., 1.6→1.9 g/L). Viscoelastic testing demonstrated only moderate

impairment of clot strength, with functional fibrinogen indices improving after plasma exchange, highlighting a discordance between INR and whole-blood haemostasis. All procedures were completed without major bleeding complications.

Conclusions:

In Budd–Chiari syndrome complicated by acute-on-chronic liver failure and extreme hyperbilirubinemia, sequential plasma adsorption and therapeutic plasma exchange achieved rapid bilirubin debulking with a reproducible post-treatment rebound. Modality-dependent INR fluctuations were not aligned with viscoelastic haemostatic profiles, supporting viscoelastic-guided interpretation within the framework of rebalanced haemostasis in advanced liver disease. This strategy may serve as a practical bridge while definitive therapy is pursued.

Keyword(s) :

2704 - Differences between non alcoholic liver disease and alcoholic liver disease related HCC

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Background / Hypothesis:

Non-alcoholic liver disease and alcoholic liver disease begin as simple steatosis that may progress to steatohepatitis and ensuing liver-related complications such as cirrhosis and hepatocellular carcinoma (HCC). We investigated differences in clinical characteristics between non-alcoholic liver disease (NALD) and alcoholic liver disease (ALD)-related HCC.

Materials and Methods:

NALD and ALD patients were identified from our department’s prospective HCC database. A total of 54 and 45 patients met predefined inclusion and exclusion criteria for the NALD-HCC and ALD-HCC groups, respectively. Clinical, biochemical and tumor characteristics were studied.

Results:

NALD-HCC patients were older compared to ALD-HCC patients (71±9 vs. 65±9 years, $P<0.001$) and less male predominant (64% vs. 97%, $P<0.001$). Prevalence of diabetes mellitus (77% vs. 35%, $P<0.001$) and hypertension (79% vs. 57%, $P<0.001$) were significantly higher in the NASH-HCC group. There were no differences in alpha-fetoprotein level, lesions found at

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diagnosis (unifocal/multifocal) or prevalence of portal vein invasion. In both group, almost half of the patients were in TNM stage 4 at the time of diagnosis and more than 50% of patients were not suitable for any therapy. Median survival in the NALD-HCC and ALD-HCC groups were 13 and 7 months respectively ($P=0.114$).

Conclusions:

Despite significant differences in demography of the NALD-HCC and ALD-HCC groups, liver and tumor characteristics were comparable. Most patients were diagnosed late and were not amenable to curative or locoregional therapies. Better characterization of patients with NALD and ALD at risk of HCC is necessary to optimize screening, surveillance, and management strategies.

Keyword(s) : Hepatocellular carcinoma, Alcoholic liver disease

2705 - Early Expansion of CCR7⁺ PD-1⁺ NK Cells and HBsAg Decline Predict Interferon Induced Immune Reconversion in Chronic Hepatitis B

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Background / Hypothesis:

Objective: Pegylated interferon-alpha (Peg-IFN- α) remains a cornerstone therapeutic agent for achieving functional cure in chronic hepatitis B (CHB), yet its overall efficacy remains suboptimal. This study aimed to identify early immune response predictors during Peg-IFN- α therapy for CHB and longitudinally characterize the dynamics of peripheral blood immune cell subsets to inform precision clinical management. Methods: This retrospective study comprised two components: 1) Serological prediction analysis: Ninety-six CHB patients receiving nucleos(t)ide analogues (NAs) plus Peg-IFN- α were stratified into responders ($n=24$) and non-responders ($n=72$) based on HBsAg seroconversion at 24 weeks. Baseline and 12-week serological markers were analyzed, with independent predictors of 24-week response identified via univariate/multivariate logistic regression and receiver operating characteristic (ROC) curve analysis. 2) Immunological response profiling: A subcohort including the interferon responder group (IRG), interferon non-responder group (INRG), and nucleos(t)ide analogue non-responder group (NANRG) was assessed longitudinally (0, 12, 24, 36, 48 weeks)

using multicolor flow cytometry. Key immune cell subset frequencies were evaluated, with response-specific immunokinetic markers identified through amplitude (Δ value), fold-change analysis, and generalized estimating equation (GEE)-based trajectory modeling.

Results: 1) Serological predictors: The logarithmic reduction in HBsAg at 12 weeks (Δ IgHBsAg) emerged as the strongest independent predictor of 24-week HBsAg response (adjusted OR = 3.90, $P < 0.001$). A dynamic model incorporating Δ IgHBsAg and alanine aminotransferase reduction (Δ ALT) demonstrated excellent predictive performance (AUC = 0.87). 2) Immunological dynamics: Compared to non-responders, IRG exhibited robust multilineage immune cell activation at 12 weeks, marked by significant expansion of CCR7⁺ PD-1⁺ NK cells, M1 macrophages, and CCR7⁺ PD-1⁺ CD8⁺ T cells ($P < 0.05$). Longitudinal trajectory modeling revealed distinct evolution patterns of CCR7⁺ PD-1⁺ NKbri cells among IRG, INRG, and NANRG (interaction $P < 0.001$), identifying this subset as the most discriminative immunokinetic biomarker. 3) Mechanistic insights: Successful immune reconstitution was characterized by early innate immune activation and exhausted CD8⁺ T cell reinvigoration, serologically reflected by rapid Δ IgHBsAg decline. Conclusion: The 12-week Δ IgHBsAg level represents a robust serological biomarker for predicting short-term therapeutic response to Peg-IFN- α , functioning as a composite indicator of successful multi-lineage immune activation in deep tissue compartments. The critical determinant of interferon efficacy lies in an early immune reconstitution program characterized by the specific kinetic profile of NK cells, particularly the CCR7⁺ PD-1⁺ NKbri subset. These findings establish both theoretical and clinical foundations for developing precision treatment strategies for chronic hepatitis B based on early dynamic immunological monitoring.

Materials and Methods:

Results:

Conclusions:

Keyword(s) : Chronic Hepatitis B; Pegylated Interferon-

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alpha; Efficacy Prediction; HBsAg Clearance; CCR7; NK Cell; T Cell

2707 - Microbial metabolism dysfunction induced by transarterial chemoembolization aggravates postprocedural liver injury in HCC

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Background / Hypothesis:

Transarterial chemoembolization (TACE) is widely used to treat unresectable hepatocellular carcinoma (HCC). Liver injury induced by TACE (TACE-LI) is the most common complication and limits long-term outcomes. Beyond the conventional understanding of direct TACE-induced damage to normal liver tissue, the underlying mechanisms of TACE-LI remain unclear. We aimed to elucidate the relationship between gut microbiota disturbances and TACE-LI.

Materials and Methods:

Microbial multi-omics analysis, genetically engineered bacteria and transcriptomics were used to study microbiota disturbances and host responses in TACE-LI.

Results:

Rats with antibiotic-depleted gut microbiota, as well as rats receiving fecal transplants from donor rats or patients who had undergone TACE, exhibited more severe TACE-LI. *Limosilactobacillus reuteri* (*L. reuteri*) abundance was significantly reduced in TACE-treated rats and patients with HCC. Reduced *L. reuteri* abundance after TACE led to decreased levels of tryptophan metabolite indole-3-lactic acid (ILA), while administration of live *L. reuteri* or ILA provided effective protection against TACE-LI. Mechanistically, *L. reuteri* relied on the key enzyme phenyllactate dehydrogenase to generate ILA. ILA inhibits the ATPase activity of heat shock protein 90, thereby deactivating the NOD-like receptor protein 3-inflammasome in macrophages and suppressing hepatic pro-inflammatory responses. Reduced levels of *L. reuteri* and ILA were correlated with aggravated LI and poorer overall survival in TACE-treated patients with HCC.

Conclusions: This is the first study to identify gut microbiota disturbance, i.e. efficiency of *L. reuteri* metabolite ILA, as a significant cause of TACE-LI. Administration of *L. reuteri* or ILA may serve as a promising therapeutic strategy to mitigate TACE-related adverse effects and improve prognosis in HCC.

Keyword(s) : liver injury; transarterial chemoembolization; gut microbiota; hepatoprotective therapy.

2708 - Risk Factors and Development of a Predictive Model for Chronic Kidney Disease in Patients with Metabolic Dysfunction-Associated Steatotic Liver Disease

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Background: To identify risk factors for chronic kidney disease (CKD) among patients with metabolic dysfunction-associated steatotic liver disease (MASLD) and to develop a risk prediction model for MASLD with CKD comorbidity to support targeted prevention and early screening.

Materials and Methods: We retrospectively enrolled 8,975 MASLD patients with MASLD diagnosed by color Doppler ultrasonography at the Affiliated Hospital of Nantong University between 2021 and 2023.

Participants were categorized into CKD and non-CKD groups according to standard diagnostic criteria. Baseline biochemical parameters, including markers of glucose, lipid metabolism, inflammation, and organ function, were compared between two groups.

Independent predictors were identified using multivariable logistic regression and incorporated into a nomogram. Model performance was temporally validated in an independent MASLD cohort from 2024 using receiver operating characteristic (ROC) analysis, calibration plots, and decision curve analysis (DCA). **Results:** Compared with the non-CKD group, the CKD group exhibited significantly higher levels of systolic blood pressure, creatinine, triglycerides, and uric acid to high-density lipoprotein cholesterol ratio, but lower hemoglobin compared with the non-CKD group ($P < 0.05$). Age, systolic blood pressure, hemoglobin, triglycerides, and uric acid to high-density lipoprotein cholesterol ratio were identified as independent predictors of MASLD with CKD. The nomogram demonstrated good discrimination ability, showed consistent calibration in temporal validation, and demonstrated favorable net benefit across clinically relevant threshold probabilities in DCA.

Conclusions: A nomogram based on routinely available indicators provides an effective approach to estimating CKD risk in patients with MASLD, which may facilitate early screening and individualized interventions for high-risk individuals.

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Keyword(s) : MASLD, CKD, Risk factors, Risk Prediction model, Nomogram

2709 - Machine Learning Based Prediction of Incomplete UDCA Response in Primary Biliary Cholangitis Model Development and Assessment

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Background / Hypothesis:

In patients with Primary Biliary Cholangitis (PBC), incomplete response to ursodeoxycholic acid (UDCA) is strongly associated with adverse clinical outcomes. This study aimed to develop a machine learning-based predictive model to enhance the accuracy of the early assessment of incomplete UDCA response.

Materials and Methods:

A retrospective study was conducted on patients with PBC who were hospitalized at the Public Health Clinical Center Affiliated to Shandong University from August 2011 to August 2024. Clinical data, including demographic information, symptoms and signs, medical history, comorbidities, laboratory tests, and imaging studies were collected. Baseline variables were screened using univariate analysis, least absolute shrinkage and selection operator (Lasso) regression, and the Boruta algorithm to identify predictors. Prediction models were developed using six machine learning algorithms, and their performance was evaluated. Model interpretability was achieved using SHAP.

Results:

Patients were randomly allocated to training (n=220) and validation (n=94) sets at a 7:3 ratio. The intersection of the three feature selection methods identified seven predictors: ALP, Stages, ALBI, GGT, Complement C3, D-dimer, and Direct Bilirubin. Among the models evaluated, Random Forest demonstrated superior performance with an area under the receiver operating characteristic curve (AUC) of 0.822, while Decision Curve Analysis (DCA) indicated favorable clinical utility.

Conclusions:

We developed an interpretable Random Forest model incorporating seven baseline clinical variables to predict UDCA response in patients with PBC. This model provides clinicians with a reliable tool for early, individualized treatment decision-making at the initial stage of therapy, offering significant practical value

and potential for broader application.

Keyword(s) : PBC; UDCA; Incomplete Response; Machine Learning; Prediction Model; Random Forest; SHAP

2710 - Construction of nomogram based on inflammatory markers to predict recurrence and survival of single small hepatocellular carcinoma after radiofrequency ablation

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Background / Hypothesis:

Radiofrequency ablation (RFA) is a primary treatment for early-stage single small hepatocellular carcinoma (HCC), yet post-operative recurrence and mortality remain high. Inflammation plays a key role in HCC progression, and inflammatory markers may reflect tumor biology and prognosis. This study aimed to identify risk factors for recurrence and poor survival after RFA and to develop prognostic nomogram models based on inflammatory markers to guide clinical decisions.

Materials and Methods:

A retrospective analysis was conducted on 270 patients with early-stage single small HCC who underwent initial RFA between November 2011 and December 2020. Data included general, etiological, laboratory, and imaging information collected within one week pre-RFA. Patients were randomly split into training and verifying sets. Optimal cut-off values for inflammatory markers (NLR, RLR, PLR, LMR, PNI) were determined using X-tile based on 5-year recurrence-free survival (RFS) and overall survival (OS). Univariate and multivariate Cox regression analyses identified independent risk factors. Nomograms for RFS and OS were constructed and internally validated using Harrell's C-index, AUC-ROC, calibration, and decision curve analysis.

Results:

Multivariate analysis identified ascites, tumor diameter >2cm, AFP >400 ng/ml, and LMR <3.2 as independent predictors of RFS. For OS, male gender, AFP >400 ng/ml, early recurrence, and RLR ≥19 were independent predictors. Nomograms incorporating these factors showed good discrimination and calibration. The RFS model had C-indices of 0.706

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(training) and 0.705 (verifying); the OS model had C-indices of 0.851 and 0.841, respectively. AUCs for 1-, 3-, and 5-year predictions were consistently robust. Risk stratification based on the nomograms significantly distinguished between low- and high-risk groups in both sets ($P < 0.0001$).

Conclusions:

Inflammatory markers are significantly associated with liver function and patient demographics in HCC. NLR, LMR, and RLR are independent prognostic factors for recurrence and survival after RFA. The established nomograms, based on readily available clinical and inflammatory markers, provide accurate and practical tools for predicting individual recurrence probability and survival, aiding in risk stratification and clinical management.

Keyword(s) : hepatocellular carcinoma, radiofrequency ablation, nomogram, inflammatory markers, prognosis, recurrence, survival, neutrophil-to-lymphocyte ratio, lymphocyte-to-monocyte ratio, prognostic nutrition index

2712 - Development and Validation of a Nomogram Incorporating Thyrotropin and Metabolic Parameters for Predicting Metabolic Dysfunction Associated Steatotic Liver Disease Risk A Cross sectional study

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a growing global health concern. Thyroid dysfunction influences metabolic homeostasis, but its role in predicting MASLD risk, especially in Eastern populations and within the euthyroid range, remains unclear. This study aimed to investigate the association between low thyroid function and MASLD and to develop a validated nomogram integrating thyroid-stimulating hormone (TSH) for individualized risk prediction.

Materials and Methods:

This retrospective study enrolled 3,734 subjects as a training cohort and 865 external subjects for validation. MASLD was defined as the presence of hepatic steatosis on ultrasound and at least one cardiometabolic risk factor. Thyroid function was categorized as strict-normal (TSH 0.4–2.5 mIU/L), low-

normal (TSH 2.5–4.2 mIU/L), or subclinical hypothyroidism (TSH ≥ 4.2 mIU/L). Independent predictors identified by multivariable logistic regression were incorporated into a nomogram. The model was assessed by its discrimination (area under the curve, AUC), calibration (calibration plot, Hosmer–Lemeshow test), and clinical utility (decision curve analysis).

Results:

Low thyroid function (TSH ≥ 2.5 mIU/L) was independently associated with higher MASLD risk (adjusted OR 1.73, 95% CI 1.47–2.04), showing a dose-response relationship. The final nomogram incorporated BMI, ALT, BUA, HDL, and TSH. It demonstrated excellent discrimination, with AUCs of 0.846 (training) and 0.848 (validation), superior to the TyG index. The model was well-calibrated and provided positive net benefit on decision curve analysis.

Conclusions:

Low thyroid function is an independent risk factor for MASLD. We developed a validated nomogram integrating TSH with metabolic parameters, offering a practical tool for individualized MASLD risk assessment and potential early intervention.

Keyword(s) : MASLD; TSH; Nomogram; Cross sectional

2713 - TRAF2 Deficiency Aggravates Mitochondrial Dysfunction Through PI3K AKT Pathway Inhibition in Acetaminophen Induced Liver Injury

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Background / Hypothesis:

Acetaminophen (APAP) overdose is a major factor in drug-induced acute liver failure. Mitochondrial dysfunction plays a central role in the pathogenesis of APAP-induced liver injury (AILI). Tumor necrosis factor receptor-associated factor 2 (TRAF2), an adaptor protein involved in the NF- κ B signaling pathway. Increasing evidence suggests it plays a role in regulating mitochondrial function. However, the role of TRAF2 in AILI is poorly understood.

Materials and Methods:

The AILI mouse model was established by intraperitoneal injection of 300 mg/kg APAP. The blood and liver tissues were collected for assessment

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of liver necrosis and mitochondrial function. To further verify the mechanism, hepatocyte-specific TRAF2 knockout (TRAF2-HKO) mice were generated.

Results:

APAP overdose significantly increased the mRNA and protein level of TRAF2 in the liver tissues of wild-type mice. To investigate the role of TRAF2 in AILI, TRAF2-HKO mice were treated with APAP. TRAF2 deficiency resulted in aggravated liver necrosis, increased serum ALT and AST levels, and exacerbated liver oxidative stress. RNA-Seq showed that the PI3K-AKT pathway was significantly downregulated in the TRAF2-HKO mice with AILI, which was verified by detecting the phosphorylation of AKT downstream proteins. Increased mitochondrial permeability was found in the livers of TRAF2-HKO mice. In vitro experiments demonstrated that TRAF2 knockdown increased GSK3 β activity by inhibiting AKT, thereby increasing mitochondrial permeability and reactive oxygen species. Gene set enrichment analysis revealed a significant downregulation of the oxidative phosphorylation (OXPHOS) subunits and mtDNA maintenance pathway in TRAF2-HKO mice, suggesting that TRAF2 may be related to mitochondrial biogenesis in AILI. TRAF2 deficiency reduced the OXPHOS subunits expression and mtDNA content in AILI mice. Mechanistically, TRAF2 knockdown could reduce NRF1 expression and mitochondrial biogenesis by inhibiting AKT activity.

Conclusions:

These results indicate that TRAF2 deficiency inhibits the PI3K-AKT pathway, thereby suppressing downstream GSK3 β phosphorylation and NRF1 expression, exacerbating mitochondrial dysfunction in AILI. This finding suggests that TRAF2 may be a potential novel therapeutic target for AILI.

Keyword(s) : acetaminophen, drug-induced liver injury, mitochondria, TRAF2

2715 - Va Cur@Lips Inhibits the Metastasis of Hepatocellular Carcinoma by Targeting the Peri Tumoral Fibrotic Microenvironment

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Background / Hypothesis: The incidence of local tumor progression (LTP) remains high after microwave ablation (MWA) for hepatocellular carcinoma (HCC). This study aimed to investigate the potential mechanism by which vitamin A-modified curcumin

nanoliposomes (Va-Cur@Lips) suppress the pro-tumor microenvironment by targeting peri-tumoral activated hepatic stellate cells (aHSCs), thereby reducing LTP after MWA of HCC.

Materials and Methods: Single-cell RNA sequencing was performed on tumor tissues and adjacent non-tumorous liver tissues from 3 HCC patients to analyze phenotypic transformation and distribution patterns of HSCs in peri-tumoral tissues. Va-Cur@Lips were prepared by thin-film hydration using lecithin and cholesterol as membrane materials, and the regulatory effects of Va-Cur@Lips on HSCs activation and migration was evaluated. A mouse orthotopic HCC model with incomplete MWA was established, and the in vivo distribution and targeting efficiency of Va-Cur@Lips in the aHSCs around the tumor were observed. The anti-tumor efficacy of Va-Cur@Lips combined with MWA was evaluated by histological assessment. Peri-tumoral tissues around the ablation zone were collected for Masson staining and immunohistochemistry to assess HSCs activation and clarify the in vivo regulatory effect of Va-Cur@Lips on peri-tumoral HSCs activation.

Results: Single-cell sequencing revealed significant remodeling of the HSCs compartment during HCC progression, with specific expansion of matrix-producing aHSC subsets in the tumor microenvironment. It was also confirmed the transition of HSCs from a quiescent phenotype to a tumor-associated activated phenotype. Va-Cur@Lips exhibited favorable physicochemical properties: particle size 174.27 ± 1.00 nm, PDI < 0.3 . The zeta potential and encapsulation efficiency (EE%) were found to be -18.99 ± 0.32 mV and $99.96 \pm 0.01\%$, respectively. Immunofluorescence staining showed the largest and strongest green-blue merged fluorescence in the Va-Cur@Lips group. Vitamin A modification further enhanced the uptake of curcumin liposomes by LX-2 cells. CCK-8 and wound-healing assays demonstrated that Va-Cur@Lips exerted the most significant inhibitory effects on LX-2 cells proliferation and migration. All curcumin formulations inhibited tumor growth, among which Va-Cur@Lips combined with MWA resulted in the slowest tumor growth and optimal anti-tumor efficacy. Masson staining indicated that residual cancer after incomplete ablation could activate the tumor fibrotic microenvironment. The Va-Cur@Lips+MWA group showed the smallest percentage of collagen fiber area, and fibrosis had almost completely reversed. Immunohistochemical showed a significantly reduced percentage of α -SMA-positive cells in the Va-Cur@Lips+MWA group.

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Conclusion : Va-Cur@Lips can effectively reverse the peri-tumoral fibrotic microenvironment of HCC by targeting and inhibiting HSCs activation, thereby better assisting MWA and suppressing the occurrence and progression of residual HCC after incomplete ablation.

Keyword(s) : microwave ablation ; hepatocellular carcinoma ; local tumor progression ; curcumin ; nanoliposomes

2716 - Preoperative prediction power of radiomics and non radiomics fusion model based on US CEUS for microvascular invasion of hepatocellular carcinoma A multicenter study

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Background / Hypothesis: To establish microvascular invasion (MVI) prediction radiomics and non-radiomics fusion model based on the preoperative gray-scale ultrasound (US), Color Doppler Flow Imaging (CDFI), and contrast-enhanced ultrasound (CEUS) imaging of patients with hepatocellular carcinoma (HCC).

Materials and Methods: A total of 489 HCC patients who underwent preoperative US-CEUS examinations from 8 hospitals between July 2020 and June 2025 were enrolled. According to the MVI status, all the patients were classified: MVI-negative (M0) and MVI-positive (M1/M2). The HCCs were randomly divided into training (n=337) and internal validations (IV, n=84) cohorts with a 4:1 ratio for developing and evaluating multimodal ultrasound deep learning models. Diagnostic performance of the model was verified using external validation (EV, n=68) cohort. Based on the different US modalities, five modules were established: ModelUS, ModelCDFI, ModelCEUS, ModelUS-CEUS, and ModelUS-CEUS+non-radiomics, and compare the diagnostic accuracy of these different models in preoperative predicting MVI.

Results: The AUC of ModelUS, ModelCDFI, ModelCEUS, ModelUS-CEUS,

and ModelUS-CEUS+non-radiomics for HCC-MVI was 0.594, 0.629, 0.841, 0.853, and 0.871 in the EV cohort, respectively. The diagnostic sensitivity of ModelUS-CEUS+non-radiomics were superior to

ModelUS-CEUS (80.8% vs 69.6%, $p < 0.01$), while the specificity of ModelUS-CEUS+non-radiomics was comparable to that of ModelUS-CEUS (76.3% vs 80.0%, $p > 0.05$). The diagnostic sensitivity and specificity of ModelUS-CEUS+non-radiomics were superior to <5-year (80.8% vs 66.7%, and 76.3% vs 50.0%, both $p < 0.01$, respectively) and >5-year skilled radiologists (80.8% vs 66.7%, and 85.5% vs 34.9%, both $p < 0.01$, respectively), respectively.

Conclusions: ModelUS-CEUS+non-radiomics with high sensitivity and specificity in preoperative HCC-MVI prediction shows its potential advantages, and its diagnostic performance is significantly superior to that of the ModelUS-CEUS and senior radiologists

Keyword(s) : hepatocellular carcinoma ; microvascular invasion ; contrast-enhanced ultrasound ; deep learning

2717 - Clinical Profile Comparison Between HBeAg Positive and HBeAg Negative Hepatocellular Carcinoma Patients Highlighting Advanced BCLC Staging and Low Antiviral Treatment Uptake in an Indonesian Cohort Analysis

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Background / Hypothesis:

HBeAg status is an independent risk factor for hepatocellular carcinoma (HCC) in chronic Hepatitis B. Despite the availability of antiviral treatment, many patients progress to malignancy without prior intervention. This study aims to analyze the clinical profiles differences and treatment history between HBeAg-positive and HBeAg-negative HCC patients within an Indonesian cohort

Materials and Methods:

A cohort of 100 HCC patients was analyzed, divided into HBeAg-positive (n=50) and HBeAg-negative (n=50) groups. Data collected included demographic profiles (age and sex), liver function tests (SGOT, SGPT, Albumin, and Bilirubin), coagulation markers (PT/INR), and platelet count. Clinical and virological assessments

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were also performed, including BCLC staging, Alpha-fetoprotein (AFP) levels, presence of cirrhosis, HBV DNA, and history of antiviral treatment. Statistical analysis was conducted to compare variables between the two groups

Results:

A significant majority of the total cohort (70%) were not receiving antiviral treatment at the time of diagnosis. Specifically, 66% of HBeAg-negative and 74% of HBeAg-positive patients were untreated ($p=0.51$). HBeAg-negative patients were significantly more likely to present with advanced disease, with 90% diagnosed at an intermediate-advanced BCLC stage compared to 68% in the HBeAg-positive group ($p=0.01$). While HBeAg-positive patients showed significantly higher median AFP levels (4.53×10^2 ng/dL) than HBeAg-negative patients (1.48×10^2 ng/dL) ($p=0.03$), other variables such as age, liver enzymes, and HBV DNA levels did not show statistically significant differences

Conclusions:

In this Indonesian cohort, HBeAg-negative HCC patients are more likely to present at an intermediate-advanced BCLC stage despite having lower AFP levels compared to HBeAg-positive patients. The finding that 70% of the patients were not on antiviral treatment highlights a significant gap in chronic Hepatitis B management in Indonesia. These results emphasize the importance of early viral treatment and rigorous surveillance

Keyword(s) : hepatitis B, hepatocellular carcinoma, Indonesia

2718 - Interpretable Deep Learning model to Predict Multi Outcome Trajectories of MASLD Using Whole Slide Images and Time Series Data A Retrospective Multicentre Study

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a systemic condition linked not only to liver complications, such as cirrhosis and hepatocellular carcinoma, but also to cardiovascular, metabolic, and endocrine disorders. Current non-invasive tests provide indirect, threshold-based, and static risk stratification, limiting continuous and

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personalized prognosis. Existing models typically focus on single outcomes and rely on cross-sectional data, failing to capture longitudinal lifestyle effects. We hypothesized that integrating digital pathology from liver biopsy whole-slide images (WSIs), peri-biopsy laboratory data, and longitudinal lifestyle time-series information into an interpretable multimodal deep learning framework would enable dynamic, individualized prediction of multisystem MASLD-related outcomes and provide mechanistic insights for targeted prevention.

Materials and Methods:

This retrospective multicentre study included biopsy-confirmed MASLD patients (2005–2010) from nine Chinese centres. After exclusions, 814 patients were analyzed (546 training; 268 external validation), with a median follow-up of 203 months. Paired H&E and Sirius Red (SR) WSIs were processed using the Prov-GigaPath foundation model. Slide-level features were fused via cross-attention. Laboratory variables were normalized and embedded using a shallow Transformer. Annual lifestyle data were modeled using a Transformer-LSTM time-series module. The final trimodal framework (Prov-GigaPath-DCL) predicted 13 multisystem endpoints. Model performance was evaluated using AUROC, AUPRC, and time-dependent AUROC. Interpretability was achieved via saliency heatmaps, tile-level self-supervised clustering (24 histomorphological phenotype clusters, HPCs), and cell segmentation/classification using Hover-Net.

Results:

Dual-stain pathology (H&E+SR) improved prediction across 13 outcomes compared with H&E alone (Δ AUROC 0.013–0.144). Adding laboratory data further enhanced discrimination (Δ AUROC 0.103–0.167 for selected endpoints).

The trimodal model incorporating lifestyle dynamics achieved the best performance, with time-dependent AUROCs ranging from 0.784 to 0.853, and AUROC >0.8 for several outcomes including coronary artery disease, chronic kidney disease, arrhythmias, heart failure, insulin-treated T2DM, and hypothyroidism. Dynamic risk trajectories were sensitive to behavioral changes, including changes in exercise and smoking status. Interpretability analyses showed outcome-specific saliency regions. HPC clustering identified fibrosis-dominant clusters associated with cirrhosis and hepatocellular carcinoma, and inflammation-dominant clusters linked to cardiovascular events. Cell-level analysis confirmed higher fibroblast proportions in

fibrosis-associated clusters and inflammatory cells in vascular-event clusters.

Conclusions:

The study presents the first interpretable trimodal deep learning framework for dynamic, multi-outcome prediction in MASLD. By integrating digital pathology, laboratory data, and longitudinal lifestyle factors, the model enables individualized, time-updated risk stratification and mechanistic insight. This approach aligns with guideline-based biopsy practice and supports personalized secondary prevention in high-risk MASLD patients.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease; deep learning; whole-slide images; multisystem outcomes; time-series data; interpretable; prediction

2719 - Periportal Arginine Deficiency Drives Macrophage Senescence to Blunt Aged Liver Regeneration

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Background / Hypothesis:

Liver regeneration is a vital reparative process following injury. While it is well-established that aging significantly compromises liver regeneration, yet the specific metabolic-immune crosstalk driving this impairment remains elusive. Macrophages are crucial non-parenchymal cells within the liver that play a pivotal role in regulating regeneration. This study aims to investigate the metabolic alterations in the aged liver during regeneration and determine how these changes impede the regenerative process via macrophages.

Materials and Methods:

We established a model of regeneration after 70% partial hepatectomy (PHx) using aged (100-week-old) and young (8-week-old) mice. Single-cell transcriptomics were employed to analyze alterations in hepatocytes and macrophages during regeneration. Spatial transcriptomics and spatial metabolomics were utilized to further characterize zonal variations in arginine metabolism within the aged liver. In vivo and in vitro experiments were conducted to assess the impact of arginine deficiency on macrophages senescence and liver regeneration.

Results:



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Our analysis revealed significant abnormalities in hepatic arginine metabolism in aged mice during the regenerative phase. Spatial multi-omics data confirmed that key enzymes in the arginine metabolism, specifically CPS1 and ASS1, were significantly downregulated in hepatocytes within the periportal (PV) zone of aged mice. Consequently, the abundance of multiple metabolites involved in arginine metabolism—such as citrulline and argininosuccinate—was markedly reduced in the PV zone of the aged group compared to young controls. This metabolic deficiency significantly induced the migration and senescence of CD274+ monocyte-derived macrophages, creating a microenvironment unfavorable for regeneration. Supplementation with arginine or its precursors reversed these alterations and improved liver regeneration in aged mice.

Conclusions:

Aberrant arginine metabolism in the PV zone of the regenerating aged liver induces the migration and senescence of CD274+ macrophages, leading to impaired liver regeneration. Strategies involving the exogenous supplementation of arginine and its precursors can alleviate macrophage senescence and restore regeneration in the aged liver.

Keyword(s) : Aging, Arginine metabolism, Liver zonation, Liver regeneration

2720 - PRE EXISTING ANEMIA INVERSELY RELATED TO RIBAVIRIN INDUCED ANEMIA IN CIRRHOTIC PATIENTS WITH VIRAL HEPATITIS C INFECTION

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Background: Sofosbuvir/velpatasvir (SOF/VEL) plus ribavirin (RBV) is the current treatment regimen for cirrhotic patients with hepatitis C infection (HCV) in Thailand. Several studies have shown that ribavirin can induce anemia, leading to admissions, blood transfusions, and dose reductions. Our study was therefore designed to systematically determine the prevalence and clinical predictors of ribavirin-induced anemia (RIA) and evaluate its impact on treatment outcomes in this cirrhotic population.

Methods: This retrospective study was conducted at a tertiary care hospital in Thailand. All patients over 18 years with HCV cirrhosis treated with SOF/VEL plus RBV between January 2021 and October 2025 were included. Patients with concomitant hemologic

diseases, renal failure, malignancy, presence of other causes of anemia were excluded. We collected clinical, laboratory, and outcomes data, with a focus on RIA prevalence, the need for treatment modification, and sustained virological response at 12 weeks (SVR12). Results: A total of 76 patients were included for analysis. Of these, 61.8% were male, with a median age of 58. 85.5% had a Child-Pugh score of A and a median Model for End-Stage Liver Disease (MELD) score of 9. The prevalence of RIA, defined as new-onset anemia after treatment initiation, was high at 61.8%. Hemoglobin (Hb) level in the RIA group was significantly lower than the non-RIA group at the 12th week of treatment ($p=0.039$). However, on the 12th week post-treatment, Hb levels were comparable ($p=0.827$). In the RIA group, almost one half (46.8%) developed at least a moderate degree of anemia, 12.8% had severe to life-threatening anemia, over one fifth (21.3%) needed blood transfusions, and 4.3% needed RBV dose reductions. Of note, RBV was halted in 2.1% of the RIA group. Surprisingly, multivariable logistic regression analysis demonstrated that patients who had pre-existing anemia by World Health Organization (WHO) definition were independently protected against developing progressive anemia during RBV treatment (OR 0.19, 95%CI 0.06-0.65; $p=0.008$). Nevertheless, the rates of SVR12 post-treatment were comparable between the 2 groups ($p=0.555$).

Conclusions: In cirrhotic HCV patients, RIA is common, with a prevalence of over 60%, and necessitates intervention, including blood transfusion, dose reduction, or even withdrawal of RBV treatment. However, this effect vanished at post-treatment 12 weeks. Importantly, the presence of pre-treatment anemia was inversely associated with progressive anemia during therapy. The comparable SVR12 rates suggested that RIA, when managed, didn't compromise virologic response. This finding supports the continued use of RBV in this high-risk population, but larger studies are warranted.

Keyword(s) : Anemia, Cirrhosis, Hepatitis C, Ribavirin

2721 - Prevalence Characteristics and Prognostic Value of Pituitary Target Gland Axis Dysfunction in Decompensated Cirrhosis A Prospective Cohort Study

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Background / Hypothesis: Decompensated cirrhosis is frequently complicated by various endocrine disturbances. While existing studies have largely focused on functional alterations of single peripheral glands, the systemic dysfunction of the pituitary-target gland axes, particularly as a distinct syndrome in decompensated patients, remains insufficiently characterized. The prevalence, risk factors, and prognostic value of this multi-axial dysfunction in decompensated cirrhosis have not yet been systematically elucidated. We hypothesize that multi-axial pituitary-target gland axis dysfunction is prevalent in patients with decompensated cirrhosis and is closely associated with liver disease severity, malnutrition, and systemic inflammation. We further postulate that this systemic neuroendocrine dysfunction is independently associated with an increased incidence of hepatic encephalopathy, infection, and mortality within one year.

Materials and Methods: This prospective cohort study enrolled 188 patients with decompensated cirrhosis. Pituitary-target gland axis hormones (including IGF-1, thyroid, adrenal, and gonadal axes), pituitary MRI, and nutritional status were assessed. Dysfunction was defined as hormone levels in ≥ 1 axis below the reference range, acknowledging functional alterations like low IGF-1. Patients were followed for 12 months for mortality, hepatic encephalopathy (HE), and infection.

Results: A total of 188 patients were included. The overall prevalence of pituitary-target gland axis dysfunction was 29.8% (56/188), with gonadal axis dysfunction (22.3%) and low IGF-1 levels (19.7%) being the most frequent abnormalities. Multivariate Logistic regression identified Child-Pugh class C, MELD-Na score > 20 , malnutrition (NRS-2002 ≥ 3), and elevated CRP (> 10 mg/L) as independent risk factors (all $P < 0.01$). Abnormal pituitary MRI findings were detected in 24.5% of patients and were positively correlated with the number of affected axes. During the 12-month follow-up, the dysfunction group exhibited significantly higher rates of all-cause mortality, hepatic encephalopathy, and bacterial infection compared to the normal function group ($P < 0.01$). After adjusting for potential confounders, pituitary-target gland axis dysfunction remained an independent predictor of 1-year all-cause mortality (HR = 2.43).

Conclusions: Decompensated cirrhosis is frequently associated with pituitary-target gland axis dysfunction,

primarily involving gonadal and GH-IGF-1 axes. This represents a systemic "Hepato-Neuroendocrine Failure Syndrome" linked to liver severity and inflammation rather than isolated pituitary pathology. It serves as a robust, independent prognostic marker surpassing traditional scoring systems alone.

Keyword(s) : Liver cirrhosis; Pituitary-target gland axis; Endocrine dysfunction; IGF-1; Prognosis

2722 - The predictive value of serum ferritin for Biochemical Non resolution (BNR) in DILI

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Background / Hypothesis:

Drug-induced liver injury (DILI) is a leading cause of acute and chronic liver disorders worldwide. In China, the widespread use of herbal medicines, dietary supplements, and anti-tuberculosis drugs contributes to a high DILI burden—23.80 per 100,000 annually, exceeding rates in Iceland (19.1), France (13.9), and the USA (2.7 per 100,000). Most patients recover after drug withdrawal, yet a growing subset develops chronic DILI with persistent inflammation, bile duct injury, or cirrhosis. Biochemical non-resolution (BNR)—persistent liver biochemistry abnormalities for ≥ 6 months—remains poorly understood. We evaluated serum ferritin (FER) as a predictor of BNR and its relationship with biochemical recovery time.

Materials and Methods:

We retrospectively analyzed 226 DILI patients diagnosed by clinical and pathological criteria at Tianjin Second People's Hospital (2012–2022). Patients were stratified by 6-month outcomes: biochemical resolution (BR) versus BNR. Normally distributed data were compared using t-tests or ANOVA; non-normal data used Mann-Whitney U or Kruskal-Wallis tests. Categorical variables were analyzed by chi-square test. Univariate and multivariate Cox regression assessed FER's impact on biochemical resolution time. X-tile determined optimal FER cutoffs for BNR prediction. Biochemical non-resolution rates across FER strata were plotted using GraphPad Prism 8.

Results:

FER levels distinguished BNR from BR patients ($p < 0.032$) and correlated with AST, ALT, GGT, and TBil (all $p < 0.05$). On univariate Cox regression, FER

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predicted biochemical resolution time (HR: 0.999, 95% CI: 0.999–1.000, $p=0.011$). This held across four multivariate models with progressive covariate adjustment ($p=0.036$, 0.016, 0.04, and 0.001). X-tile identified optimal thresholds for low-, medium-, and high-FER groups. Inter-group differences were significant for BNR rate ($p=0.048$), recovery days ($p=0.000$), hospitalization days ($p=0.002$), age, ALT, AST, TBil, and DBil (all $p\leq 0.001$). A clear gradient emerged: low-FER patients recovered fastest, followed by medium- and high-FER groups.

Conclusions:

Serum ferritin independently associates with biochemical non-resolution in DILI. Higher FER levels predict delayed biochemical recovery. FER may serve as a practical prognostic marker for identifying DILI patients at risk of chronic progression.

Keyword(s) : Drug-Induced Liver

Injury(DILI)Biochemical Non-resolution(BNR) serum ferritin(FER) iron metabolism risk factors

2723 - The Impact of Metabolic Dysfunction Associated Steatotic Liver Disease on Liver Stiffness in Chronic Hepatitis B Patients A Cohort Study

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Background / Hypothesis:With the rising prevalence of metabolic dysfunction-associated steatotic liver disease (MASLD), the proportion of chronic hepatitis B (CHB) patients with MASLD is increasing annually. MASLD may accelerate the progression of liver fibrosis in CHB patients, potentially through mechanisms involving metabolic abnormalities (such as insulin resistance and fat deposition) and chronic inflammation. Therefore, this study aims to investigate the effect of MASLD on liver stiffness measurement (LSM) changes in CHB patients.

Materials and Methods:We conducted a single-center retrospective cohort study of CHB patients with at least two LSM with FibroScan at the Second Affiliated Hospital of Nanchang University. We studied the absolute change in LSM and the change in LSM stage from the first LSM to the most recent LSM among CHB patients with MASLD compared to patients without MASLD.

Results:A total of 2498 patients were included (437

with CHB + MASLD and 2061 with pure CHB). The median follow-up was 1.9 years. Compared to patients without MASLD, those with MASLD had significantly higher LSM values at both the first and last measurements (5.9 vs. 5.6 kPa and 5.9 vs. 5.4 kPa, $p < 0.001$, respectively), a lower likelihood of achieving a decrease in LSM stage (42.2% vs. 55.3%, $P < 0.001$), and a higher likelihood of experiencing an increase in LSM stage (21.8% vs. 17.4%, $P = 0.049$). After propensity score matching, the results remained consistent. Among the 625 patients (25.0%) receiving antiviral therapy (AVT), AVT was associated with a decline in LSM ($p < 0.001$). However, patients with MASLD showed similar LSM stage reductions (55.0% vs. 56.0%, $P = 1.000$) and progression rates (15.4% vs. 15.9%, $P = 1.000$) after AVT.

Conclusions:In untreated CHB patients, the presence of MASLD is independently associated with higher LSM values, and MASLD patients are more likely to experience an increase in LSM over time. After the initiation of AVT, LSM decline is less pronounced in CHB patients with MASLD. Future studies could explore the relationship between different metabolic risk factors and liver stiffness changes, particularly with the support of multicenter studies and long-term follow-up data, to better define optimal individualized treatment strategies.

Keyword(s) : Chronic Hepatitis B, Metabolic

Dysfunction-Associated Steatotic Liver Disease, Liver Stiffness, Antiviral Therapy

2724 - Silybin Capsules Protected Hepatocytes by Regulating Mitochondrial Homeostasis in the Organoid Model of Metabolic Dysfunction Associated Steatotic Liver Disease

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Background / Hypothesis: Mitochondrial homeostasis imbalance is a critical molecular mechanism underlying the progression of metabolic dysfunction-associated steatotic liver disease (MASLD). Silybin capsules, a well-known hepatoprotective agent, have been extensively studied for its mechanisms in ameliorating lipid metabolism disorders and exerting antioxidant effects. However, its impact on mitochondrial function remains unexplored. The present study was aimed to investigate the regulation of silybin capsules on mitochondrial homeostasis in MASLD.

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Materials and Methods: An organoid model known as tissue-engineered fatty liver (TEF) was utilized. Triglycerides (TG), reactive oxygen species (ROS), aspartate aminotransferase (AST) were measured using commercial kits. Relative gene detected via qPCR. Mitochondrial membrane potential (MMP) was detected by JC-1 fluorescence-labeled microprobe.

Results: Compared to TEF liver, silybin capsules treatment resulted in 35% reduction of intracellular TG, and a 54% decrease in supernatant AST levels. MMP in TEF liver was 30% lower than normal controls, silybin capsules can significantly restored MMP to near-normal levels, accompanied by a reduction in ROS. Furthermore, Silybin capsules upregulated the mRNA expression of peroxisome proliferator-activated receptor γ coactivator 1- α (PGC1 α) and mitochondrial transcription factor (mitochondrial biogenesis), mitofusin 1/2 (mitochondrial fusion) and PINK1/Parkin (mitophagy), while down-regulated dynamin-related protein 1 (mitochondrial fission).

Conclusions: Silybin capsules regulated mitochondrial homeostasis in organoid model of MASLD to reduce liver damage.

Keyword(s) : MASLD, Silybin Capsules, mitochondrial homeostasis, organoid model

2725 - Suppression of gene expression of cholesterol in liver by royal jelly

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Background :

Excess cholesterol produced in the liver poses significant health risks and is a major contributing factor to various diseases, particularly fatty liver disease. Although royal jelly (RJ) has been shown to be effective in mitigating atherosclerosis in both human and animal models, the precise molecular mechanism by which RJ suppresses cholesterol-related gene expression remains unclear. This study hypothesizes that RJ downregulates the expression of squalene epoxidase (SE), a key enzyme in the cholesterol biosynthesis pathway, thereby reducing hepatic cholesterol production.

Materials and Methods :

HepG2 cells were treated with royal jelly, and changes in the gene expression of squalene epoxidase (SE) were evaluated. Gene expression levels were measured using real-time PCR and normalized to glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as an internal control. To investigate the source of RJ's bioactivity, RJ samples from different geographical origins were compared. Furthermore, RJ was fractionated by ultrafiltration into molecular weight components, and the < 10 kDa fraction was examined for its suppressive effects, with additional separation of water-soluble and insoluble components.

Results :

Royal jelly treatment significantly decreased SE gene expression in HepG2 cells. The suppressive effect varied depending on the country of origin of the RJ. Fractionation analysis revealed that the < 10 kDa molecular weight fraction retained similar inhibitory activity on SE expression, with the water-soluble portion of this fraction showing the most pronounced effect.

Conclusions :

Our findings suggest that royal jelly suppresses hepatic cholesterol synthesis by downregulating squalene epoxidase gene expression. The bioactive components responsible for this effect are likely small, water-soluble molecules with a molecular weight of less than 10 kDa. These results provide new insights into the cholesterol-lowering mechanisms of royal jelly and highlight its potential as a functional food for managing cholesterol-related disorders.

Keyword(s) : Suppress cholesterol, Royal jelly, RT-PCR, active ingredient

2726 - Endothelial peroxisome proliferator activated receptor α prevents acetaminophen hepatotoxicity via maintenance of endothelial junctional integrity

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Background / Hypothesis: Microvascular injury represents an early event in acetaminophen (APAP)-

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induced acute liver injury (ALI), yet its underlying molecular mechanisms remain incompletely understood. Peroxisome proliferator-activated receptor α (PPAR α), a ligand-activated transcription factor highly expressed in the liver, plays significant roles in various liver diseases. However, the specific function of PPAR α in liver sinusoidal endothelial cells (LSECs) during ALI is unknown.

Materials and Methods: ALI was induced in endothelial cell-specific Ppara-knockout (Ppara Δ EC) and control mice by APAP (400 mg/kg). Liver injury was assessed histologically and biochemically. Sinusoidal barrier integrity was evaluated by scanning electron microscopy, Western blotting, and vascular permeability assays. Primary liver sinusoidal endothelial cells were subjected to RNA sequencing, and PPAR α binding to the CDH5 promoter was examined by chromatin immunoprecipitation.

Results: APAP challenge markedly reduced PPAR α expression in liver sinusoidal endothelial cells. Endothelial Ppara deficiency exacerbated liver necrosis, serum transaminase elevation, and sinusoidal hyperpermeability. Scanning electron microscopy revealed widened inter-endothelial gaps in Ppara Δ EC mice, indicating severe junctional disruption. RNA sequencing of primary endothelial cells showed that Ppara loss amplified APAP-induced transcriptional repression of adherens junction components, particularly CDH5 (encoding VE-cadherin). Quantitative PCR and Western blot confirmed pronounced downregulation of VE-cadherin in Ppara Δ EC livers. Mechanistically, chromatin immunoprecipitation identified a functional peroxisome proliferator response element in the CDH5 promoter, to which PPAR α directly bound. Reporter assays further demonstrated that PPAR α activation transactivated CDH5 promoter activity in a ligand-responsive manner. **Conclusions:** Endothelial-specific Ppara deficiency exacerbates APAP-induced microvascular injury by disrupting endothelial junctions, in part through the impaired transcriptional activation of CDH5.

Keyword(s) : Drug-induced liver injury, Liver sinusoidal endothelial cells, Peroxisome proliferator-activated receptor α

2727 - HBV enforces interferon refractoriness by hijacking LUBAC mediated IRF9 linear ubiquitination

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Background / Hypothesis:

Pegylated interferon- α (Peg-IFN α) therapy for chronic hepatitis B achieves hepatitis B surface antigen (HBsAg) loss in only a minority of patients, and the hepatocyte-intrinsic mechanisms limiting its antiviral efficacy remain incompletely understood. This study aimed to define how linear ubiquitin chain assembly complex (LUBAC) regulates interferon signaling and antiviral efficacy.

Materials and Methods:

We integrated analyses of liver biopsies from Peg-IFN α -treated patients with mechanistic studies in HBV-replicating hepatocytes and HBV mouse models. Hepatocyte-intrinsic interferon signaling and LUBAC function were interrogated using genetic perturbations, ubiquitination and protein interaction assays, together with structure-guided pharmacological targeting of the HOIL-1 C460 site.

Results:

Elevated accumulation of linear (M1-linked) ubiquitination was observed in the livers of Peg-IFN α non-complete responders compared with complete responders. Hepatic specifically inhibition of M1-linked ubiquitination, through HOIP knockdown or OUTLIN overexpression, synergistically enhanced IFN α antiviral efficacy and HBsAg loss in HBV mouse models. IFN- α transcriptionally upregulated the LUBAC subunits HOIP and HOIL-1 via STAT1- and IRF9/STAT2-dependent mechanisms, respectively, resulting in increased M1-linked ubiquitination. Mechanistically, HOIP interacted with IRF9, and selectively catalyzed its M1-linked ubiquitination at lysine 90, thereby impairing ISGF3 complex assembly and attenuating interferon-stimulated gene expression, establishing a negative feedback loop. The HBV regulatory protein HBx amplified this pathway by engaging the autoinhibitory cysteine 460 (C460) of HOIL-1, relieving autoinhibition and hyperactivating LUBAC to suppress interferon signaling. Translationally, structure-guided virtual screening and functional validation identified a small molecule, ITC, targeting the HOIL-1 C460 switch, which restored interferon signaling and synergized with IFN- α to markedly suppress serum HBsAg and intrahepatic HBcAg in vivo.

Conclusions:

These findings identify LUBAC-mediated linear ubiquitination of IRF9 as a hepatocyte-intrinsic checkpoint that constrains interferon signaling and limits Peg-IFN α antiviral efficacy. Targeting the HOIL-1-IRF9 axis represents a strategy to overcome

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interferon refractoriness in chronic hepatitis B.

Keyword(s) : Interferon refractoriness, HBV, LUBAC, IRF9, targeted therapy

2728 - Secretory ECM1a restores HBV specific CD8⁺ T Cell response via integrin β 1 blockade to promote HBsAg loss in chronic HBV infection

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Background / Hypothesis:

Antigen-specific CD8⁺ T cells are crucial effectors for hepatitis B virus (HBV) clearance but become functionally impaired in chronic infection. Extracellular matrix protein 1 (ECM1), a component of the extracellular matrix essential for hepatic environmental homeostasis, is involved in regulating the immune response. This study aims to investigate the effect of ECM1 on HBV clearance and its underlying mechanism in anti-HBV immune responses.

Materials and Methods:

Blood and liver samples were obtained from 147 chronic HBV-infected individuals and 76 healthy controls. Multi-omics approaches including single-cell RNA sequencing (scRNA-seq), co-immunoprecipitation (Co-IP), mass spectrometry, and in vitro stimulation assays were employed to explore the effect of ECM1 on anti-HBV CD8⁺ T cell response. HBV mouse model with Hepatic stellate cell (HSC)-specific ECM1 knockout were established for in vivo validation.

Results:

Screening identified six upregulated extracellular matrix components (MMP2/7, ECM1, VCAN, COL5A1/15A1) in HBV-infected livers from GEO database. Crucially, serum ECM1 was the only component correlating with disease status in our cohort, showing significantly higher levels in patients achieving HBsAg clearance versus those with persistent infection. The assays of clinical liver specimens and HBV mouse models revealed that the elevated ECM1 originates from hepatic stellate cells (HSCs).

Functionally, the secretory isoform ECM1a, but not the non-secretory isoform ECM1b, enhanced HBsAg-specific cytotoxic T cell (CTL) responses, leading to HBsAg clearance. Moreover, HSC-specific ECM1 knockdown delayed HBV clearance in mice.

Mechanistically, the P300-H314 domain unique to ECM1a (deficient in ECM1b) competitively bound integrin β 1, thereby neutralizing the suppression of

CD8⁺ T cell function of integrin β 1 and restoring anti-HBV immunity. Meanwhile, scRNA-seq revealed that the reduction of integrin β 1 on peripheral and intrahepatic CD8⁺ T cells from chronic HBV-infected patients correlated with functional cure and favorable response to pegylated interferon- α therapy. Consistent with this finding, pharmacological inhibition of integrin β 1 signaling with ATN-161 accelerated viral clearance in HBV mouse models.

Conclusions:

The secretory isoform ECM1a facilitates HBV clearance by antagonizing integrin β 1-mediated CTL exhaustion, promoting cytotoxic cytokine production and HBsAg loss. Our findings unveil a novel extracellular matrix-immunity crosstalk mechanism and identify ECM1 as a therapeutic target for chronic HBV infection.

Keyword(s) : HBsAg loss, HBV, ECM1, Antigen-specific CD8⁺ T cells, cytotoxic T cell

2730 - Association of monocyte to high density lipoprotein cholesterol ratio with liver histopathology in chronic hepatitis b

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Background / Hypothesis: Accurate assessment of liver fibrosis and histological activity is essential for clinical decision-making in chronic hepatitis B. Although liver biopsy remains the gold standard, its invasive nature has increased interest in non-invasive biomarkers. The monocyte-to-high-density lipoprotein cholesterol ratio (MHR) has been proposed as a simple marker of systemic inflammation; however, its relationship with liver histopathology in chronic hepatitis B remains unclear. This study aimed to evaluate the association between MHR and liver histopathological findings in patients with chronic hepatitis B.

Materials and Methods: This retrospective cross-sectional study included 148 patients with chronic hepatitis B who underwent liver biopsy between 2020 and 2025. Liver biopsy specimens were evaluated for fibrosis stage, histological activity index, and hepatic steatosis. Non-invasive fibrosis assessment was

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performed using the FIB-4 score. MHR was calculated from routine laboratory parameters. Associations between MHR, histopathological findings, FIB-4 score, and clinical-laboratory parameters were analyzed using correlation analyses, group comparisons, and receiver operating characteristic (ROC) analyses.

Results: No statistically significant association was observed between MHR and fibrosis stage, histological activity index, or hepatic steatosis. Although MHR values tended to be higher in patients with advanced fibrosis, this difference did not reach statistical significance. ROC analyses demonstrated limited performance of MHR in discriminating significant histological activity and advanced fibrosis. In contrast, MHR showed positive correlations with leukocyte and neutrophil counts, AST, ALT, GGT, triglyceride, and creatinine levels, and a negative correlation with total cholesterol. Higher MHR values were observed in patients with elevated HBV DNA levels.

Conclusions: MHR does not reliably reflect liver histopathology or fibrosis burden in patients with chronic hepatitis B. However, the findings suggest that MHR may primarily represent systemic inflammatory and metabolic status rather than liver-specific histological injury.

Keyword(s) : chronic hepatitis B, monocyte-to-HDL cholesterol ratio, liver biopsy, fibrosis, inflammation

2732 - Comparative evaluation of paper and digitized cognitive tests for diagnosis of hepatic encephalopathy in patients with liver cirrhosis

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Background / Hypothesis:

Minimal hepatic encephalopathy (MHE) is frequently underdiagnosed despite its impact on quality of life and risk of progression. Paper-based psychometric testing remains the reference standard, whereas digitized tools may offer practical advantages. We aimed to compare the diagnostic performance of paper-based and digitized cognitive tests in liver cirrhosis.

Materials and Methods:

This single-center study was conducted at the Institute of Gastroenterology, Hepatology and Metabolism, Interna Clinic. A total of 200 individuals were analyzed, including 100 patients with liver cirrhosis and 100 healthy controls. Hepatic encephalopathy was defined as any grade above absence according to clinical grading. Primary analysis included cirrhosis patients only (n=100; 82 with encephalopathy, 18 without). Secondary analysis focused on MHE (62 minimal/grade I vs 18 without encephalopathy; overt cases excluded). Paper-based testing included number connection tests A and B, digit symbol test, serial dotting test, and line tracing test. Digitized testing included the EncephalApp Stroop test, digitized number connection test A, and digitized digit symbol test. Multivariable logistic regression adjusted for age, sex, and education was applied. Model performance was evaluated using five-fold cross-validation with out-of-fold predictions. Diagnostic accuracy was assessed using area under the receiver operating characteristic curve, sensitivity, and specificity at optimal Youden thresholds.

Results:

Among individual paper-based tests, number connection test A demonstrated the highest diagnostic accuracy (area under curve 0.87), followed by number connection test B (0.86). Serial dotting and line tracing tests showed moderate accuracy (0.79–0.81), while the digit symbol test showed lower performance (0.76). Digitized tests showed lower discrimination. The EncephalApp Stroop test achieved area under curve 0.79 with relatively high sensitivity but reduced specificity. Digitized number connection test A and digitized digit symbol test demonstrated area under curve values of approximately 0.74 and 0.69, respectively. The combined paper-based battery demonstrated superior diagnostic performance (area under curve 0.90; sensitivity 84%; specificity 89%). The combined digitized battery showed lower accuracy (area under curve 0.79) and reduced sensitivity (~65%) despite comparable specificity.

Conclusions:

Paper-based psychometric testing provides superior diagnostic accuracy for MHE. Digitized cognitive tests demonstrate acceptable performance and may serve as screening tools; however, confirmatory assessment with validated psychometric batteries remains necessary.

Keyword(s) : hepatic encephalopathy, minimal hepatic encephalopathy, liver cirrhosis, cognitive assessment, psychometric testing, digitized testing



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2734 - A Multidimensional Prognostic Signature Integrating a 4 Gene Panel Immune Contexture and Radiomic Features Predicts Progression Free Survival in Hepatocellular Carcinoma

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Background / Hypothesis: Hepatocellular carcinoma (HCC) is characterized by high heterogeneity and frequent early progression, limiting the effectiveness of current prognostic tools. To address this, we developed a progression-free survival (PFS)-oriented, multidimensional model integrating genomic, immune, drug sensitivity, and radiomic features for improved risk stratification and biological insight.

Materials and Methods: RNA-seq and matched radiomic data of HCC were obtained from The Cancer Genome Atlas (TCGA)-Genomic Data Commons (GDC) and The Cancer Imaging Archive (TCIA) database. The co-expression modules were analyzed by Weighted Gene Co-expression Network Analysis (WGCNA), and the differentially expressed genes (DEGs) were identified by "DESeq2" R package. Next, a RiskScore was established by univariate Cox and LASSO-Cox regression. The prognostic differences were assessed by Kaplan-Meier (KM) survival analysis, and Receiver Operating Characteristic (ROC) curve was used to evaluate the predictive accuracy. Additional analyses included CIBERSORT-based immune infiltration profiling, Gene Set Enrichment Analysis (GSEA) for pathway enrichment, oncoPredict-based drug sensitivity estimation, and integration of radiomic features into a clinical nomogram.

Results: We identified a robust PFS-associated gene signature (IYD, ACADL, B3GNTL1, C1S) that effectively stratified HCC patients into high- and low-risk groups with significantly distinct progression-free survival. The high-risk phenotype was characterized by enrichment of cell cycle and proliferative pathways, a dysfunctional immune landscape (such as the elevated Tregs), and reduced sensitivity to multiple targeted agents (e.g., Ribociclib_1632, Dihydrorotone_1827). Furthermore, three radiomic features linked to tumor heterogeneity and boundary irregularity were integrated into a nomogram that demonstrated strong predictive

performance, excellent calibration, and superior clinical net benefit.

Conclusions: We developed a PFS-oriented, multidimensional prognostic model for HCC integrating transcriptomic, immune, drug sensitivity, and radiomic feature analysis. It effectively stratifies risk and reflects underlying biological mechanisms of aggressive disease, such as immunosuppression and potential therapeutic resistance, highlighting its utility for guiding future translational research.

Keyword(s) : Hepatocellular carcinoma; Progression-free survival; Radiomics; Tumor microenvironment; Drug sensitivity

2735 - Significance of fibrinogen D dimer albumin and NLR in the progression of Acute on Chronic pre liver failure

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Background / Hypothesis:

Acute-on-Chronic Liver failure is clinically characterized by coagulation disorders, jaundice, ascites, and hepatic encephalopathy, representing acute liver dysfunction that develops from chronic liver disease. Patients with Acute-on-Chronic liver failure often present with critical conditions, high mortality rates, and significant challenges in medical management. To investigate the clinical significance of fibrinogen, D-dimer, albumin, and NLR in the progression of Acute-on-Chronic pre-liver failure.

Materials and Methods:

A retrospective analysis was conducted on 239 patients with Acute-on-Chronic pre-liver failure (pre-failure group) and 36 patients with hepatic failure (hepatic failure group) and 28 patients with chronic hepatitis (chronic hepatitis group) admitted to the Ninth Hospital of Nanchang from January 2022 to June 2025. The clinical data, including coagulation parameters, were reviewed. The pre-hepatic failure group was further divided into a progressive subgroup and a non-progressive subgroup based on disease progression. Binary logistic regression analysis was used to screen independent risk factors and establish a predictive model.

Results:

Compared with the baseline clinical data of the three groups, only the indicators of TBiL, ALB, L%, INR, PT, FIB, as well as the incidence of hepatic encephalopathy and short-term prognosis showed statistically significant differences ($P < 0.05$), while other clinical

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data showed no statistically significant differences ($P>0.05$). Compared with the non-progressive subgroup, the progressive subgroup exhibited statistically significant differences only in the indicators of ALB, serum sodium, FIB, D-dimer, and incidence of hepatic encephalopathy, and short-term prognosis ($P<0.05$), while other data showed no statistically significant differences ($P>0.05$). Based on P-values and collinearity diagnostic analysis results, the relevant variables were included in the logistic multiple regression analysis, yielding the equation $\text{LogitP} = 1.022 - 0.523 \times \text{FIB (g/L)} + 3.587 \times \text{D-dimer (mg/L)} - 0.111 \times \text{ALB (g/L)} + 0.194 \times \text{NLR}$. Patients with pre-hepatic failure were divided into two groups based on the optimal cutoff value of LogitP : ≥ -0.915 and < -0.915 . Disease progression analysis revealed that the cumulative progression risk in the $\text{LogitP} \geq -0.915$ group was significantly higher than that in the $\text{LogitP} < -0.915$ group, with a statistically significant difference ($\chi^2 = 22.038$, $P = 0.000$).

Conclusions:

Fibrinogen, D-dimer, albumin, and NLR can predict disease progression in pre-hepatic failure and have certain clinical significance.

Keyword(s) : chronic hepatitis, pre-hepatic failure, D-dimer, Fibrinogen

2737 - Effect of peginterferon alfa 2b with or without nucleos(t)ide analogues on long term outcomes in chronic hepatitis B Preliminary 48 week results from the multicenter prospective VISION study in China

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Background / Hypothesis: Optimizing antiviral strategies and achieving hepatitis B surface antigen (HBsAg) loss remain major challenges in the management of chronic hepatitis B (CHB). This study aimed to evaluate the effects of 48-week peginterferon alfa-2b (Peg-IFN α -2b) therapy, with or without nucleos(t)ide analogues (NAs), in patients with CHB enrolled in the VISION study.

Materials and Methods: In May 2024, we launched the multicenter, prospective VISION study to assess the long-term effects of Peg-IFN α -2b with or without NAs in CHB patients. This 48-week analysis involved two treatment groups: Peg-IFN α -2b monotherapy and Peg-IFN α -2b in combination with NAs, with a primary focus on assessing effectiveness and safety. Baseline characteristics between the two groups were balanced using propensity score matching (PSM).

Results: A total of 1138 patients completed the 48-week follow-up. After PSM, 798 patients were included in the analysis, comprising 399 patients in the monotherapy group and 399 patients in the combination group. At baseline, there were no significant differences between the monotherapy and

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combination groups in the proportion of males (64.7% [258/399] vs. 64.2% [256/399], $p=0.882$), mean age (43.5 $\pm$ 9.6 vs. 43.4 $\pm$ 9.4 years, $p=0.958$), HBeAg positivity rate (19.8% [79/399] vs. 21.3% [85/399], $p=0.599$), HBsAg levels (2.2 $\pm$ 1.3 vs. 2.3 $\pm$ 1.2 log IU/mL, $p=0.226$), HBV DNA levels (1.9 $\pm$ 1.5 vs. 1.9 $\pm$ 1.6 log IU/mL, $p=0.636$), and ALT levels (34.1 $\pm$ 45.9 vs. 33.8 $\pm$ 31.0 U/L, $p=0.920$). At 48 weeks, HBsAg loss rates (30.1% [120/399] vs. 29.3% [117/399], $p=0.816$) and ALT levels (49.0 $\pm$ 87.3 vs. 46.0 $\pm$ 40.8 U/L, $p=0.587$) were comparable between the two groups, while HBV DNA levels were slightly higher in the monotherapy group (1.6 $\pm$ 1.4 vs. 1.2 $\pm$ 0.8 log IU/mL, $p=0.029$). Notably, subgroup analysis showed that among patients with baseline HBsAg levels of <100, 100–<500, 500–<1500, and $\geq$ 1500 IU/mL, HBsAg loss rates were 55.9% (81/145), 27.9% (24/86), 12.3% (8/65), and 6.8% (7/103) in the monotherapy group, and 56.3% (76/135), 27.8% (27/97), 10.0% (7/70), and 7.2% (7/97) in the combination group (all $p>0.05$). Both treatment groups were generally well tolerated, with no serious adverse events observed.

Conclusions: At 48 weeks, among patients with low baseline HBsAg and HBV DNA levels, Peg-IFN α -2b monotherapy and Peg-IFN α -2b plus NAs combination therapy demonstrated comparable HBsAg loss rates and ALT levels, with no evident short-term advantage of combination therapy. The long-term benefits of these regimens are currently under investigation.

Keyword(s) :

2738 - Causal Machine Learning–Guided Personalized Immunotherapy Strategies in Intrahepatic Cholangiocarcinoma

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Background / Hypothesis: Immunotherapy (IO-chemo) has become the first-line standard care for patients with advanced intrahepatic cholangiocarcinoma (iCCA), but the incremental benefit of adding IO varies greatly among individuals, making it urgent to develop systems for identifying high-benefit populations based on individualized treatment effect (ITE) estimation.

Materials and Methods: This multi-cohort study included patients with iCCA who underwent either IO-chemo or chemo. The discovery cohort (2018–2022) included patients from three centers, while an independent cohort (2017–2023) from seven other centers served for external validation. Target trial emulation was employed to obtain unbiased average treatment effect estimation. We developed a causal machine-learning (ML) model (CMICC) to estimate heterogeneous treatment effects for IO-chemo. Based on predicted ITE, patients were stratified into high-benefit, no-to-moderate-benefit, and negative-benefit groups for each treatment. Counterfactual analyses compared overall survival between model-guided treatment selection and treatment actually received. The Qini and TOC curves were used to evaluate model performance. Model interpretability was assessed using SHAP.

Results: A total of 1485 patients, 696 in the IO-chemo group and 789 in the chemo group, were included in the discovery cohort; 562 patients, 246 in the IO-chemo group and 316 in the chemo group, were included in the external validation cohort. Using a recursive feature addition approach, we selected 17 of

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55 multidimensional variables to build the CMICC model in the discovery cohort. In the high-benefit group, compared with chemo, the hazard ratio (HR) for IO-chemo was 0.39 (95% CI, 0.30, 0.52), $P < 0.001$, with mortality rates reduced by 24.1%, 31.2%, and 28.5% at 12, 24, and 36 months, respectively. In the no-to-moderate-benefit groups, IO-chemo did not significantly differ from chemotherapy (HR, 0.91; 95% CI, 0.70, 1.18; $P = 0.488$). In the negative-benefit groups, IO-chemo was associated with worse outcomes than chemo (HR, 1.91; 95% CI, 1.47, 2.48; $P < 0.001$), with 12-, 24-, and 36-month mortality increased by 29.9%, 11.0%, and 5.1%, respectively. Counterfactual analysis indicated that CMICC-guided treatment decisions could improve survival compared with actually received treatment (RecomIC-RecIC vs RecomIC-RecC: HR: 0.53 [95%CI, 0.43, 0.65], $P < 0.001$; RecomC-RecC vs RecomC-RecIC: HR: 0.62 [95%CI, 0.50, 0.77], $P < 0.001$). These results were independently confirmed in an external validation cohort.

Conclusions: This study demonstrated that the causal ML-based CMICC model estimated ITE in iCCA patients receiving IO-chemo, performing effective benefit stratification, and may improve survival through individualized, CMICC-guided treatment decisions.

Keyword(s) : intrahepatic cholangiocarcinoma, immunochemotherapy, causal inference, heterogeneous treatment effects, target trial emulation, precision medicine

2740 - Hepatocyte TrkB acts as a gatekeeper against MASH related liver fibrosis by suppressing the TGF β CCL2 axis and macrophage infiltration

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Background / Hypothesis:

Liver fibrosis is a critical pathological stage in chronic liver disease, driven by hepatic stellate cell (HSC) activation and immune cell recruitment. While our previous work showed that overexpressing tyrosine kinase receptor B (TrkB) in HSCs inhibits their activation and alleviates fibrosis, the functional role of TrkB in

hepatocytes—the main parenchymal cells responsible for liver homeostasis—remains poorly understood. Here, we investigate the mechanistic interplay between hepatocyte-specific TrkB signaling and liver fibrosis progression.

Materials and Methods:

We investigated the role of hepatocyte-specific TrkB signaling through integrated in vivo animal models and in vitro two- and three-dimensional culture systems.

Results:

Elevated TrkB expression in hepatocytes reduces pro-fibrotic and inflammatory cytokines, attenuates HSC activation via paracrine signaling, and impairs monocyte-derived macrophage (MoMF) recruitment. Mechanistically, TrkB modulates the TGF β /SMAD3 pathway by inhibiting p-SMAD3 nuclear translocation, thus suppressing the transcription of FOS. As a core component of the AP-1 transcription factor complex, FOS directly regulates CCL2, a pivotal chemokine for macrophage recruitment.

Conclusions:

These results establish TrkB as a key regulatory node in the TGF β /SMAD3/FOS/CCL2 signaling cascade, orchestrating macrophage-mediated fibrotic responses in the liver.

Keyword(s) : Liver fibrosis, TrkB, MoMFs recruitment, 3D model

2741 - Magnetic Resonance Cholangiopancreatography Based Radiologic Evaluation of Biliary Complications in Living Liver Donors A Single Center Imaging Study

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Background / Hypothesis: To characterize the imaging spectrum of biliary complications in living liver donors using magnetic resonance cholangiopancreatography (MRCP) and to examine the association between postoperative biliary complications and biliary anatomy.

Materials and Methods: In this retrospective single-center study conducted at a tertiary referral center,



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adult living liver donors who underwent transplantation between January 2017 and January 2025 were reviewed. Donors were followed clinically and with imaging according to institutional protocols, and MRCP was performed when biliary complications were suspected. Preoperative MRCP examinations were analyzed to classify biliary anatomy. Imaging characteristics of biliary complications were investigated, and biliary anatomy was compared between donors with and without complications. Numerical variables were analyzed using the independent samples t-test or Mann–Whitney U test according to data distribution, whereas categorical variables were compared using the chi-square test or Fisher's exact test. A p value <0.05 was considered statistically significant. Donors who underwent interventional procedures were reported descriptively.

Results: A total of 181 living liver donors were included, comprising 77 females (42.5%) and 104 males (57.5%) with a mean age of 32.99 ± 8.82 years; right-lobe grafts were performed in 124 donors (68.5%) and left-lobe grafts in 57 donors (31.5%). Radiologic biliary complications were detected in 16 donors (8.8%). Early bile leakage and biloma represented the most common imaging phenotype (68.8%), while biliary strictures were observed less frequently, emerging within the first postoperative month in three donors and at 5.5 and 8 months in two donors. Although variant biliary anatomy was numerically more prevalent among donors with complications, it was not significantly associated with complication development ($p=0.282$). Hepatocyte-specific contrast-enhanced magnetic resonance imaging enabled functional confirmation of active bile leakage in selected cases. Additionally, three donors showed biochemical cholestasis despite normal MRCP findings, underscoring the value of imaging in preventing unnecessary invasive interventions.

Conclusions: Biliary anatomy variants were not associated with biliary complications, and MRCP provides a comprehensive, noninvasive tool for detecting biliary complications in living liver donors, characterized by bile leak/biloma predominance and a smaller burden of biliary strictures.

Keyword(s) : Living donor liver transplantation, Magnetic resonance cholangiopancreatography (MRCP), Biliary complicationS, Bile leak, Biloma, Biliary stricture

2743 - Immune Signatures Across Chronic Hepatitis B Phases Serum IFN γ and CD8⁺ T Cell Counts in Untreated Patients at Dr. Mohammad Hoesin Hospital Palembang Indonesia

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Background / Hypothesis:

Chronic hepatitis B virus (HBV) infection evolves through four immunologically defined phases, reflecting dynamic host-virus interactions. Innate and adaptive immune responses, particularly interferon-gamma (IFN- γ) and CD8⁺ T cells, are central to viral control and may help differentiate disease phases in untreated patients. This study investigated the association of serum IFN- γ levels and CD8⁺ T-cell counts across the phases of chronic HBV infection.

Materials and Methods:

We conducted an analytical observational study with a comparative design at Dr. Mohammad Hoesin Hospital, Palembang, Indonesia, from August to November 2024. Eligible participants were chronic HBV patients who had never received antiviral therapy and fulfilled predefined inclusion and exclusion criteria. Serum IFN- γ levels were quantified using enzyme-linked immunosorbent assay (ELISA), and peripheral blood CD8⁺ T-cell counts were measured by flow cytometry. Phase classification followed standard chronic HBV phase criteria documented in medical records and laboratory profiles. Statistical analyses were performed using SPSS version 29. Interphase comparisons were assessed using one-way ANOVA, with $p < 0.05$ considered statistically significant.

Results:

Thirty-eight untreated chronic HBV patients were included. Mean serum IFN- γ levels differed significantly across phases ($p < 0.05$), peaking in the reactivation phase (0.98 ± 0.35 pg/mL) and reaching the lowest values in the inactive carrier phase (0.40 ± 0.30 pg/mL). CD8⁺ T-cell counts also varied significantly between phases ($p < 0.05$), with the highest mean count observed in the inactive carrier phase (710.22 ± 196.08 cells/ μ L) and the lowest in the immune-active phase (415.50 ± 304.02 cells/ μ L). Together, these findings indicate distinct phase-related immune patterns

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involving both cytokine signaling and cytotoxic T-cell compartments.

Conclusions:

Serum IFN- γ levels and CD8⁺ T-cell counts show significant phase-associated differences in untreated chronic HBV infection, supporting their potential utility as immunological indicators of disease phase in clinical settings.

Keyword(s) : CD8⁺ T lymphocytes, chronic hepatitis B, disease phase classification, flow cytometry, interferon-gamma,

2744 - Paracaval Triangle A Novel Anatomic Landmark for Central Hepatectomy

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Background / Hypothesis: Central hepatectomies (resection of Segments 4, 5, and 8) are technically challenging due to the lack of clear standardized anatomic landmarks for defining the transection plane. This study aimed to describe a newly identified anatomic structure, the "paracaval triangle," and validate its value as a reliable landmark to standardize central hepatectomies.

Materials and Methods: The anatomical characteristics of the transected plane after central hepatectomy were systematically analyzed. The paracaval triangle was defined by three constituent points, and the spatial relationship with adjacent structures was clarified. A robotic-assisted central hepatectomy case is presented to demonstrate the application of this landmark.

Results: The transected plane after central hepatectomy consists of three planes: the left plane, the basal plane (paracaval triangle), and the right plane. The left plane is superficially demarcated by the falciform ligament and the intraparenchymal umbilical fissure vein when present. Using the paracaval triangle as a landmark, a robotic-assisted central hepatectomy was successfully performed with an operative time of 185 min, blood loss of 80 mL, and no postoperative complications. The patient was discharged on postoperative day 6, and no recurrence was observed during follow-up.

Conclusions: The paracaval triangle is a stable and reliable anatomic landmark for central hepatectomy. Identification of the paracaval triangle facilitates accurate localization of the transection plane, standardizes the surgical workflow, and improves the safety and efficacy of central hepatectomies, especially in robotic-assisted minimally invasive surgery.

Keyword(s) : Paracaval triangle; Central hepatectomy; Anatomical landmark; Robotic surgery; Hepatobiliary surgery

2745 - Laparoscopic Anatomical liver resection using ICG positive staining combined infrared laser guided

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Background / Hypothesis: Precise liver puncture and staining under ultrasound guidance during laparoscopic liver surgery remains a technical challenge. Currently, the success rate of puncture is less than 60%, and the location of the puncture point is usually difficult to accurately determine, as is the depth of puncture. There is a lack of effective methods for precise puncture.

Materials and Methods: We describe laparoscopic anatomical resection of the ICG-positive Portal Territory in a patient with primary hepatocellular carcinoma using laparoscopic infrared laser-guided targeted portal vein puncture.

Results: The operation time was 130 min, blood loss was 50 mL, and there were no complications. The patient was discharged 7 days after the operation. There were no complications during the 1-month follow-up.



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Conclusions: Infrared laser-guided technology can achieve precise ICG-positive puncture and accurately control the puncture position and depth. Combined with ICG-positive staining, this technology can precisely locate and significantly increase the success rate of puncture.

Keyword(s) : Infrared laser guidance; Laparoscopic ultrasound; Anatomical liver resection; ICG positive staining

2746 - Sofosbuvir Velpatasvir Voxilaprevir in Chronic Hepatitis C A Systematic Review and Meta Analysis

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Background / Hypothesis: Treatment failure occurs in a subset of chronic hepatitis C (HCV) patients, particularly those previously exposed to NS5A inhibitors, as resistance-associated substitutions (RASs) often persist and limit retreatment options. Voxilaprevir, a pangenotypic NS3–NS4A protease inhibitor, when combined with sofosbuvir/velpatasvir, provides a potent triple regimen (SOF/VEL/VOX). To better define the efficacy and safety of this regimen, we conducted a systematic review and meta-analysis of available studies.

Materials and Methods: A comprehensive literature search was conducted across PubMed, Google Scholar, DOAJ, and the Cochrane Library to identify studies evaluating the efficacy and safety of SOF/VEL/VOX in chronic HCV infection. The primary outcome was the proportion of patients achieving sustained virologic response (SVR), while secondary outcomes included reported adverse events. A random-effects model was employed to calculate the Risk Ratio (RR) with 95% Confidence Intervals (CI). Meta-analysis was performed using Review Manager version 5.4.1.

Results: A total of 19 RCTs and observational studies comprising 3339 patients were included. The pooled SVR rate was 95%. SVR rates were 94% in patients with comorbidities and 95% in those without comorbidities. Among cirrhotic patients, SVR was 93%, including 92% in those with comorbidities, compared with 97% in

non-cirrhotic patients. SVR rates were significantly lower in cirrhotics compared with non-cirrhotics (RR = 0.96; 95% CI = 0.93–0.99; p = 0.004). SVR rates were 96% for genotype 1 and 2, 92% for genotype 3, 93% for genotype 4, 95% for genotype 5, and 100% for genotype 6. Pairwise comparisons across all genotypes demonstrated that genotype 3 was associated with significantly lower SVR rates compared with other genotypes (RR = 0.95, 95% CI 0.94–0.97, p < 0.001), whereas genotype 6 achieved considerably higher SVR rates (RR = 1.05; 95% CI = 1.03–1.07; p < 0.001). Overall, adverse events occurred in 67% of patients across all studies, most commonly headache (26%), fatigue (22%), and diarrhea (18%), while abdominal pain was reported in 4%. Laboratory-related adverse events were uncommon, with hemoglobin <10 g/dl in 3%, thrombocytopenia <50,000/mm³ in 1%, and hyperglycemia >250 mg/dl in 1%.

Conclusions: Despite its high efficacy as a retreatment option for chronic HCV, SOF/VEL/VOX is still expensive in LMICs. To increase accessibility and cost-effectiveness in these contexts, tactics like generic production, price negotiations, and participation in public health initiatives are crucial.

Keyword(s) : Hepatitis C Virus, Sofosbuvir/Velpatasvir/Voxilaprevir, Sustained Virological Response, Liver

2747 - Hepatic Artery Pseudoaneurysm Presenting Overt Obscure Gastrointestinal Bleeding

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Hepatic artery pseudoaneurysm (HAP) is a rare, frequently overlooked but life-threatening cause of gastrointestinal (GI) bleeding. It is associated with hepatobiliary interventions, abdominal trauma, inflammation, or infection. Because of its rarity and variable presentation, diagnosis is often delayed, especially when initial endoscopic findings are inconclusive. We present a case of a right hepatic artery pseudoaneurysm manifesting as recurrent overt GI bleeding in a patient with prior penetrating abdominal trauma.

A 55-year-old male with a history of penetrating abdominal injury five months prior which managed

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with exploratory laparotomy revealing no significant intra-abdominal pathology, presented with a two-month history of intermittent blood-streaked stools. On the day of admission, he experienced hematochezia accompanied by vomiting, epigastric pain, and generalized weakness. On evaluation, he was found to be anemic and was admitted for further workup. Initial esophagogastroduodenoscopy (EGD) demonstrated healed gastric ulcers, while colonoscopy revealed large mixed hemorrhoids. He was discharged stable after supportive management with no immediate recurrence of bleeding.

The patient subsequently returned with persistent epigastric pain and recurrent hematochezia. Repeat colonoscopy with push ileoscopy demonstrated terminal ileal ulcers and hookworm infestation, for which antihelminthic therapy was initiated. Despite treatment, intermittent hematochezia persisted which admitted and progressively increased in volume. A repeat EGD was performed to reassess for a brisk upper GI source. Although no active ulcers or mass lesions were identified, blood clots were noted in the duodenum. Careful examination revealed blood-tinged bile emanating from the papilla.

Contrast-enhanced computed tomography angiography of the abdomen confirmed the presence of a saccular aneurysm of anterior segmental branch of the right hepatic artery. The patient underwent celiacomesenteric angiography, which confirmed the pseudoaneurysm, followed by successful coil embolization. Immediate cessation of bleeding was achieved, and no further episodes of hematochezia were observed during hospitalization and follow-up. This case underscores the importance of maintaining a high index of suspicion for vascular etiologies in patients with recurrent GI bleeding, especially in the setting of prior abdominal trauma. Endovascular embolization remains safe and effective therapy for control of bleeding for these cases.

Keyword(s) : Hepatic Artery Pseudoaneurysm, GI bleeding

2749 - Beyond the Stomach Rapunzel Syndrome Presenting with Sepsis and Multi organ Failure

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Background / Hypothesis:

Bezoars are accumulations of undigested material in the gastrointestinal tract. They are classified according to their composition: phytobezoars (undigested fruits

and vegetables), trichobezoars (hair mixed with food particles), lactobezoars (milk curds), and pharmacobezoars (medications).

Trichobezoars form because human hair is resistant to digestion and, due to its smooth surface, is not effectively propelled by peristalsis. Progressive accumulation in the stomach leads to formation of a large mass that may extend beyond the pylorus.

This condition is commonly associated with Trichotillomania, a compulsive psychiatric disorder characterized by repetitive hair pulling, often followed by trichophagia (ingestion of hair). It is frequently associated with anxiety, depression, and poor self-image.

We report a case of gastric trichobezoar with life-threatening complications to highlight the importance of early recognition and multidisciplinary management.

Materials and Methods:

This is a single-patient case report of a 12-year-old female presenting to the emergency department in a critical condition.

Clinical evaluation included:

Detailed history from attendants

Physical examination

Laboratory investigations

Contrast-enhanced CT scan of the abdomen

Ascitic fluid analysis and culture

Upper gastrointestinal endoscopy

Results:

A 12-year-old girl presented in a lethargic and gasping state with a one-year history of hair ingestion and recent worsening abdominal pain, vomiting, and distention.

On examination:

Pale and cachectic appearance



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Bald patches over the scalp

Distended abdomen with a firm, non-tender epigastric mass

Laboratory findings revealed:

Severe hypoglycemia

Metabolic acidosis

Microcytic anemia

Leukocytosis

Hypokalemia

Severe hypoalbuminemia

CT imaging demonstrated a large heterogeneous gastric mass extending into the duodenum, consistent with trichobezoar, along with ascites and mediastinal shift. Ascitic fluid culture grew *Escherichia coli*.

Upper GI endoscopy confirmed a large hair mass occupying the stomach and duodenum. Due to severe metabolic derangements and sepsis, surgical intervention was initially deferred pending stabilization.

Conclusions:

Trichobezoars are rare but potentially life-threatening if not diagnosed and treated promptly. High clinical suspicion is warranted in young female patients presenting with abdominal pain, palpable mass, and history of hair ingestion.

Early imaging and endoscopic evaluation are essential for diagnosis. Large trichobezoars require surgical removal, while psychiatric evaluation and long-term psychological support are crucial to prevent recurrence.

A multidisciplinary approach involving physicians, surgeons, and mental health professionals is fundamental for optimal outcomes.

Keyword(s) :

2750 - Role of "PAST F model" in predicting advanced liver fibrosis in Autoimmune Hepatitis

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Background / Hypothesis:

The progression of autoimmune hepatitis (AIH) can lead to liver fibrosis and ultimately cirrhosis, significantly impacting patient outcomes. Early identification of patients at risk for advanced fibrosis is crucial for optimizing management strategies and improving prognosis. Although liver biopsy remains the gold standard for diagnosing and staging liver fibrosis, it is invasive and associated with potential complications. Therefore, there is a growing need for non-invasive methods to predict advanced fibrosis in AIH patients.

Materials and Methods:

Objective:

To evaluate the diagnostic accuracy of the PAST-F model in predicting advanced liver fibrosis in patients with autoimmune hepatitis and to establish an optimal cutoff score for clinical application.

Methods:

This retrospective study included patients of either gender with features of AIH on the basis of liver biopsy from January 2016 to December 2023. Patients with concomitant liver diseases were excluded. Liver fibrosis was evaluated using the Metavir scoring system on liver parenchymal biopsy. The outcome was observed in terms of advanced fibrosis on liver biopsy. Univariate followed by multivariate logistic regression analysis was performed to identify independent predictors of advanced fibrosis in patients with AIH.

On multivariate analysis, the following independent predictors were identified and incorporated into the PAST-F model: platelet count $<135 \times 10^9/L$ (OR 1.025; 95% CI 1.015–1.035; 1 point), albumin ≤ 3 g/dl (OR 33.6; 95% CI 10.714–105.7; 6 points), seronegativity (OR 4.9; 95% CI 1.593–41.1; 3 points), total bilirubin (OR 5.134; 95% CI 3.3–8.1; 3 points), and female gender (OR 25.9; 95% CI 6.6–100.2; 4 points).

The diagnostic performance of the model was assessed using AUROC.

Results:

The PAST-F model demonstrated high diagnostic accuracy in predicting advanced liver fibrosis in patients with autoimmune hepatitis with AUROC 0.85 ($p < 0.001$). The sensitivity was 80.14%, specificity 87.67%, positive predictive value (PPV) 80.71%, negative predictive value (NPV) 87.27%, and overall

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diagnostic accuracy 84.72%.

Conclusions:

The PAST-F model shows high diagnostic accuracy and may serve as a reliable non-invasive tool for predicting advanced liver fibrosis in patients with autoimmune hepatitis.

Keyword(s) :

2751 - Fucoidan Alleviates MASH by Inhibiting JNK Phosphorylation Mediated Hepatocyte Apoptosis

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Background / Hypothesis:

Metabolic dysfunction-associated steatohepatitis (MASH) is a severe subtype liver disease, characterized by hepatic steatosis accompanied by inflammation, hepatocyte injury and apoptosis, with a high risk of progression to liver fibrosis. Fucoidan is a natural sulfated polysaccharide abundant in brown algae, and numerous studies have reported its protective effect on metabolic dysfunction-associated fatty liver disease (MAFLD), mainly focusing on improving hepatic steatosis and regulating metabolic disorders. However, studies on the effect and mechanism of fucoidan on MASH are still scarce. This study focuses on the protective effect of fucoidan on MASH and its related molecular mechanisms, filling the gap in existing research.

Materials and Methods:

First, the key c-Jun N-terminal kinase (JNK) pathway involved in the pathogenesis of MASH was screened out by transcriptome sequencing enrichment analysis. Subsequently, mechanism exploration was carried out around this molecule to observe the effect of fucoidan intervention on the pathological state, inflammatory level, steatosis degree, liver fibrosis level and liver function of liver tissue in CDAHFD MASH model mice, and to clarify the correlation between fucoidan and JNK phosphorylation, hepatocyte apoptosis as well as liver fibrosis.

Results:

The results showed that fucoidan intervention could

significantly alleviate the pathological damage of liver tissue in MASH model mice, effectively reduce the level of liver inflammation, steatosis and liver fibrosis, and improve the liver function of mice. From the perspective of molecular mechanism, fucoidan could effectively inhibit the abnormal phosphorylation of JNK, a key signaling molecule involved in regulating hepatocyte apoptosis, inflammatory response and liver fibrosis progression, whose abnormal increase in phosphorylation level is closely related to hepatocyte apoptosis and liver fibrosis progression. The inhibitory effect of fucoidan on JNK phosphorylation further reduced the activation of downstream apoptotic signaling pathways, and ultimately decreased the hepatocyte apoptosis rate, thereby alleviating liver inflammation and liver fibrosis.

Conclusions:

Fucoidan has a significant protective effect on MASH, and its mechanism is related to regulating the JNK phosphorylation pathway, inhibiting hepatocyte apoptosis and reducing liver fibrosis. It provides a new potential strategy for the prevention and treatment of MASH, and enriches the application theory of fucoidan in metabolic-related liver diseases.

Keyword(s) : Metabolic dysfunction-associated steatohepatitis; fucoidan; apoptosis; c-Jun N-terminal kinase

2752 - Protein phosphatase regulatory subunit enhances scaffold protein expression through an RNA binding protein and promotes invasion and immune remodeling in intrahepatic cholangiocarcinoma

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Background / Hypothesis:

Intrahepatic cholangiocarcinoma is an aggressive malignancy characterized by early invasion and metastasis. We identified high expression of protein phosphatase 1 regulatory subunit 1B in the more invasive large duct subtype of intrahepatic cholangiocarcinoma and hypothesized that it enhances scaffold protein expression through an RNA binding protein, thereby promoting tumor invasion and immune remodeling.

Materials and Methods:

Tumor and matched adjacent tissues from 45 patients with intrahepatic cholangiocarcinoma were subjected to single-cell transcriptomic sequencing. Malignant

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cells were stratified according to regulatory subunit expression levels. Differential gene expression and pathway enrichment analyses were performed. Functional validation was conducted in gene overexpression and gene silencing intrahepatic cholangiocarcinoma cell lines. Protein interactions were examined using immunoprecipitation and mass spectrometry. RNA–protein interactions were assessed by computational prediction and enhanced crosslinking immunoprecipitation assays. Immune infiltration and ligand–receptor interactions were analyzed using single-cell transcriptomic data and validated in an independent tissue microarray cohort by multiplex immunofluorescence staining. Results:

High regulatory subunit expression was associated with enrichment of genes involved in cell–substrate junction and cadherin binding pathways. AHNAK was identified as a key downstream effector with significant differential expression. Transcriptomic profiling confirmed that regulatory subunit overexpression increased AHNAK expression, which was further validated in tissue microarrays by immunohistochemistry.

Protein interaction analysis demonstrated binding between the regulatory subunit and the RNA binding protein HNRNP, which was confirmed by mass spectrometry of immunoprecipitated complexes. Enhanced crosslinking immunoprecipitation assays confirmed interaction between HNRNP and AHNAK messenger RNA, supporting a regulatory connection between these molecules.

Consistent with these findings, AHNAK-high malignant cells were enriched in pathways associated with extracellular matrix adhesion, epithelial–mesenchymal transition, hypoxia, glycolysis, and anoikis. Pseudotime analysis suggested progressive accumulation of AHNAK-high epithelial cells toward terminal evolutionary states, indicating a role in tumor progression.

In addition to invasive phenotypes, AHNAK expression negatively correlated with dendritic cell and PLG2-positive cytotoxic T cell infiltration. Ligand–receptor analysis identified COL6A1–CD44 as a shared interaction axis potentially mediating these effects.

Tissue microarray validation using multiplex immunofluorescence demonstrated increased alternatively activated macrophages and exhausted cytotoxic T cells in AHNAK-high tumors.

Conclusions:

These findings support a regulatory subunit–RNA binding protein–AHNAK axis that promotes invasion and contributes to the formation of an

immunosuppressive microenvironment in intrahepatic cholangiocarcinoma and may represent a potential therapeutic target.

Keyword(s) : Intrahepatic cholangiocarcinoma, Tumor invasion, Tumor microenvironment, AHNAK, HNRNP

2754 - THE CONTRIBUTION OF MRCP IN DETERMINING THE NEED FOR ENDOSCOPIC INTERVENTION IN ANASTOMOTIC BILE DUCT STRICTURES IN LIVER TRANSPLANT RECIPIENTS

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Background / Hypothesis: Magnetic resonance cholangiopancreatography (MRCP) is a valuable noninvasive imaging tool for evaluating biliary complications in liver transplant recipients. Specific MRCP-derived biliary imaging characteristics may differ between patients requiring endoscopic intervention and those managed conservatively. This study aimed to identify MRCP features associated with endoscopic intervention in liver transplant recipients with anastomotic biliary strictures.

Materials and Methods: This retrospective study included adult liver transplant recipients with MRCP findings consistent with anastomotic biliary stricture who were evaluated for suspected biliary complications. Patients were categorized into an endoscopic intervention group and a conservatively managed group with at least 12 months of follow-up. MRCP-derived biliary imaging parameters included stricture length, intrahepatic biliary dilatation grade, bile duct calibers proximal and distal to the stricture, and donor–recipient bile duct caliber dominance patterns. Logistic regression analyses were performed to identify imaging features associated with intervention.

Results: A total of 38 liver transplant recipients with MRCP findings suggestive of biliary anastomotic stricture were included: 17 patients underwent

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endoscopic intervention (referred to as the "intervention group"), while 21 patients (55.3%) were managed conservatively (referred to as the "non-intervention group"). The intervention group demonstrated significantly longer anastomotic strictures (6.4 ± 4.4 vs. 2.8 ± 3.5 mm, $p = 0.002$), more advanced intrahepatic biliary dilatation ($p < 0.001$), larger donor bile duct caliber proximal to the stricture (7.2 ± 3.4 vs. 4.7 ± 0.9 mm, $p < 0.001$), and a higher prevalence of a donor-dominant bile duct caliber pattern ($p = 0.040$). Donor bile duct caliber showed excellent discriminatory performance for predicting intervention (AUC = 0.838). In multivariable analysis, donor bile duct caliber remained independently associated with endoscopic intervention (OR = 3.09, $p = 0.013$).

Conclusions: MRCP-derived features, particularly increased donor bile duct caliber, were associated with endoscopic intervention in anastomotic biliary strictures after liver transplantation. These findings highlight MRCP as a valuable noninvasive imaging tool for identifying functionally significant strictures and guiding advanced endoscopists in clinical decision-making.

Keyword(s) : Anastomotic biliary stricture; Biliary complications; Endoscopic retrograde cholangiopancreatography; Liver transplantation; Magnetic resonance cholangiopancreatography

2755 - The Ptn Sdc2 axis regulates meningeal fibrosis and lymphatic function in hepatic encephalopathy

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Background / Hypothesis:

Meningeal lymphatic vessels are essential for CNS homeostasis, clearing metabolic waste and regulating neuroimmune interactions. Recent studies show that in hepatic encephalopathy (HE), meningeal lymphatic drainage increases compensatorily, alleviating neuroinflammation and improving motor function, yet the underlying mechanisms remain poorly understood. Here, we profiled the meningeal transcriptome to investigate how changes in the meningeal microenvironment influence meningeal lymphatic function in HE.

Materials and Methods:

We employed bioinformatics analysis of single-cell RNA sequencing and quantitative proteomics to characterize meningeal microenvironmental changes in a rat model of HE induced by 4-week bile duct ligation (BDL). Functional lymphatic drainage was assessed by tracking fluorescent tracer from the cisterna magna to deep cervical lymph nodes. Meningeal fibrosis and lymphatic changes were evaluated by whole-mount immunofluorescence. For mechanistic validation, we performed intracisternal administration of recombinant Ptn protein or AAV-Sdc2 to modulate the pathway in vivo.

Results:

Single-cell transcriptomic profiling revealed marked reduction of meningeal fibroblasts in BDL rats compared to Sham controls, accompanied by decreased expression of collagen-related genes, which was corroborated by quantitative proteomics. This fibrotic decline was attributed to downregulation of the Ptn-Sdc2 signaling axis in the meningeal microenvironment. Functionally, lymphatic drainage from the cisterna magna to deep cervical lymph nodes was significantly enhanced in BDL rats, alongside reduced collagen deposition and increased lymphatic area. Mechanistically, intracisternal administration of recombinant Ptn protein or AAV-Sdc2 restored meningeal fibrosis, suppressed lymphatic drainage, and further exacerbated the behavioral deficits, confirming that loss of Ptn-Sdc2 signaling alleviates fibrotic constraint, facilitates lymphatic function.

Conclusions:

In this study, we report for the first time that HE induces meningeal microenvironment changes characterized by reduced fibrosis, which facilitates lymphatic drainage. This fibrotic decline was driven by downregulation of the Ptn-Sdc2 axis, identified as a novel regulator mechanism of meningeal fibrosis. These findings provide a potential therapeutic target in HE by disrupting the Ptn-Sdc2 interaction.

Keyword(s) : Hepatic encephalopathy, Meningeal lymphatic vessels, Meningeal microenvironment, Meningeal lymphatic function, fibrosis, lymphatic drainage.

2757 - Diagnostic Accuracy of Leukocyte Esterase Dipstick Test in Early Detection of Spontaneous



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Bacterial Peritonitis in Cirrhotic Patients

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Background / Hypothesis:

Objective: To assess the investigative precision of the leukocyte esterase dipstick test in detecting Spontaneous Bacterial Peritonitis (SBP) among cirrhotic patients.

Materials and Methods:

During the study period. 132 cirrhotic patients were enrolled reported with grade 2 or 3 ascites. Investigative abdominocentesis was completed under stringently sanitised circumstances. A smaller sample of ascitic fluid (10mL) screened for the analysis whereas, a larger sample (30mL) was kept for the dipstick test to determine the granulocyte esterase activity. Using a color-calibrated scale, Leukocyte levels were computed. ++ and +++ dipstick results were marked as positive. Critical investigative metrics were measured discretely for positive dipstick grades.

Results:

The results of the study showed that with LERS++, the confirmation rate for SBP was 85.7% in contrast to the patients with LERS+++ which was increased to 87.5%. LERS++ threshold was calculated as follows: overall diagnostic accuracy, 79.5%, specificity, 96.7%, sensitivity, 42.9%, PPV, 85.7%, NPV, 78.4%. LERS+++ threshold was as follows: overall diagnostic accuracy 81.8%, sensitivity 50%, specificity, 80.6%, PPV 87.5%, and NPV 80.6%.

Conclusions: In order to detect SBP in cirrhotic patients, the LERS (leukocyte esterase dipstick) test appears to possess speedy, cost-economical, and sensible diagnostic benefits having affordable diagnostic precision.

Keyword(s) :

2758 - Mutation Profiles of three selected driver genes from Circulating cfDNA as Non Invasive Prognostic Biomarkers in HBV Induced

Hepatocellular Carcinoma

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Background / Hypothesis:

Chronic hepatitis B virus (HBV) infection is one of the major etiological agents of hepatocellular carcinoma (HCC), a leading cause of cancer-related mortality worldwide. Somatic mutations in TERT, TP53 and CTNNB1 genes are frequently tied-up with hepatocarcinogenesis as they drive tumor initiation, aggressiveness and define tumor phenotype and treatment response respectively. Circulating cell free DNA (cfDNA) is a promising non-invasive alternative of conventional biopsy material for detection of genetic alterations. This study is aimed to detect the hot spot mutations of the above mentioned genes from cfDNA during different stages of HCC and to predict the disease severity using bioinformatics tools.

Materials and Methods:

30 HCC patients of early, intermediate and late stages of HCC and 20 healthy controls were evaluated for TERT, TP53 and CTNNB1 gene hotspot mutations from liquid biopsy (cfDNA extracted from peripheral venous blood using QIAamp Circulating Nucleic Acid Extraction Kit) by Sanger-based cycle sequencing assay. Mutation detection frequencies were determined by MEGA 11 software comparing with specific reference sequences whereas pathogenicity prediction was determined by Predict-SNP and PhD-SNP bioinformatics tools.

Results:

: 40.0% (12/30) of HCC patients had hotspot mutations in selected three genes with 30% (3/10) in early, 44.4% (4/9) in intermediate and 45.5% (5/11) in late stages of HCC which was not statistically significant. No hotspot mutation was detected among healthy controls. TERT promoter mutations were observed 3.3% (1/30) at position c.124 C>T (p. P228L) and 6.7% (2/30) at c.146 C>T (p.L 250L). A highest mutation frequency was found 13.3% (4/30) at position c.746 G>A (p.G249S) of TP53 and then 6.7% (2/30) at c.735 C>T (p.A 245V) and 3.3% (1/30) at c.747 G>T (p.G249V) of same gene. The CTNNB1 showed mutations at position c.110 C>A (p.S37Y) and c.133 T>A (p.S45Thr), 3.3% (1/30) in each. Mutation at codon c124 and c146 of TERT; codon c735,

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c746 and c747 of TP53 and codon c133 of CTNNB1 gene predict 83% deleterious effect using Predict SNP and PhD SNP bioinformatics tool.

Conclusions::

No significant differences of mutations among early, intermediate and late stages indicate most of the mutations are occurred in early stage of HCC. Mutations detection from liquid biopsy may be helpful for early prediction and monitoring the progression of HCC.

Keyword(s) : HBV, HCC, liquid biopsy, driver mutation, cfDNA, TERT, TP53, CTNNB1

2761 - CORRELATION OF NON INVASIVE FIBROSIS TEST RESULTS WITH THE NUMBER OF METABOLIC RISK FACTORS IN PATIENTS WITH METABOLIC DYSFUNCTION ASSOCIATED STEATOTIC LIVER DISEASE

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Background / Hypothesis:

The multiplicity of metabolic risk factors is a prognostic indicator in metabolic dysfunction-associated steatotic liver disease (MASLD). We aimed to evaluate the association between the number of metabolic risk factors and liver fibrosis severity as assessed by the Fibrosis-4 score (FIB-4), NAFLD Fibrosis Score (NFS), and transient elastography among patients with MASLD.

Materials and Methods:

A retrospective review of outpatient records of MASLD patients was conducted. Patients were stratified into those with one metabolic risk factor (Group1) and those with two or more risk factors (Group2). Fibrosis severity was categorized using established cutoffs for FIB-4, NFS, and FibroScan.

Results:

A total of 1,193 MASLD patients were included and 227 were classified as Group1 and 966 as Group2. Group 2 patients were older (52.9 ± 14.9 vs 46.4 ± 15.7 years) and had a higher prevalence of obesity (76.6% vs 64.8%), with a slightly higher mean BMI (27.8 ± 4.2 vs 27.0 ± 4.6 kg/m²). Sex distribution was similar, with male predominance in both groups (~57–58%). Liver enzymes were comparable between groups (ALT ~63 U/L, AST ~45 U/L). Platelet count was modestly lower

in Group 2, while serum albumin levels were similar, indicating generally preserved synthetic function. Advanced fibrosis ($\geq F3$) was significantly higher in Group 2 than Group 1 (9.4% vs 2.6%; $p < 0.001$) by FIB-4 and NFS (7.1% vs 1.0%; $p < 0.001$), while low-risk scores were less frequent using FIB-4 (62.1% vs 78.2%) and NFS (55.0% vs 90.1%). Similar findings were observed using NFS, with more high-risk fibrosis in Group 2 (7.1% vs 1.0%) and fewer low-risk scores (55.0% vs 90.1%; $p < 0.001$). Frequency of advanced fibrosis on FibroScan did not differ significantly (Group2=29.7% vs. Group1=22.1%; $p = 0.215$).

Conclusions:

A higher metabolic burden is significantly associated with increased noninvasive fibrosis risk in MASLD, supporting targeted fibrosis screening to improve early detection and management of patients at risk for advanced liver disease.

Keyword(s) : metabolic dysfunction-associated steatotic liver disease; non-invasive fibrosis test; NFS; FIB-4; FibroScan

2763 - Comparison of Body Composition Parameters Between Cirrhotic and Non Cirrhotic MASLD patients

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease is the most prevalent chronic liver disorder globally and represents the hepatic manifestation of systemic metabolic dysfunction. It is closely associated with obesity, insulin resistance, type 2 diabetes mellitus, dyslipidemia, and cardiovascular disease, contributing significantly to long-term morbidity and mortality. Emerging evidence suggests that fat distribution, predominantly increased visceral adiposity, plays a more critical role in disease progression than overall obesity measured by body mass index (BMI).

Materials and Methods:

In the present study, 151 patients with Metabolic Dysfunction-Associated Steatotic Liver Disease were included, with a mean age of 47.99 ± 11.49 years (range: 23–76 years). The gender distribution was nearly equal, with 49.67%

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females and 50.33% males, demonstrating that MASLD affected middle-aged adults of both sexes almost uniformly in this cohort.

Results:

In the present study, patients with cirrhosis and MASLD showed clear differences in body composition compared to non-cirrhotic patients. Cirrhotic patients had a significantly lower subcutaneous fat area ($278.03 \pm 94.46 \text{ cm}^2$ vs. $369.37 \pm 130.86 \text{ cm}^2$; $p < 0.0001$) and a significantly higher visceral-to-subcutaneous fat ratio (0.61 ± 0.071 vs. 0.52 ± 0.073 ; $p < 0.0001$). However, there was no significant difference in absolute visceral fat area between the two groups (184.09 ± 47.62 vs. 196.79 ± 67.01 ; $p = 0.18$). This suggests that, disease progression is mainly associated with loss of subcutaneous fat rather than an increase in visceral fat.

Conclusions:

The Study revealed that in patients of MASLD, cirrhotic patients had a significantly lower subcutaneous fat area and a significantly higher visceral-to-subcutaneous fat ratio.

Keyword(s) :

2766 - Association of radiotherapy with atezolizumab plus bevacizumab with survival in advanced hepatocellular carcinoma

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Background and Aims: Immunotherapy is the standard first-line therapy for advanced hepatocellular carcinoma (HCC). Radiotherapy (RT) is well established for achieving local control and may also induce systemic immune effects, thereby augmenting the efficacy of immunotherapy. We evaluated the association of RT with outcomes in patients receiving Atezolizumab plus Bevacizumab (Ate+Bev).

Methods: Patients who received RT during Ate+Bev therapy ($n=88$) in 2021–2023 were compared with patients treated with Ate+Bev alone ($n=396$). Overall survival (OS) and progression-free survival (PFS) were analyzed with multivariable Cox proportional hazards models.

Results: Median follow-up was 18.3 months and the median interval from Ate+Bev initiation to RT was 32 days. Baseline tumor characteristics and liver function were comparable between the two groups. Compared with Ate+Bev alone, Ate+Bev+RT was associated with longer OS (aHR=0.71, 95% CI=0.53–0.97, $P=0.030$) and PFS (aHR=0.57, 95% CI=0.45–0.74, $P<0.001$). Effects were consistent across subgroups for PFS (all interaction $P>0.05$). In RT recipients, OS (aHR=1.38, 95% CI=0.65–2.93, $P=0.40$) and PFS (aHR=0.92, 95% CI=0.55–1.52, $P=0.73$) did not differ based on the timing of RT. In patients with extrahepatic RT targets, OS (aHR=7.91, 95% CI=2.90–21.6, $P<0.001$) and PFS (aHR=3.63, 95% CI=1.85–7.12, $P<0.001$) were inferior to those with intrahepatic RT targets. RT-related adverse events occurred in 3.4% of patients, and the overall incidence of adverse events was comparable between the two groups ($P=0.211$).

Conclusions: Combining RT with Ate+Bev was associated with longer OS and PFS, particularly in patients with intrahepatic RT targets. These findings support the potential role of RT as an effective adjunct to immunotherapy and warrant prospective validation.

Keyword(s) :

2767 - Diagnostic value of homocysteine combined with IgG in differentiating drug induced liver injury and autoimmune hepatitis

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Background / Hypothesis:

Background and Aim: Autoimmune hepatitis (AIH) and drug-induced liver injury (DILI) often present with overlapping clinical features, yet their management strategies differ significantly, posing diagnostic challenges. Currently, there is a lack of non-invasive biomarkers with high specificity for differentiating between the two conditions. This study aimed to investigate the diagnostic value of serum homocysteine (Hcy) in distinguishing AIH from DILI and to evaluate its diagnostic performance when combined with the conventional biomarker immunoglobulin G (IgG).

Materials and Methods:

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A retrospective case-control design was adopted, including 324 patients confirmed by liver biopsy (AIH: 143 cases, DILI: 181 cases). Diagnostic performance was assessed using receiver operating characteristic (ROC) curve analysis and multivariate logistic regression models.

Results:

Results showed that serum Hcy levels were significantly higher in the AIH group compared to the DILI group ($P < 0.001$). The area under the ROC curve (AUROC) for Hcy in diagnosing AIH was 0.881 (95% CI: 0.834–0.927). At the optimal cutoff value of 10.05 $\mu\text{mol/L}$, sensitivity was 88.1% and specificity was 92.3%. The AUROC for IgG was 0.732 (95% CI: 0.677–0.788). When Hcy and IgG were combined, the AUROC increased to 0.903 (95% CI: 0.864–0.942). Multivariate logistic regression analysis confirmed Hcy as an independent predictor for distinguishing AIH from DILI (OR=1.694, 95% CI: 1.404–2.043, $P < 0.001$).

Conclusions: Serum Hcy demonstrates superior diagnostic performance in differentiating AIH from DILI compared to the traditional biomarker IgG, and their combination significantly improves diagnostic accuracy. These findings suggest that Hcy may serve as a potential non-invasive tool for clinical differentiation, with important implications for clinical practice. Further validation through prospective, multicenter studies is needed to confirm its applicability across diverse populations and clinical settings.

Keyword(s) : AIH, DILI, Homocysteine, IgG, differential diagnosis

2768 - Diagnostic Value of the SS FIB VT Model Integrating Spleen Stiffness Fibrinogen and Retinal Vessel Tortuosity for High risk Esophageal Varices in Liver Cirrhosis

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Background / Hypothesis:

Gastroscopy is currently the gold standard for screening high-risk esophageal varices (HEV) in patients with liver cirrhosis, but its invasive nature limits widespread application. This study aimed to develop and validate a non-invasive diagnostic model (SS-FIB-VT) integrating spleen stiffness, the serum fibrosis marker (FIB), and retinal vascular morphology features to assess its diagnostic value in discriminating HEV.

Materials and Methods:

High-risk Esophageal Varices; Non-invasive Diagnosis; SS-FIB-VT Model; Spleen Stiffness; Retinal Vessel Tortuosity ;

Methods: This study was a diagnostic investigation. A total of 56 consecutive hospitalized patients with liver cirrhosis from December 2024 to September 2025 were enrolled, all of whom underwent gastroscopy. According to the gastroscopic findings, patients were divided into the HEV group (moderate/severe varices, $n=28$) and the non-HEV group (no/mild varices, $n=28$). Clinical serological indicators, liver and spleen stiffness values measured by transient elastography, and the average tortuosity parameters of retinal vessels in specific regions quantified by the EvisionAI fundus photography intelligent analysis system were collected and analyzed. Significant predictors were screened via univariate logistic regression analysis, and a combined diagnostic model was constructed using multivariate logistic regression. The model's performance was evaluated by indicators including the area under the receiver operating characteristic curve (AUC), sensitivity, and specificity.

Results:

Univariate logistic regression analysis identified six predictors significantly associated with HEV (all $P < 0.05$): platelet count, liver stiffness value, FIB, spleen stiffness value, average tortuosity of vessels in the inferonasal region within a 6.9 mm annular area, and average vessel tortuosity within the 9-13.5 mm annular area. Among the single-indicator diagnostic models, platelet count (AUC=0.787) and the FIB-4 index (AUC=0.795) demonstrated relatively high diagnostic value. Diagnostic efficacy was significantly improved

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through multi-parameter joint modeling. Among all tested combinations, the model integrating spleen stiffness value, FIB, and the average tortuosity of vessels in the inferonasal region within the 6.9 mm annular area was identified as the optimal diagnostic combination, named the SS-FIB-VT model. The AUC for this model was 0.886 (95% CI: 0.800-0.973). At the optimal predictive threshold of 0.46, the model maintained both high sensitivity (89.3%) and specificity (78.6%).

Conclusions:

The SS-FIB-VT model constructed in this study, by combining three types of non-invasive parameters- spleen stiffness, FIB, and retinal vessel tortuosity- demonstrates good diagnostic value (AUC 0.886) for identifying the presence of high-risk esophageal varices in patients with liver cirrhosis. This model provides an accurate and convenient new non-invasive screening strategy for clinical practice, facilitating the optimization of HEV risk stratification and management processes.

Keyword(s) : High-risk Esophageal Varices; Non-invasive Diagnosis; SS-FIB-VT Model; Spleen Stiffness; Retinal Vessel Tortuosity ;

2769 - "Diabetic Ketoacidosis After Laparoscopic Cystectomy for Hydrosalpinx A Case Illustrating Postoperative Metabolic Vulnerability"

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Background / Hypothesis:

Diabetic ketoacidosis (DKA) is a life-threatening metabolic emergency commonly associated with uncontrolled diabetes and acute illness. Its occurrence in the postoperative period following minimally invasive gynecologic surgery is rare and often unexpected. Surgical stress may precipitate metabolic decompensation, even after procedures considered low risk.

Materials and Methods:

We report a case of a 36-year-old woman with unknown diabetic history who underwent laparoscopic cystectomy for hydrosalpinx with an initially uneventful intraoperative and postoperative course. On postoperative day 7, the patient was discharged from

hospital. On postoperative day 7, the patient was readmitted to the hospital with the complaints of abdominal pain, nausea, vomiting, and unexplained metabolic acidosis. Laboratory evaluation demonstrated marked hyperglycemia (334 mg/dL), elevated anion gap metabolic acidosis, and positive ketone bodies (1.3), confirming the diagnosis of diabetic ketoacidosis. The patient was managed promptly with intravenous insulin infusion, aggressive fluid resuscitation, and electrolyte correction, resulting in progressive clinical and biochemical improvement.

Results:

This case highlights the potential role of perioperative surgical stress, counter-regulatory hormone release, and relative insulin deficiency in triggering DKA following minimally invasive gynecologic surgery. Despite smaller incisions and faster recovery profiles, laparoscopic procedures can still induce significant metabolic stress in susceptible individuals.

Conclusions:

Diabetic ketoacidosis, although rare, should be considered in the differential diagnosis of unexplained postoperative metabolic derangements following minimally invasive gynecologic procedures particularly in patients without known diabetes. Vigilant perioperative glucose monitoring and rapid intervention are key to favorable outcomes.

Keyword(s) : Diabetic Ketoacidosis, Laparoscopic Cystectomy for Hydrosalpinx, Postoperative

2770 - The Value of Serum Immature Granulocyte Levels in Predicting Disease Activation in Patients with Ulcerative Colitis

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Background / Hypothesis:Ulcerative colitis (UC) is a chronic inflammatory bowel disease affecting the colon and rectum, characterized by periods of remission and activation. Current markers used to monitor disease activity (CRP, ESR, etc.) have some limitations. This study aimed to investigate the diagnostic value of a novel inflammatory marker, the Delta Neutrophil Index (DNI), also known as Immune Granulocyte (IG) level, in determining disease activation in UC patients and differentiating active patients from those in remission.

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Materials and Methods:The study included patients diagnosed with UC and followed up in the gastroenterology clinic. Participants were divided into "active UC" and "remission" groups based on their clinical and endoscopic findings. Demographic characteristics, daily bowel movements, and laboratory parameters (IG, CRP, ESR, WBC, Hemoglobin) were examined retrospectively/prospectively. ROC curves and correlation tests were used to analyze the data.

Results:The study revealed that serum IG levels in patients with active ulcerative colitis (UC) were significantly different from those in remission. Statistically significant correlations were found between daily bowel movement frequency and laboratory parameters reflecting inflammation levels. ROC analysis results suggest that serum IG level has high diagnostic performance in predicting UC activation and can be used as a reliable marker to differentiate active disease from remission.

Conclusions:Serum IG level may be an inexpensive, readily accessible, and effective biomarker for assessing disease activity in UC patients. The use of this marker in clinical practice could contribute to the personalization of disease management and more effective planning of treatment strategies.

Keyword(s) :

2771 - Infliximab as Salvage Therapy in Multidrug Resistant Autoimmune Hepatitis A Seven Year Clinical Course

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Background / Hypothesis:Autoimmune hepatitis (AIH) is a chronic immune-mediated liver disease that typically responds to corticosteroids and azathioprine. However, approximately 10–20% of patients develop treatment-refractory disease. Management of such cases remains challenging, and data regarding biologic therapies are limited. We report a case of refractory AIH successfully controlled with infliximab after failure of multiple immunosuppressive regimens.

Case Presentation:

A 15-year-old female patient presented in 2019 with fatigue and mild scleral icterus. Laboratory evaluation revealed ALT 697 U/L, AST 438 U/L, total bilirubin 1.46 mg/dL, and elevated IgG 34.6 g/L (7-16). Antinuclear antibody (ANA) was positive. Magnetic resonance

imaging revealed no pathology other than mild hepatomegaly (165 mm). Liver biopsy demonstrated severe lymphoplasmacytic infiltration, interface hepatitis, and portal-portal bridging fibrosis, consistent with autoimmune hepatitis. The patient was diagnosed with autoimmune hepatitis, started on prednisolone 64 mg, and azathioprine 100 mg treatment was added after 2 weeks. Although an initial biochemical response was achieved, relapse occurred during steroid tapering. Over subsequent years, persistent transaminase elevation was observed despite azathioprine optimization. Anti-smooth muscle antibody (ASMA) seroconversion developed during follow-up. Mycophenolate mofetil and later tacrolimus were sequentially introduced without achieving complete biochemical remission. A repeat liver biopsy in January 2024 confirmed ongoing active interface hepatitis. Given refractory disease and sustained biochemical activity, off-label infliximab therapy was initiated in March 2025 after informed consent. Treatment was well tolerated. By January 2026, significant biochemical improvement was observed: ALT 57 U/L, AST 43 U/L, total bilirubin 0.67 mg/dL, and IgG 16.9 g/L. No major adverse events occurred during follow-up. Changes in ALT and IgG levels with treatment from the time of diagnosis are shown in Figure 1.

Conclusions:

This case highlights infliximab as a potential salvage therapeutic option in carefully selected patients with refractory autoimmune hepatitis. Biologic therapy may be considered after failure of conventional and second-line immunosuppressive agents, with close monitoring for safety and disease activity. Further prospective studies are required to define the role of anti-TNF therapy in AIH.

Keyword(s) : Autoimmune Hepatitis. AntiTNF, Infliximab, Salvage Therapy

2772 - Development of a new nutritional screening score in hospitalized patients with cirrhosis.

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Background / Hypothesis:

Malnutrition strongly predicts cirrhosis complications, including infections and hepatic encephalopathy. Nutritional risk screening in cirrhotic patients enables

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early detection of eating disorders and prompt management. Several tools are used (CNAQ, MNA-SF, RFH-NPT), but none alone adequately captures the nutritional specificities of cirrhosis. Therefore, this study aims to develop a cirrhosis-specific nutritional score combining components of the CNAQ, MNA-SF, and RFH-NPT.

Materials and Methods:

The sample consisted of 40 patients with cirrhosis, hospitalized for less than 24 hours at the time of evaluation. Patients with hepatocellular carcinoma (HCC) or with uncontrolled comorbidities (diabetes, arterial hypertension, or dyslipidemia) were excluded from the study. The following questionnaires were then administered to a sample of 40 hospitalized cirrhotic patients: the CNAQ, whose items include those of the Simplified Nutritional Appetite Questionnaire (SNAQ), the MNA-SF, and the RFH-NPT.

Results:

The mean age of the studied population was 65 years. There were respectively 6 patients, 15 patients, and 19 patients classified as Child–Pugh A, B, and C. The mean SNAQ score was 10.35 ± 3.61 , while the mean CNAQ score reached 20.28 ± 6.28 . Regarding the other nutritional measures, the total MNA-SF score was 7.40 ± 3.18 , and the RFH-NPT had a mean of 2.65 ± 1.46 .

The total SNAQ score showed a very strong correlation with the CNAQ score ($r = 0.968$, $p < 0.001$), a positive correlation with the MNA-SF ($r = 0.727$, $p < 0.001$), and a moderate negative correlation with the RFH-NPT ($r = -0.699$, $p < 0.001$).

Using the MNA-SF as the clinical reference, the CNAQ and its derivative, the SNAQ, both showed excellent discriminative ability, with an area under the curve (AUC) greater than 0.9.

However, a new combined score, composed of 7 items from the CNAQ and 5 items from the MNA-SF, presented an AUC of 0.994 (SE = 0.009, $p < 0.001$), higher than that of the SNAQ (AUC = 0.965, SE = 0.028, $p < 0.001$) and the CNAQ (AUC = 0.983, SE = 0.019, $p < 0.001$).

Conclusions:

The SNAQ demonstrates excellent psychometric properties with a strong correlation with the CNAQ and high discriminative ability. However, the superior performance of the combined score (AUC = 0.994) suggests that the CNAQ and the MNA-SF assess complementary dimensions of nutritional risk. Integrating these behavioral and anthropometric

components could optimize early screening for malnutrition while preserving clinical feasibility.

Keyword(s) : Cirrhosis, Malnutrition, Nutritional risk screening

2775 - Circadian Rhythm Disruption Induces Abnormal Serine Metabolism and Immune Suppression to Promote Hepatocellular Carcinoma Progression

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a rapidly progressing and fatal malignancy. Circadian rhythm disruption (CRD) has been shown to promote HCC by altering liver metabolism and the immune microenvironment. This study investigates the role of CRD in activating the serine metabolism pathway, leading to serine accumulation and accelerating HCC progression in mice. Single-cell RNA sequencing revealed that CRD increases neutrophil populations, especially hypoxia-induced neutrophils, with elevated expression of CCL2 in the HIF-1 α signaling pathway, indicating immune suppression in the tumor microenvironment.

Materials and Methods:

Hepa 1-6 liver cancer cells were orthotopically injected into C57BL/6N mice to establish an HCC model. Sleep deprivation was induced for 21 days. Mouse HCC tissues were analyzed by untargeted metabolomics. In vitro, CCK-8 and EdU assays were used to assess metabolite effects, and qPCR and molecular docking were employed to study the impact of circadian rhythm factors on serine synthesis enzymes. Single-cell RNA sequencing was performed to evaluate the immune microenvironment.

Results:

Sleep deprivation significantly increased tumor volume, tumor-to-body weight ratio, and mortality in HCC mice ($P < 0.01$), with no significant change in body weight. Immunohistochemistry revealed increased Ki67 and

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PCNA expression ($P < 0.05$), indicating enhanced proliferation. Untargeted metabolomics showed upregulation of DL-serine ($P < 0.05$), indicating altered serine metabolism. In vitro, DL-serine treatment ($0\mu\text{M}$, $25\mu\text{M}$, $100\mu\text{M}$) significantly increased cell proliferation ($P < 0.05$), with the $100\mu\text{M}$ group showing the strongest effect. Silencing Clock and Bmal1 with siRNA significantly increased Psat1 expression ($P < 0.01$). Molecular docking confirmed that Clock and Bmal1 bind to the Psat1 promoter, regulating its expression. Single-cell RNA sequencing identified 36,626 cells from HCC tissues, with neutrophils being the most abundant population (13,613 cells). The hypoxia-induced neutrophil subgroup exhibited the most significant differences, and CCL2 expression was upregulated in the HIF-1 α pathway, suggesting immune suppression.

Conclusions:

Circadian rhythm disruption promotes HCC progression by inducing serine accumulation and enriching immune suppressive neutrophils.

Keyword(s) : Hepatocellular carcinoma, Circadian rhythm disruption, serine metabolism, immune suppressive neutrophils

2776 - Serum Vitamin A D and B12 Levels and Their Association with Disease Severity and Treatment Response in Chronic Hepatitis C

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Background / Hypothesis:

Vitamin A and vitamin B12 are predominantly stored in the liver, whereas vitamin D undergoes hepatic 25-hydroxylation as part of its activation pathway. These liver-related vitamins participate in immune regulation, oxidative balance, and stellate cell biology, all of which are central to hepatic inflammation and fibrogenesis. Alterations in their serum levels have been reported in chronic liver diseases; however, their association with antiviral treatment response in chronic hepatitis C (CHC) remains unclear. We aimed to investigate the relationship between serum vitamin A (retinol), vitamin D, and vitamin B12 levels and disease severity, viral parameters, and sustained virologic response (SVR) in patients with CHC.

Materials and Methods:

This case-control study included 106 patients with CHC (79 treatment-naïve and 27 previous non-responders to pegylated interferon plus ribavirin) and 50 age- and sex-matched healthy controls. Serum vitamin A levels were measured using high-performance liquid chromatography, while vitamin D and B12 levels were assessed by immunoassay. HCV-RNA was quantified by real-time PCR and genotyping was performed. Associations between vitamin levels, clinical parameters, and SVR were analyzed.

Results:

The mean age of CHC patients was 56.3 ± 12.1 years, and 58.5% were female. Mean serum vitamin B12 levels were 352.3 ± 143.1 pg/mL in CHC patients and 379.0 ± 201.9 pg/mL in controls ($p > 0.05$). Mean vitamin D levels were 18.1 ± 7.2 ng/mL vs. 19.0 ± 8.8 ng/mL ($p = 0.518$), and mean vitamin A levels were 605.5 ± 242.0 ng/mL vs. 469.7 ± 219.4 ng/mL ($p = 0.328$), respectively. When stratified by vitamin A levels, patients with levels ≥ 400 ng/mL showed higher SVR rates compared to those with lower levels (mean 620.6 ± 239.8 vs. 567.5 ± 247.8 ng/mL), although this difference did not reach statistical significance ($p = 0.31$). No significant association was observed between vitamin D or B12 levels and treatment response.

Conclusions:

Higher serum vitamin A levels were associated with improved SVR rates in CHC patients, although statistical significance was not achieved. Vitamin D and B12 levels were not significantly associated with disease severity or treatment outcomes. Larger prospective studies are needed to clarify the potential predictive role of vitamin A in antiviral therapy response.

Keyword(s) : Chronic hepatitis C, vitamin A, vitamin D, vitamin B12, sustained virologic response

2777 - Profile of HSD17B13 rs72613567 TA allele Variant in Indonesian Patients With Metabolic Dysfunction-Associated Fatty Liver Disease and Type 2 Diabetes Mellitus and Its Association With Liver Steatosis Severity

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is the most common cause of chronic liver disease and is frequently observed in individuals with type 2 diabetes mellitus (T2DM). Patients with concomitant MAFLD and T2DM are at greater risk of disease progression toward liver steatosis. In addition to metabolic risk factors, genetic determinants are believed to contribute to disease severity. The HSD17B13 rs72613567 TA allele has been reported to exert a potential protective effect against hepatic inflammation, steatosis and fibrosis. However, evidence regarding the association of this genetic variant with steatosis severity among Indonesian patients with MAFLD and T2DM has never been explored previously. This research tried to determine the distribution and frequency of the HSD17B13 rs72613567 TA allele and to evaluate its association with liver steatosis severity in Indonesian patients with MAFLD and T2DM.

Materials and Methods:

This analytical observational study employed a cross-sectional design and included 268 patients with MAFLD and T2DM. Data was achieved from the Indonesia Multicenter Genomic Study Biobank of patients with diabetes mellitus conducted between November 2025 and January 2026 who met the eligibility criteria. Demographic, anthropometric, and metabolic characteristics were systematically collected. Genotyping of the HSD17B13 rs72613567 TA allele was performed using molecular techniques. Liver steatosis severity was assessed using validated non-invasive liver elastography. Statistical analyses were conducted to determine the association between the HSD17B13 rs72613567 TA allele and the degree of liver steatosis.

Results:

Most subjects were younger than 60 years, female, and obese. The majority had severe steatosis (S3), about 52,2% of population. The genotype distribution consisted of dominant homozygotes (T/T),

heterozygotes (T/A/T), and recessive homozygotes (T/A/T/A), with an overall variant frequency of 47%. No statistically significant differences were found in clinical characteristics between carriers and non-carriers of the TA allele (all $p > 0.05$), except for triglyceride levels ($p = 0.006$) and SGPT ($p = 0.002$). Bivariate analysis showed no significant association between the HSD17B13 rs72613567 TA allele and liver steatosis ($p = 0.623$, PR = 1,146, 95% CI = 0,665-1,974).

Conclusions:

In Indonesian patients with MAFLD and T2DM, the HSD17B13 rs72613567 TA allele had a frequency of 47% and was not significantly associated with liver steatosis severity.

Keyword(s) : MAFLD; type 2 diabetes mellitus; HSD17B13 rs72613567 TA allele; liver steatosis

2778 - A Novel Non Invasive Index for Predicting Non Alcoholic Fatty Liver Disease in Patients with Chronic Hepatitis B The Hatay Index

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized in patients with chronic hepatitis B (CHB) and may influence disease assessment and long-term outcomes. Accurate identification of hepatic steatosis in this population is challenging, as most non-invasive steatosis indices were developed in the general population and may be affected by viral- and treatment-related factors. Liver biopsy provides a reliable reference for detecting steatosis but is not feasible for routine clinical use. This study aimed to develop a novel non-invasive index for predicting NAFLD using a biopsy-confirmed CHB cohort, with the ultimate goal of applying this index to CHB patients with suspected NAFLD and, subsequently, to broader NAFLD populations.

Materials and Methods:

In this retrospective, single-center study, 163 adult patients with CHB who underwent liver biopsy between 2018 and 2024 were included. Hepatic steatosis exceeding 5% on histopathology was defined as NAFLD and used as the reference standard. Demographic, anthropometric, biochemical, metabolic,

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virological, and histopathological data were analyzed. Variables associated with NAFLD were first evaluated using univariate analyses, and significant parameters were incorporated into a multivariate regression model to derive the Hatay Index. The diagnostic performance of the index was assessed using receiver operating characteristic (ROC) curve analysis.

Results:

The mean age of the patients was 42.8 ± 12.3 years, and 58.3% were male. NAFLD was identified in 35.6% of the cohort. Patients with NAFLD had significantly higher TyG index, fasting glucose, HbA1c, triglyceride, and uric acid levels, and lower HDL cholesterol levels compared with those without NAFLD (all $p < 0.05$). No significant differences were observed in HBV-DNA levels, HBeAg status, histological activity index, or fibrosis stage. Multivariate regression analysis identified TyG index, uric acid, ALT, and LDL cholesterol as independent predictors of NAFLD, forming the Hatay Index. The area under the ROC curve was 0.760. At a cut-off value of 19.13, the index demonstrated a specificity of 91.4%, sensitivity of 50%, and an overall diagnostic accuracy of 76.7%.

Conclusions:

Using liver biopsy-confirmed CHB patients as a reference population, we developed the Hatay Index, a simple and non-invasive tool for predicting NAFLD. This index may facilitate the identification of hepatic steatosis in CHB patients without the need for invasive procedures and provides a foundation for future validation and application in broader NAFLD populations.

Keyword(s) : Chronic hepatitis B; Non-alcoholic fatty liver disease; Liver biopsy; Non-invasive index; Hatay Index

2780 - Antiviral therapy in treatment naive patients with hepatitis B related cirrhosis and low level or undetectable viremia long term outcomes

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Background / Hypothesis:

Current guidelines recommend antiviral therapy for all patients with hepatitis B virus (HBV)-related cirrhosis regardless of viral load. However, the clinical benefit in treatment-naïve patients with low-level (<2000 IU/mL)

or undetectable (<20 IU/mL) viremia remains uncertain. This study evaluated the long-term impact of antiviral therapy in this undertreated population.

Materials and Methods:

This retrospective study enrolled 208 treatment-naïve patients with HBV-related cirrhosis and low-level viremia or undetectable HBV DNA (antiviral therapy group, $n=150$; control group, $n=58$), followed for 1 to 5 years. Generalized estimating equations (GEE) with group-by-time interaction terms were used to analyze longitudinal changes in virological response, liver function, and clinical decompensation, and to assess between-group differences. Overall survival was evaluated by Kaplan–Meier analysis and Cox proportional hazards models.

Results:

GEE analysis revealed that the annual decline in HBeAg positivity was significantly greater in the treatment group than in controls (aOR=0.25, 95% CI: 0.10–0.62, $P=0.003$). Geometric mean annual decreases in ALT and TBIL were 7.4% and 8.8% greater, respectively, in the treatment group than in controls (ALT: $\beta = -0.032$, 95% CI: -0.062 to -0.001 , $P = 0.043$; TBIL: $\beta = -0.037$, 95% CI: -0.065 to -0.008 , $P = 0.011$). The annual risk of progression to decompensation was 29% lower in the treatment group (aOR=0.71, 95% CI: 0.56–0.92, $P=0.009$), as were annual incidences of ascites (aOR=0.72, 95% CI: 0.54–0.97, $P=0.029$) and of spontaneous bacterial peritonitis (aOR=0.70, 95% CI: 0.52–0.95, $P=0.020$). Multivariable Cox analysis identified antiviral therapy as an independent protective factor for overall survival (HR=0.215, 95% CI: 0.070–0.657, $P=0.007$); 5-year survival was higher in the treatment group than in controls (92.25% vs. 68.84%, $P<0.001$).

Conclusions:

In treatment-naïve patients with HBV-related cirrhosis and low-level or undetectable viremia, long-term antiviral therapy significantly improves virologic status, liver function, and survival, and reduces decompensation events. These findings support antiviral intervention for HBV-related cirrhosis regardless of baseline viral load.

Keyword(s) : hepatitis B virus, cirrhosis, low-level viremia, antiviral therapy, overall survival

2782 - Liver Cirrhosis Promotes Hepatocellular Carcinoma Progression by Regulating Myeloid Differentiation in Spleen

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Background / Hypothesis: Among the diverse risk factors for hepatocellular carcinoma (HCC), liver cirrhosis is widely recognized as the most critical premalignant condition. Cirrhosis is characterized by persistent hepatic inflammation, sustained immune activation, and elevated systemic levels of pro-inflammatory cytokines, which may disrupt hematopoietic homeostasis and alter the functional state of hematopoietic stem and progenitor cells (HSPCs). In particular, chronic inflammatory cues may bias HSPCs toward myeloid differentiation, thereby reshaping the hematopoietic landscape and promoting the expansion of tumor-supportive myeloid populations. However, how liver cirrhosis systemically reprograms hematopoietic lineages and drives skewed myeloid differentiation to facilitate HCC progression remains poorly understood. The key regulatory mechanisms linking cirrhosis-induced inflammatory stress to hematopoietic remodeling and tumor-promoting myelopoiesis require further elucidation. A clearer understanding of this systemic crosstalk may provide new insights into the pathogenesis of HCC and identify potential therapeutic targets.

Materials and Methods: We first collected clinical data from patients with HCC to assess the association between liver cirrhosis and prognosis. Liver cirrhosis was induced in C57BL/6 mice by carbon tetrachloride (CCl₄) injection, followed by establishment of an HCC model to evaluate tumor progression. Single-cell RNA sequencing was performed on splenic HSPCs to analyze changes in cell abundance and immune features. The role of heme in regulating cirrhosis-driven HSPC differentiation was further examined. Splenic transplantation and UBC-GFP lineage tracing were used to investigate the migration of splenic myeloid cells to the liver. Single-cell transcriptomics was also applied to characterize splenic myeloid cell profiles in cirrhosis-associated HCC.

Results: Clinical analysis revealed that patients with HCC accompanied by liver cirrhosis exhibited significantly poorer prognosis compared with non-cirrhotic HCC patients. In murine models, cirrhosis markedly accelerated HCC progression. Mechanistically, cirrhotic mice displayed elevated levels of free heme in circulation and spleen, which was associated with

increased DNA damage in splenic HSPCs. This genomic stress promoted myeloid-biased differentiation, resulting in the expansion of immunosuppressive myeloid populations. Lineage tracing and splenic transplantation experiments further demonstrated that these cirrhosis-associated myeloid cells migrated from the spleen to the liver, where they accumulated within the tumor microenvironment and facilitated HCC progression.

Conclusions: Our findings demonstrate that liver cirrhosis worsens HCC prognosis by systemically remodeling splenic hematopoiesis. Elevated free heme induces DNA damage in splenic HSPCs, driving myeloid-skewed differentiation and the expansion of immunosuppressive myeloid cells. The migration of these spleen-derived myeloid cells to the liver establishes a tumor-promoting microenvironment, thereby accelerating HCC.

Keyword(s) : Liver Cirrhosis, hepatocellular carcinoma, hematopoietic stem and progenitor cells, heme

2783 - Methyldopa induced acute liver failure successfully managed with drug withdrawal and supportive care A case report

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Background : Methyldopa is a centrally acting antihypertensive drug that is now infrequently used in general practice but remains an important option in pregnancy due to its favorable maternal and fetal safety profile. Although mild aminotransferase elevations are relatively common, severe drug-induced liver injury (DILI) and acute liver failure (ALF) are rare. Methyldopa may cause drug-induced autoimmune-like hepatitis (DI-ALH), which clinically and serologically resembles autoimmune hepatitis.

Materials and Methods:

A 40-year-old Sri Lankan woman presented with fatigue, nausea, progressive jaundice, and somnolence. She had commenced methyldopa 500 mg three times daily ten weeks earlier for newly diagnosed hypertension prior to in vitro fertilization. She denied

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alcohol use, herbal medications, previous liver diseases, or family history. Examination revealed low Glasgow Coma Scale (GCS) of 14 with flapping tremors consistent with grade 2 hepatic encephalopathy (HE). She was deeply icteric with mild right hypochondrial tenderness. Laboratory evaluation demonstrated a hepatocellular pattern of liver injury with AST 1100 U/L, ALT 1201 U/L, alkaline phosphatase of 160 u/dL, total bilirubin 18.1 mg/dL, and INR 1.8. Hepatitis A, B, E, C, cytomegalovirus, Epstein-Bar, and varicella virus screening were negative; other infectious and metabolic causes were excluded. Anti-nuclear antibody was strongly positive (titre 1:1250) with elevated IgG (1847 mg/dL). However, other immune markers such as anti mitochondrial antibody, anti smooth muscle antibody and anti neutrophilic cytoplasmic antibodies were negative. Imaging showed features of acute hepatitis, and excluded biliary obstruction, as well as intra or extra hepatic bile duct dilatation. Liver biopsy revealed moderate portal inflammation with neutrophils and eosinophils, mild fibrosis and interface hepatitis. The presence of grade II hepatic encephalopathy together with an elevated INR was consistent with early acute liver failure.

Results: Methylodopa was discontinued immediately, and a standard regimen of intravenous N-acetylcysteine was administered for 72 hours. The Roussel Uclaf Causality Assessment (RUCAM) score of the patient was 9, indicating a highly probable DILI. The patient gradually improved both clinically and biochemically over the following 2 weeks. Corticosteroid therapy or plasma exchange were not attempted. The patient was reviewed at 2 weeks and 2 months after discharge. Liver enzymes, INR, and ANA had normalized within two months.

Conclusions: Methylodopa can rarely cause severe hepatocellular injury mimicking autoimmune hepatitis which progress to acute liver failure. Early recognition and prompt withdrawal of the offending drug are critical and may prevent the need for immunosuppression, plasma exchange or liver transplantation. Early NAC may contribute to improvement of ALF due to DILI which needs more trials.

Keyword(s) : Acute liver failure, drugs induced liver injury, methylodopa, jaundice, hepatic encephalopathy

2786 - Restoring sleep in cirrhosis A prospective three arm double blind randomized clinical trial of lemborexant efficacy and hepatic safety

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Background / Hypothesis:

Liver cirrhosis is a chronic condition characterized by extensive liver tissue damage that often leads to sleep disturbances, affecting 60–80% of patients. These disturbances significantly impair quality of life and worsen disease prognosis by exacerbating cognitive dysfunction and hepatic encephalopathy. While Lemborexant—a dual orexin receptor antagonist—is effective for insomnia in the general population, data regarding its use in patients with impaired liver function is limited. This study aims to evaluate the safety and efficacy of Lemborexant in improving sleep quality among cirrhosis patients, hypothesized to be an effective treatment with a manageable safety profile.

Materials and Methods:

This study was a prospective, three-arm, double-blind, randomized clinical trial conducted at a national referral hospital. A total of 82 patients with liver cirrhosis and sleep disturbances (Pittsburgh Sleep Quality Index/PSQI > 5) were randomized into three groups: Placebo (n=28), lemborexant 5 mg (n=28), and lemborexant 10 mg (n=26). Parameters assessed included change in sleep quality scores and hepatic safety markers (aspartate aminotransferase, alanine aminotransferase, and bilirubin) over a 2-week treatment period.

Results:

At baseline, mean sleep quality scores indicated poor sleep across all groups (Placebo: 10.0; 5 mg: 12.71; 10 mg: 14.23). After 2 weeks of treatment, both lemborexant groups showed significant clinical improvement; the 5 mg group score decreased to 5.71 ($p < 0.001$) and the 10 mg group to 5.96 ($p < 0.001$). In contrast, the placebo group showed no significant change (9.0 at week 2, $p = 0.112$). Regarding safety, there were no statistically significant differences between groups in liver enzymes, albumin, or total bilirubin levels, suggesting a balanced and safe clinical profile for both doses in this population.

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Conclusions:

Lemborexant is effective in significantly improving sleep quality in patients with liver cirrhosis, as evidenced by the substantial reduction in PSQI scores compared to placebo. Furthermore, the treatment demonstrated a favorable safety profile with no adverse impact on liver function parameters during the study period. These findings suggest that Lemborexant, particularly at a 5 mg or 10 mg dose, is a viable and safe therapeutic option for managing sleep disturbances in patients with chronic liver disease.

Keyword(s) : Lemborexant, Liver cirrhosis, Sleep disturbances

2787 - Variceal bleeding due to stride immunotherapy induced or cirrhosis driven

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Background / Hypothesis:

Portal hypertension (PHTN) affects up to half of patients with hepatocellular carcinoma (HCC) and significantly worsens outcomes due to variceal bleeding. Recently approved systemic therapies for unresectable HCC—atezolizumab-bevacizumab (atezo-bev) and STRIDE (single tremelimumab + durvalumab)—have improved survival. Variceal bleeding is a known risk with atezo-bev (IMbrave150 trial), but was not observed with STRIDE in the HIMALAYA trial, likely because patients with portal vein tumor thrombosis (PVTT) were excluded. Based on this, AASLD guidelines recommend STRIDE in patients with high-risk varices or prior bleeding. We report the first fatal case of variceal bleeding following STRIDE, questioning its safety in real-world practice.

Materials and Methods:

Single-case observational report of a cirrhotic HCC patient with PVTT treated with SBRT followed by STRIDE. Clinical, laboratory, imaging, and endoscopic findings were prospectively documented. Management of bleeding followed standard endoscopic and rescue protocols.

Results:

A 53-year-old diabetic male with MASLD-related cirrhosis with Prior variceal bleed (treated with ligation + glue for GOV2); no recurrence, last endoscopy 5 days before presentation showed eradicated varices. On

carvedilol 12.5 mg/day with good HR control (55-60 bpm).

Baseline status: Child-Pugh A (6), MELD 12 Labs: Bilirubin 2.1 mg/dL, Albumin 3.6 g/dL, INR 1.33, Imaging: 15.3 × 13.5 cm HCC with main PVTT (BCLC Stage C)

Treatment: 5 sessions of SBRT, followed 6 weeks later by STRIDE (per AASLD guidance due to prior bleed).

Clinical course:

Day 8 post-STRIDE patient presented with melena on upper GI endoscopy revealed high-risk esophageal + fundal varices noted and managed with EUS-guided coil + glue for fundal varices and banding for esophageal varices. later patient Developed new-onset ascites; managed with diuretics and continued carvedilol. Two weeks later patient presented with massive hematemesis + melena; endoscopy showed post-banding ulcer bleed. Emergency DANIS stent placed, but patient died from hemorrhagic shock and renal failure.

Conclusions:

This is the first reported fatal variceal bleed after STRIDE in cirrhotic HCC with portal hypertension. While the IMbrave150 trial reported few episodes of variceal bleeding with atezolizumab bevacizumab, the landmark HIMALAYA trial on STRIDE did not report any bleeding events likely due to exclusion patients with portal vein tumoral thrombosis. Multiple factors—portal hypertension, prior interventions, portal vein thrombosis, and immunotherapy—likely contributed to refractory variceal bleed in our patient. STRIDE, though guideline-endorsed for patients with high-risk varices, may not be universally safe.

Careful patient selection, endoscopic surveillance, and individualized prophylaxis are essential. Large real world studies are needed to define bleeding risk in this population.

Keyword(s) :

2788 - Double plasma molecular adsorption system (DPMAS) for liver failure early institutional experience at a tertiary hospital in the Philippines

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Background / Hypothesis:

Acute liver failure and acute-on-chronic liver failure are associated with high mortality rates when liver

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transplantation is not feasible. Extracorporeal plasma adsorption therapies may improve early biochemical parameters, but clinical impact in mixed-etiology, real-world settings remains uncertain. We aimed to describe case-mix, baseline severity, early biochemical response, and short-term clinical outcomes after double plasma adsorption therapy (DPMAS) at a tertiary hospital in the Philippines.

Materials and Methods:

We conducted a single-center, retrospective descriptive cohort including adults (18 years or older) with acute liver failure or acute-on-chronic liver failure who underwent at least one complete index session of double plasma adsorption therapy. Baseline demographics, comorbidities, etiology, and prognostic scores (Model for End-Stage Liver Disease score with sodium correction, Child-Pugh score, hepatic encephalopathy grade) were summarized. Paired pre-post index-session laboratory outcomes included total bilirubin, international normalized ratio, creatinine, sodium, albumin, platelets, and hemoglobin. Clinical outcomes included intensive care unit length of stay, number of sessions during admission, and disposition. Analyses were descriptive (medians with interquartile ranges; proportions with 95% confidence intervals).

Results:

Thirteen patients were included (median age 36 years, 76.9% female) with heterogeneous etiologies. Baseline severity was high (median Model for End-Stage Liver Disease score with sodium correction 34 [27–38]; Child-Pugh 12 [12–13]; hepatic encephalopathy grade 3 [2–3]); 69.2% had encephalopathy grade three or higher, 69.2% required vasopressors, and 53.8% required invasive ventilation. Median total bilirubin decreased from 17.38 to 12.12 mg/dL (median percent change –16.9%); Median international normalized ratio improved from 2.47 to 1.32 (median change –0.88; paired data available for 11 patients). Median intensive care unit length of stay was 17 days (12–19), average sessions per admission was 2 (2–3), and in-hospital mortality was 61.5%.

Conclusions:

Plasma adsorption therapy was feasible and associated with early improvements in total bilirubin and international normalized ratio in a severely ill cohort, but mortality remained high. Larger prospective studies with standardized scoring and safety documentation are needed to establish whether early biochemical changes and timing of therapy correlate with clinical

outcomes.

Keyword(s) : acute liver failure, acute-on-chronic liver failure, extracorporeal support, plasma adsorption, bilirubin

2790 - Prevalence and clinical correlates of metabolic burden and fibrosis severity among non cirrhotic HBsAg positive patients

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Background / Hypothesis:

Metabolic dysfunction has emerged as an important modifier of liver disease progression across chronic liver diseases. In patients with chronic hepatitis B, the impact of cumulative metabolic risk on liver fibrosis severity, particularly in non-cirrhotic individuals, remains incompletely characterized. This study aimed to determine the prevalence of metabolic burden and to evaluate its clinical correlates in non-cirrhotic HBsAg-positive patients.

Materials and Methods:

This retrospective cross-sectional study included adult non-cirrhotic HBsAg-positive patients followed in gastroenterology outpatient clinics between January 2024 and December 2025. Metabolic burden was defined as the cumulative presence of obesity, type 2 diabetes mellitus, hypertension, and dyslipidemia, and was analyzed as both a continuous score and a categorical variable (low vs. high burden). Liver fibrosis severity was assessed using the Fibrosis-4 (FIB-4) score. Associations between metabolic burden and clinical, biochemical, and liver-related parameters were evaluated using appropriate univariable and multivariable statistical analyses.

Results:

A total of 110 patients were included (mean age 51.9 ± 12.1 years; 63.6% male). High metabolic burden was observed in 22.7% of patients and was associated with higher FIB-4 scores in univariable analyses. In multivariable linear regression analysis, age ($B = 0.029$, standardized $\beta = 0.464$, $p < 0.001$) and type 2 diabetes mellitus ($B = 0.424$, standardized $\beta = 0.221$, $p = 0.020$) were independently associated with increased fibrosis severity, whereas hypertension showed an inverse association with fibrosis severity ($B = -0.406$, standardized $\beta = -0.226$, $p = 0.035$).

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Conclusions:

Metabolic burden is common among non-cirrhotic HBsAg-positive patients and is associated with greater fibrosis severity. These findings highlight the importance of systematic metabolic risk assessment in the early management of chronic hepatitis B.

Keyword(s) : Metabolic burden; Chronic hepatitis B; HBsAg positivity; Liver fibrosis; FIB-4; Diabetes mellitus; Hepatic steatosis

2791 - Research on Resveratrol loaded Tetrahedral Framework Nucleic Acids and Their Therapeutic Mechanisms for Metabolic Dysfunction Associated Fatty Liver Disease

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Background / Hypothesis:

To address the inherent limitations of resveratrol (RSV), such as poor aqueous solubility and low bioavailability, a novel nanodrug delivery platform (tFNAs-RSV) was engineered using tetrahedral framework nucleic acids (tFNAs). This study aims to investigate the therapeutic potential of this system against Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and to elucidate the underlying molecular mechanisms through which it modulates the NF- κ B signaling pathway.

Materials and Methods:

Tetrahedral framework nucleic acid (tFNA) nanoscaffolds were precisely engineered via DNA molecular self-assembly, achieving high-efficiency loading of resveratrol (RSV). The morphology, particle size, and structural stability of tFNAs-RSV were systematically characterized using polyacrylamide gel electrophoresis (PAGE), dynamic light scattering (DLS), and transmission electron microscopy (TEM). A high-fat diet (HFD)-induced mouse model of MAFLD was established, and the animals were randomly assigned to different groups to receive intervention with equivalent doses of either free RSV or tFNAs-RSV via oral gavage. Therapeutic efficacy was evaluated by assessing hepatic lipid deposition, serum biochemical parameters, and histopathological analysis.

Results:

Physicochemical characterization confirmed that tFNAs-RSV exhibited high structural uniformity and superior aqueous dispersibility, which significantly

enhanced the stability of RSV under physiological conditions. In vivo pharmacodynamic evaluation revealed that, benefiting from the exceptional carrier properties of tFNAs, tFNAs-RSV significantly reduced the degree of hepatic steatosis and serum transaminase levels compared to free RSV. Histological analysis demonstrated that tFNAs-RSV intervention effectively suppressed the pathological infiltration of macrophages and neutrophils within the liver. Molecular mechanistic studies confirmed that tFNAs-RSV downregulated the phosphorylation levels of the nuclear transcription factor p65, thereby inhibiting the transcriptional expression of endothelial adhesion molecules and subsequently blocking the chemotaxis and recruitment of inflammatory cells toward hepatic lesions.

Conclusions:

In this study, a smart nanodrug delivery platform with excellent biocompatibility was successfully constructed, effectively overcoming the physicochemical limitations of RSV. This platform achieved precise modulation of the hepatic inflammatory microenvironment and provides a novel nanomedicine strategy for the clinical intervention of MAFLD.

Keyword(s) : Tetrahedral framework nucleic acids (tFNAs), Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), NF- κ B signaling pathway

2792 - Risk of Hepatitis B Reactivation Among HBsAg Negative Anti HBc-Positive Breast Cancer Patients Undergoing Chemotherapy

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Background / Hypothesis:

Hepatitis B virus (HBV) reactivation is a recognized complication of immunosuppressive and cytotoxic therapies. Although traditionally associated with chronic HBV infection, clinically significant reactivation may also occur in patients with resolved infection (HBsAg-negative, anti-HBc-positive). Breast cancer represents a clinically relevant context, as systemic chemotherapy remains a cornerstone of treatment in both early-stage and metastatic disease. However, real-world data regarding HBV reactivation risk in breast

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cancer patients with resolved infection are limited. This study evaluated the incidence, clinical characteristics, and determinants of HBV reactivation in HBsAg-negative, anti-HBc-positive breast cancer patients receiving systemic therapy without antiviral prophylaxis.

Materials and Methods:

In this retrospective multicenter cohort study, adult patients (≥ 18 years) with confirmed breast cancer treated at two tertiary hospitals in Ankara, Türkiye, were screened. Eligible patients were HBsAg-negative and anti-HBc-positive at baseline, received at least one cycle of systemic chemotherapy or targeted therapy, and had a minimum follow-up of six months after treatment initiation. HBV reactivation was defined according to international guideline criteria. The primary outcome was the incidence of HBV reactivation during or after systemic therapy. Secondary outcomes included time to reactivation, virological kinetics, and clinical severity.

Results:

A total of 139 anti-HBc-positive patients were included (mean age 60.1 ± 10.7 years). At baseline, 89.2% were anti-HBs positive, with a mean anti-HBs titer of 278.0 ± 378.4 IU/L. The mean follow-up duration was 11.1 ± 6.7 months. Most patients had early-stage (43.2%) or locally advanced disease (30.9%).

HBV reactivation occurred in one patient (0.7%). This 61-year-old woman, with anti-HBs < 10 IU/L and undetectable baseline HBV DNA, received anthracycline-based chemotherapy (doxorubicin-cyclophosphamide). Approximately four months after treatment initiation, she developed acute hepatitis with marked transaminase elevation (ALT 1533 U/L, AST 1247 U/L), hyperbilirubinemia, HBsAg seroreversion, and high-level viremia (HBV DNA 6.9×10^7 copies/mL). Entecavir therapy was promptly initiated, resulting in rapid virological decline and complete viral suppression by six months. Follow-up at ten months demonstrated HBsAg clearance without development of protective anti-HBs antibodies.

Conclusions:

HBV reactivation was rare in this multicenter cohort of HBsAg-negative, anti-HBc-positive breast cancer

patients managed without antiviral prophylaxis. The single event occurred in a patient lacking anti-HBs immunity who received anthracycline-based chemotherapy, supporting emerging evidence that both serologic status and regimen intensity influence reactivation risk. These findings favor a risk-adapted strategy incorporating baseline serologic profiling and targeted monitoring rather than universal prophylaxis in routine breast cancer chemotherapy settings.

Keyword(s) :

2794 - Extending Resectability in Fibrolamellar Hepatocellular Carcinoma Through Associating Liver Partition and Portal Vein Ligation for Staged Hepatectomy (ALPPS) A Case Report

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Background / Hypothesis:

Fibrolamellar hepatocellular carcinoma (FL-HCC) is a rare histologic subtype of hepatocellular carcinoma, accounting for approximately 1% of primary liver malignancies. It predominantly affects adolescents and young adults without underlying chronic liver disease or cirrhosis and is characterized by distinct clinical, histopathologic, and molecular features. Despite preserved baseline hepatic function, FL-HCC often presents at an advanced stage with large tumor burden, frequently limiting upfront surgical resectability because of an inadequate future liver remnant (FLR). Complete surgical resection remains the most important determinant of long-term survival, underscoring the need for strategies that expand resectability in select patients. Associating liver partition and portal vein ligation for staged hepatectomy (ALPPS) is an advanced two-stage surgical technique that induces rapid hypertrophy of the FLR, allowing resection of otherwise unresectable hepatic tumors; however, its use is limited by concerns regarding increased perioperative morbidity, and evidence in FL-HCC remains scarce.

Materials and Methods:

A 25-year-old man presented with a 19-month history of recurrent epigastric pain and was found to have a large, heterogeneous hepatic mass involving segments IVb, V, and VIII on contrast-enhanced computed tomography. Percutaneous biopsy confirmed fibrolamellar hepatocellular carcinoma. Multidisciplinary evaluation determined that the extent

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of disease precluded safe single-stage hepatectomy due to an inadequate FLR. The patient underwent multimodal therapy, including transarterial chemoembolization, selective internal radiation therapy, and systemic treatment with lenvatinib, with the intent of disease control and potential downstaging. Despite these interventions, interval imaging demonstrated progressive tumor enlargement. Salvage surgical management with ALPPS was therefore pursued.

Results:

Stage 1 ALPPS involved portal vein ligation with in situ parenchymal transection and was complicated by ischemia-reperfusion injury and acute kidney injury, both of which were managed conservatively. Follow-up imaging demonstrated marked hypertrophy of the remnant liver, permitting successful completion of stage 2 hepatectomy without additional major complications.

Conclusions:

This case demonstrates that ALPPS may serve as an effective salvage strategy to achieve curative resection in carefully selected patients with advanced fibrolamellar hepatocellular carcinoma and insufficient future liver remnant. Given the associated perioperative risks, ALPPS should be considered only after thorough multidisciplinary evaluation and performed in experienced centers, with careful patient selection remaining critical to optimizing outcomes.

Keyword(s) : Fibrolamellar hepatocellular carcinoma, Associating liver partition and portal vein ligation for staged hepatectomy, ALPPS, Future liver remnant, Liver resection, Salvage surgery

2795 - Achieving Recompensation in Hepatitis B Cirrhosis via Targeted Albumin Infusion A Multicenter Randomized Controlled Trial Protocol

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Background / Hypothesis: China bears a high burden of hepatitis B, and effective management of hepatitis B-related cirrhosis remains a major clinical challenge. Although "cirrhosis recompensation" has recently been recognized as a distinct disease stage and carries substantial theoretical and societal value, practical treatment guidelines are lacking. To further evaluate the efficacy of different human albumin treatment regimens in achieving recompensation at week 72, we established a prospective cohort and are currently conducting this randomized controlled trial.

Materials and Methods: This is a 72-weeks, randomized (1:1), multicenter clinical trial based on human albumin treatment. Patients with decompensated HBV-related cirrhosis were recruited from 17 centers across China and centrally randomized to an intensive or standard treatment group. Based on the validation of Baveno VII criteria for recompensation in entecavir-treated patients with hepatitis B-related decompensated cirrhosis, an albumin target of 34 g/L was set for the intensive group. Albumin was infused at baseline, with levels rechecked after 3–5 days and discontinued once ≥ 34 g/L was achieved. Albumin levels were reassessed at weeks 8, 16, and 24, and additional infusions were given if targets were not met. In the standard group, the albumin target was 30 g/L, with other interventions identical to the intensive group.

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Results: The primary objective of the trial is to evaluate the proportion of subjects with decompensated hepatitis B cirrhosis who achieve recompensation at week 72 under two different human albumin treatment regimens. Secondary objectives include assessing the effects of the two treatment regimens on liver and renal function, Child-Pugh score, and MELD score, as well as the incidence of cirrhosis-related decompensation events and liver disease-related mortality and adverse events between the two treatment groups.

Conclusions: The results of this trial are expected to identify an optimized albumin administration regimen for patients with decompensated hepatitis B cirrhosis.

Keyword(s) : Hepatitis B cirrhosis, Recompensation, Human albumin, Randomized controlled trial protocol

2797 - Incidental Multifocal Hepatic Masses in an Asymptomatic Patient A Neuroendocrine Tumor in Disguise

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Background / Hypothesis:

Incidental hepatic lesions are increasingly detected with the widespread use of abdominal imaging for screening and unrelated indications. Although the majority of these lesions are benign, the presence of multiple hepatic masses often raises concern for malignancy and necessitates comprehensive diagnostic evaluation. Differentiating among primary hepatic tumors, metastatic disease, infectious processes, and benign conditions can be particularly challenging when imaging findings are atypical and patients are asymptomatic. Well-differentiated neuroendocrine tumors (NETs) are frequently indolent and nonfunctional, often resulting in delayed or incidental diagnosis. These tumors may demonstrate variable and nonspecific imaging characteristics, occasionally mimicking infectious, inflammatory, or metastatic hepatic lesions. Furthermore, their low proliferative activity may limit the sensitivity of fluorodeoxyglucose positron emission tomography (FDG PET), increasing the risk of diagnostic uncertainty when imaging is relied upon in isolation.

Materials and Methods:

A 64-year-old man was incidentally found to have multiple hepatic masses during a routine executive health evaluation. He was asymptomatic, with no constitutional or gastrointestinal complaints. His medical history was notable only for hypertension and dyslipidemia. Physical examination was unremarkable, with no stigmata of chronic liver disease. Four-phase contrast-enhanced computed tomography of the liver demonstrated multiple ill-defined hepatic lesions with central hypodensity and peripheral venous enhancement, without arterial hyperenhancement or delayed washout. Magnetic resonance imaging with hepatobiliary contrast revealed T2 hyperintense and T1 iso- to hypointense lesions with thick peripheral enhancement, persistent central necrosis, and associated restricted diffusion, raising concern for infectious, inflammatory, or neoplastic etiologies. FDG PET-CT demonstrated no hypermetabolic hepatic or extrahepatic lesions.

Results:

Given persistent diagnostic uncertainty and potential implications for management, an image-guided liver biopsy was performed. Histopathologic examination revealed atypical epithelial cells with neuroendocrine morphology. Immunohistochemical staining was positive for cytokeratin, CK20, MOC-31, INSM1, chromogranin, synaptophysin, and CD56, with a Ki-67 proliferation index of approximately 2.25%, consistent with a well-differentiated neuroendocrine tumor, World Health Organization Grade 1.

Conclusions:

Well-differentiated neuroendocrine tumors may present as incidental, asymptomatic hepatic lesions with misleading imaging features and absent FDG uptake. Restricted diffusion and peripheral enhancement are nonspecific findings and should not be assumed to represent infection or aggressive malignancy. When imaging findings are inconclusive, histopathologic confirmation remains essential for accurate diagnosis and appropriate management. This case underscores the importance of maintaining neuroendocrine tumors in the differential diagnosis of incidental hepatic lesions and highlights the limitations of imaging alone in their evaluation.

Keyword(s) : Neuroendocrine tumor, Incidental liver

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lesion, Multiple hepatic masses, Imaging pitfalls, Liver biopsy

2798 - Small Bowel Obstruction Secondary to Post Operative Adhesions and Gastrointestinal Vasculitis in Systemic Lupus Erythematosus A Rare and Challenging Case

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Background / Hypothesis:

Small bowel obstruction (SBO) is a common cause of acute abdominal pain and emergency hospitalization, most frequently resulting from post-operative adhesions. In the absence of strangulation or ischemia, adhesive SBO is typically managed conservatively. However, coexisting gastrointestinal pathology may complicate the clinical presentation, delay definitive diagnosis, and alter management. Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease in which gastrointestinal involvement is common but often nonspecific. Rarely, lupus-related gastrointestinal vasculitis affects the small intestine, leading to bowel wall ischemia, ulceration, stricture formation, and intestinal obstruction. These manifestations may closely mimic mechanical SBO on clinical and radiologic evaluation, posing a diagnostic challenge in gastroenterology practice.

Materials and Methods:

A 46-year-old woman with no known comorbidities presented with an 18-day history of intermittent epigastric pain and vomiting, initially responsive to treatment for *Helicobacter pylori*-associated gastric ulcer. Symptoms recurred and progressed to severe, crampy abdominal pain with bilious vomiting, unintentional weight loss, and intermittent fever. She also reported inflammatory arthralgia and had a remote history of cesarean section. On examination, she was tachycardic with hypoactive bowel sounds and localized abdominal tenderness without peritoneal signs. Laboratory evaluation revealed anemia, electrolyte abnormalities, and elevated inflammatory markers. Contrast-enhanced computed tomography demonstrated multiple dilated small bowel loops with a transition point at the distal jejunum-proximal ileum, associated mesenteric fat stranding, and moderate ascites, consistent with SBO, with post-operative adhesions considered the most likely etiology. Despite an initial trial of conservative management, symptoms persisted, prompting exploratory laparotomy. Intraoperative findings revealed a strictured proximal

ileal segment densely adherent to the anterior abdominal wall with proximal bowel dilatation, necessitating segmental resection with primary anastomosis.

Results:

Histopathologic examination showed chronic ileitis with mucosal necrosis and ulceration, serosal adhesions, and small- to medium-vessel vasculitis with fibrinoid necrosis. Subsequent immunologic evaluation demonstrated hypocomplementemia, positive antinuclear antibodies, elevated anti-double-stranded DNA titers, and lupus anticoagulant, fulfilling the 2019 EULAR classification criteria for SLE. A diagnosis of adhesive SBO complicated by SLE-associated gastrointestinal vasculitis was established, and the patient improved following immunosuppressive therapy.

Conclusions:

This case highlights the rare coexistence of adhesive small bowel obstruction and lupus-related gastrointestinal vasculitis. In patients with SBO who exhibit persistent symptoms, systemic inflammatory features, or unexpected histopathologic findings, gastroenterologists should consider underlying autoimmune disease. Early recognition of SLE-associated gastrointestinal vasculitis and prompt immunosuppressive therapy are essential to improving outcomes and preventing serious gastrointestinal complications.

Keyword(s) : Small bowel obstruction, Post-operative adhesions, Systemic lupus erythematosus, Gastrointestinal vasculitis, Lupus enteritis

2800 - An observational study to evaluate the incidence clinical profile risk factors of biliary complications in adult post-liver transplant recipients.

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Background / Hypothesis:

Biliary complications remain the most frequent technical cause of morbidity after liver transplantation, particularly in settings where living donor liver transplantation (LDLT) predominates and multiple

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small duct anastomoses are common. These complications, including anastomotic strictures, bile leaks, and non-anastomotic strictures, lead to recurrent cholangitis, prolonged hospital stay, repeated interventions, and may jeopardize graft survival, making their systematic evaluation crucial.

Materials and Methods:

This was a prospective observational study of adult post-liver transplant patients (age ≥ 18 years) who underwent transplantation and subsequently developed biliary complications. Detailed donor, recipient, perioperative, and follow-up data were collected, including demographics, etiology of liver disease, comorbidities, graft-to-recipient weight ratio (GRWR), cold and warm ischemia times, biliary anatomy, type and number of ductal anastomoses, type and timing of biliary complications. Statistical analysis was performed using SPSS, with appropriate parametric and non-parametric tests and a significance level of $p < 0.05$.

Results:

A total of 100 post-transplant recipients with biliary complications were analysed; most grafts were from LDLT donors (97%) with a mean donor age of 38.35 ± 8.17 years and predominantly ABO-compatible grafts (95%). Among recipients, 81% were male, middle-aged (majority 51–60 years), and largely overweight/obese (85%), with decompensated chronic liver disease as the main indication for transplant (84%) and alcohol-associated liver disease (40%) and MASLD (39%) as leading etiologies. The mean GRWR was 0.996 ± 0.156 , with acceptable cold (74.3 ± 5.86 min) and warm (22.36 ± 2.87 min) ischemia times, and most patients had two or more ductal anastomoses reflecting complex LDLT anatomy. Overall incidence of biliary complications was 29.4% in LDLT versus 12.0% in DDLT recipients. Anastomotic strictures were the predominant complication (86%), followed by bile leaks (11%) and non-anastomotic strictures (3%). Clinically, 88% of patients were symptomatic, most commonly with fever (39%) and pruritus (28%), and symptoms were predominantly late in onset (81%), correlating with the typical course of anastomotic strictures.

Conclusions:

Biliary complications after liver transplantation were common in this LDLT-predominant cohort, with a higher incidence in LDLT compared with DDLT and anastomotic strictures forming the major burden. High frequency of complications was seen in recipients with complex biliary anatomy and multiple duct anastomoses.

Keyword(s) : Biliary complications, liver transplantation, anastomotic strictures

2801 - A Rare Cause of Massive Painless Hematochezia Acute Hemorrhagic Rectal Ulcer Syndrome in a Critically Ill Patient

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Background / Hypothesis:

Lower gastrointestinal bleeding (LGIB) is a common cause of hospitalization among elderly patients and is associated with significant morbidity and mortality, particularly in those with advanced age and multiple comorbidities. Frequently encountered etiologies include diverticular disease, ischemic colitis, colorectal neoplasia, and anorectal disorders. However, uncommon causes of LGIB may be overlooked in critically ill patients, resulting in delayed diagnosis and recurrent hemorrhage. Acute hemorrhagic rectal ulcer syndrome (AHRUS) is an underrecognized condition characterized by sudden-onset, painless, and often massive hematochezia arising from rectal ulcerations unrelated to malignancy, inflammatory bowel disease, or infectious colitis. AHRUS predominantly affects elderly, immobilized, or systemically ill patients and is commonly associated with sepsis, diabetes mellitus, cerebrovascular disease, chronic kidney disease, and antithrombotic use. Endoscopically, it is typified by distal rectal ulcers with exposed vessels or active arterial bleeding and is notable for a high risk of rebleeding despite initial hemostatic therapy. Optimal management remains challenging, particularly when endoscopic therapy fails.

Materials and Methods:

A 76-year-old woman with multiple comorbidities, including invasive breast carcinoma status post modified radical mastectomy, peripheral arterial disease, chronic cerebrovascular disease, and type 2 diabetes mellitus, was admitted after cardiopulmonary arrest secondary to septic shock and hyperosmolar hyperglycemic state. During hospitalization, she



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developed coffee-ground output via nasogastric tube, prompting initial evaluation for upper gastrointestinal bleeding. Despite conservative management, she subsequently developed recurrent episodes of painless hematochezia accompanied by progressive anemia and intermittent hemodynamic instability. Contrast-enhanced abdominal computed tomography demonstrated rectal wall thickening suggestive of inflammatory changes.

Results:

Flexible sigmoidoscopy revealed two distal rectal ulcers approximately 4 cm from the anal verge, including a large semi-circumferential ulcer with active pulsatile bleeding. Endoscopic hemostatic interventions, including hemoclippping, epinephrine injection, and thermocoagulation, were attempted but failed to achieve sustained hemostasis. Given persistent hemorrhage and clinical instability, angiographic evaluation was performed. Superselective catheterization identified abnormal hyperemia involving branches of the superior rectal artery, and particle embolization was successfully performed. Following embolization, bleeding ceased, hemoglobin levels stabilized with transfusion support, and no recurrence of hematochezia was observed.

Conclusions:

AHRUS is an uncommon but clinically significant cause of massive LGIB in elderly and critically ill patients. Diagnosis is challenging due to nonspecific presentation and the presence of competing etiologies of gastrointestinal bleeding. While endoscopic hemostasis remains first-line therapy, angiographic embolization is an effective rescue modality in refractory cases. Early recognition and a multidisciplinary approach, with attention to both local hemostasis and optimization of underlying systemic conditions, are essential to improving outcomes.

Keyword(s) : Acute hemorrhagic rectal ulcer syndrome, Lower gastrointestinal bleeding, Hematochezia, Rectal ulcer, Angiographic embolization

2802 - Melatonin Ameliorates LPS Induced Acute Liver Injury via Upregulating Sirtuin 4(SIRT4) Expression and Inhibiting Macrophage Extracellular Trap(MET) Formation

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Background / Hypothesis:

Sepsis is a life-threatening systemic inflammatory response syndrome, which often leads to multiple organ dysfunction including sepsis associated acute liver injury(SALI). This study aimed to investigate whether melatonin confers protection against SALI by regulating macrophage extracellular trap(MET) formation, and to further delineate the molecular mechanisms underlying this protective effect.

Materials and Methods:

In this study, we established a lipopolysaccharide(LPS)-induced murine model of sepsis-associated acute liver injury(SALI) for in vivo experiments, and employed a murine macrophage cell line for in vitro assays. We first investigated the functional role of macrophages in the pathogenesis of SALI, as well as the hepatoprotective efficacy of melatonin against SALI in the established model. Subsequently, we systematically delineated the molecular mechanisms underlying melatonin-mediated regulation of macrophage extracellular trap(MET) formation using RNA sequencing, mitochondrial functional assessments, and adeno-associated virus serotype 2(AAV2)-mediated targeted gene manipulation.

Results:

In this study, we demonstrated that melatonin has a significant hepatoprotective effect against LPS-induced acute liver injury, which was achieved by suppressing the inflammatory activation of intrahepatic macrophages and the formation of macrophage extracellular traps(METs). Mechanistically, melatonin promoted the nuclear translocation of the transcription factor Forkhead box M1(FoxM1), which in turn transcriptionally upregulated sirtuin 4(SIRT4) expression. We further identified that SIRT4 directly interacted with mitofusin 2(Mfn2) and modulated its acetylation status, thus facilitating mitochondrial fusion, restoring mitochondrial homeostasis and normal function, and ultimately inhibiting MET formation. Furthermore, knockdown of SIRT4 markedly abrogated the hepatoprotective effect of melatonin, as well as its inhibitory action on MET generation, in both in vivo and in vitro models.



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Conclusions:

This study pharmacologically confirmed that melatonin inhibited the formation of METs and ameliorated LPS-induced acute liver injury by activating the FoxM1/SIRT4 signaling pathway, regulating Mfn2 acetylation, and enhancing mitochondrial fusion and function. These findings provide a theoretical foundation for the development of melatonin as a potential therapeutic agent for sepsis-associated acute liver injury(SALI).

Keyword(s) : Sepsis-associated acute liver injury(SALI), Melatonin, Macrophage extracellular traps(METs), Sirtuin 4(SIRT4), Forkhead box M1(FoxM1), Mitochondrial fusion

2803 - Medical Knowledge and Therapeutic Practices Regarding Hepatitis B and C A Study Among Lebanese Physicians

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Background / Hypothesis: Viral hepatitis B (HBV) and C (HCV) remain major public health concerns worldwide and in the region. In Lebanon, the ongoing socio-economic crisis and disparities in access to care may hinder screening, diagnostic confirmation, access to treatment, and prevention strategies, particularly in the perinatal setting. In this context, assessing physicians' knowledge, attitudes, and practices (KAP) regarding HBV and HCV prevention and management is essential to identify gaps and guide targeted interventions.

Materials and Methods: This cross-sectional observational study was conducted in Lebanon among practicing physicians involved in viral hepatitis care (gastroenterology/hepatology, internal medicine, infectious diseases, pediatrics, and obstetrics–gynecology). Participation was voluntary; medical students and non-practicing physicians were excluded. Data were collected using an anonymous self-administered questionnaire based on recent literature and international guidelines. Results were summarized

using descriptive statistics (percentages and means/medians). Comparisons across specialties used chi-square or Fisher's exact tests

Results: In total, 115 physicians participated, mainly gastroenterologists (38.3%), pediatricians (26.1%), and obstetrics–gynecology physicians (18.3%), with most practicing in Beirut (32.2%) and Mount Lebanon (35.7%) and having ≥ 10 years of experience. Among 64 respondents who completed the HCV section, perceived prevalence was most often 0–0.1%, with most reporting diagnosing 0–3 cases/year. Treatment was frequently considered from early fibrosis stages (F0–F1), yet the annual number of treated patients remained low. Reported DAA use was polarized (42.2% estimated treating 0–10% of patients vs 35.9% estimating 80–100%), and DAA utilization differed significantly by specialty ($p=0.015$), with high-use responses predominantly from gastroenterology. Sofosbuvir/velpatasvir was the most cited widely used regimen (35.9%), and very high use was reported only by gastroenterologists ($p=0.011$). SVR after HCV therapy was generally high (SVR $>60\%$ reported by 65.6%), although 28.1% reported SVR $\leq 20\%$. For HBV (64 respondents), perceived prevalence was around 1–1.5%; entecavir was most commonly reported as widely used (40.6%), followed by tenofovir/TDF (15.6%). In the perinatal setting, birth-dose HBV vaccination was perceived as relatively high, whereas early HBIG administration was mostly reported as low; most gynecologists did not repeat pregnancy screening if premarital testing had been performed.

Conclusions: This study highlights a gap between theoretical knowledge of viral hepatitis management strategies and their real-world implementation in Lebanon, in a context shaped by economic, logistical, and organizational constraints. The findings support pragmatic measures, including simplifying and standardizing the HBV, HCV care cascade (screening–confirmation–treatment–follow-up), strengthening institutional perinatal protocols (screening during pregnancy, access to HBV DNA testing, HBIG availability and delivery pathways, checklists), and improving follow-up documentation (including SVR).

Keyword(s) : Hepatitis B; Hepatitis C; Lebanon; Physicians; Knowledge-attitudes-practices (KAP); Perinatal prophylaxis.



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2804 - Sequential liver transplantation after combined therapy for hepatocellular carcinoma rejection risk and management

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Background / Hypothesis: The safety of immune checkpoint inhibitors (ICIs) plus targeted therapy as bridging or downstaging before liver transplantation (LT) for hepatocellular carcinoma (HCC) remains debated, particularly regarding post-LT rejection risk.

Materials and Methods: We retrospectively analyzed nine HCC patients who achieved significant pathological response (pathological complete response (pCR) or partial response (PR)) after combined therapy and underwent LT (2021–2025). Clinical data, dynamic alpha-fetoprotein (AFP), washout period, immunosuppression, and outcomes were assessed.

Results: Median age was 54 years (range 43–67), and 88.9% were male. Median washout period was 31 days (range 11–237). All rejection episodes (3/9, 33.3%) occurred in the short washout (≤ 30 days) group (3/5, 60%) vs. none in the long washout group (0/4). Profound AFP decline (median reduction 93% in pCR group vs. 46.8% in PR group) was associated with pCR. All patients received tacrolimus sustained-release capsules (FK506) plus mycophenolate sodium enteric-coated tablets (EC-MPS) as baseline immunosuppression, with mammalian target of rapamycin inhibitor (mTORi) added after 30 days post-LT if wound healed. Among five short-washout patients, three developed rejection requiring high-dose steroids; two rejection-free patients successfully added mTORi without prolonged steroids. Survival analysis showed a trend toward worse overall survival (OS) in the rejection group (log-rank $P=0.157$), though recurrence-free survival (RFS) was similar ($P=0.637$).

Conclusions: Short washout (≤ 30 days) is a key risk factor for post-LT rejection. AFP deep response may predict pathological benefit. In this cohort, successful mTORi addition without prolonged steroids was observed in rejection-free short-washout patients, suggesting mTORi-based maintenance may benefit this high-risk group. Larger studies are warranted.

Keyword(s) : Hepatocellular carcinoma, Liver transplantation, Immune checkpoint inhibitors, Rejection, mTOR inhibitor

2805 - Evaluation of Factors Affecting Bone Mineral Density in Chronic Hepatitis B Patients Receiving Oral Antiviral Therapy "HEBDEM Study"

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Background / Hypothesis: Declining bone mineral density (BMD) is a clinically important long-term issue in chronic hepatitis B (CHB), with potential implications for fracture risk and physical functioning. In routine practice, factors shaping BMD over time are not fully defined, particularly the contribution of antiviral regimens alongside hepatic parenchymal changes detected on ultrasound. Using the HEPDEM retrospective cohort, we examined baseline and 5-year longitudinal correlates of lumbar BMD indices, focusing on tenofovir disoproxil fumarate (TDF), tenofovir alafenamide (TAF), and entecavir (ETV).

Materials and Methods: We included adult CHB patients with baseline (Visit 0) and up to 5 years of follow-up. Lumbar T- and Z-scores were the primary outcomes. Baseline associations were analysed with multivariable linear regression using robust standard errors. Repeated measures over time were assessed with population-averaged models accounting for within-patient correlation (GEE), adjusting for follow-up time, baseline age and sex, body mass index (BMI), liver enzymes, comorbidity burden (≥ 2 comorbidities), ultrasound steatohepatosis, coarse hepatic echotexture, baseline osteopenia, and time-varying antiviral regimen (ETV as reference).

Results: At baseline, mean age was 46.2 ± 12.1 years; 60% were male; mean BMI was 25.7 ± 2.2 kg/m². Mean lumbar T-score and Z-score were -1.23 ± 0.62 and -1.25 ± 0.60 , respectively. In baseline models, increasing age was associated with lower T- and Z-scores, whereas male sex was positively associated with T-score. Over 5 years, both indices declined, with baseline age and follow-up time showing the most consistent independent associations (annual change: T-

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score $\beta = -0.099$; Z-score $\beta = -0.094$; both $p < 0.001$). Relative to ETV, TDF exposure was linked to lower T-score ($\beta = -0.187$; $p = 0.011$) and Z-score ($\beta = -0.178$; $p = 0.020$). Ultrasound-defined steatohepatitis contributed little after adjustment, while coarse hepatic echotexture showed a stronger and more consistent association with lower BMD indices. Patients with ≥ 2 comorbidities also had lower scores. Baseline osteopenia was associated with lower T- and Z-scores after multivariable adjustment. Conclusions regarding TAF were limited by smaller numbers and/or heterogeneous follow-up.

Conclusions: In this real-world CHB cohort, lumbar BMD indices declined over five years, largely reflecting aging and time. TDF exposure, coarse hepatic echotexture, higher comorbidity burden, and baseline osteopenia identified patients with a less favourable BMD profile. These findings support more focused bone health monitoring, particularly for patients receiving TDF and for those with parenchymal changes on ultrasound and multiple comorbidities.

Keyword(s) : Chronic hepatitis B; Bone mineral density; Tenofovir disoproxil fumarate

2806 - Prevalence and Impact of Cirrhotic Cardiomyopathy on adverse outcomes following Liver Transplantation A systematic review and a Meta analysis

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Background / Hypothesis:

Approximately one-third of liver transplant patients have underlying Cirrhotic cardiomyopathy (CCM), which may contribute to post-transplant complications such as mortality and heart failure. However, the results are subject to various contradictions. Therefore, we aim to conduct a systematic review and a meta-analysis to determine the prevalence of CCM in Liver Transplant patients

Materials and Methods:

Electronic databases such as PubMed, Google Scholar and Cochrane were searched. Articles were selected based on predefined criteria. New Castle Ottawa score

was used to estimate the quality of included studies. Comprehensive Meta-analysis and Review Manager were used to pool prevalence and Hazard ratio (OR) with a 95% confidence interval.

Results:

A total of 6 studies evaluating 1370 patients were included. The pooled prevalence of CCM in liver transplants is 40% (95% CI 22.2% – 60.8%). Additionally, we found that Pre-transplant CCM was significantly associated with post-transplant Major Adverse Cardiovascular Events MACE (OR 1.75 [95%CI 1.23, 2.43]; $p = 0.002$), Heart failure (OR 2.58 [95%CI 1.37, 4.84]; $p = 0.003$), Arrhythmias (OR 2.47 [95%CI 1.63, 3.75]; $p < 0.0001$), and Atrial fibrillation (OR 3.20 [95%CI 1.89, 5.42]; $p < 0.0001$). In contrast, there was no significant relationship between CCM and post-transplant mortality.

Conclusions:

Approximately 40% of individuals who underwent liver transplants had an underlying CCM. Pre-transplant CCM was significantly associated with post-transplant MACE, Heart failure, Arrhythmias, and Atrial fibrillation. However, CCM was not related to post-transplant mortality.

Keyword(s) : Liver transplant; Cirrhotic Cardiomyopathy; Meta-analysis; Prevalence; Outcomes

2807 - Awareness of Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD) in Healthcare Workers A KAP Study

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Background / Hypothesis: Metabolic dysfunction-associated steatotic liver disease (MASLD), affecting about one-third of the global population, is the most common liver disorder and a key contributor to liver morbidity, cardiovascular disease, and mortality. Despite its rising prevalence, awareness of MASLD among healthcare professionals remains limited across medical specialties. This knowledge gap contributes to missed diagnoses and suboptimal patient management. Hence, the objective of the study is to assess the existing knowledge regarding MASLD across healthcare specialties in Pakistan.

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Materials and Methods:

A multi-centre, hospital-based cross-sectional survey was conducted from January to May 2025 among general practitioners, internists, cardiologists, gastroenterologists, and endocrinologists working as consultants or residents across Pakistan. Students, nurses, paramedics, and dieticians were excluded. A total of 237 participants were recruited through non-probability consecutive sampling after obtaining informed consent. Data were collected using a structured questionnaire assessing demographics, area of expertise, awareness of MASLD prevalence and risk factors, and current screening, diagnostic, and management practices. Data were analyzed using SPSS version 19. Descriptive statistics were computed, and chi-square tests were used for significance testing post-stratification. The study was approved by the institutional ethical review committee, and all data were kept confidential to uphold ethical standards

Results:

A total of 237 physicians participated, including gastroenterologists (n=85), internists (n=77), endocrinologists (n=25), cardiologists (n=24), and family physicians (n=26). Most were consultants (67.9%) and based in Sindh (64.6%). Only 33.8% felt very comfortable diagnosing MASLD, with gastroenterologists contributing 67.1% of those confident ($p<0.001$). Awareness of global MASLD prevalence was highest among gastroenterologists (71.8%) and lowest among endocrinologists (48.0%) ($p<0.001$). Obesity (94.9%), diabetes (81.0%), and dyslipidemia (79.3%) were the most recognized risk factors, though dyslipidemia awareness varied significantly across specialties ($p=0.005$). BMI ($p=0.001$) and waist circumference ($p=0.006$) were more accurately identified as diagnostic criteria by endocrinologists and gastroenterologists. Diagnostic modality preference differed, with ultrasound (39.2%) and FibroScan (38.0%) most common. Only 18.1% felt very confident managing MASLD, with cardiologists reporting the lowest confidence (0%, $p<0.001$). While 56.5% used guidelines most of the time, 13.9% were unaware of any. Barriers to screening included lack of awareness (84.8%) and limited understanding of disease reversibility (50.2%).

Conclusions:

Marked variation in MASLD-related knowledge and clinical confidence exists across specialties with

gastroenterologists showing the highest awareness. Targeted, specialty-specific educational interventions are needed to enhance early detection and standardized management of MASLD in Pakistan.

Keyword(s) : MASLD, AWARENESS, DIAGNOSIS

2808 - HYPOALBUMINEMIA AS A PREDICTOR OF COMPLICATIONS IN PATIENTS WITH LIVER ABSCESS A SYSTEMIC REVIEW AND META ANALYSIS

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Background / Hypothesis:

Pyogenic liver abscess (PLA) remains a serious infectious condition associated with high morbidity and mortality. Serum albumin, a marker of nutritional and inflammatory status, has been proposed as a prognostic indicator in infectious diseases. This systematic review and meta-analysis evaluated the impact of hypoalbuminemia on adverse clinical outcomes in PLA.

Materials and Methods:

A comprehensive literature search of PubMed, Google Scholar, and Cochrane databases was conducted from inception to May 1, 2025, following PRISMA guidelines. Studies were included if they compared PLA patients with low versus normal serum albumin levels and reported adverse outcomes. Data extraction and quality assessment using the Newcastle–Ottawa Scale were performed independently by two reviewers. Pooled odds ratios (ORs) were calculated using fixed- or random-effects models based on heterogeneity.

Results:

Five retrospective studies involving 1,779 patients met inclusion criteria, of whom 860 had hypoalbuminemia. Pooled analysis demonstrated that low serum albumin was significantly associated with increased adverse outcomes in PLA (OR 2.64; 95% CI 1.27–5.48; $p = 0.009$), with moderate heterogeneity.

Hypoalbuminemia was also associated with acute kidney injury, prolonged hospitalization, and, in some studies, mortality and metastatic complications. Although albumin cut-off values varied (<2.5 – 3.5 g/dL), the direction of association was consistent. All included

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studies were of high methodological quality.

Conclusions:

Hypoalbuminemia is a significant predictor of poor outcomes in patients with PLA, reflecting systemic inflammation and disease severity. Routine assessment of serum albumin may improve early risk stratification and guide clinical decision-making. Prospective studies are required to determine whether albumin correction improves outcomes.

Keyword(s) : Hypoalbuminemia, Liver abscess, Systematic review

2811 - COMPARATIVE POTENTIAL OF DIFFERENT EDIBLE OILS TO IMPROVE THE STATUS OF NONALCOHOLIC STEATOHEPATITIS

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Background / Hypothesis:

In world's population around 25% recently supposed to have NAFLD. Nonalcoholic steatohepatitis NASH is the advanced stage of fatty liver disease and it is the phase of chronic liver damage and can grow to cirrhosis may lead to hepatocellular carcinoma (HCC). International guidelines describe the health alimentary regimen, based on the lifestyle changes. The main component of the Mediterranean diet MD is their fat choices mainly the olive oil. Mustard oil ensure balanced ratio of monounsaturated, polyunsaturated and small amount of saturated fatty. The oils demonstrated antioxidant and the anti-inflammatory possessions

Materials and Methods:

The current study was carried out to evaluate the effect of edible oils on the different complicated condition of NASH, among the age group of 25-60 years. Then the included subjects were further divided into three groups such as G0, G1 and G2 respectively for the period of 12 weeks. Control group G0 receiving no treatment consumed conventional oil (20g), G1 consumed olive oil (20g) and G2 consumed mustard oil (20g)

Results:

It was observed that there was significant decrease in USG, BMI, CRP, and HbA1c in both treatment groups

along with the significant difference between values of ALT of G1 group consumed olive oil (Mean value= 69.66, SD= 10.5) (Mean value= 49.73, SD= 17.64) at week1 and week 12 respectively, and G2 group that was consumed mustard oil (Mean value= 73.73, SD= 16.16) (Mean value= 54.20, SD= 11.10) at week 1 and week 12 respectively. And There was also significant difference between ALP of G1 group (Mean value = 165.33, SD= 17.82) (Mean value= 127.40, SD= 19.37) at week 1 and week 12 respectively, and G2 group (Mean value= 158.26, SD=18.30) (Mean value = 134.26, SD=14.82) at week1 and week 12 respectively.

Conclusions:It has been evaluated from the current research that showed significant reduction in the treatment groups (G1and G2) compared to control group consuming conventional oil. This was directed to the concluded that the edible oils have beneficial potential against the above mentioned parameters in the condition of NASH.

Keyword(s) : Nonalcoholic steatohepatitis, Mediterranean diet, Olive oil, Mustard oil and liver enzyme

2812 - Plasma Signal Regulatory Protein a a potential biomarker to predict corticosteroid response and prognosis in patients with severe alcohol associated hepatitis

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Background / Hypothesis:

Alcohol-associated hepatitis (AH) has a high short-term mortality in patients with a severe episode (sAH), defined by MELD score > 20. Corticosteroids (CS), the only available medication in patients with sAH, currently is a sub-optimal treatment, as 40-50% of patients are ineligible and a response (CS-R) rate of 50-60% in those who are eligible to receive CS. Further, response can only be determined after administration of CS for at least 7 days (Lille Score <0.45), with a risk of bacterial and fungal infections especially in non-responders to CS (CS-NR). Signal regulatory protein alpha (SIRPa) is an immune cell receptor that regulates efferocytosis and inflammatory responses. Cleavage of SIRPa from ADAM10 depends upon macrophage polarization, with its cleavage and release into the plasma in pro-inflammatory or M1 macrophage. The role of SIRPa has not been studied in patients with AH.

Materials and Methods:

A prospective cohort of sAH patients (2019-2024) enrolled in the Alchep Network study and treated with CS were studied. Plasma samples before administration of CS were analyzed by ELISA for several cytokines and SIRPA levels (ng/mL). Patients were followed for up to 180 days from the time of CS treatment.

Results:

Of 134 sAH patients treated with CS, 88 (mean age 46.4 yrs., 39% females, 86% NH White, mean MELD 23.7, mean CTP score 10.4, mean albumin 2.56, mean WBC 9.7) with available Lille score at d-7 were analyzed. CS-R (n=52) vs. NR (n=36) were younger with lower MELD (22.6 vs. 25.4), BUN (11.9 vs. 18.8), and WBC (8.6 vs. 11.3). Baseline mean plasma SIRPa levels were higher in CS-NR vs. CS-R (216.6 vs. 176.2, Fig. 1A). A total of 18 (20.5%), 29 (33%), and 32 (36.4%) died by 28, 90, and 180-d respectively. Stratifying the data at a median SIRPa level of 187, probability of survival at d-180 was higher in 44 patients with SIRPa levels <187 vs. ≥187 ng/mL (Fig. 1B). In a cox regression model controlled for MELD score, WBC, SIRPa, and Lille score, strongest predictor of death at d-180 was Lille score at d-7, with HR (95% CI) of 16.6 (2.8-99.7), p=0.002.

Conclusions:

Plasma SIRPa is a potential biomarker to predict CS-R, and can personalize CS use in patients with sAH. Future pre-clinical studies are suggested to examine how loss of SIRPa expression or its cleavage contributes to AH pathogenesis. Prospective studies are suggested to

validate SIRPa as a biomarker in use of CS for sAH patients.

Keyword(s) : AUD, MetALD

2813 - Differential therapeutic effectiveness of atezolizumab plus bevacizumab in unresectable HCC by CHB viral activity

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Background/Aims: Atezolizumab plus bevacizumab (Atezo/Bev) is recommended as the first-line therapy for unresectable hepatocellular carcinoma (uHCC). Previous studies suggested differential immunotherapy responses between viral and non-viral HCC; however, the mechanisms remained unclear. We investigated the impact of hepatitis B virus (HBV) DNA detectability, an indicator of viral replication activity that may influence the intrahepatic immune microenvironment, on Atezo/Bev outcomes.

Methods: This retrospective study included patients with uHCC, initially treated with Atezo/Bev at three hospitals in Korea. Patients were categorized as chronic hepatitis B (CHB) with undetectable HBV DNA (CHB/undetectable-DNA), CHB with detectable HBV DNA (CHB/detectable-DNA), or non-viral. Primary and secondary outcomes were progression-free survival (PFS) and overall survival (OS).

Results: We included 549 consecutive patients: CHB/undetectable-DNA (n=216), CHB/detectable-DNA (n=171), and non-viral groups (n=162). After propensity score matching, baseline characteristics were well-balanced. CHB/undetectable-DNA group had significantly longer PFS than both CHB/detectable-DNA (hazard ratio [HR]=0.66, 95% confidence interval [CI]=0.44–0.98, P=0.04) and non-viral groups (HR=0.61, 95% CI=0.37–0.98, P=0.048). PFS did not differ between CHB/detectable and non-viral groups (HR=1.28, 95% CI=0.86–1.91, P=0.23). CHB/detectable-DNA group showed significantly shorter OS than both CHB/undetectable-DNA (HR=1.75, 95% CI=1.04–2.94, P=0.03) and non-viral groups (HR=2.48, 95% CI=1.35–4.52, P=0.003). OS was comparable between CHB/undetectable-DNA and non-viral groups (HR=0.79, 95% CI=0.34–1.84, P=0.58).

Conclusions: Undetectable HBV DNA was associated with longer PFS and OS than detectable HBV DNA in CHB patients with uHCC receiving first-line Atezo/Bev. These findings suggest that HBV DNA detectability may negatively influence immunotherapy outcomes. Antiviral treatment may mitigate this effect and

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improve the therapeutic effectiveness of Atezo/Bev.

Keyword(s) : immunotherapy, atezolizumab plus bevacizumab, metabolic dysfunction-associated steatohepatitis, HBV DNA, liver cancer

2816 - Role of HbA1C in predicting Liver Fibrosis in MASLD patients A Systematic Review and Meta Analysis

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is closely related to dysglycemia and Insulin resistance. While an increase in glycated hemoglobin (HbA1C) is associated with liver fibrosis in individual studies, result remains inconsistent. Therefore, we aim to conduct a systematic review and a meta-analysis to evaluate the association between an increase in HbA1C and the presence of severity of liver fibrosis in MASLD patients

Materials and Methods:

Electronic databases such as PubMed/Medline, Google Scholar, and Cochrane were searched from inception till 30th September, 2025. All studies evaluating the relationship between HbA1C and liver fibrosis in MASLD were included. Pooled Odds ratios (ORs) and mean difference (MD) with 95% confidence intervals (CI) were calculated with a fixed and random effects model as appropriate. Study quality was assessed using the Joanna Briggs Institute checklist.

Results:

Fifteen studies were included evaluating 4864 patients. An increase in HbA1C was significantly associated with increased fibrosis risk (OR 1.56 [0.78, 3.15], $p = 0.21$). Patients with fibrosis and advanced fibrosis had significantly higher mean HbA1C levels compared to those without fibrosis (MD 1.17 and 0.38, respectively). However, there was no association between fibrosis progression and elevated HbA1C. Subgroup analysis revealed a significant association between HbA1C and fibrosis when evaluated by Liver biopsy, but not by Transient Elastography

Conclusions:

An increase in HbA1C level is associated with the presence and severity of liver fibrosis in MASLD patients. These findings suggest that HbA1C is a potential marker that can be used for fibrosis risk stratification in MASLD patients.

Keyword(s) : MASLD; HbA1C; NAFLD; Metabolic dysfunction-associated steatotic liver disease

2817 - Efficacy of Sofosbuvir and Velpatasvir in Achieving Sustained Virological Response in Patients with End Stage Renal Disease Infected with Hepatitis C Virus

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Background / Hypothesis:

Hepatitis C virus (HCV) infection remains a critical concern globally, especially in populations with end-stage renal disease (ESRD) on hemodialysis. This study aims to evaluate the efficacy and safety of a fixed-dose combination of Sofosbuvir and Velpatasvir (SOF/VEL) in achieving sustained virological response at 12 weeks post-treatment (SVR12) among ESRD patients infected with genotype III HCV.

Materials and Methods:

A prospective cohort study was conducted at the Sindh Institute of Urology and Transplantation, Karachi, from January 2023 to December 2024. Inclusion criteria were patients aged ≥ 18 years with ESRD on maintenance hemodialysis for ≥ 3 months and genotype III HCV infection. Patients received SOF/VEL (400 mg/100 mg) daily with ribavirin (400 mg) once weekly for 12 weeks. Primary outcome measured was SVR12. Statistical analyses involved Chi-square and Student's t-tests, with significance set at $p < 0.05$.

Results:

Of 109 enrolled patients (mean age 30.45 ± 8.98 years; 75.2% male), 105 (96.3%) achieved an end-of-treatment response (ETR), while SVR12 was attained in

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91 (84.3%) patients. The mean duration of dialysis was 3.1 ± 1.2 years. The most common adverse effects were fatigue (28%), headache (18%), and nausea (12%). Significant biochemical improvements at treatment completion were noted in ALT ($p=0.001$) and AST ($p=0.021$), although biochemical changes were not significantly correlated with SVR12.

Conclusions:

The combination therapy of Sofosbuvir and Velpatasvir demonstrated good efficacy (84.3% SVR12) and a manageable safety profile in ESRD patients on hemodialysis infected with HCV genotype III. Further multi-center studies with larger sample sizes are necessary to validate and generalize these results, emphasizing the potential of SOF/VEL therapy in this challenging patient population.

Keyword(s) : End-stage renal disease; Hepatitis C; genotype III; ETR; SVR

2819 - Human leukocyte antigen DQ 2 and DQ 8 expression among renal transplanted patients with chronic diarrhea

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Background / Hypothesis:

The prevalence of positive celiac serology among patients with solid organ transplant is low due to the ongoing immunosuppression. However, it is important to determine the HLA DQ expression in this population to rule out celiac disease particularly if the index of suspicion is high. However, no data regarding Asian population has been mentioned. Therefore, our study is an attempt to identify the HLA DQ patterns in our population. This study therefore aims to determine the frequency of expression of human leukocyte antigen among patients with duodenal villous atrophy and negative celiac serology.

Materials and Methods:

It was a cross-sectional prospective study. All renal transplanted patients of both gender and aged between 18 to 60 years with villous atrophy on duodenal biopsy and having negative celiac serology were included in the study. Patients with positive celiac

serology, infectious cause of villous atrophy were excluded from the study. HLA typing was performed in all the included patients.

Results:

A total of 61 patients were included in the study. Among them, 41 (68.3%) were males. The most common presenting complaint was diarrhea seen in 55 (91.7%) followed by weight loss and abdominal pain seen in 49 (81.7%) and 32 (53.3%) respectively. Immunoglobulin A (IgA) deficiency was noted in 49 (82.7%) patients. On laboratory investigations, 40 (67%) patients had microcytic anemia. On histopathology, 30 (50%) patients had sprue marsh class IIIa. Both HLA DQ2 and DQ8 was noted in 31 (51.7%) patients while only HLA DQ2 and HLA DQ8 were observed in 54 (90%) and 37 (61.7%) patients respectively.

Conclusions:

HLA DQ2 was more commonly observed and expressed in renal transplant recipients with villous atrophy suggestive of increased prevalence of seronegative celiac disease in this population.

Keyword(s) : Seronegative; Celiac disease; HLA typing; Renal Transplant; HLA DQ2; HLA DQ8

2820 - Association of Low Vitamin D Levels with Non Alcoholic Fatty Liver Disease in Lean Individuals A Systematic Review and Meta Analysis

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Background / Hypothesis:

Non-alcoholic fatty liver disease (NAFLD) is a growing global health concern, affecting approximately 25% of the global population. While traditionally linked to obesity and metabolic syndrome, NAFLD is increasingly recognized in individuals with normal body mass index (BMI), termed 'lean NAFLD.' Identifying risk factors for lean NAFLD is vital, as these individuals often escape routine screening yet may still progress to advanced liver disease. Vitamin D, known for its immunomodulatory and anti-inflammatory effects, has been implicated in liver metabolism and fibrogenesis. Several observational studies have suggested a link

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between low serum vitamin D levels and NAFLD, but its association in the lean population remains underexplored. This meta-analysis aims to systematically assess the relationship between serum vitamin D levels and NAFLD in lean individuals.

Materials and Methods:

A comprehensive literature search was conducted using PubMed, Semantic Scholar, and Google Scholar from January 2002 to May 2025. A total of 276 studies were initially identified. After screening and eligibility assessment, 7 studies were included in the final analysis. Study designs included observational, cross-sectional, and cohort studies. The total number of patients across all studies was 258,354. The effect size was measured using both odds ratios (OR) with 95% confidence intervals (CI) and mean differences in serum vitamin D levels between NAFLD and non-NAFLD lean individuals. A random-effects model was applied to account for between-study heterogeneity. Statistical heterogeneity was assessed using the I^2 statistic, and publication bias was evaluated using funnel plots.

Results:

The pooled analysis demonstrated a significant association between low vitamin D levels and the presence of NAFLD in lean individuals. The mean difference in serum vitamin D levels between lean NAFLD and non-NAFLD groups was -4.28 ng/mL (95% CI: -6.52 to -2.04 , $p < 0.001$), indicating significantly lower levels in NAFLD patients. The odds ratio for vitamin D deficiency (<20 ng/mL) being associated with NAFLD was 1.47 (95% CI: 1.22–1.77). Substantial heterogeneity was observed among studies ($I^2 = 91\%$), which could be attributed to differences in geographic regions, population demographics, and diagnostic thresholds. Funnel plot inspection suggested minimal publication bias.

Conclusions:

This meta-analysis indicates a significant association between low serum vitamin D levels and the risk of non-alcoholic fatty liver disease in lean individuals. Given the emerging prevalence of lean NAFLD and its potential progression to more severe liver diseases, screening for vitamin D deficiency may serve as a valuable adjunct in identifying at-risk individuals.

Keyword(s) : Vitamin D, Lean NAFLD, Non-obese, Liver steatosis, Systematic review

2821 - TM6SF2 rs58542926 Gene Polymorphism and Liver Fibrosis in the Type 2 Diabetes Mellitus Population in Indonesia

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Background / Hypothesis: Along with the high prevalence of MAFLD in type 2 DM, hepatic complications such as liver cirrhosis are predicted to increase. One factor considered to play a role in MAFLD is the presence of mutations in the TM6SF2 rs58542926 gene. The aim of this study is to determine the frequency of the TM6SF2 rs58542926 gene variant and its association with liver fibrosis in patients with type 2 DM. We hypothesized that the TM6SF2 rs58542926 polymorphism is associated with the incidence of liver fibrosis

Materials and Methods:

A cross-sectional study using data from the Multicenter Genomic Study Biobank on type 2 Diabetes Mellitus patients from July to September 2025. Subjects were individuals with type 2 DM aged 18 years or older. Subjects with malignancies, autoimmune diseases, use of certain medications (methotrexate, phenytoin, isoniazid, and amiodarone), and incomplete data were excluded. Examination of the TM6SF2 rs58542926 gene variant was conducted using WGS, and liver fibrosis was assessed using transient elastography.

Results: Out of 307 subjects, 61 were found to have the TM6SF2 rs58542926 gene mutation, consisting of 50 subjects with partial mutations (C/T) and 11 subjects with complete mutations (T/T). The minor allele frequency of TM6SF2 rs58542926 was found to be 11.7%. A total of 41 subjects (13.4%) experienced significant fibrosis (F2-F4), and bivariate analysis showed that the TM6SF2 rs58542926 gene variant was not associated with liver fibrosis ($p=0.357$)

Conclusions: The frequency of the TM6SF2 rs58542926 gene in the type 2 DM population in Indonesia is 11.7%, and this variant is not associated with the incidence of liver fibrosis.

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Keyword(s) : TM6SF2 rs58542926, Liver fibrosis, MAFLD, Type 2 DM

2822 - Tumor mutational burden and circulating tumor DNA mutation analysis using liquid biopsy samples from Filipino patients with hepatocellular carcinoma

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Background / Hypothesis:

Genetic alterations in cancer genes of patients with hepatocellular carcinoma (HCC) can be potentially used for screening, diagnosis, detecting recurrence of the disease, monitoring treatment response, and prognostication. We present preliminary data on the circulating tumor DNA (ctDNA) mutational profile and tumor mutational burden (TMB) of a small cohort of Filipino HCC patients using liquid-biopsy (blood) samples.

Materials and Methods:

Cell-free DNA (cfDNA) was purified from plasma samples of 14 newly diagnosed, treatment-naïve HCC patients from five sites across the Philippines in an ongoing cohort study. Samples that met the required cfDNA concentration for library preparation (≥ 20 ng) were sequenced using a commercial 523-gene pan-cancer next-generation sequencing (NGS) assay. Bioinformatic analysis was subsequently performed using a proprietary NGS analysis pipeline with an integrated germline variant filtering step and predefined quality metrics to identify single-nucleotide variants (SNVs) and TMB analysis. Samples that had a median exon fragment coverage of ≥ 1300 and with $\geq 1000\times$ coverage in at least 80.0% of exonic regions were included in the analysis.

Results:

All samples met the minimum requirements for small variant calling and TMB analysis. Sequencing data identified 141 genes with somatic SNVs in all samples.

The most frequently mutated genes include the three HCC-associated genes TP53, CTNNB1, and ARID2. Two patients, diagnosed with BCLC stages 0/A and D, respectively, had a high TMB (≥ 10 mutations/MB). Both patients expired within one and two months following diagnosis, respectively.

Conclusions:

Our findings present preliminary evidence of recurrent ctDNA genetic alterations in a small cohort of Filipino HCC patients, warranting further research in a larger cohort with matched tissue samples and clinical correlations.

Keyword(s) : hepatocellular carcinoma, next-generation sequencing, tumor mutational burden, liquid biopsy

2823 - Continuous Terlipressin Infusion Plus Norepinephrine Versus Norepinephrine Alone in Acute Kidney Injury Associated With Acute on Chronic Liver Failure and Septic Shock A Randomized Controlled Trial

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Background / Hypothesis: Acute kidney injury (AKI) in acute-on-chronic liver failure (ACLF) with septic shock is driven by severe systemic vasodilatation and splanchnic pooling, often limiting the efficacy of norepinephrine (NE) alone. Terlipressin, a vasopressin analogue, may provide complementary splanchnic vasoconstriction and improve renal perfusion. We evaluated whether combining continuous low-dose terlipressin infusion with NE improves renal and hemodynamic outcomes compared with NE alone after fulfilling exclusion criteria and added at day 1 of randomisation.

Materials and Methods: Single-center open-label RCT of 126 ACLF patients with less-severe septic shock (NE < 0.05 $\mu\text{g/kg/min}$) and AKI randomized to NE group (n=63) or terlipressin infusion (1 mg/day, escalated to 4 mg/day by Day 4) plus NE (NE + Terli group n=63). MAP ≥ 70 mmHg targeted; 20% albumin given. The primary endpoint was day-4 shock reversal with ≥ 1 -

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stage AKI improvement; secondary endpoints were - day-7 AKI outcomes, albumin dose, Renal replacement therapy (RRT), 28day mortality, and adverse events.

Results: Baseline characteristics were comparable: NE group vs NE + Terli group, age (46.36 ± 10.09 vs 44.21 ± 12.57 years), AARC score (10.53 ± 1.52 vs 10.46 ± 1.17), eGFR (49.92 ± 20.53 vs 51.03 ± 19.49 mL/min/1.73 m²), MAP (63.47 ± 2.29 vs 62.56 ± 2.22 mmHg; $p=0.085$), lactate (2.35 ± 0.62 vs 2.26 ± 0.55 mmol/L; $p=0.502$). Pneumonia was the commonest sepsis source (47.2% vs 43.6%), followed by SBP (19.4% vs 33.3%) in NE vs NE+TERLI. Renal biomarkers were similar: urine NGAL (307.3 ± 237.4 vs 246.6 ± 200.1 ng/mL; $p=0.20$) and cystatin-C (2.19 ± 1.15 vs 1.90 ± 0.59 mg/L; $p=0.14$). NE + Terli group showed significantly higher day-4 shock reversal (92.3% vs 52.8%; $p<0.001$) and ≥ 1 -stage AKI improvement (74.4% vs 44.4%; $p=0.008$) were higher with NE+TERLI. Day-7 AKI resolution was also higher in NE + Terli group (71.8% vs 44.4%; $p=0.004$) with lower progression (12.8% vs 16.7%; $p=0.610$). Day-4 NE equivalent dose was lower (0.01 ± 0.03 vs 0.06 ± 0.08 ; $p=0.001$). Albumin dose (100.6 ± 60.7 vs 96.9 ± 62.2 g; $p=0.78$), RRT (15.8% vs 20.6%; $p=0.63$) and 28-day mortality (22.2% vs 28.2%; $p=0.55$) were comparable. AARC score (adjusted HR 3.01, CI 1.76–5.15; $p<0.001$) and baseline lactate (adjusted HR 14.39, CI 2.75–75.32; $p=0.002$) independently predicted mortality. Adverse events were higher with NE+TERLI (13.3% vs 4.3%; $p=0.2$). Baseline eGFR (OR 1.06, CI 1.02–1.11; $p=0.002$) and NE+TERLI (OR 3.60, CI 1.04–12.47; $p=0.043$) independently predicted day-7 AKI recovery.

Conclusions: In ACLF patients with septic shock-associated AKI, early continuous terlipressin plus norepinephrine significantly improved day-4 shock reversal and renal recovery, reduced vasopressor requirement, and independently predicted day-7 AKI resolution, with comparable mortality and acceptable safety versus norepinephrine alone.

Keyword(s) : ACLF, NE, NE+Terli

2824 - Resveratrol Loaded Tetrahedral Framework Nucleic Acids Ameliorate MAFLD via the NF κ B Signaling Pathway

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Background / Hypothesis:

To address the inherent limitations of resveratrol (RSV), such as poor aqueous solubility and low bioavailability, a novel nanodrug delivery platform (tFNAs-RSV) was engineered using tetrahedral framework nucleic acids (tFNAs). This study aims to investigate the therapeutic potential of this system against Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) and to elucidate the underlying molecular mechanisms through which it modulates the NF- κ B signaling pathway.

Materials and Methods:

Tetrahedral framework nucleic acids (tFNAs) were precisely engineered via DNA molecular self-assembly, achieving high-efficiency loading of resveratrol (RSV). The morphology, particle size, and structural stability of tFNAs-RSV were systematically characterized using polyacrylamide gel electrophoresis (PAGE), dynamic light scattering (DLS), and transmission electron microscopy (TEM). A high-fat diet (HFD)-induced mouse model of MAFLD was established, and the animals were randomly assigned to different groups to receive intervention with equivalent doses of either free RSV or tFNAs-RSV via oral gavage. Therapeutic efficacy was evaluated by assessing hepatic lipid deposition, serum biochemical parameters, and histopathological analysis.

Results:

Physicochemical characterization confirmed that tFNAs-RSV exhibited high structural uniformity and superior aqueous dispersibility, which significantly enhanced the stability of RSV under physiological conditions. In vivo pharmacodynamic evaluation revealed that, benefiting from the exceptional carrier properties of tFNAs, tFNAs-RSV significantly reduced the degree of hepatic steatosis and serum transaminase levels compared to free RSV. Histological analysis demonstrated that tFNAs-RSV intervention effectively suppressed the pathological infiltration of macrophages and neutrophils within the liver. Molecular mechanistic studies confirmed that tFNAs-RSV downregulated the phosphorylation levels of the nuclear transcription factor p65, thereby inhibiting the transcriptional expression of endothelial adhesion molecules and subsequently blocking the chemotaxis and recruitment of inflammatory cells toward hepatic lesions.

Conclusions:

In this study, a smart nanodrug delivery platform with

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excellent biocompatibility was successfully constructed, effectively overcoming the physicochemical limitations of RSV. This platform achieved precise modulation of the hepatic inflammatory microenvironment and provides a novel nanomedicine strategy for the clinical intervention of MAFLD.

Keyword(s) : Tetrahedral framework nucleic acids (tFNAs), Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), NF- κ B signaling pathway

2825 - APRI Tracks FIB 4 Not Imaging Based Liver Metrics in Diabetic MASH A Cross Sectional Analysis

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Background / Hypothesis:

Non-invasive biomarkers are widely used in assessing liver fibrosis and steatosis.

APRI is one such accessible index; however, its correlation with elastography-derived fibrosis grading and steatosis parameters remains uncertain. This study evaluates the association between APRI and FibroScan-based liver stiffness measurement (LSM), controlled attenuation parameter (CAP), fatty liver grading, and compares its correlation with FIB-4 score in Diabetic Patients.

Materials and Methods:

In this cross-sectional analysis, 400 patients with type 2 diabetes mellitus underwent APRI calculation, FibroScan assessment (LSM and CAP), and ultrasound-based fatty liver grading. Associations between APRI and categorical fibrosis/steatosis grades were analyzed using Kruskal-Wallis and Wilcoxon-Mann-Whitney U tests. Correlation with continuous variables (LSM, CAP, FIB-4) was assessed via Spearman's rank correlation.

Results:

No significant association was observed between APRI and FibroScan fibrosis grades ($\chi^2 = 0.764$, $p = 0.858$) or ultrasound-based fatty liver grades ($\chi^2 = 1.351$, $p = 0.717$). Similarly, APRI showed weak, non-significant

correlations with LSM ($p = 0.02$, $p = 0.629$) and CAP ($p = 0.03$, $p = 0.525$). However, a strong and statistically significant correlation was observed between APRI and FIB-4 score ($p = 0.88$, $p < 0.001$). APRI levels were also significantly higher in patients with FIB-4 > 2.67 compared to those with intermediate scores (mean APRI 1.07 vs. 0.52, $p < 0.001$).

Conclusions: APRI demonstrates a strong and clinically meaningful correlation with FIB-4 score but fails to show significant associations with elastographic or sonographic parameters of liver fibrosis or steatosis. These findings suggest that while APRI may be valuable as a biochemical fibrosis marker, it may not reflect imaging-based liver pathology with comparable accuracy.

Keyword(s) : APRI, FIB-4, FibroScan, LSM, CAP, Fatty Liver, Liver Fibrosis, NonInvasive Biomarkers

2826 - Association Between Duration of Type 2 Diabetes Mellitus and Hepatic Steatosis and Fibrosis Assessed by Transient Elastography.

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Background / Hypothesis:

Type 2 Diabetes Mellitus (T2DM) is strongly associated with metabolic dysfunction-associated steatotic liver disease (MASLD). Chronic hyperglycemia may promote hepatic fibrogenesis; however, the independent relationship between duration of diabetes and liver disease severity remains uncertain.

Materials and Methods:

This cross-sectional study included patients with T2DM who underwent vibration-controlled transient elastography for assessment of liver stiffness measurement (LSM, kPa) and controlled attenuation parameter (CAP, dB/m). Duration of diabetes was recorded in years. As at least one variable was not normally distributed, Spearman's rank correlation coefficient was used to assess associations. Linear regression equations were derived to describe trends. A p-value < 0.05 was considered statistically significant.



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Results:

There was a weak positive correlation between LSM and duration of T2DM ($\rho = 0.08$; 95% CI: -0.02 to 0.18), which was not statistically significant ($p = 0.120$). LSM increased by 0.12 kPa per year of diabetes. No meaningful correlation was observed between CAP and duration of T2DM ($\rho = 0.01$; 95% CI: -0.09 to 0.11 ; $p = 0.880$). CAP increased by 0.85 dB/m per year of diabetes, which was not statistically significant.

Conclusions: Duration of T2DM showed no significant association with hepatic fibrosis or steatosis severity. These findings suggest that metabolic burden and disease control may play a more critical role in liver disease progression than duration of diabetes alone.

Keyword(s) : Type 2 Diabetes Mellitus, MASLD, NAFLD, Transient Elastography, Liver Stiffness Measurement, CAP

2827 - Medical Management of Symptomatic Polycystic Liver Disease Using Intermittent Short Acting Octreotide A Cost Effective Alternative

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Background / Hypothesis:

Polycystic liver disease (PCLD) is an autosomal dominant disorder characterized by multiple non-communicating hepatic cysts. Symptomatic patients may require intervention. Long-acting somatostatin analogues reduce liver cyst volume but are often limited by high cost. We report the use of intermittent short-acting octreotide as a cost-effective alternative in symptomatic PCLD.

Materials and Methods:

A 38-year-old male presented with dull aching right upper quadrant abdominal pain for four months. Laboratory investigations were unremarkable. Ultrasound abdomen revealed multiple hepatic cysts in both lobes, some with internal echoes. Triphasic CT abdomen showed multiple cysts in both lobes, the largest measuring 8×7 cm in segment 4a, without high-risk features (no enhancing septations, mural nodules, calcifications, or solid components).

Given symptomatic disease with a dominant large cyst, surgical fenestration with or without aspiration followed by long-acting octreotide was offered. Due to financial constraints, the patient opted for medical

therapy and was initiated on short-acting octreotide $100 \mu\text{g}$ subcutaneously daily for 5 days every month.

Results:

At 4-month follow-up, the patient reported significant symptomatic improvement. Repeat imaging demonstrated marked reduction in cyst size, with the dominant cyst decreasing from 8×7 cm to 4×5.7 cm. The patient tolerated therapy well without major adverse effects. He was counseled regarding potential long-term side effects, including cholelithiasis, steatorrhea, and bloating.

Conclusions:

Intermittent short-acting octreotide resulted in significant clinical and radiological improvement in this patient with symptomatic PCLD. In resource-limited settings where long-acting formulations or surgery are not feasible, intermittent short-acting octreotide may serve as a practical and cost-effective therapeutic option in selected patients.

Keyword(s) :

2828 - Pipecolic Acid Attenuates MASH by Suppressing Monocyte derived Macrophage Recruitment via Activating GABA A receptor to Inhibit CaMKII STAT1 Signaling

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) shows heterogeneity in pathogenesis, with gut microbiota dysbiosis and disturbed amino-acid metabolism contributing to disease progression. Metabolic dysfunction-associated steatohepatitis (MASH) is characterized by persistent hepatic and systemic low-grade inflammation and related to higher risk for adverse outcomes including cirrhosis, hepatocellular carcinoma and extrahepatic malignancies and cardiovascular disease. Macrophages are central part of MASH. Injured hepatocytes release cytokines that recruit and polarize monocyte-derived macrophages, amplifying inflammation and activating hepatic stellate cells. In this study, we investigated the mechanism that pipecolic acid alleviate MASH.

Materials and Methods:



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We established MASH model in vitro using 3D liver spheroids treated with free fatty acids and in vivo using C57BL/6J mice fed GAN diet. Pipecolic acid was administered. Histological staining and flow cytometry were used to estimate liver injury and macrophage populations. RNA-seq and western blotting were used to investigate the mechanism.

Results:

Pipecolic acid treatment ameliorated steatohepatitis features in both 3D spheroids and GAN-fed mice, including steatosis, inflammatory infiltration and fibrosis. In vivo, pipecolic acid selectively decreased the number of infiltrating monocyte-derived macrophages in liver while showed no significant influence on Kupffer cells. Transcriptomic and protein analyses revealed that pipecolic acid acts as a GABA-A receptor agonist, leading to reduced phosphorylation of CaMKII and suppressing STAT1 transcriptional activity, lowers expression of STAT1-dependent inflammatory cytokines and chemokines implicated in monocyte recruitment.

Conclusions:

Pipecolic acid alleviate MASH by agonizing GABA-A receptor to reduce CaMKII phosphorylation, thereby inhibiting STAT1-mediated transcription of inflammatory cytokines and chemokines, which suppresses monocyte-derived macrophage recruitment and hepatic inflammation.

Keyword(s) : Pipecolic Acid, Metabolic Dysfunction-Associated Steatohepatitis, Gut Microbiota, Monocyte-derived Macrophage, Gamma-aminobutyric Acid

2829 - Trends and disparities in China's metabolic associated fatty liver disease burden from 1990 to 2021

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Background / Hypothesis:

There is a rising number of metabolic-associated fatty liver disease (MAFLD) among Chinese adolescents and young people. To identify concrete prevention strategies for MAFLD, a systematic assessment of its risk factors is required. This study uses Global Burden of Disease (GBD) data to analyse, in a systematic manner, the long-term trends and epidemiological

features of MAFLD in Chinese youths and young adults.

Materials and Methods:

We utilised data from GBD 1990–2021 to undertake a comprehensive analysis of the MAFLD burden among Chinese adolescents and young adults, covering prevalence, incidence, mortality, and disability-adjusted life years (DALYs). Time trends were examined using Joinpoint regression, age–period–cohort (APC) modelling, and a Bayesian APC (BAPC) approach to project incidence and prevalence. Finally, we analysed the attributable burden of MAFLD.

Results:

The prevalence of MAFLD is rising sharply. In China, the number of MAFLD cases amongst those aged 15-39 in 2021 increased by 140.02% compared with 1990. Younger generations are at significantly greater risk than earlier generations at the same age; the prevalence in the 35-39 age group is approximately four times that of the 15-19 age group. This suggests the window for disease management needs to shift earlier, to adolescence and young adulthood. While age-standardised mortality rates generally show a downward trend, they have risen again since 2017, indicating a possible increase in the risk of late-stage complications.

Conclusions:

Against the backdrop of China's vast population, MAFLD poses a significant public health burden for adolescents and young people, and there is an urgent need to develop scientifically sound public health policies to tackle this health challenge.

Keyword(s) : Metabolic-associated fatty liver disease; Global burden; Public health; Joinpoint regression; Age-period-cohort model

2830 - Temporal change of hepatitis C virus testing and treatment coverage in people who inject drugs in Hong Kong

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Background / Hypothesis:

In Hong Kong, with the expansion of HCV treatment provision since October 2020, the primary challenge for achieving HCV micro-elimination in people who inject drugs (PWID), a population with over 80% anti-HCV prevalence, is shifted to case identification and linkage to treatment. This study aims to examine the temporal change and factors associated with known HCV positive status and history of HCV treatment in PWID.

Materials and Methods:

Data from 2021-2024 annual surveys on the HIV awareness and risk behaviours were collated and analysed. PWID were recruited by well-trained interviewers of non-governmental organisations either in the neighbourhood of methadone clinics or in nearby urban parks, for completion of questionnaires. Factors, including survey year, associated with each outcome variable (self-reported anti-HCV positive status and history of HCV treatment) were examined in multivariable logistic regression models.

Results:

In the four annual surveys (total 2924 responses), 63% of respondents were aged 50 or above, 92% were male, and 90% were local Chinese. Ninety percent reported injection behaviour (18% with and 64% without needle sharing, and 9% no current injection), of whom 59% have injected for over 20 years. Most (89%) have attended the methadone clinics frequently for at least 22 days in the past 1 month, including 72% full attendance), and 89% were aware that drug injection could transmit HCV. From 2021 to 2024, the proportion of respondents ever tested for HCV increased significantly from 15% to 48%, among whom 49%-74% were aware of their anti-HCV positive status, and history of HCV treatment increased significantly from 6% to 48%. History of drug injection (adjusted odds ratio (aOR)=1.07, $P<0.001$), local Chinese (aOR=2.84, $P=0.015$), and older age (aOR=1.07 per year, $P<0.001$) were associated with self-reported anti-HCV positivity. Known HCV positive respondents aged 31-50 (aged >60 as reference, 31-40 aOR=0.09, 41-50 aOR=0.36, $P<0.001$) and infrequent methadone clinic attendees (full attendance as reference, aOR=0.28, $p=0.003$) were less likely to have received HCV treatment.

Conclusions:

While HCV testing and treatment coverage has increased significantly over the years, treatment uptake was disproportionately low among younger and infrequent methadone clinic attendees despite PWIDs' known HCV positive status. To achieve HCV micro-elimination in this key population, efforts would need to go beyond expanding testing coverage. Promoting awareness of the importance and effectiveness of new HCV treatment, coupled with simplifying referral and linkage pathway would be important to enhance treatment coverage.

Keyword(s) : HCV; PWID; testing and treatment

2831 - Early implementation experience in developing a Research Electronic Data Capture (REDCap) based liver disease biobank platform in a lower middle income country

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Background / Hypothesis:

Liver diseases remain a major and growing public health burden in the Philippines, driven by chronic viral hepatitis, metabolic dysfunction-associated steatotic liver disease, alcohol-related liver disease, and hepatocellular carcinoma. In many lower-middle income countries, translational research is limited by fragmented data systems and inadequate infrastructure for systematic biospecimen collection. As part of a nationally funded multidisciplinary liver research program, we established the Promoting Healthy Filipino Liver through Interdisciplinary Innovative Research (PHIL LIVER) Biobank as foundational infrastructure supporting multiple component studies. We evaluated early implementation focusing on governance, digital architecture, workflow integration, and initial operational performance.

Materials and Methods:

We conducted a prospective implementation study during the establishment of a liver disease biobank within a public academic referral center. The PHIL



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LIVER Biobank accepts biospecimens and associated clinical data from three component projects. A centralized Research Electronic Data Capture (REDCap) digital repository was developed as a single unified project integrating data across project components. The REDCap architecture utilized study arms, data access groups, and role-based permissions to ensure appropriate participant assignment, data segregation, and controlled access. Standard operating procedures were implemented for patient identification, consent, specimen collection, processing, storage, and retrieval. Pilot testing focused on cross-project variable harmonization, refinement of electronic forms, and alignment with routine clinical workflows.

Results:

Within 12 months, governance structures and operational procedures were established. The unified REDCap platform enabled structured data capture and barcode-based specimen tracking across projects, while study arms and data access groups supported secure multi-project functionality within a controlled-access environment.

As of reporting, a total of 37 participants were enrolled, all of whom consented to biobanking. From these participants, 74 plasma aliquots and 74 serum aliquots were processed and stored (two per participant). Additionally, 18 fecal aliquots were collected. All specimens were assigned unique coded identifiers with complete digital traceability in REDCap. No specimen mislabeling events were documented during pilot implementation.

Operational challenges included cross-project variable harmonization, iterative e-form refinement and workflow efficiency.

Conclusions:

Early implementation demonstrates the feasibility of establishing a centralized, multi-project liver disease biobank supported by a unified REDCap architecture in a resource-constrained setting. Integrating multiple studies within a controlled-access digital environment enhances coordination and efficiency but requires substantial effort in harmonization and workflow alignment. The PHIL LIVER Biobank provides a scalable model for strengthening multidisciplinary research infrastructure in developing health systems.

Keyword(s) : liver disease, biobank implementation

translational research, biospecimen collection, health systems strengthening, registry linkage, developing countries, Philippines

2835 - Predictive value of oxygen resaturation rate and MAFLD for left ventricular hypertrophy in obstructive sleep apnea

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Abstract

Background: Left ventricular hypertrophy (LVH) is a prevalent cardiac complication of obstructive sleep apnea (OSA). The oxygen resaturation rate (ORR), a novel metric quantifying post-apneic oxygen recovery, may provide insights into LVH. This study examined the association between ORR and LVH and whether metabolic dysfunction-associated fatty liver disease (MAFLD) influences this relationship.

Methods: This retrospective study included OSA patients without pre-existing LVH who underwent polysomnography from January 2014 to May 2017 at Affiliated Huai'an No.1 People's Hospital of Nanjing Medical University. LVH was assessed via echocardiography and blood tests from December 2023 to May 2024. ORR was calculated from obstructive respiratory events recorded during overnight polysomnography. For each event, Δt was defined as the interval from the SpO_2 nadir to recovery, and ΔSpO_2 as the magnitude of desaturation. ORR, calculated as $\Delta SpO_2 / \Delta t$, represents the rate of reoxygenation and serves as an index of reoxygenation efficiency. Multivariate Cox regression and Kaplan-Meier analyses were performed to examine associations among ORR, MAFLD, and LVH.

Results: The sample include 490 participants (79.0% male) with a median age of 53.0 [42.0,62.0] years). Over a median follow-up of 8.4 years, 85 (17.3%) were diagnosed with LVH. Compared with the lowest tertile, the highest ORR tertile was associated with a higher risk of LVH (adjusted HR 4.93 ; 95% CI 2.28-10.70, $p < 0.001$).

Notably, participants with NAFLD/High-ORR had a higher incidence of LVH than the Non-NAFLD/Low-ORR group (adjusted HR 5.16 ; 95%CI 2.13-12.53, $p < 0.001$). Subgroup analyses indicated stronger associations in participants aged < 65 years or with BMI ≥ 30 kg/m². MAFLD in combination with high AHI or ORR increased the risk of LVH, whereas no increase was observed for T90.

Conclusion: ORR is a novel, sensitive indicator

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associated with the presence of LVH in patients with OSA. A significant interaction between ORR and MAFLD was observed, which was associated with an increased risk of LVH.

Keyword(s) : Oxygen Resaturation Rate (ORR) ; Left Ventricular Hypertrophy (LVH) ; Obstructive Sleep Apnea (OSA) ; Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD)

2836 - Effectiveness and safety of transcatheter arterial chemoembolization with EqualSpheres beads in patients with unresectable hepatocellular carcinoma : a single arm multi center prospective study

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Background / Hypothesis:

This prospective study aimed to investigate the short-term efficacy and safety and outcomes of unresectable hepatocellular carcinoma (uHCC) treated by transarterial chemoembolization (TACE) using EqualSpheres beads (ETACE) .

Materials and Methods:

Eligible patients were given ETACE with graded embolization strategy, until either disease progression or intolerable toxicities emerged. Toxicity was assessed using NCI CTCAE v5.0. Response was assessed using mRECIST criteria. Objective response rates (ORR) and progression-free survival (PFS) was the primary endpoint. Overall survival (OS), disease control rates (DCR), and treatment-related adverse events (TRAEs) were secondary endpoints.

Results:

Total, 302 patients with HCC were successfully enrolled. Median follow-up lasted 14.5 months (95% CI: 11.8–20.5 months). 3 month, 6 month, and 12 month ORR and DCR after the first DEB-TACE were 55.3% and 65.3%, 26.2% and 90.3%, 70.7% and 26.2%, respectively. One-year PFS and OS rate were 54.8 and 90.3%. Median PFS was 9.1 months (95% CI: 8.2–9.6 months) and the median OS was 19.6 months (95% CI: 18.7–NA months). Impressively, 5 (1.7 %) patients were converted to resectable HCC and underwent curative hepatectomy. Majority of TRAEs were mild and manageable. ETACE-related adverse events (AEs) was commonly embolism syndrome such as fever, pain, gastrointestinal reactions, nausea, vomiting, and new ascites. Liver abscess was special phenomena in the study.

Conclusions:

In this prospective study, high ORR and long PFS are observed after ETACE with graded embolization strategy with encouraging results and manageable safety.

Keyword(s) :

2837 - Effects of miR 124 3p Knockdown on Hepatocyte Apoptosis and Protein C Synthesis in Mice with Portal Vein Obstruction

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Background / Hypothesis:

This study employed a mouse model of partial portal vein ligation (PVL). On this basis, a model of miR-124-3p low expression was established. The aim was to investigate the effect of miR-124-3p inhibition on the expression of Protein C (PROC), along with its potential regulatory mechanisms, within a pathological environment simulating portal vein thrombosis.

Materials and Methods:

Forty male C57BL/6J mice (6–8 weeks old) were equally randomized into four groups: Sham+NC, Sham+Inhibitor, PPVL+NC, and PPVL+Inhibitor. PPVL was induced via extrahepatic partial portal vein ligation; sham controls underwent laparotomy with vein isolation only. Model validation involved serum collection at days 7, 14, and 21 post-surgery to measure Protein C (ELISA) and platelet counts. Inhibitor groups received tail vein injections of miR-124-3p-sponge AAV; controls received empty virus. Four weeks post-injection, mice were sacrificed. Liver injury was assessed via serum ALT/AST/LDH. qRT-PCR quantified miR-124-3p and PROC mRNA; Western blot measured PROC, Bax, Bcl-2, and cleaved caspase-3. Apoptosis was evaluated by TUNEL staining.

Results:

Routine blood tests confirmed successful PPVL modeling, revealing significantly lower platelet counts in the PPVL group than in the Sham group. From day 14 post-surgery, Protein C (PROC) levels dropped notably in the PPVL group. Compared to Sham controls, PPVL mice showed reduced PROC mRNA and protein, alongside increased Bax and cleaved caspase-3, and decreased Bcl-2, indicating liver apoptosis. Inhibiting miR-124-3p reversed these changes: PROC rose, apoptosis-related proteins normalized to near-Sham levels, with no difference from Sham controls.



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The inhibitor alone showed no off-target effects in normal liver. TUNEL staining confirmed fewer apoptotic cells in PPVL+Inhibitor vs. PPVL+NC, comparable to Sham, with no effect from inhibitor only.

Conclusions:

This study demonstrated that in a partial portal vein thrombosis model, inhibition of miR-124-3p alleviated hepatocyte apoptosis by modulating the Bcl-2/Bax/caspase-3 apoptotic pathway, thereby promoting PROC synthesis in hepatocytes. These findings provide novel molecular mechanistic insights into the link between hepatocyte injury and coagulopathy in portal hypertension. Consequently, miR-124-3p represents a promising therapeutic target for preventing portal vein thrombosis by enhancing endogenous anticoagulant pathways.

Keyword(s) : miR-124-3p ; Protein C ; Obliterative portal venopathy ; Cell apoptosis

2838 - ONWARD Strengthening the clinical management of zoonotic hepevirus infections through coordinated European network with global linkages

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Background: Hepatitis E virus (HEV) is currently the leading cause of acute viral hepatitis worldwide. While most infections are self-limited, chronic HEV infection has become a significant clinical concern in immunocompromised individuals, particularly solid-organ transplant recipients and patients receiving immunosuppressive therapies. Extrahepatic manifestations, including neurological and renal complications, further complicate diagnosis and management. More recently, human infections caused by rat hepatitis E virus (Rocahepevirus rattii) have expanded the recognized clinical spectrum of zoonotic hepevirus infections in Europe and Asia. Despite these developments, diagnostic algorithms, screening strategies and therapeutic approaches remain heterogeneous across centers and regions. Given growing population mobility and shared clinical challenges, there is a clear need for stronger inter-regional alignment between Europe and the Asia-Pacific in clinical practice and evidence generation. Methods: ONWARD (COST Action CA24140) is a European Union-funded initiative under the COST (European Cooperation in Science and Technology) framework, launched in October 2025 to improve coordination and preparedness in zoonotic hepevirus infections. The Action brings together hepatologists, infectious disease specialists, transplant physicians, microbiologists, veterinarians and public-health experts from 30 European countries and is structured to facilitate global scientific exchange through open dissemination activities, shared protocols and collaborative analyses. Clinical-focused activities include: ii) harmonising molecular and serological testing approaches; (iii) defining shared clinical case definitions and outcome measures; and (iv) facilitating structured data exchange to support multicentre analyses, with emphasis on high-risk populations (transplant and other immunosuppressed patients). Results: The network enables systematic assessment of variability in clinical practice across Europe. Preliminary mapping demonstrates marked differences in chronic HEV screening among transplant recipients, ribavirin dosing strategies, duration of therapy, and follow-up protocols. Consensus-based frameworks for diagnostic algorithms and minimum clinical data sets are being developed, designed to be transferable and comparable across regions. Key research priorities identified include predictors of chronic infection, relapse after antiviral therapy, and the clinical relevance of ratHEV infection in humans. Conclusions: ONWARD represents the first coordinated EU-supported effort to address the clinical challenges

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posed by zoonotic hepevirus infections with an explicit goal of supporting inter-regional convergence in clinical management. By aligning diagnostic standards, strengthening multicenter collaboration and fostering cross-sector communication, the initiative aims to improve patient management and support evidence-based clinical decision-making. This model can serve as a platform for Europe-Asia dialogue on HEV clinical practice, research priorities and preparedness.

Keyword(s) : Hepatitis E, ratHEV

2840 - Ultrasound Based Assessment of Hepatic Steatosis in Rheumatoid Arthritis Patients A Cross Sectional Study

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Background / Hypothesis:

Steatotic liver disease is highly prevalent in the general population. Emerging evidence suggests a possible association between hepatic steatosis (HS) and rheumatoid arthritis (RA), although this relationship has not been sufficiently investigated.

The study aimed to determine the prevalence of hepatic steatosis among patients with rheumatoid arthritis using liver ultrasonography.

Materials and Methods:

This cross-sectional study included 154 patients with RA evaluated between July 2024 and February 2025. Hepatic steatosis was assessed by abdominal ultrasonography. All participants underwent detailed history taking, clinical examination, and anthropometric measurements, including body mass index (BMI), waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR). Disease activity was evaluated using the Disease Activity Score-28 (DAS28). Laboratory investigations included liver biochemical profile, lipid profile, glycated hemoglobin (HbA1c), and inflammatory markers (C-reactive protein and erythrocyte sedimentation rate).

Results:

The majority of patients were female (88.3%), with a median age of 45 years and a median disease duration of 96 months. Hepatic steatosis was detected in 45.5% (70/154) of patients, of whom 70% had moderate-to-severe steatosis. Compared with patients without steatosis, those with steatosis were older and had longer disease duration, higher disease activity, higher prevalence of diabetes mellitus and hypertension, and greater cumulative methotrexate exposure. Patients with steatosis had significantly higher BMI, WC, HC, WHR, WHtR, HbA1c, and lipid profile parameters (triglycerides, total cholesterol, and low-density lipoprotein cholesterol) ($P < 0.001$).

Conclusions:

Hepatic steatosis is common among patients with rheumatoid arthritis. Older age, central obesity, metabolic abnormalities, and diabetes mellitus are associated with increased risk of hepatic steatosis in this population.

Keyword(s) : Keywords: Prevalence; Hepatic Steatosis; Ultrasonography; Rheumatoid Arthritis

2842 - Machine learning-based mortality prediction models for non alcoholic fatty liver disease in the general United States population

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Background / Hypothesis:

Nowadays, the global prevalence of non-alcoholic fatty liver disease (NAFLD) has reached about 25%, which is the most common chronic liver disease worldwide, and the mortality risk of NAFLD patients is higher. Our research created five machine learning (ML) models for predicting overall mortality in ultrasound-proven NAFLD patients and compared their performance with conventional non-invasive scoring systems, aiming to find a generalizable and valuable model for early mortality prediction in NAFLD patients.

Materials and Methods:

National Health and Nutrition Examination Survey (NHANES)-III from 1988 to 1994 and NHANES-III related mortality data from 2019 were used. 70% of subjects were separated into the training set ($N = 2262$) for development, while 30% were in the testing set ($N = 971$) for validation. The outcome was all-cause death at the end of follow-up. Twenty-nine related

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variables were trained as predictor features for five ML-based models: Logistic regression (LR), K-nearest neighbors (KNN), Gradient-boosted decision tree (XGBoost), Random forest (RF) and Decision tree. Five typical evaluation indexes including area under the curve (AUC), F1 score, accuracy, sensitivity and specificity were used to measure the prediction performance.

Results:

3233 patients with NAFLD in total were eligible for the inclusion criteria, with 1231 death during the average 25.3 years follow up time. AUC of the LR model in predicting the mortality of NAFLD was 0.888 (95% confidence interval [CI] 0.867-0.909), the accuracy was 0.808, the sensitivity was 0.819, the specificity was 0.802, and the F1 score was 0.765, which showed the best performance compared with other models (AUC were: RF, 0.876 [95%CI 0.852-0.897]; XGBoost, 0.875 [95%CI 0.853-0.898]; Decision tree, 0.793 [95%CI 0.766-0.819] and KNN, 0.787 [95%CI 0.759-0.816]) and conventional clinical scores (AUC were: Fibrosis-4 Score (FIB-4), 0.793 [95%CI 0.777-0.809]; NAFLD fibrosis score (NFS), 0.770 [95%CI 0.753-0.787] and aspartate aminotransferase-to-platelet ratio index (APRI), 0.522 [95%CI 0.502-0.543]).

Conclusions:

ML-based models, especially LR model, had better discrimination performance in predicting all-cause mortality in patients with NAFLD compared to the conventional non-invasive scores, and an interpretable model like Decision tree, which only used three predictors: age, systolic pressure and glycated hemoglobin, is simple to use in clinical practice.

Keyword(s) : Non-alcoholic fatty liver disease; mortality; machine learning; Logistic regression; Decision tree; NHANES-III.

2843 - IRE1 α mediated downregulation of CYP7A1 drives MASH HCC progression and immune evasion via regulation of cholesterol metabolism

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Background / Hypothesis:

Metabolic dysfunction-associated steatohepatitis (MASH)-related hepatocellular carcinoma (HCC) is rising globally, exhibiting unique features like

metabolic dysregulation and a poorer prognosis than viral HCC. Cholesterol is key in MASH-HCC progression, regulated by CYP7A1, the rate-limiting enzyme in bile acid synthesis. Clinically, CYP7A1 is significantly downregulated in MASH-HCC tumors and linked to worse outcomes, yet its regulatory mechanisms and functional roles are unclear. This study aims to clarify these aspects to identify novel therapeutic targets for MASH-HCC.

Materials and Methods:

Paired tumor/adjacent tissues from MASH-HCC patients were analyzed by metabolomic sequencing, scRNA-seq, and spatial transcriptomics to identify cholesterol metabolism alterations and confirm CYP7A1 downregulation. MASH-HCC mouse models were generated using hepatocyte-specific IRE1 α -KO, Cyp7a1-KI, and Smo-KO mice. ChIP-seq, RNA-seq validated IRE1 α -mediated CYP7A1 regulation. IP/MS, LLPS and FRAP assays characterized IRE1 α lactoylation modification and RIDD activity. In vivo/in vitro experiments assessed the impact of IRE1 α -CYP7A1 axis on cholesterol metabolism and Hedgehog signaling. Therapeutic effects of PD-1 mAb alone or combined with IRE1 α inhibitor/CYP7A1-adenovirus were evaluated in MASH-HCC models to explore clinical translation.

Results:

CYP7A1 is markedly downregulated in MASH-HCC tumor tissues and strongly associated with unfavorable prognosis. Lactoylation of IRE1 α induces LLPS, augmenting its RIDD activity and resulting in direct degradation of CYP7A1 mRNA. Reduced CYP7A1 expression causes aberrant cholesterol accumulation, which activates Hedgehog signaling in both tumor cells and Treg cells through SMO cholesterol modification, thereby accelerating tumor proliferation and immune evasion. Therapeutic targeting of the IRE1 α /CYP7A1 axis effectively restores cholesterol metabolic homeostasis and potentially suppresses tumor progression.

Conclusions:

This study reveals a core mechanism driving MASH-HCC progression: hypoxia-induced IRE1 α lactoylation enhances its LLPS and RIDD activity, specifically degrading CYP7A1 to cause cholesterol accumulation; this accumulation exacerbates ER stress, transactivating IRE1 α to form a self-reinforcing loop that ultimately activates Hedgehog signaling via SMO cholesterol modification to promote carcinogenesis and immune evasion. The study elucidates a novel "metabolism-stress" crosstalk paradigm, offering new targets for MASH-HCC diagnosis and therapy.

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Keyword(s) : MASH-HCC; Endoplasmic reticulum stress; CYP7A1; Cholesterol metabolism; Hedgehog signaling

2845 - Effectiveness and Safety of Glecaprevir Pibrentasvir Combination Therapy in Patients Diagnosed with Chronic Hepatitis C

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Background / Hypothesis: The study aimed to evaluate the effectiveness and safety of glecaprevir/pibrentasvir (GLE/PIB) treatment in patients infected with hepatitis C virus (HCV).

Materials and Methods: Forty-four patients who applied to the Infectious Diseases and Clinical Microbiology Clinic between December 2019 and December 2022 were infected with HCV genotype 1–6 and came regularly for treatment and follow-up visits were included in the study. The study was conducted retrospectively by accessing patient data through the hospital information management system. In patients receiving GLE/PIB treatment, data regarding the effectiveness and reliability of the treatment were recorded both during the treatment process and after the treatment was terminated.

Results: Of the 44 patients, 47.7% were genotype 1b, 18.2% were genotype 1a, 15.9% were genotype 2, 13.6% were genotype 4, and 4.5% were infected with genotype 3. Early virological response (EVR) was achieved in 81.8% of 44 patients. End-of-treatment response and sustained virological response at the 12th week were achieved in all patients. No treatment failures were observed. There was no significant difference between EVR rates according to genotypes. There was a significant improvement in the liver function tests of the patients from the 4th week of treatment. The most common adverse events were fatigue and itching.

Conclusions: The GLE/PIB combination is an effective and safe treatment option in the treatment of HCV infection. Due to the high EVR rates, more comprehensive studies need to be conducted to keep the duration of GLE/PIB treatment shorter in some patient groups in patients diagnosed with CHC.

Keyword(s) : Effectiveness, glecaprevir, hepatitis c, pibrentasvir, safety

2846 - Prevalence of Sarcopenia in Cirrhotic Patients and Its Association with Prognosis

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Background / Hypothesis: Sarcopenia is closely associated with quality of life, the development of complications, and mortality in patients with liver cirrhosis, and is considered a systemic reflection of hepatic functional reserve. In this study, we aimed to evaluate the prevalence of radiologically defined sarcopenia in patients with liver cirrhosis and to investigate its associations with cirrhosis stage, sex, body mass index (BMI), presence of ascites, etiology, and metabolic markers (vitamin D, IGF-1). **Materials and Methods:** A total of 90 patients with cirrhosis were included in this retrospective single-center study, comprising 30 patients each with Child-Pugh class A, B, and C disease. Demographic data, body mass index (BMI), presence of ascites, cirrhosis etiology, MELD-Na score, and metabolic markers (vitamin D, IGF-1) were recorded for all patients. Sarcopenia was assessed using the skeletal muscle index (SMI) measured by computed tomography at the level of the third lumbar vertebra (L3) and was coded according to radiological definition as present = 1 and absent = 0. Vitamin D levels were classified as normal (>30 ng/mL), insufficient (20–30 ng/mL), deficient (10–20 ng/mL), and severely deficient (<10 ng/mL). IGF-1 levels were categorized as low or normal.

Statistical analyses were performed using the chi-square test, independent samples t-test, and Pearson correlation analysis, with $p < 0.05$ considered statistically significant.

Results: Sarcopenia was identified in 61.8% of patients (55/90). Its prevalence increased with advancing Child-Pugh stage (46% in Child A, 60% in Child B, and 78% in

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Child C; $p = 0.04$). Sarcopenia was significantly more frequent in men (73%; $p = 0.03$). No significant associations were observed between sarcopenia and BMI, MELD-Na score, or presence of ascites ($p > 0.05$).

Vitamin D levels were <30 ng/mL in 92% of patients and were lower in the Child C group; however, the association between vitamin D categories and sarcopenia was borderline significant ($p \approx 0.06$). Although low IGF-1 levels were common, they were not significantly associated with sarcopenia ($p > 0.05$). SMI showed a moderate negative correlation with age ($r = -0.38$; $p = 0.02$), indicating decreased muscle mass with advancing age.

Conclusions: Radiologically assessed sarcopenia demonstrates a high prevalence in patients with cirrhosis and is significantly associated with advanced Child-Pugh stage and male sex. Although vitamin D deficiency and low IGF-1 levels are common, their associations with sarcopenia require confirmation in larger cohorts. These findings suggest that integrating CT-based muscle mass assessment into routine clinical follow-up may facilitate early identification of high-risk patients in the management of

Keyword(s) : Sarcopenia, Liver Cirrhosis, Vitamin D, IGF-1

2847 - influential factors associated with the reduction of peripheral blood neutrophils by PegIFN α and predictive analyses

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Background / Hypothesis:

The aim of this study was to understand the effect of PegIFN α on peripheral blood NE, to analyse the relevant influencing factors of moderate to severe NE reduction, and to establish a corresponding prediction model.

Materials and Methods:

Using the data from the Pegabine IIa, IIb and III clinical trials, the clinical data were collected and analysed at baseline and during PegIFN α treatment. All patients were categorised into a moderately severely reduced NE group and a non-moderately severely reduced group according to whether the absolute value of NE decreased to $<0.75 \times 10^9/L$ during treatment.

Results:

A total of 2039 subjects (mean age 34.4 years, 62% male) were included in the clinical trial, 98.4% of patients with The decrease in absolute NE value from baseline appeared in the first 4 weeks of starting treatment, and after the first injection of PegIFN α , 91% showed a decrease in absolute NE value, of which 42.9% showed a decrease of more than 50% from baseline, and then it stabilised at a lower level and gradually returned to the baseline level after discontinuation of the drug.

Throughout the course of treatment, 17.8% of patients experienced moderate-to-severe reductions in absolute NE values ($<0.75 \times 10^9/L$), with a mean time to first moderate-to-severe reduction in NE of 11.0 weeks (standard deviation: 12.2), of which those who experienced moderate-to-severe reductions in NE in the first 12 weeks accounted for 72.9% of all patients with moderate-to-severe reductions in NE.

A prediction model was developed according to the above indicators and age, gender, and patient weight at week 12, with an AUC of 0.89 (cutoff value of 0.283), sensitivity of 0.74, and specificity of 0.88 (Figure 2).

Conclusions:

The vast majority of patients showed an absolute reduction in NE early in PegIFN α treatment, with only a few showing a moderate-to-severe reduction, and the vast majority of the first moderate-to-severe reductions occurred in the first 12 weeks. Baseline low BMI, white blood cell count and absolute NE values, alkaline phosphatase values, the time of occurrence of the lowest NE values, and the decreases in NE at weeks 4 and 12 were associated with the occurrence of moderate-to-severe decreases in NE during the course of treatment, and the prediction model developed on this basis could more accurately identify patients with moderate-to-severe decreases in NE during the course of PegIFN α treatment.

Keyword(s) : PegIFN α , severe NE reduction

2849 - A COMPUTATIONAL INTELLIGENCE APPROACH TO PREOPERATIVE LIVER FUNCTION ASSESSMENT

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Background / Hypothesis:

Assessing liver function is a prerequisite for safe hepatobiliary surgery. This study explores a computational intelligence approach to provide a more precise preoperative evaluation.

Materials and Methods:

In this study, artificial intelligence methods were used to assess liver function. A Random Forest algorithm was applied to a clinical dataset of biochemical markers sourced from a liver patient dataset. The model evaluated the diagnostic significance of various liver enzymes to establish a predictive framework.

Results:

The computational model demonstrated exceptional performance in risk assessment, yielding a predictive accuracy of 98.8% and an AUC-ROC of 0.9975. Beyond global metrics, the Random Forest feature importance analysis revealed that SGPT (Alanine Aminotransferase) and Total Bilirubin were the most statistically significant markers, accounting for the highest computational weight in determining pathological status. The model also showed a high F1-score of 0.99, indicating a balanced precision-recall trade-off that is crucial for minimizing false negatives in preoperative settings

Conclusions:

A computational intelligence approach significantly enhances the reliability of preoperative liver function assessment. This technology offers a data-driven tool for clinicians to optimize surgical risk management.

Keyword(s) :

2850 - Comparative efficacy of ursodeoxycholic acid and s adenosyl methionine in reducing transaminases in patients with non alcoholic steatohepatitis a retrospective prospective cohort study

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Background / Hypothesis:Non-alcoholic steatohepatitis (NASH) is a progressive form of fatty liver disease associated with hepatic inflammation and fibrosis, for which standardized pharmacologic therapies remain limited. Ursodeoxycholic acid and s-adenosyl methionine have demonstrated hepatoprotective effects in non-alcoholic steatohepatitis. This study

compared the efficacy of these agents in reducing serum transaminases among patients with non-alcoholic steatohepatitis.

Materials and Methods: This retrospective-prospective cohort study included adult patients with clinically suspected non-alcoholic steatohepatitis managed at a tertiary care center. Patients received either ursodeoxycholic acid for three month or s-adenosyl methionine for one month. Serum alanine aminotransferase, aspartate aminotransferase, platelet count, and fibrosis-4 index were measured at baseline and after treatment. Descriptive statistics were used to summarize baseline characteristics. Between-group comparisons were performed using independent t-tests or Mann-Whitney U tests, while within-group changes were assessed using Wilcoxon signed-rank tests. A two-tailed p value <0.05 was considered statistically significant.

Results: A total of 120 patients were included, with 64 receiving ursodeoxycholic acid and 56 receiving s-adenosyl methionine. Baseline demographic characteristics and comorbidities were comparable between groups. Baseline alanine aminotransferase and aspartate aminotransferase levels were higher in the s-adenosyl methionine group. After therapy, both treatment groups demonstrated significant reductions in alanine aminotransferase and aspartate aminotransferase compared with baseline (all p<0.001). Post-treatment alanine aminotransferase levels were significantly lower in the ursodeoxycholic acid group compared with the s-adenosyl methionine group (p=0.019), while post-treatment aspartate aminotransferase levels showed a similar trend without reaching statistical significance. Fibrosis-4 index decreased significantly from baseline in both groups, with no significant difference between treatments.

Conclusions:Both ursodeoxycholic acid and s-adenosyl methionine were associated with significant improvement in serum transaminases and fibrosis surrogate markers in patients with non-alcoholic steatohepatitis. While both agents demonstrated biochemical efficacy, ursodeoxycholic acid was associated with lower post-treatment alanine aminotransferase levels. These findings support the use

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of either therapy in clinical practice, with further studies needed to evaluate long-term outcomes.

Keyword(s) : Non-alcoholic steatohepatitis, fatty liver disease, transaminases, ursodeoxycholic acid, s-adenosyl methionine, FIB-4

2852 - Homocysteine is a promising biomarker for predicting acute kidney injury in patients with cirrhosis

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Background / Hypothesis:

Acute kidney injury (AKI) occurs very commonly in cirrhotic patients and is associated with poor prognosis. Early identification of AKI is an unmet clinical need. Homocysteine (Hcy) is a classic biomarker for vascular dysfunction, which plays a key role in the pathophysiology of AKI in cirrhosis. Herein, we aimed to assess the predictive value of serum Hcy for AKI in cirrhotic patients.

Materials and Methods:

A prospective series of 463 hospitalized cirrhotic patients was included. Serum samples were collected at admission, and Hcy was measured. The presence of AKI during hospitalization was carefully assessed. Multivariate regression and propensity score matching (PSM) were conducted to examine the association between Hcy and AKI. ROC analysis was then employed to assess the predictive value of Hcy for AKI. Integrated discrimination improvement (IDI) was calculated to assess the impact on published AKI prediction models with the addition of Hcy. Biomarkers of renal dysfunction, such as neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, and liver fatty acid binding protein, were also measured for comparison.

Results:

During hospitalization, 35 cirrhotic patients developed AKI. PSM and multivariate analyses showed that among four biomarkers, only serum Hcy was significantly associated with AKI (OR=1.165, 95% CI: 1.035-1.337). Moreover, serum Hcy could be used to predict the development of AKI in cirrhosis, with an AUC of 0.768 (95% CI: 0.686-0.850). Incorporating Hcy

into existing AKI models substantially enhanced their predictive efficacy in patients with cirrhosis, with the IDI values ranging from 0.072 to 0.106.

Conclusions:

The application of serum Hcy may help to identify cirrhotic patients at risk for developing AKI at an early stage. Exploration of the new role for some old biomarkers may be a cost-effective way for the prediction of AKI.

Keyword(s) : Homocysteine, Acute kidney injury, Cirrhosis, Predictive

2853 - Metabolic Dysfunction Associated Fatty Liver Disease Signalling and Organ Dialogue within the Systemic Metabolic Network

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a multisystem disorder driven primarily by metabolic dysfunction, involving coordinated dysregulation across the liver, adipose tissue, skeletal muscle, gut, and cardiovascular systems. The disease progresses dynamically from steatosis to cirrhosis and hepatocellular carcinoma. The prevailing 'multiple hit' hypothesis fails to explain the stable transmission of cross-organ pathological signals and their cell-specific targeting. Exosomes, as lipid bilayer vesicles measuring 30–150 nm in diameter, possess structural stability, organ-targeting capacity, and information complexity by carrying active molecules such as proteins and nucleic acids. This renders them a pivotal link connecting the multi-organ pathological networks of MAFLD. Research indicates markedly elevated liver-specific miRNAs in circulating exosomes from MAFLD patients, whilst hepatogenic exosomes induce myocardial insulin resistance and renal podocyte apoptosis. Given the multi-organ collaborative nature of MAFLD imposing stringent demands on signal transmission, systematic investigation of exosomal mechanisms is not only theoretically imperative but also provides a critical breakthrough for developing novel therapeutic strategies targeting inter-organ communication.

Materials and Methods:None

Results:None



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Conclusions:None

Keyword(s) : MAFLD; exosomes; multi-organ crosstalk;

2854 - Nanotherapy based on Mesenchymal stem cell derived extracellular vesicles target ADAM17 mediated TREM2 shedding on macrophages to promote efferocytosis for acute liver failure treatment

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Background / Hypothesis :

Acute liver failure (ALF) is a life-threatening condition with high mortality and limited therapeutic options. Impaired macrophage efferocytosis contributes to unresolved inflammation and liver injury during ALF. Mesenchymal stem cell-derived extracellular vesicles (MSC-EVs) represent a promising cell-free therapy, but their mechanisms remain unclear. We hypothesized that adipose-derived MSC-EVs (AMSC-EVs) alleviate ALF by restoring macrophage efferocytosis through regulation of TREM2 signaling.

Materials and Methods:

ALF was induced in mice using lipopolysaccharide/D-galactosamine (LPS/D-GalN). Liver injury, inflammatory responses, and macrophage efferocytosis were evaluated following AMSC-EV treatment. TREM2 knockout macrophages and myeloid-specific TREM2-deficient mice were used to determine TREM2 dependency. Mechanistic studies assessed TREM2 expression, ADAM17 activity, and NF- κ B (p65)-mediated transcriptional regulation.

Results:

AMSC-EVs significantly attenuated liver injury and promoted inflammatory resolution in ALF. Treatment restored macrophage efferocytosis and prevented the loss of full-length TREM2 in liver tissue and on macrophage membranes after inflammatory stimulation. These protective effects were abolished in TREM2-deficient macrophages and mice, demonstrating a TREM2-dependent mechanism. Mechanistically, AMSC-EVs suppressed ADAM17-mediated TREM2 cleavage by inhibiting p65-dependent ADAM17 transcription, leading to enhanced efferocytosis and increased production of anti-inflammatory and pro-resolving cytokines.

Conclusions:

AMSC-EVs ameliorate ALF by restoring macrophage efferocytosis. This study elucidates the mechanism of AMSC-EVs in targeting macrophage dysfunction in ALF through inhibition of p65-dependent ADAM17 transcription and subsequent suppression of ADAM17-mediated TREM2 shedding, highlighting AMSC-EVs as a potential nanotherapeutic strategy for ALF.

Keyword(s) : acute liver failure, macrophages, mesenchymal stem cells, extracellular vesicles, TREM2

2855 - Construction of an Intelligent Early Warning System for Chronic Liver Disease Comorbidities Based on Multimodal Data Fusion and Traditional Chinese Medicine Constitution

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Background / Hypothesis: Chronic liver disease (CLD) has a high global prevalence and frequently coexists with metabolic disorders, making early prevention and control challenging. Traditional Chinese Medicine (TCM) constitution theory posits that individual constitutional differences determine disease susceptibility. We hypothesize that integrating TCM constitution identification with modern multimodal data to establish a "liver-gut homeostasis" assessment system may provide a novel strategy for early warning and stratified intervention in CLD comorbidities.

Materials and Methods: Drawing upon TCM theory and contemporary advances in hepatology, microecology, and systems biology, we propose a risk prevention and control framework for chronic liver disease based on multimodal data fusion. This framework incorporates artificial intelligence techniques to integrate heterogeneous data sources and construct a comprehensive risk evaluation model.

Results: We propose an integrative prevention and control paradigm centered on "liver-gut homeostasis," combining Eastern and Western medical perspectives. A multimodal risk stratification model was developed using tongue and pulse characteristics, imaging data, microbiome profiles, and metabolomics signatures. Dynamic network biomarker analysis was applied to identify pathological transition windows, such as the

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acceleration phase of liver fibrosis. This approach establishes a closed-loop pathway of “constitution identification – risk prediction – window-based intervention – outcome feedback.”

Conclusions: The integration of TCM constitution theory with multimodal data provides both a conceptual framework and a technical pathway for the “pre-disease prevention” of chronic liver disease comorbidities. Future efforts should focus on developing intelligent early-warning systems and stratified intervention strategies, thereby transforming liver disease management from single-disease treatment toward personalized and precision health management.

Keyword(s) :

2856 - Association of Pre transplant Abstinence Duration

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Background / Hypothesis:

This study aimed to evaluate the post-transplant patient survival, graft survival, and alcohol relapse rates in recipients with alcohol-related liver disease (ALD) who underwent early liver transplantation (LT) with pre-transplant abstinence <6 months. Furthermore, we sought to investigate the correlation between the duration of pre-transplant alcohol abstinence and post-transplant prognosis.

Materials and Methods:

A single-center retrospective cohort study was performed. A total of 104 consecutive adult patients diagnosed with ALD who underwent primary LT at the Affiliated Hospital of Qingdao University from July 2016 to December 2024 were enrolled. The cohort was stratified into two groups based on pre-transplant abstinence duration: the early liver transplantation (ELT) group (abstinence <6 months, n=41) and the standard liver transplantation (SLT) group (abstinence ≥6 months, n=63). Survival curves were constructed using the Kaplan–Meier method and compared via the log-rank test. A propensity score-based inverse probability of treatment weighting (IPTW) adjusted

Cox proportional hazards regression model was applied to assess the independent impact of short abstinence duration on the risks of post-transplant mortality and graft failure.

Results:

The study cohort comprised 103 males and 1 female, with a median age of 52 years (range: 31–70 years) at transplantation. The median follow-up period was 605 days (interquartile range: 263–1378 days). The 1-, 5-, and 8-year cumulative patient survival rates were 97.6%, 95.1%, and 95.1% in the ELT group, compared with 93.7%, 93.7%, and 93.7% in the SLT group, respectively (log-rank P=0.730). After IPTW adjustment, pre-transplant abstinence <6 months was not associated with an increased risk of mortality (hazard ratio [HR]=0.46, 95% confidence interval [CI]: 0.08–2.62, P=0.380) or graft failure (HR=0.96, 95%CI: 0.20–4.61, P=0.961). Overall, 14.4% (15/104) of patients experienced post-transplant alcohol relapse, predominantly mild relapse. No significant intergroup difference was observed in the incidence of alcohol relapse (P=0.892).

Conclusions:

In adult LT recipients with ALD, a pre-transplant abstinence duration of less than 6 months is not a significant independent predictor of patient mortality or graft failure. Additionally, it does not significantly elevate the risk of post-transplant alcohol relapse.

Keyword(s) : Alcohol-Related Liver Disease ; Liver Transplantation ; Abstinence Duration; Prognosis ; Alcohol Relapse

2857 - Integration of Disease Susceptibility State Theory and Multimodal Artificial Intelligence A Novel Paradigm for Early Warning of Chronic Liver Disease

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Background / Hypothesis: The Traditional Chinese Medicine (TCM) theory of “treating disease before its onset” proposes that diseases undergo a latent, preclinical stage known as the “pre-disease” (Wei Bing) state, characterized by increased susceptibility but absence of overt clinical manifestations. Chronic liver diseases (CLDs), including metabolic dysfunction–

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associated steatotic liver disease (MASLD), cirrhosis, and hepatocellular carcinoma, typically develop insidiously and are difficult to detect at an early stage. We hypothesize that the TCM-defined “pre-disease” state in liver disorders has a specific biological basis. Elucidating its underlying “disease susceptibility state theory” may provide novel insights for early warning and precision prevention of CLD.

Materials and Methods: An interdisciplinary literature synthesis was conducted across modern hepatology, systems biology, microecology, and artificial intelligence. Based on this integration, we constructed a multidimensional knowledge framework that links the TCM “pre-disease” theory to liver pathophysiology and AI-driven early-warning models.

Results: The TCM “pre-disease” state in liver disorders corresponds to a dynamic and multidimensional phase of heightened disease susceptibility. Its biological underpinnings include redox imbalance and lipid peroxidation, disruption of the gut microbiota, and dysfunction of the gut–liver axis. Dynamic network biomarkers can identify critical pathological transition windows, such as the pre-decompensation stage of cirrhosis. Furthermore, multimodal deep learning models integrating tongue and pulse features, imaging data, and metabolomic profiles enable individualized risk prediction for chronic liver disease.

Conclusions: The disease susceptibility state theory establishes a novel paradigm for precision prevention of chronic liver disease. Future research should focus on developing dynamic monitoring networks that integrate macroscopic TCM phenotypic features with multi-omics molecular data, as well as artificial intelligence–driven early warning and intervention decision systems. Such advances may shift liver disease management from reactive treatment toward proactive prevention at the pre-disease stage.

Keyword(s) :

2858 - Compound Kushen Injection inhibits the formation of malignant ascites by modulating metabolites in malignant hepatocellular carcinoma ascites and influencing immune cells.

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Background / Hypothesis:

Malignant ascites in advanced hepatocellular carcinoma lacks effective treatments. Compound Kushen Injection, a safe traditional Chinese medicine, may inhibit ascites by regulating phosphatidylcholine metabolism and immune function. This study investigates its mechanism in a mouse model to support clinical application.

Materials and Methods:

This study established a Balb/c mouse model of malignant hepatocellular carcinoma ascites. Mice were divided into model and CKI low- (50μL), medium- (100μL), and high-dose (150μL) groups, treated for 15 days. In vivo imaging on days 3,7,11,15 tracked ascites and tumors. LC-MS analyzed metabolites; H&E staining assessed peritoneal invasion. Immunohistochemistry and flow cytometry evaluated T cells, macrophages, and NK cells.

Results:

Compared with the model group, medium/high-dose CKI reduced ascites (11.6%/19.9%) and prolonged survival (22.5%/37.8%). Metabolomics revealed downregulated CHKa and choline phosphotransferase in COP-choline pathways. H&E staining showed reduced peritoneal hyperplasia, fibrosis, and immune infiltration. VEGF expression decreased. Flow cytometry indicated increased CD8+ T cells and an elevated M1/M2 macrophage ratio.

Conclusions:

Compound Kushen Injection reduces malignant ascites in HCC mice by modulating choline metabolism and enhancing immunity, supporting its therapeutic potential and future clinical application.

Keyword(s) : hepatocellular carcinoma malignant ascites, Compound Kushen Injection, immune cells, phosphatidylcholine

2861 - Diagnosis of acute hepatitis C

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Ktu, Gastroenterology, Trabzon-Türkiye ¹

Gastroenterology, Gastroenterology, Trabzon-Türkiye ²

Background / Hypothesis:

Diagnosing acute hepatitis C is difficult in daily practice because there is no specific serological marker. We present a case of a patient who presented with elevated liver enzymes and jaundice and an acute HCV infection diagnosed.

Materials and Methods:

Case

A 42-year-old male patient, who underwent surgery in 2017 for a cholecystectomy and in 2023 for a meningioma and who has been using levetiracetam, presented with elevated liver enzymes and jaundice. Laboratory tests performed at the time of the admission to determine the etiology of the acute hepatitis showed negative results for viral hepatitis (anti-HAV IgG, HBs, anti-HBs, anti-HBc IgG, anti-HCV). Autoimmune hepatitis markers were weakly positive for ANA and negative for the others (SMA, SLA/LP, LKM-1, LC1, AMA, AMA M2, ASMA, LKM). The ceruloplasmin level was 0.26 g/L. Abdominal imaging was negative for acute hepatitis etiology. Although levetiracetam is generally considered to have a low risk of hepatotoxicity because it undergoes minimal metabolism in the liver compared to many other antiepileptic drugs, cases of drug-induced liver injury (DILI) and even acute liver failure have been documented. Given all these findings, we admitted the patient suspecting DILI, monitored them under supportive care, and initiated pregabalin therapy in consultation with neurology instead of levetiracetam. Although the patient's clinical course remained stable during follow-up, no significant improvement was observed in liver function tests. Therefore, diagnoses other than DILI were reevaluated, and acute viral hepatitis markers were requested again. Surprisingly, the anti-HCV test was reported as positive in this case. The patient, who has been monitored since 2017 and whose anti-HCV results were negative in the system, also had no suspicious contact history in his history. However, the patient who tested positive for HCV RNA was diagnosed with acute hepatitis C, and treatment was initiated with a combination of 100 mg glecaprevir and 40 mg pibrentasvir (Table).

Conclusions:

Anti-HCV antibodies typically become positive 6–12 weeks after infection; therefore, tests performed during the acute phase may be negative. In this case, a preliminary diagnosis of toxic hepatitis was made due to the initial negativity of anti-HCV and other diagnostic tests, but the diagnosis of acute hepatitis C

was confirmed by positivity in repeated serology tests and HCV RNA. As in this case, it would be beneficial to keep in mind that viral markers that are negative in the early stages should be rechecked, especially in cases where another definitive diagnosis cannot be

Keyword(s) : Acute hepatitis C

2862 - Based Prediction of Metabolic Dysfunction Associated Fatty Liver Disease A Meta Analysis of Algorithmic Performance

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a leading cause of chronic liver disease globally. Increasing availability of large-scale clinical and metabolic datasets has facilitated the development of machine learning models for early detection and risk stratification. However, reported predictive performance varies across studies. This meta-analysis evaluates algorithmic approaches for MAFLD classification and prediction

Materials and Methods:

A systematic search of PubMed, Embase, and Web of Science (2014–2025) identified studies applying ML or deep learning algorithms for MAFLD detection without experimental intervention. Extracted data included model type, dataset characteristics, feature categories, and AUC values. A random-effects meta-analysis was performed to calculate pooled diagnostic performance. Subgroup analyses compared traditional regression, tree-based ensemble models, and deep learning approaches.

Results:

Twenty-eight studies met inclusion criteria. The pooled AUC for ML-based MAFLD prediction was 0.87 (95% CI: 0.84–0.90). Tree-based ensemble models demonstrated

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slightly higher pooled performance (AUC 0.89) compared to logistic regression (AUC 0.81). Deep learning approaches showed high accuracy in imaging-based datasets but greater heterogeneity. Feature engineering and dataset size significantly influenced performance stability

Conclusions:

Machine learning algorithms demonstrate robust predictive capability for MAFLD detection, with ensemble and deep learning models outperforming conventional statistical methods. Standardized validation frameworks and transparent reporting are necessary to enhance reproducibility and facilitate broader implementation.

Keyword(s) :

2863 - Artificial Intelligence Driven Imaging Analytics in Liver Disease A Systematic Review

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Background / Hypothesis:

AI-based imaging analytics has become a transformative tool in hepatology. Automated interpretation of ultrasound, CT, and MRI reduces observer variability and improves early detection of liver pathology.

Materials and Methods:

A systematic review of studies (2015–2025) was conducted in PubMed and IEEE Xplore. Included studies evaluated deep learning or machine learning models for liver lesion classification or fibrosis staging. Performance indicators such as AUC, accuracy, and sensitivity were extracted and compared.

Results:

Convolutional neural networks (CNNs) demonstrated

pooled AUC values above 0.90 in differentiating benign and malignant lesions. Ensemble approaches improved robustness across imaging modalities. However, external validation datasets were limited, and model interpretability remained insufficient.

Conclusions:

AI-driven imaging systems show strong diagnostic performance in hepatology. Standardized multicenter datasets and explainable AI frameworks are required to enhance clinical reliability

Keyword(s) : Artificial Intelligence; Deep Learning; Liver Imaging; CNN; Diagnostic Accuracy

2864 - Natural Language Processing for Hepatology Knowledge Mining A Review of Algorithmic Advances

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Background / Hypothesis:

The rapid growth of hepatology literature necessitates automated knowledge extraction systems. NLP and transformer-based models offer scalable solutions for text classification and semantic analysis.

Materials and Methods:

Peer-reviewed studies (2016–2025) applying NLP in liver disease research were analyzed. Methods included BERT-based classification, topic modeling, and knowledge graph construction. Evaluation metrics included F1-score and precision

Results:

Transformer architectures consistently outperformed traditional ML models in text classification tasks (F1 >0.88). Knowledge graph approaches improved semantic retrieval and cross-disciplinary mapping. Limitations included domain-specific vocabulary challenges

Conclusions:

NLP technologies significantly enhance knowledge



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synthesis in hepatology. Future research should emphasize multilingual corpora and domain-adapted pretrained models.

Keyword(s) : Natural Language Processing; Large Language Models; Text Mining; Hepatology; Knowledge Graph

2865 - Machine Learning Models for Autoimmune Liver Disease Classification A Meta Analysis

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Background / Hypothesis:

Immune-mediated chronic liver diseases, including autoimmune hepatitis (AIH) and overlap syndromes, present diagnostic complexity due to heterogeneous serological and biochemical features. Increasingly, machine learning (ML) algorithms have been developed to improve classification accuracy and reduce reliance on subjective scoring systems. However, reported model performance varies substantially across studies. This meta-analysis aims to systematically evaluate algorithmic performance in AIH classification and risk prediction.

Materials and Methods:

A systematic literature search (2013–2025) was conducted in PubMed, Web of Science, and Embase. Studies were included if they developed ML-based classification or prediction models for immune-mediated liver diseases without interventional design. Extracted data included model type, feature categories (autoantibodies, laboratory indices, demographic data), validation methods, and AUC values. A random-effects meta-analysis was applied to calculate pooled diagnostic performance. Subgroup analyses compared logistic regression, support vector machines, and ensemble tree-based methods.

Results:

Twenty-four studies met inclusion criteria. The pooled AUC across ML models was 0.86 (95% CI: 0.83–0.89). Ensemble algorithms demonstrated superior stability (AUC 0.89) compared to traditional regression models (AUC 0.81). Feature selection techniques significantly influenced performance heterogeneity ($P = 0.048$).

Models integrating serological and biochemical features outperformed single-modality datasets.

Conclusions:

Machine learning algorithms provide robust classification performance in immune-mediated chronic liver diseases. Standardized feature engineering and transparent reporting are essential to enhance reproducibility and cross-population applicability.

Keyword(s) :

2866 - Computational Network Modeling in Immune Mediated Liver Diseases A Systematic Review

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Background / Hypothesis:

Immune-mediated liver diseases involve complex inflammatory and signaling cascades that are difficult to interpret using linear analytical methods. Computational network modeling has emerged as a powerful approach to identify key regulatory hubs and pathway interactions. This review summarizes algorithm-driven network analyses in immune-mediated liver disorders.

Materials and Methods:

A structured review of studies (2012–2025) applying protein-protein interaction networks, pathway enrichment analysis, graph theory metrics, and multi-omics integration was performed. Data sources included PubMed and Scopus. Analytical approaches were categorized into topological network analysis, modular clustering, and integrative multi-layer networks. Performance was assessed based on pathway enrichment consistency and hub gene reproducibility.

Results:

Network centrality analyses consistently identified inflammatory hubs such as TNF signaling, IL-6 pathways, and JAK-STAT cascades. Multi-omics

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integration improved detection of cross-pathway interactions compared to single-layer transcriptomic models. Graph-based clustering revealed immune-regulatory modules linked to fibrosis progression. However, reproducibility was influenced by dataset heterogeneity.

Conclusions:

Computational network modeling enhances mechanistic interpretation of immune-mediated liver diseases and supports biomarker prioritization. Future research should emphasize standardized data preprocessing and dynamic network modeling approaches.

Keyword(s) : Network Analysis; Systems Biology; Autoimmune Liver Disease; Bioinformatics; Multi-omics Integration

2867 - Superior Survival with Direct Surgery Over TACE Based Strategies in Hemodynamically Stable Patients with Spontaneously Ruptured HCC A Multicenter Propensity Score Matched Cohort Study

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Background / Hypothesis:

Spontaneously ruptured hepatocellular carcinoma (srHCC) is a life-threatening emergency. For hemodynamically stable patients with early-stage (BCLC A) disease, whether to proceed with direct surgical resection or initial transarterial chemoembolization (TACE) remains a critical, evidence-scarce clinical dilemma.

Materials and Methods:

In this multicenter, retrospective cohort study of 244 patients (189 after propensity score matching), patients treated with direct surgery had significantly better overall and recurrence-free survival than those whose treatment began with TACE (with or without subsequent surgery).

Results:

After 1:1:1 propensity score matching (n=189), the direct surgery group had significantly improved OS and RFS compared to both the TACE-plus-surgery group (OS: $P < 0.001$; RFS: $P = 0.007$) and the TACE-alone group (both $P < 0.001$). Initial treatment strategy was the strongest independent prognostic factor. A clinical prediction model incorporating treatment

modality and postoperative markers showed robust accuracy for recurrence (area under the curve: 0.85 in training, 0.79 in validation). Translational analyses suggested that TACE-associated hypoxia may promote a pro-recurrence tumor microenvironment, potentially through upregulation of the PLAUR gene.

Conclusions: In this study, direct surgical resection was associated with significantly better long-term survival than strategies beginning with TACE for patients with BCLC A srHCC. These findings suggest that upfront TACE should not be routine for this specific patient population and support prioritizing direct surgery when feasible.

Keyword(s) : Spontaneously ruptured hepatocellular carcinoma; Transarterial chemoembolization; HIF-1 α ; PLAUR; M2 macrophage polarization; Tumor recurrence.

2868 - High prevalence of coronary atherosclerosis in HBV related hepatocellular carcinoma and liver cirrhosis a cross sectional study

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Background / Hypothesis:

Chronic liver disease (CLD) is increasingly linked to cardiovascular mortality. However, coronary artery disease (CAD) risk stratification across the CLD spectrum—specifically chronic hepatitis B (CHB), liver cirrhosis (LC), and hepatocellular carcinoma (HCC)—remains inadequately defined. Coronary artery calcification (CAC), measured by Agatston score (CACS), is a robust predictor of CAD. This study aimed to compare CAC prevalence and burden across CHB, LC, and HCC cohorts to evaluate CAD risk stratification, with particular emphasis on the HCC population.

Materials and Methods:

1,244 consecutive HBV-related CLD patients were retrospectively enrolled and stratified: CHB (n=575), LC (n=543), and HCC (n=126). Baseline clinical and laboratory data were collected. All participants underwent coronary computed tomography angiography for CAC assessment. Primary outcomes included CAC prevalence, mean CACS, severity grades,

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and major vessel involvement. CAD risk was stratified by CACS (0, 1-99, 100-400, >400). Statistical comparisons utilized Kruskal-Wallis and Chi-square tests.

Results:

CAC prevalence increased progressively: 32.9% (CHB), 47.3% (LC), and 61.9% (HCC). Moderate-to-severe risk (CACS>100) was significantly higher in HCC (19.0%) versus LC (10.5%) and CHB (4.7%) ($p<0.001$). HCC patients showed the highest multi-vessel involvement (≥ 2 arteries: 39.7% vs. 20.5% LC vs. 15.1% CHB), particularly in LAD (54.0%) and RCA (39.7%) ($p<0.001$). Additionally, HCC patients exhibited severe liver, hematological, and metabolic derangements, synergistic risk factors for atherosclerosis.

Conclusions: Patients with HCC exhibit substantially elevated CAD risk compared to LC or CHB, evidenced by higher CAC prevalence, burden, and multi-vessel involvement. These findings highlight a critical need to prioritize cardiovascular assessment in HCC management. The confluence of systemic inflammation and metabolic dysfunction in HCC likely accelerates atherosclerosis. We advocate for routine CAC screening in HCC patients and suggest LC and HCC status be considered significant modifiers in CAD risk stratification.

Keyword(s) : hepatocellular carcinoma; coronary atherosclerosis; Coronary artery calcification (CAC); liver cirrhosis; Agatston score (CACS)

2869 - Hepatitis B Reactivation During Leflunomide Therapy The Importance of Pre Immunosuppression Screening

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Background / Hypothesis: Hepatitis B virus (HBV) reactivation is a well-recognized complication of immunosuppressive therapy and may occur even in patients considered inactive carriers. While high-risk biologic agents are clearly defined, the reactivation risk associated with certain disease-modifying antirheumatic drugs, such as leflunomide, is less clearly established. In clinical practice, inactive HBV carriers are sometimes perceived as low risk, which may lead to insufficient viral assessment before immunosuppressive

therapy. We present a case of significant HBV reactivation following leflunomide therapy, underscoring the importance of appropriate viral screening and multidisciplinary evaluation prior to treatment initiation.

Materials and Methods: A retrospective review of clinical, laboratory, and treatment data of a patient with chronic hepatitis B was performed. Viral markers, HBV DNA levels, liver function tests, and immunosuppressive treatment history were evaluated.

Results: A 50-year-old female with chronic hepatitis B, classified as Phase 3 (HBeAg-negative chronic infection), was under routine follow-up. She was HBsAg positive and anti-HBe positive, with previously low HBV DNA levels ($<2,000$ IU/mL). She presented to rheumatology with persistent joint pain. Autoimmune serologic markers, including RF, ANA, and ENA, were negative. Despite negative serology, leflunomide 20 mg/day was initiated due to clinical arthritis.

After two months of therapy, routine laboratory tests revealed elevated liver enzymes (ALT 170–180 U/L) without accompanying hepatic symptoms. The patient was referred to our clinic for further evaluation. Repeat testing demonstrated a marked increase in HBV DNA to 20,000,000 IU/mL, consistent with HBV reactivation.

Leflunomide was discontinued, and tenofovir disoproxil fumarate was initiated. At one-month follow-up, liver enzymes normalized and viral load decreased, accompanied by significant improvement in joint symptoms.

Conclusions: HBV reactivation may occur in inactive carriers receiving leflunomide. Careful viral screening and multidisciplinary evaluation are essential before immunosuppressive therapy.

Keyword(s) : Hepatitis B; HBV reactivation; leflunomide; immunosuppressive therapy; antiviral prophylaxis

2870 - Risk Stratification of Liver Fibrosis Progression Using Machine Learning A Meta Analytical Evaluation

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Background / Hypothesis:

Progression from fibrosis to cirrhosis remains a major determinant of liver-related morbidity. Machine learning models have been developed to predict fibrosis stages using non-invasive datasets.

Nevertheless, performance heterogeneity across studies limits standardization. This meta-analysis evaluates algorithm-based fibrosis prediction models.

Materials and Methods:

A systematic search (2014–2025) identified studies developing ML models for fibrosis staging or progression prediction without experimental intervention. Extracted metrics included AUC, sensitivity, specificity, dataset size, and model type. A random-effects model calculated pooled AUC values. Subgroup analyses compared gradient boosting, neural networks, and conventional regression.

Results:

Thirty studies were included. The pooled AUC for ML-based fibrosis prediction was 0.88 (95% CI: 0.85–0.91). Gradient boosting methods demonstrated the highest pooled performance (AUC 0.90). Neural networks showed high accuracy but greater heterogeneity. Dataset size and feature diversity significantly influenced model stability.

Conclusions:

Machine learning algorithms provide strong predictive capability for fibrosis progression and may complement traditional non-invasive scoring systems. Future work should prioritize external validation and standardized evaluation protocols.

Keyword(s) : Liver Fibrosis; Cirrhosis; Machine Learning; Risk Stratification; Meta-analysis

2872 - Bibliometric and Topic Modeling Analysis of Global Research Trends in Liver Fibrogenesis

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Background / Hypothesis:

The volume of research on liver fibrogenesis has grown substantially, necessitating systematic mapping of thematic evolution. Artificial intelligence-assisted bibliometric analysis enables large-scale identification of research hotspots and emerging directions.

Materials and Methods:

Publications indexed in Web of Science (2000–2025) were extracted. Latent Dirichlet Allocation (LDA) topic modeling, co-citation network analysis, and keyword co-occurrence clustering were applied. Topic coherence scores and citation density were evaluated to identify dominant research themes.

Results:

Topic modeling identified major clusters including hepatic stellate cell activation, immune-mediated fibrosis, metabolic-associated fibrosis, and antifibrotic signaling pathways. Citation networks demonstrated increasing interdisciplinary integration between immunology and metabolic research. Emerging trends since 2018 included metabolic dysfunction and inflammasome-related pathways.

Conclusions:

AI-assisted bibliometric analysis provides structured insight into research evolution in liver fibrogenesis. These approaches support strategic research planning and identification of future interdisciplinary directions

Keyword(s) : Bibliometrics; Topic Modeling; Liver Fibrosis; Research Trends; Latent Dirichlet Allocation

2873 - Integrative Metabolomic and Cytokine Profiling Reveals a "Metabolic Inflammatory" Signature in Chronic Hepatitis B Combined with Metabolic Dysfunction Associated Fatty Liver Disease

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Background / Hypothesis:

The coexistence of chronic hepatitis B (CHB) and metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly prevalent worldwide. This dual condition accelerates disease progression, increases the risk of hepatocellular carcinoma, and complicates treatment response. However, non-invasive biomarkers for distinguishing CHB-MAFLD from CHB alone remain lacking

Materials and Methods:

A cross-sectional study was conducted enrolling 406 participants: healthy controls (HC, n=30), CHB alone (n=149), and CHB-MAFLD (n=227). CHB was defined as HBsAg positivity for >6 months with detectable HBV DNA. MAFLD was diagnosed based on hepatic steatosis on imaging combined with metabolic abnormalities. Patients with other liver diseases (hepatitis A, C, E), malignancies, significant organ dysfunction, systemic diseases, or pregnancy/lactation were excluded. Demographic characteristics, clinical parameters, and laboratory tests were collected. Serum cytokine levels (IL-17, TNF- α) were measured using Luminex multiplex assay, and non-targeted metabolomics was performed using LC-MS/MS. Comprehensive comparisons were conducted among the three groups.

Results:

Compared to the CHB alone group, patients with CHB-MAFLD exhibited significantly higher levels of ALT, AST, GGT, BMI, and uric acid (all $P < 0.05$). Cytokine profiling revealed that serum IL-17 and TNF- α levels were significantly elevated in the CHB-MAFLD group compared to the CHB alone group (both $P < 0.05$). Metabolomic analysis identified 110 differential metabolites between the CHB-MAFLD and CHB alone groups. The top three most significantly altered metabolites were Ethyl (S)-3-hydroxybutyrate

glucoside, Indole-3-propionic acid, and Hippuric acid. Additionally, L-Leucine and L-Tyrosine levels were significantly decreased in CHB-MAFLD patients. KEGG pathway enrichment analysis identified ketone body metabolism, tryptophan metabolism, branched-chain amino acid metabolism, and purine metabolism as the core perturbed pathways.

Conclusions:

CHB-MAFLD patients present a distinct "metabolic-inflammatory" signature characterized by mitochondrial dysfunction-driven energy metabolic reprogramming, gut microbiota dysregulation, and pro-inflammatory cytokine activation. Serum cytokines IL-17 and TNF- α , along with metabolites Ethyl (S)-3-hydroxybutyrate glucoside, Indole-3-propionic acid, and L-Leucine, may serve as potential biomarkers for distinguishing CHB-MAFLD from CHB alone. These findings provide scientific evidence for understanding the pathogenic mechanisms and inform precision management strategies for this dual condition.

Keyword(s) : Chronic hepatitis B; Metabolic dysfunction-associated fatty liver disease; Metabolomics; Cytokines; Metabolic-inflammatory signature; Biomarkers

2875 - Integrative Metabolomic and Cytokine Profiling Reveals a "Metabolic Inflammatory" Signature in Chronic Hepatitis B Combined with Metabolic Dysfunction Associated Fatty Liver Disease

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Background / Hypothesis:

The coexistence of chronic hepatitis B (CHB) and metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly prevalent worldwide. This dual condition accelerates disease progression, increases the risk of hepatocellular carcinoma, and complicates treatment response. However, non-invasive biomarkers for distinguishing CHB-MAFLD from CHB alone remain

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lacking.

This study aimed to analyze the clinical characteristics of patients with CHB-MAFLD compared to those with CHB alone, characterize their differential metabolic and cytokine profiles, elucidate the unique "metabolic-inflammatory" signature, and explore potential diagnostic biomarkers. Liver Disease

Materials and Methods:

A cross-sectional study was conducted enrolling 406 participants: healthy controls (HC, n=30), CHB alone (n=149), and CHB-MAFLD (n=227). CHB was defined as HBsAg positivity for >6 months with detectable HBV DNA. MAFLD was diagnosed based on hepatic steatosis on imaging combined with metabolic abnormalities. Patients with other liver diseases (hepatitis A, C, E), malignancies, significant organ dysfunction, systemic diseases, or pregnancy/lactation were excluded. Demographic characteristics, clinical parameters, and laboratory tests were collected. Serum cytokine levels (IL-17, TNF- α) were measured using Luminex multiplex assay, and non-targeted metabolomics was performed using LC-MS/MS. Comprehensive comparisons were conducted among the three groups.

Results:

Compared to the CHB alone group, patients with CHB-MAFLD exhibited significantly higher levels of ALT, AST, GGT, BMI, and uric acid (all $P < 0.05$). Cytokine profiling revealed that serum IL-17 and TNF- α levels were significantly elevated in the CHB-MAFLD group compared to the CHB alone group (both $P < 0.05$). Metabolomic analysis identified 110 differential metabolites between the CHB-MAFLD and CHB alone groups. The top three most significantly altered metabolites were Ethyl (S)-3-hydroxybutyrate glucoside, Indole-3-propionic acid, and Hippuric acid. Additionally, L-Leucine and L-Tyrosine levels were significantly decreased in CHB-MAFLD patients. KEGG pathway enrichment analysis identified ketone body metabolism, tryptophan metabolism, branched-chain amino acid metabolism, and purine metabolism as the core perturbed pathways.

Conclusions:

CHB-MAFLD patients present a distinct "metabolic-inflammatory" signature characterized by mitochondrial dysfunction-driven energy metabolism

reprogramming, gut microbiota dysregulation, and pro-inflammatory cytokine activation. Serum cytokines IL-17 and TNF- α , along with metabolites Ethyl (S)-3-hydroxybutyrate glucoside, Indole-3-propionic acid, and L-Leucine, may serve as potential biomarkers for distinguishing CHB-MAFLD from CHB alone. These findings provide scientific evidence for understanding the pathogenic mechanisms and inform precision management strategies for this dual condition.

Keyword(s) : Chronic hepatitis B; Metabolic dysfunction-associated fatty liver disease; Metabolomics; Cytokines; Metabolic-inflammatory signature; Biomarkers

2880 - Albumin Bilirubin (ALBI) score in predicting complications in patients with HCC undergoing curative Resection a systemic review and Meta analysis

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) remains a major cause of cancer-related mortality globally, with hepatectomy being a curative option for selected patients. Accurate prognostication following surgery is critical to guide postoperative management. The Albumin-Bilirubin (ALBI) score, a simple and objective indicator of liver function, has been proposed as a potential prognostic marker. However, its predictive value in patients undergoing hepatic resection for HCC remains to be systematically evaluated. To determine the prognostic role of the ALBI score in predicting postoperative outcomes, including overall survival, recurrence-free survival, microvascular invasion, and post-hepatectomy liver failure in patients with hepatocellular carcinoma undergoing resection.

Materials and Methods:

A systematic search of PubMed/MEDLINE and the Cochrane Library databases was conducted. Studies assessing the ALBI score as a prognostic factor in HCC patients undergoing curative hepatectomy were included. Out of an initial 28 articles identified, 11

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studies fulfilled the inclusion criteria and were incorporated into the final meta-analysis. The outcomes analyzed included overall survival (OS), recurrence-free survival (RFS), prediction of microvascular invasion (MVI), and prediction of post-hepatectomy liver failure (PHLF).

Results:

Among the 11 included studies, seven studies evaluated the ALBI score's role in predicting overall survival, and six studies investigated its association with recurrence-free survival. One study assessed the ALBI score's predictive value for microvascular invasion, and another evaluated its ability to predict post-hepatectomy liver failure. The pooled analysis demonstrated that a higher ALBI grade was significantly associated with poorer overall survival and increased recurrence rates following hepatectomy. Moreover, a higher ALBI score correlated with the presence of microvascular invasion and higher rates of post-hepatectomy liver failure, reinforcing its role as a reliable prognostic biomarker.

Conclusions:

The ALBI score is a valuable, non-invasive tool for predicting postoperative prognosis in patients with HCC undergoing hepatic resection. Higher ALBI grades are significantly associated with poorer overall survival, increased risk of recurrence, microvascular invasion, and post-hepatectomy liver failure. Incorporating the ALBI score into routine preoperative assessment may facilitate better risk stratification and postoperative management strategies in this population.

Keyword(s) : Hepatocellular carcinoma; Albumin-bilirubin score; Hepatectomy; Prognosis; Recurrence; Liver failure.

2882 - Hypoalbuminemia as a predictor of mortality in patients with acute cholangitis a systemic review and meta analysis

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Background / Hypothesis:

Acute cholangitis (AC) is a life-threatening condition characterized by biliary tract infection, often

necessitating urgent intervention. Despite advances in diagnostics and therapy, mortality remains significant, especially in high-risk subgroups. Hypoalbuminemia, a marker of systemic inflammation and malnutrition, has emerged as a potential predictor of poor outcomes. This systematic review and meta-analysis aimed to evaluate the prognostic value of hypoalbuminemia for mortality in patients with acute cholangitis.

Materials and Methods:

This systematic review and meta-analysis was conducted in adherence to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. A comprehensive search of multiple databases was performed to identify studies evaluating the association between hypoalbuminemia and mortality in patients with acute cholangitis. Eligible studies included adult patients with confirmed acute cholangitis, reported serum albumin levels either as continuous variables or with defined cut-off values, and provided mortality outcomes. Case reports, reviews, pediatric studies, and articles lacking relevant data were excluded. Two independent reviewers screened studies, extracted data, and assessed methodological quality using the Newcastle-Ottawa Scale. Studies scoring ≥ 7 were considered high quality. Statistical analysis was conducted using a random-effects model to calculate pooled odds ratios (ORs) with 95% confidence intervals (CIs). Heterogeneity was assessed using the I^2 statistic, and publication bias was evaluated through funnel plots and Egger's test.

Results:

A total of eight studies comprising 3,210 patients with acute cholangitis were included in the meta-analysis. The pooled analysis revealed a significant association between hypoalbuminemia and increased mortality risk. In the subgroup where serum albumin was treated as a continuous variable, the pooled odds ratio (OR) for mortality was 2.85 (95% CI: 1.93–4.20), indicating that each unit decrease in albumin level was associated with nearly a threefold increase in mortality risk. In the subgroup using albumin cut-off values (most commonly ≤ 2.5 g/dL or ≤ 2.8 g/dL), the association was even stronger, with a pooled OR of 4.08 (95% CI: 2.58–6.45). Heterogeneity across studies was low to moderate ($I^2 = 0\%$ in the cut-off-based group and $I^2 = 47\%$ in the continuous variable group), suggesting consistency in the effect estimate, particularly when defined thresholds were used.

Conclusions:

This meta-analysis highlights hypoalbuminemia as a strong and independent predictor of mortality in patients with acute cholangitis. Early recognition of low

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serum albumin levels should prompt aggressive clinical intervention and close monitoring. Incorporating albumin into existing risk stratification tools may enhance prognostication and guide therapeutic decision

Keyword(s) : hypoalbuminemia; mortality; cholangitis; predictors

2883 - Real World Effectiveness and Safety of Sorafenib in Advanced Hepatocellular Carcinoma A Prospective Study from Fayoum Egypt

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is the most common primary malignancy of the liver. Sorafenib was the first systemic therapy to be approved for the treatment of HCC after having demonstrated a survival benefit in patients with advanced HCC in the first-line setting. Our study evaluated the efficacy and safety of sorafenib as first-line therapy in the treatment of HCC in real-life practice by evaluating overall survival (OS) and progression-free survival (PFS).

Materials and Methods:

A prospective study included 112 patients with advanced HCC treated with sorafenib at viral hepatitis units in Fayoum city, Egypt, from February 2022 to February 2025 according to the National Committee for Control of Viral Hepatitis, which restricts systemic therapy to patients with preserved liver function (Child-Pugh A and advanced-stage disease).

Results:

The median age of patients was 67 years, with male predominance (77%). Hepatitis C virus infection was the main underlying etiology (82.1%). Median overall survival (OS) was 7 months, and progression-free survival (PFS) was 3 months. Higher OS was observed in patients with BCLC stage C, initial sorafenib doses of

800 mg or 200 mg daily, tumor size <6 cm, absence of extrahepatic spread (EHS) or macrovascular invasion (MVI), and AFP levels <400 ng/mL.

Regarding safety, 58.9% tolerated the standard dose (400 mg twice daily), while 41.1% required dose modification, mainly due to adverse events. The most common grade 1–2 adverse events were fatigue (41%), diarrhea (30.3%), and hand-foot skin reaction (23.2%). Other events included anorexia (19.6%), mild liver enzyme elevation (18%), hypertension (7.1%), and hypothyroidism (3.5%). Severe adverse events occurred in 19.8%, including marked liver enzyme elevation and ischemic events. Treatment discontinuation was primarily due to disease progression (71.4%), followed by toxicity (19.6%) and loss to follow-up (8.9%).

Conclusions:

Sorafenib therapy was found to have an acceptable efficacy and safety profile, with predominantly mild to moderate side effects.

Keyword(s) : Hepatocellular carcinoma; Sorafenib; Overall Survival; Progression Free Survival; Safety.

2884 - Comparison of hepatic and extra hepatic scores in predicting mortality in patients with Hepatitis C related cirrhosis

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Background / Hypothesis:

Certain prognostic scores such as Child Turcotte Pugh (CTP) score and MELD-Sodium (MELD-Na) scores are available showing independent predictors of mortality in patients with chronic liver disease. Cirrhotic patients are frail and increasingly vulnerable to psychological effects and increased rates of decompensation in cirrhotic patients. Recently, studies have shown the correlation of liver frailty index (LFI) with CTP and MELD-Na score in cirrhotic patients. Therefore, our aim was to compare the utility of CTP, MELD-Na and LFI in predicting mortality in patients with HCV associated cirrhosis.

Materials and Methods:

It was a prospective cohort study which was conducted at the Department of Hepatogastroenterology, Sindh

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Institute of Urology and Transplantation from January 2019-Decmeber 2021. All patients with HCV associated cirrhosis as per operational definition were enrolled in the study. While, those patients with conditions over estimating frailty such as cardiopulmonary disease osteoarthritis etc. or those with altered mentation, history of hepatocellular carcinoma were excluded from the study. Statistical analysis was performed using the SPSS software version 22. Area under the receiver operating curve(AUROC) was obtained for each CTP, MELD-Na and LFI individually and also for combination of these scores for the prediction of one month,three months and six months mortality in cirrhotic patients.

Results:

A total of 145 patients were included in the study. Among them, 98(67.2%) were males. Seventy four (51%) patients belonged to CTP class B. Esophageal varices were noted in 83(57%) patients. One month mortality was noted in 11(7.6%), Three months mortality in 23(15.9%) and six months mortality in 41(39.4%) patients respectively. AUROC was highest for the combination of LFI and CTP (AUROC-0.845; $p<0.001$) followed by the combination LFI plus CTP plus MELD -Na (AUROC-0.838; $p<0.001$) in predicting six months mortality as compared to each of these scores individually while the AUROC was highest for combination of all the scores(LFI+CTP+MELD-Na) in predicting one month (AUROC-0.942; $p<0.001$) and three months (AUROC-0.858; $p<0.001$) mortality as compared to the other scores.

Conclusions:

Extrahepatic component (LFI) has shown a significant impact in predicting mortality in cirrhotic patients and should also be incorporated along with hepatic scores (CTP and MELD-Na) in predicting the prognosis in this population. This will not only help in prioritizing the patients' management strategies but will also improve the overall survival in this population.

Keyword(s) : Predictors; mortality; hepatic; extrahepatic; Frailty

2885 - Comparative prognostic utility of acute change in Liver stiffness measured using splenic versus liver probe in patients of Acute on chronic liver failure

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Background / Hypothesis: Early identification of acute on chronic liver failure (ACLF) patients with high risk of mortality is important to prioritize the need for ICU monitoring and liver transplantation (LT). In ACLF, inflammation and an acute rise in portal pressure drive the outcome and can raise liver (LSM) and spleen stiffness (SSM). Liver stiffness measurement (LSM) using the liver probe of transient elastography is increasingly explored for prognostication in ACLF. However, the conventional liver probe (LP) has a ceiling effect, with an upper limit of 75 kPa. The splenic probe(SP) with an upper limit of 100 kPa may overcome this limitation. We prospectively evaluated the prognostic performance of liver stiffness measured using the splenic probe versus the conventional liver probe at baseline and during early follow-up in ACLF patients

Materials and Methods: Patients of ACLF (APASL criteria) between 18 to 70 years of age were included. The study aimed to compare the prognostic utility of the acute change in LSM measured using splenic versus liver probe on days 7 and 14 for predicting transplant-free survival at day 90 in patients of ACLF

Results: Study included 56 patients(mean age 40.9 ± 8.6 yrs, males 88.9%, alcohol associated 83.76 %) with ACLF. At day-90, 38 (68.4%) patients had LT free survival, with 15 (26.78%) deaths and 3 (1.68%) LT. Mean baseline AARC score among survivors and non-survivors was 7.68 ± 1.37 and 10.14 ± 1.25 , respectively ($p<0.001$). Baseline LSM measured through SP(82.67 ± 17.7 kPa) was significantly higher than LP(70.95 ± 9.13 kPa) among survivors($p=0.005$) and non-survivors(SP = 90.96 ± 12.79 vs LP = 73.56 ± 9.13 kPa, $p<0.001$). On day 7, mean change in LSM by SP(-7.09 ± 3.48) was significantly higher than LP(-0.69 ± 0.63) among survivors($p<0.001$) and non- survivors (SP = -2.46 ± 1.29 vs LP = -0.11 ± 0.3 kPa) ($p<0.001$). Similarly, on day 14, the mean change in LSM by SP (-13.14 ± 7.21 kPa) was significantly higher than LP(-1.83 ± 0.95 kPa) among survivors($p<0.001$) and non-survivors (SP = -5.89 ± 1.2 vs LP = -0.08 ± 0.12 , $p<0.001$)

Conclusions: LSM measured using a splenic probe provides superior prognostic stratification compared to

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conventional liver probe in ACLF. Serial assessment of LSM using the splenic probe during the first two weeks identifies patients at high risk of short term mortality.

Key words: Transient elastography, Liver stiffness, Splenic probe, Liver Probe, Survival, ACLF

Keyword(s) :

2887 - Role of "MANLATA model" in predicting recurrence in patients with Hepatocellular carcinoma undergoing Trans Arterial Chemo embolization

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) represents the sixth most common neoplasm in terms of incidence and the third leading cause of cancer death. Unfortunately, most of patients with HCC are diagnosed at an advanced stage and are treated by the palliative options. Trans-arterial chemoembolization (TACE) is the best therapeutic option in this situation. Our aim was to identify the non-invasive predictive factors and to build a model using these predictors suggestive of post TACE recurrence of HCC .

Materials and Methods:

It was a retrospective study which included all the patients aged 18-65 years with HCC undergoing TACE from January 2010 to December 2018. While, the patients with early stage HCC undergoing resection, advanced staged HCC bearing portal vein thrombosis, metastatic HCC, patients with poor ECOG status and advanced child class were excluded. SPSS version 26.0 was used for the data analysis and R software was utilized for building a predictive model. Univariate and multivariate logistic regression analysis was performed to identify the independent predictors of post TACE recurrence. A nomogram was then built using R software and a model was then developed ("MANLATA model) for which AUROC was obtained and at an optimal cutoff, the diagnostic accuracy of the model was obtained. The model was then internally validated for the prediction of post-TACE HCC recurrence.

Results:

A total of 323 patients were included in the study. Among them, 281 (87%) were males. Baseline characteristics of all patients undergoing TACE were recorded. Post TACE recurrence was observed in 182 patients. On multivariate analysis, male gender, age > 50 years, tumor size > 5cm, serum albumin <2.8 g/dl , serum alpha-fetoprotein ≥300 ng/ml, neutrophil count >60 % and lymphocyte count <25% were independent predictors of post TACE recurrence. Using these variables, a model "MANLATA model" was developed which had an AUROC of (0.924) and at a cutoff >60, it had an excellent sensitivity of 91.76%, specificity of 85.82% and diagnostic accuracy of 90.98% in predicting post TACE recurrence. In validation cohort, this model showed an excellent sensitivity of 90.21%, specificity of 83.29% and diagnostic accuracy of 89.16% in predicting post-TACE HCC recurrence.

Conclusions:

This non-invasive model had an excellent diagnostic accuracy in predicting post TACE recurrence. However, further studies comprising of large sample sizes are required to validate this model.

Keyword(s) : TACE, HCC recurrence, non-invasive model

2889 - Hepatitis B Reactivation and the Effectiveness of Antiviral Therapy in Patients Receiving Immunosuppressive Therapy A Real World Study

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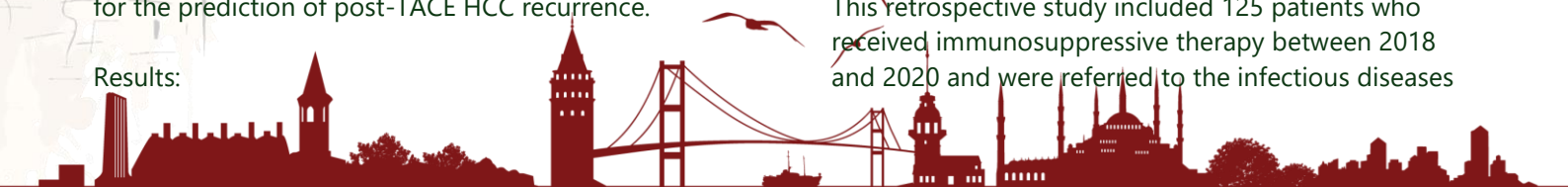
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Background / Hypothesis:

Hepatitis B virus (HBV) persists as cccDNA in the host genome, posing a permanent risk of reactivation (HBVr) during immunosuppression. HBVr not only causes hepatic damage but also disrupts the management of primary diseases. We aimed to evaluate the necessity and real-world effectiveness of antiviral prophylaxis, comparing different nucleos(t)ide analogues (NAs) in patients receiving immunosuppressive therapy.

Materials and Methods:

This retrospective study included 125 patients who received immunosuppressive therapy between 2018 and 2020 and were referred to the infectious diseases



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clinic. Patients were stratified according to baseline HBV serological status (HBsAg and anti-HBc) and immunosuppressive therapy risk. Demographic, clinical, and laboratory data including HBV DNA and liver function tests were collected. Antiviral prophylaxis with potent nucleos(t)ide analogues (TDF, TAF, or ETV) was administered according to risk stratification. Patients were followed every 3 months with biochemical and virological assessments to evaluate HBV reactivation and treatment outcomes.

Results:

The cohort included rheumatological (88%) and solid organ malignancy (7.2%) patients. At baseline, 12% (n=15) were HBsAg(+) and 88% (n=110) were HBsAg(-)/anti-HBc(+). Antiviral prophylaxis was initiated in 90.4% (n=113). No HBVr occurred during active antiviral prophylaxis. However, 4 patients (all HBsAg-positive, 26.6% of the HBsAg(+) group) developed reactivation. Two cases were due to delayed antiviral initiation, and two occurred 4–11 months after prophylaxis cessation (one post-COVID-19 steroid therapy). Anti-HBs positivity was protective: reactivation occurred in 13% of anti-HBs(-) vs. 1% of anti-HBs(+) patients. TAF was preferred over TDF in patients with higher baseline creatinine (p=0.015).

Conclusions:

Potent antiviral prophylaxis is highly effective. HBsAg positivity remains the strongest risk factor. Standard 12 month post-IST prophylaxis may be insufficient for high-risk individuals. Early referral and individualized duration are essential.

Keyword(s) : antiviral prophylaxis, Hepatitis B reactivation, immunosuppressive therapy

2890 - Correlation of Abdominal Girth with Liver Shear Wave Elastography in Non Diabetic Population with Metabolic Dysfunction–Associated Fatty Liver Disease (MAFLD)

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Background / Hypothesis:

Metabolic dysfunction-associated fatty liver disease (MAFLD) is increasingly recognized among non-diabetic population; mainly due to central obesity and the subsequent accumulation of visceral fat. Abdominal

girth is a simple bedside marker of central adiposity; while shear-wave elastography (SWE) serves as a quantitative, non-invasive tool for the estimation of liver stiffness. Therefore, in this study our aim to evaluate the correlation of abdominal girth and liver stiffness as measured by SWE in non-diabetic population with MAFLD.

Materials and Methods:

This cross-sectional study was conducted at the department of gastroenterology, Bahrain Specialist Hospital and included all the non-diabetic patients with age > 18 years and diagnosed with fatty liver on ultrasound. Patients with diabetes, excessive alcohol use, hepatitis, cirrhosis of liver and heart failure were excluded from the study. At presentation, demographic information including age (years), height, weight, Body Mass Index (BMI), blood pressure and the metabolic profile including liver function test and fasting lipid profile were recorded. Abdominal girth was measured at the level of the iliac crest by the standardized technique. Shear wave elastography (SWE) was performed using Siemens Acuson S2000 and liver stiffness was recorded as shear-wave velocity. Pearson correlation coefficient test was utilized to assess the association between abdominal girth and SWE measurements.

Results:

A total of 487 patients with fatty liver were enrolled in the study. Among them, 295 (60.6%) were females and 192 (39.4%) were males. Serum alanine transaminase was 37.9 ± 38.9 (IU); aspartate transaminase was 51.9 ± 26.9 (IU) and gamma glutamyl trans-peptidase was 47.7 ± 10.3 (IU). The mean abdominal girth was 112.4 ± 19.6 cm and the mean value for shear-wave elastography (SWE) was 1.12 ± 0.82 m/s. On Pearson correlation analysis, the abdominal girth was observed to have a significant positive correlation with liver stiffness ($r = 0.716$) ($p < 0.001$). Fasting insulin also demonstrated a strong positive correlation with SWE values ($r = 0.648$) ($p < 0.001$). A weaker correlation was also observed between the post-prandial insulin levels and liver stiffness ($r = 0.4470$) ($p < 0.001$).

Conclusions:

This study showed an excellent correlation of abdominal girth with liver stiffness measured by SWE. This suggests that central adiposity plays a pivotal role in early fibrotic change even in the absence of diabetes. Incorporating abdominal girth into routine assessment may help identify patients needing early elastography-based evaluation and can serve as an

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excellent non- invasive tool in predicting advanced liver fibrosis.

Keyword(s) : abdominal girth; shear-wave elastography; non-diabetic; fatty liver; metabolic-dysfunction

2891 - Factors associated with clinical success of endoscopic management of Post Cholecystectomy Bile Duct Injury A Retrospective Analysis

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Background / Hypothesis:

Laparoscopic cholecystectomy is currently the gold standard technique for the removal of gall bladder. However, it is still associated with complications like iatrogenic bile duct injury (BDI). Currently, due to less invasiveness, endoscopic techniques including Endoscopic retrograde cholangiopancreatography are the initial therapy used for the management of BDI. However, the local data from Pakistan is scarce regarding the effectiveness of endoscopic intervention in the management of BDI. Therefore, the aim of this study was to evaluate the clinical outcome of endoscopic intervention and the factors associated with clinical success in the endoscopic management for bile duct injuries following cholecystectomy.

Materials and Methods:

A retrospective study was conducted at the Department of Hepatogastroenterology, SIUT Karachi, on adult patients aged ≥ 18 years who developed BDI after laparoscopic cholecystectomy and who had ERCP from January 2018 until June 2023. Demographic details, nature of injury (Strasberg classification), time of ERCP, type of intervention, and outcome were analyzed. Clinical success was defined as resolution of bile leakage, reduced drain output or biloma, and resolution of cholestatic liver enzymes. Statistical analysis included univariate and multivariate logistic regression to find the independent predictors of clinical success.

Results:

Out of 56 patient who underwent endoscopic intervention for BDI, 33 (58.9%) were females. Mean age was 40.3 ± 14.2 years. According to the Strasberg classification, type A injuries were the most frequent

that were observed in 40 (71.4%) patients, while type D and type E BDI accounted for 9% and 19.6% of the cases respectively. In 50 (89.3%) patients, ERCP was performed three days after undergoing laparoscopic cholecystectomy. Most common intervention was combined sphincterotomy and stent placement (55.3%). Clinical success was achieved in 46 (82.1%) patients. On multivariate logistic regression analysis, presence of Strasberg type A injury (OR 7.4; 95% CI 2.01–27.2; $p = 0.003$) and combined endoscopic sphincterotomy and stenting (OR 2.58; 95% CI 1.17–5.7; $p = 0.02$) were the factors independently associated with clinical success in the endoscopic management of BDI.

Conclusions:

Endoscopic retrograde cholangiopancreatography is a successful and safe therapeutic measure in managing post-cholecystectomy bile duct injuries, particularly Strasberg type A leaks. Early therapy, accurate classification of the injury, and use of combined sphincterotomy with stent placement were independent predictors with positive outcomes.

Keyword(s) : Bile duct injury, laparoscopic cholecystectomy, ERCP, endoscopic therapy, Strasberg classification, bile leak.

2892 - Non invasive predictors of Hepatorenal syndrome in decompensated chronic liver disease

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Background / Hypothesis:

Hepatorenal syndrome (HRS) is a frequent complication of advanced decompensated chronic liver disease (DCLD) that is associated with high morbidity and mortality. Our aim was to identify non-invasive predictors of Hepatorenal syndrome in DCLD patients.

Materials and Methods:

It was a retrospective study which was carried out at the department of Hepatogastroenterology, Sindh Institute of Urology and Transplantation. All patients admitted with DCLD from June 2018 to December 2021 were included in the study. Univariate analysis

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followed by the multivariate analysis was performed to identify non-invasive predictors of HRS.

Results:

The total number of patients included in the study were 199. Out of them, 124 (62.1%) were males. Most common cause of chronic liver disease was hepatitis C noted in 57 (28.6%) patients. Ascites was noted in 165 (82.9%) patients. HRS was noted 78 (39.2%) patients while SBP was seen in 65 (32.7%) patients. Age greater than 40 years, Child Turcotte Pugh score > 13, Hemoglobin less than 10 g/dl, Total leucocyte count > 11 x10⁹/L and urea > 30 mg/dl were found to be independent predictors for the development of HRS.

Conclusions:

These strong predictors of HRS can help in prevention, early diagnosis and prompt treatment of the patients with HRS and improving the overall survival in DCLD including urgent referral for liver transplantation.

Keyword(s) : Decompensated Chronic Liver Disease; Hepatorenal Syndrome; Predictors

2893 - Role of PAST F model in predicting advanced liver fibrosis in Autoimmune Hepatitis

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Background / Hypothesis:

Liver biopsy remains the gold standard for diagnosing and staging liver fibrosis. However, it is invasive and associated with potential complications. Consequently, there is a growing need for non-invasive methods to predict advanced fibrosis in AIH patients. To evaluate the diagnostic accuracy of the PAST-F model in predicting advanced liver fibrosis in patients with autoimmune hepatitis and to establish an optimal cutoff score for clinical application.

Materials and Methods:

This Retrospective cohort study was conducted at the Hepatogastroenterology unit, Sindh Institute of Urology and Transplantation, Karachi from January 2016 to December 2023. All the patients diagnosed with AIH on the basis of liver biopsy were enrolled in the study. Patients with concomitant liver diseases were excluded. Liver fibrosis was evaluated using the Metavir scoring system on the liver parenchymal

biopsy. Statistically significant variables on univariate analysis then underwent multivariate logistic regression analysis to identify independent predictors of advanced liver fibrosis in autoimmune hepatitis. Using these variables a model was proposed and area under the receiver operating curve was obtained. At an optimal cutoff, diagnostic accuracy was obtained for the model in predicting advanced liver fibrosis in AIH.

Results:

A total of 360 patients were included in the study. On multivariate analysis, Female gender, advanced age, raised ANA (1:160), negative serology, increased total bilirubin, alkaline phosphatase, Gamma Glutamyl Transpeptidase, INR and decreased TLC and platelet count were the factors significantly associated with advanced fibrosis. Using the variables with higher odds ratio on multivariate analysis, a model was proposed named PAST-F model comprising of platelet count ($p \leq 0.001$) (O.R.1.025), decreased serum albumin levels ($p \leq 0.001$) (O.R.33.6), negative autoimmune serology ($p \leq 0.001$) (O.R.4.933), raised bilirubin ($p \leq 0.001$) (O.R.5.199) and female gender ($p \leq 0.001$) (O.R.25.91). AUROC was obtained for PAST-F model in predicting advanced liver fibrosis and at an optimal cutoff of score of >8, it had a good sensitivity of 80.14%, specificity of 87.67% and diagnostic accuracy of 84.72% in predicting advanced liver fibrosis in patients with AIH.

Conclusion:

The PAST-F model, incorporating variables such as platelet count, serum albumin levels, autoimmune serology status, bilirubin levels, and gender, demonstrates a high diagnostic accuracy in predicting advanced liver fibrosis in patients with autoimmune hepatitis. Therefore PAST-F model offers a valuable non-invasive tool for clinicians to identify patients at higher risk for advanced fibrosis, potentially guiding more tailored therapeutic interventions and monitoring strategies.

Keyword(s) : autoimmune hepatitis; fibrosis; non-invasive; model

2895 - Risk of hepatitis b virus reactivation in patients negative for hepatitis b surface antigen and positive for hepatitis b core antibody receiving biological agents

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Background / Hypothesis:

In HBsAg-negative, anti-HBc IgG-positive individuals, the risk of hepatitis B virus reactivation (HBVr) associated with immunosuppressive therapy varies depending on the immunosuppressive potency of the biological agent used. This study aimed to evaluate the development of HBVr in HBsAg-negative, anti-HBc IgG-positive patients receiving moderate- or high-risk biological agents.

Materials and Methods:

A total of 172 HBsAg-negative, anti-HBc IgG-positive patients were retrospectively analyzed. Patients were classified into moderate- and high-risk groups according to the HBVr risk associated with the biological agents used. Rituximab and ocrelizumab were categorized as high-risk agents, whereas anti-TNF- α agents (adalimumab, infliximab, golimumab, certolizumab, etanercept), the IL-6 inhibitor tocilizumab, and the IL-12/23 inhibitor ustekinumab were considered moderate-risk agents. Demographic and clinical characteristics, treatment details, concomitant immunosuppressive therapies, and antiviral prophylaxis status were recorded. Follow-up virological and serological data were reviewed, and HBVr occurrence was assessed. HBVr was defined as newly detectable HBV DNA or reappearance of HBsAg.

Results:

The mean age of the study population was 52.3 ± 11.0 years. The most common indications for biological agent therapy were ankylosing spondylitis (30.2%) and rheumatoid arthritis (29.7%). Overall, 20.9% ($n = 36$) of patients received high-risk agents, while 79.1% ($n = 136$) received moderate-risk agents. The median follow-up duration was 28.5 months (range: 12–60) in the high-risk group and 36 months (range: 12–96) in the moderate-risk group. All patients in the high-risk group received antiviral prophylaxis at treatment initiation, and no HBVr events were observed. In the moderate-risk group, 33.1% ($n = 45$) received antiviral prophylaxis, whereas 66.9% ($n = 91$) were monitored without prophylaxis. Virological reactivation occurred in only one patient (1.1%) among those without prophylaxis and was not accompanied by clinical or biochemical hepatitis. The patient had a baseline anti-HBs level ≥ 1000 IU/L, which remained stable throughout follow-up.

Conclusions:

Antiviral prophylaxis appears to be effective in preventing HBV reactivation in HBsAg-negative, anti-

HBc IgG-positive patients receiving moderate- or high-risk biological agents. In patients treated with moderate-risk agents, regular virological monitoring may be a safe alternative to universal prophylaxis, enabling early detection and timely management of rare reactivation events. These findings support risk-adapted management strategies in this patient population.

Keyword(s) : Hepatitis B Virus Reactivation, Biological Agents, Antiviral Prophylaxis

2896 - Usefulness and Yield of Whole Exome Sequencing in Paediatric Genetic and Metabolic liver disorders in a Tertiary Care Centre in North India

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Background / Hypothesis:

Paediatric genetic and metabolic liver disorders exhibit significant genetic and phenotypic heterogeneity. In North India, where parental consanguinity is a known clinical factor, identifying monogenic etiologies via Whole-Exome Sequencing (WES) is critical for tailored management and genetic counseling. This study evaluates the diagnostic yield and mutational spectrum of WES in these disorders.

Materials and Methods:

We analyzed a combined cohort of 327 unique pediatric patients from January 2017 to January 2026 categorized into five clinical groups: Chronic Liver Disease (CLD, $n=138$), Neonatal Cholestasis ($n=95$), Storage Disorders ($n=55$), Disorders of Bilirubin Metabolism ($n=23$), and Acute Liver Failure (ALF, $n=16$). Clinical data and consanguinity were recorded. WES data was analysed. Genetic variants were interpreted according to ACMG consensus guidelines.

Results:

The overall diagnostic yield was 43.4% ($n=142$), with an additional 38.2% harboring Variants of Uncertain Significance (VUS). The most frequently involved genes were ABCB4 ($n=13$), UGT1A1 ($n=11$), JAG1 ($n=10$), and ABCB11 ($n=9$). Diagnostic yields were highest in bilirubin metabolism (73.9%) and storage disorders (56.4%). Consanguinity was present in 16.5% ($n=54$) of

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the cohort with a 53.7% positive yield compared to 41.3% in non-consanguineous families. Missense mutations were the predominant variant type (n=150). Zygosity analysis revealed 136 heterozygous (31 of which were significant autosomal dominant variants), 85 homozygous (strongly correlated with consanguinity), 37 compound heterozygous, and 7 hemizygous cases (confirming X-linked disorders such as GSD IXa).

Conclusions:

WES provides a high diagnostic yield in paediatric genetic and metabolic liver disorders. This approach prevents unnecessary and harmful surgeries (like Kasai for Alagille), and allows families to access precision treatments (dietary for storage disorders, chelation for Wilson's disease) that prevent irreversible liver damage and transplant. It also empowers families with accurate genetic counseling for future children. The increasing availability of Next-Generation Sequencing (NGS) in developing countries has significantly improved the diagnosis of paediatric genetic and metabolic liver disorders.

Keyword(s) :

2898 - Circulating miR 184 and miR 206 as Predictive Biomarkers for Early Recurrence in HBV Related Hepatocellular Carcinoma A Prospective Study

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Background / Hypothesis: Early recurrence after curative resection remains a major challenge in hepatocellular carcinoma (HCC), particularly in hepatitis B virus (HBV)-related cases. Liquid biopsy using circulating microRNAs (miRNAs) offers a non-invasive approach to identify molecular markers predictive of recurrence.

Materials and Methods: We prospectively enrolled 30 individuals with HBV-related HCC presenting with a single tumor (<5 cm) and no vascular invasion or metastasis. Blood samples were collected preoperatively and on postoperative day 7. Expression of 20 selected miRNAs from circulating cell-free

DNA/RNA and exosomes was analyzed. Participants were categorized into early recurrence (within 1 year, n=6) and non-recurrence (n=24) groups. Differentially expressed miRNAs were identified, and target genes of significant miRNAs were retrieved from miRTarBase. Protein-protein interaction (PPI) networks were constructed using STRING and visualized in Cytoscape. Enrichment analysis was performed using Gene Ontology and KEGG pathway databases.

Results: On postoperative day 7, expression of miR-184 and miR-206 was significantly lower in the early recurrence group than in the non-recurrence group ($p < 0.05$). Other miRNAs showed no significant differences. Target gene analysis revealed 16 key hub proteins—CCND1, CCND2, KLF4, NOTCH3, BDNF, MET, CDK4, BCL2, AKT2, IGF1R, MYC, HDAC4, ESR1, KRAS, SMARCB1, and AGO2—enriched in cancer-related pathways and involved in HCC progression.

Conclusions: Reduced postoperative expression of miR-184 and miR-206 may predict early recurrence in individuals with HBV-related HCC. Their associated regulatory networks suggest possible mechanisms of recurrence and represent potential biomarkers for postoperative surveillance. Further studies are needed to validate their prognostic value.

Keyword(s) : Liquid biopsy, hepatocellular carcinoma, early recurrence, HBV

2899 - Association of Metabolic dysfunction Associated Steatotic Liver Disease with Dementia An Updated Systematic Review and a Meta analysis

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Background / Hypothesis:

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide and is increasingly recognized as a systemic metabolic disorder. Emerging evidence suggests a potential link between MASLD and neurodegenerative diseases, including dementia. However, prior meta-analyses have reported inconsistent findings and were limited by small sample

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sizes. Therefore, we performed an updated systematic review and meta-analysis to evaluate the association between MASLD and dementia, including its major subtypes.

Materials and Methods:

This systematic review was conducted according to PRISMA guidelines. Electronic databases, including PubMed/MEDLINE, Cochrane Library, and Google Scholar, were searched from inception to November 25, 2025. Studies evaluating the association between MASLD and dementia in adults were included. Hazard ratios (HRs) with 95% confidence intervals (CIs) were pooled using fixed- or random-effects models based on heterogeneity. Subgroup analyses were performed for Alzheimer's disease and vascular dementia.

Results:

Fifteen studies comprising 3,381,435 participants were included. The overall prevalence of dementia was 4.6%, and 27.7% of participants had MASLD. MASLD was significantly associated with an increased risk of all-cause dementia (HR 1.09; 95% CI 1.01–1.18; $p = 0.03$; $I^2 = 90\%$). Subgroup analysis demonstrated a significant association between MASLD and Alzheimer's disease (HR 1.08; 95% CI 1.01–1.15; $p = 0.02$; $I^2 = 80\%$). No statistically significant association was observed between MASLD and vascular dementia (HR 1.16; 95% CI 1.00–1.35; $p = 0.05$; $I^2 = 83\%$).

Conclusions:

MASLD is associated with a modest but statistically significant increased risk of all-cause dementia and Alzheimer's disease, but not vascular dementia. These findings support the concept of MASLD as a systemic disorder with potential neurocognitive implications. Further prospective studies are needed to clarify underlying mechanisms and assess whether targeted metabolic interventions may reduce dementia risk.

Keyword(s) :

2900 - Spectrum effects and threshold drift of 24 hour urinary copper excretion in hypercupriuric referrals implications for Wilson disease diagnosis

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Background / Hypothesis: Twenty-four-hour urinary copper excretion (24-h UCE) is a core test in Wilson disease (WD) evaluation, but it is commonly elevated in non-WD liver diseases, particularly with cholestasis and

advanced liver dysfunction. In referral cohorts pre-selected for hypercupriuria, conventional UCE thresholds may exhibit spectrum effects and threshold drift. We quantified the diagnostic performance of UCE across a hypercupriuric spectrum and evaluated whether ceruloplasmin (Cp)-anchored strategies improve clinical interpretability.

Materials and Methods: We retrospectively analyzed 538 consecutive patients evaluated for suspected WD with 24-h UCE $\geq 60 \mu\text{g}/24 \text{ h}$ (institutional upper limit of normal). WD was adjudicated using a prespecified genotype-informed reference standard (biallelic pathogenic/likely pathogenic ATP7B variants with Leipzig score ≥ 4); non-WD etiologies were adjudicated by structured chart review. We assessed discrimination (AUROC) and prespecified threshold performance overall and across bilirubin/INR-based severity strata. We additionally evaluated a prespecified two-step Cp triage with a predefined grey zone and a leave-one-out cross-validated secondary model.

Results: The cohort included 62 WD and 476 non-WD patients (WD prevalence 11.5%). Overall discrimination of 24-h UCE was moderate (AUROC 0.76; 95% CI 0.69–0.82) and operating characteristics shifted across the hypercupriuric severity spectrum, consistent with threshold drift. A prespecified Cp triage (rule-out Cp $> 0.20 \text{ g/L}$; rule-in Cp $\leq 0.10 \text{ g/L}$) classified 83.6% of patients at stage 1; the remaining grey-zone patients underwent a secondary model using $\ln(24\text{-h UCE})$, total bilirubin, and ALT, yielding overall sensitivity 96.8% and specificity 97.3%.

Conclusions: In hypercupriuric liver-disease referrals, 24-h UCE alone provides only moderate discrimination and is sensitive to referral enrichment and severity-related phenotype. These data support using UCE primarily as a higher-threshold rule-in component rather than a stand-alone rule-out test once evaluation is conditioned on hypercupriuria. Cp-anchored and integrated approaches are more transportable across the hypercupriuric spectrum and may reduce indeterminate evaluations; external validation is needed.

Keyword(s) : Wilson disease; 24-hour urinary copper; hypercupriuria; ceruloplasmin; diagnostic accuracy; referral cohort

2901 - Spectrum effects and threshold drift of 24 hour urinary copper excretion in hypercupriuric referrals implications for Wilson disease diagnosis

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Materials and Methods:

We retrospectively analyzed 538 consecutive patients evaluated for suspected WD with 24-h UCE ≥ 60 $\mu\text{g}/24$ h (institutional upper limit of normal). WD was adjudicated using a prespecified genotype-informed reference standard (biallelic pathogenic/likely pathogenic ATP7B variants with Leipzig score ≥ 4); non-WD etiologies were adjudicated by structured chart review. We assessed discrimination (AUROC) and prespecified threshold performance overall and across bilirubin/INR-based severity strata. We additionally evaluated a prespecified two-step Cp triage with a predefined grey zone and a leave-one-out cross-validated secondary model.

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indeterminate evaluations; external validation is needed.

Keyword(s) : Wilson disease; 24-hour urinary copper; hypercupriuria; ceruloplasmin; diagnostic accuracy; referral cohort

2902 - Integrating Clinical Biomarkers with Machine Learning to Predict Hepatocellular Carcinoma in NAFLD

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Background / Hypothesis:

Hepatocellular carcinoma (HCC) is a significant complication of non-alcoholic fatty liver disease (NAFLD), with high mortality rates due to delayed diagnosis. Identification of clinical biomarkers integrated with machine learning approaches has the potential to improve prediction and early detection.

Materials and Methods:

This study used three public datasets: TCGA-LIHC (379 patients, clinical data), GSE49541 (72 samples, gene expression data), and UCI-HCC (165 patients, clinical and survival data). Descriptive analyses were performed on patient characteristics and gene expression patterns. UCI-HCC data were used to build predictive models using Logistic Regression and Random Forest. Model performance was evaluated using accuracy, F1-score, ROC-AUC, and feature importance analysis.

Results:

The age distribution of TCGA-LIHC patients shows that most cases occur in middle-to-late age. The GSE49541 heatmap demonstrates heterogeneity in gene expression between patients. In the UCI-HCC dataset, differences in patient status provide relevant survival insights for prediction. Logistic Regression performed best with an accuracy of 87.9% and an F1-score of 0.90, while Random Forest achieved an ROC-AUC of 0.835. Feature importance analysis identified 10 key clinical variables, including liver biomarkers and laboratory parameters, as significant predictors of HCC risk.

Conclusions:

Integrating clinical biomarkers and molecular data with machine learning has shown promising results in predicting HCC risk in NAFLD patients. This approach can improve early detection strategies and

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personalised therapy, although further validation in a broader population is needed.

Keyword(s) : Machine Learning; Hepatocellular Carcinoma; Liver Oncology and Cancer Management

2903 - Single Center Experience in Complex Liver Transplantation

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Background / Hypothesis:

To summarize and analyze the clinical experience of complex liver transplantation.

Materials and Methods:

A retrospective analysis was conducted on the clinical data and follow-up outcomes of 402 patients who underwent complex liver transplantation at the Affiliated Hospital of Qingdao University. Patient survival rates and the incidence of postoperative complications were analyzed.

Results:

From July 2013 to July 2024, a total of 1,400 liver transplantations were performed at this single center,

among which 402 cases (28.7%) were classified as complex liver transplantation. These cases included patients with grade III or higher portal vein thrombosis, a history of upper abdominal surgery, repeat or multiple liver transplantations, massive diseased livers (e.g., polycystic liver disease), and those with multiple organ dysfunction. The 1-, 3-, and 5-year survival rates in the complex transplantation group were 81.3%, 61.3%, and 52.6%, respectively, whereas those in the conventional transplantation group were 88.4%, 78.2%, and 71.2%, respectively. The survival rate in the complex group was significantly lower than that in the conventional group ($P < 0.001$). The incidence of massive intraoperative bleeding in complex liver transplantation was 8.9%, and the anhepatic phase lasted 54.1 ± 15.7 minutes. Early postoperative complications included reoperation due to intra-abdominal bleeding (4.0%), vascular complications (1.7%), severe infection (16.9%), and acute renal failure (12.4%). Long-term postoperative complications mainly included hepatocellular carcinoma recurrence, biliary complications, and chronic renal impairment, with incidence rates of 48.5%, 7.2%, and 4.9%, respectively.

Conclusions:

Technical challenges in complex liver transplantation remain a major limiting factor in the development of liver transplantation programs. Improvements in surgical techniques and perioperative management may help improve the prognosis of patients undergoing complex liver transplantation.

Keyword(s) : complex liver transplantation; technical challenges; management



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