HEPATOLOGY

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1695 | A CASE OF STEALTH NIACIN INDUCED LIVER INJURY

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Introduction: Niacin, a B vitamin once recommended for dyslipidemia under National Cholesterol Education Program Adult Treatment Panel III (NCEP ATPIII 2001), fell out of favor after the 2013 ACC/AHA guidelines centered management on statins. A systematic review for the 2018 AHA/ACC/Multisociety Cholesterol Guidelines further showed niacin conferred no ASCVD benefit while raising risk of adverse effects including worsening insulin resistance and hepatotoxicity. It remains available by prescription and over the counter, particularly for statin-intolerant patients. We present chronic niacin use causing undiagnosed clinically significant drug-induced liver injury (DILI) over 10 years. **Case Presentation:** A 63-year-old man with dyslipidemia was referred for mild mixed hepatocellular-cholestatic enzyme elevation. He had 10 years of fluctuating elevation (R=0.65; alkaline phosphatase peak 160 IU/L, AST peak 49 IU/L). His medication regimen over this interval included aspirin 81 mg daily, as-needed ibuprofen, and extended-release niacin 1500 mg daily, started after statin-induced rhabdomyolysis. He denied supplements, drank rare alcohol, and had no diabetes or autoimmune history.Workup showed thrombocytopenia (89 K/uL), hypoalbuminemia (3.0 g/dL), and coagulopathy (INR 1.8). Viral serologies were negative. Iron saturation was elevated (79%) with normal ferritin; ANA was positive (1/160) and ACE elevated (105 U/L). HFE testing (H63D, C282Y) was negative. FibroScan showed liver stiffness of 10.0 kPa and CAP 387 dB/m. Liver biopsy revealed mild portal lympho-histiocytic inflammation, interface and lobular activity with feathery degeneration, acidophil bodies, ductular reaction, and focal cholestasis, without steatosis, ballooning, or bile duct injury; trichrome showed stage 1-2 fibrosis, with negative iron and copper stains (Figure 1). The differential diagnosis from pathology was Wilson disease versus DILI; 24-hour urine copper was negative.Of his three medications, niacin was deemed most likely to cause hepatotoxicity. Following discontinuation, liver enzymes, albumin, platelets, and INR normalized within 27 days and remained stable for 3 years (RUCAM 8, probable). **Discussion:** The AASLD Practice Guidance on DILI defined three scenarios for DILI, one of which is an INR > 1.5 with elevated liver enzymes. Per LiverTox, niacin toxicity can reduce protein and coagulation factor synthesis, causing hypoalbuminemia and coagulopathy. Here, INR was not checked initially, and the modest enzyme elevation would not typically prompt hepatology referral. However, biopsy showed stage 1-2 fibrosis indicating likelihood to progress if untreated. **Conclusion:** Though prescription niacin use has declined in light of the 2018 ACC/AHA/Multisociety systematic review, for patients requiring it, monitoring of albumin, platelets, and INR alongside liver enzymes may facilitate earlier recognition of hepatotoxicity.

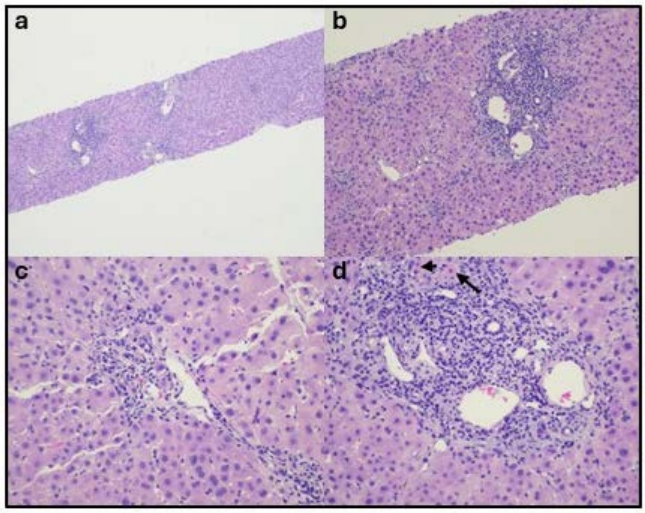


Figure 1. Liver biopsy histology, H&E stain. (a) 4x, At low magnification, sections show liver parenchyma with portal and lobular inflammatory inflammation, predominately lymphohistiocytic. No obvious necrosis, significant steatosis, or fibrosis is identified. (b) 10x, Interface hepatitis is present, where the inflammatory infiltrates cross the limiting plate of the portal tract. Bile ducts are intact. Within the parenchyma, multiple foci of lobular inflammation is identified. (c) 20x, Pericentral lobular inflammation is highlighted with focal acidophil bodies (arrow) identified. The inflammation is predominately lymphohistiocytic with rare neutrophils and plasma cells. (d) 20x, High-power view of a portal tract highlights ductular reaction and interface hepatitis with rare associated acidophile bodies (arrows). Rare eosinophils (stars), neutrophils and plasma cells are identified.

1696 | CHRONIC USE OF KHAT WITH DILI LEADING TO CIRRHOSIS.

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Introduction: Khat (catha edulis) are fresh leaves from an evergreen plant native to East Africa andthe Arabian peninsula that have stimulant properties. These leaves are chewed causingmild euphoria. Public health estimates that 10-20 million people use Khat regularly,predominately in eastern African nations where the plant is grown. While legal andaccepted in nations like Ethiopia, Somalia, and Yemen, the active agents causingeuphoria (cathinone and cathine) are classified as illegal in the United States. Casereports of Khat induced auto immune hepatitis with positive autoantibodies have beenreported with improvement in liver tests with cessation of Khat use along with somepatients requiring immunosuppression. Case studies of advanced liver disease andcirrhosis have been limited. **Case Presentation:** This case study involves a 44-year-old who was initially diagnosed with Khat druginduced liver injury 3 years prior to admission

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following two liver biopsies confirming thatthe injury was not autoimmune in nature and stage II fibrosis. Comprehensive liverevaluation unrevealing for other cause with labs indicating mixed cholestatic andhepatocellular injury. He was advised complete cessation of Khat use with reportedresuming use within 3 weeks of discharge. A year following discharge, he was admittedwith evidence of decompensated liver disease with ascites, hepatic encephalopathy andSBP. At that time, he was noted to have ANA+ (320), AMA+ and + IgG (2600). He waslost to follow up for two years and again presented with hepatic encephalopathy,anasarca and ascites. Abdominal imaging with a shrunken liver, splenomegaly,mesenteric edema and small ascites. No masses were seen on imaging and his AFPwas 33. Repeat IgG was 2102. Repeat liver biopsy was performed with HVPG of 11mmHg. Liver biopsy confirmed Khat induced liver injury leading to cirrhosis with alteredhepatic architecture and regenerative nodule formation representing stage 4 fibrosis. **Discussion:** This patient now has evidence of advanced liver disease from continued use of a herb that triggers an active heptitis. Repeat biopsy was performed due to more rapid progression to cirrhosiswithin a 3 year period than expected without cessation. **Conclusion:** This case highlights use of Khat causing acute hepatitis leading to cirrhosis withpersistent use. Recognizing other culturally accepted herbalproducts that may be contributing to advancing liver disease or autoimmune appearing elevated liver testsshould be considered when interviewing patients.

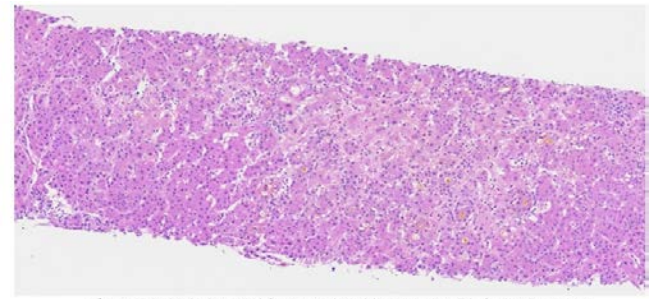
1697 | THE MANY FACES OF ACUTE LIVER INJURY IN PREGNANCY: FROM AFLP TO DELAYED CHOLESTATIC DILI

Camelia Ciobanu[1] Lauren Grinspan[1] Madeline Karsten[1] Lauren MS. Ferrara[1] Thomas Schiano[1]

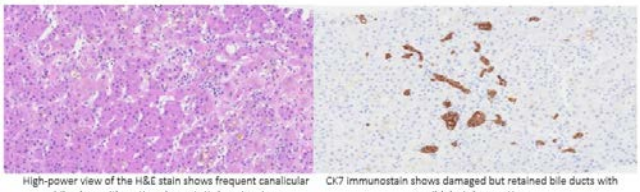
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Introduction: Acute fatty liver of pregnancy (AFLP) is a rare and potentially life-threatening entity, affecting approximately 1 in 10,000 pregnancies and typically occurring in the third trimester or early postpartum period. We describe a patient who met clinical criteria for AFLP based on the Swansea criteria but subsequently developed a second liver injury. Liver biopsy was consistent with drug-induced liver injury (DILI) and showed no hepatic steatosis. **Case Presentation:** A 30-year-old G1P0 woman at 35 weeks with a twin pregnancy presented with contractions and underwent cesarean delivery for a Category III fetal tracing. Labs showed platelets 110 ×10³/μL, creatinine 2.73 mg/dL, glucose 63 mg/dL, AST 431 IU/L, ALT 649 IU/L, and total bilirubin 5.7 mg/dL (direct 4.2 mg/dL). Postoperatively, she had lethargy, hypoglycemia, hypertension, and coagulopathy (INR 2.0, fibrinogen <60 mg/dL), with elevated ammonia, LDH, bile acids and low haptoglobin. She met >=7 Swansea criteria for AFLP. Viral hepatitis and other causes of acute liver injury were excluded. Ultrasound showed diffuse hepatic echogenicity suspicious for steatosis without biliary obstruction.Anemia, schistocytes, low C3, and persistent kidney injury raised concern for atypical hemolytic uremic syndrome (aHUS). Eculizumab was initiated empirically with penicillin prophylaxis. ADAMTS13 activity was normal and anti-factor H antibodies were negative. Mental status, renal function, transaminases, ammonia, and coagulatopathy improved.On hospital day 9, she developed a second, predominantly cholestatic liver injury with ALP 1,310 IU/L, GGT 1,087 IU/L, total bilirubin 7.0 mg/dL, AST 196 IU/L, and ALT 166 IU/L. MRI/MRCP showed no biliary obstruction or vascular abnormality. Liver biopsy demonstrated cholestatic injury consistent with delayed DILI, possible related to eculizumab, penicillin or ibuprofen without hepatic steatosis. Ursodiol was initiated, with gradual improvement in cholestatic liver biochemistries. **Discussion:** This case highlights the diagnostic challenge of AFLP and the importance of evaluating for additional causes when liver injury worsens after initial improvement. The absence of steatosis on a postpartum biopsy does not necessarily exclude AFLP. Microvesicular steatosis may be difficult to detect routine H&E staining, and tissue handling may influence its identification. Current pathology guidance recommends that, when AFLP is suspected, tissue be submitted fresh for frozen-section review of fat, with Oil Red O or Sudan staining, as routine formalin fixation and paraffin processing may remove lipid and limit its detection. **Conclusion:** AFLP remains primarily a clinical diagnosis.This case raises an important question regarding how rapidly hepatic microvesicular steatosis resolves following delivery and emphasizes the importance of biopsy timing and appropriate tissue-processing techniques when interpreting postpartum liver histology.

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H&E stain shows lobular disarray with frequent canalicular bile plugs, hepatocellular feathery degeneration, and scattered apoptotic hepatocytes.



High-power view of the H&E stain shows frequent canalicular bile plugs with scattered apoptotic hepatocytes.

CK7 immunostain shows damaged but retained bile ducts with a mild ductular reaction.

1698 | SEVERE TRANSAMINITIS WITHOUT CLASSIC FEATURES: AN ATYPICAL PRESENTATION OF ACUTE FATTY LIVER OF PREGNANCY IN A TWIN GESTATION

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Introduction: Acute fatty liver of pregnancy (AFLP) is a rare life-threatening disorder marked by microvesicular fatty infiltration of hepatocytes, thought to arise from impaired fetal-maternal fatty oxidation and typically presents in the third trimester. We describe an atypical AFLP presentation in a dichorionic-diamniotic twin gestation with limited symptoms despite a rapidly progressive hepatic decline. **Case Presentation:** A 32-year-old G2P1001 female at 32 weeks 2 days gestation with a dichorionic-diamniotic twin pregnancy was admitted for suspected preeclampsia with headache. On admission, she had no abdominal pain, nausea, or vomiting, and vital signs, including blood pressure, were stable. However, she had new-onset transaminitis (AST/ALT 168/115 U/L) that progressed to 910/734 over a week. WBCs were 14.8k cells/µL, ammonia was 65 µmol/L, bilirubin was 1 mg/dL, uric acid was 8.4 mg/dL, fibrinogen was 246 mg/dL, INR was normal, and there was no proteinuria or thrombocytopenia. Peripheral blood smear showed burr cells. Viral hepatitis, HIV, EBV, CMV, HSV, lipase, TSH, and CK studies were unremarkable. Abdominal ultrasound showed heterogeneous hepatic echotexture consistent with fatty infiltration without biliary obstruction. Given these findings, AFLP became the leading diagnosis. She met 6/15 Swansea criteria (SC) for AFLP with: bilirubin > 0.8, uric acid > 5.7, WBCs > 11k, AST/ALT > 42, ammonia > 47, and ascites or "bright liver" on abdominal ultrasound. She underwent cesarean delivery at 32 weeks 1 day gestation. Following delivery, AST/ALT improved rapidly (120/229) within 72 hours postpartum and normalized to 34/20 at the 1-month hepatology follow-up. **Discussion:** This case highlights how AFLP can cause rapidly deteriorating hepatic injury before classic clinical features emerge. Despite the subclinical presentation of AFLP due to a lack of vomiting, abdominal pain, polydipsia/polyuria, and encephalopathy, she still met SC with lab and imaging data to secure the diagnosis. Due to the association between AFLP and fetal fatty-acid oxidation defects, particularly LCHAD deficiency, screening of the patient's children was recommended to identify a potential underlying metabolic disorder. **Conclusion:** AFLP should remain in the differential diagnosis of rapidly worsening liver injury in late pregnancy, even without classical signs. Prompt recognition of subtle abnormalities may permit timely delivery before severe maternal or fetal complications develop.

1699 | ALCOHOLIC LIVER DISEASE: THE IMPORTANCE OF SEEKING APPROPRIATE CARE WITH EARLY SIGNS/SYMPTOMS

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Introduction: Alcoholic liver disease is a spectrum of liver dysfunction ranging from asymptomatic jaundice to acute on chronic liver failure. Reversibility of liver dysfunction is possible in some cases with early medical management and complete abstinence from alcohol. Delays in seeking care are associated with higher rates of decompensated liver failure, organ transplantation, and mortality. Patients may not be aware of the need to seek care at the earliest signs of liver dysfunction, i.e. development of jaundice, and online sources such as generative artificial intelligence tools may not reflect the most up-to-date guidance. **Case Presentation:** A 27-year-old man with no significant past medical history presented to our academic medical center with several days of worsening abdominal distention and pain with scleral icterus. He admitted to several years of ongoing heavy alcohol use and endorsed an episode of jaundice around 8 months prior for which he did not seek medical care. Instead, he consulted a large language model (LLM) which advised that abstaining from alcohol alone would resolve his jaundice. He abstained for about 40 days and the jaundice re-

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solved. He then resumed drinking about 80 ounces of malt liquor per day until about 2 weeks prior to admission when he began to develop jaundice and abdominal distention. Abdominal ultrasound revealed hepatomegaly, portal hypertension, splenomegaly, and massive ascites. Admission labs showed hyponatremia, hypoalbuminemia, hyperbilirubinemia, coagulopathy, and thrombocytopenia. His MELD-Na score was 36. He was admitted to the ICU for management of acute on chronic liver failure and prednisolone and N-acetylcysteine were started based on his MDF score of 151. Paracentesis revealed a serum ascites-albumin gradient greater than 1.1, confirming portal hypertensive ascites and empiric antibiotics were started for SBP. Subsequently, he underwent evaluation for early liver transplantation. **Discussion:** This case illustrates the severe sequelae of a late presentation of alcoholic hepatitis. The patient had an earlier episode of jaundice which he managed on his own by following online advice from an artificial intelligence LLM chatbot to abstain from alcohol until the jaundice disappeared. Early and appropriate medical care at the time of asymptomatic jaundice might have enabled him to make a full recovery with medical management alone (i.e. steroids) as well as obtain support and resources for his alcohol use disorder rather than progressing to acute on chronic liver failure and necessitating evaluation for early liver transplantation. **Conclusion:** The medical community must ensure that patient-facing resources available on the Internet and through generative artificial intelligence tools reflect guideline-directed recommendations to patients to seek care at the earliest sign of liver damage to maximize the potential for reversibility of disease.

1700 | NOVEL HETEROZYGOUS ABCB4 INTRAGENIC DUPLICATION UNMASKED BY CRYPTOGENIC CHOLESTATIC LIVER DISEASE IN A YOUNG ADULT

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Introduction: ABCB4 encodes multidrug resistance protein 3 (MDR3). While biallelic pathogenic variants are classically associated with PFIC 3, heterozygous variants may present later in life with a broad and often underrecognized spectrum of cholestatic liver disease. We report a young woman with chronic cholestatic liver injury, fibrosis, and severe pruritus ultimately diagnosed with a novel likely pathogenic ABCB4 duplication. **Case Presentation:** A 27-year-old woman referred for evaluation of elevated LFT and hepatic fibrosis of unclear etiology. History of cholecystectomy at age 24, anorexia nervosa in remission, and alcohol use disorder with sustained abstinence. Family history of intrahepatic cholestasis of pregnancy and abnormal LFT in her mother. Laboratory evaluation noted a progressive cholestatic pattern with alkaline phosphatase (ALP) 311 IU/mL, alanine aminotransferase (ALT) 160 IU/mL, aspartate aminotransferase (AST) 78 IU/mL, gamma-glutamyl transferase (GGT) 260 IU/mL, and serum bile acids 40.5 µmol/L. Extensive autoimmune, viral, and metabolic testing was unrevealing. MRCP with no biliary obstruction or features of primary sclerosing cholangitis. Liver biopsy demonstrated grade 2, stage 2 chronic hepatitis with periportal fibrosis without an identifiable etiology. Given elevated GGT, persistent cholestasis, severe pruritus, and suggestive family history, a cholestatic genetic panel was obtained. Testing revealed a novel heterozygous 33.8-kb intragenic duplication involving ABCB4 exons 7-20, predicted to result in loss of function and classified as likely pathogenic. Treatment was initiated with weight-based ursodeoxycholic acid (UDCA). Because baseline pruritus was severe (5D Itch Score 23/25), maralixibat was subsequently added. After six months, ALP improved from 311 to 183 IU/mL, AST from 78 to 29 IU/mL, ALT from 160 to 43 IU/mL, GGT from 260 to 54 IU/mL, and bile acids from 40.5 to 13 µmol/L. The 5D Itch Score improved from 23 to 9. Mild loose stools were reported but remained tolerable. **Discussion:** This case highlights the diagnostic challenge of ABCB4-related liver disease in adults. Despite years of abnormal liver biochemistries and fibrosis on biopsy, conventional evaluation failed to identify a cause. Elevated GGT, early gallbladder disease, severe pruritus, and a family history suggestive of ABCB4-associated disease prompted genetic testing, which established the diagnosis. The marked biochemical and symptomatic response to combined UDCA and maralixibat further supports the potential utility of targeted therapy in selected adults with ABCB4-related cholestatic disease. **Conclusion:** Genetic testing should be considered in young adults with unexplained cholestatic liver disease, elevated GGT, and refractory pruritus. Identification of a novel likely pathogenic ABCB4 duplication provided a unifying diagnosis and enabled targeted treatment resulting in substantial clinical improvement.

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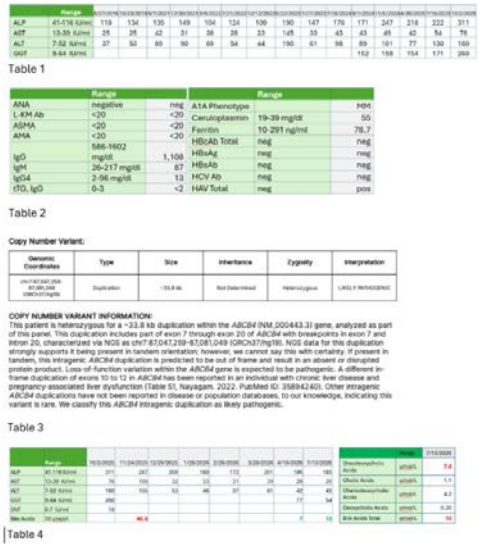


Table 1

Range			Range	
ANA	negative	neg	A1A Phenotype	MM
L-KM Ab	<20	<20	Ceruloplasmin	19-39 mg/dl	55
ASMA	<20	<20	Ferritin	10-291 ng/ml	78.7
AMA	<20	<20	HBcAb Total	neg	neg
IgG	586-1602 mg/dl	1,108	HBsAg	neg	neg
IgM	26-217 mg/dl	87	HBsAb	neg	neg
IgG4	2-96 mg/dl	13	HCV Ab	neg	neg
tTG, IgG	0-3	<2	HAV Total	neg	pos

Table 2

Copy Number Variant:

Genomic Coordinates	Type	Size	Inheritance	Zygosity	Interpretation
chr7:87,047,259-87,081,049 (GRCh37/hg19)	Duplication	~33.8 kb	Not Determined	Heterozygous	LIKELY PATHOGENIC

COPY NUMBER VARIANT INFORMATION:
This patient is heterozygous for a ~33.8 kb duplication within the *ABCB4* (NM_000443.3) gene, analyzed as part of this panel. This duplication includes part of exon 7 through exon 20 of *ABCB4* with breakpoints in exon 7 and intron 20, characterized via NGS as chr7:87,047,259-87,081,049 (GRCh37/hg19). NGS data for this duplication strongly supports it being present in tandem orientation; however, we cannot say this with certainty. If present in tandem, this intragenic *ABCB4* duplication is predicted to be out of frame and result in an absent or disrupted protein product. Loss-of-function variation within the *ABCB4* gene is expected to be pathogenic. A different in-frame duplication of exons 10 to 12 in *ABCB4* has been reported in an individual with chronic liver disease and pregnancy-associated liver dysfunction (Table S1, Nayagam, 2022, PubMed ID: 35894240). Other intragenic *ABCB4* duplications have not been reported in disease or population databases, to our knowledge, indicating this variant is rare. We classify this *ABCB4* intragenic duplication as likely pathogenic.

Table 3

Table 4

5 D Itch Score	
Date	Score
10/2/2025	23
3/27/2026	15
7/6/2026	9

Interpretations:
0 for no itch
1-11 for mild itch
12-17 for moderate itch
18-21 for severe itch
>22 for very severe itch

Table 5

2699 | TWO DAA FAILURES. DECOMPENSATED LIVER DISEASE. LIMITED RESOURCES. WHAT NEXT? A MULTIDISCIPLINARY APPROACH TO HCV RETREATMENT IN PAKISTAN

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Introduction: Direct-acting antivirals (DAAs) have transformed hepatitis C virus (HCV) treatment, yet Pakistan carries one of the world's largest HCV burdens. Salvage therapy in treatment-experienced patients with advanced liver disease remains challenging where recommended regimens, resistance testing, and affordable monitoring are inaccessible. We describe a pharmacist-integrated multidisciplinary approach to guideline-supported HCV salvage therapy in a resource-limited setting. **Case Presentation:** A 54-year-old woman (83 kg) with seronegative rheumatoid arthritis, without diabetes or hypertension, presented on January 8, 2024, with genotype 3 HCV after two previous courses of sofosbuvir/daclatasvir/ribavirin for 3 and 6 months, respectively, followed by virologic relapse. HCV RNA was 1,010,000 IU/mL. Ultrasound showed cirrhotic changes with massive ascites. She was Child-Pugh B9 with MELD 12; albumin 2.5 g/dL, bilirubin 1.7 mg/dL, INR 1.33, and platelets 58×10³/µL. Resistance testing and elastography were inaccessible. Glecaprevir/pibrentasvir was considered but was not feasible in decompensated disease. A multidisciplinary team of hepatologists, physicians, and clinical pharmacist selected sofosbuvir/velpatasvir plus ribavirin for 6 months, consistent with AASLD/IDSA guidance. VIKTANA (sofosbuvir/velpatasvir), manufactured in Pakistan under Gilead's authorized licensing framework, was accessed. Local availability reduced treatment cost, while AIMS welfare support and reduced laboratory costs further lowered the patient's financial burden. **Discussion:** Within 15 days, HCV RNA fell to <13.7 IU/mL. It became undetectable by June 8, 2024, and remained undetectable until February 6, 2026. Child-Pugh improved from B9 to A6 and MELD from 12 to 9. Albumin increased to 3.1 g/dL, bilirubin decreased to 0.7 mg/dL, and AFP decreased from 20.64 to 11.39 IU/mL. Follow-up ultrasound no longer demonstrated the previously reported cirrhotic changes, although hepatomegaly with increased echogenicity and coarse echotexture persisted. This case demonstrates how pharmacists can translate evidence into achievable care by coordinating therapeutic decisions, treatment access, and monitoring. **Conclusion:** Pharmacist-integrated multidisciplinary care can bridge the gap between guideline-supported HCV salvage therapy and real-world access in high-burden LMICs. Local authorized production and access partnerships can reduce treatment costs and improve affordability; similar initiatives from other manufacturers could expand access to salvage therapy in countries such as; Pakistan.

2700 | TO RESECT OR REDUCE FLOW? MANAGEMENT OF AN ENLARGING IDIOPATHIC EXTRAHEPATIC PORTAL VEIN ANEURYSM

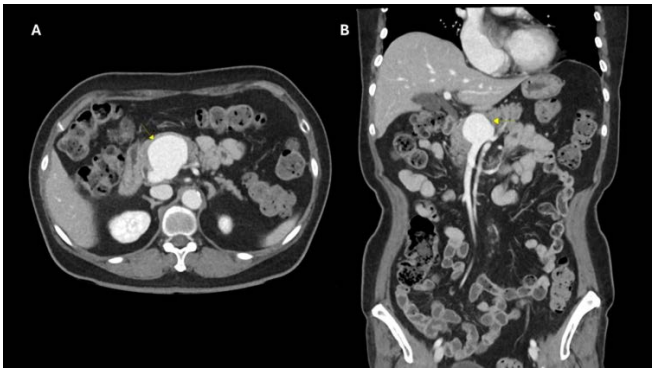
Jimyeong Son[1] Lawrence Lau[1] Nabil N. Dagher[1] James Park[1]

[1]North Shore University Hospital, Northwell Health

Introduction: Portal vein aneurysms (PVAs) are rare, and management is not standardized. Most asymptomatic lesions undergo surveillance, but interval growth may shift the risk-benefit balance, especially when repair is anatomically complex. We describe an enlarging idiopathic extrahepatic PVA in a patient without cirrhosis or portal hypertension. **Case Presentation:** A 63-year-old woman with coronary artery disease, HFpEF, and type 2 diabetes was referred for an incidentally identified aneurysm of the main portal vein. The patient had no risk factors such as cirrhosis, surgery, or trauma. There was no history of pancreatitis. The aneurysm was first noted on CT in 2022, measuring approximately 3.4 × 3.9 cm. MR venography and Doppler ultrasonography showed normal liver morphology, hepatopetal flow, and patent portomesenteric veins. She was asymptomatic, with no abdominal pain,

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ascites, splenomegaly, thrombosis, or other features of portal hypertension; liver enzymes, bilirubin, albumin, INR, and platelet count were normal. CT in January 2026 showed enlargement, with repeat imaging in June 2026 measuring 4.2 × 4.0 cm. With continued enlargement, hepatobiliary surgery considered aneurysm resection with portal vein reconstruction. Because of its retropancreatic location, adequate surgical exposure and safe reconstruction would require pancreatectomy. The plan subsequently shifted to splenic artery embolization to potentially reduce splenic venous inflow, followed by surveillance for stability. **Discussion:** Recent natural-history data suggest that most PVAs, including lesions greater than 3 cm, remain stable on surveillance.[1] Long-term observational data further suggest that large size alone should not mandate surgical intervention.[2] This case illustrates why serial behavior and anatomy may be more informative than size or symptoms alone. Despite preserved hepatic function and no portal hypertension, the lesion enlarged after relative stability. At the same time, its location behind the pancreas makes definitive reconstruction potentially morbid, with risks of pancreatic leak, bleeding, and difficult glycemic control. The shift from surveillance to a staged, flow-modifying strategy reflected individualized assessment of growth, vessel patency, anatomy, and procedural risk. **Conclusion:** The treatment approach to an asymptomatic idiopathic portal vein aneurysm in a noncirrhotic patient is not well defined. Interval growth warrants renewed multidisciplinary review, while the choice between continued surveillance, endovascular therapy, and surgery should be guided by anatomy and procedural risk rather than diameter alone.References - Binko MA, Andraska EA, Reitz KM, et al. J Vasc Surg Venous Lymphat Disord. 2025;13:102163. - Ahmed O, Ohman JW, Vachharajani N, et al. HPB (Oxford). 2021;23:127-133.



3599 | HEPATITIS C GENOTYPE 3 TREATMENT FAILURE IN PREGNANCY: CONSIDERATIONS FOR FIRST-LINE TREATMENT

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Introduction: A 27-year-old Rohingya woman presented with a new diagnosis of hepatitis C virus (HCV) in pregnancy and consented to participate in the Safety, Tolerability, and Outcomes of Sofosbuvir/Velpatasvir (SOF/VEL) in Treatment of Chronic Hepatitis C Virus During Pregnancy (STORC) trial. At screening, she had a viral load of 101,000 IU/mL with genotype 3 infection. She completed treatment, reported good adherence, but was found to have detectable HCV RNA at delivery and subsequently confirmed to have treatment failure. She was the second Rohingya refugee in the trial; the other participant also had genotype 3 infection but achieved SVR with SOF/VEL treatment. **Case Presentation:** This participant initiated SOF/VEL at 22 weeks of gestation without evidence of cirrhosis. She had follow-up visits every 4 weeks as per the STORC protocol, including HCV RNA evaluation at week 4, 8 and 12 (EOT). Treatment adherence was closely monitored within the trial, and she reported missing no doses. She was not being prescribed or taking any other medications or substances while on treatment. HCV RNA was not detectable during on-treatment monitoring, or at the end of treatment. She had a healthy baby by Caesarean section, 4 weeks after treatment completion. Her HCV RNA at delivery was 1,540,000 IU/mL. The participant did not have any active risks for HCV acquisition/reinfection, and both her partner and her other child were tested and were confirmed to be HCV antibody negative within the last six months. At the SVR12 timepoint, her viral load was 74,700 IU/mL and the genotype was confirmed to again be 3. Samples were sent for routine resistance testing which confirmed likely resistance to VEL, and sequencing for SOF resistance could not be completed. Full-genome sequencing and genotype subtyping are in progress. The infant was tested at 12 weeks of age, and is HCV RNA undetectable. **Discussion:** This relapse was not anticipated. Further inquiries about HCV treatment outcome in the local Rohingya community, found other treatment relapses/failures. All relapses located to date were recorded in people with genotype 3 infection. Resistance testing profiles available for others were similar to this patient. In the era of pangenotypic therapy,

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resistance testing delays treatment initiation, and in most cases, does not alter clinical management,thus not needed. However, in the context of pregnancy, where shared-decision making for use of off label medications and one of the major goals of treatment being to prevent vertical transmission, choosing the most appropriate therapy is critical. **Conclusion:** For Rohingya refugees, given the resistance profiles seen in genotype 3 infection, alternative strategies for treatment in pregnancy may need to be considered, With limited data for other regimens, treatment with SOF/VEL may still be appropriate, however, it may be prudent to continue treatment until delivery to reduce the chance of viremia and vertical transmission at delivery.

3600 | SEVERE HYPERBILIRUBINEMIA WITH HCV THERAPY

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Introduction: We report a unique case of acute liver injury in a young patient without cirrhosis 3 weeks after starting glecaprevir/pibrentasvir (GP) with a prolonged and protracted course of hyperbilirubinemia. **Case Presentation:** A 21 yo male with a documented prior SVR with sofosbuvir/velpatasvir had HCV reinfection and started GP. Past medical history was significant for injection drug use. He had a documented penicillin allergy. At baseline there was no evidence of cirrhosis with AST 23, ALT 23, total bilirubin (Tbili) 1.3, platelet 263k, HCV RNA 222 IU/mL, HIV antibody (ab) negative, HAV IgG reactive, HBcAb negative, HBsAg negative, HBsAb negative. He was on no other medications and denied on-going illicit drug use. He started GP according to a simplified treatment approach. Within approximately 3 weeks he complained of itching. Laboratories at treatment week 4 were drawn: AST 43, ALT 38, Tbili 12.5, and alkaline phosphatase (ALK) 276. He was noted to have a normal abdominal exam and in good spirits albeit itchy. Repeat labs done 1 week after stopping GP indicated total bilirubin 20.7 (direct 8.6), ALK 344, albumin 4.7, INR 1.09, AST 52, ALT 44. Physical exam was unremarkable except for jaundice. He was subsequently sent for CT and US which were read as "normal except for splenomegaly" with a spleen size of 15.8 cm. MRCP was negative for obstructive cholelithiasis or dilation of biliary collecting system.A few days later, he was noted to be nauseated and unable to tolerate food or water. At that time he was admitted to a hospital where labs continued to deteriorate with ALK 380, AST 80, ALT 70, albumin 3.5, ALK 450, Tbili 28.4 mg/dL (direct 20.5), and an INR of 1.2. Viral hepatitis serologies were again negative. A liver biopsy was performed and was consistent with drug induced liver injury. He was subsequently discharged. Total bilirubin was 25.1 (direct 19.3). At 4 weeks post, Tbili increased to 40.4, INR 1.42, and ALK peaked at 524. An autoimmune workup was negative. Repeat HAV, HBV, HCV and EBV negative. At approximately 5 weeks post, INR was 1.63 and a trial of vitamin K was started with resolution within days to a level of 1.05. At nearly 7 weeks post, Tbili peaked at 49.1 and he was started on ursodiol 300 mg PO BID. By 12 weeks post, his Tbili was 17.9. He continued to have repeat labs with complete resolution of his Tbili to baseline at 22 weeks post stopping GP. HCV RNA remains undetectable. **Discussion:** The potential for GP to expose familial hyperbilirubinemia was considered however genetic testing was inconclusive. The CFTR gene was identified as heterozygous and is associated with autosomal recessive cystic fibrosis. The SERPINA-1 gene was also noted as heterozygous with low penetrance and is associated with recessive alpha-1-antitrypsin deficiency. Additional variants of uncertain significance were identified. **Conclusion:** This case exposed a rare hyperbilirubinemia with GP which continued to worsen after stopping therapy. Improvement was seen with ursodiol therapy.

3601 | WHEN IT ISN'T AFLP: METASTATIC BREAST CANCER PRESENTING AS ACUTE LIVER FAILURE IN PREGNANCY

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Introduction: Acute liver failure (ALF) in pregnancy is most often attributed to pregnancy-specific conditions such as acute fatty liver of pregnancy (AFLP) or HELLP syndrome. Diffuse malignant infiltration of the liver is a rare, frequently fatal mimic. We describe a pregnant patient initially thought to have AFLP whose evolving course ultimately revealed presumed metastatic breast cancer. **Case Presentation:** A 35-year-old woman at 30 weeks gestation, with HIV diagnosed at a prenatal visit three months prior (on bictegravir/emtricitabine/tenofovir, CD4 150 cells/μL), was transferred from an outside hospital with nausea, vomiting, abdominal pain, and hepatomegaly. Outside

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imaging suggested Budd-Chiari syndrome, but repeat Doppler ultrasound and CT angiography demonstrated patent hepatic veins. Admission labs showed AST 141 U/L, ALT 544 U/L, and hemoglobin 8.1 g/dL, with a 26 cm liver span. Hypercalcemia (14.4 mg/dL) and diffuse osteolytic lesions on CT prompted evaluation for multiple myeloma. On hospital day 7, AST rose to 4,258 U/L with INR 1.48, ammonia 66 µmol/L with asterixis, and new ascites. Given concern for AFLP with evolving ALF, she received betamethasone and was transferred to a liver transplant center; breast examination before transfer revealed a firm 3-cm right breast mass with cervical and supraclavicular adenopathy. She underwent emergent cesarean delivery at 31 weeks for worsening maternal status. Myeloma and lymphoma evaluations were negative, while imaging demonstrated innumerable hepatic lesions with a pseudocirrhosis pattern, diffuse lytic osseous lesions, and bilateral breast masses with axillary adenopathy, consistent with metastatic breast cancer. Critical illness precluded tissue biopsy and systemic therapy. Her course was complicated by hyperammonemia requiring renal replacement therapy, prolonged mechanical ventilation, Candida auris fungemia, spontaneous bacterial peritonitis, and hemorrhagic shock from hemoperitoneum requiring embolization. She transitioned to inpatient hospice and died five weeks after presentation; her infant survived. **Discussion:** Malignant infiltration accounts for under 1% of ALF cases but is nearly universally fatal without treatment of the underlying malignancy; breast cancer is a leading culprit and can produce a pseudocirrhosis pattern mimicking chronic liver disease. Pregnancy compounds the diagnostic challenge: physiologic breast changes obscure masses, imaging is limited, and clinicians anchor on obstetric etiologies such as AFLP and HELLP. Hypercalcemia with lytic lesions warrants evaluation beyond myeloma, and ALF that progresses despite delivery should prompt reconsideration of pregnancy-specific diagnoses. **Conclusion:** Malignant infiltration should be considered in ALF with disproportionate hepatomegaly, even in pregnancy; its early exclusion is essential, as it contraindicates liver transplantation and reframes goals of care.

3602 | CRYPTOCOCCUS IN DISGUISE: PROGRESSIVE HEPATIC LESIONS MIMICKING MALIGNANCY IN AN IMMUNOCOMPETENT PATIENT

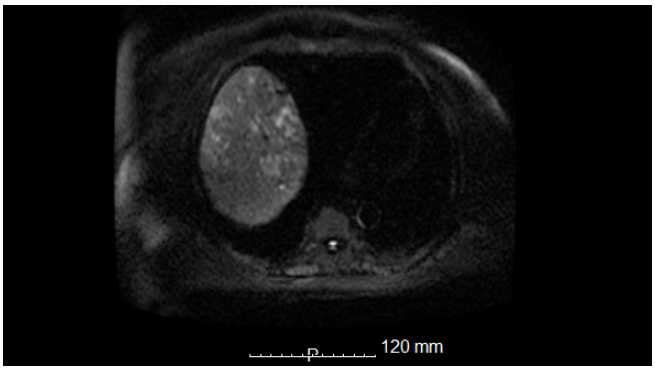
Rafi Haddad[1] Rhiannon Bou Deleh[1] Roopa Velaga[1] Carl Akel[1]

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Introduction: Hepatic cryptococcosis is rare, particularly in immunocompetent hosts, and may mimic primary or metastatic malignancy. We present progressive mass-like hepatic lesions with caseating granulomatous inflammation and persistent cryptococcal antigenemia. **Case Presentation:** An 80-year-old immunocompetent woman with remote renal cell carcinoma (RCC) status post partial nephrectomy presented after a fall and was incidentally found to have elevated liver enzymes: aspartate aminotransferase (AST) 441 U/L, alanine aminotransferase (ALT) 244 U/L, alkaline phosphatase (ALP) 501 U/L, and total bilirubin 2.1 mg/dL. Ultrasound demonstrated a 1.8-cm common bile duct (CBD) and suspicious hepatic dome lesion. Magnetic resonance cholangiopancreatography (MRCP) revealed choledocholithiasis with biliary dilation and a 10×9.7-cm mass-like process in the right hepatic dome concerning for malignancy. CBD stones were removed by endoscopic retrograde cholangiopancreatography (ERCP). Carcinoembryonic antigen (CEA) was 2.8 ng/mL; alpha-fetoprotein and carbohydrate antigen 19-9 were unremarkable. Chest computed tomography (CT) and brain magnetic resonance imaging showed no metastases. A core liver biopsy showed caseating granulomatous inflammation without malignancy. Acid-fast bacilli (AFB) and Grocott methenamine silver (GMS) stains were negative; tissue and AFB cultures showed no growth. Human immunodeficiency virus testing was nonreactive. Serum cryptococcal antigen (CrAg) was positive at 1:5. Coccidioides immunoglobulin G was initially positive with negative immunoglobulin M; subsequent serology and complement fixation were negative. Beta-D-glucan was negative. Fluconazole was initiated.Two months later, she was readmitted after another fall. Despite near-normalization of liver enzymes (AST 28 U/L, ALT 7 U/L, ALP 130 U/L, bilirubin 0.4 mg/dL), CT showed multiple hepatic lesions increased in size and number, again concerning for malignancy. CEA increased to 6.5 ng/mL and serum CrAg remained positive. Cerebrospinal fluid CrAg and culture were negative. Given progressive hepatic lesions, caseating granulomas, persistent antigenemia, and no malignancy on biopsy, hepatic cryptococcosis was favored and therapy escalated to liposomal amphotericin B. **Discussion:** This case highlights malignancy-like hepatic lesions despite biochemical improvement and granulomatous histology. Persistent CrAg and exclusion of other diagnoses supported hepatic cryptococcosis. Negative fungal stains should not exclude infection when clinical, radiographic, and pathologic findings are discordant. **Conclusion:** Hepatic cryptococcosis should remain in the differential of granulomatous hepatic lesions even in immunocompetent hosts with negative fungal stains. Integrating histopathology, fungal testing, and serial imaging may prevent an infectious hepatic pseudotu-

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mor from being mistaken for malignancy.



3603 | A RARE CAUSE OF ANICTERIC HEPATITIS AND HLH: DONOR-DERIVED DISSEMINATED HSV-1 FOLLOWING LUNG TRANSPLANTATION

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Introduction: Herpes simplex virus (HSV) hepatitis is a rare complication of disseminated HSV infection. More than half of cases occur in immunocompromised patients and can manifest with an anicteric hepatitis pattern contributing to delays in diagnosis and high mortality.1,2 In rare cases, disseminated HSV infections in immunocompromised patients may lead to a hyperinflammatory syndrome with hemophagocytic lymphohistiocytosis (HLH) progressing to life-threatening multiorgan failure.3,4 We report a case of a lung transplant patient with an acute liver injury (ALI) attributed to HSV hepatitis and secondary HLH who improved with early initiation of empiric acyclovir. **Case Presentation:** A 67-year-old male with a history of end-stage interstitial lung disease status post bilateral lung transplant was found to have acute elevations in transaminases to >1000 U/L on post-operative day 14. Liver function was intact with platelet count >300 K/UL, INR 1.2, and Factor V activity 83%. Infectious and autoimmune workup for acute hepatitis were initially unrevealing, and empiric IV acyclovir was started pending send-out serologies. Additional labs obtained revealed significantly elevated ferritin >100,000 ng/mL, fibrinogen 570 mg/dL, and triglycerides 429 mg/dL with bicytopenia and an HScore of 204, concerning for HLH. ALT/AST reached a maximum of 1977 U/L and 3984 U/L and started to downtrend to <1000 U/L within 3 days of starting acyclovir. On post-operative day 18, HSV-1 PCR and CSF were positive concerning for disseminated HSV infection suspected to be donor-derived due to previously negative HSV IgG. A bone marrow biopsy demonstrated hemophagocytes, and additional testing revealed elevated sIL-2R and low NK cell activity supporting a diagnosis of HLH. The patient was continued on a 21-day acyclovir course with subsequent improvement in his transaminases and HLH-associated labs, favoring a diagnosis of donor-derived HSV-1 hepatitis with secondary HLH. **Discussion:** HSV hepatitis poses a challenge in early diagnosis and treatment due to its nature of rapid progression and delays in laboratory confirmation. Previous cases have been described with delayed diagnosis in solid organ transplant patients leading to severe complications including disseminated intravascular coagulation and death.5 Moreover, disseminated HSV infection in solid organ transplant patients may lead to secondary HLH, contributing to a widespread inflammatory process and confounding the clinical picture. **Conclusion:** This case highlights 1) the potential utility in considering disseminated HSV infection in solid organ transplant cases with HLH-like syndromes, and 2) the importance of initiating early empiric acyclovir in solid organ transplant patients presenting with severe hepatitis without other identifiable causes.

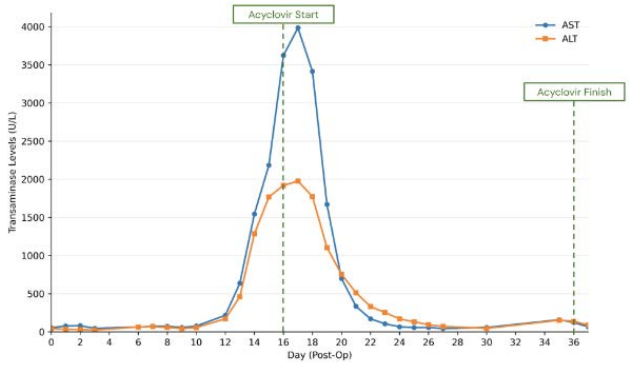


Figure 1. Transaminase (AST and ALT) trends from post-operative day 0 to post-operative day 36. IV acyclovir initiated on post-operative day 16 and completed on day 36.

3604 | FUNCTIONAL CURE OF CHRONIC HEPATITIS B IN AN INFANT TREATED WITH TENOFOVIR ALAFENAMIDE MONOTHERAPY

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Introduction: Functional cure of chronic hepatitis B (CHB) remains a major unmet clinical need. Recent studies suggest that the rate of clinical cure in children is inversely associated with age, indicating that

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younger patients may have a greater likelihood of achieving clinical cure. However, the clinical characteristics, antiviral response, and functional cure of infants with CHB under 1 year of age remain largely unknown. **Case Presentation:** We report the case of an 8-month-old infant with CHB acquired through mother-to-child transmission, who exhibited features of the immune clearance phase, with a hepatitis B surface antigen (HBsAg) level of 8,875 IU/mL, positive hepatitis B e antigen (HBeAg), a hepatitis B virus (HBV) DNA level of 9.94×10^7 IU/mL, and an alanine aminotransferase (ALT) level of 189 U/L. Because lamivudine was no longer available in Henan Province, and the infant was unable to tolerate entecavir or tenofovir, developing vomiting and choking after administration, treatment with tenofovir alafenamide (TAF, 1 mg/kg/day) was initiated following approval by the institutional ethics committee. After 5 months of antiviral therapy with TAF tablets, the infant achieved undetectable HBV DNA and normalization of ALT. At 8 months of treatment, both HBeAg and HBsAg became negative. The patient then received hepatitis B vaccination according to the "0-, 1-, and 6-month" schedule and subsequently developed high levels of hepatitis B surface antibody (682 mIU/mL). Consolidation therapy was continued until month 12, after which treatment was discontinued. Ultimately, clinical cure was achieved 6 months after treatment cessation. **Discussion:** This case challenges the traditional concept that young children with chronic HBV infection, particularly infants, are almost exclusively in the immune-tolerant phase and therefore do not require antiviral therapy. Because infants with CHB have historically received limited clinical attention, the true proportion of those entering the immune clearance phase remains uncertain, and antiviral treatment in this population may have been underestimated.The virological response observed in this infant was remarkably rapid. HBV DNA declined from approximately 7 log IU/mL at baseline to approximately 2 log IU/mL after only 1 month of therapy, with undetectable HBV DNA documented at month 5, although viral suppression may have occurred even earlier because of the interval between follow-up visits. HBsAg and HBeAg also declined rapidly and subsequently became undetectable. **Conclusion:** This case suggests that even an infant with CHB can exhibit features of the immune clearance phase and unexpected rapid virological response during treatment. To our knowledge, this is the first report demonstrating the safe use of TAF resulting in functional cure in an infant with CHB. Larger studies are needed to validate these findings and further evaluate the safety and efficacy of TAF in this population.

3605 | STRIKE WHILE THE VIRUS IS HOT: NAVIGATING HEPATITIS C VIRUS-ASSOCIATED CRYOGLOBULINEMIC GLOMERULONEPHRITIS WITH CONCURRENT HEPATOCELLULAR CARCINOMA

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Introduction: In the era of highly effective direct-acting antiviral (DAA) therapy for hepatitis C virus (HCV), extrahepatic manifestations of untreated HCV are less commonly observed and may be overlooked when competing complications of advanced liver disease are present. In patients with newly diagnosed hepatocellular carcinoma (HCC), DAA therapy is often deferred due to lower rates of sustained virologic response (SVR) in presence of active malignancy. We present a case of HCV-associated cryoglobulinemic glomerulonephritis (GN) that altered the timing of antiviral therapy in a patient with decompensated cirrhosis and HCC. **Case Presentation:** A 56-year-old woman presented with dyspnea on exertion, back pain, and lower extremity edema. Through imaging and laboratory data, she was diagnosed with decompensated cirrhosis attributed to untreated HCV infection with additional metabolic risk factors. Magnetic resonance imaging identified multiple intrahepatic lesions consistent with multifocal HCC.During recurrent admissions for hepatic hydrothorax, she was found to have acute kidney injury, microscopic hematuria and proteinuria. Serologic testing demonstrated low C4, normal C3, trace cryoglobulins, negative rheumatoid factor, and atypical ANCA positivity with negative PR3/MPO. Despite the absence of classic systemic manifestations of cryoglobulinemia and only trace circulating cryoglobulins, renal biopsy demonstrated polyclonal deposition of immunoglobulins M and G consistent with HCV-associated cryoglobulinemic GN, type III.For the GN, she was treated with corticosteroids and two doses of rituximab, which led to a marked improvement in kidney function, removing the need for plasmapheresis. A 12-week course of sofosbuvir-velpatasvir was initiated while she underwent loco-regional therapy for multifocal HCC. At her last follow-up, her HCV viral load was undetectable, renal function had stabilized, and thoracentesis needs were decreasing. Systemic HCC therapy is being considered following completion of DAA therapy. **Discussion:** This case highlights two important considerations: first, kidney biopsy should be pursued in patients with HCV and nephritic features even with minimal circulating cryoglobulins and absent systemic manifestations; second, extrahepatic HCV disease alters the risk-benefit assessment of postponing antiviral

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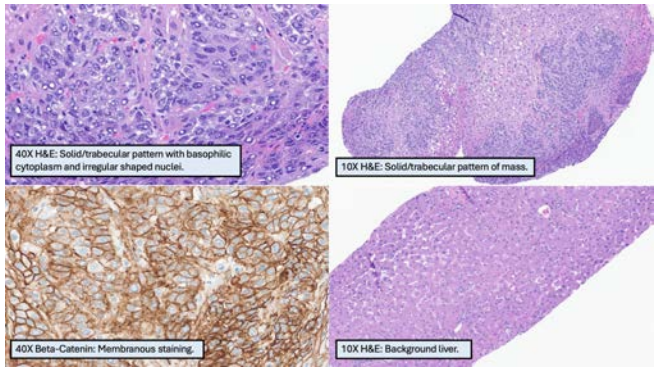
therapy in the setting of active HCC. Prompt initiation of DAA-mediated SVR halts the viral antigenic stimulation and immune-complex formation that underlie HCV-associated cryoglobulinemia, while B-cell depletion with rituximab provides adjunctive control of severe glomerulonephritis. **Conclusion:** Ultimately, individualized multidisciplinary coordination is essential when balancing HCV eradication with HCC-directed treatment.

3606 | HEPATOCELLULAR CARCINOMA IN A 17-YEAR-OLD WITH HISTORY OF GESTATIONAL ALLOIMMUNE LIVER DISEASE

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Introduction: Gestational Alloimmune Liver Disease (GALD) is a maternal-fetal alloimmune disorder that causes neonatal acute liver failure and may result in severe hepatic injury, extrahepatic siderosis (neonatal hemochromatosis, [NH]), and death. Histologically, affected infants have extensive parenchymal fibrosis with near-complete loss of hepatocytes. Treatment with intravenous immunoglobulin (IVIg) and double-volume exchange transfusion (DVET) can promote native liver recovery and fibrosis regression.Unlike other liver diseases in which chronic inflammation and fibrosis increase the risk of hepatocellular carcinoma (HCC), children with GALD who recover with their native liver are thought to regenerate healthy hepatic parenchyma without persistent liver injury or malignancy risk. However, long-term adult outcomes remain poorly characterized because GALD was first recognized in the early 2000s. **Case Presentation:** A neonate presented with acute liver failure in 2008. MRI and buccal biopsy demonstrated extrahepatic siderosis consistent with GALD after genetic, metabolic, infectious, and structural causes were excluded. She was treated with intravenous immunoglobulin (IVIg), N-acetylcysteine, alpha-tocopherol, selenium, and prostaglandin E1. She did not receive double-volume exchange transfusion (DVET) as this was not yet standard of care. After treatment she achieved complete recovery with restoration of native liver function. During three years of hepatology follow-up, she had normal liver enzymes and synthetic function without evidence of chronic liver disease. At 17 years of age, she re-presented with epigastric pain, nausea, and weight loss and was found to have a 25.7 × 13.4 cm hepatic mass. Biopsy confirmed hepatocellular carcinoma (HCC), with next-generation sequencing identifying an NRASp.Gln61Lys mutation (Figure 1). Background liver histology was normal, and evaluation for other chronic liver diseases, including viral hepatitis and alpha-1 antitrypsin deficiency, was negative. She is currently receiving chemotherapy and immunotherapy for HCC. **Discussion:** This is the first reported case of hepatocellular carcinoma (HCC) in a patient with a history of Gestational Alloimmune Liver Disease (GALD). Given the absence of chronic liver disease in the background liver, this most likely represents two rare, unrelated conditions. However, as the earliest GALD survivors reach adulthood with their native liver, continued reporting and long-term follow-up are needed to determine whether this is an isolated event or a previously unrecognized complication that could change current assumptions about disease resolution and surveillance. **Conclusion:** As increasing numbers of GALD survivors reach adulthood with their native liver, reporting outcomes is essential to guide long-term monitoring. Current experience continues to support full resolution of liver injury in patients with recovery from GALD.



3607 | BEYOND GVHD AND SOS: RAPID-ONSET HYDRALAZINE-INDUCED LIVER INJURY AFTER ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANTATION

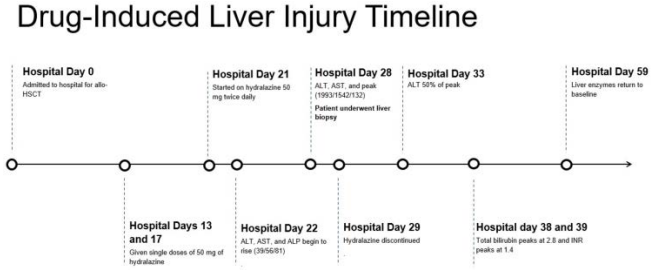
Eric O'Hara[1] Jason Lewis[1] Omar Y. Mousa[1] Kristen Ryland[1] Rasha Rashan[1]

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Introduction: Acute liver injury after allogeneic hematopoietic stem cell transplantation (allo-HSCT) warrants urgent evaluation for graft-versus-host disease (GVHD), sinusoidal obstruction syndrome (SOS), infection, and drug toxicity. We describe a case of rapid-onset acute hepatocellular injury associated with hydralazine in a patient following allo-HSCT, with clinical features overlapping those of several transplant-re-

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lated complications. **Case Presentation:** A 50-year-old man with acute myeloid leukemia and persistent hypertension was admitted following allo-HSCT. He received single doses of hydralazine 50 mg on hospital days 13 and 17, followed by escalation to a scheduled regimen of 50 mg twice daily on hospital day 21. His liver enzymes were at baseline prior to initiating hydralazine (ALT 12 U/L, AST 11 U/L, ALP 93 U/L). On hospital day 22, 9 days after his first dose of hydralazine, his liver enzymes began to gradually increase, peaking on hospital day 28 at ALT 1993 U/L, AST 1542 U/L, and ALP 132 U/L.During this period, the patient reported nonspecific symptoms, including intermittent fever, nausea, and diarrhea, raising concern for graft-versus-host disease (GVHD), viral reactivation, or hepatic sinusoidal obstruction syndrome (SOS). An extensive evaluation for alternative etiologies, including acute viral hepatitis, HSV, EBV, autoimmune liver disease, and other potential hepatotoxic exposures, was unrevealing. Medication reconciliation confirmed that the patient was taking no other medications or herbal supplements. Abdominal ultrasound showed no acute abnormalities. A liver biopsy performed on hospital day 28 demonstrated lobular hepatitis without evidence of GVHD or SOS, findings consistent with drug-induced liver injury (DILI). The R value was 45.3, indicating a hepatocellular pattern of injury, and the Roussel Uclaf Causality Assessment Method (RUCAM) score was 10, indicating a high probability of DILI. Following discontinuation of hydralazine, ALT declined by 50% within 4 days and normalized approximately 30 days after discontinuation. **Discussion:** This case highlights the diagnostic challenge of identifying DILI in an immunocompromised patient with multiple competing causes of liver injury in the setting of nonspecific symptoms. The temporal association between hydralazine initiation and the development of liver injury, exclusion of competing causes, compatible histology, and subsequent improvement following drug discontinuation supported hydralazine DILI as the likely cause. Although hydralazine DILI is established, the teachable feature is its post-HSCT context: transplant-specific diagnoses can obscure a newly introduced medication. **Conclusion:** In post-HSCT liver injury, medication chronology should be reviewed in parallel with GVHD, SOS, and infection. Early recognition and culprit withdrawal can facilitate recovery and may prevent unnecessary treatment.



3608 | EOSINOPHILIC HEPATITIS AND EOSINOPHILIC CHOLANGIOPATHY IN A PATIENT WITH HYPEREOSINOPHILIC SYNDROME

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Introduction: Eosinophilic hepatitis and eosinophilic cholangitis are rare manifestations of hypereosinophilic syndrome. We present a case of cholestatic hepatitis and biliary strictures from eosinophilic hepatitis and eosinophilic cholangitis respectively. **Case Presentation:** A 31-year-old man was referred to hepatology for evaluation of liver enzymes elevation (AST 61 U/L, ALT 121 U/L, ALP 374 U/L). He was asymptomatic. CBC with differentials showed significant peripheral eosinophilia of 59%. Autoimmune, infectious, genetic, and metabolic evaluation were unremarkable. MRI-MRCP demonstrated mild multifocal peripheral intrahepatic biliary dilatation, but no biliary strictures were noted. Liver biopsy showed portal and lobular eosinophilic infiltration consistent with eosinophilic hepatitis. The patient was started on budesonide resulting in biochemical improvement. After being lost to follow up, he presented a year later with abdominal pain and fever. Liver biochemistries were AST 286 U/L, ALT 264 U/L, ALP 564 U/L, CBC showed WBC 19.5 10(3)/uL and eosinophils of 61.6% (absolute eosinophil count: 12.0 x 10(3)/uL). MRI- MRCP showed multifocal biliary strictures. ERCP revealed diffuse multifocal biliary strictures with sludge and purulent debris. EUS showed enlarged porta hepatis lymph nodes, and fine-needle aspiration cytology demonstrated predominant eosinophils and occasional plasma cells. IGG4 immunostaining was negative. Antibiotic was started and after resolution of acute cholangitis, prednisone 40 mg/day was started. Mepolizumab was initiated for long-term management of hypereosinophilic syndrome. Liver enzymes and hypereosinophilia normalized completely and prednisone was successfully tapered to 5 mg daily. Repeat MRI-MRCP showed improvement in biliary strictures. **Discussion:** Eosinophilic cholangitis may mimic PSC and IGG-4 sclerosing cholangitis. Evaluation for hypereosinophilia must be performed in a patient with biliary structuring disease. In a patient with hypereosinophilic syndrome, presence of significant eosinophils in liver biopsy and/or biliary strictures would confirm eosinophilic hepatitis and/or cholangitis. **Conclusion:** Eosinophilic hepatitis and eosinophilic cholangitis must be considered in a patient with hypereosinophilic syndrome pre-

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senting with cholestatic liver injury. Eosinophilic hepatitis and eosinophilic cholangiopathy seem to respond well to corticosteroid therapy and management of hypereosinophilic syndrome.

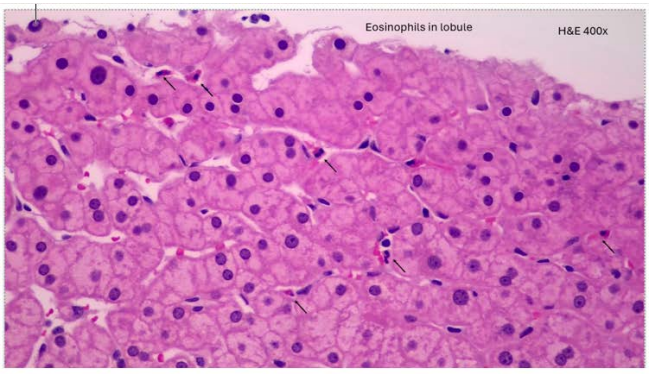


Figure 1 – Liver biopsy: Eosinophils in lobule

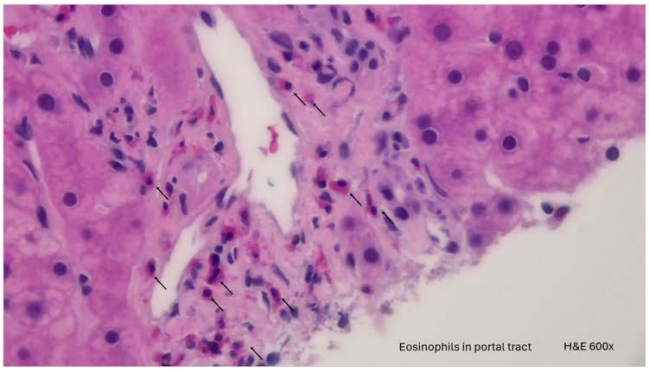


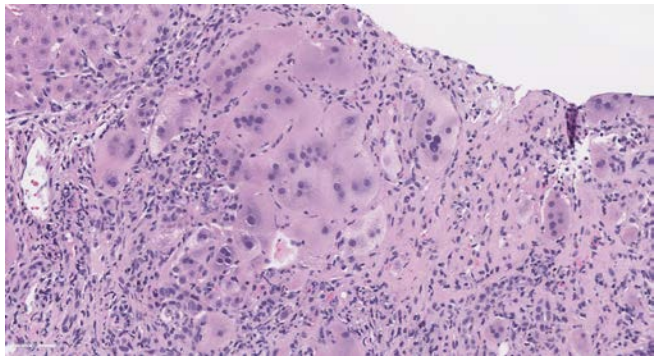
Figure 2 – Liver biopsy: Eosinophils in portal tract

3609 | POST-INFANTILE GIANT CELL HEPATITIS REVEALING AUTOIMMUNE HEPATITIS DESPITE TACROLIMUS IMMUNOSUPPRESSION

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Introduction: Giant cell hepatitis (GCH) is a histopathologic diagnosis characterized by large multinucleated hepatocytes and parenchymal inflammation in liver injury. GCH is predominantly described in pediatric populations, while post-infantile giant cell hepatitis (PIGCH) is exceedingly rare in adults. **Case Presentation:** A 21-year-old man with immune complex-mediated glomerulonephritis (IC-MPGN) on tacrolimus presented to the hospital for nausea and acute abdominal pain. Lab evaluation demonstrated severe hepatocellular injury and coagulopathy with ALP 259 U/L, AST 1,350 U/L, ALT 1843 U/L, Total Bilirubin 4.0 mg/dL, Direct Bilirubin 3.1 mg/dL, albumin 2.3 g/dL, and INR 3.29. A year prior liver tests were normal, but four months prior patient had evidence of injury with ALP 150 U/L, AST 146 U/L, ALT 193, Total Bilirubin 0.6 mg/dL, Albumin 3.3 g/dL, and INR 1.30. Imaging demonstrated hepatosplenomegaly. He had no hepatic encephalopathy, history of liver disease, substance use, or new medications or supplements. Evaluation for viral hepatitis, including HEV, Wilson disease, and other infectious etiologies was unrevealing. Given concern for an immune-mediated process, autoimmune hepatitis (AIH) remained on differential. ANA and anti-mitochondrial antibody were negative; however, anti-smooth muscle antibody was elevated at 1:640 with IgG 3,787 mg/dL. Subsequent liver biopsy demonstrated severely active hepatitis with extensive parenchymal collapse and syncytial hepatic giant cells, compatible with PIGCH in the setting of AIH. Despite treatment with intravenous methylprednisolone, hepatic dysfunction progressed, and he underwent orthotopic liver transplantation. Explant pathology revealed cirrhosis with active hepatitis, syncytial giant cells, and extensive lobular injury with marked ductular reaction. **Discussion:** PIGCH is not itself a disease, but a histopathologic pattern signaling severe liver injury. A rare manifestation in adults, PIGCH is most often associated with autoimmune processes, particularly AIH. Other causes include viral infections, drug and toxin injury, malignancy, metabolic disease, and post-transplantation states. Identifying the underlying etiology is imperative, as outcomes range from recovery to progressive hepatic dysfunction requiring transplantation. This case highlights that AIH-associated PIGCH can occur despite pre-existing calcineurin-inhibitor immunosuppression, negative ANA, and may progress despite corticosteroid therapy. **Conclusion:** PIGCH in adults warrants expedited evaluation for an underlying etiology, including AIH despite negative ANA. This case demonstrates that AIH-associated PIGCH may progress despite pre-existing immunosuppression and corticosteroid therapy. Early recognition or progressive hepatic dysfunction and timely transplantation assessment are essential.



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3611 | BEYOND SHOCK LIVER: SECONDARY SCLEROSING CHOLANGITIS AFTER VV-ECMO

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Introduction: Secondary sclerosing cholangitis in critically ill patients (SSC-CIP) is a progressive ischemic cholangiopathy characterized by persistent cholestasis, biliary casts, and multifocal stricturing after critical illness. Bile colonization, cholangitis, and abscess formation is a prognostically significant feature. Early disease may be difficult to distinguish from other causes of liver injury. We describe a case of SSC-CIP after VV-ECMO, diagnosed among competing etiologies of elevated liver enzymes. **Case Presentation:** A 44-year-old man with status asthmaticus required intubation and 17 days of VV-ECMO with hospital course complicated by shock, HSV viremia, rhabdomyolysis, and acute kidney injury. Liver tests were normal at ECMO initiation. During ECMO, AST/ALT increased to 526/338 U/L with normal alkaline phosphatase (ALP) amid shock and severe rhabdomyolysis (CK ~64,000 U/L). Near decannulation, AST/ALT rose to 615/921 U/L with new elevations in ALP and bilirubin. Over subsequent weeks, aminotransferases fluctuated and normalized while ALP and bilirubin progressively increased. Hepatology was consulted when ALP reached 1127 U/L. Differential diagnoses included critical illness-associated cholestasis, cholestatic drug-induced liver injury from ceftriaxone, ischemic hepatopathy, and rhabdomyolysis-associated aminotransferase elevation. MRI/MRCP demonstrated multifocal intrahepatic stricturing. ERCP showed a dominant left hepatic duct stricture; biliary casts were extracted and two plastic stents placed. Persistent cholestasis prompted repeat ERCP two weeks later, demonstrating severe diffuse stricturing of the right and left hepatic ducts and intrahepatic branches, consistent with SSC-CIP. In line with SSC-CIP's infection predilection, his hospitalization was complicated by *E. faecalis* bacteremia and cholangitis which progressed to form hepatic abscesses requiring several weeks of Zosyn therapy. Despite antibiotics/stenting, bilirubin kept rising and he developed post-ERCP ampullary bleeding requiring a covered metal biliary stent, critical illness neuropathy, and dialysis initiation. He is being considered for simultaneous liver-kidney transplantation. **Discussion:** Normalization of aminotransferases should not reassure providers when cholestatic markers continue to rise. Divergent improvement in AST/ALT with progressive ALP and bilirubin elevation in patients on ECMO should prompt evaluation for SSC-CIP. Ischemic cholangiocyte injury, biliary cast formation, and progressive stricturing impair biliary drainage and predispose patients to recurrent cholangitis and hepatic abscesses which may require prolonged antibiotic therapy. **Conclusion:** SSC-CIP has poor transplant-free survival, highlighting the importance of early recognition and timely transplant evaluation.

HD #	Milestone	AST (U/L)	ALT (U/L)	ALP (U/L)	T. bili (mg/dL)
1	Admission / ECMO start	31	33	90	0.4
10	Peak transaminases on ECMO	526	338	82	0.4
18	ECMO decannulation (–HD 17)	615	921	272	3.5
31	Post-ECMO recovery	199	141	268	1.4
55	Hepatology consulted	179	199	1,127	2.5
62	ERCP #1 (L hepatic duct stent, HD 61)	179	184	1,095	4.2
76	ERCP #2 (diffuse strictures, bilateral stents, HD 75)	90	78	1,017	10.2
78	E. faecalis bacteremia / cholangitis	48	44	773	10.6
88	EGD/ERCP: ampullary bleed, covered metal stent	123	65	847	20.3

Note: HD = hospital day (HD 1 = admission/ECMO start, 5/22). HD 18, 62, and 76 are the first available draws after ECMO decannulation and each ERCP, respectively.

3612 | COMPLEX CAVOATRIAL RECONSTRUCTION FOR ACQUIRED SUPRAHEPATIC INFERIOR VENA CAVA OBSTRUCTION AFTER CONGENITAL DIAPHRAGMATIC HERNIA REPAIR: A BRIDGE TO LIVER TRANSPLANTATION

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Introduction: Acquired suprahepatic inferior vena cava (IVC) obstruction following congenital diaphragmatic hernia (CDH) repair is an unusual cause of chronic hepatic venous outflow obstruction and may substantially increase the technical complexity of liver transplantation. We describe staged cavoatrial reconstruction to establish a favorable venous outflow pathway before transplantation in a pediatric patient with cirrhosis and complex caval anatomy. **Case Presentation:** A 16-year-old male with right-sided CDH repaired neonatally with a Gore-Tex patch and congenital heart

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disease developed progressive liver disease. His neonatal course was complicated by pleural and pericardial effusions and refractory ascites requiring a pericardial window. During atrial septal defect repair in infancy, the IVC was noted to be absent at its expected diaphragmatic entry into the right atrium, consistent with cavoatrial obstruction. He subsequently developed chronic hepatic venous outflow obstruction, portal hypertension, progressive fibrosis, and cirrhosis.Magnetic resonance angiography demonstrated complete suprahepatic IVC occlusion between the hepatic vein confluence and right atrium with extensive azygos and hemiazygos collateralization (Fig 1A), creating a significant challenge for hepatic venous outflow reconstruction. Following multidisciplinary evaluation, a staged surgical approach was undertaken. Through sternotomy and laparotomy, a 20-mm Gore-Tex graft was placed from the infrahepatic IVC above the renal veins to the right atrium, establishing a cavoatrial conduit for subsequent transplantation (Fig 1B). Completion angiography demonstrated unobstructed flow. Postoperative non-occlusive graft thrombosis was successfully treated with catheter-directed thrombectomy.The patient subsequently underwent successful orthotopic liver transplantation with a size-matched whole liver using bicaval technique. The recipient IVC was replaced, with the suprahepatic anastomosis incorporating the immediately subdiaphragmatic portion of the cavoatrial graft. **Discussion:** Staged cavoatrial reconstruction transformed complex venous anatomy into a more favorable configuration for liver transplantation. Staging permitted recovery and assessment of conduit patency while reducing the technical complexity of the definitive operation. Incorporation of the graft into the bicaval reconstruction provided a practical hepatic venous outflow strategy in the setting of suprahepatic caval obstruction. **Conclusion:** In selected patients with complex suprahepatic caval obstruction, staged cavoatrial reconstruction can facilitate liver transplantation by establishing a favorable pathway for hepatic venous outflow. This case highlights the importance of multidisciplinary planning and individualized vascular reconstruction in patients with challenging venous anatomy.

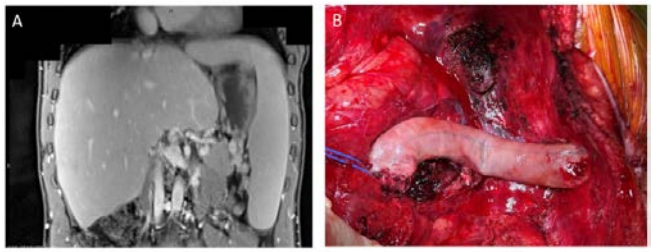


Figure 1: (A) MR angiography showing complete suprahepatic IVC occlusion with extensive venous collateralization. **(B)** Intraoperative view after native hepatectomy at liver transplantation showing the 20-mm Gore-Tex graft connecting the infrahepatic IVC to the right atrium. IVC, inferior vena cava; MR, magnetic resonance.

3613 | IMMUNOSUPPRESSION FREE LIVING DONOR LIVER TRANSPLANTATION BETWEEN MONOZYGOTIC TWINS FOR ALCOHOL ASSOCIATED LIVER DISEASE

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Introduction: Living donor liver transplantation (LDLT) between monozygotic twins is rare, with fewer than ten cases reported worldwide. Their genetic identity offers a rare opportunity to avoid lifelong immunosuppression, a benefit long established in twin kidney transplantation but not yet replicated in the liver, where most prior twin LDLT recipients still received at least one perioperative steroid dose. This report describes a monozygotic twin LDLT in which the recipient received no perioperative steroid or induction agent and remains immunosuppression-free. **Case Presentation:** A 45-year-old female with alcohol-associated cirrhosis (MELD-3.0 score of 26) presented for liver transplant evaluation. Her monozygotic twin sister was evaluated as donor. Donor evaluation showed a right lobe graft of 780 mL (52.2% of standard liver volume; graft recipient weight ratio 0.99), 7.2% steatosis, a single hepatic artery, and a type I portal vein. Zygosity was evaluated by Short Tandem Repeat (STR) profiling across 16 loci (Promega Wizard) demonstrating all alleles were identical (Figure 1A), indicating greater than 99 percent probability of monozygosity. HLA typing showed complete Class I and Class II concordance with no donor specific antibody (Figure 1B), the immunologic basis for pursuing an immunosuppression-free course. The patient underwent right lobe LDLT without steroid or induction agent. Explant showed stage 4 cirrhosis without malignancy. AST/ALT declined from 781 and 512 U/L (normal <35 and <45 U/L, respectively) to 36 and 25 U/L, respectively, with total bilirubin declining from 10.9 to 0.8 mg/dL (normal <1.2 mg/dL). The postoperative course was uncomplicated with no clinical or biochemical evidence of rejection. Graft function, six weeks after LDLT, at the time of submission, remains excellent with normal liver chemistries. **Discussion:** Monozygotic twin LDLT leverages complete HLA identity to avoid lifelong immunosuppression, whose cumulative burden includes nephrotoxicity, hypertension, diabetes, dyslipidemia, cardiovascular mortality, and malignancy. The liver is also uniquely immunotolerant

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among solid organs, promoting T cell anergy and clonal deletion, which explains the comparatively frequent spontaneous tolerance seen even in non-twin recipients. This biologic plausibility, combined with a fully immunosuppression-free course from the outset, distinguishes this case from prior twin reports, which relied on at least one perioperative steroid dose. **Conclusion:** Molecularly confirmed monozygotic twin LDLT can be performed without perioperative steroids or induction immunosuppression, establishing complete HLA identity as sufficient grounds to forgo immunosuppression entirely in carefully selected donor-recipient pairs.

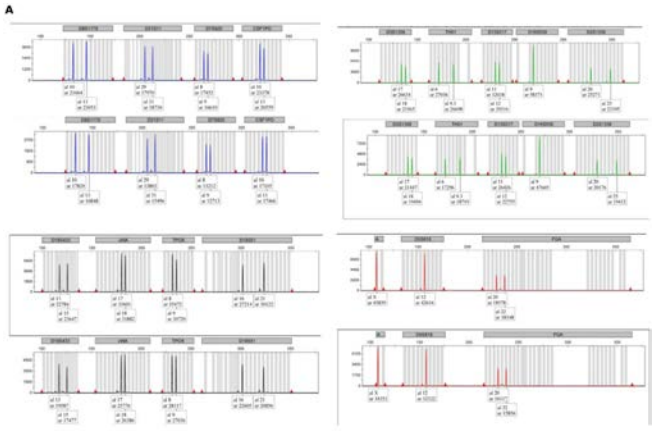


Figure 1. **(A)** Short tandem repeat (STR) electropherograms across the 16 loci profiled (D8S1179, D21S11, D7S820, CSF1PO, D3S1358, TH01, D13S317, D16S539, D2S1338, D19S433, vWA, TPOX, D18S51, D5S818, FGA, and amelogenin); each labeled panel shows the donor (upper trace) and recipient (lower trace), with peak position along the x-axis corresponding to allele fragment size. Identical peak positions between donor and recipient at every locus indicate shared alleles. **(B)** HLA and MHC typing table listing recipient and donor allele calls across classical HLA Class I (A, B, C), classical Class II (DPA1, DPB1, DQA1, DQB1, DRB1, DRB3, DRB4, DRB5), non-classical (E, F, G, H), and MHC class I chain-related (MICA, MICB) loci. Identical allele calls at every locus constitute complete donor-recipient concordance.

3614 | SHORT GUT TO FAST FIBROSIS: ACCELERATED RECURRENT ALLOGRAFT MASH IN ABSENCE OF METABOLIC RISK FACTORS

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Introduction: We present a case of rapidly progressive biopsy-proven recurrent allograft MASH cirrhosis following OLT in a middle-aged female with TPN-dependent intestinal failure due to short gut syndrome, culminating in definitive management with multivisceral transplantation. **Case Presentation:** Severe steatosis with fibrosis was present as early as six months post-transplant, progressing to bridging fibrosis within fourteen months, and biopsy-proven early cirrhosis by four years post-transplant. With decompensation as ascites and esophageal varices, cirrhosis was again confirmed on biopsy fourteen years post-transplant (Table 1). Notably, our patient lacked traditional metabolic risk factors, with hemoglobin A1c never exceeding 5.6%, normal lipid indices, and a peak post-transplant BMI of only 23.9 with declining trajectory. There was no evidence of ACR or chronic rejection, hepatitis B and C serologies were negative, and negative PEths confirmed no recurrent alcohol use. She underwent DBD transplant of stomach, small bowel, pancreas, and liver with thyroglobulin/rituximab induction from HCV positive donor, completing Epclusa treatment with achieving SVR. Immediate postoperative course was complicated by anastomotic leak, but four years post MVT, she remains with functioning liver and intestinal allografts, enteral autonomy, and no primary disease recurrence, steatohepatitis, rejection, or portal hypertensive complications. **Discussion:** Fibrosis present by six months progressing to bridging fibrosis by fourteen months and cirrhosis by four years exceeds the typical timeline of recurrent post-transplant MASH. Moreover, this unusually aggressive progression occurred in a patient without traditional metabolic risk factors but rather unique intestinal-failure physiology and TPN dependence. Known IFALD-implicated mechanisms such as intestinal dysbiosis, bacterial translocation, altered enterohepatic signaling, and parenteral nutrition-associated steatotic liver injury may have accelerated fibrogenesis. **Conclusion:** Chronic intestinal failure, short gut syndrome, and prolonged TPN dependence may represent underrecognized contributors to accelerated allograft steatohepatitis and fibrosis.

Table 1. Rapid Histologic Progression of Fibrosis

Time post OLT	Liver Biopsy Key Findings
6 months	Severe steatosis (>66%) with fibrosis present
14 months	Bridging fibrosis (Stage 3)
4 years	Severe steatohepatitis, NAS 5, Stage 3-4 fibrosis/early cirrhosis
14 years	Mild steatohepatitis, NAS 3, Stage 4 fibrosis (cirrhosis)

3615 | BEYOND PNPLA3: NOVEL PTPN6 AND SH3RF2 VARIANTS IN A CASE OF MASH WITH DISPROPORTIONATE FIBROSIS

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Introduction: In a subset of patients with metabolic dysfunction-associated steatohepatitis (MASH), fibrosis progression occurs rapidly in comparison to the majority that progress over decades. When fibrosis is

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disproportionate to a patient duration in disease state and cardiometabolic risk factors, additional risk modifiers should be suspected. Genetic variants such as *PNPLA3* Ile148Met (C>G) are established modifiers, but rarer variants likely also contribute to fibrosis risk in MASH. **Case Presentation:** A 41-year-old woman presented with ALT 251 U/L, AST 381 U/L, and alkaline phosphatase 67 U/L, with hepatic steatosis on imaging. Liver biopsy confirmed moderately active MASH with extensive centrilobular sinusoidal fibrosis and early bridging fibrosis (stage 2-3 of 4, NASH CRN). Workup for alternate liver disease etiologies was negative, and she reported no alcohol use. This degree of fibrosis was felt to be out of proportion to her cardiometabolic risk factors - stage 1 obesity (BMI 28, of Asian descent) and a history of gestational diabetes. Therefore, she was offered whole exome genetic testing through the Mayo Clinic Tapestry DNA Sequencing Research Study. **Discussion:** The patient was identified to be a carrier of the *PNPLA3* Ile148Met (C>G) variant, which is an established intermediate-magnitude fibrosis risk modifier. Two additional predicted-damaging variants that were not previously known to associate with MASH were identified: *PTPN6* c.1264del and *SH3RF2* c.45C>A. Both of these genes have been shown in pre-clinical models to modify the diet-induced MASH phenotype. However, there are no current therapies to target these variants. The patient achieved a 20% total body weight loss via diet and physical activity changes over the next 1 year, which led to normalization of liver enzymes and improved liver stiffness on MR elastography (from 3.1 kPa to 2.0 kPa). **Conclusion:** Routine genetic testing is not currently recommended in MASH but should be considered when fibrosis severity is disproportionate to cardiometabolic risk factors and time in disease state. This case highlights two candidate rare variants in *PTPN6* and *SH3RF2* that may warrant further study as fibrosis modifiers. Despite not having targeted therapies against these variants, lifestyle intervention achieved meaningful biochemical and liver stiffness improvement in this patient highlighting the fundamental importance of enviroment and host interactions in MASH progression.

3616 | BEYOND THE RETINA: CYP4V2-ASSOCIATED CRYSTALLINE HEPATOPATHY IN BIETTI CRYSTALLINE DYSTROPHY

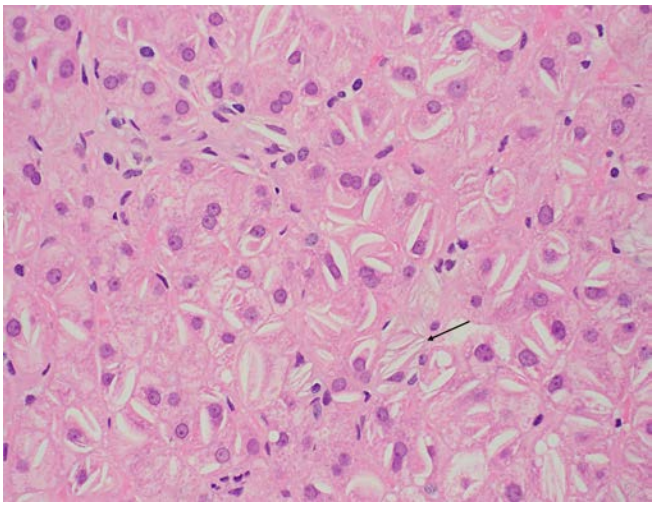
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Introduction: Bietti crystalline dystrophy (BCD) is a rare autosomal recessive retinal disorder caused by biallelic *CYP4V2* variants and traditionally considered ocular. Recent reports identify crystalline hepatopathy as an extraocular manifestation.[1,2] CYP4V2 is expressed in the liver and participates in fatty-acid omega-hydroxylation.[2,3] **Case Presentation:** A 54-year-old man with BCD and homozygous *CYP4V2* c.802-8_810del17insGC was evaluated for progressive aminotransferase elevation. AST/ALT increased from 35/55 U/L in April 2025 to 127/288 U/L by May 2026, while alkaline phosphatase remained 99-126 U/L, bilirubin stayed normal, and hepatic synthetic function was preserved. Viral hepatitis studies were negative. Ultrasound showed hepatomegaly and steatosis. FibroScan suggested F4 fibrosis, and MRE showed liver stiffness of 5.4 kPa, compatible with advanced fibrosis.[9] Liver biopsy demonstrated diffuse intracellular and extracellular needle-shaped crystalline clefts within hepatocytes and portal stroma, with histiocytic/giant-cell reaction and portal fibrosis. There was no bridging fibrosis, cirrhosis, significant steatosis, or cholestasis. **Discussion:** The biopsy findings closely resemble crystalline hepatopathy first described in three patients with BCD.[1] The crystalline material is thought to represent lipid-related deposits lost during processing due to abnormal fatty-acid metabolism in CYP4V2 deficiency.[1,5] Chronic deposition may provoke macrophage recruitment, giant-cell reaction, and fibrosis. The c.802-8_810del17insGC variant disrupts normal splicing near exon 7, but whether this genotype increases hepatic risk is unknown.[6-8] The marked discordance between elastography and biopsy suggests that crystalline deposition and tissue reaction may increase liver stiffness independently of fibrosis. Infiltrative processes and inflammation are recognized confounders of liver-stiffness measurements,[6,7] although this has not been systematically studied in BCD. **Conclusion:** BCD may have clinically relevant hepatic manifestations. In patients with BCD and unexplained liver-test abnormalities, crystalline hepatopathy should be considered. Discordant elastography and histology should be interpreted cautiously. Longitudinal follow-up is needed to define natural history, genotype associations, and management of hepatic CYP4V2 de-

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ficiency.

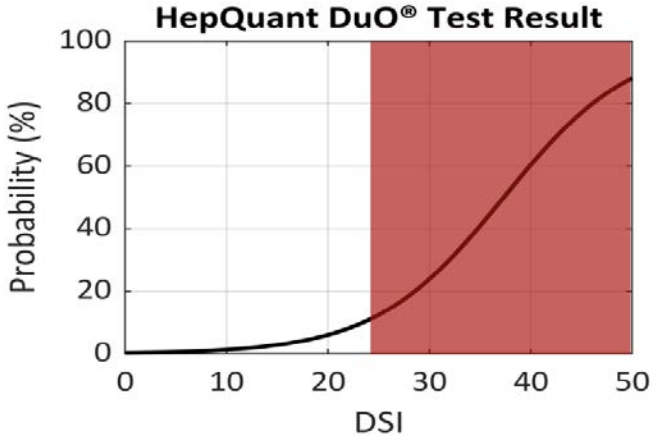


3617 | UTILIZING FUNCTIONAL HEPATIC ASSESSMENT (HEPQUANT DUO) TO STRATIFY VARICEAL RISK IN A COMPLEX PATIENT WITH HCV, ALCOHOL ABUSE, AND OBESITY

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Introduction: Accurate staging of compensated advanced chronic liver disease (cACLD) is critical to determine the risk of clinically significant portal hypertension (CSPH) and the necessity of screening esophagogastroduodenoscopy (EGD). The standard Baveno VII criteria rely on transient elastography (TE) and platelet counts to safely spare endoscopies. However, severe obesity frequently limits the technical acquisition of reliable liver stiffness measurements (LSM), creating a critical diagnostic gap. **Case Presentation:** A 24-year-old male diagnosed with chronic hepatitis C and obesity (BMI 43.8) recently started on antiviral therapy. FibroScan was technically limited due to body habitus, and no result was obtained. FIB-4, ELF score, and platelet count suggested significant fibrosis. Medications: Zoloft and Risperdal.Focused physical examination: BP 108/73 HR 76 Weight 280 lbsSkin: no jaundiceAbdomen: obese with no ascites and no organomegalyExtremities: no edema, spider angioma/palmar erythema Neuro: alert, oriented without asterixisSocial History: remote history of IVDU, cocaine use, and prison-related tattoos; significant history of alcohol abuse, including 4-6 beers daily (ages 14-17) and heavy liquor intake of 700-1500 ml daily starting at age 18. He now reports sobriety since diagnosis of liver disease Labs: Total bilirubin 0.9, serum albumin 4.1, INR 1.1AST 190, ALT 269; Alk Phos 107Platelet count 96,000. HCV RNA 7,490,000ELF 11.71, FIB-4 7.92.Imaging:Abd U/S from 10/22/26 noted hepatomegaly; normal spleen size; no ascites; AFP and MRI pendingThe patient started on antiviral therapy 8/22/2026. Since inability to assess the need for EGD with Baveno criteria, the provider ordered a HepQuant DuO test: for the EGD decision and assessment of the liver disease severity:DSI 30 (normal <11.6)Risk for Large Esophageal Varices: 24%2SHUNT%: 46% (normal 23.7%) Hepatic Reserve: 52% (normal >97%) **Discussion:** The HepQuant DuO test, along with the patient history and the other laboratory data, confirmed the need for an EGD. Antiviral therapy was started on 8/22/2026 and frequent follow up was initiated. Repeat DSI 3-6 months to assess functional improvement.Patient likely has MetALD; reinforced benefit from continued sobriety as well as weight loss. **Conclusion:** Teaching points: - Standard "liver function tests" change late in the disease process and can underestimate the degree of liver injury - The DSI of the HepQuant DuO test demonstrates stepwise increases in liver disease severity from normal to cirrhosis with LEV even when "LFTs" are WNL. - The HepQuant DuO test can assess need for EGD and severity of liver disease even in obesity. - Baseline DSI and serial DSI can predict risk for liver related events and change in this risk over time.



3618 | THE H63D PARADOX: SEVERE HEPATIC IRON OVERLOAD IN A HETEROZYGOUS PATIENT

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Introduction: Hereditary hemochromatosis (HH) is an autosomal recessive disorder of iron overload, most strongly associated with HFE C282Y homozygosity. In

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contrast, isolated H63D heterozygosity has low penetrance and is rarely associated with clinically significant iron overload [2,3]. We report severe biopsy-confirmed hepatic iron overload in an H63D heterozygote, highlighting an unusual genotype-phenotype mismatch. **Case Presentation:** A 61-year-old asymptomatic woman was referred for chronic elevated liver enzymes (AST 139 U/L, ALT 232 U/L, ALP 152 U/L). She denied alcohol use and had a BMI of 21.94 kg/m². The physical examination was unremarkable. Ferritin was 2,100 ng/mL, serum iron 84 µg/dL, TIBC 238 µg/dL, and transferrin saturation 35%. Viral hepatitis testing and evaluation for other chronic liver diseases were unrevealing. She had received 4 units of blood transfusions for acute gynecological bleeding several years earlier. HFE testing detected heterozygous H63D; C282Y and S65C were absent. Liver biopsy demonstrated severe hepatocellular iron deposition with additional Kupffer-cell hemosiderosis. Hepatic iron concentration was 11,748 µg/g dry weight (reference 200-1,800) with a hepatic iron index (HII) of 3.5. Therapeutic phlebotomy was initiated every two weeks, with ferritin decreasing to approximately 900 ng/mL and improvement in liver enzymes. **Discussion:** Clinically significant iron overload is exceptionally uncommon in H63D heterozygotes. In a prospective 12-year population study, no documented iron overload occurred among H63D heterozygotes [4]. Rare clinically significant iron overload has been reported in isolated H63D heterozygotes, including MRI-confirmed hepatic and pancreatic iron deposition [1]. ACG guidance does not consider isolated H63D to confer increased risk of iron overload and recommends evaluating alternative causes in patients without C282Y homozygosity [2]. Despite a transferrin saturation of only 35%, our patient had unequivocal tissue iron overload, demonstrated by severe hepatocellular iron deposition, markedly elevated hepatic iron concentration, and an HII of 3.5. The severity was unexplained by secondary causes of iron overload. Because testing non-HFE hemochromatosis genes was not performed; an additional genetic contributor cannot be excluded. **Conclusion:** This case demonstrates severe hepatic iron overload despite isolated H63D heterozygosity and highlights the importance of evaluating phenotype independently of HFE genotype, particularly when other causes of iron overload have been excluded.

3619 | A NOVEL FOUNDER MUTATION IN HEMOJUVELIN IDENTIFIED IN AN INDIGENOUS NORTHERN ALBERTAN POPULATION LEADING TO TYPE 2A JUVENILE HEMOCHROMATOSIS

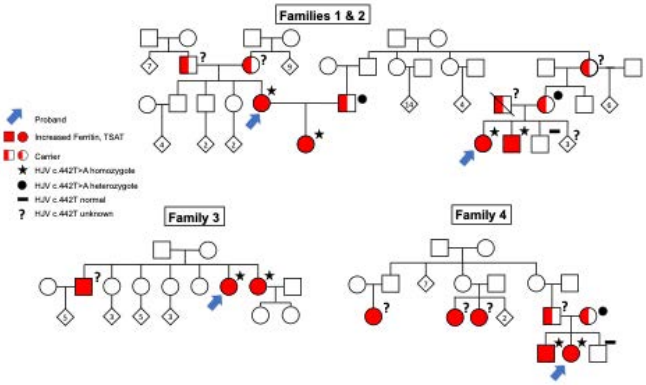
Scott A. MacKay[1] Jeffery M. Patterson[1] Alison Eaton[1] Sherryl Taylor[1] Anna Serebrin[1] Juan G. Abraldes[1] Malcolm Wells[1]

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Introduction: Juvenile hemochromatosis most commonly occurs due to mutations in the hemojuvelin (HJV) gene. Rare HJV mutations have been previously reported with varying pathogenesis ranging from asymptomatic disease to cirrhosis, cardiomyopathy, and hypogonadism. A novel mutation in HJV, c.442T>A, was first identified by our team in 2022 in patients in a remote Northern Albertan Cree community. Patients have since been followed in a joint clinical genetics, hematology and hepatology clinic to provide comprehensive care. **Case Presentation:** This prospective cohort study aims to collect serial ferritin and transferrin saturation (TSAT) levels, FibroScan results, abdominal ultrasounds, and additional relevant imaging (e.g., cardiac and abdominal MRIs) of all individuals identified to be HJV c.442T>A homozygous. Genetic analysis and interpretation included single nucleotide variant analysis by Sanger sequencing of exons 2-4 of the HJV gene and assessment of pathogenicity per ACMG criteria. To date, four families with 9 confirmed HJV c.442T>A homozygous and 3 confirmed heterozygous individuals have been identified in this community. An additional 3 first-degree relatives of affected individuals have self-identified to our study without laboratory or genetic abnormalities detected. An autosomal recessive pattern of inheritance has been identified and the presence of disease within geographically-linked non-kindred patients suggests a founder mutation. HJV c.442T>A homozygous individuals experience elevated ferritin and TSAT levels, hepatic iron deposition and have developed decompensated cirrhosis and HCC. Heterozygous individuals show no laboratory or clinical abnormalities. **Discussion:** There is significant phenotypic variability in the small population of patients with JH secondary to HJV c.442T>A homozygosity that we have identified. The most severe manifestations we have observed include the development of decompensated cirrhosis complicated by HCC and iron overload which was non-responsive to phlebotomy. Conversely, the majority of patients (57.1%) had uncomplicated disease which responded to phlebotomy. Disease within geographically-linked non-kindred patients suggests a founder mutation of the c.442T>A variant. We continue cascade

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genetic testing by identifying, informing, and testing at-risk family members. In addition, given the seemingly high prevalence, a potential founder effect, and a high penetrance of the HJV c.442T>A variant, we advocate for population-based screening within the northern Alberta Cree community. **Conclusion:** The c.442T>A variant in HJV leads to varying levels of disease. The high likelihood this is a founder variant necessitates an ongoing effort for both advocacy and increased healthcare accessibility for this Cree community to identify and treat further cases of juvenile hemochromatosis.



3620 | THE SPECTRUM OF PRESENTATION IN USP53-ASSOCIATED PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS TYPE 7: TWO CASE COMPARISON

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Introduction: Progressive familial intrahepatic cholestasis (PFIC) syndromes are a heterogenous group of autosomal recessive genetic disorders of abnormal bile secretion and/or production. USP53-associated PFIC 7 is recently described, with fewer than 40 cases reported worldwide and variable disease presentation and progression. We present 2 unrelated cases of cholestatic liver disease ultimately diagnosed with PFIC 7. **Case Presentation:** Patient #1 An ex-full-term male presented at 8 months old with 4 months of persistent scleral icterus, jaundice, fussiness, pruritis, and malnutrition. Laboratory evaluation showed total bilirubin 22.1, direct bilirubin 14.9, AST 118, ALT 46, and gamma-GT 22. Liver biopsy demonstrated severe cholestasis with obstructive patterns and advanced fibrosis; ERCP excluded biliary atresia. Next-generation sequencing revealed a bi-allelic variant in USP53. Treatment with ursodiol and rifampin resolved pruritus, with subsequent resolution of cholestasis and malnutrition and improved fibrosis scoring on elastography. Patient #2 A 16-year-old female presented with acute jaundice and severe pruritis. She had been hospitalized at 3 months of age in Syria for infection and bile obstruction responsive to steroid therapy, followed by normal growth and no recurrent jaundice until presentation. Laboratory evaluation showed total bilirubin 6.6, direct bilirubin 5.1, AST 37, ALT 47, and gamma-GT 25. Liver biopsy showed intracytoplasmic and intracanalicular cholestasis without significant fibrosis. Next-generation sequencing revealed bi-allelic USP53 mutations. Ursodiol and trials of hydroxyzine, cholestyramine, naltrexone, and rifampin provided limited efficacy for pruritis. After genetic diagnosis, she transitioned to an ileal bile acid (IBAT) inhibitor with resolution of cholestasis and pruritis on monotherapy. **Discussion:** As demonstrated through our distinct patient cases, presentation of PFIC 7 is variable, and diagnosis should be entertained at any age. The usual clinical course follows neonatal jaundice with intermittent relapsing episodes. Prior identified cases have described age at diagnosis ranging from 4-87 months. Mangement targets cholestasis and pruritus, which historically has been accomplished by ursodiol, cholestyramine, and rifampin. However, IBAT inhibitors now FDA approved for cholestatic pruritus in PFIC have demonstrated excellent clinical response and should be considered in PFIC 7 cases. **Conclusion:** Neonatal jaundice is likely shared between our patients; however, the diagnosis was delayed in Patient #2 due to unavailability of next generation sequencing. Our case comparison highlights the value of early genetic testing in any patient with normal gamma-GT cholestasis, as disease presentation and progression vary significantly, and delayed recognition may postpone effective treatment.

3621 | THE LIVER KNEW FIRST: ACUTE HEPATITIS AS THE PRESENTING AND PERSISTING MANIFESTATION OF STILL'S DISEASE ASSOCIATED MACROPHAGE ACTIVATION SYNDROME IN AN ADOLESCENT

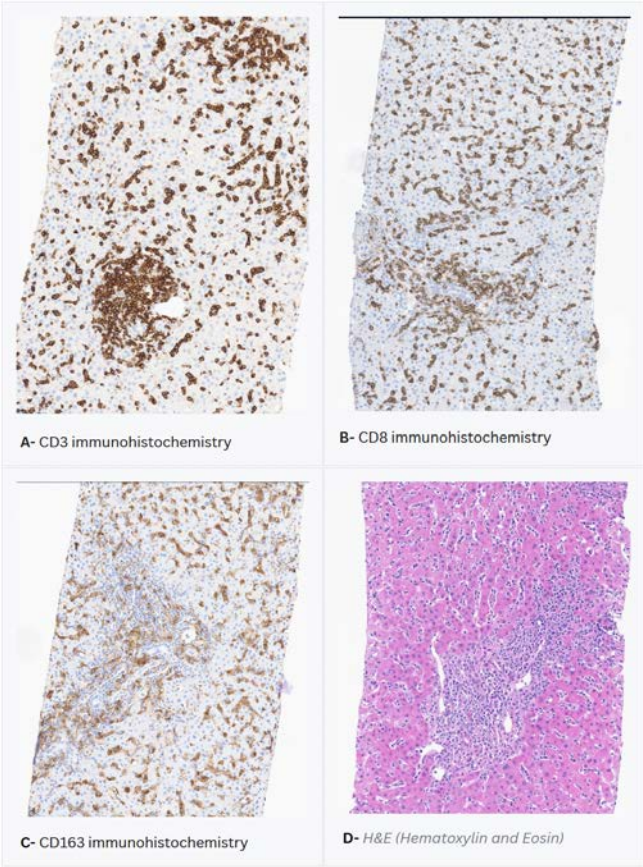
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Introduction: Hemophagocytic lymphohistiocytosis/macrophage activation syndrome

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(HLH/MAS) can present with hepatitis, preceding overt cytopenias. We present an adolescent in whom overlapping clinical and laboratory features initially favored infectious or autoimmune hepatitis (AIH), before the hepatitis was attributed to Still's disease associated MAS (SD-MAS) triggered by acute Epstein-Barr virus infection (EBV). **Case Presentation:** A previously healthy 17-year-old male presented with five days of fever, headache, myalgias, and pruritic rash. Labs showed cholestatic hepatitis with mild coagulopathy (ALT 296 U/L, AST 207 U/L, total bilirubin 2.5 mg/dL, direct bilirubin 1.8 mg/dL, INR 1.5) without hepatic encephalopathy, leukocytosis (12.5 x 10^3/µL), thrombocytopenia (92 x 10^3/µL), CRP 12.5 mg/L, and markedly elevated ferritin (20,829 ng/mL). Ultrasound showed hepatosplenomegaly. Workup showed positive EBV PCR/serologies and positive CMV IgM (IgG negative). AIH workup was unremarkable (ANA 1:80, anti-smooth muscle 1:80, negative anti-LKM1, normal IgG, simplified AIH score 2). With fever, hyperferritinemia, and splenomegaly, HLH was considered but less favored (met only 3/8 HLH-2004 criteria). After defervescence, he was discharged with close follow-up.He was readmitted within 48 hours with recurrent fever, worsening cytopenias, and elevated triglycerides. Rheumatology established SD-MAS, as he had met SJIA/SD-MAS 2016 criteria on initial presentation, supported by markedly elevated CXCL9 (39,887 pg/mL), IL-18 (167,109 pg/mL), and sCD25 (10,237 U/mL) despite improving ferritin and liver enzymes. Twice-daily anakinra partially improved systemic inflammation (fever, ferritin, thrombocytopenia); however, weaning to daily dosing led to liver-localized inflammation, producing a liver-confined MAS. Liver biopsy showed acute hepatitis with spotty necrosis and a sinusoidal-predominant infiltrate of CD3+CD8+ lymphocytes and CD163+ macrophages, with portal lymphocytes admixed with macrophages and no significant plasma cells; electron microscopy showed occasional macrophages with intracytoplasmic lymphocytes, and EBV/CMV were negative. **Discussion:** This atypical hepatitis-predominant SD-MAS was likely EBV-triggered. Absent arthritis and evanescent rash initially favored infectious/autoimmune etiologies; SD-MAS emerged with recurrent fever, hyperferritinemia disproportionate to hepatitis, hepatosplenomegaly, and an IFN-γ biomarker profile. Presentation without arthritis is not uncommon and should not deter diagnosis; elevated IL-18 is a valuable clue. Biopsy CD3+CD8+ T cells admixed with CD163+ macrophages engulfing lymphocytes is the MAS/HLH signature. **Conclusion:** MAS can present as isolated hepatitis mimicking infectious or autoimmune hepatitis. Clinicians should have a low threshold to send IFN-γ pathway markers when hepatitis persists or recurs despite improving ferritin.



3622 | AN UNUSUAL CAUSE OF KIDNEY DYSFUNCTION IN A PATIENT WITH PBC: HEPATORENAL SYNDROME IS NOT ALWAYS THE ANSWER

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Introduction: Acute kidney injury (AKI) and chronic kidney disease (CKD) are common in cirrhotic patients. The most common causes are multifactorial pre-renal injury and hepatorenal syndrome (HRS). Approximately 1% of kidney injury in CLD is caused by glomerulonephritis (GN), most commonly membranoproliferative GN (MPGN) or IgA nephropathy. We present an example of such with this case of a patient with primary biliary cirrhosis (PBC), who was found to have worsening CKD caused by MPGN secondary to cryoglobulinaemia (CG). **Case Presentation:** A 64-year-old woman with a history of PBC was noted to have an asymptomatic AKI at routine follow up, with creatinine rising by 60µmol/L over a nine month period. Examination revealed hypertension, peripheral oedema and blanching papules on the hands. Serial LFTs, coagulation studies, and CBCs were stable. Liver US showed cirrhosis with trace ascites. HBV, HCV and

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HIV screening were negative. CG testing was found to be positive for polyclonal IgM alone. C3 and C4 values were below the reference range. The patient was seen by Renal Medicine nine months following presentation. ANA, dsDNA, EPP, free light chains and Anti-GBM testing were negative. Renal biopsy was performed and favoured an immune complex mediated MPGN. Given the clinical picture, the diagnosis was secondary mixed (type III) cryoglobulinaemic MPGN, on a background of PBC, with HCV negative. As the patient was largely asymptomatic with stable renal function and no significant proteinuria, treatment with immunosuppression was withheld. **Discussion:** CG causes a small and medium vessel vasculitis, with a clinical syndrome involving joint pain, skin rashes and kidney dysfunction. Heterogenous clinical findings and rate of progression increase diagnostic challenges in this condition. Increased CG IgM can be found in the majority of PBC patients. CG-related MPGN remains uncommon, though there is an increased risk in PBC patients compared to the general population. Patients with CKD and a known diagnosis of PBC should initially be screened for drug related ATN and HRS. In cases such as this, where no offending nephrotoxic can be found, and clinical picture is inconsistent with HRS, GN should be considered. Extended GN and vasculitis screens should be performed to exclude alternative causes of renal dysfunction in cirrhotic patients, including CG-related MPGN and IgA nephropathy. Early referral to nephrology may facilitate timely diagnosis and initiation of proper management. **Conclusion:** This case serves to emphasise the importance of early consideration of alternative diagnoses to pre-renal or HRS AKI in cirrhotic patients who are presenting atypically. As AKI caused by CG-related MPGN is readily reversible with immunosuppression, early identification and referral may vastly improve outcomes in cirrhotic patients with renal dysfunction.

3623 | TRUST THE DISCORDANCE: ASCITES WITHOUT CIRRHOSIS REVEALING EXTRAHEPATIC MALIGNANCY

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Introduction: Ascites and portal hypertensive physiology typically prompt evaluation for advanced liver disease. However, discordance between clinical findings and non-invasive fibrosis testing should prompt further investigation. We present a patient with MASLD, ascites, discordant fibrosis assessments, and a pancreatic lesion, ultimately diagnosed with metastatic high-grade serous Müllerian carcinoma. **Case Presentation:** A 66-year-old female with MASLD, morbid obesity (BMI 58), and remote hysterectomy with bilateral salpingo-oophorectomy (BSO) for endometrial intraepithelial neoplasia underwent preoperative evaluation for bariatric surgery for elevated liver enzymes. FIB-4 was 0.97 (AST 34, ALT 44, Platelets 347). Ultrasound elastography showed stiffness of 12.3 kPa suggesting advanced fibrosis. MR elastography (MRE) showed severe steatosis (15%) without advanced fibrosis (3.8 kPa), but revealed ascites and a 4.3-cm multicystic pancreatic head lesion with normal spleen size. Due to ascites despite normal fibrosis assessment, she underwent liver biopsy which showed steatohepatitis without fibrosis. Transjugular pressure measurements demonstrated no trans-sinusoidal gradient. Her ascites was not amenable to a diagnostic paracentesis, so it was not sent for studies or cytology. EUS-FNA of the pancreatic lesion was consistent with IPMN with high-grade dysplasia. CT and PET demonstrated omental and abdominal wall nodules concerning for metastatic disease, but the pancreatic lesion was not FDG-avid. Biopsy of an umbilical wall mass revealed metastatic high-grade serous carcinoma of Müllerian origin, establishing stage IV primary peritoneal carcinoma. She completed carboplatin/paclitaxel with ongoing multidisciplinary consideration of cytoreductive surgery and HIPEC. **Discussion:** This case highlights important diagnostic considerations when utilizing non-invasive testing to risk-stratify patients with MASLD. Given BMI>35 along with discordant FIB-4 and ultrasound elastography, MRE was performed which showed ascites without advanced fibrosis providing further evidence of discordance. This prompted consideration of noncirrhotic and extrahepatic causes of ascites, rather than a premature diagnosis of MASH-related cirrhosis with portal hypertensive complications. Malignancy can cause malignant ascites via peritoneal carcinomatosis or ascites via portal hypertension due to vascular obstruction, sinusoidal infiltration, or extrinsic compression. In this case, she likely had malignant ascites with tissue diagnosis establishing metastatic Müllerian carcinoma. Her remote BSO raises the possibility of occult Müllerian precursor disease with peritoneal dissemination during bariatric surgery, although the mechanism is uncertain. **Conclusion:** Ultimately, diagnostic discordance should prompt further evaluation when ascites, portal pressures, liver stiffness, and histology do not align.

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3624 | CYCLOSPORIASIS AS A PRECIPITANT OF HEPATIC ENCEPHALOPATHY IN A PATIENT WITH MASLD CIRRHOSIS

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Introduction: *Cyclospora cayetanensis* is an intestinal protozoan that causes protracted watery diarrhea, particularly in immunocompromised hosts. Though more common in tropical or sub-tropical regions, a recent outbreak has made cyclosporiasis an emerging cause of foodborne diarrheal illness in the United States. However, no reports to date have described cyclosporiasis precipitating hepatic encephalopathy (HE). Thus, we describe a patient with Metabolic dysfunction-Associate Steatotic Liver Disease (MASLD) cirrhosis who presented with his first episode of HE secondary to Cyclospora infection. **Case Presentation:** A 48-year-old man with MASLD cirrhosis, esophageal varices treated with transjugular intrahepatic portosystemic shunt, type 2 diabetes, and depression presented to the emergency department with confusion, nausea, and vomiting. Two weeks prior, the patient began experiencing persistent nausea, non-bloody emesis, and worsening acute-on-chronic diarrhea. The patient subsequently developed confusion 24 hours prior to presentation.On examination, the patient appeared lethargic and jaundiced with scleral icterus. He was disoriented to time and situation and exhibited psychomotor slowing, asterixis, and a positive milkmaid's grip. Abdominal exam was unremarkable without ascites. Laboratory evaluation demonstrated pancytopenia, predominantly indirect hyperbilirubinemia, hypoalbuminemia, severe hypokalemia of 2.7 mmol/L, and non-anion gap metabolic acidosis. Imaging with RUQ US and CTA revealed no acute findings. The patient was admitted and treated for HE (West Haven Criteria Grade II) with lactulose and rifaximin, along with IV fluids and potassium repletion, with subsequent improvement in mental status and resolution of jaundice and asterixis. On day 3 of admission, stool multiplex PCR detected cyclospora and TMP-SMX was initiated. The patient recovered back to his baseline mentation on day 4 and was discharged on day 5 with close follow-up. **Discussion:** Infections are a common precipitant of HE, yet enteric parasitic infections have not been described as a distinct precipitant. In our patient, HE was likely driven by hypokalemia and dehydration which are well-established independent precipitants of HE through multiple converging mechanisms that amplify hyperammonemia at the renal, hepatic, and cerebral levels. Cyclosporiasis invades jejunal enterocytes, replicates intracellularly, and produces villous atrophy, crypt hyperplasia, and a mixed inflammatory infiltrate. Beyond metabolic derangements, systemic inflammation, which has emerged as an important mechanism of HE through the gut-liver-brain axis, may have contributed. **Conclusion:** In patients with decompensated cirrhosis presenting with GI upset or increased epidemiologic risk, clinicians should consider early stool multiplex PCR to identify uncommon pathogens and facilitate targeted therapy, potentially preventing progressive HE.

3625 | HYPONATREMIA REFRACTORY TO LIVER TRANSPLANTATION: A RESET OSMOSTAT AND CIRRHOSIS-ASSOCIATED IGA AFTER TRANSPLANTATION

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Introduction: Hyponatremia in decompensated cirrhosis typically improves after liver transplantation (LT) as effective arterial blood volume and hepatic function are restored. Persistent hyponatremia after LT is unusual and may reflect altered osmoregulation. We present symptomatic persistent hyponatremia after LT attributed to Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) with reset osmostat. **Case Presentation:** A 40-year-old man with alcohol-associated decompensated cirrhosis developed hyponatremia and acute kidney injury requiring transient renal replacement therapy. He underwent deceased-donor LT with excellent graft function. Post-transplant, he developed a pericaval fluid collection requiring drainage; however, his hyponatremia persisted and he was readmitted 31 days later with altered mental status, dysarthria, and visual and auditory hallucinations. Serum sodium was 116 mmol/L, and mentation normalized after correction with hypertonic 3% saline. Evaluation revealed nephrotic-range proteinuria, and kidney biopsy demonstrated IgA nephropathy attributed to antecedent liver disease. Nephrology diagnosed reset osmostat secondary to SIADH, with stable sodium in the mid-120s.Several further admissions for symptomatic hyponatremia followed. Tolvaptan was withheld because of hepatotoxicity concerns. He was managed with 1.2 liters fluid restriction plus oral urea. By three months, graft and kidney function were preserved, proteinuria had fallen below 0.5 g/g, and he was asymptomatic at his new reset baseline. **Discussion:** Persistent hyponatremia after LT should not au-

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tomatically be attributed to residual portal hypertensive physiology. In this patient, restoration of graft function and marked improvement in proteinuria occurred despite persistent hyponatremia, suggesting a distinct disturbance in osmoregulation. The stable sodium set point in the mid-120s supported a reset osmostat. Its persistence after LT may reflect prolonged pre-transplant hyponatremia and chronic illness-associated osmoregulatory adaptation. The divergent improvement in IgA nephropathy and persistent hyponatremia suggests these processes followed independent trajectories after transplantation. Recognition of reset osmostat is clinically important because treatment should focus on preventing symptomatic or severe sodium excursions rather than pursuing normalization of a chronically defended set point. This is particularly relevant in patients at increased risk of osmotic demyelination syndrome, including those with alcohol use, malnutrition, and recent transplantation. Oral urea provided an alternative when conventional therapies were unsuitable. **Conclusion:** Persistent hyponatremia after LT may reflect reset osmostat rather than residual portal hypertensive physiology. Recognizing this entity can guide individualized management and help avoid unnecessary or overly rapid correction.

3626 | CECAL DIEULAFOY LESION IN NEWLY DIAGNOSED ALCOHOLIC CIRRHOSIS AND SUSPECTED HEPATIC MALIGNANCY: A CASE REPORT AND DISCUSSION OF POSSIBLE CIRRHOSIS- AND MALIGNANCY-RELATED ASSOCIATIONS

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Introduction: Dieulafoy lesions cause 1-2% of GI bleeds; cecal lesions are rare, representing ~17% of colonic cases. Cirrhosis has been noted in passing as a contributing factor, but never mechanistically explored. We report a cecal Dieulafoy lesion revealing previously occult alcoholic cirrhosis and suspected hepatocellular carcinoma, and propose two possible vascular links: cirrhosis-associated angiogenesis and tumor-derived VEGF from an underlying malignancy. **Case Presentation:** An 82-year-old man with small-bowel AVMs, diverticulosis, and a Schatzki ring presented with a week of hematochezia culminating in syncope. Hemoglobin was 10.9 g/dL with an ALT-predominant transaminitis. CT angiography for the bleed incidentally revealed undiagnosed cirrhosis with two hepatic masses concerning for malignancy. Hemoglobin nadired at 8.4 g/dL without transfusion. Colonoscopy was technically difficult; in a narrow-angled cecum seen only in retroflexion, a pulsating Dieulafoy lesion was found adjacent to the ileocecal valve. Clip placement failed given the retroflexed position; hemostasis was achieved with epinephrine injection and gold-probe cautery. EGD showed duodenal bulb ulcers without bleeding stigmata. MRI confirmed cirrhosis and the hepatic masses, with AFP >6,000 ng/mL and a pulmonary nodule consistent with metastasis; the patient declined biopsy and further workup, so malignancy type was never confirmed. **Discussion:** This patient's cirrhosis was previously unrecognized. A prior capsule endoscopy had also missed the Dieulafoy lesion. Cirrhosis creates a hyperestrogenic, pro-angiogenic state that explains findings such as spider angiomata and may similarly contribute to Dieulafoy lesion formation. This patient also had an unconfirmed hepatic malignancy. Tumor-derived VEGF, a proliferative hallmark of cancer, may independently contribute to lesion formation as well, and this mechanism would extend to malignancy of any origin. Alcohol use remains a plausible confounder. **Conclusion:** A cecal Dieulafoy lesion unmasked occult alcoholic cirrhosis and suspected hepatocellular carcinoma. If substantiated, either association could support hepatic evaluation or an occult malignancy workup (CT-CAP) in patients with an unexplained Dieulafoy lesion, and may eventually inform anti-VEGF therapy such as bevacizumab; further comparative study is needed.



3627 | A PRESSURE PARADOX: BLEEDING DUODENAL VARIX IN CIRRHOSIS DESPITE LOW PORTAL PRESSURE

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Introduction: Portosystemic collaterals are a well-recognized marker of clinically significant portal hypertension (CSPH), defined as hepatic venous pressure gradient (HVPG) >=10 mmHg. In patients with cirrhosis, esophageal varices are at risk of rupture and bleeding once HVPG reaches 12 mmHg. However, ectopic varices, including splenic and duodenal varices, may develop at lower portal pressures and can rupture despite an HVPG <10 mmHg, resulting in life-threatening bleeds. **Case Presentation:** A 58-year-old male with a history of type 2 diabetes, and alcohol use disorder, presented to a regional campus after he was found unresponsive on the bathroom floor with evidence of hematochezia. Initial laboratory evaluation was notable for a hemoglobin of 7.4, anion gap metabolic acidosis, and mild elevation in transaminases (AST 45 and ALT 43) with bilirubin of 1.55 and INR of 1.29. CT angiography demonstrated active bleeding in the duodenum. Urgent esophagogastroduodenoscopy (EGD) revealed a single clean-based duodenal ulcer and small varices in the middle third of the esophagus without stigmata of recent bleeding. The patient subsequently developed hematemesis, requiring multiple blood product transfusions and endotracheal intubation for airway protection. He was transferred to the network's tertiary hospital for escalation of care and was found to be in hemorrhagic shock on arrival. Emergent repeat EGD identified a protruding lesion in the duodenal sweep with an overlying adherent clot. Gentle irrigation dislodged the clot, resulting in brisk bleeding from a small, ulcerated opening consistent with a bleeding duodenal varix. Hemostasis was achieved with ethanolamine injection and placement of two endoscopic clips. Given the high risk of recurrent bleeding, he underwent emergent transjugular intrahepatic portosystemic shunt (TIPS) placement. The initial measured HVPG was 8 mmHg prior to TIPS creation and decreased to 2 mmHg following the procedure. **Discussion:** Duodenal varices are an uncommon but important cause of upper gastrointestinal bleeding in patients with cirrhosis. Unlike esophageal varices, which typically rupture at HVPG >=12 mmHg, ectopic varices may rupture at lower pressures. Variations in portosystemic collateral anatomy and shunting may partially decompress the portal venous system, potentially contributing to bleeding despite a relatively low HVPG. **Conclusion:** The AASLD recommends reducing HVPG to <12 mmHg or a by >=50% after TIPS creation in patients with esophageal variceal bleed. Although the HVPG targets for ectopic varices are not well characterized, this case highlights that similar principles can be applied when managing ectopic variceal bleeds. Proceduralists should therefore aim for a >=50% reduction in portal pressure during TIPS creation in cases of ectopic variceal hemorrhage when portal pressure measurements fall below traditional thresholds for esophageal variceal rupture.

3628 | PORTAL VEIN RECANALIZATION RESOLVES A CHRONIC CASE OF MINIMAL HEPATIC ENCEPHALOPATHY REFRACTORY TO THERAPEUTIC INTERVENTION

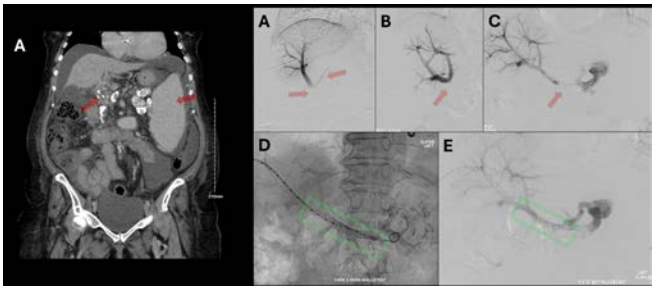
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Introduction: Hepatic encephalopathy (HE) is a spectrum of reversible neurocognitive impairments associated with cirrhosis and can be secondary to portal-systemic shunting and chronic portal vein thrombosis (PVT), resulting in portal hypertension (pHTN). The exact pathogenesis of HE is unclear. Two major contributing factors are systemic inflammation and ammonia accumulation. HE treatment focuses on supportive care and correcting inciting factors, potentially followed by ammonia-reducing medications. Yet, some cases are refractory to these therapeutics. Here we present a case of HE resolved by recanalization of the portal vein (PV). **Case Presentation:** A 74-year-old female with chronic PVT, pHTN, cavernous transformation of the PV, monoclonal gammopathy, and a heterozygous prothrombin mutation presented to an emergency department for a week of generalized weakness, difficulty ambulating, and dizziness. She was treated for acute cholecystitis, during which she developed ascites, undergoing paracentesis. Symptom recurrence and new speech difficulty led to a stroke code with no evidence of cerebrovascular accident. Outpatient hepatology was consulted.Neurocognitive impairment in the context of pHTN and recent ascites raised concern for HE and cirrhosis. Physical exam noted asterixis and abdominal superficial collaterals. She started on lactulose and rifaximin. Labs were consistent with her history; significant thrombocytopenia (16 K/uL) and normal liver function tests. Due to bleeding risk, transjugular liver biopsy was deferred; thus, MRI with elastography showed noncirrhotic, mild liver fibrosis (stiffness score of 3 kPa, Stage I-II fibrosis), with persistent evidence of PVT, with the right and left portal venous vasculature supplied by diminutive collaterals and abdominal varices. Splenorenal shunts and splenomegaly were consistent with prior studies (Figure 1).Mental fogginess continued to interfere with her work; therefore, interventional radiology assessed options. A prior attempt to embolize the splenorenal shunts proved technically challenging. Thus, stent (10 mm) placement within the PV was performed. Pre- and post-stenting CT imaging showed improved patency of left and right

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PV branches (Figure 2). At follow-up, she reports symptom improvement, with no mental fogginess or dizziness, and continues on rifaximin. **Discussion:** Treatment of HE relies on medical intervention, yet refractory cases skip minimally invasive procedures. This case exemplifies a difficult-to-diagnose, refractory-to-treatment covert HE that was effectively treated with PV stenting. Because clinical trials and meta-analyses are lacking, AASLD recommendations for HE treatment have lagged and do not include stenting as an option for these difficult-to-treat cases. **Conclusion:** We hope this case presents a new avenue for alleviating HE symptoms secondary to chronic PVT in the setting of significant thrombocytopenia.

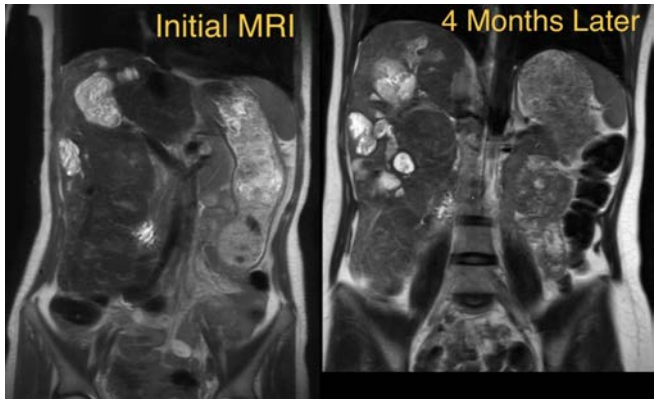


3629 | A RARE INDICATION FOR LIVER TRANSPLANTATION: ISCHEMIC BILIARY NECROSIS AS A COMPLICATION OF HEREDITARY HEMORRHAGIC TELANGIECTASIA

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Introduction: Hereditary hemorrhagic telangiectasia (HHT) is a rare autosomal dominant disorder characterized by mucocutaneous telangiectasia and systemic vascular malformations. Hepatic vascular malformations are present in up to 70% of patients but are symptomatic in only 5-8%. Biliary ischemia leading to biliary necrosis is an uncommon hepatic complication and a recognized, though rare, indication for liver transplantation. We report the case of a 30-year-old woman with HHT complicated by ischemic biliary necrosis who underwent liver transplantation, representing, to our knowledge, the first such case at our institution. **Case Presentation:** A 30-year-old woman presented with a three-month history of right upper quadrant and epigastric pain, nausea, vomiting, anorexia, and weight loss. She had recurrent epistaxis, with a first-degree family history of mucocutaneous telangiectasia, and pulmonary AVM. Examination revealed multiple telangiectasia and tender hepatomegaly. Her liver enzyme were elevated in a cholestatic profile with alkaline phosphatase level in the 200's and MRI demonstrated inflammatory cholangiopathy with multiple biloma and mildly dilated right posterior hepatic lobe bile duct. Her abdominal pain was also thought to have functional component and was prescribed Gabapentinoids, and TCAs. Persistent abdominal pain despite high dose neuromodulators prompted a follow-up MRI imaging which showed arteriovenous and arterioportal shunting, dilated hepatic veins and numerous branching hepatic lesions with bile duct dilatation and multiple new biliomas consistent with ischemic cholangiopathy. Given her significant family history and all four Curaçao criteria met, genetic tesing confirmed an ACVRL1 mutation. Despite low MELD score, the patient underwent orthotropic living-donor liver transplantation with MELD exception. Explant pathology showed vascular malformations, telangiectasias, with associated changes including ischemic cholangiopathy, nodular regenerative hyperplasia, and portosinusoidal vascular disease. Postoperatively, the patient had significant improvement in her abdominal pain and was subsequently tapered off neuromodulators. **Discussion:** High-output cardiac failure is the leading indication for liver transplantation in HHT. Although biliary ischemia is a rare presentation, it is an important transplant indication, and a high index of suspicion is required in HHT patients presenting with abdominal pain. Hence prompt transplant hepatology referral, follow-up imaging, and recognition of progressive ischemic biliary necrosis is paramount in such group of patients. **Conclusion:** Ischemic cholangiopathy with biliary necrosis, though a rare hepatic manifestation of HHT, is an important and potentially life-threatening indication for liver transplantation and should be highly suspected in the setting of persistent abdominal pain and rapidly progressing bilomas.



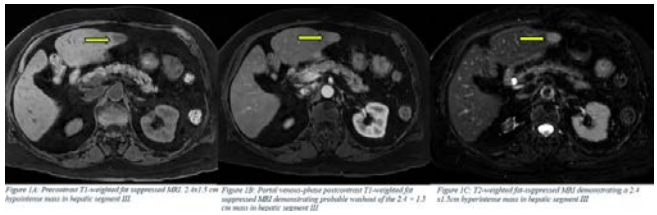
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3630 | A SPLEEN IN DISGUISE: ECTOPIC SPLENIC TISSUE PRESENTING AS A LIVER MASS

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Introduction: Intrahepatic splenosis (IHS) is a rare condition arising from autotransplantation of splenic tissue into the liver parenchyma after splenic trauma or splenectomy, and it is frequently mistaken for hepatocellular carcinoma (HCC) or metastatic disease on imaging. Correctly identifying IHS matters because unnecessary hepatic resection can remove tissue that is providing partial immune protection in a patient who is already asplenic. We describe a case of IHS diagnosed by liver biopsy, presenting nearly two decades after splenectomy. **Case Presentation:** A 69-year-old woman with a history of coronary artery disease, hypertension, cholecystectomy, and Roux-en-Y gastric bypass two decades prior to presentation complicated by iatrogenic splenic injury requiring splenectomy presented with right upper quadrant abdominal pain. Liver function tests were within normal limits. The patient denied nausea, vomiting, unintentional weight loss, or jaundice. Abdominal ultrasound with elastography demonstrated hepatic steatosis and a 1.7cm hypoechoic hepatic lesion without internal vascular flow.Contrast-enhanced MRI demonstrated a 2.4x1.5cm, T1-hypointense, T2-hyperintense rounded mass in hepatic segment III with mild diffusely uniform enhancement, no internal fat, and probable washout on venous phase images, raising concern for HCC versus a benign cystic lesion. Given the indeterminate imaging findings, she was referred for image-guided liver biopsy. Histopathology showed a well-circumscribed lesion with vascular structures and lymphoid follicles resembling splenic red and white pulp. Littoral cells were positive for CD8 and ERG; lymphoid follicles were positive for CD20/PAX5; Ki-67 was low. AE1/AE3, CAM5.2, and arginase-1 were negative within the lesion, excluding carcinoma. These findings confirmed benign intrahepatic splenosis. **Discussion:** This case is notable not only for its atypical demographics, as IHS classically affects younger men (83.1% male, median age 51), but for its etiology: to our knowledge, IHS following iatrogenic splenectomy following bariatric surgery, a complication occurring in 3% of such procedures, has not been previously reported. CT and MRI show only modest accuracy (50-70%) for identifying ectopic splenic tissue which can mimic a hypervascular neoplasm, explaining why biopsy was pursued despite indeterminate imaging. Awareness of this washout pattern in a patient with remote splenectomy could prompt earlier inclusion of IHS in the differential, avoiding biopsy. Non-invasive tools such as Tc-99m scintigraphy may help avoid unnecessary resection and preserve residual immune function in asplenic patients. However, littoral cell positivity for CD8 and ERG was essential in distinguishing this lesion from malignancy. **Conclusion:** IHS should be considered in the differential of a hepatic mass in any patient with prior splenic trauma or splenectomy, regardless of age or time elapsed.



3631 | SEVERE INTRAHEPATIC CHOLESTASIS AND SUBACUTE LIVER FAILURE IN EARLY-STAGE CLASSICAL HODGKIN LYMPHOMA: A PARANEOPLASTIC PRESENTATION

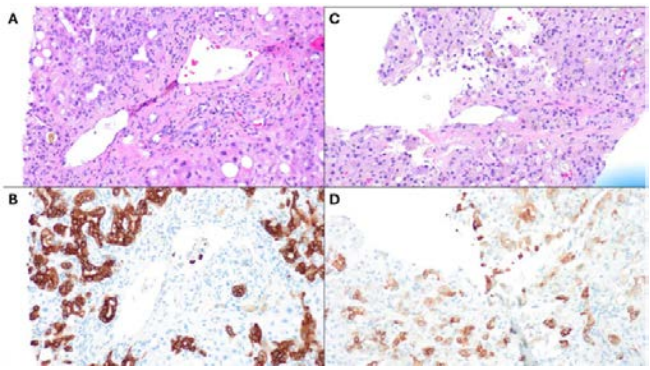
Srija Reddy[1] Pooja Mallipaddi[2] Hunter Hines[2] Matthew Posen[3] Jan Petrasek[2] Eugenia Tsai[2] Joseph Gosnell[2] Fabian Rodas[2] Krishna Venkata[3] Nazeer Ahmed[3] Carmen Landaverde[2] Fred Poordad[2] Lisa Pedicone[3]

[1]University at Texas, San Antonio, [2]UTSA, [3]Texas Liver Institute

Introduction: Paraneoplastic intrahepatic cholestasis and vanishing bile duct syndrome (VBDS) are rare, potentially fatal complications of classical Hodgkin lymphoma (cHL). We report stage IA cHL with progressive hepatic dysfunction from a pre-ductopenic to ductopenic phenotype despite favorable interim lymphoma response. **Case Presentation:** A 26-year-old man with mild ulcerative colitis developed progressive jaundice, marked hyperbilirubinemia, and volume overload during evaluation of severe cholestatic liver disease. Extensive evaluation was unrevealing. ERCP, MRCP, and cross-sectional imaging showed no biliary dilatation, obstruction, or focal hepatic lesion.Initial liver biopsy showed marked bland lobular/canalicular cholestasis, mild mixed portal inflammation, preserved interlobular bile ducts, ductular proliferation, stage 2 perisinusoidal/pericellular fibrosis, and CK7-positive intermediate hepatocytes; cholestasis was disproportionate to hepatocellular injury. Repeat biopsy two months later showed reduced ductular proliferation and progressive loss of interlobular bile ducts, consistent with ductopenic cholangiopathy/VBDS.During transplant evaluation, left cervical lymph node biopsy established stage IA cHL; bone marrow biopsy was negative. He

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received ABVD with 50% dose reduction of doxorubicin and vinblastine because of hyperbilirubinemia. Interim PET/CT showed favorable cervical nodal response (Deauville 3), but liver failure progressed: bilirubin 39.6 mg/dL, INR 2.5, MELD-Na 34, encephalopathy, acute kidney injury, and respiratory failure requiring mechanical ventilation. He was ineligible for transplantation because of active lymphoma and critical multiorgan illness and transitioned to hospice. **Discussion:** This case supports severe intrahepatic cholestasis as a paraneoplastic manifestation of cHL within the Hodgkin-associated VBDS spectrum. Serial histology captured a pre-ductopenic phase with bland cholestasis, preserved ducts, stage 2 fibrosis, and CK7-positive intermediate hepatocytes before bile duct loss. The discordance between limited stage IA cHL and profound hyperbilirubinemia/high MELD is notable. Irreversible hepatic deterioration despite favorable PET response challenges the assumption that hepatic recovery follows lymphoma response. Dose-reduced ABVD constrained definitive therapy. Tumor-directed radiotherapy, brentuximab-based strategies, and anti-PD-1 therapy were not used. **Conclusion:** Consider paraneoplastic cholestasis in cHL with disproportionate cholestatic injury after excluding obstruction, infection, autoimmune disease, drug injury, and hepatic infiltration. Serial biopsy may identify pre-ductopenic disease and progression to VBDS. Favorable lymphoma response does not ensure hepatic recovery; progressive liver failure may preclude curative therapy and transplantation.



3632 | GENETICALLY CONFIRMED CITRULLINEMIA TYPE 1 PRESENTING AS FULMINANT ACUTE LIVER FAILURE AT 20 WEEKS' GESTATION: AN ACUTE FATTY LIVER OF PREGNANCY MIMIC

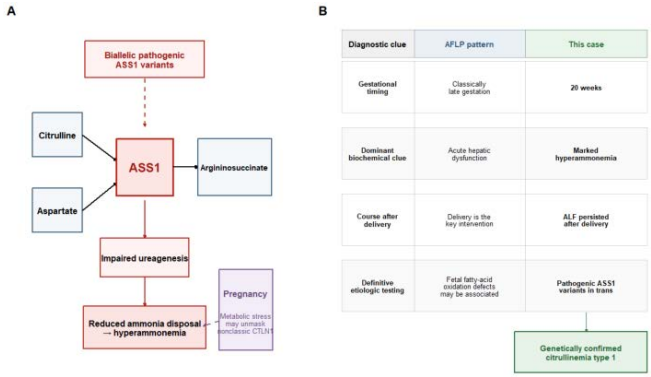
Assem Al Refaei[1] Makeda Dawkins[1] Naveena Sunkara[1] Raey Gesese[2] Yifan Gao[2] Bernard Partiula[1]

[1]Westchester Medical Center, [2]New York Medical College

Introduction: Acute fatty liver of pregnancy (AFLP) typically occurs in late gestation. Citrullinemia type 1 (CTLN1) is caused by biallelic pathogenic variants in ASS1, impairing argininosuccinate synthetase activity, ureagenesis, and ammonia detoxification. We report genetically confirmed CTLN1 presenting with fulminant acute liver failure (ALF) at 20 weeks' gestation and initially mimicking AFLP. **Case Presentation:** A woman in her early 30s, gravida 1 para 0, at 20 weeks' gestation presented after one week of nausea, vomiting, and dark urine. She had prior partial hepatectomy for benign hepatic adenomas but no known chronic liver disease. Initial studies showed AST 2,027 U/L, ALT 3,855 U/L, total bilirubin 8.0 mg/dL, ammonia 178 µmol/L, INR >10, and factor V activity 21.3%. Within hours she developed confusion and asterixis with progressive hepatic encephalopathy. Doppler ultrasonography showed patent hepatic vasculature without biliary obstruction. A broad ALF evaluation for viral, autoimmune, toxic, and vascular causes was unrevealing. Liver biopsy was deferred due to coagulopathy, and head CTs remained without acute intracranial pathology. Pregnancy-associated ALF, particularly AFLP, became the leading working diagnosis despite the atypically early gestation. Pregnancy-associated ALF, particularly AFLP, became the leading working diagnosis despite the atypically early gestation. CVVHD was initiated for hyperammonemia with N-acetylcysteine, hypertonic saline, neurocritical monitoring, and correction of coagulopathy. Multidisciplinary consensus favored expedited previable delivery for maternal rescue. Despite delivery, ALF persisted: ammonia peaked at 267 µmol/L, INR again exceeded 10, and factor V fell to 11.5%. She was listed Status 1A and underwent orthotopic liver transplantation, followed by biochemical and neurologic recovery and discharge home. Post-transplant testing identified pathogenic ASS1 variants c.535T>C (p.W179R) and c.1087C>T (p.R363W); parental testing confirmed one variant from each parent, establishing variants in trans and CTLN1. Fetal fatty-acid oxidation testing showed no reportable variants in HADHA, HADHB, or ACADVL. **Discussion:** This case demonstrates CTLN1 presenting as pro-

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found hyperammonemic ALF in mid-pregnancy and clinically resembling AFLP. The unusually early gestational age, marked hyperammonemia, and persistent hepatic failure despite delivery were key clues to an alternative metabolic etiology. Physiologic stress may precipitate hyperammonemic decompensation in previously asymptomatic CTLN1, making continued metabolic evaluation critical when pregnancy-associated ALF follows an atypical course. **Conclusion:** In pregnancy-associated ALF, atypical timing, marked hyperammonemia, or failure to improve after delivery should prompt evaluation for a urea-cycle disorder while aggressive ammonia control and early transplant evaluation proceed.

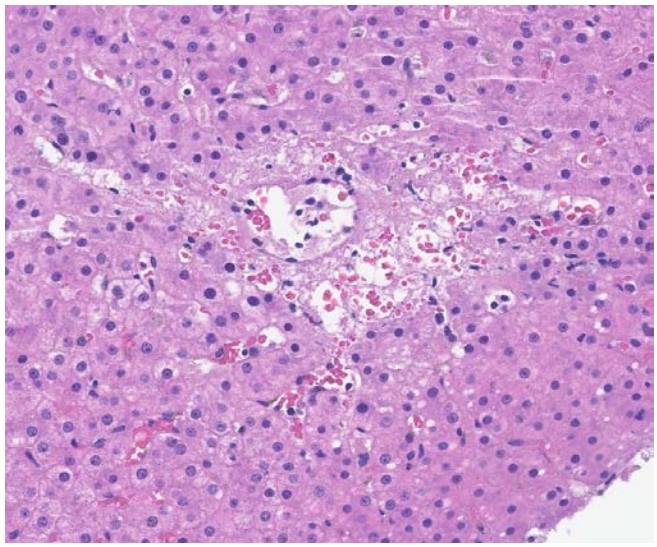


3633 | RECURRENT ACETAMINOPHEN-INDUCED ACUTE LIVER FAILURE AND CYP2D6 GENETIC VARIATION: A CASE REPORT

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Introduction: Acetaminophen (APAP) hepatotoxicity is dose-dependent, mediated by the reactive metabolite NAPQI, classically generated by CYP2E1, CYP1A2, and CYP3A4. CYP2D6 also bioactivates APAP, accounting for up to ~12.6% of hepatic NAPQI formation, with greater contributions in ultrarapid metabolizers. We report two episodes of APAP-induced acute liver failure (ALF), in a patient with a CYP2D6 profile consistent with increased enzymatic activity, highlighting pharmacogenetic susceptibility as a risk factor for recurrent APAP hepatotoxicity. **Case Presentation:** A 37-year-old woman with obesity status post Roux-en-Y gastric bypass, hypothyroidism, and migraines presented with abdominal pain, vomiting, and lethargy after taking 2-3 325 mg APAP tablets every 4-6 hours for one week following a motor vehicle accident. She was tachycardic and hypotensive with AST 3500, ALT 2860, INR 6.15, ammonia 136, lactate 13.4, and APAP level 23. She developed encephalopathy requiring CVVH and intubation; received N-acetylcysteine (NAC) and recovered without transplant.Two years later, after starting oxycodone/APAP 325 mg twice daily for one week for chronic pain, she re-presented with lethargy, hypoglycemia, and lactic acidosis. Labs showed AST 9091, ALT 6274, INR 7.58, ammonia 54, and APAP level 34. She developed asterixis and psychomotor slowing. She received NAC and recovered without transplant. Genotyping revealed heterozygosity for the no-function CYP2D6*4 allele plus a CYP2D6 gene duplication, raising suspicion for an ultrarapid-metabolizer phenotype. **Discussion:** Recurrent ALF after separate, modest APAP exposures suggest host predisposition to hepatotoxicity. CYP2D6 duplication typically confers an ultrarapid-metabolizer phenotype when the amplified allele is functional, potentially enhancing NAPQI generation and lowering the hepatotoxicity threshold to near therapeutic dosing. Prior studies implicate other metabolizing-gene variants in APAP-ALF risk, supporting a multifactorial genetic contribution. The second episode involved an oxycodone/APAP product that is disproportionately represented in APAP-ALF due to unintended exposure to APAP. **Conclusion:** This case illustrates a pharmacogenetic mechanism, CYP2D6-mediated ultrarapid APAP bioactivation, underlying recurrent APAP-ALF. Clinicians should consider pharmacogenomic evaluation after unexplained/recurrent APAP-ALF and counsel at-risk patients to avoid APAP, including in combination of opioid products.



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3634 | A STEPWISE DIAGNOSTIC APPROACH TO ISOLATED CONJUGATED HYPERBILIRUBINEMIA: MOLECULAR CONFIRMATION OF DUBIN-JOHNSON SYNDROME

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Introduction: Isolated conjugated hyperbilirubinemia in an otherwise healthy young adult presents a broad differential including Dubin-Johnson syndrome (DJS), Rotor syndrome, and other inherited cholestatic disorders. Without a structured workup, these conditions are often misattributed or remain undiagnosed. This case highlights a stepwise diagnostic approach using biochemical testing, urine porphyrin fractionation, and multigene panel testing to establish a definitive molecular diagnosis without liver biopsy. Confirming the diagnosis also allows for counseling on relevant drug interactions. **Case Presentation:** A 24-year-old male with no significant medical history was referred for isolated hyperbilirubinemia with total bilirubin levels as high as 5.0 mg/dl since 2024. He was asymptomatic aside from mild scleral icterus noted by his spouse. He denied pruritus, dark urine, alcohol use, hepatitis exposure, medications or supplements, and family history of liver disease. Laboratory evaluation showed total bilirubin 2.68 mg/dL with direct bilirubin 1.9 mg/dL. Liver enzymes, albumin, CBC, and total bile acids were normal. Abdominal ultrasound demonstrated mild hepatomegaly and small gallbladder polyps without biliary dilation. Given the predominantly conjugated pattern with preserved synthetic function, urine porphyrin fractionation was obtained. Urine coproporphyrin I was elevated at 11 umol/mol CRT with a normal coproporphyrin III, consistent with an inverted ratio. A 112-gene cholestasis panel identified two pathogenic ABCC2 variants on opposite chromosomes, confirming DJS by genomic analysis. **Discussion:** This case demonstrates how a targeted diagnostic approach can efficiently distinguish DJS from other inherited causes of conjugated hyperbilirubinemia. In DJS, dysfunction of the multidrug resistance-associated protein 2 (MRP2) transporter redirects coproporphyrin I from bile into urine, inverting the normal urinary coproporphyrin I:III ratio from approximately 30:70 to greater than 80% coproporphyrin I, as seen in this patient. Fractionated urine porphyrins offer a valuable noninvasive step that narrows the differential before genetic testing. **Conclusion:** Establishing the diagnosis of DJS avoided unnecessary liver biopsy and provided prognostic reassurance. As DJS follows a benign course requiring no treatment or lifestyle modification, counseling should focus on caution when using estrogen-containing contraceptives and other drugs that rely on MRP2-mediated biliary excretion, as they may precipitate or worsen hyperbilirubinemia in these patients. This case highlights the diagnostic and counseling value of a stepwise, noninvasive evaluation in patients with isolated conjugated hyperbilirubinemia.

Molecular Diagnosis of Dubin-Johnson Syndrome

Cholestasis Gene Panel (112 Genes)

Gene	Variant	Protein Change	Zygosity	Classification
ABCC2	c.2302C>T	p.Arg768Trp	Heterozygous	Pathogenic
ABCC2	c.2370del	p.Val791Trpfs*5	Heterozygous	Likely Pathogenic

Key Finding

Two pathogenic/likely pathogenic ABCC2 variants were identified, consistent with compound heterozygosity and confirming a molecular diagnosis of Dubin-Johnson syndrome.

Figure 1. Cholestasis gene panel demonstrating disease-causing ABCC2 variants.

3635 | CYTOMEGALOVIRUS HEPATITIS COMPLICATED BY SPLENIC INFARCTION IN AN IMMUNOCOMPETENT ADULT: A CASE FOR INDIVIDUALIZED ANTIVIRAL THERAPY

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Introduction: Cytomegalovirus (CMV) hepatitis has been associated with immunocompromised hosts. However, few studies have described its appearance in immunocompetent adults. We describe a case of CMV hepatitis in a previously asymptomatic immunocompetent host, with appearance of extrahepatic manifestations. **Case Presentation:** A 71-year-old woman without previously known medical conditions presented to the emergency department with one week of nocturnal fevers and a six-month history of non-bloody diarrhea. She denied abdominal pain, nausea, or vomiting, and reported no recent travel or sick contacts. At presentation she was febrile (102°F) without hepatosplenomegaly, lymphadenopathy, or rash. Initial labs showed a mixed hepatocellular-cholestatic pattern (AST 244, ALT 341, ALP 450 U/L; normal total bilirubin) and mild thrombocytopenia (144 × 10^3/μL). CT of the abdomen and pelvis showed an incidental finding of a 1.8-cm splenic infarction. Workup for viral hepatitis A/B/C, HIV, atypical endocarditis organisms, and stool pathogens was unrevealing. After a week of recurrent fevers, uptrending liver enzymes, and conservative

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management, CMV IgM and EBV viral capsid antigen IgM tested positive, but EBV DNA PCR was negative; quantitative CMV PCR confirmed active infection with a viral load of 31,600 copies/mL, establishing the diagnosis of CMV hepatitis. Given persistent fever and rising aminotransferases, and after shared decision-making, intravenous ganciclovir (2.5 mg/kg every 12 hours) was started on hospital day 4. Fever resolved within 3 days; aminotransferase elevation and thrombocytopenia (204 K/uL) subsequently improved, with a 97% decline in CMV viral load (to 987 IU/mL) by day 11. She was discharged on oral valganciclovir 450 mg twice daily to complete a 14-day course. **Discussion:** CMV in immunocompetent adults is usually self-limited and it is rare to cause manifestations. Despite the rising liver enzymes, our differential did not include CMV hepatitis initially, given patient history of no previous medical conditions or overt risk factors. Moreover, splenic infarction is rare, with only two prior reported cases currently in the literature. **Conclusion:** This case highlights that CMV hepatitis should likely be in the differential of clinicians seeing patients with fevers and rising liver enzymes. Extrahepatic manifestations of the disease should be better characterized. Individualized antiviral therapy appears to be warranted in this condition, despite its immunological status.

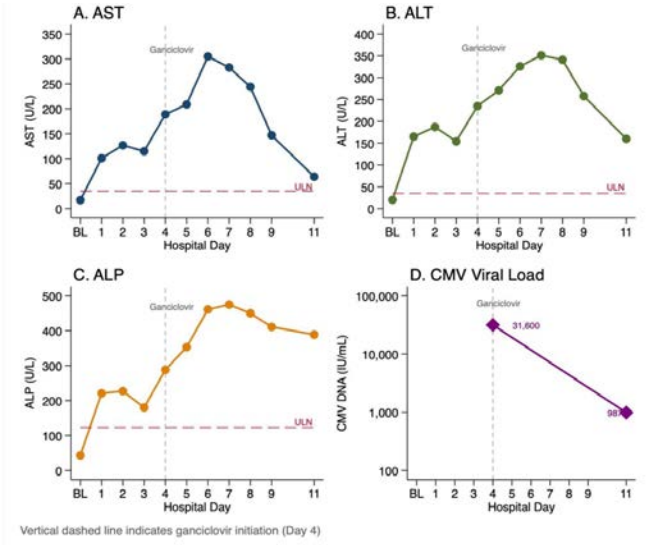


Figure 3. Laboratory trends during hospitalization for cytomegalovirus-associated hepatitis.

(A) Aspartate aminotransferase, (B) alanine aminotransferase, (C) alkaline phosphatase, and (D) CMV DNA viral load over the hospital course. The vertical dashed line indicates initiation of intravenous ganciclovir on hospital day 4. Horizontal dashed lines in panels A–C represent the upper limit of normal.

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BL, baseline; CMV, cytomegalovirus; ULN, upper limit of normal.

3636 | HEPATITIS D IN A PATIENT WITH CROHN'S DISEASE RECEIVING INFLIXIMAB: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE

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Introduction: Abnormal liver biochemistries in Crohn's disease have a broad differential, including primary sclerosing cholangitis (PSC), drug-induced liver injury, metabolic dysfunction-associated steatotic liver disease, and viral hepatitis. HDV is an important but potentially overlooked cause of liver injury in patients with chronic HBV. The intersection of chronic HDV infection and biologic therapy for Crohn's disease is poorly characterized. To our knowledge, we report the first case of chronic HDV infection identified in a patient with Crohn's disease receiving infliximab. **Case Presentation:** A 49-year-old man with chronic HBV, advanced hepatic fibrosis, and small- and large-bowel Crohn's disease complicated by perianal fistula and cutaneous involvement presented with persistent hepatocellular liver injury. He was receiving tenofovir alafenamide 25 mg daily with low-level HBV DNA. AST/ALT remained persistently elevated, reaching 120/128 U/L, while total bilirubin remained normal. Given competing etiologies, including infliximab-associated liver injury and small-duct PSC, further evaluation was pursued. HDV antibody was positive with HDV RNA 224,000 IU/mL (5.35 log IU/mL), establishing active infection; HBV DNA was 32 IU/mL. Prior VCTE and MR elastography demonstrated advanced fibrosis, with subsequent VCTE showing increased liver stiffness without steatosis. EUS-guided liver biopsy was planned to further characterize fibrosis and evaluate for small-duct PSC. Bulevirtide 8.5 mg subcutaneously daily was initiated while tenofovir was continued for HBV suppression. **Discussion:** The diagnostic challenge was distinguishing HDV-associated liver injury from other causes in a patient with Crohn's disease receiving biologic therapy. Infliximab exposure, IBD-associated hepatobiliary disease, and possible small-duct PSC were plausible alternative explanations. HDV testing identified a treatable viral etiology despite suppressed HBV replication. Concurrent HDV infection and necessary biologic therapy also complicated management. **Conclusion:** This case highlights an underrecognized intersection of chronic HDV infection, Crohn's disease, and biologic therapy. HDV should be considered in HBV-infected patients with unexplained hepatocellular injury, particularly when competing IBD- and medication-related etiologies exist. Recognition of active HDV enabled targeted antiviral therapy while maintaining HBV suppression and necessary Crohn's treatment.

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3637 | FROM AMYLOID TO MALIGNANCY: A RARE CASE OF ALECT2-ASSOCIATED CIRRHOSIS WITH HEPATOCELLULAR CARCINOMA

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[1]HCA Florida Largo

Introduction: Leukocyte cell-derived chemotaxin 2 (LECT2) amyloidosis (ALECT2) is a systemic amyloidosis and a leading cause of hepatic amyloid deposition in the US. Hepatic ALECT2 is generally indolent, though advanced fibrosis and portal hypertension are rarely reported. Hepatocellular carcinoma (HCC) in ALECT2-associated cirrhosis has rarely been described. We report a biopsy-confirmed case. **Case Presentation:** A 59-year-old Hispanic woman with systemic ALECT2, hypertension, and type 2 diabetes was referred for liver transplant evaluation. She had biopsy-proven cirrhosis with portal hypertension complicated by ascites and pleural effusions, for which TIPS was placed. MRI showed a segment 4A lesion growing to 4.0 × 4.5 cm (from 2.6 × 3.1 cm), with arterial hyperenhancement but no definitive washout (LI-RADS 4). Given interval growth despite indeterminate imaging features, biopsy was performed, showing well-differentiated HCC with solid and focal trabecular growth, mild nuclear atypia, and near-complete reticulin loss. Immunohistochemistry showed diffuse HepPar-1, focal glypican-3, and focal polyclonal CEA with a canalicular pattern, supporting hepatocellular differentiation. Serum AFP was normal. The lesion was within Milan criteria and MELD was 26; transplant evaluation is ongoing given systemic ALECT2 and dialysis-dependent renal involvement. Viral, autoimmune, metabolic, and genetic workups were unrevealing, with no alcohol use. MASLD was unlikely given low body weight and absent steatosis, though diabetes may be an independent HCC risk factor. **Discussion:** Hepatic ALECT2 is traditionally indolent but can progress to advanced fibrosis and portal hypertension. LECT2 has a complex role in hepatocarcinogenesis: it can act as a tumor suppressor but is also a Wnt/β-catenin transcriptional target and may be elevated in β-catenin-activated HCC. The well-differentiated morphology raises a possible pathway relationship, though β-catenin activation was not assessed, as additional tissue was unavailable. Chronic amyloid deposition, inflammation, and architectural distortion may promote fibrogenesis and a permissive oncogenic environment. Causality cannot be established from a single case, and diabetes or other metabolic cofactors may have contributed. **Conclusion:** This case expands the recognized spectrum of hepatic ALECT2 and highlights the potential for HCC in ALECT2-associated cirrhosis. Although generally indolent, ALECT2 may progress to cirrhosis, at which point standard HCC surveillance should be performed per current cirrhosis guidelines. Further study of the Wnt/β-catenin pathway is needed to clarify whether ALECT2-associated hepatic injury contributes to hepatocarcinogenesis.

3638 | TUMOR LYSIS SYNDROME FOLLOWING TRAUMATIC RUPTURE OF HEPATOCELLULAR CARCINOMA

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Introduction: Tumor lysis syndrome (TLS) results from rapid destruction of tumor cells, leading to release of intracellular components. Nearly all cases of TLS in hepatocellular carcinoma (HCC) have occurred following transarterial chemoembolization, transarterial radioembolization, or systemic therapy.4-6 To our knowledge, we present the first reported case of TLS developing after traumatic rupture of undiagnosed HCC, with tumor embolization precipitating tumor lysis. **Case Presentation:** A 68-year-old male with compensated alcohol-associated cirrhosis presented to the emergency department following blunt abdominal trauma during a physical altercation. Computed tomography (CT) of the abdomen and pelvis (A/P) demonstrated cirrhotic morphology with an infiltrative hepatic lesion involving the bilateral portal vein (Figure 1C). CT angiography A/P was concerning for ruptured malignancy with active extravasation of contrast (Figure 1A, B). The patient underwent emergent embolization of the left, right, and middle hepatic arteries that supplied the mass. Following the procedure, nephrology was consulted for acute kidney injury (AKI). Laboratory evaluation was notable for K-5.4 mmol/L, PO_4 - 6.5 mg/dL, Ca - 8.1 mg/dL, Uric Acid - 8.7 mg/dL, Cr - 1.7 mg/dL, and LDH -1426 U/L, consistent with Cairo-Bishop diagnostic criteria for laboratory and clinical TLS.3 Treatment with aggressive intravenous fluids and allopurinol was initiated with close monitoring and planned escalation to rasburicase pending a serum G6PD. Subsequent triphasic CT was diagnostic for HCC. Given bilateral portal invasion, the tumor was consistent with Barcelona Clinic Liver Cancer-Stage C disease. The patient's AKI resolved with normalization of electrolyte abnormalities. The patient was discharged on hospital day six. **Discussion:** TLS is a life-threatening oncologic emergency that can lead to severe metabolic de-

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rangements and ultimately progress to fatal complications such as cardiac arrhythmias and seizures. The majority of TLS occurs in hematologic malignancies following induction of chemotherapy. In high-risk hematologic cancers, 15-20% of patients developed laboratory TLS and 5% developed clinical TLS.1 In contrast, TLS in solid tumors is uncommon. A systematic review identified 132 reported cases of TLS associated with solid tumors, of which 22 cases (17%) involved HCC.2 Recent reviews demonstrate the vast majority of HCC-associated TLS cases occur following locoregional therapies, radioembolization, or systemic anticancer treatment.4 Although traumatic rupture prompted the diagnosis of HCC, the temporal relationship between the embolization and subsequent development of TLS favors cellular lysis secondary to ischemic tissue necrosis rather than trauma alone. **Conclusion:** We believe this is the first reported case of TLS occurring after traumatic rupture of an undiagnosed HCC, with embolization acting as the inciting mechanism for tumor lysis.

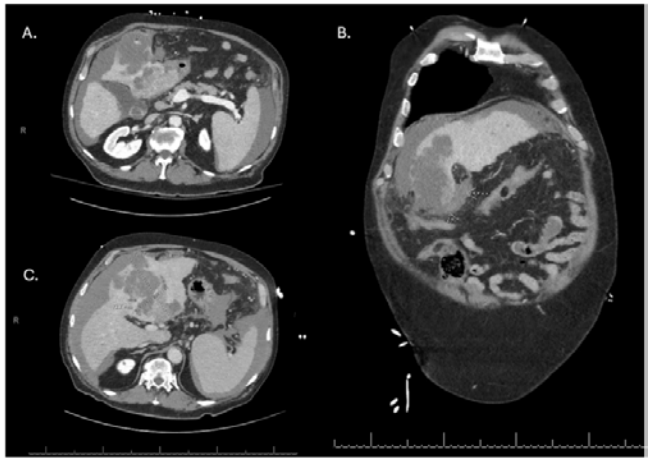


***Figure 1A** - focal hyperattenuation within the collateral component of the mass which protrudes from the hepatic capsule, which raises concern for active extravasation, concerning for ruptured mass. **Figure 1B** – extensive perihepatic blood products with areas of subcapsular hematoma*

*(Grade III injury). **Figure 1C** - large infiltrative, heterogenous dominant mass centered in segment 4A/B measuring approximately 13 x 8.5 x14 cm*

3639 | RECURRENT ACUTE HEPATOCELLULAR INJURY FOLLOWING REPORTED OCCUPATIONAL HCFC-123 EXPOSURE

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Introduction: We describe recurrent HCFC-123-induced hepatocellular injury with a documented positive rechallenge, a rarely observed but highly persuasive indicator of causality in toxicant-induced liver injury. Re-exposure after recovery precipitated a more severe recurrence within 10 days. **Case Presentation:** A 24-year-old previously healthy man developed malaise, fatigue, RUQ abdominal pain, nausea, and vomiting one day after occupational exposure while removing refrigeration and cooling system components, with three days of symptoms before admission on 3/10/26. He denied hepatotoxic medications, supplements, or recreational drug use, and reported sporadic, light alcohol use (approximately one shot of liquor occasionally). Admission labs showed severe hepatocellular injury: AST 1,426 U/L, ALT 1,507 U/L, ALP 146 U/L, GGT 322 U/L, total bilirubin 5.6 mg/dL, albumin 3.6 g/dL, platelets 181 x10^9/L, INR 1.6 (R-ratio 23.6). Serologic workup for viral, autoimmune, Wilson disease, and metabolic liver disease was negative. MRCP showed gallbladder wall thickening/edema without obstruction. He received standard IV N-acetylcysteine (3/11-3/15/26), improved, and was discharged on 3/16/26, with liver tests trending toward normal by 3/23/26. He returned to the same occupational environment and duties and was readmitted 3/26/26 with recurrent, more severe hepatocellular injury (AST 2,294 U/L, ALT 2,841 U/L, ALP 183 U/L, total bilirubin 5.4 mg/dL, albumin 4 g/dL, platelets 373 x10^9/L, R-ratio 38.8). Liver biopsy (3/30/26) showed mild resolving hepatitis with a macrophage-predominant infiltrate, no fibrosis (trichrome), intact lobular architecture (reticulin), and negative iron, PAS-D, AFB, GMS, CMV, HIV1/2, and EBV-EBER stains. Clinical course followed positive rechallenge with worsening aminotransferase elevation than at presentation before trending down from 3/31/26 and normalizing by 4/20/26. **Discussion:** The patient reported exposure to HCFC-123 (R-123) based on information relayed by his employer; no documentation (safety data sheet, industrial hygiene records) was available to independently confirm this. Given the temporal association with reported occupational exposure, an explicit positive rechallenge, exclusion of infectious, autoimmune, and metabolic etiologies, and biochemical recovery after removal from exposure, toxicant-induced liver injury, reportedly related to HCFC-123, was considered the most likely diagnosis. **Conclusion:** This case illustrates a defined positive rechallenge with occupational refrigerant exposure causing recurrent acute hepatocellular injury, and underscores both the value of detailed occupational history-taking in unexplained acute hepatitis and the limitations of relying on unverified employer-reported exposure information.

Date	Day	AST (U/L)	ALT (U/L)	ALP (U/L)	T. Bili (mg/dL)	Albumin (g/dL)	Platelets (x10^9)	INR	R-ratio	Clinical Event
3/10/2026	Day 0	1426	1507	146	5.6	3.6	181	1.6	23.6	Admission 1
3/11/2026	Day 1	1536	1646	109	4.5	2.7	147	1.7		IV NAC
3/12/2026	Day 2	1968	2528	111	4.8	2.5	150	1.2		IV NAC
3/13/2026	Day 3	1433	2598	119	4.4	2.4	153	1.2		IV NAC
3/14/2026	Day 4	572	2056	129	3.1	2.5	154	1.1		IV NAC
3/15/2026	Day 5	392	1701	153	2.3	2.6	185	1.1		IV NAC
3/16/2026	Day 6	250	1336	154	2.1	2.8	215	1.2		Discharge 1
3/23/2026	Day 13	253	762	167	1.4	3.9	431	N/A		Low point, near normalization
3/26/2026	Day 16	2294	2841	183	5.4	4.0	373	1.1	38.8	Admission 2
3/30/2026	Day 20	202	1060	155	0.8	3.1	312	1.1		Liver biopsy
3/31/2026	Day 21	113	872	148	0.8	3.1	308	N/A		LFTs trending down
4/6/2026	Day 26	40	237	132	0.9	3.7	N/A	N/A		LFTs trending down
4/20/2026	Day 41	31	79	109	0.7	4.2	N/A	N/A		Full normalization

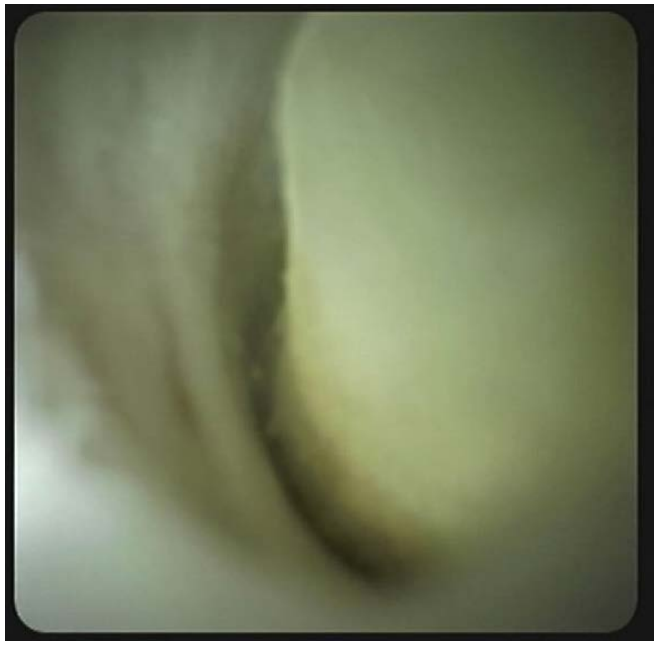
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3640 | LEFT-LOBE HEPATOLITHIASIS IN A YOUNG WOMAN: FROM RECURRENT BILIARY SYMPTOMS TO HEPATECTOMY

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Introduction: Hepatolithiasis, defined as calculi within the intrahepatic bile ducts, is common in East Asia but rare in Western populations (<1% of biliary stones) and particularly uncommon in young adults. Its rarity and nonspecific presentation can delay diagnosis and complicate management. **Case Presentation:** A 34-year-old woman presented with recurrent right upper quadrant pain, nausea, and vomiting over several years. Despite extensive evaluation and cholecystectomy, her symptoms persisted without a definitive diagnosis. During her most recent admission, she developed scleral icterus and right upper quadrant tenderness with cholestatic liver injury (ALP 290 U/L, total bilirubin 3.2 mg/dL), elevated transaminases, and leukocytosis. ERCP revealed dilated left-lobe intrahepatic ducts containing stones. Smaller stones and sludge were removed, but an impacted pigment stone could not be retrieved due to ductal narrowing. Cholangioscopy and EUS confirmed hepatolithiasis. She improved with antibiotics and supportive care and was referred for surgical evaluation. Given localized left-lobe disease and incomplete endoscopic clearance, she underwent successful left hepatectomy in December 2025. **Discussion:** This case demonstrates that hepatolithiasis can occur in young adults in Western populations and remain undiagnosed despite years of biliary symptoms. To our knowledge, no prior cases have been reported from Puerto Rico. Chronic hepatolithiasis is associated with recurrent cholangitis, biliary strictures, and an increased risk of cholangiocarcinoma, particularly with persistent stones and chronic inflammation. Management should be individualized according to stone location, disease extent, and underlying biliary anatomy. Endoscopic therapy can provide biliary decompression and stone clearance; however, impacted stones and intrahepatic ductal narrowing may preclude complete clearance. In selected patients with localized disease, hepatic resection offers definitive treatment by removing affected biliary segments and residual stone burden, thereby limiting prolonged exposure to chronic inflammation-a particularly relevant consideration in younger patients with longer life expectancy. **Conclusion:** Hepatolithiasis should remain in the differential diagnosis of recurrent biliary symptoms and unexplained cholestatic liver injury, even in young patients from Western populations. This case highlights the importance of recognizing hepatolithiasis outside endemic regions and tailoring therapy to disease distribution and response to endoscopic management. When disease is localized and complete endoscopic clearance cannot be achieved, surgical resection may provide definitive treatment and reduce recurrent complications



3641 | ALCOHOL BIOMARKER TESTING IN A HOSPITALIZED PATIENT WITH CIRRHOSIS AND RECENT TRANSFUSIONS RAISES A "PETH-ORA" OF QUESTIONS

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Introduction: PEth (phosphatidylethanol) testing is routinely obtained in those undergoing liver transplant evaluation. PEth is formed by a transphosphatidylation reaction that substitutes ethanol for water on RBC membranes. In general, PEth thresholds of < 20ng/mL indicate abstinence, 20-200 ng/mL alcohol use, and > 200 ng/mL suggests chronic, excessive alcohol intake. We describe marked PEth elevation secondary to massive transfusion protocol (MTP) and discuss the implications for hospitalized patients seeking liver transplant. **Case Presentation:** 42YM with no medical history was transferred to a tertiary liver center after a failed attempt at TIPS shunt placement for hematemesis from esophageal variceal bleed requiring MTP x2

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(13u pRBC). After intubation for airway protection, he underwent EGD with variceal banding x6. On transfer, laboratory testing showed Hgb 10, PLT 123, AST 3439, ALT 974, TB 3.2, Cr 3.1, INR 2, lactate 3.5, MELD 28. Imaging revealed an enlarged liver with surface nodularity, patent vessels and sequela of portal hypertension. TIPS shunt was again attempted but failed, and transhepatic variceal embolization was performed. The patient was successfully extubated. Based on negative workup and risk factor assessment, his cirrhosis was initially attributed to MASH, however, PEth 16:0/18:1 drawn on transfer returned at 254ng/mL, suggesting heavy alcohol use. The patient was given the AUDIT-C and scored 1. He and his family strongly denied significant alcohol use (1 drink ~ 1x/month). **Discussion:** PEth testing is specific and sensitive for alcohol intake requiring ~2-4 drinks daily for several weeks to form on RBC surfaces. Artificial elevations can occur by transfusion as PEth resides within donor RBC membranes. One study found that over 40% of donors have PEth >=10 ng/mL (max 587) and processing whole blood into RBC raises PEth levels.1 In a cohort of 143 patients, even one PEth+ unit changed 19.6% of patients from negative to positive (>20 ng/mL) with highest post-transfusion value of 83.4.2 Transfusion can also dilute PEth so interpretation can be skewed in both directions. The implications of PEth testing are life altering for liver transplant candidates as false positives may limit transplant chances. No formal consensus, guidance or protocol exists for interpreting post-transfusion PEth. **Conclusion:** PEth level should be drawn before transfusion or performed on prior stored EDTA sample. Blood transfusion is common in hospitalized patients with cirrhosis, and positive PEth should prompt a 28-day blood transfusion history. A single unit transfusion may result in moderate elevation, but interpretation of PEth >200 is difficult in the setting of MTP. Ethyl glucuronide from urine or hair is not affected by transfusion but detection window is short for urine (3-5 days) and hair assay is less accessible. Appreciating factors which impact PEth results is essential before prioritizing its role in clinical decision-making.

3642 | DISEASE-MODIFYING THERAPY WITH AN IBAT INHIBITOR IN A PATIENT WITH ANTIBODY-INDUCED BSEP DEFICIENCY AFTER LIVER TRANSPLANTATION AND REFRACTORY CHOLESTATIC PRURITUS: A CASE REPORT

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Introduction: Antibody-induced bile salt export pump (BSEP) deficiency is a rare and potentially graft-threatening recurrence of the progressive familial intrahepatic cholestasis type 2 (PFIC2) phenotype after liver transplantation (LT). Current treatment strategies target the underlying humoral immune response but may fail to control cholestasis and pruritus or prevent progressive graft injury. Ileal bile acid transporter (IBAT) inhibition reduces pruritus and improves clinical outcomes in patients with PFIC2. Whether these therapeutic effects can be translated to anti-BSEP antibody-mediated disease after LT remains unknown. **Case Presentation:** A male patient homozygous for the *ABCB11* variant c.2178+1G>C underwent LT at age 12 years for refractory pruritus and excessive biliary loss following partial external biliary diversion. Two years later, severe pruritus and low-gamma-glutamyl transferase cholestasis recurred. Liver biopsy demonstrated giant-cell hepatitis, collapse necrosis, fibrosis and canalicular-bound antibodies, while circulating anti-BSEP antibodies confirmed antibody-induced BSEP deficiency. Progressive graft fibrosis (F4) prompted listing for retransplantation at age 15. Despite six years of treatment with immunoadsorption, plasma exchange, intravenous immunoglobulin, rituximab, intensified immunosuppression and multiple antipruritic therapies, disease control could not be achieved. At age 20, odevixibat, an IBAT inhibitor, was started at 40 µg/kg/day and increased to 60 µg/kg/day while plasma exchange was continued at an unchanged frequency. Within 2 weeks, serum bile acids decreased from 223 to 35 µmol/l, AST from 202 to 42 U/l and ALT from 299 to 64 U/l, accompanied by rapid and complete resolution of pruritus. After 42 weeks, sustained clinical and biochemical improvement permitted discontinuation of plasma exchange without recurrence of pruritus or biochemical deterioration during the subsequent 20 months of follow-up. **Discussion:** To the best of our knowledge, this is the first reported use of IBAT inhibition for antibody-induced BSEP deficiency after LT. The rapid and sustained clinical and biochemical response despite advanced graft fibrosis and failure of

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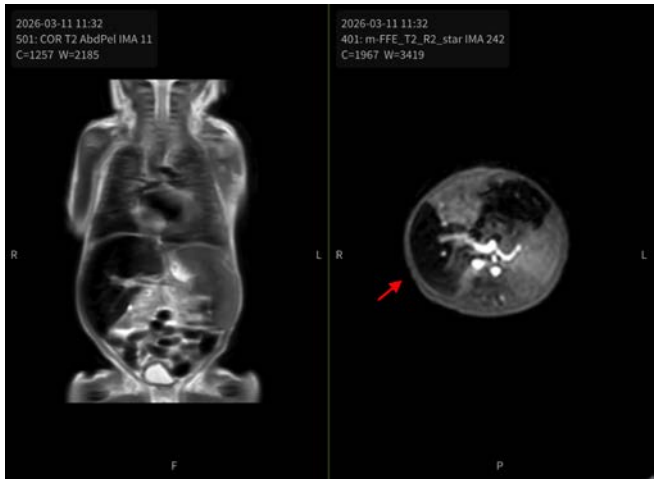
multiple therapies suggests that interruption of the enterohepatic bile acid circulation may remain effective even in severe antibody-mediated BSEP disease. **Conclusion:** In PFIC2 recipients with recurrent cholestasis and pruritus, antibody-induced BSEP deficiency should be considered. IBAT inhibition reduces intestinal bile acid reuptake without restoring BSEP function or suppressing antibody production and may therefore represent a mechanistically complementary approach to immunotherapy. Thus, IBAT inhibitors, such as odevixibat, may improve refractory pruritus and reduce the need for apheresis in patients with refractory antibody-induced BSEP deficiency.

3643 | MIMICKING GALD: CHOLESTASIS AND COAGULOPATHY IN ANTI-E HEMOLYTIC DISEASE OF THE NEWBORN

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Introduction: Hemolytic disease of the fetus and newborn (HDFN) results from maternal IgG alloantibodies against fetal red cells, leading to hemolysis and potentially hydrops or fetal demise. While cholestasis is a known complication of HDFN, coagulopathy is exceptionally rare. When signs of liver dysfunction are seen alongside HDFN, the presentation can closely mimic gestational alloimmune liver disease (GALD), a condition in which maternal IgG causes complement-mediated fetal hepatocyte injury and neonatal acute liver failure. **Case Presentation:** A male infant was born at 35 weeks following a pregnancy complicated by anti-E alloimmunization, fetal anemia requiring two intrauterine transfusions, and a maternal history of fetal demise. Postnatally, he exhibited cholestasis (total bilirubin 16.8 mg/dL, direct 9.2 mg/dL), coagulopathy (INR 2.0), thrombocytopenia (platelets 4.6×10³/µL), hypoalbuminemia (2.0 g/dL), and alanine aminotransferase 76 U/L. He received vitamin K, phototherapy, intravenous immunoglobulin (IVIG), and exchange transfusion by 10 hours of life. He had improvement of INR and cholestasis with multiple blood products and doses of IVIG over the following week, but he then developed a rebound coagulopathy on day of life 9. Evaluation for GALD showed elevated ferritin (2,882 ng/mL) and transferrin (99%), but normal alpha-fetoprotein (AFP 10,706 ng/mL). Extensive evaluation for metabolic, infectious, and hemophagocytic etiologies, including whole genome sequencing, were otherwise unrevealing. A T2-weighted MRI revealed siderosis isolated to the right hepatic lobe, with normal signal in the pancreas and spleen, ruling out GALD. Coagulopathy resolved by day 17, and his prolonged cholestasis improved over subsequent months with supportive care and ursodeoxycholic acid. **Discussion:** This case highlights the diagnostic challenge when severe HDFN presents with features of GALD. The infant's signs of liver dysfunction, including clinical coagulopathy and cholestasis, low-normal ALT, thrombocytopenia, hypoglycemia, and a maternal history of stillbirth were highly suspicious for GALD. The hyperferritinemia and hypersaturated transferrin were evidence of iron overload that overlap both HDFN and GALD. However, AFP was far below expected GALD levels, and the absence of extrahepatic siderosis on MRI definitively ruled it out. While 10-13% of HDFN cases feature mild cholestasis, coagulopathy is exceedingly rare. Proposed mechanisms for this phenotype include iron-mediated hepatocellular injury and downregulated bile salt pumps. **Conclusion:** Although HDFN and GALD share initial therapies like IVIG and exchange transfusion, distinguishing them is crucial. GALD carries a high recurrence risk in future pregnancies that can be mitigated with antenatal IVIG.



3644 | INDUCED HYPERNATREMIA, HEMODIALYSIS DEBULKING, AND DUAL-CIRCUIT CRRT FOR EXTREME HYPERAMMONEMIA IN ACUTE LIVER FAILURE

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Introduction: Intracranial hypertension (ICH) in acute liver failure (ALF) has declined with earlier renal replacement therapy but remains lethal in severe hyperammonemia. Arterial ammonia independently predicts encephalopathy and ICH, which develops in 55% of patients with levels >200 µmol/L. Guidelines support

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early continuous renal replacement therapy (CRRT) for severe hyperammonemia even without renal indications, but clearance may be inadequate when ammoniagenesis exceeds single-circuit capacity. We present an adult patient presenting with ALF complicated by extreme hyperammonemia refractory to high-dose CRRT, managed with hemodialysis debulking followed by simultaneous dual-circuit CRRT as a bridge to successful transplantation. **Case Presentation:** A previously healthy 34-year-old man presented with three days of vomiting, abdominal pain, jaundice, and shock with ALF. Studies showed pH <6.93, lactate >17 mmol/L, AST 8,036 U/L, ALT >10,000 U/L, bilirubin 11 mg/dL, INR >10, creatinine 3.9 mg/dL, ammonia 665 µmol/L, and MELD 52. ALF etiologic evaluation was unrevealing; he was listed Status 1A.Despite intubation, triple vasopressors, N-acetylcysteine, and CRRT at 60 mL/kg/hr, ammonia rose to 906 µmol/L with progressive obtundation, indicating ammoniagenesis exceeded single-circuit clearance. Given extreme cerebral edema risk, neuroprotective RRT was escalated. Custom 7.5% saline raised serum sodium to 145-150 mEq/L before 2 hours of iHD for rapid ammonia debulking (Qb 300/Qd 600 mL/min, chilled dialysate with sodium 150 mEq/L, MAP >80 mmHg), followed by simultaneous dual-circuit CRRT at 75 mL/kg/hr per circuit for sustained clearance. Ammonia fell to 768, 459, then 300 µmol/L without disequilibrium or ICH. He underwent liver transplantation on hospital day three and recovered neurologically intact with excellent graft function at five months. **Discussion:** Conventional CRRT delivers 20-25 mL/kg/hr; at 60 mL/kg/hr, ammonia continued to rise, indicating ammoniagenesis exceeded single-circuit clearance. At ultra-high doses, clearance may be limited by dialysate-to-blood flow relationships, membrane capacity, and machine flow limits; a second circuit adds membrane area with additional blood- and effluent-processing capacity, increasing achievable clearance. Induced hypernatremia, sequential iHD, and dual-circuit CRRT have been described for hyperammonemia management; however, to our knowledge, deliberate induction of hypernatremia followed by neuroprotective iHD for ammonia debulking and immediate simultaneous dual-circuit CRRT for sustained clearance has not been reported in adult ALF. **Conclusion:** When ammoniagenesis exceeds single-circuit CRRT capacity, this integrated neuroprotective RRT strategy-controlled hypernatremia, iHD for rapid ammonia debulking, and dual-circuit CRRT for sustained clearance-may provide an effective bridge to transplantation and warrants further study.

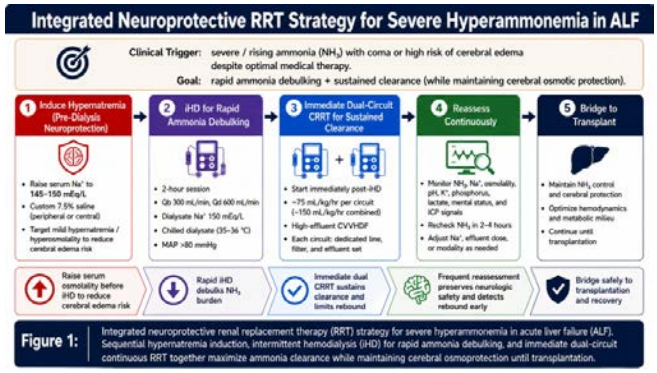


Figure 1: Integrated neuroprotective renal replacement therapy (RRT) strategy for severe hyperammonemia in acute liver failure (ALF). Sequential hypernatremia induction, intermittent hemodialysis (iHD) for rapid ammonia debulking, and immediate dual-circuit continuous RRT together maximize ammonia clearance while maintaining cerebral osmoprotection until transplantation.

3645 | WHEN THERAPEUTIC BECOMES TOXIC: ACUTE LIVER FAILURE FOLLOWING ACETAMINOPHEN USE AFTER ALCOHOL BINGE

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Introduction: Acetaminophen is the leading cause of acute liver failure (ALF) in the United States and is classically associated with overdose. However, severe hepatotoxicity may occur at therapeutic doses in patients with specific risk factors, including chronic or recent excessive alcohol use. Recognition of acetaminophen-associated ALF may be challenging when serum drug levels are low. We present a case of fulminant ALF following therapeutic acetaminophen use in the setting of recent alcohol binge. **Case Presentation:** A 23-year-old woman with no known chronic liver disease presented with one day of nausea, vomiting, and right upper quadrant abdominal pain. She reported intermittent acetaminophen use for two to three weeks, typically 600 mg per dose, with increased intake to approximately 3-3.6 g daily following a weekend binge of approximately 30 beers. Initial laboratory testing revealed AST 766 U/L, ALT 481 U/L, INR 1.9, total bilirubin 2.4 mg/dL, thrombocytopenia, and ammonia 156 µmol/L. Serum acetaminophen level obtained approximately 24 hours after last ingestion was 16 µg/mL. Imaging demonstrated hepatic steatosis without cirrhosis or biliary obstruction. Despite supportive care, she rapidly progressed to ALF with peak AST 6,976 U/L, ALT 6,036 U/L, INR 16.1, ammonia 248 µmol/L, acute kidney injury, shock, and hepatic encephalopathy. An extensive evaluation excluded viral, autoimmune, metabolic, and vascular etiologies. She was treated with intravenous N-acetylcysteine, continuous renal replacement therapy, and therapeutic plasma exchange. Liver transplantation was not pursued. **Discussion:** This case highlights fulminant ALF resulting from therapeutic acetaminophen use in the

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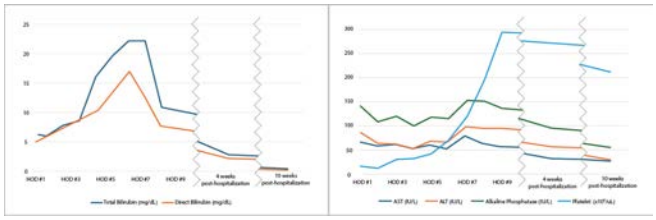
setting of recent alcohol binge and prior alcohol exposure. Alcohol induces cytochrome P450 2E1, increasing production of toxic acetaminophen metabolites while depleting hepatic glutathione, rendering hepatocytes vulnerable even at recommended doses. Importantly, low or therapeutic serum acetaminophen levels do not exclude toxicity, particularly in delayed or repeated supratherapeutic ingestion. Early initiation of N-acetylcysteine and aggressive supportive management, including extracorporeal therapies, may facilitate recovery without liver transplantation. Clinicians should maintain high suspicion for acetaminophen-associated ALF in patients with recent alcohol use, regardless of reported dosing or serum drug levels. **Conclusion:** Alcohol-related susceptibility can amplify acetaminophen hepatotoxicity despite reported therapeutic dosing. Recognition of this high-risk phenotype and timely implementation of liver failure-directed therapies may improve transplant-free survival.

3646 | JAUNDICE AND ACUTE KIDNEY INJURY IN RECOMPENSATED ALCOHOL-RELATED LIVER DISEASE

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Introduction: Alcohol abstinence has been shown to prevent alcohol-related liver disease (ALD) progression and, in some cases, resolve clinical manifestations of hepatic fibrosis and portal hypertension. A history of ALD can bias clinicians toward recurrent alcohol-related hepatitis, but infectious mimics should remain on the differential. **Case Presentation:** A 36-year-old man (NB) with a history of decompensated ALD presented with fever, abdominal pain, nausea, and vomiting. He was previously listed for liver transplant in 2022 with a MELD of 29, delisted in 2024 due to clinical improvement after alcohol cessation. He reported last drinking alcohol in 2022 and denied any acetaminophen use, new medications, herbal supplements, vitamins, or unprescribed substances.Physical exam was notable for scleral icterus, a soft and nontender abdomen without bulging flanks, localized petechial rash, and no asterixis. Labs showed mixed liver injury, conjugated hyperbilirubinemia, and thrombocytopenia (Fig 1), INR 1.2, WBC 12.6, Cr 3.4, Na 130, K 3.2, CK 519. MELD on admission of 30.Hepatology was consulted on admission given suspected acute on chronic liver disease (ACLD) and recurrent alcohol-related hepatitis. Our initial differential included leptospirosis given concomitant thrombocytopenia, myalgias, and acute kidney injury (AKI); leptospira serologies and Infectious Disease (ID) consultation were recommended. ID agreed with empiric treatment while awaiting serologic confirmation for severe leptospirosis/Weil Disease.On HOD 4, qualitative PCR of blood and urine detected leptospira. On further interview, NB reported removing rodent droppings from his dog's mouth. PEth <20ng/mL. NB completed a 7-day course of ceftriaxone with symptomatic improvement during his hospitalization. He was seen in Hepatology clinic post-hospitalization, with complete normalization of his liver enzymes, platelet count, and creatinine. **Discussion:** Leptospirosis is a zoonotic disease spread through urine of infected animals. While typically infection is self-limited, 10% of cases can present with Weil disease, characterized by fever, renal failure, and jaundice. Weil Disease may also affect the respiratory, cardiac, and musculoskeletal systems.In this case, an initial concern for recurrent ALD risked diagnostic anchoring, but the combination of fever, AKI, thrombocytopenia, and jaundice prompted evaluation for leptospirosis, confirmed by PCR. While alcohol relapse can be common in ALD, this patient reported abstinence since 2022 and had PEth <20ng/mLduring the hospitalization, making ACLD secondary to ALD unlikely. **Conclusion:** Maintaining a broad differential for fever and jaundice is crucial, even in patients with a history of ALD. Leptospirosis and other zoonotic diseases may also present with thrombocytopenia, elevated liver enzymes, and AKI-and may be overlooked when alcohol-related organ injury is presumed.



3647 | WHEN C. DIFFICILE GOES OFF-SCRIPT: AN EXTRAINTESTINAL HEPATIC ABSCESS AFTER APPENDECTOMY, COLON PERFORATION AND PERITONITIS

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Introduction: Clostridioides difficile is a leading cause of healthcare-associated colitis but is only rarely identified as a cause of extraintestinal infection. Hepatic abscess is among its rarest manifestations, with only a handful of cases reported in the literature. We describe a case of C. difficile hepatic abscess following laparoscopic appendectomy complicated by colonic perforation and polymicrobial peritonitis. **Case Presentation:** A 55-year-old woman with no significant medical history presented with periumbilical pain migrating to the right lower quadrant, leukocytosis and CT abdomen showing appendicitis. Given a reported penicillin allergy, she received levofloxacin/metronidazole and underwent laparoscopic appendectomy.On postoperative day one, she developed persistent pain, fever, tachycardia, and dyspnea; CT abdomen showed a loculated peri-resection collection. CTA chest (negative for PE) on day two incidentally revealed large-volume pneumoperitoneum. Laparoscopy showed diffuse purulent peritonitis with an intact appendiceal staple line, prompting laparotomy, which revealed a 2 cm hepatic flexure perforation, peritoneal fluid sample were collected and right hemicolectomy was performed.Peritoneal fluid grew Candida albicans, non-albicans yeast, E. coli, and Streptococcus viridans; blood cultures were negative. She received IV micafungin, vancomycin, aztreonam, and metronidazole, yet leukocytosis and thrombocytosis kept worsening and later developed sinus tachycardia and fever. Repeat CT abdomen showed a new subcapsular hepatic collection, IR drainage yielded 80 mL of fluid, which grew C. difficile and Candida Albicans. Oral vancomycin was started for decolonization, and Anti-fungal agent was continued for concurrent Candida growth. Imaging showed abscess improvement with normalizing labs; she was discharged on oral metronidazole, ceftriaxone, and fluconazole. **Discussion:** Extraintestinal CDI is uncommon, and hepatic abscess is exceptional, with prior reviews identifying only a few number of such cases.Proposed mechanisms include translocation of C. difficile across a disrupted bowel wall into the peritoneal cavity. Our patient's presentation is notable because hepatic seeding followed a colonic perforation discovered only on re-exploration, and occurred alongside concurrent fungal and enteric bacterial peritonitis. No standardized empiric regimen exists for C. difficile-related hepatic abscess; management in this and prior cases centered on drainage, anaerobic therapy, and gut decolonization. **Conclusion:** C. difficile hepatic abscess is rare but should be considered in atypical hepatic abscesses after enteric perforation. Absent formal guidelines, management can be informed by existing case reports and should include source control drainage, systemic anaerobic antibiotics, and oral vancomycin for decolonization to address the likely colonic origin of infection.



3648 | MALIGNANT TRANSFORMATION IN AN EXCLUDED BILIARY SEGMENT: A 25-YEAR LATENCY FOLLOWING IATROGENIC BILE DUCT INJURY

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Introduction: Iatrogenic bile duct injury (IBDI) carries well-documented acute and perioperative risks, but the ultra-long-term oncogenic potential of chronically excluded biliary segments remains underrecognized. Persistent biliary stasis and chronic inflammation in surgically excluded or oversewn ducts may establish a pro-carcinogenic microenvironment analogous to primary sclerosing cholangitis. **Case Presentation:** A patient who underwent open cholecystectomy complicated by intra biliary duct injury in 2001 received a Roux-en-Y hepaticojejunostomy repair, during which an accessory right bile duct was surgically oversewn. The patient remained clinically stable for over two decades; however, surveillance imaging in 2015 revealed chronic segmental ductal dilatation with localized hepatic atrophy. In 2026-25 years following the index surgery-the patient presented with right upper quadrant pain, nausea, and chills. Cross-sectional imaging demonstrated a 6.3 × 5.4 cm mass originating precisely within the chronically isolated and dilated biliary segment. Liver biopsy confirmed adenocarcinoma favoring a biliary primary against a background of dense fibrous stroma. Biliary instrumentation was avoided given the anatomical distortion, segmental isolation, and high risk of infectious seeding. **Discussion:** The malignant potential of excluded biliary segments underscores the need for proactive clinical monitoring. Chronic stasis and subsequent inflammation within isolated ducts act as a potent oncogenic stimulus, necessitating a high index of suspicion when evaluating re-

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mote IBDI cases. Early detection of ductal changes or localized atrophy may allow for intervention before progression to advanced malignancy. **Conclusion:** Lifelong surveillance is essential for patients with previous IBDI, particularly those with surgically excluded segments. Consistent radiologic follow-up is vital to identify early indicators of neoplastic transformation, ultimately improving long-term outcomes in this unique patient population.

3649 | UNDIFFERENTIATED EMBRYONAL SARCOMA OF THE LIVER IN ADULT POST-PARTUM WOMAN

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Introduction: Undifferentiated embryonal sarcoma of the liver (UESL) is the third most common pediatric hepatic malignancy but is exceedingly rare in adults. Tumors are typically large (10-30 cm) and present with non-specific symptoms including fever, anorexia, and abdominal pain. We present the largest known case of UESL, developing during pregnancy with continued growth and resection post-partum. **Case Presentation:** A 35-year-old woman presented 6 weeks post-partum with abdominal pain, early satiety, low-grade fevers, and a firm right upper quadrant mass, reporting she felt "still pregnant." CT showed a hypoattenuating mass (24.0 × 17.8 × 25.6 cm) with internal septations centered in segments 5 and 6, initially thought to be a septated cavernous hemangioma. The tumor grew rapidly with compression of the right portal vein, prompting transfer to our institution for worsening pain. Repeat CT showed a hypodense lobulated mass (34.5 × 26.0 × 32.9 cm) arising from the inferior right lobe, favoring biliary cystadenoma or cystadenocarcinoma. Biopsy showed necrotic tissue with scattered atypical ghost cells concerning for neoplasm; PET/CT showed metabolically active peripheral tumor despite central necrosis. Right portal and right hepatic vein embolization was performed to promote left lobe hypertrophy. She then underwent extended right hepatectomy, cholecystectomy, bile duct resection, and Roux-en-Y hepaticojejunostomy. Pathology confirmed a 50.4 × 44.6 × 16.3 cm UESL weighing 16,002 g, with negative margins except focal parenchymal involvement. Immunohistochemistry supported UESL (desmin+, SMA+, glypican-3+, AE1/AE3+, OSCAR+; SOX10−, myogenin−, MyoD1−). Post-operative left hepatic vein stenosis was treated with endovascular stenting. Multidisciplinary care addressed ascites, bacterial peritonitis, intra-abdominal abscesses, and pulmonary embolism. At 8 weeks, the hepatic remnant showed moderate regeneration with normalization of aminotransferases and synthetic function. **Discussion:** To our knowledge, this is the largest reported UESL and the first in a post-partum woman. The tumor mimicked biliary cystadenoma or cystadenocarcinoma, posing diagnostic and therapeutic challenges. Rapid growth, unprecedented size, and ghost cells on histology raised concern for this rare entity. **Conclusion:** The case underscores the need for management at experienced centers, where shared decision-making between hepatobiliary and transplant teams enabled curative-intent resection.



3650 | RAPIDLY PROGRESSING PRIMARY LUSC WITH A MASSIVE SOLITARY HEPATIC METASTASIS RESPONDING TO NON-SURGICAL CHEMOIMMUNOTHERAPY AND RADIATION: A CASE REPORT

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Introduction: Non-small cell lung cancer (NSCLC), particularly lung squamous cell carcinoma (LUSC), accounts for ~85% of lung cancer cases, with over one-third of patients presenting with distant metastasis at diagnosis. Among metastatic sites, liver involvement is relatively uncommon yet carries an unfavorable prognosis with median survival around 4-10 months

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and rapid clinical deterioration. Hepatic metastases typically present as multiple small lesions; presentation as a solitary massive necrotic mass is clinically rare. Here, we report an atypical case of primary LUSC with a rapidly progressing isolated hepatic mass achieving near-complete radiologic resolution following non-surgical chemoimmunotherapy and adjunct palliative radiation. **Case Presentation:** A 71-year-old female with stage IIIb CKD and a 10 pack-year smoking history presented for follow-up of an incidental 1.3 cm right upper lobe (RUL) pulmonary nodule. Four months later, following new-onset hemoptysis, a repeat CT showed significant progression: the RUL nodule enlarged to 3.1 cm, alongside a newly developed 6.5 × 10.0 × 9.3 cm centrally necrotic mass in the right hepatic lobe. Subsequent FDG-PET confirmed a hypermetabolic RUL mass (3.5 × 3.3 cm), a massive necrotic hepatic lesion (10.0 × 7.1 cm), and an incidental right adrenal lesion. Biopsy of the liver mass confirmed metastatic LUSC. The patient initiated systemic chemoimmunotherapy (carboplatin, paclitaxel, pembrolizumab) with adjunct palliative radiation. PET scans at 6 months showed marked tumor regression. Follow-up at 12 months confirmed sustained, near-complete resolution with minimal residual FDG avidity in the lung and liver. **Discussion:** The rapid tumor evolution in this case highlights the high metastatic potential and aggressive volume expansion characteristic of LUSC. Standard predictive guidelines rely on Volume Doubling Time (VDT) to predict malignancy, noting a 26% diameter increase doubles volume; NELSON trial data shows VDT <400 days carries a 9.9% malignancy risk. LUSC mean VDT is 126+-58 days. Here, the primary nodule nearly tripled in diameter in <180 days (~13-fold volume increase), coinciding with a 10 cm solitary liver metastasis. While isolated hepatic metastases are typically managed with surgical resection and chemotherapy, our patient was successfully treated non-surgically. The hepatic microenvironment ("liver immune sink") upregulates PD-L1 and recruits Tregs, secreting IL-10 and TGF-β to trigger CD8+ T-cell exhaustion and resistance to single-agent ICIs. However, combining chemo-radiation induces tumor cell death and antigen release, overriding local tolerance to synergize with anti-PD-1 therapy. **Conclusion:** For patients with underlying comorbidities, such as Stage IIIb CKD, multimodal chemoimmunotherapy and radiation provide an effective non-invasive alternative for durable disease control.

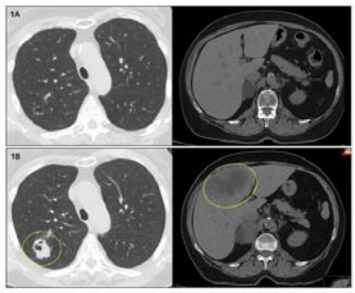


Figure 1. Low dose CT Chest without IV contrast five months prior to initial diagnosis of SCC of the lung demonstrated stable RUL lung mass (1.3cm) and no significant liver mass (1A), compared to repeat CT Chest at time of diagnosis showing a significant increase in size of RUL nodule (3.1cm) and newly visualized mass in the right hepatic lobe (1B).

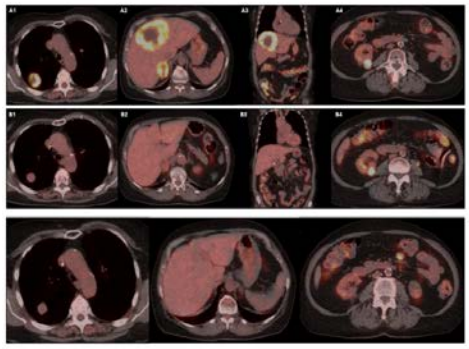


Figure 2. Evolution of tumor burden at initial time of diagnosis (Panel A) compared to six months (Panel B) and one year (Panel C) of treatment as demonstrated on routine positron emission tomography (PET) scans. Areas with high intake of F-18 fluorodeoxyglucose (FDG) appear as bright yellow (10 standardized uptake value (SUV)) compared to areas of low FDG uptake appearing as subdued red (0-5 SUV).

Panel A demonstrates large masses with high FDG uptake on the axial view of the right upper lobe of the lung (A1), right hepatic lobe (axial view (A2), coronal view (A3)), and right adrenal gland (A4). Panel B demonstrates these same regions after six months of treatment with carboplatin, paclitaxel, and pembrolizumab immunotherapy with adjunct palliative radiation therapy. Panel C demonstrates decreased size and low FDG uptake in the RUL of the lung (C1), liver (C2), and right adrenal gland (C3).

3651 | A FATAL PRESENTATION OF SYSTEMIC AL AMYLOIDOSIS: PROGRESSIVE CHOLESTATIC LIVER FAILURE AND DIAGNOSTIC CHALLENGE

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Introduction: Amyloidosis is the deposition of misfolded protein in various organs, leading to organ dysfunction. [1]Hepatic involvement is common on biopsy but is usually clinically silent. [2] AL amyloidosis is the most prevalent form, accounting for about 75% of systemic cases.[3] We present a case of systemic AL amyloidosis presenting with cholestatic liver injury and progression to organ failure. **Case Presentation:** A 65-year-old female who presented to an outside hospital with two months of weakness and jaundice. She had recently taken nitrofurantoin for a UTI. Initial laboratory studies showed cholestatic liver injury (Table 1). MRCP noted hepatomegaly, gallbladder sludge, and no biliary obstruction. ERCP with biliary stent failed to improve the jaundice. Doppler ultrasound ruled out vascular thrombosis. Further chronic liver disease workup was unrevealing (Table 1). A working diagnosis of DILI was

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made based on chronic lobular cholestasis with advanced fibrosis that was noted on the initial liver biopsy read from the outside hospital. However, she was readmitted for persistent jaundice and worsening liver tests (Table 1). Repeat pathological review at our tertiary liver transplant center identified extensive hepatic amyloid deposition. Serum studies revealed elevated IgA, two monoclonal IgA-kappa bands on serum protein electrophoresis, and elevated kappa free light chains.Cardiac MRI showed infiltrative cardiomyopathy, and bone marrow biopsy confirmed a plasma cell neoplasm with Congo red-positive amyloid, confirming multiorgan AL amyloidosis. Despite treatment with daratumumab and dexamethasone, she developed shock, coagulopathy, and multiorgan failure and ultimately died. **Discussion:** Hepatic amyloidosis is often difficult to diagnose due to its subtle presentation, sometimes necessitating liver biopsy. [2] In this case, DILI from nitrofurantoin was initially suspected, while the original biopsy misinterpreted amyloid deposition for fibrosis. However, pathological reevaluation confirmed hepatic amyloidosis. An abnormal plasma cell clone produces the misfolded protein that deposits in the organs, making liver transplantation ineffective, as the underlying clones are located in the bone marrow. [4] In contrast, liver transplantation can be effective in hATTR because the liver is the main source of the transthyretin, and replacing it markedly reduces production of the mutant protein. [5] AA amyloidosis, however, is primarily managed by controlling the underlying inflammatory process to reduce hepatic serum amyloid A production, and liver transplantation may be beneficial only in selected cases where the liver is central to the underlying inflammatory process. [6] **Conclusion:** Consider hepatic amyloidosis in unexplained cholestatic liver injury when routine workup is unrevealing. Biopsy and amyloid typing are key to diagnosis, treatment, and transplant decisions.

		06/07/2026(First admission)	06/18/2026(Readmission)	07/07/2026(Before passing)
Aspartate Aminotransferase (AST)		145	200	422
Alanine Aminotransferase (ALT)		48	75	101
Alkaline Phosphatase (ALP)		397	560	578
Total Bilirubin		8.3	12.1	16.9
Direct Bilirubin		4.4	7.2	9.7
INR		2.1	1.7	4.1
Antinuclear Antibody	Negative			
Anti-Smooth Muscle Antibody	Negative			
liver-kidney microsomal Antibody	Negative			
Anti-Mitochondrial Antibody	Negative			
Hepatitis A/B/C	Negative			
HIV	Negative			
Hemochromatosis DNA	Negative			
PEth	Negative			

Table 1: Laboratory results

Abbreviations:

OSA: Obstructive sleep apnea

MRCP: Magnetic resonance cholangiopancreatography

ERCP: endoscopic retrograde cholangiopancreatography

DILI: drug-induced liver injury

UTI: urinary tract infection

hATTR: hereditary transthyretin amyloidosis

3652 | SUCCESSFUL LIVER TRANSPLANTATION FOR IMMUNE CHECKPOINT INHIBITOR-INDUCED ACUTE LIVER FAILURE: A FIRST-IN-LITERATURE CASE REPORT

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Introduction: Immune checkpoint inhibitor (ICI) hepatitis is usually responsive to medical therapy and progresses to acute liver failure (ALF) in <1% of patients. Liver transplantation (LT) has not been previously considered because of underlying malignancy and acute

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rejection risk. Emerging data in advanced hepatocellular carcinoma (HCC) suggest LT can be performed safely after ICI downstaging when washout exceeds 3 months. Here, we report the first successful LT for ICI-induced ALF. **Case Presentation:** A 58-year-old man with metastatic melanoma of unknown primary presented with jaundice and acute liver injury. He had undergone radical neck dissection for cervical nodal disease and remained on nivolumab for 9 years without toxicity or recurrent disease. His last dose was 6 weeks before presentation. Initial labs showed total bilirubin 15.8 mg/dL, AST 1,299 U/L, ALT 656 U/L, ALP 236 U/L, and INR 1.62. Serologic evaluation was unrevealing; liver biopsy showed moderate-to-severe immune-mediated hepatitis with foci of bridging necrosis. Staging imaging showed no recurrent melanoma. Methylprednisolone 125 mg daily was started, followed by mycophenolate mofetil (MMF) 1,000 mg twice daily. After transient improvement, he deteriorated with obtundation, worsening coagulopathy, and rising lactate and was listed Status 1A. Peak total bilirubin was 43.8 mg/dL, INR 4.40, lactate 9.4 mmol/L, and MELD 45. He underwent deceased-donor LT 8 weeks after nivolumab. To mitigate rejection risk, he received basiliximab induction, high-dose MMF (1500mg BID), tacrolimus (trough 8-12 ng/mL), and corticosteroids. Explant pathology confirmed severe immune-mediated hepatitis with bridging necrosis. At 12 weeks, he has normal transaminases and no rejection. **Discussion:** LT after ICI therapy has been studied mainly in advanced HCC after downstaging. Acute rejection and graft loss occur in approximately 20% and 4% of patients, respectively. Longer ICI washout is associated with lower rejection risk, with approximately 3 months generally recommended before LT. Our patient was unique because LT was required for ICI-induced ALF itself rather than malignancy, after failure of medical therapy and despite recent nivolumab exposure (8 weeks). His prolonged melanoma remission and absence of active disease favored LT despite substantial oncologic and acute rejection risks. **Conclusion:** Recent ICI exposure should not be considered an absolute contraindication to LT in otherwise appropriate patients with irreversible ICI-induced ALF. As ICI use expands across malignancies, transplant teams may increasingly encounter similar patients in whom recent exposure complicates candidacy and post-transplant management. Thorough oncologic assessment, individualized risk-benefit evaluation, augmented immunosuppression, and close rejection surveillance may permit successful salvage transplantation in carefully selected patients.

3653 | LIVER TRANSPLANTATION IN REFRACTORY ACUTE INTERMITTENT PORPHYRIA: WHEN MAXIMAL MEDICAL THERAPY FAILS

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Introduction: Acute intermittent porphyria (AIP) is a rare hepatic porphyria with a prevalence of 5-10 cases per 100,000 and <1% clinical penetrance among heterozygotes. [1] Treatment options include glucose, hemin, and givosiran, with additional therapies targeting triggers and symptoms. [2] Liver transplantation is curative for refractory AIP but is rarely performed, with 38 cases reported to the European Liver Transplant Registry between 2002 and 2019.3 **Case Presentation:** A 42-year-old woman received liver transplantation for refractory AIP, diagnosed at age 33 after presenting with abdominal pain, hypertension, hyponatremia, and dark urine. Urine PBG was 991.8 and ALA 318, with elevated stool porphyrins and a heterozygous HMBS mutation. Flares progressed despite trigger avoidance, carbohydrate supplementation, and weekly prophylactic hemin. Chronic hemin therapy resulted in secondary hemochromatosis requiring phlebotomy. Hormonal therapy was limited by intolerance or flare recurrence, and givosiran was stopped due to minimal clinical benefit. She developed worsening chronic migraines, peripheral sensory neuropathy, and opioid-dependent abdominal pain. Eight years after diagnosis, monthly hospitalizations and severe deconditioning prompted referral for liver transplantation. Hepatic function remained preserved with no evidence of cirrhosis. Given medically refractory AIP with recurrent debilitating attacks despite optimized therapy, an exception request was submitted to the National Liver Review Board, which approved a MELD exception score equivalent to the median MELD at transplant (MMaT). At 3 months post-transplant, she continues to require analgesia for abdominal and neuropathic pain but has not needed readmission for porphyria-related disease. **Discussion:** AIP remains a rare indication for liver transplantation. It has a mean diagnostic delay of 15 years, reflecting its nonspecific presentation.2 Givosiran, approved in 2019, reduces attack frequency, hemin requirement, and chronic pain, potentially further decreasing the need for transplant.4 Delayed transplant referral in severe disease may reflect AIP's rarity and the initial focus on trigger avoidance and medical therapy. Although transplantation prevents further acute attacks, data regarding recovery from established disease-related morbidity is limited. In a post-transplant cohort of 24 patients, neuropathic pain improved, although the timing of assessment was not

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reported.3 In this patient with recent transplantation, porphyria-related hospitalizations have ceased, and quality of life is gradually improving. **Conclusion:** Although timely transplantation may limit progression of irreversible disease-related morbidity, early diagnosis and transplant referral remain challenging in AIP. Recognition of refractory disease despite maximal medical therapy is critical to facilitate timely transplantation and improve long-term outcomes.

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3654 | GRANULICATELLA ADIACENS BACTEREMIA COMPLICATED BY LUMBAR SPONDYLODISCITIS FOLLOWING ELECTIVE ENDOSCOPIC VARICEAL LIGATION: A CASE REPORT

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Introduction: Endoscopic variceal ligation (EVL) is widely used to prevent and treat esophageal variceal bleeding. Bacteremia after elective EVL is uncommon and usually transient. We report a rare case of *Granulicatella adiacens* bacteremia complicated by lumbar spondylodiscitis shortly after EVL in a patient with decompensated cirrhosis. **Case Presentation:** A 60-year-old man with decompensated alcohol-related cirrhosis (Child-Pugh C10), morbid obesity and type 2 diabetes underwent elective EVL after surveillance endoscopy showed interval progression to large grade II-III (F2-F3) esophageal varices without active bleeding. Seven days later, he was admitted with severe lumbar pain and acute gait impairment, without fever or focal neurologic deficit. C-reactive protein was 158 mg/L, leukocyte count 13 × 10^9/L, creatinine 2.20 mg/dL and sodium 126 mmol/L. *Granulicatella adiacens* was recovered from 3 of 4 blood-culture bottles from 2 peripheral sets and was susceptible to ceftriaxone. Lumbar MRI demonstrated L2-L3 spondylodiscitis with extensive circumferential epidural inflammatory involvement and severe canal stenosis. Two transthoracic echocardiograms showed no vegetations. Follow-up blood cultures cleared within 48 hours. Intravenous ceftriaxone was administered for 6.5 weeks, with resolution of pain and inflammation and no neurologic sequelae **Discussion:** *G. adiacens* is a fastidious Gram-positive coccus historically classified among nutritionally variant streptococci and is part of the oral and gastrointestinal microbiota. It is a recognized cause of bacteremia, infective endocarditis and, rarely, vertebral infection. Clinically significant infection after elective EVL is uncommon, and routine antibiotic prophylaxis is not generally recommended. In this case, the close temporal relationship between EVL and subsequent *G. adiacens* bacteremia with spondylodiscitis strongly suggests a procedure-related bacteremic event. **Conclusion:** Severe back pain with systemic inflammation after EVL should prompt blood cultures and spinal imaging. Although rare, clinically significant bacteremia after elective EVL may lead to metastatic infection in vulnerable patients with advanced cirrhosis.

3655 | PERSISTENT COMA AFTER GASTRIC VARICEAL HEMORRHAGE IN ADVANCED ALCOHOL-ASSOCIATED LIVER DISEASE: IDENTIFYING A BENZODIAZEPINE CONTRIBUTION WITH A TITRATED FLUMAZENIL CHALLENGE

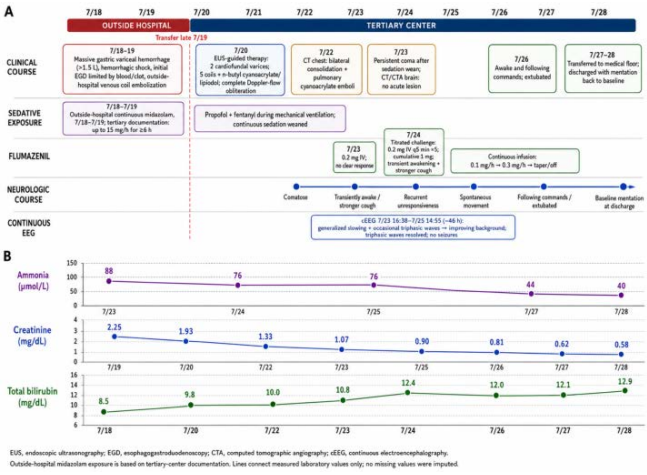
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[1]Westchester Medical Center

Introduction: Persistent coma in patients with advanced liver disease is often attributed to hepatic encephalopathy (HE), but sedative accumulation may be overlooked when hepatic and renal clearance are impaired. We report prolonged coma after massive gastric variceal hemorrhage with a clinically meaningful response to flumazenil. **Case Presentation:** A 43-year-old woman with alcohol use disorder and portal hypertension presented with >1.5 L hematemesis and hemorrhagic shock. Imaging showed large gastric varices and a splenorenal shunt. Initial endoscopy was limited by clot burden; recurrent bleeding required massive transfusion, vasopressors, and outside-hospital venous coil embolization. After transfer, EUS identified two cardiofundal varices arising from separate feeding vessels. Five coils plus n-butyl cyanoacrylate/lipiodol achieved complete Doppler-flow obliteration without recurrent major bleeding. Her course was complicated by pneumonia, hypoxemic respiratory failure, AKI, hyperammonemia, and bilateral pulmonary cyanoacrylate emboli.Referring-hospital

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records documented continuous midazolam over 2 days, reportedly reaching 15 mg/hour for at least 6 hours. Despite sedative discontinuation, she remained comatose with reactive pupils and corneal reflexes but no eye opening or motor response. CT/CTA brain showed no acute lesion. Forty-six hours of continuous EEG showed reactive generalized slowing with triphasic waves that resolved; no seizures occurred. Ammonia peaked at 88 µmol/L and creatinine at ~2.6 mg/dL. An initial 0.2-mg flumazenil dose produced no clear response. The next day, 0.2-mg doses every 5 minutes to a cumulative 1-mg challenge produced transient awakening and improved coughing followed by recurrent unresponsiveness. A flumazenil infusion was started at 0.1 mg/hour, increased to 0.3 mg/hour, then tapered. Over 48 hours she developed spontaneous movement, followed commands, and was extubated without clinical or electrographic seizures. She was subsequently transferred to the medical floor and discharged with mentation back to baseline, expressing excitement about returning to teaching. **Discussion:** The coma was multifactorial, including HE, infection, shock, AKI, and sedative exposure. However, prolonged midazolam exposure, impaired hepatic/renal clearance, and transient improvement after titrated flumazenil supported a clinically important benzodiazepine component. Sustained recovery coincided with renal recovery, infection treatment, and ammonia improvement; flumazenil alone cannot explain recovery. **Conclusion:** In patients with liver disease and persistent coma, medication accumulation should be considered before attributing neurologic failure solely to HE. A carefully selected, monitored flumazenil challenge may identify a reversible benzodiazepine contribution when prolonged exposure and impaired clearance are present.



3656 | RE-DO LIVER TRANSPLANTATION FOR RECURRENT FACTOR VIII DEFICIENCY, 19 YEARS AFTER INITIAL TRANSPLANT

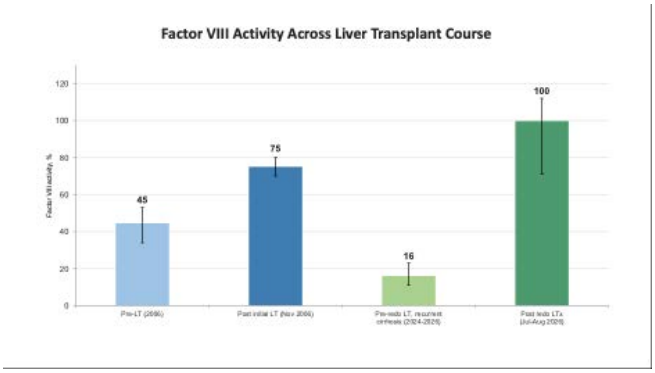
Elina Stoffel[1] Jiyoon Park[1] Rebecca Roediger[2] Benjamin Samstein[2] Anish Sharda[3] Robert S. Brown[2] Cary Caldwell[4]

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Introduction: Patients with Hemophilia A (HemA) who develop cirrhosis from HCV-contaminated transfusions have undergone liver transplantation (LT) with improved Factor VIII (FVIII) production by sinusoidal endothelial cells (SEC) in the donor liver. However, most succumb to recurrent HCV cirrhosis with poor survival. We report the first case of a re-do LT in a HemA patient with successful restoration of FVIII levels, 19 years after initial LT. **Case Presentation:** In 2006, a 23-year-old male underwent LT for HCV cirrhosis and HCC, resulting in improved FVIII levels (75%, from a pre-op 20%). Fibrosis progressed before HCV eradication with direct-acting antivirals, and recurrent FVIII deficiency developed (<20%). By 2023 he developed clinically significant portal hypertension with esophageal varices, massive splenomegaly, and profound thrombocytopenia, and he was listed for re-do LT at a low MELD. He required repeated endoscopic band ligation and TIPS in 2024, reducing HVPG from 22 to 11 mmHg. Post TIPS, the FVIII paradoxically dropped to <10%. On efanesoctocof alfa, standardized trough FVIII was monitored, with no inhibitor detected. Weekly FVIII replacement became necessary. A median MELD at transplant (MMaT) exception was granted for FVIII deficiency conferring mortality not captured by physiologic MELD. In July 2026 he underwent re-do LT (intra-operative blood loss 20L) with perioperative octocog alpha replacement, discontinued on POD4 given stable endogenous FVIII. Mean post-op FVIII was 99.7% (71-112) versus a pre-op mean of 16% (11-23, 2024-26). **Discussion:** Our patient underwent a successful re-do LT for cirrhosis and recurrent FVIII deficiency of the initial allograft, accomplished by appropriate MMaT. The FVIII did not improve after TIPS to our surprise, suggesting SEC dysfunction independent of portal hypertension. Endogenous FVIII normalized after each LT (2006 and 2026), confirming donor SEC as the source of restored production. Without durable alternative to costly weekly replacement therapy (>$20,000 per week), restoration of endogenous FVIII via re-transplant under MMaT exception was the only viable option. **Conclusion:** This is the first reported case of re-do LT for recurrent FVIII deficiency in

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HemA. Improved understanding of SEC-derived FVIII enhances recognition and management of hepatic sequalae in surviving HemA patients.



3657 | DELAYED DIAGNOSIS OF CHRONIC BUDD-CHIARI SYNDROME MASQUERADING AS CIRRHOSIS: A CASE REPORT

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Introduction: Diagnostic delay in chronic Budd-Chiari syndrome (BCS) can span decades, with multiple case series reporting intervals up to 24 years from symptom onset to diagnosis. We report the case of a young male with over 15 years of unexplained hepatosplenomegaly and liver nodularity who was ultimately diagnosed with chronic BCS from intrahepatic inferior vena cava (IVC) and hepatic vein stenosis. **Case Presentation:** A 24-year-old male with Hirschsprung's disease had multiple abdominal surgeries in the first five years of his life. He was evaluated around the age of 8 years for hepatosplenomegaly and nodular liver. Evaluation demonstrated normal hepatic synthetic function and a liver biopsy which was remarkable only for portal fibrosis. At the age of 11 years, he had grade 1 esophageal varices on upper endoscopy but was subsequently lost to follow-up. He was referred to our clinic at the age of 24 years following a motor vehicle collision when computed tomography (CT) reported nodular liver, caudate hypertrophy, a small-caliber IVC, enlarged azygos/hemiazygos veins, and abdominal wall varices. Clinical exam showed engorged bilateral lateral abdominal wall serpiginous veins suspicious for IVC stenosis and right thigh varicose veins. Based on preserved hepatic synthetic function, significant abdominal venous collaterals, caudate lobe hypertrophy, and clinical exam findings, we were highly suspicious for chronic BCS. Venogram showed intrahepatic IVC and hepatic vein stenosis confirming the diagnosis of BCS. There was evidence of suprarenal IVC stenosis and bilateral iliac vein stenosis which explained lower extremity varicose veins. Percutaneous liver biopsy showed no hepatic fibrosis and normal liver parenchyma. Hypercoagulable work-up was negative. Iliac vein and suprarenal IVC angioplasty was performed, but direct intrahepatic portosystemic shunt could not be performed due to the proximity of stenosed intrahepatic IVC to the right atrium. There were multiple intrahepatic venous collaterals and enlarged azygos vein which likely helped decompress the venous system. **Discussion:** Budd-Chiari syndrome (BCS) results from hepatic venous outflow obstruction anywhere from small hepatic veins to the cavoatrial junction. Chronic BCS develops insidiously, causing hepatosplenomegaly, thrombocytopenia, and portal hypertension leading to collateral formation, caudate lobe hypertrophy, and nodular regenerative hyperplasia. Since imaging may show nodular liver, chronic BCS is often initially misdiagnosed as cirrhosis. **Conclusion:** The teaching point in this case is that chronic BCS should be considered when portal hypertension coexists with preserved hepatic synthetic in the setting of caudate lobe hypertrophy and abdominal venous collaterals. Dilated lateral abdominal veins should raise suspicion for IVC stenosis. Prompt recognition will prevent misdiagnosis of cirrhosis.

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3658 | WHEN THE HEART SILENCES THE LIVER: SEVERE ISCHEMIC HEPATOPATHY REVEALING LEFT VENTRICULAR NONCOMPACTION CARDIOMYOPATHY

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Introduction: Ischemic hepatopathy (IH) results from a critical mismatch between hepatic oxygen delivery and demand. In a systematic review, cardiac events preceded 78.2% of cases, while hypotension occurred in only 52.9%, demonstrating that overt shock is not required. Left ventricular noncompaction (LVNC), characterized by excessive trabeculation, can cause HF and arrhythmias. **Case Presentation:** A 73-year-old man with HF, DM, HTN, and CKD3 presented with dyspnea, hypoxemic respiratory failure, and tachycardia. He remained normotensive (SBP 105-131 mmHg). His rhythm was initially interpreted as AF with RVR, prompting amiodarone and beta-blockade; subsequent review favored sinus tachycardia. Initial AST/ALT were 158/211 U/L, creatinine 1.58 mg/dL, and lactate 4.1 mmol/L. Within 24 hours, AST/ALT rose to 2,909/2,714 U/L and later peaked at 4,434/3,954 U/L, with LDH 2,336 U/L, INR 7.87, lactate 5.9 mmol/L, and creatinine 2.22 mg/dL. Newly initiated apixaban was held; he had no encephalopathy or bleeding. Acetaminophen level and viral hepatitis serologies were negative; hepatic Doppler showed no vascular obstruction. TTE demonstrated a moderately dilated LV with apical hypertrabeculation and deep recesses consistent with LVNC, global hypokinesis, LVEF 30%, grade III diastolic dysfunction, elevated filling pressures, and moderate MR/TR. Chest CT showed concordant LV morphology. Normotensive low-output cardiogenic shock complicated by IH and AKI was diagnosed. Dobutamine, IV diuresis, and vasodilators were initiated; NAC and vitamin K were administered. Inotropes were stopped after AF with RVR developed. After hemodynamic optimization and restoration of sinus rhythm, lactate fell to 2.0 mmol/L, creatinine to 1.32 mg/dL, INR to 1.88, and AST/ALT to 206/1,185 U/L. He was discharged stable. **Discussion:** Reduced cardiac output, compounded by elevated filling pressures and hepatic venous congestion, can produce centrilobular hypoxic injury despite preserved systemic BP. In LVNC, impaired systolic function reduces forward flow, while venous congestion limits hepatic oxygen diffusion, creating a "two-hit"

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insult to zone 3 hepatocytes. The abrupt aminotransferase and LDH elevations, lactic acidosis, AKI, severe reversible coagulopathy, negative evaluation for competing hepatic etiologies, and parallel biochemical recovery after restoration of perfusion supported IH. IV amiodarone hepatotoxicity was considered; however, the concurrent multiorgan hypoperfusion and coordinated improvement in hepatic, renal, and metabolic indices with cardiac stabilization favored a predominantly hemodynamic mechanism. **Conclusion:** Normal BP does not exclude clinically significant hypoperfusion. Severe unexplained hepatocellular injury should prompt evaluation for occult low-output cardiac disease, as early hemodynamic correction may reverse hepatic injury and prevent multiorgan dysfunction.

3659 | HEPATITIS A-ASSOCIATED ACUTE LIVER FAILURE REQUIRING LIVER TRANSPLANTATION

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Introduction: Hepatitis A virus (HAV) causes more than 150 million infections annually, ranging from asymptomatic disease to acute liver failure (ALF). Most cases are self-limited. We present a healthy 31-year-old man with HAV-associated ALF requiring liver transplantation. **Case Presentation:** A 31-year-old man without prior liver disease or alcohol use presented with one week of nausea, vomiting, dark urine, poor oral intake, and chills. He denied acetaminophen use. Laboratory studies showed AST 4,195 U/L, ALT 6,905 U/L, alkaline phosphatase 121 U/L, total bilirubin 5.6 mg/dL, INR 12.5, and lactate 11.6 mmol/L. Acetaminophen was undetectable and acute hepatitis B serologies were negative. Additional testing included HSV, HEV, CMV, and EBV serologies, ceruloplasmin, and autoimmune serologies, all of which were negative. Duplex ultrasound showed a mildly echogenic liver with patent hepatic vasculature, no ascites, and normal spleen size. He rapidly developed hypotension and unresponsiveness requiring intubation and ICU care. Arterial pH was 7.39 and ammonia 306 mmol/L. Hepatitis A IgM subsequently returned positive, establishing HAV as the etiology of his hyperacute liver failure. He was urgently transferred to a liver transplant center and underwent successful deceased donor liver transplantation two days later. **Discussion:** HAV is usually self-limited, and many infections are asymptomatic. Although ALF occurs in only 0.015-0.5% of HAV infections, it carries substantial morbidity and mortality. Among patients who develop ALF, approximately 55% recover spontaneously, 35% require transplantation, and 10% die. HAV is primarily transmitted by the fecal-oral route and is associated with poor sanitation, although our patient had no identifiable risk factors. This case demonstrates that HAV can rapidly progress to fulminant hepatic failure in an otherwise healthy young adult. **Conclusion:** HAV is a rare but potentially devastating cause of ALF. Clinicians should recognize the potential for rapid deterioration and promptly transfer patients with HAV-associated ALF to a liver transplant center for evaluation.

3661 | A CURIOUS CASE OF CHOLESTASIS: WHEN COPPER OVERLOAD IS NOT WILSON DISEASE

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Introduction: *HNF1B* variants can cause cholestatic liver disease resembling progressive familial intrahepatic cholestasis (PFIC), including PFIC-8 caused by kinase family member 12 (*KIF12)* loss-of-function variants. We describe chronic cholestasis in a patient with a 17q12 microdeletion involving *HNF1B* initially diagnosed with Wilson disease due to hepatic copper overload. **Case Presentation:** A 32-year-old woman with diabetes and bilateral renal cysts re-established hepatology care for cholestatic-predominant liver injury. She had episodic jaundice since age 8 and was hospitalized at age 25 for jaundice and sepsis from a skin and soft tissue infection, with bilirubin peaking at 13 mg/dL. Evaluation for drug-induced liver injury, viral, autoimmune, PBC, and PSC-related disease was unrevealing. Imaging showed splenomegaly and possible biliary irregularities. Liver biopsy demonstrated ductopenia without features specific for autoimmune, PBC, or PSC. Hepatic copper was elevated to 1600 µg/g, prompting concern for Wilson disease despite elevated ceruloplasmin, elevated alkaline phosphatase, and negative *ATP7B* testing. A 24-hour urine copper was 241 µg, and Trientine was initiated. Genetic testing identified a 17q12 microdeletion encompassing the entire *HNF1B* gene. Ursodiol provided incomplete relief of pruritus, so Maralixibat was initiated, with improvement in pruritus and fatigue at 2 month follow-up. **Discussion:** *HNF1B* variants cause a spectrum of liver disease, from neonatal jaundice to adult-onset cholestasis. Reduced intrahepatic bile ducts and cilia on bile duct epithelial cells may contribute to cholestasis and fibrosis. *KIF12*, a direct *HNF1B* target, is implicated in *PFIC-8*, suggesting a shared pathway for cholestasis involving impaired ciliary function. Our patient's 17q12 microdeletion likely explains her milder phenotype. *HNF1B*-associated disease is a multisys-

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tem disorder characterized by renal abnormalities, including renal cysts, and maturity onset diabetes of the young (MODY), both of which were present in our patient.Wilson disease was initially suspected due to copper overload; however, cholestasis can cause secondary hepatic copper accumulation and elevated urinary copper. High ALP and ceruloplasmin argued against Wilson disease, prompting alternative evaluation. Chelation therapy was discontinued, and an ileal bile acid transporter inhibitor was initiated for pruritus, with marked symptomatic improvement. **Conclusion:** This case illustrates a rare etiology of chronic cholestasis likely attributed to an 17q12 microdeletion. Secondary copper accumulation may mimic Wilson disease. PFIC subtypes should be considered in the differential diagnosis of unexplained chronic cholestasis, particularly in patients with persistent cholestatic pruritus despite conventional therapies, and genetic testing should be considered to facilitate diagnosis and guide targeted treatment.

Table 1. Laboratory Data

Lab Parameters Reference Range	ALP (U/L) 37 - 113	AST (U/L) 13 - 62	ALT (U/L) 8 - 70	Total bilirubin (mg/dL) 0.2 - 1.2	Direct bilirubin (mg/dL) <=0.3	GGT (U/L) 7 - 68	Ceruloplasmin (mg/dL) 14 - 45	Copper Plasma (ug/dL) 80 - 155	Copper, 24-Hour Urine (mcg/24 h)
9/10/19	344	62	57	13.9	10.8	231	49	-	-
9/23/19	422	72	64	5.7	4.2	-	-	-	-
1/27/20	271	129	208	4.1	-	-	-	-	-
2/20/20	327	179	234	3.5	2.1	-	-	-	-
5/12/20 [a]	220	86	136	2.2	1.5	-	-	-	-
6/18/20	219	104	205	1.9	-	-	-	-	-
7/7/20	188	88	172	2.2	-	-	-	-	-
11/13/20 [b]	227	106	200	1.9	-	-	-	-	-
11/16/20	-	-	-	-	-	-	-	-	241
12/21/20	274	146	244	1.6	-	-	-	-	-
2/17/21	263	110	220	1.6	-	-	-	-	-
4/13/21	240	93	154	1.6	-	-	-	-	-
7/26/21	316	60	97	1.6	0.5	-	34	172.8	-
10/4/21	564	160	252	2.4	0.5	-	-	-	-
1/25/22	361	60	84	1.5	-	-	-	-	-
3/31/23	376	58	84	1.5	0.7	-	-	-	-
4/23/24	376	125	169	2.0	-		33	-	-
12/15/25	569	68	134	2.4	0.9	-	38	-	-
3/16/26	349	56	91	2.0	-	163	35	178.3	225
7/10/26	397	80	125	2.7	-	-	27	-	-

ALP: Alkaline phosphatase, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, GGT: Gamma-glutamyl transferase
a – Ursodiol initiation
b – Trientine initiation

Figure 1. Extended cholestasis genetic panel

MOLECULAR GENETICS REPORT:
Cholestasis Panel

SUMMARY OF RESULTS: **Positive**

Copy Number Variant:

Genomic Coordinates	Type	Size	Inheritance	Zygosity	Interpretation
chr17:34,746,412-36,285,884 (GRCh37/hg19)	Deletion	~1.5 Mb	Not Determined	Heterozygous	PATHOGENIC

3662 | WHEN A TICK TARGETS THE LIVER: EHRLICHIOSIS-TRIGGERED HLH PRESENTING WITH MENINGOENCEPHALITIS AND ACUTE LIVER INJURY

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Introduction: Human monocytic ehrlichiosis (HME), caused by Ehrlichia chaffeensis, commonly produces cytopenias and liver biochemical abnormalities, reported in 68.1% of cases. Rarely, HME triggers secondary hemophagocytic lymphohistiocytosis (HLH), a fulminant hyperinflammatory syndrome associated with multiorgan failure and 17.6% mortality. When neurologic and hepatic manifestations predominate, HME may mimic viral meningoencephalitis or primary acute liver disease, delaying potentially lifesaving doxycycline. **Case Presentation:** A 63-year-old woman with HTN, bipolar I disorder, Lewy body dementia, and MASLD presented in May with fever, agitation, and encephalopathy after gardening. No rash or known tick bite was documented. Medications included quetiapine, memantine, escitalopram, trazodone, and diazepam. Labs showed Plt 50 K/µL, AST 420 U/L, ALT 133 U/L, ALP 327 U/L, Tbili 2.4 mg/dL, and INR 2.11. CSF showed 57 leukocytes/µL (76% lymphocytes) and protein 97 mg/dL; cultures and a multiplex meningoencephalitis panel were negative. Viral meningoencephalitis was favored. She developed leukopenia, thrombocytopenia, and liver injury (AST 1,040 U/L, ALT 234 U/L, ALP 606 U/L, Tbili 3.5 mg/dL). Toxic, acute viral, autoimmune, and obstructive etiologies were excluded. The syndromic pattern prompted doxycycline and NAC. She then developed hypoglycemia, shock, and lactic acidosis. Ferritin >100,000 ng/mL, TG 349 mg/dL, fibrinogen <60 mg/dL, and hepatosplenomegaly yielded an HScore indicating 99% probability of HLH. Dexamethasone and etoposide were initiated. Despite ventilation, vasopressors, transfusions, and attempted CRRT, she developed DIC, anuric AKI, and refractory shock. AST exceeded 7,000 U/L, consistent with superimposed ischemic hepatitis, and she died on day 6. E. chaffeensis IgM was positive with IgG >1:1024. **Discussion:** Neurologic ehrlichiosis may produce inflammatory CSF, obscuring diagnosis. Experimental models identify the liver as an early target, with infection of mononuclear phagocytes, endothelial cells, and hepatocytes; HLH then magnifies immune-mediated injury. Her biphasic course distinguished antecedent ehrlichial/HLH injury from terminal

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ischemic hepatitis. In a 46-patient PCR-confirmed Ehrlichia/Anaplasma cohort, delayed doxycycline was associated with ICU transfer (39.3% vs 0%) and ventilation (28.6% vs 0%). Adult HLH guidance supports prompt trigger-directed and immunomodulatory treatment during evolving organ dysfunction. **Conclusion:** Fever with cytopenias, encephalopathy, and liver injury in an endemic setting should trigger immediate doxycycline, even without rash or recalled tick exposure. Extreme ferritin and falling fibrinogen should prompt urgent HLH evaluation before shock becomes irreversible.

3663 | CHRONIC HEPATITIS E PRESENTING AS PROGRESSIVE HEPATITIS AND CIRRHOSIS IN A KIDNEY TRANSPLANT RECIPIENT: AN OFTEN-OVERLOOKED CAUSE OF LIVER DISEASE IN THE UNITED STATES

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Introduction: Hepatitis E virus (HEV) is an increasingly recognized cause of chronic hepatitis in immunosuppressed patients. Traditionally associated with endemic regions, HEV is increasingly recognized in developed countries. Solid organ transplant recipients are at risk for chronic infection, fibrosis, and cirrhosis. We report a kidney transplant recipient with five years of unexplained hepatitis ultimately diagnosed with chronic HEV infection. **Case Presentation:** A 49-year-old Vietnamese woman underwent kidney transplantation in September 2018 for end-stage renal disease secondary to lupus nephritis and was maintained on tacrolimus, mycophenolate mofetil, and prednisone. Laboratory testing in October 2018 demonstrated elevated aminotransferases with preserved synthetic function. Evaluation including hepatitis B and C serologies, iron studies, autoimmune testing, and abdominal ultrasound was unrevealing, and liver enzymes normalized by year-end.In May 2020, she developed recurrent liver enzyme elevations that persisted despite discontinuation of supplements and other potential hepatotoxic agents. FibroScan in 2022 showed no significant fibrosis or steatosis. Liver biopsy demonstrated active hepatitis with portal and lobular inflammation, interpreted as possible drug-induced liver injury.Given persistent mild aminotransferase elevations (<2× ULN), FibroScan performed in 2025 revealed liver stiffness of 10 kPa (F3 fibrosis). Repeat liver biopsy demonstrated active hepatitis with spotty necrosis and cirrhosis with nodularity. She was referred to Mayo Clinic, where positive HEV IgG and IgM antibodies and detectable HEV RNA (140,000 IU/mL) confirmed chronic hepatitis E. Ribavirin therapy and immunosuppression reduction were initiated. HEV RNA became undetectable after three months and remained so throughout treatment. Therapy was discontinued after seven months with sustained virologic response and preserved hepatic function. **Discussion:** Chronic HEV infection is an often-overlooked cause of liver injury in immunosuppressed patients. Transplant recipients may fail to clear the virus and rapidly progress to advanced fibrosis or cirrhosis. Diagnosis is often delayed because aminotransferase elevations are mild and histologic findings are nonspecific.This case highlights how chronic HEV can mimic drug-induced liver injury or autoimmune hepatitis, resulting in years of diagnostic uncertainty and progressive fibrosis. Recognition of HEV led to antiviral therapy, immunosuppression reduction, and viral clearance. **Conclusion:** Chronic hepatitis E should be considered in immunosuppressed patients with unexplained aminotransferase elevations. Increased awareness is needed as HEV emerges as a cause of chronic liver disease in the US, particularly among transplant recipients and those with epidemiologic risk factors. Early HEV RNA testing may prevent progression to irreversible liver disease.

3664 | HERPES SIMPLEX VIRUS TYPE 2 HEPATITIS IN AN IMMUNOCOMPETENT WOMAN: EARLY RECOGNITION BEFORE ACUTE LIVER FAILURE

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Introduction: Herpes simplex virus (HSV) hepatitis is a rare but potentially fatal manifestation of disseminated HSV infection, primarily described in immunocompromised patients and during pregnancy, but occasionally in immunocompetent individuals. Early recognition is challenging because manifestations are nonspecific and hepatitis is often anicteric despite marked hepatocellular injury. Delayed diagnosis may result in progressive acute liver failure. We report HSV-2 hepatitis in an immunocompetent woman diagnosed and treated before development of hepatic synthetic dysfunction. **Case Presentation:** A 30-year-old immunocompetent woman presented with fever, progressive abdominal pain, nausea, and abdominal distension. Initial evaluation demonstrated leukopenia and mild aminotransferase elevation, followed by progressive hepatocellular injury, with AST 642 U/L, ALT 523 U/L, and

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LDH 969 U/L. Despite biochemical progression, total bilirubin remained 0.20 mg/dL and INR 1.07.Because of worsening abdominal pain, pelvic inflammatory disease was initially suspected. Computed tomography demonstrated free intraperitoneal fluid, prompting diagnostic laparoscopy, which unexpectedly revealed multiple irregular whitish hepatic lesions. Liver biopsy and peritoneal fluid were obtained. Preliminary histopathology demonstrated multifocal hepatic necrosis with viral inclusions suggestive of HSV, while peritoneal fluid PCR detected HSV-2. Intravenous acyclovir 600 mg every 8 hours was initiated. Definitive histopathology confirmed multifocal necrotizing hepatitis with areas positive for herpesvirus (Figure 1). Within three days, AST decreased to 84 U/L and ALT to 208 U/L. The patient remained without hepatic synthetic dysfunction, transitioned to oral antiviral therapy, and was discharged in stable condition. **Discussion:** HSV hepatitis is diagnostically challenging because fever, abdominal symptoms, leukopenia, and aminotransferase elevation are nonspecific, and mucocutaneous herpetic lesions may be absent. In this case, preserved bilirubin and INR and an abdominal presentation suggestive of a gynecologic process obscured the diagnosis. Unexpected hepatic lesions at laparoscopy prompted tissue and molecular testing, enabling rapid HSV-2 identification and antiviral treatment. When HSV hepatitis is suspected, PCR testing and empiric acyclovir should not be delayed while awaiting histopathologic confirmation. **Conclusion:** HSV hepatitis should be considered in unexplained progressive anicteric hepatocellular injury, even in immunocompetent patients without mucocutaneous lesions. Prompt HSV testing and early acyclovir before hepatic synthetic dysfunction may prevent progression to acute liver failure and improve outcome.

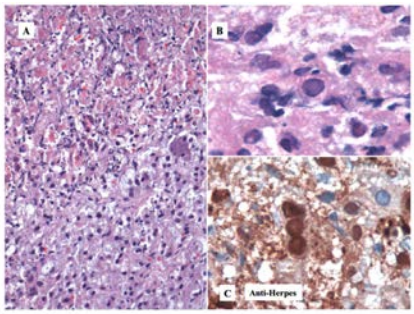


Fig 1. Subsequent histopathological examination of the liver biopsy. (A) Acute and chronic hepatitis with multifocal necrosis, with viable tissue located at the base. (B) Hepatocytes show eosinophilic intranuclear inclusions surrounded by a clear halo, nuclear enlargement, chromatin margination, and occasional multinucleation (Cowdry bodies). (C)Immunohistochemistry with anit-herpes antibodies shows strong nuclear positivity.

3665 | HEPATIC INJURY IN DISSEMINATED HISTOPLASMOSIS IN AN IMMUNOCOMPETENT PATIENT

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Introduction: Disseminated histoplasmosis (DH) is an infection caused by the dimorphic fungus, *Histoplasma capsulatum,* classically seen in immunocompromised populations. While all organs may be clinically involved, DH commonly seeds the reticuloendothelial organs, including the liver, via the hematogenous route. Rare cases of DH have been reported in immunocompetent patients, though its biochemical profile is poorly characterized. **Case Presentation:** A 54-year-old male with a history of right neck mass presented with generalized weakness, diffuse maculopapular rash, and a week of jaundice. He was treated with a one-week course of amoxicillin/clavulanic acid (Augmentin) prior to presentation. Initial labs showed an aspartate aminotransferase (AST) of 303 U/L, alanine aminotransferase (ALT) of 539 U/L, alkaline phosphatase (ALP) of 149 U/L, total bilirubin (TB) of 9.8 mg/dL, and direct bilirubin (DB) of 5.7 mg/dL. Initially, elevated liver tests were attributed to drug-induced liver injury (DILI) secondary to Augmentin. On discontinuation of Augmentin, livers tests improved (AST 65 U/L, ALT 124 U/L, ALP 66 U/L), however, TB paradoxically rose (TB 14.8 mg/dL, DB9.2 mg/dL). Further workup was positive for Epstein-Barr virus (EBV) DNA (1,180 copies/mL) and cytomegalovirus (CMV) DNA (73 copies/mL). The remainder of the infectious and autoimmune workup was unremarkable. Contrast-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) of the abdomen revealed scattered hepatic cysts, splenomegaly with multiple ill-defined masses up to 4 cm, and bilateral adrenal masses. Subsequent liver biopsy on hospital day 11 was complicated by hemorrhagic shock requiring two hepatic artery embolizations. TB progressively rose to 33.7 mg/dL with a direct component of 19.4 mg/dL on day 21. Biopsy showed lymphohistiocytic inflammation with intracellular yeast forms. Grocott-Gomori methanamine silver (GMS) and periodic acid-Schiff (PAS) stains were positive, consistent with hepatic histoplasmosis. Histoplasmosis urine antigen returned positive at 9.6 Ng/mL. The patient was started on liposomal amphotericin B with improvement in clinical status and downtrend in bilirubin. **Discussion:** Biochemical profiles for hepatic histoplasmosis are poorly characterized in current literature. Our patient was found to have disseminated histoplasmosis without pulmonary

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involvement with superimposed DILI secondary to Augmentin. His initial hepatocellular pattern of liver tests in the setting of DILI and immunocompetent status served as a red herring resulting in a delayed diagnosis of DH. **Conclusion:** DH can occur in immunocompetent patients without pulmonary involvement. It should still be considered a differential diagnosis despite immunocompetent status. While hepatic involvement in DH is well recognized, its pattern of hepatic injury is not well-characterized, leading to delayed diagnoses of DH.

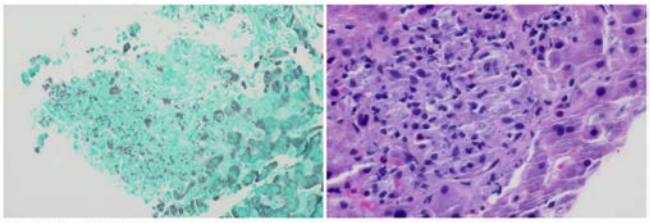


Figure 1A. Grocott–Gömöri's methenamine silver (GMS) stain Figure 1B. Periodic acid–Schiff (PAS) stain

3666 | HIDDEN IN THE BIOPSY: INCIDENTAL CMV ESOPHAGOGASTRITIS WITH REACTIVE HEPATITIS

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Introduction: Cytomegalovirus (CMV) infection has a broad clinical spectrum that varies with host immune status.1 CMV commonly causes gastrointestinal disease in immunocompromised patients but is generally asymptomatic or presents as a mononucleosis-like syndrome in immunocompetent hosts.1-2 Although less common, severe tissue-invasive disease can still occur in the absence of immunosuppression.2 We present a case of biopsy-proven CMV gastritis in an immunocompetent patient with minimal viremia and concurrent hepatitis without direct hepatic CMV infection. **Case Presentation:** A 69-year-old male was referred to hepatology for newly elevated liver chemistries in the setting of fatigue, nausea, and unintentional 25-pound weight loss. Laboratory evaluation showed mixed hepatocellular and cholestatic liver injury with negative acute viral hepatitis serologies. MRI/MRCP revealed gastroesophageal junction thickening with adjacent paraesophageal lymphadenopathy. Upper endoscopy showed a normal-appearing stomach and duodenum; however, gastric cardia biopsies unexpectedly revealed CMV inclusions confirmed by immunohistochemistry. Liver biopsy showed hepatocellular cholestasis with focal hepatocyte dropout without fibrosis, consistent with resolving acute hepatitis. CMV and Epstein-Barr virus stains were negative. Serum CMV DNA was detected at very low levels on two occasions, HIV testing was negative, and leukocyte counts were normal. There was no other evidence of immunodeficiency. He was treated with valganciclovir with resolution of symptoms, stabilization of weight, and normalization of liver chemistries. Repeat CMV DNA was undetectable, and follow-up gastric biopsies were negative for CMV. **Discussion:** This case highlights two major diagnostic pearls. First, low-level CMV viremia does not exclude clinically significant tissue-invasive disease.3 Second, reactive CMV-associated liver injury may occur without histologic evidence of direct hepatic invasion.4 Despite minimal viremia and normal-appearing gastric mucosa, this patient had biopsy-confirmed CMV gastritis. His concurrent acute hepatitis without hepatic CMV suggested a reactive process rather than direct viral cytopathic injury. His improvement in symptoms and liver chemistries with viral clearance following antiviral therapy further supports an association with systemic CMV, although direct causality cannot be established. It is important to note that reliance on serum CMV DNA alone may have precluded further workup and underestimated the extent of disease in this case. **Conclusion:** CMV infection should be considered in immunocompetent patients with otherwise unexplained liver injury and systemic symptoms, even when viremia is minimal. This case highlights that serum viral burden may not accurately reflect tissue-invasive disease, and that CMV-associated hepatic injury may occur without histologic evidence of direct hepatic infection.

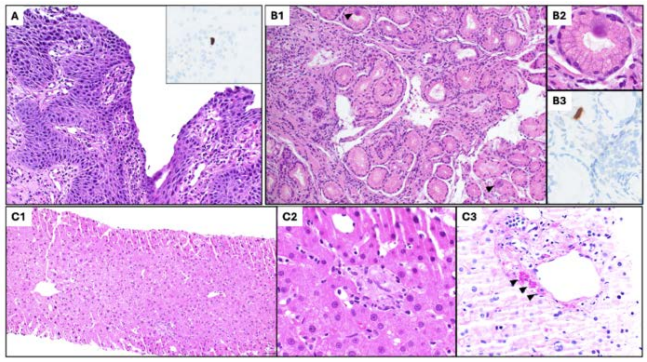


Figure 1: The sampled esophageal mucosa (**A**) appeared reactive; convincing cytopathic effect was not identified on H&E, though CMV immunostain on a deeper level revealed a rare focus of reactivity suspicious for an inclusion (**inset**). No fungal forms were seen on GMS. **A.** Rare epithelial cells in the gastric cardia exhibited cytopathic effect (**B1**, arrowheads), including cytomegalic change and classic "owl-eye" intranuclear inclusions, best appreciated at higher magnification (**B2**). CMV immunostain (**B3**) was confirmatory. *H. pylori* immunostain was negative. Liver needle biopsy demonstrated a small focus of lobular lymphohistiocytic inflammation (**C1**) as well as occasional debris-laden macrophages within the portal tracts, best appreciated on PAS-diastase stain (**C3**, representatively designated by arrowheads); CMV immunostain and EBER in situ hybridization (ISH) were negative. There was no steatosis. The findings, while nonspecific, were compatible with a resolving mild acute hepatitis.

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3667 | BEYOND GAVE: AMPULLARY BLEEDING PRESENTING AS HEMATOCHEZIA IN RECURRENT CHOLANGIOCARCINOMA

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Introduction: Hematochezia usually prompts evaluation for a lower gastrointestinal source; however, brisk upper gastrointestinal bleeding may present similarly. Gastric antral vascular ectasia (GAVE) causes up to 4% of nonvariceal upper gastrointestinal bleeding, whereas hemosuccus pancreaticus occurs in approximately 1 in 1,500 upper gastrointestinal bleeds. Although the incidence of hemobilia is not well defined, approximately two-thirds of contemporary cases are iatrogenic. We describe recurrent hematochezia in a patient with several competing bleeding sources **Case Presentation:** An 84-year-old man with recurrent intrahepatic cholangiocarcinoma treated with chemotherapy and radioembolization, an internal biliary stent and percutaneous transhepatic drain, GAVE treated with argon plasma coagulation, and atrial fibrillation on rivaroxaban presented with hypotension. He developed two large-volume episodes of hematochezia, hemorrhagic shock requiring vasopressors, with a hemoglobin of 6.8 g/dL requiring multiple packed red blood cell transfusions. Computed tomography angiography demonstrated a small hyperdense focus consistent with clot in the second portion of the duodenum without active extravasation. Esophagogastroduodenoscopy revealed no active bleeding, and previously treated GAVE showed no evidence of recurrent hemorrhage. Colonoscopy was deferred because of hemodynamic instability and inability to tolerate bowel preparation. Capsule endoscopy from a prior admission in 2025 had documented active bleeding from the major papilla. Rivaroxaban was withheld. As bleeding ceased and no active target was identified, embolization was deferred, and his hemoglobin stabilized. **Discussion:** Known GAVE and the presentation with hematochezia created dual diagnostic anchors. However, prior capsule endoscopy had documented active papillary bleeding, and the current CTA demonstrated a clot within the second portion of the duodenum, anatomically adjacent to the major papilla, supporting recurrent papillary bleeding as the likely source despite the absence of active extravasation. The differential included hemobilia and hemosuccus pancreaticus. The absence of pancreatitis or other pancreatic pathology favored hemobilia, potentially resulting from malignant vascular-biliary erosion related to recurrent cholangiocarcinoma or iatrogenic injury from Y-90 therapy, biliary stenting, sphincterotomy, or percutaneous drainage. Rivaroxaban likely amplified the hemorrhage, while its intermittent nature explained the negative endoscopy. **Conclusion:** In patients with hepatobiliary malignancy or biliary instrumentation, recurrent hematochezia should not be assumed to be lower gastrointestinal or attributed solely to a previously diagnosed lesion. Documented papillary bleeding is a critical localization clue and should prompt differentiation between hemobilia and hemosuccus pancreaticus.



3668 | GASTRIC ADENOCARCINOMA WITH CONCOMITANT HYPERBILIRUBINEMIA RAISING SUSPICION FOR HILAR CHOLANGIOCARCINOMA: A RARE DIAGNOSTIC DILEMMA

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Introduction: Gastric adenocarcinoma is the most common and aggressive subtype of gastric cancer, accounting for over 90% of cases. The poorly differentiated histology is particularly prevalent and carries a considerably poor prognosis. Obstructive jaundice is an uncommon presentation and biliary imaging may show circumferential hilar bile duct stenosis visually indistinguishable from hilar cholangiocarcinoma. We report this rare and diagnostically challenging presentation. **Case Presentation:** A 67-year-old woman with a history of hyperlipidemia and obesity presented to the ED

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with epigastric pain for 4 weeks, associated with bloating, poor appetite, jaundice, dark urine, generalized body itching but no weight loss. Initial laboratory results showed WBC 5.3, hemoglobin 10.6, platelets 351, total bilirubin 7.6, direct bilirubin 5.5, ALT 306, AST 240, ALK 676. CEA and CA 19-9 were 380 and 518, respectively, but alpha fetoprotein was normal. CT abdomen demonstrated dilatation of intrahepatic bile ducts and common bile duct, a distended gallbladder with circumferential wall thickening coinciding with adjacent gastric antrum wall thickening, omental thickening, and abdominopelvic ascites. EGD and diagnostic paracentesis revealed a 10 mm cratered non-bleeding gastric ulcer with surrounding edema and erythema. MRCP showed a stricture at the hilum of the right and left hepatic ducts, likely from a hilar tumor.Histology revealed a poorly differentiated gastric adenocarcinoma against a background of H. pylori-associated chronic gastritis. While ascitic fluid cytology favored metastatic adenocarcinoma, endoluminal brush biopsy showed unspecified malignant cells. Although improving liver enzyme levels were observed, the patient progressively worsened, developing multiorgan failure with hypoxia and renal impairment. Her clinical condition remained too unstable to consider definitive chemotherapeutic or surgical interventions. In light of her advanced cancer, comfort and end-of-life care was initiated, and she died two days later. Her family declined an autopsy. **Discussion:** Gastric adenocarcinoma remains an aggressive cancer often notorious for poor outcomes, especially when diagnosed late in the disease stage. Diagnostic dilemmas exist when poorly differentiated metastatic gastric adenocarcinomas present with concomitant features of hilar cholangiocarcinoma including jaundice, pruritus, hyperbilirubinemia, hyperbilirubinuria, and transaminitis. In these circumstances, it is important to rule out clinical suspicion for a synchronous hilar cholangiocarcinoma, as therapeutic direction often hinges on diagnostic accuracy. **Conclusion:** This case highlights how these distinctive patterns in late-stage clinical presentation may hinder histology with immunohistochemical marker confirmation, and prove difficult to distinguish between an advanced gastric adenocarcinoma or a co-occurring Klatskin tumor.

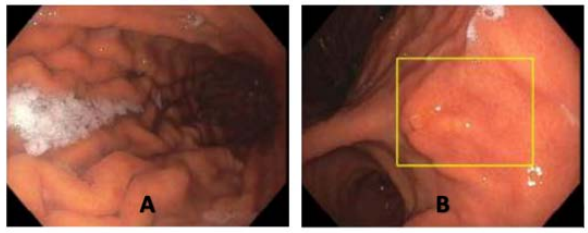


Figure 1. Esophagogastroduodenoscopy (EGD) images demonstrating (A) mild inflammation of the gastric body with evidence of edema and erythema of gastric rugae, and (B) a 10 mm non-bleeding, non-obstructing cratered gastric ulcer (yellow box) with a clean base located at the gastric antrum, with associated mild surrounding edema and erythema. Using cold forceps, biopsy was collected for histology and immunohistochemistry.



Figure 2. Biliary imaging demonstrating (A) MRCP revealing a distended gallbladder with large calculi measuring about 2.4 cm x 1.7 cm and 1.8 cm x 1.4 cm. Right and left intrahepatic duct dilatation with small caliber of the right and left hepatic ducts at the biliary hilum (yellow arrow) is concerning for wall thickening and the presence of a hilar tumor. (B) Interventional radiology-guided external percutaneous biliary Fr 8 drainage to relieve biliary sludge, followed by endoluminal brush biopsy of the common bile duct.

3669 | AN AGGRESSIVE FIBROLAMELLAR CARCINOMA WITH EXTENSIVE PORTAL TUMOR THROMBOSIS MIMICKING HEPATOBILIARY INFECTION

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Introduction: Fibrolamellar carcinoma (FLC) is a rare variant of hepatocellular carcinoma, accounting for <1% of primary liver tumors. It mainly affects adolescents and young adults without chronic liver disease and usually with normal alpha-fetoprotein (AFP). We report a case mimicking cholangitic abscesses. **Case Presentation:** An 18-year-old man with no history of liver disease, alcohol use, or viral hepatitis presented with 7 days of severe right upper quadrant pain radiating to the ipsilateral lumbar region, nausea, vomiting, diaphoresis, and dizziness, with an unintentional 20-kg weight loss during the preceding month. He was afebrile, without jaundice or peritoneal signs. Laboratory tests showed hemoglobin 12 g/dL, leukocytes 12×10^9/L, AST 70 U/L, ALT 96 U/L, alkaline phosphatase 651 U/L, and total bilirubin 0.9 mg/dL. Contrast-enhanced CT demonstrated multiple hypodense

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hepatic lesions, predominantly in the right lobe, with portal and superior mesenteric vein thrombosis and cavernous transformation. Findings were initially interpreted as multiple cholangitic abscesses, and broad-spectrum antibiotics were started. After 6 days without improvement, triphasic CT revealed a heterogeneous solid right-lobe mass with arterial enhancement, multiple satellite lesions, hilar lymphadenopathy, and tumor thrombus extending into both portal branches and the superior mesenteric vein. AFP was normal (5.4 ng/mL). Liver biopsy confirmed FLC, with immunohistochemistry positive for arginase-1, cytokeratin 7, and CD68. Because of bilobar disease and extensive vascular invasion, including an approximately 14-cm portal tumor thrombus, the tumor was deemed unresectable and unsuitable for locoregional therapy. Within 3 months, the patient developed ascites and large esophageal varices requiring endoscopic band ligation. **Discussion:** Fibrolamellar carcinoma (FLC) predominantly affects young patients without chronic liver disease and often with normal AFP. In this case, multiple hypodense lesions mimicked cholangitic abscesses, prompting empiric antibiotics. Persistent symptoms led to repeat multiphasic imaging, revealing arterial enhancement, satellite lesions, and extensive tumor thrombus involving both portal branches and the superior mesenteric vein. Histopathology confirmed FLC. This case highlights how an infectious-appearing presentation may delay diagnosis of aggressive FLC. **Conclusion:** FLC should be considered in young patients with atypical hepatic lesions and normal AFP, especially when presumed hepatobiliary infection does not respond to antibiotics. Early reassessment with multiphasic imaging and histopathology may reduce diagnostic delay.

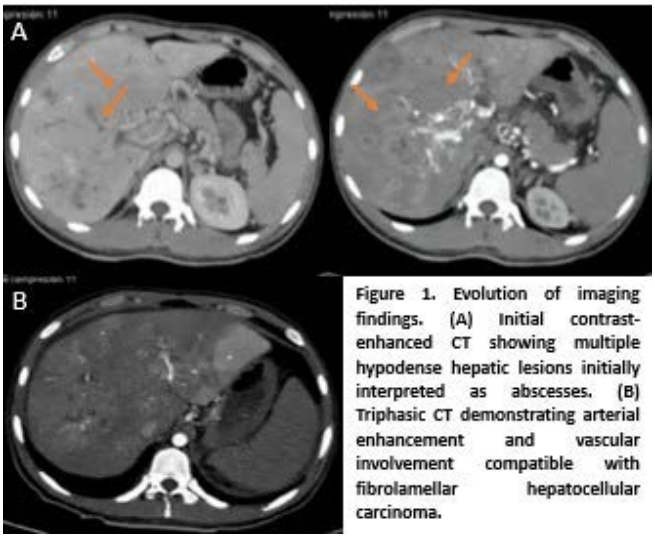


Figure 1. Evolution of imaging findings. (A) Initial contrast-enhanced CT showing multiple hypodense hepatic lesions initially interpreted as abscesses. (B) Triphasic CT demonstrating arterial enhancement and vascular involvement compatible with fibrolamellar hepatocellular carcinoma.

3670 | LATENT AMIODARONE-ASSOCIATED HEPATOTOXICITY WITH MALLORY-DENK BODIES IN THE ABSENCE OF STEATOSIS

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Introduction: Amiodarone, a class III antiarrhythmic used in the management of ventricular arrhythmias, is an established cause of clinically apparent liver injury. We present a case of amiodarone-associated liver injury (AILI) with evolving cirrhosis in the absence of steatosis complicated by clinically significant cholestatic injury, portal hypertension, and recurrent hepatic hydrothorax. **Case Presentation:** A 74-year-old male with ischemic cardiomyopathy, CABG, ventricular tachycardia status post ICD/ablation, CKD G3a, HFimpEF presented with diarrhea and dyspnea. Labs revealed AST 240 U/L, ALT 80 U/L, ALP 125 U/L, and total bilirubin 1.1 mg/dL with progressive elevation in the preceding 3 months. CT abdomen pelvis with contrast showed large right pleural effusion, trace ascites, and mild liver hyperdensity and no biliary duct dilatation. Pleural studies were transudative. Right heart catheterization showed no elevated right sided pressures. Workup was unrevealing with negative hepatitis serologies, ASMA, Anti-LKM, IgG, IgM, and PEth with no A1AT or ceruloplasmin deficiency. The patient was on daily 400mg amiodarone for the preceding15 months with previously normal liver enzymes. Liver biopsy demonstrated prominent periportal ballooning degeneration, abundant Mallory hyaline, and focal neutrophilic satellitosis without steatosis. Trichrome staining highlighted mild pericellular and periportal fibrosis and early nodule formation. EUS revealed a portosystemic pressure gradient of 15mmHg with liver stiffness measuring 15kPa via elastography. Amiodarone was discontinued with subsequent normalization in liver tests. However, the patient continued to have recurrent ascites and pleural effusions and ultimately transitioned to hospice care. **Discussion:** Our patient developed mild transaminitis several months after starting amiodarone, with peak elevations over a year later after approximate cumulative exposure of 180g. Characteristic histopathology findings, along with negative PEth, exclusion of alternate etiologies, lab improvement after discontinuation, and a RUCAM score of 7 suggest probable AILI. Despite mild aminotransferase elevations, our patient developed progressive fibrosis with clinically significant portal hypertension, recurrent ascites, and hepatic hydrothorax requiring repeated hospitalizations. **Conclusion:** AILI can progress to advanced fibrosis and clinically significant

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portal hypertension despite mild laboratory abnormalities and the absence of steatosis. Chronic amiodarone use should remain on the differential of unexplained liver injury even months to years after initiation. The pattern of injury typically resembles that of alcoholic liver disease clinically and histologically, including the presence of Mallory hyaline. However, the absence of steatosis in AILI can help distinguish these diagnoses from each other when imaging and laboratory workup alone are nondiagnostic.

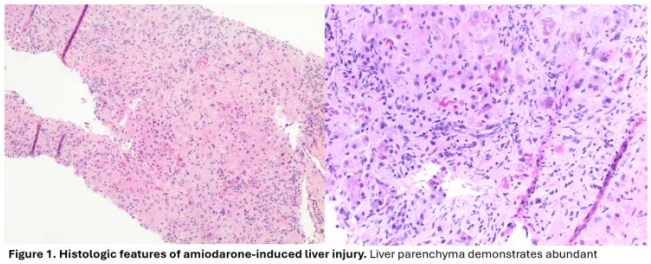


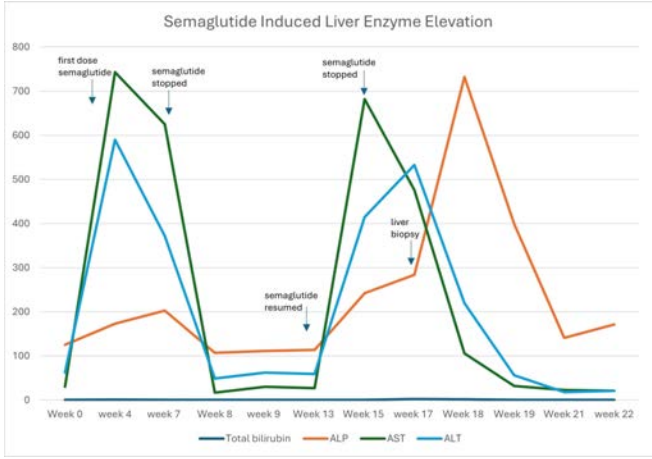
Figure 1. Histologic features of amiodarone-induced liver injury. Liver parenchyma demonstrates abundant Mallory-Denk bodies in hepatocytes with minimal to absent hepatocellular ballooning, a characteristic pattern of amiodarone-associated hepatotoxicity (H&E).

3671 | SEMAGLUTIDE INDUCED LIVER INJURY

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Introduction: Semaglutide is efficacious in reducing hepatic fibrosis and steatohepatitis in patients with metabolic dysfunction-associated steatohepatitis. With increased use, there have been a few case reports of drug induced liver injury from semaglutide. This case report demonstrates a drug induced liver injury to semaglutide with positive rechallenge test. **Case Presentation:** This is a 76-year-old woman with obesity, pre-diabetes, heart failure preserved ejection fracture, atrial fibrillation, rheumatoid arthritis, prior cholecystectomy who was started on semaglutide 0.25mg weekly for obesity and cardiac health. Four weeks later, she presented to the emergency department with acute abdominal pain and was found to have elevated liver function tests in hepatocellular pattern. Semaglutide was held and liver enzymes repeated within one week demonstrated improvement. She underwent a thorough workup including abdominal ultrasound, MRCP, and endoscopic ultrasound with a mildly dilated biliary duct to 10mm without an obstructive process identified. Serologic workup ruled out an autoimmune or infectious pathology. Six weeks following normalization in labs, she was rechallenged with Semaglutide 0.25mg weekly. Six weeks following the rechallenge, liver enzymes increased in a mixed hepatocellular and cholestatic pattern. Semaglutide was stopped and labs normalized within five weeks. Liver biopsy showed multiple ceroid laden macrophages predominantly in the portal areas, with no significant lymphoplasmacytic inflammation or granuloma and bile ducts uninvolved. Findings most consistent with drug induced liver injury. Unfortunately, her liver biopsy was complicated by cholangitis and ecoli bacteremia due to a blood clot obstructing the bile duct which resolved with common bile duct stenting. **Discussion:** GLP-1 agonists are widely used medications for a variety of multisystem conditions related to metabolic dysfunction. Phase 3 clinical drug trials and large meta-analysis of observational cohort studies evaluating semaglutide for metabolic dysfunction associated liver dysfunction with F2-3 fibrosis have demonstrated a beneficial safety profile with no discontinuations due to elevated liver enzymes. There have been rare case reports of drug induced liver injury secondary to Tirzepatide and Semaglutide with only one study demonstrating positive rechallenge test. There remains a knowledge deficit in the mechanism of GLP-1 induced hepatocyte injury. Two case reports demonstrating cholestatic liver injury, two with hepatocellular liver injury and this case demonstrating a mixed pattern injury. **Conclusion:** Although a rare complication, it is important to consider DILI in patients even on low dose semaglutide with rising liver function tests, and if recognized early, liver biopsy can be avoided.



3672 | HIDDEN HAZARDS ON THE SHELF: SEVERE DRUG-INDUCED LIVER INJURY FROM UNREGULATED HERBAL AND DIETARY SUPPLEMENTS

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Introduction: Drug induced liver injury (DILI) is commonly associated with acetaminophen (APAP) and an-

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tibiotics however, between 2004 and 2013 DILI due to herbal and dietary supplements (HDS) increased from 7 to 20 percent. We present a case of a patient taking multiple HDS resulting in severe acute liver injury. **Case Presentation:** A 47-year-old male with history of opiate use disorder in remission on suboxone presented to our tertiary care center with malaise and abdominal pain. His labs were significant for a mixed pattern of liver injury and INR 1.6 (Table 1). Liver tests were normal three months prior. The patient reported kratom use for 1 year and recently started a supplement "O.R. Reset" containing a synthetic mu-opioid receptor agonist "Essar 17" or SR-17018. Additionally, he was taking multiple supplements including lion's mane, green tea leaf extract, maca root extract amongst others. There was no history of recent antibiotics, APAP, or alcohol use. The patient was started on N-acetylcysteine for suspected non-APAP DILI. Chronic liver disease workup including autoimmune, genetic, and infectious causes were negative. He underwent a liver biopsy that was consistent with DILI and showed thirty percent hepatocyte necrosis. He was discharged on hospital day seven. He had close monitoring and bilirubin peaked at 29.8 and INR 1.9. He was considered for liver transplantation however his most recent labs 1.5 months since discharge show marked improvement (Table 1) with a full recovery expected. **Discussion:** The HDS industry is a multi-billion-dollar industry with hundreds of new products emerging every year. Some studies show that as many as 50% of Americans consume HDS. Ingredients are often underreported due to little regulatory oversite. Supplements are not necessarily Food and Drug Administration (FDA) approved, but they are well advertised on social media platforms and major retail stores. Studies assessing the safety of ingredients is not required unless the product contains a novel compound discovered after the 1994 Dietary Supplement Health and Education Act.Our patient was using a variety of supplements including Kratom, a known cause of liver injury, typically causing cholestatic or mixed pattern of liver injury within 1 to 8 weeks of starting use. Given our patient's long-term use of kratom, with previously normal liver tests, there was low concern it was the culprit of his DILI. SR-17018 the main ingredient in "OR-Reset" is an experimental compound never studied in humans. As of August 27,2026, the FDA issued a temporary order to schedule SR-17018 to "avoid an imminent hazard to public safety." We suspect SR-17018 or a combination of his herbal supplements as a source of DILI. **Conclusion:** As the HDS industry continues to grow and advertise, consumers should be aware of the potentially dangerous and deadly side effects of such products. Our case highlights the need for increased public health, safety, and regulatory efforts with regards to supplements.

Table 1: Liver Tests & INR Trend

Date	Alkaline Phosphatase	Alanine Aminotransferase (ALT)	Aspartate Aminotransferase (AST)	Total Bilirubin	INR
8/19/26	104	262	168	6.2	1.2
8/5/26	122	642	365	13.7	1.2
7/27/26	113	982	660	29.8	1.3
7/20/26	129	910	544	20.7	1.5
7/10/26	146	959	529	13.5	1.7
7/6/26	119	1,338	626	10.0	1.7
7/5/26	100	1,453	632	8.6	1.9
7/4/26	105	1,682	719	8.1	1.9
7/3/26	103	1,791	771	7.9	1.9
7/2/26	101	1,889	867	6.5	1.9
7/1/26	83	1,879	957	4.7	1.9
6/30/26	102	1,867	903	4.5	1.6
3/26/26	69	17	20	0.6	

3673 | FROM CHOLESTASIS TO CIRRHOSIS: A RARE CASE OF AMOXICILLIN/CLAVULANATE RELATED LIVER INJURY PROGRESSING TO CHRONIC LIVER DISEASE

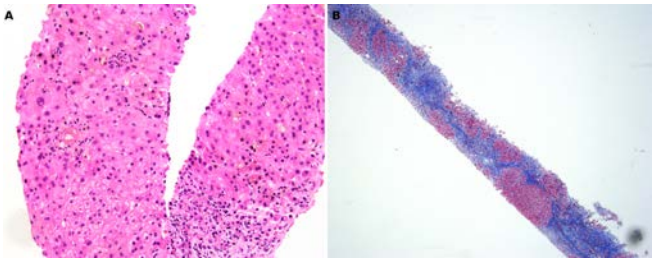
Udochukwu Anwaegbu[1] Judy Dinh[1] Naziheh Assarzadegan Assarzadegan[1] Nimy John[1]

[1]University of Florida

Introduction: Amoxicillin/clavulanate is a well known cause of drug-induced liver injury (DILI). Ususally, the acute liver injury resolves once the offending agent is stopped. We highlight a case of amoxicillin-clavulanate-associated DILI with progression to decompensated cirrhosis over a two-year period. **Case Presentation:** A 44-year-old man with type 2 diabetes and hypertension presented with one week of pruritus. Laboratory testing showed a cholestatic pattern of liver injury: AST 110 U/L, ALT 164 U/L, total bilirubin 4.9 mg/dL, and alkaline phosphatase (ALP) 548 U/L. Two weeks earlier, he had completed four days of amoxicillin-clavulanate. CT and ultrasound showed no biliary obstruction. Evaluation for viral, autoimmune, and hereditary liver disease was unrevealing, and he denied alcohol use. He was readmitted one month later for recurrent symptoms and worsening cholestatic injury. EUS-guided liver biopsy revealed a cholestatic hepatitis pattern with no fibrosis on trichrome stain. There was presence of severe cholestasis characterized by bile plugs in the canaliculi and hepatocytes, feathery degeneration of hepatocytes, and bile ductular proliferation. Given the recent history of amoxicillin-clavulanate and absence of bile duct obstruction, DILI was favored. Despite steroid treatment, he continued to have increased liver enzymes with ALP >2000 U/L. He denied any new medications, supplements, or alcohol use. He then developed ascites, peripheral edema, thrombocytopenia, and splenomegaly. MRCP

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demonstrated no biliary ductal dilation or irregularity. Repeat autoimmune evaluation remained negative. Repeat liver biopsy at two years showed cirrhosis with cholestasis and brisk bile ductular proliferation without histologic features of primary biliary cholangitis or autoimmune hepatitis. He was referred for liver transplant evaluation. **Discussion:** DILI presents with elevated liver enzymes hepatocellular, cholestatic, or mixed pattern. Diagnosis requires recognition of a temporal relationship with a potential offending agent and exclusion of alternative causes. Although amoxicillin-clavulanate-associated DILI is generally self-limited, this case demonstrates a striking progression from severe cholestatic hepatitis without significant fibrosis to biopsy-proven cirrhosis in two years. **Conclusion:** DILI should not be assumed to resolve after stopping the causal agent. Continued monitoring to ensure normalization of lab work is essential to avoid persistent injury and progression to chronic liver disease.

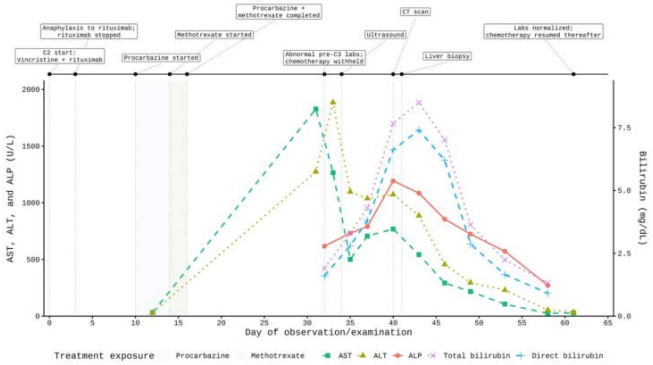


3674 | IDIOSYNCRATIC DRUG INDUCED LIVER INJURY FROM PROCARBAZINE: A RARE COMPLICATION OF R-MVP IN PRIMARY CNS LYMPHOMA

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Introduction: Clinically apparent drug-induced liver injury (DILI) from procarbazine is rare and almost always reported as hypersensitivity. We describe idiosyncratic DILI during R-MPV (rituximab, methotrexate, procarbazine, vincristine) for primary CNS lymphoma, in which interface hepatitis raised concern for autoimmune hepatitis (AIH) and selective drug withdrawal allowed curative therapy to continue. **Case Presentation:** A 63-year-old woman with no known liver disease and normal baseline liver tests received R-MPV, Precycle 3 testing showed AST 1827 U/L, ALT 1274 U/L, ALP 629 U/L, bilirubin 1.9 mg/dL, and INR 1.33, without abdominal pain, jaundice, rash, fever, or eosinophilia. R factor was 7.1 (hepatocellular) and RUCAM 7 (probable). Chemotherapy was withheld. Hepatitis A, B, C, acetaminophen, alcohol, and herbal exposure were excluded; imaging of the liver and biliary tree showed no bile duct obstruction, thrombosis, or metastases. Aminotransferases fell but plateaued as bilirubin rose to 8.5 mg/dL, meeting Hy's law criteria. A liver biopsy was performed that showed portal lymphocytic inflammation with few neutrophils and eosinophils, focal interface hepatitis, apoptosis, and mild canalicular cholestasis, without plasma cell clusters, rosettes, confluent necrosis or fibrosis. Serum IgG was normal and autoantibodies were negative apart from low-titer anti-smooth muscle antibody (1:20). Procarbazine was permanently discontinued; liver tests normalized within 3 weeks without corticosteroids, and she completed seven cycles of rituximab/methotrexate-based therapy without recurrence of liver injury. **Discussion:** DILI is a potentially serious complication of chemotherapy and identification of the culprit in multidrug chemotherapy may be challenging. Identification of the offending agent (Procarbazine) was based on the known hepatotoxicity profiles of each drug and their temporality. Methotrexate DILI is dose-related and transient, rituximab is linked to hepatitis B reactivation, and vinca alkaloids lack well-characterized hepatotoxicity, leaving procarbazine as the only agent whose profile and latency fit. Dechallenge produced full recovery while the other agents were tolerated over seven cycles, confirming Procarbazine as the likely culprit agent. Prior reports describe hypersensitivity features which were absent here, suggesting procarbazine DILI can occur without hypersensitivity. **Conclusion:** Procarbazine warrants consideration in marked hepatocellular injury during multiagent chemotherapy even without hypersensitivity. Interface hepatitis with normal IgG and negative autoantibodies does not establish AIH or drug-induced autoimmune-like hepatitis. Timing, exclusion of competing causes, dechallenge, and tolerance of nonculprit agents can secure the diagnosis of idiosyncratic DILI while allowing chemotherapy and management to continue.



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3675 | MARKED HEPATOCELLULAR INJURY IN HYPEREMESIS GRAVIDARUM: MORE THAN MORNING SICKNESS

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Introduction: Elevation of transaminases is seen in roughly 50% of hospitalized women with hyperemesis gravidarum (HG). Although enzyme elevation in HG is usually less than 200 U/L, levels as high as 1,600 U/L have been reported. We present a case of HG complicated by AST and ALT elevations to 640 and 820 U/L, respectively. This case highlights that severe hepatocellular injury, while uncommon, can occur in HG and should prompt a broad evaluation for pregnancy-specific and non-pregnancy-related causes of liver injury. **Case Presentation:** A 29-year-old woman (G2P0010) presented to an outside facility at 8 weeks' gestation with HG and was initially managed with intermittent parenteral fluids. Four weeks later, she was admitted because she could not tolerate oral intake or tube feedings and was started on total parenteral nutrition. On admission, AST and ALT were greater than 500 U/L and total bilirubin was 1.4 mg/dL; these abnormalities did not improve with parenteral fluid and nutrition support. Evaluation with antimitochondrial antibody, anti-smooth muscle antibody, tissue transglutaminase level, hepatitis panel, and Monospot testing was unremarkable. Ceruloplasmin was 45. EGD was unremarkable, and MRI of the liver showed mild hepatosplenomegaly and hepatic steatosis. She was transferred to our facility after a 2-week hospitalization for persistent hepatocellular injury. On transfer, AST was 640 U/L, ALT was 820 U/L, and total bilirubin was 1.3 mg/dL. TPN was continued. Liver ultrasound with Doppler was unremarkable. HSV PCR, EBV PCR, CMV PCR, hepatitis E testing, and serum drug screen were negative. Autoimmune hepatitis was considered unlikely given negative serologies and lack of response to previously administered dexamethasone. Acute drug-induced liver injury was also considered unlikely because she had been taking sertraline consistently for 10 years. The patient was treated empirically with ursodiol, and efforts were made to transition her to oral feeding while avoiding parenteral overfeeding. She remained clinically stable and was tapered off TPN over the next 2 weeks. At discharge, AST had improved to 49 U/L and ALT to 243 U/L. **Discussion:** The differential diagnosis of transaminase elevation during pregnancy includes both pregnancy-specific and non-pregnancy-specific etiologies. Pregnancy-specific disorders include hyperemesis gravidarum, intrahepatic cholestasis of pregnancy, HELLP syndrome, and acute fatty liver of pregnancy, each with characteristic timing and clinical features. Non-pregnancy-specific causes include viral hepatitis, systemic viral infections, autoimmune hepatitis, drug-induced liver injury, and Wilson disease. **Conclusion:** In this case, the temporal association of symptoms with severe hyperemesis, negative viral and autoimmune workup, unrevealing hepatic imaging, and improvement following nutritional stabilization favored HG as the most likely etiology of the hepatocellular injury.

3676 | AUTOIMMUNE HEPATITIS PRESENTING AS A HEPATIC PSEUDOTUMOR

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Introduction: Autoimmune hepatitis (AIH) presentation ranges from asymptomatic elevation of liver enzymes to acute severe hepatitis. However, AIH presenting with acute severe hepatitis and an infiltrative liver mass has not been reported in the literature. We present a rare case of AIH presenting as an infiltrative hepatic mass with retroperitoneal lymphadenopathy, radiographically mimicking hepatic malignancy or lymphoma. **Case Presentation:** A 30-year-old woman with no prior liver disease presented with a two-month history of progressively worsening hepatocellular liver injury and a one-month history of jaundice, dyspnea, and abdominal distention. Admission labs were AST 338, ALT 243, total bilirubin 8.7 mg/dL (direct 5.3), albumin 1.5 g/dL, Alkaline Phosphatase 180, and INR 2.7. MRI revealed an 11.5 x 9.7 x 7.6 cm infiltrative right hepatic mass with retroperitoneal lymphadenopathy, concerning for primary hepatic malignancy, lymphoma, or metastatic disease (Figure 1a). No definitive history of dietary herbal supplements or significant alcohol intake. Workup revealed ANA 1:80, ASMA 37, and elevated IgG 2,636. The remainder of the workup, including viral hepatitis serologies, AMA, ceruloplasmin, AFP, CEA, CA19-9, and PEth, was negative. CT-guided biopsy of the mass and background liver both showed portal lymphoplasmacytic infiltrates and severe interface hepatitis without malignancy. Based on the revised original diagnostic scoring system, the patient was diagnosed with definitive AIH. Prednisone 60 mg daily was initiated. Over the next two weeks, AST, ALT, and total bilirubin improved (Figure 1b). To better characterize the liver lesion on treatment, a repeat liver biopsy and flow cytometry were performed. Results were negative for dysplasia, malignancy, and lymphoproliferative disorder, but revealed resolution of the lymphoplasmacytic infiltrates. The patient was dis-

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charged on a prednisone taper and mercaptopurine (TPMT was within normal range). Labs after 3 weeks revealed continued improvement of bilirubin, transaminases, INR, and albumin (Figure 1b). Contrast-enhanced CT showed small multiple hyperdense lesions in the liver suggesting regenerative nodules, but no infiltrative mass. **Discussion:** The diagnosis of AIH was established based on the revised diagnostic scoring system, despite the unusual picture on contrast imaging. Therapeutic biochemical, radiological, and histologic responses point toward AIH-related pseudotumor in this case. Follow-up imaging in 3-6 months would be helpful to assess lesion resolution. **Conclusion:** An infiltrative hepatic pseudotumor with retroperitoneal lymphadenopathy is a rare presentation of AIH mimicking malignancy or lymphoma.

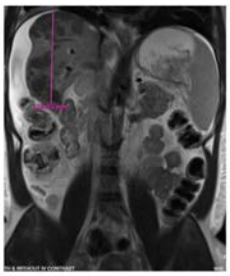


Figure 1a: Infiltrative liver lesion in the right hepatic lobe with smooth contour of liver

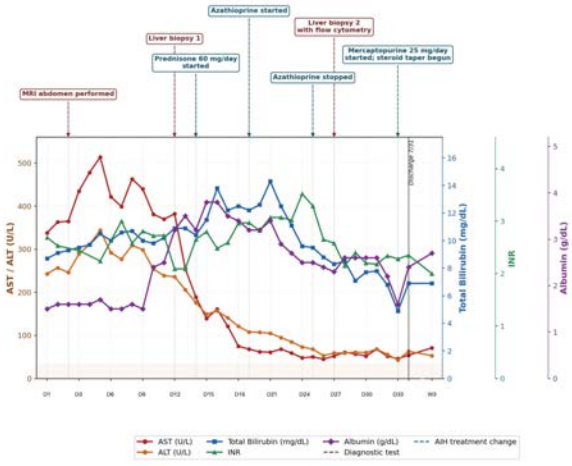


Figure 1b: Biochemical profile at baseline and on treatment

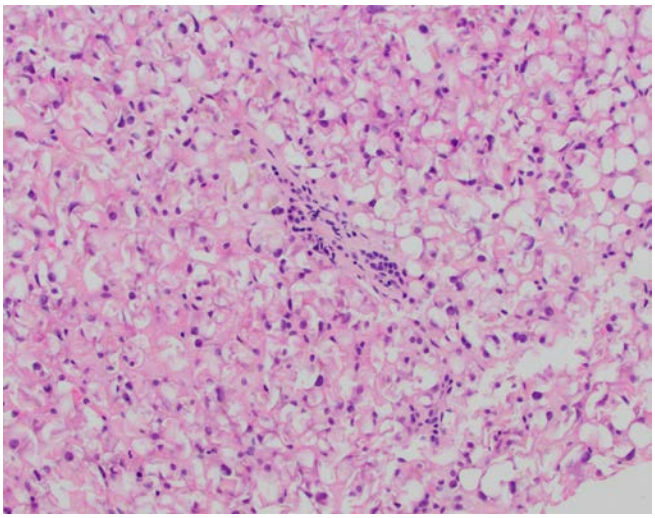
3677 | ACUTE MACROVESICULAR STEATOSIS IN ACUTE SEVERE ULCERATIVE COLITIS: A MALNUTRITION-ASSOCIATED LIVER INJURY

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Introduction: Rapid development of severe macrovesicular hepatic steatosis is unusual. Protein-calorie malnutrition is an uncommon but recognized cause and may be overlooked in patients with traditional metabolic risk factors. **Case Presentation:** A 64-year-old woman with obesity and recently diagnosed ulcerative colitis (UC), without known prior MASLD or alcohol use, presented after several months of poor oral intake, 13% weight loss, and persistent bloody diarrhea found to have acute severe ulcerative colitis flare. Two months earlier, liver enzymes and albumin were normal (AST 25 U/L, ALT <7 U/L, alkaline phosphatase [ALP] 66 U/L, albumin 4.1 g/dL), and abdominal imaging showed no steatosis. By January 2026, CT demonstrated new severe diffuse hepatic steatosis with hepatomegaly (consider putting a picture of the ct). During admission for severe UC flare, liver tests rose to AST 192 U/L, ALT 218 U/L, and ALP 413 U/L, while bilirubin remained normal. Albumin fell from normal just a few months prior to 1.3 g/dL and total cholesterol decreased from 164 to 55 mg/dL. MRCP showed no biliary abnormality, and ANA, ASMA, and AMA were negative. Liver biopsy demonstrated >90% large-droplet macrovesicular steatosis without steatohepatitis or fibrosis and no significant portal or lobular inflammation, bile duct injury, or cholestasis. Stool α1-antitrypsin clearance, obtained after initiation of UC flare treatment and during clinical improvement, was mildly elevated at 33 mL/24 h, supporting intestinal protein loss. During nutritional rehabilitation, hypophosphatemia and hypokalemia developed, consistent with refeeding physiology. With treatment of colitis and nutritional recovery, albumin increased to 3.1 g/dL and AST/ALT/ALP decreased to 83/137/173 U/L by 3/6/26. Follow MRI 3 months later showed moderate hepatic steatosis. LFTs remained normal. **Discussion:** The abrupt appearance of near-total bland macrovesicular steatosis, profound hypoalbuminemia and hypolipidemia, absence of fibrosis or steatohepatitis, and subsequent biochemical recovery support malnutrition-associated hepatic steatosis with intestinal protein loss. Background MASLD cannot be completely excluded given obesity, but it does not explain the rapid radiographic and lab evolution. **Conclusion:** Severe nutritional depletion should be considered in patients with rapidly developing steatosis, including those with obesity.

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3678 | A THERAPEUTIC CATCH-22: SIMULTANEOUS LIVER TRANSPLANTATION AND MITRAL VALVE REPLACEMENT IN DECOMPENSATED CIRRHOSIS

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Introduction: Concomitant decompensated cirrhosis and severe valvular disease may pose a therapeutic "catch-22," as cirrhosis increases perioperative risk for cardiac surgery, while uncorrected severe cardiac valvular disease may complicate liver transplantation (LT). A multidisciplinary approach is therefore essential when neither operation can be safely pursued alone. **Case Presentation:** A 48-year-old man with decompensated alcohol-associated cirrhosis complicated by hepatic encephalopathy and esophageal varices, as well as severe mitral regurgitation (MR) status post transcatheter edge-to-edge repair (TEER) presented 5 months after TEER with 30-lb weight gain and anasarca. Transesophageal echocardiography showed a flail anterior leaflet, severe eccentric MR, and a mean transmitral gradient of 11 mmHg, consistent with severe post-TEER mitral stenosis. Notably, this gradient persisted at a heart rate of 60-70 beats per minute, limiting further medical optimization. While the severity of his valvular disease precluded transcatheter re-intervention, isolated valve surgery was deemed too risky because of his liver disease. He was therefore referred for multidisciplinary cardiology and hepatology evaluation. Labs included creatinine 1.3 mg/dL, INR 1.5, total bilirubin 4.6 mg/dL, albumin 2.2 g/dL, and platelets 120 K/µL; MELD 3.0 was 22 and he was Child-Pugh Class C. VOCAL-Penn estimated 30- and 90-day postoperative mortality at 11.1% and 13.6%. Given the risks of either isolated cardiac surgery or LT with untreated severe mitral valve disease, a combined operative approach was chosen.Nine months later, he underwent simultaneous LT and mitral valve replacement on cardiopulmonary bypass with ASD closure and left atrial appendage clipping. Postoperative complications included acute kidney injury requiring temporary CRRT, atrial fibrillation, chyle leak, and transient hypotension. He was extubated and weaned from vasopressors on postoperative day (POD) 2, and he was discharged on POD 16. At 6 months, he had no rehospitalizations and excellent allograft function with normal liver enzymes. **Discussion:** Coexisting advanced liver and cardiac disease can create a therapeutic dilemma when the severity of each condition increases the risk of addressing the other in isolation. Although cirrhosis-specific tools, such as VOCAL-Penn, are helpful to assess risk, their performance remain unvalidated in these populations since they were derived from non-transplant data. Previously published single-center studies report 1-year survival of 73.3-74.2% following combined LT and cardiac surgery, but improvements in post-operative management mean outcomes are likely better currently. **Conclusion:** Multidisciplinary coordination can overcome the therapeutic "catch-22" of concurrent advanced liver and cardiac disease, allowing definitive treatment when sequential procedures are deemed unsafe.

Table 1. Perioperative Trends in Liver Function and Mitral Valve Hemodynamics

	At time of referral	Pre-op	Discharge (post-op day 16)	1-month follow-up	6-month follow-up
Total bilirubin (mg/dL)	4.6	4.9	0.8	0.5	0.4
Platelets (x10⁹/ µL)	120	182	210	153	149
INR (PT, sec)	1.5 (17.5)	1.5 (17.0)	—	—	—
Creatinine (mg/dL)	1.3	1.2	1.7	1.9	1.3
Albumin (g/dL)	2.2	2.4	1.9	3.4	4.2
Aspartate aminotransferase (U/L)	88	76	30	27	16
Alanine aminotransferase (U/L)	57	38	34	86	12
MELD 3.0	22	22	—	—	—
Mean mitral valve gradient (mmHg)	11	7	6	—	—

3679 | A CASE OF SUSPECTED TRANSPLANT ASSOCIATED THROMBOTIC MICROANGIOPATHY IN A PEDIATRIC LIVER TRANSPLANT RECIPIENT

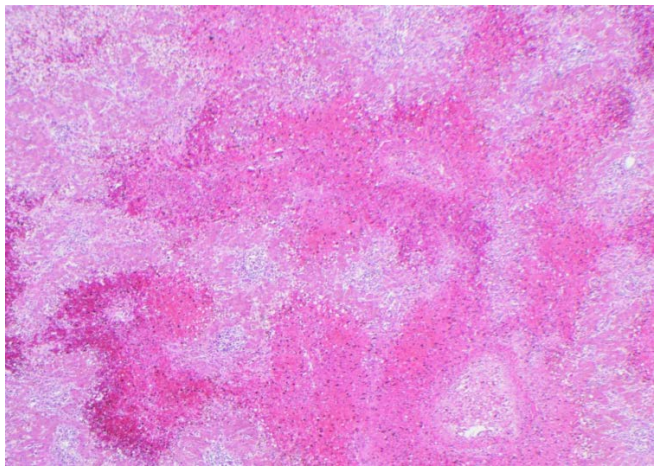
Forum Patel[1] Alyssa Kriegermeier[2]

[1]Ann & Robert H. Lurie Children's Hospital of Chicago, [2]Ann and Robert H. Lurie Children's Hospital of Chicago

Introduction: Transplant-associated thrombotic microangiopathy (TA-TMA) is characterized by endothelial injury, microvascular thrombi, thrombocytopenia, microangiopathic hemolytic anemia, and multiorgan dysfunction. Although well described after hematopoi-

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etic stem cell transplantation (HSCT), TA-TMA may also occur following solid organ transplantation. Potential triggers include ischemia-reperfusion injury, infection, calcineurin inhibitors and complement activation. We describe a case of suspected TA-TMA post liver transplant leading to graft loss and re-transplantation. **Case Presentation:** A 3-year-old male with maple syrup urine disease underwent a deceased-donor left lateral segment liver transplant for recurrent metabolic crises. Organ cold ischemia time was approximately 7 hours, with a warm ischemia time of 30 minutes. Immediately after reperfusion, he developed severe ischemia-reperfusion injury with hemodynamic instability, coagulopathy, and acidosis. On postoperative day (POD) 1, he developed hypertension, thrombocytopenia, anemia, elevated LDH (3370), AKI, and 2+ schistocytes. He had persistent hepatofugal portal vein (PV) flow and hyperammonemia requiring CRRT. Complement levels demonstrated low C3/C4 and CH50, negative factor H antibody with markedly elevated soluble C5b-9 (1730 ng/mL). Liver biopsy on POD2 demonstrated 30-50% centrilobular necrosis without microvascular thrombi. Evaluation for rejection, infection, primary non-function, and vascular complications was unrevealing. These clinical and laboratory findings strongly supported TA-TMA and therapeutic plasma exchange (TPE) and eculizumab were initiated. Despite 3 doses of eculizumab, repeated TPE, and maximal CRRT, he had persistent graft failure and repeat biopsy showed persistent centrilobular necrosis, ultimately requiring repeat transplantation on POD 12. **Discussion:** This case highlights TA-TMA as a rare cause of early graft failure. Proposed TMA diagnostic criteria include histologic microvascular thrombi/endothelial injury or >= 4 of the 7 following features: elevated LDH, schistocytes, de novo thrombocytopenia, de novo anemia, hypertension, proteinuria and elevated sC5b9. Our patient met all 7 of the proposed clinical criteria for TMA, and we hypothesize that severe ischemia-reperfusion injury triggered endothelial injury and complement dysregulation, leading to progressive microvascular injury, necrosis and persistent hepatofugal PV flow. Eculizumab, a monoclonal antibody against C5, has demonstrated improved outcomes and graft recovery in TMA, however, did not result in meaningful graft recovery in this patient. **Conclusion:** TA-TMA is a rare but graft and life-threatening complication post liver transplant. Clinical presentation may overlap with several other etiologies of graft dysfunction and clinicians but maintain a high degree of suspicion for TA-TMA post-transplant.



3680 | INTERFERON-GUIDED JAK1/2 INHIBITION WITH RUXOLITINIB FOR STEROID-REFRACTORY REJECTION AFTER PEDIATRIC LIVER TRANSPLANTATION

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Introduction: Steroid-refractory acute cellular rejection (ACR) after pediatric liver transplantation is difficult to treat, and lymphodepletion carries substantial infectious and metabolic toxicity. Ruxolitinib, aJAK1/2 inhibitor that blocks signaling downstream of type I and II interferons, is approved for steroid-refractory graft-versus-host disease, but experience in solid-organ rejection is limited. Wereport on its use for refractory ACR. **Case Presentation:** A 15-year-old girl underwent orthotopic liver transplantation for acute liver failure; explant pathology revealed Autoimmune Hepatitis/Primary Sclerosing Cholangitis overlap. At 5 months post-transplant, she developed steroid-refractory ACR requiring methylprednisolone and antithymocyte globulin (ATG). She remained on tacrolimus, mycophenolate, and prednisone but could not be weaned off steroids. Immune profiling revealed persistent innate and adaptive activation with Th1-predominant inflammation and mixed type I/II interferon activation; IFN-γ elevated at 13.3 pg/mL with CXCL9 in the reference range. At 18 months she re-presented with AST 182 and ALT 250 U/L; biopsy confirmed ACR (Banff rejection activity index [RAI] 5/9). Despite repeated pulse steroids, she had an inadequate response, with enzymes worsening during taper. Repeat biopsy 2 weeks later showed RAI 5/9. Allograft tissue showed a CD8+/CD103+ tissue-resident memory T-cell infiltrate. ATG was considered, however there were concerns for infection risk, given obesity (BMI 40.4 kg/m²), and steroid-induced diabetes. Ruxolitinib 10 mg twice daily

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was started with antimicrobial prophylaxis and increased to 15 mg twice daily. ALT fell from 199 to 68 U/L within 10 days. Transaminases normalized by month 5 and remained normal through 8 months. Ruxolitinib was reduced to 10 mg twice daily and prednisone tapered from 60 mg twice daily to 5 mg daily, without further episode of rejection or opportunistic infection. **Discussion:** Ruxolitinib was chosen to attenuate a broad, cytokine-mediated inflammatory response rapidly and reversibly. JAK1/2 blockade interrupts with interferon signaling directly downstream of the receptor; while corticosteroids and lymphodepletion act further upstream. Profiling showed type I interferon and IFN-γ elevation without a rise in CXCL9, suggesting a broader inflammatory phenotype and supporting potential benefit. **Conclusion:** In recurrent steroid-refractory pediatric liver allograft rejection with mixed type I/II interferon activation, mechanism-directed JAK1/2 inhibition was associated with durable biochemicalremission and substantial steroid sparing without opportunistic infection. Prospective evaluation in refractory solid-organ rejection is warranted.

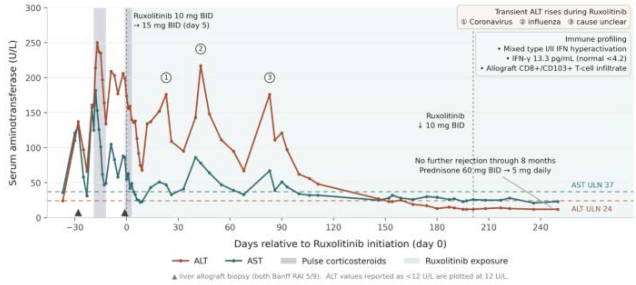


Figure 1. Serum aminotransferase trajectory before and after Ruxolitinib initiation. Two liver allograft biopsies (▲) five weeks apart each showed Banff RAI 5/9 despite pulse corticosteroids. Ruxolitinib was added on day 0, one day into a repeat methylprednisolone pulse, and increased to 15 mg twice daily on day 5. Three later transient ALT rises (①②③) coincided with coronavirus infection and influenza; the third had no identified cause. AST and ALT normalized by month 5 and remained normal through 8 months, permitting Ruxolitinib de-escalation and a prednisone taper from 60 mg twice daily to 5 mg daily. Timeline is relative to Ruxolitinib initiation.

3681 | RECURRENT GENERALIZED LYMPHATIC ANOMALY IN A HEPATIC ALLOGRAFT

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Introduction: Generalized Lymphatic Anomaly (GLA), formerly hepatic lymphangiomatosis, is a rare disorder of soft tissues, viscera, and bone caused by multifocal, cystic dilation of lymphatic channels [1, 2]. It is often misdiagnosed as Polycystic Liver Disease (PLD) on imaging, but has histology positive for lymphatic endothelial markers, such as D2-40 or PROX-1 [2, 3]. Here, we report the second case of GLA recurrence in a hepatic allograft [4]. **Case Presentation:** A 61-year-old woman underwent deceased donor orthotopic liver transplant (OLT) for symptomatic PLD. Explant histology unexpectedly demonstrated GLA. Post-transplant, she was maintained on sirolimus and mycophenolate mofetil. CT scan at 21 months post-transplant revealed numerous round low-density lesions throughout the allograft and a 6.5cm known splenic lesion which was originally 5.1cm 2 years prior. Serial imaging over the following two years demonstrated disease progression with extra-hepatic lesions in the spleen, mesentery and lung, consistent with multifocal GLA. Allograft biopsy at 47 months post-transplant confirmed recurrent GLA with ductular proliferation and possible cirrhosis. Immunohistochemistry was positive for D2-40 and PROX1 lymphatic markers confirming lymphatic endothelial immunophenotype. Due to concern for cirrhosis, a repeat allograft biopsy at 48 months was performed which showed stage 2 fibrosis with chronic passive congestion. Echocardiogram and hepatic venogram were unremarkable, with a portosystemic gradient of 1. Lanreotide was initiated, and due to the patient's extrahepatic disease, she was not a re-transplant candidate. **Discussion:** To our knowledge, this is the second reported case of GLA recurrence in a hepatic allograft. In the present case, disease recurrence happened significantly earlier, 2 years versus 19 years post-transplant [4]. GLA is a developmental anomaly of the lymphatic system caused by the activation of the PI3K-AKT-mTOR signaling pathway by somatic mosaic variants of genes including PIK3CA, PIK3CB, and AKT3 [1]. Recurrence and progression occurred despite maintenance sirolimus, an mTOR inhibitor, which is the mainstay treatment for GLA [2, 5]. This is consistent with reported outcomes, in which only about half of patients achieve a partial radiographic response and none achieve a complete response [5]. **Conclusion:** Cystic liver disease without supporting evidence, such as family history or renal cysts, should increase concern for GLA and warrants further investigation, including tissue diagnosis and lymphatic endothelial marker testing. As one of only two reported cases of allograft reoccurrence, post-transplant GLA is poorly understood and requires future study to guide management.

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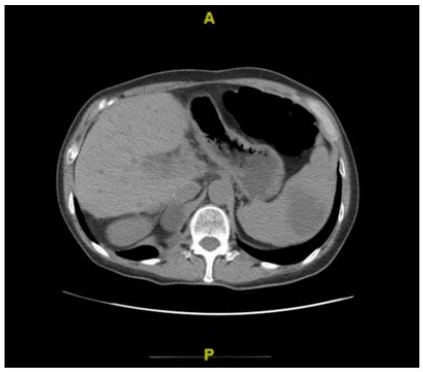


Image 1. CT scan of the abdomen with numerous round low-density lesions in the allograft and a 6.5 cm splenic cyst.

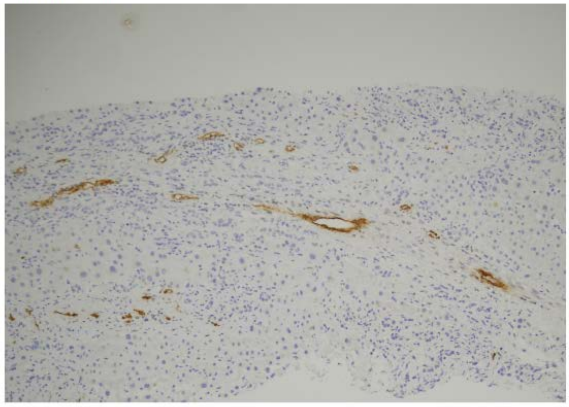


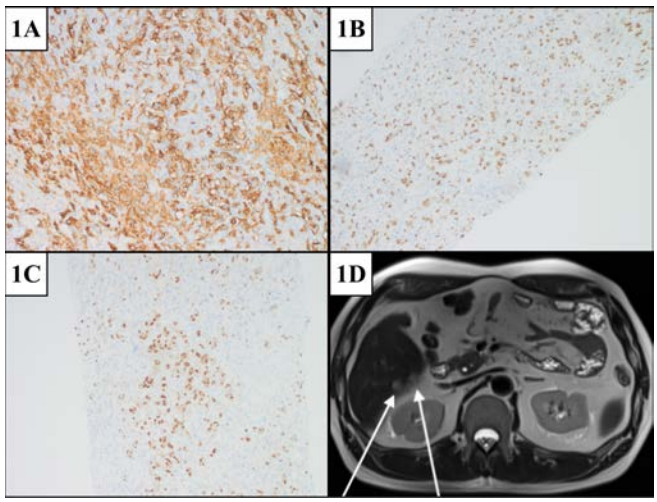
Image 2. D2-40 immunohistochemical stain highlighting abundant, focally dilated lymphatic spaces. [D2-40 10X]

3682 | LIVING DONOR LIVER TRANSPLANTATION FOR HEPATIC EPITHELIOID HEMANGIOENDOTHELIOMA

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Introduction: Hepatic epithelioid hemangioendothelioma (HEHE) is a rare malignant vascular tumor with an incidence of 1-2 cases per million, variable behavior, and no standardized treatment. Management includes systemic therapy, resection, and liver transplantation. **Case Presentation:** A 48-year-old male without liver disease was found to have a 16 cm segment IVb liver mass on MRI obtained for isolated alkaline phosphatase elevation (131 U/L; reference 34-104) with biopsy revealing bilobar HEHE. Initial therapy with bevacizumab was discontinued for nonresponse. Absence of macrovascular invasion, hilar lymphadenopathy, and disease progression over 120 days suggested low recurrence risk, prompting transplant evaluation. The initial Model for End-Stage Liver Disease (MELD) score was low, but his course was complicated by worsening liver function and progressive portal hypertension, including new ascites with superimposed *Aspergillus* peritonitis. MELD ultimately peaked at 29 prompting living donor liver transplantation before further decompensation.Explant pathology showed bridging fibromyxoid stroma with spindle and atypical epithelioid cells positive for CD34, ERG, and CAMTA1, consistent with HEHE, without features of sarcoma (Figure 1A-C). Liver biopsy at one month for elevated liver enzymes was negative for acute cellular rejection or malignancy. The patient recovered well and was transitioned to tacrolimus and sirolimus therapy for renal sparing and potential antitumor effect. At 19 months, quarterly surveillance MRI revealed multiple new hyperintense, hypoenhancing peripheral liver lesions typical of HEHE and concerning for recurrence (Figure 1D), prompting pazopanib initiation. The patient remains on pazopanib at 25 months post-transplant. **Discussion:** HEHE management remains non-standardized given its rarity and variable behavior, with systemic therapy showing modest efficacy. Transplant candidates with HEHE often have disease too extensive for resection, so selection bias likely explains lower survival versus non-transplant approaches (1- and 3-year survival 63% and 25% in transplantation versus 98% and 91% with medical and surgical treatment). In select candidates, 5-year disease-free survival after transplant can reach 80%, with a 25% recurrence rate. Long-term outcomes parallel those after transplant for hepatocellular carcinoma. **Conclusion:** Extensive or bilobar disease alone should not exclude candidates from transplant consideration. Given the risk of recurrence, lifelong surveillance is essential for early detection and treatment.



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3683 | REGRESSION OF ADVANCED FIBROSIS FOLLOWING INCRETIN-BASED THERAPY IN PATIENTS WITH MASLD/MASH: A TWO-CASE SERIES

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Introduction: Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have transformed the management of metabolic disease and obesity and are increasingly recognized for their beneficial effects in metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH). Emerging clinical trial data demonstrate improvement in hepatic steatosis, inflammation, and fibrosis with GLP-1RA therapy. The ESSENCE trial evaluating semaglutide in patients with MASH and F2-F3 fibrosis reported superior rates of MASH resolution and fibrosis improvement compared with placebo over 72 weeks. Similarly, tirzepatide has demonstrated histologic improvement in MASH and fibrosis regression in phase 2 studies. However, real-world histopathologic evidence of regression of advanced fibrosis and cirrhosis remains limited. **Case Presentation:** Case 1: A 62-year-old woman with type 2 diabetes mellitus underwent liver biopsy in 2022, demonstrating steatohepatitis with stage 4 fibrosis (NASH CRN NAS 3/8). She was treated sequentially with semaglutide for 4 years followed by tirzepatide for 1.5 years. Repeat liver biopsy showed patchy mild portal inflammation with marked reduction in fibrosis, consistent with regression from cirrhosis to stage 2-3 fibrosis. Case 2: A 58-year-old woman with a past medical history significant for type 2 diabetes mellitus was initially treated with exenatide and later transitioned to dulaglutide in 2015. Initial liver biopsy performed in 2020 demonstrated steatohepatitis with evolving inflammatory grade 2 disease, NAS 5/8, and extensive bridging fibrosis suspicious for evolving cirrhosis (stage 3-4 fibrosis). Repeat liver biopsy in 2026 demonstrated patchy bridging fibrosis with nodularity, chronic hepatitis with minimal activity, and no significant steatotic liver disease. The fibrosis pattern, in conjunction with the absence of ongoing steatotic liver disease and lack of elevated corrected sinusoidal pressure, was consistent with incomplete septal fibrosis of partially regressed cirrhosis. **Discussion:** These cases highlight the potential ability for incretin-based therapy-mediated reversal of cirrhosis. Data from bariatric cohorts have shown potential reversal of F4/Cirrhosis in some cases (ie Lassailly et al., Gastroenterology, October 2020), but there have not been reports of biopsy-proven reversal of MASH Cirrhosis with patients on Incretin therapy before. **Conclusion:** This case series highlights the potential for prolonged GLP-1RA therapy to induce meaningful histologic regression of advanced fibrosis and cirrhosis in patients with MASLD/MASH. These findings support emerging evidence that incretin-based therapies may alter the natural history of advanced steatotic liver disease and underscore the need for larger longitudinal studies evaluating fibrosis reversal and long-term hepatic outcomes in such patients.

3684 | PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS 10 DIAGNOSED IN AN INFANT: IDENTIFYING THIS RARE VARIANT AND MITIGATING FURTHER COMPLICATIONS WITH EARLY INTERVENTIONS

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Introduction: Progressive familial intrahepatic cholestasis (PFIC) comprises of a group of rare genetic liver disorders that result in cholestasis to progressive liver disease. We present a case of an infant with PFIC 10 whose genetic cholestasis panel reported the heterozygous variant in MYO5B gene. **Case Presentation:** A 4 week old infant presented with new onset jaundice and labs reported total/direct bilirubin of 8.3/5.6, Alanine Aminotransferase (ALT) 65, Aspartate Aminotransferase (AST) 84. Gamma Glutamyl Transferase (GGT), thyroid function tests, coagulation studies,newborn screen, urine analysis, Alpha 1 antitrypsin phenotype, ferritin were normal. Hepatobiliary scan (HIDA scan) was reported as negative for biliary atresia. Genetic cholestasis panel reported heterozygous pathogenic MYO5B gene variant. Patient was started on ursodiol and repeat labs improvement of bilirubin. **Discussion:** Advances in next-generation and whole-exome sequencing have expanded the classic classification of PFIC into distinct genetic subtypes. Among these, PFIC type 10 (PFIC10)-previously designated as PFIC6-is driven by pathogenic variants in the MYO5B gene on chromosome 18q21.1. Loss of function variants in MYO5B. impair the biliary bile acid secretion and cause toxic intracellular bile acid retention. Biliary bile acid transport defects in PFIC10 may be less absolute than in PFIC2, particularly in patients carrying biallelic missense variants, making them receptive to interventions that block intestinal bile acid reabsorption.Clinically, patients with PFIC10 can present with isolated cholestatic liver disease with potential to progress to severe hepatic and extra hepatic manifestations including severe congeni-

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tal enteropathies.Early precise molecular identification is vital to mitigate liver damage, support growth and treat severe pruritus symptoms with ursodeoxycholic acid (UDCA) or Rifampicin. When conventional medical therapies fail, surgical options such as partial external biliary diversion (PEBD) or liver transplantation are historically utilized.The therapeutic landscape for pediatric PFIC has evolved significantly with the introduction of selective ileal bile acid transporter (IBAT) inhibitors, such as odevixibat and maralixibat. Recent real-world pediatric data evaluating odevixibat specifically in PFIC10 children with refractory pruritus demonstrated robust clinical efficacy: odevixibat therapy led to rapid normalization of serum bile acids and total bilirubin within 3-6 months in the majority of patients. **Conclusion:** Documenting pediatric PFIC10 cases highlights the critical role of early genetic testing in low GGT cholestasis, allowing for prompt initiation of targeted IBAT inhibitor therapy and avoiding complications.

3685 | NODULAR REGENERATIVE HYPERPLASIA AND NON-CIRRHOTIC PORTAL HYPERTENSION ASSOCIATED WITH IGM HYPERGAMMAGLOBULINEMIA: A CASE REPORT

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Introduction: Nodular regenerative hyperplasia (NRH) is a rare hepatic vascular disorder marked by diffuse micronodular transformation of the parenchyma without fibrous septation. It represents a histologic correlate of porto-sinusoidal vascular disorder and an underrecognized cause of non-cirrhotic portal hypertension (NCPH), often associated with hematologic malignancy, autoimmune disease, or microvascular injury. We describe a case of NRH associated with marked IgM hypergammaglobulinemia due to smoldering Waldenström macroglobulinemia (WM), suggesting IgM-mediated microvascular obstruction as a contributing mechanism. **Case Presentation:** A 73-year-old man with remote alcohol use (>30 years abstinent) was referred for presumed cryptogenic cirrhosis after presenting with new ascites and an incarcerated umbilical hernia requiring emergent repair, alongside a 30-lb unintentional weight loss and marked muscle wasting. Ascitic fluid showed a serum-ascites albumin gradient >1.1 with low protein, and imaging revealed splenomegaly (15 cm) and paraesophageal varices. Serologic workup for autoimmune, metabolic, and viral liver disease was negative. MR elastography showed stage 2-3 fibrosis (3.95 kPa) without steatosis. Serum IgM was markedly elevated at 2,920 mg/dL (normal 37-286), prompting a hematologic diagnosis of smoldering WM without indication for treatment. Transjugular biopsy demonstrated NRH with mild zone 3 fibrosis and focal small portal vein attenuation, without significant inflammation. Hepatic venography showed diminutive hepatic veins with an HVPG of 7 mmHg. The patient improved on carvedilol, torsemide, and spironolactone. **Discussion:** This case illustrates NRH as a cause of clinically significant NCPH with preserved synthetic function. The temporal association between markedly elevated IgM and biopsy-confirmed NRH supports paraprotein-mediated microvascular injury and compensatory nodular hyperplasia, consistent with prior reports linking NRH to lymphoproliferative and monoclonal gammopathic disorders. Distinguishing NRH from cirrhosis carries important prognostic implications, given the risk of variceal hemorrhage, ascites, and spontaneous bacterial peritonitis despite normal liver synthetic function. **Conclusion:** NRH should be considered in patients with non-cirrhotic portal hypertension and unexplained hypergammaglobulinemia. This case supports IgM-mediated microvascular obstruction as a mechanism in smoldering WM and underscores the value of combined hepatologic and hematologic evaluation.

3686 | REFRACTORY ASCITES AS AN INITIAL PRESENTATION OF POEMS-CASTLEMAN DISEASE

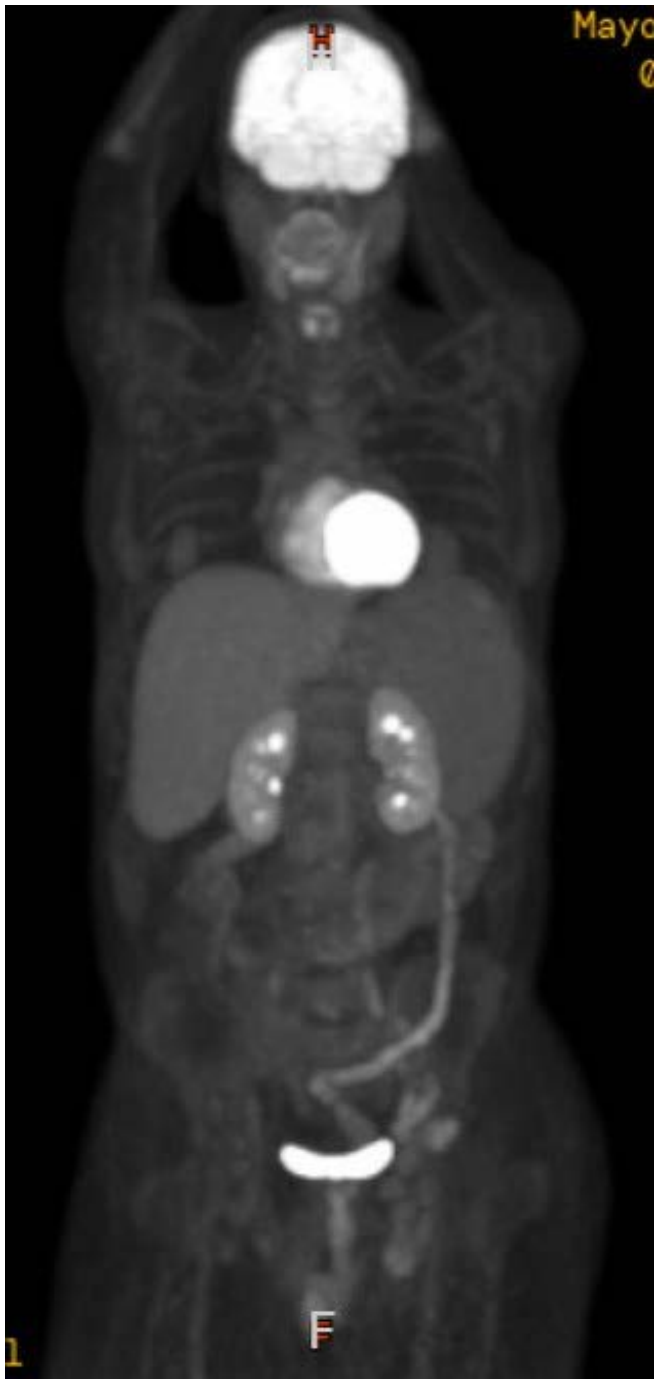
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[1]Mayo Clinic Rochester

Introduction: Ascites is a common clinical finding prompting referral to hepatology for evaluation of liver disease. Although ascites most commonly occurs in the setting of cirrhosis and portal hypertension, a comprehensive evaluation requires consideration for other etiologies. POEMS syndrome is a rare paraneoplastic disorder arising from a plasma cell neoplasm. Features of this syndrome include polyradiculoneuropathy, organomegaly, endocrinopathy, monoclonal protein elevation, and skin changes. Although the mechanism is not fully understood, ascites in POEMS syndrome is thought to arise from systemic vascular permeability related to cytokine release and elevated vascular endothelial growth factor. **Case Presentation:** A 44-year-old male presented for evaluation of recurrent ascites requiring frequent large-volume paracentesis and up to 60-pound weight loss. Physical examination revealed anasarca with notable cachexia and skin hy-

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perpigmentation. Laboratory testing showed normal liver chemistries and INR. Serum albumin was 3.2 g/dL. Imaging showed hepatosplenomegaly, abdominopelvic lymphadenopathy, and findings of volume overload with body wall edema and moderate ascites. Ascitic fluid studies showed albumin 2.3, total protein 3.6, and negative cytology. The calculated serum-ascites albumin gradient was 0.9. A transthoracic echocardiogram was unremarkable with normal right-sided pressures. An upper endoscopy demonstrated portal hypertensive gastropathy and endoscopic lymph node biopsies were unrevealing. Liver biopsy demonstrated minimal sinusoidal dilation and mild perivenular fibrosis and was otherwise unremarkable. PET-CT demonstrated normal FDG-avidity of mildly prominent mediastinal and abdominopelvic lymph nodes, liver, and spleen. Bone marrow biopsy noted normocellular bone marrow, increased erythropoiesis and 1% blasts. Fat aspirate was negative for amyloid. Ultimately, an excisional biopsy of a 1.8 cm left inguinal node revealed reactive lymphoid tissue with increased plasma cells, occasional follicles with nonspecific Castleman-like changes and high endothelial venules. Additional staining was negative for EBV and HHV-8. **Discussion:** Multidisciplinary review of the case with hematology, rheumatology, dermatology, and endocrinology culminated in a diagnosis of POEMS syndrome and idiopathic multi-centric Castleman disease. The patient received daratumumab, bortezomib, lenalidomide, and dexamethasone (D-VRd) with resolution of ascites and was referred for autologous stem cell transplantation (SCT). **Conclusion:** While myeloma-directed treatment followed by SCT has improved survival, volume overload related to capillary leak is a highly fatal manifestation of POEMS syndrome. Hepatologists may be the first to encounter patients with refractory ascites seen in POEMS syndrome and should be aware of the diagnostic considerations of this hematologic disorder.



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3687 | CONGENITAL INTRAHEPATIC PORTOSYSTEMIC SHUNT MASQUERADING AS HEPATIC ENCEPHALOPATHY IN AN ELDERLY PATIENT

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Introduction: Congenital intrahepatic portosystemic shunts (CIPSS) are rare vascular anomalies that divert portal blood directly into the systemic circulation, bypassing the liver metabolism. Most cases are diagnosed in childhood, whereas presentation with overt hepatic encephalopathy in an elderly patient without cirrhosis is exceedingly uncommon. **Case Presentation:** A 74-year-old man with hypertension and toxic multinodular goiter presented to the emergency department with unresponsiveness, impaired communication, and gait disturbance. Neurological evaluation revealed no structural or other neurological abnormality sufficient to explain the encephalopathy, except for hyperammonemia with a peak of 206 µg/dL. Liver function tests, viral and autoimmune hepatitis serology, and tumor markers were all normal, with no clinical findings suggestive of cirrhosis. Abdominal MRI/MRCP showed a 3x2 cm segment VII lesion with delayed enhancement, a relatively small left lobe, and normal biliary tree. Triphasic CT reclassified it as a portohepatic vascular shunt with aneurysmal dilatation (~37x30 mm), with segment IV atrophy and caudate hypertrophy consistent with chronic portosystemic shunting. Doppler confirmed a fistula between the right posterior portal branch and right hepatic vein with aneurysmal dilation; no ascites noted. Biopsy showed preserved architecture without fibrosis, excluding cirrhosis as the cause of HE. With antibiotics and HE therapy, consciousness improved, but HE recurred with intercurrent infections. After multidisciplinary review, resection and transplantation were unsuitable, and embolization was recommended as the definitive treatment. **Discussion:** CIPSS may present in advanced age with recurrent hyperammonemic HE despite preserved liver function and the absence of cirrhosis. The clinical presentation may closely resemble decompensated cirrhotic HE, while cross-sectional imaging may also initially raise concern for a focal hepatic neoplasm. Recognition of the vascular nature of the lesion, together with secondary morphological findings such as segmental hepatic atrophy and caudate lobe hypertrophy, was essential for establishing the diagnosis. Liver biopsy further demonstrated preserved hepatic architecture without fibrosis, helping exclude cirrhosis as the underlying cause. **Conclusion:** CIPSS is classically diagnosed in childhood, it may remain clinically silent for decades and first become symptomatic in elderly patients. In patients with otherwise unexplained hyperammonemic encephalopathy and no evidence of cirrhosis, CIPSS should therefore be considered in the differential diagnosis. Careful evaluation with vascular-phase imaging, supplemented by histopathological assessment when clinically indicated, can facilitate accurate diagnosis of this potentially treatable cause of non-cirrhotic hepatic encephalopathy.



3689 | HEPATIC AMYLOIDOSIS PRESENTING AS ABNORMAL LIVER ENZYMES WITH COEXISTENT NODULAR REGENERATIVE HYPERPLASIA

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Introduction: Hepatic amyloidosis is a rare, underrecognized cause of abnormal liver enzymes that can be missed on noninvasive testing. LECT2-type amyloidosis, from deposition of leukocyte chemotactic factor 2, disproportionately affects Hispanic patients and is predominantly reported as renal disease. NRH, a noncirrhotic vascular injury from microvascular obliteration, has rarely been described with hepatic amyloidosis, and coexistence with LECT2 amyloidosis is not well

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characterized. **Case Presentation:** A 62-year-old Hispanic woman with obesity, hyperlipidemia, hypertension, CKD stage 3, and rheumatoid arthritis (RA) was referred for chronically abnormal liver enzymes. ALT/AST peaked at 116/119 U/L, then normalized, while ALP persisted at 110-137 U/L. She was clinically compensated without jaundice, ascites, or stigmata of chronic liver disease. Ultrasound was unremarkable, but transient elastography showed grade 3 steatosis and F4-range liver stiffness, discordant with clinical status. ANA was 1:160, IgG 1797 mg/dL, and PBC workup was negative. Given inconclusive testing and concern for AIH, MASLD, or AMA-negative PBC, liver biopsy was performed, showing globular eosinophilic portal and lobular deposits with apple-green birefringence on Congo red stain, confirming amyloid. Immunostaining for serum amyloid A, kappa, and lambda light chains was negative, excluding AL and AA subtypes; findings were consistent with LECT2-type amyloidosis. Reticulin staining showed alternating hypertrophic and atrophic hepatocyte plates diagnostic of NRH, with no significant fibrosis, contrasting with the elastography-predicted F4 stage. **Discussion:** Amyloid deposition preferentially involves portal tracts and perisinusoidal spaces, producing a cholestatic pattern without hepatocellular necrosis, mirroring this patient's enzyme pattern. Noninvasive evaluation was misleading: F4-range elastography was histologically unsubstantiated, likely reflecting amyloid-driven viscoelastic change rather than true fibrosis. NRH has been linked to autoimmune disease, including RA, and rarely to systemic amyloidosis, but to our knowledge, this is the first reported case of coexistent LECT2 amyloidosis and NRH. RA and amyloid may have acted synergistically here, and absence of fibrosis should not reassure clinicians of low portal hypertension risk. **Conclusion:** Hepatic amyloidosis should be considered in Hispanic patients with unexplained cholestatic liver enzymes, especially when noninvasive fibrosis assessment is discordant with the clinical picture. This appears to be the first reported instance of these two entities occurring together, a finding with clinical relevance since noncirrhotic portal hypertension may develop independently of fibrosis stage. Liver biopsy remains essential when noninvasive evaluation is inconclusive.

3690 | A RARE AND LIFE-THREATENING CAUSE OF MULTISYSTEM INFLAMMATION: HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS

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Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a rare and potentially fatal hyperinflammatory syndrome characterized by uncontrolled activation of macrophages, histiocytes, and cytotoxic lymphocytes, resulting in progressive systemic inflammation and multiorgan dysfunction. HLH may occur as a primary genetic disorder or as secondary HLH triggered by malignancy, infection, autoimmune or autoinflammatory disease, immunodeficiency, or other underlying conditions. Although the HLH-2004 diagnostic criteria remain widely used, their application in adults presents challenges because of substantial clinical overlap with other critical illnesses, including sepsis and multiorgan dysfunction syndromes. **Case Presentation:** A 67-year-old man with hypertension and hyperlipidemia presented with recurrent seizure-like activity following one week of weakness, fatigue, and diarrhea. Evaluation revealed pancytopenia (WBC 2.1 K/μL, hemoglobin 9.9 g/dL, platelets 23 K/μL), hemolysis (haptoglobin <10 mg/dL, LDH 942 U/L), severe hyperbilirubinemia (total bilirubin 23.1 mg/dL), hepatic dysfunction, coagulopathy (INR 1.9), hypertriglyceridemia (692 mg/dL), and acute kidney injury (creatinine 3.49 mg/dL), raising concern for acute liver failure. Neurologic and infectious evaluations were unrevealing. Ferritin was 15,169 ng/mL; bone marrow biopsy showed hypercellularity with possible hemophagocytosis, while CT showed mild splenomegaly and ascites. Secondary HLH was suspected (H-score 220; 93-96% probability). Despite dexamethasone and etoposide, he developed progressive multiorgan failure requiring hemodialysis, mechanical ventilation, and vasopressors, ultimately dying after transition to comfort care. **Discussion:** HLH is a rare, rapidly progressive hyperinflammatory syndrome requiring early recognition to prevent irreversible organ damage and death. In adults, it may mimic sepsis, malignancy, infection, or acute liver failure (ALF). Hepatic involvement is common and may present with hepatocellular, cholestatic, or mixed injury. In patients with severe hepatic dysfunction, concurrent cytopenias, marked hyperferritinemia, hypertriglyceridemia, splenomegaly, or hemophagocytosis should prompt consideration of

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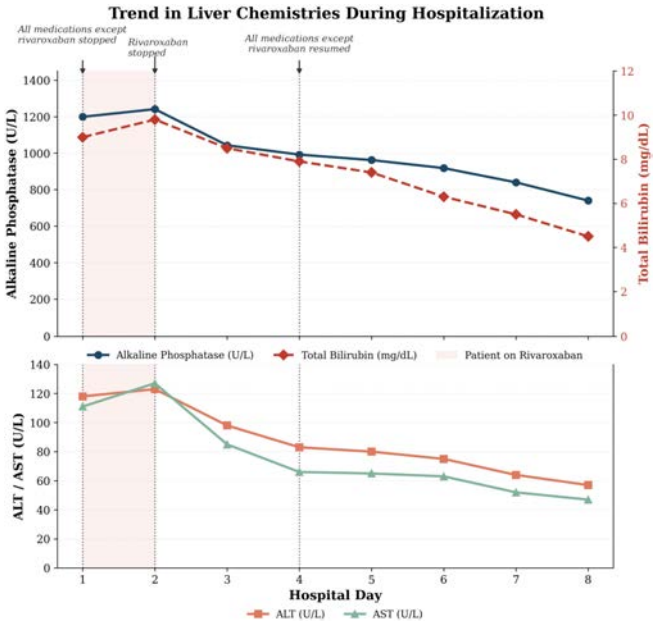
HLH. Diagnostic tools, including HLH-2004 criteria and the HScore, support early recognition. Treatment targets immune hyperactivation and underlying triggers, with dexamethasone and etoposide used in severe disease. **Conclusion:** Hemophagocytic lymphohistiocytosis is a potentially fatal hyperinflammatory disease whose main issue is that it can be difficult to recognize in adults due to its ability to mimic other types of illnesses. Earlier evaluation may prevent organ dysfunction or death in many, high-risk patients.

3691 | 3.5 YEARS LATER: AN UNUSUALLY DELAYED CASE OF RIVAROXABAN-INDUCED CHOLESTATIC LIVER INJURY

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Introduction: Idiosyncratic rivaroxaban-induced liver injury (DILI) is rare and classically rapid: published cohorts report onset between 2 and 180 days (median 15 days). We describe cholestatic hepatotoxicity emerging after 3.5 years of stable rivaroxaban use, exceeding the longest previously reported latency of 180 days. **Case Presentation:** A 56 year old woman with type 2 diabetes, hypertension, and peripheral arterial disease on rivaroxaban 20 mg daily for 3.5 years presented with jaundice and abdominal pain. She had been hospitalized 3 weeks earlier for similar findings, at which time all her medications were held (rivaroxaban 20 mg daily, insulin, lisinopril 40 mg daily, amlodipine 10 mg daily, no statin due to myalgias, and a recently started multivitamin). Liver function tests improved modestly; the culprit was thought to be the recently started multivitamin, which was thereafter discontinued. However, jaundice recurred within 2 weeks. Labs showed a hyper-cholestatic pattern: total bilirubin 9.0 mg/dL, GGT 1,919 U/L, alkaline phosphatase 1,199 U/L, ALT/AST 118/111 U/L, R-factor 0.4. Viral hepatitis serologies, AMA, and SMA were negative; ANA was weakly positive (1:80). Magnetic resonance cholangiopancreatography (MRCP) excluded biliary obstruction. Even during the second admission, rivaroxaban was initially dismissed as a cause given 3.5-year duration of use, but was incidentally held for liver biopsy. Biopsy showed neutrophilic cholangitis, ductal proliferation, and stage 2 fibrosis consistent with a biliary-pattern drug reaction. Within 48 hours of holding rivaroxaban, bilirubin and alkaline phosphatase improved dramatically, and continued to improve and normalize after resuming all other home medicines; she was transitioned to apixaban without recurrence of liver injury. **Discussion:** The offending agent here was identified only by accident: rivaroxaban was held for a procedure, not on clinical suspicion, since its long duration of use had already taken it off the differential. This incidental hold functioned as a de facto dechallenge and revealed the true cause after a prior admission had wrongly implicated a multivitamin. Once obstruction was ruled out on MRCP, biopsy showing neutrophilic cholangitis and ductal proliferation suggestive of biliary pattern drug reaction, along with the timeline of LFT resolution while rivaroxaban was held and all other medicines resumed, were essential to make the diagnosis. Published case series only report rivaroxaban-associated DILI latency up to 180 days; this case exceeds that window by over 1,000 days. **Conclusion:** Duration of use alone should not be used to rule out rivaroxaban from the differential for cholestatic liver injury once obstruction is ruled out; drug hold, for a procedure or otherwise, can function as diagnostic dechallenge and should prompt reassessment of causality. Biopsy is warranted whenever a biliary-pattern injury mimics cholangitis and imaging is unrevealing.



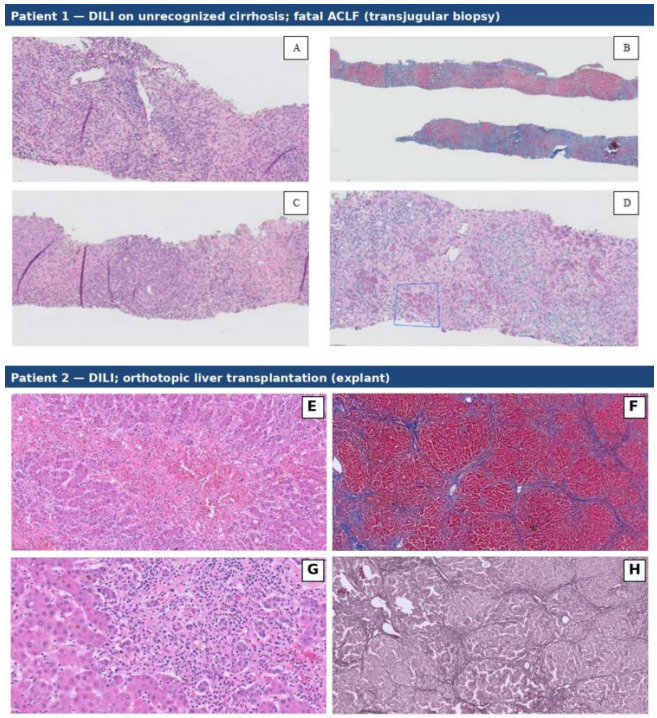
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3692 | VETERINARY ANTIPARASITICS FOR A SOCIAL-MEDIA "ANTICANCER" PROTOCOL: A TWO-PATIENT SERIES OF FENBENDAZOLE-ASSOCIATED ACUTE LIVER FAILURE

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Introduction: Drug-induced liver injury (DILI) is a leading cause of acute liver failure (ALF), yet veterinary antiparasitics are rarely implicated and are unapproved for human use. We present two women who developed severe DILI after self-administering fenbendazole (one with concurrent ivermectin) for a social-media "anticancer" protocol, a misinformation-driven hazard. **Case Presentation:** Both were women in their 60s with hypothyroidism. Patient 1 took ivermectin paste 1.87% for ~6 months and fenbendazole 444 mg daily for 2 months for cancer prevention, then developed severe acute hepatocellular injury (aspartate/alanine aminotransferase [AST/ALT] 2,924/3,396 U/L; total bilirubin 18.7 mg/dL; international normalized ratio [INR] 3.0). Autoimmune and infectious workups were unremarkable. Liver biopsy showed active hepatitis with ~70% confluent necrosis and stage 4 fibrosis; hepatic venous pressure gradient was 21 mmHg, revealing unrecognized cirrhosis (Figure 1). She progressed to acute-on-chronic liver failure (ACLF) with encephalopathy, renal and respiratory failure, and septic shock, was deemed transplant-ineligible, and died. Patient 2, with rheumatoid arthritis, took fenbendazole 444 mg daily for several months and presented with ALF (AST/ALT 1,079/767 U/L; INR 2.32). Endoscopic ultrasound and endoscopic retrograde cholangiopancreatography excluded obstruction and malignancy despite incidental gastric thickening and adenopathy; viral serologies were negative and antinuclear antibody was low-titer. She underwent liver transplantation; explant confirmed hepatocyte dropout necrosis and bridging fibrosis (Figure 1). **Discussion:** Fenbendazole, a veterinary benzimidazole, lacks human approval or efficacy data, yet online promotion as cancer therapy drives self-use; reported hepatotoxicity is rare and usually reversible. Ivermectin is generally hepato-safe but may injure the liver at prolonged veterinary doses. These cases extend the spectrum to biopsy-confirmed, life-threatening injury and show that outcome hinges on background liver health: DILI on silent cirrhosis precipitated fatal ACLF in Patient 1, whereas ALF in Patient 2, without cirrhosis, was salvaged by transplantation. Concurrent agents preclude single-agent causality but reflect real-world self-dosing. Management rests on early recognition, medication reconciliation probing supplements and veterinary products patients deem "natural," prompt drug withdrawal, and timely transplant evaluation. **Conclusion:** Unsupervised veterinary antiparasitics can cause DILI leading to acute liver failure and death. In unexplained liver injury, clinicians should suspect hepatotoxic exposures, take a supplement and veterinary-product history, stop offending agents, and engage hepatology early, while public-health efforts counter the driving misinformation.



3693 | CATASTROPHIC ECTOPIC VARICEAL HEMORRHAGE IN DECOMPENSATED ALCOHOL-RELATED CIRRHOSIS

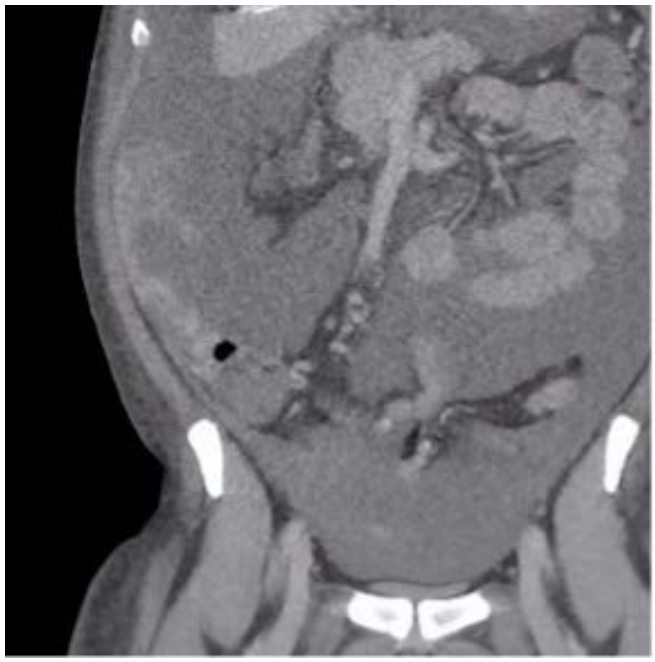
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Introduction: Ectopic varices are portosystemic collaterals outside the gastroesophageal region caused by portal hypertension. Though they account for only 1-5% of variceal bleeds, hemorrhage can be life-threatening and difficult to localize or manage. We present a case of ectopic colonic variceal hemorrhage following treatment of rectal varices. **Case Presentation:** A 45-year-old male with hepatitis C, decompensated alcohol-related cirrhosis with ascites, grade III esophageal varices, and rectal varices presented with

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lower GI bleeding, hemoglobin of 3, abdominal distension, nausea, emesis, and hematochezia. Sigmoidoscopy at an outside hospital showed bleeding rectal varices treated with band ligation. He was transferred for ongoing bleeding. Initial vitals: blood pressure 99/67, heart rate 119, hemoglobin 6 despite 2 units transfused, MELD 33. Course was complicated by shock and worsening distension, with repeat hemoglobin of 5 unresponsive to transfusion. Computed tomography angiography (CTA) showed hemoperitoneum with active extravasation along the right paracolic gutter, concerning for ectopic ascending colonic variceal hemorrhage. He underwent emergent embolization of varices surrounding the cecum. His abdomen then became markedly distended and rigid; drain placement evacuated 13 liters of blood. Despite massive transfusion, he expired of hemorrhagic shock. **Discussion:** This case highlights the challenges of ectopic intra-abdominal variceal hemorrhage. Unlike gastroesophageal varices, ectopic colonic varices are often endoscopically inaccessible and may evade angiographic localization due to diffuse collateralization. No established guidelines exist; treatment typically requires multidisciplinary collaboration among gastroenterology, interventional radiology, and surgery. Options include embolization, TIPS, and balloon-occluded retrograde transvenous obliteration; our patient was not a TIPS candidate given MELD of 33. This case illustrates the limits of embolization in advanced portal hypertension: extensive embolization risks bowel ischemia, while isolated embolization without portal decompression may raise collateral pressures and precipitate recurrent hemorrhage. **Conclusion:** Ectopic colonic varices are a rare but potentially fatal cause of GI hemorrhage in decompensated cirrhosis, requiring heightened suspicion, early multidisciplinary care, and recognition of limited options when portal decompression is not feasible.



3694 | AN UNUSUAL CASE OF OBSTRUCTIVE JAUNDICE SECONDARY TO CLONORCHIASIS IN A NON-ENDEMIC SETTING

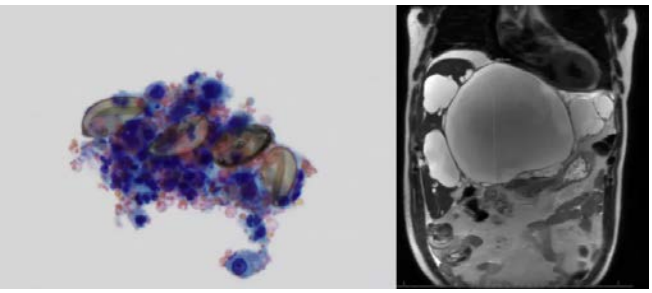
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Introduction: *Clonorchis sinensis* is a fish-borne trematode endemic to East/Southeast Asia, transmitted via raw or undercooked freshwater fish. Chronic infection causes mechanical and inflammatory injury of the intra- and extrahepatic bile ducts and is associated with obstructive jaundice, cholecystitis, cholelithiasis, cholangitis, and is a well-established risk factor for cholangiocarcinoma. **Case Presentation:** A 69-year-old Cantonese-speaking male who immigrated from Southern China in 2018 with a history of chronic Hepatitis B, cholelithiasis, and active tobacco use, was referred to Hepatology for elevated LFTs (AST 357, ALT 142, ALP 607, Tbili 2.7) and ultrasound showing numerous large hepatic cysts in the setting of recent travel to China. CT showed hepatic cysts up to 15cm and pancreatic duct enlargement. Initial workup was notable for equivocal *E. histolytica* IgG and positive *Echinococcus* IgG with mild eosinophilia. Infectious Disease felt *E. histolytica* IgG was a false positive, and confirmatory *Echinococcus* EIA/western blot was neg-

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ative. No treatment was given, and he was eventually lost to follow-up.Seven months later, he presented with a week of epigastric discomfort, nausea, vomiting, and diarrhea. Exam showed epigastric tenderness and scleral icterus. Initial Tbili was 16.9, peaking at 32.4 during this admission. MRI showed hepatic cysts (largest 17cm) with marked bilateral intrahepatic ductal dilation, likely from mass effect. IR drainage of the largest cyst yielded 2.3L of non-purulent bilious fluid and Tbili fell to 14.9 by discharge. Cytology showed eggs consistent with *Clonorchis* or *Opisthorchis*, though travel history to China supported a diagnosis of clonorchiasis. He completed 3 days of praziquantel, with eventual Tbili normalization. The drain was removed two months later and follow-up MRI and CT showed stable to mildly enlarged cysts. **Discussion:** We report a case of obstructive jaundice due to clonorchiasis in a Chinese immigrant presenting in a non-endemic region, illustrating the diagnostic challenge posed by parasitic biliary disease outside endemic areas. *C. sinensis* infection can be diagnosed by detection of eggs on stool O&P or cytologic examination of bile, serology, or PCR. Praziquantel remains first-line management, with procedural intervention indicated for hepatobiliary complications. Long-term cholangiocarcinoma surveillance is also important given the malignant potential of chronic infection. **Conclusion:** While less common in non-endemic regions, clonorchiasis remains an important diagnostic consideration for obstructive jaundice, especially among immigrant or refugee populations of the US that may carry chronic, undiagnosed infections from their country of origin, particularly given its strong association with malignancy.



3695 | A PLASMA CELL-RICH DIAGNOSTIC TRAP: HEPATIC BARTONELLOSIS MIMICKING AUTOIMMUNE HEPATITIS

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Introduction: Hepatic bartonellosis is rare, and predominantly described in the literature as a granulomatous hepatitis or bacillary peliosis. This case is notable for a distinctly different plasma cell-rich, autoimmune hepatitis (AIH)-like pattern that could have led to inappropriate escalation of immunosuppression during occult infection. **Case Presentation:** A 40-year-old woman with immunoglobulin (Ig) A nephropathy on tacrolimus and mycophenolate presented with 3 weeks of daily fever to 40.5°C and headache. Chest and abdominal computed tomography showed lower-lobe consolidation, mediastinal/hilar and periportal adenopathy, and no biliary obstruction. Vancomycin/piperacillin-tazobactam did not improve fever. Alkaline phosphatase (ALP) rose from 188 to 470 U/L as aminotransferases fell and bilirubin remained normal. The differential included infection, AIH, drug injury, and malignancy. Given concern for infection, mycophenolate was held and immunosuppression was not intensified. Pruritic wheals developed, and skin biopsy showed leukocytoclastic vasculitis. Liver biopsy showed plasma cell-rich portal/lobular hepatitis, interface activity, and central perivenulitis supporting AIH, plus a rare non-necrotizing granuloma suggesting infection. Three weeks post-discharge, further history revealed >15 cats, home fleas, and a possible cat scratch. B. henselae IgG was >1:1024 and IgM 1:128. Blood polymerase chain reaction was negative, supporting probable hepatic B. henselae infection. Doxycycline initially improved fever. Six weeks later, recurrent fever, headache, and weakness prompted azithromycin. Four months later, persistent fever, fatigue, and headaches despite dual therapy prompted rifampin and thus tacrolimus dose escalation. After 11 months of total therapy, symptoms resolved and aminotransferases normalized. **Discussion:** Most prior reports of hepatic bartonellosis describe granulomatous hepatitis or bacillary peliosis, whereas this case was dominated by plasma cell-rich portal and lobular hepatitis with interface activity and central perivenulitis, closely resembling AIH. The cholestatic biochemical pattern, persistent fever, adenopathy, vasculitis, and rare granuloma created a clinicopathologic mismatch that favored infection over primary AIH. Because Bartonella was not demonstrated in liver tissue, the diagnosis remains probable rather than microbiologically confirmed. **Conclusion:** This case highlights an underrecognized AIH-like phenotype of hepatic bartonellosis. In immunosuppressed patients, interpreting plasma cell-rich interface hepatitis as primary AIH without reconciling discordant systemic and cholestatic findings may lead to inappropriate escalation of immunosuppression and worsening of occult infection. Recognition of this mimic should prompt careful exposure history and targeted Bartonella evaluation before immunosuppression is intensified.

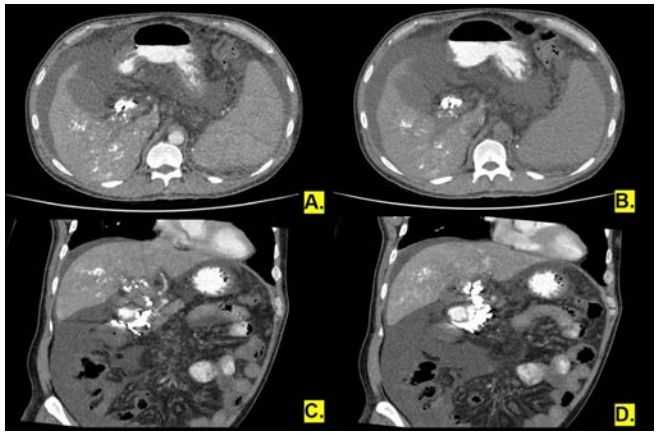
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3696 | SPONTANEOUS HEMOPERITONEUM REVEALING MULTIFOCAL HEPATOCELLULAR CARCINOMA IN A PATIENT WITHOUT PREVIOUSLY RECOGNIZED CIRRHOSIS

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Introduction: Spontaneous hemoperitoneum is an uncommon but severe presentation of hepatocellular carcinoma (HCC), particularly in patients without previously recognized cirrhosis. Rapid identification of neoplastic bleeding is essential, as clinical stability may mask significant intraperitoneal hemorrhage. **Case Presentation:** A 70-year-old man with hypothyroidism and remote alcohol use presented with sudden abdominal pain and rapidly progressive distension. He was pale but hemodynamically stable. Laboratory evaluation showed acute anemia (Hb 7.8 g/dL), marked transaminase elevation (AST 880 U/L, ALT 762 U/L), cholestasis (ALP 310 U/L, GGT 420 U/L), and INR 1.4. Viral hepatitis serologies were negative, and no clinical features of cirrhosis were present.Contrast-enhanced CT revealed a large irregular hepatic mass involving multiple segments with heterogeneous arterial enhancement, portal-phase hyperenhancement, and delayed washout. Multiple smaller lesions with similar characteristics were scattered throughout the liver. High-density intraperitoneal fluid consistent with hemoperitoneum was present, with a subcapsular segment VI lesion suspected as the bleeding source. Figure 1. AFP measured 125 ng/mL.Because transarterial embolization was unavailable and the patient remained stable, conservative management was pursued. Biopsy of the dominant lesion confirmed well-differentiated HCC. Given multifocal tumor burden and recent hemorrhage, curative options were not feasible, and care focused on supportive management. **Discussion:** Spontaneous hemoperitoneum due to HCC rupture is rare and may be the first manifestation of underlying liver disease. Subcapsular hypervascular tumors are particularly prone to rupture. In noncirrhotic patients, multifocal hepatic lesions may initially mimic metastatic disease, and LIRADS criteria cannot be applied, making histologic confirmation essential. This case also illustrates challenges in resource-limited settings where embolization is unavailable, requiring reliance on conservative stabilization even in potentially life-threatening hemorrhage. **Conclusion:** Spontaneous hemoperitoneum may reveal multifocal HCC in patients without previously recognized cirrhosis. Maintaining a broad differential and recognizing imaging patterns of tumor rupture are critical to avoid misdiagnosis and guide timely management.



3697 | STAGE IVB CLASSIC HODGKIN LYMPHOMA PRESENTING AS ACUTE CHOLESTATIC LIVER FAILURE: DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN FRAIL COMORBID PATIENTS

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Introduction: Hepatic involvement in classic Hodgkin lymphoma (cHL) as the primary manifestation of acute cholestatic liver failure is rare and associated with a dismal prognosis. Diagnosing lymphomatous liver infiltration in elderly multimorbid patients is clinically complex, as features can mimic post-cardiac passive congestion, sepsis, or drug-induced liver injury. **Case Presentation:** An 82-year-old male with end-stage renal disease on hemodialysis, JAK2/TET2 polycythemia vera, and severe aortic stenosis status-post transcatheter aortic valve replacement (TAVR) presented with progressive jaundice, dark urine, and abdominal distension. Laboratory workup revealed marked cholestatic liver failure with total bilirubin rising rapidly from 5.8 to 19.5 mg/dL, alkaline phosphatase of 655 U/L, AST 73 U/L, ALT 57 U/L, and coagulopathy (INR 1.6). Leukocytosis surged from 15.1 to 46.2 × 10^3/µL, with persistent thrombocytopenia (68-95 × 10^3/µL). Viral hepatitis serologies were negative. Abdominal imaging showed mild hepatomegaly, severe splenomegaly, portal hypertension (portal vein 14 mm), and extensive retroperitoneal/periportal lymphadenopathy without biliary ductal dilatation. A unifying diagnosis of Stage IVB classic Hodgkin lymphoma with hepatic infiltration was established. Given multi-organ failure and frailty, curative-intent chemoimmunotherapy (nivolumab-AVD/gemcitabine) was unsafe. Management focused on a time-limited dexam-

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ethasone prephase (20 mg daily) to mitigate hepatic infiltration, alongside rasburicase for high-risk tumor lysis syndrome (uric acid 14.3 mg/dL) and hemodialysis. **Discussion:** Managing hepatic cHL in elderly patients with multi-organ failure presents a clinical paradox: the necessity for rapid cytoreduction to reverse liver failure versus the high risk of treatment-related mortality with standard chemotherapy. Diagnostic vigilance is required to identify lymphomatous infiltration when imaging is non-specific. Multidisciplinary coordination between hepatology, oncology, and palliative care is essential to navigate these complexities in frail populations. **Conclusion:** In cases of acute cholestatic liver failure due to suspected lymphoma in fragile patients, a corticosteroid prephase serves as a critical diagnostic and therapeutic bridge. This approach facilitates stabilization and helps clinicians assess the potential for organ recovery before committing to more intensive, curative-intent interventions.

3698 | WHEN TIMING MATTERS: REASSESSING CEFTRIAXONE AS THE CAUSE OF SEVERE ACUTE HEPATOCELLULAR INJURY

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Introduction: Acute severe hepatocellular injury after antibiotic exposure is often labeled drug-induced liver injury (DILI), but causality can be misleading when multiple medications are administered over a short interval. **Case Presentation:** A 46-year-old woman with type 2 diabetes, dyslipidemia, prior Roux-en-Y gastric bypass, and cholecystectomy presented with abdominal pain, nausea, and vomiting. CT showed ileus versus early partial small-bowel obstruction with hepatomegaly and steatosis. Admission liver chemistries were normal (AST 17 U/L, ALT 25 U/L, ALP 99 U/L, total bilirubin 0.2 mg/dL). Within 24 hours, AST increased to 1,633 U/L and ALT to 2,013 U/L, with ALP 241 U/L and bilirubin 2.2 mg/dL (R ratio 20.9). INR remained normal, and there was no encephalopathy. Hepatitis A-C serologies, ANA, smooth-muscle antibody, ceruloplasmin, CK, lactate, cultures, and hepatic Doppler were unrevealing. Ceftriaxone was initially suspected; however, the first dose preceded the measured peak by only 11.5 hours, and a second dose was administered after the peak while aminotransferases were already declining, without rebound. A serum acetaminophen concentration was detectable at 11.3 µg/mL despite no inpatient acetaminophen administration, suggesting unrecognized pre-admission exposure. **Discussion:** Updated RUCAM classified ceftriaxone only as possible, not probable, DILI. The short latency, continued biochemical improvement despite repeat ceftriaxone exposure, and concurrent acetaminophen exposure weakened the case for ceftriaxone causality. Liver chemistries improved without specific therapy to AST 122 U/L, ALT 472 U/L, ALP 155 U/L, and bilirubin 0.7 mg/dL by discharge as the bowel process resolved. **Conclusion:** This case highlights the importance of reconstructing medication timing and enzyme kinetics before assigning DILI to the most recently administered drug. A detectable late acetaminophen level should be interpreted in clinical context and should not be dismissed solely because it is below a laboratory toxic threshold.

3699 | HIDE AND SEEK: AN ASYMPTOMATIC HEPATIC SARCOID-LIKE REACTION AFTER ICI TREATMENT IN RENAL CELL CARCINOMA

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Introduction: Immune checkpoint inhibitors (ICIs) have revolutionized the landscape of cancer treatment. However, many patients develop immune-related adverse events (iRAEs) including rare sarcoid-like reactions (SLR) which may present with histologic and organ involvement patterns similar to sarcoidosis. Hepatic SLRs present as a nonspecific granulomatous hepatitis which carry a broad differential including infectious and noninfectious etiologies. We present a unique case of an asymptomatic hepatic SLR after pembrolizumab treatment in a patient undergoing treatment for renal cell carcinoma (RCC). **Case Presentation:** A 63-year-old woman with a history of RCC received 4 cycles of adjuvant pembrolizumab (Oct 2024-Dec 2024) and stopped treatment due to ICI-induced edema. She was found to have sub-centimeter liver lesions on abdominal MRI in March 2026. She had no evidence of portal hypertension on imaging, and she remained asymptomatic. On exam, she was afebrile, without jaundice, with a soft, non-distended abdomen. Liver biopsy demonstrated coalesced granulomas with surrounding bands of fibrosis and mild macrovesicular steatosis without steatohepatitis. Infectious workup was negative for QuantiFERON tuberculosis testing and hepatitis serologies. Her laboratory tests were significant for AST 22 U/L, ALT 11 U/L, total bilirubin 0.5 mg/dL, platelet count 260 x 109/L, and ACE enzyme 34 U/L. Given her asymptomatic presentation with negative infectious work-up and normal liver

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chemistries, a decision was made to defer steroids and monitor with surveillance imaging. **Discussion:** Hepatic granulomas require histopathology for diagnosis and treatment is guided by underlying etiology, impact on liver function, and clinical symptoms. The pattern of granulomas seen on pathology may guide clinical work up. Per guidelines, patients found to have incidental granulomas on biopsy may undergo monitoring of liver enzymes and synthetic function every 6 to 12 months. Our patient did not meet Common Terminology Criteria for Adverse Events (CTCAE) for ICI-related liver toxicity. Therefore, ongoing multidisciplinary monitoring was pursued, and steroids were deferred. She previously experienced a mediastinal lymph node SLR based on CT imaging, which resolved without intervention. SLRs secondary to immunotherapy have previously been correlated with improved overall survival due to improved response to immunotherapy. However, it is still important to investigate hepatic granulomas with tissue biopsy to confirm suspected ICI-related SLRs and rule out progression of malignancy or other etiologies. **Conclusion:** To our knowledge, this is the first reported case of a patient with a history of renal cell carcinoma previously treated with pembrolizumab found to have granulomatous hepatitis consistent with a hepatic SLR with normal liver chemistries. Ongoing studies are needed to determine long-term outcomes related to asymptomatic hepatic SLR.

3700 | ELAFIBRANOR-ASSOCIATED DRUG-INDUCED LIVER INJURY IN PRIMARY BILIARY CHOLANGITIS: A CASE REPORT

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Introduction: Primary biliary cholangitis (PBC) is an autoimmune cholestatic liver disease for which ursodeoxycholic acid (UDCA) remains first-line therapy, with additional agents used in inadequate responders. Elafibranor is a newer therapeutic option for patients with uncontrolled disease despite UDCA. We present a patient with PBC who developed progressive liver injury while receiving elafibranor, raising concern for drug-induced liver injury (DILI). **Case Presentation:** A 55-year-old female with elevated liver enzymes (LFTs) in a mixed pattern since 2020 was diagnosed with PBC in 03/2024 (Table 1) based on elevated ALP and AMA. The patient was intolerant to UDCA at 13-15 mg/kg/day due to dizziness and was maintained on a lower dose. Obeticholic acid (OCA) was added in 06/2024 with improved LFTs but was stopped due to worsening pruritus. Elafibranor 80 mg daily was started in 06/2025. Routine labs in 04/2026 and 08/2026 showed progressive worsening of LFTs on admission. Patient had Roux-en-Y gastric bypass surgery in 5/2026, and no antibiotics were given at that time. Viral, hereditary, and autoimmune serologies were negative. No new medications or supplements were reported. Elafibranor was discontinued, and LFTs subsequently started improving. MRI and HIDA showed no biliary obstruction and mild biliary dyskinesia, which was not deemed clinically significant by Transplant Surgery. Liver biopsy (Figure 1) showed a disproportionate degree of inflammation consistent with PBC with bridging fibrosis but with an overlapping process such as DILI, without features of autoimmune hepatitis. The patient was treated with a taper of oral prednisone 40 mg, with improving liver enzymes. An attempt was made to increase UDCA to 13-15 mg/kg/day (1500 mg), but she remained intolerant and was continued at a 1000 mg dose. **Discussion:** The temporal association, lack of other compelling etiologies, improvement in liver enzymes after drug discontinuation, and liver biopsy findings all support the diagnosis of elafibranor-induced DILI. It is too early in the clinical course to determine the long-term outcome of the drug in this patient. While prior trials have raised suspicion for DILI associated with elafibranor, none have confirmed it. To our knowledge, this is the first published real-world case of clinically significant elafibranor-induced DILI in a patient with established PBC and reinforces the need for close monitoring of patients starting this medication. This case highlights the complexities of PBC treatment and the risk of progression of fibrosis if inadequately treated. Further reports may help better define the expected course after drug discontinuation and the mechanism of DILI. **Conclusion:** This case highlights the challenge of recognizing DILI in patients with PBC and the importance of monitoring liver biochemistry after initiation of new therapies. DILI should be considered when liver enzymes worsen unexpectedly after other causes are excluded.

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Date	AST (U/L)	ALT (U/L)	ALP (U/L)	Total Bilirubin (mg/dL)	Context
03/08/24	99	76	404	0.8	PBC diagnosed, Urso started 1500mg daily
06/2024	No labs	No labs	No labs	No labs	OCA started, UDCA 500mg daily
4/21/25	48	60	174	0.5	OCA stopped
6/5/2025	71	63	201	0.5	Elafibrinor 80mg/d started, UDCA 1000mg
9/9/25	49	52	216	0.5	No med changes
4/14/26	106	119	515	0.8	No Med changes
7/29/26	450	491	919	2.0	Elafibranor discontinued, UDCA 100
8/4/26	276	389	835	2.1	Liver Biopsy reported, Started Prednisone 40mg daily, UDCA 1000
8/5/26	242	358	777	1.7	Patient discharged with advice to get labs following week on Prednisone taper

Table 1. Serial liver biochemistry from diagnosis through treatment course. AST, ALT, ALP, and total bilirubin are shown alongside contemporaneous medication changes, illustrating an initial biochemical response to ursodeoxycholic acid (UDCA) and obeticholic acid (OCA), a subsequent trial of elafibranor, and a biochemical flare after elafibranor was discontinued, prompting liver biopsy and initiation of prednisone with UDCA dose escalation.

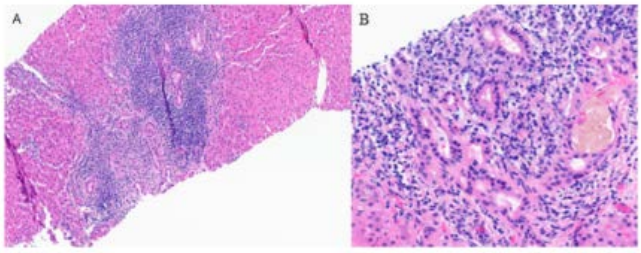


Figure 1. Liver biopsy, H&E stain. (A) Low-power view showing an adequate sample with multiple portal tracts, several of which show dense lymphocytic portal inflammation with a mild ductular reaction at the interface and preserved lobular architecture. (B) High-power view of a portal tract demonstrating a predominantly lymphocytic infiltrate with lymphocytic cholangitis of the bile duct epithelium, without convincing duct loss, granulomas, or significant plasma cell population. Trichrome staining (not shown) demonstrated fibrous expansion of most portal tracts with focal portal-to-portal bridging, consistent with Ishak stage 3 fibrosis.

4613 | UNUSUAL PRESENTATION OF KRATOM TOXICITY CAUSED RHABDOMYOLYSIS, COMPARTMENT SYNDROME, ACUTE LIVER FAILURE AND RENAL FAILURE

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Introduction: Kratom is an herbal extract derived from *Mitragyna speciosa*, a tropical evergreen tree native to Southeast Asia. It has dose-dependent stimulatory and sedative effects and is increasingly used in the United States for pain management and opioid withdrawal. We report a rare case of H09 Kratom-associated rhabdomyolysis complicated with compartment syndrome, acute liver failure and renal failure (AKI), highlighting potential mechanisms of severe multisystem toxicity. **Case Presentation:** A 32-year-old male with substance use disorder in sustained remission, who initiated H09 Kratom use 6 months ago (reportedly twice a week), was brought into the ER after being found unresponsive at home. On arrival, he was awake, alert, and complained of severe lower back and bilateral extremity pain. Initial Labs were notable for WBC 33.12, hemoglobin 18.2, Creatine Kinase (CK) of 25854 units/L, alanine aminotransferase/aspartate aminotransferase (ALT/AST) 988/1400 units/L, alkaline phosphatase (ALP) 78 units/L, creatinine 1.65. The patient subsequently developed hepatic encephalopathy, West--Haven grade 1, with peak ALT/AST 825/2188 units/L, ALP 50 units/L, elevated INR 1.48, and peak CK of 45508 units/L. Hospital course further complicated by AKI, reduced urine output, peak creatinine 8.2 (Figure 1). Despite aggressive intravenous fluid resuscitation, the patient developed progressive paresthesia, numbness, and inability to dorsiflex both feet. CT pelvic showed extensive intramuscular and peri-muscular edema involving the bilateral gluteal, deep back, and adductor musculature, concerning for compartment syndrome. Emergent fasciotomy of the gluteal-iliotibial region was performed. Hemodialysis was initiated because of progressive renal failure. **Discussion:** The mechanism of kratom causes rhabdomyolysis remains unclear. It has been proposed that mitragynine and 7-hydroxy mitragynine, the main compounds in Kratom, may stimulate α-adrenergic receptors, leading to vasoconstriction, muscle ischemia, and subsequent rhabdomyolysis. There are two proposed mechanisms for rhabdomyolysis causing acute liver injury: 1) release of AST, ALT, and LDH from damaged skeletal muscle rather than true hepatocellular injury, with AST disproportionately elevated because of higher skeletal muscle concentration (Figure 1A); 2) True hepatic injury caused by oxidative stress, ischemia/hypoperfusion, and proteases released from injured muscle. AKI is likely due to synergistic mechanisms including intrarenal vasoconstriction, direct tubular toxicity, and tubular obstruction. **Conclusion:** This case highlights a rare presentation of Kratom toxicity complicated by severe rhabdomyolysis, compartment syndrome, acute liver failure, and AKI requiring hemodialysis. It is essential for physicians to recognize the effect of Kratom overdose and be familar with treatment. There is no antidote for kratom. The treatment is mainly supportive care.

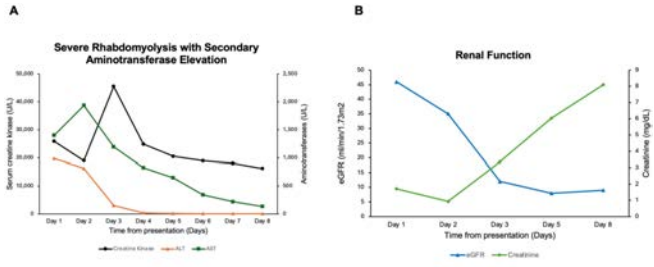


FIGURE1: (A) Severe rhabdomyolysis associated with marked aminotransferase elevation, with AST predominance (AST > ALT). (B) Elevated creatinine and declined eGFR consistent with acute kidney injury.

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4614 | BEYOND BILIARY OBSTRUCTION: PERSISTENT CHOLESTATIC JAUNDICE LEADING TO LIVER TRANSPLANTATION IN AUTOIMMUNE HEPATITIS AND METABOLIC DYSFUNCTION-ASSOCIATED STEATOHEPATITIS

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Introduction: Persistent cholestatic jaundice can present a diagnostic challenge when biliary abnormalities do not fully explain progressive liver dysfunction. Autoimmune hepatitis (AIH) may present atypically, while concomitant metabolic dysfunction-associated steatohepatitis (MASH) can further obscure the underlying etiology. We present a patient initially managed for biliary obstruction whose persistent cholestasis ultimately revealed advanced liver disease requiring transplantation. **Case Presentation:** A 63-year-old woman with obesity, diabetes mellitus, hypertension, and dyslipidemia presented with progressive jaundice and a cholestatic pattern of liver injury. Initial magnetic resonance cholangiopancreatography (MRCP) demonstrated biliary strictures, prompting endoscopic retrograde cholangiopancreatography (ERCP) with stone removal and stent placement. Despite intervention, hyperbilirubinemia persisted. She was discharged but subsequently returned with worsening jaundice and total bilirubin >14 mg/dL. Repeat MRCP showed no evidence of persistent obstruction. Given progressive cholestasis despite biliary intervention, an intrahepatic etiology was suspected. Liver biopsy and clinical evaluation supported AIH with concomitant MASH. Her liver disease progressed to advanced hepatic dysfunction with a MELD score of 26, prompting evaluation for transplantation. She subsequently underwent orthotopic liver transplantation. Explant pathology demonstrated cirrhosis with cholestasis, bile plugs, bile ductular proliferation, chronic portal inflammation, and macrovesicular steatosis, without viral cytopathic effect, granulomas, or neoplasia. Her post-transplant course was complicated by a biliary anastomotic stricture requiring hepaticojejunostomy, with subsequent normalization of liver synthetic function and excellent graft function. **Discussion:** This case highlights the importance of reconsidering the underlying diagnosis when cholestasis persists despite appropriate treatment of an identified biliary process. However, initial imaging demonstrated biliary abnormalities, persistent hyperbilirubinemia after endoscopic intervention suggested that obstruction alone did not explain the progressive liver dysfunction. AIH may present atypically, and coexisting MASH can further complicate recognition of the underlying liver disease. Liver biopsy was pivotal in identifying an intrahepatic process and facilitating timely referral for transplantation. **Conclusion:** Persistent cholestatic jaundice despite adequate biliary intervention should prompt evaluation for concomitant intrahepatic liver disease. This case demonstrates how AIH with coexisting MASH may masquerade as persistent biliary disease and underscores the role of liver biopsy when the clinical course is not adequately explained by biliary findings.

4615 | BEYOND A VARIANT OF UNCERTAIN SIGNIFICANCE: LIVER DISEASE ASSOCIATED WITH A HETEROZYGOUS ABCB4 VARIANT IN AN ADULT

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Introduction: In adults, persistently elevated liver function tests (LFT) may remain unexplained despite standard evaluations. Although biallelic ABCB4 variants cause Progressive Familiar Intrahepatic Cholestasis (PFIC) type 3, heterozygous variants are increasingly recognized as part of the spectrum of adult cholestatic liver disease. **Case Presentation:** A 27-year-old woman with hypothyroidism was evaluated for abnormal LFT since age 16. She had no history of jaundice, decompensated liver disease, significant alcohol use, hepatotoxic supplements, or risk factors for viral hepatitis. No history of gastrointestinal symptoms. Initial testing showed alkaline phosphatase (ALP) 182 U/L, aspartate aminotransferase (AST) 142 U/L, alanine aminotransferase (ALT) 240 U/L, total bilirubin 0.5 mg/dL, and albumin 4.7 g/dL. Abdominal ultrasound was normal. Antinuclear antibody was negative. Anti smooth muscle antibodies were transiently positive 1:20, with normal IgG, and negative IgG4. Tissue transglutaminase immunoglobulin A (tTG-IgA) was >110 U/mL. Endoscopy with duodenal biopsy confirmed celiac disease, and a gluten-free diet (GFD) was initiated. Magnetic resonance cholangiopancreatography with elastography showed no fibrosis and mild, nonspecific peripheral intrahepatic duct dilatation. Endoscopic ultrasound and retrograde cholangiopancreatography confirmed focal right intrahepatic duct dilatation (2-3 mm); peripheral or small-duct primary sclerosing cholangitis was considered. Repeat liver biopsy was nondiagnostic, showing possible patchy sinusoidal dilatation. LFT remained elevated after 1 year of GFD despite histologic resolution of celiac disease. Ursodiol 500 mg nightly was started, followed by progressive LFT normalization. Genetic testing was or-

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dered to rule out PFIC. It identified a heterozygous pathogenic variant in ABCB4 gene, c.475C>T, p.Arg159*, which has been described in homozygous and compound heterozygous state in PFIC type 3. **Discussion:** Although autosomal recessive PFIC3 requires biallelic ABCB4 variants, there is some evidence that heterozygous variants in ABCB4 are associated with increased risk for liver diseases with variable penetrance and expressivity. This case emphasizes that variants in genes traditionally associated with pediatric PFIC should not be dismissed in adults solely because they are heterozygous, as these mutations are increasingly recognized as clinically relevant contributors to adult-onset cholestasis, biliary injury, fibrosis, and intrahepatic cholestasis of pregnancy. **Conclusion:** This case supports incorporating a cholestasis-focused gene panel into the evaluation of selected adults with otherwise unexplained persistently elevated LFT, as heterozygous PFIC-associated variants may still be clinically meaningful, and therapeutic options may be available.

	07/17/2024	08/01/2024	11/4/2024	01/07/2025	12/08/2025	06/16/2026	8/12/2026
Total bilirubin (mg/dL)	0.2	0.2	0.2	0.2	0.3	0.3	0.3
Alkaline phosphatase (U/L)	252	229	95	103	79	81	92
AST (U/L)	123	119	40	36	29	27	29
ALT (U/L)	252	308	53	49	35	35	34
Total protein (g/dL)	7.2	7.2	6.9	7.4	6.8	7.6	7.6
Albumin (g/dL)	4.8	4.7	4.5	4.5	4.5	4.6	4.8

Table 1. Liver function tests before (pink) and after (green) initiation of ursodiol.

4616 | WHEN TREATMENT FUELS RISK: A CASE OF GRAFT VERSUS HOST DISEASE AFTER LIVER TRANSPLANTATION

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Introduction: Graft versus host disease (GVHD) is a rare but devastating complication following liver transplant, characterized by donor-derived T-cell recognition of recipient tissues as foreign. The reported incidence ranges from approximately 0.1% to 2%, but mortality often exceeds 75%. We present a case of GVHD following liver transplantation complicated by severe infectious sequelae. **Case Presentation:** A 47-year-old woman with a history of Roux-en-Y gastric bypass complicated by portal vein thrombosis, non-cirrhotic portal hypertension, and portal biliopathy, status post failed transjugular intrahepatic portosystemic shunt placement due to cavernous transformation of the portal vein, presented for living-donor liver transplantation. Initial transplant was complicated by hemorrhagic shock requiring laparotomy, hematoma evacuation, and right hemicolectomy for ischemic bowel. She was subsequently found to have primary graft failure, necessitating plasma exchange and repeat liver transplantation with a brain-death donor on post-op day two. Intraoperatively, additional ischemic small bowel was resected with end ileostomy creation. She remained critically ill on vasopressors and continuous renal replacement therapy. Initial complications included cytomegalovirus related to massive blood transfusion, Klebsiella pneumoniae bacteremia secondary to pneumonia, and an intra-abdominal infection. After initial treatment she developed Klebsiella bacteremia from her hemodialysis catheter and peri-hepatic abscess. Subsequently she experienced two tonic clonic seizures. Imaging findings revealed posterior reversible encephalopathy syndrome and subarachnoid hemorrhage, presumed to be tacrolimus induced. Hospital course was further complicated by GVHD requiring prednisone and ruxolitinib, with severe pancytopenia necessitating bone marrow biopsy. She later developed MSSA bacteremia from her surgical incision and a cutaneous abdominal wall abscess. The lesions initially raised concern for varicella-zoster virus due to their dermatomal distribution and were treated with three weeks of antiviral therapy. She continued to develop numerous abscesses. Cultures grew Klebsiella pneumoniae, Candida glabrata/krusei, Enterococcus faecium, and and vancomycin-resistant Enterococcus, necessitating repeated surgical debridement and prolonged antimicrobial therapy. After nearly four months of hospitalization, she was discharged but returned days later to the intensive care unit with recurrent septic shock. **Discussion:** Infections are among the leading causes of mortality in patients with post-transplant GVHD and complicate decisions regarding escalation of immunosuppressive therapy. **Conclusion:** This case highlights the heightened vulnerability to life-threatening conditions during GVHD treatment.

4617 | KAPOSI SARCOMA PRESENTING WITH LYMPHADENOPATHY MIMICKING PTLD AFTER LIVER TRANSPLANTATION: A CASE REPORT

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Introduction: Kaposi sarcoma (KS) is a rare complication of immunosuppression following solid organ transplantation associated with HHV-8. KS may present with nonspecific systemic symptoms and lymphadenopathy in the absence of characteristic mucocutaneous lesions, creating a diagnostic challenge and

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mimicking post-transplant lymphoproliferative disorder (PTLD). Recognition of this atypical presentation is particularly important in liver transplant recipients, in whom timely tissue diagnosis may alter immunosuppressive and oncologic management. **Case Presentation:** A 61-year-old female with a history of MASH and hemochromatosis presented with fatigue, hypotension, and low-grade fevers 5 months after orthotopic liver transplantation. Her immunosuppressive regimen included tacrolimus and prednisone. Examination revealed no oral or cutaneous lesions, rash, or palpable hepatosplenomegaly. Laboratory workup revealed anemia, thrombocytopenia, hypoalbuminemia, and hyponatremia. There was initial concern for PTLD.CT showed new enlarged upper abdominal and retroperitoneal (RP) lymph nodes and splenomegaly. PET demonstrated hypermetabolic lymphadenopathy above and below the diaphragm (Fig. 1A). RP lymph node biopsy revealed KS with positive CD31, CD34, ERG, and HHV-8 staining. CRP was 139 and HHV-8 PCR was positive with a viral load exceeding 1 million copies/mL. Immunosuppression was switched from tacrolimus to sirolimus.Given the marked systemic inflammation, high HHV-8 viral load, cytopenias, and hyponatremia in the setting of biopsy-proven KS, her presentation was consistent with Kaposi sarcoma-associated herpesvirus inflammatory cytokine syndrome (KICS). Liposomal doxorubicin, tocilizumab, and rituximab were initiated with rapid hyponatremia resolution. She completed 6 cycles of liposomal doxorubicin and rituximab with remission of KICS and KS, undetectable HHV-8, and stable allograft function. End-of-treatment PET showed resolution of hypermetabolic lymphadenopathy (Fig. 1B), and MRI 1.5 years later showed normal-appearing lymph nodes without evidence of recurrence, supporting durable remission. **Discussion:** KS can closely mimic PTLD in liver transplant recipients, particularly when systemic symptoms and diffuse lymphadenopathy occur without mucocutaneous lesions. Because imaging cannot reliably distinguish these entities, tissue diagnosis is critical and directly informs immunosuppressive and systemic therapy. Recognition of concomitant KICS further informed management and contributed to rapid clinical improvement. **Conclusion:** KS should remain in the differential diagnosis of post-transplant lymphadenopathy despite absent mucocutaneous disease. Timely biopsy distinguishes KS from PTLD and guides management, enabling durable remission with preserved allograft function.

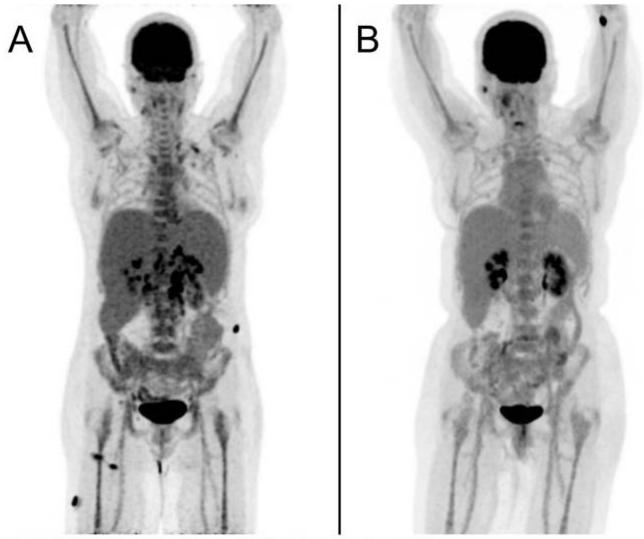


Figure 1. PET at the time of diagnosis (A) and post-treatment (B)

4618 | ACUTE GRAFT-VERSUS-HOST DISEASE MASQUERADING AS HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS AFTER LIVER TRANSPLANTATION

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Introduction: Graft-versus-host disease (GVHD) is a rare complication after liver transplantation, affecting 0.1-2% of recipients with mortality over 70%. Usually occurring 1-8 weeks post-transplant (median 28-30 days), it results from donor T lymphocytes attacking recipient tissues. Symptoms such as fever, diarrhea, rash, and pancytopenia overlap with common post-transplant complications. This delay and a lack of consensus on optimal treatment lead to high morbidity and mortality. We present a case of acute GVHD that initially mimicked hemophagocytic lymphohistiocytosis (HLH), highlighting the diagnostic and therapeutic challenges of marrow-predominant disease. **Case Presentation:** A 53-year-old man with alcohol-related cirrhosis underwent a deceased-donor liver transplant in March 2026. Three weeks post-transplant, he developed an erythematous, desquamating rash on his upper torso along with encephalopathy, diarrhea, tremors, hyponatremia, acute kidney injury, and pancytopenia, prompting ICU admission. His course was complicated by refractory pancytopenia, neutropenic fever, upper GI bleed, hyperferritinemia (> 30,000), lymphadenopathy, splenic lesions, and EBV viremia. Hyperferritinemia and hemophagocytosis on bone marrow biopsy were initially concerning for HLH; he was treated with dexamethasone, IVIG, and G-CSF with poor response. However, his chimerism studies

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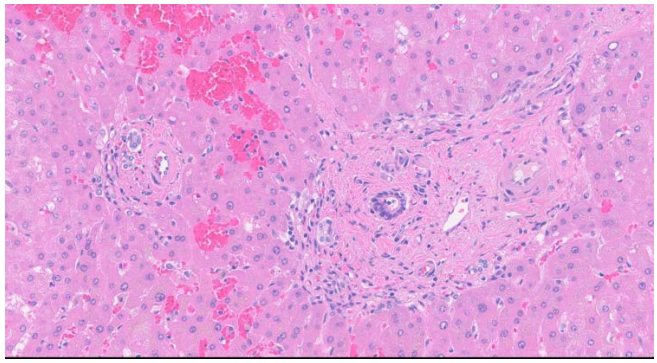
showed elevated donor T-cell populations (up to 80%), and skin biopsy confirmed GVHD, shifting the diagnosis toward transplant-associated GVHD. He received methylprednisolone, tacrolimus, ruxolitinib, thymoglobulin, and thrombopoietin receptor agonists, as well as supportive transfusions and rituximab for EBV viremia. He was evaluated for allogeneic hematopoietic stem cell transplantation (HSCT), but this was deferred due to high predicted treatment-related mortality, comorbidities, and possibility of autologous hematopoietic reconstitution. After 87 days in hospital, he was discharged to multidisciplinary follow-up with transplant hepatology, hematology/oncology, and the stem cell transplant team. **Discussion:** Liver transplant-associated GVHD can mimic HLH, making early tissue biopsy and chimerism testing essential for refractory cytopenias that are unexplained by infection or other etiologies. Prompt diagnosis and treatment are crucial before irreversible bone marrow failure. Unfortunately, there is a lack of data regarding the safety and efficacy of HSCT in patients who recently received a solid organ transplant and have multiple comorbidities. **Conclusion:** GVHD should remain high on the differential diagnosis in the first two months after liver transplantation in patients presenting with desquamating rash and refractory pancytopenia, highlighting the need for standardized management strategies and multicenter studies for patients ineligible for HSCT.

4619 | PROGRESSIVE PORTO-SINUSOIDAL VASCULAR DISEASE AND END-STAGE LIVER DISEASE IN NOONAN SYNDROME REQUIRING LIVER TRANSPLANTATION

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Introduction: Noonan syndrome (NS) is a RASopathy caused by germline dysregulation of the RAS/MAPK signaling pathway. Hepatic manifestations such as hepatomegaly, cholestasis, and autoimmune hepatitis are increasingly recognized, yet progressive liver disease requiring liver transplantation has not been reported. We present the first such case, with explant pathology demonstrating advanced porto-sinusoidal vascular disease (PSVD). **Case Presentation:** A 33-year-old man with NS presented in 2016 with acute abdominal pain. Imaging revealed portal hypertension with splenomegaly, varices, and acute portal vein and superior mesenteric vein thrombosis. Hypercoagulable workup was unrevealing. Serologic evaluation was negative including viral hepatitis, ANA, ASMA, AMA, and alpha-1 antitrypsin. FibroScan showed liver stiffness of only 8 kPa despite significant portal hypertensive findings, characteristic of noncirrhotic portal hypertension. Transjugular liver biopsy demonstrated mild chronic inflammation without cirrhosis. He subsequently developed progressive decompensation with lower extremity edema and worsening portal hypertension requiring transjugular intrahepatic portosystemic shunt. Surveillance imaging identified a persistent LI-RADS 4 lesion with normal alpha-fetoprotein and worsening synthetic function (MELD 14). Evaluation revealed mild hepatopulmonary syndrome (PO2 71 mmHg) with intrapulmonary shunting and reduced diffusion capacity. At age 41 he underwent deceased donor orthotopic liver transplantation using a donation after circulatory death graft with normothermic machine perfusion. Explant pathology demonstrated diffuse focal nodular hyperplasia (FNH)-like lesions, nodular regenerative hyperplasia, obliterative portal venopathy, prominent hepatic arterialization, and bridging fibrosis without malignancy; the dominant lesion was a large FNH-like nodule. Post-transplant liver function normalized. **Discussion:** These explant findings fulfill diagnostic criteria for advanced PSVD rather than conventional cirrhosis. Low liver stiffness despite severe portal hypertension early in the disease course supports a primary vascular process. Emerging reports describe similar pathology across multiple NS genotypes, including obliterative portal venopathy in MRAS-related NS and biopsy-confirmed PSVD in LZTR1-related NS, suggesting hepatic vascular disease may be an underrecognized RASopathy phenotype. The RAS/MAPK pathway is central to endothelial function and angiogenesis, providing a plausible mechanism for portal vascular remodeling. **Conclusion:** This is the first reported case of liver transplantation for end-stage liver disease in Noonan syndrome. PSVD in NS can progress to end-stage liver disease requiring transplantation. Clinicians should consider PSVD in NS patients with unexplained portal hypertension and low liver stiffness.



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4620 | IMPROVEMENT IN MAGNETIC RESONANCE ELASTOGRAPHY-DEFINED ADVANCED HEPATIC FIBROSIS DURING LONG-TERM INCRETIN THERAPY DESPITE MODEST WEIGHT LOSS

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Introduction: Metabolic Dysfunction-Associated Steatohepatitis (MASH) is a leading cause of chronic liver disease worldwide and can progress to cirrhosis. The extent and progression of fibrosis in MASH are strongly associated with liver-related morbidity and mortality. Incretin-based therapies, including glucagon-like peptide-1 (GLP-1) receptor agonists and newer dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 agonists, have demonstrated histologic improvement and reduction in fibrosis among patients with F2-F3 fibrosis. However, evidence of regression in advanced F3-F4 fibrosis with incretin-based therapy remains limited, particularly longitudinal data collected via serial MRI elastography. **Case Presentation:** We report a 44-year-old woman with class III obesity and type 2 diabetes mellitus who demonstrated marked regression in MRI elastography-defined liver fibrosis after long-term incretin therapy. The patient underwent MRI elastography in February 2021, which demonstrated a mean liver stiffness of 4.9 kPa (consistent with stage F3-F4 fibrosis) and an estimated fat fraction of 30% (corresponding to moderate steatosis), supporting the clinical diagnosis of MASH. Beginning in December 2022, she was initiated on a GLP-1 agonist and over the next 3.5 years, continued GLP-1-based incretin therapy (liraglutide, semaglutide, then tirzepatide) alongside diabetes therapy including basal insulin, metformin, and SGLT-2 inhibitors. During this time, her weight fell from 131 kg to 120 kg (8.4% weight loss) and hemoglobin A1c improved from 11.3% to 8.5%, with a minimum A1c of 7.6% during this time period. A repeat MRI elastography in May 2026 showed a mean liver stiffness of 3.37 kPa, corresponding to stage F1-F2 fibrosis, and a fat fraction of 3.2%. These findings demonstrated substantial improvement in liver stiffness and hepatic fat during long term incretin therapy. **Discussion:** This case describes marked regression of MRI elastography-defined liver fibrosis from F3-F4 to F1-F2 and hepatic fat from 30% to 3.2% during long-term GLP-1-based incretin therapy. This improvement occurred with 8.4% total body weight loss, below the >=10% weight loss generally associated with fibrosis regression in MASH and the weight loss typically reported with contemporary GLP-1 receptor agonist therapies. Glycemic control also remained suboptimal, with a minimum A1c of 7.6%. Notably, serial MRI elastography provided a noninvasive method for monitoring liver stiffness, highlighting the value of longitudinal imaging in assessing treatment response and reducing the need for liver biopsy. **Conclusion:** This case emphasizes the potential role of incretin therapies in regression of MRI elastography-defined advanced liver fibrosis and steatosis in MASH, even with modest weight loss. It also demonstrates the importance of serial MRI elastography for noninvasive monitoring of treatment response in patients with advanced MASH.

4621 | REAL WORLD DATA FOR RESMETIROM USE IN A LARGE ACADEMIC CENTER: CASE SERIES

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Introduction: Metabolic dysfunction-associated steatohepatitis represents a growing public health burden affecting an estimated 86 million U.S. adults. In the landmark MAESTRO-NASH trial, a selective thyroid hormone receptor-β agonist, Resmetirom, demonstrated histologic improvement in MASH including fibrosis regression and has been approved by the FDA for treatment of MASH with fibrosis severity of F2-F34. We aim to describe real world data on Resmetirom efficacy in relation to weight loss. **Case Presentation:** We conducted a retrospective case series of three adults with MASH diagnosed noninvasively via transient elastography in conjunction with MASH metabolic criteria who were treated with Resmetirom 100 mg once daily for an average duration of 21 months in a single center hepatology clinic. These patients were specifically selected after serologic workup for secondary etiologies of liver disease were ruled out as recommended by AASLD guidelines. Baseline and follow up data included demographics, metabolic comorbidities, liver chemistries, and serial transient elastography metrics. Patients were followed longitudinally with serial laboratory and noninvasive fibrosis measurements at yearly clinical intervals. Outcomes were summarized descriptively focusing on changes in biochemical and noninvasive fibrosis parameters. **Discussion:** Three patients (mean age 45.7 +- 17.0 years; 2 males, 1 female; Korean, Hispanic/Latino, and Egyptian/Middle Eastern backgrounds) with baseline fibrosis stages F2-F3 and steatosis grade S3 were treated with Resmetirom 100 mg daily for a mean of 21.0 +- 1.0

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months (Table 1). Mean BMI at initiation was 38.3 +- 1.0 kg/m². Weight remained stable throughout follow-up. All three patients (100%) achieved >=1-stage fibrosis regression. Mean LSM declined from 10.6 +- 0.9 to 7.6 +- 1.4 kPa (Δ −3.0 +- 0.5 kPa; −28.2%; p=0.009). Mean CAP declined from 391.0 +- 6.1 to 281.7 +- 5.8 dB/m (Δ −109.3 +- 5.5 dB/m; −28.0%; p=0.0008), with steatosis grade improvement in all patients (S3→S0 in two patients; S3→S1 in one patient). Mean ALT declined from 105.7 +- 18.0 to 31.7 +- 0.6 U/L (−70.0%; p=0.018) and mean AST from 98.3 +- 9.5 to 37.3 +- 1.2 U/L (−62.0%; p=0.006). No adverse events, gastrointestinal intolerability, or drug-drug interactions were observed in any patient. **Conclusion:** This real-world case series demonstrates Resmetirom 100 mg daily produced statistically significant and clinically meaningful improvements in all hepatic endpoints without weight change. Resmetirom was well-tolerated without significant adverse events in our case series. Small sample size and retrospective design, the large effect sizes and consistent directional findings across all three patients lend credibility to these exploratory results. Larger prospective real-world studies with histologic confirmation are warranted given limitations of small sample size and non-generalizability.

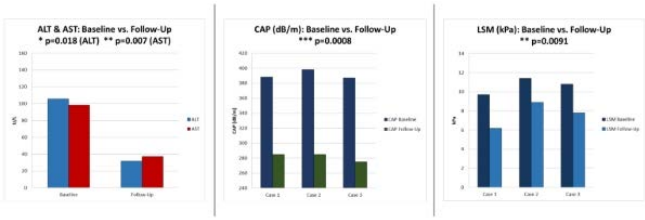


Figure 1. Laboratory markers, Controlled Attenuation Parameter, and Liver Stiffness Measurement.

Endpoint	Baseline Mean ± SD	Follow-Up Mean ± SD	Mean Δ ± SD	95% CI (lower)	95% CI (upper)	% Change	t-statistic (df=2)	P-value
ALT (U/L)	105.7 ± 18.0	31.7 ± 0.6	-74.0 ± 17.5	-117.53	-30.47	-70.0%	7.315	0.0182
AST (U/L)	98.3 ± 9.5	37.3 ± 1.2	-61.0 ± 8.5	-82.22	-39.78	-62.0%	12.366	0.0065
CAP (dB/m)	391.0 ± 6.1	281.7 ± 5.8	-109.3 ± 5.5	-123.01	-95.65	-28.0%	34.384	0.0008
LSM (kPa)	10.6 ± 0.9	7.6 ± 1.4	-3.0 ± 0.5	-4.24	-1.76	-28.2%	10.392	0.0091
Weight (lbs)*	220.3 ± 15.5	219.3 ± 13.9	-1.0 ± 4.4	-11.83	9.83	-0.5%	0.397	0.7295

Table 1. Pre-Treatment Baseline versus Follow-up (~12 months) After Resmetirom Initiation.

4622 | RAPID DEVELOPMENT OF CIRRHOSIS IN ACQUIRED GENERALIZED LIPODYSTROPHY: A 25-YEAR COURSE OF A 2-YEAR-OLD CHILD

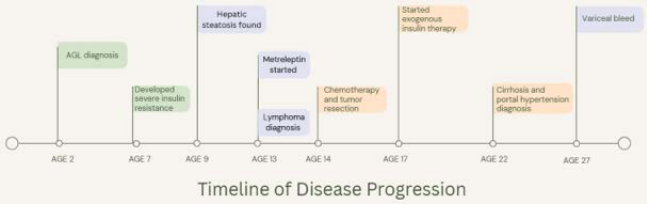
Sai Chalasani[1] KiBeom Kwon[1] Asif A. Hitawala[1] Christopher Koh[2] Marissa Lightbourne[1] Rebecca Brown[1] Theo Heller[1]

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Introduction: Lipodystrophy comprises a rare group of disorders defined by loss of subcutaneous adipose tissue secondary to the inability to store triglycerides in adipose tissue. It can be divided into congenital or acquired and further categorized into partial or generalized loss. It can lead to insulin resistance and metabolic dysfunction-associated steatohepatitis (MASH). The onset varies widely and prompt diagnosis can slow sequelae. We describe a 27-year-old woman with acquired generalized lipodystrophy (AGL) diagnosed at age 2 now with decompensated MASH cirrhosis. **Case Presentation:** A 27-year-old woman presents to our clinic with decompensated cirrhosis. She was diagnosed with acquired generalized lipodystrophy at age 2. Her initial symptoms were development of facial panniculitis along with generalized subcutaneous fat loss. Genetic studies were negative, consistent with AGL. She later developed severe insulin resistance, acanthosis nigricans, and hypertriglyceridemia. At age 9, ultrasound showed fatty liver and percutaneous liver biopsy showing macrovesicular steatosis, mild portal chronic inflammatory infiltrate, and fibrosis difficult to discern with NASH score 2. Leptin therapy was initiated at age 12, and repeat biopsy showed more steatosis but less apparent fibrosis and no evidence of cirrhosis with NASH score 8. At age 13, she was diagnosed with ALK-positive anaplastic large cell lymphoma achieving remission with chemotherapy and resection. By age 17, her severe insulin resistance progressed requiring insulin up to 500 U/day, and she developed hepatosplenomegaly.Her steatohepatitis progressed to advanced fibrosis. At age 22, she presented with an upper GI bleed caused by esophageal varices. A transjugular liver biopsy confirmed cirrhosis with portal hypertension and NASH score 2. Most recent liver-associated enzymes include ALP 72 (40-150 U/L), ALT 63 (0-55 U/L), AST 46 (5-34 U/L), Bilirubin 0.9 (0.2-1.2 U/L), INR 1.63 (0.8-1.2 U/L), albumin 3.7 (3.5-5.2 U/L) and GGT 163 (9-36 U/L). Last fibroscan in 2025 showed CAP 268 and elastography 44.3 kPa (IQR 16). Her subsequent course was defined by variceal bleeding requiring endoscopic intervention and treatment with carvedilol. **Discussion:** This case illustrates accelerated liver disease reflecting lipodystrophy pathophysiology and how swift diagnosis can

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slow progression. Development of decompensated cirrhosis despite treatment for AGL necessitates discussion of liver transplant, which is challenging especially with a history of cancer (lymphoma in our case). Her disease progressed despite metformin and leptin replacement.



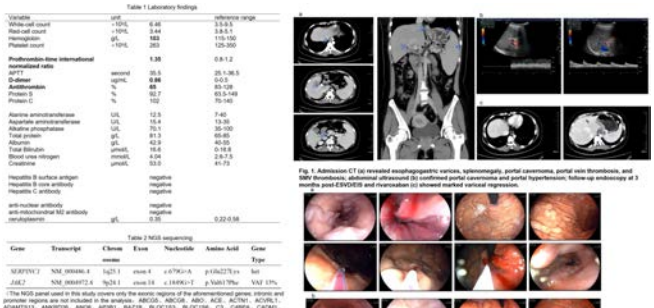
Timeline of Disease Progression

4623 | HEREDITARY ANTITHROMBIN III DEFICIENCY WITH LOW-BURDEN *JAK2 V617F* MUTATION CAUSING CAVERNOUS TRANSFORMATION OF THE PORTAL VEIN AND ESOPHAGOGASTRIC VARICEAL BLEEDING: A CASE REPORT

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Introduction: *SERPINC1* mutation-induced hereditary antithrombin III (AT III) deficiency impairs protein N-glycosylation and confers high risk of splanchnic vein thrombosis (SVT). Unresolved chronic portal vein thrombosis (PVT) progresses to cavernous transformation of the portal vein (CTPV) and non-cirrhotic portal hypertension complicated by variceal hemorrhage. *JAK2 V617F* denotes myeloproliferative neoplasm (MPN)-related hypercoagulability. Concurrent hereditary AT III deficiency plus low-allelic-burden *JAK2* clone is extremely rare. **Case Presentation:** A 30-year-old Chinese female was referred with a 3-month history of hematemesis and melena. She was diagnosed with PVT and CTPV 3 years after acute pancreatitis, with prior transient thrombocytosis (peak 720×10^9/L). Endoscopy identified severe esophageal varices and gastric varices. Serum AT activity dropped to 65% (ref 83-128%), accompanied by mild elevated D-dimer, INR and mild anemia; platelet count was normal. Genetic testing detected heterozygous *SERPINC1* c.679G>A p.Glu227Lys and *JAK2 V617F* (VAF=13%). Bone marrow biopsy excluded MPN morphologic changes. Intervention & Follow-up: Endoscopic selective varices devascularization (ESVD) combined with injection sclerotherapy (EIS) was performed to treat the esophageal and gastric fundus varices. Carvedilol 6.25 mg twice daily was administered for secondary prophylaxis of variceal rebleeding. Rivaroxaban 10 mg once daily was safely resumed 1 week post-endoscopy. At 3-month follow-up, endoscopy showed significant regression of the varices, with no recurrent bleeding or new thrombotic events. **Discussion:** This case highlights hereditary AT III deficiency as the primary thrombophilic driver, with low-burden *JAK2 V617F* (VAF=13%) not fulfilling MPN criteria. Although the two defects may synergize, the current portal cavernoma and variceal bleeding are largely attributable to AT III deficiency. However, long-term surveillance for clonal evolution is mandatory, as the *JAK2* clone may expand over time and convert to overt MPN. Endoscopic eradication (ESVD+EIS) plus early rivaroxaban achieved complete variceal regression and no rebleeding at 3 months. Implications: (1) screen young CTPV patients for inherited thrombophilia and MPN drivers; (2) monitor *JAK2* VAF, blood counts, and marrow periodically; (3) early anticoagulation after radical endotherapy is safe. Multidisciplinary collaboration is essential for long-term thrombosis-bleeding balance. **Conclusion:** Young adults with non-cirrhotic portal hypertension and CTPV require simultaneous screening for congenital thrombophilia and MPN gene variants. Sequential endoscopic eradication combined with carvedilol and early rivaroxaban achieves favorable safety and efficacy in hypercoagulable states, mandating cross-specialty collaborative management.



4624 | WHEN TWO SILENT KILLERS COLLIDE: PROFOUND ANEMIA FROM MYELODYSPLASTIC SYNDROME AND OCCULT SMALL-BOWEL BLEEDING IN A PATIENT WITH CARCINOID TUMOR.

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Introduction: Myelodysplastic syndrome (MDS) commonly produces chronic anemia through ineffective hematopoiesis, whereas gastrointestinal bleeding from small-bowel neuroendocrine tumors (NETs) is uncommon and may evade conventional endoscopic evaluation. Their coexistence can obscure the etiology of worsening anemia and delay definitive hemostasis. **Case Presentation:** An 85-year-old man with MDS, stable small-bowel carcinoid tumor, atrial fibrillation on apixaban (eliquis), and heart failure with reduced ejection fraction (HFrEF) presented with dizziness, dyspnea, and weakness. Hemoglobin (hgb) was 4.1 g/dL, with hypotension, atrial fibrillation with rapid ventricular response (Afib w RVR), positive fecal occult blood testing, and melena. Troponin increased from 2,865 to >50,000 despite absence of chest pain, consistent clinically with severe supply-demand myocardial injury in the setting of profound anemia, hypotension, and tachyarrhythmia. Apixaban was withheld and multiple packed red blood cell transfusions increased hemoglobin to 9.1 g/dL. Esophagogastroduodenoscopy revealed no bleeding source. Because occult small-bowel hemorrhage remained suspected in the setting of a known carcinoid tumor, the patient was transferred for interventional radiology evaluation and underwent mesenteric angiography with selective transcatheter arterial embolization (TAE). Hemoglobin subsequently remained stable at 9.1 g/dL without recurrent overt bleeding. **Discussion:** Anemia in patients with MDS is often attributed to ineffective hematopoiesis, but a rapid or disproportionate decline in hemoglobin should prompt evaluation for gastrointestinal blood loss. In this case, melena, positive fecal occult blood testing, hemodynamic instability, and a known small-bowel NET strongly suggested active bleeding despite a nondiagnostic upper endoscopy. Small-bowel NETs are an uncommon and often elusive source of gastrointestinal hemorrhage because they may not be detected by conventional endoscopy. When bleeding is severe and a culprit vessel can be localized, transcatheter arterial embolization offers an effective, minimally invasive treatment. This case underscores the importance of considering a bleeding small-bowel NET when anemia worsens unexpectedly, particularly in a patient receiving anticoagulation. **Conclusion:** Severe anemia in MDS should not automatically be attributed to ineffective hematopoiesis. Concurrent gastrointestinal blood loss, including bleeding associated with small-bowel NETs, should be investigated when clinical evidence suggests an additional source.

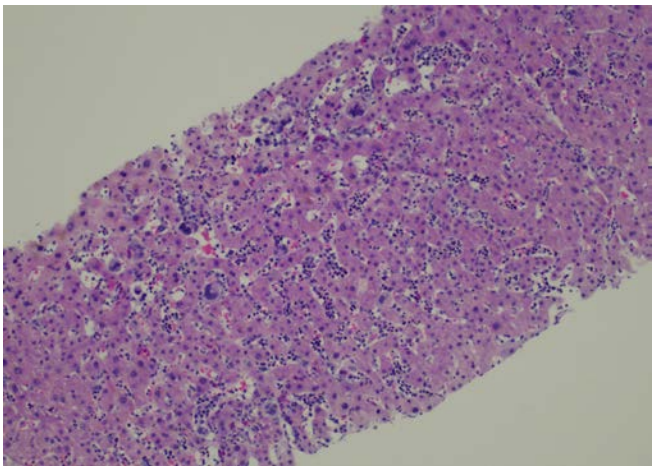
4625 | MARKED HEPATIC EXTRAMEDULLARY HEMATOPOIESIS IN A PATIENT WITH CHRONIC CHOLESTATIC LIVER TEST ABNORMALITIES

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Introduction: Extramedullary hematopoiesis (EMH), the production of hematopoietic cells outside the bone marrow, is most commonly associated with chronic hematologic disorders. Hepatic EMH is uncommon and may be an unexpected finding on liver biopsy. We describe marked hepatic EMH identified during the evaluation of otherwise unexplained chronic liver test abnormalities. **Case Presentation:** A 66-year-old man with heart failure with preserved ejection fraction, atrial fibrillation, ischemic stroke, hypertension, and subdural hematoma was evaluated for persistent liver test abnormalities first noted in 2025. He had no significant alcohol use or features of metabolic syndrome. Laboratory studies demonstrated a cholestatic pattern of liver injury with hyperbilirubinemia. Evaluation for drug-induced, viral, and autoimmune liver disease was unrevealing. Contrast-enhanced MRI with elastography demonstrated hepatomegaly with cirrhotic morphology, splenomegaly, trace ascites, and small splenorenal varices, without focal hepatic lesions. An endoscopic ultrasound-guided liver biopsy was performed to clarify the etiology of the liver disease. Histology demonstrated marked EMH within hepatic sinusoids, including erythropoietic islands, immature myeloid cells, and atypical megakaryocytes. CD61 and myeloperoxidase highlighted megakaryocytes and myeloid cells, respectively; CD34 showed no significant increase in blasts. Additional findings included focal mild portal inflammation, periportal and perisinusoidal fibrosis, and less than 5% steatosis. **Discussion:** Marked hepatic EMH raised concern for an underlying hematologic disorder or a reactive leukoerythroblastic state. Peripheral blood findings included persistent leukocytosis, mild anemia, elevated red cell distribution width, nucleated red blood cells, and circulating metamyelocytes and myelocytes. Hematology evaluation documented iron-deficiency anemia and anemia of chronic disease, but no definitive hematologic disorder. The combination of marked hepatic EMH and leukoerythroblastic peripheral blood findings warranted further hematologic evaluation. **Conclusion:** Hepatic EMH may provide an important clue to an underlying hematologic process, even in the absence of a previously diagnosed hematologic disorder. Recognition of marked hepatic EMH on liver biopsy, particularly in conjunction with leukoerythroblastic peripheral blood findings, should prompt targeted hematologic evaluation.

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4626 | OCCULT LUNG ADENOCARCINOMA PRESENTING AS ACUTE LIVER FAILURE AND TROUSSEAU SYNDROME

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Introduction: Diffuse metastatic infiltration is a rare cause of acute liver failure (ALF) that may remain undetected on imaging, complicating transplant evaluation. We report occult lung adenocarcinoma presenting as ALF initially attributed to drug-induced liver injury (DILI). **Case Presentation:** A 72-year-old nonsmoking man was transferred for transplant evaluation 6 days after abrupt jaundice. His history included prostatectomy for prostate cancer in 2025 and postoperative deep vein thrombosis (DVT) treated with apixaban. A contralateral DVT developed after a motor-vehicle collision five weeks prior to presentation. Amoxicillin-clavulanate had been prescribed for pneumonia one month prior. At transfer, he had grade 2 encephalopathy, INR 2.4, bilirubin 14 mg/dL, alkaline phosphatase 2,121 U/L, and a MELD 3.0 score of 29. CT showed hepatomegaly without cirrhosis or focal lesions. Toxicologic, infectious, autoimmune, and metabolic testing were unrevealing. CT angiography identified pulmonary emboli, bilateral DVTs, portal vein thrombosis, and splenic and renal infarcts. TTE demonstrated mitral and tricuspid vegetations. Blood cultures were negative. Exposure timing and cholestatic injury initially favored amoxicillin-clavulanate DILI. However, transjugular liver biopsy showed metastatic adenocarcinoma favoring pulmonary origin (CK7+/TTF-1+; Figure 1), establishing a contraindication to transplantation. Thrombosis progressed despite heparin and argatroban, and he passed after transitioning to comfort care. Autopsy confirmed extensive hepatic infiltration from pulmonary lymphangitic adenocarcinoma without a gross lung mass, and sterile platelet-fibrin vegetations diagnostic of nonbacterial thrombotic endocarditis (NBTE), supporting Trousseau syndrome. **Discussion:** This case highlights occult lung adenocarcinoma as a rare cause of ALF. In an ALF registry, malignant infiltration caused 1.4% of cases; only 44% of affected patients had imaging evidence of infiltration or masses. The absence of focal hepatic lesions does not exclude malignant infiltration, as tumor cells may spread diffusely along the hepatic sinusoids. The evolving thrombotic pattern provided an additional diagnostic clue. Progressive venous and arterial thromboses with valvular vegetations should heighten suspicion for occult malignancy with Trousseau syndrome and possible NBTE, even when a plausible drug exposure initially favors DILI. **Conclusion:** Malignant infiltration is a rare cause of ALF that may lack focal hepatic lesions, making recognition challenging. Unexplained multiterritory thrombosis should heighten suspicion and prompt timely liver biopsy to establish diagnosis and clarify transplant eligibility.

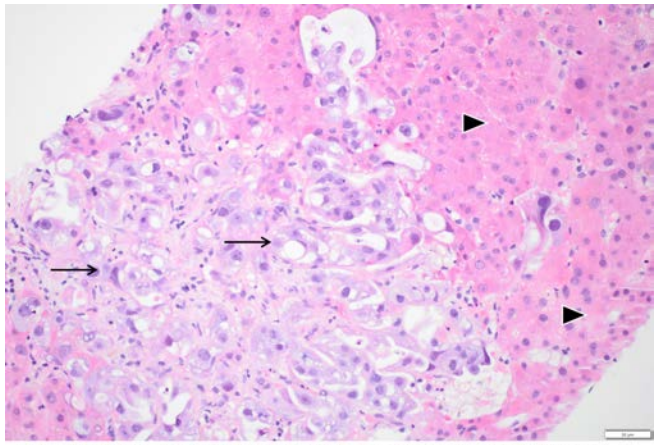


Figure 1. Hematoxylin and Eosin (H&E) stain demonstrating a high-power view of the liver lesion from the original liver biopsy in relation to background hepatocytes (arrowhead). There are infiltrating cells that are poorly gland forming, consistent with an adenocarcinoma (arrow). Image was captured with a 20x objective.

4627 | A CASE REPORT OF LIVER FAILURE COMPLICATED WITH RHABDOMYOLYSIS TRIGGED BY VORICONAZOLE-ESOMEPRAZOLE INTERACTION

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Introduction: A 65-year-old female with primary biliary cholangitis and autoimmune hepatitis overlap syndrome developed acute-on-subacute liver failure. Voriconazole combined with esomeprazole triggered rhabdomyolysis. After drug discontinuation and supportive care, she improved. **Case Presentation:** A 65-year-old female was admitted on July 22, 2024, with 16-year history of abnormal liver function and

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2-month fever. In 2008, she had elevated transaminases after Chinese herbs, which normalized. In 2023, enzymes recurred. On April 12, 2024, ALT/AST rose to 934.2/659.3 U/L. Biopsy showed AIH-PBC overlap. She received steroids and UDCA, discharged April 30. On May 26, she developed fever and jaundice. Readmission: ALT/AST 210.7/744.6 U/L, TBIL 97.9 μmol/L. NGS detected Trichosporon asahii and CMV. Voriconazole was started. Discharged July 17. Four days later, she had severe fatigue and weakness. Labs: TBIL 128.1 μmol/L, CK 5822 IU/L, serum myoglobin 2189.5 ng/ml, and urine myoglobin 68.1 ng/ml, confirming rhabdomyolysis. Voriconazole-esomeprazole interaction was the likely cause. Voriconazole was stopped; prednisolone and MMF were started. CK normalized. By November 2024, liver function normalized. **Discussion:** First, the etiology of liver failure requires careful differentiation between AIH-PBC overlap and drug-induced liver injury, integrating history, autoantibodies, and pathology. Second, opportunistic infections are common in immunosuppressed liver failure patients, necessitating rapid pathogen detection. Third, drug interactions, particularly via CYP2C19, can cause rhabdomyolysis; clinicians should monitor drug levels and CK. Fourth, a multifactorial approach is essential, as rhabdomyolysis often results from combined insults. Finally, comprehensive management with multidisciplinary collaboration is key to favorable outcomes. **Conclusion:** In patients with liver failure complicated by rhabdomyolysis, clinicians must maintain a high index of suspicion for drug-drug interactions, particularly when combining agents metabolized via the CYP450 system. Early recognition, prompt withdrawal of offending drugs, and a multidisciplinary approach addressing both hepatic and extrahepatic organ dysfunction are paramount to achieving favorable outcomes in this critically ill population.

4628 | HISTOTRIPSY OF CHOLANGIOCARCINOMA NESTLED WITHIN THE PORTAL CONFLUENCE: A NONINVASIVE ALTERNATIVE FOR A CHALLENGING LOCATION

Leonardo L. Diaz-Gandica[1] Joshua Boone[1] Justin Drake[1] Emmanuel Zervos[1] Nasreen Vohra[1] Carlos Marroquin[1]

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Introduction: Histotripsy (HT) is a nonthermal, noninvasive technique that uses focused ultrasound to mechanically destroy and liquify tissue through cavitation. It avoids the heat-sink effect and risk of vascular injury associated with thermal ablation and provides an alternative to major hepatic resection and associated comorbidities and arduous recovery. Its use in intrahepatic cholangiocarcinoma (iCCA), particularly for perivascular or periductal lesions, remains sparsely reported. **Case Presentation:** A 77-year-old woman with multiple comorbidities was found to have a 2.8-cm enhancing mass in segment 4B during evaluation of elevated liver function test results. The lesion was nestled within the portal venous (PV) origins with the confluence at its inferior border, the left PV coursing along the left border and the anterior sectoral branch along the right margin of the lesion. Staging CT of the chest, abdomen, and pelvis revealed no evidence of extrahepatic malignancy.Biopsy confirmed a moderately differentiated adenocarcinoma consistent with iCCA. On the day of the procedure, targeting ultrasound again demonstrated a 2.9 × 2.6 × 2.3-cm isoechoic mass accessible through a subcostal approach with minimal transducer angulation, confirming a favorable acoustic window. She subsequently underwent histotripsy using the Edison System (HistoSonics). Following post-procedural observation and monitoring, she was discharged home the same day. No immediate complications were observed. At 2.5 months of follow-up, she remained without procedure-related complications and was undergoing chemotherapy. **Discussion:** Unlike other ablative techniques, histotripsy uses mechanical, non-thermal mechanisms that mitigate heat-sink effects near the portal vein and avoid thermal or mechanical injury to adjacent bile ducts and vascular structures. These characteristics make it particularly suitable for centrally located tumors, in which thermal modalities carry a greater risk of vascular or biliary injury and incomplete ablation. Histotripsy also avoids the morbidity associated with major hepatic resection. In this patient, curative resection would have required an extended right or left hepatectomy, with substantial complexity and morbidities. Published experience with histotripsy for iCCA remains limited compared with that for hepatocellular carcinoma and metastatic liver disease. Evidence is particularly sparse for centrally located, perivascular, or periductal lesions such as the one described here. **Conclusion:** Histotripsy provides a locoregional treatment option for perivascular or periductal iCCA when tumor location or a patient's comorbid risk profile makes resection or thermal ablation less attractive.



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4629 | LAMOTRIGINE DOSE ESCALATION LEADING TO ACUTE LIVER INJURY AND REVERSE TAKOTSUBO CARDIOMYOPATHY: AN UNUSUAL DIAGNOSTIC CHALLENGE

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Introduction: Acute liver injury (ALI) has multiple causes, with drug-induced liver injury (DILI) and hypoxic hepatitis among the most common. Differentiating between them is challenging because both may present with marked aminotransferase elevations. Lamotrigine is a recognized but uncommon cause of idiosyncratic DILI. Reverse Takotsubo cardiomyopathy (rTCM) is a rare stress cardiomyopathy that can also cause ischemic liver injury. We report a patient in whom concurrent lamotrigine and rTCM created a significant diagnostic dilemma. **Case Presentation:** A 26-year-old woman with epilepsy presented with nausea, vomiting and breakthrough seizures while taking lamotrigine 400 mg/day and topiramate 200 mg/day. Following an inpatient lamotrigine dose increase to 500 mg/day, AST and ALT acutely rose to >7000 U/L and 6677 U/L with INR 2.42.This was consistent with severe hepatocellular injury. CT showed hepatomegaly with periportal edema. Echocardiography for suspected cardiogenic hypoxic hepatitis showed rTCM with EF 30-35%; coronary CT angiography was normal. Extensive infectious, autoimmune, metabolic, vascular, and toxic evaluation was unrevealing. RECAM score for lamotrigine was 7 (probable DILI). Lamotrigine was discontinued and liver tests improved rapidly. At 2-month follow-up, liver biochemistries normalized and EF recovered to 64%. **Discussion:** The main challenge was distinguishing lamotrigine-induced liver injury from hypoxic hepatitis secondary to rTCM. Although ischemic hepatitis was considered, the patient remained hemodynamically stable without sustained cardiac or respiratory failure. The close temporal relationship to lamotrigine dose escalation, exclusion of competing etiologies, probable RECAM score, and rapid biochemical improvement after drug withdrawal strongly supported DILI. Because the dose escalation occurred during hospitalization, liver injury was recognized within 24 hours and lamotrigine was promptly discontinued. We propose that severe DILI served as the physiological stressor precipitating rTCM rather than rTCM causing the liver injury and that lamotrigine dose-escalation period may represent a window of increased vulnerability during which selected patients could benefit from closer biochemical monitoring **Conclusion:** This case highlights the challenge of diagnosing DILI when concurrent cardiac dysfunction is present. Despite the absence of liver biopsy, the clinical course favored DILI. Inpatient lamotrigine dose escalation enabled prompt recognition and drug withdrawal. Whether targeted biochemical monitoring during dose escalation in selected higher-risk patients improves early detection warrants further study.

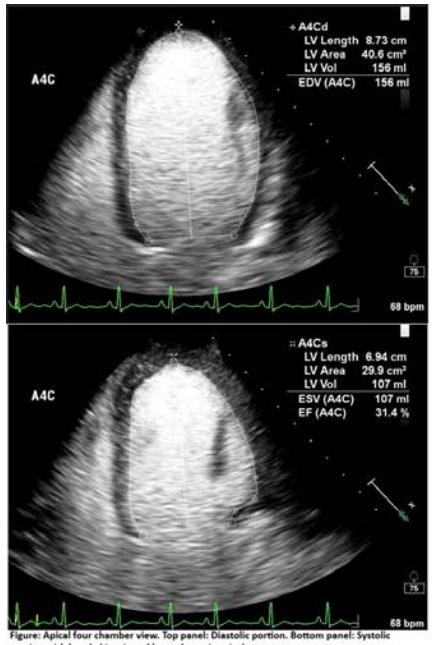


Figure: Apical four chamber view. Top panel: Diastolic portion. Bottom panel: Systolic portion with basal akinesis and hyperdynamic apical segments

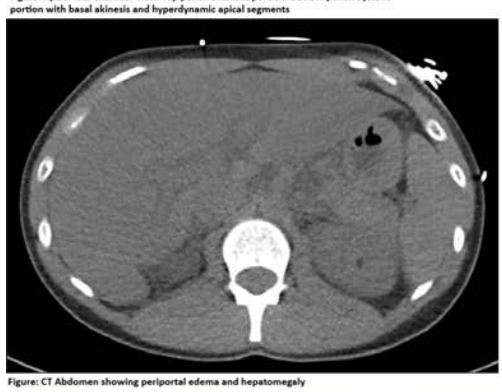


Figure: CT Abdomen showing periportal edema and hepatomegaly

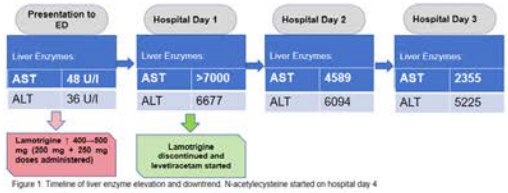


Figure 1: Timeline of liver enzyme elevation and downtrend. N-acetylecysteine started on hospital day 4

HSV1,2, HIV, EBV, CMV, VZV	IgM Ab, PCR testing
Hep A,B,C,E	IgM Ab, Surface AG/AB
Autoimmune Hepatitis	ANA, Anti-SM,Anti-mitochondrial ab
Wilsons and Hemochromatosis	ceruloplasmin, 24h-urine copper, ferritin, TIBC
Acetaminophen, salicylate	Individual level checks
Ethanol	Serum ethanol
Malignant infiltration	CT and MRI abdomen pelvis

Figure 2: Other etiologies which were ruled out with corresponding testing

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4630 | VORICONAZOLE-INDUCED ACUTE HEPATITIS IN A PATIENT WITH INVASIVE PULMONARY ASPERGILLOSIS

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Introduction: Voriconazole is first-line therapy for invasive pulmonary aspergillosis. Although severe hepatitis from voriconazole is rare, it is a well-known cause of drug-induced liver injury (DILI). Injury typically manifests within the first month of therapy. We report biopsy-proven acute hepatitis presenting two weeks after completing a 12-week course of treatment. **Case Presentation:** A 58-year-old man with a history of cavitary mycobacterium abscessus infection, bronchiectasis, and recently diagnosed invasive pulmonary aspergillosis was initiated on voriconazole 200 mg twice daily. A trough level on day 6 was therapeutic (3.8 mg/L); however, no subsequent levels were measured due to missed follow-up laboratory appointments. The total duration of treatment was extended to 12 weeks from 6 as he remained symptomatic.Two weeks after completing therapy, the patient developed painless jaundice and was admitted for acute hepatitis (Table). Autoimmune and viral panels were negative. MRCP showed no liver or biliary pathology. Liver biopsy demonstrated active panlobular hepatitis with centrilobular necrosis (Image 1). The patient improved with conservative care after stopping the medication. Table Admission Discharge 2 months later RefALT 1480 978 89 10-50 U/LAST 1705 656 129 0-50 U/LALP 208 203 147 40-129 U/LBilirubin 13.1 8.5 2.2 0-1.2 mg/dLINR 1.4 1.1 1.1 0.9-1.1AFP 65.8 5.8 <10 ng/mLCa 19-9 1114 172 <37 U/mL **Discussion:** This clinical timeline is atypical. Hepatic injury is usually reported within the first month of voriconazole therapy, whereas jaundice in our case developed 3 months after the first dose and 12 days after the last dose. This finding indicates that completion of therapy does not exclude voriconazole as a potential cause. The R value was approximately 21, and the marked transaminase elevation with hyperbilirubinemia met Hy's Law criteria for severe DILI. Panlobular hepatitis with centrilobular necrosis is a nonspecific but recognized histologic finding of severe hepatocellular DILI. In combination with negative serologies, and improvement of liver profile after stopping voriconazole, these findings favored DILI over infectious or autoimmune hepatitis. One trough level was obtained on day 6 to verify steady state; no additional levels were measured during the subsequent 11 weeks. Given voriconazole's saturable metabolism, a single in-range level cannot confirm safe exposure throughout prolonged therapy. Missed follow-up possibly delayed recognition of accumulating risk. Elevated AFP and CA19-9 raised concern for malignancy, but both markers can increase nonspecifically with hepatocyte regeneration and cholestasis. Recognizing this avoided unnecessary workup. **Conclusion:** Voriconazole-induced hepatitis should remain on the differential for liver injury after aspergillosis treatment, even after therapy is complete. Close monitoring of voriconazole levels during prolonged courses may help reduce this risk.

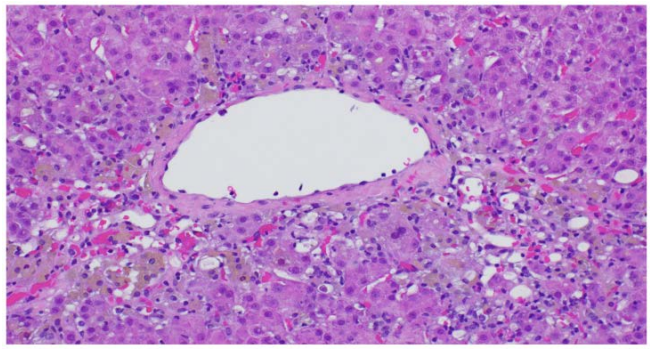


Image 1: Liver biopsy pathology: active panlobular hepatitis with centrilobular necrosis. Hematoxylin and eosin staining 20x pericentral view.

4631 | SEVERE HEPATOCELLULAR INJURY AFTER THERAPEUTIC AMOXICILLIN-CLAVULANATE AND ACETAMINOPHEN EXPOSURE: A CASE OF DRUG-INDUCED LIVER INJURY WITH UNDERLYING METASTATIC CANCER

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Introduction: Drug-induced liver injury (DILI) is defined as hepatic damage caused by medications, herbs, or dietarysupplements. Most cases improve after withdrawal of the offending agents. However, diagnosis remainschallenging because other etiologies must be excluded. We discussed a patient who developed acute liver injury after therapeutic doses of amoxicillin-clavulanate and acetaminophen-containing cold medications,and a later hospital course, metastatic cancer was identified. **Case Presentation:** A 56-year-old man with a history of a benign lung nodule presented with nausea, vomiting, abdominal pain,and jaundice for one day. Two weeks prior, he had completed a 7-day course of amoxicillin-clavulanate for anupper respiratory infection and had taken recom-

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mended doses of acetaminophen-containing coldmedications. Laboratory evaluation showed marked hepatocellular injury with normal blood counts andcoagulation functions (Table 1). He reported social alcohol use and denied tobacco, recreational drug, or herbal supplement use. Family history was notable for leukemia, colon, bladder, thyroid, lung, and throat cancers in first-degree relatives. Physical examination revealed scleral icterus and mild right upper quadrant tenderness.Further evaluation for viral hepatitis, autoimmune hepatitis, hemochromatosis, Wilson disease, and alpha-1antitrypsin deficiency was unremarkable (Table 1). MRCP showed no acute findings. Liver biopsy showed moderate-to-severe portal inflammation with necroinflammatory activity, consistent with DILI and without significant autoimmune features (Figure 1). Intravenous N-acetylcysteine was discontinued due to an allergic reaction. The patient was treated with ursodiol, with gradual improvement in liver function and jaundice. Subsequent hospital course, left ankle metastatic cancer was identified. **Discussion:** Augmentin is typically associated with cholestatic or mixed DILI, whereas acetaminophen causes dose-dependent hepatocellular injury. Our patient developed unusually severe hepatocellular injury despite therapeutic dosing, with liver biopsy supporting DILI. The subsequent discovery of metastatic malignancy, together with an extensive family history of cancer, raises the possibility that underlying genetic or oncologic factors increased susceptibility to hepatotoxicity. **Conclusion:** This case underscores that severe DILI can occur even at therapeutic doses of commonly used medications. Because DILI remains a diagnosis of exclusion, careful evaluation for alternative etiologies is essential. Early recognition, prompt withdrawal of suspected agents, and close clinical monitoring are critical to minimizing liver injury and improving outcomes.

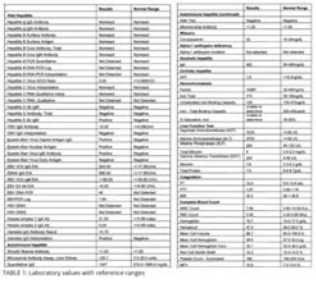


TABLE 1: Laboratory values with reference ranges

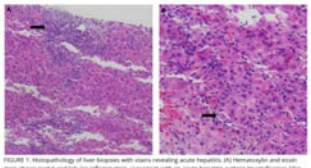


FIGURE 1: Histopathology of liver biopsies with stains revealing acute hepatitis. (A) Hematoxylin and eosin stain shows portal and lobular inflammation, consistent with an acute hepatitis pattern (magnification 10x). (B) Hematoxylin and eosin stain indicates lobular necroinflammatory activity. The acidophil body highlights active liver injury or inflammation (magnification 20x).

4632 | SURVIVAL AFTER NEAR-FATAL ABIRATERONE-ASSOCIATED ACUTE LIVER FAILURE COMPLICATED BY CARDIAC ARREST AND DIALYSIS-DEPENDENT ACUTE KIDNEY INJURY

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Introduction: Abiraterone is used for metastatic castration-resistant prostate cancer and may cause aminotransferase elevation, but acute liver failure is rare. Subsequent shock may obscure whether severe liver injury was primary or ischemic. We report near-fatal abiraterone-associated acute liver failure in which hepatocellular injury preceded cardiac arrest. **Case Presentation:** A 61-year-old man with metastatic castration-resistant prostate cancer presented with malaise and abdominal pain two months after starting abiraterone; prior liver tests were reportedly normal. He was cognitively intact and hemodynamically stable without hypotension. Laboratory testing showed marked elevation in liver biomarkers (aspartate aminotransferase [AST] 42,256 U/L, alanine aminotransferase [ALT] 10,131 U/L, total bilirubin 7.0 mg/dL, ammonia 63 µmol/L), international normalized ratio (5.73), serum lactate (26.3 mmol/L), and serum creatinine (8.34 mg/dL). He subsequently developed severe metabolic acidosis and cardiac arrest, with return of spontaneous circulation after 5 minutes. Mechanical ventilation and hemodialysis were required. Evaluation for viral and autoimmune hepatitis, acetaminophen toxicity, biliary obstruction, hepatic metastasis, and alcohol-associated liver disease was unrevealing, supporting abiraterone-associated acute liver failure. Abiraterone was discontinued. Within one week, AST fell to 260 U/L and ALT to 708 U/L. Renal function recovered, and he was extubated neurologically intact and discharged for rehabilitation. **Discussion:** This case highlights the importance of chronology in distinguishing drug-induced from ischemic liver injury. Profound hepatocellular injury preceded hemodynamic collapse, arguing against ischemic hepatitis as the initiating event. The two-month latency also supports abiraterone as the culprit, as reported hepatotoxicity typically emerges within 4-8 weeks after treatment initiation. Prompt drug withdrawal, exclusion of competing etiologies, and intensive organ support may permit recovery despite multiorgan failure. **Conclusion:** Abiraterone-associated acute liver failure should be considered when severe hepatocellular injury precedes

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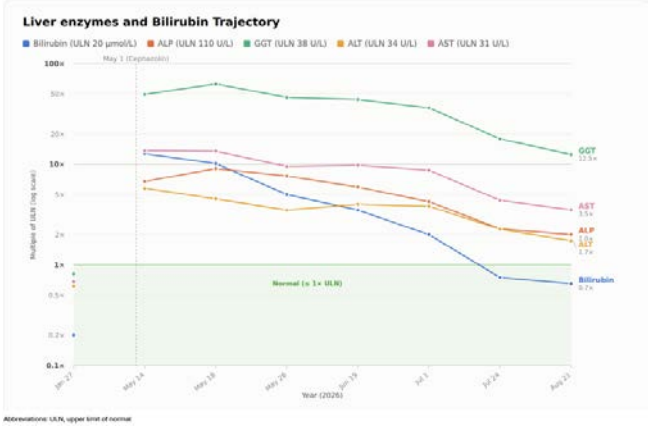
shock, even several weeks after treatment initiation.

4633 | AN EYE FOR DETAIL: UNCOVERING INTRACAMERAL CEFAZOLIN-INDUCED LIVER INJURY

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[1]Royal Brisbane Women's Hospital

Introduction: DILI is a diagnosis of exclusion requiring recognition of likely causative agents and exclusion of competing causes. Cefazolin-associated DILI is usually seen after systemic administration and has a classic cholestatic picture presenting 1-3 weeks after exposure. We describe an unusual case of immediate reaction following a second hit of 1 mg of intracameral cefazolin where exposure was only mentioned in the perioperative anaesthetic record, highlighting the importance of perioperative medication reconciliation. **Case Presentation:** A 75-year-old woman with metabolic syndrome presented with two weeks of night sweats, jaundice, dark urine, pale stools and intermittent right upper quadrant pain, beginning one day after uncomplicated left cataract surgery on May 1, 2026. Biochemistry demonstrated marked cholestatic liver injury (R-ratio 0.8; Fig. 1). ANA was elevated (1:640), with otherwise unremarkable autoimmune and viral hepatitis serology. Liver ultrasound and MRCP demonstrated cholelithiasis without biliary dilatation, obstruction or suspicious masses. Her regular medications were reviewed, and no hepatotoxic drugs or herbal supplements were identified. Endoscopic ultrasound was performed to exclude biliary pathology and obtain liver biopsy; however, no safe biopsy window was identified. The patient declined further inpatient procedures and was discharged with outpatient biopsy and hepatology follow-up planned.Two weeks later, meticulous review of the perioperative record identified 1 mg intracameral cefazolin, documented only in the anaesthetic record. Further history revealed identical exposure during right cataract surgery eight months earlier. Given the compatible cholestatic pattern, exclusion of alternative causes and known hepatotoxicity of cefazolin, cefazolin-induced hypersensitivity DILI was considered most likely. RUCAM was 9, indicating highly probable DILI. Liver biochemistry and symptoms continued to improve over three months (Fig. 1). **Discussion:** This case highlights two unusual features of cefazolin DILI: significant cholestatic injury following only 1 mg intracamerally, a rarely reported route, and jaundice within 24 hours, substantially earlier than the typical 1-3 week latency. Prior exposure eight months earlier may have contributed to an accelerated response on re-exposure. Cholelithiasis and positive ANA initially suggested biliary or autoimmune disease, but imaging and serology excluded these alternatives. Spontaneous improvement without immunosuppression further supported DILI. **Conclusion:** Intracameral medications should be considered in unexplained postoperative liver injury. Cefazolin was hidden in plain sight and identified only in the anaesthetic record, highlighting the importance of perioperative medication review to prevent misdiagnosis, unnecessary investigation and re-exposure.



4634 | FROM F4 TO F2: REVERSIBLE LIVER STIFFNESS AFTER TREATMENT OF THYROTOXICOSIS IN PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS

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Introduction: Hepatic biochemical abnormalities are common in untreated hyperthyroidism, but clinically significant liver disease is uncommon. Thyrotoxicosis-associated advanced liver fibrosis is rarely described, particularly with concomitant chronic liver disease. **Case Presentation:** A 24-year-old man presented with abdominal pain, diarrhea, 20-lb unintentional weight loss, and severe jaundice. Evaluation for presumed biliary pancreatitis led to cholecystectomy and endoscopic retrograde cholangiopancreatography without relevant findings. Persistent cholestasis prompted further evaluation; viral hepatitis and autoimmune serologies were negative. Congestive heart failure was excluded. Laboratory testing showed direct hyperbilirubinemia, aminotransferase elevation, hypoalbuminemia, persistent cholestasis, and ascites. Magnetic resonance cholangiopancreatography demonstrated chronic liver injury with fibrotic areas and splenomegaly. Liver biopsy showed acute hepati-

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tis with parenchymal collapse, severe cholestasis, and copper-containing hepatocytes. Wilson disease was excluded. Genetic testing established progressive familial intrahepatic cholestasis (PFIC), but treatment resulted in no clinical or biochemical improvement. Profound thyrotoxicosis was subsequently identified and treated with radioiodine. After treatment of hyperthyroidism, liver biochemistries and bilirubin improved while transient elastography decreased from F4 to F2. **Discussion:** Advanced fibrosis or cirrhosis associated with thyrotoxicosis is rarely reported. Proposed mechanisms include increased hepatic oxygen demand with hypoxia and oxidative stress, triiodothyronine-mediated mitochondrial injury, accelerated catabolism, and cholestasis. Despite genetically confirmed PFIC, the lack of response to initial treatment and subsequent biochemical and elastographic improvement after thyroid control suggests that thyrotoxicosis substantially contributed to liver injury. The reduction from F4 to F2 is particularly relevant because inflammation and cholestasis can increase liver stiffness independent of structural fibrosis, indicating a potentially reversible component. **Conclusion:** The coexistence of hyperthyroidism and PFIC represents an unusual clinical scenario in which thyrotoxicosis may exacerbate underlying liver disease. In this patient, control of hyperthyroidism was essential to achieve improvement in liver biochemistries and was accompanied by regression of liver stiffness from F4 to F2. This case highlights thyrotoxicosis as a potentially reversible contributor to hepatic injury and fibrosis assessment, even in patients with an underlying genetic cholestatic disorder. Achieving euthyroidism and reassessing liver disease severity after thyroid control may be crucial to accurately determine prognosis and guide management.

Parameter	During thyrotoxicosis	After restoration of euthyroidism
Thyroid-stimulating hormone, mIU/L	<0.008	0.59
Free thyroxine, [unit]	7.49	1.38
Total bilirubin, mg/dL	26.14	2.72
Direct bilirubin, mg/dL	18.54	0.76
Aspartate aminotransferase, U/L	537*	22
Alanine aminotransferase, U/L	608*	20
Alkaline phosphatase, U/L	338*	224
Albumin, g/dL	2.46*	4.95
International normalized ratio	1.60*	1.35
Liver fibrosis stage by transient elastography	F4	F2

Table 1. Biochemical and elastographic response following treatment of hyperthyroidism. *Peak liver injury values.

4635 | HYPERTHYROIDISM UNMASKING A COMPLEX GENETIC CHOLESTATIC PHENOTYPE: A CASE OF CONCURRENT UGT1A1 AND VPS33B VARIANTS IN A YOUNG ADULT

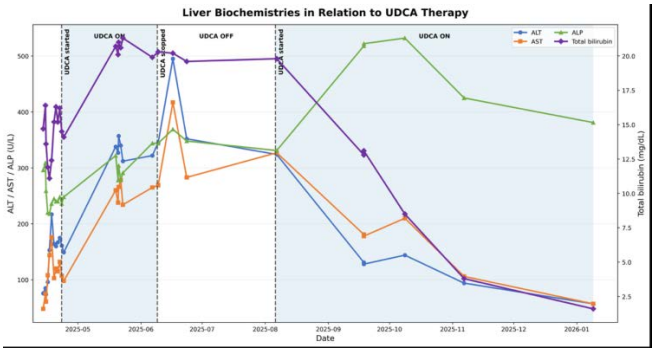
Riya Gupta[1] Aalam Sohal[2] Mohanad Al-Qaisi[3] Nilofar Najafian[3]

[1]Creighton University Phoenix, [2]Creighton Univeristy Phoenix, [3]St. Joseph Hospital and Medical Center

Introduction: Progressive familial intrahepatic cholestasis (PFIC) is a group of rare autosomal recessive disorders that result in cholestasis, which, if left untreated, results in end-stage liver disease. Up to 13 PFIC subtypes have been described, each caused by variants in different genes involved in canalicular bile transport. Although predominantly observed in the pediatric population, heterozygous variants in PFIC-associated genes have been reported to contribute to adult-onset cholestatic phenotypes. We report adult-onset cholestasis from synergistic heterozygosity involving UGT1A1 and VPS33B precipitated by hyperthyroidism. **Case Presentation:** A 25-year-old male presented with gallstone pancreatitis, jaundice, and choledocholithiasis (ALT 76, AST 48, ALP 296, lipase 815 IU/L, total bilirubin (TB) 14.7 mg/dL). CT showed dilated intra/extrahepatic ducts from an obstructive 0.6 cm ampullary stone. ERCP/sphincterotomy revealed a patulous ampulla and dilated CBD. Cholecystectomy showed a discolored, nodular liver suggestive of cirrhosis. Post-op MRCP showed biliary ductal dilation and mild enhancement of the bile duct wall. He was readmitted within a month with worsening TB 20.7, ALT 338, AST 260, ALP 322 and bile acid 364. Workup for viral hepatitis, autoimmune disease, Wilson's, hemochromatosis, and A1AT deficiency was negative. Liver biopsy showed cholestatic hepatitis with lymphocytic inflammation, elevated hepatic copper (311.3 µg/g; ref 15-55), urine copper 140 µg/24h, minimal fibrosis, and no cirrhosis or ductopenia. MRI showed portal hypertension (perigastric/perisplenic varices). He also had weight loss, pruritus, and tachycardia; TSH <0.008, fT4 7.4, and elevated TPO antibodies confirmed Graves' disease. A cholestatic gene panel revealed heterozygous VPS33B and homozygous UGT1A1 variants. He received I-131 and UDCA, with notable improvement; stopping UDCA caused a rebound in bilirubin, which reversed on reinitiation. After 1 year of ursodiol and treatment for Graves', labs showed bilirubin 1.6, ALT 57, AST 57, and ALP 381 IU/L. **Discussion:** This case suggests a synergistic genetic contribution to cholestatic disease with concurrent hyperthyroidism. While thyrotoxicosis can be associated with cholestatic injury, it is unlikely to fully ex-

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plain this patient's persistent cholestasis and portal hypertension. We hypothesize that homozygous UGT1A1 and heterozygous VPS33B variants increased cholestasis risk, with hyperthyroidism as a superimposed trigger for clinical expression. Marked response to UDCA, with worsening after its discontinuation, further supports an underlying cholestatic process, and residual ALP elevation despite treatment suggests residual chronic cholestasis. Elevated hepatic copper levels raised suspicion for Wilson's disease; however, ATP7B was negative. **Conclusion:** This case underscores the importance of genetic testing in young adults with unexplained cholestasis.



4636 | REAL-WORLD EXPERIENCE WITH ODEVIXIBAT IN ADULT-ONSET GENETIC CHOLESTASIS: A CASE SERIES OF FOUR PATIENTS

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Introduction: Adult-onset genetic cholestatic disorders are increasingly recognized through expanded genetic testing. Patients may experience recurrent cholestatic episodes with severe pruritus and impaired quality of life (QoL). Treatment remains challenging when conventional therapies are ineffective. Treatment with Odevixibat, a selective ileal bile acid transporter (IBAT) inhibitor might be an option in this population. **Case Presentation:** We present four adult patients (2 male, 2 female; age 17-41 years) who were treated with Odevixibat. Clinical, biochemical, and genetic data were retrospectively collected at two Belgian tertiary centers after obtaining informed consent. Three patients had genetically confirmed BRIC type 2 with compound heterozygous ABCB11 variants, while one had a BRIC-like phenotype with negative cholestasis panel testing. All had previous symptomatic cholestatic episodes. They again presented with jaundice, pruritus and serum bile acid (sBA) levels between 127 and 699 µmol/L. Odevixibat was administered at 2400-6000 µg/day for 4-12 weeks. All patients experienced rapid clinical improvement. Pruritus improved within 1-6 weeks, accompanied by better sleep, daily functioning, and QoL. Marked biochemical improvement was observed in all cases. All patients had sBA levels <10 µmol/L within 8 weeks. Bilirubin and liver enzyme abnormalities also improved. **Discussion:** Odevixibat, an IBAT inhibitor, is effective in pediatric progressive familial intrahepatic cholestasis, but data in adults is limited. We report four patient with genetic cholestasis experiencing a bout, who were treated with Odevixibat and had excellent clinical response. No serious adverse events, hepatotoxicity, or clinically significant gastrointestinal side effects were reported, and no patient discontinued treatment because of toxicity. One patient relapsed after treatment interruption but responded to re-initiation. The remaining three remained recurrence-free for up to seven months after discontinuation. **Conclusion:** In this real-world case series, odevixibat was associated with rapid and clinically meaningful improvements in pruritus, sBA levels, liver biochemistry, and QoL in adults with recurrent genetic cholestasis. Treatment was well tolerated, with no new safety signals. These findings support further evaluation of IBAT inhibition as a treatment option for selected adults with recurrent genetic cholestasis and severe pruritus.

Patient	Genotype	Clinical presentation	Odevixibat dose	Treatment duration	Odevixibat response	Follow-up
Case 1 27y old male	ABCB11 compound heterozygous	Severe recurrent cholestatic episodes with pruritus, fatigue, jaundice, steatorrhea, and weight loss	6000 µg/day	12 weeks	Normalization of sBA and liver tests, marked QoL improvement	Recurrence after interruption, controlled with maintenance therapy
Case 2 17y old female	ABCB11 compound heterozygous	Recurrent estrogen-triggered cholestasis with pruritus, fatigue, weight loss and jaundice	4800 µg/day	6 weeks	Resolution of pruritus, normalization of sBA and liver enzymes	No recurrence
Case 3 36y old male	Genotype unresolved	Recurrent cholestasis with severe pruritus and jaundice	2400 µg/day	4 weeks	Rapid symptom relief; normalization of sBA within 4 weeks	Event-free 7 months after discontinuation
Case 4 41y old female	ABCB11 compound heterozygous	Recurrent cholestasis with pruritus and jaundice	3000 µg/day	4 weeks	Rapid reduction of sBA within 2 weeks and complete symptom resolution	Event-free 7 months after discontinuation

Abbreviations: sBA = serum bile acids, QoL= quality of life

4637 | A CHOLESTATIC CLUE: HEPATIC AMYLOIDOSIS UNMASKING MULTIPLE MYELOMA

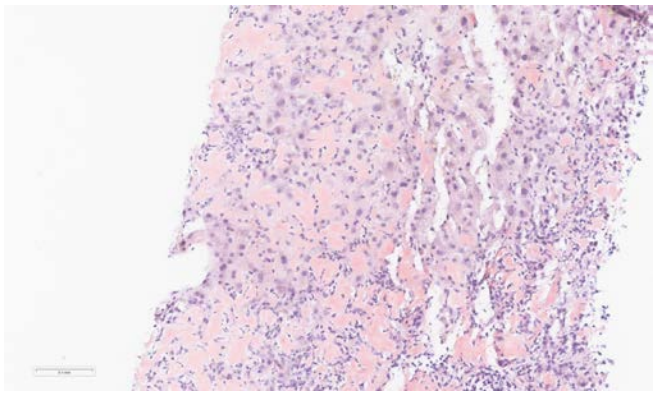
Odai Mansour[1] Callie Lilley[1] Wei Chen[1] Sendhilnathan Ramalingam[1] Kara Wegermann[1]

[1]Duke University

Introduction: In a patient with fatigue, anorexia, and new liver injury, the differential is broad and includes infectious, autoimmune, malignant, and drug-induced etiologies. For presentations with initial workups not elucidating a clear etiology, uncommon hepatic manifestations of systemic disease should be considered. **Case Presentation:** A 75-year-old male presented

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with 2 months of fatigue, unintentional weight loss, dark urine, and new mixed hepatocellular/cholestatic liver injury. Past medical history was notable for hypertension, hyperlipidemia, and chronic back pain. Relevant medical history included recent use of doxycycline, acetaminophen, and rosuvastatin. Physical exam showed scleral icterus without hepatomegaly. Initial labs were notable for a predominantly cholestatic pattern of liver injury: total bilirubin 2.8 mg/dL, alkaline phosphatase 571 U/L, AST 148 U/L, and ALT 77 U/L. Other labs included serum protein 7.5 g/dL, albumin 2.9 g/dL, GGT 1,166 U/L, ESR 95 mm/hr, and CRP 1.5 mg/dL. A broad workup evaluated infectious, drug-induced, autoimmune, and malignant etiologies. HIV and Viral hepatitis testing was negative. Salicylate level returned low, and doxycycline and rosuvastatin seemed unlikely culprits. Autoimmune workup included negative ASMA, AMA, and ANA. Kappa/Lamba ratio was 0.04. IgA was elevated to 1,603 with low IgG/IgM. Liver ultrasound showed no new findings, and MRCP revealed non-specific heterogenous liver enhancement. CT Abdomen and Pelvis showed bilateral external iliac lymphadenopathy and enlarged gastrohepatic, periportal, and periaortic lymph nodes. Pathology from a liver biopsy revealed extensive AL lambda amyloid deposition, suggestive of a hepatic manifestation of systemic disease. A bone marrow biopsy and imaging subsequently confirmed systemic AL lambda amyloidosis secondary to multiple myeloma. **Discussion:** Although the patient's history of low back pain, fatigue, and anorexia is consistent with multiple myeloma, a diagnosis of hepatic amyloidosis would have otherwise been low on the differential diagnosis. The conditions considered most likely based on history and exam were immune mediated conditions (PSC, PBC, IgG4 disease) and drug-induced liver injury. A liver biopsy revealed hepatic amyloidosis from a likely extra-hepatic source, later found to be multiple myeloma. This diagnosis would have been difficult without liver biopsy-highlighting the importance of histopathologic examination. **Conclusion:** Hepatic amyloidosis is an uncommon but important cause of otherwise unexplained liver injury and may be the presenting manifestation of an underlying plasma cell disorder. This case highlights the importance of considering infiltrative disease and pursuing liver biopsy when unexplained liver injury is accompanied by systemic features such as fatigue and weight loss.



4638 | WHEN THE BILIARY TREE CANNOT RECOVER: A CASE OF RAPIDLY PROGRESSIVE SECONDARY SCLEROSING CHOLANGITIS REQUIRING LIVER TRANSPLANTATION

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[1]Duke University Medical Center, [2]Duke University School of Medicine, [3]Duke University Hospital, [4]Duke University

Introduction: Secondary sclerosing cholangitis (SSC) occurs when an insult triggers progressive cholestatic injury, leading to biliary inflammation, fibrosis, strictures, and ductal destruction. A subset of cases of SSC may progress rapidly to irreversible biliary destruction, recurrent infection, and liver failure. **Case Presentation:** A 70-year-old man with no prior liver or biliary disease presented with *Clostridium perfringens* bacteremia and septic shock due to emphysematous cholecystitis and ascending cholangitis. Initial ERCP demonstrated a dilated common bile duct with debris but no strictures. He underwent percutaneous cholecystostomy and subtotal cholecystectomy. Liver biopsy demonstrated cholestatic biliary injury without cirrhosis. Over the subsequent 8 weeks, he developed recurrent cholangitis and rapidly progressive multifocal intrahepatic strictures despite repeated ERCPs and biliary stenting. After transfer for LT consideration, he developed septic shock from recurrent cholangitis requiring percutaneous biliary drainage. Cholangiography demonstrated diffuse biliary irregularity with multifocal stricturing, dilation, and extensive central biliary necrosis (Image 1). He developed coagulopathy, with an INR rising to 2.6, and hepatic encephalopathy. Following control of biliary sepsis, he was listed at MELD 3.0 of 25. At listing, labs notable for total bilirubin 16

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mg/dL, AST 132 U/L, ALT 192 U/L, alkaline phosphatase 1,035 U/L, albumin 2.0 g/dL, INR 1.5. He underwent LT 10 weeks after presentation. Explant showed cirrhosis with dilated large bile ducts, biliary infarcts, abscess formation, bile plugs, and marked cholestasis (Image 2). His early post-transplant course was complicated by polymicrobial peritonitis requiring surgical washouts. At last follow-up, he was improving on antimicrobials with preserved allograft function. **Discussion:** This case demonstrates remarkably rapid progression of SSC following severe biliary sepsis and ischemic injury. Despite repeated endoscopic interventions, biliary injury progressed to recurrent infection, central biliary necrosis, and hepatic dysfunction within weeks. Cirrhosis on explant despite a recent biopsy without cirrhosis further highlights the aggressive course. Recurrent biliary sepsis presents a particular challenge, as LT may provide the only definitive treatment while uncontrolled infection simultaneously threatens transplant candidacy. **Conclusion:** SSC can progress from acute biliary injury to irreversible ductal destruction and hepatic dysfunction within weeks. Early referral for LT should be considered when biliary destruction and recurrent cholangitis progress despite endoscopic therapy, as recurrent sepsis may create a narrow window for transplantation.

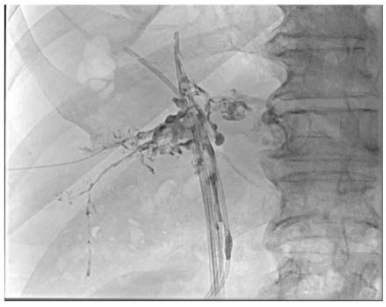


Image 1: Percutaneous transhepatic cholangiogram obtained at DUH (Week 8)

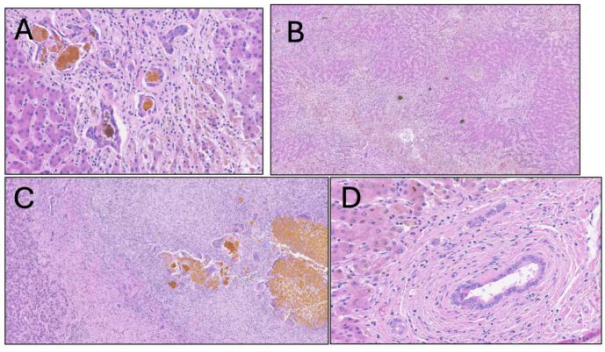


Image 2: Liver histology shows bile duct proliferation, dilation and cholestasis with A. cholangitis and B. biliary cirrhosis. C. Bile abscess with prominent bile lakes and giant cells. D. Periductal fibrosis with canalicular cholestasis.

4639 | AN UNDER-RECOGNIZED PHENOTYPE OF PRIMARY BILIARY CHOLANGITIS

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[1]Geisinger

Introduction: Primary biliary cholangitis (PBC) is an autoimmune cholestatic liver disease that predominantly affects women and is typically recognized by persistent alkaline phosphatase (ALP) elevation and anti-mitochondrial antibody (AMA) positivity. Although PBC with normal ALP has been described, this phenotype may be under-recognized, particularly when accompanied by atypical hepatocellular enzyme elevations. We present an asymptomatic man with AMApositive, biopsy-confirmed PBC, normal ALP, and unexpectedly advanced liver disease. **Case Presentation:** A 66-year-old man with chronic kidney disease, diabetes, and dyslipidemia was referred for chronically elevated aminotransferases. AST and ALT had been elevated for several years, peaking at 129 U/L and 125 U/L, respectively, while ALP remained persistently normal. He was asymptomatic and denied alcohol use, viral hepatitis, herbal supplement use, or a family history of liver disease. Subsequent evaluation demonstrated positive AMA and anti-smooth muscle antibody (ASMA), and AST/ALT normalized and remained normal.VCTE demonstrated liver stiffness of 13.2 kPa, concerning for advanced fibrosis. Given the atypical biochemical profile and ASMA positivity, autoimmune hepatitis-PBC variant disease was considered. Liver biopsy demonstrated portal inflammation with bile duct injury and a lymphocytic infiltrate resembling a florid duct lesion, supporting PBC. Hepatic venous pressure gradient was 11 mm Hg, consistent with clinically significant portal hypertension. His metabolic risk factors and hepatic steatosis raised concern for concomitant metabolic dysfunction-associated steatohepatitis. Ursodeoxycholic acid was initiated with planned longitudinal biochemical and fibrosis assessment, and carvedilol was started for portal hypertension. **Discussion:** This case illustrates an under-recognized PBC phenotype characterized by male sex, absence of symptoms, persistently normal ALP, and an atypical hepatocellular biochemical profile. Despite the absence of conventional biochemical evidence of cholestasis, the patient had AMA positivity, characteristic PBC histology, advanced fibrosis, and clinically significant portal hypertension. The case highlights the potential limitations of relying on ALP and symptoms alone to identify and monitor PBC, particularly in patients with atypical presentations. **Conclusion:** PBC should remain a diagnostic consideration in patients with unexplained liver enzyme abnormalities and positive AMA, even when ALP is persistently normal. This

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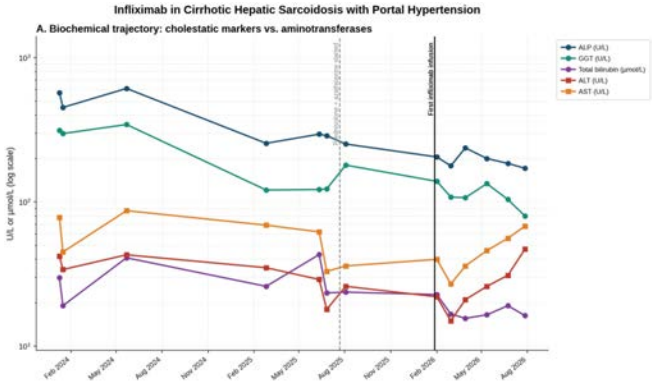
case underscores that normal ALP and minimal symptoms do not exclude clinically significant PBC and highlights the importance of histologic assessment and fibrosis staging in this under-recognized phenotype.

4640 | INFLIXIMAB FOR REFRACTORY CONSTITUTIONAL SYMPTOMS IN CIRRHOTIC HEPATIC SARCOIDOSIS WITH PORTAL HYPERTENSION: A CASE REPORT

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Introduction: Sarcoidosis is a multisystem granulomatous disorder of unknown cause. We describe a patient with sarcoidosis confined to the liver at presentation and recognised over eighteen months, whose constitutional symptoms proved refractory to corticosteroids and azathioprine and thereafter entered complete remission after infliximab therapy, in the setting of established cirrhosis and portal hypertension. **Case Presentation:** A 57-year-old woman presented with six months of dry cough and four months of fever reaching 40°C, with night sweats and weight loss. Liver biochemistry was markedly cholestatic (alkaline phosphatase 572 U/L, total bilirubin 29.8 µmol/L, ALT 42 U/L, AST 78 U/L). Abdominal CT showed findings consistent with early cirrhotic change, and transient elastography measured 23.3 kPa (F4). Liver biopsy showed granulomatous hepatitis with bile duct injury and noncaseating granulomas; no fibrosis was seen in the sampled core. The leading diagnosis was initially primary biliary cholangitis. She was readmitted with worsening fever, jaundice and pruritus. Repeat chest CT showed faint ground-glass opacities, and she developed inflammatory arthritis; sarcoidosis with predominantly hepatic involvement was diagnosed. Prednisolone and azathioprine were started, but fever continued for a further fifteen days, after which infliximab 5 mg/kg was started. Chest CT six days before the first infusion showed resolution of the ground-glass opacities, yet the fever continued until infliximab was started. After the first infusion, the fever resolved completely and never returned. Bilirubin normalised and GGT fell by 42%, whereas ALP fell by only 17%. Six infusions were given over six months without an infusion reaction, infection or hepatic decompensation, though aminotransferases rose progressively (ALT 15→47 U/L; AST 27→68 U/L) while the cholestatic markers continued to fall. **Discussion:** Hepatic sarcoidosis with cirrhosis and portal hypertension is at the edge of available evidence, a group in which conventional immunosuppression performs least well. The patient is informative due to organ responses separated in time: pulmonary disease remitted while the constitutional symptoms persisted. The resolution of fever can therefore be attributed to infliximab with reasonable confidence. Experience with anti-TNF agents in liver disease is scant, with none described in cirrhotic hepatic sarcoidosis. **Conclusion:** Infliximab produced a sustained and complete remission of constitutional symptoms in a woman with hepatic sarcoidosis, cirrhosis and portal hypertension, who underwent six infusions over six months without decompensation, infusion reaction or infection. As pulmonary disease had already resolved on corticosteroids and azathioprine, the constitutional response is attributable to anti-TNF therapy. This case report supports consideration of biologic therapy where conventional immunosuppression has failed.



4641 | HNF1B-ASSOCIATED CHOLANGIOPATHY IN AN ADULT WITH 17Q12 DELETION SYNDROME: A CASE REPORT

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Introduction: The 17q12 recurrent deletion syndrome, encompassing the *HNF1B* gene, is classically associated with renal cysts and diabetes (RCAD) syndrome. While hepatic involvement has been described in developmental contexts, chronic liver disease in adults with this deletion remains poorly characterized. We present a case of progressive hepatopathy in an adult

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with 17q12 deletion. **Case Presentation:** A 31-year-old woman with a confirmed pathogenic 17q12 deletion (1,377 kb) encompassing *HNF1B* presented with chronically fluctuating transaminitis (ALT intermittently exceeding 200 U/L), markedly elevated gamma-glutamyl transferase (GGT up to 521 U/L, 13 times the upper limit of normal), elevated alkaline phosphatase (up to 355 U/L) and elevated serum bile acids for 3 years. Additional clinical features involved recurrent abdominal pain, bilateral renal cysts, and severe hypomagnesemia requiring intravenous supplementation. Comprehensive chronic liver disease workup was unrevealing. Two liver biopsies performed three years apart demonstrated progression from resolving acute hepatitis without fibrosis (2023) to mild, nonspecific resolving hepatitis with new mild portal fibrosis (2026). Immunohistochemistry with CK19 on the second biopsy demonstrated the absence of bile ducts in several portal tracts, suggestive of mild ductopenia, and CK7 showed periportal biliary metaplasia findings consistent with a developmental cholangiopathy attributable to *HNF1B* haploinsufficiency. The biochemical and histologic profile was most consistent with a nonspecific hepatitis not classifiable as primary biliary cholangitis, primary sclerosing cholangitis, or any recognized autoimmune or metabolic biliary disease. **Discussion:** *HNF1B* is essential for bile duct morphogenesis, cholangiocyte differentiation, and ciliary function. HNF1B haploinsufficiency is recognized in pediatric hepatobiliary disease, yet in adults, manifestations are presumed static. This case challenges that assumption, demonstrating histologic progression over 3 years. This likely reflects developmental bile-duct paucity compounded by impaired cholangiocyte regeneration. This chronic hepatitis was unclassifiable as any recognized biliary disease but due to HNF1B-driven cholangiopathy. CK19 and CK7 immunohistochemistry proved essential for detecting these findings. Extrahepatic features such as renal cysts and diabetes should prompt consideration of HNF1B deficiency in unexplained chronic liver disease. **Conclusion:** 17q12 deletion syndrome may manifest as chronic hepatopathy with histologically demonstrable ductopenia and may lead to portal fibrosis. This is consistent with HNF1B's established role in biliary development. Longitudinal surveillance should be considered in affected patients.

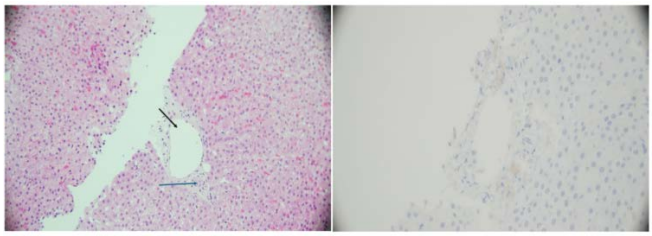


Figure : a) The portal tracts lack bile ducts, portal venules (black arrow) and hepatic arterioles (blue arrow) seen (HE, 20x)

Figure b): The portal tracts lack bile ducts, CK19 is negative. (IHC CK19, 40X)

4642 | NEW SUBMISSION

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Introduction: Progressive familial intrahepatic cholestasis is a group of rare genetic conditions with mutations that impair bile secretion. Progressive familial intrahepatic cholestasis 3 (PFIC3) is due to a mutation in ABCB4, a gene encoding MDR3 that is crucial to phosphatidylcholine excretion. PFIC3 often manifests in early childhood with symptoms of cholestasis. Given patients present with symptoms similar to other biliary diseases, adult presentations are easy to misdiagnose. **Case Presentation:** A 34-year-old woman with compensated cirrhosis was referred to hepatology for presumed small-duct PSC. She had a longstanding history of pruritus and abnormal liver studies since the age of 10, when she was started on ursodiol. She underwent liver biopsies at ages 14 and 16, which showed nonspecific chronic biliary obstruction and portal fibrosis of unclear etiology. Around the time of her second biopsy, she also developed symptomatic cholelithiasis requiring cholecystectomy. Despite being on ursodiol, her liver disease progressed, with liver biopsy at age 26 showing liver cirrhosis with nonspecific histologic features that could not exclude autoimmune disease. She was trialed on 4 months of steroids, which did not improve her liver enzymes and thus discontinued. MRCP at that time also revealed a normal-appearing biliary tract without beading or strictures. She was subsequently diagnosed with ulcerative colitis and, at age 29, began having MRCPs showing mild intrahepatic biliary duct dilation and irregularities. As a result, she was diagnosed and managed as having small-duct primary sclerosing cholangitis (PSC). Upon reassessment in 2026, it was discovered that her brother had passed at age 33 from esophageal variceal hemorrhage. Given childhood onset of symptoms with progression to cirrhosis along with family history of liver disease, genetic testing for cholestatic liver pathologies was ordered. The panel resulted with a pathogenic ABCB4 variant (Pro726Leu), which supported a finding of PFIC3. She was continued on ursodiol and also started on an ileal bile acid transport inhibitor. **Discussion:** This case highlights the diagnostic difficulty of PFIC3. It can commonly be misdiagnosed as other cholestatic liver diseases such as autoimmune cholangiopathy, small-duct PSC, and cryptogenic cholestatic liver disease, given nonspecific radiographic and histologic findings along with varying degrees of illness severity. Young age of symptom onset along with a family history can help point to an inherited cholangiopathy, with genetic testing being diagnostic. **Conclusion:** Adult patients with early-onset

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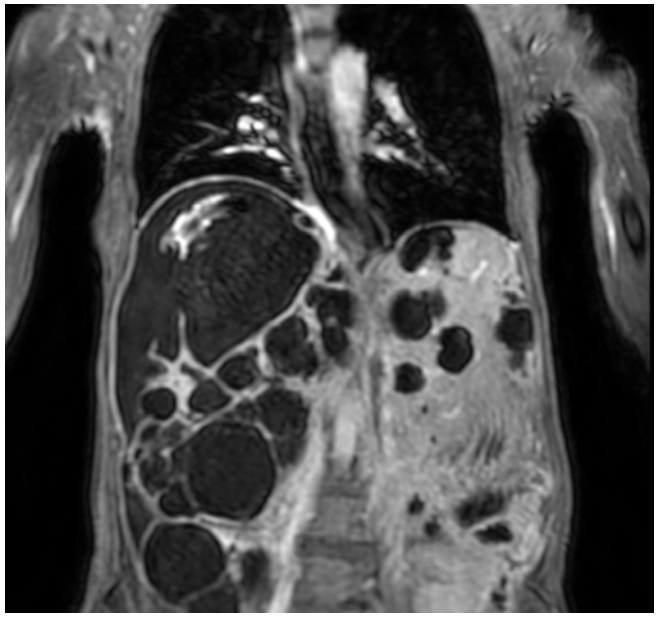
pruritus, cholelithiasis, abnormal liver enzymes, and cirrhosis along with a family history of liver disease should be evaluated for an inherited cholangiopathy, such as PFIC3. Early genetic testing can decrease time to diagnosis and influence medical care and family counseling.

4643 | WHEN THE LIVER SILENCES THE ESOPHAGUS: PROGRESSIVE DYSPHAGIA FROM GASTROESOPHAGEAL MASS EFFECT OF MASSIVE POLYCYSTIC LIVER DISEASE

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Introduction: Autosomal dominant polycystic kidney disease (ADPKD) may be accompanied by polycystic liver disease (PLD), causing marked hepatomegaly and mass effect on adjacent structures. Dysphagia caused by gastroesophageal compression is extremely rare and primarily reported in isolated case reports. We describe a case with severe PLD presenting with progressive oral intolerance and nutritional compromise initially attributed to primary esophageal disease. **Case Presentation:** A 64-year-old woman with ADPKD, PLD, and prior esophageal narrowing developed months of progressive dysphagia from solids to liquids, regurgitation, nausea, vomiting, and inability to maintain oral intake. Over four months, she lost approximately 30 lb and developed malnutrition, with a body mass index of 17.7 kg/m², requiring multiple hospitalizations. Computed tomography and upper gastrointestinal contrast imaging showed a dilated, fluid-filled esophagus with long-segment distal narrowing across the gastroesophageal junction (GEJ). Stricture, achalasia, and superior mesenteric artery syndrome were taken into consideration after esophagogastroduodenoscopy revealed distal food impaction, proximal esophageal dilatation, and traversable GEJ narrowing. Subsequent magnetic resonance imaging revealed severe cystic hepatomegaly, abrupt esophageal cutoff at the GEJ, and gastric compression with lateral displacement by the enlarged liver. Following multidisciplinary evaluation, she underwent laparoscopic fenestration of four dominant hepatic cysts with evacuation of approximately 3 L of fluid, resulting in temporary improvement in oral tolerance. Dysphagia recurred, and percutaneous aspiration was deferred because diffuse cyst burden was unlikely to permit durable decompression. An open feeding jejunostomy was placed for enteral nutrition. The patient remains under evaluation for liver transplantation. **Discussion:** This case demonstrates an unusual mechanical complication of diffuse PLD that mimicked primary esophageal disease. An extrinsic mechanism was supported by traversable narrowing without a fixed intraluminal lesion, the anatomic mass effect, and the transient response to cyst fenestration. The limitations of localized therapy are demonstrated by the recurrence of dysphagia following treatment when the mass effect of residual cyst burden persists. Severe nutritional compromise combined with persistent compressive symptoms may require long-term nutritional support and liver transplant evaluation. **Conclusion:** Massive PLD should be considered in unexplained progressive dysphagia when endoscopy does not identify a sufficient intraluminal cause and imaging demonstrates compatible gastroesophageal mass effect. Recognizing this rare extrinsic mechanism can redirect care toward hepatobiliary intervention, nutritional optimization, and liver transplant evaluation.



4644 | INCIDENTAL DIAGNOSIS AND TREATMENT OF HCV CRYOGLOBULINEMIA HALTING NEED FOR URGENT LIVER-KIDNEY TRANSPLANT EVALUATION

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[1]OHSU, [2]Oregon Health And Science University

Introduction: Acute-on-chronic kidney injury (AKI/CKD) in cirrhosis often reflects prerenal azotemia, hepatorenal syndrome, or bile cast nephropathy, but atypical causes-including HCV (hepatitis C)-associated cryoglobulinemia-must be considered. 40-60% of pa-

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tients with HCV have circulating cryoglobulins; but only approximately 0.36% develop membranoproliferative glomerulonephritis (MPGN), with cryoglobulinemia renal involvement being a poor prognosis. **Case Presentation:** A 41-year-old man with alcohol-associated decompensated cirrhosis complicated by recurrent hepatic encephalopathy (HE), variceal bleeding in 2023 with banding, trace ascites, portal hypertensive gastropathy, severe thrombocytopenia, and CKD (presumed HTN vs HRS type 2, baseline creatinine 3.0) presented with altered mental status that resolved with lactulose and rifaximin, consistent with hepatic encephalopathy. Labs showed metabolic acidosis (pH 7.4, pCO2 25, HCO3 15), hyperkalemia with AKI/CKD (Cr 3.49, BUN 90, K 5.9), acute-on-chronic anemia (Hb 6.3), chronic thrombocytopenia (Plt 25), MELD 20 predominantly from AKI, total bilirubin 1.2, with proteinuria and hematuria (20 RBCs, protein 100 mg/dL, no casts). During liver transplant evaluation, HCV RNA was 632,000 IU/mL. Serologies showed elevated rheumatoid factor (141), C3 of 39, low C4 (<8), and low-titer ANA (<1:80), anti-smooth muscle (1:40), and antimitochondrial (1:80) antibodies. Cryoglobulins returned positive. Sofosbuvir/velpatasvir (Epclusa) was started 7/11, with creatinine improving rapidly from 5.1 (7/9) to 3.7 (7/12) to 2.8 (7/15) at discharge, temporizing the need for urgent simultaneous liver-kidney (SLK) transplant. **Discussion:** While undergoing SLK transplant evaluation in the setting of MELD > 15, and glomerular function < 30%, the decision was made to initiate HCV treatment to reduce potential cryoglobulinemia-related kidney failure and impending dialysis. The creatinine began improving 1-2 days prior to Epclusa, but given continued resolution without additional fluids or medications, we favor HCV treatment as a contributing factor the continued and sustained improvement in his acute kidney injury. **Conclusion:** An atypical renal presentation in a patient with cirrhosis particularly with HCV viremia -hematuria, proteinuria, and hypocomplementemia rather than a bland AKI-should prompt broader renal evaluation before attributing renal failure to HRS or CKD. Engaging nephrology colleagues and checking for cryoglobulinemia can change both the diagnosis and the transplant plan. Because direct-acting antiviral therapy can rapidly reverse glomerular injury, treating active HCV first may recover renal function and eliminate the kidney indication for SLK transplant.

4645 | ACUTE WILSONIAN CRISIS UNMASKING PREVIOUSLY UNRECOGNIZED WILSON DISEASE IN A YOUNG WOMAN WITH CRYPTOGENIC CIRRHOSIS

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[1]Vanderbilt University Medical Center

Introduction: Wilson disease (WD), an autosomal recessive disorder of copper metabolism due to ATP7B mutations, causes toxic hepatic copper accumulation. WD can present as chronic liver disease, neuropsychiatric illness, or acute Wilsonian crisis, an often fatal presentation with Coombs-negative hemolytic anemia, coagulopathy, encephalopathy, and low alkaline phosphatase (ALP) relative to bilirubin. Without transplantation, mortality approaches 100%. We describe previously unrecognized WD, initially labeled cryptogenic cirrhosis, that progressed to acute Wilsonian crisis. **Case Presentation:** A 38-year-old woman with recently diagnosed decompensated cirrhosis, initially considered cryptogenic, presented with lower extremity edema, abdominal pain, and nausea. Autoimmune and viral workups were negative; ceruloplasmin was low (13 mg/dL) with elevated 24-hour urine copper (773.5 mcg), suggesting WD. She was discharged after diuretic optimization but returned weeks later with worsening jaundice, leukocytosis, and coagulopathy (INR 3), then decompensated with hypotension, altered mentation, and hemoglobin drop from 12.3 to 8 g/dL without a source, suggesting hemolysis. Labs showed INR 2.9, bilirubin 29.6 mg/dL, ALP 40 U/L (ALP:bilirubin ratio 1.4), AST 193, ALT 52 (AST:ALT ratio 3.7); MELD 3.0 31. She was transferred to the ICU, started on plasma exchange, and urgently underwent liver transplantation. Post-transplant, explant copper quantification confirmed markedly elevated hepatic copper (1656 mcg/g dry weight; reference <50); ATP7B sequencing showed heterozygous pathogenic (p.His1069Gln) and uncertain-significance (p.Cys985Arg) variants. **Discussion:** This case illustrates how acute Wilsonian crisis can unmask previously undiagnosed WD in a patient labeled with cryptogenic cirrhosis. Her presentation fulfilled the classic diagnostic pentad: hemolysis, coagulopathy, encephalopathy, low ALP:bilirubin ratio (<4), and elevated AST:ALT ratio (>2.2), prompting urgent transplant listing before confirmatory testing resulted. Diagnosis was later confirmed by explant copper quantification and a pathogenic ATP7B variant; her second allele was a variant of uncertain significance, underscoring that genetic testing should not delay time-sensitive transplant evaluation. Diagnosis instead integrates clinical, biochemical, and genetic data (Leipzig score), while the New Wilson Index helps identify such pa-

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tients early. **Conclusion:** Acute Wilsonian crisis should be considered in any patient with rapidly progressive liver failure, hemolytic anemia, and coagulopathy, even when cirrhosis has been attributed to another or unknown cause. A low ALP:bilirubin ratio with elevated AST:ALT ratio should prompt urgent hepatology involvement andtransplant listing without awaiting genetic confirmation. A variant of uncertain significance should not exclude WD when clinical and biochemical findings are diagnostic.

4646 | PORTAL VEIN RECANALIZATION IN UMBILICAL CATHETERIZATION-ASSOCIATED THROMBOSIS: WHEN BLEEDING CHALLENGES ANTICOAGULATION

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Introduction: Portal vein thrombosis from umbilical catheterization causes pediatric portal hypertension. While bare-metal stent placement enables portal vein recanalization, postoperative anticoagulation is essential to prevent thrombotic occlusion. Severe thrombocytopenia and concurrent drug anaphylaxis can render anticoagulation impossible, forcing individualized decision-making. **Case Presentation:** A 16-year-old male with umbilical catheterization-associated portal vein thrombosis presented with acute variceal hemorrhage after elastic band ligation. Laboratory findings: hemoglobin 6.8 g/dL, platelets 17,000/μL, INR 1.89. Interventional radiology performed portal vein recanalization with bare-metal stent placement (10 × 80 and 12 × 60 mm) and collateral embolization.Critical conflict emerged: anticoagulation was contraindicated due to platelets <50,000/μL. Additionally, propranolol-the standard vasoactive bridge-was contraindicated by prior anaphylaxis history to milk protein with requirement for multiple epinephrine doses.The patient remained hemodynamically stable without recurrent bleeding. Platelets recovered to 35,000-47,000/μL by week 2, yet anticoagulation was withheld. Serial imaging confirmed stent patency without thrombosis. At 3-month follow-up, he remained asymptomatic with platelets 35,000/μL and patent stent on ultrasound. **Discussion:** This case demonstrates a rare but instructive clinical impasse: the collision between two absolute contraindications (anticoagulation-thrombocytopenia and propranolol-anaphylaxis) that together prevent implementation of guideline-recommended therapies. While observational management without anticoagulation after bare-metal stent placement contradicts standard practice, close hematologic monitoring and serial vascular imaging identified no thrombotic complications during 9 months of follow-up. The favorable outcome likely reflected dual anatomic/clinical factors: (1) successful embolization of high-pressure portosystemic collaterals reduced portal pressure gradient, theoretically diminishing stent thrombosis risk, and (2) gradual platelet recovery to >30,000/μL may have provided sufficient hemostasis for endothelial healing without antiplatelet activation.This case emphasizes that rigid protocol adherence may prove unsafe when competing contraindications exist, and that multidisciplinary teams (pediatric hepatology, interventional radiology, hematology, allergy-immunology) must weigh competing risks individually rather than defaulting to guidelines. **Conclusion:** Complex pediatric portal hypertension cases demand individualized risk-benefit assessment when standard therapies become medically contraindicated. Careful hemodynamic monitoring and serial imaging surveillance may provide acceptable alternatives to conventional anticoagulation in selected high-risk scenarios.

4647 | CRYPTOSPORIDIUM-INDUCED SCLEROSING CHOLANGITIS IN A PEDIATRIC LIVER TRANSPLANT RECIPIENT

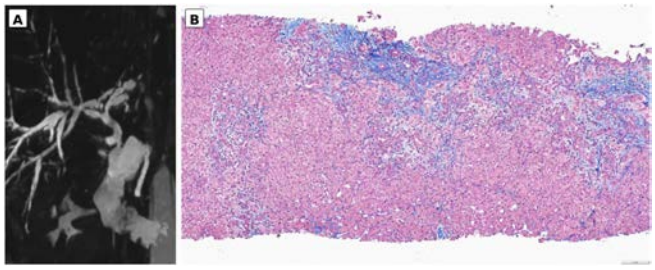
Ahmed Elhatw[1] Nawras Habash[2] Samar H. Ibrahim[3]

[1]Mayo Clinic , Rochester, [2]Mayo Clinic, Rochester, [3]Mayo Clinic Rochester

Introduction: Biliary complications after pediatric liver transplantation (LT) may lead to graft dysfunction or loss. While most are anastomotic or ischemic, infectious causes are uncommon but potentially reversible. Cryptosporidium can cause severe biliary disease in immunocompromised patients. We report Cryptosporidium-associated secondary sclerosing cholangitis in a pediatric LT recipient **Case Presentation:** An 8-year-old female underwent LT at 3 months of age for gestational alloimmune liver disease, with choledochoduodenostomy for biliary reconstruction. Her subsequent course was complicated by acute cellular and chronic ductopenic rejection, which resolved with intensified immunosuppression. Maintenance immunosuppressive therapy consisted of tacrolimus and sirolimus.Seven years after transplantation, she presented during a diarrheal illness with a cholestatic-predominant elevation in liver enzymes: ALT 220 U/L, AST 216 U/L, GGT 501 U/L, and total bilirubin 0.6 mg/dL. Laboratory evaluation also demonstrated a white blood cell count of 10.5 × 10^9/L, thrombocytosis with a platelet count of 635 × 10^9/L, and elevated CRP, with preserved hepatic synthetic function. One year earlier, she had Cryptosporidium infection associated

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with diarrhea and acute cellular rejection, treated with pulse methylprednisolone and nitazoxanide. She was discharged on a prednisone taper, sirolimus, and tacrolimus. She subsequently moved to a rural area, was lost to follow-up, and did not complete scheduled stool studies to confirm clearance of Cryptosporidium.Ultrasound demonstrated patent hepatic vasculature and central bile duct dilation. MRCP showed multifocal intrahepatic and extrahepatic biliary beading and irregularity with a patent choledochoduodenostomy (Figure 1A). Liver biopsy demonstrated a biliary obstructive pattern with portal and focal bridging fibrosis without acute cellular rejection (Figure 1B). Stool testing was positive for Cryptosporidium. She was treated with nitazoxanide and ursodiol. Recent laboratory studies showed normal direct bilirubin and ALT, with downtrending AST (44 U/L) and GGT (109 U/L). **Discussion:** Biliary cryptosporidiosis is a rare but potentially serious complication after pediatric LT. Persistent infection in the setting of intensified immunosuppression may promote biliary epithelial injury, leading to progressive cholangiopathy and fibrosis. **Conclusion:** Cryptosporidium should be considered in pediatric LT recipients on multiple immunosupressive therapy presenting with cholestatic liver enzyme abnormalities and biliary strictures, particularly with diarrheal illness. Prompt recognition and treatment may prevent progressive biliary injury and graft loss



4648 | FLYING UNDER THE RADAR: A CASE OF OCCULT METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS BACTEREMIA IN A PATIENT WITH NEW TRACE ASCITES AND ACUTE-ON-CHRONIC HYPONATREMIA

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[1]Montefiore Medical Center

Introduction: Patients with cirrhosis are immunocompromised and high-risk for developing occult infections. We present a case of methicillin-resistant Staphylococcus aureus (MRSA) bacteremia with spinal abscesses in a patient with cirrhosis who presented without fever, leukocytosis, or hemodynamic instability. **Case Presentation:** A 55-year-old man with alcohol-associated cirrhosis complicated by ascites (on furosemide 20 mg daily), large non-bleeding esophageal varices, and chronic hyponatremia presented with one week of dull, aching mid-thoracic back, left shoulder, neck, and left hip pain that started after throwing out a 30-pound garbage bag. Pain worsened with movement and was minimally relieved with acetaminophen, methocarbamol, and topical analgesic creams. He denied fever, chills, cough, abdominal pain, urinary symptoms, or alcohol or drug use. On arrival, his vitals were normal, and he was afebrile. Examination was notable for lower extremity edema, a soft, non-distended abdomen, and mild tenderness over the left deltoid and cervical paraspinal muscles. Laboratory studies revealed sodium of 123 mEq/L (baseline 130 mEq/L) likely secondary to hypervolemic hyponatremia from cirrhosis and total bilirubin of 3.5 mg/dL (baseline 1.6 mg/dL). He had baseline anemia, thrombocytopenia, and coagulopathy but no leukocytosis or renal injury. Chest radiography was unremarkable. Liver ultrasound revealed a cirrhotic liver with new trace ascites compared to two months prior.Due to worsening hyponatremia and new ascites, urine and blood cultures were obtained. On hospital day two, he developed a fever of 101.7 F and blood cultures grew MRSA. He was started on vancomycin. Orthopedic surgery was consulted, and clinical suspicion for septic arthritis of hip and shoulder was low. Magnetic resonance imaging of the spine revealed a C4-C5 epidural abscess, a lateral S1-S2 abscess, and an L5-S1 phlegmon. Transthoracic echocardiography showed no vegetations. Transesophageal echocardiography was deferred given patient was not a surgical candidate. Per neurosurgery and infectious disease, he was treated with antimicrobials alone with subsequent clearance of blood cultures. **Discussion:** A patient with cirrhosis presented with musculoskeletal pain following an injury and had no clinical signs of sepsis. Based on guidelines, worsening baseline hyponatremia and new trace ascites prompted an infectious workup that revealed MRSA bacteremia with multiple spinal abscesses. Due to gut microbial translocation, bacteremia in cirrhosis is mostly caused by enteric gram negative organisms; however, a high index of suspicion should be maintained for gram positive infections with unexplained musculoskeletal symptoms. **Conclusion:** Patients with cirrhosis can present with subtle signs of MRSA bacteremia. A low threshold for obtaining blood cultures should be maintained to evaluate worsening hyponatremia, new trace ascites, or unexplained pain.

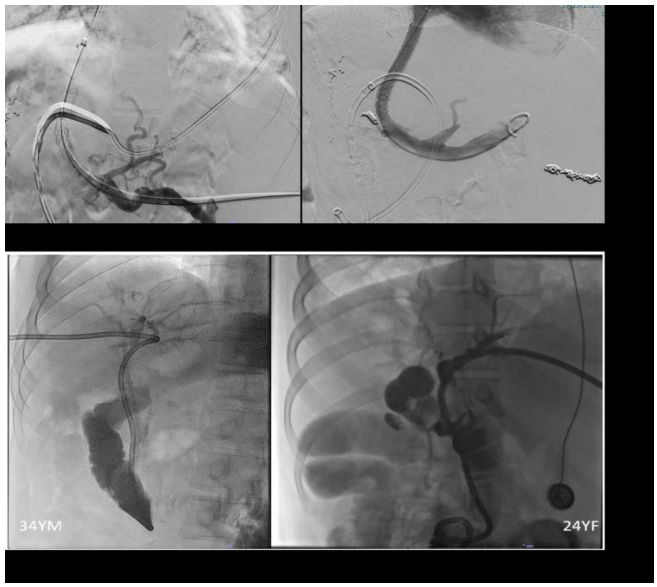
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4649 | AN EXIT OPTION: A CASE SERIES OF PERCUTANEOUS TRANSHEPATIC BILIARY DRAIN PLACEMENT FOR COMPLICATED PORTAL CAVERNOMAS

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Introduction: A portal cavernoma is a compensatory network of hepatopetal collateral veins shunting around chronic portal vein obstructions. We describe two cases of portal cavernomas resulting in portal bilopathy, recurrent cholangitis and hemobilia. Both required percutaneous transhepatic biliary drain (PTBD) for decompression but with divergent management. **Case Presentation:** 24YF with biliary atresia s/p liver transplant at age 2 with hepaticojejunostomy (HJ) anatomy, history of acute cellular rejection (no fibrosis on biopsy) and portal vein thrombosis (~10 yrs) with cavernous transformation, presented with epigastric pain, fever and vomit. Lab studies were INR 1.7, TB 2.6, AST 47, ALT 36, ALP 239. She had similar uncomplicated presentations for 2 years, resolving with antibiotics. CT scan showed new bile duct dilation. ERCP was unsuccessful but revealed hemobilia. Percutaneous cholangiogram performed with HJ balloon dilation to 10mm and intrabiliary ultrasound showed multiple portal collaterals surrounding the HJ narrowing. Intermittent bile duct "obstruction" was suspected due to anastomotic fibrosis and extrinsic compression from cavernoma. A PTBD was placed across the HJ for decompression. TIPS shunt was performed for portal vein recanalization. After coil embolization of large splenorenal shunt to optimize flow, her cavernoma decompressed. PTBD was removed 1 month later with TB 1.8, ALP 182 and ALT 25.34YM with ALL in remission, MASLD cirrhosis (varices and portal cholangiopathy), and chronic portal, superior mesenteric, and splenic vein thrombosis with cavernous transformation, presented with RUQ pain, jaundice and fever. Had 3 prior cholangitis admissions with hemobilia and stent occlusion from extrinsic biliary compression 2/2 portal cavernomas; last ERCP 3 weeks prior where covered metal and plastic stent placed in CBD. On presentation, TB 4.5, ALP 458, ALT 116, and AST 108. CT showed intrahepatic biliary ductal dilatation. MRI showed portal cavernoma and biliary biliopathy. Plastic stent was removed during ERCP; cultures grew E. faecium and yeast. To prevent further stent occlusion, the patient underwent PTBD. TIPS to the splenic and portal veins failed due to highly organized clot. Currently, PTBD remains in place and the patient is pursuing multivisceral transplant. **Discussion:** Both patients underwent PTBD placement due to recurrent cholangitis and hemobilia occurring secondary to extrinsic biliary compression from extensive portal cavernomas. One had successful decompression of cavernoma with portal vein recanalization via TIPS shunt and PTBD removal. The other patient unfortunately had lost splenic access about 6 years prior. **Conclusion:** There is no clear guidance of portal cavernoma complications that are refractory to TIPS. PTBD can act as a bridge to allow stabilization of these patients and prevent cholangitis, especially in immunosuppressed patients, while working towards definitive management.



4650 | PORTO SINUSOIDAL VASCULAR DISEASE MIMICKING HEPATOCELLULAR ADENOMA IN A YOUNG MAN

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Introduction: Porto sinusoidal vascular disease (PSVD) can occur without portal hypertension and may produce focal hepatic lesions. We report focal PSVD mimicking hepatocellular adenoma (HCA) in a young man. **Case Presentation:** A 21-year-old man was incidentally found to have hypervascular liver lesions during evaluation of nonspecific abdominal pain. He denied anabolic steroid use. MRI showed a 1.1 cm arterially enhancing segment 3 lesion interpreted as HCA. Liver tests and platelet count were normal, with no clinical or imaging signs of portal hypertension. Serial MRI showed persistence with slight enlargement to 1.5 cm. CT guided core biopsy showed benign liver parenchyma without adenoma. Endoscopic ultrasound (EUS) guided fine needle aspiration (FNA) showed mildly

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atypical hepatocellular fragments with diffuse sinusoidal CD34 staining, raising suspicion for HCA, although interpretation was limited by fragmented sampling. Multidisciplinary review favored HCA. Given the malignant potential of HCA in men, resection was recommended. Left lateral segmentectomy was performed. The targeted lesion showed sinusoidal dilatation, ectatic vessels, mild nodular regenerative hyperplasia, and sinusoidal CD34 staining consistent with PSVD. No HCA, focal nodular hyperplasia (FNH), or malignancy were identified. **Discussion:** Focal hepatic lesions in PSVD are most often FNH like, making a discrete lesion that mimicked HCA without a hepatocellular neoplasm an unusual presentation. The initial core biopsy likely sampled nonlesional liver, while FNA captured abnormal hepatocytes but lacked the architecture needed to recognize PSVD. This limitation is important because PSVD changes can be subtle and unevenly distributed and diagnosis depends on adequate tissue. In this case, sinusoidal CD34 staining supported HCA on FNA but was also present in the resection specimen, showing that this finding was not specific for adenoma. PSVD may be diagnosed without portal hypertension when a specific histologic lesion such as nodular regenerative hyperplasia is present. **Conclusion:** PSVD should be considered when a presumed HCA remains discordant across imaging and limited tissue sampling. Adequate tissue that preserves architecture and includes adjacent liver may be needed to distinguish vascular regenerative change from hepatocellular neoplasia.

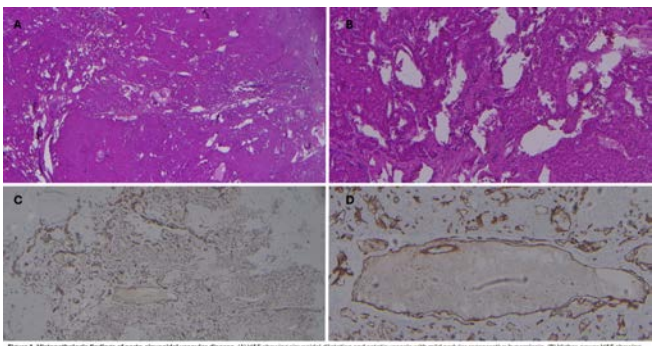


Figure 1. Histopathologic findings of porto-sinusoidal vascular disease. (A) H&E showing sinusoidal dilatation and ectatic vessels with mild nodular regenerative hyperplasia. (B) Higher-power H&E showing sinusoidal dilatation and ectatic vessels. (C) CD34 immunohistochemistry showing sinusoidal staining. (D) Higher-power CD34 immunohistochemistry showing sinusoidal staining.

4651 | BEYOND THE THRESHOLD: DIPS IN A PATIENT WITH BUDD CHIARI SYNDROME AND EJECTION FRACTION < 50%

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Introduction: Clinically significant portal hypertension (CSPH) is a common complication of Budd Chiari Syndrome (BCS). Interventional therapies such as transjugular intrahepatic portosystemic shunt (TIPS) placement and direct intrahepatic portosystemic shunt (DIPS) placement are frequently used to manage CSPH. **Case Presentation:** A 56 y/o female with prior DCIS s/p bilateral mastectomy is diagnosed with catastrophic antiphospholipid syndrome (CAPS) shortly after undergoing elective bilateral axillary liposuction and left breast implant placement with a plastic surgeon. She develops pulmonary embolism, left and middle hepatic venous thrombosis consistent with Budd Chiari Syndrome (BCS) and new inflammatory myocarditis with an ejection fraction (EF) of 35%. She has positive cardiolipin antibodies and B2 glycoprotein antibodies. The patient is initially treated with heparin, IVIG, and prednisone. She is discharged on prednisone taper, anticoagulation with warfarin and GDMT for new HFrEF. She receives rituximab as an outpatient. Post-hospitalization imaging shows persistent hepatic vein thromboses despite therapeutic anticoagulation. The patient develops CSPH with large varices that require banding and refractory ascites requiring weekly paracenteses. The patient is not a candidate for revascularization due to concern for thromboembolism. The patient's EF improves to 43% on cardiac MRI post-discharge, below AASLD guidelines for TIPS/DIPS. After multidisciplinary discussion, the decision is made to proceed with DIPS with close hemodynamic monitoring via pulmonary artery catheter. Pre-procedural left and right heart catheterization showed no coronary artery disease and normal right heart pressures. To decrease the magnitude of preload increase with shunt creation, a shunt diameter of 8 mm was utilized. Postoperative course was uncomplicated. The patient's hemodynamics were monitored closely in an ICU setting. The patient's CSPH has improved; she has not required paracentesis in 4 weeks. She has lower extremity edema which is being treated with oral diuretics. **Discussion:** Current AASLD guidelines list EF less than 50% as a contraindication to TIPS/DIPS placement, likely due to concern for cardiac decompensation with shunt placement. Heart failure has a heterogeneous clinical phenotype, and current guidelines may be precluding compensated patients (such as ours) who otherwise benefit from TIPS/DIPS. Work by Billey et al (2019) and a systematic review by Tomassoni et al (2025) did not identify EF as a predictor of post-TIPS cardiac decompensation. This suggests that there are other factors that better characterize the risk of cardiac decompensation. **Conclusion:** Our case highlights that compensated patients with an EF less than 50% may tolerate TIPS/DIPS for BCS management. Further study into cardiac risk stratification in this population is warranted to better identify suitable candidates for TIPS/DIPS.

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4652 | CONGENITAL INTRAHEPATIC PORTOSYSTEMIC SHUNT AS A RARE MANIFESTATION OF NOONAN SYNDROME: A CASE REPORT

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Introduction: Noonan syndrome (NS) is an autosomal dominant genetic disorder caused by gain-of function mutations in the RAS-MAPK signaling pathway, classically presenting with congenital heart disease. Although rare, emerging evidence links NS and the broader category of RASopathies to congenital hepatic vascular abnormalities. The most common is a congenital extrahepatic portosystemic shunt called Abernathy malformation. We present the case of a 29-year-old female with NS who developed intermittent hyperbilirubinemia and imaging findings of portal hypertension and was subsequently diagnosed with a congenital intrahepatic portosystemic shunt (IHPSS). **Case Presentation:** A 29-year-old female with a past medical history of NS and splenomegaly noted on imaging since birth presented with intermittent hyperbilirubinemia (total bilirubin 1.4 mg/dL). A follow-up abdominal ultrasound showed a dilated main portal vein without significant liver parenchymal disease. MRI obtained one year later showed evidence of a venovenous shunt between the right hepatic vein and right portal vein branches as well as a newly dilated inferior vena cava. Overall, her imaging was concerning for congenital IHPSS and evolving noncirrhotic portal hypertension. Concurrent serological testing was negative for an alternate cause of these findings, including alpha-1-antitrypsin phenotyping, ANA, ceruloplasmin, smooth muscle antibody, mitochondrial antibody, IgG, ferritin, and hepatitis serologies. **Discussion:** This case highlights IHPSS as a rare hepatic vascular manifestation of NS. While Abernathy malformations are increasingly recognized in RASopathies, IHPSS reports in NS remain exceedingly rare. The pathophysiological basis likely involves impaired embryonic vascular remodeling due to dysregulated RAS-MAPK signaling, preventing normal regression of fetal portosystemic communications.It is important for clinicians to recognize IHPSS as a potential manifestation of NS, as these congenital vascular malformations are associated with porto-sinusoidal vascular disease as well as liver lesions such as adenomas and hepatocellular carcinoma in the absence of cirrhosis. These patients are also at risk for the complications associated with portal hypertension, including hepatic encephalopathy, variceal bleeding, portopulmonary hypertension, ascites, and lower extremity edema. The increased risk of these complications indicates increased need for monitoring and surveillance in the NS patient population. **Conclusion:** This case highlights the importance of recognizing congenital IHPSS as a rare but clinically significant hepatic vascular manifestation of NS. Early identification through vascular imaging is essential to enable timely surveillance and management of associated complications of portal hypertension, including hepatic encephalopathy, variceal bleeding, portopulmonary hypertension, and liver lesions.



Image 1. CT abdomen and pelvis coronal view showing splenomegaly along with multiple splenic artery aneurysm (red arrows), largest measuring 2.3 cm, secondary to underlying portal hypertension.

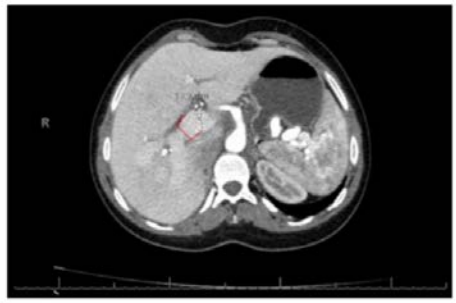


Image 2. CT abdomen and pelvis axial view showing dilated main portal vein (red bracket), measuring 18.9 mm, as evidence of portal hypertension.

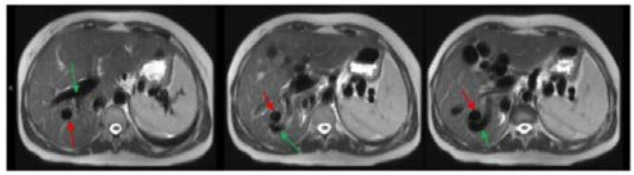


Image 3. Contrast enhanced MRI of liver showing axial views of large shunt between the right hepatic vein (red arrow) and the right portal vein (green arrow).

4653 | STATUS EPILEPTICUS AS AN UNUSUAL PRESENTATION OF RECURRENT HEPATIC ENCEPHALOPATHY IN A PATIENT WITH A LARGE SPONTANEOUS SPLENORENAL SHUNT

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Introduction: Hepatic encephalopathy (HE) is a neuropsychiatric manifestation of liver dysfunction and/or portosystemic shunting. In patients with recurrent or persistent HE despite optimized medical therapy, large spontaneous portosystemic shunts (SPSS) may be present, allowing portal blood to bypass hepatic detoxification. We describe a patient with recurrent HE despite lactulose and rifaximin who developed severe hyperammonemia with status epilepticus in the setting of a large splenorenal shunt. **Case Presentation:** A 44-year-old man with alcohol-associated cirrhosis, MELD 3.0 of 15, prior HE, and no seizure history presented with status epilepticus requiring intubation and treatment with several antiepileptics. Family described witnessing four generalized seizure episodes without interval return to baseline. Despite adherence to lactulose and rifaximin, he had had five prior hospitalizations for HE. Of note, he had never had ascites or variceal hemorrhage. Infection, hepatic tumor, clots, gastrointestinal bleeding, and sedative medications were ruled out as possible contributors at every hospitalization. Ammonia level was 322 µmol/L at presentation, and he was aggressively treated with lactulose and rifaximin. Brain imaging was normal and continuous EEG showed severe diffuse encephalopathy with triphasic waves. Neurology opined that status epilepticus and seizures were provoked by severe hyperammonemia. Abdominal CT demonstrated cirrhosis with a large SPSS. Venography further characterized a large, high-flow splenorenal shunt draining into a hypertrophied but patent left renal vein with multiple collaterals. Interventional radiology performed successful retrograde transvenous obliteration using an Amplatzer plug at the shunt-renal vein confluence reinforced by coils, additional coils occluding secondary outflow branches, and embolic/sclerosant material filling the shunt lumen (Figure 1). Post-embolization venography showed complete shunt stasis with preserved left renal vein patency. His mentation improved dramatically after the procedure, returning to baseline by the following day. **Discussion:** Spontaneous portosystemic shunts (SPSS) are increasingly recognized contributors to recurrent or refractory HE by diverting ammonia-rich portal blood away from hepatic metabolism. In our patient, recurrent HE occurred despite lactulose and rifaximin, and without other precipitants of HE, making the large splenorenal shunt a likely driver of his episodes. Severe hyperammonemic HE provoked seizures with status epilepticus. **Conclusion:** Large SPSS should be actively considered in patients with recurrent or medication-refractory HE, particularly when HE is the predominant or only decompensation in a patient with cirrhosis. Identification of an underlying large SPSS may reveal a treatable anatomic driver for which embolization can provide a targeted therapeutic strategy.



4654 | SYPHILITIC HEPATITIS: A CASE OF ACUTE HEPATOCELLULAR LIVER INJURY

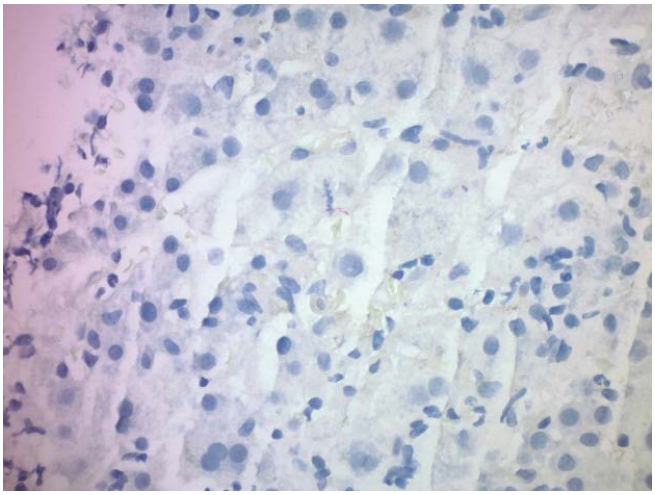
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Introduction: Syphilis has a wide clinical spectrum and can mimic many conditions; hepatic involvement is uncommon and rarely the initial presentation. We describe a rare case of early syphilis presenting as acute hepatocellular liver injury, emphasizing the importance of considering treponemal infection in patients with unexplained acute hepatitis. **Case Presentation:** A 33-year-old female with no medical history presented with two days of jaundice and malaise. She denied alcohol use, new medications, recent travel, or sick contacts. Laboratory evaluation revealed marked hepatocellular injury: AST 1426 U/L, ALT 2524 U/L, alkaline phosphatase 231 U/L, and total bilirubin 6.7 mg/dL (direct bilirubin 3.8 mg/dL). INR and creatinine were normal. She exhibited no encephalopathy. Right upper quadrant ultrasound demonstrated a normal liver without biliary dilation, patent vasculature, and normal echogenicity. Additional workup included a negative toxicology screen, negative autoimmune serologies, ferritin 1681 ng/mL, and normal ceruloplasmin, immunoglobulins, and alpha-1 antitrypsin levels. Viral

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testing for EBV, CMV, HSV, acute hepatitis B, hepatitis C, and HIV was negative. Notably, rapid plasma reagin (RPR) was positive at 1:32. Liver biopsy revealed severe acute hepatitis with portal and lobular inflammation. Immunohistochemical staining identified spirochetes, confirming syphilitic hepatitis. The patient received intramuscular penicillin G, and her liver enzymes normalized within four weeks. **Discussion:** Syphilitic hepatitis represents a critical diagnostic "pearl" due to its ability to mimic more common causes of acute liver injury. Patients may not present with classic features of syphilis including weight loss, fevers, or rash. Without biopsy, syphilitic hepatitis can be diagnosed by abnormal liver tests, serologic markers of syphilis, and exclusion of an alternative diagnosis. Syphilitic hepatitis more commonly presents with markedly elevated ALP, but it can present with markedly elevated transaminases and positive autoantibodies, causing misdiagnosis. In this case, a sexual history, positive RPR, and otherwise negative work up promoted a liver biopsy which clinched the diagnosis. Administration of intramuscular penicillin G typically results in rapid, complete resolution of hepatic injury. This case underscores the necessity of maintaining a high index of suspicion and screening for syphilis in all atypical presentations of acute hepatitis to provide prompt treatment. **Conclusion:** Syphilitic hepatitis is a rare but important cause of acute liver injury. Clinicians should obtain a sexual history and screen for syphilis in patients with unexplained acute hepatitis, even in the absence of rash or fever. Liver biopsy may be essential when diagnostic uncertainty persists.



4655 | LARGE HEPATIC GUMMA IN AN IMMUNOCOMPETENT PATIENT: A RARE TERTIARY SYPHILIS MANIFESTATION

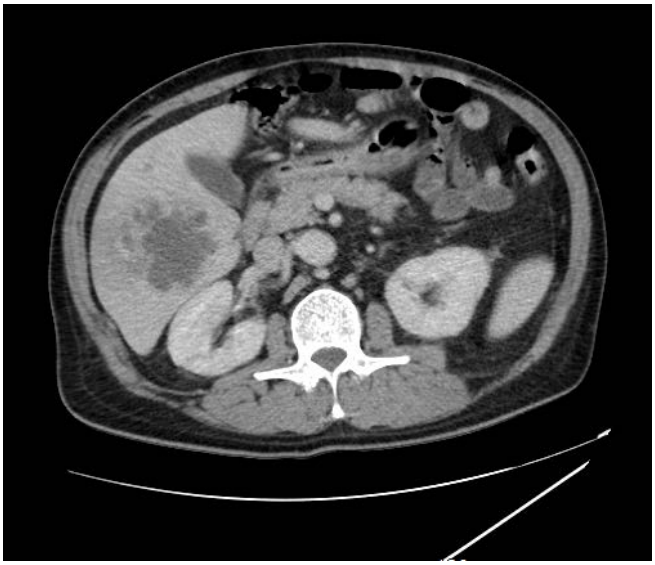
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Introduction: Hepatic gumma remains a very rare form of tertiary syphilis that mimics pyogenic abscess or hepatocellular carcinoma on imaging. In the era preceeding antibiotics, gummatous syphilis affected about 15% of untreated patients, 1 to 46 years after infection, but hepatic involvement was rare. In the largest contemporary series (14 cases), syphilis was the leading clinical suspicion in only one patient. With rising syphilis rates and benzathine penicillin G shortages persisting into 2026, awareness of uncommon presentations and alternative therapy matters. **Case Presentation:** 66-year-old male, immunocompetent and HIV-negative, with a history of penicillin allergy (causing a rash), who had a temperature of 36.6 degrees and some tenderness in the right upper quadrant. Laboratory work included leukocytosis (15.7 × 10^3/μL, with neutrophils 82%), alkaline phosphatase of 275 U/L, AST of 42 and ALT of 43 U/L, albumin was 2.3 g/dL, and bilirubin was normal. AFP, CEA and CA 19-9 were all negative.CT showed an 11-cm heterogeneous hypodense right-lobe mass with irregular margins and septations; the differential was hepatocellular carcinoma versus abscess. MRI confirmed a T2-hyperintense necrotic-appearing lesion. Ultrasound-guided drainage yielded 30 mL of purulent fluid, but bacterial, fungal, mycobacterial, and blood cultures and amoeba serology were all negative. RPR returned at 1:256 with reactive FTA-ABS; HIV and hepatitis B and C were nonreactive.A clinical diagnosis of hepatic gumma was made on the basis of high-titer serology, a sterile purulent aspirate, compatible imaging, and exclusion of malignancy and pyogenic and amoebic abscess. Given the penicillin allergy and drug shortage, the patient received doxycycline 100 mg twice daily for 28 days-the CDC-recommended alternative for late latent syphilis without neurologic involvement. Symptoms improved after drainage and therapy, with serologic follow-up planned at 6, 12, and 24 months. **Discussion:** This case shows that hepatic gumma can be diagnosed clinically when histopathology is unavailable, since aspirates often yield only necrotic debris.[5,9] It also illustrates real-world management of tertiary syphilis in a penicillin-allergic patient during a benzathine penicillin G shortage.[7,11] Most reported hepatic syphilis occurs in HIV-positive patients; this case demonstrates that a large gumma can arise in an otherwise healthy host.[13,4] **Conclusion:** Hepatic gum-

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ma can be diagnosed clinically through high-titer serology, sterile cultures despite a purulent aspirate, compatible imaging, and exclusion of alternatives, and treated with doxycycline when penicillin is unavailable. As syphilis rates rise, clinicians should suspect this treatable condition in culture-negative hepatic masses, but CSF examination should precede therapy for tertiary syphilis to exclude neurosyphilis.



4656 | MULTIFOCAL HEPATIC NODULES MIMICKING TUBERCULOSIS: A CASE REPORT OF ADULT HEPATIC LANGERHANS CELL HISTIOCYTOSIS

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Introduction: Langerhans cell histiocytosis (LCH) is a rare histiocytic disorder characterized by the accumulation and proliferation of Langerhans-type dendritic cells. Hepatic involvement is uncommon and may occur as part of multisystem disease. The clinical manifestations of hepatic LCH are variable and may include hepatomegaly, abdominal pain, cholestatic liver injury, jaundice, and, in advanced disease, hepatic dysfunction. Establishing the diagnosis requires histopathologic examination and immunophenotypic confirmation. We present an adult patient with multifocal hepatic nodules, abdominal pain, jaundice, and intra-abdominal lymphadenopathy who was initially managed for presumed hepatobiliary tuberculosis. **Case Presentation:** The patient is an adult with no known comorbidities and no previous hospitalizations who initially developed severe abdominal pain in June 2025. The patient was subsequently treated empirically for presumed schistosomiasis with praziquantel. Over the succeeding months, the patient continued to experience intermittent episodes of abdominal pain. Progressive increase in abdominal girth was subsequently noted. In December 2025, the patient developed severe abdominal pain accompanied by jaundice, prompting admission. At that time, the patient was evaluated for liver disease, with hepatobiliary tuberculosis and fungal hepatitis considered among the differential diagnoses. Abdominal imaging demonstrated hepatomegaly with multiple hepatic nodules/lesions involving both hepatic lobes. A liver biopsy was performed with specimens submitted for histopathologic examination and subsequent immunohistochemical evaluation. In March 2026, the immunohistochemical results became available and demonstrated a staining profile consistent with Langerhans cell histiocytosis. The patient was subsequently evaluated and recommended chemotherapy. **Discussion:** Langerhans cell histiocytosis is an uncommon clonal histiocytic disorder characterized by the proliferation of Langerhans-type dendritic cells. Disease may be limited to a single organ or may involve multiple organ systems. The diagnostic challenge was further complicated by the patient's chronic dry cough and the endemic nature of tuberculosis and schistosomiasis in the Philippines. The case also demonstrates an important diagnostic pitfall: hepatic LCH may present as multiple hypodense nodules and therefore mimic infection or metastatic disease radiologically. **Conclusion:** This case highlights an unusual presentation of hepatic Langerhans cell histiocytosis as multifocal hepatic nodules mimicking hepatobiliary tuberculosis and fungal hepatitis. This case emphasizes the importance of obtaining tissue diagnosis in patients with unexplained multifocal hepatic lesions. Hepatic Langerhans cell histiocytosis should be considered as a potential diagnostic mimic of multifocal hepatic infection.

4657 | PATHOLOGY-CONFIRMED METASTATIC HEPATOCELLULAR CARCINOMA PRESENTING AS AN ISOLATED CEREBELLAR METASTASIS WITHOUT AN IDENTIFIABLE HEPATIC PRIMARY

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Introduction: Brain metastases from hepatocellular carcinoma (HCC) are rare, occurring in approximately 1-2% of patients with advanced HCC and typically in the setting of extensive intrahepatic disease. Reports of pathology-confirmed metastatic HCC without an identifiable hepatic primary are exceedingly uncom-

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mon. We present a case of isolated cerebellar metastasis from HCC occurring after resection of a radiographic HCC mimic and years of negative hepatic surveillance. **Case Presentation:** A 61-year-old male with a history of atypical meningioma status post resection and chronic hepatitis B cirrhosis on entecavir was found in 2022 to have a 4.9-cm segment 7 liver mass meeting radiographic criteria for HCC. Multidisciplinary review supported a diagnosis of localized HCC, and he underwent partial hepatectomy. Surprisingly, pathology revealed only benign findings - an organizing fibrosing nodule and bile duct hamartoma.Subsequent liver surveillance with semiannual MRI demonstrated no suspicious hepatic lesions through May 2026. In September 2025, an incidental cerebellar lesion was identified on spine MRI. Follow-up brain MRI demonstrated a 2.7-cm enhancing right cerebellar mass, initially suspected to represent recurrent meningioma. Following suboccipital craniotomy in January 2026, pathology revealed metastatic HCC, confirmed by positive HepPar-1 and Arginase-1 immunostaining. Postoperative PET/CT and repeat liver MRI demonstrated no intrahepatic or extrahepatic disease. **Discussion:** This case highlights two uncommon phenomena: benign hepatic lesions mimicking HCC in a cirrhotic liver and pathology-confirmed metastatic HCC occurring in the absence of an identifiable primary tumor despite four years of negative surveillance imaging. Possible explanations include an occult microscopic HCC present at the time of resection, spontaneous regression of an undetected primary lesion, or disease remaining below the threshold of radiographic detection. Rarely, cases of HCC presenting as craniospinal metastases in the absence of obvious hepatic disease have been reported. **Conclusion:** Metastatic HCC may rarely present isolated brain metastasis despite years of negative hepatic surveillance and no detectable hepatic primary, underscoring limitations of imaging-based diagnosis and surveillance in cirrhosis.

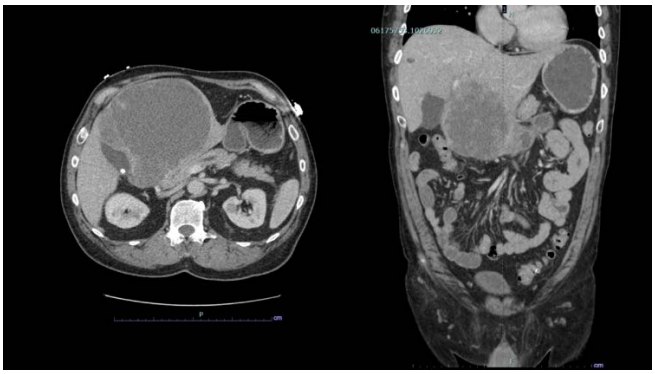
4658 | CYSTIC HEPATOCELLULAR CARCINOMA IN A NON-CIRRHOTIC PKHD1 HETEROZYGOTE WITH A DUCTAL PLATE MALFORMATION: AN UNREPORTED ASSOCIATION IN THE FIBROPOLYCYSTIC SPECTRUM

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Introduction: Hepatocellular carcinoma (HCC) typically arises in cirrhosis. Approximately ~20% occur in non-cirrhotic livers where cystic, hypovascular variants are and mimic benign cysts, biliary cystadenoma, or cystic metastasis. Malignancy within the fibropolycystic/ductal plate malformation spectrum is overwhelmingly cholangiocarcinoma, not HCC. We describe a poorly differentiated cystic HCC in a non-cirrhotic PKHD1 heterozygote with coexisting ductal plate malformation, an unreported association. **Case Presentation:** A 63-year-old man with hypertension, hyperlipidemia, and type 2 diabetes presented with 2.5 weeks of right-sided abdominal pain, nausea, vomiting, and 9.5-lb weight loss. CT showed a 16.0 x 14.0 cm cystic, lobulated mass in segments III-IV with mural soft-tissue nodules and a posteriorly displaced but patent portal vein. Viral hepatitis A/B/C serologies were negative with no history of prior liver disease. Labs showed mild normocytic anemia and preserved hepatic function (AST 37, ALT 17, total bilirubin 0.4, albumin 3.8). AFP was 9,022 ng/mL, CA 19-9 mildly elevated (49 U/mL), and CEA was normal. MRI confirmed a large hypovascular left-lobe mass (15.2 x 15.1 cm) without portal vein thrombus, biliary dilatation, or ascites. He underwent open left hepatectomy, cholecystectomy, and segment 8 wedge biopsy. Pathology showed poorly differentiated HCC (G3, 15 cm), margin-negative, with one node positive for metastatic HCC and without histologic signs of cirrhosis. The segment 8 lesion was a bile duct hamartoma (von Meyenburg complex), a ductal plate malformation within the PKHD1-associated spectrum. Germline testing identified a heterozygous PKHD1 variant. AFP fell to 79.4 ng/mL postoperatively. **Discussion:** On the delayed/equilibrium phase, the mass is large, lobulated, and predominantly cystic/hypoattenuating relative to the background liver, without an enhancing capsule. The mildly elevated CA 19-9 further suggested a biliary lesion. The markedly elevated AFP (high specific at ~400 ng/mL) was the anchoring clue and its postoperative decline supported hepatocellular origin and a complete resection. Arginase-1 focal positivity on microscopic examination also supports hepatocellular origin. PKHD1 encodes fibrocystin and heterozygous carriers, once considered unaffected, are now recognized to manifest ductal plate malformations. Malignancy in this setting is characteristically cholangiocellular, whereas HCC is seldom described. We raise the possibility that PKHD1 heterozygosity, together with metabolic risk factors, lowered the threshold for hepatocarcinogenesis. **Conclusion:** Poorly differentiated histology with nodal involvement despite R0 resection mandates close surveillance. Clinicians should maintain suspicion for HCC in non-cirrhotic patients with atypical cystic masses and integrate tumor markers with imaging to avoid diagnostic delay.

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4659 | A RARE CAUSE OF PAINLESS JAUNDICE: LEFLUNOMIDE-INDUCED VANISHING BILE DUCT SYNDROME

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Introduction: Leflunomide is a commonly used disease modifying antirheumatic drug (DMARD) for the treatment of autoimmune conditions. Although leflunomide-associated hepatotoxicity is a recognized adverse effect, clinically significant cholestatic liver injury and vanishing bile duct syndrome (VBDS) are rare complications. **Case Presentation:** 71 year old female with paroxysmal atrial fibrillation on Dabigatran and rheumatoid arthritis on Leflunomide presenting with painless jaundice, papular pruritic rash, and dark urine after transitioning from metoprolol tartrate to succinate. Admission lab work showed cholestatic versus mixed pattern of liver injury with AST 105 IU/L, ALT 113 IU/L, total bilirubin 8.6 mg/dL, direct bilirubin 7.5 mg/dL. MRCP was negative for obstructive jaundice. Hepatitis panel, anti-nuclear antibody, anti-mitochondrial antibody, cytomegalovirus, strongyloides antibody were all negative. Epstein barr virus showed prior infection. Liver biopsy showed chronic cholestasis and ductopenia most likely from drug-induced VBDS. Consensus between Radiology and Gastroenterology indicated chronic leflunomide use as the culprit. **Discussion:** Leflunomide liver injury most commonly occurs within the first months of therapy. Previously tolerated exposure however should not exclude Leflunomide when evaluating unexplained cholestatic liver injury. [1,2] VBDS is characterized by loss of interlobular bile ducts and may be accompanied by cholestasis, portal inflammation, and progressive fibrosis. Liver biopsy is valuable when cholestatic injury persists without an identifiable cause. [2-4] The mechanism underlying Leflunomide VBDS is unknown. Leflunomide is converted to its active metabolite Teriflunomide (A77 1726), which undergoes extensive enterohepatic recirculation and has a prolonged elimination half-life. Experimental studies have implicated mitochondrial dysfunction and endoplasmic reticulum stress in Leflunomide hepatotoxicity, while drug-induced VBDS is thought to involve direct or immune-mediated cholangiocyte injury leading to progressive bile duct loss. [3,5,6] Management begins with withdrawal of the offending agent. Cholestyramine can accelerate drug elimination. Whether accelerated elimination changes the course once ductopenia and established VBDS are present remains uncertain. Similarly, Ursodeoxycholic acid and corticosteroids have been used in drug-induced VBDS, but evidence that either prevents progression of bile duct loss is limited. [2,3] **Conclusion:** This case highlights an important manifestation of Leflunomide DILI, particularly when the evaluation for obstructive, infectious, and autoimmune causes is unrevealing. Liver biopsy remains central to recognizing ductopenic injury, while persistent hyperbilirubinemia after drug withdrawal should raise concern for established bile duct loss and the potential for chronic cholestatic liver disease.



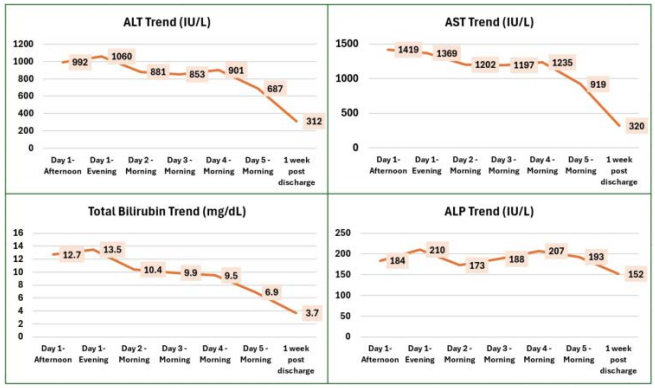
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4660 | SEVERE HEPATOCELLULAR INJURY FROM A TURMERIC-PIPERINE HAIR-GROWTH SUPPLEMENT: EXPANDING THE PHENOTYPE OF HERBAL DILI

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Introduction: Herbal and dietary supplements (HDS) are now the second most common cause of drug-induced liver injury (DILI) in the US, and turmeric (curcumin) is an increasingly implicated culprit-typically hepatocellular, latency 1-4 months, linked to HLA-B*35:01, and often co-formulated with black pepper (piperine) (Halegoua-DeMarzio et al., Am J Med, 2023). Piperine inhibits P-glycoprotein, CYP3A4, and glucuronidation, raising curcumin bioavailability up to 20-fold (Stati et al., Front Pharmacol, 2021). **Case Presentation:** A 53-year-old African American woman on metoprolol and escitalopram presented with 8 days of painless jaundice and malaise. Two to three weeks earlier she had concurrently begun three online products: a weight-loss tea, a multivitamin, and a hair-growth supplement whose main constituents were turmeric and piperine. She reported social alcohol use; a sister had cryptogenic cirrhosis. Exam showed scleral icterus without encephalopathy. Labs: ALT 992, AST 1419 U/L, total bilirubin 12.7 mg/dL, ALP 184 U/L (R 16.2, hepatocellular), INR 1.3, IgG 3060 mg/dL, SMA 1:80, ferritin 2250 ng/mL, transferrin saturation 61%, CA 19-9 229 U/mL. Hepatitis A IgG positive; HAV IgM, HCV, CMV, EBV, HSV, acetaminophen, phosphatidylethanol, alpha-1 antitrypsin, and AMA negative. Imaging showed patent vasculature without steatosis, obstruction, cirrhosis, or infiltration. Enzymes improved after cessation without corticosteroids; biopsy was deferred. She was discharged on day 5 with continued recovery. **Discussion:** The hepatocellular pattern, latency, temporal association, and rapid recovery after cessation are characteristic of turmeric-associated DILI, supported by a probable RUCAM score and exclusion of viral, ischemic, biliary, and infiltrative causes. Reactive ferritin/transferrin saturation reflect acute hepatocellular injury, not hemochromatosis. Modest SMA and IgG elevation raised autoimmune hepatitis, but remission without immunosuppression favors DILI over AIH, which relapses off treatment. Concurrent products preclude isolating the supplement-a recognized limitation of HDS causality assessment. **Conclusion:** This case adds a hair-growth product to the widening range of turmeric-piperine supplements causing hepatocellular DILI and underscores meticulous supplement history-taking.



4661 | FATAL HEPATITIS-ASSOCIATED APLASTIC ANEMIA AFTER SUPPLEMENT-INDUCED DILI

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Introduction: Hepatitis-associated aplastic anemia (HAAA) is a rare variant of aplastic anemia developing with, or within 6 months of, acute hepatitis. It mainly affects young males and is usually linked to seronegative hepatitis, though rare cases follow drug-induced liver injury (DILI). Immune cross-reactivity between liver and marrow is proposed. We report a rare HAAA case following severe liver injury in a 66-year-old woman with long-term herbal/supplement use. **Case Presentation:** A 66-year-old woman presented with one week of jaundice and pruritus. Labs showed severe hepatocellular injury: AST 2,114, ALT 2,940, alkaline phosphatase 298 U/L, bilirubin 28.1 mg/dL, R factor 29, normal CBC, INR. Autoimmune/viral workup and imaging, including MRCP, were unrevealing.She reported long-term use of multiple supplements: collagen powder, an immune-support supplement with serum-derived IgG concentrate, a biotoxin blend (molybdenum, yucca, humic shale), beet juice powder, green tea extract (GTE), and schisandra fruit extract. Liver biopsy showed severe lobular-predominant acute hepatitis- pattern of injury (Figure 1) characterized by acidophil bodies, lobular disarray, hepatocellular swelling with patchy perivenular cholestasis and 5-10% parenchymal collapse, consistent with severe

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DILI.Over the next month, the liver injury resolved with supportive treatment, but she developed profound pancytopenia. Bone marrow biopsy revealed markedly hypocellular marrow (<5% cellularity), without evidence of hematologic malignancy, consistent with HAAA secondary to DILI. She was administered anti-thymocyte globulin, cyclosporine, and prednisone; the clinical course was complicated by recurrent bacteremia requiring prolonged antibiotics. While being evaluated for bone marrow transplant, she deteriorated and died of multiorgan failure. **Discussion:** This case highlights the diagnostic challenge of HAAA following supplement-associated DILI. Years of multi-supplement use made it hard to identify the offending agent, and products were proprietary blends with undisclosed components, though ingredients like GTE are recognized causes of DILI. Epigallocatechin-3-O-gallate (EGCG), the predominant GTE catechin, induces a CD4+ T-cell response via MHC class II presentation, possibly explaining the elevated CD4:CD8 ratio on bone marrow biopsy, contrasting with the low/inverted ratio typical of immune-mediated injury. This may reflect early EGCG-driven clonal expansion, though significant marrow hypoplasia limits our assessment. Her bacteremia and advanced age likely contributed to the poor outcome, consistent with the literature. **Conclusion:** In the US, older women have high rates of herbal/supplement use and related adverse events. This case shows the need to document supplement use, counsel against non-FDA-approved products, and monitor blood counts after DILI.

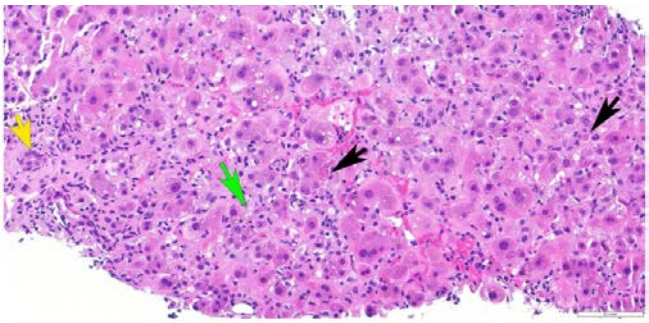


Figure 1: Lobular-predominant acute hepatitis pattern of injury with numerous foci of lobular inflammatory activity, including acidophil bodies (black arrows). There is lobular disarray along with hepatocellular cytoplasmic swelling and foci of cholestasis (green arrow). Cholestasis was noted in the perivenular region. Patchy interlobular bile duct injury was also observed (yellow arrow).

4662 | NOT THE LYMPHOMA: TRIMETHOPRIM-SULFAMETHOXAZOLE-INDUCED LIVER INJURY MASQUERADING AS A BARRIER TO CHEMOTHERAPY IN NEW DLBCL

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Introduction: Drug-induced liver injury (DILI) is a diagnosis of exclusion and a leading cause of unexplained transaminitis. Incidental liver enzyme elevation discovered during pre-chemotherapy evaluation is clinically important, because the underlying cause determines whether cancer treatment can proceed safely and on schedule. We report a case of trimethoprim-sulfamethoxazole (TMP-SMX)-induced hepatocellular injury identified before initiation of chemotherapy for diffuse large B-cell lymphoma (DLBCL). **Case Presentation:** An 88-year-old woman with Sjogren's syndrome, rheumatoid arthritis, and vitiligo was admitted with nasal congestion and dyspnea and found to have DLBCL of the left nasal/maxillary sinus with intracranial extension. Pre-chemotherapy labs revealed new hepatocellular injury (peak AST 1,228 U/L, ALT 768 U/L; normal alkaline phosphatase and bilirubin; R-value ~30). AST/ALT had been normal weeks earlier. She had started TMP-SMX on 6/12 for post-cataract pre-septal cellulitis, giving a latency of ~11 days. She denied alcohol, herbals, supplements, and acetaminophen. Hepatitis A/B/C serologies were negative; ANA was 1:640 and IgG 1,734 mg/dL. Ultrasound showed a normal liver with normal portal flow. After TMP-SMX was stopped, transaminases normalized by 7/15. **Discussion:** The hepatocellular pattern, temporal link to a single new drug, and resolution after withdrawal supported idiosyncratic TMP-SMX DILI with RUCAM (9, highly probable). Autoimmune hepatitis was considered given her autoimmune history, but she had no prior transaminase elevation and was on high-dose prednisone, which would suppress a flare. Lymphomatous hepatic infiltration and biliary obstruction were excluded by normal alkaline phosphatase, bilirubin, and imaging. Viral hepatitis was serologically excluded, and stable hemodynamics argued against ischemic injury. With no synthetic dysfunction and improving enzymes, N-acetylcysteine, steroid escalation, and biopsy were deferred. **Conclusion:** Very high transaminases in a patient awaiting chemotherapy should never be attributed to a single cause without a broad, systematic workup, because treatment-altering diagnoses (malignant infiltration, biliary obstruction, viral or autoimmune hepatitis) must first be excluded. Here, that structured approach (medication history, injury-pattern recognition, targeted serologies and imaging, and RUCAM-supported causality) confidently identified TMP-SMX DILI. Drug withdrawal alone produced full resolution, avoiding biopsy or hepatotoxic therapy and preventing any delay of lymphoma treatment.

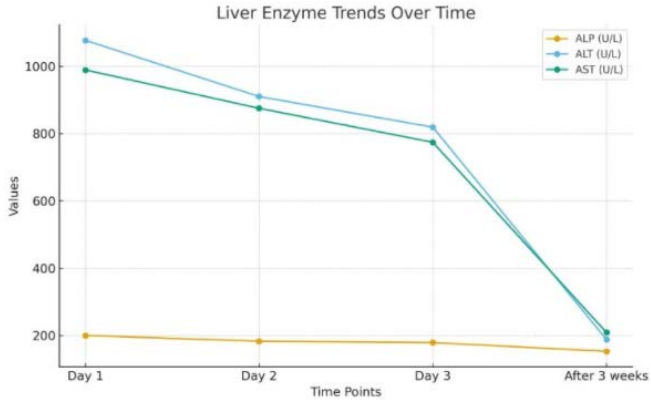
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4663 | A RARE CASE OF RAPID ONSET HEPATOCELLULAR INJURY BY SHORT-TERM FLUCONAZOLE THERAPY

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Introduction: Drug induced liver injury (DILI) is a complex diagnosis that requires ruling out multiple alternative causes. Fluconazole, a commonly used antifungal, is generally safe but can rarely cause acute hepatocellular injury. We present a rare case of rapid onset acute hepatocellular injury, following short-term fluconazole therapy. This case highlights the need for clinical vigilance even after minimal exposure. **Case Presentation:** - A 55-year-old female presented with one week of jaundice along with dark-colored urine. She denied abdominal pain,alcohol-use, or prior liver diseases. On further evaluation of her medical history, it was found that she had taken two doses of fluconazole 150 mg for a fungal infection in addition to her oral estrogen therapy for two days. - Investigations showed ALT 1077 U/L, AST 989 U/L, ALP200 U/L, total bilirubin 19.6 mg/dL, and a direct bilirubin of12.2 mg/dL. Workup for viral, autoimmune, and metabolic causes of liver disease was negative, and both ultrasound and MRCP revealed no significant abnormalities. The R-ratio was 16.1, consistent with a hepatocellular pattern of injury,with a RUCAM score of 9 indicating a probable relationship to fluconazole. - Estrogen-induced liver injury was considered less likely given its typical cholestatic presentation and short exposureduration. The patient was managed with supportive care, IV fluids and continuous outpatient follow-up. Her jaundice resolved and liver enzymes progressively normalized after discontinuation of fluconazole. **Discussion:** - Fluconazole-induced hepatotoxicity is extremely rare (<1 in10,000 exposures). The mechanism is idiosyncratic and not dose-dependent. - Most cases occur after prolonged therapy, but this case emphasizes that even brief exposure can trigger severehepatocellular injury. Prompt discontinuation and supportive management are crucial for recover. - Estrogen is known to induce a cholestatic pattern of liver injury, rather than a hepatocellular injury. In this case, the pattern of liver function test abnormalities is consistent with fluconazole as the causative agent. **Conclusion:** - Clinicians should maintain awareness of potential fluconazole-induced hepatotoxicity, even with short-term use. - Early recognition and prompt discontinuation are vital toprevent progression to severe hepatic dysfunction.



4664 | ACUTE LIVER INJURY FOLLOWING COMBINED IMMUNE CHECKPOINT INHIBITOR AND CHEMOTHERAPY IN A PATIENT WITH UNTREATED HEPATITS C: A DIAGNOSTIC AND MANAGEMENT CHALLENGE

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Introduction: Immune checkpoint inhibitors (ICIs) have transformed treatment of non-small cell lung cancer (NSCLC), but hepatotoxicity is a recognized complication. Patients with chronic hepatitis C virus (HCV) were largely excluded from pivotal ICI trials, leaving limited data regarding safety in this population. We present severe liver injury in a patient with untreated, high-burden HCV receiving combination ICI and chemotherapy. **Case Presentation:** A 73-year-old man with metastatic lung adenocarcinoma, untreated chronic HCV (HCV RNA >100 million IU/mL), prior CVA, and diabetes presented with abdominal and chest pain. Seven weeks after receiving nivolumab, ipilimumab, pemetrexed, and carboplatin, laboratory evaluation showed mixed hepatocellular-cholestatic liver injury (ALT 463 U/L, AST 380 U/L, ALP 359 U/L, bilirubin 16.3 mg/dL; R factor 3.9). Aminotransferases had been elevated for approximately six weeks. INR was 1.2 and ammonia was normal. He denied alcohol and acetaminophen use. CT and ultrasound showed no biliary obstruction or hepatic metastases. He subsequently developed refractory hypotension requiring norepinephrine and ICU transfer. The differential included ICI-mediated hepatitis, HCV-related injury, chemotherapy-related DILI, and metastatic infiltration. Viral studies were obtained, and liver biopsy was recommended after stabilization to distinguish etiologies. **Discussion:** The mixed liver injury pattern and untreated high-burden HCV complicated attribution of hepatotoxicity. The temporal relationship to ICI therapy

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raised concern for immune-mediated hepatitis, while active HCV and chemotherapy represented competing etiologies. Combination nivolumab/ipilimumab regimens carry a reported grade > =3 hepatotoxicity rate of 8-18.8%, while emerging real-world data suggest higher rates of hepatic failure among HCV-positive patients receiving ICIs.1,2,4 The mixed pattern (R factor 3.9) is also atypical for classic ICI hepatitis and may reflect cholangitic or alternative drug-induced injury. Liver biopsy was recommended to distinguish these etiologies, as treatment differs substantially. Corticosteroids, the mainstay of ICI hepatitis, may increase HCV replication, making accurate diagnosis critical before immunosuppression. This diagnostic distinction is particularly important when liver injury occurs soon after ICI initiation in patients with untreated viral hepatitis. **Conclusion:** This case highlights the importance of assessing HCV status before ICI therapy and considering antiviral treatment when appropriate. In patients with active HCV receiving ICIs, careful evaluation of competing causes of liver injury is essential before initiating immunosuppression.

4665 | OXALIPLATIN-INDUCED SINUSOIDAL INJURY WITH CONCOMITANT GILBERT SYNDROME: A DIAGNOSTIC CHALLENGE

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Introduction: Oxaliplatin-associated sinusoidal obstruction syndrome and nodular regenerative hyperplasia are well-established hepatotoxic side-effects. Severe cholestatic liver injury with hepatosplenomegaly and fibrosis is uncommon. Concurrent causes of hyperbilirubinemia may further obscure clinical interpretation of the underlying hepatic injury. We present a case of severe oxaliplatin-induced liver injury with concomitant Gilbert syndrome. **Case Presentation:** A 31-year-old male with a history of sigmoid intramucosal adenocarcinoma underwent five cycles of neoadjuvant FOLFIRINOX. Cycle six was held due to new cholestatic liver injury with alkaline phosphatase 627 U/L, total bilirubin 4.1 mg/dL, and direct bilirubin 0.7 mg/dL with normal aminotransferases. Magnetic resonance imaging revealed new hepatosplenomegaly compared to prior imaging two weeks prior, raising concern for an acute intrahepatic process. Trans-jugular liver biopsy demonstrated a hepatic venous pressure gradient of 2 mmHg and sinusoidal dilation predominantly around central veins without significant steatosis. Trichrome staining revealed periportal and perivenular stage 1 fibrosis. Given the temporal relationship to oxaliplatin exposure and sinusoidal changes, oxaliplatin-associated sinusoidal injury was suspected. After two months his liver enzymes normalized, and his chemotherapy regimen was changed to CAPEOX. Unfortunately, he develop recurrent indirect hyperbilirubinemia to 5.3 mg/dL soon after initiation, feared to be due to Capecitabine-related hemolysis. However, UGT1A1 genotyping was conducted and confirmed the diagnosis of Gilbert syndrome, explaining his indirect hyperbilirubinemia. The benefit of resuming chemotherapy with close monitoring was thought to outweigh the risk of recurrent injury, and he completed six additional cycles without complication. **Discussion:** This case reveals the potential for oxaliplatin-induced sinusoidal injury to present with cholestatic liver injury, hepatosplenomegaly, and fibrosis. This case also highlights the importance of excluding alternative etiologies when determining whether a potentially hepatotoxic but necessary drug can be safely resumed. **Conclusion:** Severe cholestatic liver injury may represent a clinically important manifestation of oxaliplatin injury. Gilbert syndrome may coexist with chemotherapy-related liver injury and should be considered when indirect hyperbilirubinemia complicates assessment of treatment-related hepatotoxicity. Distinguishing inherited bilirubin metabolism abnormalities from chemotherapy-associated hepatic injury is critical in determining when chemotherapy can be safely resumed.

4666 | HNF1B MUTATION AS A NOVEL GENETIC RISK FACTOR FOR SEVERE INTRAHEPATIC CHOLESTASIS OF PREGNANCY

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Introduction: The hepatocyte nuclear factor 1β (HNF1B) mutation is a pathogenic mutation associated with abnormal embryonic development of epithelia, predominantly affecting the kidneys and pancreas with significant phenotypic variation. A few studies have reported liver disease manifestations associated with HNF1B mutations, often presenting as chronic elevation of transaminase levels or cholestatic disease due to abnormal bile duct development. However, association with intrahepatic cholestasis of pregnancy (ICP) has not been reported1. **Case Presentation:** A 23-year old G1P0 female presented with elevated bile acids (28.3 umol/L) and pruritus at 13 weeks gestation.

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The patient was known to be heterozygous in the HNF1B gene for sequence variant c.913A>G, which results in the amino acid substitution p.Lys305Glu. At 8 weeks of life, she was jaundiced and had severe intrahepatic cholestasis secondary to bile duct paucity found on liver biopsy, a reported neonatal manifestation of HNF1B-related disease2. She has not had further hepatobiliary complications since her infancy, but developed progressive chronic kidney disease, maturity-onset diabetes of the young (MODY), and panhypopituitarism, ns3. Updated genetic testing revealed no other known genetic risk factors for ICP. At diagnosis, the patient was prescribed 900mg ursodiol (UDCA) daily. Her bile acids and liver tests (LTs) initially continued to rise, with bile acids peaking at 52.6 umol/L, but then declined (Figure 1). At 22 weeks, her dose was titrated to 1200 mg daily; her bile acids stabilized between 13.5-22.6 umol/L and pruritus improved. At 36 weeks 5 days, labor was induced due to worsening kidney function. At this time, her bile acids were 31.4 umol/L, and LTs were normal. She gave birth to a healthy neonate, bile acids dropped to within the normal range (5 umol/L) immediately after delivery, and UDCA was discontinued. One week postpartum, however, her bile acids worsened (26.6 umol/L) and ALT and AST were mildly elevated to 54 IU/L and 48 IU/L, respectively. Bile acids continued to rise, three weeks later peaking at 56.5 umol/L, ALT and AST rose to 76 IU/L and 57 IU/L, respectively, and she developed severe pruritus. UDCA was re-initiated at 900 mg daily to good response. MRCP imaging showed significant bile duct abnormalities consistent with HNF1B. **Conclusion:** Although currently classified as a variant of unknown significance to ICP, the HNF1B mutation may be a risk factor for atypical and severe ICP, in this case appearing early in pregnancy and persisting after delivery. Counseling of this risk should be provided to women of reproductive age with HNF1B mutations. Furthermore, consideration for continued use of UDCA post-delivery, rather than discontinuing at time of delivery, should be made for women with HNF1B.

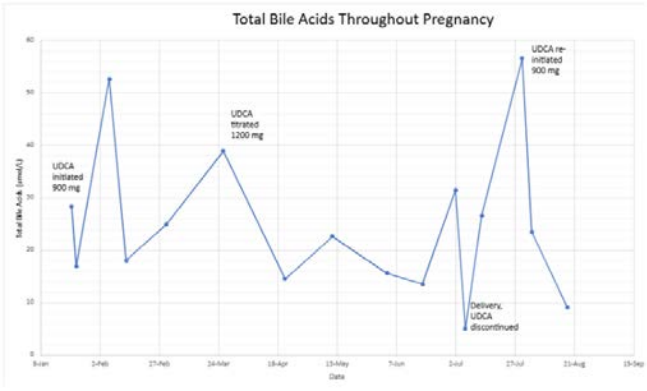


Figure 1. Total bile acids throughout pregnancy and postpartum, annotated with changes in UDCA total daily dose and time of delivery.

4667 | ADULT-ONSET PFIC PRESENTING AS RECURRENT POSTPARTUM CHOLESTASIS

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Introduction: Progressive familial intrahepatic cholestasis (PFIC) is a rare inherited disorder of bile transport classically presenting in childhood. Partial-loss-of-function variants may cause milder adolescent or adult-onset disease. We present an atypical adult presentation following pregnancy. **Case Presentation:** 19-year-old woman, eight months postpartum, developed jaundice, dark urine, and severe pruritus after cholecystectomy for symptomatic cholelithiasis. ERCP demonstrated biliary sludge/debris; repeat ERCP/EUS showed no obstruction but nonspecific pancreatic changes, leading to empiric steroids for suspected autoimmune pancreatitis. Symptoms and cholestasis persisted. Total bilirubin peaked at 4.5 mg/dL (direct 3.3), alkaline phosphatase at 220 U/L, and AST/ALT initially at 985/1161 U/L before improving. MRI/MRCP showed no biliary dilation, and Doppler ultrasound excluded vascular thrombosis. Infectious, autoimmune, and metabolic evaluation was unrevealing. Repeat biopsy demonstrated bland canalicular cholestasis with mild lobular inflammation and no fibrosis or obstruction. Findings raised concern for PFIC; genetic testing was initiated. She received ursodiol, cholestyramine, antihistamines, and gabapentin for pruritus; maralixibat was pursued. **Discussion:** PFIC can present beyond childhood, particularly with partial-loss-of-function variants. ABCB4-related PFIC3 is typically associated with elevated GGT and may manifest as intrahepatic cholestasis of pregnancy or adult-onset disease. This case demonstrates how inherited cholestatic disease may be overlooked amid competing explanations such as gallstone disease or nonspecific pancreatic findings. Persistent cholestasis despite exclusion of obstruction and acquired etiologies should prompt consideration of genetic cholestatic disorders, even without a known family history. Recognition is important because genotype-informed therapies, including ursodeoxycholic acid and ileal bile acid transporter inhibitors, may improve symptoms. **Conclusion:** Adult-onset PFIC should be considered in unexplained cholestasis and severe pruritus. This case highlights the role of liver biopsy and genetic testing when conventional evaluation is unrevealing and underscores expanding therapeutic options for inherited cholestatic disease.

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4668 | SPONTANEOUS BACTERIAL PERITONITIS SECONDARY TO LISTERIA MONOCYTOGENES IN A POST-LIVER TRANSPLANT PATIENT

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[1]University of Miami

Introduction: Spontaneous bacterial peritonitis (SBP) is a common complication of advanced hepatic cirrhosis. Listeria monocytogenes, a gram-positive bacillus, is an established cause of SBP in immunosuppressed patients; however, it has rarely been reported in post-liver transplant patients. Given the high mortality associated with delayed/absent treatment for Listeria SBP, early recognition is crucial. **Case Presentation:** A 69 year old female with primary biliary cholangitis and prior liver transplantation (2007) presented with a 2-week onset of abdominal pain, distension, and watery diarrhea. Her abdomen was diffusely tender and distended with shifting dullness without guarding. Labs were significant for hyponatremia (Na 125 mmol/L), acute kidney injury (Cr 1.9 mg/dL, baseline 0.8 mg/dL), hypoalbuminemia (2.6 g/dL), thrombocytopenia (126 × 10^9/L), anemia (Hgb 9.1 g/dL), and elevated C-reactive protein (24.5 mg/L). CT with contrast imaging demonstrated cirrhotic liver morphology, large-volume ascites, portal hypertension, and splenomegaly. Initial paracentesis fluid studies revealed WBC 4815, PMN 58% consistent with SBP. Empiric IV ceftriaxone was initiated. Three paracenteses were performed after antibiotic initiation. A repeat paracentesis on hospital day 3 yielded an ascitic fluid culture positive for Listeria monocytogenes, with WBC 7337, PMN 67%. Therapy was changed to IV ampicillin/gentamicin, with clinical improvement and negative repeat cultures. She was transitioned to a 3-week course of oral amoxicillin and discharged on trimethoprim-sulfamethoxazole secondary prophylaxis. **Discussion:** Listeria is a rare, atypical cause of SBP in liver transplant patients. Ascitic fluid cultures are often negative due to poor sensitivity; diagnosis is based on ascitic fluid PMN count >250/mm3. Adequate ascitic fluid diagnostic yields (10 mL) increase culture sensitivity. SBP infections are typically enteric, monobacterial, and gram-negative (E. coli and K. pneumoniae). Only three prior cases of Listeria SBP have been reported. Our patient's history increases her risk for Listeria infection, with multiple paracenteses/cultures being required to obtain a positive result. Third-generation cephalosporins, the standard empiric therapy for SBP, lack activity against Listeria. Delayed/missed diagnosis results in serious consequences. **Conclusion:** Listeria monocytogenes is a rare but potentially fatal cause of SBP in post-liver transplant patients. Standard third-generation cephalosporin empiric regimens do not provide adequate coverage, requiring repeat paracenteses to elicit a positive culture, and delaying treatment. Early transition to ampicillin-based therapy and timely antibiotic tailoring improves patient survival. Listeria should be suspected when clinical deterioration continues despite appropriate therapy, and clinicians should maintain a low threshold for broadening coverage in refractory SBP.

Reference	Year	Demographic	Comorbidities	Presentation	Ascitic fluid (AF) WBC	# of AF Cultures needed before diagnosis (dx)	Dx Method	Empiric Treatment (Tx)	Narrowed Tx	Outcome
Current Manuscript	2025	69F	Liver transplant, cirrhosis, PBC	Abdominal pain, distention, watery diarrhea	1732 (60% PMN)	3	Ascitic fluid culture (cx)	IV Ceftriaxone	IV Ampicillin + gentamicin then PO amoxicillin	survived
Jayaraj et. al.	1998	52F	Liver transplant, hepatitis C related cirrhosis, 1 prior episode of SBP	Anorexia, fever, abdominal pain, diarrhea	1180	2	Ascitic fluid cx	Cefotaxime 2g IV Q8H	ampicillin/sulbactam 2g IV Q4H and gentamicin 3 week duration	survived
Gaspar et. al.	2017	51M	Kidney transplant, heart failure with severe impaired left ventricular function, chronic liver disease due to congestive hepatopathy	abdominal distention, pain	11390 (81% PMN)	2	Ascitic fluid cx	Ceftriaxone 2g QD	Ampicillin 2g/day	survived

4669 | NORMAL HVPG, ABNORMAL OUTFLOW: A CASE OF PIGGYBACK CAVAL STENOSIS

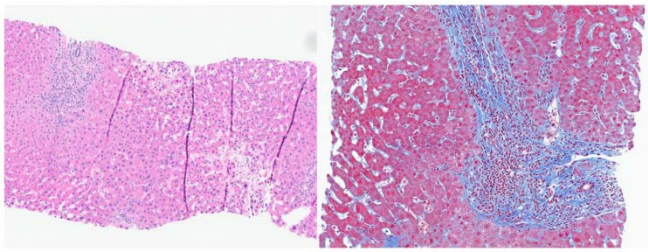
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Introduction: Hepatic venous outflow obstruction (HVOO) is an uncommon but important complication of piggyback liver transplantation. When obstruction is distal to the hepatic veins, wedged and free hepatic venous pressures may rise together, yielding a deceptively normal hepatic venous pressure gradient (HVPG). **Case Presentation:** A 32-year-old male underwent donation-after-circulatory-death liver transplantation for alcohol-associated cirrhosis using a piggyback caval anastomosis. Day 6 Doppler showed portalized waveforms in all hepatic veins and a right pleural effusion, attributed to graft edema. Five months later, he developed large-volume ascites and a right hydrothorax requiring daily chest tube drainage. Ascitic protein was 3.7 g/dL with a serum-ascites albumin gradient of 1.3 g/dL, a high-protein, high-gradient pattern characteristic of post-sinusoidal congestion rather than portal hypertension from graft disease. AST was 29 U/L, ALT 11 U/L, and bilirubin 0.8 mg/dL. Biopsy showed moderate centrilobular sinusoidal dilatation and congestion, hepatocyte atrophy with focal dropout, no rejection, and Ishak stage 2 portal fibrosis. HVPG was 5 mmHg, but wedged and free hepatic vein pressures were both elevated at 28 and 23 mmHg; right atrial pressure was 3 mmHg. CT venography demonstrated marked IVC narrowing at the piggyback anas-

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tomosis. Direct measurements confirmed 12- and 14-mmHg gradients from the hepatic veins to the IVC. Dilation with a 12-mm balloon achieved expansion of the stenotic segment and reduced wedge pressures to 20 mmHg. Ascites and hydrothorax improved, permitting chest tube removal. He was discharged on day 12 and had lost 7.8 kg by follow-up on day 16, without dyspnea or clinically evident ascites. **Discussion:** A normal HVPG does not exclude post-transplant HVOO because downstream caval obstruction can elevate wedged and free pressures in parallel. When Doppler abnormalities, high-protein ascites, or biopsy suggest congestion, direct hepatic vein-to-IVC pressure gradients should be obtained. Portalized hepatic venous waveforms and early pleural effusion were likely the initial clues in this case. Ishak stage 2 fibrosis within five months raises concern that chronic unrecognized congestion contributed to early allograft injury. Persistent or recurrent narrowing after angioplasty warrants close Doppler surveillance and consideration of stenting. **Conclusion:** Piggyback caval stenosis can present with severe venous congestion and early allograft fibrosis despite normal liver biochemistry and a normal HVPG. In transplant recipients with new ascites or hydrothorax, direct hepatic vein-to-IVC pressure measurement can establish the diagnosis and guide timely endovascular treatment, potentially limiting further allograft injury.



4670 | A CASE SERIES HIGHLIGHTING A RARE TRIAD OF COMPLICATIONS IN MALNOURISHED PATIENTS FOLLOWING GASTRIC BYPASS

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Introduction: Bariatric surgery is an effective treatment for severe obesity, but it carries the risk of serious nutritional complications. Hepatic steatosis, severe protein malnutrition with hypoalbuminemia and anasarca, and exocrine pancreatic insufficiency (EPI) are individually recognized complications of gastric bypass surgery. Here we present three cases illustrating this clinical triad that has not been well characterized previously. **Case Presentation:** Patient 1 was a 40-year-old male (BMI 18.54 kg/m²) with a history of Roux-en-Y gastric bypass who presented with ascites and edema. Laboratory evaluation revealed severe hypoalbuminemia, pan-fat-soluble vitamin deficiencies, and low fecal elastase, confirming EPI as shown in Table 1. Liver biopsy demonstrated hepatosteatosis with stage II fibrosis. Management with medium-chain triglyceride (MCT)-based enteral feeds via nasojejunal tube led to improvement. Patient 2 was a 50-year-old female (BMI 29.00 kg/m²) with a history of duodenal switch and non-adherence to vitamin supplementation. She presented with altered mental status and volume overload and was found to be in acute liver injury. She had biopsy-proven hepatic steatosis in the setting of micronutrient deficiencies. This progressed to acute liver injury following ingestion of an herbal supplement. EPI was suspected based on imaging and a pattern of fat-soluble vitamin deficiency as shown in Table 1. The patient was not a transplant candidate and died secondary to complications of liver failure. Patient 3 was a 44-year-old female (BMI 22.68 kg/m²) with a history of gastric bypass and a history of pancreatic insufficiency who was on outpatient pancreatic enzyme replacement therapy and multivitamin supplementation. She was found obtunded at home from septic shock secondary to acute on chronic pancreatitis, ultimately dying from cardiac arrest. Table 1 highlights hypoalbuminemia, low fecal elastase, and hepatic steatosis identified on ultrasound. **Discussion:** This case series identifies a significant triad of hepatic steatosis, hypoalbuminemia with anasarca, and exocrine pancreatic insufficiency in malnourished post-gastric bypass patients. The pathophysiology is likely synergistic: EPI drives fat malabsorption and vitamin deficiency, which exacerbates hepatic steatosis; protein malnutrition impairs hepatic synthetic function, causing hypoalbuminemia. Two of the three patients died, highlighting the severity of this triad and the importance of early nutrient supplementation to prevent these complications. **Conclusion:** Early recognition and aggressive nutritional management of hepatic steatosis, protein malnutrition, and EPI after bariatric surgery may prevent progression to life-threatening complications.

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Table 1. Patient laboratory results at or prior to hospital admission

Laboratory Test	Patient 1	Patient 2	Patient 3	Reference Range
Albumin	1.6 gm/dL	1.0 gm/dL	1.9 gm/dL	3.4 - 5.0 gm/dL
Prealbumin	8 mg/dL	Not Collected	Not collected	18.0 - 38.0 mg/dL
Total Protein	4 gm/dL	2.0 gm/dL	5.5 gm/dL	6.0-8.0 gm/dL
AST	25 u/L	5779 u/L	104 u/L	10.0 – 40.0 u/L
ALT	29 u/L	988 u/L	25 u/L	7.0 - 50.0 u/L
ALP	82 u/L	198 u/L	115 u/L	40.0 – 125.0 u/L
Total Bilirubin	0.3 mg/dL	7.9 mg/dL	4.0 mg/dL	0.0-1.2 mg/dL
Direct Bilirubin	0.1 mg/dL	5.3 mg/dL	3.0 mg/dL	<= 0.2 mg/dL
INR	1.8	7.8	2.7	0.8 - 1.2
Vitamin A (retinol)	0.08 g/L	<0.06 g/L	<0.06 g/L	0.3 – 1.2 g/L
Thiamine	<1 ng/mL	83 ng/mL	<1 ng/mL	4.0 – 15.0 ng/mL
Vitamin B9	3.9 ng/mL	3.5 ng/mL	12.9 ng/mL	> 5.8 ng/mL
Vitamin B12	773.0 pg/mL	>2000 pg/mL	>2000 pg/mL	181.0 – 194.0 pg/mL
Vitamin E - Alpha-Tocopherol	9.0 mg/L	1.3 mg/L	2.0 mg/L	5.5 – 18.0 mg/L
Vitamin K1	1.02 nmol/L	0.58 nmol/L	0.30 nmol/L	0.22 – 4.88 nmol/L
Copper	29.7 ug/dL	44.6 ug/dL	32.0 ug/dL	70.0 - 140.0 ug/dL
Zinc	40.6 ug/dL	34.6 ug/dL	20.1 ug/dL	60.0 - 120.0 ug/dL
Ionized calcium	4.1 mg/dL	3.6 mg/dL	4.7 mg/dL	4.2 - 5.4 mg/dL
Pseudocholinesterase	900 u/L	Not collected	Not collected	2900- 7100 u/L
Fecal elastase	< 10 ug/g	Not collected	< 10 ug/g	> 100.0 ug/g
Transferrin	< 75 mcg/dL	< 75 mcg/dL	< 75 mcg/dL	200.0 – 400.0 mcg/dL
Lipase	4.0 u/L	118 u/L	<4 u/L	11.0 - 82.00 u/L
Iron	67.0 mcg/dL	27 mcg/dL	56 mcg/dL	50.0 – 180.0 mcg/dL
Ferritin	778 ng/mL	332 ng/mL	454 ng/mL	10 –259 ng/mL
Ceruloplasmin	29 mg/dL	11 mg/dL	10 mg/dL	17.0 - 54.0 mg/dL
Selenium	62.7 ug/L	30.1 ug/L	58.3 ug/L	23.0 - 190.0 ug/L
Pathology from liver biopsy	Transjugular liver biopsy showed steatotic liver disease. Mild bile ductular reaction, and incomplete septal fibrosis compatible with fibrosis stage II of IV.	Transjugular liver biopsy showed severe steatosis (80%) with F2-3 fibrosis.	No history of liver biopsy.	
Imaging	Liver ultrasound showed heterogenous echogenic coarse hepatic parenchyma.	Magnetic Resonance Cholangiopancreatography showed fatty atrophy of pancreas with no ductal dilatation.	Liver ultrasound showed diffuse hepatic steatosis.	

4671 | DELAYED RECURRENT CHYLOUS ASCITES FOLLOWING RADICAL NEPHRECTOMY FOR RENAL CELL CARCINOMA

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Introduction: Chylous ascites is a rare cause of abdominal ascites, with a historical incidence as low as 1 in 20,000 cases1. It may occur as a postoperative complication due to disruption of the lymphatic vasculature during intra-abdominal surgery, including nephrectomy, though reported postoperative rates remain low (up to 5.1%2). **Case Presentation:** A 75-year-old male without prior cirrhosis underwent radical open left nephrectomy for pT3aN0 grade 3 renal cell carcinoma. The operative intervention was uncomplicated. He was discharged on postoperative day 2 and initiated on adjuvant pembrolizumab. No complications were noted at follow-up. At 9 weeks postoperatively, he developed progressive abdominal distention, bilateral lower extremity edema, and weight gain. Computed tomography of the chest incidentally revealed large-volume ascites. He underwent a diagnostic and therapeutic paracentesis with five liters of turbid, gray-white fluid removed. Ascitic fluid analysis demonstrated triglycerides of 1,555 mg/dL, confirming chylous ascites. No spontaneous bacterial peritonitis was identified. Doppler ultrasound did not show hepatic or portal vein thrombosis. He was asymptomatic at discharge after a brief hospitalization. He developed rapid fluid re-accumulation after four days requiring repeat paracentesis and removal of seven liters of cloudy, white fluid confirmed again as chylous ascites. Given rapid accumulation of chylous ascites, he was readmitted for treatment with octreotide and total parenteral nutrition (TPN). Lymphangiogram showed a leak on the left lateral margin of L3, and he was treated with glue embolization. **Discussion:** Chylous ascites remains an uncommon etiology of ascites but should be considered in post-operative patients with recent abdominal interventions. Alternative differentials include malignancy, cirrhosis, and tuberculosis. This case highlights a delayed presentation of postoperative chylous ascites with rapid recurrence after initial paracentesis, emphasizing clinical suspicion and early diagnostic testing in postoperative patients presenting with new ascites. Diagnosis is confirmed by an elevated ascitic triglyceride level (>200 mg/dL). Management includes dietary modification with medium-chain triglyceride supplementation, periodic therapeutic paracentesis3, and consideration of TPN. If symptoms persist, next steps may include lymphangiography with interventional radiology to evaluate and intervene upon the lymphatic leak. **Conclusion:** When evaluating and managing postoperative chylous ascites, optimal outcomes depend on close coordination between internal medicine, gastroenterology, and interventional radiology, from the time of initial diagnosis through definitive intervention.References: - Press OW et al, Ann Intern Med 1982;96:358-64. - Kim BS et al, J Urol 2010;184:570-4. - Lizaola B et al, Aliment Pharmacol Ther 2017;46:816-24.

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Figure 1. Syringe from diagnostic paracentesis demonstrating turbid, milky-white ascitic fluid.



Figure 2. Lymphangiogram showed a leak along the left lateral margin of the L3 vertebral body.

4672 | PORTOSYSTEMIC ENCEPHALOPATHY UNMASKING RECURRENT METASTATIC RENAL CELL CARCINOMA WITH ASSOCIATED PORTAL VEIN TUMOR THROMBUS

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Introduction: Non-cirrhotic portosystemic encephalopathy (PSE) caused by portal vein tumor thrombus (PVTT) is a recognized but rare complication of hepatocellular carcinoma and has rarely been reported in non-hepatocellular malignancies including colon, gastric, and pancreatic cancers. To our knowledge, its occurrence in renal cell carcinoma (RCC) has never been described. We present the first documented case of PSE precipitated by PVTT as the presentation of recurrent metastatic RCC. **Case Presentation:** A 68-year-old male with non-clear cell RCC was diagnosed in 2024 after presentation with extensive deep vein thromboses and a malignant inferior vena cava tumor thrombus. He underwent radical nephrectomy and thrombectomy and more recently presented with subacute progressive cognitive decline over several months, characterized by intermittent confusion, disorientation, and decreased responsiveness. On admission, workup for altered mental status revealed an elevated ammonia of 151 with normal liver chemistries and no history of liver disease. MRI of the brain demonstrated punctate acute and subacute infarcts of the left cerebellum and right frontal lobe but these findings were insufficient to explain his intermittent confusion and disorientation, more consistent with encephalopathy versus focal deficits. Infectious, toxic, and autoimmune workups were negative, and EEG showed no seizure activity. Abdominal MRI identified a new occlusive thrombus in the main and right portal vein extending into the left portal vein with cavernous transformation, concerning for tumor thrombus. Biopsy of the distal portal vein confirmed recurrent metastatic RCC, with tumor demonstrating similar morphology to prior resected kidney tumor. The patient's encephalopathy resolved with lactulose and rifaximin, supporting the diagnosis of PSE. The underlying mechanism is attributed to malignant portal vein tumor thrombosis causing portal hypertension, development of collateral vascularization and portosystemic shunting, resulting in impaired hepatic ammonia clearance despite preserved intrinsic liver function. The patient has completed radiation therapy to the portal vein and is now pending systemic treatment with pembrolizumab and lenvatinib for progressive disease on his most recent PET scan. **Discussion:** This is the first case attributing non-cirrhotic PSE to PVTT from metastatic RCC. It highlights the importance of considering PSE in cancer patients with unexplained altered mental status and negative standard workups, particularly those with prior vascular tumor extension, since recognition enables targeted treatment and can uncover otherwise occult malignant recurrence. **Conclusion:** This case emphasizes the importance of considering portal hypertensive physiology in patients with unexplained hyperammonemia and altered mental status, even in the absence of liver disease and particularly in patients with history of cancer.

4673 | NOT ALL VARICES ARE ESOPHAGEAL: RARE PRESENTATION OF PARASTOMAL VARICES

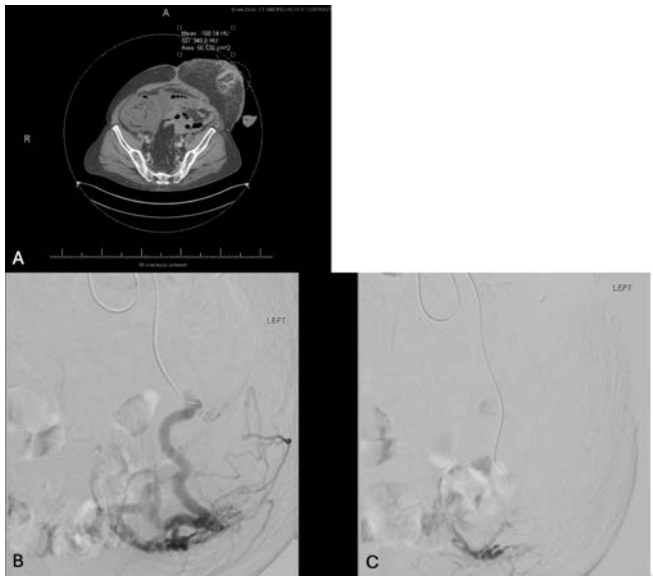
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Introduction: Parastomal variceal bleeding is a rare, life-threatening complication of portal hypertension that can occur in patients with cirrhosis and surgical stomas. We present a case of parastomal variceal bleeding in a patient with decompensated cirrhosis successfully treated with percutaneous transhepatic coil embolization. **Case Presentation:** A 57-year-old male with past medical history of decompensated alcohol related Child Pugh C cirrhosis with ascites and hepatic encephalopathy, perforated diverticulitis status post Hartmann's procedure with end colostomy complicated by large parastomal hernia presented with significant bloody output in his ostomy bag. He was noted to have stomal bleeding that was successfully treated

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with suture ligation. He returned a few days later with recurrent bleeding. Computed tomography angiography was negative for active arterial bleeding and showed multiple portosystemic and body collaterals with a prominent ostomy varix (Figure 1A). Upper endoscopy did not reveal evidence of esophageal varices or other sources of bleeding. While preparing for colonoscopy, active spurting was seen from the stoma. General surgery emergently performed suture ligation in the endoscopy suite. Subsequently, the patient underwent percutaneous transhepatic coil embolization of the parastomal inferior mesenteric varices(Figure 1B, 1C). There were no further signs of bleeding. **Discussion:** Parastomal varices occur due to the formation of portosystemic collaterals between the abdominal wall and stoma. Endoscopy and CT imaging are typically unremarkable due to the superficial location of venous pathology. Careful inspection of the stoma site after removal of the appliance is paramount as it will reveal the characteristic bluish-purple hue of engorged vessels around the stoma. Treatment modalities include transjugular intrahepatic portosystemic shunt (TIPS), percutaneous transhepatic embolization, EUS guided coiling, sclerotherapy or surgical revision of the stoma if possible. The multidisciplinary team discussed further management of portal hypertension via TIPS; however, the patient had prior complications with hepatic encephalopathy and poor compliance, so this was avoided. **Conclusion:** There is a paucity of evidence dictating optimal treatment approach due to the rarity of parastomal variceal bleeding. TIPS effectively manages portal hypertension and rebleeding risk, with success rates up to 95% when paired with percutaneous antegrade transhepatic venous obliteration. Surgical repair is avoided due to comorbidities and increased rates of poor healing and postoperative mortality. Minimally invasive approaches are often preferred in this group due to concerns about hepatic encephalopathy and the need for general anesthesia. Despite TIPS's strong success rate, a 20% rebleed rate is still reported in the literature. Treatment must be individualized following multidisciplinary team discussions.



4674 | NON-CIRRHOTIC PORTAL HYPERTENSION IN A PATIENT WITH HEPATIC ARTERIOVENOUS MALFORMATIONS SECONDARY TO PTEN HAMARTOMA TUMOR SYNDROME

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Introduction: PTEN hamartoma tumor syndrome (PHTS) is a rare autosomal dominant disorder caused by germline mutations in the PTEN (phosphatase and tensin homolog) tumor suppressor gene leading to unopposed PI3K/AKT/mTOR signaling. This results in vascular abnormalities in ~54% of patients, most commonly in the soft tissues of the extremities, chest, and axilla. Hepatic arteriovenous malformations (AVMs) are exceedingly rare, with only one prior published report identified. We present a case of a young woman with PHTS complicated by severe non-cirrhotic portal hypertension secondary to hepatic AVMs who was treated with therapy targeting the PI3K/AKT/mTOR pathway while awaiting liver transplantation (LT). **Case Presentation:** A 33-year-old woman with a known pathogenic PTEN variant (c.48T>A; p.Y16*) and multisystem manifestations of PHTS (breast cancer, multinodular goiter, dysplastic colon polyps, and lipomas) presented with recurrent bleeding esophageal varices, refractory high SAAG ascites, spontaneous bacterial peritonitis, mild hepatic encephalopathy, and non-occlusive mesenteric thrombosis. Imaging showed multiple large hepatic AVMs with liver biopsy negative for cirrhosis or porto-sinusoidal vascular disease. Hepatic venous pressure gradient was 4 mmHg, consistent

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with a presinusoidal etiology. She did not meet criteria for hereditary hemorrhagic telangiectasia. Her portal hypertension was therefore attributed to PHTS. She was listed for LT but has not been transplanteddue to severe malnutrition and sarcopenia (BMI 14.8 kg/m2). Subsequent imaging revealed additional high-flow mesenteric AVMs and bowel edema. A trial of medical AVM management was preferred over TIPS or AVM embolization. She was started on the AKT inhibitor capivasertib for upstream PI3K/AKT/mTOR blockade. Serial D-dimers and Doppler AVM flow dynamics will be used to assess response to treatment. **Discussion:** Hepatic AVMs are a rare potential visceral manifestation of PHTS. To our knowledge, there appears to be only one other published report linking hepatic AVMs to this syndrome. While LT is the definitive management of diffuse AVMs causing severe NCPH, a case series from 2024 demonstrated clinical benefit of the mTOR inhibitor sirolimus in 6 of 7 patients with PHTS-associated vascular anomalies. Outcomes included reduction in malformation size and improved pain control over a 2.5-year period. Furthermore, a 2025 preclinical study showed that use of the AKT inhibitor capivasertib effectively blocked vascular lesion growth in PHTS mouse models. This rationale supports the empiric trial of capivasertib in our patient. **Conclusion:** This case highlights severe non-cirrhotic portal hypertension in a patient with hepatic AVMs secondary to PTEN hamartoma tumor syndrome with refractory ascites, severe malnutrition, and sarcopenia treated with novel PI3K/AKT/mTOR-targeted therapy as a potential bridge to transplant.

4675 | HETEROZYGOUS ABCB4 VARIANT IN AN ADULT WITH UNEXPLAINED CHRONIC CHOLESTASIS, EARLY CHOLELITHIASIS, AND INTRAHEPATIC CHOLESTASIS OF PREGNANCY

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Introduction: Although biallelic pathogenic variants classically cause progressive familial intrahepatic cholestasis type 3, heterozygous ABCB4 variants are increasingly recognized in adults with intrahepatic cholestasis of pregnancy (ICP), early cholelithiasis, and chronic cholestatic liver disease. **Case Presentation:** A 38-year-old woman was referred for abnormal liver tests dating to 2012, when she developed gallstone pancreatitis requiring cholecystectomy. She later developed ICP during her second pregnancy and persistent liver enzyme abnormalities in 2022. In 2024, she sustained acetaminophen-related liver injury, with persistent abnormalities after recovery. She denied alcohol/drug use, herbal supplements, or family history of liver disease. Labs showed AST 100 U/L, ALT 221 U/L, alkaline phosphatase 138 U/L, and bilirubin 0.5 mg/dL, with preserved albumin and INR. Hepatitis B/C testing, ceruloplasmin, ANA, smooth muscle and antimitochondrial M2 antibodies, SP100, GP210, tissue transglutaminase IgA, and IgG/IgM were unrevealing; total IgA and alpha-1-antitrypsin were normal (MM phenotype). MRI was normal; MR elastography showed no fibrosis (2.2 kPa). A cholestasis panel identified a heterozygous ABCB4 c.1294G>A (p.Gly432Ser) VUS absent from ClinVar and gnomAD and predicted damaging in silico. A heterozygous likely pathogenic DGUOK variant lacked a second allele. Ursodiol 500 mg twice daily was started; biochemical follow-up is pending. **Discussion:** Monoallelic ABCB4-associated disease challenges the traditional view of ABCB4 disease as exclusively recessive and expands the role of genetic testing in otherwise unexplained chronic cholestasis, particularly with ICP or early cholelithiasis. In this patient, early gallstone disease, ICP, and persistent unexplained liver enzyme abnormalities support a clinically meaningful ABCB4-related phenotype despite a novel heterozygous variant and absent fibrosis. Given the concordant phenotype, unrevealing evaluation for competing etiologies, and absence of fibrosis on MR elastography, ursodiol with biochemical and noninvasive fibrosis monitoring was favored, reserving liver biopsy for inadequate response or disease progression. **Conclusion:** Genetic testing should be considered in adults with unexplained cholestatic liver disease, particularly with ICP or early gallstone disease. Monoallelic ABCB4 variants should not be dismissed; genotype-phenotype correlation may identify clinically meaningful disease.

4676 | SEVERE ASHWAGANDHA INDUCED LIVER INJURY AT A STANDARD DOSE

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Introduction: Ashwagandha is among the most widely used herbal supplements in US adults. LiverTox grades it as a likely cause of clinically apparent liver injury (likelihood score B), yet controlled trials report no hepatotoxic signal at doses up to 2,000 mg daily. Reported cases are typically cholestatic or mixed, arise 2 to 12 weeks after initiation, and the highest ALT catalogued by LiverTox is 3,400 U/L, in a case requiring

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transplantation. **Case Presentation:** A previously healthy 29 year old man presented with abdominal pain and vomiting two days after an otherwise social drinker who consumed approximately 10 alcoholic drinks on two consecutive nights. RUQ US and CT demonstrated diffuse hepatic inflammation and parenchymal edema without biliary obstruction. Peak ALT/AST reached 14,100/12,200 U/L with ALP 130 U/L and total bilirubin peaking after 3 days at 7.3 mg/dL, meeting Hy's Law. INR rose to 6.2 though he was not encephalopathic during admission. Hepatitis A-E, ANA, AMA, and anti-smooth muscle antibody were negative; CT angiography excluded Budd-Chiari syndrome. He reported intermittent ashwagandha use over several years without injury, with a 5 month gap before resuming 2 weeks prior at 600 mg daily (Nutricost). All supplements were stopped and he received N-acetylcysteine per standard acetaminophen protocol. RUCAM score was 7 (probable), supporting ashwagandha as causative even accounting for alcohol. ALT fell more than 50% of the excess above ULN within 4 days and he was discharged without transplant evaluation. **Discussion:** The patient's peak ALT is roughly four fold the highest value catalogued by LiverTox. Injury occurred at 600 mg daily, the dose most trials describe as well tolerated, after years of uneventful use. Despite an INR of 6.2, preserved mental status placed him in severe acute liver injury rather than acute liver failure, preventing need for transplant evaluation. Injury followed two nights of binge drinking. While RUCAM supported ashwagandha as causative independent of alcohol, we hypothesize that alcohol-mediated glutathione depletion may have aggravated, rather than confounded, this hepatotoxicity. **Conclusion:** Ashwagandha can produce hepatocellular injury far exceeding the severity described in trials or prior reports, at a standard dose, in a young patient without liver disease. Injury here followed years of intermittent uneventful use and coincided with binge drinking in an otherwise social drinker. Patients should be counselled that prior uneventful use and reassuring trial data do not ensure safety.

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