LIVER

S2904 ACG Case Reports Journal Award (Trainee)

A Superfood With a Dark Side: A Case of Severe Liver Injury Due to Turmeric

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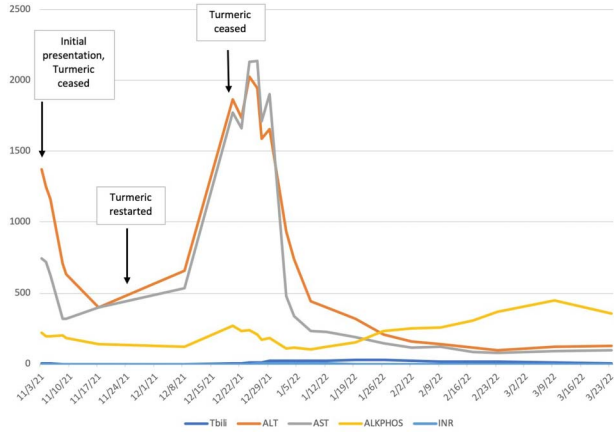
Introduction: Herbal and dietary supplements are a common cause of drug induced liver injury. Turmeric is a supplement used for anti-inflammatory and anti-neoplastic effects believed to have a good safety profile. Turmeric induced severe liver injury is a rare entity that has not been well-described.
Case Description/Methods: A 49 year old female with no significant medical history was admitted for acute liver injury with total bilirubin 2.0, alanine aminotransferase (ALT) 1300, aspartate aminotransferase (AST) 745, alkaline phosphatase (ALP) 220, international normalized ratio (INR) 1.1. Workup of her liver injury with serologic workup and imaging were negative. She admitted to taking a 1000 milligram daily dose of turmeric in formulation with black pepper for the past 3 months. Her liver tests improved and she was discharged with instructions to avoid further herbal supplements. Two weeks later, outpatient labs showed improving liver tests with total bilirubin 0.9, ALT 397, AST 320, ALP 138, INR 1.0 One month later, she was readmitted and was found to have total bilirubin 3.9, ALT 1865, AST 1770, ALP 269, INR 1.2. Repeated serologic workup and imaging were again unremarkable. Her liver tests continued to increase, peaking with total bilirubin 27.8, ALT and AST above 2000, INR 2.3 without encephalopathy. Percutaneous liver biopsy showed severe acute hepatitis with hepatocyte dropout and focal parenchymal collapse without significant fibrosis, concerning for drug-induced liver injury. She admitted that a few weeks after her first hospitalization, she resumed taking turmeric. Due to rising INR, she was treated with prednisone and monitored closely. After an extended hospitalization, she was discharged home with a prednisone taper. On discharge, total bilirubin was 23, ALT 445, AST 231, ALP 124, INR 1.6. Over the next 6 months, her liver tests significantly improved, including normalization of bilirubin and INR. She continues to avoid turmeric. (Figure)
Discussion: While studies have reported mild liver function test abnormalities with turmeric use, this case illustrates a rare example of severe turmeric induced liver injury in the setting of a positive rechallenge. The risk of severe liver injury may be increased when turmeric is taken in formulation with black pepper, which aids intestinal absorption of turmeric's active ingredient curcumin. As turmeric is a highly unregulated supplement, physicians should be aware of patients who are taking this supplement and the potential adverse effects associated with it.



[2904] **Figure 1.** Liver tests over time in the setting of turmeric cessation and rechallenge.

S2905 Presidential Poster Award

HBV-Host Junctional Sequences: A Novel Urine Tumor Marker for Recurrent Hepatitis B-Associated Hepatocellular Carcinoma With Low Alpha-Fetoprotein

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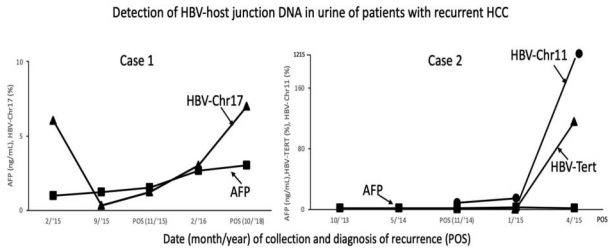
Introduction: Hepatocellular carcinoma (HCC) is one of the most common and deadly cancers worldwide. A common risk factor for liver cancer is chronic infection with the hepatitis B virus (CHB). HCC screening and surveillance is limited by diagnostic accuracy of current testing modalities. Prior studies suggest the potential of urine-based biomarkers for HBV-associated HCC (HBV-HCC), as DNA fragments from both virus and tumor have been detected in urine from those with CHB and HCC. As most HBV-HCC tumors contain integrated components of the HBV genome, unique HBV-host junctional sequences (HBV-JS) represent a viable molecular signature to identify HCC. This study evaluated a novel urine-based biomarker platform, utilizing HBV-JS DNA as a possible marker of tumor recurrence in patients with HBV-HCC.
Case Description/Methods: Urine sample from HBV-HCC patients with HCC recurrence confirmed by MRI was obtained. HBV-JS were detected by an HBV-targeted NGS assay (JBS Science Inc., Doylestown, PA) followed by ChimericSeq for junction detection. The most abundant NGS-detected junction sequences were then validated by PCR-Sanger sequencing. Quantitative junction-specific PCR assays were developed to track dynamic changes of HBV-JS in the urine specimens. HBV-JS sequences were detected from 2 cases of HBV-HCC with tumor recurrence (Figure). **Case 1**: A 78-year-old female with HBV-related cirrhosis was diagnosed with HCC in 2015. After microwave ablation, follow-up MRI revealed a new LI-RADS 3 lesion 1 year later. Subsequent imaging remained radiographically indeterminate until 2018 when the lesion was classified as definite HCC (LI-RADS 5). While serial AFP levels were negligible and MRI results variable, the unique HBV-JS DNA, HBV-Chr17, steadily increased from initial diagnosis to HCC recurrence. **Case 2:** A 74-year-old male with HBV-related cirrhosis was diagnosed with HCC in 2014, which recurred with a LI-RADS 5 lesion after 1 year despite loco-regional therapy. While AFP levels were negligible, two HBV-JS DNA, HBV-Chr11 and HBV-Tert, rapidly increased after initial HCC diagnosis.
Discussion: Unique HBV-JS sequences were detectable in the urine of patients with HBV- HCC. These sequences were detectable at increasing levels prior to diagnosis of recurrence of HCC by MRI imaging or AFP elevation. Together, our data suggest that HBV-JS DNA in urine maybe a further biomarker for the detection of HCC recurrence in patients with HBV-HCC.



[2905] **Figure 1.** HBV-host junctional sequences (HBV-JS) were detected from 2 cases of HBV-associated HCC with tumor recurrence

S2906 Presidential Poster Award

Metastatic Ocular Melanoma Mimicking Budd-Chiari Syndrome

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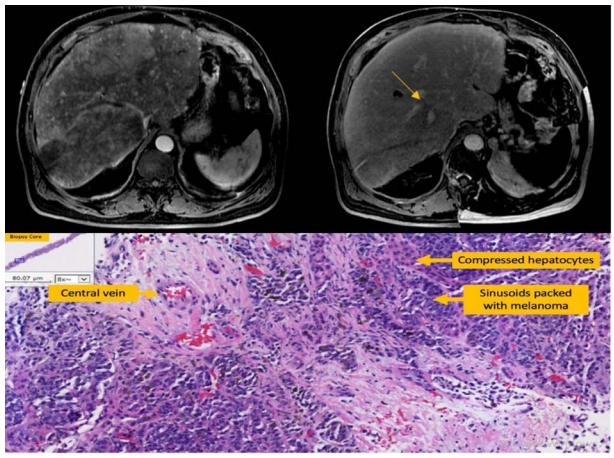
Introduction: Budd-Chiari syndrome (BCS) is hepatic venous outflow obstruction at the hepatic venules, hepatic veins or IVC that causes right-upper quadrant (RUQ) abdominal pain, hepatomegaly and ascites. It can present with a wide spectrum of imaging findings. We present a novel case of metastatic ocular melanoma that mimicked several clinical and radiographic features of BCS
Case Description/Methods: 78-year-old man with a history of uveal melanoma treated with radiation therapy 8 years ago presented with abdominal pain, distention and jaundice. He had no history of liver disease, new medications or heavy alcohol use. Physical examination showed jaundice, RUQ abdominal pain and abdominal distention with bulging flanks. Labs showed elevations in total bilirubin (7.8 mg/dL), alkaline phosphatase (131 U/L), alanine aminotransferase (158 U/L), aspartate aminotransferase (272 U/L) and prothrombin time (15.6 seconds). Doppler ultrasound showed hepatomegaly, ascites and no blood flow in the right hepatic vein. MRI of the abdomen revealed hepatomegaly with heterogeneous enhancement of the liver parenchyma, several hyper-enhancing hepatic nodules and evidence of thrombosis in the posterior right lobe hepatic veins (Figure 1). Given a high suspicion for BCS based on this, venography was done, showing nonocclusive thrombus in the peripheral branches of the right hepatic vein and patent central and left hepatic veins. The calculated sinusoidal pressure gradient was 12 mmHg. Liver biopsy was done and showed an effaced liver architecture from multi-nodular malignant pigmented cell infiltrates preferentially distributed in the centrilobular sinusoids compressing adjacent hepatocytes, and displaying histopathological features indicative of metastatic melanoma (Figure 1). The patient had progressive liver failure and was discharged to hospice care
Discussion: Imaging findings and clinical presentation are often needed to definitively diagnose BCS. Typical imaging findings include occlusion of the hepatic veins and IVC, caudate lobe hypertrophy, inhomogenous liver enhancement and intrahepatic collateral vessels and hypervascular nodules. But, as in this case, other causes, like invasive intrahepatic malignancy, can present with similar radiographic findings and venography may be needed to definitively diagnose BCS. Uveal melanoma, albeit rare, commonly metastasizes to the sinusoids in the liver and can mimic clinical and radiographic features of BCS, especially when the metastases preference the centrilobular sinusoids



[2906] **Figure 1.** Top left panel shows diffuse enhancement of liver parenchyma and multiple enhancing liver lesions; Top right panel suggestive of posterior right hepatic vein occlusion (arrow). Bottom panel shows nests of malignant tumor cells infiltrating the centrilobular sinusoids between hepatocytes.

S2907 Presidential Poster Award

COVID-19 Vaccine-Induced Liver Injury: A Case Series

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Introduction: The coronavirus 2019 (COVID-19) pandemic has caused over 248 million cases and over 5 million deaths worldwide. Given its high morbidity and mortality, there was a rapid effort to develop a vaccine within a year of the commencement of the pandemic. Although these vaccines have excellent safety profiles, there is a risk of adverse effects such as fever, fatigue, arthralgias, injection site pain, and, less commonly, anaphylactic reaction. Drug-induced hepatotoxicity (DIH) is a rare side effect of vaccines that has rarely been reported. We report a cohort of four patients who presented with DIH following COVID-19 vaccination.
Case Description/Methods: Our cohort includes four patients, aged 39-70, who presented between 12 to 82 days after their second dose of Pfizer COVID-19 vaccine. Only one patient had a history of COVID-19 infection. One patient had a history of hepatic steatosis. All four patients had normal alanine transaminase [ALT] and aspartate transaminase [AST] within one year before the current presentation. All patients demonstrated a hepatocellular pattern of liver injury (peak ALT range of 57 to 904 U/L and AST range of 51 to 828 U/L). Tests for acute viral hepatitis were negative. One patient was found to have positive antinuclear antibodies and anti-smooth muscle antibodies. One patient was found to have an elevated antimitochondrial antibody. Liver stiffness measurement in three patients showed no evidence of fibrosis. Three patients underwent liver biopsy of which two suggested evidence of toxic/drug induced liver injury while the other demonstrated nonspecific inflammation. (Figure)
Discussion: DIH leads to 10% of all cases of acute hepatitis and up to 50% of all cases of liver failure, making it one of the common reasons for withdrawal of medications from the market. We observed that our patients developed hepatic injury after Pfizer COVID-19 vaccination. Vaccine-induced immune-mediated hepatitis is a known phenomenon thought to be secondary to the COVID-19 spike protein triggering an auto-immune-like hepatic condition. This could explain the findings seen in our patients, raising the question of inflammatory response in patients with underlying autoimmune conditions. It is important that these patients receive pre and post vaccination laboratory monitoring, especially given the emergence of booster vaccinations. There is a need to follow these patients in the long-term to monitor for changes in clinical and laboratory studies in order to assess the risk of complications and outcomes.

Patients' characteristics									Peak lab values					Relevant work up (labs, imaging, pathology)			
Case	Age, Sex	Race	BMI	Alcohol use (per week)	Co-morbid conditions	Liver disease co-morbidities	Presenting symptoms	Timing of presentation (days) after 2nd vaccine *	ALT (U/L)	AST (U/L)	ALP (U/L)	Total Bilirubin (mg/dL)	Ferritin (ng/mL)	Antibody Panel	Transient Elastography	Liver Biopsy	Liver Imaging	Return of ALT & AST to baseline
1	58, M	White	25.4	<1 drink	HLD	Hepatic Steatosis	Fatigue, arthralgias	12	101	51	77	0.8	478.12	ANA: - AMA: + ASMA: - IgM: 78 IgG: 882	CAP 231 kPa 5.1	Mild porto-sinusoidal vascular disease. Mild ferritin and hemosiderin deposition. No evidence of autoimmune hepatitis or steatohepatitis.	Not performed	Yes
2	46, F	White	27.4	2-4 drinks	Migraines, OSA	None	Fever, fatigue, arthralgias	42	57	58	147	0.4	662	ANA: - AMA: - ASMA: -	Not performed	Not performed	Not performed	Yes
3	70, F	Black	28.9	7 drinks	HTN, HLD, Grave's disease	None	Incidental elevation of liver function tests	82	904	828	85	0.8	604	ANA: + AMA: - ASMA: + IgM: 66 IgG: 2127	CAP 152 kPa 5.5	Mixed lymphoplasmacellular infiltrates with mild to moderate interface hepatitis, focal bridging necrosis and focal multiacinar necrosis. Consistent with AIH superimposed by DILI or DILI-initiated AIH.	US: Normal	Yes
4	39, F	White	19.6	<1 drink	HSV2	None	Fever, chills, myalgias, LAD	81	596	454	213	1.7	358	ANA: AMA: ASMA: IgM: IgG: cANCA: +	CAP 155 kPa 3.4	Focal portal fibrosis. Perivenular necroinflammation with hepatocyte dropout, mild lymphocytic inflammation and steroid-containing macrophages. Mild predominantly centrilobular small-droplet steatosis. Consistent with acute drug /toxin induced liver injury.	US: mildly echogenic liver MRCP: Normal	Yes

* All patients received Pfizer vaccination.

BMI: Body Mass Index, HTN: Hypertension, HLD: Hyperlipidemia, OSA: obstructive sleep apnea, HSV2: herpes simplex virus, LAD: Lymphadenopathy, ALT: alanine transaminase, AST: aspartate aminotransferase, ALP: Alkaline phosphatase, ANA: antinuclear antibodies, ASMA: anti-smooth muscle antibody, AMA: Antimitochondrial antibodies, IgM: Immunoglobulin M, IgG: Immunoglobulin G, cANCA: Antineutrophil Cytoplasmic Autoantibody, CAP: controlled attenuation parameter, kPa: kilopascal, AIH: autoimmune hepatitis, DILI: Drug Induced Liver Injury, US: ultrasound, MRCP: Magnetic resonance cholangiopancreatography.

[2907] **Figure 1.** Patient demographics and characteristics

S2908 Presidential Poster Award

Liver Transplant for Management of Neurological Wilson Disease in a Patient With Preserved Liver Function

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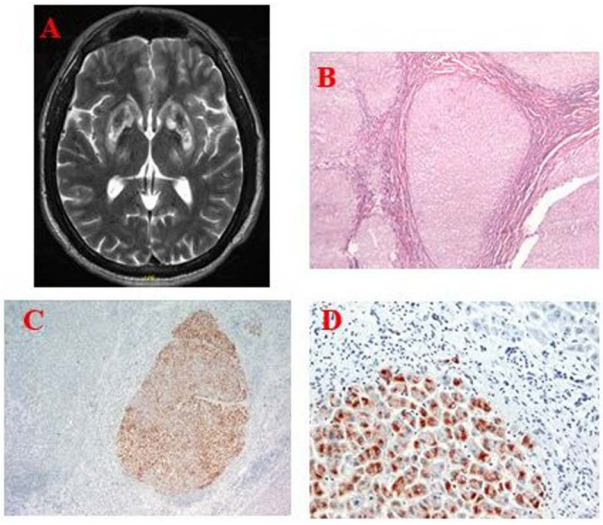
Introduction: The role of orthotopic liver transplant (OLT) solely for the management of neurological Wilson disease (WD) is unclear. We present a patient with WD who underwent OLT for neurological deterioration with compensated cirrhosis.
Case Description/Methods: A 19-year-old male presented with three-month history of dysarthria, akinesia, and fine tremors. His past medical history was remarkable for unexplained splenomegaly and thrombocytopenia. He denied history and risk factors for liver disease. Physical exam showed slurred speech, bilateral hand tremors, spasticity of the lower extremities, and absence of Kayser-Fleischer ring. Labs were unremarkable, except for platelets of 79 k/uL. MRI brain showed bilateral, symmetric hyper-intensity in the basal ganglia, lateral thalami and midbrain on T2-weighted images. (Figure) CT liver revealed cirrhosis, portal hypertension, and mild splenomegaly. Copper metabolism study confirmed low serum copper (8 μmol/L) and high 24-hr urine copper (234 μg/d) consistent with diagnosis of WD. He was promptly started on Zinc and Trientine 250 mg daily. After one month, the patient was re-admitted for worsening dysphagia, dysarthria and rigidity, eventually becoming bedridden with inability to talk and eat requiring G-tube placed). Liver biopsy showed cirrhosis with little inflammatory activity and abundant copper on Rhodanine stain (>600 mcg/g). The patient underwent OLT after being granted a Metabolic MELD exception of 40 due to medically unresponsive neurological impairment, with preserved liver function (calculated MELD 8). Post-OLT, he had gradual neurological improvement, except persistent dystonia and spasticity. Serum ceruloplasmin levels normalized but repeat MRI showed persistent signal alterations at the basal ganglia and brainstem. At 16 month follow-up, his neurological recovery remained largely stagnant with minimal improvement in extrapyramidal symptoms.
Discussion: OLT for management of neurological WD remains highly debatable. Most data on neurological recovery after LT are based on patients with mixed liver and neurological involvement with success rate of 62%. In our patient, he failed to display complete neurological recovery 1.5 years after OLT despite initial improvement. The rapid neurologic deterioration that led to OLT occurred after initiation of chelation therapy which may indicate paradoxical worsening. Additional research is needed to determine the timing of OLT and role of medical optimization prior to OLT in patients with neurological WD.



[2908] **Figure 1.** (A) MRI Brain with contrast showing (T2/FLAIR) hyperintensity and mild edema symmetrically in the caudate heads, putamina, ventral lateral thalami, and midbrain. Findings are suspicious for Wilson Disease. (B) Cirrhotic nodules accompanying mild chronic inflammation (Hematoxylin and eosin, 100X). (C) The liver cells of this cirrhotic nodule are extensively filled with copper (Rhodanine stain, 100X) (D) The liver cells show bright red granules of copper in the cytoplasm (Rhodanine stain, 200X).

S2909 Presidential Poster Award

Severe Immune-Mediated Liver Injury Following COVID-19 Vaccination Necessitating Liver Transplantation in a Patient With Cirrhosis

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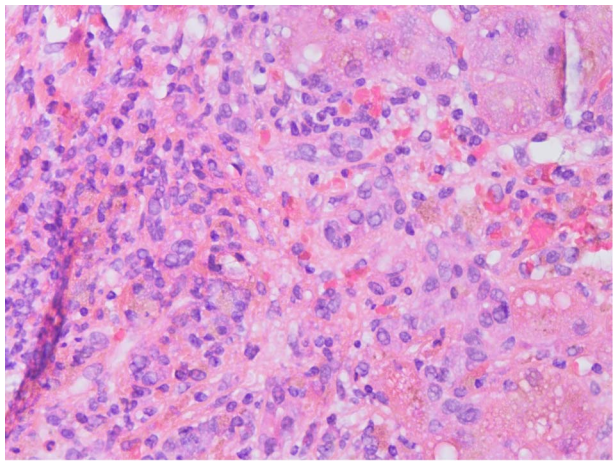
Introduction: Pfizer COVID-19 vaccine (C19V) uses mRNA encoding SARS-CoV-2 spike protein to elicit antibody response. Recent literature documented temporal correlation between C19V and immune-mediated liver injury (ILI). The proposed mechanisms are molecular mimicry and immune cross-reactivity in genetically susceptible individuals. All had an excellent response to corticosteroids (CS). We present a case of severe ILI post C19V in a patient (pt) with compensated alcohol-associated cirrhosis who failed CS and needed liver transplantation (LT).
Case Description/Methods: 65 yo Caucasian male with epilepsy and coronary artery disease presented with jaundice and ascites on day 10 after first C19V. He had 30+ years of excessive alcohol use. No known liver disease and had normal liver enzymes prior. On levetiracetam, metoprolol, ramipril, aspirin and omeprazole for years. Denied any new medications or herbals. No family history of autoimmune diseases.

Admission labs: AST 1,786, ALT 1,613, ALP 182, TB 9, INR 2.1, Cr 0.7; negative hepatitis A, B, C, CMV, EBV, HIV; positive ANA, ASMA, AMA; total IgG of 1310. CT abdomen: heterogeneous liver with steatosis and mild nodularity, splenomegaly, ascites, patent vasculature, and no biliary dilation. Liver biopsy: marked portal and lobular inflammation with interface hepatitis and lymphoplasmacytic infiltrates, confluent necrosis with hemorrhage, balloon degeneration, cholestasis and reticulin collapse. (Figure) Pt deteriorated and developed hepatic encephalopathy and acute kidney injury (Cr 2.6) despite high dose CS. MELD-Na peaked at 45 and pt had deceased donor LT 53 days post C19V. Explant: cirrhosis with cholestasis and chronic inflammation.

Discussion: mRNA-based vaccine has been associated with increased risk of immune mediated disease, attributed to intrinsic immunostimulatory effect and proinflammatory cytokine activation. C19V associated ILI in this case is supported by time to onset, lack of autoimmune risk factors or other causes of liver injury, liver histopathology and case reports of similar findings. Our pt did not respond to CS. We postulate C19V induced ILI triggered intense systemic inflammation and immune dysfunction that led to acute hepatic decompensation and organ failure in this pt with compensated cirrhosis (i.e acute on chronic liver failure). To our knowledge, this is the first report of severe ILI following C19V necessitating LT in a cirrhotic pt. We wish to raise awareness of ILI as a potential complication of C19V but not to discourage vaccination.



[2909] **Figure 1.** Liver biopsy: marked portal and lobular inflammation with interface hepatitis and lymphoplasmacytic infiltrates

S2910 Presidential Poster Award

Transcatheter Aortic Valve Replacement Restoring Candidacy for Liver Transplant in Cirrhotic Patients

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Introduction: Current guidelines for preoperative workup for an orthotopic liver transplant (OLT) recommend echocardiogram and cardiac testing that classically rule-out patients with superimposed severe aortic stenosis as transplant candidates. We evaluated the potential of transcatheter aortic valve replacement (TAVR) as a bridging therapy to restore the candidacy of cirrhotic patients with severe AS for liver transplant.

Case Description/Methods: A retrospective chart review was performed on 472 patient records that underwent liver transplants at a single large tertiary care center between 2017 and mid-2021. We identified patients who had a history of severe aortic stenosis corrected by TAVR. We extracted demographic and long-term outcome data. Two patients underwent the TAVR procedure which alleviated AS and allowed for reinstatement of eligibility for OLT. Evaluation at 1-month, 3-months and 1-year post-liver transplant showed no complications in the function of or related to the prosthetic aortic valves. Patient survival at 1 year and 2 years post-transplant was 100%. Mean time of follow-up post-transplant was 27 months. Graft survival at 1 year and 2 years was 100% with no evidence of rejection. Both 1 year and 2-year follow-up labs showed that patients were normotensive and had lipid profiles within the normal range. One of the patients had baseline high HbA1c consistent with their history of type 2 diabetes that continued to show in their follow-up labs while the other had normal glucose and HbA1C throughout. Noteworthy post-transplant complications included a left lower quadrant hematoma in one patient which resolved spontaneously and alcoholic recidivism managed through transplant psychiatry care. One patient did have a left-sided facial droop and right-sided pronator drift two days following the liver transplantation. CTA showed 74% stenosis of the right ICA and MRI confirmed hypoxic ischemia. Facial droop and drift resolved upon revaluation a few hours after onset. (Table)

Discussion: In the setting of these growing contraindicated problems of AS and liver cirrhosis, we wanted to contribute to the currently scarce compilation of case reports that illustrates the effect that the TAVR procedure has on relieving the pre-transplantation risks of aortic stenosis in high-risk patients. The patients do have the potential for other cardiovascular diseases long-term and should be monitored closely with modification of risk factors.

Table 1. This is a comparison of echocardiogram parameters of aortic valve morphology and function for both patients #1 and #2 before and after TAVR as well as after OLT

Parameters	Patient #1			Patient #2		
	Before TAVR	After TAVR	After OLT	Before TAVR	After TAVR	After OLT
Aortic valve peak gradient (mmhg)	16.9	12.2	16.6	67.7	51.4	50.0
Aortic valves mean gradient (mmhg)	8.4	5.8	9.8	38.3	24.5	25.5
Left ventricular ejection fraction (%)	61	46	55	>70	67	63
Aortic valve area, Vmax (cmÂ2)	2.22	1.53	3.12	0.92	0.98	1.18

The post-TAVR parameters occurred 2 days after the procedure for patient #1 and <6 months for patient #2 due to care referral to our offices. The post OLT echo parameters were measured two months after patient #1's transplant and 4 days after patient #2 transplant.

S2911 Presidential Poster Award

The Spectrum of Spontaneous Bacterial Empyema: An Elusive Disease

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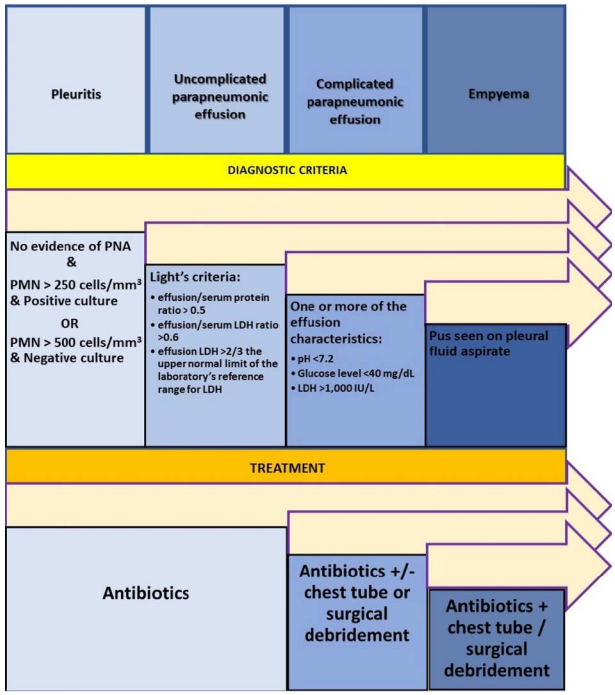
Introduction: Spontaneous bacterial empyema (SBE) is a potentially fatal complication of hepatic hydrothorax (HH). Despite high mortality rate, guidelines do not clearly outline the clinical trajectory and management of SBE. We aim to present a case of SBE to familiarize clinicians with the spectrum of SBE presentations.

Case Description/Methods: A 58-year-old male with a history of alcohol use disorder and alcohol-associated cirrhosis decompensated with ascites and left-sided HH presented with fever of 38.1°C. Physical exam was notable for blood pressure 80/57, tachypnea at 28 breaths/minute, and decreased breath sounds in left lung fields. Labs were notable for leukocytosis and lactic acidosis (**Table**). Infectious workup was negative for spontaneous bacterial peritonitis (SBP) or urinary infection. Pleural fluid analysis is shown in **Table**. Blood and pleural fluid cultures grew Clostridium perfringens. CT chest showed loculated fluid in

the left pleural space. 12 French pigtail chest tube was placed. In addition to metronidazole and ceftriaxone, 6 doses of intrapleural fibrinolytics were administered. Chest tube was removed once the output had decreased. He was discharged on oral antibiotics and remained asymptomatic on 10-week follow up.

Discussion: SBE should be regarded as a spectrum where it can present as "simple" pleuritis (akin to peritonitis in SBP), uncomplicated parapneumonic effusion (PE), complicated PE or frank empyema similar to our case. It is important to delineate where your patient lies on that spectrum as treatment modalities will differ. To diagnose PE, the pleural effusion has to be exudative per Light's criteria. A complicated PE is diagnosed by the presence of exudative effusion that has one or more of the following characteristics: (i) pH $<$ 7.2, (ii) glucose level $<$ 40 mg/dL, or (iii) LDH $>$1000 IU/L. In complicated PE, identifying complex septations or loculations by imaging is pertinent, either by bedside ultrasound or CT. Frank empyema is diagnosed when pus is seen on pleural fluid sampling. Pleuritis and uncomplicated PE, is treated like SBP. Source control of the infection may be warranted in complicated PE/empyema. Chest tube drainage is the least invasive method. Chest tubes are relatively contraindicated in HH but have a role in SBE. Intrapleural fibrinolytics may assist in drainage. Surgical debridement is indicated if recurrence of complicated PE/empyema despite adequate drainage occurs. **Figure 1** highlights the SBE spectrum. Secondary prophylaxis is indicated after treatment.



[2911] **Figure 1.** Spectrum of Spontaneous Bacterial Empyema

Table 1. Laboratory results on admission

	Reference range, adults	On admission
Complete Blood Count		
White Blood Cells (/μL)	4,000-11,000	17,580
Hemoglobin (g/dL)	12.0-15.3	9.2
Platelet Count (/μL)	150,000-450,000	108,000
Comprehensive Metabolic Profile		
Lactic acid (mmol/L)	0.5-2.0	6.9
Sodium (mmol/L)	135-146	135
Potassium (mmol/L)	3.4-5.2	4.5
Blood Urea Nitrogen (mg/dL)	7-24	31
Creatinine (mg/dL)	0.5-1.1	2.5
Albumin (g/dL)	3.4-5.2	2.4
Aspartate Aminotransferase (IU/L)	11-40	28
Alanine Aminotransferase (IU/L)	4-35	8
Alkaline Phosphatase (IU/L)	30-115	72
Total Bilirubin (mg/dL)	0.0-1.2	3.2
Direct Bilirubin (mg/dL)	0.1-0.5	1.9
C-Reactive Protein (mg/L)	$<$ 5.0	43.5
Pleural fluid analysis		
White Blood Cells (/μL)		2,195
Glucose		$<$ 5
Lactate dehydrogenase		3,261
pH		6.87

S2912 Presidential Poster Award

Transjugular Intrahepatic Portosystemic Shunt as a Cure for Macrohematuria

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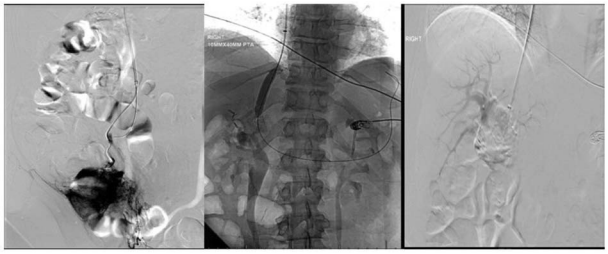
Introduction: Varices most commonly form in the esophagus or stomach, however, they can form anywhere along the gastrointestinal tract. Ostomy-related varices can be challenging to diagnose and treat. Here, we discuss a rare case of recurrent bleeding from an ileal conduit urostomy due to ectopic varices successfully treated with a transjugular intrahepatic portosystemic shunt (TIPS).
Case Description/Methods: A-59-year-old male with a history of ileal conduit due to spina bifida complicated by neurogenic bladder, and alcoholic cirrhosis presented with a 3-weeks of recurrent bleeding from the urostomy site. The patient presented three times with bleeding from his urostomy site. Each time he received a blood transfusion and was seen by urology, and was subsequently treated with silver nitrate, cauterization, and suturing. The bleeding would stop temporarily and then recur. He eventually presented with reports of "fist-sized" blood clots pouring into his ostomy bag. Sutures were placed to tamponade the bleeding by Urology. Physical exam was significant for hypotension and palor. Laboratory tests were significant for hemoglobin of 6.0 g/dl. Gastroenterology was consulted and bleeding was thought to be due to ectopic variceal hemorrhage at the stoma site. TIPS was performed by Interventional Radiology. During the procedure, it was noted that there were tortuous dilated peristomal varices in the distal superior mesenteric vein corresponding to the ileal conduit, which were subsequently sclerosed and embolized (Figure 1). The patient was monitored for 48 hours after the procedure. On follow-up after 2 months, he continued to do well and without recurrence of bleeding.
Discussion: Hemorrhage from stomal varices is a rare but serious complication of portal hypertension. While the bleeding sometimes may be limited to one area and thus respond to manual compression, more commonly, the bleeding is diffuse and requires further intervention. There are a few treatment modalities that can be explored in these cases including decompression with TIPS, endoscopic band ligation, endoscopic sclerotherapy, glue injection, or surgery. Decompression with TIPS carries a lower rate of complication and re-bleeding rate compared to other lines of management. Our patient underwent TIPS which resulted in the cessation of bleeding without complications.



TIPS placement with reduction of portosystemic gradient from 21 mmHg to 5 mmHg. Tortuous dilated peristomal varices are seen in the distal superior mesenteric vein at the level of the ileal conduit. Sclerosis and embolization of the peristomal varices were performed.

[2912] **Figure 1.** TIPS placement with reduction of portosystemic gradient from 21 mmHg to 5 mmHg. Tortuous dilated peristomal varices are seen in the distal superior mesenteric vein at the level of the ileal conduit. Sclerosis and embolization of the peristomal varices were performed.

S2913 Presidential Poster Award

Tocilizumab as an Effective Steroid-Sparing Agent for the Treatment of Recurrent and Steroid-Dependent Immune Checkpoint Inhibitor-Mediated Hepatotoxicity: A Case Study and Insight into Pathophysiology

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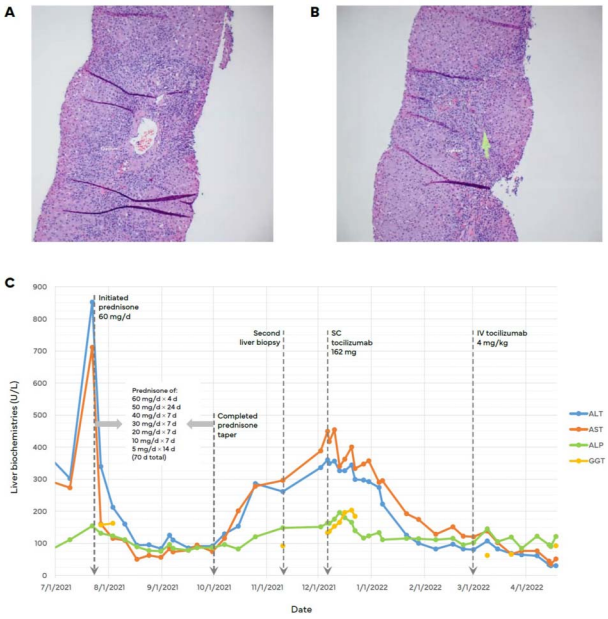
Introduction: Immunotherapy-mediated hepatotoxicity (IMH) is a well-recognized immune-mediated adverse event (irAE) in patients who undergo treatment with immune checkpoint inhibitors. An effective pathway for addressing steroid-resistant or steroid-dependent cases of IMH remains an area of ongoing investigation. Limited published experience has introduced tocilizumab, an IL-6 receptor antagonist, as a viable steroid-sparing agent to manage challenging cases of IMH. We describe a successful case of utilizing tocilizumab in both subcutaneous (SC) and intravenous (IV) forms, without concurrent steroids, to treat a case of recurrent and steroid-dependent IMH.
Case Description/Methods: A 32-year-old woman with a history of Hodgkin lymphoma and no underlying liver disease was evaluated for elevated liver enzymes. Her lymphoma was managed with autologous stem cell transplant (SCT) (2011), allogenic SCT (2012), nivolumab (2016), and lenalidomide (2019). She started pembrolizumab in 3/2020; the dose was increased by 12/2020. Due to CTCAE grade 3 liver toxicity in 1/2021, pembrolizumab was held. A liver biopsy performed in 2/2021 confirmed IMH (instead of hepatic graft-vs-host disease). Initial treatment with prednisone (total of 66 days) yielded biochemical remission. By the end of 4/2021, she was re-challenged with one dose of pembrolizumab but soon developed grade 4 liver enzyme elevations, so prednisone was reintroduced (induction dose 60 mg/d), but liver enzymes exhibited only partial improvement after 70 days of steroids. Liver enzymes increased again after stopping steroids. Repeat liver biopsy reaffirmed IMH. To avoid prolonged exposure to systemic corticosteroids, SC tocilizumab 162 mg was given, leading to subsequent improvement in liver enzymes. She developed a brief episode of shingles that was treated. IV tocilizumab 4 mg/kg was given about 2 months later to bring her to biochemical remission that was achieved 1.5 months thereafter, without need for steroids. (Figure)
Discussion: IL-6 plays an important role in liver biology. Limited reports feature tocilizumab as a feasible and effective option to treat select cases of IMH, including cholangiohepatitis phenotypes and steroid-refractory cases. In this case, both SC and IV tocilizumab conferred efficacy in treating IMH without concurrent systemic steroids. Our example further highlights the unmet clinical need to study steroid-sparing strategies in order avert a protracted course of steroids and to allow patients to engage sooner in additional cancer treatment.



[2913] **Figure 1.** (A) and (B): Histologic evaluation (hematoxylin & eosin) from the second liver biopsy, which was performed before initial tocilizumab administration. The key features include panlobular hepatitis with bridging/centrilobular necrosis. The portal tracts show slight expansion with a mixed inflammatory infiltrate, comprised mostly of lymphocytes and occasional neutrophils and plasma cells, interface activity, bile duct injury, and bile ductular proliferation, without bile ductopenia, and no florid duct lesions are identified. No granulomas are seen. (C): Timeline of liver biochemical tests and associated treatments. Although induction steroids with prednisone 60 mg/d was able to yield improvement in liver enzymes, subsequent elevation of liver enzymes after completion of steroid taper suggests she was steroid-dependent. Subcutaneous tocilizumab was administered, yielding significant response but not yet ALT normalization before a second dose of tocilizumab (now intravenous route) was administered to eventually attain biochemical remission. No additional corticosteroids were prescribed. Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; SC, subcutaneous; IV, intravenous; d, day.

Table 1. Chronological biochemical lab trends referenced from the time of initial tocilizumab (subcutaneous) administration

Lab (Reference range)	Day 0 (SC toci given)	Day 1	Day 14	Day 21	Day 30	Day 85	Day 93 (IV toci given)	Day 130
ALT (N ≤ 33 U/L)	360	349	344	297	274	80	107	30
AST (N ≤ 32 U/L)	449	417	400	347	290	120	138	35
ALP (N = 35 – 104 U/L)	163	162	165	116	133	102	145	90
GGT (N = 5 – 36 U/L)	133	137	203	-	-	-	62	-
IL-6 (N = 0 – 5 pg/mL)	102	89	-	-	-	-	222	-

No systemic corticosteroids were administered in the time frame displayed. Intravenous tocilizumab (4 mg/kg) was administered at 2.8 months from the initial tocilizumab dose, at which time there was already significant improvement in the transaminase levels, and biochemical remission (normalization of ALT) was achieved an additional 1.5 months thereafter. There was no correlation of liver biochemical response with the serum IL-6 level. Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; IL-6, interleukin-6; SC, subcutaneous; IV, intravenous; toci, tocilizumab.

S2914 Presidential Poster Award

Clinical Course and Diagnostic Challenge of Jamestown Canyon Virus Infection in Post Liver Transplant Recipient With Altered Mental Status

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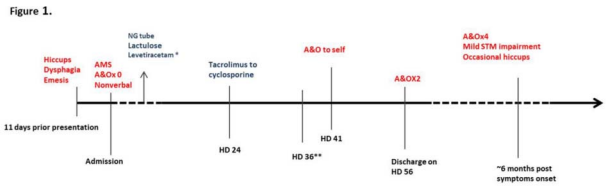
Introduction: Jamestown Canyon Virus (JCV) is an increasingly recognized mosquito-transmitted arbovirus found in North America, most prevalent in April-September. Infection ranges from asymptomatic to rarely neuroinvasive disease. Although mortality rates are low morbidity can be prolonged and diagnosis is challenging. Our case describes a liver transplant (LT) recipient with prolonged altered mental status (AMS) secondary to JCV encephalitis.
Case Description/Methods: A 72-year-old man from New Hampshire who had a LT for cryptogenic cirrhosis 2 years prior to presentation, on tacrolimus and mycophenolate mofetil for immunosuppression, presented with new, nonspecific neurological symptoms: hiccups, oropharyngeal dysphagia, AMS, and intermittent nonrhythmic twitching of facial muscles and legs (Fig 1). On admission, he had stable vitals and was alert, but disoriented, aphasic, and not following commands. He moved all extremities spontaneously with no focal motor neurological deficit. CBC, BMP, LFTs, brain MRI, and initial CSF analysis were normal (Table 1). Extensive viral, bacterial and fungal tests were negative on multiple occasions. There was no significant improvement with treatment for possible seizures, hepatic encephalopathy, or with switching tacrolimus to cyclosporine. After 1 month, new fever prompted repeat CSF analysis. CSF was notable for lymphocytic pleocytosis. Serum and CSF samples were sent to the State Public Health Laboratory for further testing. Mental status gradually improved. After discharge, serum JCV plaque reduction neutralization titer resulted positive at 1:640 consistent with recent infection. CSF JCV IgM capture ELISA and PRNT were negative. By six months after presentation, the patient had returned baseline except for mildly impaired short-term memory, fatigue, and occasional hiccups.
Discussion: JCV encephalitis may cause prolonged AMS in LT recipients. Diagnosis in LT recipients can be challenging. Symptoms may be attributed to other causes like immunosuppressant side effects. Diagnosis is based on exclusion of other etiologies and positive JCV serology. To our knowledge, this case is the first to describe the natural progression of JCV infection in a LT recipient. Although Intravenous immunoglobulin was previously used in another LT recipient with JCV, there remains no proven treatment for JCV[1].



[2914] **Figure 1.** Timeline of JCV-specific testing and treatment over disease course. Abbreviations: A&O, Alert and oriented; HD, hospital day; JCV, Jamestown Canyon virus; STM, short term memory. *Discontinued on HD 30. ** JCV testing performed and resulted after 2 months.

Table 1. CSF analysis data of test tube number 4

Hospital day (HD)	protein (15-45 mg/dL)	Glucose (40 to 70 mg/dL)	White Blood Cell count (< 10 cells/uL)	Differentials %
HD 7	117	84	4	Not available
HD 33	118	100	26	Lymphocytes 92%; Monocytes 8%; Neutrophils 0%

REFERENCE

1. Emily J Ciccone et al, Encephalitis Caused by Jamestown Canyon Virus in a Liver Transplant Patient, North Carolina, USA, 2017, O-F-I-D, Volume 9, Issue 3, March 2022, ofac031

S2915 Presidential Poster Award

A Case Report of Ivermectin-Induced Liver Failure

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Introduction: Ivermectin is an antiparasitic medication that is primarily metabolized by the liver. During the COVID-19 pandemic, researchers demonstrated that Ivermectin successfully inhibited the replication of SARS-COV-2 in vivo, but current research has failed to demonstrate clinical benefit for treatment of COVID-19. Despite this, misinformation campaigns have misled patients to ingest Ivermectin at concentrations meant for domestic animals. Here, we present a case of acute liver failure secondary to the use of Ivermectin.
Case Description/Methods: A 61-year-old man with medical history of ischemic cardiomyopathy with last echocardiogram showing ejection fraction at 21%, atrial fibrillation on warfarin for oral anticoagulation, and previously treated Hepatitis C presented with generalized weakness and yellowish discoloration of the skin worsening over the last two weeks. The patient denied significant alcohol use, acetaminophen use, or illicit drugs. He admitted to injecting himself with two doses of weight-based horse ivermectin, for COVID prophylaxis, two weeks prior to his presentation. Physical exam was pertinent for scleral icterus and hepatomegaly with no abdominal tenderness. Initial labs revealed elevated liver chemistries in a mixed pattern (Figure 1). Acute hepatitis panel, HSV, and CMV were negative. Hepatitis C antibodies were positive, but the patient was in sustained virologic response. Full workup for chronic liver disease was unremarkable. Ultrasound revealed hepatosplenomegaly with patent portal and hepatic vasculature. Subsequently, the patient developed hepatic encephalopathy along with his coagulopathy, raising concern for acute hepatic failure. The patient was transferred to the ICU and started on N-Acetylcysteine, rifaximin, and supportive care. The patient recovered well and fortunately did not require liver transplant.
Discussion: While the FDA recommends against the use of Ivermectin for COVID-19, many continue to inappropriately consume it. Ivermectin-induced liver failure is a rare but deadly side effect. Given our patient's rapid onset of symptoms post-self injection of Ivermectin, his liver injury was presumed to be related to Ivermectin. The drug interaction between Ivermectin and warfarin had worsened the patients coagulopathy. Physicians should be aware of the ways Ivermectin overdose may clinically present to avoid delayed treatment. This case demonstrates the detriments of perpetuation of medical misinformation to care.

Table 1. Relevant lab values throughout hospitalization

	Day 0	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 12	Day 22
Alkaline Phosphatase (u/l)	252 (H)	199 (H)	180 (H)	165 (H)	183 (H)	186 (H)	171 (H)	208 (H)	133	171 (H)	179 (H)	145 (H)
AST (u/l)	997 (H)	1,375 (H)	1,324 (H)	973 (H)	860 (H)	654 (H)	503 (H)	283 (H)	115 (H)	96 (H)	49 (H)	24
ALT (u/l)	1,036 (H)	1,146 (H)	1,106 (H)	979 (H)	975 (H)	865 (H)	745 (H)	595 (H)	304 (H)	350 (H)	206 (H)	47
Bilirubin,Total (mg/dl)	6.7 (H)	6.6 (H)	6.1 (H)	5.3 (H)	6.8 (H)	6.7 (H)	7.1 (H)	5.8 (H)	3.3 (H)	5.0 (H)	3.0 (H)	1.9 (H)
INR	12.6 (AA)*}"<<12.6 (AA)	12.6 (AA)*}"<<12.6 (AA)	8.2 (AA)	4.7 (H)	3.4 (H)	2.9 (H)		2.1 (H)	1.8 (H)	1.4 (H)	1.3	1.4

S2916 Presidential Poster Award

A Rare Case of Refractory Drug Induced Liver Injury Following Ocrelizumab Use for Multiple Sclerosis

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Introduction: Ocrelizumab, a humanized anti-CD20 monoclonal antibody, has been recently approved for use in Multiple Sclerosis (MS). Thus far, it has not been associated with liver enzyme elevations during therapy or idiosyncratic liver injury, but has been linked to cases of reactivation of Hepatitis B in susceptible patients. We present a case of severe liver injury in a patient with relapsing-remitting MS who received Ocrelizumab.
Case Description/Methods: A 34-year-old Hispanic woman with newly diagnosed MS was planned to start treatment with Ocrelizumab. Pre-treatment acute and chronic Hepatitis B labs were negative. During the first infusion session, the patient developed an infusion reaction with rash and pruritis, which resolved spontaneously. Three weeks later, she presented to the ED with abdominal pain, nausea, fatigue, diminished appetite, and dark urine. Labwork revealed ALT 2209 U/L, AST 2261 U/L, Tbili 10.6 mg/dL, ALP 232 U/L (LFTs two months prior were all within normal limits). INR was elevated to 1.5, but she had no signs of hepatic encephalopathy. Viral and toxic etiologies of acute hepatitis were thoroughly ruled out. Autoimmune markers, including those specific for autoimmune hepatitis, were negative. Abdominal US and MRCP revealed no abnormalities other than a surgically absent gallbladder. As LFTs continued to rise despite drug withdrawal, she was started on IV steroids. INR further rose to 1.8, and inpatient liver transplant evaluation was initiated. Liver biopsy revealed acute hepatitis with hepatocyte trabecular disarray and parenchymal collapse, perivenular zone 3 confluent necrosis, and mixed inflammatory infiltrate with no lymphocytes/plasma cells identified, suggestive of drug-induced liver injury (DILI). Considering the above workup and timeline of symptoms and lab abnormalities in relation to initiation of Ocrelizumab, DILI secondary to Ocrelizumab was favored as the culprit. The patient's symptoms resolved and LFTs steadily improved with IV steroids. She was discharged on PO steroids with close follow-up and monitoring.
Discussion: We report what is, to our knowledge, the first case of DILI (4+ on DILI Network severity scale) related to Ocrelizumab. As it is a high-efficacy disease-modifying therapy now approved for various subtypes of MS, its use will continue to increase. Therefore, characterizing, monitoring, and reporting Ocrelizumab-related hepatotoxicity is important. However, further studies are needed to shed light on the mechanisms underlying DILI from Ocrelizumab.

Table 1. **Characteristics of polyps in patients with advanced adenoma (n=31) as compared to all other sub-types (n=117)**

		Total		Advanced Adenoma		Non-Advanced Adenoma		p value
		N=148		n=31		n=117		
# Polyps	median(IQR)	2	1-2	2	1-4	1	1-2	< **0.001**
Largest polyp size	mean(SD)	7.0	4.8	14.1	5.1	5.1	2.2	< **0.001**
Polyp Location(s)	n(%)							
Terminal Ileum		1	0.3%	0	0.0%	1	0.3%	0.608
Cecum		35	9.5%	8	25.8%	27	8.0%	0.752
Ascending Colon		38	10.3%	10	32.3%	28	8.3%	0.349
Hepatic Flexure		3	0.8%	2	6.5%	1	0.3%	**0.050**
Transverse Colon		32	8.6%	13	41.9%	19	5.6%	**0.002**
Splenic Flexure		1	0.3%	0	0.0%	1	0.3%	0.608
Descending Colon		17	4.6%	2	6.5%	15	4.4%	0.326
Sigmoid		44	11.9%	9	29.0%	35	10.3%	0.925
Rectum		26	7.0%	3	9.7%	23	6.8%	0.197
Anal Verge		0	0.0%	0	0.0%	0	0.0%	
Surgery	n(%)	3	0.8%	3	9.7%	0	0.0%	< **0.001**
Mortality	n(%)	11	3.0%	2	6.5%	9	2.7%	0.235

S2917 Presidential Poster Award

Acute Alcoholic Hepatitis With Portal Hypertension Complicated by Tuberculous Peritonitis and Ascites

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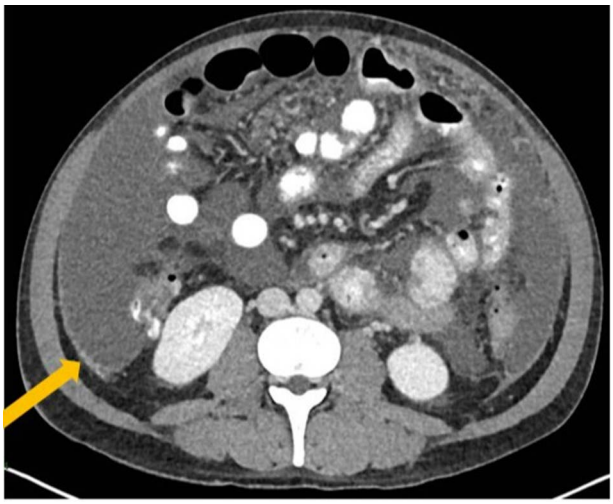
Introduction: Peritonitis related to tuberculosis (TB) infection is rare in the US and normally associated with immunocompromised states. We present a patient found to have TB peritonitis during evaluation of alcoholic hepatitis without evident lung disease.

Case Description/Methods: The patient is a 39 yo male born in Mexico with no medical history who presented with three days of abdominal distention, jaundice, and fevers in the setting of alcohol use disorder. He had no other historic risk factors for TB or cough. Exam was remarkable for a distended abdomen. Laboratory results showed leukocytosis, total bilirubin 3.5 (mg/dL), alkaline phosphatase 295 (U/L), AST 147 (U/L), ALT 62 (U/L), and positive quantiferon gold. Chest x-ray had no pulmonary lesions. Ascites fluid studies were significant for high white count (2214 k/uL) with lymphocytic predominance (91%). The serum ascites albumin gradient was consistent with portal hypertension, and ascites total protein was elevated (3.6 g/dL). Fluid ADA was positive. Cross sectional imaging of the abdomen showed omental caking, peritoneal lining nodularity, and hepatosplenomegaly. Fine needle aspiration of the peritoneum showed necrotizing and non-necrotizing granulomas. Sputum gram stain was negative for acid fast bacilli although sputum and ascites cultures ultimately grew TB (Figure). The patient was started on rifampin, isoniazid, pyrazinamide and ethambutol for TB and abstained from further alcohol use. Three months after discharge, jaundice and ascites had resolved and his liver tests had significantly improved.

Discussion: TB peritonitis makes up 4-10% of extrapulmonary TB infections. Though rare in the US, it has a high mortality rate (50-60%). Diagnosis can be complicated by difficulty isolating the organism and culture results can take weeks. TB peritonitis can be supported by ascites fluid studies with elevated lymphocyte count, total protein, and positive ADA; although for more definitive diagnosis peritoneal biopsy should be considered. This patient represents a unique confluence of portal hypertension related ascites, induced by acute alcoholic hepatitis, along with TB peritonitis. It is possible that transient immunosuppression from alcoholic hepatitis served as a risk factor in reactivation and dissemination of latent TB. An index of suspicion for TB infection, based on his foreign birth, along with his abnormal ascites fluid studies, led to the peritoneal biopsy and ultimate culture diagnosis of his infection that might otherwise have been overlooked.



[2917] **Figure 1.** Nodularity of the peritoneal lining.

S2918

Autoimmune Hepatitis-Like Syndrome After Coronavirus Disease 2019 Vaccine

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Introduction: The coronavirus disease 2019 (COVID-19) pandemic has brought a wave of devastation, resulting in six million deaths worldwide. This has prompted the rapid development of anti-COVID vaccines: mRNA vaccines BNTb262 and mRNA-1273. A wide range of autoimmune diseases are increasingly being reported following COVID-19 vaccination. We describe a rare case of autoimmune hepatitis-like syndrome following the mRNA-1273 SARS-CoV-2 vaccination.
Case Description/Methods: A 56 year-old female received her second dose of the mRNA-1273 SARS-CoV-2 vaccine on the 29th of April 2021. One week post-vaccination, she developed severe fatigue, myalgia, and arthralgia. Subsequently, she noted jaundice, upper abdominal discomfort, and dark urine one month post-vaccination. She had a history of depression for which she was on sertraline 25mg/day. She did not use herbal remedies or alcohol. On examination, she was jaundiced with mild right upper quadrant tenderness. Laboratory results were markedly abnormal: AST of 1377 U/L, ALT of 2035 U/L, alkaline phosphatase of 435 U/L, and bilirubin of 3.8 mg/dL. Further investigation showed a positive 1:320 anti-nuclear antibody. Serology for viral hepatitis and Epstein-Barr virus were negative. Ceruloplasmin and α1-antitrypsin levels were normal. Anti-smooth muscle antibody, anti-liver-kidney microsomal microsome type 1 antibody, and anti-mitochondrial antibody were negative. Abdominal ultrasound showed hepatic steatosis without cholelithiasis or biliary dilatation. A liver biopsy revealed portal, peri-portal, and lobular inflammation consisting of lymphocytic infiltrates, focally prominent plasma cells, and ceroid-laden macrophages. The pathology was compatible with AIH. The patient was started on budesonide 9 mg/day with improvement in liver function tests.
Discussion: Although 5.16 billion people have received at least one dose of a COVID-19 vaccine globally, COVID-19 vaccine-induced AIH remains extremely rare with only 32 cases documented in the literature. While the causality of the vaccine leading to AIH is not established, the absence of hepatotoxic agents, negative viral serology, supportive histology, and response to treatment in our case suggest the association is not a mere coincidence. Clinicians should be vigilant for vaccine-induced AIH in patients who received vaccination and present with jaundice and abdominal pain in the setting of elevated liver enzymes.

S2919

Bile Duct Injury Due to Ketamine Use

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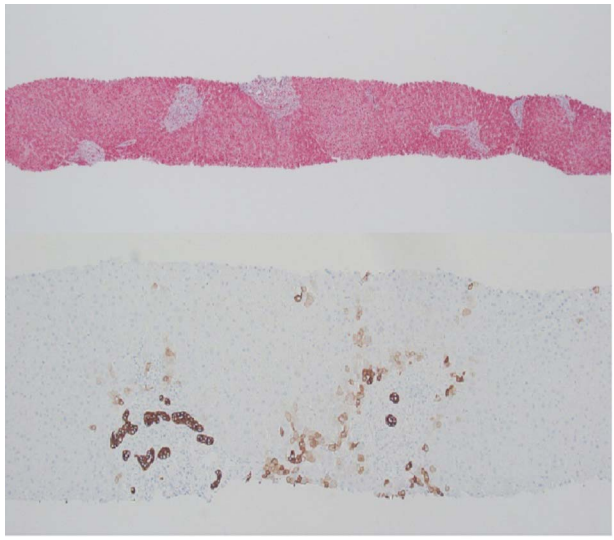
Introduction: Ketamine is used routinely in the hospital for anesthesia, but is also used recreationally for its hallucinogenic and dissociative side effects. Chronic recreational use has been linked to bile duct damage and ulcerative cystitis. We report a case of ketamine induced sclerosing cholangiopathy.
Case Description/Methods: A 42-year-old Cantonese woman with past medical history of bilateral hydronephrosis with stents leading to chronic kidney disease presented with abdominal pain associated with abnormal transaminases. Alkaline phosphatase was high at 1,017 IU/L with gamma glutamyl transferase of 2,310 IU/L with normal bilirubin. Abdominal imaging showed diffuse dilatation of the common bile duct. Viral serologies and anti-mitochondrial antibody were negative. She reported chronically elevated liver tests of unknown etiology. Social history was notable for prior alcohol abuse. She denied family history of liver diseases. Her pain resolved and plan was for outpatient follow-up, but she re-presented with sepsis with a rise in bilirubin to 2.9 mg/dL (Table 1). Given prior abnormal imaging, a magnetic resonance cholangiopancreatography was obtained, which showed increased intra- and extrahepatic bile duct dilation with irregular appearance of the central intrahepatic ducts with possible stricture. Differential diagnoses were recurrent pyogenic cholangitis, sclerosing cholangitis, and IgG4 disease. Her IgG level was high at 2,779 mg/dL with IgG4 elevated at 213 mg/dL. Liver biopsy showed chronic cholestatic liver injury consistent with sclerosing cholangitis (Image 1). IgG4 stain was negative. Upon further investigation, patient admitted to at least a decade of daily ketamine use, which was likely the culprit of both her kidney and liver diseases.
Discussion: Due to her age, race, gender and rarity of ketamine induced cholangiopathy, she was not directly asked about ketamine use. Since the early 2000s, ketamine has emerged as the illicit drug of choice in Hong Kong and has seen increased use throughout Asia. The biliary damage from ketamine has been studied by a Chinese group that found 62% of the 257 chronic ketamine users had biliary tract anomalies. The greater the alkaline phosphatase, the higher the likelihood of finding biliary tract anomalies on imaging. Ketamine cessation has resulted in normalization of liver tests and imaging, however worsening cholangiopathy has been seen despite abstinence. In patients who present with unexplained cholangiopathy, chronic ketamine use should be considered.



[2919] **Figure 1.** Liver biopsy: A mild lymphoplasmacytic inflammation is noted in some triads with focal and mild interface activity. The bile ducts are injured (irregular contour, unevenly distributed nuclei, intracytoplasmic vacuoles) with focal periportal cholestasis. Immunohistochemical stain for keratin 7 confirms predominantly periportal cholestatic hepatocytes. Keratin 19 shows widespread loss of canals of Hering and focal bile duct loss. Rhodanine stain confirms periportal hepatocellular copper accumulation compatible with a chronic cholestatic process.

Table 1. Patient's hepatic function panels during initial encounter, subsequent encounter, and at least 6 months after subsequent encounter

Liver Tests	Reference Range & Units	Initial Hospitalization	Subsequent Hospitalization	Most Recent Liver Tests
Total bilirubin	0.2 – 1.2 mg/dL	0.8	2.9	4.5
Direct bilirubin	0 – 0.5 mg/dL	0.7	1.8	3.3
Aspartate aminotransferase	5 – 34 IU/L	75	104	88
Alanine transaminase	0 – 37 IU/L	74	129	91
Alkaline phosphatase	40 – 150 IU/L	1017	1106	845
Gamma glutamyl transferase	12 – 43 IU/L	2310	2516	1897

S2920

New Drugs and New Toxicities: Liraglutide-Induced Liver Injury

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Introduction: Liraglutide is a glucagon-like peptide-1 (GLP-1) receptor agonist. It has demonstrated efficacy as an antidiabetic and anti-obesity drug. However, several common adverse events have also been reported, including nausea, vomiting, loose stools, and rarely pancreatitis. To our knowledge, this report represents only the third case of drug-induced liver injury (DILI) after the use of liraglutide.
Case Description/Methods: A 43-year-old obese female was initiated on liraglutide 3 months ago for poorly controlled diabetes mellitus (HbA1c: 10.7%). She developed dull, right upper abdominal pain for 12 days. It was associated with fatigue and appetite loss. The patient was a nonsmoker, nonalcoholic, and drug-free. She did not take any over-the-counter or herbal supplements. Before liraglutide initiation, her LFTs were within normal ranges. Physical examination revealed tenderness in the upper abdomen. Laboratory studies revealed ALT 1837 U/L, AST 1062 U/L, ALP 373 U/L, total bilirubin 1.8 mg/ dL, and INR 1.0. The R ratio was 10.73, indicating a category 2 – moderate hepatocellular injury. Ultrasonography of the abdomen revealed fatty changes in the liver, but ruled out biliary abnormalities. The workup for infectious hepatitis (A, B, C, D, and E), cytomegalovirus, and Epstein-Barr virus were all negative. The investigations for autoimmune disorders, hemochromatosis, Wilson's disease, and alpha-1 antitrypsin deficiency were also negative. After exclusion of probable causes, the patient was diagnosed with liraglutide-induced DILI. Liraglutide was immediately discontinued and N-acetylcysteine was administered. The clinical response was excellent, with resolution of symptoms in 4 days. Her LFTs also showed a downward trend. At discharge, her ALT 586 U/L, AST 115 U/L, ALP 258 U/L, and total bilirubin was 1.1 mg/dL. At the follow-up visit after 3 months, her LFTs were within normal limits.
Discussion: Liraglutide-induced DILI remains an extremely rare adverse drug reaction. In our patient, the temporal relationship between DILI onset with liraglutide initiation and resolution of DILI with drug cessation indicated liraglutide-related DILI. On literature review, we found only 2 previously reported case reports. As in our case, the clinical outcomes in both cases were good and the patients recovered without any complications. Liraglutide is a safe drug, but patients should be monitored for newer adverse events like DILI. Prompt detection and drug cessation may spare patients from potential morbidity.

S2921

Flood Syndrome: A Challenge for the Clinician

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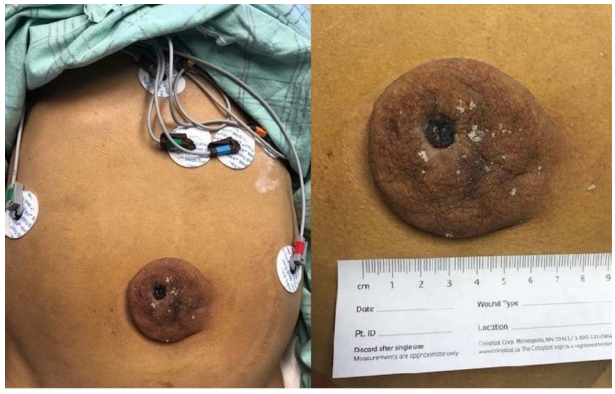
Introduction: Flood syndrome is a rare life-threatening complication of spontaneous paracentesis from a ruptured umbilical hernia typically in the setting of refractory ascites and end-stage liver cirrhosis. While conservative management is associated with higher mortality compared to surgical management, herniorrhaphy in patients with end-stage liver cirrhosis remains difficult to plan due to a high-risk of morbidity and mortality. We present a case of flood syndrome and our multidisciplinary approach for management.
Case Description/Methods: A 59-year-old male, with decompensated alcoholic cirrhosis including esophageal varices, recurrent ascites, and hepatocellular carcinoma, presented with hepatic encephalopathy and a ruptured umbilical hernia leaking ascites. (Figure) He was hemodynamically stable and afebrile. His MELD-Na score on admission was 30. A CT abdomen was negative for incarcerated hernia or obstruction. After obtaining cultures he was started on cefepime and vancomycin. Additionally, he received albumin 25g every 6 hours, lactulose and rifaximin. Medical and surgical planning included evaluation by gastroenterology, infectious disease, and surgery for medical optimization with the aim of pursuing elective repair when the patient was at his lowest MELD score. Unfortunately, he deteriorated clinically with worsening encephalopathy and hemodynamic instability. Ultimately, he was considered too unstable for herniorrhaphy. His hospital course was complicated by *Staphylococcus lugdunensis* and vancomycin-resistant *Enterococcus faecium* peritonitis and sepsis despite multiple antimicrobials. He eventually developed a massive rectal bleed and transitioned to comfort measures per family wishes.
Discussion: Flood syndrome has a poor prognosis with a high mortality rate. Patients with end-stage liver disease and recurrent ascites are at higher risk due to increased intra-abdominal pressure, weakened abdominal muscle and fascia, and malnutrition. There is no standard of care for management of flood syndrome, but literature favors early surgical intervention. Our case shows the difficulty and complexity of managing these patients. Despite utilization of a multidisciplinary approach to care for our patient, the severity of our patient's liver disease and his frailty made him ineligible for surgical intervention. Early surgical intervention to prevent rupture of these umbilical hernias may ensure the best chance at positive patient outcomes.



[2921] **Figure 1.** A. Large 6.5cm umbilical hernia.

S2922

Non Cirrhotic Portal Hypertension as a Long-Term Consequence of Oxaliplatin

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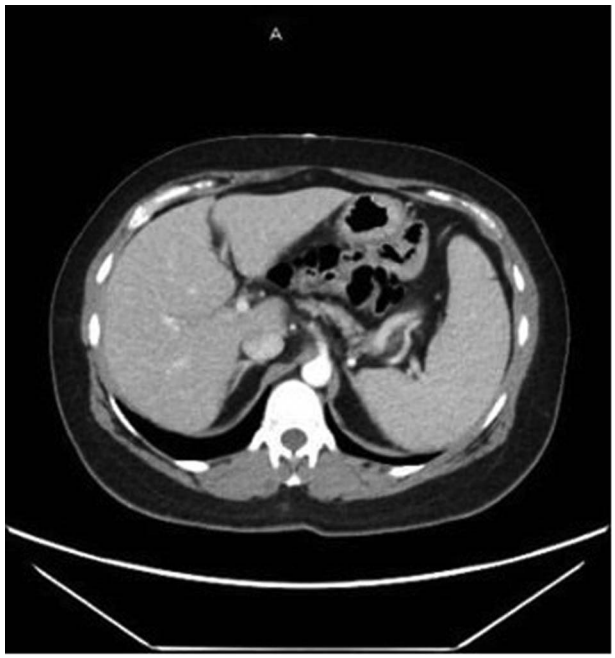
Introduction: Oxaliplatin, a platinum-containing alkylating drug used to treat advanced colorectal cancer, is known to cause hepatotoxicity, a rare potentially fatal long-term side effect. Here, we present a case of Non Cirrhotic Portal Hypertension caused by oxaliplatin based regimen.
Case Description/Methods: A 46 -year-old female presented to the emergency department with complaints of coffee-ground emesis, melena and one episode of syncope. No reported use of NSAIDs, excessive alcohol or anticoagulants. Past history is significant for Lynch Syndrome, adenocarcinoma of colon status post right hemicolectomy and adjuvant chemotherapy comprising leucovorin calcium(folinic acid), fluorouracil, and oxaliplatin (FOLFOX), cholecystectomy and appendectomy. Family history was significant for colon cancer in father. She was tachycardic and hypotensive, physical examination revealed guaiac positive black stool per rectum. Lab results showed low hemoglobin of 9.5 g/dL, elevated white blood cell count of 14.4 10*3/uL, deranged coagulation profile, and elevated BUN of 38 mg/dL with normal creatinine, liver, and cardiac enzymes. Her chest x-ray was normal, contrast CT abdomen pelvis revealed significant splenomegaly. (Figure) Endoscopy identified two Grade II esophageal varices status post bands, widened Schatzki ring in the distal esophagus, and benign non-bleeding gastric ulcers. Since her comprehensive liver workup came out negative, a liver biopsy was performed, demonstrating changes of nodular regenerative hyperplasia, leading to noncirrhotic portal hypertension. There is no significant portal or lobular inflammation or fibrosis, ruling out cirrhosis as a cause.
Discussion: Oxaliplatin is known to cause Nodular Regenerative Hyperplasia (NRH) leading to Non Cirrhotic Portal Hypertension (NCPH). NRH has been linked to drugs as well as rheumatoid arthritis, myeloproliferative disorder, and vasculitis. Changes of sinusoidal obstruction syndrome (SOS) and local disturbances in hepatic perfusion have been suggested as a cause of NRH in patients receiving oxaliplatin-based chemotherapy. Our patient was treated with Oxaliplatin for colon cancer, which led to the development of NRH-related NCPH, which manifested as esophageal varices associated with splenomegaly. There have been reports that Bevacizumab may help prevent NRH, which should be investigated further. More research is needed to determine the mortality differences between chemo regimens with and without Oxaliplatin.



[2922] **Figure 1.** Significant splenomegaly on CT scan.

S2923

De Novo Autoimmune Hepatitis Following COVID Vaccination: A Case Series

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Introduction: We present a case series of patients with de novo autoimmune hepatitis following COVID vaccination.
Case Description/Methods: A 68-year-old female with a past medical history of GERD and diverticulitis presents with the sudden onset of dark urine three days after receiving the second Moderna COVID vaccine. This is followed by right upper quadrant pain, jaundice, nausea, vomiting, and acholic stools. She denies any other associated symptoms. She endorses a family history of rheumatoid arthritis but denies any family history of liver disease/cancer. The patient is admitted and is found to have AST: 1,650, ALT: 1,604, alkaline phosphatase: 225, and total bilirubin: 4.1. Labs include positive IgG: 2,160 and positive ANA. Liver biopsy reveals intense lymphocytic, plasmacellular, eosinophilic, and neutrophilic hepatitis with bridging necrosis. Trichrome stain shows portal/periportal fibrosis. These findings are consistent with autoimmune hepatitis, and she is subsequently started on mycophenolate mofetil (MMF) and prednisone with improvement in her liver enzymes and symptoms. A 43-year-old female with a past medical history of keratoconus presents to the clinic with painless jaundice for one week. She endorses feeling fatigued as well for the past three months after receiving her second Moderna COVID vaccine. She denies any other associated symptoms. She endorses occasional alcohol use and denies any family history of liver disease/cancer or autoimmune disease. Her home medications include oral contraceptives and ibuprofen. Laboratory evaluation is remarkable for AST: 579, ALT: 800, alkaline phosphatase: 130, and total bilirubin: 6.7. MRI/MRCP is unremarkable for biliary obstruction or cirrhosis. She is found to have anti-smooth muscle antibody titers of 1:160 and IgG of 1,629. Liver biopsy reveals lymphoplasmacytic infiltrate with eosinophils, interface hepatitis, triaditis, necroinflammation with necrosis, and periportal septate fibrosis. The patient is diagnosed with autoimmune hepatitis and is started on MMF and prednisone with clinical improvement.
Discussion: These cases demonstrate that COVID vaccination may play a role in inciting de novo autoimmune hepatitis in some patients. This can be a challenging situation for many clinicians to navigate, as COVID remains a significant threat to patients' health. Patients with autoimmune risk factors may benefit from closer laboratory evaluation and monitoring for any symptoms surrounding COVID vaccination.

S2924

Hidden in Plain Sight: A Rare Case of Hepatosplenic T-Cell Lymphoma

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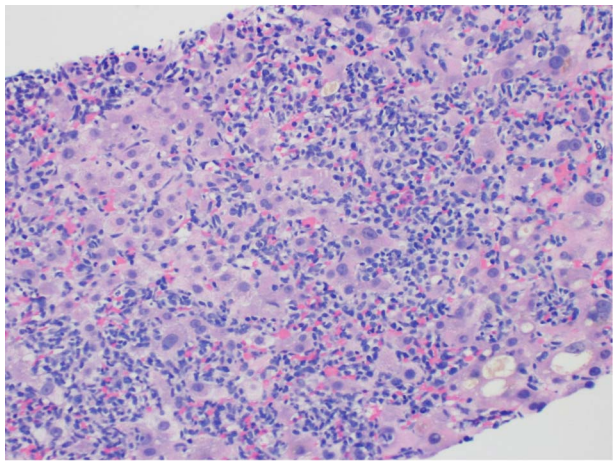
Introduction: Hepatosplenic T-cell lymphoma (HSTCL) is a highly aggressive subgroup of non-Hodgkin's lymphomas affecting 1.4% of the world's population. It is characterized by clonal expansion and infiltration of cytotoxic γδ T-cells typically in young male adults, presenting with constitutional B symptoms, cytopenias, elevated liver enzymes, hepatosplenomegaly, and lack of lymphadenopathy. We present a rare case of an αβ variant of HSTCL in an elderly woman.
Case Description/Methods: A 79-year-old female with type 2 diabetes mellitus and hyperlipidemia presented with generalized weakness and weight loss. Home medications included atorvastatin and metformin. Physical exam did not show stigmata of chronic liver disease. Initial laboratory work-up was notable for aspartate aminotransaminase (AST) 860 U/L, alanine aminotransaminase (ALT) 938 U/L, alkaline phosphatase (ALP) 416 U/L, total bilirubin 5.8 mg/dl with a direct predominance, international normalized ratio (INR) 1.1, albumin 4.1 g/dL, and platelets 129 10*3/mcL. Serologic workup was negative for common and rare viral hepatitis, autoimmune, toxic and metabolic etiologies. MRI abdomen with magnetic resonance cholangiopancreatography (MRCP) revealed patent hepatic vasculature with mild hepatosplenomegaly. Liver enzymes continued to worsen, with a peak AST 1419 U/L, ALT 1048 U/L, and total bilirubin 24.5 mg/dl. Liver biopsy revealed an atypical T-cell infiltrate with a high Ki-67 index negative for CD56 and CD5. Immunohistochemistry showed alpha beta positivity (Figure 1). Molecular testing confirmed the presence of monoclonal T-cell receptor (TCR) beta and gamma populations highly suggestive of hepatosplenic T-cell lymphoma with αβ phenotype.
Discussion: Since its discovery in 1990, only several hundred cases have been described for the γδ variant of HSTCL and less than 40 cases have been reported for the αβ variant. The median age of diagnosis is 34 for γδ cases. The αβ variant can be seen in women over 50 years of age and has been associated with worse prognosis. Most cases occur de novo. Approximately 20% of cases arise in a setting of immunosuppression, often associated with autoimmune disorders and inflammatory bowel disease. Outcomes are poor with a 5-year-suvival of < 10% without bone marrow transplant. Albeit rare and difficult to diagnose, HSTCL should be considered in the differential for elevated liver enzymes, as early diagnosis is crucial for imminent induction chemotherapy.



[2924] **Figure 1.** Liver biopsy showing infiltrate of atypical T-cells with high Ki67 index and αβ positivity.

S2925

Fibrosing Cholestatic Hepatitis Masquerading Acute Cellular Rejection in HCV-Positive Donor Liver Graft

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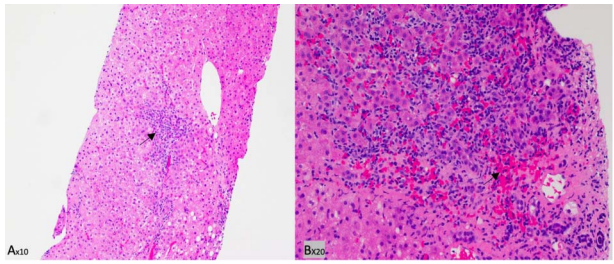
Introduction: Direct-acting antiviral (DAA) therapy has made it possible to include HCV+ donors to the organ donor pool. Cholestatic hepatitis post-transplant is quite rare with HCV viremia. We present a rare case of fibrosing cholestatic hepatitis appearing in an HCV+ donor liver.
Case Description/Methods: A 48-year-old female with history of end stage liver disease due to alcohol abuse underwent deceased donor, orthotopic liver transplant on 2/22/22 (HCV NAT +). Patient did well post operatively with no complications. However, she continued to have up-trending liver enzymes along with elevated bilirubin without any ductal abnormalities on imaging. She subsequently underwent liver biopsy on 3/02/22, showing sinusoidal pressure of 8mmHg indicating no obstruction, though the tissue histology revealed moderate acute cellular rejection, for which she received pulse dose solumedrol with improving LFTs. However, her LFTs started to rise again in a cholestatic pattern and therefore she underwent a repeat liver biopsy on 3/8/2022 which revealed no evidence of rejection but showed bile duct injury with cholestasis. She was started on ursodiol as a result. Considering the donor was HCV+, Hepatitis C RNA, PCR was done on 3/1/22 which was >100000000 IU/mL, at this point patient was started on EPCLUSA (sofosbuvir-velpatasvir). Her repeat viral PCR subsequently showed continued improvement and became undetectable on 4/27/2022. Of note, her LFTs showed significant improvement with the continued use of antiviral therapy. Patient remained stable throughout her post operative course and did undergo repeat liver biopsy twice thereafter on 3/22/22 and 6/8/22 showing no acute cellular rejection, though it showed bile ductular proliferation with mixed inflammatory infiltrate. Table-1 shows the trend in LFTs, and HCV viral load seen post operatively.
Discussion: DAA has proven to be pivotal in helping meet the ever-growing demand for organ donors. The safety of HCV+ liver donor is well established, though developing fibrosing cholestatic hepatitis 2-4 weeks post-transplant is a rare possibility. Our case (see Figure-1), shows portal inflammation and cholestasis; is a solid example of such pathology. In the absence of anatomic complication and acute cellular rejection, prompt testing for viral load should be employed. Thus timely initiation of DAA can lead to improved clinical outcomes as seen above.Further long-term studies are needed to establish treatment protocols in patients with cholestatic type injury in seropositive donors.



[2925] **Figure 1.** (H&E) A: Arrow pointing at portal tract and ductular reaction which is not diagnostic of acute cellular rejection. B: Arrow pointing towards pigment deposition showing intrahepatic cholestasis.

Table 1. Trend in LFTs and HCV RNA viral load over hospital course following deceased donor HCV+ OLT

Date	Hepatitis C Virus RNA PCR IU/mL	Total Bilirubin mg/dL	Alkaline Phosphatase U/L	AST U/L	ALT U/L
3/1/20222	100000000	13	480	110	101
3/7/2022	2310000	5.8	438	91	80
3/28/2022	248	2.4	285	132	176
4/27/2022	0	0.7	80	23	17

S2926

Hepatic Steatosis and Acute Liver Injury in Chronic Arsenic Exposure

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Introduction: Arsenic toxicity may not be apparent on initial presentation given its myriad of effects across many body systems and its rarity in developed countries.
Case Description/Methods: A 29-year-old woman with history of gastroesophageal reflux disease, irritable bowel syndrome, gastroparesis, post-traumatic stress disorder, obsessive-compulsive disorder, and generalized anxiety disorder presented with abdominal pain, nausea, and vomiting. Her recent medical history included 7 months of progressive sensorimotor polyneuropathy, bowel and bladder incontinence, vision decline, skin rash and flaking, poor oral intake with 25% weight loss, and recent hospitalization for stress-induced cardiomyopathy and ventricular tachycardia. She was found to have coagulopathy, hemolytic anemia, and elevated transaminases, alkaline phosphatase, direct and indirect bilirubin, and serum ammonia. She developed encephalopathy with rising ammonia requiring scavenger therapy. She underwent diagnostic evaluation for alcohol-related, metabolic, inflammatory, and vascular causes of liver disease. Liver biopsy revealed non-cirrhotic portal fibrosis, severe and mostly macrovesicular steatosis, ductular proliferation, and canalicular cholestasis. Initial labs showed high urine orotic acid and low serum citrulline, so empiric treatment for ornithine transcarbamylase deficiency was initially given. Whole exome sequencing was negative. Urine and serum heavy metal testing was negative. However, arsenic was highly elevated in hair and nail samples, most consistent with chronic arsenic exposure.
Discussion: We present a case of hepatic steatosis and acute liver injury in chronic arsenic exposure. Recent work has associated arsenic toxicity with non-cirrhotic portal fibrosis and elevated risk of non-alcoholic fatty liver disease in humans, previously seen in mice.[1] In this patient, arsenic exposure may explain the hepatic steatosis, either directly or indirectly via rapid weight loss. Arsenic toxicity also explains the hepatic injury and portal fibrosis, encephalopathy, polyneuropathy, hair loss, and skin changes. Recognizing metal toxicity as a cause of liver injury was crucial, as arsenic testing dramatically altered our course of management and patient care.

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S2927

Persistent Spontaneous Bacterial Peritonitis in a Patient With Cardiogenic Ascites

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Introduction: Ascites is defined as a collection of more than 25mL of fluid in the peritoneal cavity, most commonly caused by liver cirrhosis. Cardiogenic ascites from heart failure contributes to 5% of cases. Spontaneous bacterial peritonitis (SBP) is a common complication of ascites, defined as an infection of ascitic fluid in the absence of a surgically treatable source. This is more commonly associated with hepatic cirrhosis. Here we present a case of SBP in a patient with cardiogenic ascites.
Case Description/Methods: Patient is a 37-year-old male with right sided heart failure secondary to bronchopulmonary dysplasia with no history of liver disease. The patient was found to have dyspnea and anasarca, so he was admitted for heart failure exacerbation. CT of the abdomen showed large volume ascites and hepatomegaly. While being diuresed, 5L of ascitic fluid was removed via paracentesis. Analysis revealed a serum albumin-ascites gradient (SAAG) of 0.7, protein 4.5, absolute neutrophil count (ANC) 948, and negative culture. The patient was then started on ceftriaxone for SBP. An additional 4.5L was removed via paracentesis 3 days later due to worsening ascites. Analysis at that time showed SAAG 0.6, protein 4.2, and ANC 4,808. No cultures were reported. Antibiotics were escalated to piperacillin-tazobactam due to worsening ANC. A third paracentesis was performed 3 days later after reaccumulation of ascites (3.2L) and to assess therapeutic response. Peritoneal fluid analysis revealed SAAG 0.8, protein 3.7, ANC 2,931, and negative culture. At this point, the infectious disease team was consulted, recommending discontinuation of antibiotics and close monitoring. Patient was discharged 6 days later as he was afebrile and without leukocytosis.
Discussion: The pathophysiology of SBP is thought to be related to bacterial translocation and impaired immunity, specifically deficiencies in complement protein neutrophil function. This is reflected by low ascites protein. However, SBP is exceedingly rare in cardiogenic ascites with only 8 cases reported. This is likely secondary to the high protein and opsonic character in this type of ascites, developing from elevated intrahepatic pressures and congestive hepatopathy. This composition of ascites fluid yields antimicrobial activity similar to normal peritoneal fluid. Perhaps this was the reason for the patient's elevated ANC despite negative cultures. Future studies could focus on the role of antibiotics and prophylaxis in these patients.

S2928

A Novel Case of Drug-Induced Acute Liver Injury Following Intravenous Iron Therapy

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Introduction: Acute Liver injury attributed to dose-related acute iron poisoning has been a phenomenon with limited description in medical literature. Intravenously high doses of iron have shown serious liver toxicity, however cases have been predominantly shown in children. Here we present an adult case of drug induced liver injury after intravenous use of IV iron.
Case Description/Methods: An 86-year-old male with a past medical history of Stage I Colon Cancer status post resection in 1986, Heart Failure with Reduced Ejection Fraction, Atrial Fibrillation on Coumadin, and Coronary Artery Disease who presented to the ED for dyspnea on exertion and melena for one month with concern for hemodynamically unstable GI bleed. Vitals revealed mild hypotension. Labs were significant for hemoglobin 7.2 g/dL, INR 4.5. GI was consulted regarding this patient's acute GI bleed, and they followed the patient for the entirety of his admission. The patient was started on a PPI drip and frequent CBC monitoring and received a total of two transfusions as well as IV iron for a total of five days. On EGD, mild antral gastritis was identified however with biopsy taken to rule out H. pylori being negative. His hemoglobin and hematocrit would improve but his hospitalization would be complicated with progressive waxing and waning lethargy and worsening transaminitis, with maximal AST/ALT values being 2638/1929 (on admission AST 45, ALT 36). Iron studies were remarkable for elevated Iron 195, Iron Saturation 53%, and Ferritin elevated at 553. CT abdomen and pelvis revealed no acute process, only an 8 mm focus of enhancement in the liver dome. Autoimmune, metabolic and infectious including hepatitis panel workup were negative. After Intravenous iron was discontinued, his liver function tests began to improve and normalize within two weeks.
Discussion: Iron is both an essential micronutrient and toxic to cellular processes in excess. The proposed mechanism of iron-toxicity induced tissue damage is free radical production and lipid per-oxidation. Systemic toxicity usually occurs from injury to the Liver and the cardiovascular system with cause of death from iron poisoning usually being shock or liver failure. Onset of Hepatotoxicity usually occurs within 12-96 hours of oral ingestion of iron however, intravenous iron overdoses are rare and limitedly reported in medical literature. We presented a case of a patient with worsening transaminitis secondary to IV Iron-induced acute liver injury.

S2929

AST > 7500, ALT > 4500: Can It Be Anaplasmosis?

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Introduction: Human granulocytic anaplasmosis (HGA) is a tick-borne rickettsial disease caused by Anaplasma phagocytophilum. The clinical presentation of HGA is nonspecific with symptoms such as fever, chills, headache, nausea and fatigue. HGA is known to present with mild transaminases, severe transaminases are rare. We present a case of a 41-year female with severe transaminases > 4500 due to anaplasmosis.
Case Description/Methods: A 41-year old female with no comorbidities from upstate New York presented to the hospital with lightheadedness associated with nausea, vomiting, fever and right upper quadrant abdominal pain. She was hemodynamically stable. On exam she had mild tenderness in RUQ, otherwise normal exam findings. Lab revealed normal CBC but AST >7500, ALT >4500, Alk Phos of 99, total bilirubin of 3.1, PT/INR 28.9/2.47. Abdominal ultrasound and CT abdomen with IV contrast showed fatty liver otherwise no abnormality. Serum acetaminophen level was negative. Viral hepatitis panel, herpes simplex, autoimmune panel were unremarkable. Hemochromatosis and Wilson's disease were ruled out. She was treated conservatively without any significant improvement. On day 3, she mentioned that she might have had a tick bite on camping few weeks ago. Tick-borne panel came back positive for anti- anaplasmosma phagocytophilum antibodies of 1:1024. Patient was started on doxycycline and discharged on day 7 with significant improvement of her symptoms. Liver functions normalized in 6 weeks.
Discussion: Tick borne infections are common zoonotic diseases of significant global morbidity burden. HGA is a black legged deer tick transmitted infection caused by Anaplasma phagocytophilum. First reported in 1994, HGA is now endemic in the Northeast and upper Midwest states of the USA. CDC notes that up to 15% of cases may be subclinical. Clinically, HGA presents with nonspecific viral symptoms which usually resolve spontaneously. Up to 70% of HGA may have elevated transaminases in 100s which have a direct correlation with disease severity and organ damage. According to the literature reviewed, no cases of HGA with transaminases >1000 without causes such as acetaminophen toxicity, herpes hepatitis and shock have been reported. Our case highlights an atypical occurrence of severe transaminases due to HGA. Thus, HGA should be considered as a possible differential for severe transaminases in patients with risk factors.

S2930

Calciphylaxis Due to Alcoholic Liver Disease

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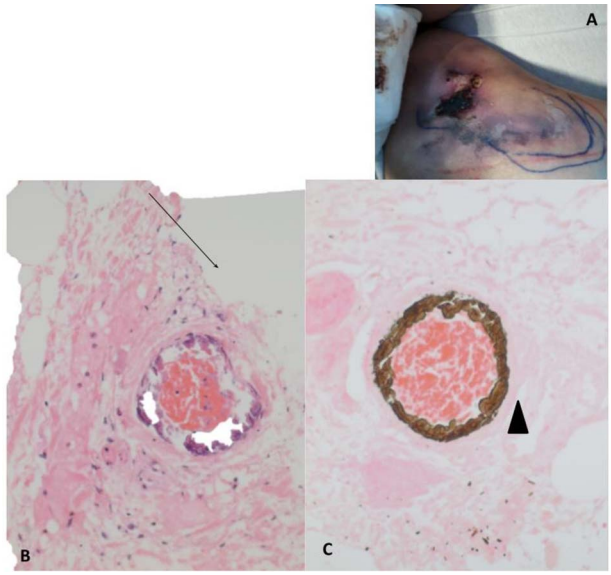
Introduction: Advanced liver disease often has multiorgan involvement, including dermatological findings. These range from spider angiomas to palmar erythema. Here we present a case of calciphylaxis as a manifestation of decompensated alcoholic liver disease.
Case Description/Methods: A 38 year-old female with decompensated alcoholic cirrhosis (portal vein thrombosis, ascites, hepatic encephalopathy, and hepatic hydrothorax) reported to the emergency room complaining of new right thigh wounds. Her vital signs were unremarkable and her exam demonstrated ascites and bilateral lower extremity edema with right medial thigh erythema, induration, and superficial ulceration (Figure 1). Labs showed sodium 125 mEq/L, Creatinine 0.95 mg/dL, WBC 16.2 x10^9, and total bilirubin 1.6 mg/dL. CT of her thigh demonstrated bilateral, symmetric subcutaneous induration consistent with anasarca and possible superimposed cellulitis. She was admitted and treated with IV antibiotics. As the lesions became duskier and more painful dermatology was consulted who initially were concerned for a vasculopathic process versus calciphylaxis. The patient was placed on empiric treatment for calciphylaxis with sodium thiosulfate. Skin biopsies were performed and showed non-uremic calciphylaxis (Figure 2). The patient then developed an AKI thought to be from hepato-renal syndrome and was trialed on albumin, midodrine, and octreotide with no improvement in renal function. She then developed acidosis, eventually requiring a bicarb drip, and her wounds continued to progress (Figure 1). When she started becoming repeatedly hypotensive her family transitioned her to comfort care and she was discharged to hospice.
Discussion: Calciphylaxis is a rare disease that was first described in 1961 by Selye et al and is largely thought to be related to uremia. Here we describe a case of calciphylaxis in a patient with alcoholic liver disease and initial normal renal function. Our review of the literature found fewer than 11 case reports of calciphylaxis associated with liver disease. The mechanism in these cases appears to be poorly understood though is believed to be connected to protein C and protein S deficiency. As in those with advanced renal disease, calciphylaxis has a poor prognosis with high mortality often reported as greater than 50% even despite treatment. Thus it is important that clinicians recognize its association with alcoholic liver disease to aid in diagnosis and ensure prompt initiation of treatment.



[2930] **Figure 1.** A: Right medial thigh wound B: High power of calcium deposits and thrombus (arrow) within the subcutaneous fat C: corresponding von kossa stain (arrowhead).

S2931

Benign Recurrent Intrahepatic Cholestasis (BRIC) Managed With Plasmapheresis

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Introduction: Benign recurrent intrahepatic cholestasis (BRIC) and Progressive Familial Intrahepatic Cholestasis (PFIC) are rare conditions of defective bile salt transport, each with an estimated prevalence of 1/50,000-100,000. Symptoms include episodic jaundice and severe pruritus. Pruritus management is difficult due to limited clinical experience and disease rarity. Treatment typically focuses on symptomatic relief of pruritus. We present a case of BRIC refractory to conventional medical treatment managed with plasmapheresis.
Case Description/Methods: A 32-year-old woman with a past medical history of BRIC type 1 diagnosed at age 14 with ATP8B1 mutation with clinical concern for overlapping PFIC type 1 presented with intractable pruritus and jaundice. She experienced seven episodes of similar presentation previously controlled with conventional medical therapy. Physical exam was remarkable for jaundice and diffuse excoriations. Pertinent lab values are shown in Table 1. Liver ultrasound was unremarkable and negative for acute biliary pathology. Liver biopsy demonstrated panlobular hepatocanalicular cholestasis with mild perivenous, perisinusoidal, and periportal fibrosis, compatible with chronically impaired biliary drainage. This episode was refractory to conventional treatment with ursodiol, cholestyramine, diphenhydramine, sertraline, rifampin, naltrexone, and odevixibat. Five sessions of plasmapheresis with albumin were performed every other day with improvement in pruritus and jaundice.
Discussion: BRIC and PFIC are on opposite ends of a clinical spectrum and overlap can be present. Subtype 1 is an autosomal recessive mutation within the ATP8B1 gene, encoding phospholipid flippase. The defect results in impaired bile salt transport. Patients with PFIC may present in the neonatal or early childhood period and usually progress to end-stage liver disease, while the typical patient with BRIC presents later with episodic cholestasis without hepatic fibrosis. In our case, the patient's symptoms were refractory to multiple lines of conventional therapy but responsive to plasmapheresis, which is presumed to remove circulating cholestatic pruritogens from the blood. Subjective assessment of symptom relief is recommended when evaluating treatment response, as serum bile acid levels may not correlate with cholestatic symptoms. Our case highlights the potential usefulness of this promising modality in improving pruritus and shortening the duration of attacks in BRIC/PFIC patients.

Table 1. Labs on admission versus post-plasmapheresis sessions

	Labs on admission	Labs post-plasmapheresis
Total bilirubin (mg/dL)	30.6	15.1
Direct bilirubin (mg/dL)	25.6	11.6
AST (U/L)	39	28
ALT (U/L)	20	10
Alkaline phosphastase (U/L)	184	67
GGT (U/L)	18	—
Bile acids (umol/L)	388.5	432

S2932

Autoimmune Hepatitis, SLE, and Leukocytoclastic Vasculitis Following the Moderna COVID Vaccine

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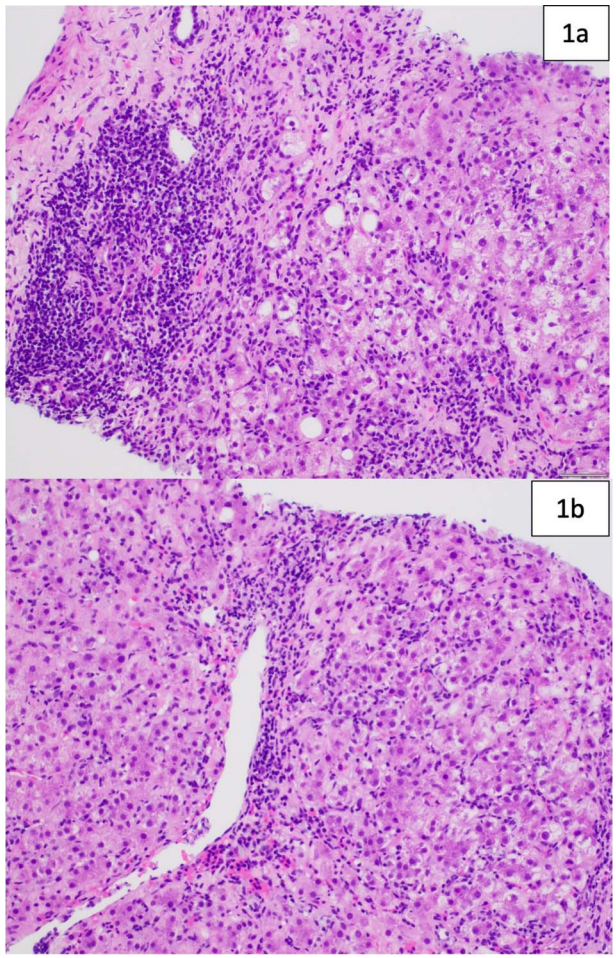
Introduction: There have been a small number of case reports of patients who developed autoimmune hepatitis following vaccination against COVID. (1) The following case report details the development of multiple autoimmune conditions, including AIH, SLE, and leukocytoclastic vasculitis in a patient shortly after she received the Moderna vaccine against COVID.
Case Description/Methods: A 59-year-old woman with a history of morbid obesity, CAD, HTN, HLD, and prior cholecystectomy presented to the emergency department complaining of petechial rash and fatigue that developed 10 days after receiving the Moderna COVID vaccine. Physical exam was notable only for a petechial rash involving both legs. A biopsy of the rash revealed LCV. She was found to have abnormal liver enzymes with ALT 259 U/L, AST 382 U/L, TBili 1.9 mg/dL, and Alk Phos 224 U/L. Further testing revealed an ANA with 1:1280 titer and elevated IgG level at 2,815 mg/dL. RUQ ultrasound revealed no evidence of biliary obstruction and mild CBD dilation due to cholecystectomy. Viral hepatitis, CMV, celiac panel, ceruloplasmin, ferritin, AMA, ASMA, and alpha-1 antitrypsin testing were all negative. Both her rash and liver injury improved with steroid administration but worsened with tapering. She then underwent liver biopsy, which revealed lymphoplasmacytic infiltrate and interface hepatitis that were compatible with autoimmune hepatitis. She was also found to have low complement and positive anti-dsDNA, and was diagnosed with SLE. Her liver injury improved with IV steroids, and she was then transitioned to azathioprine and tapered off of steroids without further flares (Figure).
Discussion: The diagnosis of autoimmune hepatitis was not able to be confirmed until the liver biopsy. Our patient received 7 points for the IAHG AIH diagnostic criteria, indicating definitive AIH. This case report adds to this small but growing series of patients who developed autoimmune hepatitis after receiving a COVID-19 vaccine. Because of vaccine hesitancy, providers must be able to have an informed discussion with patients about the benefits and risks. This case highlights the need for greater awareness on the part of clinicians of this rare complication.



[2932] **Figure 1.** 1a: Image shows moderate portal-based chronic inflammation with conspicuous plasma cells (left side of the image) and at least moderate interface activity (right side of the image). Apoptotic hepatocytes are also identified. 1b: Inflammation rich in plasma cells, lymphocytes, and histiocytes extends throughout the lobule with areas of centrivenular accentuation. Increased fibrosis is also evident (not shown here). The overall findings are compatible with autoimmune hepatitis in the clinical context.

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1. Bril F. Autoimmune hepatitis developing after coronavirus disease 2019 (COVID-19) vaccine: One or even several swallows do not make a summer [published online ahead of print, 2021 Aug 10]. J Hepatol. 2021;S0168-8278(21)01965-6. doi:10.1016/j.jhep.2021.08.001

S2933

BRIC Type 3? A Case of Benign Recurrent Intrahepatic Cholestasis Unlinked to ATP8B1 and ABCB11

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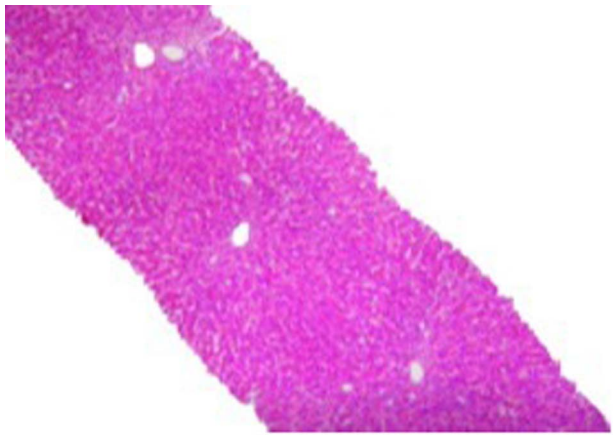
Introduction: Benign Recurrent Intrahepatic Cholestasis (BRIC) is a genetic disorder with two common subtypes: BRIC I (mutated ATP8B1 gene), and BRIC II (mutated ABCB11 gene). Diagnosis requires high degree of suspicion and is confirmed by genetic testing. There are rare cases where patients lack both of the typical mutations. We present a patient with clinical, lab and histological evidence of BRIC without the usual mutations.

Case Description/Methods: A 17-year-old male with no medical history presented with yellow eyes, pruritus, and acholic stools for six weeks, without triggering illness or stress. He denied fatigue, fevers, rash, dizziness, nausea, vomiting, diarrhea, constipation, arthralgias, melena, hematochezia, or hematemesis. He had no tattoos, blood transfusions, or allergies. He denied medications, alcohol, smoking, and drug use. On exam, he was icteric, with normal abdominal exam, and Alkaline Phosphatase 370 U/L, ALT 96 U/L, AST 66 U/L, and conjugated bilirubin of 6.7 mg/dL. Serological tests were normal, including antibodies for hepatitis A/B/C, CMV, EBV, HIV; alpha-1 antitrypsin; ceruloplasmin; iron, transferrin, C28Y/H63D mutations; TSH; antinuclear, anti-smooth muscle, anti-liver kidney microsomal, anti-mitochondrial antibodies; serum tissue transglutaminase; urine chlamydia and gonorrhea. Liver ultrasound had no biliary dilation. Genetic testing revealed negative ATP8B1 and ABCB11. Nonfocal liver biopsy showed bland cholestasis with coarse granular material in dilated bile canaliculi, compatible with a disorder of intrahepatic cholestasis, with morphologically similarities to biopsies seen in BRIC. He received ursodiol and symptoms improved. He stopped ursodiol after discharge and presented six months later with similar lab abnormalities. Repeat serological workup was normal. He received ursodiol and symptoms resolved again (Figure).

Discussion: Diagnostic criteria for BRIC include episodes of jaundice separated by a symptom-free interval of at least six months, lab and histological evidence of non-inflammatory cholestasis, non-dilated biliary ducts, and no other explanatory causes. Our patient satisfied these diagnostic criteria. Some evidence indicates the presence of additional disease loci for low γ-GT BRIC, explaining why some patients diagnosed with BRIC on clinical and histopathologic evidence lack usual mutations in ATP8B1 or ABCB11. This disorder is informally termed "BRIC 3"; it is unclear which genes are implicated in this form and further research is needed.



[2933] **Figure 1.** Histopathologic appearance of the liver biopsy. Bland centrilobular cholestasis with coarse granular biliary material within dilated bile canaliculi, without any significant inflammation, bile duct damage, or ductular reaction.

S2934

Budd-Chiari Syndrome as the Sole Presentation of SARS-CoV-2 Infection

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Introduction: The SARS-CoV-2 infection has been associated with a significant risk of venous thromboembolism. We present a case of a 22-year-old man with no prior medical history who is diagnosed with Budd-Chiari syndrome due to SARS-CoV-2 infection.
Case Description/Methods: The patient had one month of dry cough and abdominal distention. He had worsening abdominal discomfort, dyspnea, orthopnea, and decreased urine output for a week prior to admission. At admission, the patient was in distress due to abdominal discomfort. He was hemodynamically stable, icteric, and pale, had a distended abdomen that was dull on percussion and had a fluid thrill with shifting dullness. His labs showed a picture of liver failure with INR 1.9 HI (0.9-1.1 HI), total bilirubin 2.8 mg/dl (0.2-1.2 mg/dl), albumin 3.3 g/dl (3.5-5.0 g/dl), ammonia 55 mcmol/l (11-32 mcmol/l), AST 94 U/L (10-43 U/L), ALT 58 U/L (13-41 U/L), and Alk Phos 264U/L (42-119 U/L). CBC, and CMP were normal. His SARS-CoV-2 reverse transcription-polymerase chain reaction (RT/PCR) was negative and the SARS-CoV-2 IgG antibody was positive. Inflammatory markers were elevated. Imaging of the abdomen showed a 10 cm mass-like area in the left hepatic lobe suspicious of hepatic malignancy versus altered vascularity and edema, thrombosis of all the hepatic veins, left portal vein, marked portal hypertension, liver failure, abdominal pelvic varices, and severe ascites. These findings were consistent with Budd Chiari Syndrome. A hypercoagulability workup was done as mentioned in the table. He underwent paracentesis three times during his hospital stay and underwent a liver mass biopsy for a definitive diagnosis. Biopsy showed bridging necrosis, central hepatic vein thrombosis, and congestion, negative for malignancy. The patient's hypercoagulability, malignancy, and autoimmune workup were negative. Other tests like hepatitis panel, and HIV were negative. It was concluded that the Budd Chiari syndrome was due to mildly symptomatic SARS-CoV-2 infection. The patient was discharged on diuretics and was asked to follow up at a liver transplant center (Figure).
Discussion: Budd-Chiari syndrome is an unusual and sometimes the sole manifestation of SARS-CoV-2 infection. These patients might have no or minimal respiratory symptoms and usually have no underlying liver disease. The usual workup should involve ruling out hypercoagulable state, malignancy, and autoimmune disorder. Unfortunately, it can be fatal and a liver transplant is the only definitive management for such patients.

Hypercoagulable work up	Value	Reference range
Protein S activity (%)	76	70- 150
Protein C activity (%)	47	70- 180
Antithrombin III assay (%)	79	80- 135
Factor V Leiden mutation	Not detected	
Prothrombin gene mutation	Not detected	
Lupus anticoagulant	Not detected	
Anicardiolipin IgG (GLP)	Negative	

[2934] **Figure 1.** Anticoagulation workup.

S2935

Autoimmune Hepatitis After COVID-19 Vaccine: An Unusual Complication

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Introduction: Vaccination against SARS-Cov2 has represented a major milestone in our ability to ameliorate the COVID-19 pandemic. The vaccines are generally safe and effective, but some adverse events have been reported, including recent reports of autoimmune-like hepatitis. This case report presents a rare diagnosis of autoimmune hepatitis after administration of an mRNA COVID-19 vaccine.
Case Description/Methods: An 18-year-old man with no medical history presented after receiving the Pfizer COVID-19 vaccine in April 2021, and second dose in May 20221. In June of 2021, about two weeks after the second dose, the patient was found with AST 120 u/L, ALT 181 u/L, Platelets 90. See Table 1 for further lab values. Per family, prior liver chemistries were normal. Initial serological workup was completed, including viral hepatitis (Hepatitis A, B, C, E), CMV IgG and IgM, EBV IgG and IgM, ceruloplasmin, alpha-1-antitrypsin, iron studies, all of which were unremarkable. Patient was also found to have ANA titer 1:640, positive anti-smooth-muscle antibody of 1:640, IgG level of 1,845, and positive antibody to soluble liver antigen of 2.321. Abdominal MRI was completed with findings of splenomegaly 20 cm, radiographic evidence of advanced hepatic fibrosis Patient underwent a liver biopsy with foci of interface and lobular hepatitis, areas of bridging necrosis, and scarring and focal nodularity concerning for transition to cirrhosis. Given these findings and it's timing close to administration of COVID-19 vaccine, the patient was diagnosed with autoimmune hepatitis associated with the vaccine. Patient was started on steroids in November 2021, and later, azathioprine, with improvement of liver enzymes.
Discussion: Autoimmune hepatitis is a rare complication of COVID-19 vaccines, reported in few case reports in the literature. The pathophysiology remains unclear but is hypothesized to be related to molecular mimicry. The mRNA vaccines developed against COVID-19 leads to the production of the spike protein, and its antibodies. Antibodies to the spike protein have high affinity toward other human tissue proteins, which can lead to autoimmune tissue injury. In most reports, patients improved after treatment with steroids, though two have been reported to have passed away of liver failure. No liver

transplants have yet been reported. Given the serious implications of this disease and the number of vaccines against COVID-19 given world-wide, further research is needed to better understand this entity and its pathophysiology.

Table 1. Lab Values

	6/6/2021	11/15/2021	12/2/2021	1/12/2022	2/11/2022	4/18/2022	5/30/2022
AST U/L	120	323	31	35	48	37	50
ALT U/L	181	519	87	58	74	44	48
ALK P U/L	265	292	165	101	109	116	124
PLATELET COUNT	90	86	93	82	96	111	98

S2936

Be Suspicious: A Unique Case of Herpes Simplex Virus Hepatitis

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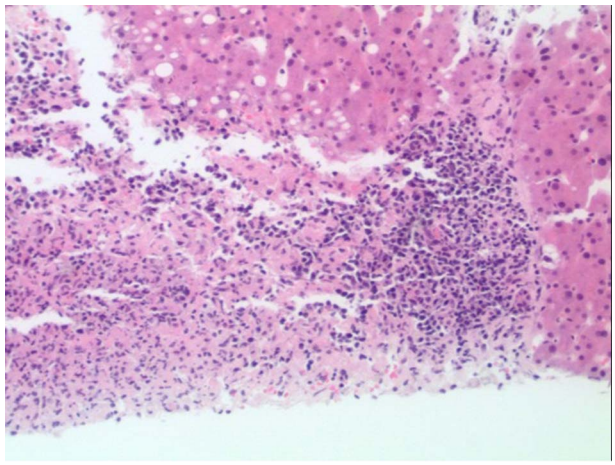
Introduction: We present a case of disseminated HSV infection leading to acute liver failure and encephalitis in the setting of a history of autologous stem cell transplant.
Case Description/Methods: A 66-year-old female with a past medical history of amyloidosis status post chemotherapy and autologous stem cell transplant in 2018 and chronic kidney disease was transferred to a tertiary care facility for concern of acute liver failure. On initial presentation, she was encephalopathic. Laboratories demonstrated an AST of 5264 IU/L, ALT of 3237 IU/L, INR of 3.1, and creatinine of 5.92. No other significant personal or family history was found. Workup was negative for toxins. The patient was considered immunocompromised. Her infectious workup was initially positive for E. coli in urine culture and Fusarium species in respiratory culture. The patient was initially thought to have liver hypoperfusion in the setting of severe sepsis. Serum testing for HSV1 and HSV2 was performed, and the HSV1 qualitative PCR was found to be positive. The patient's liver enzymes remained elevated. Lumbar puncture was performed, and the patient was initiated on intravenous Acyclovir given a high index of suspicion for HSV hepatitis and HSV encephalitis. Ultimately, the cerebrospinal fluid studies demonstrated a positive HSV DNA. Liver biopsy demonstrated confluent liver necrosis involving the portal tract extending to Zone 2 with plasma cell predominance, and immunohistochemical staining was positive for HSV1 (Figure 1). Treatment was continued with improvement in the patient's liver enzymes and mental status. Serial HSV quantitative serum PCRs were eventually undetectable. The patient recovered and was found to be at her baseline on outpatient follow-up.
Discussion: Patients who receive stem cell transplantation are considered immunocompromised for up to six weeks post-transplant and are at a higher risk of infection for one year. During this period, dormant viral infections such as HSV and VZV can be reactivated. This patient was immunocompromised, putting her at risk for reactivation of HSV. HSV hepatitis is an uncommon presentation of HSV that can cause fulminant hepatic failure in immunocompromised patients. Definitive diagnosis of HSV hepatitis can often delay diagnosis and increase mortality. Providers should maintain a high clinical suspicion for HSV hepatitis in immunocompromised patients presenting with acute liver failure and maintain a low threshold for starting anti-viral therapy if clinically indicated.



[2936] **Figure 1.** H&E stain of liver biopsy showing confluent liver necrosis involving the portal tract and extending to Zone 2. A moderate inflammatory infiltrate with plasma cell predominance located at the periphery of the necrotic area. No interface hepatitis or lobular inflammation is identified. Rare hepatocyte nuclei show smudge chromatin pattern, suggestive of a viral cytopathic effect.

S2937

Antiphospholipid Syndrome as a Cause of Nodular Regenerative Hyperplasia Presenting as Refractory Ascites

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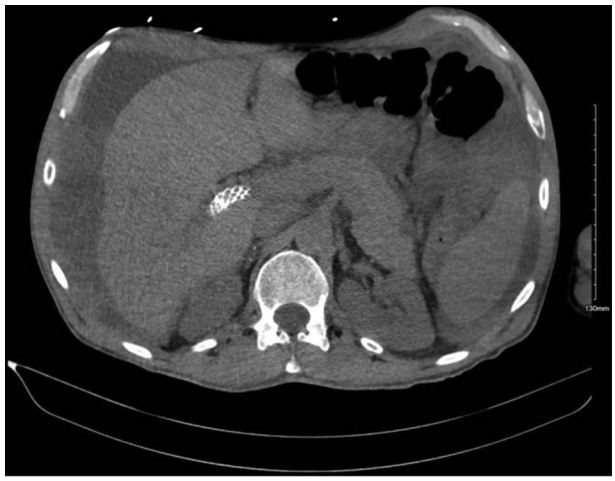
Introduction: Nodular regenerative hyperplasia (NRH) and obliterative portal venopathy (OPV) are two causes of non-cirrhotic portal hypertension (NCPH), which is a vascular liver disease in which clinical signs of portal hypertension (PHT), such as esophageal varices, ascites, and splenomegaly develop in the absence of cirrhosis and portal vein thrombosis. The etiology often remains unidentified, but herein we present a case of a male with antiphospholipid syndrome who developed NRH and OPV.
Case Description/Methods: We present a case of 56-year-old-male with NCPH and refractory ascites who underwent liver biopsy confirming NRH and OPV. Etiological work-up revealed beta-2 glycoprotein-1 and anticardiolipin antibodies, concerning for APS despite no prior history of thrombosis. The patient underwent a transjugular intrahepatic portosystemic shunt (TIPS) procedure for his refractory ascites and was started on prophylactic anticoagulation due to concern for APS with clinical improvement in his ascites and shortness of breath. (Figure)
Discussion: NCPH is a rare disease that typically presents with complications of PHT, such as ascites or variceal bleeding, and is commonly misdiagnosed as cirrhosis. The pathophysiology is unknown but both intrahepatic vascular obstruction and increased splanchnic blood flow have been suggested to explain NCPH. At minimum, the diagnosis of NCPH requires the presence of portal hypertension, the absence of cirrhosis, the presence of advanced fibrosis or other causes of chronic liver diseases, and the absence of thrombosis of the hepatic or portal veins on imaging. NCPH is a diagnosis of exclusion and an extensive work-up is typically recommended to first evaluate for other causes of liver disease, such as alcoholic and nonalcoholic steatohepatitis, autoimmune hepatitis, and viral hepatitis. For confirmation, liver biopsy findings can be associated with NRH and OPV, which are two causes of NCPH. Different conditions have been associated with NRH and OPV, including immunological disorders such as Lupus or APS, viral infections such as HIV, and immunosuppressive medications such as azathioprine. Pursuing TIPS earlier in the setting of refractory ascites, as well as offering anticoagulation therapy for patients with possible APS to prevent the development of potential thromboses, could be appropriate recommendations to prevent complications in the disease course. This case report highlights the need for further investigations on the etiologies, diagnosis pathways, and treatment options for NCPH.



[2937] **Figure 1.** Ascites with visible transjugular intrahepatic portosystemic shunt.

S2938

Autoimmune Hepatitis Triggered by Acute Liver Failure Caused by Hepatitis A

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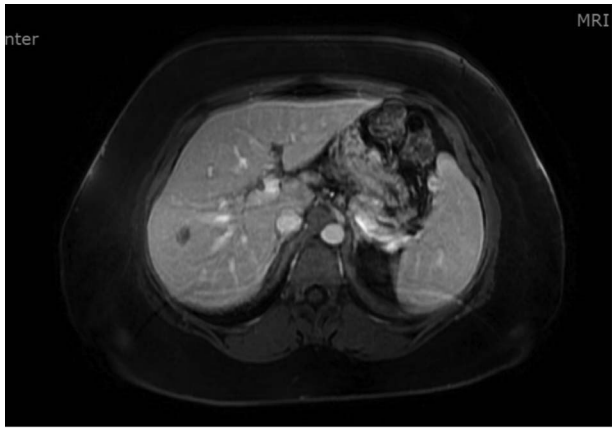
Introduction: Autoimmune hepatitis is a disease with autoantibodies and elevated serum globulin levels that can present either as acute or chronic hepatitis. It has been theorized that autoimmune hepatitis is unveiled when there is an environmental trigger in a genetically predisposed individual. There has been some evidence that various viruses can be a trigger as well. This is a patient who developed autoimmune hepatitis shortly after an episode of hepatitis A.
Case Description/Methods: A 37-year-old female with no medical history was previously hospitalized with acute liver failure secondary to acute hepatitis A in a restaurant-related outbreak. She recovered after a four-day hospital stay and liver enzymes normalized after three months from > 7000 U/L. After another three months, she presented with complaints of dark-colored urine, right-sided abdominal pain, bilateral pedal edema, and fatigue and was also found to have elevated liver enzymes (AST 1612 U/L, ALT 1347 U/L). Viral hepatitis labs were negative. Antinuclear antibody (ANA) and anti-liver-kidney microsome (anti-LK) antibodies were negative but anti-smooth muscle antibody (ASMA) was 1:80 and IgG was 3893. A liver biopsy performed revealed chronic moderately active hepatitis with abundant plasma cells and stage 2 fibrosis which combined with lab findings was suggestive of autoimmune hepatitis. Prednisone 20 mg every day was started and liver enzymes normalized in three weeks. At that time, azathioprine was started and prednisone was weaned off (Figure).
Discussion: The exact pathogenesis of autoimmune hepatitis is not clear but it appears that environmental triggers can cause the disease in genetically susceptible patients. It is thought to be a molecular interaction between the antigen, the major histocompatibility complex, and the T cell receptor which forms a complex. Triggers can include viruses, herbs, and immunizations. The major autoantibodies involved in autoimmune hepatitis include ANA, anti-LK, ASMA, and anti-mitochondrial antibody. The goal of therapy is to suppress the immune system and often starts prednisone plus/minus azathioprine which can help reduce the doses of steroids required. This often leads to remission, at which point prednisone can be tapered and maintenance doses of azathioprine can be used.



[2938] **Figure 1.** MRI showing hepatomegaly and a lesion in the right hepatic lobe consistent with benign hemangioma.

S2939

Ashwagandha Toxicity: A Rare Case of Drug Induced Liver Injury (DILI)

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Introduction: The pursuance of natural and herbal remedies by many has brought with it further knowledge of the many causes of Drug Induced Liver Injury (DILI). Many of these "natural" over-the-counter (OTC) supplements may not be benign due to the lack of scientific analysis and unregulated production. Ashwagandha is a supplement used for memory enhancement, management of anxiety, and general increase in vitality. We present a case of Ashwagandha DILI in a healthy 36-year-old man.
Case Description/Methods: A 36-year-old male presented to our institution with 1 week of fatigue, jaundice, nausea and subjective fevers. Upon presentation he was hemodynamically stable and afebrile. Physical exam was notable for jaundice without other stigmata of liver disease or mental status changes. He was found to have acute liver injury with lab chemistries remarkable for AST= 1482 U/L, ALT= 1375 U/L, Tbili= 22.3 mg/dL, alkaline phosphatase= 202 U/L and an INR of 1.6. He denied alcohol and drug use, recent travel or sick contacts. Medications list was notable for cetirizine, diphenhydramine (both taken as needed), OTC testosterone supplements, OTC apple cider vinegar gummies BID and OTC ashwagandha gummies BID. Workup was negative for autoimmune, infectious, metabolic, obstructive and

vascular causes of liver injury. Acetaminophen level and urine drug screen were negative. Trans-jugular liver biopsy revealed evidence of acute portal and lobular hepatitis and cholestasis, suggestive of DILI. Patient's liver enzymes continued to downtrend (AST=929, ALT=765, Tbili=22.5) after withdrawal of his supplements and was discharged (Figure).

Discussion: Ashwagandha causing DILI has been infrequently reported in the literature. One case series of 5 patients in Iceland and a single case in Japan reported that ashwagandha is an uncommon culprit linked to acute liver injury. None of the reported cases necessitated liver transplant. Average time to resolution and normalization of LFTs was 3.5 months and R values usually ranged in the mixed range (R value 2-5). The case series also revealed that ashwagandha's toxicity could be dose dependent, as higher peaks in LFT's were tied to recent increases in ashwagandha dosing. Although ashwagandha has been a rarely reported cause of DILI, a thorough history of medication and OTC supplements in patients with undifferentiated acute liver injury may yield more cases. Further study and reporting of DILI in the ever-growing supplement market appears to be required to minimize potential injury.

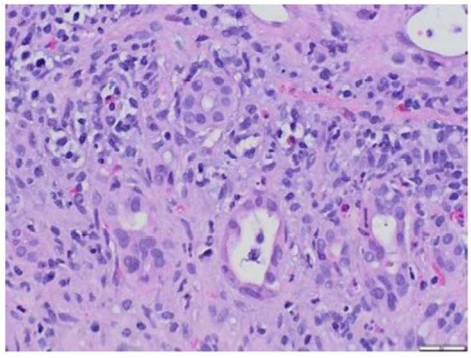

Figure 1: Mixed Portal Inflammation with Bile Ductular Proliferation

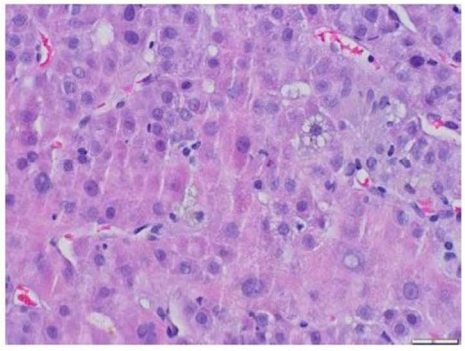

Figure 2: Cholestasis with Feathery Degeneration

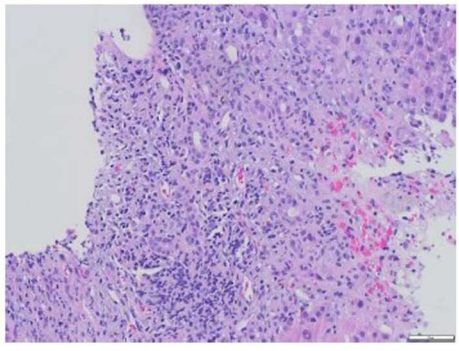

Figure 3: Portal Inflammation

[2939] **Figure 1.** Tranjugular Liver Biopsy Findings.

S2940

Bupivacaine-Induced Hepatotoxicity in a Healthy Patient Without Chronic Liver Disease After Shoulder Arthroscopy

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Introduction: Bupivacaine is a local anesthetic which has been increasingly used in the post-operative state for pain control. Hepatotoxicity is a rare complication, and few cases are reported in patients with chronic liver disease. We present a case of acute liver injury from bupivacaine use in a healthy patient without prior history of liver disease.

Case Description/Methods: A 68-year-old female with a past medical history of primary hypertension and recent nontraumatic complete tear of the right rotator cuff, presents to the hospital with fatigue, loss of appetite, and nausea. She recently underwent an arthroscopy of the right shoulder with repair of the rotator cuff two weeks prior. Her surgery was uncomplicated, and patient was started on bupivacaine ONQ pump infusion at 5 ml/hr for three days for post-operative pain. Further history reveals patient is non-alcoholic without prior liver disease, including cirrhosis. Review of systems is concerning for associated generalized abdominal discomfort. Physical exam demonstrated jaundice with scleral icterus with mild periumbilical tenderness to palpation without hepatosplenomegaly or ascites. Labs demonstrated elevated total bilirubin of 10.2 mg/dL with Alkaline phosphatase, ALT, and AST being 924 U/L, 429 U/L, and 279 U/L, respectively. Imaging studies including CT abdomen and pelvis with contrast, abdominal ultrasound, MRCP, and portal vein doppler were negative. Additional work up for underlying liver disease including acetaminophen and ethanol levels, SARS-CoV2, Hepatitis panel, EBV antigen, and urine

toxicology were negative. It was determined patient had bupivacaine induced hepatotoxicity. Patient's health improved with conservative management and she was discharged with instructions for close monitoring of her LFTs.
Discussion: Bupivacaine is an amino-amide anesthetic which binds to the intracellular portion of voltage-gated sodium channels and prevents depolarization of pain signals. It is metabolized by the liver and thus reports of hepatotoxicity, although rare, occur in patients with underlying liver pathology. Our patient became symptomatic with acute rise in LFTs. An extensive workup for other etiologies of acute liver toxicity was negative. Rapid vascular uptake of the drug is the most common reason for bupivacaine toxicity; and this remains a possibility for the mechanism of toxicity in our patient. A prior case report of bupivacaine hepatotoxicity demonstrated a cholestatic pattern, which is consistent with our findings.

S2941

Cardiac Tamponade Presenting as Acute Liver Injury

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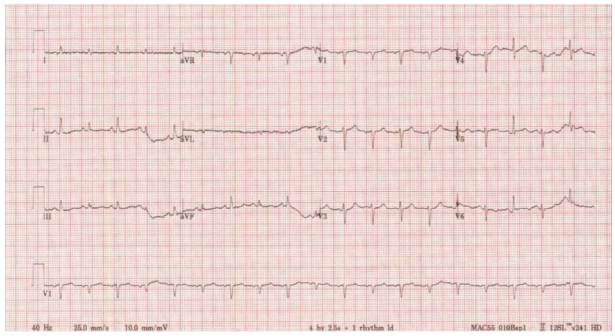
Introduction: Acute liver injury (ALI) has an extensive differential diagnosis list which can make diagnosis challenging. A few causes of elevation of AST (aspartate transaminase), ALT (alanine transaminase) in thousand is ischemic, toxic and viral hepatitis. One such rare cause is cardiac tamponade that can present as an acute liver injury. The literature of cardiac tamponade causing ALI is limited to few case reports and we present one such case.
Case Description/Methods: A 55 year old male with squamous cell lung cancer on durvalumab, diabetes mellitus presented with worsening shortness of breath for the last one week. On presentation his vitals were stable and physical exam revealed distant heart sound with electrical alternans noted in electrocardiogram. Blood work was noted for elevated ALT 1481 U/L (units per liter), AST 803 U/L, ALP (alkaline phosphatase) 227 U/L, total bilirubin 1.3 mg/dl. Patient has had a normal LFT (liver function test) in blood work done 3 weeks ago. Initial chest x-ray showed large right pleural effusion. The next day, repeat lab revealed worsening of liver enzymes with INR (international normalized ratio) 3, ALT 4146 U/L, AST 4302 U/L, ALP 189 U/L, total bilirubin 2 mg/dl and LDH (lactate dehydrogenase) 3879 U/L. Serum creatinine was also noted to worsen to 2.06 mg/dL from baseline 0.89 mg/dL. CT chest revealed large right sided pleural effusion with possible underlying pneumonia or atelectasis in the right middle and lower lobe with moderate to large pericardial effusion. Patient underwent an echocardiogram which revealed a 3.1 cm circumferential pericardial fluid collection with tamponade physiology. He was taken for urgent pericardiocentesis with removal of 760 mL of fluid. Pericardial fluid cytology revealed adenocarcinoma. Post pericardiocentesis patients LFT improved and he was discharged after 6 days (Figure).
Discussion: ALI can be a presenting sign of cardiac tamponade, a prompt diagnosis and treatment can be life saving. Cardiac tamponade is thought to cause liver injury by hepatic venous congestion and decreasing cardiac output causing ischemic Injury. 50% cases of ischemic hepatitis do not have documented hypotension. A few lab abnormalities often seen with ischemic hepatitis are acute kidney injury, massive rise in LDH, hyperglycemia. The prognosis of ischemic hepatitis depends on the inciting cause rather than severity of liver injury. A 50% improvement in liver enzymes within 72 hour of correction of inciting cause is usually seen.



[2941] **Figure 1.** Electrical alterans.

S2942

Bleeding IGV2 from Left-Sided Portal Hypertension in a Decompensated Cirrhotic

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Introduction: Variceal hemorrhage is one of the leading causes of morbidity and mortality in cirrhosis. While esophageal varices (EVs) are more likely to bleed, bleeding from gastric varices is typically more severe and carries higher mortality. IGV2, isolated gastric varices located outside of the cardiofundal region, are very rarely seen in patients with cirrhosis and do not frequently result in bleeding. Here we discuss a complex case of bleeding IGV2 in a cirrhotic patient who developed left-sided portal hypertension due to splenic vein thrombosis.
Case Description/Methods: A 69-year-old white male with decompensated Child-Pugh B cirrhosis presented with acute onset hematemesis and hematochezia. He has a known chronic portal vein thrombus for which he was not anticoagulated, as he was not deemed a liver transplant candidate. Initial vitals were in normal range. Initial labs were notable for hemoglobin 12.5 g/dL, blood urea nitrogen 37 mg/dL, and MELD-Na 16. He was administered octreotide and ceftriaxone and taken for upper endoscopy. Upper endoscopy revealed small, nonbleeding EVs and copious gastric blood. Large IGV2 with stigmata of recent bleeding were identified in the gastric body. Multiphasic computed tomography showed several portosystemic shunts, cavernous transformation of the portal vein, and thrombus extending into the superior mesenteric vein (SMV) and splenic vein. After multidisciplinary discussion, the patient was taken for splenectomy which resulted in variceal decompression. Hepatic function remained at baseline following surgery and bleeding did not recur.
Discussion: This case highlights the multidisciplinary and collaborative approach which is required for management of bleeding IGV2. Ultimately, approach depends on the patient's vascular anatomy, which in cases of cirrhosis may be quite complex due to portosystemic shunts and collateral vessels. As such, diagnostic radiology, interventional radiology, gastroenterology, and surgery were all involved in this case. While transjugular intrahepatic portosystemic shunt (TIPS) and balloon occluded retrograde transvenous obliteration (BRTO) play big roles in managing most gastric varices, TIPS would not have decompressed the left side of the portal system due to thrombus. BRTO, while perhaps more efficacious, would carry risk of intestinal ischemia in the setting of SMV thrombus, as occlusion of collateral vessels may disrupt drainage of the small bowel. Splenectomy was therefore felt to be the most definitive management.

S2943

Budd-Chiari Syndrome Diagnosis Achieved With High Clinical Suspicion Raised by EUS-Guided Portal Pressures and Liver Biopsy

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Introduction: Budd Chiari syndrome (BCS) is a rare syndrome due to hepatic venous obstruction in the absence of cardiac cause, mostly secondary to thrombosis. As high as 80%of patients present with ascites. The diagnosis should be suspected in patient with acute or chronic liver disease without identified cause. Venous obstruction can be seen on Doppler ultrasound, CT, or MRI of the hepatic veins and inferior vena cava. In some cases, hepatic venogram is needed for its diagnostic and therapeutic advantages. We report a 50-year-old woman with BCS who underwent successful wire recanalization, angioplasty and thrombolysis of the right hepatic vein.
Case Description/Methods: 50-year-old woman with no significant past medical history presented with abdominal distension and bilateral lower extremity swelling of two weeks duration. On exam, abdomen was distended with positive signs of fluid collection with +3 bilateral lower extremity edema. Labs revealed total bilirubin 3.3 direct bilirubin 1.4, ALP 477, ALT 38, AST 96, PT 14.4, INR 1.35. Ultrasound showed Heterogeneous appearance of liver and large volume ascites. Doppler study showed normal hepatic and portal venous flow. Abdominal MRI showed hepatic steatosis and caudate lobe hypertrophy. Large

volume paracentesis was done with analysis showing SAAG of 3.1, WBC 364, Neutrophils 8. EGD revealed large esophageal varices. Viral hepatitis panel, ceruloplasmin, alpha-1antitripsin, ASMA, AMA antibodies were negative. EUS guided liver biopsy and portal pressure measurement was done showing venous pressure gradient of 13 consistent with portal hypertension. Liver biopsy showed grade 2 fibrosis and zone 3 congestion. Hepatic venogram showed chronically occluded right hepatic vein on which successful wire recanalization was done. Pt was worked up for hypercoagulable state and was found to have JAK 2 mutation and Protein C deficiency. Apixaban, Beta blocker and diuretics were started. Patient showed significant improvement with ascites and MELD score.
Discussion: First line investigation for diagnosis of BCS is doppler ultrasound. Doppler US and MRI venography didn't reveal the hepatic venous thrombosis in our patient. However, high index of suspicion, finding of elevated IVC pressure on EUS, and caudate lobe hypertrophy found on imaging as well as congestion liver biopsy findings led us to further investigate with hepatic venogram which confirmed the diagnosis and provided therapeutic advantages.

Table 1. Prothrombotic risk factors for BCS ACG Clinical Guideline

A. Acquired thrombophilia	B. Inherited thrombophilia	C. Systemic factors	D. Hormonal factors
Myeloproliferative disorder	Factor V Leiden	Sarcoidosis	OCP use
Polycythemia vera	Prothrombin gene G20210A mutation	Vasculitis	Pregnancy
Essential thrombocytosis	MTHFR C677T mutation	Behcet's disease	
Idiopathic myelofibrosis	Thalassemia	Connective tissue disease	
JAK 2 mutation	PC deficiency	Inflammatory bowel diseases	
PNH	Protein C deficiency		
Hyperhomocysteinemia	Antithrombin deficiency		

Disorders of the Hepatic and Mesenteric Circulation Simonetto, Douglas A.; Singal, Ashwani K.; Garcia-Tsao, Guadalupe; Caldwell, Stephen H.; Ahn, Joseph; Kamath, Patrick S. Official journal of the American College of Gastroenterology | ACG115(1):18-40, January 2020. doi: 10.14309/ajg.0000000000000486

S2944

Autoimmune Hepatitis Presenting With Concomitant Chronic Pancreatitis

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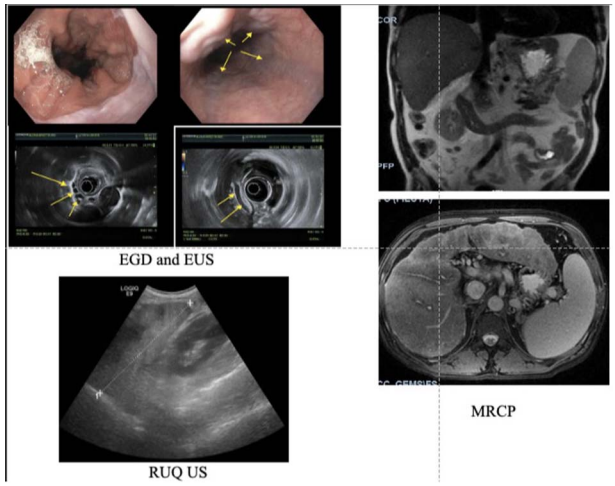
Introduction: Autoimmune Hepatitis (AIH) is a progressive form of chronic hepatitis, with periods of remissions and exacerbations. Diagnosis includes abnormally high levels of immunoglobulins and multiple autoantibodies, with female predominance. Clinical presentation is variable, with a spectrum extending from asymptomatic cases to fulminant liver failure. Presenting symptoms may include abdominal pain, malaise, fatigue, and small joint arthralgia. We present a case of a 36 YO M with PMH of alcohol dependence and acute pancreatitis who was diagnosed with AIH.
Case Description/Methods: A 36 YO AA M with PMH of alcohol dependence (in remission for 2 yrs), tobacco use, and pancreatitis, presented to the ED with non-radiating mid-epigastric abdominal pain 10/10 in severity, associated with NBNB emesis, exacerbated by movement for 2 days. The patient was hemodynamically stable and on exam he had icteric frenulum, abdominal distension, with liver span 12cm at mid-clavicular line, and absence of fluid wave, shifting dullness, rebound tenderness, or voluntary guarding. Labs notable for pancytopenia, elevated lipase, elevated ALP, elevated AST and ALT with 2:1 ratio, and hyperbilirubinemia. MRCP showed cirrhotic liver with splenomegaly and varices, as well as with free fluid in the lesser sac along the pancreatic head, duodenum, and right retroperitoneum (compatible with acute pancreatitis). Patient received IV fluids for pancreatitis. Additional labs were remarkable for elevated actin smooth muscle antibody at 26U (ref range: 0-19), ANA positive, with high alpha-1-antitrypsin levels and normal ceruloplasmin levels. The patient left against medical advice; and was given resources for Hepatology with referral for liver transplant (Figure).
Discussion: There is limited data regarding patients with concomitant AIH and pancreatitis. Our pt presented with a AIH with secondary acute on chronic pancreatitis, in the absence of additional autoimmune manifestations. Mechanism of AIH remains poorly understood; however, there is an association between the HLA gene and AIH. Genetic studies have shown HLA-DRB1*0301 and HLA-DRB1*0401 as primary and secondary genotypes susceptible to AIH, as well as genetic variants with CARD10 and SH2B3. Products secondary to metabolism of ETOH such as alcohol dehydrogenase, malondialdehyde, and acetaldehyde, can lead to development of autoantibodies. Additional research is indicated to evaluate the relationship between AIH and acute pancreatitis.



[2944] **Figure 1.** A closer look at the case with images from the EGD, EUS, Right upper quadrant ultrasound and MRCP.

S2945

Check Point SOS! A Case of Cemiplimab-Associated Sinusoidal Obstruction Syndrome

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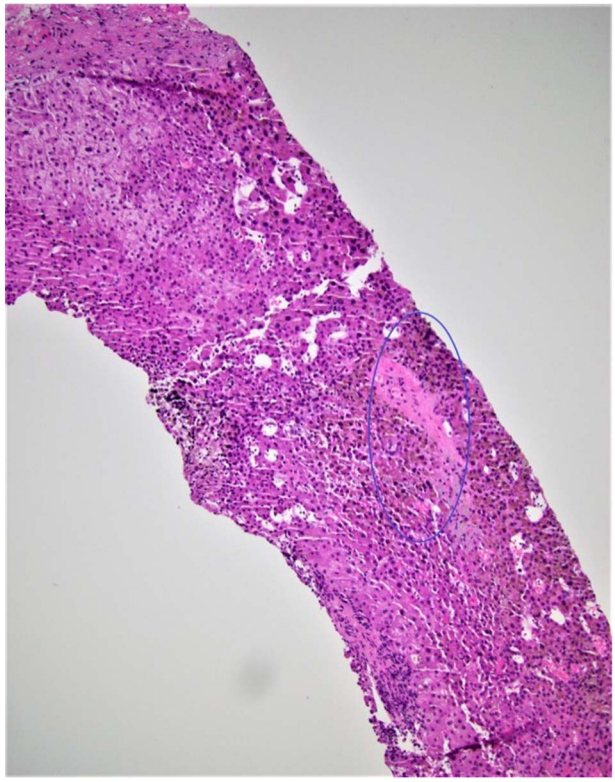
Introduction: Hepatic veno-occlusive disease (VOD) or sinusoidal obstruction syndrome (SOS), is a clinical syndrome characterized by hepatomegaly, right-upper quadrant pain, and ascites that occurs most commonly in the setting of high-dose chemotherapy or hematopoietic stem cell transplantation (HSCT). The diagnosis can be confirmed on biopsy. Cemiplimab is an immune checkpoint inhibitor recently approved for the treatment of cutaneous squamous cell carcinoma. There are currently no known reports of immune checkpoint inhibitor-related VOD/SOS.
Case Description/Methods: A 58-year-old female with a history of locally advanced basal cell carcinoma of the left eye treated with six months of Cemipilimab presented with ascites. On admission, labs were notable for a total bilirubin of 1.2, mildly elevated liver function tests, alkaline phosphatase 884, and international normalized ratio 2.1. A diagnostic tap revealed a high SAAG ascites that was negative for infection. A comprehensive serological workup for viral, metabolic and autoimmune causes was unrevealing. A transjugular liver biopsy demonstrated a hepatic venous pressure gradient of 18mmHg, nodular regenerative hyperplasia (NRH), and portal venopathy. The patient was discharged on steroids but returned one month later for recurrent ascites and worsening bilirubin to 12.6 (direct 7.3); COVID PCR was

negative. A full rheumatologic and vasculitis workup was unremarkable. Repeat biopsy (Figure 1) demonstrated moderate NRH changes, prominent central vein sclerosis with fibrous obliteration, signs of SOS/VOD and central venulitis with fibrotic changes with sinusoidal portal hypertension.

Discussion: VOD occurs most often with hematopoietic stem cell transplantation, and chemotherapeutic agents. Here we present the first case of checkpoint inhibitor-induced VOD/SOS. Despite discontinuation of the offending agent and a trial of steroids, the patient's clinical course continued to deteriorate. She eventually developed refractory ascites and portosystemic encephalopathy. She was deemed not a candidate for liver transplant given her underlying malignancy. She was transitioned to home hospice before further treatment, such as Defibrotide could have been pursued. VOD associated with immune checkpoint inhibition should be considered in the differential of patients who develop new onset liver dysfunction and ascites while receiving these medications.



[2945] **Figure 1.** Sinusoidal dilatation and congestion alongside central vein sclerosis. These are common findings of sinusoidal obstruction syndrome on liver biopsy.

S2946

Chronic Graft versus Host Disease Masquerading CMV Colitis After Liver Transplantation: A Clinical Challenge

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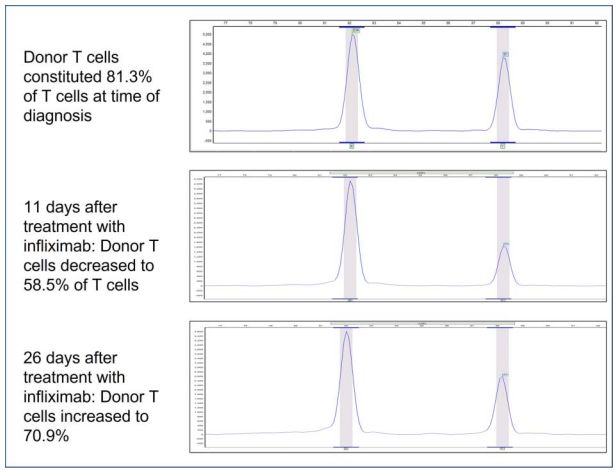
Introduction: Graft versus host disease (GVHD) after solid organ transplantation is a rare but serious complication with high mortality that typically occurs 1-8 weeks post liver transplantation (LT). Steroids are first line treatment and data on second line treatment are less widely available.

Case Description/Methods: 54-years-old woman with history of alcohol-related cirrhosis went under LT with a positive cytomegalovirus (CMV) and HCV male donor. She was discharged home without any major complications. Immune suppression included tacrolimus, mycophenolate mofetil, (MMF) and prednisone taper. She was admitted to hospital 3 months later with diarrhea and acute kidney injury. C. difficile PCR was negative, CMV PCR was positive, and biopsies obtained during colonoscopy revealed severe CMV colitis. She was started on ganciclovir. Tacrolimus was reduced and MMF was discontinued. Fever and diarrhea were continued despite CMV treatment and later she developed a diffuse skin rash. This initially thought to be due to ganciclovir, but skin biopsy was concerning for GVHD. Gastric and colon biopsies and peripheral blood chimerism (donor T cell 81.3%; normal $< 1\%$) confirmed GVHD. She was started on steroid therapy with minimal improvement in diarrhea as well as minimal improvement after the addition of topical budesonide and octreotide. Her course was complicated by posterior reversible encephalopathy syndrome and status epilepticus and was intubated. Tacrolimus was stopped. She then developed pancytopenia with severe neutropenia despite discontinuation of ganciclovir. Bone marrow biopsy showed 10.7% donor alleles. Infliximab was initiated. Stool output decreased and neutropenia resolved after 1 week. Seizures were controlled and she was extubated. Chimerism analysis of peripheral blood after 11 days showed significant reduction of donor T cells (58.5%). However, after 2 weeks, diarrhea worsened and donor T cells increased to 70.9% in blood. Ruxolitinib and cyclosporin were started in addition to infliximab but she developed septic shock and expired (Figure).

Discussion: Persistent diarrhea and skin rash after liver transplantation may be signs of GVHD but these symptoms can also be seen in more common conditions such as C. difficile infection, drug reaction, and CMV colitis. Biopsy and chimerism analysis might both be needed for diagnosis of GVHD in patients with low pretest probability. Post LT GVHD that is refractory to steroid therapy may respond to infliximab. Sepsis is the most common cause of mortality in LT GVHD.



[2946] **Figure 1.** Sample micrographs from short tandem repeat (STR) testing (chimerism analysis) on isolated T cells from peripheral blood.

S2947

Case Report: Multitarget HCC Blood Test in the Early Detection of HCC

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Introduction: Intro: Liver cancer is the fastest growing cause of cancer-related deaths in the U.S. Guidelines recommend biannual HCC screening using ultrasound (US), with or without serum alpha-fetoprotein (AFP) measurement in at-risk patients. HCC screening in at risk individuals is associated with improved early detection, greater likelihood of curative treatment, and improved outcomes. The sensitivity of recommended tools for early-stage cancer detection remain sub-optimal. Better screening techniques have been developed and are currently under investigation. One such commercially available approach is the multi-target HCC blood test (mt-HBT, Oncoguard® Liver), analyzes blood-based biomarker, (methylated DNA and AFP) to aid in the detection of early-stage HCC. The real-world benefits of these new approaches are being seen. We report a case demonstrating the benefit of mt-HBT for diagnosis & early treatment of HCC.
Case Description/Methods: Case report: 69 yo male, with cirrhosis, was referred for evaluation and treatment. Patient's etiology of cirrhosis was presumed to be related to NASH due to negative serologic testing and patient's history of obesity with BMI over 40 for greater than 30 years. Liver biopsy was refused by the patient. HCC screening guidelines were initiated. Initial screening, with US and AFP were negative. Six months later, an US and mt-HBT, was used for screening. US was negative, but mt-HBT was positive. Follow-up MRI with and without contrast was negative for HCC. Per patient's request, he was referred to an academic transplant center, where a repeat MRI imaging study and GALAD calculations were negative for HCC. Because of positive mt-HBT, patient had repeat imaging studies 4 months later. A 1.3 cm HCC lesion was found in the right lobe of the liver. Radiofrequency ablation of the HCC was performed and follow up imaging studies one month later showed no evidence of residual tumor.
Discussion: The overall goal of HCC screening is to detect disease earlier, reducing morbidity and mortality. This case demonstrates the benefit using the mt-HBT. While further study is needed, given the initial negative standard HCC screening technique (AFP, US, MRI, & GALAD), an isolated positive mt-HBT, may warrant more frequent HCC screening. This patient demonstrates benefits of early diagnosis; less invasive treatment options leading to cure of HCC. How to use the mt-HBT in post HCC cure needs further investigation, but this case demonstrates the benefit of the mt-HBT.

S2948

Challenges in Recognizing and Diagnosing Wilson Disease

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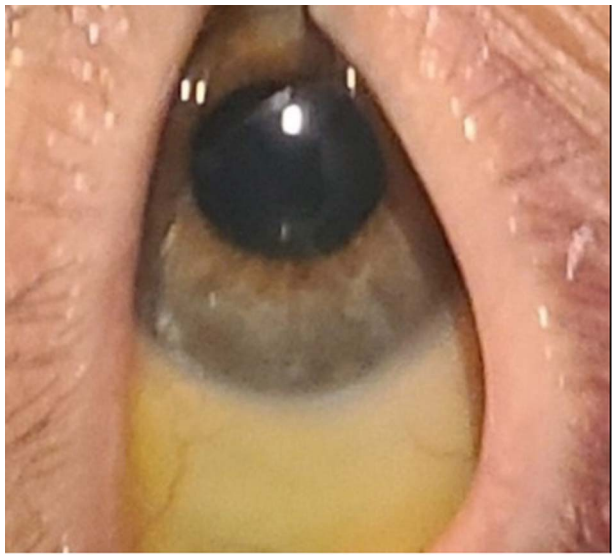
Introduction: *Wilson disease* is a rare autosomal recessive disease leading to impaired copper homeostasis and accumulation in the liver, brain, and other organs. It is manageable, and physicians must familiarize themselves with the diversity of its presentation. Our case sheds light on the challenges; health care providers face in establishing the diagnosis.
Case Description/Methods: A fifty-eight-year-old female was admitted to the hospital for altered mental status who had a dramatic hospital course with acute liver failure complicated by shock and multiorgan failure. Her past medical history is significant for COPD, liver cirrhosis. Family history is positive for Alpha-1 Antitrypsin deficiency. She presented with acute psychosis and altered mental status. Patient was admitted with suspected hepatic encephalopathy. She developed acute liver failure followed by shock and multiorgan failure. Her work up came back as Hg 6.7, Plt 27, INR 2.1, Haptoglobin $<$ 8 mg/dl, Coombs negative, Ceruoplasmn 5.9, copper serum 32, 24 hours urine copper level 42 and urine copper level 351. Previously, she complained of tremor and slurred speech. She was evaluated by neurology team for seizure like activity, but it was found that her movements were compelling choreiform movements (Figure). Liver cirrhosis evaluation started on 2019 when she was evaluated for new onset ascites. Her exam showed abdominal distension and lower limb edema, labs showed ALP 247, AST/ALT 51/16, total bilirubin 0.7, ANA $>$1: 1280, alpha 1 antitrypsin low 72 with MZ phenotype, Ceruplasmin level 13. Liver Biopsy Showed findings consistent with moderately active steatohepatitis with cirrhosis. It was presumed that her liver cirrhosis was related to A-1AT deficiency/NASH. The low ceruplasmin has slipped and the diagnosis of Liver Cirrhosis secondary to A-1AT deficiency carried on.
Discussion: Wilson disease caused by mutations in the ATP7B gene responsible for intracellular copper transporter's function, led to impairment in biliary copper excretion leading to the deposit of copper in several organs. The diagnosis is based on the score developed at the 8th International Meeting on Wilson Disease in Leipzig (8). If the score is $\geq$4, Wilson disease is highly likely. In our patient, points are given as follows: Neuropsychiatric symptoms suggestive of Wilson disease (2), Coombs-negative hemolytic anemia with high serum copper (1), Serum ceruloplasmin $<$ 10 mg/dL (2) with a total score of 5 points, which made her diagnosis with Wilson disease is highly likely.



[2948] **Figure 1.** Kayser-Fleisher ring.

S2949

Case of Cholangiogram-Induced Acute Liver Injury Disease

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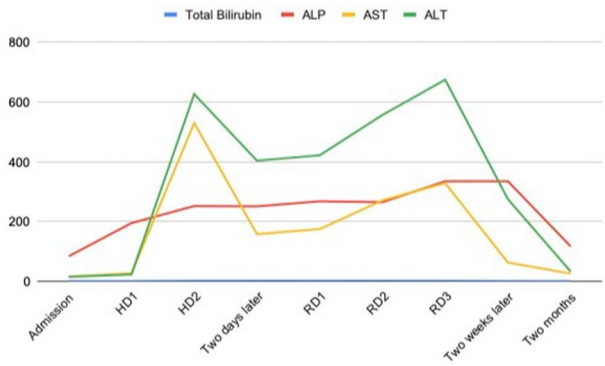
[1]University of Tennessee- Nashville, St. Thomas Midtown, Nashville, TN; [2]St. Thomas West, Nashville, TN.

Introduction: We present a female who had prolonged acute hepatocellular liver injury after undergoing a cholangiogram using Omnipaque contrast. About 10 cases of cholestatic liver injury have been reported after ERCP, however our case is unique being predominantly hepatocellular injury.
Case Description/Methods: Initially presenting with abdominal pain then diagnosed with cholecystitis, she underwent cholecystectomy with cholangiogram. She subsequently underwent ERCP due to a retained stone and discharged home the next day without notable complications. She presented to the hospital two days later with pruritus. After ruling out infectious cause and further mechanical hepatobiliary pathology, she was discharged home due to improved symptoms with symptomatic management. Transaminases gradually resolved with close outpatient follow-up at two weeks and two months.
Discussion: The exact mechanism remains unclear, liver injury due to idiosyncratic reaction from contrast was postulated. This is possibly due to local hepatic injury from the contrast agent in the biliary system. The contrast material being infused under high pressure, can have a toxic effect on the liver with disruption of canalicular plasma membranes. Systemic distribution of the contrast medium from the bile duct and the spreading of the agent extracellularly to the nearby tissues might be responsible for direct toxic liver injury. Awareness of this reaction is helpful in preventing unnecessary repeat ERCP to confirm bile duct clearance as well as liver biopsies. Prednisone may be considered if persistently elevated liver enzymes.



[2949] **Figure 1.** Title: Liver Function Tests during clinical course Caption: Patient's liver function test over the hospitalization, readmission, two-week outpatient follow-up, and two-month outpatient follow-up revealing fluctuating elevation in transaminases after cholangiogram performed on day of admission while total bilirubin remains within normal limits. Gradual resolution of elevated transaminases over two-week and two-month follow-up. ERCP occurred on hospital day 1. Legend: HD- Hospital day RD- Readmission day ALP- alkaline phosphatase AST- Aspartate transaminase ALT- Alanine Transaminase.

S2950

Cirrhosis-Associated Immune Dysfunction: Diffuse Curvulvaria Infection, CMV Viremia and PJP Pneumonia in an End-Stage Liver Disease Patient

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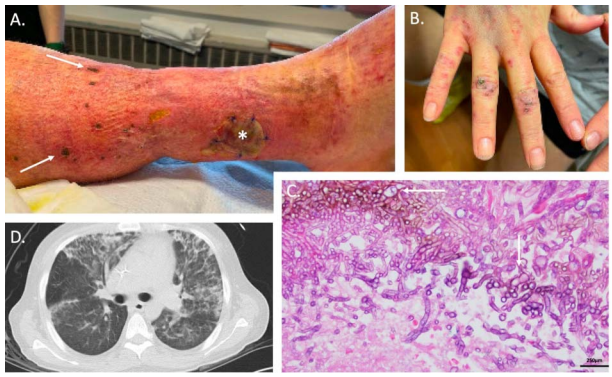
Medstar Georgetown University Hospital, Washington, DC.

Introduction: Infections are a major cause of morbidity and mortality in end-stage liver disease (ESLD). Cirrhosis-associated immune dysfunction (CAID) refers to the immune system dysregulation observed in ESLD. Innate and adaptive immunity dysfunction is clinically evident by increased susceptibility to bacterial, fungal, and viral infections. We present the case of a young patient with multiple concomitant opportunistic infections attributed to immune dysfunction secondary to ESLD.
Case Description/Methods: A 36-year-old male with a prior medical history of asthma and alcohol-related cirrhosis presented with abdominal distension, jaundice, and lower extremity swelling. Physical examination was notable for lower extremity scabbed black lesions. Model for End-Stage Liver Disease with sodium (MELD-Na) score was 34, and he was admitted for liver transplant evaluation. Admission serologies demonstrated high CMV IgG/IgM titers with a positive CMV PCR, and treatment was initiated. Over the first week, a coalescing petechial rash developed around the leg wounds and gradually spread to the upper extremities. Swab cultures grew Curvularia, a facultative pathogen mold found in decaying plants. Deep tissue sampling demonstrated branching hyphae with evidence of angioinvasion, and cultures

confirmed Curvularia, prompting antifungal therapy initiation. Immunodeficiency work-up was negative for known conditions but was notable for low CD4, CD19, CD16+56+ counts, and low total IgG (normal on admission). The patient developed a productive cough, and chest imaging demonstrated multifocal pneumonia. Bronchoalveolar lavage was positive for PJP. The patient's clinical condition progressively deteriorated, requiring transfer to the intensive care unit for continuous renal replacement therapy and mechanical ventilation. Ultimately, after a two-month-long hospitalization, the patient passed away from sepsis-associated multi-organ failure (Figure).

Discussion: Herein, we present a case of severe immune dysregulation attributed to ESLD. The multiple concomitant opportunistic infections and observed immune abnormalities support global immune dysfunction. Notably, this is the first description of diffuse curvularia infection in the setting of cirrhosis. This case demonstrates the spectrum of CAID, which can be severe enough to mimic profound immunodeficiency states. More research is called for to define the incidence of various opportunistic infections and elucidate the liver's role in maintaining immune homeostasis in health and disease.



[2950] **Figure 1.** A. Picture of lower extremity lesions. Diffuse lower extremity confluent petechial rash with necrotic lesions covered by black eschar (white arrows). The (*) demonstrates the site of deep tissue biopsy sampling. B. Representative lesions on the hand. C. H & E stain of deep tissue biopsies demonstrating branching hyphae with angioinvasion (white arrows). D. Chest computed tomography (lung window) demonstrating multifocal bilateral ground-glass opacities.

S2951

Case Report of Community Acquired Methicillin-Resistant *Staphylococcus aureus* Liver Abscess in a 38-Year-Old Immunocompetent Male With No Comorbidities

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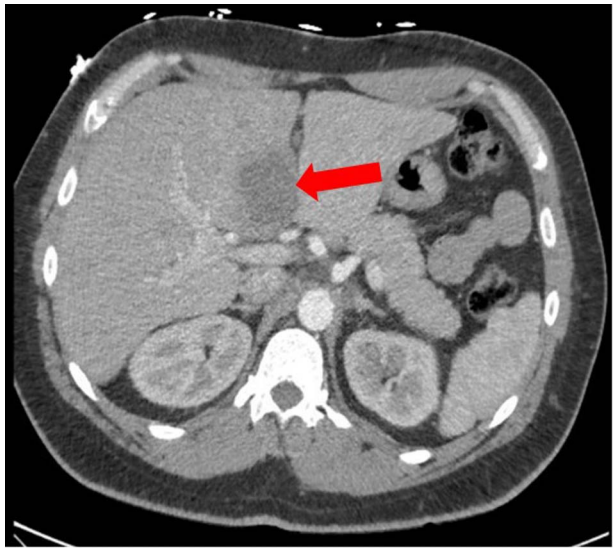
[1]St. Luke University Health Network, Easton, PA; [2]St. Luke's University Health Network, Bethlehem, PA; [3]BJ Medical College, Easton, PA.

Introduction: Pyogenic liver abscess (PLA) is a rare entity with annual incidence between 2.3 to 3.6 per 100,000 population in United States and Canada. Less than 10% of all PLA infections are caused by Staphylococcus aureus and very rarely by community acquired methicillin-resistant Staphylococcus aureus (MRSA). Herein, we present a rare case of PLA in a young immunocompetent 38-year-old man with no comorbidities, to our knowledge is only the third case of community-acquired MRSA reported in the United States.

Case Description/Methods: A 38-year-old Caucasian man with past medical history of hypertension presented with 3 days of fever, jaundice, and right upper quadrant (RUQ) abdominal pain. He denied any recent travel, diarrhea, weight changes, alcohol, or intravenous drug history. He was immunocompetent and non-diabetic. He reported having leg infection treated with oral antibiotics 2 months prior to presentation. Physical examination showed presence of fever, jaundice, and RUQ tenderness. Blood tests revealed neutrophilic leukocytosis, elevated bilirubin and CRP, normal liver enzymes and alkaline phosphatase and sterile blood cultures. Imaging revealed 4.4 x 2.7 x 3.1 cm right liver lobe abscess (Figure 1) without any intra-abdominal source of infection. He was managed with Ultrasound (US) guided percutaneous drainage along with intravenous (IV) daptomycin followed by oral doxycycline for a total duration of 8 weeks based on cultures and sensitivities of aspirates. Transthoracic echocardiogram did not show any vegetations. He was found to have complete resolution of abscess with significant improvement in general condition on follow-up imaging.

Discussion: PLA is an uncommon cause of hospitalization and potentially life-threatening disease caused by enteric and anaerobic species of bacteria. Risk factors like diabetes mellitus, liver transplant, malignancy, hepatobiliary or pancreatic diseases are typically present. Men are usually more affected with predominant involvement of right lobe of liver. The mode of pathogenesis is usually by direct liver injury or hematogenous spread of bacteria via portal vein and rarely through hepatic artery. Symptoms like fever, chills, RUQ pain with lab tests showing low albumin, elevated liver enzymes, bilirubin, and leukocytosis commonly present. CT with contrast or US are the imaging modality of choice, these can also be used for image guided treatment. First line therapy continues to be IV vancomycin or daptomycin with total duration of antibiotic for around 4-6 weeks.



[2951] **Figure 1.** CT scan shows Hypo enhancing lesion in right hepatic lobe (red arrow).

S2952

Cefepime-Induced Liver Injury

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Introduction: Drug induced liver injury can be a result of many medications and remains as one of the most challenging disorders faced by GI specialists. The most common causes in the Western world include antimicrobials, antiepileptics, anticancer medications, herbal and dietary supplements. It is important to make the distinction between intrinsic and idiosyncratic types of DILI. Intrinsic DILI is capable of causing injury in a predictable pattern in humans when given in high doses. Idiosyncratic DILI only affects susceptible individuals and has less of an association to dosing. The latter being the more difficult type to diagnose and treat. An important value, the R-factor, is used to define hepatotoxicity injury patters. An R-factor < 2 suggests a cholestatic pattern, R-factor > 5 suggests a hepatocellular pattern, and in between a mixed pattern of injury. Early withdrawal of the offending agent is the treatment of choice for any cause of DILI as this prevents progression of acute liver failure.
Case Description/Methods: We present a case of a 61 year old male (S, J) with acute elevations in his liver enzymes and alkaline phosphatase. Patient was admitted for alcohol withdrawal. The following day, patient was intubated secondary to hypercapnic respiratory failure and days later developed ventilator associated pneumonia for which he was started on antimicrobial therapy, cefepime and vancomycin. After 9 days of receiving cefepime, patient's alkaline phosphatase and liver transaminases acutely increased. Patient's baseline levels were normal on arrival to the hospital. His lab values were the following: ALT 263, AST 507, and alkaline phosphatase 279 with an R factor of 2.8 showing a mixed injury pattern. A right upper quadrant ultrasound showed hepatomegaly and an acute hepatitis panel was ordered which resulted negative. No episodes of significant hypotension were reported. The antibiotic was immediately discontinued with resolution of patient's levels back to normal.
Discussion: Though not as commonly reported, increased ALT, AST or alkaline phosphatase can be a result of cefepime use. Our patient on admission did not have abnormal liver chemistry tests despite his history of alcohol use. R-factor for our patient showed a mixed pattern of liver injury. First line tests when suspecting DILI such as acute viral hepatitis serologies and imaging studies were ordered. Our patient was not re-started on cefepime and his liver enzymes returned to baseline.

S2953

Disseminated Histoplasmosis: Extra-Pulmonary Hepatic Histoplasmosis and HLH!

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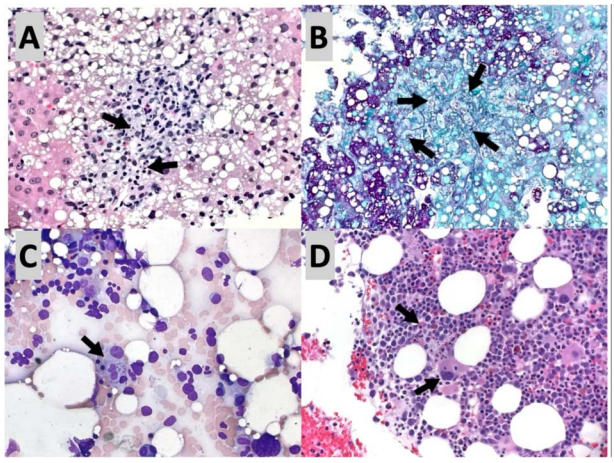
Introduction: Histoplasmosis is the most prevalent endemic mycosis in the United States. While most infections are asymptomatic or self-limiting, some individuals develop acute pulmonary infections or severe and progressive disseminated infections. Disseminated histoplasmosis (DH) is a progressive extra-pulmonary infection. Liver is rarely the primary site of infection without evidence of pulmonary disease. Most patients who develop DH are immunosuppressed (eg, AIDS, solid organ transplantation, treatment with tumor necrosis factor-alpha inhibitors) or are at the extremes of age. DH very rarely presents with severe acute liver injury or hemophagocytic lymphohistiocytosis (HLH).
Case Description/Methods: Our subject is a 61 year old female with past medical history significant for rheumatoid arthritis treated with a TNF-alpha inhibitor who presented with few weeks history of fatigue, night sweats and fever. The patient was found to be in acute liver failure on admission to the hospital. She also developed cytopenias and was found to have elevated elevated IL-2 receptor levels raising the concern for HLH. A liver biopsy was obtained and showed acute hepatitis and marked steatosis. Scattered foci of lobular inflammation were also noted and with vaguely granulomatous appearance. Special stains for fungal organisms (PAS, GMS) showed numerous budding yeasts, consistent with histoplasmosis (Figures A&B). A bone marrow biopsy was noted with frequent hemophagocytic histiocytes and rare monocytic cells with intracytoplasmic inclusion bodies consistent with presumed HLH and histoplasmosis (Figures C&D). The patient was started on treatment with antifungals soon after diagnosis, with gradual improvement in liver function and blood counts and eventual normalization.
Discussion: Disseminated histoplasmosis presenting as granulomatous liver disease is rare. Hepatic histoplasmosis as the primary manifestation of disseminated disease and with no evidence of pulmonary involvement is rarely seen. Patients may present with stigmata of chronic liver disease, portal hypertension, ascites, varices, and occasionally with severe liver injury leading to acute failure as shown in this patient. Histoplasmosis-associated HLH is an uncommon disorder for which data are limited regarding the optimal treatment and clinical outcomes in adults. Providers should keep a high index of suspicion for this uncommon manifestation of fairly common infections in patients who present with cryptogenic liver injury and fever, specially in endemic areas.



[2953] **Figure 1.** A- H&E stained section of liver demonstrating lobular inflammation consisting of lymphocytes, macrophages, and eosinophils in a vaguely granulomatous pattern with surrounding steatosis. Although challenging to interpret, hemophagocytosis is identified within the inflammation (arrow) B- PAS stain of the liver demonstrating numerous budding yeast forms surrounded by granulomatous inflammation, consistent with histoplasmosis. C- Bone marrow aspirate: More readily identifiable in the bone marrow aspirate sample, hemophagocytosis is easily appreciated (arrow) D- Bone marrow clot: More readily identifiable in the bone marrow clot sample, hemophagocytosis is easily appreciated (arrow).

S2954

COVID-19 Infection as a Trigger for Seronegative Autoimmune Hepatitis: A Case Report

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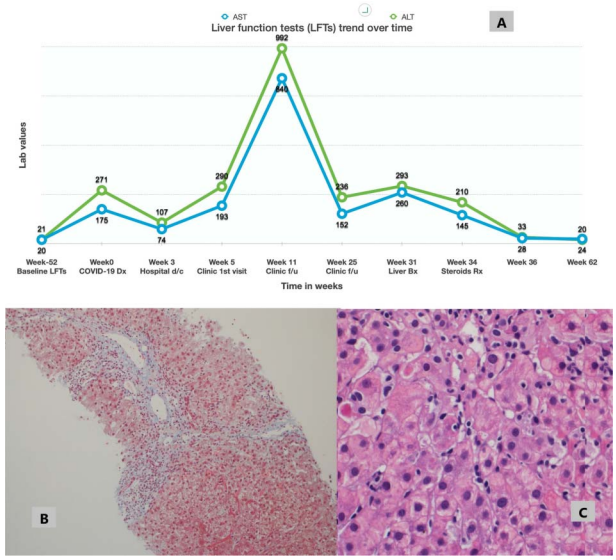
Introduction: Autoimmune hepatitis (AIH) is an immune-mediated liver disease that commonly develops in a genetically predisposed patient after exposure to an environmental trigger. These triggers include viruses, immunization and drugs. Both COVID-19 infection and vaccination have been linked to the development of AIH.
Case Description/Methods: A 58-year-old male patient, known to have vitiligo and mild rheumatic mitral stenosis presented to our hospital complaining of flu-like symptoms. The patient denied any history of alcohol intake, substance abuse, over-the-counter or herbal medications, or family history of liver or autoimmune disease. His chronic medications included aspirin and bisoprolol. He tested positive for COVID-19 PCR, so he was admitted for observation. Chest X-ray showed no abnormality, and he was not started on any medication. His laboratory investigations (Table 1) were remarkable for ALT of 271 and AST of 175 U/L. During hospitalization, liver enzymes were trending down but remained elevated. The hepatitis workup (Table 1) was notable for an elevated IgG level of 16.9 g/l (normal range 7-16). His radiological

investigations included US abdomen that revealed mildly increased echotexture, MRI liver that was only remarkable for tiny cyst 2 mm, and liver elastography that showed a stiffness average of 5.77 kPa indicating mild stiffness. The patient was offered a liver biopsy, but he was reluctant. During follow-up, his LFTs remained elevated but were fluctuating (Figure 1a). On week 31, he finally agreed for liver biopsy that showed moderate interface and portal tract inflammatory cell infiltrate composed mainly of lymphocytes with occasional eosinophils (Figure 1b,c). The histopathological findings were suggestive of AIH. On week 34, he was started on prednisone 40 mg. LFTs on week 36 were completely normal. The prednisone was tapered over the following two months. Six months later, repeat LFTs were also normal.

Discussion: Our patient had an elevation of LFTs after COVID-19 infection that persisted for weeks and promptly responded to steroids therapy. Drug-induced liver injury was less likely as he didn't receive any hepatotoxic medications prior to or during his hospitalization. In our patient, the Revised Original Score for Autoimmune Hepatitis (AIH) was 17, indicating definite AIH. The proposed mechanism of AIH development after COVID-19 infection is the molecular mimicry between spike protein S1 and multiple human tissue proteins. More studies are needed to examine this association.



[2954] **Figure 1.** a: Trend of LFTs over time. b,c: histopathological images showing interface and portal tract inflammation. Abbreviations alphabetically: Bx: biopsy - d/c: discharge - Dx: diagnosis - f/u: followup - Rx: treatment

Table 1. shows extensive hepatitis workup

Laboratory test	result	Reference
ALT	271	0-40 U/L
AST	175	0-37 U/L
ALP	38.9	40-129 U/L
Antinuclear ab	Negative	Negative
Anti Mitochondrial ab	Negative	Negative
Anti Mitochondrial M2 ab	Negative	Negative
Anti Smooth Muscle ab	Negative	Negative
Anti Liver Kidney Microsomes	Negative	Negative
Hepatitis B Core ab	Reactive	non-reactive
Hepatitis B Core Ab IgM	non-reactive	non-reactive
Hepatitis B Surface Antigen	Non-reactive	Non-reactive
Hepatitis B e Ab	Non-reactive	Non-reactive
Hepatitis B e Antigen	Non-reactive	Non-reactive
Hepatitis A Ab IgM	Non-reactive	Non-reactive
Hepatitis C Ab	Non-reactive	Non-reactive
Hepatitis E IgG	Reactive	Non-reactive
Hepatitis E IgM	Non-reactive	Non-reactive
Herpes Simplex virus Ab IgM	Non-reactive	Non-reactive
Parvovirus Ab IgM	Non-reactive	Non-reactive
Alpha-1 anti-trypsin	37	20-53 μmol/L
Ceruloplasmin	33	14 to 40 mg/dL
CMV PCR	Negative	Negative
EBV PCR	Negative	Negative
HBV PCR	Negative	Negative

S2955

Drop-It: A Quest to Reduce Wine Sulfites Leads to Drug-Induced Liver Injury

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Introduction: Drug-induced liver injury (DILI) accounts for 50% of acute jaundice and 10% of acute liver failure. Most cases are idiosyncratic rather than intrinsic. Drop-It (hydrogen peroxide, sunflower lecithin, natural egg white protein) is a supplement marketed for reducing wine sulfites/tannins. To date, DILI due to Drop-It or its contents has never been described.
Case Description/Methods: A 39-year-old healthy male presented with four days of painless jaundice, fatigue, dark urine, and acholic stools. He consumed 1-3 glasses of wine daily, having used Drop-It for "months" to curb "wine headaches", and had lost 15 lbs over 9 months. None of his medications were known hepatotoxins (multivitamin, co-enzyme Q, magnesium, vitamin D). He denied any chronic liver disease, though 4 months prior had an aspartate aminotransferase (AST) 73, alanine aminotransferase (ALT) 74, alkaline phosphatase (ALP) 138, and total bilirubin (TB) 0.8. Exam showed a flat nontender abdomen, no edema, jaundice, and no asterixis. Initial labs showed AST/ALT 1452/972, ALP 211, TB 10.8 (direct fraction 7.7), and prothrombin time 17.9. Viral/autoimmune hepatitis, hereditary hemochromatosis, Wilson's, primary biliary cholangitis, and alpha-1 anti-trypsin deficiency were ruled out. Liver dopplers showed steatosis and patent perihepatic vessels. Magnetic resonance imaging was unremarkable with no ascites. Liver biopsy reviewed by internal and external pathologists showed "cholestatic hepatitis consistent with DILI"; "mild steatosis, macrovesicular and microvesicular"; and grade 1 hepatocellular hemosiderosis. A second external pathologist found "no ballooning degeneration", making alcoholic hepatitis less likely, and agreed that "consideration for a drug or toxin related injury may be given". He received N-acetylcysteine infusion and supportive care. On day 6, he was discharged with alcohol counseling and Drop-It cessation. 6 weeks post-discharge, his labs normalized to AST/ALT 22/21, ALP 77, TB 0.5.
Discussion: Given the temporal association between drug exposure and acute liver injury, then resolution of injury with cessation, it seems highly likely that Drop-It was the offending agent. Whether there was an idiosyncratic or intrinsic DILI to Drop-It's listed ingredients, or to its undisclosed inactive agents, remains unclear. This first published report of DILI related to Drop-It underscores caution in monitoring herbal and dietary supplement use, the second most common cause of DILI in the US.

S2956

Delayed Onset Drug-Induced Acute Liver Failure Caused by Glatiramer Acetate (GA) in Multiple Sclerosis Requiring Liver Transplantation

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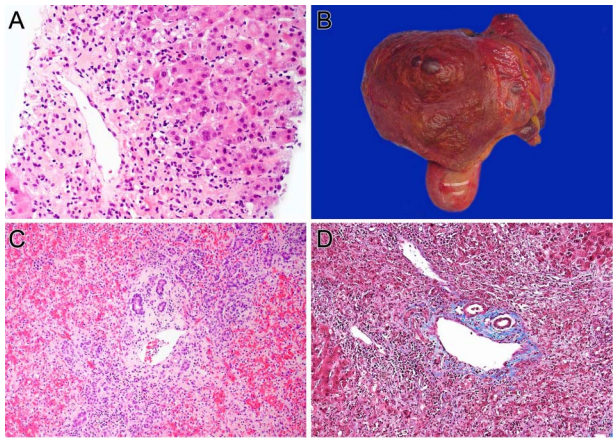
Introduction: Glatiramer acetate (GA) has been used for multiple sclerosis (MS) since 1996. Regular liver function test monitoring is not required for the medication because there have not been any reported cases of liver toxicity.[1]Here, we report the first case of delayed onset GA-induced ALF requiring liver transplantation.
Case Description/Methods: A 59-year-old woman with well-controlled MS presented to the emergency department with three weeks of jaundice and dark-colored urine. Initial labs showed AST 2527 IU/L (81x ULN), ALT 2512 IU/L (81x ULN), total bilirubin 22.7 mg/dL (19x ULN), ALP 210 IU/L (2x ULN), INR 2.65. Further workup showed normal ceruloplasmin, alpha-1-antitrypsin, and IgG. Hepatitis, HSV, VZV, T-spot, HIV, ANA, anti-mitochondrial antibody, anti-LKM antibody were negative. Anti-smooth muscle antibody was 1:40. Her liver enzymes were normal one year prior to presentation, and she had no family history of autoimmune or liver disease. Computed tomography abdomen/pelvis showed patent vasculature and no evidence of cirrhosis. MRI/MRCP showed no biliary obstruction. Her only medication was GA, which she had been taking since 2008. Physical exam was notable for mild asterixis, and she was started on IV N-acetyl cysteine for 3 days. Liver biopsy demonstrated submassive necrosis (70%) consistent with drug induced liver injury (Fig. 1A). GA was thought to be the most likely etiology of her liver injury and was held. Liver transplant was ultimately deferred because her liver enzymes and her mental status improved. She was discharge on day 6. Four days later she developed severe abdominal pain, lethargy and re-presented to the hospital. She was slow to respond, had scleral icterus and asterixis with AST 909 IU/L, ALT 839 IU/L, total bilirubin 27.2 mg/dL, INR 2.94. The patient underwent expedited liver transplant evaluation and was listed on the same day. She successfully underwent deceased donor liver transplant three days later. Liver explant showed mixed zone 1 and 3 liver necrosis (50%) with associated inflammation, mild lobular inflammation and cholestasis (Fig. 1B-D).
Discussion: Our case illustrates the first case of GA-induced ALF requiring liver transplantation. Previous reports showed that patients with GA drug-induced-liver injury recovered completely in 1-5 months after drug withdrawal and time of presentation ranges from 1 to 8 months, as opposed to 14 years in our case. Patients on GA should have long term regular liver monitoring.



[2956] **Figure 1.** Histological analysis of pre-transplant and explant liver. A. Hematoxylin and eosin (H&E) stain of the pre-transplant biopsy showed extensive necrosis including zone 3 (x100). B. Gross picture of explant (weight 500.3 gram). The surface showed wrinkles, which were characteristics of acute liver failure with massive hepatocyte necrosis. C. H&E stain of explant showed no viable hepatocytes in the worst area. (x200). D. Masson trichrome stain showed no fibrosis, which confirms the acute process (x200).

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S2957

Cytarabine-Induced Hyperbilirubinemia in a Pregnant Patient With Acute Myeloid Leukemia

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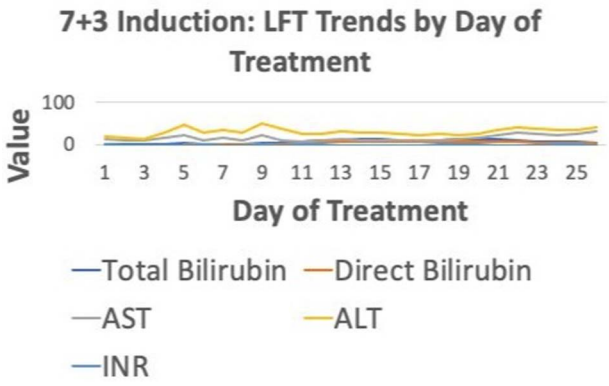
Introduction: We present a case of a 33-year-old pregnant patient with inversion 16 Acute Myeloid Leukemia (AML) that developed cytarabine-induced hyperbilirubinemia without other signs of synthetic liver dysfunction.
Case Description/Methods: A 33-year-old patient, G2P1, at 19 weeks gestation with a medical history of gestational hypertension presents with a chief complaint of dyspnea on exertion over the past four days. In the emergency department, laboratory findings were notable for a white blood cell count of 86.6, hemoglobin of 5.5, and a platelet count of 18. Bone marrow biopsy was performed and confirmed the diagnosis of inversion 16, +22 AML. Once on the hematology service, maternal fetal medicine (MFM) was consulted for assistance regarding contraindications to chemotherapy. A chemotherapeutic regimen consisting of daunorubicin 60 mg/m^2 and cytarabine 100 mg/m^2 was chosen. On Day 5 of treatment, the patient's total bilirubin was above 2 mg/dL for the first time and coincided with the development of jaundice. This prompted a right upper quadrant ultrasound that revealed mild non-obstructive cholestasis. The total and direct bilirubin continued to rise to a peak of 13.4 mg/dL and 8.5 mg/dL, respectively, on Day 14 (Figure 1). There were no associated rises in INR. Total and direct bilirubin continued to downtrend until discharge on day 32, after adequate recovery of neutrophils (Absolute neutrophil count $>$ 1000) and platelets. The patient received four more maintenance cycles of high dose cyatarbine (HiDAC) at a reduced dose in the setting of hyperbilirubinemia during the induction phase. Mild elevations in total and direct bilirubin that normalized after each cycle were observed. Most recent bone marrow biopsy after maintenance cycles revealed no evidence of disease.

Discussion: In this report, a pregnant patient exhibited cytarabine induced isolated hyperbilirubinemia during induction chemotherapy without any other evidence of liver dysfunction; in addition, decreasing cytarabine dosage during the consolidation phase resulted in lower elevations of bilirubin. Our findings add to the existing literature on cytarabine induced hyperbilirubinemia by highlighting its effects in pregnancy, while also showing that dose adjustments can be utilized not only to prevent high bilirubin levels but to achieve clinically significant outcomes.



[2957] **Figure 1.** Laboratory trends during chemotherapeutic induction phase with Cytarabine and Daunorubicin. Peak elevation in total and direct bilirubin can be seen on Day 14.

S2958

Do Not Trust the Internet: Acute Liver Failure Secondary to DILI From Online Hormones Versus Stimulant Use in a Transgender Patient

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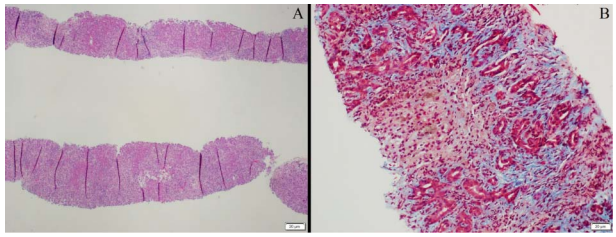
Introduction: Approximately half of acute liver failure cases in the United States are from drug-induced liver injury (DILI), a process modulated by the interplay between host, environment, and agent. Here, we present a case of DILI from unsupervised feminizing hormones versus stimulant use.
Case Description/Methods: A 27 year old transgender (M to F) woman with history of anxiety, depression, and attention deficit hyperactivity disorder (ADHD) presented with 3 weeks of jaundice, abdominal discomfort, acholic stools, and memory issues after consuming several drugs purchased from international websites. Due to severe anxiety, she did not seek endocrinology evaluation for hormone guidance, instead ordering estrogen and anti-androgen therapy to transition on her own. She had been on estradiol for 5 years and cyproterone acetate intermittently for 4 years, restarted 9 months prior with N-methyl cyclazodone for ADHD. All 3 agents were discontinued at the onset of jaundice, but she then began copious water and laxative intake—"to flush the bilirubin out of [her] body"—along with that of milk thistle and delta-8 THC extract. Pertinent labs included AST 485, ALT 668, T. Bili 52.8, AP 140, INR 3, and K of 2.3. Chronic liver disease workup was negative. Biopsy revealed severe hepatocyte dropout with associated marked cholestasis and mixed portal inflammatory cell infiltrate including eosinophils, features consistent with DILI (Figure). Hospital course involved hematemesis, spontaneous bacterial peritonitis, Ogilvie's syndrome, acute tubular necrosis requiring continuous renal replacement therapy, paraphimosis, and, ultimately, acute liver failure. Given severely compromised hepatic function with transplant the only option for viable recovery, patient was listed as category 1a with a MELD score of 37. She underwent orthotopic liver transplantation complicated by hemorrhage from a vein near the porta hepatis, which was repaired. She was started on tacrolimus, mycophenolate, prednisone, fluconazole, valganciclovir, and bactrim with recommendations to defer hormone replacement therapy for 1 year post-transplant.
Discussion: Those with gender dysphoria may turn to the internet to relieve the distress caused by mismatch between biological sex and gender identity. Unfortunately, the online market is rife with unregulated information and products. In consuming such, physician supervision is imperative, and lack thereof lead our self-medicating patient to incur multiorgan insult, notably dire hepatic dysfunction requiring transplant.



[2958] **Figure 1.** Patient's histopathology with hematoxylin and eosin staining (A) versus trichrome staining (B) demonstrating severe hepatocyte dropout with inflammatory cell infiltrate.

S2959

COVID-19-Induced Liver Injury: A Rare Cause of Acute Cholestasis in a High Risk Population

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Introduction: Within the COVID-19 pandemic, hepatic dysfunction is common in the elderly population and ICU patients. Hepatic injury may be multifactorial including direct cytopathic effects after entry via host angiotensin-converting enzyme 2 (ACE2) receptors which are highly expressed in cholangiocytes. We describe an 82 year old female presenting with sudden jaundice and pruritus with marked elevation in bilirubin and transaminitis secondary to COVID-19 infection.
Case Description/Methods: Patient presented with sudden jaundice and pruritus without respiratory distress, pain, fevers, or bowel movement changes. She tested positive for COVID-19 however didn't require supplemental oxygen, steroids or antiviral therapy. Notable labs included AST 260 IU/L, ALT 260 IU/L, ALP 1015 IU/L, total bilirubin 28 umol/L, direct bilirubin over 15 umol/L and INR 1.03. All toxicology was negative. ANA was positive at 1:320, anti-smooth muscle antibody level 22 and normal total IgG. Viral hepatitis panel, EBV, CMV, TTG, AMA were not detected. MRI/MRCP demonstrated mild fatty liver, with normal gallbladder and pancreas without biliary dilatation or obstruction. Liver biopsy demonstrated cholestatic changes including bile plugging, portal fibrosis, and scattered inflammatory cells. She was prescribed cholestyramine and Ursodeoxycholic acid and her condition was self limiting with continued monitoring as an outpatient.
Discussion: Liver injury ranges from 2.5-76.3% of all COVID-19 cases. In vitro studies identify that coronavirus enters host cells via the ACE2 host receptor. Immunohistochemistry shows high ACE2 expression in vascular endothelium, type 2 alveolar cells, gastrointestinal tract and cholangiocytes. COVID-19 liver injury is likely multifactorial, including direct viral cytopathic injury, hypercoagulation and thrombosis in the porta-hepatic system, and hypoxia-induced reactive oxygen species. Innate immune response dysregulation contributes to pulmonary and extrapulmonary injuries and hepatotoxic agents such as antivirals are known to also cause drug-induced liver injury (DILI). COVID-19 liver histology shows moderate microvesicular steatosis, mild inflammatory infiltrates in the hepatic lobule and portal tract with instances of portal fibrosis and acute liver necrosis, as seen in our patients pathology report. Covid-19 liver injury is mainly self limiting however risk of severe injury have been seen in elderly and the critically ill.

S2960

Diagnosing Wilson Disease: Perseverance Is a Virtue

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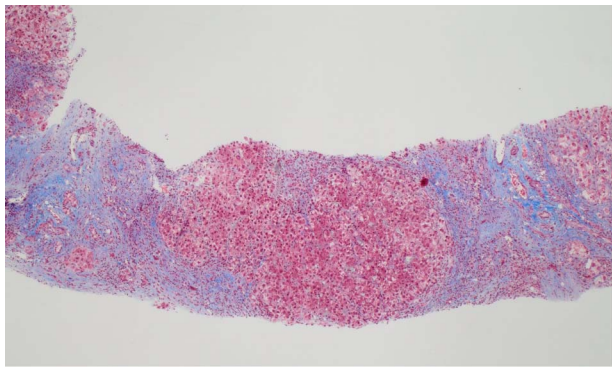
Introduction: Wilson disease is an autosomal recessive disorder with constellation of hepatic,neurologic and psychiatric symptoms.Prevalence is varies ranging from 1:30,000 to 1:66,000 patients with 15% of patients presenting with isolated hepatic dysfunction.We present a case of a male with decompensated cirrhosis found to have Wilson disease via cumulative copper staining on liver biopsy
Case Description/Methods: 35-year-old male presented with decompensated cirrhosis.Previously diagnosed at outside hospital with etiology presumably non-alcoholic steatohepatitis(NASH) given body mass index 48,unremarkable alcohol use,viral,genetic,and autoimmune labs.On admission, AST 318, ALT 160, alkaline phosphatase 180, total bilirubin 16, and MELD-Na 25.Labs revealed ceruloplasmin level 27.4mg/dL(normal 20-60), negative slit lamp for Kayser Fletcher(KF) rings, and 24-hour urine copper 137mcg/day(normal 15-60).Given the presumed diagnosis of decompensated cirrhosis secondary to NASH, the elevated transaminases were unusual.Transjuglar liver biopsy showed marked cholestasis, hepatocyte injury, moderate portal/septal inflammation and stage 4 nodule formation.After discussion at liver disease meeting, a trial of prednisone and ursodiol was started for possible autoimmune hepatitis.Given concern for other etiologies, cumulative copper stain on liver biopsy revealed copper level 531mcg/g(normal < 50).Prior to starting copper chelating agent, patient was readmitted with MELD-Na 32 and ultimately underwent a successful liver transplant.He has followed up in clinic without complications (Figure).
Discussion: Wilson disease should be considered in adults with NASH < 35 years old,AST:ALT ratio >2,significant family history,or neurologic/psychiatric symptoms.Initial work up includes serum ceruloplasmin levels, slit lamp exam,and 24-hour urine copper excretion.Liver biopsy is recommended when there is isolated 24-hour urine copper elevation.KF rings are present in 90-99% with neurologic symptoms but 50% patients with hepatic dysfunction.Leipzig scoring system uses clinical and laboratory findings with 4 points establishing diagnosis.Our patient met criteria with 24 hour urine copper >2 times and total liver copper >5 times, upper limit of normal.Cumulative copper level on liver biopsy >250mcg/g has an 83% sensitivity to diagnosis Wilson disease.Our case demonstrates the need for further evaluation in patients with newly diagnosed cirrhosis of unclear etiology as Wilson Disease can be diagnosed without KF rings and normal ceruloplasmin level.



[2960] **Figure 1.** Sections show marked cholestasis with associated hepatocyte swelling. Large septal areas show bile ductular reaction with mixed septal inflammation. Trichrome stain highlights cirrhotic nodules (stage 4/4).

S2961

Delayed Hepatitis Following COVID 19 mRNA Vaccine and an Accidental Booster

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Parkview Medical Center, Pueblo, CO.

Introduction: Covid 19 vaccination has become mandatory in many if not all healthcare institutions ; it is our possible gateway out of this pandemic however a small number of patients have developed unpleasant to life threatening adverse reactions. We share with you a case of acute onset of hepatitis with symptoms lasting for 4-5 weeks following covid 19 initial vaccine and an accidental booster dose.
Case Description/Methods: 32 yr old healthy male, physician by profession, finally decided its time for Pfizer/BioNTech BNT162b2 mRNA vaccine ; 3 days later patient experienced acute onset of lethargy weakness, nausea, loss of appetite and subjective fever. These symptoms resolved within a day only to be followed by acute onset of right upper quadrant abdominal pain which progressively worsened over the next 2 weeks when patient finally got a RUQ US and LFTS. RUQ US was positive for hepatomegaly; LFTs reported AST 212 , ALT 446 , GGT 101 - > lfts further peaked at day 20 with ALT at 622 ,AST 232 and slowly resolved over the next week along with resolution of pain. Patient had the booster after 2 months with negligible symptoms. However 2 months later (4months from the initial dose) patient went into a pharmacy to obtain a Flu vaccine when he was accidentally adminsistered a dose of covid 19 BNT162b2 mRNA vaccine . Following this dose; patient had similar pattern of profound weakness, lethargy and body pains on day 2 with and similar pattern of delayed lft elevation and RUQ pain that would last over the next 4-5 weeks. Complete hepatic workup including acute hepatitis panel, EBV, CMV, autoimmune markers ANA, anti-smooth muscle antibodies, Anti-liver/kidney microsomal antibodies, IgG were found to be negative. Patient has never had alcohol and did not take any OTC or herbal medications. It has been over a year after the last dose of the vaccine and patient has not had any symptoms thereafter.
Discussion: With 2 instances of similar pattern of symptoms and liver injury and following vaccine administration, it seems unlikely to be just a coincidence and more likely an association. 2 - 3 weeks is the optimal time at which immune system is at its peak following vaccination and also the exact time when the patients lfts peaked. Can it be speculated that vaccine may be triggering an autoimmune response through cross reactivity leading to liver injury? We hope to raise awareness of this potential side effects which could be benign and self resolving but cause unrelenting pain for up to 4-5 weeks following vaccination.

S2962

Crypto Can Be Confusing: Cryptogenic Cirrhosis Occurring in Isolated Heterozygous Alpha1 Antitrypsin Deficiency

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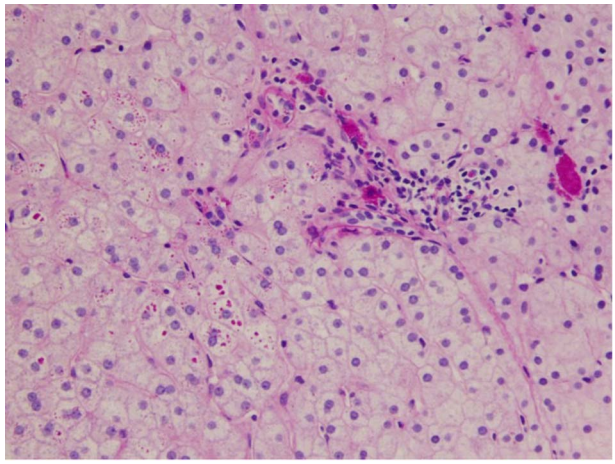
Introduction: Alpha 1 Antitrypsin deficiency (AATD) is an autosomal co-dominant disorder involving the SERPINA1 gene occurring in individuals who inherit an gene from each parent. The homozygous ZZ variant is the well-known variant linked to severe emphysema, chronic hepatitis, and decompensated cryptogenic cirrhosis. This is a case of decompensated cirrhosis occurring in a heterozygous M, Z individual. The insidious onset, rapid progression, and lack of potentiating etiologies and risk factors add to the uniqueness of this case.
Case Description/Methods: Our patient is a forty-seven-year-old non-smoker, non-alcoholic, female with a history of menorrhagia and well-controlled hypertension. The patient reports to the clinic with complaints of abdominal distention, intermittent brain fog, confusion, and altered sleep cycles. The patient was admitted to evaluate and treat new-onset decompensated cirrhosis. Initial imaging revealed mild parenchymal changes and subtle nodular changes suggestive of Cirrhosis. Additionally, labs were negative for common infectious, metabolic, immunological, and genetic etiology. A biopsy revealed fibrotic changes consistent with cirrhosis, along with patchy intracytoplasmic inclusions of PAS-D positive spherical globules in hepatocytes consistent with alpha 1 antitrypsin deficiency. Additional testing for alpha 1 antitrypsin revealed an MZ genotype, and the serum levels of AATD were within the normal range. Symptoms or signs indicative of lung disease were never present. The patient was followed by transplant hepatology. Progression of disease was evidenced by Model for End-stage Liver Disease scores worsening from nine to seventeen four months after diagnosis. A successful transplant of the liver was completed five months after the initial diagnosis. There haven't been any signs of rejection in the first six months post-transplant (Figure).
Discussion: Alpha 1 antitrypsin (A1AT) deficiency is an underrecognized and underdiagnosed disease due to its profound clinical variability. Patients present during infancy or adulthood, and the effect of disease modifiers, such as smoking, alcohol, hormonal effects, and allele variability are not thoroughly understood. Additionally there's limited knowledge regarding occurrence of disease processes in

heterozygous individuals. Individuals arent routinely screened for A1AT, however a biopsy may aide in arriving to the correct diagnosis. Early detection, allows for early transplant evaluation, genetic counseling and education.



[2962] **Figure 1.** The biopsy shows a core liver biopsy with overall preserved architecture and few portal tracts seen. PAS with diastase highlights a very patchy distribution of bright, magenta-colored globules in mainly periportal hepatocytes (black arrows).

S2963

Congenital Riedel's Lobe of the Liver: A Case Report

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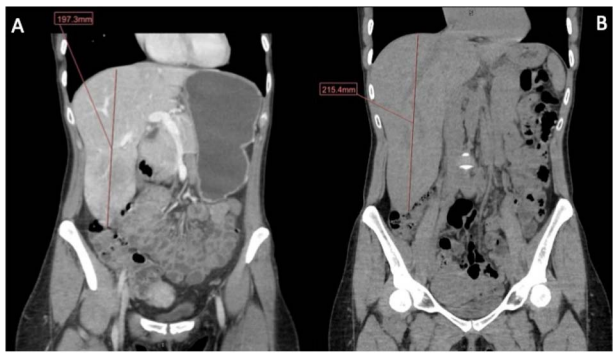
Introduction: Riedel lobe of the liver is a rare anatomical variation with a reported incidence to be between 3.3% and 14.5%. We report a case of a 43-year-old female with an incidental finding of non-palpable Riedel's lobe.
Case Description/Methods: A 43-year-old female was referred for evaluation of hepatomegaly, which was revealed on MRI and CT scan dating back to 2016. Medical history notable for Irritable Bowel Syndrome (IBS), uterine fibroids, and a history of a tumor removal from her right breast. Patient denies any history of alcohol, illicit drugs, hepatotoxic medications, or pre-existing liver disease. Physical exam was unremarkable and abdominal exam did not reveal any mass or abnormalities. Routine blood examination, including LFTs and iron studies, was within normal limits. Hepatitis panel (A, B, C), anti-smooth muscle antibody, and LKM-1 IgG antibody was negative. ANA, alpha-1 antitrypsin, and tissue transglutaminase were all negative. The only lab abnormality was an elevated IgG mitochondrial M2 antibody 54.5 (normal less than 20 units). CT abdomen was significant for an enlarged liver with the right lobe extending into the pelvis and 'not completely included in the study'. MRI abdomen revealed a markedly enlarged liver measuring up to 23.8cm in its craniocaudal dimension with extension into the pelvis with the pancreas deviated to the left, likely secondary to the prominent hepatomegaly. Venous duplex significant for normal directional flow in the portal and hepatic veins with no evidence of portal hypertension. Liver biopsy revealed signs of sinusoidal dilatation nonspecific for veno-occlusive outflow obstruction with no signs of inflammation, steatosis, or fibrosis. The patient was diagnosed with Riedel's lobe of the liver. She was discharged from the hospital without treatment with a recommendation to repeat an MRI in 1 year, as torsion is a reported complication of Riedel's lobe over time. Patient will be recommended to repeat LFTs and anti-mitochondrial antibody to determine progression/significance prior to follow up in 6 months.
Discussion: Riedel's lobe of the liver is a rare anatomical variant that is often incidentally found on imaging or the presence of hepatomegaly on physical exam. Although patients are usually asymptomatic, its presentation can vary, ranging from nonspecific symptoms to more severe symptoms such as torsion, obstruction, rupture, and bleeding. The range of symptoms highlights its importance in diagnosis and surveillance in this patient population.



[2963] **Figure 1.** (A) CT of the abdomen/pelvis from 2016. Liver measuring up to 197.3mm in the sagittal plane. (B) CT of the abdomen/pelvis from 2020. Liver measuring up to 215.4mm in the sagittal plane.

S2964

COVID-19 Vaccination May Increase the Risk of Autoimmune Hepatitis in Patients With Underlying Autoimmune Disease

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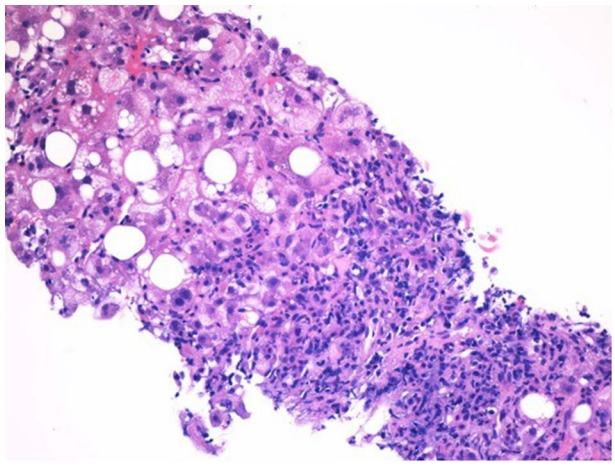
Introduction: It is established that COVID-19 viral infection is associated with many autoimmune processes, especially in predisposed patients, such as autoimmune hepatitis AIH [1,2]. Molecular mimicry is one of the suggested mechanisms behind such phenomena. mRNA COVID-19 vaccination can plays the same role as the infection does.
Case Description/Methods: 55 year old Hispanic male with ulcerative colitis on home sulfasalazine who presented to the ED with complaints of a vague, non-cramping, and dull RUQ abdominal pain for 3 weeks duration. Negative ROS. The patient's social history is unremarkable. No recent travel, occupational exposures, herbal supplements use, or acetaminophen use. He received 2 doses of Pfizer COVID -19

vaccines , last dose was around 21 days before admission. VS were normal, physical examination was remarkable for jaundice and mild RUQ tenderness. laboratory tests were remarkable for: Liver Function Tests: AST/ALT 1621/1476 units per liter (U/L) , T. bilirubin 6.0 milligrams per deciliter (mg/dL), D.bilirubin 4.5 (mg/dL), Alkaline phosphatase 167 (U/L) , GGT: 339 , ESR: 99 mm, CRP: 3.40 (mg/dL). Abdominal ultrasound and MRI were unremarkable. EUS showed a diffuse abnormal echotexture in the visualized portion of the liver. No significant ductal pathology. The patient was treated with azathioprine and prednisone 60 mg daily with slow tapering and with continuation of his home dose sulfasalazine (Figure).

Discussion: Few case reports were reported over billions of doses of vaccine administration. The increasing number of case reports associating autoimmune processes with COVID-19 related infection and mRNA vaccines might allow for more data analysis to determine risk factors that predispose patients for AIH. It is likely that patients with pre-existing autoimmune diseases are more vulnerable to develop autoimmunity after exposure to spike-protein based COVID-19 vaccines. If more data confirms causality of COVID-19 vaccine induced AIH, this will help providers to identify patient at high risk to develop AIH as a side effect, and also may encourage scientists to find out other types of vaccines that has low/no chance of molecular mimicry.



[2964] **Figure 1.** Liver biopsy - histopathology.

S2965

Decerebrate Posturing and Convulsions: A Rare Hepatic Encephalopathy Presentation

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Introduction: Hepatic encephalopathy (HE) is an acute neuropsychiatric syndrome complicating liver failure. Posturing is a medical emergency and an uncommon sign in the setting of hyperammonemia. Our report details a patient acutely progressing, within hours, from grade 0 HE to grade IV comatose HE with posturing and convulsions hours after revision of TIPS.

Case Description/Methods: A 62-year-old Hispanic man with past medical history of treated hepatitis C infection, decompensated liver cirrhosis secondary to previous excessive alcohol use, hepatocellular carcinoma on chemotherapy, type 2 diabetes mellitus and peptic ulcer disease. He was on oral lactulose as a home medication. He underwent TIPS placement due to recurrent variceal bleeding and had no previous history of hepatic encephalopathy. He presented due to hematemesis. At time of admission he was fully alert and oriented and without focal neurological findings. Esophagogastroduodenoscopy was done and varices were banded. Forty-eight hours later he was found to have a partial thrombus in the shunt on imaging and underwent TIPS revision. Overnight the patient became acutely altered, hypertensive, tachycardic and had witnessed convulsions. He was then upgraded to the intensive care unit. Both Stat CT head and abdomen/pelvis were ordered and both revealed no acute abnormalities. Labs were significant for ammonia 387 (upper limit 38 μmol/L), lactate 8 (upper limit 2 mmol/L) and bicarbonate 13 (range 23-30 mEq/L). Physical exam revealed jerking movements and decerebrate posturing (arms/legs extended at sides with head/neck arching back) with a fixed left upper gaze. EEG was indicative encephalopathy of severe nature. Given he was already on oral lactulose from admission additional treatment with lactulose enema was begun resulting in multiple bowel movements. He had improved mentation within 1 day, allowing him to be discharged home within 4 days.

Discussion: Fortunately our patient responded well to conservative therapy with lactulose. Variceal bleed and/or recent sedation could possibly be inciting factors for his HE, however we acknowledge it is impossible to pinpoint the exact cause. Globally a rising number of patients are developing cirrhosis and its complications. There is a possibility we may see an increasing number of patients presenting with this manifestation of HE. Early recognition of posturing as a rare sign in the setting of HE can improve both early diagnosis and treatment along with decreasing excessive workup and length of hospital stay.

S2966

Cystic Fibrosis-Associated Liver Disease: A Rare Etiology of Cirrhosis

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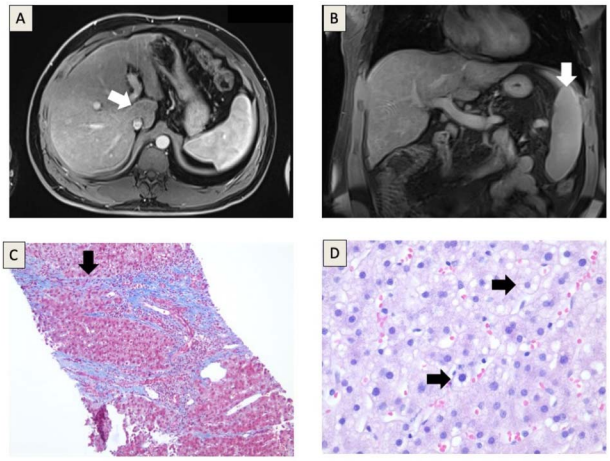
Introduction: Cystic fibrosis (CF) is a rare etiology of cirrhosis. Alteration in bile hydration and alkalinity cause bile duct injury and CF-associated liver disease (CFLD). Factors associated with rapid progression of CFLD include class I-III mutations in CF transmembrane conductance regulator (CFTR), male sex, history of pancreatic insufficiency and meconium ileus. Approach to prevention and management of CFLD is unclear in current guidelines. We present a patient with CF who progressed to cirrhosis.

Case Description/Methods: A 24-year-old man with history of CF complicated by Staphylococcal and Pseudomonal lung colonization, pancreatic exocrine insufficiency was evaluated for chronic liver disease. He was diagnosed with CF at age of 14 years with c.489+1G >T/c.579+1G >T mutations on genetic testing. Two years later, he developed cholestatic pattern of elevated liver enzymes that progressively worsened. At time of evaluation, the patient was asymptomatic with unremarkable examination. Laboratory work showed total bilirubin 1.62 mg/dL, alkaline phosphatase 259 U/L, AST 88 U/L. ALT 246 U/L and thrombocytopenia (126 x109/L). Abdominal ultrasound showed coarse hepatic echotexture and splenomegaly. MRCP revealed markedly nodular hepatic contour, splenomegaly to 17 cm, caudate lobe hypertrophy, diffuse hepatic steatosis with morphological changes of cirrhosis and fatty replacement of pancreas. Workup for other causes of liver diseases was unremarkable. Liver biopsy demonstrated regenerative nodules, prominent ductular reaction and thick biliary secretions in the lumen consistent with cirrhosis due to CFLD. He was started on ursodeoxycholic acid (UDCA) with recommendation to continue multivitamin, pancreatic enzyme supplements, dornase alfa and follow up in 6 months for hepatocellular cancer screening. (Figure) (Table).

Discussion: A high index of clinical suspicion is required for early identification of CFLD in patients with cholestatic pattern of elevated liver enzymes, male sex, class I-III mutation of CFTR, and history of pancreatic insufficiency. There is no effective treatment of CFLD. Efficacy of UDCA is controversial as it improves biochemical parameters but its effects on outcomes of CFLD remain unknown. Further, while steatosis has been described in patients with CFLD, its impact on CFLD outcomes need to be addressed in large cohorts. Primary goals in managing CFLD is close monitoring of liver function, early identification of cirrhosis, its complications and liver transplant in progressively liver failure.



[2966] **Figure 1.** Magnetic resonance cholangiopancreatography: transverse (A) and coronal (B) view shows hypertrophic caudate lobe (arrow) with nodular contour of liver, splenomegaly (arrow). Liver histology: trichrome stain (C) shows cirrhotic nodule at 100x (arrow) and hepatic steatosis (D) at 400x (arrow).

Table 1. Liver enzyme patterns from age of CF diagnosis to current age

Patient age (years)	AST (IU/L)	ALT (IU/L)	ALP (IU/L)	Total Bilirubin (mg/dL)	INR	Platelets (x10^9 /L)	Sodium (mmol/L)
14	**68 (H)** (< 40)	**97 (H)** (< 41)	280 (100-390)	**1.7 (H)** (0.2-1.6)	1.3	179	142
15	23 (12-43)	34 (4-51)	204 (79-446)	0.5 (0.2-1.0)	1.0	202	143
16	24 (12-43)	47 (4-51)	136 (58-331)	0.9 (0.2-1.0)	1.2	223	142
18	**52 (H)** (12-43)	**97 (H)** (4-51)	270 (58-331)	**1.7 (H)** (0.2-1.0)	1.3	181	140
19	**160 (H)** (10-50)	**61 (H)** (< 41)	**193 (H)** (40-129)	1.31 (0.0-1.60)	1.0	213	140
20	**68 (H)** (< 40)	**131 (H)** (4-51)	**242 (H)** (40-129)	1.2 (0.0-1.60)	-	187	141
21	27 (< 40)	**71 (H)** (4-51)	**177 (H)** (40-129)	**5.6 (H)** (0.0-1.60)	1.2	**116 (L)**	143
22	**57 (H)** (< 40)	**95 (H)** (4-51)	**266 (H)** (40-129)	1.6 (0.0-1.60)	**1.4 (H)**	**109 (L)**	143
23	**81 (H)** (< 40)	**149 (H)** (10-50)	**322 (H)** (40-129)	**2.41 (H)** (0.0-1.60)	**2.1 (H)**	**102 (L)**	141
24	**88 (H)** (< 40)	**246 (H)** (10-50)	**159 (H)** (40-129)	**1.62 (H)** (0.0-1.60)	1.0	**126 (L)**	141

(H) indicates abnormal high laboratory value relative to reference range. (L) indicates low laboratory value relative to reference range. Where appropriate, reference laboratory ranges are included. Hyphens are included where laboratory values are not available. AST: aspartate transaminase; ALT: alanine transaminase; ALP: alkaline phosphatase; INR: international normalized ratio.

S2967

Drug-Induced Autoimmune Hepatitis Post Acute Liver Injury From Skullcap Supplements: An Unfortunate Case of Herbal Toxicity

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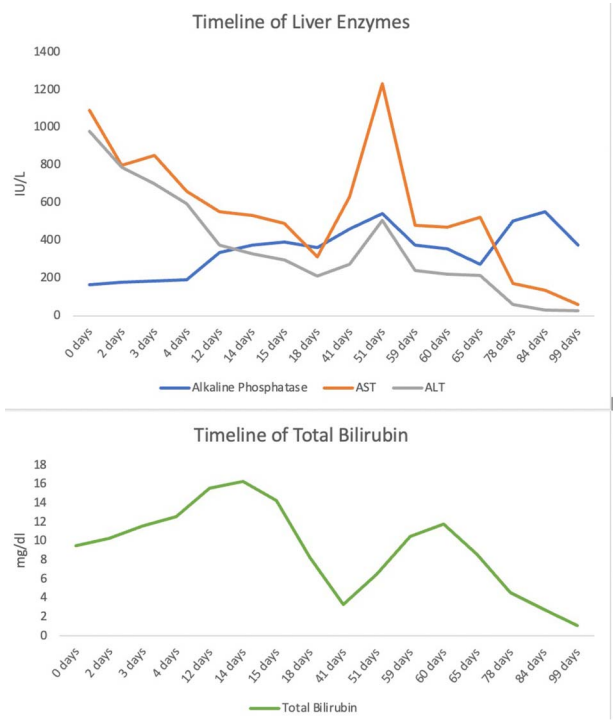
[1]University of Kentucky, Lexington, KY; [2]Memorial Healthcare System, Pembroke Pines, FL.

Introduction: *Scutellaria lateriflora*, also known as Skullcap, is widely used in alternative medicine for menstrual, nervous, digestive and kidney problems. There have been rare instances of acute liver injury (ALI) attributed to its use. However, the occurrence of de-novo plasma cell hepatitis has not been reported before. We present a case of severe acute liver injury and drug-induced autoimmune hepatitis resulting from Skullcap usage.

Case Description/Methods: A 62-year-old female, with a history of Sjogren's disease presented with new onset jaundice. Her labs were - Alkaline Phosphatase (Alk Phos) 164 IU/L, AST 1091 IU/L, ALT 980 IU/L, Total Bilirubin (T bili) 9.5, INR 2.4. MRCP showed did not show cirrhosis or biliary obstruction. The patient's LFTs were normal 4 months ago. She denied any history of liver disease but affirmed taking Skullcap supplements over the last month secondary to insomnia. Initial testing for causes of liver disease was negative for Hepatitis A, B, and C, CMV, EBV, and HSV. ANA was chronically positive given her history of Sjogren's disease. IgG was elevated at 2573 mg/dl, but was thought to be secondary to ALI. Initial liver biopsy showed resolving centrilobular necrosis with predominant eosinophilic inflammation. Given downtrending LFTs over the next 72 hours, the patient was discharged with outpatient follow-up (Figure). She was admitted a month later with worsening jaundice and acute kidney injury. Her LFTs were Alk Phos 619 IU/L, AST 1222 IU/L, ALT 540 IU/L, and T bili 6.6 mg/dl. Infectious workup was negative. Over the next few days, transaminases downtrended but the T bili continued to rise, peaking at 11.8 mg/dl. Repeat biopsy showed extensive plasma cells consistent with drug-induced autoimmune hepatitis. The patient finally did improve with resolution of jaundice. She was subsequently listed for a simultaneous liver kidney transplant but was later deactivated due to overall improvement in status.

Discussion: The toxic effects of Skullcap are thought to be mediated secondary to flavonoids present in its root. However, the contribution of adulterants cannot be excluded as there are no regulatory bodies governing herbal supplement production. To our knowledge, this is the first case of drug-induced autoimmune hepatitis secondary to Skullcap supplements.



[2967] **Figure 1.** TImeline of Liver Function Tests.

S2968

DRESS Syndrome and Drug-Induced Liver Injury: A Product of Antibiotic Cocktail

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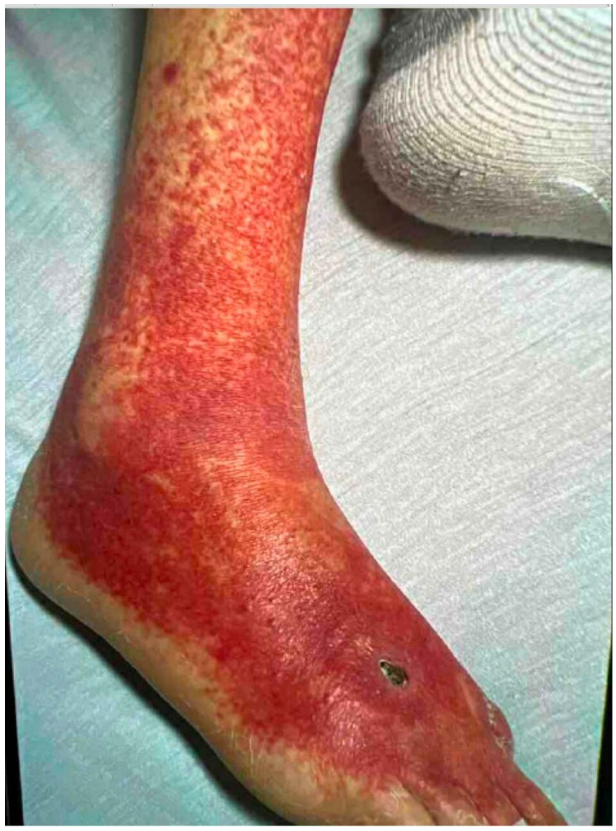
Louisiana State University Health Sciences Center, Shreveport, LA.

Introduction: Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome is a severe cutaneous drug reaction characterized by the presence of a maculopapular rash, fever, lymphadenopathy, eosinophilia, and visceral involvement such as hepatitis, pneumonitis, pericarditis, nephritis, and colitis. The Liver is the most commonly involved organ and the most common cause of death.
Case Description/Methods: Patient is a 63-year-old male admitted with right toe gangrene status post amputation with cultures growing E. coli and staphylococcus lugdunensis treated with a prolonged course of multiple IV antibiotics. Patient was initially treated with Ampicillin/Sulbactum. On day 17, Xray of the foot was obtained & showed signs of osteomyelitis of the right foot. Antibiotics were switched to Vancomycin, cefepime and metronidazole. Shortly after, antibiotics were switched to Vancomycin and Clindamycin for better coverage. On day 36, the patient tested positive for flu, Tamiflu was started, and clindamycin was discontinued. Liver enzymes elevated in a cholestatic pattern with alkaline phosphatase levels continuously trending up. Ertapenem was later added to vancomycin. Eventually, ertapenem and vancomycin were held due to drug induced liver injury. Oral doxycycline and Augmentin were started on day 48. MRCP was negative and was suggestive of normal common bile duct and pancreatic duct dilation. Over the course of several days, patient developed a maculopapular rash with eosinophilia, fever, and severe elevation of ALP levels reaching 1159 IU/ L and 2 episodes of hematemesis. CT imaging was suggestive of acute pancreatitis. All antibiotics were stopped at this point. Patient underwent EGD which was not significant for any source of bleeding. Patient was started on prednisone 1 mg/kg which was eventually tapered over 4-6 weeks and the patient's symptoms resolved (Figure).
Discussion: Even though elevated ALP levels more commonly suggest obstructive jaundice, drug induced liver injury can also portray a cholestatic pattern of liver enzymes and should always be in the differential. DRESS syndrome is a unique diagnosis and recognition of the offending agent in a prolonged inpatient setting, and its withdrawal can halt the progression earlier in the disease course and prevent further organ damage.



[2968] **Figure 1.** Maculopapular rash seen on patient's right foot.

S2969

Distinction Between Mitochondrial Antibody Positive and Negative Primary Biliary Cholangitis: Case Report and Literature Review

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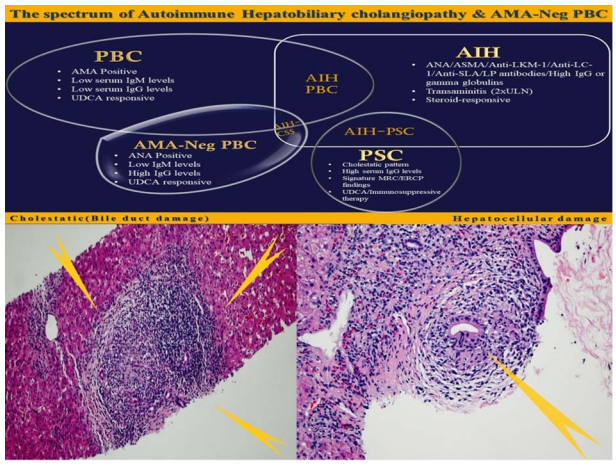
[1]Christus Highland Medical Center, Shreveport, LA; [2]LSUHSC, Shreveport, LA.

Introduction: Anti-mitochondrial antibody-positive primary biliary cholangitis (AMA-pos PBC) is an autoimmune disorder in which monoclonal antibodies are produced against epitopes with in the mitochondrial membranes of biliary epithelial cells, resulting in progressive non-suppurative biliary cholangitis. Up to 5 % of PBC patients lack these auto antibodies, termed as anti-mitochondrial antibody negative (AMA-neg) PBC. This is a somewhat new entity and a variant of AMA-pos PBC but not an overlap syndrome. There have not been good studies describing this phenomenon or associated terminology in the literature.
Case Description/Methods: An 87-year-old woman was referred to our clinic after her medical oncologist found elevated isolated levels of serum alkaline phosphatase (714 units/L). She reported fatigue but denied any other symptoms. The physical examination was benign, except for bilateral lower extremity swelling secondary to lymphedema. Her serum alkaline phosphatase level decreased to 413 units/L after an initial dose of prednisone 40 mg daily, and she was maintained on 10 mg daily. Her antinuclear antibody titer was greater than 1:2560 in a centromere pattern. Anti- mitochondrial antibody was not detected. Total IgG level was 871 mg/dL (normal, $<$ 1600 mg/dL), serum anti-smooth muscle antibody was negative, and the hepatitis panel was normal. Computed tomography of the abdomen and pelvis without contrast showed normal liver parenchyma and no acute intra-abdominal pathology. Histopathological examination indicated florid duct lesions. Background parenchyma showed no significant steatosis, and the inflammatory changes were limited primarily to the portal areas. Periodic acid-Schiff staining highlighted the intact hepatic parenchyma and architecture. The patient was diagnosed with AMA-neg PBC and responded well to Urosdeoxycholic acid therapy. (Figure) (Table).
Discussion: This case highlights the importance of recognizing AMA-neg PBC as a variant of AMA-pos PBC and being able to differentiate them. Autoimmune cholangitis is a vague and imprecise term that cannot be used in this context. All AMA-negative PBC patients should be tested for other PBC-specific autoantibodies. Although prognosis and bile duct damage and loss are worse in AMA-neg PBC for unknown reasons, treatment remains the same for both.



[2969] **Figure 1.** A: The position of AMA-neg PBC in the spectrum of autoimmune hepatobiliary cholangiopathy PBC, primary biliary cholangitis; ANA, antimitochondrial antibody; IgM, immunoglobulin M; UDCA, ursodeoxycholic acid; AMA-neg, antimitochondrial antibody negative; ANA, antinuclear antibodies; IgG, Immunoglobulin G; ASMA, anti-smooth muscle antibody; anti-LKM-1, anti-liver kidney microsomal antibodies; Anti-LC-1, anti-liver cytosol antibodies; anti-SLA/LP antibodies, anti-soluble liver antigen/liver pancreas antibodies; MRCP, magnetic resonance cholangiogram; ERCP, endoscopic retrograde cholangio-pancreatogram. B and C:Two photomicrographs showing histologic features typical of the florid duct lesion seen in primary biliary cholangitis.. The portal areas show granulomatous inflammation and lymphocytic cholangitis of the bile ducts (pointed yellow arrows) (hematoxylin and eosin stain, 200X original magnification).

Table 1. Diagnostic criteria of AMA-Pos PBC The diagnosis of AMA-pos PBC can be established when two of the criteria listed in Table 1 are met[5]

Evidence	Criteria
1) Biochemical evidence of cholestasis	Elevated alkaline phosphatase levels
2) Serological: AMA-Pos PBC AMA-Neg PBC	Mitochondrial antibody positive Mitochondrial antibody negative/Presence of other PBC specific autoantibodies i.e., sp100 or gp210
3) Histopathological	Nonsuppurative destructive cholangitis and destruction of interlobular bile ducts.

Cytomegalovirus-Associated Spontaneous Splenic Infarct in an Immunocompetent Patient

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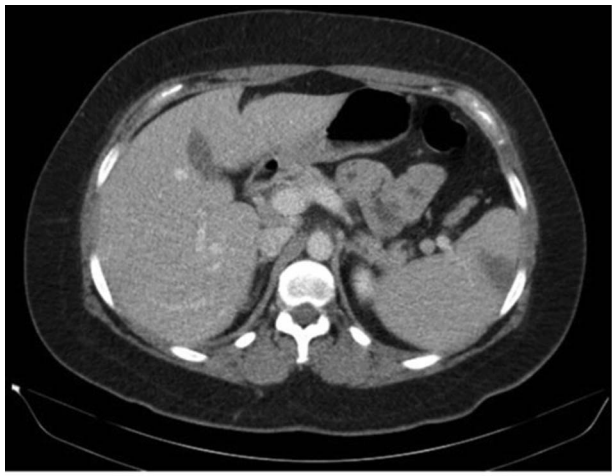
Introduction: Cytomegalovirus (CMV) associated thrombosis has been reported in medical literature and mainly in the immunocompromised population. However, CMV-associated splenic infarcts have rarely been reported.

Case Description/Methods: A 36-year-old Caucasian woman with no significant past medical history who presented to the emergency department with fever, chills, night sweats, palpitation, and mild abdominal pain was found to have acute CMV infection. The patient mentioned that she has used oral contraceptives prior to the presentation. No family history of thrombophilia has been reported. CT scan of the abdomen and pelvis showed a splenic infarct and an ultrasound of her spleen with doppler showed the same findings, i.e., splenic infarct with the normal flow in the splenic artery. However, Echocardiography showed no vegetation, PFO, or mural thrombus. Hypercoagulable workup was negative. Anticoagulation was deferred, oral contraceptives were stopped, and a copper IUD was recommended, patient was managed conservatively due to involvement of small vessels only and lack of related symptoms. She continued to do well on follow-up evaluations (Figure).

Discussion: Acute CMV infections with various thrombotic manifestations have been reported in the medical literature. It is a transient risk factor for both arterial and venous thromboembolism and can occur in immunocompetent patients in the absence of other hypercoagulable factors. The patient in our case had two risk factors that might explain the reason behind splenic infarction: the first, being on oral contraceptives and the second is the acute CMV infection. A meta-analysis on 97 patients in 2011 reported that the incidence of thrombosis among acute CMV infection in hospitalized patients was 6.4%, and the incidence of acute CMV infection among hospitalized patients with thrombosis was 1.9-9.1%. Several mechanisms have been described explaining the role of CMV in thrombosis. In vitro, CMV activates factor X and stimulates the production of factor VIII and vWF. It also binds to platelets via Toll-Like Receptor 2 and causes systemic endotheliitis at various sites in the body, leading to the expression of tissue factors. These mechanisms result in platelet and leukocyte aggregation, adhesion, and thrombin formation. In vivo, a transient increase in antiphospholipid antibodies and a decrease in protein C activation have been reported. In conclusion, patients with unexplained arterial or venous thrombosis should be tested for CMV infection as a possible etiology.



[2970] **Figure 1.** CT scan of the abdomen showing splenic infarction.

S2971

Cryoglobulinemic Vasculitis After 6 Years of Sustained Virologic Response in a Post-Liver Transplant Patient

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Introduction: Cryoglobulinemic vasculitis (CV) is caused by cryoglobulins that bind to polyclonal IgG and can damage multiple organ systems. It is associated with active hepatitis C virus (HCV) infections and the primary therapy is usually treatment of the underlying HCV. We present a rare case of CV occurring in a post-liver transplant (LT) patient who was treated for HCV with sustained virologic response (SVR) for more than 6 years.
Case Description/Methods: A 67-year-old with history of HCV cirrhosis and hepatocellular carcinoma treated with LT in 2011 on tacrolimus, chronic kidney disease stage 3, hypertension, hyperlipidemia initially presented to an outside hospital with nausea, vomiting, and diarrhea which resolved with supportive care. She later developed arthralgias, a maculopapular rash, and left eye pain with orbital swelling. She was evaluated by ophthalmology without findings of retinitis but with bilateral elevated intraocular pressures. MRI orbits revealed lacrimal gland adenitis treated with topical medications including erythromycin and systemic acetazolamide. Dermatology evaluated patient with a skin punch biopsy which showed vascular/perivascular C3 and fibrinogen staining suggestive of early vasculitis without evidence of IgA vasculitis. Notably, she was treated with ledipasvir/sofosbuvir and ribavirin which was completed 7/2015 with SVR achieved 12/2015. HCV RNA viral load was again negative as well as other viruses. Due to rapidly worsening renal function with creatinine rising to 3.8 from baseline of 1.3-1.5, a renal biopsy was performed which revealed cryoglobulinemic glomerulonephritis—establishing a diagnosis of CV. Patient's course was complicated by a biopsy-related renal hematoma requiring embolization. She was treated with pulse dose solumedrol and plasmapheresis. Shortly after treatment, her rash, vision, and arthralgias improved. Her creatinine returned to baseline and she was started on prednisone 60mg daily.
Discussion: CV is typically associated with active HCV infection, but in rare cases can be found in treated patients with SVR. This case is also unique in that it involves a post-LT patient on tacrolimus, raising the question of the impact of tacrolimus in developing CV in a patient with SVR. Moreover, multiple modalities of diagnosis may need to be pursued if initial biopsies are not conclusive to establish effective treatment plans. Overall, CV should remain on the differential in patients with a treated HCV and SVR.

S2972

Disseminated Herpes Simplex Virus: A Diagnostic Enigma

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Introduction: Disseminated Herpes Simplex Virus (HSV) is rare but is associated with up to 50% mortality, especially in immunocompromised patients and pregnant women in whom the diagnosis is often missed. In pregnant women, it can cause fulminant liver failure and can lead to fetal demise in some cases. We describe a case of disseminated HSV in a pregnant woman with no known active disease prior to pregnancy.
Case Description/Methods: A 28-year-old healthy female presented at 26 weeks gestation with fever, chills and back pain. Initial labs were concerning for pyelonephritis and antibiotics were initiated. Despite broad spectrum antibiotics, she had persistent fever and workup for infectious etiology was broadened to include HIV, CMV, EBV, toxoplasma, syphilis, which were all negative. She developed worsening transaminases with AST 384 U/L and ALT 220 U/L. Viral hepatitis serologies and, due to her recent travel to Lyme endemic area, arbovirus serology was tested, which were all negative. She subsequently developed thrombocytopenia and worsening hypertension raising concern for HELLP syndrome and underwent emergent cesarean section. Postpartum course was complicated by persistent fevers and concern for chorioamnionitis; however, she continued to have worsening transaminases (AST 2632, ALT 1000). Computed tomography abdomen found numerous liver lesions concerning for abscesses or metastases. Liver biopsy found microvesicular steatosis consistent with HELLP syndrome. She developed worsening thrombocytopenia, coagulopathy (INR 2) and rising LDH, which was concerning for disseminated intravascular coagulation. She was started on plasma exchange and N-acetylcysteine. HSV PCR serology was tested which returned positive for HSV-2 and she was started on acyclovir. Lumbar puncture returned positive for HSV confirming disseminated HSV. Her course was complicated by encephalopathy that eventually improved and she was able to be discharged on a 21-day course of acyclovir. Transaminases and coagulopathy improved with treatment of disseminated HSV.
Discussion: Disseminated HSV has a high mortality in pregnancy but can be missed due to the rarity of the condition. Early diagnosis and administration of acyclovir decreases mortality. A high index of suspicion is necessary, and it should remain in the differential diagnosis in pregnant and postpartum patients with acute hepatitis and even considered in patients with elevated transaminases without any overt history or skin lesions concerning for HSV as in this patient.

S2973

Cryoglobulin-Mediated Duodenitis in an HCV Patient With Sustained Virologic Response

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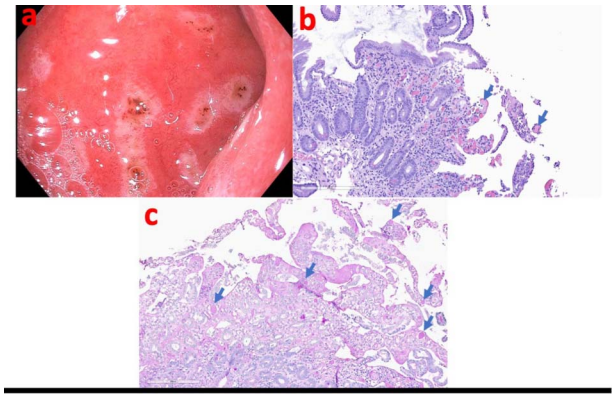
Introduction: Mixed cryoglobulinemia syndrome (MCS) is caused by chronic hepatitis C virus (CHC) in >90% of cases. The classic presentation of MCS includes palpable purpura, renal dysfunction, and arthralgias, but other life-threatening manifestations can occur, including pulmonary, hepatic, and mesenteric involvement. Nearly all patients experience resolution of MCS after sustained virologic response (SVR), but relapse can rarely occur. We present a patient who was found to have asymptomatic cryoglobulin-mediated duodenitis after years of SVR.
Case Description/Methods: A 62-year-old man with a history of type 2 diabetes, hypertension, and prior kidney transplant was treated with an unknown direct acting antiviral for CHC at an outside center in 2018. He then presented after likely cirrhosis was incidentally diagnosed on an abdominal CT. He appeared clinically compensated with no complaints, and vital signs and laboratory testing were consistent with compensated disease. Lack of active viral hepatitis was confirmed. A screening endoscopy found no varices but was notable for scattered mucosal discoloration and erosion throughout the duodenum. Biopsies

demonstrated numerous hyaline Periodic acid-Schiff-diastase positive thrombi consistent with intravascular cryoglobulin deposits. Serum cryoglobulins were not detected, another cause of persistent cryoglobulinemia was not identified, and there was no prior diagnosis of MCS. Therapy was deferred, as the patient was asymptomatic (Figure).
Discussion: Mesenteric involvement is a rare (2.6% of all extrahepatic CHC) but potentially devastating complication of CHC-associated MCS. It can cause intestinal ischemia and perforation, and its presence is independently associated with increased mortality. Treatment of underlying CHC generally induces remission, but this patient demonstrated active MCS-related duodenitis years after achieving SVR. Persistent MCS after SVR due to sustained clonal production of cryoglobulins has been rarely reported, although our patient had no evidence of systemic involvement. Persistent MCS after SVR should prompt HCV testing to assess for relapse. If no relapse is found, as in this patient, a different underlying condition should be considered, including B-cell lymphoma, HIV, HBV, or autoimmune disease. While MCS incidence is decreasing concurrently with HCV burden, this case illustrates that MCS is possible even years after SVR, an unusual finding warranting further investigation.



[2973] **Figure 1.** a) Scattered mucosal discoloration and erosion seen in the duodenum consistent with ischemic injury. b) Duodenal mucosa with intravascular hyalin thrombi indicated by the arrows. c) Periodic acid-Schiff-diastase stain showing multiple intravascular thrombi indicated by the arrows.

S2974

Common Variable Immune Deficiency With Atypical Hepatic Disease

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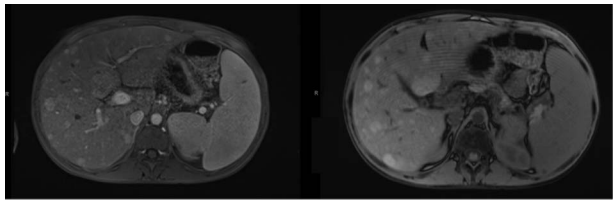
Introduction: Common variable immunodeficiency (CVID) is a primary B-cell immunodeficiency disorder leading to hypogammaglobulinemia. Liver involvement may rely on immune dysregulation and includes abnormal liver biochemistries, primarily elevation of alkaline phosphatase (ALP), nodular regenerative hyperplasia (NRH) leading to chronic cholestasis, portal hypertension, and disruption of liver function. We present a case of CVID with atypical liver involvement on imaging and biopsy revealing focal nodular hyperplasia.
Case Description/Methods: A 24-year-old female with a past medical history of CVID on Intravenous immunoglobulin (IVIG) present for follow-up with chronically elevated liver enzymes that is a cholestatic pattern (ALP 1050 U/L, AST 114 U/L, ALT 214 U/L, Total bilirubin 1.0 mg/dL) along with normal synthetic liver function. Previous comprehensive work up including autoimmune (Antinuclear, Anti-smooth muscle, anti-mitochondrial antibodies) were negative. Viral hepatitis including Hepatitis B, Hepatitis C, Herpes simplex, Varicella zoster, and Epstein Barr viruses polymerase chain reaction were all negative. other causes of elevated liver enzymes including genetic and metabolic work up all came back negative. The medications list included Ursodiol and Multivitamins. She reports no alcohol, recreational or herbal drugs use. She underwent liver protocol magnetic resonance imaging and magnetic resonance cholangiopancreatography that showed no biliary tree abnormalities, Innumerable hepatic lesions with characteristics suggestive of multiple focal nodular hyperplasias largest measures up to 3.8 cm, two benign hepatic hemangiomas with the larger measuring 1.9 cm and splenomegaly. Ultrasound-guided biopsy of the left hepatic lobe showed concern for focal nodular hyperplasia (confirmed by an outside pathologist at another liver transplant center) with unremarkable trichrome and PAS stains (Figure).
Discussion: At least 10% of CVID patients present with liver involvement, including anicteric cholestatic liver enzymes (65%) and portal hypertension (50%). Histological analysis in recent studies revealed non-fibrosing architectural abnormalities consistent with nodular regenerative hyperplasia in 84% of CVID and less commonly, typical histologic features of primary biliary cholangitis. Our case liver imaging and biopsy read by 2 pathologists (2 institutions) revealed focal nodular hyperplasia, a new entity not described in previous studies.



[2974] **Figure 1.** Liver protocol MRI showing focal nodular hyperplasia.

S2975

Decompensated Liver Failure in a Post-Partum Patient With Hepatitis C-Related Liver Cirrhosis: Balancing on a Thin Line

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Introduction: Decompensated liver cirrhosis is a common reason for admission to intensive care units, often with high mortality rates. However, pregnancy among cirrhotic patients is rare, carrying a significant risk for variceal bleeding and acute decompensation, leading to adverse maternal and fetal outcomes.
Case Description/Methods: We report a case of a 38-year-old multigravid at 34 weeks gestation, with known liver cirrhosis secondary to chronic hepatitis C, admitted for observation due to short cervix on ultrasound. Workup on admission revealed anemia (Hgb 8.9 mg/dL) and thrombocytopenia (platelet 126,000 mm/mL), with mildly elevated transaminases (ALT 60 U/L, AST 65 U/L), altered synthetic liver function (low albumin 2.7 g/dL) but normal bilirubins and INR. She underwent an emergency cesarean section due to non-reassuring fetal status and had a delivery of a 2.1 kgs preterm baby with an APGAR score 7-8. However, postoperatively, she presented with hematemesis and hypotension. She was transferred to the intensive care unit, where she developed overt hepatic dysfunction with AST 2,260 U/L and ALT 3,388 U/L. Other workup showed hyperbilirubinemia, leukocytosis, thrombocytopenia, and prolonged INR. In addition, she developed metabolic acidosis, eventually requiring hemodialysis. Esophagogastroduodenoscopy revealed portal hypertensive gastropathy and tortuous esophageal varices to which rubber band ligation was performed. Despite maximal supportive medical management, she had decreasing sensorium and shock. She was intubated requiring four vasopressors. Her clinical status progressively declined post-delivery as she developed worsening liver failure and grade IV encephalopathy. Finally, the patient expired on the 3rd postoperative day due to hepatorenal failure.
Discussion: Pregnant women with hepatic cirrhosis have altered reproductive hormone levels and deranged systemic physiology. Pregnant cirrhotic women should be closely monitored due to high maternal and fetal morbidity with associated mortality. It is recommended that known cirrhotic patients, regardless of etiology, be appropriately assessed and managed before any planned pregnancy. The decision for

cirrhotic patients to become pregnant should be a balanced family decision with a multidisciplinary team. Further studies are needed to develop protocols for prevention, prognostication, and management of postpartum liver failure as current data are limited.

S2976

Cry Me a Liver: Ashwagandha-Induced Liver Toxicity

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Introduction: Ashwagandha is a derivative of the medicinal plant Withania somnifera. It has been used for centuries as a tonic to increase energy, reduce fatigue and as anti-aging in Ayurvedic medicine. Herbal medicines and dietary supplements (HDS) are categorized as food by the FDA and, unlike prescription medicines, are presumed to be safe unless otherwise reported. However, a recent case series has implicated this herb to cause clinically apparent liver injury. This case illustrates the hepatotoxic potential of ashwagandha.
Case Description/Methods: A 20-year-old healthy college student presented to our ER with complaints of yellowish discoloration of skin and worsening abdominal pain for 3 days. The discoloration was initially noted in his eyes, associated with excessive itching of skin but no stool changes. He also had right upper quadrant abdominal pain, 6/10 in intensity, without any associated aggravating or relieving factors. He denied recent travel, alcohol or drug consumption and his last sexual encounter was 4 months ago. His physical examination was significant for a normal BMI, icteric sclera, soft and non-tender abdomen. Initial labs showed AST of 659 and ALT of 415 and direct bilirubinemia of 8.6. A thorough autoimmune, infective hepatitis panel and hemolytic anemia work up was negative. USG and CT abdomen/pelvis showed no abnormalities. On further questioning he endorsed consuming over the counter (OTC) Ashwagandha 450mg every day for the past 30 days to "calm his nerves". Over the course of his hospitalization, he symptomatically improved with down trending liver enzymes and bilirubin. He was advised to avoid ashwagandha containing supplements in the future. Post-discharge 3 week follow up showed a normal hepatic function test.
Discussion: Withanolides are the active components of Ashwagandha. The liver injury presents 2 to 12 weeks after ingestion with a cholestatic or mixed pattern of injury, jaundice and pruritus. Jaundice tends to be a dominant feature but ultimately resolves after discontinuation of the offending drug without fatalities or chronic injury. Biopsy can be considered if no clinical improvement to evaluate for uncommon etiologies. Benefit of ursodeoxycholeic acid use in drug induced liver injury remains controversial. OTC medication and herbal supplements have been associated with liver injury and it is important to obtain a thorough history in a patient with elevated transaminases.

S2977

Early Use of Tocilizumab as an Effective Steroid-Sparing Strategy for the Treatment of Immune Checkpoint Inhibitor-Mediated Cholangiopathy: Building Foundations for Personalized Management

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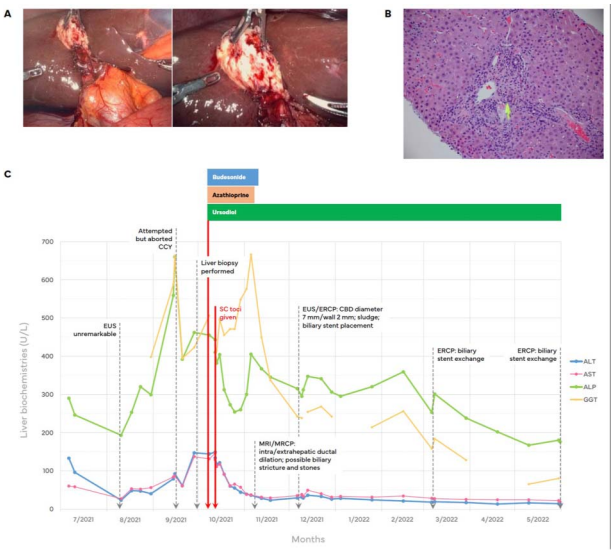
Introduction: Immune-mediated cholangiopathy (IMCp) is an increasingly recognized complication of immune checkpoint inhibitor therapy, often associated with exposure to anti-PD-1/L1 agents. Both intra- and extrahepatic manifestations can occur. Potential sequelae include biliary strictures and acute cholangitis. The management of IMCp remains undefined across multiple society guidelines, but published cases have offered insight into formulating effective treatment strategies. We present a complex case of a patient with IMCp successfully treated with budesonide and early tocilizumab, an IL-6 receptor antagonist.
Case Description/Methods: A 70-year-old man with a history of urothelial carcinoma (treated with neoadjuvant carboplatin/gemcitabine, followed by 3 cycles of an anti-PD-1 agent, pembrolizumab), pre-diabetes, and no underlying liver disease, presented with abnormal liver enzymes. ERCP revealed some biliary sludge but no biliary stricture. Endoscopic ultrasound 3 months later was normal. ALT normalized, but alkaline phosphatase (ALP) remained elevated; both then increased to CTCAE grade 2. R factor was 0.5. Elective cholecystectomy was attempted but aborted: He had a firm, contracted gallbladder with dilated extrahepatic biliary ducts, and the surgeon could not demarcate borders of the cystic and common bile ducts. MRCP showed intrahepatic biliary ductal dilation, ductal thickening and enhancement, and CBD dilation with a new stricture. Liver biopsy showed cholestatic hepatitis with portal and lobular inflammation and hepatocyte necrosis. A diagnosis of immune-mediated cholangiohepatitis with IMCp was made. Oral budesonide/azathioprine/ursodiol were prescribed. Five days later, subcutaneous tocilizumab 162 mg was administered without complication. After completion of budesonide/azathioprine, ursodiol was continued long-term for persistent elevation of ALP. Serial ERCPs for plastic biliary stent placement were performed for biliary sludge and for an unresolved CBD stricture. ALP level improved to < 180 U/L about 7-8 months after tocilizumab (Figure).
Discussion: Cases of IMCp could be potentially overlooked because ALP is not featured in the society guidelines' assessments for liver toxicity. Although steroids can be used to initially treat IMCp, aggressive escalation to alternative treatments such as tocilizumab is important to mitigate progression to biliary sequelae that might arise. As use of anti-PD-1/L1 agents expand, IMCp may become increasingly common, and prompt treatment is of paramount importance.



[2977] **Figure 1.** (A): Intra-operative photographs revealing a white/firm contracted gallbladder and dilated extrahepatic biliary ducts, with inability to demarcate the cystic duct and common bile duct; due to distortion of the extrahepatic biliary anatomy, cholecystectomy was not performed and aborted. (B): Histologic evaluation (hematoxylin & eosin) from liver biopsy, showing chronic cholestatic hepatitis with portal and moderate lobular inflammation, bile duct injury, bile ductular proliferation, moderate cholestasis, and scattered hepatocyte necrosis; no significant fibrosis seen. (C): Timeline of liver biochemical tests and associated treatments. Budesonide was selected as the induction steroid, at 9 mg/day, for 5 days total, followed by 6 mg/day for 14 days, and 3 mg/day for 14 days. Azathioprine adjunct of 100 mg/day was briefly prescribed. Subcutaneous tocilizumab 162 mg was administered; serum ALT normalized at 31 days thereafter. Ursodiol was administered long-term in an attempt to address and to mitigate further cholangiopathic consequences. Abbreviations: EUS, endoscopic ultrasound; CCY, cholecystectomy; SC, subcutaneous; toci, tocilizumab; MRI/MRCP, magnetic resonance imaging/magnetic resonance cholangiopancreatography; ERCP, endoscopic retrograde cholangiopancreatography; CBD, common bile duct; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase.

S2978

End-Stage Liver Disease in the Setting of Type IIIa Glycogen Storage Disease

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Introduction: Glycogen storage diseases (GSD) are genetic defects in glycogen metabolism and utilization. Features of GSD 3 are attributed to defective glycogenolysis, including hepatomegaly, transaminitis, hypoglycemia, myopathic changes and elevated creatinine kinase. For confirmation, AGL gene mutation testing and measurement of debrancher enzyme activity are available. Symptomatic therapy includes high-protein feedings and high-protein snacks. Although rare, there are reports of GSD 3 progressing to cirrhosis due to abnormally structured glycogen damaging hepatocytes. We highlight the management of GSD 3 and the importance of therapeutic adherence for cirrhosis prevention.
Case Description/Methods: This is a 35-year-old female who presented with encephalopathy, jaundice, and hematemesis. She was diagnosed with GSD 3a at 18 months due to hepatomegaly. Diet therapy was recommended at this time, including high-protein and frequent small meals with corn starch supplementation. Diet therapy in GSD 3a is typically effective and has improved hypoglycemia, hepatomegaly, and overall development. Given dietary noncompliance including low protein and high carbohydrates, she developed cirrhosis and underwent OLT at 15-years-old. After a 16-year period of loss to follow up updated labs demonstrated a past Hepatitis C infection. Imaging demonstrated cirrhotic liver transplant, liver biopsy indeterminate for acute rejection and EGD with portal hypertension. Admission labs included hyperammonemia, hyperbilirubinemia, liver function with cholestatic and hepatocellular involvement, oliguric AKI and a MELD of 35. Physical exam was significant for hepatic encephalopathy grade III, jaundice and asterixis. Her admission was complicated by hepatorenal syndrome type I treated per protocol and oliguria managed with hemofiltration, encephalopathy complicated by failure to protect airway requiring intubation and severe malnutrition. Retransplant was performed and she was discharged on Tacrolimus and Cellcept.
Discussion: Patients with GSD 3a typically follow a benign course, symptoms resolving by early adulthood. In our case, hepatocellular failure predominated and progressed to require a liver transplant. This case highlights diet therapy being the primary treatment for GSD 3a. Whereas the course after liver transplant is typically benign, the importance of longitudinal outpatient follow-up post-liver transplant is imperative for monitoring chronic rejection and/or retransplant requirement.

S2979

Endotipsitis: An Atypical Risk Factor for *Lactobacillus* Bacteremia

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Introduction: Transjugular intrahepatic portosystemic shunt (TIPS) is a procedure commonly performed to decompress portal venous pressure in patients with liver cirrhosis. Infection of TIPS, called endotipsitis, is a rare and underreported complication of the TIPS procedure. Bacteremia due to endotipsitis carries a mortality of 32%. It is a diagnosis of exclusion, as definitive diagnosis requires removal of TIPS and culture. There are only three reported cases of endotipsitis due to lactobacilli. Here, we present a rare case of endotipsitis due to *L*actobacillus bacteremia.
Case Description/Methods: A 64-year-old female with a past medical history of nonalcoholic steatohepatitis (NASH), cirrhosis, portal hypertension, recurrent ascites, esophageal varices with bleeding, status post TIPS procedure 9 months ago who presented to the ED with fever, chills, somnolence, and progressive lethargy. Given concern for possible septic shock on presentation, a full infectious workup was performed including echocardiogram, ultrasound, and computed tomography of the abdomen and pelvis revealing a patent TIPS. Two sets of blood cultures grew Lactobacilli. She was started on broad spectrum antibiotics. Blood cultures were repeated on day 3, which showed no bacterial growth. Considering the history of TIPS and no other identifiable source of infection, a "probable" diagnosis of endotipsitis was made. The patient declined further inpatient work up; given her rapid clinical improvement, she was discharged on a 21-day course of IV piperacillin-tazobactam, based on culture sensitivities, with a plan to repeat blood cultures at that time.
Discussion: Sanyal et al. defined the term endotipsitis for the first time in 1998, indicating an infection of the TIPS procedure. A "definite" infection was referred to as a "continuous, clinically significant bacteremia, with vegetations or thrombi inside the TIPS, whereas a "probable" infection was described as "bacteremia in a patient with an apparently normal TIPS without an identified site of infection elsewhere in the body." However, there continues to be a lack of consensus on the definition of endotipsitis, resulting in unclear guidelines and the diagnosis becoming one of exclusion. It is imperative that clinical practice guidelines regarding definition and diagnosis be established. Definitive treatment of endotipsitis is removal of TIPS with liver transplantation, but given the clinical difficulty of this, it is often treated with antimicrobial agents.

S2980

Drug-Induced Liver Injury (DALI) Due to Levofloxacin, Presenting as Acute Onset of Cholestatic Jaundice: A Case Report

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Introduction: Fuoroquinolones generally have good safety profile however, there are reported cased of Drug-induced liver injury (DALI) due to their use. Clinicians should be aware of this rare but serious adverse effect for early recognition in order to prevent catastrophic liver injury requiring liver transplant.
Case Description/Methods: A 63 year old woman was hospitalized for pneumonia and started on Levofloxacin 750mg IV daily for treatment. On admission total bilirubin, AST, ALT, ALKP, CBC, alcohol and renal function tests were normal. On third day sudden onset of severe jaundice and scleral icterus was noted with no associated rash, fever, chills or abdominal pain. Repeat lab work revealed cholestatic pattern of liver injury with total bilirubin of 9.5mg/dL, direct bilirubin of 6.2mg/dl and only minimal hepatocellular injury with AST of 43 IU/L, ALT 52 IU/L, and normal ALKP, albumin, prothrombin time/INR, platelets, hemoglobin, haptoglobin and LDL levels. Viral hepatitis serology was negative and auto-immune hepatitis was ruled out with negative anti-nuclear antibody (ANA), anti-smooth muscle and anti-mitochondrial antibodies. Ultrasound liver/gallbladder and CT scan of abdomen did not show and intra or extrahepatic bile ducts dilatation or any other abnormalities. Levofloxacin was discontinued and patient was treated with IV fluids resulting in dramatic improvement in bilirubin levels from as high as 9.5mg/dL to 2mg/dL within three days.
Discussion: DILI can present in hepatocellular, cholesteric or mixed liver injury patterns. Fluoroquinolones can cause idiosyncratic DILI due to unpredictable response in few patients even at therapeutic doses. It is important for clinicians to be cognizant of rare but potentially life threatening adverse effects of fluoroquinolones. Prompt recognition of the offending agent, Levofloxacin in this case, and to discontinue it early on prevents fulminant hepatic failure requiring transplant. Interestingly, in this case hepatocellular and synthetic functions remained normal, including ALKP and patient presented with very sudden and significant cholestatic jaundice only. Other causes of cholestatic jaundice including hematological disorder, infectious hepatitis, autoimmune and structural liver diseases need to be ruled out before blaming the drug as potential culprit of the liver injury. Offending drug should be discontinued and should never be administered to the patient again.

S2981

Drug-Induced Autoimmune-Like Hepatitis Due to HemoHIM in a 28-Year Old Pregnant Woman

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Introduction: Drug-induced autoimmune-like hepatitis (DIAIH) is a pathologic immune response against proteins in the liver that can be triggered in susceptible individuals by drugs or herbal remedies. While any supplement can cause idiosyncratic hepatotoxicity, up to 16% of drug-induced liver injuries are attributable to herbal supplements. To date, there are still limited data on herbal medicine-related hepatotoxicity. This topic is particularly understudied in pregnant patients, with most current clinical information coming from case reports. While use of supplements is common during pregnancy, there is still limited information regarding their safety.
Case Description/Methods: A previously healthy G4P2A1 28-year-old presented at 5 weeks of pregnancy with right upper quadrant pain and fevers, and was presumed to have acute cholecystitis resulting in a laparoscopic cholecystectomy. Despite this, her liver enzymes continued to worsen (AST 2893, ALT 2664, TBili 2.6, Alk Phos 460, INR 1.3). The patient also had laboratory evidence of AIH including Anti-Mi+ 34.5, U1-RNP/RNA-A68 2.4, Chromatin Abs 1.6. She underwent liver biopsy showing drug-induced liver injury and autoimmune hepatitis (AIH). Given that AIH often presents during pregnancy, it was unclear if this presentation represented DIAIH or AIH. Notably, the patient had recently started taking an herbal supplement with purported immune support called HemoHIM that was thought to be a possible driver of DIAIH, The supplement was discontinued and she was started on a 60mg prednisolone with subsequent clinical and laboratory improvement. Despite improvement, the patient suffered spontaneous abortion early in the treatment course and in retrospect underwent unnecessary cholecystectomy. Given concern for possible AIH and slow normalization of LFTs, she was started on azathioprine, but self-discontinued after 7 months. Her liver enzymes remained stable 6 months later; supporting a diagnosis of DIAIH due to HemoHIM rather than AIH which would have likely rebounded with such early treatment withdrawal.

Discussion: While DIAILH is an underreported disease in pregnant women this case demonstrates the importance of early diagnosis and treatment. Additionally, it illustrates he challenges in distinguishing between DAILH and AIH especially in patients taking herbal supplements.

S2982

Drug-Induced Liver Injury Secondary to Gabapentin

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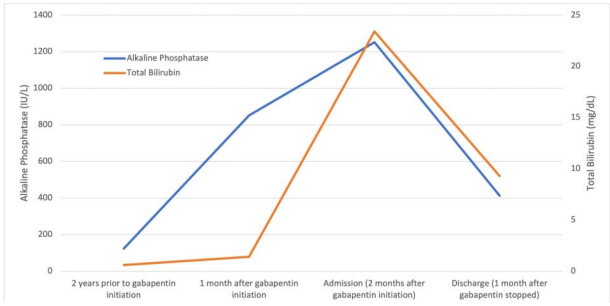
Introduction: We are reporting a case of drug induced liver injury (DILI) secondary to gabapentin therapy with risk factors for underlying non-alcoholic fatty liver disease (NAFLD).
Case Description/Methods: A 56-year-old male with hypertension, hyperlipidemia, diabetes with neuropathy, obesity, and chronic kidney disease stage 3 presented as an outside hospital (OSH) transfer for evaluation of liver transplantation after discovery of acute liver injury. He initially presented to the OSH for jaundice. Admission labs were notable for AST 87, ALT 61, ALP 1252, total bilirubin (Tbili) 23.4, INR 1.1, and creatinine 1.98 (baseline 1.2). Prior lab review showed liver enzymes within normal limits until one month prior to admission, when his ALP was 851. He started taking gabapentin, without introduction of any other medications, one month prior to the initial rise in ALP . Evaluation for viral, inherited, and metabolic causes of liver disease were negative. Liver biopsy showed multifocal hepatocyte cholestasis predominantly involving zone 3 with associated hepatocyte feathery degeneration and neutrophil and lymphocyte infiltration. There was patchy portal edema, mild portal inflammation with neutrophil and lymphocyte infiltrates, and bile duct injury. Trichrome stain highlighted periportal and focal bridging fibrosis appearing to be unrelated to cholestasis, most likely due to underlying NAFLD. The leading differential for cholestasis was drug induced liver injury (DILI) versus biliary obstruction. Magnetic resonance cholangiopancreatography showed no biliary abnormalities. After gabapentin was discontinued, liver enzymes began to downtrend with discharge values of AST 16, ALT 35, ALP 413, Tbili 9.3 and INR 1.1 (Figure).
Discussion: Gabapentin induced liver injury is rare with few reported cases, many of which did not exclude other etiologies. In this case, the key elements of diagnosing DILI were met including gabapentin initiation closely preceding liver injury, other etiologies excluded, and discontinuation of gabapentin leading to improvement. The severity of this patient's DILI and general recommendation to avoid future exposure precluded him from being rechallenged with gabapentin. Given the extensive use of gabapentin in medical practice, this case represents an uncommon, but severe, complication of its use. This is also an example of DILI with suspected underlying NAFLD. While NAFLD has not been shown to predispose to DILI, it is suspected to be associated with more serious liver injury in patients who develop DILI.



[2982] **Figure 1.** The figure above depicts the patient's trend in alkaline phosphatase and total bilirubin starting at a baseline obtained 2 years prior to initiation of gabapentin through to his discharge (one month after gabapentin was stopped). The x-axis show chronology of gabapentin administration. The left y-axis shows alkaline phosphatase levels in IU/L. The right y-axis shows total bilirubin levels in mg/dL.

S2983

Drug-Induced Liver Injury Due to Chronic Kratom Use

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Introduction: Kratom (Mitragyna speciosa) is an herb with opiate and stimulant like properties. It is marketed as an herbal supplement. It can be exploited as a recreational drug and is popular for self-treatment of opiate withdrawal and pain. It was previously unknown what pathological changes could occur with chronic Kratom use. New evidence suggests that Kratom can cause a drug induced liver injury (DILI). There is little published evidence of the toxic effects of Kratom, yet there is increasing self-treatment of opioid withdrawal and reports of adverse events and lethal overdoses. We describe a case where chronic use of Kratom caused severe DILI.
Case Description/Methods: A 39-year-old male with no significant medical history presented to the emergency department with sharp abdominal pain and diarrhea. Physical exam revealed jaundice, scleral icterus, and left lower quadrant tenderness. Initial labs were AST 791 units/L ALT 1841 units/L, total bilirubin 7.4 mg/dL, alkaline phosphatase 449 units/L and INR of 1.2. A CT demonstrated scattered porta hepatis lymph nodes. The patient reported no medication or recreational drug use. He underwent extensive testing including viral hepatitis panel, stool studies, toxicity screen, anti-smooth and anti-mitochondrial antibodies which all produced negative results. Magnetic resonance cholangiopancreatography and right upper quadrant ultrasound were unremarkable. A liver biopsy revealed grade-III inflammatory activity with significant eosinophilia, highly suspicious for drug interaction. On hospital day 5, the patient admitted to several months of kratom consumption. His AST level peaked at 1190 units/L, ALT peaked at 2261 units/L, total bilirubin peaked at 20.2 mg/dL and alkaline phosphatase continually declined. Given the strong correlation with his biopsy results, the likely etiology of this liver injury was determined to be hepatocellular toxicity due to chronic Kratom use. The patient symptomatically improved and in outpatient follow-up there was normalization of liver function after discontinuation of Kratom.
Discussion: Kratom's mechanism and scope of toxicity is not well understood. It remains legal in our state, Georgia, but is banned in some other states and countries around the world. As it remains commercially available, policy regarding this substance should be reviewed closely and studies should be conducted to evaluate its toxicity. Fortunately, this case demonstrates reversible toxic effects to the liver.

S2984

Drug-Induced Liver Failure Masquerading as Late Onset Wilson Disease: A Case Report

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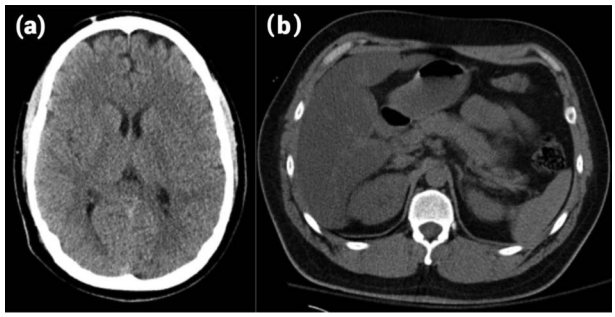
Introduction: The use of herbal and dietary supplements has increased occurrences of drug-induced liver failure. Acute liver failure (ALF) can rapidly progress to multiorgan failure with about 50% of patients requiring a liver transplant. About 10% of all ALF cases have been due to drug injury while only about 2% have been due to Wilson disease (WD). This article provides a detailed case review of ALF due to the use of alkaline water that was originally thought to be due to WD.
Case Description/Methods: A 35-year-old male with no known past medical history is brought into the emergency department (ED) with altered mental status for the past 2 to 3 weeks. The patient was found to be in acute renal failure and ALF. Computed tomography (CT) of the head was unremarkable and a CT of the abdomen showed hepatomegaly and hepatic steatosis without evidence of cirrhosis. Abdominal ultrasound was negative for portal vein thrombus, and laboratory findings were significant for reduced ceruloplasmin. Due to the absence of other causes for his ALF, the patient's presentation was thought to be due to WD. An ophthalmologist conducted a slit-lamp exam, which was negative for Kayser-Fleischer rings. A liver biopsy showed focal liver cholestasis, bile ductular reaction, and mixed portal inflammation suggestive of drug-induced liver injury. Examination of the liver core biopsies displayed acute and chronic periportal inflammatory infiltrate with eosinophils. The patient's acute encephalopathy completely resolved after six days, and the patient subsequently reported drinking an alkaline water daily 10-days before the onset of symptoms. After ten days, the patient was transferred to a transplant center, however, with improving liver function and resolution of symptoms, the patient was discharged home without the need for further treatment. (Figure) (Table).
Discussion: Due to increased consumption of herbal and dietary supplements in the United States, the rates of drug-induced liver failure have increased. Many cases of drug-induced liver failure due to these sources are underreported and when they are discovered have been linked to increased rates of liver transplant. This case not only highlights drug-induced acute liver failure and its complicated presentation, but

also brings into consideration alkaline water as a potential source for ALF. Cases such as this demonstrate that a thorough history and further understanding of herbal and dietary supplements cannot be overlooked when attempting to elucidate a source in instances of ALF.



[2984] **Figure 1.** Computed tomography of the head and abdomen. (a) Axial view of head. No abnormalities in the basal ganglia. (b) Coronal view of abdomen.

Table 1. Laboratory test results of the patient a day before admission, on the day of admission, five days after admission, ten days after admission, and two days after transfer to an outside facility

Variable (normal range)	Two days before admission	Day of admission	Five days after admission	Ten days after admission	Two days after transfer
White Blood Cell k/mm3 (3.10-10.20)	11.13	26.27	14.75	12.20	13.17
Hemoglobin g/dL (13.1-16.8)	14.9	16.3	11.0	9.2	8.9
Platelet k/mm3 (119-332)	314	354	182	163	274
Lactic Acid mmol/L (0.50-1.90)		> 11.70	1.08	1.39	
BUN mg/dL (5-26)	9	18	102	30	38
Creatinine mg/dL (0.55-1.30)	0.81	2.11	11.11	1.97	2.0
Bilirubin, Total mg/dL (0.0-1.2)	0.5	2.1	5.0	1.7	1.3
Bilirubin, Direct mg/dL (< = 0.5)		1.6	2.9	1.1	
AST U/L (8-34)	40	11,867	193	90	65
ALT U/L (10-49)	< 7	1,001	171	394	289
Alk-Phos U/L (46-116)	89	152	135	165	126
INR (0.80-1.2)		5.37	1.19	0.95	1.0
Prothrombin time (9.3-12.4 seconds)		53	12.6	10.2	11.8
Ceruloplasmin mg/dL (16.0-31.0)		8.7	13.5		24.6
Copper ug/dL (72-166)		52	82		76
Urine Copper Level ug/L		2787			
Urine Copper mcg/24hrs (9-71)					40
Alk-Phos (IU/L) to total bilirubin (mg/dL) ratio		72.38			

AST, aspartate transaminase; ALT, alanine transaminase; Alk-Phos, alkaline phosphatase; INR, International Normalization Ratio; aPTT, activated partial thromboplastin

S2985

Drug-Induced Autoimmune-Like Hepatitis Secondary to Gymnema Sylvestre

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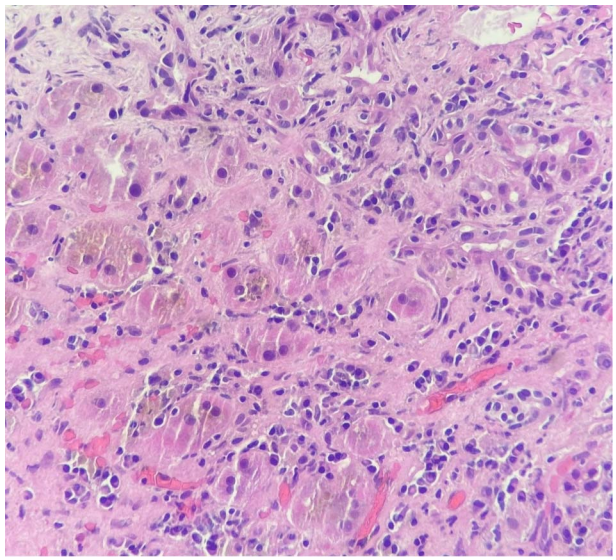
Introduction: The use of complementary and alternative medicine remains attractive despite not undergoing the same rigorous evaluations as the Federal Drug Administration-Approved medications. Gymnema sylvestre (GS) is an herbal supplement used for its hypoglycemic effects in patients with diabetes mellitus (DM), despite little evidence supporting its benefits. However, we present a patient who developed acute liver injury, via an autoimmune modality, while initiating therapy with GS.

Case Description/Methods: A 67-year-old male with a history of hypertension and type II DM, who arrived at the Emergency Department with jaundice and acute elevation of liver enzymes. Laboratory workup was remarkable for the marked elevation of liver function tests (LFTs), with AST 2247 U/L and ALT 2100 U/L, elevated total bilirubin levels at 19.8 mg/dL with direct bilirubin predominance. R-factor at that time was 16, suggestive of hepatocellular injury. Further workup including urine toxicology, anti-mitochondrial antibody, HIV, CMV, EBV, HSV, and ceruloplasmin levels were unremarkable. However, the anti-smooth muscle antibody and HAV antibody were positive. Abdominopelvic MRI demonstrated absence of hepatic pathology. Drug-induced liver injury (DILI) was suspected from GS, which the patient was taking for glucose control. In addition, IgG and IgA levels were found to be elevated, at 2952 and 663 respectively, resulting in an autoimmune hepatitis score of 4. LFTs started to improve after the discontinuation of GS. Interventional Radiology was consulted for liver biopsy, with findings of necrosis and a marked inflammatory infiltrate pattern of injury, as is seen in autoimmune hepatitis or DILI. High-dose steroid therapy was started along with initiating workup for liver transplant (Figure).

Discussion: GS is commonly used for glucose control in patients with DM, but its reportedly beneficial effects have only been supported in a small amount of non-randomized trials. Although LFTs were on a downward trend after discontinuation of the offending agent, the patient's synthetic liver function continued to deteriorate. Biopsy and lab work were suggestive of classic autoimmune hepatitis (AIH) vs drug-induced autoimmune-like hepatitis (DI-AIH). Prednisone was started and, considering poor prognostic factors on histology (necrosis), workup for liver transplant was initiated. Steroid therapy response may determine if the patient's acute liver injury is due to AIH or DI-AIH. Nevertheless, GS played a leading role in this patient's disease process.



[2985] **Figure 1.** Liver biopsy shows portal inflammation with marked increase in plasma cells. Marked interface activity is seen with areas of necrosis.

S2986

Fomepizole as an Adjunctive Treatment in Severe Acetaminophen Poisoning

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Introduction: Acetaminophen is the most common cause of acute liver failure (ALF) in the United States. We report a case of acetaminophen overdose treated with N-acetyl cysteine (NAC) and fomepizole.
Case Description/Methods: A 58-year-old female presented with altered mental status. Her past medical history was significant for fibromyalgia and chronic alcoholism. She had been experiencing abdominal pain over the last two days and had taken copious amounts of extra-strength acetaminophen. She was unable to specify the exact number or last ingestion of acetaminophen. Her last alcohol consumption was multiple days ago with no signs of withdrawal. Initial labs were significant for an acetaminophen level of 126.3 ug/ml, ethanol < 0.01 gm/dl %, AST of 2175U/l, ALT of 896 U/l and total bilirubin of 2.6 mg/dl, INR 1.6 (baseline of 1), lactic acid 3.9 mmol/L, phosphorus 2.5 mg/dl. NAC rescue was immediately initiated. In the following days, the transaminitis worsened to AST 34,665 U/l, and ALT 13,995 U/l. Additionally, there was an increase in INR to 4.8, bilirubin to 8.3 mg/dl, and Creatinine increased to 2.6 mg/dl (baseline 1mg/dl). Irrespective of these values, her encephalopathy improved already one day after admission. A viral and autoimmune panel remained unremarkable, imaging indicated patent veins and trace perihepatic ascites. King's College criteria were calculated daily but did not indicate the need for transplant evaluation. State poison control recommended the addition of fomepizole to the existing NAC regimen after 48 hours secondary to worsening transaminitis. The patient's liver and kidney function improved significantly returning to baseline 4 weeks after admission.
Discussion: NAC is known as the primary antidote for acetaminophen toxicity, but there might be a potential benefit to adding fomepizole, especially in massive acetaminophen overdoses. The proposed hepatoprotective effect is likely explained by an oversaturation of the regular glucuronidation pathway and a fomepizole-driven inhibition of CYP2E1. This results in a decrease of the toxic metabolite N-acetyl-p-benzoquinone imine (NAPQI). Wong et al. suggested in a retrospective data analysis that the multiplication of the concentration of acetaminophen (μg/mL) and ALT (IU/L) is correlating with the probability to develop significant hepatotoxicity. As a value of >10,000 μg/mL * IU/L predicts hepatotoxicity with a very high likelihood, the addition of fomepizole should be considered and discussed with state poison control.

S2987

Esophageal Varices in Myelofibrosis Associated Non-Cirrhotic Portal Hypertension: A Call to Screening

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Introduction: Myelofibrosis is characterized by bone marrow hematopoetic failure, leading to extramedullary hematopoiesis and can be secondary to various causes such as primary myelofibrosis or chronic proliferative neoplasms i.e. polycythemia vera myelofibrosis and essential thrombocythemia.$_1$ Mylefibrosis in the setting of primary polycythemia can led to non-cirrhotic portal hypertension with esophageal varices as demonstrated in this clinical presentation. Although esophageal varices are a relatively uncommon complication in this setting given its increased mortality, some have recommended these patients undergo screening and surveillance upper endoscopy.
Case Description/Methods: A 62 year old man presented to emergency department with hematemesis. Past medical history included secondary myelofibrosis from polycythemia vera on daily aspirin and ruxolitinib. Physical examination was pertinent for splenomegaly and hepatomegaly without ascites. Upper endoscopy (EGD) revealed diffuse gastric erosions and small esophageal varices with no high risk stigmata. Initial laboratory evaluation revealed WBC of 2.8 x 10^3/mcL, hemoglobin 7.0 g/dL, platelets 250 x 10^3/mcL, and normal liver associated enzymes. An MRI of his liver showed massive splenomegaly up to 27.4 cm, hepatomegaly up to 27.6 cm, esophageal varices, and an otherwise normal appearing liver. He had an unremarkable abdominal doppler ultrasound evaluation with normal hepatic and hepatopetal flow in the portal venous system. Fibroscan was obtained with kPa of 12. Chronic liver disease workup including infectious, autoimmune, and metabolic causes was unremarkable. The patient declined liver biopsy and the plan was made for follow-up EGD in 1 year to survey the small varices seen on the original EGD.
Discussion: While the exact mechanism of portal hypertension secondary to myelofibrosis is still under investigation, several pathophysiologic mechanisms have been proposed including: hepatic infiltration of hematopoietic stem cells, sinusoidal fibrosis, increased pre-hepatic flow as a result of profound splenomegaly, and portal venous insults.$_2$ Portal hypertension has been observed in 10% to 17% of patients with myelofibrosis.3 Given the increased risk of developing esophageal varices, patients in this population may benefit from variceal screening to proactively treat and prevent variceal hemorrhage. The role of fibroscan in risk stratifying these patients remains to be established with most studies limited to small retrospective case series.

S2988

Flavimonas Oryzihabitans: Not Your Typical Organism in a Patient With Chronic Liver Disease

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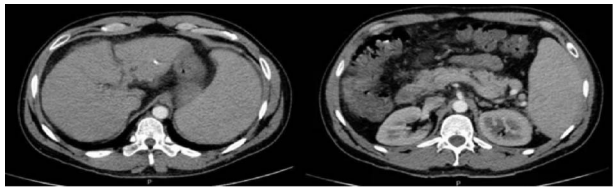
Introduction: Flavimonas Oryzihabitans is an organism typically found in rice paddies, stagnant water, and soil. There have been very few cases as a human pathogen described in literature. It has been seen in infections associated with catheters, respiratory therapy, mechanical ventilation, and other instrumentation in immunocompromised patients, particularly those with neoplastic conditions. We describe a rare case of a bacteremia by F. Oryzihabitans as a suspected contributor to the decompensation of a chronic liver disease (CLD) patient.

Case Description/Methods: A 41-year-old incarcerated male with PMH of cirrhosis, HIV (2010) on HAART, hypertension, and Hepatitis C (2017), presents to the ED with fatigue, abdominal pain and distention, and altered mental status. History was mostly from the correctional report due to altered mental state. He began experiencing abdominal pain and subjective fevers 2 days prior with associated nausea and constipation several days prior. Other symptoms included a 10lb weight loss in the past 2 months, orthopnea, leg swelling, easy bruising and slurred speech. Notably, he had a remote history of IVDA use. PE was remarkable for slurred speech, confusion, lower extremity pitting edema, hematomas in all extremities, mild abdominal distension, and abdominal pain with palpation. Laboratory workup was notable for leukopenia, predominant mild neutropenia (ANC 1042), lymphopenia (ALC 486), normocytic anemia, thrombocytopenia and borderline direct hyperbilirubinemia. Abdominal US and Abdominopelvic CT scan (Figure 1) showed splenomegaly, mild to moderate amount of ascites, colonic thickening, edema, and fat stranding suggestive of typhlitis versus changes of portal hypertension colopathy. Paracentesis was not performed due to lack of a safe, viable pocket. Yet, blood cultures revealed a gram-negative bacillus, Flavimonas Oryzihabitans. Piperacillin/ tazobactam was started to complete 14 days of therapy, and later, he was discharged with significant improvement to continue therapy for his chronic conditions.
Discussion: Flavimonas Oryzihabitans, although extremely rare, has been implicated in infections of immunocompromised patients. A history of HIV, with no known CD4 count, and CLD status may be elements to regard an immunocompromised state. It may also be plausible to consider organisms such as this as contributing factors for spontaneous bacterial peritonitis or decompensated CLD in resource limited settings and marginalized populations with the aforementioned conditions.



[2988] **Figure 1.** Abdominopelvic CT scan with IV contrast demonstrating splenomegaly, free peritoneal fluid, colonic thickening, edema, and fat stranding.

S2989

Excessive Hormonal Therapy in a Male Transitioning to a Female Causing Cirrhosis

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Introduction: High doses of progestins/estrogens can cause elevated liver enzymes, serum aminotransferase elevations with no changes in alkaline phosphatase or bilirubin, 1 to 2 weeks after treatment. These side effects are usually transient and resolve with dose modification or discontinuation. We present a unique case where a male in-transition to female was found to be cirrhotic from hormonal dose therapy. Gastroenterologists should be aware of the importance of starting hormonal therapy in the transgender process as exogenous estrogen therapy may lead to underlying liver disease.
Case Description/Methods: This is a 63 year male on hormone replacement therapy who presented to the hospital for evaluation of hyperlipidemia, hypertension, diabetes, and gender dysmorphia with concerns of questionable abdominal pain and early findings of cirrhosis. During evaluation, he complained of abdominal pain, jaundice, and dark urine. His LFTs were unremarkable prior to the visit. Upon inquiry of a prior history of fatty liver disease, he admitted not following up with his doctor. The patient then described how he had recently begun hormonal therapy to transition to a female. He reported first starting on an estrogen patch with a transition to estradiol 2mg daily. He denied having a prior history of alcohol abuse or other risk factors of cirrhosis. His liver function tests were noted to be T. Bili of 6.3, D. Bili of 3.2, ALK phos of 263, AST of 51, ALT of 176 and lipase of 100. Given his elevated LFTs an ultrasound and MRCP were performed which showed cirrhosis with portal hypertension and cholelithiasis. He did not have any synthetic liver dysfunction. Upon discontinuation of his estradiol his LFTS resolved back to baseline after five days.
Discussion: Gastroenterologists should be aware of gender-affirming hormonal therapies in the transgender population as they can lead to long term sequale such as developing cirrhosis. Evidence in the literature is sparse with no consensus on the long term effects of high dose hormone therapy. Further studies should focus on the risk factors of starting hormonal therapy using demographic characteristics, BMI, and alcohol use as further parameters for gauging cirrhosis. We present the case of a 63-old trans-female patient whose hormone therapy can be associated directly with liver cirrhosis.

S2990

Fitz Hugh Curtis Syndrome: The Zebra Amongst the Horses With Right Upper Quadrant Abdominal Pain

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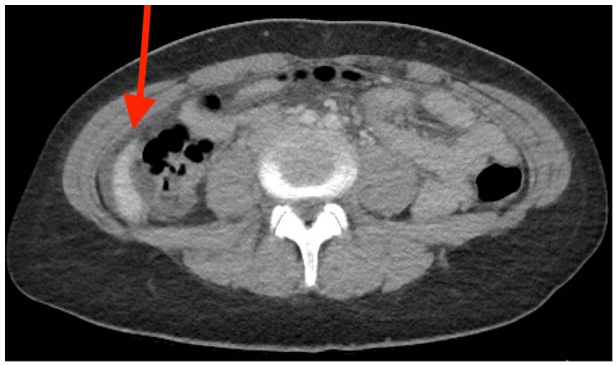
Introduction: Fitz-Hugh -Curtis syndrome (FHCS) is a perihepatitis caused by inflammation of the liver and adjacent peritoneal structures due to genital tract infections. Chlamydia trachomatis and Neisseria gonorrhoeae are commonly isolated microbes. FHCS usually manifests as right upper quadrant (RUQ) pain masquerading like other hepatobiliary and gastrointestinal conditions. Hence, FCHS can be missed without a high index of suspicion. FCHS occurs more often in women of reproductive age. The complications of untreated patients with Fitz-Hugh-Curtis syndrome include infertility and bowel obstruction due to adhesions in the peritoneum. We present a case of RUQ pain in a patient with no reported vaginal discharge diagnosed with Fitz-Hugh -Curtis syndrome.
Case Description/Methods: A 22year old young woman with a past medical history of anxiety and panic disorder presented to the emergency room with a one-month history of intermittent right upper quadrant pain, which had worsened in the last three days before presentation. She described a sharp pain, 10/10 in intensity, that radiated to her right shoulder. She also reported episodes of nonbloody and non bilious vomiting. The patient denied a history of PUD, hepatitis, vaginal discharge, or pelvic pain. Initial vital signs showed tachycardia with a heart rate of 104. On examination, she had mild tenderness in the right upper quadrant of the abdomen. Initial labs showed no leukocytosis, white blood cell count of 10,000, hemoglobin 12, platelet count of 272,000, liver function test was normal with AST 9, ALT of 7, alkaline phosphatase of 62, total bilirubin of < 0.2, lipase 20, acute hepatitis panel was negative, electrolytes were within normal limits, and pregnancy test was negative. Ultrasound RUQ was unremarkable. A computerized tomography scan of the chest and abdomen showed small ascites adjacent to the inferior aspect of the right lobe of the liver and in the dependent portion of the lower pelvis (See Fig 1). The patient was started on PPI daily. Upper endoscopy was performed, which showed mild gastritis negative for H. pylori. NAAT for gonorrhea and chlamydia was positive for chlamydia. The patient and partner were treated with doxycycline with complete resolution of her symptoms.
Discussion: FCHS is an infrequent cause of RUQ abdominal pain in women of reproductive age. Physicians should maintain a high index of suspicion in young women with referred RUQ abdominal pain to the right shoulder to help prevent extraneous workup and treatment.



[2990] **Figure 1.** A computerized tomography scan of the abdomen with an arrow showing small ascites adjacent to the inferior aspect of the liver.

S2991

Fontan Liver Disease Associated Splenic Aneurysms

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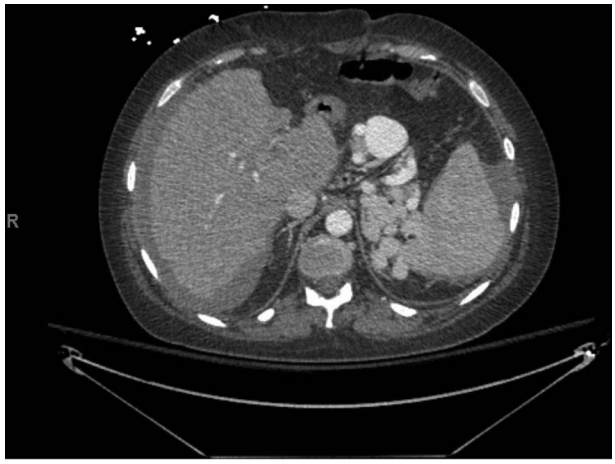
Introduction: The Fontan procedure results in liver fibrosis in all recipients and can result in many disastrous complications. Here we present a patient with hemoperitoneum from a splenic artery pseudoaneurysm.
Case Description/Methods: A 25 year old female with a history of a hypoplastic left heart status post Fontan Procedure, Atrial Flutter on rivaroxaban, cirrhosis complicated by downhill varices presented to the hospital with diffuse crampy abdominal pain. She initially attributed her symptoms to constipation, however her symptoms did not resolve after she took stool softeners which resulted in a bowel movement. Patient underwent a CT Abdomen Pelvis at an outside hospital which showed a 5.9 cm left cystic ovarian mass with concern for active hemorrhage due to surrounding fluid. On transfer, she underwent a CT Angiogram of the Abdomen and Pelvis which was notable for increased hemoperitoneum and as well as a "3.5 cm pseudoaneurysm arising from the proximal aspect of the splenic artery." This was thought to be the source of the hemoperitoneum. Patient subsequently underwent embolization of the mid splenic artery with a 10mm amplatzer plug, embolization of the splenic artery aneurysm with concerto detachable coils and embolization of the proximal splenic artery with a 12mm amplatzer plug. (Figure)
Discussion: Patient's who have undergone the Fontan procedure have universal development of liver fibrosis. Splenic artery pseudoaneurysms are an extremely rare entity and are typically associated with chronic pancreatitis, although can be associated with trauma, post-operative complications and peptic ulcer disease. Fontan associated liver disease has been reported to be associated with splenic artery aneurysms in only one other case report. This can be a potentially fatal complication.



[2991] **Figure 1.** CTA with Splenic Artery Pseudoaneurysm.

S2992

Extensive Portomesenteric Venous Thrombosis Due to *JAK2* V617F Mutation as an Indication for Multivisceral Transplantation

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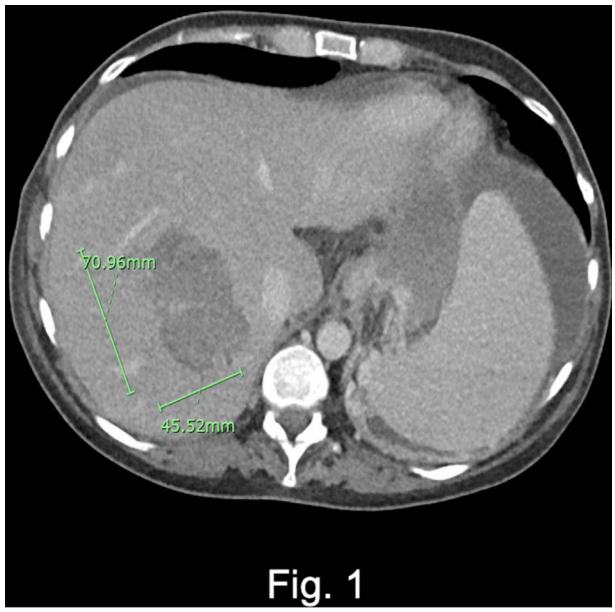
Introduction: Cavernous transformation of the portal vein (CTPV) involving periportal or intrahepatic venous collateral network is a sequelae of chronic portal vein thrombosis (PVT). This adaptation increases the risk of complications and poor outcomes from revascularization procedures. In cases with diffuse portomesenteric venous thrombosis, multivisceral transplantation (MT) may be indicated.
Case Description/Methods: An otherwise healthy female was diagnosed with Janus kinase 2 V617F (JAK2) mutation at age 43 during work up for PVT of unknown etiology. Her disease progressed with bleeding esophageal varices, portal hypertensive gastropathy, and splenomegaly. Liver biopsy was negative for cirrhosis. Bone marrow biopsy was negative for leukemia or fibrosis. She developed mesenteric vein thrombosis (MVT) at age 45 and was started on rivaroxaban, but then transitioned to high dose enoxaparin due to worsening thrombus progression. At age 47 she underwent thrombolysis for new MVT. Four months later, on current presentation, she endorsed worsening abdominal pain. Physical exam demonstrated ascites and pitting lower extremity edema. Abdominal ultrasound with venous duplex demonstrated absent flow in the portal vein. Computed tomography angiography (CTA) revealed progressive portal, superior mesenteric, and splenic vein thromboses despite anticoagulation (AC) compliance. Transjugular intrahepatic portosystemic shunt (TIPS) procedure was unsuccessful. Post-procedure CTA demonstrated an enlarging hepatic hematoma treated by embolization (figure 1). AC was discontinued resulting in worsening clot burden and hepatic ischemia. Given the extent of clot burden, the patient was transferred to another facility for a MT.
Discussion: Presented is a rare case of chronic PVT in a patient with JAK2 mutation leading to CTPV refractory to anticoagulation or endovascular intervention, eventually requiring MT. Though JAK2 is associated with myeloproliferative disorders, it is an independent risk factor for the development of PVT. With worsening thrombus chronicity and burden, cavernous transformation may occur which decreases

the chance of successful TIPS. When organ ischemia develops due to extensive portomesenteric venous thrombosis, orthotopic liver transplantation is no longer an option. Multivisceral transplantation replaces the liver, small bowel, and other abdominal organs. Replacement of the thrombosed portomesenteric system may be the only recourse to reverse portal hypertension and address the primary disease.



[2992] **Figure 1.** Computed tomography angiography of hematoma in liver segments VII and VIII, demarcated by green lines. A complication from attempted portal vein access.

S2993

Extensive Hepatic Fibrosis Mimicking Primary Liver Malignancy

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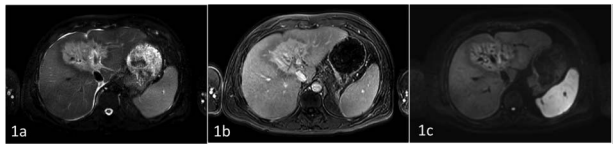
Introduction: Liver cirrhosis is a pathophysiological outcome of sustained liver inflammation. Sustained hepatic insults generate a process of fibrogenesis, in which an excessive extracellular matrix deposition replaces normal liver parenchyma. We present a patient with newly diagnosed liver cirrhosis who was found to have a liver mass suggestive of primary liver malignancy on imaging; however, multiple biopsies confirmed the mass to be fibrosis with mild hepatitis.
Case Description/Methods: A 40-year-old male presented for the evaluation of new bilateral lower extremity edema. He reported a long history of alcohol use but otherwise denied prior medical history and no medication use. Laboratory work on admission was pertinent for a total bilirubin of 3.5, AST 162, ALT 57, alkaline phosphatase 176, albumin 3.0, and a non-reactive viral hepatitis panel. CT abdomen/pelvis revealed a heterogeneous liver suggestive of cirrhosis along with a 6.8 cm x 8.7 cm mass with irregular margins situated between the right and left lobes of the liver. On 3-phase MR imaging, the lesion demonstrated progressive enhancement in the portal venous phase and hepatic venous phase, with retention of contrast on 5-minute delayed images [Image 1a, b, c]. Overall, these findings suggested infiltrative cholangiocarcinoma. Additional imaging ruled out multifocal disease, and tumor markers CEA and AFP were unremarkable. Subsequently, a diagnostic laparotomy with liver biopsy was performed. The biopsy demonstrated fibrosis with atypical ductular proliferation, but the presence of low-grade infiltrative cholangiocarcinoma could not be ruled out. To rule out biopsy sampling error, two additional biopsies later that month concurred cirrhotic liver segments with areas of mild hepatitis. Tumor markers CK20 and CDX2 were negative, and no atypical cells were present. It was concluded that this liver lesion represented extensive hepatic fibrosis and mild hepatitis due to ongoing alcohol use, but no malignancy.
Discussion: Liver cirrhosis is a major risk factor for the development of primary liver cancers such as hepatocellular carcinoma and cholangiocarcinoma. Triple-phase CT or MR imaging plays a key role in diagnosing these cancers; however, as demonstrated in our case, certain benign lesions such as liver fibrosis can mimic primary liver malignancy. Improved recognition of these benign lesions is necessary to avoid unnecessary and potentially harmful interventions.



[2993] **Figure 1.** 1a) Pre-contrast MR T2 weighted image showing a large central hepatic mass and lobular contour of liver consistent with cirrhosis. 1b) Post-gadolinium T1 fat-saturated MR image demonstrating enhancement of the liver lesion in the hepatic venous phase. The lesion showed high retention index of contrast on 5-minute delayed images (not included). 1c) Diffusion-weighted MR image showing the liver lesion with significantly restricted diffusion suggestive of infiltrative type of cholangiocarcinoma.

S2994

Fulminant Liver Failure Due to Budd-Chiari Syndrome Triggered by Strenuous Exercise

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Introduction: This case illustrates the complexity of diagnosis of Budd-Chiari syndrome especially with an atypical presentation.
Case Description/Methods: A 27-year-old man in his usual good state of health attended an all-day police academy fitness test. That evening he developed abdominal pain, nausea, vomiting, and diarrhea. He left work the next day due to these symptoms and progressive confusion. His family called EMS and he was transported to a local emergency department where he was found to be hypothermic, tachycardic, and hypoxemic. Labs were notable for acute kidney injury with creatinine 1.7, lactate 11.8, total bilirubin 1, AST 116, and ALT 95. He was begun on 2 vasopressors and empiric antibiotics. CT Abdomen noted a possible mesenteric thrombus; he was taken emergently to the operating room, but no bowel ischemia was found. His laboratory tests increased over the next 3 days reaching total bilirubin 3.0, AST 8560, ALT 5880, alkaline phosphatase 92, albumin 2.7, creatinine 3.1, INR 5.06, WBC 34.5, and ferritin 18,724. He was subsequently transferred to a tertiary center where his mental status worsened, and he was intubated. A transjugular liver biopsy demonstrated perivenular coagulative necrosis involving 80% of the hepatic parenchyma, and a hepatic venogram noted extensive portal vein thrombus. He had no history of taking any medications on a regular basis and did not take acetaminophen. An acetaminophen level was negative. Orthotopic liver transplantation was performed on the sixth day after his initial hospital presentation.

During the surgery the patient was noted to have extensive thrombus involving the portal system including the main portal vein as well as the superior mesenteric vein, and thrombus in the hepatic veins. Testing for hypercoagulable state was positive for JAK2 V617F mutation, and he was begun on treatment with hydroxyurea.
Discussion: His development of Budd-Chiari syndrome is felt to be due to this hypercoagulable state combined with dehydration from strenuous exertion. There was no hepatomegaly noted on imaging which had led to confusion regarding the diagnosis of Budd-Chiari syndrome, but the occlusion of the portal vein in this patient resulted in decreased blood flow to the liver and prevented the development of hepatomegaly. His initial development of lactic acidosis, leukocytosis, and elevated ferritin is felt to be the result of marked hepatic necrosis from the Budd-Chiari syndrome but had led to confusion over the possibility of septic shock or bowel ischemia.

S2995

Gallstone Ileus: An Unusual Cause of Small Bowel Obstruction After Liver Transplantation

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Introduction: Liver transplantation is the only curative treatment for patients with decompensated liver disease. The prevalence of biliary complications after a deceased donor liver transplant is estimated between 10 to 15%. Biliary leaks and strictures are the most common biliary complications. We are presenting a rare case of gallstone ileus at the biliary limb in a patient with a complicated post-liver transplant course.
Case Description/Methods: A 78-year-old female with a history of NASH cirrhosis for which she underwent a deceased donor liver transplant ten years before presentation. Post-transplant course was complicated by anastomotic biliary strictures, recurrent choledocholithiasis, and recurrent cholangitis requiring multiple ERCPs with placement of multiple biliary stents. She underwent Roux-en-Y hepaticojejunostomy three years before presentation with marked improvement in the number of cholangitis episodes. She presented with intractable nausea and vomiting. Physical exam showed abdominal distention and periumbilical tenderness but no rebound tenderness or rigidity. Labs were significant for a mild rise in serum creatinine but no leukocytosis and liver chemistries were normal. CT abdomen revealed small bowel obstruction with a transition point at the right lower quadrant anastomosis suspecting phytobezoar at the jejuno-jejunal anastomosis. The patient underwent push enteroscopy to decompress the obstructed limb and remove the suspected food material. The scope was advanced to the jejuno-jejunal anastomosis. About 15 cm beyond the afferent limb, the lumen was entirely occluded by a giant stone measuring (3.5 cm x 2.5 cm). The stone had sequential rings and appeared as a cholesterol stone. The stone was fragmented using grasping forceps, snares, and dilating balloons. It was successfully fragmented into 15-20 smaller stones over three hours duration. The scope was advanced beyond the fragmented stones. A nasogastric tube was inserted for decompression. Patient symptoms resolved following the procedure, and she was discharged home with no recurrence. (Figure)
Discussion: Biliary leaks and strictures are the most common biliary complications following liver transplant. Gallstone ileus should be considered in liver transplant patients presenting with small bowel obstruction, especially if they have a history of recurrent biliary complications. Prompt recognition of this complication is key, endoscopic lithotripsy should be attempted first in these patients as they are usually high risk surgical candidates.

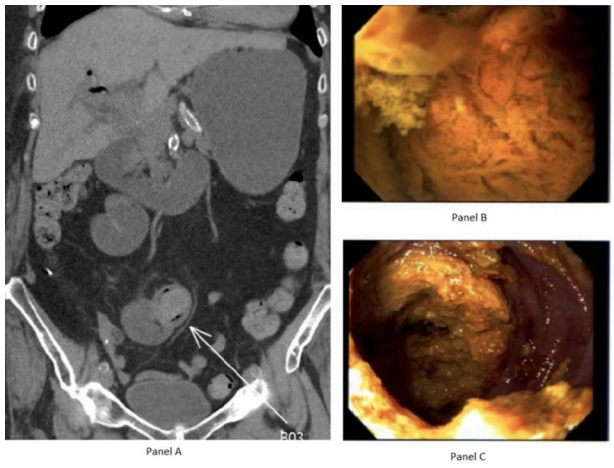

Image 1

[2995] **Figure 1.** Panel A: CT abdomen and pelvis showing dense luminal material at the transition point. Panel B: Endoscopic image of the cholesterol stone occluding the lumen before lithotripsy. Panel C: Endoscopic image of the cholesterol stone after the lithotripsy.

S2996

Erdheim-Chester Disease Associated With Aggressive Sclerosing Cholangitis

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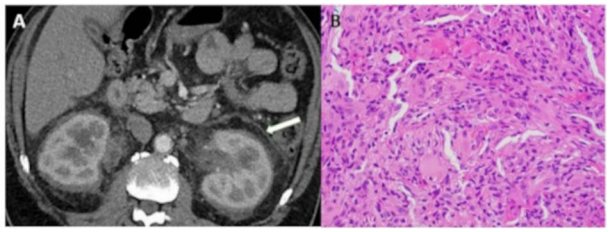
Introduction: Erdheim-Chester disease (ECD) is a rare histiocytosis recently recognized as a neoplasm due to discovery of MAPK pathway mutations. ECD can involve multiple systems, but hepatic involvement is exceedingly rare.
Case Description/Methods: A 64-year-old male presented with fatigue, pruritis, 15-pound weight loss, bilirubin 1.8 mg/dL (0.3-1), and alkaline phosphatase (ALP) 1671 IU/L (44-147). MRCP showed multifocal, short segment dilation of intrahepatic ducts only. Liver biopsy showed periportal and focal bridging fibrosis suggesting primary sclerosing cholangitis (PSC). Anti-smooth muscle, anti-mitochondrial, antineutrophil cytoplasmic antibodies (ANCA) were negative, IgG subclasses normal. A month later he developed emesis, weakness, and leukocytosis. CT scan showed perinephric and periaortic soft tissue stranding suggestive of ECD. ^{18}FDG PET/CT showed a 2.1x2.9 cm highly FDG avid mediastinal mass, follicular lymphoma on biopsy. He improved with antibiotics but returned after a few days with emesis, leukocytosis, and bilirubin 10.7 mg/dL, ALP 1277 IU/L. CT scan showed progressive perinephric and periaortic soft-tissue encasement (Figure1A). Endoscopic retrograde cholangiopancreatography showed a common bile duct (CBD) stricture and non-filling right hepatic duct. Despite CBD stenting bilirubin continued to rise. He now had molluscum like skin lesions which on biopsy revealed dermal proliferation of CD68+, Factor XIIIa+, CD1a- histiocytes and multiple giant cells characteristic of ECD (Figure1B). Targeted therapy with BRAF or MEK pathway inhibitors was considered though there was concern that he may be too sick to receive these. Patient and family opted for comfort care.
Discussion: Despite symptoms, cholestatic liver function and biopsy suggestive of PSC, several factors make us question this diagnosis; such as lack of inflammatory bowel disease or positive ANCA which are seen in 60-80% patients with PSC. PSC is usually indolent, time from diagnosis to death or liver transplant generally being 10-12 years. This patient was transitioned to hospice within 6 months. This makes us suspect hepatic involvement with ECD or an overlap syndrome, or that PSC associated with ECD runs a much more aggressive course. Though liver and CBD biopsies did not show ECD, it is not unusual for biopsy findings in ECD to be obscured by non-specific fibrosis. We want to highlight that a negative biopsy should not delay diagnosis of ECD if imaging is indicative, especially as effective therapy is now available.



[2996] **Figure 1.** 1A: CT abdomen showing perinephric soft tissue stranding and encasement. B: extensive histiocytic infiltration (majority of cells) seen on skin biopsy.

S2997

Fifteen-Year Evolution of a Rare Hepatic Cystic Lesion: A Case Report

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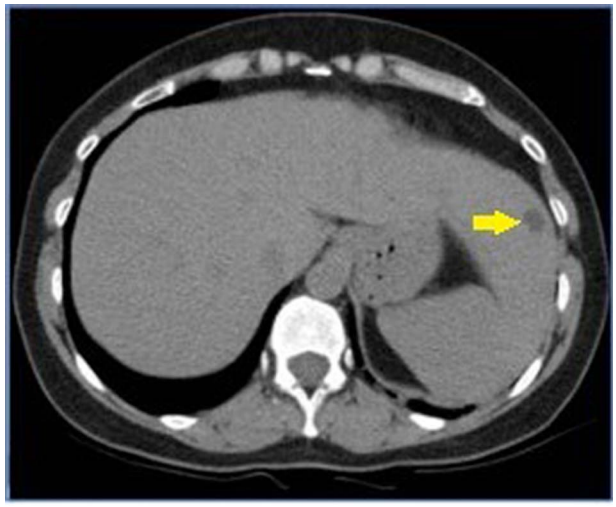
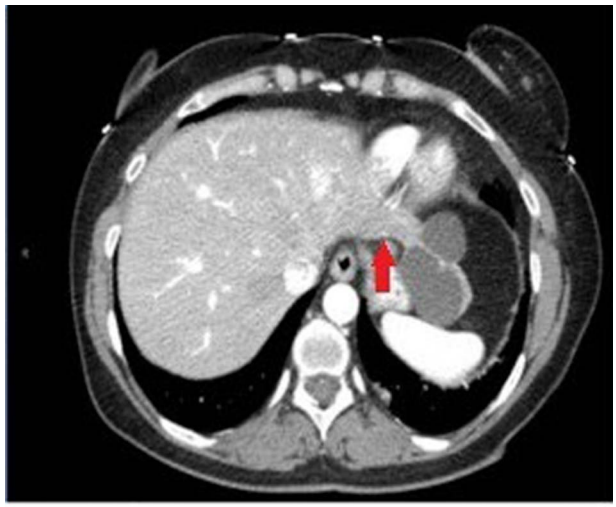
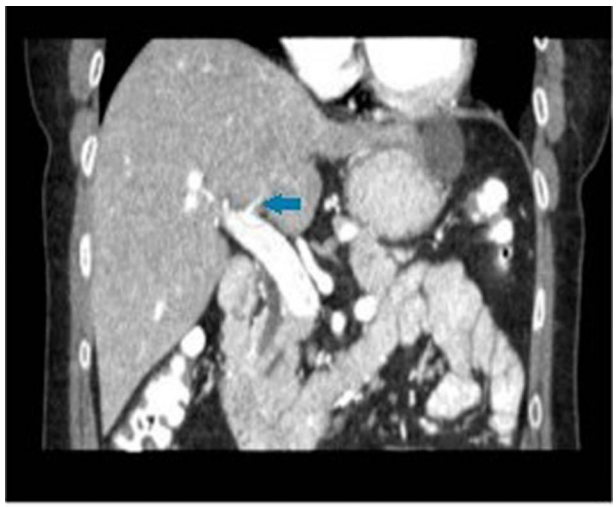
Introduction: Hepatic Cysts (HC) are often common incidental findings on Computed Tomography (CT) scans which enlarge silently for years before identification. Here we describe a rare etiology in a patient with hypercoagulable status.
Case Description/Methods: 54 year old female with recurrent deep vein thrombosis due to elevated Factor VIII levels, on oral anticoagulation, presents in 2019 for evaluation of incidentally found abdominal cystic lesion. She was asymptomatic. Detailed review of the previous imaging was performed. Initial CT in 2006 showed a 1.1cm hypodense lesion in an enlarged left lobe that extended far to left of midline to anterior to the spleen. (Fig 1). A 2014 MRI showed evidence of a diminutive left portal vein with associated atrophy of the hepatic parenchyma (Fig 2.). This was followed with serial imaging and by 2021, the lesion measured 7.7 x 8.5cm (Fig 3). Due to gradual increase in size and mass effect on the stomach (Fig 4) endoscopic ultrasound (EUS) was performed for further evaluation, which showed a well-defined, lobulated, anechoic and cystic lesion with no associated solid mass (Fig 5). Fine needle aspiration (FNA) yielded a clear, colorless fluid devoid of malignant cells. Fluid Carcinoembryonic antigen (CEA) level was 15.2ng/mL. Immunohistology positive for Epithelial Antigen.
Discussion: HCs are found in 5% of the population and remain clinically insignificant. Larger cysts can present with pain, biliary obstruction, hemorrhage or intraperitoneal rupture. On ultrasound, HCs are anechoic fluid-filled lesions with imperceptible walls and posterior acoustic enhancement. FNA can help identify HCs with atypical sonographic appearance as they are lined by cuboidal epithelium similar to bile duct cells, have elevated carbohydrate antigen (CA) 19-9 levels and low CEA levels. In this patient, we hypothesize that hepatic atrophy occurred due to portal vein thrombosis(PVT) given that a study showed that elevated factor VIII levels were found in 84% of patients with idiopathic PVT. Additionally, diversion of portal venous flow from hepatocytes causes loss of hepatotrophic substances leading to atrophy as previously reported by *Hann et al*[2]. Considering the evolution of the cyst morphology on serial imaging, non-bilious fluid on aspiration, and a low fluid CEA, this cyst was considered a benign, non-mucinous simple cyst that enlarged over time in the background of left portal vein thrombosis (PVT) due to her hypercoagulable state and resultant to left lobar atrophy.



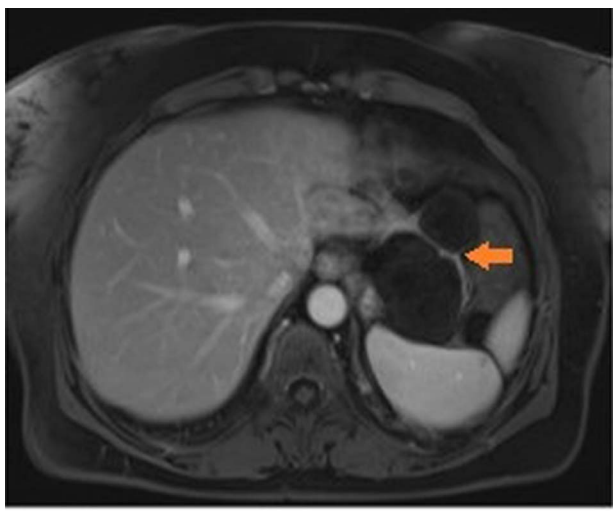
[2997] **Figure 1.** Left to Right.CT A/P without contrast (Mar 2006): The left hepatic lobe is large and extends anterior to the spleen in the left upper quadrant. A 1.1cm well-circumscribed hypodensity in hepatic segment II is present and likely represents a small hepatic cyst (yellow arrow).



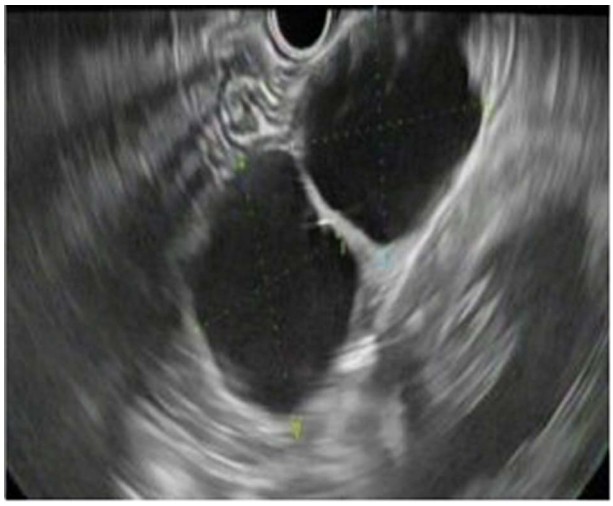
[2997] **Figure 2.** Axial image (Fig 2) from a CT with contrast from June 2014 demonstrates interval left hepatic lobar atrophy; atrophic parenchyma is observed bridging to a now larger cystic structure (red arrow).



[2997] **Figure 3.** Coronal image (Fig 3) from the same examination shows a diminutive vs absent left portal vein (blue arrow).



[2997] **Figure 4.** 2021 MRI Abdomen: Notable for a 7.7 x 8.5 x 4.0cm multiloculated, encapsulated T2 hyperintense and T2 hypointense cystic structure (orange arrow) present along the greater curvature of the stomach and splenic hilum in the left subphrenic space.



[2997] **Figure 5.** EUS 2022 An anechoic and multi-cystic mass measured 48 mm by 45 mm in maximal cross-sectional diameter. The endosonographic borders were well-defined.

S2998

Fatal Drug-Induced Liver Injury in a Patient With Known Chronic Liver Disease Secondary to Ethanol and Hemochromatosis

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Introduction: Drug-induced liver injury (DILI) is defined as a liver injury caused by various medications, leading to abnormalities in liver tests or liver dysfunction. Amoxicillin–clavulanate (AC) is the most frequent cause of idiosyncratic DILI, and the clavulanic acid component is the likely injurious agent. Pre-existing liver disease is associated with higher rates of severe DILI and three times higher risk of mortality in comparison to those without prior liver disease.
Case Description/Methods: 73-year-old male with a history of chronic liver disease secondary to hemochromatosis and alcohol abuse presented with progressive jaundice, pruritus and choluria for a week. He received a 14-day course of Amoxicillin-Clavulanate for right gluteal abscess 2 weeks prior to presentation. Physical examination pertinent for jaundice and stigmata of chronic liver disease. Laboratories displayed a disproportionate elevation in alkaline phosphatase (417 U/L) compared to serum aminotransferases (AST 112U/L, ALT 92 U/L). Bilirubin levels were significantly elevated at 18.1 mg/dl with direct predominance >10.0mg/dl; all labs consistent with cholestatic injury. Serologies revealed a negative HAV IgM, HBSAg, HB core IgM, and HCV PCR. MRCP showed no evidence of choledocholithiasis or intra or extrahepatic biliary ductal dilatation. No evidence of an active infection. The patient was managed with rifaximin, lactulose, spironolactone, furosemide, and cholestyramine powder. Clinical scenario was then complicated by acute disseminated intravascular coagulation (DIC) and multiorgan failure. Despite aggressive management with blood products, antibiotics, vasopressors, rewarming, fluids, colloid administration, and critical care therapy, the patient did not survive admission. Pathology autopsy report with macronodular cirrhosis, ductular reaction, macrovesicular steatosis, stasis, hemorrhage, and iron depositions with determination of cirrhosis and hepatic failure as primary cause of death.
Discussion: This case report emphasizes the importance of identifying potential hepatotoxic agents such as AC in patients with already established chronic liver disease as they can precipitate life threatening complications such as DILI or DIC. Physicians should also be aware that susceptibility to drugs or chemical compounds can be altered in iron-overloaded livers such as this patient with known history of Hemochromatosis.

S2999

Flood Syndrome: A Herniating Complication of Liver Cirrhosis

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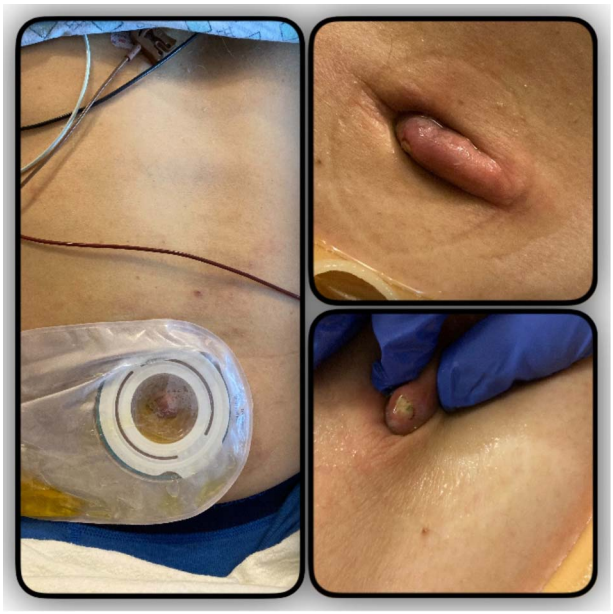
Introduction: Flood syndrome is a rare complication of patients with end-stage liver cirrhosis characterized by coinciding ventral hernia with spontaneous umbilical hernia rupture and extrusion of ascitic fluid through the defect. We present a unique case of Flood syndrome in a patient with decompensated cirrhosis and umbilical hernia.
Case Description/Methods: A 51-year-old Caucasian male with a past medical history of an alcohol use disorder, end-stage cirrhosis [Model for End-Stage Liver Disease (MELD) score of 27], portal hypertensive gastropathy, and esophageal varices was presented to the hospital after noticing gushing of an around a liter of fluid through his umbilicus. Physical exam was significant for the stigmata of liver disease with visible scleral icterus, jaundiced skin, spider angiomata, and distended abdomen. Also evident was a compressible umbilical hernia (4 cm x 2 cm) with draining straw-colored serous fluid (Figure). The patient was placed on fluid restriction, and a drainage bag was placed over the draining umbilicus. A total of 4 liters of fluid was drained from his umbilicus for the first two days of admission, which decreased to 1 liter daily by day four of his hospitalization. Fluid analysis of the ascitic fluid did not show any evidence of spontaneous bacterial peritonitis. Due to the high MELD score, a transjugular intrahepatic portosystemic shunting procedure (TIPS) was initially not recommended. After a multidisciplinary discussion, an orthotopic liver transplant evaluation was planned while focusing on stabilizing renal function and decompensated liver failure in the hospital.
Discussion: An increase in intraabdominal pressure from worsening ascites leads to the hernia rupture and ultimate leakage of peritoneal fluid through the weakened abdominal wall at the site of the herniation. Patients with end-stage liver disease and long-standing ascites are susceptible to the rare and potentially life-threatening flooding syndrome. Treatment for flood syndrome is complex and multifactorial. Due to its rarity, there is no consensus on therapeutic guidelines. However, an impending rupture can be predicted by skin color changes, excoriation, ulcers, or necrosis over the umbilical hernia, which often requires aggressive intervention. Physicians may attempt TIPS, elective herniorrhaphy, portal venous decompression, and peritoneovenous shunts as an alternative to failed conservative methods with diuretics and regular paracentesis.



[2999] **Figure 1.** A large umbilical hernia (4 cm x 2 cm) with overlying erythema and excoriation draining ascitic fluid.

S3000

Ezetimibe-Induced Autoimmune Hepatitis: An Uncommon Offender

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Introduction: Drug-induced autoimmune hepatitis (DIAIH) is a subset of autoimmune hepatitis (AIH) leading to a seropositive hepatitis caused by an inciting drug. Ezetimibe is a cholesterol absorption inhibitor within the intestinal tract that very rarely causes DIAIH. The proposed mechanism of Ezetimibe-induced DIAIH is thought to be due to interactions between drug metabolites and CYP450 within hepatic cells. Statins are another class of medication known to cause hepatotoxicity that are commonly used in combination with ezetimibe for hyperlipidemia. Highlighting cases of drug-related autoimmune hepatitis can be useful as routine liver function testing for statin-induced hepatitis is currently not recommended.
Case Description/Methods: This case describes a 67 year old man with hyperlipidemia and Hashimoto's thyroiditis admitted for abdominal pain and biochemical evidence of hepatitis after starting ezetimibe 6 weeks prior. He had also been on atorvastatin but had been on a stable dose for the previous 7 years. He did not have a history of alcohol, herbal medication or Tylenol use and had entirely normal liver function tests 6 months prior to admission. Admission labs were pertinent for AST of 2159, ALT of 3011, Alkaline phosphatase of 245, total bilirubin of 2.7 (direct bilirubin 1.3), INR of 1.3, and a creatinine kinase of 497. Autoimmune serologies were notable for a positive ANA (1:320) and anti-smooth muscle antibody (1:320). An ultrasound was negative for hepatic lesions or evidence of advanced cirrhosis. Ezetimibe and atorvastatin therapy were discontinued on admission and the patient was eventually discharged after his liver function tests improved.
Discussion: Ezetimibe is a common 2nd line agent used for patients with statin-refractory hyperlipidemia which has also been known to cause hepatic injury typically with a latency period of 1-2 months into treatment. When used in combination with a statin the risk of hepatic injury increases. Current guidelines recommend against regular surveillance of liver function tests during statin treatment given that significant hepatotoxicity is exceedingly rare and routine testing did not prevent these events. However, in patients taking both a statin and ezetimibe it may be beneficial to obtain pre-treatment and in-treatment liver function testing towards the end of the latency period, especially if the incidence of ezetimibe-induced autoimmune hepatitis continues to rise.

S3001

Fighting the Odds: A 31-Year-Old Long-Term Survivor of a Kasai Procedure With a Native Liver

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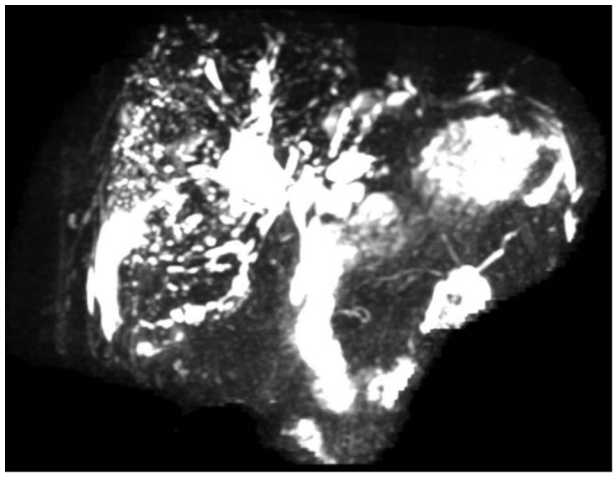
Introduction: Congenital biliary atresia is a leading cause of neonatal cholestasis and is a common indication for liver transplantation world wide. The Kasai is a surgical procedure (hepatoportoenterostomy) involving radical excision of the biliary ducts and helps reestablish bile flow and attempts to prevent biliary cirrhosis. Surgical correction can prolong life but almost 80% of patients will require a liver transplant and almost half require surgery before the age of 2. The 20 year survival rate with native liver is less than 45%. We present a unique case of a 31 year old male with biliary atresia who underwent the Kasai Procedure and has still not required a liver transplant despite his age.
Case Description/Methods: This is a 31 year old male with a known history of having biliary atresia status post Kasai procedure performed at (5 weeks old) presented for evaluation of abdominal pain. On the physical exam, he had jaundice and abdominal distention. His labs were pertinent for a total bilirubin of 3.1, alkaline phosphatase 187, AST 50, ALT 84, platelets 88, INR of 1.07 and albumin 4.2. An MRCP was performed to rule out cholangitis which showed postoperative changes of Kasai procedure, minimal intrahepatic duct dilation with marked left lobe atrophy and sequelae of hypertension. He was treated with a course of intravenous antibiotic therapy and was diagnosed with cholangitis secondary to biliary stasis from his previous Kasai procedure. He was later followed by a Hepatologist who performed a fibroscan and diagnosed the patient with F4 fibrosis with Steatosis of S1 consistent with cirrhosis. His MELD score was noted to be 10 and he was placed on ursodiol. A screening EGD was performed which showed grade 2 esophageal varices. The patient's plan is to eventually undergo a liver transplant in the near future. (figure)
Discussion: Common complications of patients with a prior Kasai include recurrent cholangitis, portal hypertension, and synthetic liver dysfunction which was seen in our patient. Prior case reports have shown that very few patients have survived longer than 20 years without a liver transplant. It is important for gastroenterologists to know the long term side effects and complications that can be seen in these patients as they transition to adulthood. Our case highlights a rare case of a 31 year old male who survived against all odds and still has not required a liver transplant. To our knowledge and after an extensive literature review, this is the longest survivor to date in the US.



[3001] **Figure 1.** MRCP showing evidence of Kasai Procedure.

S3002

Gastrointestinal Variant of Lemierre's Syndrome Due to Fusobacterium Nucleatum: A Case Report

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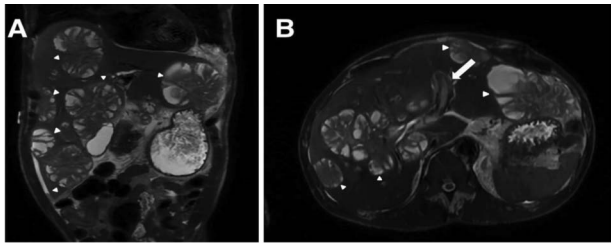
Introduction: Fusobacterium species are an extremely uncommon cause of pyogenic liver abscess (PLA) and are rarely isolated in the clinical setting. Herein, we report a rare case of cryptogenic Fusobacterium nucleatum-associated liver abscess and septic thrombophlebitis in an apparently immunocompetent patient.
Case Description/Methods: A 51-year-old male patient presented to the emergency department with a two-week history of abdominal pain, distension, nausea and chills. His physical exam revealed a distended abdomen with tenderness to palpation over the right upper and lower quadrants, and palpable hepatomegaly. Laboratory evaluation revealed WBC of 13,360 cells/mm3, lactic acid of 5.2, hemoglobin 8.0 g/dL, ALT of 73 IU/L, AST of 171 IU/L, ALP 625 IU/L, total bilirubin of 1.9 mg/dL and direct bilirubin 1.3 mg/dL. Magnetic resonance imaging (MRI) of the abdomen with and without contrast revealed innumerable multiseptated cystic hepatic masses with an associated portal vein thrombosis. The largest of these cystic lesions was measured at 9.0 cm x 5.4 cm. Patient received empiric intravenous antibiotics and therapeutic intravenous heparin. He underwent a CT-guided liver biopsy with aspiration of abscess material. Blood cultures and aspirate culture were negative. Next generation sequencing 16S PCR of the aspirate was positive for Fusobacterium nucleatum. Unfortunately, the patient passed away due to cardiac arrest before the etiology of the liver lesions could be established. (Figure)
Discussion: Fusobacterium nucleatum is a rare cause of PLAs with only 20 cases reported in literature. Risk factors for development include recent pharyngitis, periodontal disease or otherwise cryptogenic. Fusobacterium can cause a unique GI variant of Lemierre's syndrome (LS) presenting with an intra-abdominal infection and associated septic thrombophlebitis of the portal venous system known as pylephlebitis. Main presenting symptoms in most patients include fever, chills, right upper quadrant abdominal pain, vomiting and shortness of breath. The gold standard for diagnosis of PLA is fine needle aspiration for culture, however aspirate cultures are positive in only 70-80% of cases. Ribosomal RNA (rRNA) gene PCR can be used for detection and identification of bacterial pathogens, as shown in this case. We provided this case report to increase awareness of Fusobacterium- species associated GI variant of LS and to add knowledge to the literature about its presentation, diagnosis, and available treatment options.



[3002] **Figure 1.** MRI of the abdomen, showing numerous cystic hepatic lesions throughout the grossly enlarged liver indicated by white arrowheads (A). White arrows point to an area of increased attenuation within the portal vein indicative of thrombus (B).

S3003

Granulomatous Hepatitis as Primary Manifestation of Disseminated Histoplasmosis in an Immunocompetent Host Diagnosed on EUS FNA and Liver Biopsy

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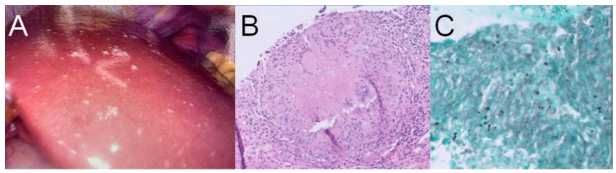
Introduction: Disseminated histoplasmosis (DH) presents as primarily lung manifestations with extrapulmonary involvement in immunocompromised hosts. Granulomatous hepatitis as first presentation of DH in an immunocompetent host is uncommon.
Case Description/Methods: 25-year-old female presented with one month of fever, fatigue, myalgias, 30-pound weight loss, cough, nausea, vomiting, and epigastric pain. She has lived in the Midwest and southwestern US. Presenting labs: TB 1.9 mg/dL, AP 161 U/L, AST 172 U/L, ALT 463 U/L. Workup was negative for COVID, viral/autoimmune hepatitis, sarcoidosis, tuberculosis, and HIV. CT scan showed suspected gallstones and 9 mm left lower lobe noncalcified nodule. EUS showed a normal common bile duct, gallbladder sludge and enlarged porta hepatis lymph nodes which underwent fine needle aspiration (FNA). She was diagnosed with biliary colic and underwent cholecystectomy, with white plaques noted on the liver surface (A). Liver biopsy/FNA showed necrotizing granulomas (B) and fungal yeast on GMS stain (C). Although histoplasmosis urine and blood antigens were negative, histoplasmosis complement fixation was >1:256. She could not tolerate itraconazole for DH, requiring amphotericin B. She then transitioned to voriconazole, discontinued after 5 weeks due to increasing AP. However, her symptoms resolved with normal transaminases. At one year follow up, she is asymptomatic with normal liver function tests.
Discussion: DH is a systemic granulomatous disease caused by Histoplasma capsulatum endemic to Ohio, Mississippi River Valley, and southeastern US. DH more commonly affects immunocompromised hosts with AIDS, immunosuppressants, and organ transplant. Gastrointestinal involvement is common in DH (70-90%) with liver involvement in 90%. However, granulomatous hepatitis as primary manifestation of DH is rare (4% of liver biopsies). Hepatic granulomas are seen in < 20%. Patients may present with nonspecific systemic symptoms. Serum/urine antigens may be negative. Gold standard for diagnosis is identifying yeast on tissue stains. Recommended treatment is amphotericin B followed by 1 year of itraconazole. However, shorter treatment duration may be effective in immunocompetent hosts.

This case is unique in that granulomatous hepatitis was the first presentation of DH in our immunocompetent patient diagnosed on EUS FNA and liver biopsy. Clinicians must have a high degree of suspicion for DH in patients with fever of unknown origin especially in endemic areas regardless of immunologic status.



[3003] **Figure 1.** Liver surface, liver biopsy, porta hepatis lymph node FNA.

S3004

Giant Hepatic Hemangioma: Dulling the Blade of Ockham's Razor

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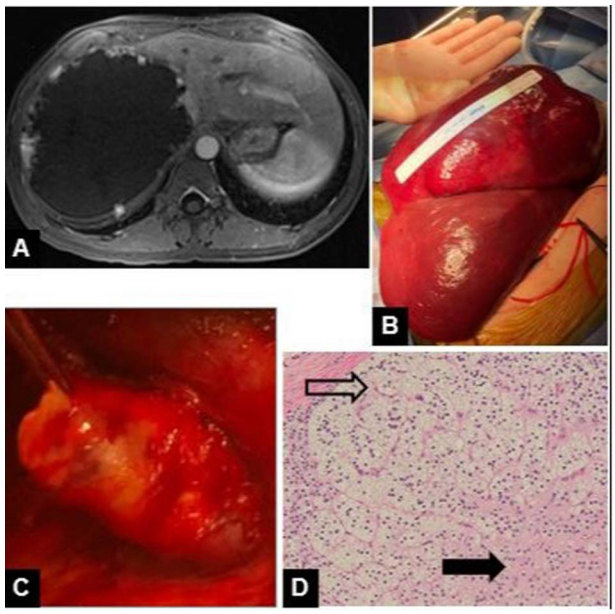
Introduction: *Ockham's razor* suggests the simplest diagnosis is likely correct. *Hickman's dictum* says multiple diagnoses may occur in one patient. We present a patient with abdominal pain due to giant hepatic hemangioma, as well as secondary hypertension due to adrenal mass masked by giant hepatic hemangioma.
Case Description/Methods: A 38-year-old female with abnormal uterine bleeding due to cervical ectropion developed right-sided abdominal pain and hypertension two years prior to presentation. She reported increased facial hair and acne which improved on spironolactone. Other medications included amlodipine, irbesartan, and progestin intrauterine device. Physical exam showed normal vital signs, facial hirsutism and acne, and abdominal distension with tenderness to palpation in the right upper and lower quadrants. Laboratory studies including serum adrenocorticotropic hormone, cortisol, aldosterone, and renin, and plasma and urine norepinephrine, epinephrine, and dopamine levels were unremarkable (Table 1). Magnetic resonance imaging with intravenous gadobutrol showed a 13.9 x 14.1 x 20.2 centimeter (cm) mass replacing the right hepatic lobe, compatible with hemangioma (Figure 1A), normal left adrenal gland, and patent renal vessels. The right adrenal gland was not visualized. She underwent right hepatic resection of hemangioma with enucleation (Fig. 1B). This allowed visualization and palpation of the right adrenal gland, revealing a 3 cm mass on the inferior portion (Fig. 1C). Right adrenalectomy was performed. Pathology revealed a well-demarcated vascular neoplasm consistent with liver hemangioma. Right adrenalectomy specimen revealed nodular cortical hyperplasia (Fig. 1D), correlating with aldosterone hypersecretion. Following surgery, she was normotensive without medication, and hirsutism resolved. Hepatic hemangioma alone is not known to cause hypertension. Her hypertension was attributed to adrenal hyperplasia, with potential elevation of serum aldosterone masked by spironolactone.
Discussion: Our patient presented with concurrent onset of abdominal pain and hypertension, secondary to two distinct, unrelated entities – hepatic hemangioma and unilateral adrenal hyperplasia. This unique case of a giant hepatic hemangioma obscuring radiographic views of an adrenal mass reminds us that patients can have as many diseases as they please.



[3004] **Figure 1.** A, MRI of the abdomen with intravenous gadobutrol shows 13.9 x 14.1 x 20.2 cm hemangioma replacing the right lobe. B, Giant liver hemangioma specimen. C, Intraoperative photo of right adrenal mass. D, Adrenal cortical hyperplasia with enlarged lipid-rich cells (black open arrow) and lipid-depleted cells (black solid arrow. Hematoxylin and Eosin stain, magnification 200X.

Table 1. Laboratory Data

Serologies	Patient Values	Reference Range
White blood cell count (K/μL)	4.38	3.98-10.04
Hematocrit (%)	10.9	11.2-15.7
Hemoglobin (g/dL)	33.3	34.1-44.9
Platelet count (K/μL)	185	173-369
Sodium (mmol/L)	139	136-145
Potassium (mmol/L)	3.7	3.4-5.1
Chloride (mmol/L)	101	98-107
Total CO2 (mmol/L)	26	22-29
Urea nitrogen (mg/dL)	14	6-20
Creatinine (mg/dL)	0.60	0.51-0.95
Glucose (mg/dL)	88	74-99
Alkaline phosphatase (U/L)	66	35-105
Alanine aminotransferase (U/L)	19	0-33
Aspartate aminotransferase (U/L)	16	0-32
Bilirubin, total (mg/dL)	0.5	0.0-1.2
Bilirubin, direct (mg/dL)	< 0.2	0.0-0.3
Activated partial thromboplastin time (sec)	31.2	25.3-37.3
Prothrombin time (sec)	14.9	11.6-15.2
Hemoglobin A1c (%)	5.6	4.0-6.0
Albumin (g/dL)	4.2	3.5-5.2
Calcium (mmol/L)	2.30	2.15-2.55
Magnesium (mmol/L)	0.81	0.66-1.07
Phosphorus (mg/dL)	3.1	2.5-4.5
Thyroid stimulating hormone (mcIU/mL)	1.24	0.27-4.2
Cortisol, serum	5.9	5.0-25.0
Follicle stimulating hormone (U/L)	3.5	< 21
Luteinizing hormone (U/L)	3.9	< 77
Estradiol, serum (pg/mL)	212.1	15-350
Dehydroepiandrosterone sulfate (mcg/mL)	0.85	0.35-4.30
Norepinephrine, plasma (pg/mL)	286	112-750
Epinephrine, plasma (pg/mL)	21	0-50
Dopamine, plasma (pg/mL)	< 25	0-29
Fractionated normetanephrine (pg/mL)	70	18-112
Fractionated metanephrine (pg/mL)	14	12-61
Aldosterone, serum (ng/dL)	6.6	< 44.5
Renin, plasma (ng/mL/h)	0.8	0.6-4.3
Adrenocorticotropic hormone (pg/mL)	8.2	5.0-46.0
Alpha fetoprotein (ng/mL)	3.7	0.6-6.6
Sex hormone binding globulin (nmol/L)	100	18-114
Testosterone, total (ng/dL)	< 20	< 81
Vitamin D, 1,25-dihydroxy (pg/mL)	64	20-79
Parathyroid hormone, intact (pg/mL)	48.1	15-65
Serotonin, whole blood (ng/mL)	87	< 330
Chromogranin A (ng/mL)	38	< 93
Human chorionic gonadotropin (IU/L)	< 1	0-5
Epinephrine, urine (mcg/24h)	8.3	< 21
Norepinephrine, urine (mcg/24h)	61	15-80
Dopamine, urine (mcg/24h)	250	65-400

S3005

Glycogenic Hepatopathy in a Type 1 Diabetic

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Introduction: Glycogenic hepatopathy (GH) results from excessive intrahepatic glycogen accumulation and is a rare and underdiagnosed complication of longstanding uncontrolled type 1 diabetes mellitus. However, due to the potential reversibility, greater awareness of this condition should help increase diagnosis and help further to establish guidelines concerning management, outcomes, and screening.

Case Description/Methods: A 24-year-old Caucasian male with a history of type 1 diabetes mellitus with recurrent hospitalizations for Diabetic ketoacidosis (DKA) presented to the emergency department with nausea, vomiting, and decreased p.o. intake. The patient was found to be in DKA and started on an Insulin drip in the emergency department. HbA1c was >14.0, indicating medication non-compliance at home. AST and ALT were initially mildly elevated at 149 and 349, respectively, then peaked on hospital day two at 2596 and 693. Acetaminophen, acute hepatitis, AA panel, and urine toxin screen were all negative. Right upper quadrant Ultrasound showed worsening hepatomegaly and findings consistent with hepatic steatosis. The patient was transitioned to subcutaneous insulin, AST and ALT were improving without additional intervention and discharge was prepared with outpatient follow-up. After an extensive record review from The Mayo Clinic, the patient had a liver biopsy consistent with GH. Due to an otherwise negative workup, it was determined that the patient had recurring GH, which spontaneously resolved over the next month. This was the fourth visit at our facility with GH, each with worsening hepatomegaly and a higher degree of AST and ALT elevation.
Discussion: GH results from excessive intrahepatic glycogen accumulation and is a rare and underdiagnosed complication of longstanding uncontrolled type 1 diabetes mellitus. Progression to end-stage liver disease has never been reported, but in the setting of recurrent admissions with worsening hepatomegaly and liver enzymes consistent with acute hepatitis, declining liver function is only a matter of time. Greater awareness will result in more cases helping to establish further guidelines concerning management, outcomes, and screening.

S3006

Granulomatous Hepatitis: The Search for a Culprit

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Introduction: Hepatic granulomas are found in 2-10% of liver biopsies. Granulomatous hepatitis (GH) has numerous etiologies and is often associated with an underlying systemic disease. We present a case of GH after extensive testing for liver disease returned normal.
Case Description/Methods: A 53-year-old Caucasian female with a history of obesity presented with 4 days of right upper quadrant (RUQ) abdominal pain, jaundice, nausea, pale stools and dark urine. She was raised on a farm with animals. Her mother died of an unknown liver disease. Liver enzyme tests revealed AST 258 U/L, ALT 236 U/L, alk phos 631 U/L, T. bili 5.5 mg/dL and D. bili 3.2 mg/dL. Comprehensive testing for liver disease was unremarkable. CT scan revealed hepatomegaly and magnetic resonance cholangiopancreatography showed hepatomegaly with diffuse hepatic steatosis. Atorvastatin was discontinued. Patient had improvement in her liver enzymes. She was discharged home after 6 days and scheduled for an outpatient liver biopsy. She returned to the hospital 5 days later with similar symptoms, persistently elevated alk phos and an increase in Tbili to 8.5 mg/dL (DBili 5.5 mg/dL). She underwent endoscopic ultrasound-guided liver biopsy which revealed granulomatous hepatitis. A thorough investigation was performed to assess the underlying cause. The patient was discharged home in stable condition while awaiting results of reference labs.
Discussion: Granulomatous hepatitis (GH) has many causes including infectious (i.e. bacterial, fungal, viral, parasitic), autoimmune (i.e. primary biliary cholangitis [PBC]), drug-induced (i.e. sulfonylureas, allopurinol), metals (i.e. beryllium, copper, gold), extrahepatic malignancy or idiopathic. Sarcoidosis, mycobacterial infection, PBC and drug-induced GH represent the most common etiologies. GH may be asymptomatic, can present with hepatobiliary symptoms or with constitutional symptoms from systemic disease. Imaging may be normal or reveal hepatomegaly as in our case. Liver biopsy is the best diagnostic tool for GH. Symptomatic and idiopathic GH often responds to corticosteroids once infection has been excluded. Methotrexate or infliximab have also been used. Offending agents should be discontinued. It is speculated that our case may have been due to exposure to a zoonotic infection. Complications of GH include fibrosis, portal hypertension and cirrhosis. GH should be considered in a patient with RUQ abdominal pain, fever, hepatomegaly and elevated LFTs in the appropriate clinical context.

S3007

Glucosamine-Induced Hepatitis - A Case Report

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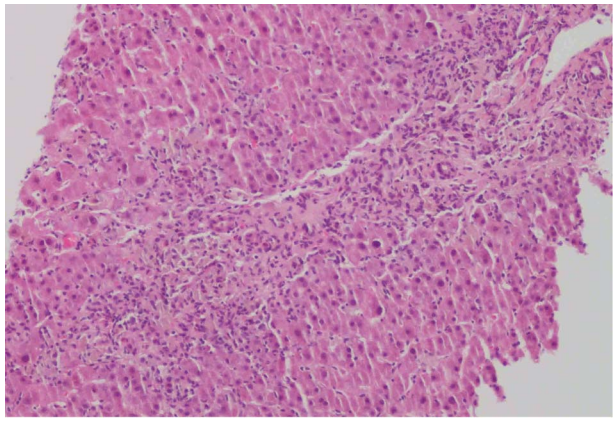
Introduction: Drug-induced liver injury (DILI) is an adverse reaction to drugs that occurs either as a predictable event when an individual is exposed to toxic doses of some compounds or as an unpredictable event at therapeutic dosing. The diagnosis of DILI relies on the exclusion of other etiologies of liver disease as specific biomarkers are still lacking. We present a case of acute hepatocellular hepatitis in the setting of recent glucosamine use.
Case Description/Methods: 57-year-old female without significant medical history presents with markedly elevated transaminases noted on routine annual physical. The patient denies any past history of liver disease or abnormal liver enzymes. Baseline weight is around 114 lbs, stable for years. She exercises regularly by spinning and walking. Pt drinks alcohol socially, average 1 glass of wine daily for 10 years, which she has withheld since onset of illness. Pt denies any history of blood transfusion, IVDU, tattoo, new medications or antibiotics. She started taking glucosamine for joint pain on 4/12. Patient had LFTs drawn on 4/22 as a part of annual evaluation by PCP which were elevated. She was having her LFTs checked daily as an outpatient with progressive rise in her AST and ALT. AST/ALT peaked at 2322 and 3335 around 4/28 accompanied by ALP elevation at 364 with TB 3.4. INR remained within normal limit and mental status has been intact. Workup included normal TSH, normal IgA/IgM/IgG, neg HAV/HBV/HCV serologies, normal Tylenol level, negative ANA/ASMA/Anti LKM and normal ceruloplasmin. RUQUS: contracted gallbladder without gallstones. MRCP: no biliary obstruction. Liver biopsy: pan lobular mixed inflammatory infiltrate with patchy bile duct damage suggestive of DILI. She was recommended to discontinue glucosamine. LFTs slowly down trended during hospitalization without any intervention and she was d/c home on 5/4. On follow up, LFTs continued to improve with the most recent LFTs on 6/15: AST/ALT 43/62, ALP and TB normal. (Figure)
Discussion: Glucosamine is used as a nutritional supplement to decrease the pain and cartilage loss in osteoarthritis. Recently, a few case reports of clinically apparent liver injury attributed to glucosamine have been published. The time to onset is usually 1 to 4 weeks and the pattern of injury is typically hepatocellular or mixed. Most patients were reported to recover within 4 to 8 weeks. We want to demonstrate and raise awareness of glucosamine as a potential cause of DILI and to consider it in the differentials for acute hepatitis.



[3007] **Figure 1.** Panlobular mixed inflammatory infiltrate, mild ductular reaction, patchy bile duct damage, hepatocanalicular cholestasis, prominent kupffer cell aggregates and acidophil bodies.

S3008

HCC Recurrence Post Liver Transplant With Rapid Progression in a Patient Presenting With Elevated Transaminases

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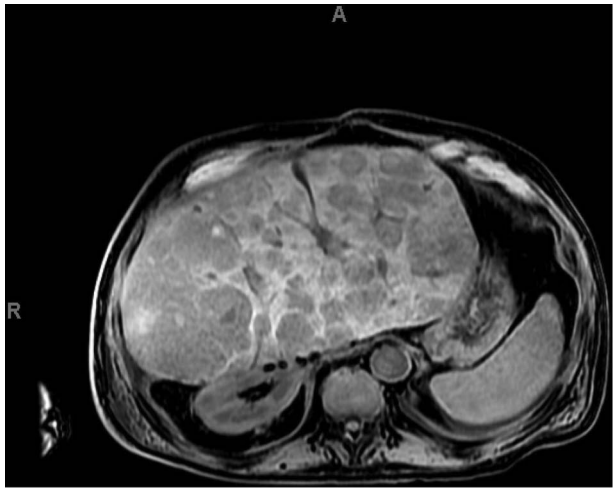
Introduction: Hepatocellular carcinoma (HCC) recurrence post liver transplant is seen in 6-18% of patients despite stringent inclusion criteria. In recurrence post-transplant, mortality is high, with median survival between 10 and 27 months. Current therapies include resection, loco-regional intervention, external beam radiation, and systemic therapy. Here we present a case of rapid progression of post-transplant HCC recurrence despite systemic chemotherapy in a patient presenting with elevated transaminases.

Case Description/Methods: A 72 year old male with a history of HCV cirrhosis complicated by HCC underwent three rounds of loco-regional therapy prior to OLT. Alpha-fetoprotein (AFP) before transplant peaked at 138 and was 26 prior to transplant. Explant pathology showed moderately differentiated HCC within Milan criteria. Vascular or perineural invasion was not noted. His post-transplant course was complicated by recurrence of HCC to the right adrenal gland and liver. He underwent resection of segments 6 and 7 of the liver and right adrenal gland. He was later noted to have widespread metastatic disease involving the liver, bones, peritoneum, and left adrenal gland and was started on Lenvatinib. Due to progression of disease after six months, he was switched to Cabozantinib. After one month on Cabozantinib, he presented to the hospital due to elevated LFTs on outpatient labs. ALP was over 1000, AST was 454, ALT 190, total bilirubin 0.7, and direct 0.5. Viral hepatitis panel was unremarkable. MRI of his abdomen was notable for a significant increase in the number of hepatic metastases, with greater than 90% of the liver replaced by tumor. Portal and hepatic veins were noted to be patent without evidence of invasion. Non-targeted liver biopsy showed HCC without evidence of acute rejection. Given the limited effective options in the setting of rapid progression on Levatinib and Cabozantinib, and current recommendations against immunotherapy in transplanted patients, he was transitioned to hospice. The patient died one week following admission. (Figure)

Discussion: Despite advancements in systemic therapies for HCC in general, therapies for patients with HCC recurrence post-transplant are limited. Our case is important as it highlights the unmet needs for systemic therapy in post-transplant patients and the need to maintain a high level of suspicion for rapid progression of diffusely infiltrative cancer in the differential diagnosis for dramatic elevation of the transaminases in select patient populations.



[3008] **Figure 1.** MRI abdomen with and without contrast demonstrating extensive tumor burden of HCC in transplanted liver.

S3009

Gastrointestinal Complications of COVID-19: A Rare Case of Intestinal Perforation, Budd-Chiari Syndrome and Liver Failure Requiring Liver Transplant

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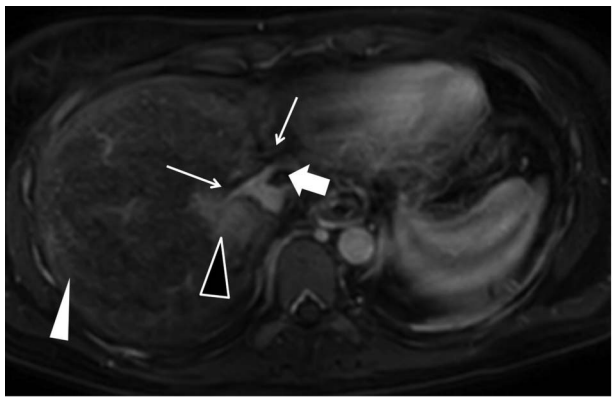
Introduction: COVID-19 commonly presents with upper respiratory illness, while some patients experience GI manifestations of nausea, vomiting or diarrhea. Rarely, other complications include transaminitis, cholecystitis, ileus, pancreatitis, or mesenteric ischemia. We discuss the unique presentation of a COVID-19 positive woman with severe abdominal pain due to duodenal perforation and Budd-Chiari syndrome. This is the first documented case in the medical literature of COVID-19 associated Budd-Chiari syndrome progressing to liver failure and requiring liver transplantation.

Case Description/Methods: A 46-year-old Female presented to the ER with severe epigastric abdominal pain, nausea, and vomiting. She was under the care of a naturopathic doctor for COVID-19 infection and received homeopathic treatment and Ivermectin. The patient reported that the ivermectin severely exacerbated her abdominal pain, and she decided to visit the ER. Physical examination revealed an ill-appearing woman with conjunctival icterus, in distress due to abdominal pain. She was tachycardic and hypertensive. Abdominal exam revealed diffuse tenderness to palpation, hepatomegaly, and hypoactive bowel sounds. Labs revealed leukocytosis, hyponatremia, lactic acidosis, hyperbilirubinemia, marked transaminitis, and an elevated INR. Acute viral hepatitis panel, along with alpha-1 antitrypsin, anti-smooth muscle antibody, and Phosphatidylethanol (PETH) were all found to be negative. CT scan revealed pneumoperitoneum, ascites, and heterogeneous liver enhancement consistent with acute hepatic injury. Emergency laparotomy was performed, where a perforated duodenal ulcer was identified and repaired. 2L of ascitic fluid along with hepatomegaly and venous congestion and diffuse oozing consistent with coagulopathy were also identified. Given acute liver failure and COVID-19 hypercoagulable state, Budd-Chiari syndrome was suspected, MRI imaging was obtained which revealed periportal edema, hepatomegaly, and hepatic vein thrombosis. Due to progressive coagulopathy, jaundice, and hepatic encephalopathy despite anticoagulation, she ultimately underwent liver transplant with no complications and has since made a complete recovery. (Figure)

Discussion: There is a growing incidence of GI perforation and thromboembolic events associated with COVID-19. A high index of suspicion for Budd-Chiari Syndrome and GI perforation should be kept in mind in patients with a history of COVID-19, as early recognition of abdominal pain or transaminitis could be lifesaving.



[3009] **Figure 1.** Axial T1FS post contrast image during portal-venous phase demonstrates recognizable imaging findings of acute Budd-Chiari syndrome with diminutive (<3 mm in diameter) left and middle hepatic veins (thin white arrows) and non visualized right hepatic vein. There is heterogeneous hepatic parenchymal enhancement with predominantly central hyper enhancement around IVC (black arrowhead) and hypoenhancing periphery (white arrowhead) that correlates with relatively preserved central liver perfusion and drainage versus congested hypoperfused periphery. Large white arrow indicated additional finding of non occlusive suprahepatic IVC thrombus.

S3010

Getting to the Bottom of the Effects of Alcohol and Kratom: A Possible Synergistic Mechanism to Hepatocellular Liver Injury

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Introduction: Kratom is a herbal derivative of an evergreen species, Mitragyna speciosa. Extracts have been used as an opioid replacement in treating chronic pain as they contain partial mu-opioid receptor activity (Schimmel et al.). Although rare, chronic kratom use has been seen to cause a cholestatic pattern of liver injury with severe hyperbilirubinemia. Our case presents a 36-year-old male who presented with drug-induced hepatocellular liver injury due to chronic kratom use.
Case Description/Methods: We report a case of a 36-year-old male with a history of alcohol dependence who presented to the hospital for evaluation of intermittent chest pain. On admission, the patient's vital signs were stable. Physical examination revealed mild epigastric tenderness. Electrocardiogram showed normal sinus rhythm without ST-T wave changes. Clinical laboratory results showed significant transaminitis in a pure hepatocellular pattern with aspartate aminotransferase (AST) 1162 and alanine aminotransferase (ALT) 913, representing an R factor of 55.9. Gamma-glutamyl transferase (GGT) level was 208. Total and direct bilirubin levels were normal. The coagulation profile and hepatitis panel were unremarkable. Prior evaluation four months ago showed AST of 111 and ALT of 132. Computed Tomography (CT) of the abdomen and ultrasonography showed evidence of hepatic steatosis. Discontinuation of kratom during the hospital course showed improvement in transaminase levels, and the patient was discharged with continued liver function monitoring outpatient.
Discussion: The interaction of alcohol and kratom has not been well studied. The literature review demonstrated case reports showing a cholestatic pattern of liver injury; however, our patient's case did not align with these findings, most notably with normal total and direct bilirubin levels. Drug-induced liver injury is usually dose-dependent, as seen with improved liver function with kratom abstinence in our case. The mechanism of injury due to regular kratom use has not been well established; however, recent studies show hepatic upregulation of a ligand-gated transcription factor leading to increased toxic metabolite formation. We hypothesize that the combination of kratom and alcohol is potentially synergistic in causing acute drug-induced liver injury. Robust medical profiles of herbal supplementation are lacking yet crucial in clinical awareness and patient education.

S3011

Got Milk? A Complex Case of Chylous Ascites

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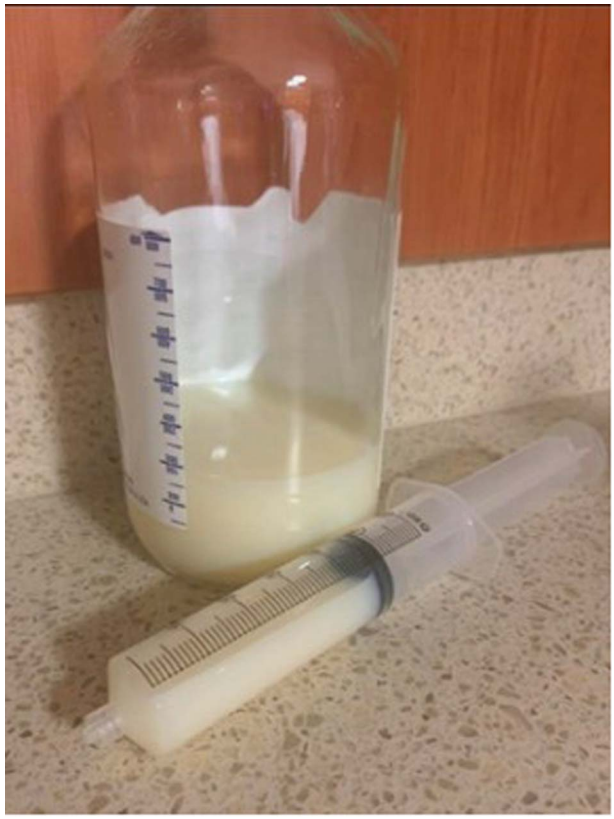
Introduction: Chylous ascites (CA) is characterized by peritoneal fluid rich in triglycerides and is rarely encountered in clinical practice. Herein, we report a case of recurrent CA suspected to be caused by multiple abdominal surgeries.
Case Description/Methods: A 58-year-old female with history of obesity status post Roux-en-Y gastric bypass, and ventral wall hernia incarceration presented with abdominal ascites, lower extremity edema, and abdominal pain. The patient had her bypass surgery 14 years earlier, and had undergone ventral herniorrhaphy one year prior to presentation. Abdominal paracentesis removed 7 liters of cloudy fluid with a triglyceride level of 592 mg/dL (reference range < 110 mg/dL), consistent with chylous ascites. Liver biopsy showed low grade nodular regenerative hyperplasia without evidence of bridging fibrosis or cirrhosis. Patient had thrombocytosis (platelet count of 555,000) and cytology testing for underlying myeloproliferative disorder was performed and was negative. Patient underwent CT imaging of chest/abdomen/pelvis along with further infectious workup and was ruled out for overt malignancy and tuberculosis. Lymphatic duct injury or leakage from prior surgery was considered highly on the differential and lymphoscintigraphy obtained, however this showed no abnormal accumulation of tracer in chest or abdomen. Patient was placed on high protein and low-fat diet with medium chain triglycerides to control accumulation of ascitic fluid. (Figure)
Discussion: Chylous Ascites (CA) is a rare form of triglyceride-rich ascites that typically occurs from underlying malignancy, cirrhosis, or lymphatic disruption after abdominal surgery. The etiology of our patient's CA was most likely from prior abdominal surgery as there was no evidence of underlying malignancy or cirrhosis. Lymphangiography can be performed to help identify source of leak, however it was not performed in our patient due to risks from the study including tissue necrosis and fat embolism. The goal of treatment is to address underlying causes, and when this is elusive management revolves around control of the accumulation of chylous ascites. Diuretics have no role in management, and a high protein and low-fat diet with medium chain triglycerides is recommended to slow accumulation of ascites.



[3011] **Figure 1.** Patient's ascitic fluid sample.

Table 1. Notable Labs

Lab Test	Values
WBC x 10^3	8.22
RBC x 10^6	3.76 (L)
HgB	9.7 (L)
HCT	30.8 (L)
MCV	81.9
RDW-SD	52.9 (H)
PLT x 10^3	624 (H)
Na	131 (L)
Total Bilirubin	0.2
Calcium	7.3 (L)
Albumin	1.9 (L)
Alkaline Phosphatase	123 (H)
ALT	57 (H)
AST	70 (H)
Cytology (Paracentesis Fluid)	Inflammation, predominantly lymphohistiocytic. Negative for malignant cells
Cultures	No AFB, fungal or bacterial growth

S3012

Hepatic Epithelioid Hemangioendothelioma – A Rare Mimicker of Hepatic Metastasis

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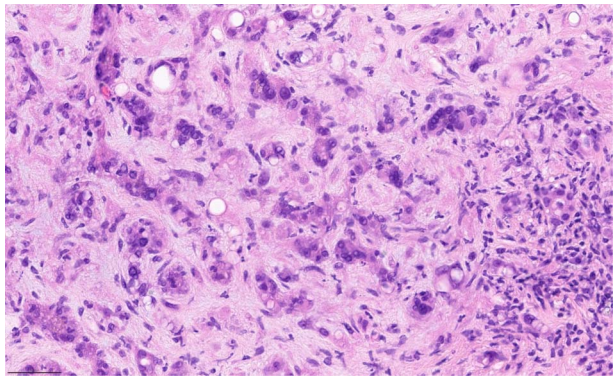
Introduction: Hepatic epithelioid hemangioendothelioma (EHE) is a malignant tumor of vascular origin with an estimated incidence of one in a million. The tumor derives its name from the characteristic composition of dendritic and endothelial cells with epithelioid morphology. Mostly asymptomatic, EHE is usually an incidental radiographic diagnosis that can mimic other liver tumors such as metastasis, hepatocellular carcinoma (HCC), angiosarcoma, or cholangiocarcinoma. We present a case of incidental diagnosis of hepatic EHE masquerading as multifocal liver metastasis.
Case Description/Methods: An 80-year-old man with coronary artery disease presented with 5 days of diarrhea and black stools. He was taking bismuth subsalicylate in an attempt to ease diarrhea. Labs showed mild anemia with hemoglobin (Hb) of 13.1 g/dL with normal platelets and INR. A CT abdomen with contrast showed mild hepatomegaly and multiple space-occupying lesions in the liver concerning for metastatic cancer. The patient thereby underwent an EGD and colonoscopy which was negative for a primary GI malignancy. Ultrasound-guided liver biopsy was then pursued. Immunohistochemical stains performed on the core biopsy showed that the cells were positive for CD31, CD34 and ERG and were negative for cytokeratin-20, CDX-2, TTF-1, arginase, and glypican-3. Focal staining was noted with cytokeratin AE1/AE3 and cytokeratin-7. Special stain for mucicarmine was negative. These findings were consistent with diagnosis of hepatic EHE. His diarrhea resolved and Hb remained stable. He was discharged with outpatient oncology follow-up. (Figure)
Discussion: Hepatic EHE is typically diagnosed incidentally. However, the course of EHE may range from indolent to aggressive disease with distant metastases. Suspicion for EHE may arise when a patient is discovered to have multiple liver lesions with no identifiable primary cancer. Liver biopsy is required for diagnosis as EHE has unique histologic, immunohistochemical, and molecular characteristics. Treatment options are typically chosen based on the extent of the disease. EHE confined within the liver has been treated with liver resection, liver transplantation, radiofrequency ablation, or simply a wait and watch method. Patients with extrahepatic involvement have been treated with systemic therapy including a variety of cytotoxic chemotherapy, immune therapy, or targeted therapy. As EHE remains exceptionally rare, prompt diagnosis should be followed by a multidisciplinary discussion for individualized care.



[3012] **Figure 1.** Hematoxylin and Eosin staining of liver biopsy in Hepatic EHE.

S3013

Home Brew Gone Wrong: A Case Report on Potential Auto-Brewery Syndrome Sequelae

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Introduction: Auto-Brewery Syndrome (ABS) is a condition not often seen in medical practice. It involves the conversion of carbohydrates to alcohol by the intestinal microbiome in a subset of patients. However, when these patients deny any consumption of alcohol, they are often brushed off or worked up for other etiologies, including stroke or hypoglycemia. There are also many possible medical sequelae that are not as widely reviewed due to the rarity of this disease. These include symptoms of chronic alcohol consumption such as increased alcohol cravings addiction, possible neuropathy, and liver disease such as fatty liver disease or cirrhosis.

Case Description/Methods: In this case, we present a 26-year-old male with a history of auto-brewery syndrome presenting with bilateral lower extremity paralysis, sensation loss, and neuropathy. These symptoms were similar to an episode which was inconclusive. He consumed alcohol occasionally but was sober at that time. During that stay, he was diagnosed with auto-brewery syndrome. In ED, his blood alcohol was 493mg/dL, although he was sober for 1 year, and his liver function tests were significantly elevated with AST twice the level of ALT, consistent with findings of alcoholic liver injury. There was some return of neurological and hepatic function over the hospital stay with supportive care. He was placed on a consistent carbohydrate diet and his LFTs subsequently improved.
Discussion: While there is data on potential causes and causative organisms for this condition, there is little in the literature on the potential sequelae associated with auto-brewery syndrome. Chronic alcoholism and its effects are widely seen and studied. From alcohol neuropathy to cirrhosis, the effects of consistent alcohol consumption are deleterious to one's health. The question that needs to be asked is: are patients with chronic exposure to endogenous alcohol at risk for the same complications seen in chronic alcoholism? Cirrhosis, which is seen as a risk factor for ABS, may actually be a consequence of ABS. In this patient, his AST and ALT were mildly elevated, even during previous admission. Therefore, the patient may have been experiencing chronic liver injury from alcohol use while sober, placing him at increased risk for NASH and eventually cirrhosis at a much earlier onset than expected. It would be imperative in this case to ensure that the patient is on a limited intake of carbohydrates, or antifungals if necessary.

S3014

Hepatotoxic Effects of Whey Protein Supplementation

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Introduction: Currently, more than 50% of Americans take dietary supplements. Whey is a commonly used protein supplement that is often viewed as safe, with only rare reports of drug-induced liver injury (DILI). Here, we report an unusual case of severe liver injury following whey protein consumption.
Case Description/Methods: A 40-year-old female with obesity and well-controlled hypothyroidism presented with 3 days of nausea and painless jaundice. Her only home medication was levothyroxine. Two months prior, she began drinking whey protein shakes (each containing 20 grams of protein) twice daily to aid with weight loss. She denied use of alcohol, recreational drugs, herbal supplements, and recent medication changes. On physical exam, she was alert and oriented x4, with jaundice and scleral icterus present. Lab work was significant for ALT 2,502, AST 1,434, total bilirubin 11.0, INR 1.42, and platelets 217,000. Acetaminophen and salicylate levels were unremarkable. Her whey protein supplementation was immediately stopped and a workup for transaminitis was initiated. RUQ ultrasound was unremarkable. Evaluation for other causes of acute liver injury, including viral hepatitis (A, B, C, E), EBV, CMV, HIV, hemochromatosis, Wilson's disease, and autoimmune hepatitis was unremarkable. A liver biopsy was performed, with pathology showing diffuse ballooning degeneration of hepatic parenchyma, numerous acidophil bodies, and dominant pericentral hepatocyte drop-out. A diagnosis of DILI (with predominant hepatocellular injury) was made. Following discontinuation of whey protein supplementation, the patient's labs and clinical symptoms improved, and she was discharged home on hospital day 8.
Discussion: This case highlights a rare occurrence of DILI secondary to whey protein consumption and emphasizes the importance of taking a thorough history when working up liver injury. Furthermore, in the treatment of DILI, we recommend cessation of all possible offending agents that were recently started, including supplements that are reputably marketed to be safe for consumption.

S3015

Hepatic Abscesses and Portal Vein Thrombosis Due to *Fusobacterium Nucleatum* Septicemia: Lemierre Syndrome Variant

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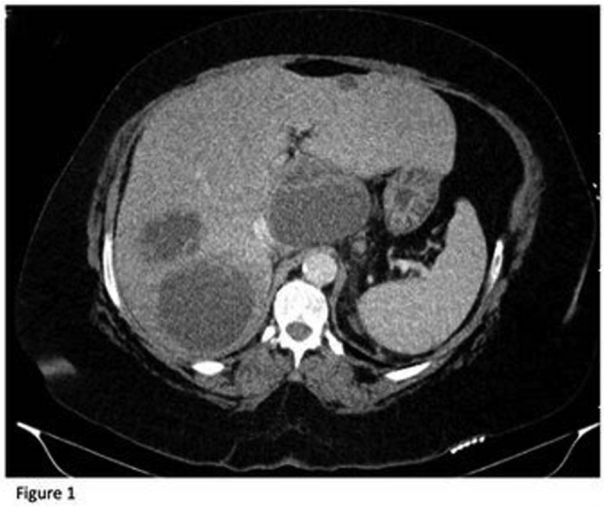
Introduction: *Fusobacterium* species are well known to cause Lemierre syndrome. However, few cases had linked *Fusobacterium nucleatum (F. nucleatum)* to gastrointestinal (GI) variant of Lemierre syndrome. We report a rare case of *Fusobacterium nucleatum* septicemia with septic pylephlebitis and multiple liver abscesses, most likely from mild gastrointestinal primary infection.
Case Description/Methods: A 62-year-old female with a medical history of hypertension, and heart failure with reduced ejection fraction presented as a transfer from an outside hospital for evaluation of liver lesions. Patient presented with generalized malaise, confusion, low-grade fever, and body aches. One week prior to presentation, she had left-sided cramping abdominal pain associated with decreased appetite. Labs on presentation were significant for leukocytosis, microcytic anemia with elevated ferritin, elevated alkaline phosphatase, and elevated creatinine. CT abdomen and pelvis with contrast (Figure 1) demonstrated numerous large hepatic lesions up to 9cm in size, subocclusive thrombus in the superior mesenteric and portal vein, several enlarged para-aortic lymph nodes, and chronic diverticulitis. Infectious work-up was significant for blood culture that grew fusobacterium nucleatum. Patient was started on enoxaparin and ampicillin-sulbactam. MRCP redemonstrated multiple predominantly cystic hepatic masses suspicious for abscess versus metastatic disease. CT chest and Brain MRI were unrevealing. Colonoscopy showed moderate, benign-appearing, 10cm stenosis in the distal sigmoid colon, and extensive diverticula. Tumor markers were negative. Echo was negative for vegetations. Interventional radiology performed aspiration of the left large and right hepatic cysts and placed 2 drains which yielded purulent fluid with cytology showing degenerative cells, neutrophils, and negative for malignancy. Patient was switched to ertapenem outpatient and apixaban. Repeat CT abdomen/pelvis (Figure 2) showed a decrease in size of the multiple hepatic abscesses and mild residual non-occlusive thrombus. Hepatic drains were removed, and patient was switched to oral Augmentin.
Discussion: Our patient had portal vein thrombosis, splenic vein thrombosis and hepatic abscesses presumably from primary GI infection likely mild diverticulitis. This picture may mimic metastatic liver cancer with portal vein thrombosis. Exclusion of malignancy and distinguishing clinical features should be identified early to initiate antimicrobial therapy and anticoagulation.



[3015] **Figure 1.** numerous large hepatic lesions up to 9 cm in size.

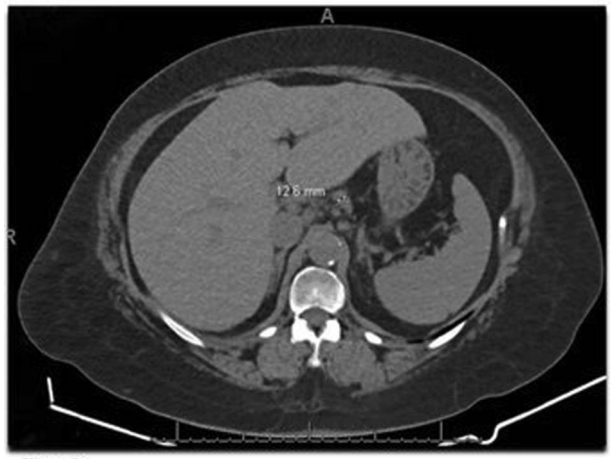


Figure 2

[3015] **Figure 2.** Significant decrease in size of multiple hepatic lesions.

S3016

Hepatocellular Carcinoma as an Uncommon Cause of PTHrP-Mediated Hypercalcemia

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Introduction: Humoral hypercalcemia of malignancy driven by PTHrP is a syndrome frequently associated with squamous cell carcinoma. We present a case of humoral hypercalcemia as a rare manifestation of hepatocellular carcinoma and describe the impact of early diagnosis on patient care.
Case Description/Methods: A 67-year-old male with HCC presented to the emergency department with subacute confusion, constipation, and lower extremity weakness. Physical exam revealed tangential thinking and no asterixis. Serum calcium was 14.8 (corrected to 15.3), creatinine 1.36 (baseline 0.8), AST 141, ALT 32, and AFP 135 (all previously normal). Imaging revealed new liver lesions in multiple segments, enlarged lymph nodes in the porta hepatis, widespread lytic lesions in the spine, calvarium, and facial bones with a 3.4 cm x 2.4 cm left orbital wall mass consistent with metastatic disease. Serum calcium levels normalized with IV fluids, calcitonin, and zolendronic acid. The PTHrP was elevated at 79. Vitamin D levels, SPEP and UPEP were normal. He returned one week later with recurrent severe hypercalcemia. No further cancer treatment options were available so the patient elected for hospice services and died at home.
Discussion: Malignancy is the most common cause of hypercalcemia in the inpatient setting. Malignancy-associated hypercalcemia occurs through three distinct mechanisms – humoral (PTHrP mediated), lytic bone lesions, and increased absorption due to excess 1,25 vitamin D production by the cancer. Most cases of HCC-associated hypercalcemia are due to metastatic lytic bone lesions. Less than 10% of cases are attributable to PTHrP, which is severe and often refractory. The mechanism for this is unknown but may be related to an underlying paraneoplastic syndrome and downstream metabolic derangements. Humoral hypercalcemia in patients with HCC is associated with a more advanced TNM stage and higher tumor burden. In a study of 534 patients with HCC, 6.3% had humoral hypercalcemia and had worse prognoses by Child-Pugh scores. Another study of 165 patients with HCC and PTHrP-mediated hypercalcemia found a median survival time of 15 days. Early recognition of humoral hypercalcemia in patients with HCC is essential given its association with higher mortality. Doing so can help providers expedite locoregional therapy or guide goals of care discussions with their patients.

S3017

HIV-Associated Iron Overload: A Rare Cause of Elevated Transaminases in Patients With HIV

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Introduction: Hereditary hemochromatosis can lead to elevated transaminases due to iron deposition in liver. In this case, we discuss a patient with advanced HIV, who in the absence of HFE gene mutation or history of blood transfusions, had drastically elevated ferritin levels and evidence of hemosiderosis in the liver.
Case Description/Methods: A 44-year-old man with a medical history notable for HIV (diagnosed 15 years ago, not on antiretroviral therapy, CD4 count 0, viral load 799000) presented with a transient ischemic attack which resolved after tPA. He was incidentally found to have elevated transaminases and admitted for workup. Patient denied alcohol use for the past 4 years. Physical exam was unremarkable with BMI 17. Labs were notable for ALT 150 u/L, AST 320 u/L, ALP 431 u/L, GGT 1056 u/L, and total serum bilirubin 0.4 mg/dL. His liver enzymes were within normal limits until 3 months prior to presentation, when he was incidentally found to have ALT 165 u/L, AST 100 u/L, ALP 156 u/L, and total serum bilirubin 0.3 mg/dL. Of note, at that time, the patient had received a course of amoxicillin-clavulanate for pneumonia. Hepatitis panel was negative for active infection. DILI was suspected due to history of receiving amoxicillin-clavulonate; however, his liver enzymes were still uptrending 3 months after receiving the medication which is uncommon with DILI. AIDS cholangiopathy, a chronic development of biliary strictures caused by infection, was also suspected. MRCP showed no abnormality of biliary system but revealed iron deposition in the liver and spleen consistent with hemosiderosis. Iron studies were then performed showing ferritin 8260 ug/L, serum iron 157 ug/dL, TIBC 272 ug/dL, transferrin saturation 58%. HFE allele genetic testing was negative for mutation, and thus hereditary hemochromatosis was ruled out. Patient was sent home with outpatient hepatology follow-up.
Discussion: HIV commonly targets cells that modulate iron metabolism such as macrophages, and consequently excessive iron accumulates in various organs including liver causing tissue damage, and thus the use of iron chelators has been proposed [1][2]. Our case illustrates the necessity of suspecting HIV-related iron overload in patients with HIV with elevated transaminases of unclear etiology as well as reiterates the potential implications of iron chelation therapy in such cases.

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S3018

Hypocalcemia-Induced Right Heart Failure: A Rare Cause of Acute Liver Failure

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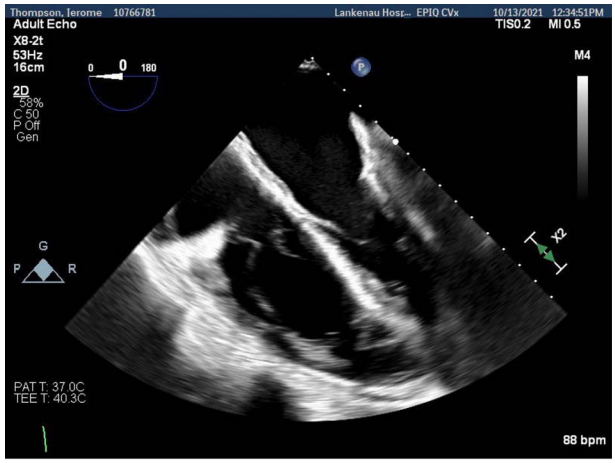
Introduction: Cardiogenic shock is an uncommon cause of acute liver failure (ALF). Hypocalcemia is a rare, but reversible cause of acute cardiomyopathy. We present the case of a patient with severe hypocalcemia after parathyroidectomy causing right ventricular (RV) failure and ALF.
Case Description/Methods: A 64-year-old male with a history of primary hyperparathyroidism status post parathyroidectomy 2 months prior presented with hypoxia and confusion. On exam he was obtunded with abdominal distension and peripheral edema. Labs showed calcium 4.8 mg/dL, ionized calcium 0.63 mmol/L, INR 5.1, AST 2478 IU/L, ALT 1999 IU/L, alkaline phosphatase 304 IU/L, total bilirubin 1.6. CT

abdomen noted nodular liver and cardiomegaly. He was intubated and started on IV calcium gluconate and N-acetylcysteine. Urine drug screen, acetaminophen level, salicylate level, and viral and autoimmune liver workup all unrevealing. A transthoracic echocardiogram (TTE) showed severely decreased RV systolic function with normal LV function. Serum aminotransferases and INR normalized prior to discharge. (Figure)

Discussion: In evaluating ALF, it is important to rule out unusual causes of liver injury. In addition to drug-induced and viral etiologies, other causes include ischemic injury, neoplastic infiltration, metabolic diseases, and autoimmune diseases. Treatment consists of early recognition, identifying and addressing complications, supportive care, and transplant if appropriate. In the case of our patient, the workup for the typical causes of ALF was unrevealing. He had no known underlying liver disease, suggesting an acute process. Hypocalcemia is a rare, but known reversible cause of acute dilated cardiomyopathy. As calcium plays a vital role in myocyte contractility, low levels can lead to decreased myocardial performance, cardiogenic shock, and end-organ damage including congestion and ischemia of the liver. The cases of hypocalcemic cardiomyopathy that have been reported affected the left ventricle (LV). This is likely because the oxygen requirement of the RV is lower than the LV, thus the RV is less susceptible to ischemic insults. It is important to keep a broad differential in evaluating causes of acute liver failure, including severe electrolyte disturbances causing global ischemia. Our patient's hepatic and cardiac function quickly improved with intravenous calcium supplementation.



[3018] **Figure 1.** Severely dilated and akinetic right heart, severely decreased RV function.

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S3019

Hepatic Infarction Associated Antiphospholipid Syndrome and HELLP in Pregnancy

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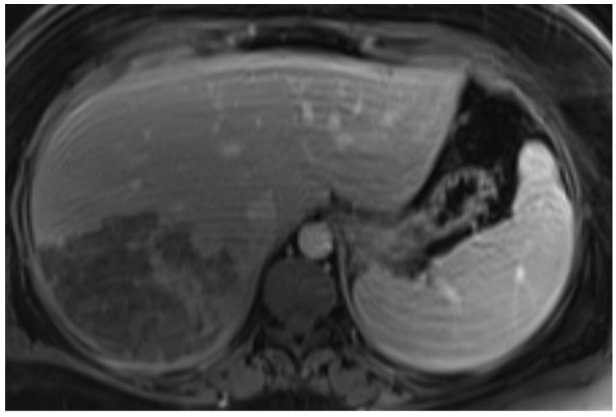
Introduction: Pregnant patients with antiphospholipid syndrome (APLS) are at risk for thromboembolic complications. They are more likely to present with hemolysis, elevated liver enzymes, low platelets (HELLP) syndrome. Hepatic infarction is a rare complication which can lead to hepatic rupture, fulminant liver failure and death. We present a case of a pregnant patient with known history of APS who presented with HELLP and a large liver infarction despite treatment with anticoagulation.

Case Description/Methods: A 36-year-old pregnant female with history of pulmonary embolism, prothrombin gene mutation and APLS, presented to an outside hospital with right upper quadrant pain. Labs showed AST 255, ALT 274 and platelets 116. Labs also showed evidence of hemolytic anemia. Her medications included enoxaparin and aspirin. Due to concern for HELLP syndrome she underwent an emergent cesarean section. Post-delivery her labs worsened with AST 2712, ALT 2783, and platelets 43. Abdominal CT showed a large ill-defined hypodensity within segment 3 of the liver concerning for hepatic infarcts. She was treated with plasmapheresis and methylprednisolone. Given concern for impending acute liver failure she was transferred to a liver transplant center. After transfer, MRI abdomen confirmed a large infarction of the liver which involved the entirety of segments 6 and 7, as well as adjacent portions of segments 5 and 8 (Figure 1). The hepatic and portal veins appeared normal. She was observed in the ICU and over several days her abdominal pain and labs improved, with repeat showing AST 577, ALT 975 and platelets 51. Additional workup including viral hepatitis panels were normal. She was continued on enoxaparin and transitioned to warfarin for long term anticoagulation. Her hepatic panel 1 month later showed AST 39, ALT 29.

Discussion: Hepatic infarction is a rare complication of APS due to the dual blood supply of the liver. 93% of reported cases of hepatic infarction in pregnant women with APS were associated with HELLP syndrome. Even when patients are on anticoagulation it is important to consider hepatic infarction as a complication in patients with APS and HELLP who presents with abdominal pain, worsening lab values and hepatic failure. Multidisciplinary care with maternal-fetal medicine, hematology, and hepatology, and transfer to a transplant center should be considered given the high morbidity associated with this condition.



[3019] **Figure 1.** Venous phase MRI with contrast showing large infarct in the liver.

S3020

Hepatitis From a Sexually Transmitted Bacterial Infection

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Introduction: Syphilis is a multisystemic, sexually transmitted infection (STI) that is caused by the spirochete *Treponema pallidum*. Hepatic involvement is rare. We present a patient who was diagnosed with syphilitic hepatitis (SH).
Case Description/Methods: A 54-year-old male presented to the hospital with one week of sharp upper abdominal pain, jaundice, poor appetite, and a 25-lb. weight loss. He also reported multiple sexual partners and a rash that developed 2 months ago. Vital signs were within normal range. Physical examination revealed scleral icterus, a soft, non-tender abdomen, a plaque-like penile lesion and small, circular lesions on his palms and soles of feet. Laboratory studies revealed aspartate aminotransferase (AST) 164 U/L, alanine aminotransferase (ALT) 207 U/L, alkaline phosphatase (ALP) 1395 U/L, total bilirubin 5.6 mg/dL, gamma-glutamyl transferase 782 U/L. Hepatitis panel, acetaminophen and alcohol levels were negative. Chronic liver disease work-up was unremarkable. Treponemal antibody and rapid plasma reagin test were both positive. HIV test and cerebrospinal fluid venereal disease research laboratory (VDRL) test were negative. Abdominal ultrasound and CT scan of the abdomen were unremarkable. Magnetic resonance cholangiopancreatography was notable for hepatomegaly and hepatitis. Patient received 1 dose of penicillin G 2.4 million units intramuscularly with remarkable improvement in his symptoms and liver function tests. He was discharged home with close follow-up with gastroenterology and infectious disease.
Discussion: SH occurs in 0.2-9.7% of patients with syphilis and is most commonly seen in the primary and secondary disease stages. Clinical manifestations include a maculopapular rash (involving the trunk, palms and/or soles of feet), fatigue, poor appetite, hepatomegaly and icterus. Liver function tests (LFTs) may reveal a marked increase in ALP and GGT in comparison to ALT and AST. SH is diagnosed with abnormal LFTs, serological evidence of syphilis, exclusion of other liver diseases and LFTs returning to normal after antibiotic therapy as in our patient. The histological features of SH include inflammatory infiltration of the bile duct and hepatic granulomas. Spirochetes are often difficult to identify in liver tissue. Penicillin is the mainstay of treatment. Clinicians should consider syphilitic hepatitis in a patient with abnormal LFTs (especially ALP) and a rash involving the palms and soles of feet in the appropriate clinical context.

S3021

Hepatotoxicity From Marijuana Gummies

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Introduction: We report a case of drug-induced liver injury (DILI) induced by cannabis gummies containing Corydalis Rhizome.
Case Description/Methods: A 37-year-old female presented to her primary care clinic with recurrent fevers, night sweats, and myalgias for 7 weeks accompanied by eye redness, brain fog, headache, nausea, and abdominal pain. She denied rashes, tick-bites, cough, dyspnea, chest pain, joint swelling, or genitourinary symptoms. Past medical history was notable for IBS, migraines, and anxiety. She reported edible marijuana use four times a week, rare alcohol use, and denied tobacco use. She denied a family history of liver disease. Physical exam was notable for tachycardia to 110 and scleral injection with the remainder of vitals and exam unremarkable. Initial labs were notable for AST 61, ALT 44 and CRP of 12. CBC, BMP, urinalysis, ESR, blood cultures, blood smear for parasite screen, tests for Lyme disease, Babesia, Tularemia, Anaplasma, Ehrlichia, Rickettsia, EBV, HIV, RPR, ANA, CMV, parvovirus B19, and chest x-ray were all negative. The patient was referred to infectious disease with further testing for West Nile, Leptospira, lymphocytic choriomeningitis virus, and COVID-19 returning negative. Repeat LFTs showed worsening transaminitis with ALT 979 and AST 712, alkaline phosphatase 88, total bilirubin 0.7, and albumin 4.9. Hepatitis workup including hepatitis A, B, and C, HSV, EBV, VZV serologies, AMA, ASMA, antiLKM Ab, acetaminophen level, INR, iron panel, CPK, TSH, and abdominal ultrasound were all normal. It was later discovered that her marijuana gummies contained Corydalis rhizome extract known to be hepatotoxic. Cessation of this drug was strongly advised. She was discharged with hepatology follow-up and underwent a liver biopsy showing patchy periportal and lobular inflammation with extension across the limiting plate, hepatocyte injury and apoptosis, and increased lipofuscin for age compatible with mild to moderate hepatitis. She had complete recovery after cessation of Corydalis-containing gummies. (Figure)
Discussion: Our patient consumed '1906 Midnight', an American cannabis brand containing Corydalis rhizopus 100 mg, advertised to improve sleep, pain, and have a liver protective effect. A Korean systematic review on herbal-induced liver injury reported that Corydalis was the 3rd most frequent causative herb, with 36 cases. Although there are several personal accounts on social networking sites and other websites, there are no American-based publications reported on DILI from Corydalis.

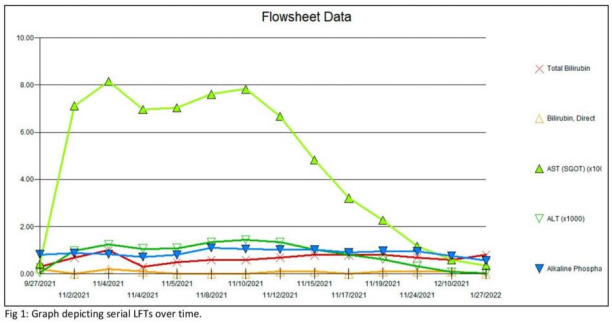

Fig 1: Graph depicting serial LFTs over time.

[3021] **Figure 1.** Patient's serial LFTs depicted over time.

S3022

Hepatic Cerebrospinal Fluid Pseudocyst: A Rare Complication of Ventriculoperitoneal Shunt

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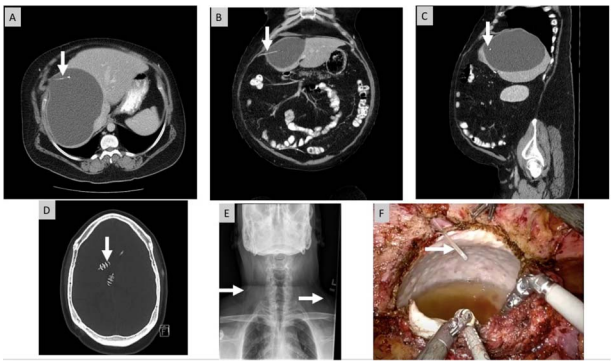
Introduction: Ventriculoperitoneal shunts (VPS) are commonly used in the management of hydrocephalus to drain cerebrospinal fluid (CSF) into the peritoneal cavity. The placement of VPS has prolonged the survival of patients with hydrocephalus. Abdominal pseudocysts are long-term complication of VPS that are identified later in life. Hepatic CSF pseudocyst is a rare long-term complication of VPS that should be differentiated from other cystic lesions of liver.
Case Description/Methods: A 49-year-old man with intellectual disability from congenital hydrocephalus s/p placement of right and left-sided VPS at age of 3 months and 7 years respectively presented with exertional dyspnea and abdominal distension. On presentation his vitals were unremarkable except tachycardic (115/min)., He had abdominal distention, hepatomegaly but no tenderness. Initial labs showed D-dimer 2.20 mcg/mL, AST 27 u/L, ALT 38 u/L, alkaline phosphate 126 u/L and total bilirubin 0.5 mg/dL. Chest CT with IV contrast was negative for pulmonary embolism, however revealed a large 18x13x13.5 cm cyst in right hepatic lobe. CT abdomen and pelvis demonstrated a 17.5x12.6x12.7 cm cystic lesion in the right hepatic lobe with the tip of VP shunt catheter within cyst cavity. Hepatobiliary nuclear scan was unremarkable for any biliary leak or sphincter of oddi dysfunction. CT head was negative for any acute abnormalities. Shunt series x-rays were negative for disruption of VPS catheter. Robotic laparoscopic cyst fenestration with partial hepatectomy was performed and catheter was repositioned to the right lower quadrant of abdomen. Patient was discharged home two days later with significant reduction of cyst size on follow up imaging. (Figure)
Discussion: This case illustrates a rare complication of VPS that result in hepatic CSF pseudocyst. If hydrocephalus is absent, patients with hepatic CSF pseudocyst are asymptomatic at earlier stages., or they may present with abdominal pain, distention, or palpable right upper quadrant abdominal mass. A subset of patients with hepatic CSF pseudocyst are complicated with bacterial or parasitic infection and present with abdominal pain, distention, or right upper quadrant mass. Abdominal ultrasound and CT scan assist in diagnosis by identifying the tip of VPS catheter in the pseudocyst cavity. Asymptomatic patients are managed conservatively while surgical repositioning of VPS catheter with or without cyst fenestration or surgical excision of cyst may be required for complete resolution of symptoms.



[3022] **Figure 1.** CT of abdomen showing a large right hepatic lobe cyst with evidence of tip of right VPS catheter within the cavity of the cyst (arrows) on transverse (A), axial (B) and lateral (C) views. CT scan of the head (D) showing VPS catheters both in right and left lateral ventricles without evidence of ventricular dilation. Shunt series x-ray (E) shows now distortion of VPS catheter. Robotic laparoscopic cyst fenestration (F) shows VPS catheter within cyst cavity containing CSF.

S3023

Hepatotoxicity From Anabolic Steroids: A Case of Ruptured Liver

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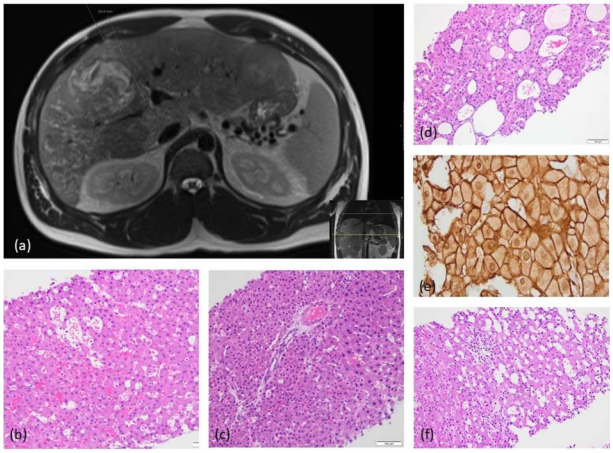
Introduction: Anabolic steroid use can lead to a spectrum of hepatic injuries from abnormal liver function panel to neoplasia. Here we report a middle-aged man who developed a wide range of hepatic manifestations attributable to steroids.
Case Description/Methods: A 30-year-old healthy male presented with six days of fever, right upper quadrant abdominal pain and distension. Social history included 3 years of intravenous anabolic steroid use. On physical exam, he had non-tender hepatomegaly. Labs were remarkable for ALT of 541 U/L, AST of 77 U/L, elevated inflammatory markers, and alpha-fetoprotein < 4. Infectious workup was negative. MRI liver showed enlarged liver (27 cm) with cavernous cystic changes, multiple T2 hyperintense hemorrhagic lesions in the right lobe, the largest measuring 6.8x6.5x7.7 cm, and multiple other hypodense lesions. Random liver biopsy showed areas of sinusoidal dilation, peliosis hepatis, pseudoglandular areas, pseudo portal tracts, and nodular regenerative hyperplasia. Biopsies from hemorrhagic and solid liver masses revealed well-differentiated hepatocellular adenoma which was beta-catenin activated. Malignant transformation could not be excluded in the biopsied liver masses hence surgical resection was recommended. The patient abstained from steroid use however, delayed the surgery due to social reasons. He presented to the hospital four months later with worsening abdominal pain and distension. CT abdomen revealed subcapsular hematoma and hemoperitoneum. He underwent exploratory laparotomy which showed a ruptured hepatic capsule, and a friable right lobe of the liver which had dissected from its ligamentous attachments. He underwent right hepatectomy and resection of adenomas in a staged manner. (Figure)
Discussion: This case illustrates that anabolic steroids can lead to a spectrum of histological diagnoses in the same patient. Multiple biopsies of the liver with samples inclusive of masses as well as random tissue are crucial. Large adenomas (>5 cm) have a high risk of malignant transformation and rupture. Pathology after resection is the only way to definitively diagnose or exclude hepatocellular carcinoma (HCC) harboring within adenomatous tissue. Compared to non-users, HCC from steroid users often carries a better prognosis, as it presents earlier in life in the absence of cirrhosis. However, HCC if diagnosed after hepatic rupture, the tumor will be staged metastatic. Hence surgery should be considered in a time-sensitive manner for large adenomas secondary to anabolic steroid use.



[3023] **Figure 1.** (a) MRI Liver - cystic changes and adenoma (b) Peliosis hepatis (c) Pseudoportal tract (d) Pseudoglandular area (e) Abnormal Beta-catenin stain in Hepatic Adenoma (f) Sinusoidal dilation.

S3024

High, High Ammonia, How High Can We Go? A Case of Encephalopathy in the Setting of Hemodialysis

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Introduction: The incidence of Urea Cycle Disorders (UCDs) is extremely rare with very few cases being reported. estimated to be less than 0.00001% of live births. Ornithine transcarbamylase (OTC) deficiency is the most common of the UCD, with an incidence estimated to be 1 in every 60,000 births. Classical presentation is at birth with a newborn male, resulting in coma and demise. Here, we describe a mysterious case of rising ammonia levels in a 66-year-old female.
Case Description/Methods: A 66-year-old woman with a past medical history of developmental delay and recent ischemic stroke presented to the hospital from her extended care facility for decreased responsiveness. Initial work-up revealed an ammonia of 252 umol/L, for which rifaximin 550mg twice daily and lactulose 30mg four times daily were initiated. Hospitalization was complicated by acute hypoxic respiratory failure, resulting in intubation. Despite rifaximin and lactulose, the ammonia level increased to 280umol/L and continuous renal replacement therapy (CRRT) was initiated with twice daily lactulose

enemas. Despite these interventions, ammonia continued to rise, peaking at 785umol/L on the tenth day of admission despite CRRT. During hospitalization, the patient experienced multiple seizures. Electroencephalogram (EEG) revealed seizures emanating from the left anterior mid temporal head region. Liver workup consisted of right upper quadrant (RUQ) ultrasound, biopsy, drug toxin level and hepatitis workup. Biopsy demonstrated stage III fibrosis. Negative hepatitis panel and normal salicylates and acetaminophen levels. UCD workup included urine amino acids analysis and genetic sequencing. Urine showed severely elevated orotic and aconitic acid levels. Furthermore, genetic sequencing revealed heterozygous gene for c.944T >G (p.Val351Gly), leading to the diagnosis of Ornithine transcarbamylase (OTC) deficiency. Unfortunately, the patient passed eighteen days after admission.
Discussion: OTC is a X-linked recessive disorder with a survival rate to the age of 12 is approximately 20%. Accumulating ammonia levels, results in encephalopathy, coma and eventual death. Treatment consists of low protein diet, and the use of nitrogen scavenging agents. The use of lactulose and rifaximin are popular and effective. Clinicians should be aware of atypical presentations of hyperammonemia in the setting of ultrafiltration given the late onset and poor outcomes.

S3025

Hepatic Sarcoidosis Presenting as Decompensated Liver Cirrhosis

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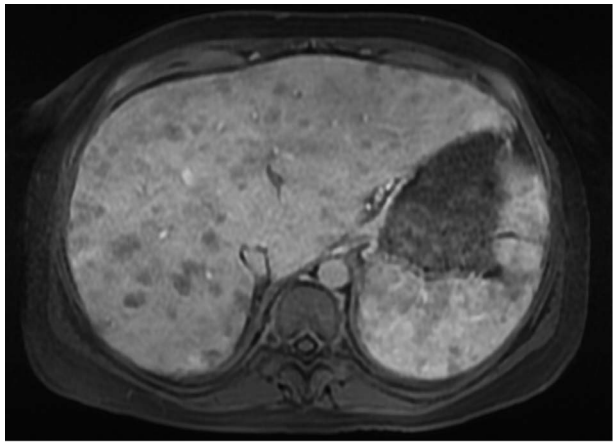
Introduction: Sarcoidosis is a disorder characterized by the formation of non-caseating granulomas in multiple organ systems. While pulmonary manifestation is the most common presentation, hepatic involvement can be seen in many patients. Most patients with liver involvement are asymptomatic; however, some can present with symptoms of liver injury. The presentation of hepatic sarcoidosis can resemble many other disease processes. Since definite diagnostic criteria have not yet been formalized for hepatic sarcoidosis, it can often be challenging for providers to make a prompt diagnosis.
Case Description/Methods: We present the case of a 36-year-old female who was evaluated for generalized abdominal pain and left flank pain. On presentation, labs were notable for calcium at 13 mg/dl, ionized calcium at 1.62 mmol/l, PTH at 8 pg/ml, 1,25-dihydoxyvitamin D at 96.4 pg/ml, alkaline phosphatase at 192 IU/L, AST at 51 IU/L and ALT at 47 IU/L. Eight months prior, the patient was admitted to an outside hospital in the setting of hematemesis. EGD with variceal banding was performed, and she was diagnosed with cryptogenic liver cirrhosis. CT A/P demonstrated marked heterogeneity of the liver. MRI of the abdomen showed mild hepatosplenomegaly and "diffuse small areas of altered signal intensity throughout the liver suspicious for diffuse hepatic metastatic disease" (Figure A). The patient just had her first visit with an oncologist prior to her presentation to our hospital. Her physical exam was notable for hepatomegaly, abdominal distension and RUQ tenderness. Her hypercalcemia was treated with fluids. Infectious and autoimmune workup came back negative. Different tumor markers including CA 19-9, CA 27-29 and CA 125 were normal. Liver biopsy showed hepatic parenchyma with numerous non-necrotizing granulomas and associated fibrosis. IL-2R receptor-serum came back elevated at 2916.1 pg/ml. The patient was then started on 20 mg prednisone daily for treatment of hepatic sarcoidosis.
Discussion: Hepatic sarcoidosis can have a wide spectrum of clinical presentation, ranging from incidental finding to end-stage liver disease. When a patient is diagnosed with a new hepatic disease, keeping hepatic sarcoidosis on the differential is very important. In case of hepatic sarcoidosis, assessing symptoms of liver involvement and biochemical evidence of cholestasis is crucial. While observation is indicated for asymptomatic liver disease, corticosteroids and/or ursodeoxycholic acid are the first line agents for symptomatic disease.



[3025] **Figure 1.** Hypointense liver lesions on MRI, initially presumed suspicious for diffuse hepatic metastatic disease, later confirmed to be sarcoid lesions.

S3026

Hepatic Sarcoidosis Hiding Beneath Mesenteric Panniculitis

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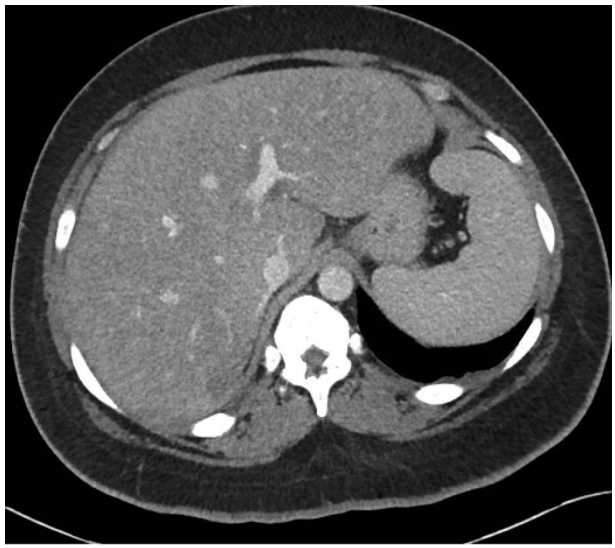
Introduction: Mesenteric panniculitis is a rare inflammatory disease that affects the adipose tissue of intestinal mesentery. The incidence of mesenteric panniculitis ranges from 0.16%-3.4%. Though its etiology is unknown, it has been linked to a range of conditions including vasculitides, malignancies, abdominal trauma, and autoimmune diseases. We present a case of a 32-year-old male with a history of chronic abdominal pain and unintentional weight loss who presented with worsening transaminitis eventually found to have mesenteric panniculitis and hepatic sarcoidosis.
Case Description/Methods: A 32-year-old male with a history of sarcoidosis and pulmonary embolism, not compliant with medication, presented to the emergency department with a six-month history of sharp, intermittent, and diffuse abdominal pain. He denied any fevers, nausea, vomiting, diarrhea, changes in medications or diet, recent travel, or sick contacts. He also endorsed a 20-pound weight loss. On presentation, he was tachycardic to 121 and febrile to 100F. Labs were significant for a hemoglobin of 8.2, AST 105 U/L, ALT: 81U/L, and ALP 51 U/L. On physical exam, the abdomen was diffusely tender to palpation in the epigastric region and right upper quadrant. He was started on IV Ceftriaxone. Computed Tomography (CT) of the abdomen and pelvis revealed a hazy appearance of mesentery with numerous mesenteric lymph nodes suspicious of mesenteric panniculitis, hepatomegaly, multistational upper abdominal lymphadenopathy, and a 2.0 cm ill-defined hepatic lesion (Figure 1). Percutaneous liver biopsy revealed hepatic sarcoidosis and mesenteric panniculitis. He was initiated on a two-week tapered course of prednisone. Azathioprine was later added with marked improvement in his symptoms.
Discussion: Mesenteric panniculitis is a rare fibroinflammatory condition of unknown etiology. There is limited understanding of the pathogenic mechanism, but it has been linked to autoimmune processes. Presenting symptoms are vague and highly variable, but abdominal pain is reported to be the most common. CT scan is the imaging modality preferred, however, diagnosis is established by histological confirmation. No specific treatment exists, but steroids have demonstrated promising results. Our case illustrates the need for maintaining a high index of suspicion for mesenteric panniculitis when evaluating patients with a history of autoimmune disease presenting with vague abdominal pain in addition to transaminitis.



[3026] **Figure 1.** CT scan (axial view) showing focal lesion in the liver and mesenteric lymphadenopathy.

S3027

Hepatocellular Carcinoma Presenting as a Clival Mass

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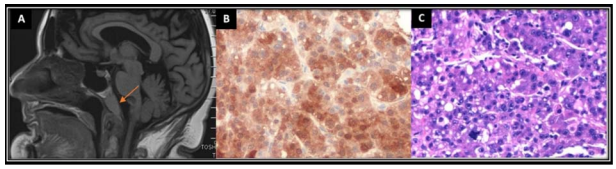
Introduction: Hepatocellular carcinoma (HCC) comprises the majority of primary liver cancer and has a poor prognosis. Clivus metastasis is rare with only a few reported cases in the medical literature. We report a case of a patient who presented with clival mass found to have metastatic HCC.
Case Description/Methods: A 63-year-old woman presented for neurosurgical evaluation after she was found to have a skull base mass on computerized tomography (CT) of the head at an outside hospital. She endorsed dysphagia for three months, however denied headaches or visual disturbances. A magnetic resonance imaging (MRI) revealed a 5.4 cm by 2.9 cm by 3.6 cm mass in the clivus, which was deemed as the cause of dysphagia (Figure 1a). The patient subsequently underwent an endoscopic transsphenoidal resection of the clival mass. Histopathology from the tissue revealed a hepatoid carcinoma, concerning for metastatic HCC (Figure 1b and 2c). Immunohistochemical strains were positive for hepatocytic marker arginase-1 (Figure 1d). Laboratory studies revealed alpha fetoprotein (AFP) of 56,344 ng/mL, CA-125 of 376 ng/mL, normal B-HCG and carcinoembryonic antigen (CEA). Thereafter, a triple phase CT of the liver revealed two LI-RADS 5 lesions suggestive of HCC as the primary malignancy. Patient's case was discussed at multidisciplinary tumor board with recommendations for systemic immunotherapy with atezolimumab plus bevacizumab and radiation therapy to the clivus.
Discussion: The incidence of HCC has almost tripled since the 1980s making it the fastest rising cause of cancer related deaths. Metastasis to the brain comprises 0.26% to 2.2% of cases and the skull base is the most rarely affected anatomical site. Although CNS presentation is rare, we may see more neurological manifestations of metastatic HCC with the persistence of chronic hepatitis infections, the rise of metabolic diseases such as NASH, and an increase in alcohol-related liver disease during the COVID-19 pandemic. Although exceedingly rare, metastasis to the clivus should be considered in the differential diagnosis of skull base masses. Despite detection and treatment, prognosis remains poor and emphasis should be placed on consistent HCC surveillance. This case emphasizes that skull masses must be evaluated diligently as they can be the first sign of underlying liver malignancy. Given the morbidity and mortality associated with HCC, recognition of atypical manifestations of HCC can lead to a prompt diagnosis and initiation of life-saving treatment.



[3027] **Figure 1.** A. Sagittal view of the MRI brain revealing clivus mass (red arrow). B. H&E stain at high power showing proliferation of malignant epithelial cells with hepatoid morphology arranged in sheets of nests and trabeculae. D. Positive immunohistochemical stain for hepatocytic marker arginase-1.

S3028

Identifying and Screening At-Risk Patients for Hepatitis Delta Virus

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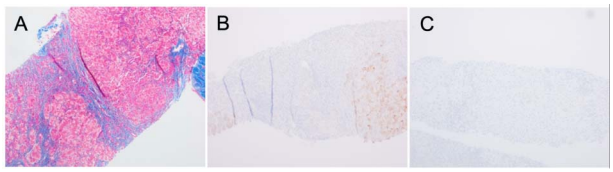
Introduction: Hepatitis delta virus (HDV) is associated with the most severe forms of viral hepatitis with rapid progression to cirrhosis and hepatocellular carcinoma. The prevalence of HDV may be significantly higher than formerly acknowledged in the United States, making it doubly important to identify at-risk individuals.
Case Description/Methods: A recently emigrated 29-year-old Afghani male with chronic hepatitis B virus (HBV) infection presented with abdominal pain, nausea and vomiting to an outside facility. He underwent a laparoscopic cholecystectomy for symptomatic cholelithiasis. He developed postoperative abdominal distension with imaging initially concerning for a large bile leak however ERCP and a diagnostic laparoscopy were unrevealing. Four liters of fluid were drained, and a surgical drain was placed which continued to drain up to two liters of fluid daily. Upon transfer to our hospital, diagnostic workup showed AST 583 U/L, ALT 452 U/L, alkaline phosphatase 88 U/L, total bilirubin 1.8 mg/dL, and albumin 2.8 g/dL. HBV DNA PCR was < 10 IU/mL. HBV core antibodies, HBV e antibody, and HBV surface antigen were reactive. HBV e antigen and HBV surface antibody were non-reactive. Ascites fluid studies showed a high serum ascites albumin gradient and low protein, prompting a liver biopsy that showed cirrhosis (image 1). The patient was ultimately found to be positive for HDV antibody, confirming a diagnosis of cirrhosis due to HDV superinfection. The patient had his drains removed and was discharged on oral diuretics and entecavir for treatment of HBV infection. Diuretic uptitration is ongoing with plans to enroll in a trial in the future pending clinical status.
Discussion: In patients with chronic HBV who acquire HDV as a superinfection, 70-80% develop cirrhosis or hepatocellular carcinoma within 5-10 years. Superinfection is the likely cause of cirrhosis in our patient, as cirrhosis in a 29-year-old would not be expected with HBV infection alone. Early recognition of at-risk individuals can aid in faster diagnosis and earlier treatment. Screening should be considered in all individuals with a reactive HBV surface antigen, and especially in migrants from endemic areas, hemodialysis patients, healthcare employees, and IV drug users. Total HDV antibodies should be obtained for screening, with diagnosis confirmation by serum RT-PCR. Currently, therapeutic options are only available for compensated patients, with pegylated interferon alpha approved in the United States and buleviritide in Europe.



[3028] **Figure 1.** (A) Trichrome stain showing markedly active chronic hepatitis consistent with cirrhosis. (B) Immunohistochemical stains demonstrate patchy staining with HBV surface antigen but (C) negative for HBV core antigen.

S3029

Hydralazine-Induced Liver Injury With Autoimmune Features: A Case Report and Literature Review

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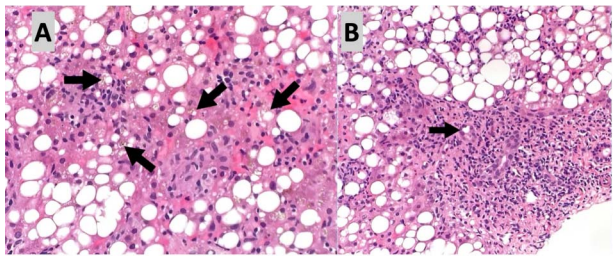
Introduction: Autoimmune drug induced liver injury (AI-DILI) is a syndrome characterized by liver injury accompanied by laboratory evidence of autoimmunity due to the ingestion of a drug or herbal product. DILI accounts for approximately 15% of the cases of acute liver failure. Many drugs have been associated with DILI such as Amoxicillin-clavulanate, Diclofenac, Azathioprine, Infliximab. However, very rare cases have been reported on hydralazine-induced DILI. A response to treatment with steroids supports the diagnosis.
Case Description/Methods: We present a case of a 59 year old female with no reported past history of liver disease. Patient was admitted for workup of jaundice, fatigue, and abdominal dissension of one month duration. Labs were significant for liver enzymes elevation with a cholestatic pattern. ERCP and EUS were done and results came back unremarkable. An extensive infectious and autoimmune workup was also negative. On further clarification of medication history, patient appeared to have had started hydralazine roughly 3 months prior to presentation. A liver biopsy was done and showed cholestatic injury and granulomatous inflammation (Figures A&B). Hydralazine was presumed the cause of patient's liver injury, it was immediately stopped and patient was started on oral prednisone with significant improvement in symptoms and liver function test.
Discussion: Autoimmune features and autoantibody reactivity in such cases of DILI vary according to liver injury severity and disease progression. The presentation of AI-DILI is classically indistinguishable from idiopathic autoimmune hepatitis (I-AIH). The presence of autoimmune features of liver injury due to hydralazine is not associated with the typical HLA alleles found in I-AIH such as HLA-DRB1*03:01 and DRB1*04:01 alleles. Histologically, AI-DILI shows changes that cannot be definitively distinguished from I-AIH without clinical correlation. Features on liver biopsy favoring DILI with autoimmunity include portal inflammation, fibrosis, portal neutrophils and plasma cells, and intracellular cholestasis. Liver biopsy is not performed in many cases of DILI, but may be useful if the liver injury is prolonged and does not promptly resolve with discontinuation of the drug and to also decide on need for treatment with steroids. In the attached table we present a review of the body of research on hydralazine-induced DILI with autoimmune features.



[3029] **Figure 1.** A- H&E stained section of the liver demonstrates moderate cholestasis (arrows) with surrounding inflammation. B- H&E stained section of the liver demonstrating a portal triad with mixed inflammation composed of predominately lymphocytes with rare neutrophils and plasma cells and mild interface activity. Moderate bile duct injury is seen (arrow).

Table 1. Literature review for hydralazine-induced DILI with autoimmune features

Author	Number of cases	Average age	Signs/Symptoms	Sex	Type	Average LFTs	Markers	Course	Outcome
Our case (2022)	1	59	Jaundice, ascites, abdominal distension	F	Cholestatic	ALT 100s AST 200s Bili 17	ANA- ASMA- AMA -	Moderate	Resolved
de Boer (2016)	7	60 (42-76)	Jaundice, itching, rash, fever, eosinophilia	5 F 2 M	Hepatocellular (3) Mixed (2) Cholestatic (2)	ALT 500s AST 1000s ALP 200s Bili 2	-	Mild (2) Moderate (3) Severe (2)	Liver transplant (1) Death (0)
deLemos et al. (2014)	1	42	Flu-like symptoms, jaundice, ascites	M		ALT 1000s AST 1000s ALP 200s INR 2	ANA+ ASMA- AMA-	Very severe	Liver transplant
Donald Rice, MD (1983)	1	51	Nausea, vomiting, malaise, fever, dark urine	F	Granulomatous hepatitis	AST 500s ALP 200s Bili 5	Lupus -	mild	Resolved

Abbreviations: F:female, M:male, LFT: liver function test, ANA:anti-nuclear antibody, ASMA:anti-smooth muscle antibody, AMA:anti-mitochondrial antibody.

S3030

Hepatic Portal Venous Gas Secondary to Ischemic Gastritis

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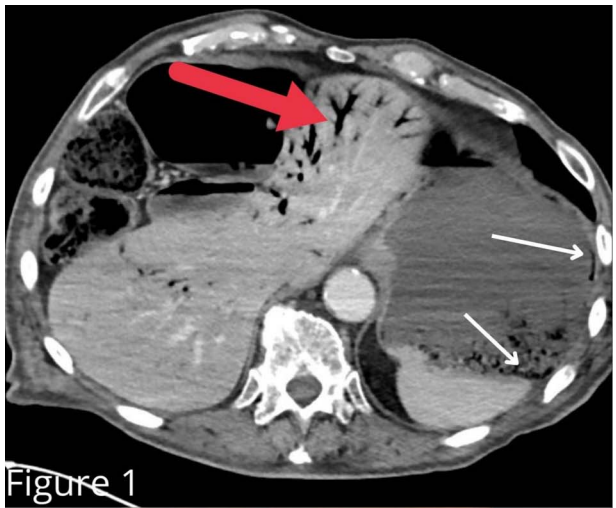
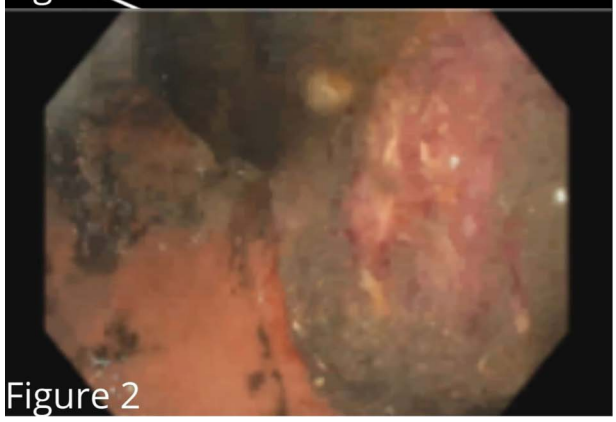
Introduction: Hepatic portal venous gas is an uncommon but potentially fatal radiographic finding caused by various disease processes with an overall in-hospital mortality rate of 27.3%. Herein we report a case with an uncommon cause of portal venous gas secondary to severe acute ischemic gastritis associated with gastric pneumatosis.
Case Description/Methods: An 86-year-old male with a past medical history of Parkinsons disease presented with persistent coffee ground emesis for one day. Initial laboratory workup revealed a hemoglobin of 8.9. The patient continued to experience multiple bouts of intractable vomiting unresponsive to antiemetics. He was subsequently intubated and placed on mechanical ventilation for airway protection and transferred to ICU. A CT abdomen and pelvis with IV contrast showed large amounts of portal venous gas in the liver accompanied by air in the mesenteric veins adjacent to the stomach and pneumatosis

involving the gastric fundus. Patient was started on a Pantoprazole drip and a nasogastric tube was placed with low intermittent suctioning for gastric decompression. Endoscopy revealed friable hemorrhagic and ulcerated mucosa; biopsy returned positive for active gastritis in the absence of H.pylori. This elucidated the underlying cause to be most likely secondary to ischemic gastritis. Following multiple blood transfusions and ultimately symptom resolution, the patient was extubated and repeat endoscopy revealed an improvement in gastritis from prior examination. (Figure)
Discussion: Hepatic portal venous gas is considered an imaging manifestation of various etiologies and cannot be used as a predictor of mortality by itself. Most cases are caused by intestinal ischemia. Other causes can largely be divided into iatrogenic and non-iatrogenic such as infection, trauma, and ulceration in the former to endoscopic procedures in the latter. Our case is unique in that ischemia is relatively uncommon in stomach, particularly in patients such as this one with relatively little atherosclerotic disease. It is important to differentiate the etiology of portal venous gas due to its varied presentation from benign to life-threatening, its mortality being determined by the underlying pathology.





[3030] **Figure 1.** CT scan of abdomen showing large amount of portal venous gas in the liver (red arrow) with pneumatosis involving the gastric fundus (white arrows). Figure 2: Endoscopy showing severe mucosal changes characterized by dusky discoloration, friability, hemorrhagic appearance and ulceration along the lesser curvature of the stomach.

S3031

Hepatic Angiosarcoma Masquerading as Benign Venous Malformation of the Liver: A Commonly Underdiagnosed Case

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Introduction: Hepatic angiosarcoma (HA) is uncommon yet notoriously deadly, accounting for 0.1- 2% of total primary liver malignancies. It is clinically challenging to diagnose due to its nonspecific presentation and absence of tumor markers.
Case Description/Methods: A 75 y/o male with a history of COPD and GERD presented with complaints of chronic fatigue. A routine CT chest identified an incidental liver mass 11 x 8 mm along with lower esophageal thickening and pulmonary nodules. Initial lab work-up showed AST/ALT-44/45 u/l, ALP-94 u/l, T Bil- 0.6 mg/dl, and acute hepatitis panel was negative. The patient was discharged for outpatient follow-up. Due to persistent symptoms, two months later, he was then referred to gastroenterology. He denied exposure to vinyl chloride, arsenic, thorium dioxide, and anabolic steroids. Tumor markers came back normal, CA 19-9 -8 u/ml, & AFP- 1.82 ng/ml. An MRI showed benign venous malformation, but a liver biopsy confirmed HA. Eventually, the patient started chemotherapy with a suspicion of metastasis based on prior CT imaging. He could not tolerate the side effects of Paclitaxel after two doses, so he opted for hospice care and was discharged.
Discussion: Hepatic angiosarcoma rapidly progresses with a potential for metastases increasing mortality. Often, radiological findings can be misleading in the initial stages, warranting an invasive approach, liver biopsy, to confirm the diagnosis. Due to its rare occurrence and absence of specific symptoms, we encourage a multi-disciplinary approach, particularly involving gastroenterology during the initial stages, to decrease the mortality in these cases. Also, additional research is required to improve diagnostic accuracy, establish treatment guidelines, and guide our future therapies.

S3032

Hiding in Plain Sight: An Atypical Presentation of Metastatic Breast Cancer as Acute Decompensated Cirrhosis

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Introduction: Liver is a common site of breast cancer metastases, which are typically identified as hypervascular lesions on imaging. We present a rare case of breast cancer leading to diffuse, intra-sinusoidal, radiographically occult metastases in a patient presenting with new hepatic decompensation.

Case Description/Methods: A 65-year-old woman with a remote history of grade IIIa invasive ductal carcinoma of the right breast status post lumpectomy, chemotherapy, and local radiation therapy was referred to liver clinic for 1 month of abdominal pain and newly elevated AST 144 U/L, ALT 143 U/L, total bilirubin 5.2 mg/dL, conjugated bilirubin 3.7 mg/dL, and alkaline phosphatase 652 U/L. She denied use of alcohol, substances, new medications, or supplements. Physical exam did not demonstrate stigmata of chronic liver disease. Work up for infectious, autoimmune, and common infiltrative etiologies of liver disease were negative; several tumor markers were elevated (Table 1). A routine surveillance PET-CT and subsequent MRCP demonstrated diffuse heterogeneous liver attenuation with cirrhotic morphology, splenomegaly, trace ascites, and intrabdominal lymphadenopathy. No discrete masses were seen on either study. The following week the patient was admitted for rapidly progressive failure to thrive, worsening ascites, jaundice, and oliguria. Inpatient labs demonstrated worsening hyperbilirubinemia (total bilirubin 14.8 mg/dL, conjugated bilirubin 12.2 mg/dL), hyponatremia, and AKI. Admission MELD-Na score was 31 and CLIF-C ACLF score was 46. Paracentesis demonstrated SAAG 1.6 with ascites protein 1.8 g/dL confirming Child Pugh Class B Cirrhosis. Transjugular liver biopsy measured hepatic venous pressure gradient 12mmHg and pathology demonstrated metastatic breast carcinoma of the sinusoidal and vascular spaces with background steatosis and non-bridging fibrosis. Despite aggressive treatment with fulvestrant and abemaciclib, she had worsening multiorgan failure. Given her overall poor prognosis, the patient elected to transition to hospice.
Discussion: Metastatic breast cancer uncommonly presents with diffuse, intra-sinusoidal metastases that are radiographically occult. This case highlights the importance of maintaining a high index of suspicion for metastatic disease in patients with a history of malignancy presenting with new hepatic decompensation even in absence of typical radiographic findings. In such cases, liver biopsy may be required for a definitive diagnosis. (Table)

Table 1. Work up for etiologies of chronic liver disease

Autoimmune markers	
ANA	Positive (1:320, speckled)
Anti-mitochondrial Ab	< 1:20
dsDNA	< =200
Smooth muscle Ab	< 20
RNP Ab	< 20
SSA Ab	68 (Elevated)
SSB Ab	< 20
Rheumatoid Factor	< 10
Thyroid Peroxidase Ab	135 (Elevated)
Beta-2-Glycoprotein IgA/IgG/IgM	< 10
Cardiolipin IgA/IgG/IgM	< 20
C-ANCA Myeloperoxidase Ab P-ANCA Proteinase-3 Ab	< 1:20 < 1:20 < 1:20 < 1:20
Infectious studies	
Hep A Ab	Nonreactive
Hep C Ab	Nonreactive
Hep B surface Ab	< 10
Hep B surface Ag	Nonreactive
Hep B core Ab	Nonreactive
MTB-Quantiferon-Gold	Negative
Other causes of Liver Disease	
Alpha-1-antitrypsin	Negative
SPEP/UPEP	No monoclonal bands observed
Ceruloplasmin	Elevated
Copper	Elevated
Ferritin	Elevated
Tumor Markers	
AFP	4.2 ng/mL, decreased to < 1.82 ng/mL
CEA	84 ng/mL, increased to peak 3042 ng/mL (Elevated)
CA 125	930 U/mL
CA 19-9	1166 U/mL
CA 27.29	7450.1 U/mL (Elevated)

S3033

HIV-AIDS: A Rare Cause of Cholestatic Cholangiopathy

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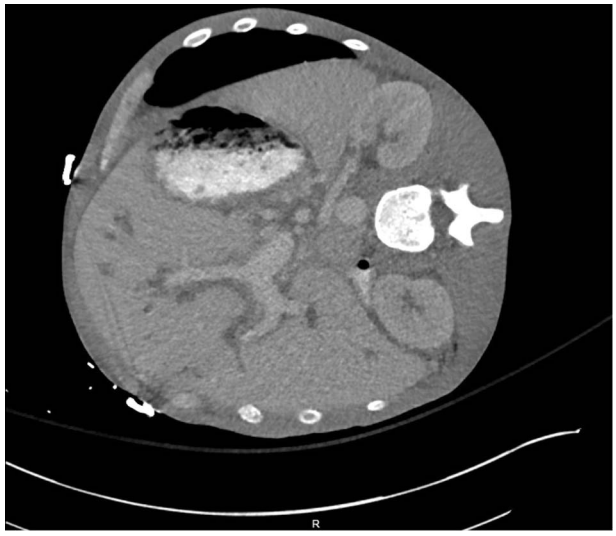
Introduction: HIV/AIDS has been shown to affect hepatic parenchyma and biliary tree leading to inflammation and biliary strictures. With the advent of highly active antiretroviral therapy (HAART), it is rarely encountered, except in cases of resistance or medication non-compliance.
Case Description/Methods: A 48-year-old man with history of HIV and poor compliance with HAART presented with abdominal pain, nausea and diarrhea. Physical exam showed scleral icterus and mild abdominal tenderness, with labs showing a T.bili. 16.9, AST 212, ALT 133, and ALP 3885. MRCP revealed intrahepatic biliary ductal dilatation of both lobes of liver and mild dilatation of the proximal extrahepatic CBD (9 mm) without cholelithiasis, biliary sludge or ductal stone suggesting nonspecific cholestasis. ERCP was performed which revealed papillary stenosis and moderately enlarged CBD (12 mm). Irregularities were noted in bilateral intrahepatic branches but not in ducts. A pancreatic and biliary sphincterotomy was performed. With minimal decrease in T.bili. and ALP, repeat MRCP was performed 3 months later showing persistent intrahepatic biliary ductal dilatation with normal CBD. Repeat ERCP showed patent prior sphincterotomy and the biliary tree was swept with nothing found. Further workup revealed a CD4 count of 17 and serology positive for cryptosporidium antigen. A diagnosis of HIV-cholangiopathy was made and the patient was restarted on HAART and nitazoxanide with gradual decline in T.bili and ALP. (Figure)
Discussion: HIV/AIDS-cholangiopathy is a rarely encountered biliary syndrome, usually manifesting in severely immunosuppressed AIDS patients. Several opportunistic pathogens have been implicated in its pathogenesis with Cryptosporidium parvum being the most commonly associated pathogen. C. parvum predominantly affecting the biliary tract leading to apoptotic cell death of the cholangiocytes. Biliary involvement has been described in four cholangiographic patterns – papillary stenosis, extrahepatic strictures, intra and extrahepatic sclerosing lesions, and acalculous cholecystitis. Diagnosis can be established

usually by MRCP or ERCP which shows papillary stenosis with intrahepatic ductal dilatation. Alternating multifocal intrahepatic strictures can also be seen giving a beaded appearance. The cornerstone of management involves the management of opportunistic infections with the initiation of HAART. The prognosis is usually poor due to advanced stages of immunosuppression and multiple opportunistic infections.



[3033] **Figure 1.** CT Abdomen pelvis showing intrahepatic biliary ductal dilation.

S3034

Histoplasmosis Infection Presented as Cholestatic Liver Disease

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Introduction: Histoplasmosis is the most common mycosis in the United States with peak incidence in the Midwest region. In ~21% of patients with systemic histoplasmosis hyperbilirubinemia can be detected. However, cholestatic presentation of histoplasmosis infection is very rare. Here we present a case of immunocompromised patient with predominant cholestatic liver disease caused by Histoplasma Capsulatum.
Case Description/Methods: This is the case of 56- years- old female who presented with– sided abdominal pain, shortness of breath, chills, hot flushes, nausea, vomiting and diarrhea. She is suffering from rheumatoid arthritis. Her medications are adalimumab, celecoxib, hydroxychloroquine. She denied alcohol intake. Her initial workup including liver function tests (LFTs) was positive for a total bilirubin 3.5 mg/dl, conjugated bilirubin 2.7 mg/dl, alkaline phosphatase – 547 U/L, AST – 152 U/L, ALT-142 U/L, and platelet count – 55 k/ul. During hospitalization her abdominal pain was getting progressively worse, and her LFTs continued to trend up. Eventually liver biopsy was performed. The results showed multiple granulomas with central necrosis, and associated portal and lobular inflammation. The fungal stain highlighted yeasts suggestive of histoplasma. Her urinary histoplasma Ag Quantitative EIA eventually came back positive -8.87 ng/ml. Viewing the normal chest x-ray and ongoing hypoxia, chest CT scan with IV contrast was performed showing 1.1 cm lung nodule in the left lower lobe. A subsequent bronchoscopy revealed budding yeasts. Patient was started on liposomal amphotericin B, and her LFTs trended down. The patient was treated with amphotericin B for 21days subsequently changed to twice-daily oral Itraconazole 200mg. At the outpatient follow up visit, patient admitted a complete resolution of her abdominal pain and improvement of her breathing status.
Discussion: Liver histoplasmosis is a rare cause for cholestatic liver disease. Over the past two decades, a limited number of cases with similar clinical scenario were described. Despite of the rarity of this condition, hepatic histoplasmosis should be considered in a selected population of immunocompromised patients living in an endemic area (ex: Ohio). Inclusion of urine antigen testing for histoplasma in early workup can potentially prevent diagnostic delay.

S3035

Hepatocellular Carcinoma in a Non-Cirrhotic Patient Treated With Left Hepatic Trisegmentectomy: A Case Report

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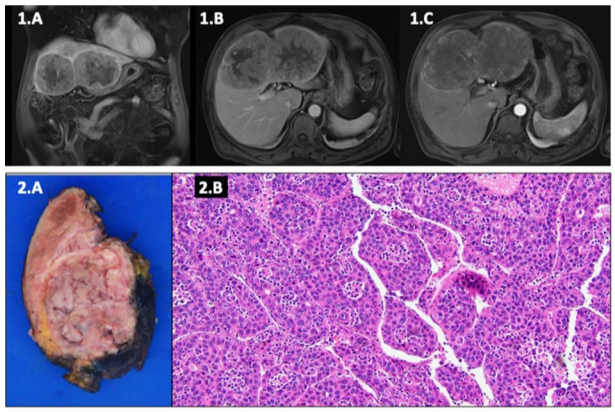
Introduction: Hepatocellular carcinoma (HCC) is the 5th-most common cancer and the 3rd-most common cause of cancer-related mortality. Chronic liver disease is the most important risk factor for HCC and 80% of cases are in patients with cirrhosis. HCC has an insidious presentation in patients without cirrhosis and is often found incidentally.
Case Description/Methods: A 68-year-old male presented with acute right upper quadrant pain. History was notable only for daily 12-beer consumption for the preceding 6 months. Laboratory tests revealed normal liver profile and international normalized ratio. Ultrasound demonstrated hepatomegaly and a heterogenous mass. Computed tomography (CT) with contrast of the abdomen demonstrated a large mass involving segments 2, 3, 4, 5, and 8 measuring 19.8 cm in largest dimension. Magnetic resonance imaging was concerning for fibrolamellar variant HCC without imaging findings suggestive of cirrhosis (fig. 1). CT scan from 1 year prior demonstrated non-cirrhotic liver morphology and hemangioma in the left lobe. Chronic liver disease workup was unremarkable. Alpha-fetoprotein was elevated to 150,000 ng/mL. Staging scans were negative for metastatic disease. Biopsy of normal liver parenchyma excluded underlying cirrhosis with mild mixed vesicular steatosis (20%) and stage 2 fibrosis. Volumetry confirmed adequate residual liver volume. After multi-disciplinary discussions, the patient underwent a left hepatic trisegmentecomy. Pathology demonstrated macrotrabecular-massive variant of HCC with lymphovascular invasion (Fig. 2).
Discussion: Macrotrabecular-massive variant is characterized by thick trabeculae and has been reported in as few as 5% of HCC cases. It is associated with frequent lymphovascular invasion and elevated AFP levels. If residual liver volume is ≥40% of original, curative resection is recommended in non-cirrhotic HCC patients. Unfortunately, post-resection recurrence rate is high, and risk factors include microvascular invasion, large tumor size, AFP >400 ng/mL, and cirrhosis. For patients who have HCC recurrence without macrovascular invasion or extrahepatic spread, liver transplant may be considered. The Barcelona Clinic Liver Cancer staging system and the Liver Imaging Reporting and Data System (LI-RADS) have been widely validated in patients with cirrhosis; however, neither system has been studied in non-cirrhotic HCC. This case highlights the rarity of non-cirrhotic HCC and benefit of multi-disciplinary discussions to create individualized treatment plans.



[3035] **Figure 1.** (A) Coronal and (B) axial arterial phase MRI through the abdomen demonstrating heterogenously enhancing bi-lobed mass with non-enhancing central scar. (C) Axial portal venous phase MRI demonstrating portal venous washout with non-enhancing central scars in a bilobed mass. 2: (A) Gross specimen demonstrating tumor. (B) Histology demonstrating macrotrabecular variant HCC.

S3036

Hepatoid Gastric Adenocarcinoma

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Introduction: Hepatoid gastric adenocarcinoma is a rare extrahepatic tumor, characterized by morphological similarity to hepatocellular carcinoma and production of alpha-fetoprotein. We report a case of gastric hepatoid adenocarcinoma occurring at the esophagogastric junction; a location scantily reported of this lesion subtype.
Case Description/Methods: Our patient presented with abdominal pain, regurgitation, weight loss, and bulky retroperitoneal lymphadenopathy on imaging. Later, two enlarging abdominal wall masses emerged prompting biopsy, staining positive for glypican-3, Hep-Par-1, and alpha-fetoprotein. On endoscopy, an ulcerating mass was noted at the esophagogastric junction extending into the cardia. Next-generation sequencing identified 68th percentile PD-L1 expression. Treatment was initiated with oxaliplatin, 5-fluorouracil, leucovorin, and nivolumab. After initial regression, he later succumbed.
Discussion: Hepatoid adenocarcinoma accounts for only 0.17-15% of gastric cancer subtypes[1]. Most reports involve lesions occurring solely in the stomach — very few describe occurrences involving the esophagogastric junction supplementing the uniqueness of this case. It also commonly co-occurs with metastatic liver lesions[2] which were not so in this circumstance. These tumors produce alpha-fetoprotein and may also stain positive for novel hepatocellular tumor markers: glypican-3 and Hep-Par-1. In the phase 3 Checkmate 649 trial, the PD-1 inhibitor nivolumab combined with chemotherapy was superior to chemotherapy alone[3]. There are currently no evidence-based recommendations for therapy. Current treatment strategies are extrapolated from other gastric cancers lending to a dismal prognosis.

S3037

Hepatocellular Carcinoma With Para-Celiac Lymph Node Metastasis Presenting as Unknown Primary Neoplasm in the Post-Liver Transplant Setting

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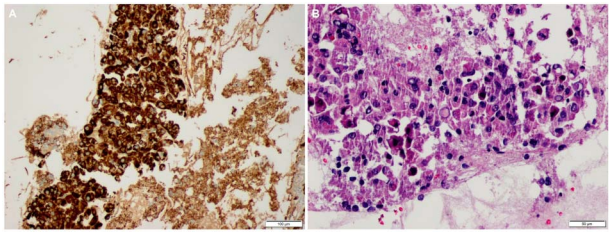
Introduction: Metastatic HCC without primary liver lesion is an exceedingly rare clinicopathologic entity. Only a handful of case reports have been reported to date, mostly involving bony structures without primary liver cancer. To our knowledge, we hereby present the first case of metastatic HCC involving the para-celiac lymph nodes in the post-liver transplant setting.
Case Description/Methods: A 73-year-old male who previously underwent liver transplant for non-alcoholic steatohepatitis was admitted to GI for the evaluation of a suspicious pancreatic tail cyst. He denied acute GI symptoms. Laboratory studies revealed normal liver enzymes. CT abdomen and pelvis showed a 2-cm lesion located under the right hemidiaphragm, posterior to the right hepatic lobe. Subsequently, the patient underwent diagnostic EUS with biopsy for both lesions. EUS revealed an irregularly shaped oval cyst in the tail, round cysts in the body and neck, and a complex of small cysts in the pancreatic head. The 45 x 55-mm para-celiac mass was irregularly shaped, hypoechoic, homogenous solid lesion, located lateral to the celiac axis. It had well-defined endosonographic borders. Immunostaining of the fine-needle biopsy (FNB) of the para-celiac mass was positive for Hep Par-1 and pancytokeratin, consistent with metastatic HCC (Figure 1; A&B). Fine-needle aspiration (FNA) of the pancreatic cysts was negative for malignancy. Alpha-fetoprotein level was 11.1 ng/mL. The diagnosis of HCC of unknown primary was finally made. The patient then underwent an uneventful surgical resection.
Discussion: To our research, this is the first reported case of metastatic HCC without primary liver lesion involving the para-celiac lymph nodes in a post-transplant patient. With regard to pathogenesis, one theory implicates the presence of ectopic liver tissue that transforms into HCC. The presence of micro-HCC that regresses spontaneously or gets destroyed by the immune system can also be a plausible explanation. Standard diagnostic protocol for HCC consists of imaging (CT or MRI) and/or biopsy. Even in patients with cirrhosis, the diagnostic modality of choice is imaging and liver biopsy is less frequently performed. Biopsy is recommended for atypical hepatic lesions on imaging or nonclassical enhancement patterns or detection in the absence of cirrhosis. No standardized treatment exists for metastatic HCC with unknown primary. This report illustrates that clinicians should consider HCC while evaluating patients for the primary origin of metastatic carcinoma.



[30371_wc] **Figure 1.** Immunostaining of the para-celiac mass. Endoscopic ultrasound-guided fine-needle biopsy (FNB) specimen indicating positive status for Hep Par-1 and pancytokeratin.

S3038

Hepatic Graft vs Host Disease in Patients Post Stem Cell Transplant

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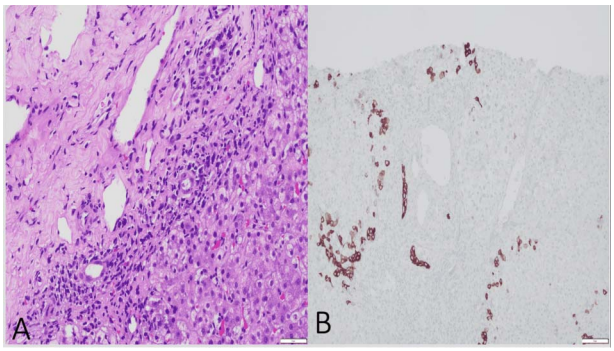
Introduction: Hepatic graft versus host disease (GVHD) is a rare but severe complication in patients who received stem cell transplant (SCT) that needs early diagnosis and management by a multidisciplinary team.
Case Description/Methods: 40-year-old female with B cell-Acute Lymphocytic Leukemia (ALL) s/p allogeneic stem cell transplant (SCT) stopped taking tacrolimus 3 months ago, and presented with fever, nausea, vomiting, and jaundice for 7 days. She also had skin rash but no diarrhea. Liver tests on presentation include AST 124, ALT 69, ALKP 1251, T. Bili 23.9. Extensive viral panels were negative (HAV, HBV, HCV, HSV, HEV, EBV, CMV, VZV, Adeno & HHV6). The autoimmune, genetic, and metabolic comprehensive workups were unremarkable except for elevated Ferritin and ASMA. MRCP did not show biliary obstruction. Histology results from the liver biopsy showed frequent bile ducts destruction with lymphocytic cholangitis and ductopenia in 60% of portal triads with moderate portal-based mixed inflammation consistent with GVHD. She also had evidence of GVHD from skin biopsy. After admission, she was restarted tacrolimus and steroids at 2 mg/kg for 5 days and then infliximab 10 mg weekly (2 doses). Given otherwise stable condition and 50% improvement in ALKP, she was discharged on ruxolitinib 5 mg as an outpatient with close follow-up. (Figure)
Discussion: Hepatic GVHD is a T-cell mediated disease in which donor T lymphocytes recognize host antigens as foreign producing tissue injury. The cumulative incidence of hepatic GVHD is 6.7% in those who underwent SCT. Hepatic GVHD present as jaundice and elevated bilirubin and ALKP. The diagnosis usually requires liver biopsy. Hallmark histologic finding is bile duct injury. Ductopenia, fibrosis, and ductular proliferation can also be seen in chronic cases. Portal inflammation with or without interface hepatitis or lobular inflammation can also be seen in some patients. It is important to rule out other causes of liver dysfunction, including drugs, infections, hepatic sinusoidal obstruction syndrome, and cancer relapse. Management for severe hepatic GVHD is high dose steroids (1-2 mg/kg) along with calcineurin inhibitor. If there is no improvement within 5 days then second-line agents (extracorporeal photopheresis, IL 2 Receptor antibodies, anti-TNF antibodies, mTOR inhibitors, and MMF) and third-line agents (mesenchymal stem cells, methotrexate, alemtuzumab, pentostatin) are used. Ruxolitinib (JAK inhibitor) is one of the newest therapies used for steroid refractory GVHD.



[3038] **Figure 1.** A. Bile duct injury identified by intraepithelial lymphocytes, cytoplasmic vacuolization, and nuclear disarray (H&E, 200x) B. CK7 immunohistochemical stain showing loss of bile duct and biliary hepatic metaplasia(CK7, 200X).

S3039

Hepatic Undifferentiated Pleomorphic Sarcoma Mimicking Liver Abscess

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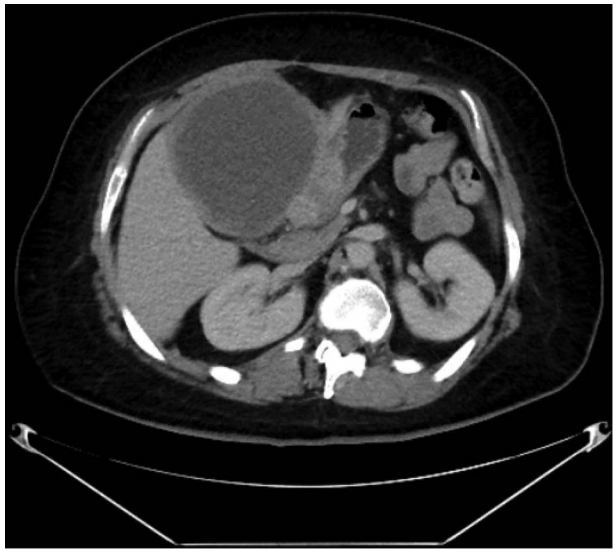
Introduction: Undifferentiated pleomorphic sarcoma (UPS) has been recognized as one of the most common malignant soft tissue tumors. It usually affects the elderly population and involves the extremities. However, hepatic UPS is rare. Fewer than 200 cases have been reported. Primary hepatic UPS occurs in late adulthood. It typically presents with a solid hepatic mass with regional lymph node involvement. Occasionally, UPS mimics hepatic infection clinically. Here we report a case of hepatic UPS, mimicking the presentations of liver abscess.
Case Description/Methods: A 44-year-old female with a past medical history of hypertension presented at the emergency department (ED) for right upper abdominal pain for one week. The pain was described as dull, 5/10 on a pain scale, non-radiating, and associated with nausea, fever, and chills. The pain was worsened by sitting up and alleviated by lying down. At ED, the patient's vital signs were within normal range. Physical examination showed right upper quadrant tenderness, without rebound, guarding, or rigidity. She was found to have slightly elevated liver enzymes and alkaline phosphate. CT of the abdomen revealed an irregular hypodense mass at the right hepatic lobe measuring 14.3 x 11.6 cm and a 2.6cm enlarged porta hepatis lymph node. The patient was admitted for further evaluation. Concerning possible liver abscess, the blood culture was collected, and the patient was treated with empiric antibiotics. Tumor markers including AFP, and CEA were within normal range. The blood culture was negative for infection. To rule out malignancy, PET scan was performed and showed a hypermetabolic mass with necrotic components in the liver. The patient underwent a liver biopsy. The pathology revealed extensively necrotic pleomorphic round cells with eosinophilic cytoplasm and variably sized, oval nuclei, indicating hepatic undifferentiated pleomorphic sarcoma. Oncology was consulted and recommended doxorubicin and ifosfamide for neoadjuvant chemotherapy. The patient was discharged to home to follow up with oncology as outpatient for chemotherapy and surgery for tumor resection. (Figure)
Discussion: Hepatic UPS is a rare malignant mesenchymal tumor. Nonspecific clinical symptoms, unmarkable laboratory findings, and rapid tumor growth lead to poor prognosis. Physicians should be aware of this rare disease as a differential diagnosis for liver mass. Surgical resection is the preferred treatment. Chemotherapy could be used as neoadjuvant or adjuvant treatment to improve the survival rate.



[3039] **Figure 1.** a large liver mass noticed on CT of abdomen with contrast.

S3040

HSV Hepatitis Resulting in Acute Liver Failure and Liver Transplantation

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Introduction: Herpes simplex virus (HSV) is a rare cause of hepatitis that can result in acute liver failure (ALF) in 75% of affected patients and is associated with a high mortality rate (90%). We present a case of a 38-year-old female with ALF from HSV hepatitis resulting in orthotopic liver transplant (OLT).
Case Description/Methods: A 38-year-old female presents with abdominal pain, persistent fever, headache, and generalized malaise. Her mental status acutely decompensates, and she is intubated. Head CT reveals no acute process for cerebral edema. She is diagnosed with ALF, having elevated AST (5,070 IU/L), ALT (3,000 IU/L), TBIL (3.0 mg/dL), and INR (1.38). A complete blood count reveals low white blood cell count (1.7 K/uL) and platelet count (25 K/uL). HSV-2 is detected in the blood via polymerase chain reaction. Given her clinical presentation and rapid deterioration, she is urgently evaluated for liver transplantation and undergoes OLT shortly after being listed. A biopsy of her explant reveals submassive hepatic necrosis with positive immunostaining for HSV, predominantly in areas of necrosis, revealing underlying HSV viremia. Her post-transplant course is complicated by acute kidney injury requiring renal replacement therapy with improvement, bile leakage requiring Roux-en-Y hepaticojejunostomy, and neutropenia. The patient is treated with intravenous acyclovir and then transitioned to oral valacylovir, upon which she develops cytomegalovirus viremia and is started on valgancyclovir treatment. She clinically improves but did have recurrent labial lesions with HSV, similarly treated with valacylovir.
Discussion: HSV hepatitis is a rare cause of ALF. Its clinical presentation often includes fever, encephalopathy, coagulopathy, and acute renal failure. Leukopenia and thrombocytopenia are also commonly associated with HSV hepatitis. Mucocutaneous lesions are not present in more than half the patients. This makes diagnosis difficult without a thorough history, lab evaluation, and histopathology. Liver biopsy is the gold standard for diagnosis of HSV hepatitis with histology revealing focal or confluent areas of acidophilic-type necrosis with little inflammation. Treatment with acyclovir early in the course may result in a better prognosis and reduce the need for OLT. Once ALF is diagnosed, OLT is the definitive treatment. Because of its rapid and aggressive progression, consideration of HSV hepatitis in the differential diagnosis and timely treatment is critical.

S3041

Hepatic Artery Dissection – A Case Report

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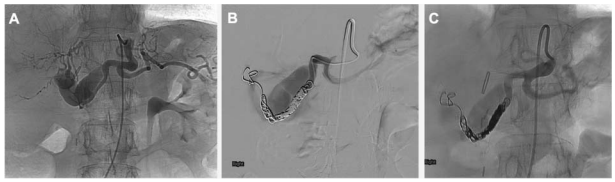
Introduction: Visceral artery aneurysms are rare pathologies with reported incidence rates between 0.01% to 0.2%; even rarer are hepatic artery dissections (HAD). Most aneurysms rupture or are incidentally found on autopsy, but imaging advancements has enabled earlier identification of aneurysms; in this case, a HAD.
Case Description/Methods: A 77-year-old female with medical history pertinent for sickle cell trait, acquired factor VIII inhibitor coagulopathy, history of type 2 thoracic aortic dissection, and hypertension presented to the emergency room with a week of mild fatigue, postprandial epigastric burning pain, and intermittent stabbing right upper quadrant pain. Right upper quadrant ultrasound demonstrated a hypervascularized right hepatic lobe mass with large tortuous internal vessels. CT abdomen & pelvis without contrast showed a large hypodensity in the right liver with curvilinear density. MRI demonstrated large arteriovenous malformation (AVM) within the right hepatic lobe and focal hepatic parenchymal hemorrhage centered within AVM nidus. Gastroenterology was consulted and CT angiogram findings were suspicious for common HAD, proper hepatic & intrahepatic pseudoaneurysms, and intrahepatic rupture. Interventional radiology was consulted. Hepatic angiogram showed a large defect in the proximal hepatic artery with brisk opacification, thought to represent a pseudoaneurysm. This prompted framing coil placement across the large defect and coils extending from the proper hepatic artery to the common hepatic artery with significant reduction in forward flow & pseudoaneurysm opacification. Per hepatobiliary & vascular surgery, no need for additional surgical intervention. Postoperative course was complicated by fever & leukocytosis, worsening kidney injury, and anemia; attributed to the procedure and resolved with supportive care. Patient discharged on postoperative day 8 with outpatient follow-up. (Figure)
Discussion: This case highlights the importance of imaging for earlier detection of hepatic artery aneurysms (HAA) in minimizing risk of catastrophic hemorrhage. HAA are rare but clinically important given they are associated with high incidence of rupture and mortality up to 20%. Although surgery is preferred for extrahepatic findings, endovascular interventions are favored for intrahepatic findings depending on size and presence of pseudoaneurysms, such as this case. They have led to more favorable short & longer-term outcomes and lower mortality & morbidity rates, particularly in poor surgical candidates.



[3041] **Figure 1.** Hepatic angiography and embolization was performed. A large defect with brisk opacification of the proper and common hepatic arteries were seen (A), prompting deploy of a framing coil. (B) Embolization was then performed on the hepatic artery with three coils (5mm, 6mm, & 8mm). Coils were examined from proper hepatic artery to the common hepatic artery. (C) Following placement of the coils, significant reduction in flow and opacification of the pseudoaneurysm was seen.

S3042

Hepatic Lymphoproliferative Lesions in the Setting of Chronic, Multi-Drug Immunosuppression

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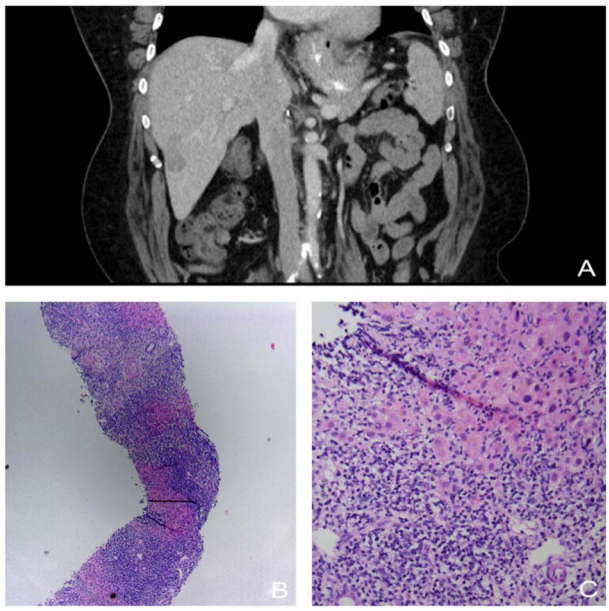
Introduction: Non-Hodgkin's lymphoma (NHL) is a relatively common cancer, however one that originates in the liver as a primary hepatic lesion is exceedingly rare, making up only 0.016% of NHLs. Understanding the course of this disease is vital to earlier intervention and management. Some researchers have found connections between biologic drugs like TNF-alpha inhibitors and lymphoma, but this association is not well understood or thoroughly documented. Methotrexate has a well-documented association with primary hepatic lymphoma as one of its most serious side effects. The purpose of this case report is to document the course of primary hepatic lymphoproliferative disease in the context of chronic, multi-drug immunosuppression.
Case Description/Methods: This is a 61-year-old African American male with a history of severe rheumatoid arthritis (RA). He was on etanercept/leflunomide therapy from 2001 until 2016. Due to a recurrent scleritis, he was switched to adalimumab in 2016. He was on methotrexate for a year and long-term prednisone therapy since 2011. In 2018, an incidental finding on CT imaging showed multiple hypodense lesions in this patient's liver, as well as splenomegaly. A repeat CT in 2019 redemonstrated the lesions and a follow-up biopsy was suspicious for B cell lymphoproliferative disorder but not confirmatory. A repeat biopsy three months later was non-diagnostic. In 2021, a CT showed scattered, new lesions and old lesions decreased in size. A follow-up biopsy demonstrated atypical lymphoid infiltrates with small lymphoid cells and irregular nuclear contour. Immunohistochemistry stained positive for CD20, mildly positive for LM02 and CD10, and negative for EBER ISH, BCL6, Cyclin D1, and CD23, along with a low Ki67 proliferative index. CEA, AFP, and liver enzyme levels were normal. A month after these results and the discontinuation of adalimumab, a PET scan showed unremarkable findings with no hypermetabolic hepatic lesions. (Figure)
Discussion: Long-term use of adalimumab is thought to have caused this patient's lesions while the prednisone simultaneously treated them, explaining the inconclusive biopsies and fluctuating CT findings. Chronically ill patients receiving long term biologic therapy are at greater risk for developing lymphomas, and treating physicians should be hypervigilant and familiar with side-effects and drug-interactions. Further studies are needed to elucidate the influence of these medications on hepatic lymphoproliferative disease processes.



[3042] **Figure 1.** (A) Liver CT from 2021 depicting an enlarging, hypodense lesion (B) Liver biopsy H&E stain, 4x (C) Liver biopsy H&E stain, 20x.

S3043

Hereditary Hemochromatosis Unmasked by Drug-Induced Liver Injury From Amoxicillin-Clavulanate

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Introduction: Drug-induced liver injury from amoxicillin-clavulanate remains one of the most frequent causes of non-acetaminophen drug-induced liver injury per prospective registries across the US and Europe. It is estimated the incidence is 14-19 cases per 100,000. Patients typically present with symptoms of cholestasis – fatigue, nausea, loss of appetite and pruritis. This translates to cholestatic liver enzyme abnormalities. It is important to rule out other causes of liver injury which makes DILI a difficult diagnosis. We present a patient with newly diagnosed hereditary hemochromatosis unmasked by drug induced liver injury secondary to amoxicillin-clavulanate use.
Case Description/Methods: 75-year-old female presented to her primary care provider for sinusitis and was prescribed amoxicillin-clavulanate two weeks prior. She began to experience nausea, vomiting, reflux and epigastric abdominal pain. She subsequently developed worsening pruritus, and scleral icterus. No other new medications or alcohol use were reported. Lab work showed an alkaline phosphatase of 574, AST 251, ALT 602, total bilirubin 4.2 and INR of 1.06. Elevated ferritin of 635 and iron saturation of 94%. Abdominal ultrasound and CT abdomen/pelvis were unremarkable. Trending of lab work showed ALP peak value of 682 and total bilirubin of 5.0 while AST and ALT were down-trending. Extended liver work up revealed a negative acute hepatitis panel, ANA positivity however anti-actin antibody, anti-smooth muscle antibody, and anti-mitochondrial antibody were all negative. Interestingly, the patient was found to be an HFE H63D homozygote. Gastroenterology follow up was arranged one week after hospital discharge with lab work at that time showing improving values ALP 571, AST 144, ALT 233, and total bilirubin of 2.7. Patient required cholestyramine, ursodiol and hydroxyzine to control the pruritus, otherwise the remainder of her symptoms were vastly improved. She was referred to Hematology-Oncology for further evaluation of Hemochromatosis.
Discussion: The exact mechanism of amoxicillin-clavulanate causing drug induced liver injury is unknown, but theorized to be in relation to an immune-allergic response. The onset of injury can last up to 8-10 weeks, fortunately there is rarely long-lasting injury. Similar to our patient, there has been a prior report of a patient with acute liver injury with jaundice secondary to amoxicillin-clavulanate which obscured underlying hemochromatosis diagnosed during the work up.

S3044

Hepatoid Adenocarcinoma - A Mimicker of Hepatocellular Carcinoma

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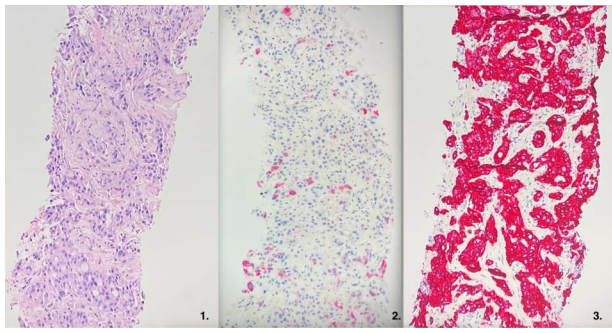
Introduction: Hepatoid adenocarcinoma of the lung (HAL) is a rare lung carcinoma that resembles hepatocellular carcinoma of the liver (HCC) on histology. Immunohistochemical staining distinguishes the two. We present a case of HAL in which treatment targeting HCC achieved significant response.
Case Description/Methods: A 69-year-old male with a history of tobacco use presented with pathologic weight loss. CT scan revealed a 31mm lung nodule with mediastinal adenopathy in addition to a 10mm liver lesion. Core needle biopsy of the pulmonary nodule was performed. Histology showed a poorly-differentiated carcinoma comprising polygonal cells containing eccentrically located nuclei and abundant eosinophilic cytoplasm with cells arranged in solid nests, resembling HCC.(Image 1) Treatment with atezolizumab and bevacizumab was initiated. However, given the patient's lack of risk factors for HCC, further pathology analysis was requested. Immunohistochemical staining was positive for Hep-Par, consistent with HCC.(Image 2) However, strong diffuse positivity for cytokeratin-7 favored lung as the primary site of the tumor.(Image 3) This led to a change in the diagnosis from HCC to HAL. Two cycles of immunotherapy led to a significant reduction in tumor burden. After two additional months of therapy, new onset back pain led to the discovery of spinal metastases. Treatment was changed to carboplatin and Taxol, but the patient only received one cycle before his decline and passing.
Discussion: This case highlights a unique pathology to be considered when histology suggests HCC. HAL resembles HCC histologically; in both there are cells with abundant eosinophilic cytoplasm arranged in sheet-like portions. Immunohistochemical staining differentiates between the two. While Hep-Par stains are positive in both, only cytokeratin-7 stains are positive in HAL.[i] The prognosis for HAL is poor. Partial disease response has been reported with platinum-based chemotherapy.[ii] This case is unique in that there was a significant response to atezolizumab and bevacizumab, which was first chosen to target HCC.



[3044] **Figure 1.** Haematoxylin and eosin stain showing poorly differentiated polygonal cells containing eccentrically located nuclei and abundant eosinophilic cytoplasm with cells arranged in solid nests. 2. Hep-par stain positivity suggestive of hepatocellular carcinoma. 3. Cytokeratin-7 stain diffusely positive favoring lung as the primary tumor site.

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S3045

Hepatitis With an Unknown Etiology with Concomitant ITP

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Introduction: Alcoholic hepatitis (AH) usually presents after decades of alcohol consumption and can even manifest with recent abstinence. The clinical presentation may be compounded by underlying liver cirrhosis and LFT's are not a reliable means of diagnosing AH due to poor sensitivity and specificity. Hepatitis is only one of the major causes of thrombocytopenia. Immune thrombocytopenic purpura (ITP) is a diagnosis of exclusion and further workup is warranted with persistent thrombocytopenia refractory to treatment for AH. Although there is limited data demonstrating a correlation between AH and ITP, both conditions respond to steroids. We present a case of a 42 YO M with an unknown cause of hepatitis and concomitant ITP who responded well to steroids.
Case Description/Methods: A 42 YO M PMH of alcohol use disorder (abstinent for 3 months), presented with acute bilateral lower extremity edema and diffuse petechiae. Vitals were within normal limits and exam revealed diffuse jaundice, scleral icterus, scattered ecchymoses on his extremities and flanks, with bilateral pitting edema. Labs were remarkable for anemia (Hb 11.5g/dL), thrombocytopenia (PLT 87K/mm3). hyponatremia (132mEq/L), hypokalemia (3.1mEq/L), abnormal LFT's (ALP 226 U/L, AST 156 U/L, ALT 86 U/L), and conjugated hyperbilirubinemia (Tbili 55.2mg/dL, Dbili 31.8mg/dL). MDF was 84.6. An extensive hepatitis workup was unremarkable. Abdominal U/S revealed a nodular and echogenic liver of 19.8 cm. CT confirmed cirrhosis with evidence for portal hypertension and splenomegaly. He was admitted for suspected AH and treated with a 7-day course of prednisolone 40 mg daily. Further workup revealed hemosiderinuria, low haptoglobin, and positive IIb/IIIa antibodies, concerning for ITP. Labs were repeated one month following discharge and he was continued on steroids for a total of 48-days with taper. He was seen in office with resolution of his ITP and presumed AH, with a total bilirubin of 1.9 mg/dL.
Discussion: Our patient was treated for ITP and concomitant AH with prednisolone, although a definitive diagnosis could not be made. His bilirubin and platelets improved with this treatment at a dosing of 1 mg/kg, which is the regimen for ITP, albeit at one-half of the suggested duration. Due to the complexity of his presentation, determining the optimal management for this patient was difficult. It is important for clinicians to remember the broad differentials of thrombocytopenia and recognize that there may be overlapping etiologies of deranged liver enzymes.

S3046

Hepatitis B Causing Severe Hemolysis and Multi-Organ Failure in Patients With Undiagnosed G6PD

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Introduction: Hepatitis B is a viral infection that attacks the liver and can cause both acute and chronic disease. WHO estimates 1.5 million new infections annually. Most people remain asymptomatic; however, some people have acute illness that lasts several weeks, which can at times be complicated with acute liver failure. Glucose-6-phosphate dehydrogenase (G6PD) X-linked deficiency is the most common human enzymopathy.
Case Description/Methods: A 73-year-old male presented with loss of appetite, and dark colored urine. On examination, vitals were within normal range. Scleral icterus was present, along with hepatomegaly. Patient was alert and oriented. Patient's lab work was concerning for AST was 3017 unit/L, ALT 2712 Unit/L, BUN 74 mg/dL, Cr 3.6 mg/dL, total bilirubin 72.4 mg/dL, direct bilirubin 41.8 mg/dL, indirect bilirubin 30.6 mg/dL, hemoglobin 12.9 g/dL, white count 17,000 per microliter and platelet count of 173,000 per microliter (mcL). Patient's hepatitis panel was positive for Hepatitis B Core Antibody Total, Hepatitis B Core Antibody IgM, Hepatitis B Surface Antibody, log 10 HBV IU/mL 2914. Hematological work up was done which was concerning for LDH 4,875, ferritin > 7500, reticulocyte, Peripheral smear did not show schistocytes. Erythrocyte G6PD level was performed which was low Patient was started on hemodialysis due to electrolyte derangement and oliguria. The patient was given intravenous fluids and was started on Entecavir for acute complicated Hepatitis B infection, Renal Biopsy was done which was suggestive of acute tubular injury. Patient liver function, labs as well as renal function continued to improve and no longer required dialysis.
Discussion: Hemolytic anemia has been associated with viral hepatitis, but the degree is usually mild to moderate. With cases of severe intravascular hemolysis, a diagnosis in addition to hepatitis should be sought out. Our case is unique since this is the first reported case of severe hemolysis and renal failure precipitated by acute HBV in an undiagnosed G6PD deficient patient and treatment with Entecavir causing marked improvement. With early recognition and establishing diagnosis of complicated hepatitis B leading to severe hemolysis as well as renal failure, prompt treatment with anti-viral as well as supportive treatment for G6PD deficiency can help with early recovery of patients and prevent life threatening organ failure.

S3047

Hepatic Amyloidosis With Multi-Organ Involvement

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Introduction: Amyloidosis, particularly with cardiac involvement, carries a significant risk for mortality that has increased from 1.77 to 3.96 per million between 1979 and 2015. Incidence is thought to be 3-5 cases per million for AL amyloid, with hepatic involvement in 9% of those. Amyloidosis most commonly affects older persons, with a mean age of 63, males and has had higher mortality in African Americans.
Case Description/Methods: A 55-year-old male presented for evaluation of worsening shortness of breath over 2-3 weeks associated with abdominal, genital and lower extremity edema. History included CLL, COPD, polysubstance abuse, CAD s/p MI, HTN, T2DM and DVT s/p IVC filter.Physical exam showed extensive bilateral pitting edema and ascites. CBC showed WBC 14.2 with lymphocytic predominance, Hgb 14.5 and platelets 450 (peaked at 507). CMP was significant for Cr 2.1, albumin 1.5, total bilirubin 1.5(predominantly direct), ALP 2229, AST 73, ALT 27, INR 0.9 and BNP 2381. CT revealed large volume ascites, anasarca and hepatomegaly. Abdominal US showed hepatic steatosis, patent vasculature, low portal vein velocities 12-17 cm/s indicative of portal hypertension and splenomegaly. Paracentesis was performed, which revealed SAAG 1.2, protein < 0.5 and < 250 PMNs. SPEP, UPEP, FLC assay, HIV, viral hepatitis testing and PLA2R Ab were unremarkable. Renal biopsy performed due to proteinuria >10g/day was positive for AL(light chain) amyloid. Ursodeoxycholic acid was started for cholestasis. Liver biopsy was not performed. Encephalopathy and severe epistaxis developed. Final chemistries were Cr 10.8, ALP 2613, AST 206, ALT 82, total bilirubin 7.6, and INR 2.1. He ultimately transitioned to comfort measures and was discharged to hospice. He expired with 1 month of discharge. (Figure)

Discussion: Our patient presented with the majority of complications seen in systemic amyloidosis with liver involvement, including abnormal liver chemistries, portal hypertension, hepatic failure, nephrotic syndrome, and congestive heart failure. Liver biopsy was not performed due to coagulopathy and risk of liver fracture. Even so, clinical, lab and imaging findings and positive kidney biopsy for Al amyloid strongly support hepatic amyloidosis. One case series following 98 patients with hepatic amyloidosis found a median survival of 8.5 months with worse outcomes seen in those with heart failure, elevated bilirubin and platelets >500. Our patient had all these features, contributing to his rapid decline.

S3048

Hyperacute Liver Failure Resulting From COVID-19 Infection

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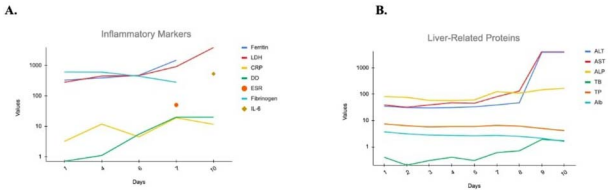
Introduction: Acute liver failure (ALF), characterized by acute liver injury, hepatic encephalopathy, and an increased international normalized ratio, is subcategorized based on the timeline as hyperacute (< 7 days) and acute (7 to 21 days), where cerebral edema is typical, or subacute (>21 days and < 26 weeks).
Case Description/Methods: A 54-year-old female with a history of diabetes complained of fevers, chills, headache, chest pain, abdominal pain, nausea, vomiting, diarrhea, body aches, and back pain for three days. Two days prior, the patient had tested positive for COVID-19. On admission, the patient did not have any respiratory compromise. Initial biochemical tests were unremarkable, except for elevated inflammatory markers and imaging suggestive of atypical pneumonia. After the third day in the hospital, the patient began developing worsening respiratory status and was transferred to critical care; the following day, the patient was intubated for acute respiratory failure. On day eight of admission, the patient began having elevated liver-related proteins and worsening inflammatory markers (Figures 1). Two days later, the patient passed despite aggressive therapeutic measures on day ten after admission.
Discussion: In COVID-19, ALF may result from the virus invasion, which directly infects cells via angiotensin-converting enzyme receptor-2 present in the liver cells, including cholangiocytes (60%) and hepatocytes (3%); and are absent in Kupffer cells, where direct viral impact, systemic inflammation, drug-induced damage, congestion abnormalities, and hypoxia-induced damage contribute to liver damage. In a cytokine storm, an acute hyperinflammatory response is responsible for critical illness in many conditions, including viral infections, cancer, sepsis, and multi-organ failure. However, the exact mechanisms of COVID-19 in the induction of ALF have not been identified. Empiric therapy is often started with the diagnostic workup when hyperacute or ALF is suspected, consisting of N-acetylcysteine (NAC). However, management is directed based on the specific clinical requirements of each patient; NAC, anticoagulants, monoclonal antibodies, plasmapheresis, and symptomatic treatment may be used concomitantly to improve outcomes. Cases of hyperacute liver failure itself are a rare disorder. Therefore, when hyperacute or ALF is suspected in COVID-19 disease, early recognition, and prompt action are required to improve patient survival.



[3048] **Figure 1.** A: Inflammatory markers trending from day one of admission to day ten. [Ferritin (blue line), Lactose Dehydrogenase (LDH; red line), C-reactive protein (CRP; yellow line), D-Dimers (DD; green line), Erythrocyte sedimentation rate (ESR; orange dot), Fibrinogen (turquoise line), Interleukin-6 (IL-6; gold diamond)]. B: Liver-related proteins trending from day one of admission to day ten. [Alanine aminotransferase (ALT; blue line), Aspartate aminotransferase (AST; red line), Alkaline phosphatase (ALP; yellow line), Total bilirubin (TB; green line), Total protein (TP; orange line), Albumin (Alb; turquoise line)].

S3049

It's Not Always Hepatorenal Syndrome

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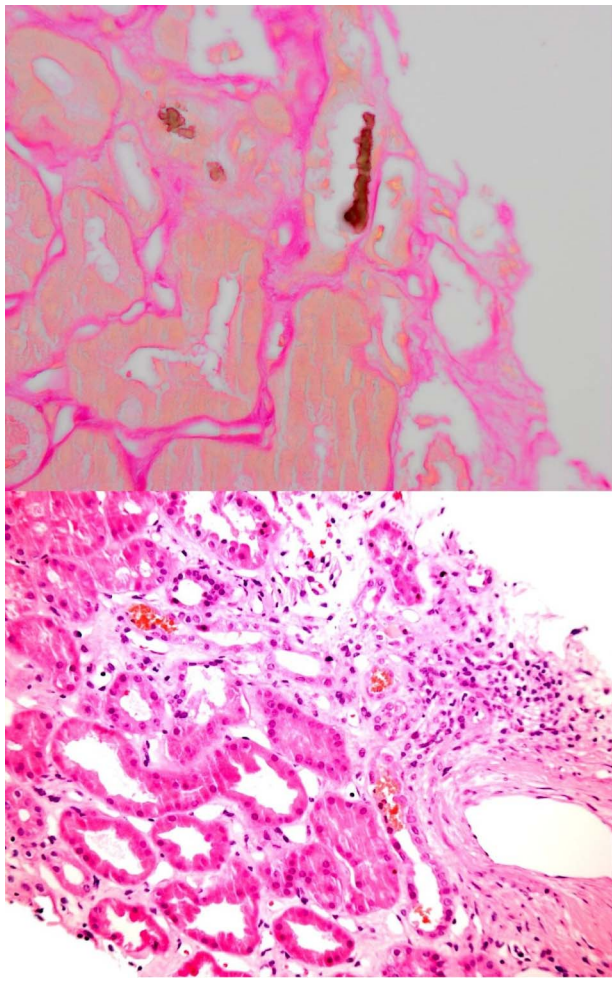
Introduction: Bile cast nephropathy (BCN) or Cholemic Nephrosis (CN) is a form of acute renal dysfunction that happens in the background of liver dysfunction and hyperbilirubinemia. We report an interesting case of BCN, in a patient who developed Acute Kidney Injury in the setting of hyperbilirubinemia due to Hepatitis A.
Case Description/Methods: A 58-year-old female presented with 4 days history of intractable nausea and vomiting. There was no associated abdominal pain or fever however she noted yellow discoloration of her skin and eyes. Her past medical history included Diabetes Mellitus Type 2, Hypothyroidism, Hypertension and Hyperlipidemia. Patient traveled to Arizona 2 months prior but denied any sick contacts, insect bites, herbal supplements, antibiotic use, drug use, alcohol/tobacco use or tattooing. On physical exam she was noted to be hemodynamically stable and afebrile with significant scleral icterus and jaundice. Abdomen was nontender without any organomegaly. Blood work showed Total bilirubin 7.9, Direct bilirubin 6.1, ALT 4792, AST 5228, ALK 313, Cr 2.8, BUN 32 and eGFR 19, INR 2.35. Elevated urobilinogen on UA. Imaging showed hepatic steatosis. Hepatitis-A IgM was positive. Management included supportive therapy. In the following days total Bilirubin levels reached above 20, Creatine >8 and GFR < 10. Patient was initiated on hemodialysis with significant improvement in her symptoms. Renal biopsy showed pigmented casts, consistent with BCN or CN. On the day of discharge, AST levels were 63, ALT was 76, alkaline phosphatase of 189 although total bilirubin was elevated at 20.5. Outpatient hemodialysis was arranged and patient was discharged home. (Figure)
Discussion: Jaundice related nephropathy can lead to renal failure which is referred to as CN. Although the pathophysiology of CN is unclear, studies have shown tubular injury in mice from excretion of toxic bile acids in urine. Severe bilirubin elevations can cause acute renal injury, exacerbated by obstructive nephropathy secondary to bile cast formation. Such patients should be investigated for the possibility of CN and a renal biopsy is indicated. Therapy involves reducing bilirubin levels, but patients commonly require hemodialysis. By presenting this case, we encourage physicians to keep a broad differential in cases with hyperbillirubenemia and acute kidney injury. We found two cases of BCN in patients with acute Hepatitis A in our literature review. To our knowledge, there has not been a specific association between these entities.



[3049] **Figure 1.** Picture above: Positive Fouchet Test Picture below: Tubular Injury and Pigmented Casts.

S3050

Intestinal Failure-Associated Liver Disease vs Non Alcoholic Fatty Liver Disease in the Setting of Short Bowel Syndrome: A Case of Rapidly Progressive Hepatic Failure

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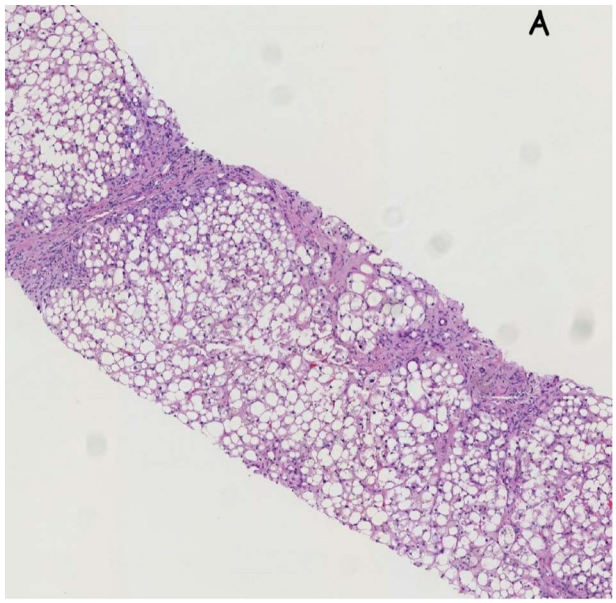
Introduction: Intestinal Failure-Associated Liver Disease (IFALD) is a progressive disease with a high mortality rate in patients dependent on parenteral nutrition (PN). It is a multifactorial entity associated with a spectrum of hepatic manifestations including cholestasis, steatosis, portal hypertension, choline deficiency and manganese toxicity. In patients with Short Bowel Syndrome (SBS), hepatic steatosis occurs in 40-55% and IFALD in 5-15%. Risk factors for IFALD in SBS include chronic PN use and length of remaining bowel. Early IFALD may present similarly to NAFLD, however pathogenic and prognostic differences make distinguishing these diseases crucial. We present a case of rapidly progressive IFALD in an adult SBS patient after PN use.
Case Description/Methods: We present a 65 year-old female with massive short bowel resection (< 30cm remaining) and malnutrition with a 12-year history of on-and-off PN use. PN was discontinued 6 months prior and she was gaining weight with enteral feeding and teduglutide. She presented with right upper quadrant pain and nausea. Ultrasound showed cholelithiasis and hepatic steatosis. She was discharged after resolution of symptoms. In two weeks, she returned with jaundice, worsening abdominal pain, weight loss, altered mentation and asterixis. Labs showed total bilirubin 11.4 mg/d L (Direct 6.0mg/d L), ALP 139 IU/L, AST 136 IU/L, ALT 65 IU/L, NH3 182 mg/L and serum carnitine 15 μmol/L. She was treated for hepatic encephalopathy with lactulose, rifaximin and carnitine. Computed tomography showed moderate ascites, mesenteric edema, and edematous bowels. Diagnostic paracentesis revealed portal hypertension. Liver biopsy showed cirrhosis with steatohepatitis and peri-cellular fibrosis consistent with TPN-associated liver disease in the setting of SBS-IF(Image A). Multifocal pneumonia with multi-organ failure led to her death. (Figure)
Discussion: This case demonstrates the potentially rapid progression of IFALD, particularly in patients with SBS. Clinicians should exercise high clinical suspicion of IFALD in patients with a history of PN use and SBS that present with hepatic manifestations. Early recognition is important to distinguish the disease from similarly presenting NAFLD (table 1). It is also vital to consider and treat other factors that may exacerbate hepatic disease including nutritional deficiencies. There may be benefit to diagnosis with liver biopsy early in the disease course to initiate prompt treatment or transplant, preventing rapid and fatal progression.



[3050] **Figure 1.** Liver biopsy showing cirrhosis with marked steatohepatitis, mild lobular inflammation, ballooning hepatocyte degeneration nodular and peri-cellular fibrosis stage IV

Table 1. Differentiating features between IFALD vs NAFLD

FEATURES	IFALD	NAFLD
Metabolic syndrome	No metabolic syndrome ,no insulin resistance, or it is improved after initiating PN, low BMI and low plasma cholesterol levels	Metabolic syndrome common with Insulin resistance, hyperlipidemia high BMI
Nutritional status and PN dependence	Severe malabsorption, mostly dependent on PN	No malabsorption, PN mostly not required
Plasma choline levels	Low plasma free choline concentration	Normal to high levels
Effect of choline supplementation	Reduction of steatosis ,improved liver tests with choline supplementation	Minimal difference to choline supplementation
Cholestasis	Highly evident with hyperbilirubinemia	Not typical
Steatosis (macro vs micro)	Macro and micro steatosis	Predominantly macro steatosis
Disease progression and cirrhosis development	Rapid progression to ESLD, cirrhosis develops within ~3-5 months after initiating PN	Longer duration ~10-20 years for cirrhosis to develop
Zone of steatosis	More common in zone -1 (periportal)	Mostly involves zone -1(peri-central)
Pattern of fibrosis	Characteristic "jig-saw" pattern fibrosis	Sinusoidal fibrosis with ballooning of hepatocytes
Treatment	Intestinal transplant is mainstay of treatment to overcome malabsorption and impending liver failure	No role of intestinal transplant as no malabsorption.
Prognosis	Rapid onset of death within 1-4 years	Rapid death extremely rare

LIVER

S3051

Invasive Liver Abscess Syndrome Leading to Klebsiella Endophthalmitis - A Possibly Reversible Debilitating Disease?

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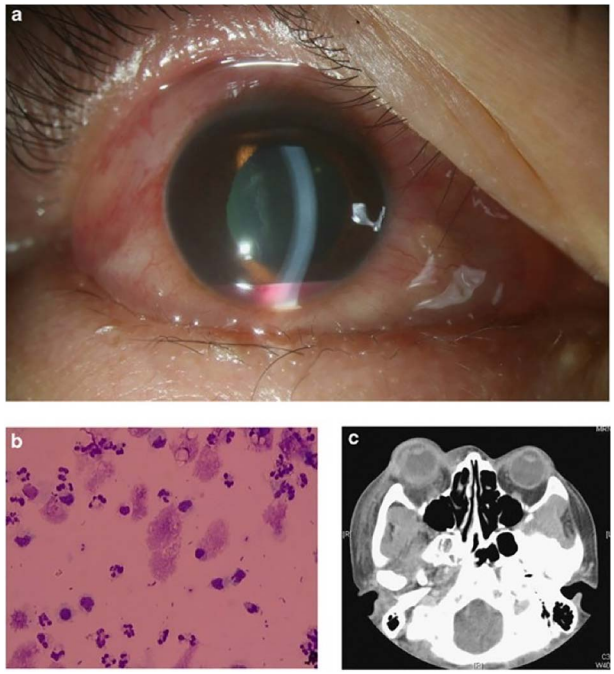
Introduction: Invasive liver abscess syndrome is a rare metastatic infection associated with primary *Klebsiella pneumoniae* liver abscesses typically seen in Asian countries. Disease progression often results in *Klebsiella pneumoniae* endogenous endophthalmitis (KPEE). Reported risk factors for KPEE include liver abscesses, diabetes mellitus, and systemic immunocompromise. Limited data exists among Hispanics through a small number of isolated case reports. We present a case of a Hispanic female diagnosed with KPEE who developed blindness, which later resolved with prompt intravitreal antibiotics, highlighting a possible treatment for an otherwise visually devastating disease.
Case Description/Methods: A 75-year-old Hispanic female with history of diabetes mellitus presented to the emergency department with weakness and flank pain. Computed tomography showed a multicystic/septated peripherally enhancing lesion in the hepatic dome near the inferior vena cava measuring 3.2 cm. Blood cultures grew *Klebsiella pneumoniae* on hospital day two likely secondary to the liver abscess. IR-guided drainage was not possible due to proximity of the Inferior Vena Cava (IVC) thus she was treated with Ceftriaxone and Metronidazole. Despite this, she developed complete vision loss in the right eye and hypopyon on hospital day three. Ophthalmology was consulted and recommended emergent transfer for intravitreal antibiotics. The patient was transferred and treated within 12 hours and upon discharge already had partial recovery of vision. (Figure)
Discussion: While our patient had a suspected *Klebsiella* liver abscess, drainage of the abscess was not possible due to its proximity to the IVC resulting in endophthalmitis and right eye blindness. Invasive liver abscess syndrome with KPEE is a rare and devastating disease with significant morbidity and mortality. Even with aggressive treatment within 24 hours, vision loss is typically permanent. However, our patient did have some restoration of vision and we propose that it was due to prompt administration of intravitreal antibiotics within 12 hours. Greater awareness of this devastating complication will result in more cases which will hopefully help to establish further guidelines concerning management and improved outcomes. It will also hopefully lead to faster treatment of patients with KPEE and potentially if intravitreal antibiotics can be administered within twelve hours, the medical community may see a higher probability of vision restoration.



[3051] **Figure 1.** Slit-lamp examination of the right eye - hypopyon with conjunctival injection and chemosis. (b) Photomicrograph of the anterior chamber aspirate shows numerous neutrophils and bacilli. (c) Orbital CT revealed panophthalmitis and right orbital cellulitis with preseptal and retroorbital involvement.

S3052

Iron Man: A Case of Non-HFE Hemochromatosis Without Significant Fibrosis

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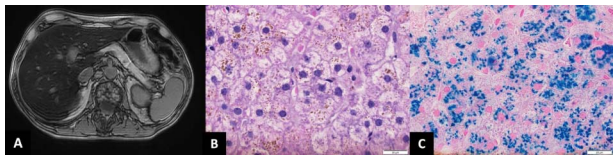
Introduction: Hereditary hemochromatosis (HH) is a rare disorder with abnormally high levels of intestinal iron absorption leading to end organ damage. It is classically associated with HFE gene mutation although there are isolated case reports of non-HFE hemochromatosis. We present a case of an elderly man evaluated for elevated ferritin found to have non-HFE hemochromatosis.
Case Description/Methods: A 74-year-old Japanese man with a medical history of colon cancer status post hemicolectomy was evaluated for ferritin above 1500 mg/dL. He was asymptomatic and laboratory studies revealed hemoglobin 10.2 g/dL, iron 239 mcg/dL, iron saturation >90%, transferrin 195 mg/dL and normal liver enzymes. Given the elevated ferritin and iron saturation, there was a concern for iron overload. Genetic testing for HFE mutation was negative. Secondary causes of iron overload such as thalassemia, or prior history of transfusions were ruled out. Magnetic resonance imaging (MRI) of the abdomen revealed iron deposition with hypointense liver on T2-weighted imaging and normal appearing spleen consistent with primary hemochromatosis (Figure 1a). Given elevated ferritin, a liver biopsy was performed to assess for fibrosis which revealed coarse iron deposits corresponding to hemosiderin, without fibrosis (Figure 1b-c). The hepatic iron index was 4.3 and hepatic iron concentration by weight was 1770 ug/g further suggesting a diagnosis of non-HFE hemochromatosis. Hematology was consulted and the patient underwent phlebotomy treatments. The patient developed symptomatic anemia therefore phlebotomy was stopped and deferasirox was initiated. He remains under close monitoring. Results of further non-HFE gene testing are pending.
Discussion: HFE gene modulates the expression of hepcidin, an iron-regulating hormone in the liver controlling the delivery of iron into the circulation. Primary mutations in HFE gene are the most recognized genetic disorders however, other rare non-HFE genes such as hemojuvelin (HJV), hepcidin (HAMP), transferrin receptor (TFR2) and ferroportin (SLC40A1) can also be involved. The diagnosis typically involves excluding secondary causes of iron overload, assessing degree of iron stores and hepatic fibrosis. It is interesting to note that our patient had no fibrosis despite the long duration of disease and degree of ferritin elevation. This case emphasizes that elevated ferritin levels should be evaluated diligently and early recognition can lead to a prompt diagnosis and initiation of life-saving treatment.



[3052] **Figure 1.** A. MRI of the abdomen showing iron deposition with hypointense liver on T2-weighted imaging and normal appearing spleen. B. H&E stain showing coarse iron deposits corresponding to hemosiderin. C. Prussian blue iron stain demonstrating blue granules of hemosiderin.

S3053

Isolated Pinworm in Ascites

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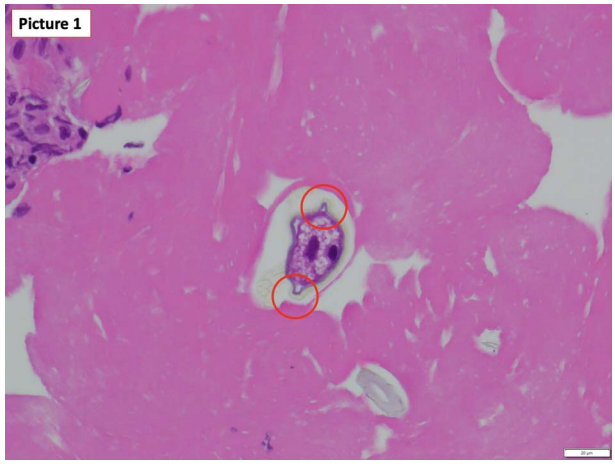
Introduction: Extra-intestinal Enterobius vermicularis infections (pinworm) are rare with sites including liver, kidney, spleen, and lung. In females, urinary tract infections and invasion of the genital tract have been described. There have been some case reports describing parasitic infections presenting as eosinophilic ascites in otherwise healthy patients. We present a case in which a cirrhotic patient presented with hepatic encephalopathy (HE) and was found to have a Enterobius vermicularis infection of ascitic fluid.
Case Description/Methods: Patient was a 67-year-old female with decompensated cirrhosis from non-alcoholic steatohepatitis (NASH) with ascites, varices, and a history of HE and chronic kidney disease who presented to an outside hospital (OSH) for concerns of HE. The patient had titrated her lactulose from three times to two times daily about one month prior. In addition, diuretics had been held for the past few months due to acute kidney injury (AKI). Abdominal radiography was unremarkable. Laboratory workup at the OSH was unremarkable. A paracentesis was attempted but couldn't be completed due to the patient's mentation. As she was an established patient, she was transferred to our hospital from the OSH for further management. She initially received lactulose enemas with which her mentation improved. She was eventually placed back on lactulose three times daily, diuretics, and prophylactic antibiotics. A diagnostic paracentesis was performed; fluid studies are shown in Table 1. Cytopathology from paracentesis fluid studies identified Enterobius vermicularis on a cell block section (Figure 1). She developed itching around the umbilical area shortly after her diagnostic paracentesis. She was seen by the infectious diseases team and was prescribed albendazole for a duration of 4 weeks, followed by paracentesis which confirmed eradication.
Discussion: Peritoneal cavity pinworm contamination has been noted presenting as chronic pelvic peritonitis due to enterobius granulomas, however pinworm isolation in ascites is rare. The source in this case is unknown, however, could include translocation from the gut or migration from the genitourinary tract. Treatment with albendazole 400 mg weekly for 4 weeks was effective in eradication of infection.



[3053] **Figure 1.** Pinworm is pictured with characteristic lateral spines in ascites.

Table 1. Ascites fluid studies

RBC	1,662/mm^3
Nucleated	287/mm^3
Neutrophils	4%
Lymph	54%
Macrophage	42%
Glucose	143 mg/dL
Albumin	1.2 g/dL

S3054

Interdisciplinary Management of a Complicated Pregnancy in a Patient With Cirrhosis

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Introduction: We present a unique case of clotting disorder related cirrhosis leading to pregnancy complications.
Case Description/Methods: A 29-year-old Caucasian female with a history of gastrointestinal bleeding, gastroesophageal reflux disease, epistaxis, and alcohol use disorder, goes for a routine gynecologic checkup when the physician notices a concerning enlargement of the caudate lobe on a computed tomography scan. Further evaluation reveals cirrhosis and Budd-Chiari syndrome. The patient starts enoxaparin and warfarin following this diagnosis. Eventually the patient is placed on the liver transplant list with a MELD (model of end-stage liver disease) score of 17 which improves to a score of 7 with sobriety. The patient attempts pregnancy and is unable to conceive. She is told she has early menopause at the age of 34. She undergoes reproductive endocrinology evaluation and achieves a successful in-vitro fertilization with a donor egg at age 36. This starts her high-risk pregnancy. The high-risk nature of this pregnancy revolves around the patient's complicated past medical history of hemorrhagic stroke of unclear etiology, cirrhosis complicated by esophageal variceal bleeding, coagulopathy, Budd-Chiari syndrome, and thrombocytopenia. The patient has close variceal and hepatic vein thrombosis surveillance, labs, and routine

checkup for vaginal bleeding with her increased risk of thrombosis during pregnancy. Endoscopy in the 1 st and 3rd trimesters shows small esophageal varices with no evidence of progression. Abdominal ultrasound displays vasa previa at approximately 28 weeks with velamentous umbilical cord insertion 2.3 cm from the internal os. After careful monitoring, the vasa previa is noted to resolve with umbilical cord insertion increasing to 4.8 cm from internal os in the following weeks. Repeated fetal echocardiograms present concern for the fetus being large for gestational age with estimated fetal weights ranging between the 95th-98th percentiles. The patient undergoes elective cesarean delivery and gives birth to a baby boy at 34 weeks with no labor complications.
Discussion: We present a unique case of pregnancy during cirrhosis requiring management with multiple specialists. Although the physiologic changes in pregnancy are well understood, the way in which cirrhosis affects pregnancy and its outcomes are not yet well understood given paucity of published data. Certainly, there is complex interplay and physiology involved with increased potential for pregnancy complications.

S3055

Late Manifestation of Heterozygous H63D Mutation as Hereditary Hemochromatosis in 89-Year-Old Man

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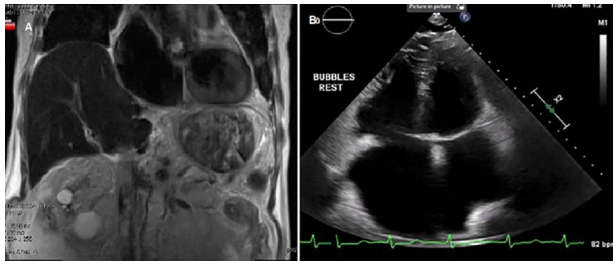
Introduction: Hereditary hemochromatosis (HH) is an autosomal recessive that usually affects middle-aged people [1]. In most patients, homozygous C282Y mutations in the HFE gene express the disease. But in some occurrences, compound heterozygous, including C282Y and H63D mutations, were also witnessed. We present an atypical case where heterozygous H63D manifested as HH in an 89-year-old man.
Case Description/Methods: An 89-year-old Caucasian man of Italian descent presented with confusion, left-sided facial droop, and slurring of speech. Past medical history included end-stage renal disease on hemodialysis and atrial fibrillation. For the past few months before presentation, the patient has been complaining of increasingly tired with intermittent episodes of slurring speech, mainly after he receives dialysis. During the initial physical examination, the patient had confusion but was alert & oriented to self and time. There was mild abdominal distension and positive for asterixis, and the skin was bronze in color. Initial labs showed in table 1. The patient had persistent hypoglycemia and required intravenous glucose administration. Brain imaging was normal. CT scan of the abdomen, pelvis, and chest showed severe cardiomegaly and a moderate amount of ascites. Iron studies were abnormal. Ascites fluid analysis was consistent with cirrhosis. Although, an ultrasound right upper quadrant was negative for any cirrhotic morphology. MRI showed hemosiderosis. 2D Echo showed a severely dilated right ventricle. The patient empirically underwent two sessions of phlebotomy. Genetic testing confirmed heterozygous H63D mutation. The patient followed up for the phlebotomy sessions and demonstrated significant improvement.
Discussion: This case highlights the consideration of HH in differential regardless of age if a patient presents with symptoms suspected of HH. Standard genetic testing includes testing C282Y and H63D mutations in the HFE gene on chromosome 6. Less frequently tested S65C, HFE4, TFR2-HHC, and FTH1 gene mutations also have the expression of HH in sporadic cases [2-3]. Previously, heterozygous C282Y carriers with iron overload are assumed to have other genetic changes or influence by environmental factors, such as alcohol or liver disease, that increase disease expression [2]. However, no such literature is available for heterozygous H63D carriers. Given the unusual age for phenotype expression and atypical disease, the presentation makes this unique case for learning the pathophysiology and genetics of HH.



[3055] **Figure 1.** A: MRI of liver showing decreased signal of the liver and spleen on in phase imaging is likely due to hemosiderosis. B: Two-dimensional transthoracic echocardiography showing severely dilated right ventricle and both atria. Moderate pulmonary hypertension was also noticed.

Table 1.

	Day 1	Day 2	Day 3	Day 4	Day 15	Day 42
Hemoglobin (g/dL)	15.6	14.8	15.1	13.8	13.6	12.2
AST (U/L)	85	102	135	125	109	71
ALT (U/L)	56	57	70	74	72	46
Bilirubin (mg/dL)	3.0	2.9	2.6	2.9	2.6	2.1
INR	1.3	1.4	1.4	1.4	1.2	1.2
Ammonia (μmol/L)		99	45	46	42	31
Platelets (103/μL)	103	86	82	63	59	91
Ferritin (ng/ml)		1283				725
Iron (μg/dL)		187				130
Transferrin Saturation (%)		94				74
Total Iron-binding Capacity (μg/dL)		200				276

S3056

Immune-Mediated Hepatitis: A Single Drug or a Class Effect?

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Introduction: While the rates of diagnosis and death due to non small cell lung cancer are decreasing, the five year relative survival rate remains 20%. Treatment of lung cancer is rapidly evolving, particularly in the metastatic setting. Immunotherapies, most notably PD-1 inhibitors, have drastically changed the landscape of treatment. Immune Hepatitis can, unfortunately, be a reason for discontinuation of the drugs. Immune Hepatitis has been generally thought to be associated with all drugs of a particular class, not specific drug agents.
Case Description/Methods: We present a case of an 84 year old female with metastatic non small cell lung cancer with a PD-1 expression of 100%. She was started on single agent Pembrolizumab due to inability to tolerate chemotherapy limiting therapeutic options. Shortly after starting therapy she developed immune hepatitis requiring steroid therapy and withdrawal of the drug with multiple drug interruptions. Ultimately, the drug was discontinued due to worsening elevation in her liver enzymes with resumption of the drug. Standard of practice in such cases is to avoid all drugs in the PD-1 inhibitor family due to similar toxicity profiles and mechanism of action observed. Given the fact that she did not have any other treatment options, she was started on Nivolumab, a different PD-1 inhibitor. Patient was able to tolerate Nivolumab without elevation of her liver enzymes or development of immune hepatitis. She has been on the drug for approximately 10 months with serial monitoring and no evidence of liver injury has been observed.

Discussion: While PD-1 inhibitors have revolutionized the treatment of metastatic NSCLC and the drugs are overall well tolerated, a small percentage of patients develop immune mediated liver injury due to reactive cytotoxic T lymphocytes as the reactivated T cells attack other tissues, including the liver. Generally, this has been seen consistently across the entire drug class, not specific to single agents. We present a rare case of immune mediated liver toxicity specific to Pembrolizumab that was subsequently not observed with Nivolumab, despite both drugs having identical mechanisms of actions. Patient has had multiple serial labs and imaging studies showing no progression of her disease, stable on Nivolumab. This case highlights the need for further investigation regarding the mechanism of Immune mediated liver toxicity/injury that may be specific to single agents and not necessarily a drug class.

S3057

Iron Overload in an H63D Homozygote: Looking Beyond the Genotype

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Introduction: Hereditary hemochromatosis (HH) is a genetic disorder in which excessive gastrointestinal absorption of iron results in iron-mediated damage to the liver and other organs. HH is inherited in an autosomal recessive fashion and is characterized by mutations in the HFE gene; however, due to low penetrance, many patients with two abnormal copies of HFE do not exhibit clinical iron overload. The most common pathologic mutations of HFE are C282Y and H63D. Over 90% of HH cases are due to homozygosity for C282Y, with the remainder attributed to C282Y/H63D compound heterozygosity, H63D homozygosity, or S65C mutation. We discuss a rare case of newly diagnosed HH in a patient with H63D homozygosity and family history of iron overload.
Case Description/Methods: A healthy 67-year-old Caucasian male with history of hyperlipidemia presented for abnormal iron studies which were checked after he reported that his father used to require phlebotomy. He drinks two alcoholic beverages daily and denies any clinical complaints. Laboratory tests were notable for serum ferritin of 1240 ng/mL, transferrin saturation of 45.6%, hemoglobin 15.5 g/dL, ALT 91 units/L, and AST 43 units/L. Genetic testing revealed homozygosity for H63D. Due to his markedly elevated ferritin, liver biopsy was performed and showed diffusely increased iron deposition, predominantly in hepatocytes. Moderate steatosis and portal fibrosis (fibrosis stage 1/4) were also seen. He was diagnosed with HH and is scheduled for phlebotomy with goal ferritin less than 50 ng/mL.
Discussion: Patients homozygous for H63D make up fewer than 10% of all HH cases and clinically significant iron overload is far less common. The likelihood of iron overload for a particular genotype is higher if a first-degree relative had iron overload which may explain our patient's phenotype. Although our patient did not have cirrhosis on biopsy, his HH in addition to hepatic steatosis puts him at higher risk of liver disease. This risk can be mitigated with control of his ferritin via phlebotomy and modification of other risk factors. Our case highlights the importance of evaluating for HH and hepatic fibrosis in patients with family history, irrespective of genotype, if they present with phenotypic signs of iron overload.

S3058

Kratom Induced Acute Liver Injury: A Case Study and Systematic Review of Liver Injury Patterns Due to Kratom

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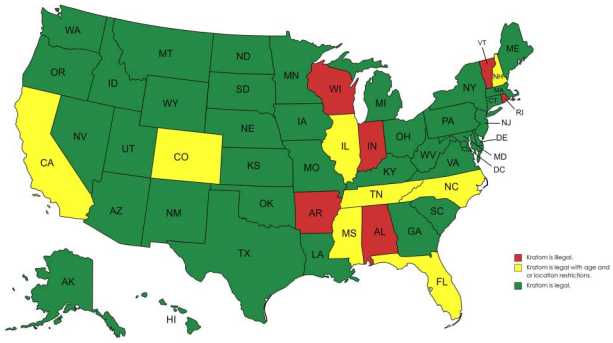
Introduction: Kratom is an herbal supplement derived from the Mitragyna speciosa tree leaves in Southeast Asia. It is currently banned in only 6 states, regulated in 8, and completely unregulated in the rest (Figure 1). Kratom is popular for its psychotropic and opioid-like activity. In addition to other side effects, it is associated with acute liver injury and in rare cases, acute liver failure. Here, we report a patient who presented with cholestatic liver injury after Kratom use. We compare this case to 53 other reports of Kratom-associated hepatotoxicity to better understand the safety of this drug.
Case Description/Methods: A 47-year-old male with a history of peripheral neuropathy and hypertension presented with 15 lbs of unintentional weight loss over a month and jaundice for 5 days. For at least three weeks, he had taken Kratom for neuropathy and hip pain. Labs were notable for cholestatic injury with an R factor of 1.8 and a urine drug screen positive for tetrahydrocannabinol and benzodiazepines (Table 1). Other workups including HIV, acetaminophen, and hepatitis labs were negative. He improved with supportive care and was advised to discontinue Kratom. After discharge, the patient reported complete resolution of his symptoms. A literature search in PubMed, Cochrane, and Embase found 69 cases of Kratom-induced DILI. We excluded cases without laboratory values, leaving 53 cases. A majority of Kratom users report taking the drug for acute or chronic pain. Males were predominant (64%) and the majority had a cholestatic liver injury pattern (80%). Unfortunately, 5.6% of these cases resulted in liver transplants. Furthermore, 9.4% of these cases experienced acute renal injury, with 60% requiring hemodialysis. Some patients also experienced rhabdomyolysis (3.7%), reversible heart failure (3.7%), acute cholecystitis (3.7%), and undifferentiated shock (1.9%). One patient suffered from Salmonella-contaminated Kratom ingestion. (Figure)
Discussion: Kratom is widely available throughout the United States with very minimal regulation. After a review of our patient's case report, as well as those of 53 other patients, we have significant safety concerns for the use of Kratom in the general population. Caution is advised in patients with pre-existing liver disease. Though its chemical properties may have interesting applications worthy of investigation, we recommend further research on the risks and mechanism of liver injury of Kratom.



[3058] **Figure 1.** A map of the United States and locations of Kratom restrictions as of 2022. California: Kratom is legal, except in San Diego and Oceanside; Colorado: Kratom is legal, except in the towns of Monument and Parker; Florida: Kratom is legal, except in Sarasota country; Illinois: Kratom is legal for adults 18 years and older, except in Jerseyville and Alton; Mississippi: Kratom is legal, except in several counties; New Hampshire: Kratom is legal, except in Franklin city; North Carolina: Kratom is legal for adults 18 years and older; Tennesee: Kratom is legal for adults 21 and older.

Table 1. Laboratory test results

Variable (normal range)	Day of admission	Day of discharge
Bilirubin, Total mg/dL (0.0-1.2)	**10.7**	**5.0**
Bilirubin, Direct mg/dL (< = 0.5)	**6.7**	
AST U/L (8-34)	**64**	**53**
ALT U/L (10-49)	**168**	**87**
Alk-Phos U/L (46-116)	**265**	**279**
GGT U/L (12-64)	**376**	
INR (0.80-1.2)	1.04	

Table 1. (*continued*)

Variable (normal range)	Day of admission	Day of discharge
Prothrombin time (9.3-12.4 seconds)	10.9	
Lipase U/L (< 78)	**36**	
CA 19-9 U/mL	< 5.30	
Acetaminophen ug/mL (0.0-10.0)	5.6	
Ferritin ng/mL (11.0-307.0)	**410.2**	
Iron ug/dL (65-175)	140	
Ceruloplasmin mg/dL (16.0-31.0)	**31.3**	
Copper ug/dL (72-166)	120	
Antinuclear Antibody	Negative	
Smooth Muscle Antibody U (0-19)	**20**	
Anti-Mitochondrial Antibody units (0.0-20.0)	< 20.0	
Alpha-1 Antitrypsin mg/dL (90.0-200.0)	186.0	

AST, aspartate transaminase; ALT, alanine transaminase; Alk-Phos, alkaline phosphatase; INR, International Normalization Ratio; aPTT, activated partial thromboplastin; GGT, Gamma-Glutamyl Transferase.

S3059

Into the Unknown: A Curious Case of Hyperammonemia

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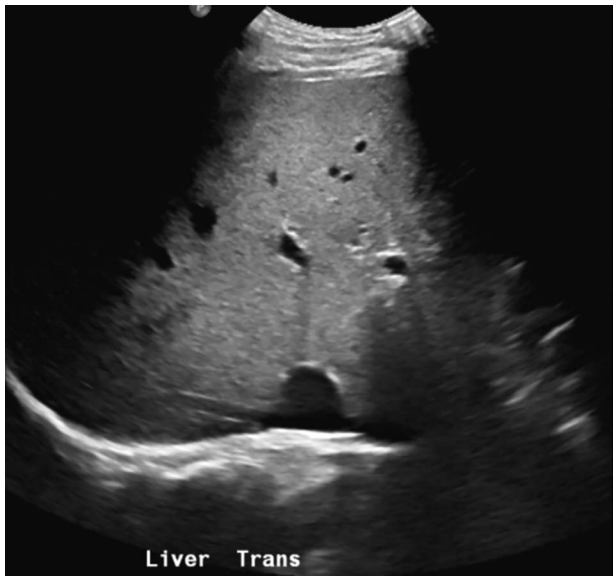
Introduction: Hyperammonemia is a metabolic disorder characterized by high levels of ammonia. Around 90% of hyperammonemia cases are due to cirrhotic liver disease. Less common causes are congenital or acquired disorders involving enzymatic defects in the urea cycle, organic acidemias, fatty acid oxidation and amino acid deficiencies. Adult onset non-cirrhotic hyperammonemia is extremely rare. We present a case of adult onset non-cirrhotic hyperammonemia and how a multidisciplinary approach allowed for successful management of his condition.
Case Description/Methods: Case of a 65-year-old male with history of diabetes mellitus type II (DM-II) and hypertension, with multiple admissions due to encephalopathy secondary to hyperammonemia. He was initially managed with lactulose, with partial response. Gastroenterology service was consulted and an extensive workup was negative for liver offenders. Imaging studies and liver chemistries were unrevealing and cirrhosis was ruled out. No offending medications were present. Due to DM-II, small intestinal bacterial overgrowth was suspected, however after a course of antibiotics he had no response. The presence of urease producing bacteria was considered but resulted negative. Due to lack of response and no identifiable cause, an interdisciplinary approach with endocrinology service was done. An extensive genetic workup was negative for the most common mutations related with acquired hyperammonemia like urea cycle disorders, amino acid deficits or fatty acids oxidation disorders. Only abnormality was elevated threonine levels with normal citrulline levels in serum but elevated in urine. Mutation for Citrullinemia type- II was negative, however, before obtaining the final result, he was medically managed as having citrullinemia type II with a high-protein, low carbohydrate diet and Arginine supplementation. After this, ammonia levels decreased and encephalopathy resolved completely. Patient continues on this diet and has not been readmitted and remains with normal ammonia levels. Case will be referred for further evaluation at the National Institute of Health for definitive diagnosis.
Discussion: Adult onset non-cirrhotic hyperammonemia is very uncommon and proper identification can be very challenging, making interdisciplinary approach the best option for diagnosis and management. This case reiterates the importance of a thorough clinical history, workup and interdisciplinary teamwork that can guide the treatment and resolution of challenging clinical cases.



[3059] **Figure 1.** Grayscale ultrasound of the liver demonstrating normal hepatic parenchyma, with no sonographic evidence of surface nodularity or other cirrhotic features.

S3060

Labetalol, a Common Antihypertensive, an Uncommon Cause of Drug-Induced Liver Injury (DILI)

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Introduction: Labetalol is an antihypertensive medication (AHM) commonly used as first line agent for non-severe hypertension in pregnancy. We present a case of DILI in a young woman transitioned to labetalol while attempting to conceive.
Case Description/Methods: A 35-year-old woman with a h/o hypertension, hashimoto's thyroiditis, anxiety, obesity (BMI 34) was admitted to our hospital for abdominal pain, jaundice, pruritus and aminotransferase (LFT) elevations. She had been admitted previously at an outside facility with nausea, anorexia, and elevated LFTs (ALT 1264, AST 1419, ALP 294, TBili 5) 20 weeks after initiation of labetalol. There were no h/o alcohol abuse or risk factors for hepatitis B/C. Home medications including labetalol were stopped. Imaging studies including MRCP showed no evidence of biliary obstruction. Infectious workup (hepatitis A/B/C/E, CMV, HSV, EBV) and drug/tox screen were negative. A liver biopsy showed acute hepatocellular injury without eosinophils, steatosis, cholestasis or fibrosis; differentials included DILI and infectious hepatitis. LFTs improved, patient was discharged and labetalol restarted. Two weeks later, she presented to our hospital with worsening jaundice. Admission labs: ALT 905, AST 1553, ALP 257, TBili 19.8, INR 1.4 and creatinine 1. On exam, there was no stigmata of chronic liver disease or encephalopathy. Labetalol was discontinued. Infectious, hereditary, metabolic were negative. ANA was positive (1:320) homogenous pattern with mild elevation in IgG. Prednisolone 40 mg daily was started with rapid improvement in LFTs. At 8 week follow up, she was asymptomatic and LFTs were near normal (Tbili 2); prednisolone was tapered.
Discussion: Mild to moderate, transient aminotransferase elevations are noted in up to 8% of patients on labetalol. But they are asymptomatic and resolve even with continuation of the medication. Severe liver injury however is rare and rapid resolution typically ensues after cessation of the drug. However acute liver failure, death and OLT have been reported. There are isolated case reports and small case series of labetalol induced DILI, mostly in pregnant women. In a case series, 3 out of 11 patients died and 1 of 5 patients who had liver biopsy had chronic active hepatitis. The mechanism of labetalol hepatic injury is believed to be idiosyncratic. Inadvertent rechallenge has been shown to cause recurrence of DILI. We present this case to increase awareness of labetalol induced DILI as it is the most commonly used AHM in pregnant women.

S3061

It Only Takes One: A Case of Drug-Induced Liver Injury Due to Cefazolin Administration

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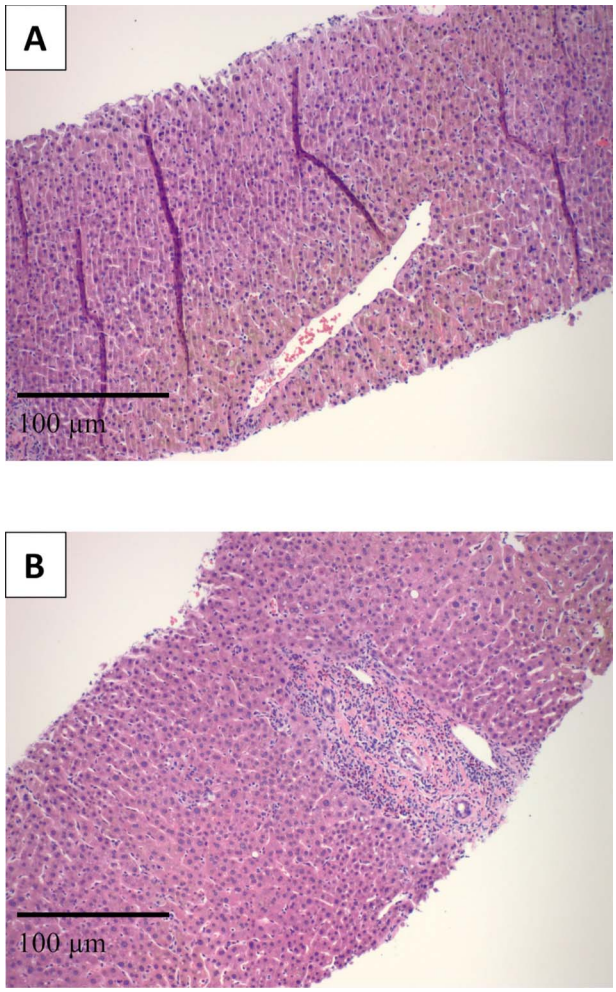
Introduction: Drug-induced liver injury (DILI) is a common cause of hepatotoxicity that has been associated with multiple medications and supplements. Herein, we present an intriguing case of DILI related to preoperative Cefazolin administration.
Case Description/Methods: A 64-year-old female with history of breast cancer presented for right upper quadrant abdominal pain, nausea, vomiting, and jaundice for 1 week. The patient underwent left mastectomy with administration of a single dose of preoperative IV Cefazolin about three weeks prior to presentation. On presentation, vital signs were within normal limits. On physical exam, the patient was noticeably jaundiced with right upper quadrant tenderness. Laboratory studies showed total bilirubin 11.9 mg/dL, direct bilirubin 8 mg/dL, alkaline phosphatase 603 IU/L, AST 165 IU/L, ALT 376 IU/L. Abdominal ultrasound found no biliary obstruction. Magnetic resonance cholangiopancreatography noted mild hepatomegaly with no intrahepatic or extrahepatic ductal dilatation. Workup for autoimmune disease, Wilson disease, and hemochromatosis was negative. Testing for hepatitis A and C was negative. The patient had positive HBcAb, however, HBsAg, HBsAb, and quantitative DNA PCR were negative. Liver biopsy showed cholestatic hepatitis, no significant fibrosis, and prominent eosinophils consistent with DILI. The patient was diagnosed with DILI secondary to Cefazolin administration. She was ultimately discharged home with improvement of symptoms and repeat lab work showing normalization of liver chemistries.
Discussion: Cefazolin is an antibiotic that is widely used for surgical prophylaxis, and it can be overlooked when working up acute liver injury, as it is usually a one-time dose. According to LiverTox, the latency period between administration and onset of liver injury is typically 1-4 weeks with most cases causing mild elevations of liver enzymes, however, significant elevations in aminotransferases greater than 5 times the upper limit of normal are rare (< 1%). Hepatotoxicity is likely due to hypersensitivity with primarily cholestatic patterns of liver enzymes, but mixed and hepatocellular patterns have been described. Our patient displayed a mixed pattern after one dose of Cefazolin, and eosinophils were present on biopsy consistent with hypersensitivity. This case highlights the importance of thoroughly reviewing a patient's history to help diagnose potential causes of liver injury, especially DILI, as it is imperative to the definitive management.



[3061] **Figure 1.** H&E stain (high power) showing intrahepatic cholestasis (A). H&E stain (high power) showing portal inflammation with prominent eosinophils (B).

S3062

Is It Cancer? Hepatic Inflammatory Pseudotumor: An Important Hepatic Mass Differential

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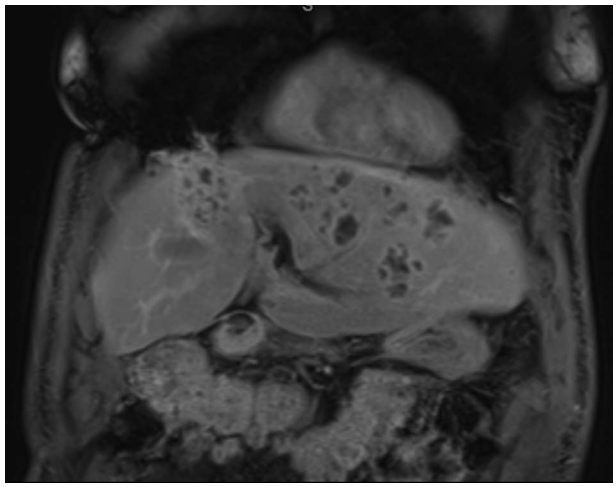
Introduction: Hepatic inflammatory pseudotumor (HIP), albeit rare, is an important pathology to be included in differentials for hepatic masses. The benign nature and treatment of this disease process should be considered especially in comparison to malignant hepatic processes.
Case Description/Methods: A 66-year-old male with pre-existing history of compensated Hepatitis C cirrhosis status post direct-acting antivirals with sustained virologic response presented in shock after a syncopal episode. Initial work up revealed leukocytosis, thrombocytopenia, acute renal injury, elevated liver enzymes, and COVID-19 positive test. Patient underwent initial liver ultrasound revealing intrahepatic and extrahepatic biliary ductal dilation. Subsequent MRCP demonstrated diffuse thickening of intra and extra hepatic bile ducts suggestive of cholangitis and several hepatic masses concerning for abscesses versus possible metastatic cholangiocarcinoma. Patient improved symptomatically with antibiotics and supportive care. A liver biopsy was performed with pathology showing lymphoplasmacytic inflammation and fibroblastic infiltration suggestive of hepatic inflammatory pseudotumor. A repeat MRCP one week later showed interval decrease in size of liver lesions and repeat liver function tests also showed improvement. Patient was discharged on a course of ciprofloxacin and metronidazole. Patient had repeat MRCP 3 months after discharge, with further significant improvement in size of liver lesions. After multi-disciplinary discussion the plan was for further surveillance with imaging and labs in 2 months.
Discussion: Inflammatory pseudotumors are benign and non-neoplastic lesions that can occur in any organ. They can appear as a malignant lesion when they arise in the liver and an accurate identification can allow for conservative management and prevent unnecessary invasive procedures. Hepatic inflammatory pseudotumors are often seen with concomitant infection or inflammatory processes. Liver biopsies distinguish these tumors from other malignant processes as they demonstrate a characteristic dense inflammatory infiltrate interspersed in stroma of interlacing bundles of myofibroblasts. This case highlights the importance of maintaining HIP on the differential diagnosis.



[3062] **Figure 1.** Coronal cross section of MRCP demonstrating numerous hepatic masses.

S3063

Jaundice? Look for Exposure to TMP-SMX!

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Introduction: Abnormal liver enzymes are commonly encountered in clinical practice and drug induced liver injury (DILI) is a common cause of hepatotoxicity. Trimethoprim-Sulfamethoxazole (TMP-SMX), a commonly prescribed antibiotic can cause serious adverse reactions. Herein, we present a rare case of a patient presenting with jaundice and transaminitis due to TMP-SMX.
Case Description/Methods: 83 y/o female with PMH of hypertension, GERD came to the emergency department for evaluation of jaundice and abdominal pain for the past 2 days. The pain was intermittent, diffuse, sharp and non-radiating associated with decreased appetite and nausea. She visited ED for dysuria 1 week ago and was discharged on TMP-SMX for presumed cystitis. She reported to have sweating and pains in multiple joints on the 3rd day of her antibiotic therapy and she stopped the medication on the third day. Over the next few days, her skin was turning yellow and she came to the ED for evaluation. She denied any recent travel, intravenous drug use, taking any herbal or OTC medications, blood transfusion history, change in bowel movements, fevers or chills. Vitals were normal and examination was unremarkable except icteric sclera. Labs were significant for elevated liver enzymes (ALT 263 U/L, AST 169 U/L, ALP 169 U/L), bilirubin (5.4mg/dl and direct bilirubin 4.3 mg/dl) and eosinophilia. Albumin was 2.9 and PT/INR was 14.7/1.25. Acetaminophen level, hepatitis panel, HIV antibody, ferritin, autoimmune workup, EBV, CMV and urine drug screen was normal. US abdomen and MRCP were unremarkable for any bile duct etiology. Based on the clinical findings with recent exposure to bactrim, it was highly suggestive of drug induced liver injury secondary to bactrim exposure. She was managed conservatively and her LFTs trended down on discharge.
Discussion: TMP-SMX induced liver damage has been attributed to three mechanisms including mixed hepatocellular cholestatic, hepatocellular and bile duct injury due to ductopenia. Thus, the liver injury may present as transaminitis and/or fulminant hepatic failure. Symptoms typically present within a few days of exposure to the medication but may even present months later. For non-paracetamol DILI, there is no strong evidence regarding the benefit of NAC. However, due minimal side effect profile, it is widely used. Clinicians must maintain a high level of suspicion when encountering acute hepatitis with unclear etiology. A detailed history of medication use may hold the answer.

S3064

Intestinal Post-Transplant Lymphoproliferative Disorder

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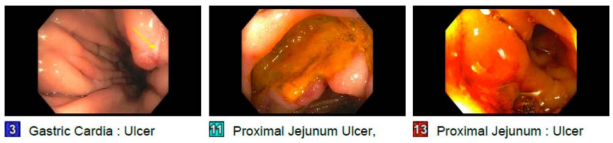
Introduction: Post-transplant lymphoproliferative disease (PTLD) is a group of lymphoid disorders arising in the setting of immunosuppression following a solid organ or stem cell transplantation. PTLD impacts 2.8% of adults and 15% of children post-orthotopic liver transplant (OLT) at a median of 8 years. The small intestine, rich in B cells, is a common site for PTLD and the treatment is chemotherapy. Here, we present a case of a patient post-OLT presenting with abdominal pain, and anemia found to have small bowel PTLD resulting in jejunal perforation with eventual resolution of symptoms after surgical resection and chemotherapy.
Case Description/Methods: 65-year old obese F with non-alcoholic steatohepatitis decompensated by hepatopulmonary syndrome with O2 dependence and hepatocellular carcinoma underwent an uneventful OLT (CMV +/-) with no immediate postoperative complications and resolution of the hepatopulmonary syndrome. Presented 2 months after the OLT with worsening LLQ abdominal pain and acute anemia. An EGD and colonoscopy completed shortly after admission showed evidence of gastric, duodenal, and descending colon non-bleeding ulcers that were biopsied. Pathology showed EBV+ atypical B cell proliferation concerning for large B cell lymphoma and monomorphic-PTLD. An EBV viral load obtained at that time was elevated and subsequent CT Abdomen/Pelvis showed scattered small sub-centimeter mesenteric and retroperitoneal lymph nodes. Her immunosuppression was minimized without a change in symptoms. She subsequently developed large volume haematochezia leading to shock and underwent an EGD and colonoscopy followed by a push enteroscopy that revealed multiple ulcers but no active bleeding site. Immediately after the procedure, she developed an acute abdomen due to a perforated Jejunal ulcer. Had an emergent exploratory laparotomy and jejunectomy with subsequent improvement in her anemia. She was treated with R-CHOP for PTLD with the resolution of her presenting symptoms.
Discussion: PTLD, however rare, remains the commonest post-transplant malignancy. Once there is suspicion (pain, weight loss, and anemia post OLT), EGD/Colonoscopy reveals suspicious malignant-looking ulcers, and mucosa or lymph node biopsy confirms PTLD. The patient ultimately underwent treatment with 4 cycles of R-CHOP with surveillance EBV PCR and imaging with resolution of complete symptoms. Immediate treatment should be started with lowering immune suppression and chemotherapy rather than waiting for self-resolution in PTLD cases.



[3064] **Figure 1.** Jejunal and Gastric ulcers (seen on prior scopes as well) were found on the Push enteroscopy just prior to the perforation of the bleeding jejunal ulcer and eventual surgical resection of the jejunum that resolved her anemia.

S3065

Ischemic Hepatitis Associated With Influenza A Infection

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Introduction: Ischemic hepatitis is one of the few differential diagnoses to consider in cases of severely elevated serum aminotransferases 20 times the upper limit of normal. Given the inherently robust nature of the liver despite ischemic states, ischemic hepatitis usually presents in patients requiring advanced care in an intensive care unit, and often carries a high rate of in-hospital mortality. Moreover, most cases occur in patients with underlying heart failure. Here we report a case of ischemia hepatitis in a patient not requiring intensive care and without apparent heart failure, who was successfully treated for COPD exacerbation in the setting of an influenza A infection.
Case Description/Methods: A 56-year-old female with COPD presented with 3 days of dyspnea with SpO2 as low as 79% on room air. She tested positive for influenza A. She was incidentally found to have elevated aminotransferases, which peaked within 12 hours of admission, with AST of 1226 IU/L and ALT of 943 U/L, and LDH of 1300 U/L. She had unremarkable INR, bilirubin, albumin, ALP, and GGT. Hepatitis panel, acetaminophen, ethanol, alpha-1 antitrypsin, antinuclear antibody, anti-smooth muscle antibody, antimitochondrial antibody, and ceruloplasmin were also all unremarkable. CT and ultrasound of the abdomen did not suggest underlying liver disease, and echocardiogram did not suggest any heart dysfunction. She denied any recent changes to her medications but did take marijuana gummy supplements. Her aminotransferases drastically fell with standard treatment of COPD exacerbation. Oxygen supplementation was limited to nasal cannula only, and she never required intensive care. At discharge, the patient's AST was 63 IU/L and ALT was 211 U/L.
Discussion: Although ischemic hepatitis is a rare diagnosis primarily diagnosed in the intensive care setting, it can be infrequently seen in the hospital ward when patients demonstrate serum aminotransferases 20 times the upper limit of normal. In the setting of infections by viruses not traditionally considered as hepatotropic, ischemic hepatitis serves as a potential mechanism for severe serum aminotransferase elevations due to a hypoxic state. Furthermore, recent research suggests that an immune-mediated response to viral infection, and not hypoxia, is what primarily drives the aminotransferase elevation.

S3066

Interferon Therapy for Chronic Hepatitis Delta Viral Infection

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Introduction: Hepatitis Delta Virus (HDV) infects those that are already infected with the Hepatitis B Virus (HBV), resulting in a serious infection that may damage hepatocytes. We present a patient that undergoes interferon therapy for chronic hepatitis delta infection with an emphasis on efficacy and side effects of treatment.
Case Description/Methods: A 20 year old previously healthy African male presents with progressive fatigue. Bloodwork demonstrates elevated liver enzymes, thrombocytopenia, positive Hepatitis B surface antigen and core antibody, and is negative for hepatitis B e-antigen. HBV DNA levels are 30 IU/mL (reference range < 10 IU/mL). Six months later, liver enzymes remain elevated (AST 100 U/L, ALT 186 U/L). Liver biopsy confirms chronic hepatitis and moderate fibrosis with Metavir grade A3, stage F2, and is consistent with viral hepatitis infection. The biopsy also reveals schistosomiasis of the liver, for which the patient is treated with two series of Praziquantel 1800 mg, 3 times per day. Schistosomiasis resolve following treatment, yet liver enzymes remain elevated. Subsequent bloodwork is positive for HDV, with very high levels of HDV RNA at 14,000,000 IU/mL. The patient is started on tenofovir 300mg/day and peginterferon 180 mcg/week. This therapy is maintained for over 4 years, with side effects of headache and fatigue. Liver biopsy near end of treatment reveals mild portal inflammatory infiltrate composed predominantly of lymphocytes with scattered foci of lobular inflammation; Metavir grade 1, stage 0. Bloodwork at this time shows improvement in liver enzymes with AST 57 U/L and ALT 39 U/L. Patient stopped interferon due to side effects after four years of suppressive treatment. The most recent bloodwork shows HBV and HDV viral suppression via PCR, and negative Hepatitis B surface antigen. Liver surveillance via ultrasound reveals no evidence hepatocellular carcinoma during the duration of therapy.
Discussion: Hepatitis delta virus (HDV) is a blood-borne virus that infects human hepatocytes. The replication of HDV depends upon a simultaneous infection with the Hepatitis B virus (HBV). Chronic HDV infections manifest as a rapidly progressing form of viral hepatitis, and can lead to cirrhosis with an increased risk of hepatocellular carcinoma. Currently, HDV has no approved treatment options from the United States Food and Drug Administration, and the only option for treatment is pegylated interferon-alpha.

S3067

Liver Histology Findings in COVID-19 Vaccine-Induced Hepatitis: A Case Series

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Introduction: There have been reported cases of hepatitis after COVID-19 vaccination. The previously described clinical presentations and histology findings have been heterogenous, making it difficult to identify a definitive pattern of liver injury. We present a series of three patients with elevated liver enzymes after Pfizer COVID-19 vaccination who underwent liver biopsy.
Case Description/Methods: Patient 1, a 32-year-old male in good health, presented with elevated liver enzymes on routine outpatient labs. He was asymptomatic and physical exam was unremarkable. He had received his second dose of the COVID vaccine 27 days prior. Serologic liver workup and biopsy findings can be found in Table 1. He was treated with ursodiol 500 mg twice daily, with significant improvement in liver enzymes after 30 days. Patient 2, a 56-year-old male with history of diabetes presented with nausea, pruritis and painless jaundice. Jaundice and diffuse excoriations were seen on exam. Liver enzymes were elevated and serologic liver workup and biopsy findings can be found in Table 1. His third COVID-19 vaccination was 5 days prior to presentation. The patient was treated with IV steroids for 5 days, with short term improvement in liver enzymes. Patient 3, an 85-year-old male with history of dyslipidemia and NSTEMI presented with dark urine and poor oral intake. Scleral icterus was noted on physical exam. Liver enzymes were elevated, and serologic liver workup and biopsy findings can be found in Table 1. He received his third COVID-19 vaccine 4 days prior. He was treated with prednisone taper with complete resolution of liver enzymes after 3 months.
Discussion: This case series suggests a possible correlation between Pfizer COVID-19 vaccination and hepatitis as demonstrated by liver biopsy findings. All three patients had no prior history of underlying liver disease, alcohol use or identifiable risk factors. Patient presentation was variable in terms of clinical symptoms, serologic workup and even histology findings. The common thread was elevated immunoglobulins suggesting an immune component without findings of autoimmune hepatitis. This is consistent with a previously published case series of COVID-19 vaccine-induced hepatitis. We share our findings to add to the collective repository of COVID-19 vaccine-induced hepatitis to add to existing literature and with the hope of increasing awareness.

Table 1. This table depicts demographic information, pertinent work up, and treatment of each case

Case	Age, Sex, BMI	Pertinent Medical History	Vaccine Type, Dose, Timing*	Associated Symptoms	Peak Lab Values					Pertinent Work Up (negative unless listed)	Liver Biopsy	Clinical Course
					AST (U/L)	ALT (U/L)	ALP (U/L)	T bili (mg/dL)	INR (ratio)			
1	32 male 25.1	None No prior liver disease	Pfizer-BioNTech Dose #2 27 days	None	222	372	666	1.4	1.1	IgG 1,670 mg/dL Cross-sectional imaging within normal limits	Chronic hepatitis with mild-moderate fibrosis (Grade 1-2, Stage 2) Mild centrilobular changes with steatosis	Treatment with ursodiol 500 mg twice daily for 30 days Improving

Table 1. (*continued*)

Case	Age, Sex, BMI	Pertinent Medical History	Vaccine Type, Dose, Timing*	Associated Symptoms	Peak Lab Values					Pertinent Work Up (negative unless listed)	Liver Biopsy	Clinical Course
					AST (U/L)	ALT (U/L)	ALP (U/L)	T bili (mg/dL)	INR (ratio)			
2	56 male 30.5	Recent acute cholecystitis, type 2 diabetes mellitus No prior liver disease	Pfizer-BioNTech Dose #3 (booster) 5 days	Nausea, vomiting, jaundice, scleral icterus, pruritis, dark urine, weight loss	474	395	860	23.6	1.4	IgA 640 mg/dL IgG 2,230 mg/dL, IgG subclass 4,153 mg/dL +ANA 1:640, +ASMA 1: 320 Ferritin 3,775 ng/mL MRCP: cholelithiasis with possible acute cholecystitis, no biliary duct dilatation, no choledocholithiasis Mild nonspecific periportal edema with mildly prominent periportal lymph nodes	Cholestatic hepatitis, stage 3 out of 4 NASH, tissue IgG4 immunostains unremarkable (1+ out of 4)	Treatment with IV methylprednisolone 100 mg x5 days, followed by 60 mg prednisone daily for 30 days with short term improvement
3	85 male 27.8	Hypo-thyroidism dyslipidemia No prior liver disease	Pfizer-BioNTech Dose #3 (booster) 4 days	Dark urine	2354	2221	313	15.9	1.2	IgG 1,770 mg/dL, IgG4 212 mg/dL ANA+, ASMA+ 1:20 CT scan A/P with IV contrast: acute interstitial pancreatitis, gallbladder wall thickening MRI: normal liver contour & size, without evidence of hepatic steatosis	Acute hepatitis Steatohepatitis, moderate inflammation, marked ballooning degeneration and no significant sinusoidal fibrosis	Treatment with prednisone 40 mg daily, then tapered over 3 months Normalized

AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; T bili, total bilirubin; IgG, immunoglobulin G; IgA, immunoglobulin A; ANA, anti-nuclear antibody; ASMA, anti-smooth muscle antibodies; MRCP, magnetic resonance cholangiopancreatography; DILI, drug-induced liver injury; NASH, non-alcoholic steatohepatitis *In association with COVID vaccine

S3068

Lifting the Veil off Weil's Disease: A Case Report of Fulminant Leptospirosis

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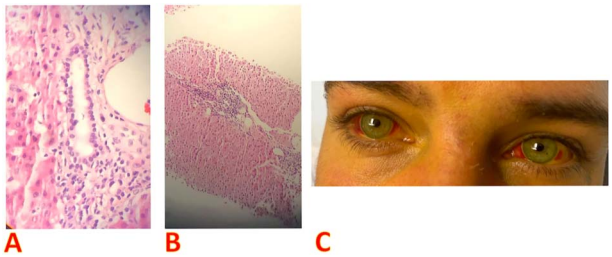
Introduction: Leptospirosis, a zoonosis caused by the spirochete Leptospira, is acquired by contact with urine of infected animals, mainly rodents, through cuts and mucous membranes. Traditionally considered as an occupational disease affecting farmers and sewer workers, incidence through recreational exposure via freshwater activities has been increasing. It is a notifiable disease, with around 150 cases reported annually in the US.
Case Description/Methods: A 32-year-old homeless man presented with fever, chills, nausea, myalgias, and headache for one day. He was an active intravenous drug user and UDS was positive for methamphetamine. Temperature was 101.2 F and heart rate 115/minute. Tooth decay and track marks were noted. Abdomen was non tender. Labs showed WBC count of 14.8k, platelet count (PC) 177k, CK 1036. TEE did not show any vegetations. CT scan showed a right lung consolidation. Patient was started on Vancomycin and Piperacillin-tazobactam. On HOD 1, patient developed watery, non-bloody diarrhea and lower limb pain. By HOD 3, PC fell to 44k, while creatinine started rising. Patient reported diffuse abdominal pain and now had scleral icterus with bilateral conjunctival congestion. Total bilirubin was 12.8. Stool studies, peripheral smear and hepatitis panel were unremarkable. Liver biopsy showed sinusoidal congestion and mild chronic inflammation within the portal tracts, with rare interface hepatitis. An infectious cause was strongly suspected in view of the bicytopenia and multi-systemic changes. Rhabdomyolysis and AKI could also be attributed to meth use. On HOD 12, Leptospira IgM Ab returned positive. The patient received a 7-day course of oral doxycycline. Eventually, labs normalised and he was discharged on HOD 20. (Figure)
Discussion: The fulminant form of leptospirosis, Weil's disease, presents with renal failure and jaundice. Conjunctival suffusion (redness without purulent exudates) is characteristic in the initial leptospiremic phase. Hepatocellular damage and disruption of intercellular junctions leads to bilirubin elevation in the immunological phase. Antibodies take 3-10 days to develop and so IgM serological tests must be done a week after symptom onset. PCR is very sensitive and specific in the acute phase. Microscopic agglutination test is considered the gold standard due to its high specificity. Starting treatment with empiric antibiotics early on without waiting for testing results can be lifesaving, especially in cases with a high degree of clinical suspicion.



[3068] **Figure 1.** A, B: Liver biopsy showing liver parenchyma with sinusoidal congestion and mild chronic inflammation within the portal tracts consisting mainly of lymphocytes and occasional plasma cells; C: Bilateral conjunctival suffusion, characteristic of leptospirosis.

S3069

Lifesaving but Tiresome Treatment for Tyrosinemia Type 1

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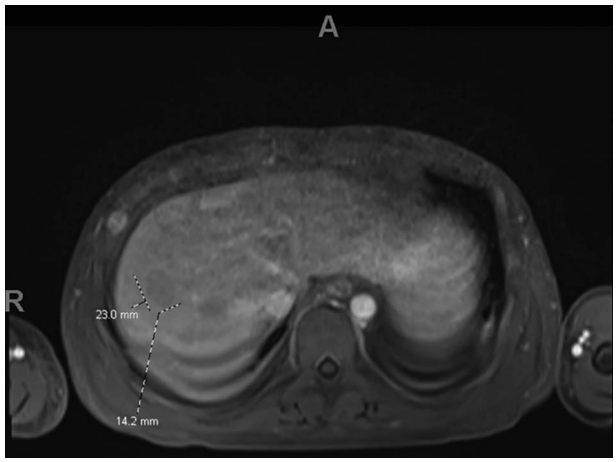
Introduction: Tyrosinemia type I (HT1) is a rare autosomal recessive condition caused by a genetic mutation disrupting proper functioning of the fumarylacetoacetate hydrolase (FAH) enzyme, the terminal enzyme in the tyrosine catabolism pathway. The below case exemplifies the difficulty for adherence as children transition to adulthood of the standard therapy that prolongs life and decreases need for orthotopic liver transplantation (OLT).
Case Description/Methods: A 23-year-old male with known tyrosinemia type I presents with progressively worsening back, lower extremity, and abdominal pain over the last six months. He had been lost to follow-up from Genetics clinic for the last ten years. He adhered to nitisinone therapy and diet modifications until around five years ago. The patient was admitted to the hospital for neurological crises due to

non-adherence to HT1 therapy. On admission, vital signs were stable and his physical exam significant for mild tenderness of abdomen at RUQ and tenderness in the lumbar and thoracic region of back. Pertinent lab findings were albumin of 3.2, alkaline phosphatase of 356. AST, ALT, Bili, CBC, BMP WNL. Amino acid panel found tyrosine level of 62. AFP level of 188. MRI Abdomen with and without contrast found cirrhotic liver morphology, developing portal hypertension (borderline enlarged spleen, collateral vasculature, but no ascites), and a lesion of hepatic segment 8 measuring 2.3cm x 1.4cm in diameter. Patient was planned to be restarted on nitisinone with pending further evaluation of liver lesion. (Figure)

Discussion: The above case demonstrates a patient with the rare genetic disorder Tyrosinemia type 1 with non-adherence to nitisinone and diet modification therapies. The presentation of neurological crises and abdominal pain with development of hepatomas/hepatocellular carcinoma (HCC) is typical for untreated or non-adherent patients with HT1. Nitisinone has been shown to be highly effective for this condition with 4-year survival rates of 94% compared to 29% of diet modification alone. Prior to nitisinone therapy, most patients required OLT. Despite the clear benefits, recent evidence is finding increasing non-adherence as patients grow older into adolescence and adulthood. This is consistent with adherence problems with early diagnosis chronic diseases with long-term therapies. More research is needed to elucidate major barriers for non-compliance and to refine guidelines regarding frequency of follow-up and monitoring of serum AFP to screen for HCC and encourage compliance.



[3069] **Figure 1.** MRI Abdomen with contrast with findings of a hepatic lesion on segment 8.

S3070

Macro AST: An Uncommon Yet Benign Entity

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Introduction: An isolated increase in serum aspartate aminotransferase (AST) can be attributed to many conditions, including alcoholic liver disease, myocardial injury or skeletal muscle injury. Persistent elevations in AST without an obvious source can lead to diagnostic confusion. When evaluating such a patient, one must consider macro-enzyme aspartate aminotransferase (macro-AST). Macro-AST is an uncommon, yet benign condition in which a macro-enzyme is formed from self-polymerization of AST molecules or by the formation of a complex with other serum proteins such as immunoglobulins (Ig). These macro-enzymes circulate in the blood stream and can lead to an accumulation along with decreased clearance from the blood stream. Below, is an example of a woman without suggestive symptoms found to have persistently elevated AST.

Case Description/Methods: The patient is a 60-year-old Hispanic female with hypothyroidism, hypertension who presented to the emergency room with complaints of lower back pain. Initial blood work revealed an AST of 404 U/L (normal 8-34 U/L) without other significant laboratory abnormalities. A computed tomography (CT) scan of the abdomen and pelvis with intravenous (IV) contrast showed lumbar spinal stenosis without other abnormalities. A right upper quadrant abdominal ultrasound with Doppler showed a normal appearing liver, biliary tree and patent vasculature. She denied any changes in her medications or use of supplements. She denied any history of liver disease, alcohol use or a family history of liver disease. Acetaminophen, ethanol and urine drug screens were negative. Testing for viral hepatitis, autoimmune hepatitis, ceruloplasmin, alpha-1-antitrypsin, ferritin, creatinine kinase, troponin I, Celiac disease and thyroid function tests were all unremarkable. Her AST level remained persistently elevated throughout hospitalization and after discharge. Given unremarkable workup and chronic AST elevation, macro-AST was the likely diagnosis.

Discussion: Macro-AST is a benign cause of persistently elevated AST. Though its prevalence is not well known, some reports suggest that they can be seen across a wide age range and have no genetic basis as relatives of the patients have normal AST levels. Some cases have suggested a means of confirming the diagnosis via polyethylene glycol precipitation or electrophoresis. As these elevations can persist for over ten years, it is important for clinicians to be aware of this entity to avoid unnecessary procedures or testing.

S3071

Leflunomide-Induced Severe Hepatotoxicity Treated With Ursodeoxycholic Acid

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Introduction: Drug induced liver injury (DILI) is common usually leading to mild elevations in liver enzymes but on rare occasions cause severe liver injury and it is regarded as the most common cause for acute liver failure in the US. Leflunomide is a disease-modifying antirheumatic agent approved for rheumatoid arthritis (RA) and psoriatic arthritis (PA). Mild elevations in the liver enzymes is a common side effect but rare severe hepatotoxicity and fatalities have been reported. We present a case of severe liver injury related to leflunomide with no preexisting liver disease, or use of other hepatotoxic drugs whose prolonged pruritus and cholestasis responded to ursodeoxycholic acid (UDCA).

Case Description/Methods: 57-year-old female who was started on Leflunomide for uncontrolled RA was referred to our clinic due to elevated liver enzymes and jaundice. The patient reported rapid improvement of joint pain with Leflunomide but on 5th and 6th week of treatment she started complaining of diarrhea, nausea, fatigue and pruritus. Her only other medications were Olmesartan and Ketorolac. Labs revealed AST 253, ALT 810, Alk Phos of 466 and Bilirubin of 4.0. Leflunomide was immediately discontinued. Work up showed negative hepatitis serologies and autoimmune markers. MRI with MRCP was normal. On week 8 and 9 she had persistent severe pruritus and progressive increase in bilirubin. UDCA 500 mg twice daily was started. A week later bilirubin decreased and itching resolved. Figure 1 shows this trend.

Discussion: Leflunomide is used for RA and PA and can be associated with symptomatic severe liver injury. The liver injury arises after 1 to 6 months of therapy and present with a cholestatic or hepatocellular pattern. On weeks 5-6 of treatment, our patient was symptomatic with diarrhea, nausea, and pruritus. A mixed hepatocellular and cholestatic pattern was noted on week 7, with an improvement of liver enzymes within a week of stopping treatment, however, with progressive elevation of bilirubin and troublesome persistent pruritus that responded promptly to UDCA. This case underscores the importance of monitoring liver enzymes in patients on Leflunomide, at least once monthly for the first six months, and immediate discontinuation when liver enzymes increase more than three times upper normal. In our patient, worsening jaundice and persistent pruritus responded promptly to UDCA which supports data that shows possible benefits of UDCA in the treatment of DILI.

Table 1. **Hepatic Panel results. The weeks correspond to the length of time after initiation of Leflunomide**

Laboratory	Week 7	Week 8	Week 9	Week 10	Week 13
AST	253	193	66	60	27
ALT	810	600	220	167	38
ALK Phos	466	413	368	286	142
T. Bilirubin	4	5.7	7.5	4.7	1.4

S3072

Liver Injury Caused by a Weight Loss Supplement Containing Green Tea Extract and Garcinia Cambogia

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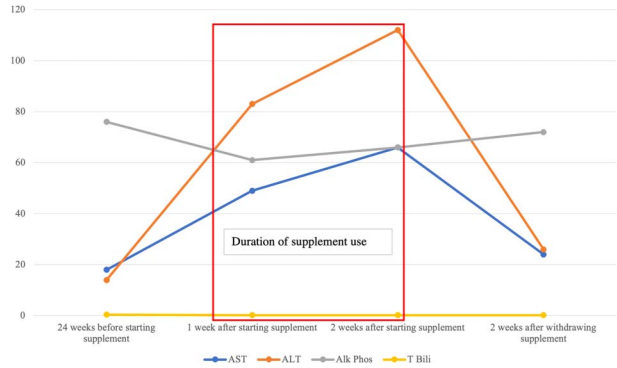
Introduction: Herbal-induced liver injury (HILI) is a growing topic worldwide, and herbal and dietary supplements (HDS) are the second leading class of compounds after antimicrobials causing liver injury in the United States. It is thought that many cases go unrecognized, as patients fail to mention their use of HDS. This is a case of mild liver injury in a young woman after taking a short course of a weight loss supplement containing a garcinia cambogia (GC) and green tea extract (GTE).
Case Description/Methods: A 45-year-old female with a history of obesity presented to clinic with elevated liver enzymes identified on routine labs from two weeks prior. Medication reconciliation revealed that one month prior, she started a 15-day course of Revert 10.0 by Natureal for weight loss. She stopped taking the supplement on her own as she felt it was not helpful. Her only prescription medication was her oral contraceptive pill. She was born in the United States and denied any prior history of liver disease. She was asymptomatic with no nausea, abdominal pain, or jaundice. Her physical exam was unremarkable. Baseline laboratory values from six months prior showed AST 18 U/L (normal < 40), ALT 14 U/L (normal < 32), ALP 76 U/L (normal 39-117), TBili 0.4 mg/dL (normal < 1.2). Her liver tests after one week of using Revert 10.0 revealed elevations of AST to 49 and ALT to 83, with normal ALP 61, and TBili 0.2. LFTs after two weeks showed further rises in AST to 66, and ALT to 112, with normal ALP 66, and TBili 0.2. Her viral hepatitis panel was non-reactive. An abdominal ultrasound and CT scan of the abdomen/pelvis showed no abnormalities with normal echogenicity and liver morphology. Two weeks after withdrawing the supplement, her LFTs had returned to normal, AST 24, ALT 26, ALP 72, and TBili 0.2. (Figure)
Discussion: This is the first report of liver injury from Revert 10.0. We encourage clinicians to consider HILI in patients with even mild laboratory abnormalities, and to conduct thorough history-taking. While GC and GTE are both known hepatotoxins, there are no cases reported of the two agents working in conjunction to cause HILI. Furthermore, the Roussel Uclaf Causality Assessment Method, the gold standard for HILI diagnosis does not consider the potential for synergy between multiple known hepatotoxic agents. Our case highlights that further research should explore how synergy of multiple supplements can affect presentation of HILI.



[3072] **Figure 1.** Liver Enzyme Trend Over Time

S3073

Liver Involvement in Autosomal Recessive Polycystic Kidney Disease

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Introduction: Autosomal recessive polycystic kidney disease (ARPKD) is an uncommon genetic disorder, which is seen in approximately 1 in 20,000 children.[1] Because this disease is so rare, the associated pathologies of ARPKD can often be overlooked. It is important to know that liver involvement is invariably present in all cases of ARPKD and the following case highlights this association.[2]
Case Description/Methods: A 36-year-old female with ARPKD status post renal transplant on mycophenolate and tacrolimus for immunosuppression presented to the hospital with melena and hematemesis. Her hemoglobin was 7.2 g/dL on admission; her baseline hemoglobin was 12.5 g/dL. EGD was performed and grade 2 esophageal varices were identified and banded. These findings prompted workup for portal hypertension (PH) etiology because the patient had no known history of liver disease. She had a repeat EGD one month later to evaluate for eradication of varices. Endoscopic ultrasound (EUS) was also performed at that time for liver biopsy and portal pressure gradient (PPG) measurements. The calculated EUS-guided portal pressure gradient was 18.3mmHg. The sonographic findings of the liver revealed a diffuse abnormal echotexture which was characterized by a heterogenous appearance. Liver biopsy revealed a benign hepatic parenchyma with multiple ductal plate malformations. This finding is consistent with congenital hepatic fibrosis which has a known association with ARPKD.
Discussion: This young patient's presentation with esophageal varices was rather perplexing considering she had no known history of liver disease, alcohol dependence, or known risk factors for PH. The key to identifying the cause of her PH was her history of ARPKD. The novel method of EUS-guided PPG measurement was used to evaluate PH severity and provided helpful information without the need of an additional procedure. Liver biopsy confirmed the diagnostic suspicion. Approximately 10% of ARPKD patients ultimately require liver transplantation.[3] The patient is being followed closely in the clinic.

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S3074

Lecithin-Cholesterol Acyltransferase Deficiency From Statin-Induced Autoimmune Hepatitis

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Introduction: A 65yr female presented to the hospital with complaints of pruritis and fatigue that had been ongoing for two months. Upon admission she was found to have transaminitis with AST and ALT at 392 and 400 respectively. (reference lab values AST 0-37 ; ALT 0-35) along with a total bilirubin of 25.1 mg/dL. (Ref 0-1.2). She reported that 1 month ago she went to a primary care doctor and was started on insulin, a calcium channel blocker and atorvastatin. A liver biopsy was done which revealed subacute liver injury- autoimmune pattern of injury (de novo versus secondary) +/- medication injury. This was confirmed by further positive autoimmune markers suggestive of autoimmune hepatitis.
Case Description/Methods: The patient presented to the ER complaining of intense pruritis, dark urine, pale stools, and jaundice. She had recently started atorvastatin, amlodipine and insulin for newly diagnosed diabetes mellitus and essential hypertension. All home medications were stopped at admission except insulin. She had no prior history of liver disease and no family history of liver disease. She did not take any supplements and had never consumed alcohol. Initial labs revealed a transaminitis with AST and ALT at 392 and 400 IU/L respectively. (reference lab values AST 0-37 ; ALT 0-35)along with alkaline phosphate at 2147 IU/L. (Ref 44-121). Initial imaging with a right upper quadrant ultrasound was normal. Patient continued to have a transaminitis and elevated alkaline phosphate that were not improving. A MRCP was done which showed hepatomegaly, mild periportal edema, slightly heterogenous enhancement-conglomerate of findings suggests nonspecific hepatitis. An EUS liver biopsy was performed which revealed subacute liver injury- autoimmune pattern of injury (de novo versus secondary) +/- medication injury. Additional laboratory testing revealed positive autoimmune markers; pointing to a drug induced autoimmune hepatitis as the likely diagnosis.
Discussion: Patient was started on prednisone and discharged on a steroid taper once her liver function tests began to improve. She followed up in liver clinic and was noted to have developed hypercholesteremia with total cholesterol of 1,375 MG/DL (Ref 170-199 MG/DL), LDL of 1220 Mg/DL and HDL of 162 MG/DL. A cholesterol esters assay was ordered and referral for genetic testing made for likely acquired lecithin cholesterol acyltransferase deficiency in the setting of statin induced autoimmune hepatitis. Previous cholesterol tests prior to starting atorvastatin were normal.

S3075

Massive Splenomegaly With Non-Cirrhotic Portal Hypertension

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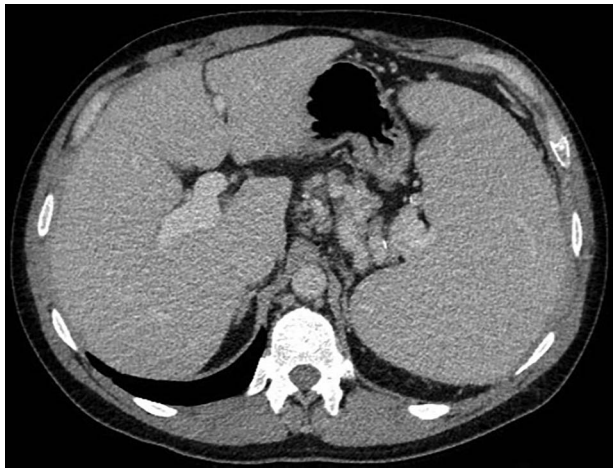
Introduction: A 42 year old male with a 12 year history of Common Variable Immune Deficiency (CVID) presented for management of "cirrhosis." His only subjective complaint was mild non-specific abdominal pain. He noted no history of jaundice, weight loss or constitutional symptoms. He did not drink alcohol. He was receiving immune globulin (Gamunex-C) infusions every 28 days.
Case Description/Methods: Physical exam revealed a middle aged male in no acute distress with stable VS with mild diffuse abdominal tenderness and "fullness." Remainder of exam was unremarkable. An abdominal CT scan was obtained that showed massive splenomegaly (Image 1), portal vein enlargement, no ascites. He had intact hepatic synthetic and excretory function, normal aminotransferases but an elevated alkaline phosphatase. An EGD done three years previously showed minimal esophageal varices. A transjugular liver biopsy was obtained that showed minimal hepatic fibrosis but features of nodular regenerative hyperplasia (NRH). (Figure)
Discussion: CVID is the most common immunodeficiency disease with a prevalence of 1 in 25,000. It is a primary B-cell immunodeficiency disorder with hypogammaglobulinemia. It can occur at any age, without gender preference and can affect multiple organ systems to include the liver with hepatic fibrosis and portal hypertension. The most common hepatic dysfunction in CVID is nodular regenerative hyperplasia (NRH) occurring in 5-10% in this population. Depending on clinical practice patterns the gastroenterologist could be presented with this scenario *de novo* or in consultation with Allergy/Immunology or Infectious Disease subspecialties. NRH in this population has been divided into three categories: Category 1; non-progressive, Category 2; NRH with portal hypertension and splenomegaly (as in our case presentation) and Category 3; NRH with features of autoimmune hepatitis. Repeated hepatic cell injury with concurrent regeneration causing compression of hepatic parenchyma, or of the portal vein and central veins is thought to result in portal hypertension, esophageal varices and splenomegaly. The patient can present anywhere along this variable clinical spectrum of development. Biopsies typically show a lack of perinodular fibrosis or intrahepetic shunts. Category II patients despite immunoglobulin replacement, can have infectious complications due to neutropenia. Category III patients can be progressive. Awareness of this entity is essential to optimize treatment strategies.



[3075] **Figure 1.** Splenomegaly calculated volume of 2000 cc, engorged splenic vein.

S3076

Marchiafava-Bignami Disease: Have You Ever Heard of This?

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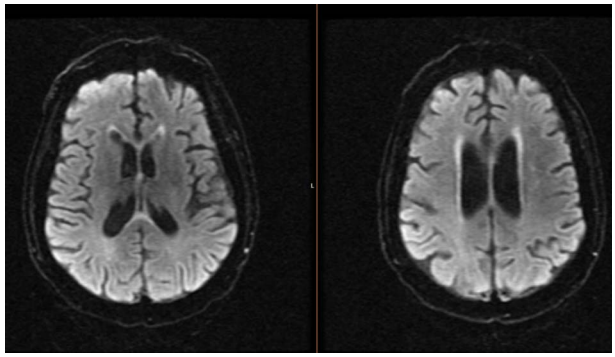
Introduction: Marchiafava–Bignami disease (MBD) is a rare disorder characterized by primary demyelination and necrosis of the corpus callosum. This disorder is mainly encountered, although not exclusively, in nutritionally depleted chronic alcoholics, resulting in usually fatal neurologic disease.
Case Description/Methods: A 55- year-old male with a past medical history significant for chronic alcoholism and who had consumed about 800 to 1000 grams of liquor for the past 15 years was admitted to the inpatient service initially for evaluation of recurrent falls and new-onset abdominal pain over the last few weeks. Further workup revealed leukocytosis, macrocytic anemia with normal vitamin B12 and folate levels, thrombocytopenia, transaminitis, hypoalbuminemia, severe hyponatremia, and mild hyperammonemia, and with normal coagulation studies. CT head without contrast was normal on admission. He was initially diagnosed and managed for acute alcoholic hepatitis. The hospital course was complicated with altered mental status, which progressed to a coma in two weeks. He was treated for hepatic encephalopathy, correction of hyponatremia, alcohol withdrawal, injection of thiamine, and empiric coverage with antibiotics for possible spontaneous bacterial peritonitis (SBP). SBP workup was normal, as well as other neurologic disorders. MRI of the brain without contrast was later performed as the patient did not improve on earlier management. MRI revealed an increased FLAIR signal in the inferior corpus

callosum and mild restricted diffusion in the genu and splenium of the corpus callosum consistent with Marchiafava-Bignami disease. The patient's clinical condition deteriorated, and he passed away subsequently after the family proceeded to honor his wishes for hospice care. (Figure)
Discussion: MBD mainly involves the corpus callosum but can affect other brain regions. The presentations of MBD are nonspecific and include dementia, altered mental status, spasticity, dysarthria, ataxia, gait abnormalities, seizures, and coma. MBD manifesting with severe neurological dysfunction has a poorer prognosis. This is a rare disorder that is not only difficult to diagnose as there are other possible etiologies; it is, therefore, imperative that gastroenterologists and hepatologists are familiar with the diagnostic and therapeutic approaches to this disorder and involvement of the multispecialty team early on.



[3076] **Figure 1.** MRI with and without contrast of the brain.

S3077

Malignancy Diagnosed via Ascites Analysis and EUS-Guided Portal Pressures

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Introduction: Ascites is the pathologic accumulation of fluid within the peritoneum and is classified by underlying etiology. Workup includes measurement of the serum ascites albumin gradient (SAAG) and ascitic protein levels with imaging modalities used as adjuvant tests. Although cirrhosis is the most common cause, other causes can include malignancy, infections, or congestive heart failure. We present a case of EUS-guided portal pressure measurements and liver biopsy being used to differentiate etiology of ascites when lab workup and imaging findings were incongruent.
Case Description/Methods: Patient is a 48-year-old female who presented with new-onset ascites. Abdominal ultrasound showed nodular contour of the liver with ascites suggestive of cirrhosis. Patient denied any alcohol use, drug use, or family history of liver disease. She underwent paracentesis and fluid analysis revealed a SAAG of 0.8, ascitic protein 4.3 gm/dL, and negative culture and cytology. Because of discordance between imaging findings and ascitic fluid analysis, she underwent EUS-guided portal pressure measurements and liver biopsy. EUS of the liver showed sharp borders and homogenous echotexture of the liver. EUS-guided portal pressure gradient was 0, indicating no evidence of portal hypertension and liver biopsy was inconsistent with cirrhosis. Further workup revealed elevated CA-125 and transvaginal ultrasound showed bilaterally complex cystic and solid adnexal masses. She underwent surgical resection and biopsy of a peritoneal wall mass which was consistent with mucin producing adenocarcinoma.
Discussion: The gold standard for diagnosis of cirrhosis is liver biopsy, but several imaging modalities have been used as reliable alternatives. Nodularity is the most common imaging finding in cirrhosis, however imaging is subject to observer variability. CT is more sensitive than ultrasound, but often appears normal at early stages. Our case demonstrates a patient in whom ascites was assumed to be due to cirrhosis based on imaging, but EUS-guided portal pressures and liver biopsy were used to rule out cirrhosis and portal hypertension. These findings prompted additional workup which led to the diagnosis of ovarian malignancy. Ruling out portal hypertension allowed for surgical risk stratification. We recommend a comprehensive workup when ascites analysis and imaging are discordant.

S3078

Liver Function Abnormalities in Adult Onset Still's Disease: A Case Report

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Introduction: Adult Onset Still's disease(AOSD) has an estimated incidence of 0.16-0.4 per 100,000 people[1]. Hence, liver dysfunction due to AOSD is a rare entity with a wide spectrum from hepatomegaly and elevated transaminases to fulminant hepatic failure. Delayed diagnosis can lead to life-threatening liver failure. We report a case of liver dysfunction due to AOSD diagnosed after extensive evaluation.
Case Description/Methods: A 27-year-old African-American woman presented to the emergency department with 2 weeks of fever, chills, myalgia, non-bloody watery diarrhea, shortness of breath, and pleuritic chest pain, which started after a recent trip to Haiti. On exam, she was febrile (103 F), tachycardic, hypoxic, with tender bilateral axillary and inguinal lymphadenopathy. Treatment with antibiotics, methylprednisolone, and anti-tubercular therapy (RIPE) was initiated. She required intubation and vasopressor support due to worsened respiratory distress. Her liver enzymes were noted to be trending up after admission. RIPE therapy was modified but transaminases continued to rise. Extensive infectious and rheumatological workup was negative. Work-up for chronic liver disease was negative as well (table 1). A sonogram with doppler and MRCP showed normal hepatobiliary anatomy. Inguinal lymph node biopsy showed reactive hyperplastic patterns. A liver biopsy showed few nonspecific small lobular foci of spotty inflammation, necrosis and ballooning degeneration of hepatocytes. The patient was diagnosed with AOSD based on Yamaguchi criteria since she met 3 major and 3 minor criteria. Given lack of improvement with methylprednisolone, interleukin-1β inhibitor canakinumab was started. Inflammatory markers and liver enzymes improved significantly and she was discharged.
Discussion: AOSD is a rare disease with nonspecific signs and symptoms. Liver function abnormalities are the most common manifestation in AOSD. Liver involvement can occur in the absence of other features of the disease and can delay diagnosis. Diagnosis is based on clinical criteria. Treatment is largely based on expert opinion since controlled trials are lacking. The most effective treatment is corticosteroids and biologic therapy. This case highlights the need for a high index of suspicion for AOSD when abnormal liver enzymes are seen in the context of nonspecific signs and symptoms.

Table 1. Laboratory Investigations during Hospitalization

TEST	ON ADMISSION	PEAK VALUE	ON DISCHARGE(POST-CANAKINUMAB)
WBC	10,870	33,710	8210
AST	91	625	40
ALT	19	294	82
ALP	48	1460	554
Total Bilirubin	0.6	12.6	1.1
Direct Bilirubin	-	8.6	0.6
GGT	-	1136	-
CRP	120	168	< 5
ESR	43	90	19

Table 1. (*continued*)

TEST	ON ADMISSION	PEAK VALUE	ON DISCHARGE(POST-CANAKINUMAB)
Ferritin	>40,000	>40,000	518
Rheumatoid Factor	12	-	-
ANA	Negative	-	-
Hepatitis A IgM	Non-reactive	-	-
Hepatitis A IgG	Reactive	-	-
HbsAg	Non-reactive	-	-
HbsAb	Reactive	-	-
HbcAb	Non-reactive	-	-
HbeAg	Non-reactive	-	-
HbeAb IgM	Non-reactive	-	-
HbeAb IgG	Reactive	-	-
HBV DNA	Not detected	-	-
Hepatitis C Antibody	Non-reactive	-	-
Alpha-1 antitrypsin	Normal	-	-
Ceruloplasmin	Normal	-	-
Anti-Mitochondrial Antibody	Negative	-	-
Anti-SMA	Negative	-	-
Anti-LKM Antibody	Negative	-	-

Abbreviations WBC - white blood cells AST - aspartate transaminase ALT - alanine transaminase ALP - alkaline phosphatase GGT- gamma - glutamyl transferase CRP - C reactive protein ESR - erythrocyte sedimentation rate ANA - antinuclear antibody IgM - Immunoglobulin M IgG - Immunoglobulin G HbsAg - Hepatitis B surface antigen HbsAb - Hepatitis B surface antibody HbcAb - Hepatitis B core antibody HbeAg - Hepatitis B e antigen HbeAb - Hepatitis B e antibody HBV DNA - Hepatitis B virus DNA SMA - smooth muscle antibody LKM- liver-kidney-microsomal

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S3079

Leaking Curiosity: Post-Operative Drainage Leading to Revelation of Noncirrhotic Portal Hypertension in a Patient With Suspected Wilson's Disease

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Introduction: In patients with an elevated serum ascites albumin gradient and low ascitic protein, the presumption is often that ascites is secondary to cirrhosis. Here, we present an interesting case of portal hypertension without significant fibrosis.
Case Description/Methods: A 61 year old male with history of "chronic encephalopathy," severe agitation, dysphagia, Roux-en-Y gastric bypass, anemia, and chronic pain presented with 2 days of confusion, nausea, emesis, and abdominal pain accompanied by distention. Imaging was notable for massive pneumoperitoneum as well as abdominopelvic ascites. He was emergently taken to surgery, undergoing repair of a perforated marginal ulcer and subsequent placement of a JP drain near the gastrojejunal anastomosis at the right upper quadrant. He was downgraded from the surgical ICU to a floor medicine team with significant output from the JP drain (500-1500 mL daily), which surgery attributed to expected leakage in the context of peritonitis and severe malnutrition; nutritional deficiencies included zinc, vitamin A, and vitamin D. On further investigation, ascitic fluid studies were consistent with portal hypertension presumed secondary to cirrhosis. Duplex abdominal ultrasound was notable for ascites and slightly nodular liver surface, prompting further evaluation. Chronic liver disease workup was notable for low ceruloplasmin at 12 mg/dL and elevated 24 hour urine copper at 165 mcg. Considering patient's overall history and concerns that his neuropsychiatric disturbances, ocular complaints, anemia, arthralgias, and presumed cirrhosis were due to Wilson's disease, Hepatology was consulted and recommended liver biopsy with quantitative copper in addition to evaluation by Neurology and Ophthalmology to add to the clinical picture. Biopsy was read as noncirrhotic portal hypertension with patchy sinusoidal dilatation and portal vein changes, suggestive of hepatoportal sclerosis/sclerosing portal venopathy; no evidence of significant fibrosis, and copper was normal at 16 ug/g. Patient was discharged from the hospital and later passed away without further evaluation.
Discussion: This patient's clinical picture and laboratory results were suspicious for late diagnosis of Wilson's disease, though lack of assessment for Kayser-Fleischer rings renders it challenging to establish this as the case. That hepatoportal sclerosis was the suggested etiology for this patient's noncirrhotic portal hypertension is curious and would require exclusion of chronic liver disease.

S3080

Metastatic Merkel Cell Carcinoma Presenting as Silent Infiltrative Liver Disease: A Lesson Emphasizing the Utility of Liver Biopsy in the Exclusion of Immune Checkpoint Inhibitor-Mediated Hepatotoxicity

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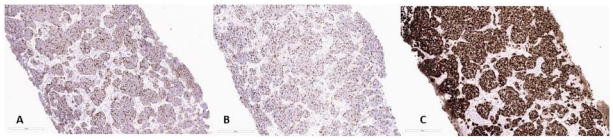
Introduction: Merkel cell carcinoma (MCC) is a rare neuroendocrine tumor that arises on skin exposed to the sun and occurs in patients who are Caucasian, elderly, and immunocompromised. The mortality of MCC is about 33% and disseminated disease suggests poor prognosis. Metastasis to the liver is exceedingly rare, with only few reported cases presenting as infiltrative liver disease. We present a case of MCC with "silent" infiltration in the liver effectively differentiated from suspected immune checkpoint inhibitor-mediated hepatotoxicity (IMH) by liver biopsy
Case Description/Methods: A 76-year-old man with a history of metastatic MCC of the left distal thigh status-post stem cell transplant presented to a cancer care center for evaluation of fatigue, nausea, and dyspnea. He reported consuming 10 alcoholic drinks a week for many years until 2 years ago. He was started on a treatment regimen of pembrolizumab and plinabulin (a phase I microtubule inhibitor) with radiation to the liver 2 months ago. On admission, serum transaminases were elevated from baseline as indicated in Table 1. After an initial period of observation, the patient was started on oral budesonide 9 mg/d and ursodiol 1000 mg/d for empiric treatment of IMH without subsequent improvement in liver enzymes. Liver imaging was unrevealing for liver lesions. A diagnostic parenchymal liver biopsy was performed to clarify the underlying diagnosis, including possible IMH, drug-induced liver injury from plinabulin toxicity, radiation-induced liver injury, or metastatic disease. Histology showed that the tumor cells were strongly positive for synaptophysin, chromogranin, and extensive metastatic MCC. The steroids were discontinued, and the worsening hepatitis was attributed to Merkel cell liver infiltration. The patient subsequently passed away 3 months later from cancer. (Figure)
Discussion: Traditionally when there is high suspicion, IMH is clinically diagnosed without expectation for liver biopsy, and steroids are often prescribed upfront. One critique regarding the role of liver biopsy may be that histological features associated with IMH are nonspecific, with features of lobular to pan-lobular hepatitis. However, the absence of an alternative findings on the biopsy confers additional confidence towards the diagnosis of IMH. This case highlights the importance of the diagnostic liver biopsy prior to immunosuppressive treatment for suspected IMH to exclude other differentials and guide the decision for empiric steroids.



[3080] **Figure 1.** Solid nests and lobules of poorly differentiated neuroendocrine cells are seen in this liver biopsy A) Merkel cell chromogranin - Tumor cells are strongly and diffusely positive for chromogranin. B) Merkel cell CK20- Tumor cells show perinuclear dot-like reactivity to Cytokeratin20 C) Merkel cell synaptophysin- Tumor cells are strongly and diffusely positive for neuroendocrine marker Synaptophysin.

Table 1. Prior to initiating immunotherapy, serum transaminases rose slowly but remained within 3x upper limits of normal (ULN)

Laboratory Value	Prior to Admission On Immunotherapy	Day 1 Of Admission	After Initiating Steroids
Aspartate Aminotransferase (AST)	41 U/L	269 U/L	281 U/L
Alanine Aminotransferase (ALT)	46 U/L	315 U/L	240 U/L
Alkaline Phosphatase (ALP)	87 U/L	226 U/L	227 U/L
Total Bilirubin	0.3 mg/dL	0.7 mg/dL	1.1 mg/dL
International Normalized Ratio (INR)	1.06	1.15	1.18

At the end of treatment, transaminases reached >5 times ULN. No improvement was seen in liver enzymes after initiating empiric steroid therapy for suspected IMH.

S3081

Newfound Glow: The First Reported Case of Drug-Induced Liver Injury Due to Tofacitinib

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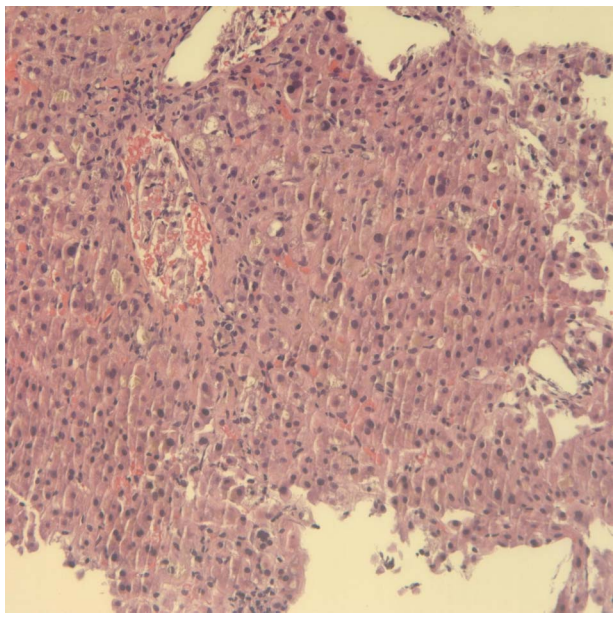
Introduction: Tofacitinib, a Janus kinase (JAK) inhibitor, is a medication used to treat autoinflammatory conditions. It has previously been associated with mild hepatocellular liver enzyme elevations, but here we describe the first reported case of a clinically apparent drug-induced liver injury (DILI) related to tofacitinib.

Case Description/Methods: A 29-year-old man with ulcerative colitis developed jaundice, malaise, anorexia, abdominal pain, diarrhea, and pruritus six months after starting tofacitinib. His only other medication was lisinopril. He consumes less than three alcoholic drinks weekly. Liver chemistries revealed aspartate aminotransferase (AST) 635 IU/L, alanine aminotransferase (ALT) 614 IU/L, alkaline phosphatase (ALP) 150 IU/L, total bilirubin (T-Bili) 3.2 mg/dL, and direct bilirubin (D-Bili) 1.7 mg/dL. Coagulation studies were normal. Abdominal ultrasound showed a diffusely hypoechoic liver and a diffusely thickened gallbladder wall but no biliary pathology. He stopped taking tofacitinib, and his liver chemistries peaked at AST 826 IU/L, ALT 1427 IU/L, ALP 182 IU/L, and T-Bili 6.5 mg/dL. Anti-smooth muscle antibody was slightly positive at 41. Additional workup included negative antinuclear antibody and negative serologies for cytomegalovirus, Epstein-Barr virus, and hepatitis A, B, and C. His symptoms improved, and he resumed tofacitinib two months after symptom onset. Four months later, the patient's symptoms recurred and labs redemonstrated acute liver injury with a new peak of T-Bili 9.1 mg/dL. Liver biopsy revealed acute hepatitis with diffuse severe hepatocanalicular cholestasis, inflammation, and necrosis, consistent with DILI. He stopped taking tofacitinib, and after one month his symptoms resolved and liver chemistries normalized. He was transitioned to vedolizumab and has remained asymptomatic. (Figure)

Discussion: This patient developed a clinically apparent liver injury with jaundice six months after initiating therapy with tofacitinib for ulcerative colitis. Other causes, such as viral hepatitis, alcohol-related liver disease, and autoimmune hepatitis, were ruled out. Biopsy results were consistent with DILI, and he was on no other culprit medications. He was rechallenged with tofacitinib and redeveloped liver injury, strongly supporting the diagnosis of tofacitinib-induced DILI. The mechanism of liver injury is not fully understood but might be attributed to drug metabolism via CYP3A4. More research should be conducted to better elucidate the mechanism of liver injury by tofacitinib.



[3081] **Figure 1.** Liver biopsy with acute hepatitis, severe hepatocanalicular cholestasis, inflammation, and necrosis, as well as a multinucleated hepatocyte, consistent with drug-induced liver injury in the setting of tofacitinib use.

S3082

Novel Approach in the Management of Large Portosystemic Shunt Causing Recurrent Hepatic Encephalopathy

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Introduction: Portosystemic shunting (PSS) is a common complication of advanced cirrhosis, and is usually a result of portal hypertension. In severe cases, it can lead to worsening hepatic encephalopathy (HE) leading to multiple hospitalizations. We present a patient with a large PSS who failed initial embolization of the PSS, requiring subsequent TIPS placement as well as re-embolization.
Case Description/Methods: A 55-year-old male with a history of non-alcoholic steatohepatitis (NASH) cirrhosis presented with altered mental status (AMS) for 2 days and worsening abdominal distension for a week. He did not report any fever, vomiting, or blood in stool. On chart review, he had multiple admissions for AMS secondary to HE. His caretaker reported compliance with home lactulose, rifaximin and zinc. A full infectious workup was negative. Abdominal ultrasound with doppler revealed no flow in the main portal vein. CT scan of the abdomen showed a massive PSS identified extending from the level of the splenic vein to the inferior vena cava, essentially shunting blood away from the portal vein. Patient underwent embolization of this PSS, however, this did not significantly decrease the blood flow through this shunt. Subsequently, a TIPS was placed to redirect flow into the portal vein and reduce flow in the shunt. Repeat embolization of the PSS was successful and resulted in almost no flow through the PSS with improved flow in the main portal vein. Since then, he has not had any further admissions with HE.
Discussion: Spontaneous PSS occurs as a result of worsening liver disease in cirrhotic patients, and their manifestations vary based on their location. In a study by Simón-Talero et al, ~50% of patients with large PSS had episodic and persistent HE. Patients with HE are usually treated medically with lactulose, rifaximin, or zinc, and treatment of the trigger (infection, GI bleeding, substance use). In a study on 25 patients with large PSS undergoing embolization, Lynn et al demonstrated a 100% success rate in immediate post-procedure improvement. However, in our patient, there was continued flow in spite of embolization of the shunt. Hence, we performed a TIPS procedure with repeat embolization of the shunt which ultimately minimized the blood flow through the large PSS and redirected flow into the portal vein. This novel modality of treatment may be beneficial in PSS which don't respond to embolization, and further studies may be needed to understand the benefit in such refractory cases.

S3083

Not All Cirrhosis Is Created Equal: A Rare Case of Regorafenib-Induced Pseudocirrhosis

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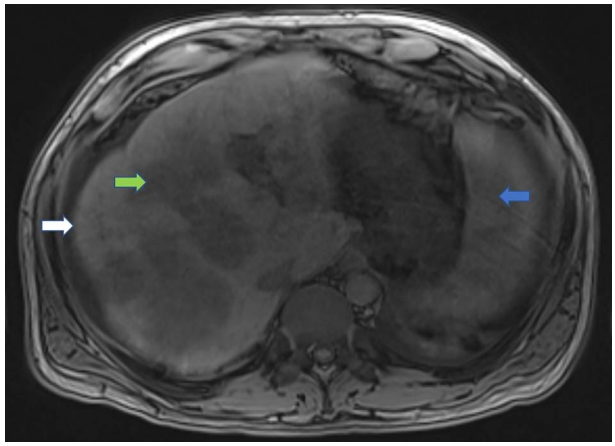
Introduction: Although chemotherapy has been shown to improve survival rates in metastatic colorectal cancer (mCRC), it can be associated with severe side effects. Regorafenib, an oral multikinase inhibitor, is used in treatment-refractory mCRC but carries a risk of developing pseudocirrhosis. We present a rare case of pseudocirrhosis resulting from regorafenib use.
Case Description/Methods: A 39-year-old male with Stage IV adenocarcinoma of the colon with metastatic liver lesions presented with a few days of diffuse abdominal pain and mild jaundice. CT abdomen showed cirrhotic liver morphology, ascites, splenomegaly, and non-obstructed biliary tract. He recently started regorafenib 4 months prior, after failing three prior chemo/immunotherapy regimens. Initial labs were – T Bili 5.5 mg/dl (direct 3.8 mg/dl), alkaline phosphatase 594 IU/L, GGT 272 IU/L, AST 55 IU/L, ALT 39 IU/L, and INR 1.4. MRCP demonstrated an interval increase in hepatic metastases burden with non-obstructed biliary tract. MRI abdomen is shown in Figure 1. Serology showed positive ANA 1:640 and elevated IgG. Infectious workup was negative. Ascitic fluid studies showed a SAAG of 2 without evidence of SBP or malignancy. Liver biopsy demonstrated normal lobular architecture, severe cholestasis, and focally prominent sinusoidal dilatation but was negative for cirrhosis, bridging fibrosis, sinusoidal obstruction, fatty change, or centrilobular necrosis. Portosystemic gradient ranged from 1-8 mmHg. He was started on spironolactone, furosemide, and ursodiol and underwent pre-emptive biliary stenting. Ultimately, the patient was diagnosed with regorafenib-induced pseudocirrhosis. Due to persistently increasing bilirubin, no further treatment options were available. He was offered hospice care.
Discussion: Pseudocirrhosis refers to the radiological appearance of cirrhotic liver morphology sans histological evidence of fibrosis. Regorafenib-induced pseudocirrhosis falls under the realm of idiosyncratic drug-induced liver injury. Although studies postulate nodular regenerative hyperplasia (NRH) as the causative pathology, no evidence of NRH was found in our case. The presence of concomitant cholestasis and sinusoidal dilation was indicative of a post-sinusoidal pathology. Given the increasing incidence of CRC, high suspicion and awareness about this condition are warranted.



[3083] **Figure 1.** MRI abdomen showing nodular liver contour (white arrow), metastatic disease (green arrow), and splenomegaly (blue arrow).

S3084

Metastatic Pancreatic Tail Adenocarcinoma Presenting With Multiple Hepatosplenic Abscesses: A Case Report

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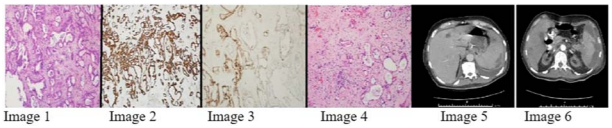
Introduction: Pancreatic cancer is the third leading cause of cancer-related deaths in the United States. It is often detected in advanced stages and may present in unusual ways such as pyogenic liver abscesses (PLA) or splenic abscesses which may be a diagnostic and therapeutic challenge.
Case Description/Methods: A 78-year-old dialysis-dependent man with a history of end stage renal disease, hypertension and cerebrovascular accident was evaluated for diffuse abdominal pain and altered mental status for one day. The examination was pertinent for generalized weakness and altered sensorium. Laboratory tests revealed transaminitis and leukocytosis. Computed tomography (CT) of the abdomen and pelvis showed multiple hypoattenuating hepatic foci with hepatosplenomegaly. A set of blood cultures grew Clostridium inoculum. A pyogenic liver abscess was diagnosed, drained percutaneously and the culture grew pansensitive Escherichia coli. A follow up CT of the abdomen and pelvis revealed a large splenic abscess. Exploratory laparotomy, splenectomy, liver biopsy and peritoneal lavage were performed. Immunostaining of the sample was positive for CK7, CK20 and CDX2. Pancreaticobiliary adenocarcinoma metastatic to the spleen and liver was confirmed by pathology. CA 19-9 and CA 125 were elevated. A repeat CT of the abdomen with pancreatic protocol showed a pancreatic tail mass. Vancomycin resistant Enterococcus faecium grew on peritoneal fluid culture. Daptomycin and ceftriaxone were started. The patient improved clinically but he was deemed unsuitable for further invasive procedures or chemotherapy. (Figure)
Discussion: Uncommon presentations of pancreatic adenocarcinoma include gastrointestinal (GI) bleeding from metastasis and acute abdomen from splenic infarct. PLA is associated with GI malignancies including pancreatic cancer. Some experts recommend cancer evaluation for GI malignancies in patients with PLA. Mucosal barrier breach by cancer cells, translocation of bacteria into the circulation and

inoculation at metastatic sites are possible explanations for developing liver and splenic abscesses. Dialysis is also a risk factor for hepatosplenic abscess. In an elderly patient with hepatosplenic abscess and elevated tumor markers, a thorough evaluation for GI malignancies including pancreatic cancer should be done.



[3084] **Figure 1.** Image 1 shows cancer in the spleen. Images 2-3 show positive immunostains for CK7 in image 2, CK20 in image 3. Image 4 shows the cancer in the liver biopsy (no normal liver is present). Image 5 shows non-enhancing hypoattenuated foci in the right and left lobes of the liver most consistent with multifocal liver abscesses. Image 6 shows splenomegaly with multiple hypoattenuated foci and a mass within the tail of the pancreas.

S3085

Molecular Adsorbent Recirculating System in Acute Liver Injury

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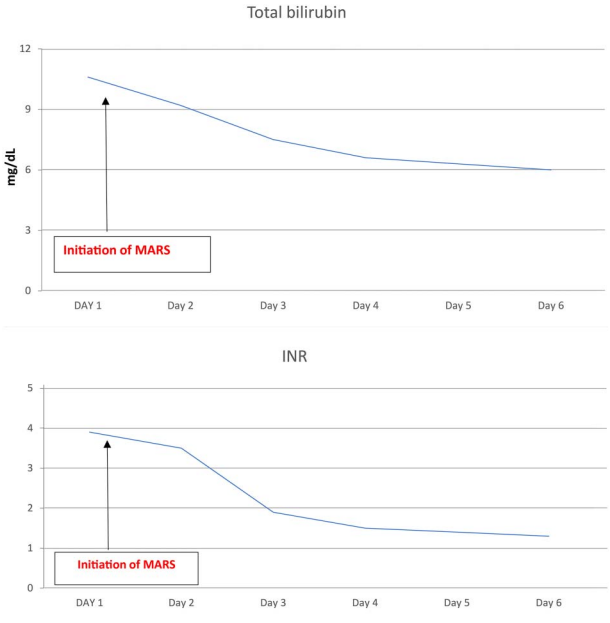
Introduction: Acetaminophen (APAP) toxicity is common and has been known as a cause of acute liver failure (ALF). ALF has high mortality rate, and in many cases, liver transplantation (LT) is the only lifesaving treatment. Unfortunately, many ALF patients are not candidates for transplant due to many factors. In addition, Hemodialysis have minimal to no effect on liver detoxification. Alternative approach could be albumin dialysis using Molecular Adsorbent Recirculation System (MARS). The evidence of effectiveness of MARS is limited with very few clinical studies. Here we report a case of ALF in an adult due to APAP toxicity who was not a candidate for LT, and treatment therefore included the use of MARS.
Case Description/Methods: A 44 y/o M with hx of heavy alcohol use presented with altered mental status. It was reported that he may have taken 40 APAP tablets over the last 1-2 days. Physical exam was significant for confusion, tachycardia and scleral icterus. CMP: total bilirubin of 10.6 mg/dL, alkaline phosphatase 195 U/L, AST 5605 U/L, ALT 7000 U/L, albumin of 2.6 g/L and INR of 3.9. Ammonia was 215 umol/L and Tylenol of 75 ug/ml. Autoimmune workup, viral hepatitis panel and a broad infectious workup were negative. Alpha 1 antitrypsin and ceruloplasmin were normal. US of the liver was normal. Etiology of ALF believed to be due to APAP toxicity. He was admitted to the ICU and started on N-acetylcysteine infusion. He was evaluated for LT and deemed to be not a candidate given psychosocial concerns. After multidisciplinary discussion, MARS was initiated and planned for 5 days. The patient was monitored with serial CMP and coagulation studies. Mental status, INR, bilirubin and hepatic transaminases rapidly improved while on MARS. Patient ultimately became clinically stable to be transferred to a regular medical floor and continued to show improvement in his liver functions. (Figure)
Discussion: In USA, ALF due to APAP toxicity is very common. LT is the only intervention with known survival benefits in ALF, but many factors can play a rule in LT evaluation and can lead to delay or might exclude patients from being candidates for LT, as seen in our patient. MARS, works on the concept of removal of albumin-bound substances accumulating in ALF, may be an ideal bridge to spontaneous recovery during liver regeneration. MARS could be a promising treatment with improvement of clinical response, laboratory parameters and liver function. Further studies are needed to better understand the efficacy of MARS on ALF outcomes.



[3085] **Figure 1.** Total Bilirubin and INR levels before, during and after MARS treatment in a patient with Acute Liver Failure. The arrows indicate the first treatment session.

S3086

New Diagnosis of Systemic Amyloidosis in a Patient With Chronic Hepatitis B and Elevated Liver Stiffness Score on MR Elastography

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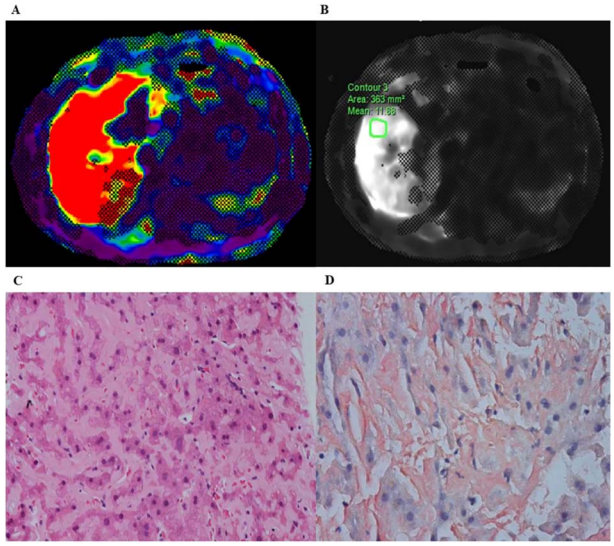
Introduction: Amyloidosis is a disorder of abnormal deposition of protein into bodily tissue. This case describes a patient with chronic hepatitis B (HBV) who was found to have systemic amyloidosis after a magnetic resonance elastography (MRE) was consistent with advanced fibrosis. This case highlights the importance of confirming concerning MRE results with a liver biopsy.
Case Description/Methods: A 58-year-old male initially evaluated for chronic HBV on Tenofovir Disoproxil Fumarate since 2009. He reported short-term memory defict, impaired concentration, weight loss, and anorexia for 2 years. Physical examination was unremarkable. Labs included normal CBC, LFTs, and INR, baseline creatinine, and undetectable HBV DNA. Shear wave ultrasound elastography (SWR) revealed increased echogenicity and mild steatosis, but liver stiffness (LS) measurements could not be obtained. After options for further testing were discussed, the patient elected to have a MRE (Fig. 1A and B). He was found to have a LS measurement of 11.75 kPa consistent with cirrhosis. Liver appeared normal on T2 without steatosis. No stigmata of chronic liver disease or contour nodularity was observed. He ultimately had a non-focal liver biopsy which revealed diffuse deposition of amorphous congophilic material consistent with amyloid involving sinusoids and portal tracts, mild increased portal and periportal fibrosis (stage I-II/IV), and mild chronic portal inflammation. Findings were consistent with amyloidosis (Fig. 1C and D). Amyloidosis was typed by laser capture liquid chromatography and tandem mass

spectrometry as AL–lambda. Further work up ruled out renal and cardiac involvement. Bone marrow biopsy revealed lambda-typic plasma cell dyscrasia. The patient is being treated with daratumumab and cyclophosphamide + bortezomib + dexamethasone.

Discussion: SWE and MRE provide noninvasive information about fibrosis in patients with chronic liver disease. This case highlights the importance of confirming the stage of fibrosis with liver biopsy, especially if the clinical picture is not consistent with LS measurements. In this case, the LS measurement was elevated due to hepatic amyloidosis rather than cirrhosis. Amyloidosis has a wide variety of presentations. The most common findings in hepatic amyloidosis are hepatomegaly and elevated alkaline phosphatase, none of which was present in our case. Gastrointestinal and hepatic involvement are usually associated with worse outcomes, thus early diagnosis is essential.



[3086] **Figure 1.** (A and B) MR elastography with (A) showing the sampleable portion of liver parenchyma demonstrating elevated stiffness as colored red. (B) is one of four liver samples used to calculate liver stiffness measurement. (C and D) Non-focal liver biopsy 20× H&E and 40× congo red stain respectively, showing deposition of amorphous congophilic material in sinusoidal tracts.

S3087

Microwave Failure

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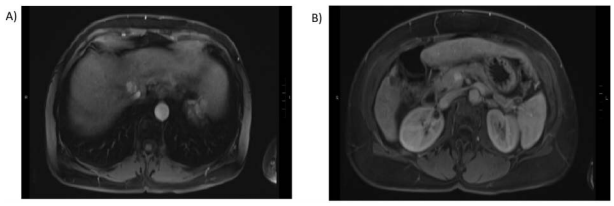
Introduction: Microwave ablation (MWA) is a nonsurgical option for patients with hepatocellular carcinoma but is not without risk. In the following patient, MWA was used to treat hepatocellular carcinoma (HCC) secondary to hepatitis C and led to acute liver failure.

Case Description/Methods: A 60-year-old Caucasian female with cirrhosis secondary to chronic untreated hepatitis C and was found to have HCC with a 3 cm mass in segment V, a 2.4 cm nodule in segment IVa on MRI. The patient's cirrhosis was well compensated with no evidence of portal hypertension, ascites, coagulopathy, encephalopathy, or gastrointestinal bleeds and her MELD score was 8. She underwent four rounds of transarterial chemoembolization for her HCC with still viable residual tumor and was recommended to have CT guided microwave ablation (MWA). Two days following her MWA the patient was found unresponsive. She was intubated and required the initiation of pressors due to persistent hypotension. On arrival and was found to have a WBC of 23.5, lactate 5.9, Cr 3.31, AST 5050, ALT 5681, ALK Phos 1489, total bilirubin 11.2, INR 8.7, calculated MELD of 47. A liver duplex demonstrated no evidence of thrombosis, and a full infectious workup was negative along with Tylenol and aspirin levels. She developed anuric renal failure and her condition continued to deteriorate until she passed away (Figure 1).

Discussion: Features of acute liver failure include liver enzymes elevated greater than 10 times the upper limit of normal, increased INR, and hepatic encephalopathy, which were all present in this patient. Differential diagnosis for acute liver failure must always include viral, autoimmune, metabolic, vascular and drug induced etiologies. MWA has well documented adverse effects including hemorrhage, thrombosis, and biliary tract manipulation, but acute liver failure without evidence of these findings is rarely found in literature. Alone, any of these adverse effects can cause liver failure, but as evidenced in this patient MWA itself is also a risk factor for acute liver failure. When presented with a patient who is actively undergoing MWA and has acute liver failure, one must consider MWA as an etiology in their differential diagnosis along with ruling out other common causes.



[3087] **Figure 1.** MRI showing two hepatocellular carcinoma lesions measuring 1.8 cm and 14 mm respectively, following 4 rounds of transarterial chemoembolization.

S3088

Nasopharyngeal Hepatocellular Carcinoma After Orthotopic Liver Transplant

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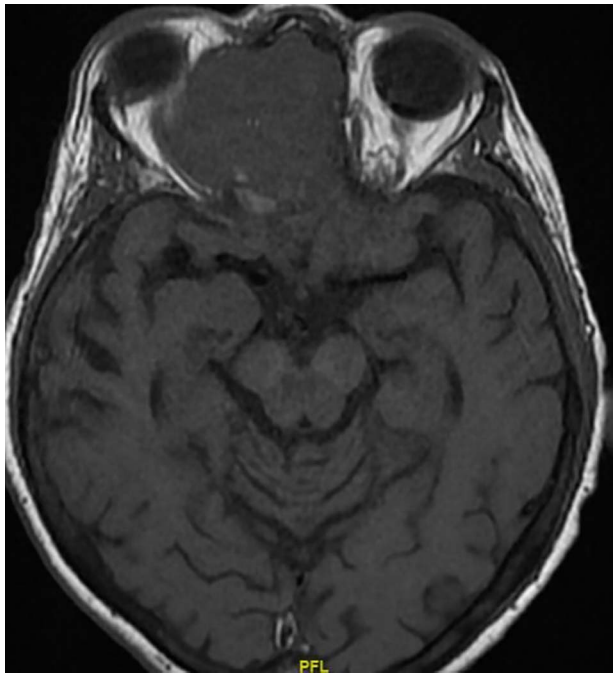
Introduction: Hepatocellular carcinoma (HCC) is the fifth-most common cancer in the world and the third leading cause of cancer-related death. It accounts for 75% of liver cancers, with rapidly rising incidence rates in the USA. Treatment includes locoregional therapy, chemotherapy, surgery, and transplant if within Milan Criteria. Extrahepatic metastases of HCC occur in 30%-50% of patients. The most common sites include the lungs, lymph nodes, bone, and brain. Here we present a rare case of nasopharyngeal metastases of HCC after liver transplant.

Case Description/Methods: A 76-year-old female with alcoholic cirrhosis complicated by HCC with known metastases to the lungs and kidney status post (s/p) locoregional therapy and orthotopic liver transplant with an HBV core antibody positive donor liver (within Milan criteria), and non-Hodgkin's lymphoma s/p XRT in remission presented to the ER with one month of right eye swelling and rhinorrhea without fever. Vital signs were significant for chronic asymptomatic bradycardia and hypertension. Physical examination with right orbital swelling without pupillary or extraocular muscle abnormalities. No

neurological deficits were noted. Liver enzymes were AST 9 U/L, ALT 3 U/L, total bilirubin 0.4 mg/ dL (direct 0.1), INR 1.2. Serum AFP one month prior to presentation was 24.5 ng/mL. CBC showed WBC of 3.7 K/UL, hemoglobin 8.2 g/dL and PLT 186 K/UL. Computed tomography showed a large expansile infiltration centered at the right ethmoid and upper nasal cavity extending to the superior medial aspect of the orbit, with associated mass effect upon the frontal lobes. Nasopharyngeal biopsies revealed poorly differentiated adenocarcinoma with immunostaining positive for heppar-1 compatible with metastatic HCC. The patient underwent bifrontal craniotomy for resection of the anterior skull base lesion, with a hospital course complicated by encephalopathy and sepsis necessitating ICU. The patient was discharged on comfort measures and hospice (Figure 1).

Discussion: HCC metastasizing to the nasopharynx is exceedingly rare. The first case report documenting an isolated nasopharyngeal metastasis from a liver primary was described by Kattepur et al in 2014. In our case, the patient reported swelling behind the right eye as the initial presentation of a metastatic HCC after liver transplant. In patients with history of HCC, clinicians should maintain a broad differential with clinical suspicion for uncommon presentations of extra hepatic metastases, even after liver transplant.



[3088] **Figure 1.** T1 weighted MRI orbit without contrast showing space occupying lesion extending through the right ethmoid sinuses with intracranial extension.

S3089

Monoclonal Immunoglobulin Deposition Disease: A Rare Case of Acute Liver Failure

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Introduction: Light chain deposition disease (LCDD) is a characterized by deposition of immunoglobulin light chain in extracellular tissue. Classically it involves the kidneys, however, can rarely affect other organs such as liver. Its manifestation is mostly non-specific and presents as mild cholestatic liver injury and, acute liver failure is extremely rare.

Case Description/Methods: An 83-year-old female with Monoclonal Gammopathy of Undetermined Significance (MGUS) was admitted to the hospital for dyspnea and anasarca. Her initial labs were remarkable for mild, predominantly cholestatic, pattern of liver injury. Additional workup was negative for common etiologies of liver injury, with normal appearance of liver and new-onset diastolic cardiac dysfunction. Initial diagnosis was acute liver injury in the setting of recent heart failure and treatment with aggressive diuresis was commenced. However, she progressed to acute liver dysfunction and multi-organ failure manifesting as encephalopathy, jaundice (T.Bili 16.25) and coagulopathy (INR 1.3). Her Alk-Phos was 1116 and AST, ALT were 289 and 95, respectively. Liver biopsy revealed deposition of pink hyaline material expanding the sinusoids. This was initially suspected to be amyloid, until a positive PAS and negative Congo-Red stain established the diagnosis of LCDD. SPEP revealed a M-protein light chain with elevated κ/λ ratio of 16.6. Chemotherapy was rejected considering poor prognosis and patient was discharged on hospice care.

Discussion: LCDD results from excessive production and deposition of monoclonal light chains into various organs including liver. Concurrent plasma cell dyscrasias and multiple myeloma are often seen in these patients. High suspicion of disease and biopsy without delay is imperative for diagnosis, which typically show granular, κ LC predominant deposits in the perisinusoidal basement membrane which unlike amyloid, appear unorganized and stain positive with PAS and negative with Congo-Red. At present, there is no consensus on LCDD treatment, however, autologous stem cell transplant following bone marrow conditioning with high dose melphalan and bortezomib based induction chemotherapy with or without systemic glucocorticoids have been reported to treat hepatic and systemic LCDD with some success.

S3090

Metformin-Induced Acute Hepatitis

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Introduction: Metformin is considered the initial oral pharmacotherapy of choice for the treatment of hyperglycemia in Type 2 Diabetes Mellitus (T2DM). Although safe in the vast majority of the population, rare side effects will come to light as the prevalence of T2DM continues to rise. We present a case of metformin induced hepatotoxicity.

Case Description/Methods: A 75-year-old male with T2DM, on metformin therapy, presented with a 1-month history of fatigue, nausea, vomiting, anorexia, and generalized abdominal pain. He had no preexisting liver disease or prior abnormal liver labs. He denied current use of alcohol, toxins, over the counter agents, herbal products, or new prescription medications. Of note, his metformin dose had been recently increased from 500 mg to 1000 mg twice daily a few weeks prior to presentation. On admission, laboratory work up was remarkable for a mixed hepatocellular and cholestatic pattern of liver injury as shown in Table 1. Abdominal imaging was negative for evidence of cirrhosis, portal hypertension, hepatic steatosis, or congestion. An extensive evaluation for other etiologies of acute hepatitis were unremarkable. Metformin was discontinued. Subsequently, patient's liver enzymes improved, the presenting symptoms resolved, and he was discharged in a stable condition. He followed up outpatient four weeks later and was found to be doing well with normalization of liver enzymes.

Discussion: Drug induced liver injury (DILI) is a well established problem and accounts for nearly 10% of all cases of acute hepatitis. Although common, diagnosing DILI can be difficult as no specific serum biomarkers or tests are available to reliably attribute liver injury to a drug. This can be especially challenging when hepatotoxicity is caused by a medication that is not considered intrinsically hepatotoxic such as metformin. In this case, a diabetic patient presented with symptoms of acute hepatitis after an increase in metformin dose, the diagnosis of metformin-induced hepatotoxicity was supported by the causal relationship between an increase in metformin and the onset of liver injury, exclusion of all other causes of liver injury, and recovery of liver function on discontinuation of metformin. With the rising burden of

T2DM worldwide, rare side effects of commonly used anti-diabetic medications will continue to emerge. Through our case report we aim to make clinicians aware of one such reaction, metformin induced severe idiosyncratic acute liver injury.

Table 1. Laboratory workup on admission was remarkable for a mixed hepatocellular and cholestatic patter of liver injury

Total Bilirubin	3.4mg/dl
Aspartate aminotransferase	3,241 units/L
Alanine aminotransferase	3,870 units/L
Alkaline Phosphatase	190 units/L

S3091

Noncirrhotic Portal Hypertension in Turner's Syndrome: A Case Report

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Introduction: Turner's syndrome is one the most common chromosomal aneuploidies in humans. Although it can affect multiple organs, involvement of gastrointestinal (GI) organs is rare.
Case Description/Methods: A 20-year-old female with history of Turner's syndrome with premature ovarian failure and mild mitral regurgitation was evaluated for easy fatigability and bruising. Laboratory assessment indicated pancytopenia. Peripheral blood smear showed a variety of red cell shape abnormalities and bone marrow biopsy showed normal cellular marrow. Computed tomography of the chest, abdomen and pelvis with and without contrast showed severe splenomegaly, tiny varices in the anterior mediastinum along the gastrohepatic ligament, and no portal vein thrombosis. Abdominal ultrasound examination revealed severe diffuse heterogeneity of the liver parenchyma with a slightly nodular hepatic contour and Fibroscan indicated mild steatosis S1 with advanced fibrosis F3. She underwent liver biopsy which showed focal mature fibrous expansion of some portal tracts, with delicate bridging fibrosis, as well as patchy increase in the number of small portal arteries with occasional increased thickness of arterial wall, subtle lobular parenchymal changes, suggestive of porto-sinusoidal vascular disease with no evidence of cirrhosis. Upper GI endoscopy revealed grade 2 esophageal varices and erythematous mucosa in stomach, suggestive of portal hypertension. Considering the definitive clinical signs of portal hypertension (gastroesophageal varices), the absence of cirrhosis on liver biopsy and histologic signs of porto-sinusoidal vascular disease, she was diagnosed with non-cirrhotic portal hypertension due to porto-sinusoidal vascular disease. She was started on propranolol for gastroesophageal varices and her heart rate is currently well-controlled. Follow-up abdominal ultrasound is negative for further changes in the liver size, morphology, or masses.
Discussion: Turner's syndrome might be associated with non-cirrhotic portal hypertension. High index of clinical suspicion can lead to early diagnosis and treatment of portal hypertension in individuals with Turner's syndrome, reducing the burden of complications of portal hypertension such as cytopenia or bleeding.

S3092

Metastatic Hepatic Epithelioid Hemangioendothelioma: A Case Report

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Introduction: Epithelioid Hemangioendothelioma (EHE) is a very rare vascular neoplasm consisting of epithelioid or histiocytoid cells that have endothelial characteristics. EHE has an incidence of 0.038/100,000/year peaking in the 4th-5th decade and a prevalence of <1/1,000,000 with a slight predominance in females. EHE can arise anywhere throughout the body but mostly involves the liver, lungs, and bone with >50% of patients presenting with metastatic disease. The variability of its nature and clinical course makes uniform staging, treatment, and prognosis difficult. Several possible causative factors have been recommended; however, they remain as loose associations or still potentially undiscovered.
Case Description/Methods: We present a noteworthy case of this rare tumor in a previously healthy 47-year-old male, non-smoker, 1-2 EtOH 2-3 nights/wk, family history of skin cancer, UC, and lung cancer, that presented with 2 weeks of nonspecific symptoms. Initial presentation was concerning for cholecystitis; therefore, an US and CT were performed finding a complex lesion in the right hepatic lobe measuring 6.5 × 5.3 cm. CT guided core needle bx was significant for atypical cells seen within a somewhat loose myxoid background, with occasional cytoplasmic vacuoles. Immunohistochemistry revealed (+) CD31/34 and a strong (+) CAMTA-1 nuclear staining which confirmed EHE. Additional CT chest showed several small bilateral pulmonary nodules, the largest measuring 1.1cm in the left upper lobe concerning for mets. He underwent a right total hepatectomy with associated cholecystectomy and a wedge bx of a suspicious lesion in the right lateral abdominal wall parietal peritoneum overlying the diaphragm.
Discussion: Given diagnosis of hepatic EHE with stage IV mets, Oncology planned for CT chest/abd/pelvis as part of standard surveillance with additional imaging only as indicated for symptoms not attributable to other causes. Given his small and indeterminate pulmonary nodules, close observation was favored with follow-up and repeat imaging q3mX1yr then q4mX1yr then q6mo presuming stable disease. With no standard of care for EHE, chemotherapy typically uses drugs for other soft tissue sarcomas or anti-angiogenic approaches and would be important if he has disease progression. 15 months post-op imaging showed unchanged bilateral pulmonary nodules, with stable to slightly small seroma, unchanged left renal lesions, unchanged omental infiltration, and nodularity particular in the right upper quadrant suspicious for sarcomatosis.

S3093

Monotherapy for Multiple Autoimmune Diseases: The Novel Use of Ofatumumab for Concurrent Multiple Sclerosis and Autoimmune Hepatitis

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Introduction: Autoimmune hepatitis (AIH) is a rare acute or chronic inflammatory liver disease, that can be associated with a variety of extrahepatic disorders, such as multiple sclerosis (MS). AIH has been reported both during and after treatment with immunomodulatory drugs for MS, such as interferon-beta and steroids, with its incidence in MS being 10-fold higher than the general population. Typically, those patients are treated with steroids and azathioprine as the first line of treatment according to AIH guidelines. However, in comparison to newer disease-modifying therapies (DMTs) for MS, azathioprine is less effective and requires further study. Notably, several studies have suggested a role for B-cell-driven autoimmune liver injury in AIH, and there have been reports of refractory AIH being successfully treated with rituximab, an anti-CD20 monoclonal antibody. Based on these results and the well-known role of B-cells in the underlying pathophysiology of MS, we postulated that anti-CD20 therapy may be effective in treating patients with concurrent MS and AIH, thereby helping to limit steroid duration and avoid the use of azathioprine.
Case Description/Methods: We report the case of a 49-year-old previously healthy female who presented with symptoms and magnetic resonance imaging (MRI) findings consistent with relapsing-remitting MS (RRMS). On routine labs at initial presentation, she was incidentally found to have ALT/AST in the 500's range. An extensive work-up was completed, including a liver biopsy that showed a centrilobular necroinflammatory injury pattern suggest of early AIH without cirrhosis. The patient was started on oral budesonide followed by ofatumumab, an anti-CD20 monoclonal antibody approved for treatment of RRMS, as steroids were weaned. The patient's liver enzymes returned to normal ranges within 12 weeks and have remained so now for approximately 8 months. Additionally, she has had no new clinical or imaging findings suggestive of MS disease activity. We present this as the first-reported case of the use of an anti-CD20 monoclonal antibody to treat both MS and AIH concurrently.
Discussion: This case furthers our understanding of AIH by suggesting that the pathophysiology contributing to AIH is predominately B-cell related as this patient's AIH was successfully treated with ofatumumab and a short course of budesonide. It highlights a possible alternative treatment modality for AIH that avoids a prolonged course of steroids and/or azathioprine. More research is still needed however.

S3094

Leptospirosis-Induced Acute Liver Injury

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Introduction: Weil's disease (Leptospirosis) is a relatively common worldwide zoonotic infection due to Leptospira spp. However, leptospirosis is underreported due to its low incidence in the United States and its variable presentation. Furthermore, leptospirosis-induced acute hepatic injury is extremely rare. The purpose of this case report to enlighten urban practitioners to consider testing for leptospirosis in patients with acute liver injury. We are reporting a rare case of leptospirosis induced acute liver injury.
Case Description/Methods: A 38 year-old-male with no past medical history presented with 6-day history of fever, abdominal pain, N/V, fatigue and myalgia. On presentation patient was vitally stable, physical exam, ill appearing man with icterus sclera, mild tenderness in RUQ abdomen. Labs: WBC 14.16, ALT 181, AST 232, Bilirubin 7.5 (direct bilirubin 6.2), platelets 41, INR 1.13, aPTT 19.7, BUN 33, Cr 3.41, GFR 21.6, CT abdomen revealed hepatic steatosis and hepatomegaly. Extensive hepatic and sepsis work up sent and patient was started on vancomycin, piperacillin/tazobactam. For next 4 days, WBC 25.22, Bilirubin 18, (direct bilirubin >10.0) with more yellow skin, however, ALT 68, AST 54, platelets 93 improved. Renal function worsened to BUN 58, Cr 4.05, GFR 17.5. All the extensive work up came back unremarkable, Leptospira IgM antibody was sent on day 5. The patient was transferred to the intensive care unit on day 6 for multi-organ failure. On day 8 leptospira IgM antibody came back positive and antibiotics were de-escalated to IV penicillin G. On day 12, patient had significant clinical and labs improvement, WBC 10.16, Platelets 347, ALT 59, AST 44, Bilirubin 4.2, (direct bilirubin 2.6), BUN 20, Cr 1.22, GFR >60. 2 weeks after hospital discharge, patient was feeling much better and his hepatic and renal function returned to baseline.
Discussion: Leptospirosis-induced acute liver injury is exceedingly rare in United States; hence, a high index of suspicion is required to make the diagnosis due to its variable clinical course. Most cases are mild, while some are severe and potentially fatal. In urban areas, the disease is mainly transmitted via rodent urine contamination of water and soil. With the increase use of various types of cycles, riders of these devices, as well as sewer workers and joggers, risk greater exposure to the disease. Providers, especially in urban settings, should have a high index of suspicion, and evaluate patients with acute liver injury for leptospirosis.

S3095

Recurrence of Hepatocellular Carcinoma After Orthotopic Liver Transplant

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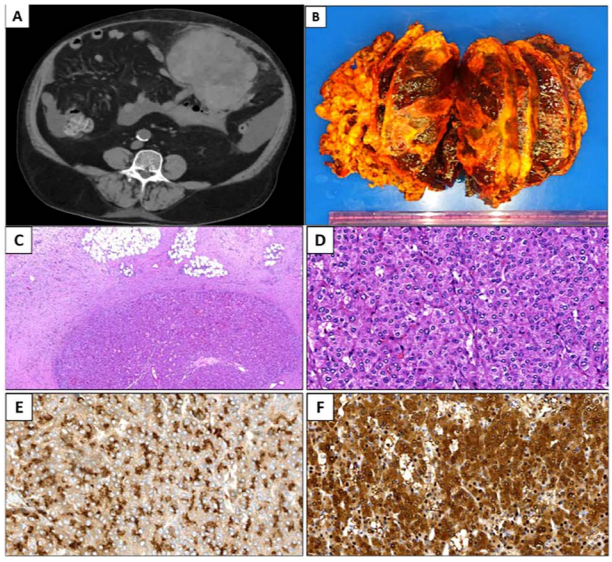
Introduction: Liver transplantation (LT) is the preferred treatment for hepatocellular carcinoma (HCC) in patients with decompensated cirrhosis. Recurrence of HCC after LT is rare, and high-risk features include viable tumor on explant, larger tumor size, elevated alpha fetoprotein (AFP), and micro/macrovascular invasion. The Risk Estimation of Tumor Recurrence After Transplant (RETREAT) score estimates HCC recurrence risk with a 5-year recurrence risk more than 75% in scores 5+. We present a case of extrahepatic recurrent HCC after LT.
Case Description/Methods: A 75-year-old male with history of alcohol-associated cirrhosis complicated by biopsy-confirmed HCC treated with radiofrequency ablation (RFA) underwent LT. His RETREAT score was 1 with negative surveillance scans through 1-year post-LT. The patient was lost to follow up and did not undergo surveillance. Three years post-LT, he developed diffuse abdominal pain and presented to a local hospital where contrast-enhanced computed tomography (CT) abdomen demonstrated an 11-centimeter omental mass with biopsy confirming HCC. This mass likely stemmed from tract seeding during RFA or pre-LT biopsy. AFP was normal. Subsequent positron emission tomography scan showed isolated uptake at the omental mass. The patient later re-presented to a local hospital with abdominal pain and fatigue and was found to be anemic with a hemoglobin 5 g/dL. The patient was transferred to our center where multidisciplinary discussion with hepatology, oncology, radiation oncology, and transplant surgery was conducted. CT abdomen revealed intraabdominal bleeding from the now enlarged mass (Fig. 1A), which necessitated resection (Fig. 1B). Pathology confirmed HCC (Fig. 1C–F). Post-resection surveillance imaging has not demonstrated evidence of disease.
Discussion: This case highlights the importance of continued HCC surveillance post-LT and the possible complications of RFA and biopsy. A diagnosis of HCC in cirrhotics can be made radiographically with the Liver Reporting and Data System (LI-RADS) criteria thus avoiding invasive diagnostics. Peak recurrence occurs within 2-3 years post-LT. The most common extrahepatic sites are lungs, bone, soft tissue, and peritoneum, and surgical resection is an independent predictor of long-term survival if metastatic disease is isolated to one organ. Systemic chemotherapy can be offered, but immunotherapy carries the risk of organ rejection in small studies. A multi-disciplinary approach should be pursued in patients with HCC recurrence post-LT.



[3095] **Figure 1.** CT image, gross image, histomorphology, and immunophenotype of the omental mass. (A) Extrahepatic HCC presenting as omental mass. (B) An omentectomy specimen was received consisting of a tan-yellow, hemorrhagic segment of omentum measuring 21.5 × 17.6 × 9.3 cm. Serial sections of the specimen revealed a 15.1 × 12.5 × 9.2 cm tan-white, hemorrhagic, thinly encapsulated mass. Low-power magnification demonstrates a cohesive, well-circumscribed lesion infiltrating the omental connective tissue (C, H&E stain, 20×). Intermediate magnification shows the lesion is composed of trabecular and solid sheets of atypical polygonal cells with nuclear pleomorphism, eosinophilic cytoplasm and thickened hepatic cell plates morphologically consistent with metastatic hepatocellular carcinoma (D, H&E, 200×). The neoplastic cells demonstrate strong and diffuse staining with Glypican-3 (E, 200×), and Arginase-1 (F, 200×), thus confirming the diagnosis.

S3096

Autoimmune Hepatitis Flare Following COVID Vaccination: A Case Series

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Introduction: We present a case series of patients with autoimmune hepatitis experiencing a flare following COVID vaccination.
Case Description/Methods: A 62-year-old male presented to the clinic with right upper quadrant pain associated with elevated liver enzymes. This patient's lab and liver biopsy results were consistent with autoimmune hepatitis. He remained stable on mycophenolate mofetil (MMF) for nine years without any acute flares. However, two weeks after receiving the first COVID Pfizer vaccine, the patient presented with jaundice and severely elevated liver enzymes - AST: 830, ALT: 1,450, and total bilirubin: 7.6. He was subsequently hospitalized and required IV solumedrol for seven days. The patient was transitioned to oral steroids and his liver enzymes normalized within a month. A 41-year-old female with a past medical history of lupus was found to have elevated liver enzymes on yearly labs. This patient's lab and liver biopsy results were consistent with autoimmune hepatitis. She was responsive to a stable regimen of prednisone and MMF for eight months. However, three weeks following her fourth COVID Pfizer vaccine, her

routine labs exhibited AST: 344, ALT: 464, and alkaline phosphatase: 265. These values were a significant increase from normal the month before. This necessitated up-titration of prednisone and MMF, which normalized her liver enzymes within two months. A 67-year-old female with a past medical history of hypothyroidism was found to have elevated liver enzymes associated with fatigue. Further lab work-up and liver biopsy revealed autoimmune hepatitis. This patient's autoimmune hepatitis was controlled on a regimen of prednisone and MMF for six months. However, three weeks after receiving the third COVID Pfizer vaccine, this patient's labs exhibited AST: 99 and ALT: 105. These values were a significant increase from normal the month before. Her immunosuppressants were up-titrated with an increase in MMF and the addition of cyclosporine. Her labs improved to AST: 73 and ALT: 87 within two months.
Discussion: These cases demonstrate that COVID vaccination may play a role in autoimmune hepatitis flares. This can be a challenging situation for many clinicians to navigate, as COVID remains a significant threat to patients' health, and there are many case reports that show that COVID infection itself can precede a flare. Patients with autoimmune liver disease may benefit from closer laboratory evaluation surrounding COVID vaccination.

S3097

A Rare Cause of Severe Hepatitis: Non-Typhoidal Salmonella

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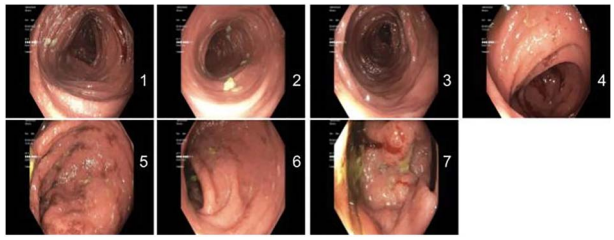
Introduction: Non-typhoidal salmonella (NTS) are food borne pathogens that often result in self-limited gastroenteritis. Derangements in transaminases, indicative of acute hepatitis are seen in 1% to 26% of enteric fever cases caused by salmonella typhi and paratyphi species. There have been very few case reports of hepatitis in non-typhoidal species. We present a case of a young man with severe gastroenteritis and elevated liver enzymes, found to have non typhoidal salmonella enteritis, hepatitis, and bacteremia.
Case Description/Methods: We report a 33-year-old man who presented to our emergency department with 1 week of nonbilious, non-bloody emesis with associated green watery diarrhea. He reported eating at a buffet in Jamaica one week prior, with his family having similar, yet milder symptoms. On physical exam he was noted to have scleral icterus and diffuse abdominal tenderness. CT of the abdomen and pelvis was unremarkable. Viral hepatitis serology was negative for hepatitis A IgM, Hepatitis B immune, Hepatitis C negative, Hepatitis E IgM and IgG negative. Trends of the patient's liver panel can be seen below (Table 1). Serum markers of autoimmune hepatitis were negative. EGD and colonoscopy were performed on day 4 due to persistent symptoms which showed gastritis as well as inflammation throughout the colon. Stool PCR resulted positive for Salmonella enterica on day 4 of hospitalization despite two negative stool cultures. He was started on Ceftriaxone as blood cultures revealed pan-sensitive group D salmonella bacteremia. He was discharged home to complete a 14 day course of antibiotics with levofloxacin 500 mg daily given symptomatic and laboratory improvement (Figure 1).
Discussion: Salmonella hepatitis is a rare, yet well documented complication of typhoid salmonella and is hypothesized to occur due to direct invasion of the organism or endotoxin mediated immune liver injury. Salmonella hepatitis can often mimic viral hepatitis. An admission ALT/LDH ratio is a good discriminator between both entities. A ratio greater than 9 can demonstrate viral hepatitis, while less than 9 can be seen in typhoid hepatitis. Our presented patient is unique in that he demonstrated salmonella hepatitis with the non-typhoid serotype. We conclude that non-typhoid salmonella may have the same capacity for hepatic injury as does typhoid salmonella likely through a similar mechanism and should be suspected as the cause of hepatitis when other sources are ruled out in patients infected with salmonella enterica.



[3097] **Figure 1.** Scattered moderate inflammation characterized by altered vascularity, congestion (edema), erythema, friability, loss of vascularity and serpentine ulcerations was found in the transverse colon (1, 2), splenic flexure (3), descending colon (4, 5), sigmoid colon (6), and rectum (7).

Table 1. Liver Panel

	Day 1	Day 4	Day 7	Day 16
AST (unit/L)	253	258	712	155
ALT (unit/L)	276	454	1,593	569
ALP (unit/L)	74	68	130	109
Total Bilirubin (mg/dL)	1.2	1.0	1.5	0.4
Direct Bilirubin (mg/dL)	0.7	0.6	-	-

AST = Aspartate transaminase, ALT = Alanine Aminotransferase, ALP = Alkaline phosphatase

S3098

Vanishing Bile Duct Syndrome in the Setting of Newly Diagnosed Hodgkin Lymphoma

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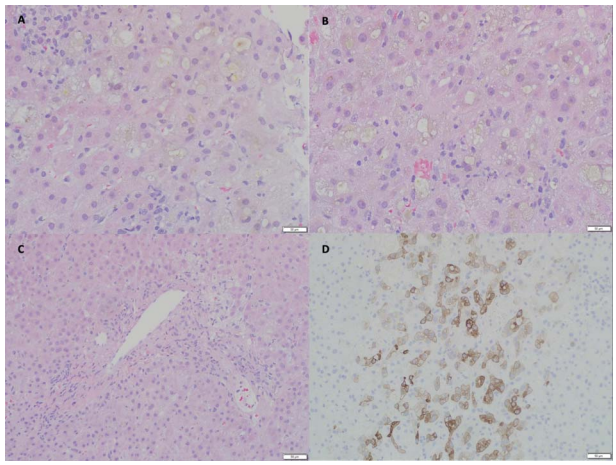
Introduction: Vanishing bile duct syndrome (VBDS) is characterized by cholestatic liver disease in the setting of disappearing intra-hepatic bile ducts. It can resemble other forms of cholestatic liver disease; however, imaging and biochemical tests will be unrevealing. We describe a case of a woman with Hodgkin lymphoma (HL) who developed cholestatic hepatitis with loss of intra-hepatic bile ducts.
Case Description/Methods: A 23-year-old woman with a history of HL who presented for evaluation and treatment of HL. Two months prior she had been treated for CMV-associated gastroenteritis and sepsis. Initial examination revealed jaundice and bilateral conjunctival icterus. Lab results showed total bilirubin of 18.1 mg/dL, ALT 363 U/L, AST 149 U/L, alkaline phosphatase (ALP) of 2392 U/L, direct bilirubin of 15.5 mg/dL, INR of 2.07, and cytomegalovirus (CMV) PCR at 670 IU/mL. Other acute viral hepatitis studies, tests for intrinsic liver disease, and autoimmune markers were negative. Computerized tomography (CT) of the abdomen and pelvis showed extensive lymphadenopathy, sclerotic changes throughout the skeleton, splenomegaly, and hepatomegaly without focal liver lesions. Magnetic resonance cholangiopancreatography (MRCP) of the abdomen showed no evidence of biliary ductal dilatation. Treatment for her HL was initiated, as well as foscarnet for her CMV viremia. Liver biopsy showed benign liver parenchyma with marked cholestasis and paucity of bile ducts, no cirrhosis, and no viral inclusions. The patient was initiated on ursodeoxycholic acid (UDCA) at 15 mg/kg/d without resolution of symptoms or cholestasis. Given no improvement in her cholestasis, the prospects of liver transplantation (LT) were discussed, but the patient was not a candidate (Figure 1).
Discussion: Liver involvement in HL typically manifests as parenchymal invasion, external compression, or paraneoplastic destruction of bile ducts (VBDS). VBDS tends to signify a poor prognosis with patients frequently progressing to liver failure. The pathogenesis of VBDS in HL is not yet defined with current evidence suggesting an immune-mediated response. Typically, viral causes should be excluded. In this case, the patient did have CMV viremia. Given her prior treatment, low viral load, and absence of viral inclusions on liver biopsy, CMV was excluded as a cause. Treatment for VBDS revolves around treating the underlying cause. UDCA can be used as a temporizing measure, but, if no improvement is observed over time, patients are referred for liver transplant.



[3098] **Figure 1.** Parenchymal liver biopsy: (A) and (B) Lobular cholestasis. The hepatic lobules show cholestasis within the hepatocytes and bile canaliculi. (C) Paucity of intrahepatic bile duct. No bile duct is seen in the portal tract. (D) CK-7 positive hepatocytes in chronic cholestasis. The hepatocytes in this case stain positive for CK-7.

S3099

A Case of Autoimmune Hepatitis Disguised as Hemochromatosis

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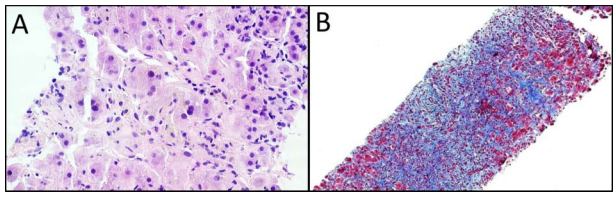
Introduction: The diagnosis of autoimmune hepatitis (AIH) requires excluding other etiologies of liver disease and the presence of autoantibodies and elevated serum globulins. Autoantibodies, however, can be absent in some cases making diagnosis difficult. We report a case of seronegative AIH and lab findings suggestive of hemochromatosis (HC) that proved misleading.
Case Description/Methods: A 65-year-old female with a history of SLE (on maintenance prednisone) and hypothyroidism presented with abdominal pain, nausea and jaundice. She had discontinued prednisone a month prior to presentation. No history of alcohol or herbal supplements use and no prior blood transfusions. Exam was unremarkable except for jaundice. Labs were significant for elevated liver enzymes, iron, ferritin and transferrin saturation (Table 1), negative acute viral hepatitis, ANA, ASMA, AMA, A1AT, and ceruloplasmin, elevated IgG, and no mutations in HFE genes. Liver biopsy was consistent with severe, active AIH (Fig. 1). She scored 4 points on Simplified Diagnostic Criteria for AIH (SDC), indicating possible AIH, and scored 18 points on Revised Diagnostic Criteria (RDC), indicating definite AIH. She was treated with steroids and maintained on azathioprine with clinical and laboratory recovery (Table 1).
Discussion: Our patient with a history of autoimmune diseases was likely genetically predisposed to AIH, which manifested after she discontinued her prednisone. She had negative autoantibodies, which is seen in ~10% of AIH cases and is associated with a more aggressive course. Delayed diagnosis can lead to rapid progression of disease. In these cases, diagnosis is mainly based on histological findings and the response to steroids. Also, supplemental use of RDC can be more helpful than SDC in seronegative AIH. Her elevated ferritin and transferrin saturation falsely suggested HC, however, given the acuity of her presentation, absent HFE genetic mutations, and biopsy findings, HC was ruled out. Ferritin, an acute phase reactant, can be elevated with AIH, however, transferrin saturation is expected to be normal. It is hypothesized that with severe liver injury causing decreased transferrin synthesis, and in presence of elevated serum iron, transferrin saturation can seem falsely elevated. This case highlights a rare presentation of seronegative AIH associated with elevated transferrin saturation, and the importance of liver biopsy and use of RDC score to avoid delay in diagnosis of a potentially aggressive disease.



[3099] **Figure 1.** (A) Severe interface hepatitis with marked acute and chronic inflammation including both neutrophils and lymphocytes and occasional plasma cell (H&E). (B) Trichrome stain showing extensive chicken-wire fibrosis (stage V of VI on Ishak's modified staging system) disturbing the architecture of liver and consistent with cirrhosis. Iron stain showed accumulation of hemosiderin predominantly in Kupffer cells consistent with siderosis (not shown).

Table 1. Trend of select liver function tests and iron studies from initial presentation to 1.5 months on immunosuppressant therapy

	Initial Presentation	1.5 Months on Treatment
Aspartate Aminotransferase (U/L)	696	39
Alanine Aminotransferase (U/L)	654	41
Alkaline Phosphatase (U/L)	258	109
Total Bilirubin (mg/dL)	8.2	1.0
Iron (mcg/dL)	219	122
Transferrin Saturation (%)	88	36
Ferritin (mcg/dL)	3,117	339