S3100

Renal Mass and Nonmetastatic Hepatic Dysfunction: Paraneoplastic or Autoimmune?

Andreas W. Bub, MD[1], Jonathan Kandiah, MD[2], Meghan Veno, MD[1].
[1]Stamford Hospital, Stamford, CT; [2]Norwalk Hospital, Norwalk, CT.

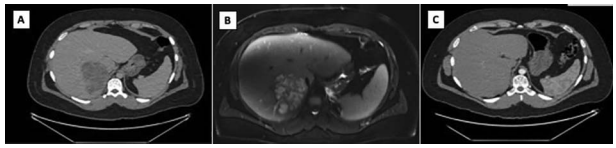
Introduction: Renal tumors fall into two classes of varied malignant potential, renal cell carcinoma (RCC) and renal epithelial stromal tumors (REST). Multilocular cystic renal neoplasm of low malignant potential (MCRNLMP), a type of RCC, shares many traits of RESTs. Adult Cystic Nephroma (ACN), a benign and incidentally found REST in adult women presents with bloating and hematuria, like RCC. RCC is typically malignant and linked to paraneoplastic disorders, such as Stauffer's Syndrome (SS). SS, a rare paraneoplastic disorder, causes cholestatic hepatitis without metastatic disease, resolving after tumor resection. Autoimmune Hepatitis (AIH) and Primary Biliary Cirrhosis (PBC) also share most characteristics but are differentiated by autoantibodies and pathology. We present a 62 year old woman with a renal mass and jaundice that improved post-nephrectomy but was later diagnosed with AIH/PBC.
Case Description/Methods: A 62 year old Woman with a 12-year history of a multicystic left renal mass presented with 10 days of jaundice. Urinalysis showed hyperpigmentation, serum studies were consistent with cholestasis and showed an autoantibody titer of 1:80. A CT scan showed the mass doubled in size as well as new portal lymphadenopathy. Liver biopsy showed nonspecific inflammation and cholestasis but no malignancy. Radical nephrectomy was performed with pathology showing ACN or MCRNLMP. The patient's liver function tests improved post-nephrectomy and were improving after 2 weeks. 6 months later she developed jaundice, hypergammaglobulinemia, and elevating autoantibodies titers of 1:160. Repeat imaging revealed no changes and she was diagnosed with AIH/PBC overlap.
Discussion: REST and RCC can be hard to distinguish, like ACN from MCRNLMP. They share a cellular origin plus histopathological and imaging findings. Moreover SS and AIH/PBC share most traits and even cause immune dysregulation in similar manners. The initial improvement post resection hints that her hepatic dysfunction was partly due to the tumor, though she suffered relapse due to an occult autoimmune disorder. The similarities between the malignancies and immune disorders involved may mean it is possible that the former led to the latter. A long history of indolent nephroma left time for a seronegative immune response to become a seropositive one. This case highlights ambiguity in diagnosing renal tumors, the need for prompt resection if paraneoplastic disorder arises, and the need to differentiate paraneoplastic disorders from autoimmune disorders.

S3101

When a Cyst Isn't Just a Cyst: A Rare Case of a Hydatid Liver Cyst Caused by *Echinococcus* in the United States

Amanda Eisinger, DO, Vishal Phogat, MBBS, Michael Sandhu, MD, Harvir Gambhir, MD, Savio John, MD.
SUNY Upstate Medical University, Syracuse, NY.

Introduction: Echinococcus Granulosus is a parasite that causes hydatid disease. It is endemic to many countries but is rarely seen in the US, particularly in the Northeast. We present a case of a 22-year-old American male with a hydatid liver cyst due to an Echinococcus infection.
Case Description/Methods: 22-year-old obese male presented to a local hospital for RUQ abdominal pain that radiated throughout his abdomen. He denied recent diet changes, travel or sick contacts.On presentation the patient had leukocytosis, transaminitis, hyperbilirubinemia and an elevated alkaline phosphatase. CT abdomen showed mild splenomegaly and a new 8.6cm diameter heterogeneous low density poorly enhancing mass in the right hepatic lobe. RUQ US revealed a heterogeneous irregularly marginated hypoechoic 9.6 × 9.5 × 5.7 cm soft tissue liver mass. Abdominal MRI showed a 9.3 × 7.3 cm complex, cystic and multilobulated lesion with thick walls and internal septations in the right hepatic lobe. He was transferred to our hospital for GI evaluation, where an US guided aspiration of the collection was recommended. 5 ccs of purulent aspirate were drained, sent for culture and a hepatic drain was placed. There was no evidence of malignancy in the aspirate. He then shared that over a year before his presentation, he served 9 months in Afghanistan with the US military. An infectious workup then showed serum positive for echinococcus antibody. Treatment was started with daily oral albendazole for Cystic Echinococcus (CE). Eight and half weeks later his pain had resolved and interval improvement with decreased abscess size was seen on CT (Figure 1).
Discussion: CE is common in South America, the Middle East, Africa and Asia. Most cases of CE in the US are found in immigrants from endemic countries. Local transmission has been reported in California, Alaska and the southwest US. Our case of an American male with CE highlights the importance of understanding the lifecycle of CE as there is often many years between ingestion of eggs and symptom onset. It also demonstrates the importance of obtaining enzyme-linked immunosorbent assay test for Echinococcus, which is 90% sensitive, prior to considering surgery for a cystic liver lesion. Rupture of hydatid cysts occurs spontaneously or during surgery and can cause multifocal dissemination leading to fever, urticaria, eosinophilia or anaphylaxis. If physicians are familiar with pathology endemic to different countries, patients will be more likely to receive timely, appropriate care.



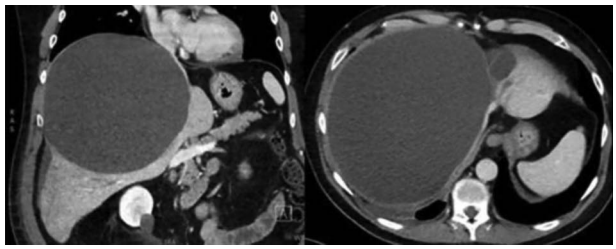
[3101] **Figure 1.** A. CT abdomen completed at time of presentation demonstrating heterogenous low density and poorly enhancing mass. B. MRI abdomen completed at time of presentation showing complex cystic and multilobulated lesion with thick walls and thick internal septations. C. CT abdomen eight and half weeks after initiation of Albendazole.

S3102

New Onset Budd-Chiari Syndrome Caused by an Abnormally Large Liver Cyst

Ashley Soler-Acevedo, MD, Jonathan Kittle, MD, Luis Velez-Colon, MD.
William Beaumont Army Medical Center, El Paso, TX.

Introduction: Simple liver cysts are one of the most common incidental findings on imaging studies. It is estimated that liver cysts occur in approximately 2.5% of the population, with a slight female predominance. Most of them are asymptomatic and usually do not cause any problems. However, if the cyst continues to grow it can cause compression of surrounding structures, increasing the risk for Budd-Chiari syndrome (BCS). BCS is caused by obstruction of hepatic venous outflow and is the leading cause of post-sinusoidal liver failure. We present the case of a patient with BCS caused by an abnormally large liver cyst.
Case Description/Methods: The patient is a 71-year-old Hispanic male with past medical history significant for a simple hepatic cyst, discovered in 2016. The lesion had remained stable at 10 cm as of 2019. Patient presented to the ED in 2021 with a chief complaint of worsening, bilateral, lower extremity edema that extended to the abdomen. CT abdomen/pelvis revealed enlargement of the cyst to 23 × 18 cm, with compression of the IVC causing secondary Budd-Chiari syndrome. Serum Echinococcus antibodies were obtained and resulted negative prior to intervention. Patient underwent a laparoscopic fenestration and partial liver cystectomy, by the General Surgery Service. Cytology obtained on the cyst was without evidence of malignancy, and fungal/bacterial cultures grew no organisms. Serum hepatitis, Strongyloides, Schistosoma, HIV, and Entamoeba antibodies resulted negative. Patient was discharged home without further complications (Figure 1).
Discussion: Simple liver cysts are saccular fluid filled structures that usually do not have septations or calcifications, and usually do not enhance with intravenous contrast. The diagnosis is usually established by clinical presentation and imaging. Ultrasound can be used to identify the characteristics of the cyst, but magnetic resonance is recommended for further characterization. Percutaneous needle aspiration can be performed, with or without the usage of sclerosing agents, but this method is associated with a high recurrence rate. Although uncommon, BCS can be acute and lead to fulminant liver failure. Abdominal pain, in the setting of a history of known liver cysts should be taken seriously, since complications including peritoneal infection, anaphylaxis, hemorrhage and acute liver failure can be catastrophic.



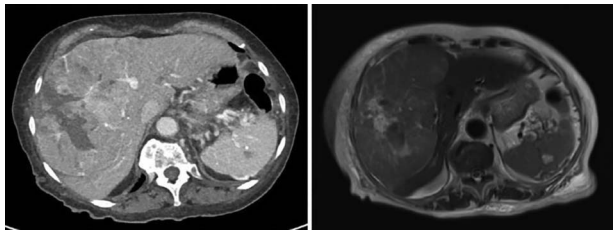
[3102] **Figure 1:** First panel shows a sagittal view of the liver cyst compared to liver parenchyma. Second panel demonstrates an axial view of the liver cyst with concomitant compression of the hepatic vasculature.

S3103

A Case of Rapidly Progressing Hepatocellular Carcinoma Hidden From Surveillance

Jay Shah, DO, MS, Soumojit Ghosh, MD, Eugene C. Nwankwo, MD, Michelle Baliss, DO, Laith Numan, MD, Hany Elbeshbeshy, MD.
Saint Louis University, St. Louis, MO.

Introduction: Hepatocellular carcinoma (HCC) accounts for over 90% of all liver cancers worldwide and carries a high mortality rate. Despite screening efforts, the incidence of HCC still remains high due to the prevalence of viral hepatitis, alcoholic cirrhosis, and non alcoholic fatty liver disease. At present, there are no screening guidelines focusing on non cirrhotic primary biliary cholangitis (PBC) patients. We herein present a case of an 84 year old female who was diagnosed with rapidly progressive HCC in the setting of PBC.
Case Description/Methods: An 84 year old female previously diagnosed with anti-mitochondrial antibody negative PBC presented with one week history of diarrhea and 20lb unintentional weight loss over the past year. Physical exam was notable for non tender hepatomegaly. Significant laboratory values include ALP 337 U/L, AST 208 U/L, ALT 52 U/L, INR 1.1, bilirubin total 3.3 mg/dL, Hgb 9.1 g/dL, platelets 309 10^3/uL, and albumin 2.4 g/dL. Social history was negative for tobacco or alcohol use. A CT of the abdomen and pelvis was obtained and revealed a 16 cm mass encompassing the entirety of the right hepatic lobe. A subsequent AFP was >20,000 ng/mL, consistent with HCC and was discharged home with hepatology follow up. She presented 2 week later with shock and was found to have a ruptured HCC which eventually was the cause of death. 6 months prior to admission, she was noted to have normal liver enzymes and AFP <2 ng/mL, but no imaging was obtained as a fibroscan done 12 months prior to admission revealed a liver stiffness of 7.8 kPa (low to moderate probability of advanced fibrosis) and CAP of 229 dB/m (no liver steatosis) (Figure 1).
Discussion: Patients with PBC have a slightly increased risk of HCC which is usually diagnosed in the setting of underlying cirrhosis. Current guidelines recommend surveillance in those patients with documented cirrhosis with imaging every 6 months. However, studies have shown that patients with PBC have a 2.4% risk of developing HCC at any histologic stage of PBC, with a male predominance. There are currently no screening guidelines for PBC, which may have led to an earlier diagnosis, and therefore treatment, in our patient who passed away only 2 weeks after presentation due to ruptured HCC. Given the outcome in this case, more aggressive screening might be beneficial even in the non-cirrhotic population who have underlying PBC.



[3103] **Figure 1.** CT and MRI visualizing HCC predominately in right hepatic lobe.

S3104

A Mysterious Case Report of Acute Liver Failure: Possible Defect in Ammonia Metabolism

Rosanna Sanchez, BA[1], Mohammad Abdallah, MBBS[2], Deena Kapadia, MD[2].
[1]Rutgers New Jersey Medical School, Clifton, NJ; [2]Rutgers NJMS, Newark, NJ.

Introduction: Acute liver failure (ALF) is defined as severe, acute liver injury causing impairment of synthetic function in a patient without pre-existing liver disease. Infections, hepatotoxic drugs, and autoimmune diseases are more commonly known causes, while, rarely, metabolic disorders may be the culprit. In some cases, however, the cause of ALF remains unknown. We present the case of a young male with ALF of unknown etiology, possibly resulting from a non-specific defect in ammonia metabolism.
Case Description/Methods: A 21-year-old male with no significant past medical history arrived at our liver transplant center with ALF. The patient was visiting New Jersey from Ohio. Two days prior to admission, he developed fatigue after eating at a restaurant. The next morning, he felt nauseated and, later that afternoon, was found to be incoherent, covered in coffee ground emesis. In the hospital, he was found to have elevated AST/ALT 938/875 U/L, extremely elevated ammonia of 2000 μmol/L, elevated lactic acid, and an INR of 4.87. He also developed acute kidney injury and cerebral edema. He was started on continuous renal replacement therapy due to the hyperammonemia and metabolic acidosis and was intubated. An extensive work-up of his ALF including viral panels to assess for CMV, HCV, EBV and HSV PCR were all negative. Ceruloplasmin, alpha-1-antitrypsin, AMA, ASMA testing were also all unyielding. His notable hyperammonemia raised suspicion for a urea cycle disorder. However, despite having an abnormal amino acid profile (see Table 1), subsequent genetic testing showed no conclusive explanation for his ALF. Mitochondrial gene sequencing for common causes was also negative. The patient ultimately underwent an orthotopic liver transplantation which resulted in significant clinical improvement in neurological status and resolution of his hyperammonemia. He was discharged on appropriate post-transplant therapy. Liver biopsy pathology revealed extensive degenerative changes and confluent necrosis.
Discussion: The etiology of ALF remains undiagnosed in approximately 20-40% of cases. Although the availability of liver transplantation has substantially advanced the management of ALF, identification of the underlying cause significantly influences determination of prognosis, management approach, and the likelihood of recurrent liver failure, especially in cases of suspected metabolic disorders. Therefore, the continued pursuit of potential causes of ALF will aid in making life-saving decisions.

Table 1. Plasma Amino Acid Profile

Amino Acid	Value	Reference Range
Aspartate	2.2 umol/L	0.0 - 7.4 umol/L
Asparagine	**86.0 umol/L**	29.5 - 84.5 umol/L
Glutamate	47.1 umol/L	18.1 - 155.9 umol/L
Glutamine	**1021.9 umol/L**	372.8 - 701.4 umol/L

Table 1. (*continued*)

Amino Acid	Value	Reference Range
Proline	**543.9 umol/L**	84.8 - 352.5 umol/L
Glycine	337 umol/L	144.0 - 411.0 umol/L
Alanine	362.9 umol/L	209.2 - 515.5 umol/L
Citrulline	**52.2 umol/L**	15.6 - 46.9 umol/L
Cystine	22 umol/L	15.8 - 47.3 umol/L
Homocitrulline	< 0.5 umol/L	0.0 - 1.7 umol/L
Cystathionine	**22.0 umol/L**	0.0 - 0.7 umol/L
Argininosuccinate	< 0.1umol/L	0.0 - 3.0 umol/L
Beta-Alanine	4.1 umol/L	1.1 - 9.0 umol/L
Ornithine	**178.1 umol/L**	30.1 - 101.3 umol/L
Lysine	**424.7 umol/L**	94.0 - 278.0 umol/L
Histidine	**123.1 umol/L**	47.2 - 98.5 umol/L
Arginine	**210.0 umol/L**	36.3 - 119.2 umol/L

S3105

Role of Hepatectomy in Patients With Liver Abscess: A Single Center Experience

Antoine Boustany, MD, MPH[1], Aiman Obed, MD, PhD[2], Mohammad AbuAssi, MD[2], Abdalla Bashir, MD[2], Anwar Jarrad, MBBS[2].
[1]Cleveland Clinic Foundation, Cleveland, OH; [2]Jordan Hospital, Amman, 'Amman, Jordan.

Introduction: Liver abscess is relatively rare and could be lethal if left untreated. Current advances in the management include diagnostic and interventional radiology that have decreased the rate of mortality. The aim of this study is to present four cases of patients with liver abscess who were cured by hepatectomy after failing CT-guided and surgical drainage.
Case Description/Methods: Four cases of patients with liver abscess due to post-radiofrequency ablation in HCC-patient (case 1), unknown etiology (cases 2 and 3), and foreign body injury from a shrapnel (case 4); were selected (Table 1). All of them were treated initially with empiric antibiotics and underwent CT-guided and surgical drainage. They subsequently underwent liver resection as their clinical condition failed to improve. On follow-up, the patients improved and repeat abdominal ultrasonography was negative for recurrence of abscess.
Discussion: Although rare, liver abscess has a high mortality rate. Hence, there is an urgent need to treat this condition upon diagnosis. Recently, percutaneous drainage in addition to empiric antibiotics have been the initial approach for treating the liver abscesses. However, surgical drainage or liver resection can sometimes be considered to treat hepatic abscess in selected cases where the abscess is not accessible to CT-guided percutaneous drainage, if it is ruptured, or if the patient failed to improve with optimized medical therapy and percutaneous drainage. These cases highlight the importance of considering liver resection in curing patients with liver abscess who did not improve with medical therapy and CT or surgical drainage.

Table 1. **Characteristics and laboratory findings of the four patients with liver abscess selected**

	Case I	Case II	Case III	Case IV
Age	75	48	44	24
Gender	Male	Female	Male	Male
Cause of liver abscess	Post-radiofrequency ablation in HCC-patient	Cryptogenic	Cryptogenic	Foreign body injury from a shrapnel
Chronic Hep	Positive	Negative	Negative	Negative
Preop CRP	249 mg/l	240 mg/l	2 mg/l	250 mg/l
Culture	E. Coli (ESBL) and Enterococcus Faecium	No growth	No growth	Pseudomonas aeruginosa and Candida

S3106

N-Acetyl Cysteine Use in the Treatment of Ischemic Hepatitis in the Setting of COVID-19 Infection

Vivian Agumadu, MT-ASCP, PA-C, MD[1], Gabriela Generette, MD[2], Altaf Dawood, MD[1], Nicole Gentile, MD[1], Luqman Baloch, MD[1], Mario Affinati, MD[1].
[1]MercyHealth Internal Medicine Residency Program, Rockford, IL; [2]MercyHealth Internal Medicine Residency Program, Schaumburg, IL.

Introduction: Acute liver injury occurs in 15-53% of COVID-19 patients. Most often, elevation in transaminases is mild, but severe liver injury has been described possibly secondary to hepatic ischemia. We present a case of ischemic hepatitis with concurrent COVID-19 infection, in which N-acetyl cysteine (NAC) administration led to clinical improvement and recovery.
Case Description/Methods: A 58-year-old male with adult congenital heart disease (ACHD), on warfarin for atrial flutter and cardiac cirrhosis presented to the ER with generalized fatigue, chills, congestion, and cough. Patient tested positive for COVID-19. He was hemodynamically stable with an unremarkable exam. He was alert and oriented times three. He initially required 8 L of oxygen which continued to improve on a daily basis. His initial laboratory workup was notable for AST 1954 U/L, ALT 1905 U/L, INR 19.1, and total bilirubin 0.9 mg/dL. At baseline, patient's liver enzymes were normal. He was admitted to the ICU due to the severity of his liver injury. Alcohol, acetaminophen, salicylate levels and urine drug screen were all within normal limits. Abdominal ultrasound revealed known liver cirrhosis and ascites. Acute hepatitis serologies including hepatitis E IgM, ANA, AMA, ASMA, anti-LKM, IGG subclasses, ceruloplasmin, alpha-1 antitrypsin, and celiac panel were done and unremarkable. In addition, HIV, VZV PCR, EBV PCR, HSV PCR and CMV PCR were also negative for acute infection. Patient was treated with a NAC protocol with decrease in elevated liver enzymes by half in the first 72 hours and >20-fold of the highest level upon discharge. Throughout his admission, his mental status remained normal.
Discussion: Ischemic hepatitis (IH) results from insufficient blood flow volume and/or oxygen content to the hepatocytes. Acute hypoxic respiratory failure as a result of COVID-19 infection is one of the potential causes of this type of liver injury. Other possible causes of liver injury include direct viral cytopathic effect, cytokinesis and drug-induced liver injury. In the setting of COVID 19 infection, prompt diagnosis and recognition of IH is critical as studies describe mortality rates as high as 50%. We describe our clinical experience with NAC in the setting of COVID 19 with severe IH and propose that NAC is considered in the treatment of COVID-19 patients with ischemic hepatitis, however further research including prospective clinical trials is needed to better validate this.

S3107

Successfully Treated Acute Liver Injury in Acute Hepatitis C With N-Acetyl Cysteine: A Case Report

Brian Sowka, DO, Padmavathi Mali, MD.
Gundersen Health System, La Crosse, WI

Introduction: Significant liver injury related to acetaminophen overdose is more common in patients with preexisting liver disease. We present a patient with newly diagnosed acute hepatitis C infection with acute liver injury treated successfully with N-acetylcysteine despite negative acetaminophen levels.
Case Description/Methods: A 29-year-old male with history of spontaneously cleared HCV two years ago and polysubstance use presented with abdominal pain, anorexia, and jaundice. He admitted to taking less than 2 g of acetaminophen for tooth ache and chronic back pain along with recent IV drug use, alcohol use, and marijuana use. Examination revealed scleral icterus and right upper quadrant tenderness. Labs revealed normal acetaminophen level, elevated bilirubin 9.4, transaminases AST 1,023; ALT 1,941; alkaline phosphatase (ALP) 202, and INR of 1.4. Hepatitis C antibody returned positive. Hepatitis C genotype was 1a or 1b with an HCV RNA level of 10,917. Evaluation for other etiologies for acute liver injury including alpha-1 antitrypsin, ceruloplasmin, autoimmune, and other infectious work up was negative. MRCP was normal without biliary pathology. He was given 140 mg/kg oral N-acetylcysteine for suspected acetaminophen overdose as the etiology for liver injury. His symptoms improved and liver tests showed an improvement in the next few days (total bilirubin 5.1, mg/dL, AST 100, ALT 731, ALKP 218). The patient was prescribed Sofosbuvir-Velpatasvir at discharge for the treatment of acute hepatitis C.
Discussion: In patients with hepatitis C infection the rate of liver injury with acute or chronic infection is about 16.7%. This case highlights the importance of having a low threshold for treating patients for acetaminophen toxicity in acute hepatitis C patients based on history. This case also highlights further studies are needed to determine the incidence and implications of acute liver injury in acute hepatitis C patients as there are no studies in this group of patients so far.

Table 1. Liver Function Tests During Admission

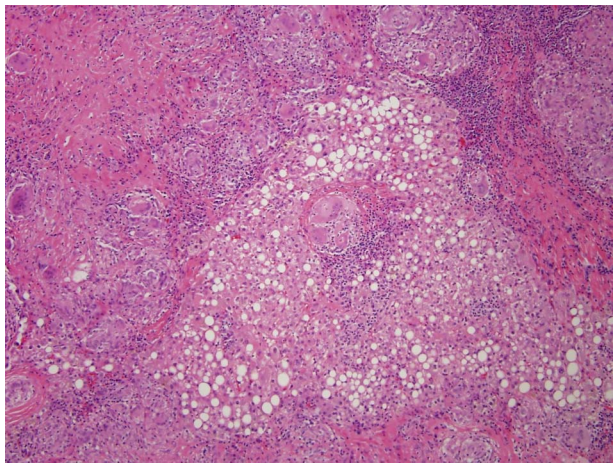
Liver Function Tests	Initial	Repeat
Total Bilirubin (mg/dL)	9.4	5.1
ALT (IU/L)	1,941	731
AST (IU/L)	1,023	100
ALKP (IU/L)	202	218
INR	1.4	1.2

S3108

Hepatic Sarcoidosis Presenting as Cholestatic Liver Injury Exacerbated by Nitrofurantoin Use

Sean Halloran, BS[1], Sara Stanley, DO[2], Jordan Burlen, MD[3], Yaseen Alastal, MD, MPH[2].
[1]University of Toledo College of Medicine and Life Sciences, Toledo, OH; [2]University of Toledo, Toledo, OH; [3]The Ohio State University, Toledo, OH.

Introduction: The liver is a common site of involvement in patients with sarcoidosis with 50-80% of patients having hepatic involvement at initial diagnosis. However, it is largely asymptomatic and less than 15% of patients present with symptoms of significant hepatic injury. We report a patient with hepatic sarcoidosis presenting as cholestatic liver injury exacerbated by nitrofurantoin use.
Case Description/Methods: A 67-year-old African American female presented due to 1 week of diffuse itching, shortness of breath, and scleral icterus with darkened urine. Medical history was significant for type 2 diabetes, hypertension, and hyperlipidemia without history of liver disease. Patient denied alcohol use and was a lifetime non-smoker. Family history was significant for sarcoidosis in father. Notably, the patient took nitrofurantoin for a urinary tract infection one week prior to presentation. Lab results showed elevated direct bilirubin 5.2, total bilirubin 8.8, ALK PHOS 950, ALT 126, and AST 229. Urinalysis was positive for glucose and moderate bilirubin. CT of chest, abdomen and pelvis showed abdominal and mediastinal lymphadenopathy. Ultrasound of the liver and MRCP showed hepatic steatosis and gallbladder sludge without acute cholecystitis or biliary dilation. EUS with FNA of a mediastinal lymph node displayed numerous granulomas. Liver biopsy showed cholestatic granulomatous hepatitis with stage 2-3 bridging fibrosis. Biopsy was negative for A1AT, acid-fast bacilli, and fungal organisms. ANA, AMA, SMA, and hepatitis panel were negative. This patient's presentation was deemed most consistent with hepatic sarcoidosis and prednisone therapy was initiated. The patient reports improvements in pruritus and scleral icterus. Nitrofurantoin is a well-known cause of hepatic injury, but has rarely been reported as causing granulomatous disease exacerbating underlying sarcoidosis (Figure 1).
Discussion: While the liver is a common site of involvement in patients with sarcoidosis, the vast majority of patients are asymptomatic and do not require treatment. Hepatotoxic drugs can exacerbate the symptoms of hepatic sarcoidosis and lead to clinical diagnosis. In those with clinical symptoms, a cholestatic pattern is most common. Glucocorticoids and antimetabolites such as methotrexate are commonly used treatments. However, there is a lack of large randomized controlled studies regarding the treatment and surveillance of hepatic sarcoidosis at this time.



[3108] **Figure 1.** Histology slide showing hepatic involvement of sarcoidosis.

S3109

***Aeromonas caviae* Bacteremia in a Patient With Spontaneous Bacterial Peritonitis**

Arunava Saha, MD.
Saint Vincent Hospital, Worcester, MA.

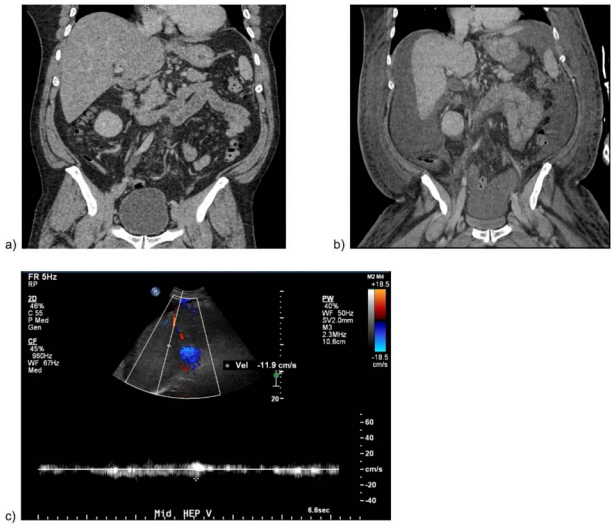
Introduction: The genus Aeromonas consists of Gram-negative rods found in aquatic environments and soil. They commonly cause diarrhea, but have been associated with extraintestinal manifestations such as bacteremia. Drug-resistant strains are being increasingly identified, which are acquired in both community and hospital settings; and can affect both immunocompetent and immunocompromised patients.
Case Description/Methods: A gentleman in his late 30 s presented with a month of high-grade fever with chills, abdominal distension, nausea with vomiting, and weight loss. He had a history of alcoholic chronic liver disease, consumed 200 ml of hard liquor daily for 15 years. On admission he was tachycardic and had a temperature of 102 F. General examination revealed pallor. The liver edge was palpable 1 cm below the right costal margin with a liver span of 12 cm. Abdominal distension with diffuse tenderness was present, but no guarding or rigidity. Free fluid was present. There was no encephalopathy. Laboratory investigations revealed anemia, thrombocytopenia and normal leukocyte count with neutrophilic predominance. Liver functions showed hyperbilirubinemia with transaminitis and hypoalbuminemia. Blood borne virus screen was negative. Ascitic fluid analysis revealed SAAG of 1.6 mg/dl, 17,448 WBCs per mm^3 with 96% polymorphonuclear cells. Ultrasound abdomen showed features of cirrhosis with ascites without any hepatic vein obstruction. He was diagnosed with spontaneous bacterial peritonitis with underlying decompensated chronic liver disease (Child Pugh-C, MeldNa-25). Blood and ascitic fluid cultures were sent, and IV Meropenem was started. The blood cultures isolated Aeromonas Caviae, which was sensitive to Levofloxacin. The ascitic fluid culture was sterile. The antibiotic was switched to IV Levofloxacin for a two-week course. A repeat ascitic fluid count was 104 WBCs/mm^3. The patient symptomatically improved, remained afebrile and started tolerating his diet. He was continued on his management for chronic liver disease and discharged. In a follow-up visit, he was found to be asymptomatic.
Discussion: Aeromonas species can cause invasive and fatal infections in immunocompromised hosts. Initial empiric therapy of suspected Aeromonas infections is with a fluoroquinolone or carbapenem, as resistance rates are high for cephalosporins around the world. Although rare, the Aeromonas species should be considered as one of the causative agents of bacteremia in patients with hepatobiliary diseases or underlying malignancy.

S3110

Collapsing Alcoholic Cirrhosis Presenting as Functional Budd-Chiari Syndrome

Joshua Diaz, MD, Nikolas St. Cyr, MD, Michael Bernstein, MD.
Coney Island Hospital, Brooklyn, NY.

Introduction: Budd-Chiari syndrome (BCS) is a rare disease involving hepatic venous obstruction, often associated with hypercoagulability. Treatment with anticoagulation (AC) and thrombolysis is imperative in BCS; however, this also carries substantial risk. Thus, additional studies are important in confirming BCS prior to initiating treatment. Here we have a case that presented similarly to BCS, but was later ruled out using ultrasonogram (US) with doppler and triphasic CT scan for confirmation, altering the management plan.
Case Description/Methods: A 49 year old male with alcoholic cirrhosis and esophageal varices presented with bilateral lower extremity edema, abdominal pain and dyspnea. Physical exam found scleral icterus, jaundice, abdominal distention with fluid wave, bilateral lower extremity edema and tenderness. Labs showed platelets of 83, ALK 268, AST 44, ALT 23, Direct bilirubin 1.4 and ammonia 48. He was admitted for decompensated liver cirrhosis and prophylaxis for spontaneous bacterial peritonitis was initiated. Patient was diuresed and underwent paracentesis. Fluid analysis showed SAAG $>$ 1.1 and Neutrophils $<$ 250, indicating portal hypertension. Doppler US showed attenuated caliber of hepatic veins and discontinuity between these and the IVC. Confirmatory study with Triphasic CT showed a shrunken liver without clots, ruling out primary BCS. Patient was deemed unsuitable for TIPS given MELD-Na of 20, degree of hepatic damage and transplant unavailability, and was discharged on medical management (Figure 1).
Discussion: This case of alcoholic cirrhosis shares clinical and physiologic similarities with BCS. Differentiating between the two is key to initiating correct intervention and minimizing complications. This patient has atrophied hepatic architecture that serves as an outflow obstruction similar to that seen in BCS. Imaging with doppler US, CT or MRI looks for direct and indirect signs of BCS; here, no diagnostic signs were observed, despite a defect of venous outflow on doppler US. Had confirmatory tests not ruled out BCS, AC likely would have been initiated, exposing him to unnecessary risks. This highlights a patient with advanced alcoholic cirrhosis presenting with signs and symptoms suspicious for BCS. Initial imaging was also concerning for BCS, but confirmatory tests proved otherwise. This illustrates the value of confirmatory triphasic CT scan in a patient with US findings that are suspicious for, but not confirmatory of, BCS, guiding treatment away from AC and its risks and complications.



[3110] **Figure 1.** (a) CT scan abdomen and pelvis illustrating hepatomegaly and nodular liver contour suggestive of cirrhosis (2019). (b) CT scan abdomen and pelvis showing significantly shrunken liver with modularity and ascites (2022) (c) US with Doppler showing attenuated middle hepatic vein with reduced flow velocity.

S3111

An Unusual Case of Acute Liver Failure Secondary to Autoimmune Hepatitis (AIH) From Drug-Induced Liver Injury (DILI)

Abid Ahmad, MD, Howard Masuoka, MD, PhD, John Guardiola, MD.
Indiana University School of Medicine, Indianapolis, IN.

Introduction: A broad differential diagnosis and detailed workup is necessary in the evaluation of acute liver failure, as the below case demonstrates.
Case Description/Methods: A 68-year-old Caucasian man presented to the ED complaining of chronic right lower quadrant pain, worsening over the last few weeks. He reported taking up to 20 tablets of naproxen a day. Review of systems was positive for diffuse pruritis of recent onset. Past medical history consisted of COPD, inguinal hernia status post mesh repair, knee osteoarthritis and a cholecystectomy. He endorsed daily tobacco use and minimal alcohol intake. Medications consisted of Albuterol inhaler and Naproxen. He had been diagnosed with a urinary tract infection one month prior that was treated with a ten-day course of Cephalexin. Exam was notable for diffuse excoriation and right upper/lower quadrant tenderness. Initial labs demonstrated creatinine 0.87, AST 2635, and ALT 887, with the remainder of his

comprehensive metabolic panel being normal. Contrasted CT scan of the abdomen and pelvis revealed no abnormalities. A right upper quadrant ultrasound was notable for left sided intrahepatic biliary dilatation. Over the next day, his mentation worsened, with repeat labs notable for bicarbonate 13, creatinine 3.08, AST 12050, ALT 3950, total bilirubin 2.4 with direct bilirubin 1.5, INR 3.11 and ammonia 164. A broad infectious workup was unremarkable including negative viral hepatitis serologies. Smooth muscle antibody was weakly positive at a low titer of 1:20. Anti-nuclear and anti-mitochondrial antibodies were negative. Serum IgG level was normal. Endoscopic retrograde cholangiopancreatography revealed a severe left intrahepatic biliary stricture with resulting stent placement into the left hepatic duct. Bile duct brushing was negative for atypical cells. Liver biopsy demonstrated centrilobular necrosis and parenchymal collapse with focal bridging necrosis and mixed inflammatory infiltrate. A diagnosis of AIH secondary to DILI leading to acute liver failure was made, with either Naproxen or Cephalexin being the inciting drugs.

Discussion: He was started on a prednisone taper and showed improvements in mental status and kidney and liver function on lab-work prior to discharge. Labs obtained on clinic follow-up one month after discharge revealed complete resolution of his kidney and liver injury. This case illustrates a rare clinical entity as neither Naproxen nor Cephalexin are classically associated with causing AIH or acute liver failure.

S3112

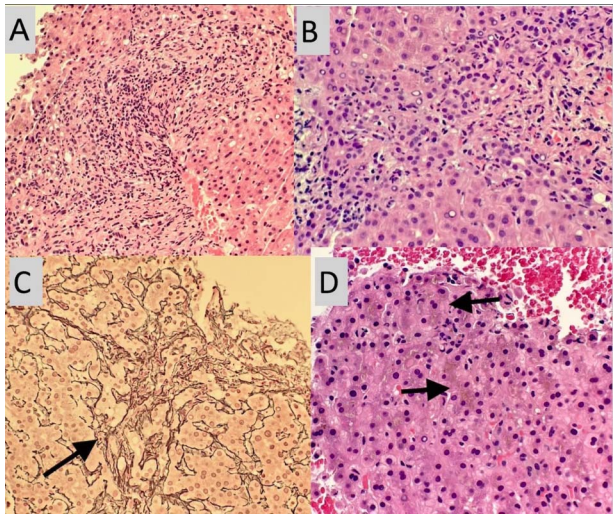
A Bactrim-Induced Liver Failure Requiring a Transplant: A Report of a Rare Side Effect of a Commonly Used Drug!

Rawan Aljaras, MD[1], Maryam Haider, MD[2], Ahmad Karkash, MD[3], Bianca Nicole Puello Yocum, MD[3], Razan Aljaras, MD[3], Gerardo Calderon, MD[1].
[1]Indiana University, Indianapolis, IN; [2]Wayne State University/Detroit Medical Center Sinai Grace Hospital, Detroit, MI; [3]Indiana University School of Medicine, Indianapolis, IN.

Introduction: Drug-induced Liver Injury (DILI) is an important cause of acute liver failure cases in the United States. Multiple drugs including prescription, over-the-counter, and herbal products can cause hepatotoxicity through a variety of mechanisms. Clinical suspicion is key in diagnosing DILI. Sulfamethoxazole with trimethoprim (Bactrim or TMP/SMX) is a fixed antibiotic combination that is very commonly used in clinical practice, and as many others, this combination has been linked to rare instances of acute liver failure, sometimes even requiring a liver transplant as represented by this case.

Case Description/Methods: We present a case of a 38 year old female patient who presented with jaundice a month after she completed a 5 day course of Bactrim for a urinary tract infection. Initial labs were notable for cholestatic pattern of liver injury. Extensive infectious and autoimmune workup came back negative. MRCP and ERCP were both done and noted unremarkable with no biliary tract abnormalities. Liver biopsy with cholestasis and liver injury pattern described in (Figures 1A–D). Patient was started on oral ursodiol and steroids, with continued trend up in liver enzymes and eventually the development of ascites. Patient had a repeat liver biopsy few months after which showed cirrhosis and cholestasis. Roughly, 18 months after exposure to Bactrim, patient got a liver transplant.

Discussion: As fascinated as the patient could be, a simple urinary tract infection ended up with her needing an organ transplant and fortunately she was able get one. Bactrim is known for its potential to cause idiosyncratic liver injury that has features of drug-allergy or hypersensitivity. Three forms of Bactrim induced liver damage have been described; hepatocellular, mixed hepatocellular cholestatic, and (more recently) bile duct injury with ductopenia or Vanishing Bile Duct Syndrome. The onset of symptoms usually occurs within a few days of ingestion but can take up to 1–2 months. Diagnosis is suspected from the clinical presentation, and absence of other causes, in addition to suggestive changes on liver biopsy. Treatment is generally supportive; liver transplantation has been successful for both fulminant hepatic failure and vanishing bile duct syndrome of varying causes.



[3112] **Figure 1.** A, H&E stained liver biopsy demonstrates moderate portal inflammatory infiltrate consisting of neutrophils, lymphocytes, and eosinophils. There is a marked ductular reaction. B, H&E stained liver biopsy demonstrates moderate portal inflammatory infiltrate consisting of neutrophils, lymphocytes, and eosinophils. There is a marked ductulaR reaction. C, Reticulin stain demonstrates mild portal fibrosis (arrow) with preserved hepatic architecture. D, H&E stained liver biopsy demonstrating marked cholestasis (arrows).

S3113

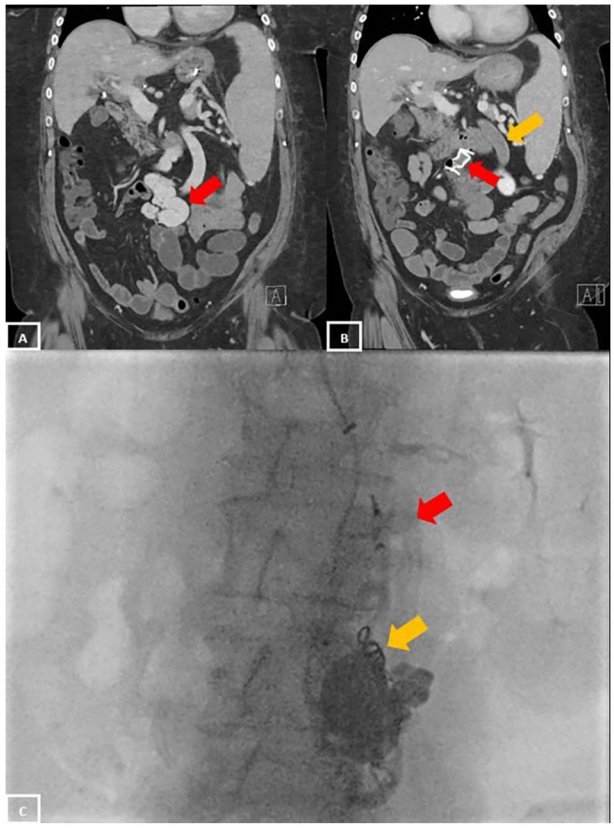
Plugging up the Leak After Transplant: Shunt-Induced Encephalopathy PARTO-ly Treated and Completed With CARTO

Carl L. Kay, MD[1], Christian Horn, MD[1], Omer Junaidi, MD[2].
[1]Brooke Army Medical Center, JBSA Fort Sam Houston, TX; [2]Methodist Hospital Specialty and Transplant, San Antonio, TX.

Introduction: Spontaneous portosystemic shunts (SPSS) are a common cause of recurrent hepatic encephalopathy (HE) in patients with cirrhosis. Liver transplant (LT) is the definitive treatment of end-stage liver disease and its complications, but SPSS may persist after LT. It is rare for SPSS to cause HE after LT; however, it is a treatable cause of encephalopathy.

Case Description/Methods: A 52-year-old female had an orthotopic liver transplantation for decompensated cirrhosis due to non-alcoholic steatohepatitis. Two months later, her post-transplant course began to be complicated by recurrent episodes of encephalopathy. She was diagnosed with HE after extensive unremarkable work-up – including metabolic, infectious, advanced brain imaging, electroencephalography, cerebrospinal fluid analysis, and neurology evaluation. The diagnosis was also supported by persistent hyperammonemia and improvement of sensorium with initiation of lactulose. In total, she had four episodes of overt HE requiring hospitalization. She had a transjugular liver biopsy which revealed stage 1 fibrosis but no evidence of graft cirrhosis or rejection. She had normal graft synthetic function. CT scan of the abdomen 8 months after transplant demonstrated a large inferior mesenteric to left renal vein shunt measuring 16 mm in diameter. This was confirmed by transjugular mesenteric venogram and excluded portal vein thrombosis or stenosis. She underwent plug-assisted retrograde transvenous occlusion (PARTO) of the shunt using 22 mm vascular plug. Coil-assisted retrograde transvenous obliteration (CARTO) technique with two 6 mm interlocked coils were used to treat residual collaterals in order to complete a successful shunt closure. In six months of follow up, she has not had any further episodes of HE or required any lactulose (Figure 1).

Discussion: Post-LT encephalopathy has a broad differential and SPSS is a rare cause of HE - especially without graft cirrhosis. After excluding alternative etiologies, it is important to recognize that SPSS can be treated with embolization. Successfully treated patients can avoid a significant amount of morbidity associated with recurrent hospitalizations and ongoing pharmacologic management. We describe the second known case of combined PARTO/CARTO technique to treat post-LT SPSS-related HE. This is only the 9th identified case of post-LT SPSS closure for treatment of HE. Not only is shunt closure feasible, but it is also safe within the 1st year of transplantation without thrombosis or need for repeat embolization.



[3113] **Figure 1.** (A) Pre-PARTO/CARTO contrasted computed tomography (coronal view) in the venous phase depicting the large tortuous shunt from the inferior mesenteric vein to the left renal vein (red arrow); (B) Post-PARTO/CARTO contrasted computed tomography (coronal view) in the venous phase depicting vascular occlusion device (red arrow) with successful thrombosis of the dilated shunt (yellow arrow); (C) Post-PARTO/CARTO angiography depicting vascular occlusion device (red arrow) and coils (yellow arrow).

S3114

Painless Jaundice With a Significantly Elevated CA 19-9: It Is Not Always Pancreaticobiliary Cancer

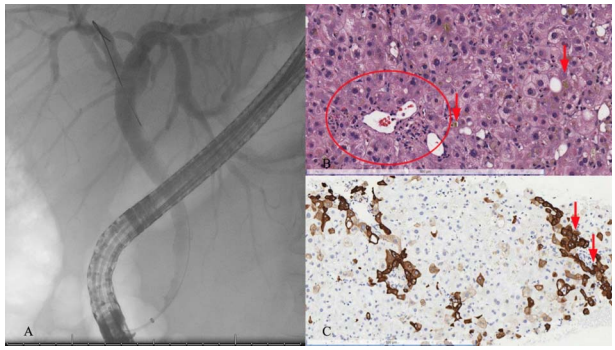
David Jonason, MD, Aisha Mohamed, MS, Nicha Wongjarupong, MD, Oyedele Adeyi, MD, Jeffrey H. Albrecht, MD, Guru Trikudanathan, MD.
University of Minnesota Medical Center, Minneapolis, MN.

Introduction: Carbohydrate antigen 19-9 (CA 19-9) is often very high in pancreaticobiliary cancers but can be substantially elevated in benign diseases too. We present a case of painless jaundice with a significantly elevated CA 19-9 from vanishing bile duct syndrome (VBDS).
Case Description/Methods: 65-year-old white male with history of obesity, hypertension, hyperlipidemia, prior cholecystectomy and no family history of pancreatic cancer presented with two weeks of painless jaundice and 10 pounds unintentional weight loss. Physical exam noted scleral icterus and jaundice with a T.Bili of 23 mg/dL (Table 1). CT scan showed intrahepatic bile duct dilation and a 10 mm common bile duct without a discrete mass. CA 19-9 was elevated to 1,480 U/mL, concerning for malignancy. ERCP found a low-grade main bile duct stricture which was stented. Bile duct brushings and fine needle aspiration of the pancreas were negative for malignancy. Biliary dilation was resolved on follow-up MRCP but his bilirubin remained high. Infectious and autoimmune workup was unremarkable. Repeat cholangiography and brushings confirmed no obstruction or malignancy (Fig. 1a). An infiltrative versus drug induced liver injury was suspected. EUS-guided liver biopsies revealed cholestasis with dilated bile canaliculi, hepatocytes with ballooning degeneration and a paucity of bile ductules. Cytokeratin-7 immunostaining demonstrated >50% ductopenia, confirming VBDS (Fig. 1b and c). Follow up at hepatology clinic determined triamterene-HCTZ, atorvastatin or omeprazole stopped during hospitalization to be the likely culprit. His CA 19-9, LFTs and symptoms improved over the following weeks (Table 1).
Discussion: VBDS is an acquired cholestatic liver disease that may mimic the presentation of pancreatic cancer. CA 19-9 can be substantially elevated through T-cell mediated destruction of small bile ducts causing impaired excretion, inflammatory production, and decreased clearance. Severity of liver damage depends on the duration of injury and degree of bile duct loss at the time of diagnosis. Prompt diagnosis is important to prevent cirrhotic progression, improve outcomes, and avoid unnecessary treatments. Recognizing its association with an elevated CA 19-9 may help with this, but is scarcely reported in the literature. CA 19-9 is not typically checked in cases of VBDS but is likely commonly elevated. Providers should be aware of this association when faced with a patient with painless jaundice and a significantly elevated CA 19-9 but no obvious malignancy.

Table 1. LFT Trends During the Course of Presentation

	Normal Range	Hospital Admission *	2 weeks	3 weeks **	4 weeks ***	7 weeks	13 weeks
Total bilirubin	0.2-1.3 mg/dL	23	22.7	22	19.7	4.7	1.3
Direct bilirubin	0.0-0.2 mg/dL	>13	n/a	14.9	14.6	4.2	0.6
AST	0-45 U/L	107	134	172	200	121	27
ALT	0-50 U/L	190	130	163	182	120	35
ALP	40-150 U/L	267	179	187	168	132	109
CA 19-9	0-35 U/mL	1480	n/a	n/a	85	49	n/a

AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; CA19-9, carbohydrate antigen 19-9; n/a, not available. * ANA, anti-AMA, anti-SMA, anti-mitochondrial, anti-TTG, anti-LKM 1/2 IgG, and EBV IgG antibodies were negative. Serum IgA, A1AT, ceruloplasmin, TSH, iron levels, hepatitis panel, serum protein electrophoresis and acetaminophen levels were normal. Acetaminophen, multivitamin, iron, triamterene- HCTZ, omeprazole and atorvastatin were stopped on discharge. ** Three weeks post hospitalization. Repeat EUS/ERCP showed biliary decompression. VBDS diagnosed on liver biopsies. *** Seen in liver clinic. Started ursodiol 2000mg daily.



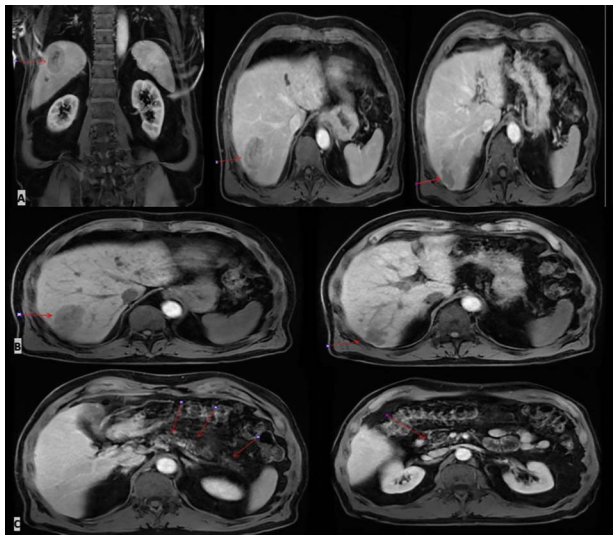
[3114] **Figure 1.** (A) ERCP with cholangiogram showing a low-grade distal biliary stricture without significant upstream dilation. (B) Liver biopsy with H&E stain, 500 μm magnification showing small portal tracts with no bile ducts (red circle); note marked cholestasis (arrows) in dilated canaliculi and lack of ductular reaction. Also visible at this magnification is the ballooning degeneration of hepatocytes. (C) Liver biopsy with cytokeratin 7 immunostain, 500 μm magnification. Small portal tracts with no terminal/small bile ducts. Early signs of ductular reaction seen by hepatocytes taking up CK7 stain (arrows).

S3115

Pancreatic Cancer Without Pancreatic Lesion: Pancreatic Acinar Cell Carcinoma (PACC) Presenting as Isolated Hepatic Lesion Without Primary Organ Involvement

Rehmat ullah Awan, MD[1], Shazia Rashid, MD[2], Ambreen Nabeel, MBBS[1], Mohammed Alsaggaf, MD[3], Hrishikesh Samant, MD[3].
[1]Ochsner Rush Health System, Meridian, MS; [2]LSUHSC, Shreveport, LA; [3]Ochsner Health System, New Orleans, LA.

Introduction: Pancreatic cancer are diagnosed in advanced stages and by that time the treatment options are often limited. We report a rare case, showing metastatic pancreatic acinar cell carcinoma (PACC) in the liver without any evidence of primary lesion in the pancreas based on imaging studies.
Case Description/Methods: A 78-year-old male with a past medical history of prostate cancer, pancreatic insufficiency due to recurrent pancreatitis and irritable bowel syndrome was seen in gastroenterology clinic for epigastric pain. Patient's family history was significant for prostate cancer and social history was pertinent for alcohol abuse in the past. He underwent endoscopic evaluation with EGD and colonoscopy, both of which were unremarkable. Thereafter, MRI abdomen with contrast revealed a 6 cm right hepatic lobe lesion concerning for malignancy along with multiple cysts approximately 15 mm in size, and no other lesion was identified in the abdomen. Lab work was also unremarkable (see Table 1). Subsequently he underwent IR guided liver biopsy of the mass. The final histopathological diagnosis of the biopsy was acinar cell carcinoma of pancreatic origin and likely metastatic lesion. Looking back at the MRI abdomen there were no findings to suggest of pancreatic lesion, consequently he underwent EUS which showed normal esophagus, stomach and duodenum, the pancreatic parenchyma was consistent with fatty infiltration however no cyst, pseudocyst or mass was identified through the entire pancreas. At this point, patient already followed with medical oncology and was started on systemic pancreatic regimen therapy. He tolerated chemotherapy well and repeat CT scans in subsequent months showed overall stable disease (Figure 1).
Discussion: Hepatologists routinely come across liver lesions suspicious for malignancy. Liver is the second most common site of metastasis after lymphatic system. Pancreatic acinar cell carcinomas (PACC) are rare, accounting for 1-2% of adult pancreatic tumors. On MRI, PACC appear as large, oval mass with moderate and heterogenous enhancement after intravenous contrast. Our case is one of its kind due to the detection of significantly sized pancreatic acinar cell tumor in the liver prior to it being detectable in the pancreas itself (see Figure-1 for imaging details). This case highlights the importance of prompt diagnosis and management especially in challenging cases which present with discordance between histopathological and imaging studies.



[3115] **Figure 1.** A, Post-contrast T1-weighted MRI; images showing predominantly peripheral enhancement of segment 6/7 hepatic lesion (red arrows). B, Pre-contrast T1-weighted MRI; images showing predominantly peripheral enhancement of segment 6/7 hepatic lesion (red arrows). C, Post-contrast T1-weighted MRI; Left image marks pancreatic body and tail, Right image marks the uncinate/head, does not show any lesion or mass in pancreas.

Table 1. **Diagnostic Lab work up**

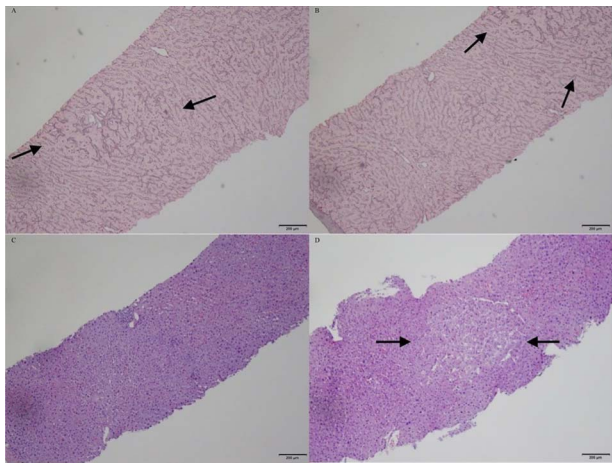
Lab Values/Reference Range &Units	Patient Result
WBC 4.50-13.50 K/uL	7.06
Hemoglobin 13.0-16.0 g/dL	13.8
Platelets 150-450 K/uL	231
Albumin 3.2-4.7 g/dL	3.3
INR 0.8-1.2	1.1
Total Bilirubin 0.1-1.0 mg/dL	0.9
Alkaline Phosphatase 89-365 U/L	98
AST 10-40 U/L	22
ALT 10-44 U/L	15
Cancer Antigen 19-9 0.0-40.0 U/Ml	13.3
CEA 0.0-5.0 ng/mL	1.7
AFP 0.0-8.4 ng/mL	< 2.0
PSA, Screening 0.00-4.00 ng/mL	< 0.01

S3116

Novel Nodules: A Previously Unreported Manifestation of Aicardi-Goutières Syndrome

Matthew Houle, MD[1], Eric Lorio, MD[2], Austin Hansen, MD[1], Martin Moehlen, MD, MPH[3].
[1]Tulane University, New Orleans, LA; [2]Tulane University, Metairie, LA; [3]Tulane University School of Medicine, New Orleans, LA.

Introduction: Aicardi-Goutières syndrome (AGS) is a rare autosomal recessive genetic disorder resulting from mutations of genes encoding multiple proteins, including DNA 3' repair exonuclease 1 (TREX1). This form of leukodystrophy is characterized by increased interferon-alpha in the cerebrospinal fluid and serum leading to immune dysfunction primarily targeting white matter myelin in the brain. Late onset AGS can affect other organs, including liver, kidneys, heart, and lungs. Hepatic inflammation is associated with the neonatal form of AGS, but the incidence of hepatitis across other ages remains unknown. To our knowledge, there have been no reported cases of nodular regenerative hyperplasia in adults with Aicardi-Goutières syndrome.
Case Description/Methods: A 63-year-old man was evaluated for proteinuria and microscopic hematuria. Laboratory workup was unrevealing and renal ultrasound was normal. Family history was significant for father with kidney transplant for unknown renal disease and brother with recurrent bilateral vitreous hemorrhage. Subsequently, the patient and brother had kidney biopsies that showed findings consistent with thrombotic microangiopathy (TMA). Familial TMA prompted whole genome sequencing that revealed a mutation in TREX1 in all three family members. Following nephrology workup, the patient was incidentally found to have a nodular hepatic contour suggestive of cirrhosis. Physical exam was absent of stigmata of liver disease, and he denied a history of alcohol use. A focused laboratory workup was unrevealing (Table 1). MRCP revealed a nodular liver consistent with fibrosis and no intrahepatic or extrahepatic biliary dilation. MR elastography showed F2-F3 fibrosis without evidence of hepatic steatosis or iron overload. Ultimately, a percutaneous liver biopsy demonstrated nodular regenerative hyperplasia (NRH) (Figure 1).
Discussion: NRH has been associated with autoimmune conditions such as systemic lupus erythematosus (SLE) and is thought to be caused by blood vessel inflammation within the liver leading to an overcompensated replication of hepatocytes. The finding of NRH in this patient is representative of the phenotypic overlap between AGS and SLE related to the common underlying feature of IFN-α up-regulation. As seen in this patient, NRH typically does not cause any overt signs or symptoms of liver disease. However, given it can eventually lead to the development of non-cirrhotic portal hypertension, further monitoring of this patient is warranted.



[3116] **Figure 1.** Percutaneous liver biopsy with nodular regenerative hyperplasia. (A, B) Liver biopsy, reticulin stain, low power ×40. Focal compression of reticulin framework with alternated areas of sinusoidal dilatation which gives rise to a vague nodular appearance in the absence of fibrosis, suggestive of NRH. (C, D) Liver biopsy, H&E stain, low power ×40. Intact hepatic architecture with focal hepatocyte atrophy and hepatocyte plate compression with areas of sinusoidal congestion. There is no significant hepatocyte apoptosis or necrosis. Hepatic lobules show mild reactivity with occasional lymphocytic inflammation. There is no significant steatosis, ground glass cells or viral inclusions.

Table 1. **Focused Laboratory Results**

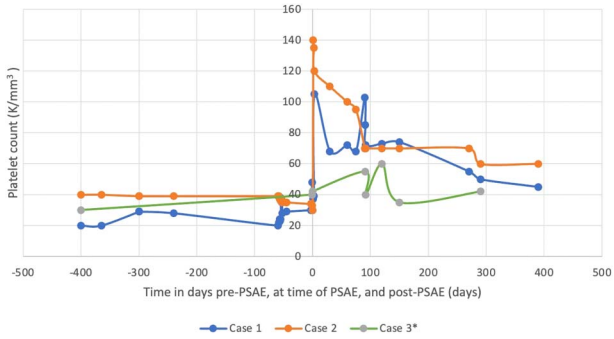
AST/ALT	Total Bilirubin	Alkaline Phosphatase	Viral Hepatitis Serologies (HAV, HBV, HCV)	Serum Total Protein	Serum Albumin	Ceruloplasmin	Anti-Smooth Muscle Antibody	Alpha-Fetoprotein	Alpha-1 Antitrypsin
14/21 units/L	0.4 mg/dL	92 units/L	Non-reactive	7.4 g/dL	3.4 g/dL	24.6 mg/dL	11 units	1.0 ng/mL	126 mg/dL

S3117

Partial Splenic Artery Embolization for Patients With Portal Hypertension Undergoing Organ Transplant Evaluation: A Case Series

Lauren Shaffer, MD, David Kaplan, MD.
University of Pennsylvania, Philadelphia, PA.

Introduction: Partial splenic artery embolization (PSAE) is an infrequently utilized approach to reduce portal hypertension (PHT) by reducing splenic arterial inflow and thereby splenic vein flow into the portal vein. We describe three patients who underwent PSAE to address diverse manifestations of PHT due to contraindications to transjugular intrahepatic portosystemic shunts (TIPS).
Case Description/Methods: **Case 1:** A 50-year-old male with CKD stage 4 and idiopathic PHT had diuretic-refractory ascites and thrombocytopenia, precluding surgery for an arteriovenous fistula (AVF) and kidney transplantation. He was not a candidate for TIPS due to extensive portal vein thrombosis but underwent PSAE leading to resolution of ascites and marked improvement in thrombocytopenia (Figure 1). The patient ultimately received an AVF and was placed on the kidney transplant waitlist. He has been followed for 30 months with sustained clinical improvement. **Case 2:** A 60-year-old male with prior hepatocellular carcinoma (HCC) status post right partial hepatectomy and hepatitis C cirrhosis complicated by severe thrombocytopenia and refractory ascites, had two previously aborted TIPS procedures due to unusual hepatic vein anatomy. He underwent PSAE that led to a reduction in frequency of therapeutic paracenteses and improvement of thrombocytopenia (Figure 1). Eight months later, TIPS was reattempted and resulted in complete ascites control for over six years. He is currently on the waitlist for liver transplantation. **Case 3:** A 60-year-old male had cryptogenic cirrhosis complicated by mild ascites, significant thrombocytopenia, and massive splenomegaly with mass effects. He underwent successful PSAE to alleviate splenic compression of the bladder. One year later, he had a repeat PSAE for diaphragmatic compression secondary to splenic hypertrophy. His thrombocytopenia also mildly improved (Figure 1). The patient was declined for liver transplant due to lack of social support. He expired from advanced HCC after two years of follow-up.
Discussion: We report three cases of PSAE in patients with PHT awaiting organ transplantation. All three cases had improvement in ascites, splenomegaly, and thrombocytopenia. Although PSAE is not the primary treatment for PHT, it may have a therapeutic role in advanced cases, particularly in transplant candidates when other interventions like TIPS are not possible. Future studies should be conducted to further evaluate the role of PSAE in patients with PHT awaiting organ transplantation.



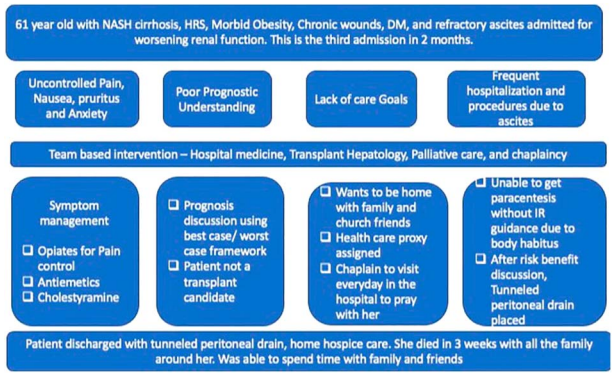
[3117] **Figure 1.** *2nd PSAE attempt in case 3 represented at time 0.

S3118

Palliative Care for Decompensated Cirrhosis

Austin Matney[1], Priya Abhyankar, MD[1], Kshitij Thakur, MD[2].
[1] University of Kentucky College of Medicine, Lexington, KY; [2]University of Kentucky, Lexington, KY.

Introduction: The total mortality of liver diseases and cirrhosis surpassed 51,000 deaths in 2020. Decompensated cirrhosis is often a terminal illness for patients that do not receive a transplant. Palliative care involvement for decompensated cirrhosis patients improves symptom management and documentation of goals of care. We present a case where palliative care consultation addressed goals of care, poor prognostic understanding, and symptom management in a patient with NASH cirrhosis complicated by refractory ascites.
Case Description/Methods: A 61-year-old female with a history of NASH cirrhosis, morbid obesity, chronic wounds, and refractory ascites was admitted for worsening renal function due to suspected hepatorenal syndrome (HRS). She required frequent paracenteses with interventional radiology (IR) due to body habitus. This was her third admission in two months. Transplant hepatology deemed she was not a liver transplant candidate. Her renal function continued to worsen despite HRS treatment. She had no clear goals of care documented and a poor understanding of her prognosis. She was managed by a multidisciplinary team involving hospital medicine, transplant hepatology, nephrology, and palliative care. Palliative care assessed the patient's unmet needs, including lack of care goals, poor prognostic understanding, and uncontrolled pain. Utilizing the best-case and worst-case framework, a family meeting was held to discuss patient prognosis. This revealed the patient's desire to return home for her remaining days. Palliative care managed her uncontrolled symptoms and discussed the utility of dialysis. She declined dialysis. Prior to discharge to home hospice, IR placed a palliative tunneled long-term peritoneal drain. She died after spending three weeks at home with family (Figure 1).
Discussion: Palliative care consultation has a positive impact on symptom management and documentation of goals of care, yet it is commonly delayed and only used for end-of-life care in decompensated cirrhosis patients. Barriers to consultation include misperceptions about palliative care, difficulty estimating prognosis, and the allure of transplant. As in our case, these patients may experience frequent procedures, disrupting quality of life. Palliative tunneled long-term abdominal drains can be an effective alternative for drainage of ascites when used in the right setting and patient population, allowing for better symptom management with continuous drainage of small amounts of ascites.



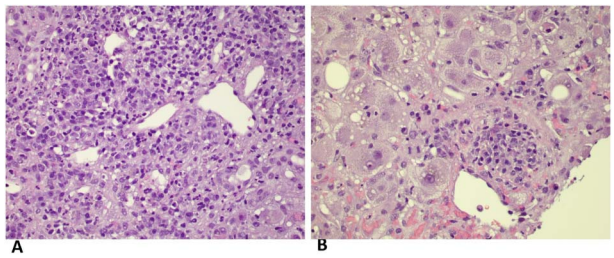
[3118] **Figure 1.** Hospital Course.

S3119

Patient With Autoimmune Hepatitis Requiring Liver Transplant: Case Report and Literature Review

Naif Hindosh, MD, Ragarupa Kotala, MD, Vikas Yellapu, MD, Kunal Bhagatwala, MD.
St. Luke's University Health Network, Easton, PA.

Introduction: Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease caused by autoimmune-induced damage to the hepatocytes. AIH is known for elevated anti-smooth muscle antibodies (ASMA) and serum globulin levels. A liver biopsy is needed for the diagnosis. Despite its favorable prognosis, few cases can progress to fulminant liver failure.
Case Description/Methods: 71-year-old male maintained on Descovy as preexposure prophylaxis for HIV for 1 year, was admitted 3 consecutive times over 1 month due to fatigue and jaundice. He was noted to have elevated transaminase and bilirubin levels. His presentation was suspected to be secondary to Descovy. Abdominal ultrasound was notable for hepatomegaly otherwise normal. He reported social alcohol consumption. He denied acetaminophen or new medication use. PE showed stable vital signs with jaundiced skin and hepatomegaly. The laboratory workup is shown below. Descovy was stopped and he was discharged. He was readmitted 1 week later for worsening symptoms. Workup showed positive ASMA for which he underwent a liver biopsy which was consistent with AIH. Infectious workup was unremarkable. He was started on a prednisone taper. Liver enzymes stabilized and he was discharged with plans to start azathioprine as an outpatient. However, he was admitted 2 weeks later for abdominal pain and encephalopathy. His labs showed worsening bilirubin, INR, and creatinine levels. CT head and abdomen were unremarkable. Doppler ultrasound demonstrated patent hepatic vasculature. He was placed on IV hydration, lactulose, and rifaximin. Due to concerns for hepato-renal syndrome, midodrine, octreotide, and albumin were added. He had a liver transplant evaluation which he underwent successfully. After prolonged hospitalization, he was discharged to post-acute rehab with a regimen consisting of tacrolimus, azathioprine, and prednisone (Figure 1).
Discussion: The initial treatment for AIH is glucocorticoid therapy. Azathioprine can be added in moderate to severe cases or when there is a contraindication from using high-dose steroids. Few studies showed that tacrolimus can be effective in some cases that do not respond to steroids. Response to treatment is assessed by improvement of symptoms, laboratory tests, and liver histology. A liver transplant remains a rare indication in AIH and is reserved for patients who do not respond to immunosuppressants or progress to fulminant liver failure. The recurrence rate is reported to be 20-40% with a possibility of developing de novo AIH in a few cases.



[3119] **Figure 1.** Image A is showing portal tract with reactive bile ducts, abundant plasma cells, and interface hepatitis (H&E stain, 10×). Image B is showing Lobular aggregate of plasma cells adjacent to a central vein and involving hepatocytes which show reactive changes including ballooning (H&E stain, 20×).

Table 1.

Test	Admission #1	Admission #2	Admission #3	Post-transplant	Reference range
AST	481	952	264	32	5-45 U/L
ALT	1048	1284	501	61	12-78 U/L
ALP	236	184	166	66	46-116 U/L
Total bilirubin	15.29	36.00	42.02	0.68	46-116 U/L
Direct bilirubin	11.47	26.99	30.82	0.24	0.00 - 0.20 mg/dL
Total protein	7.5	7.2	6	7	6.4-8.2 g/dL
Albumin	3.3	2.7	2.1	3.7	3.5-5.0 g/dL
PT	14.5	17.9	44	13.5	11.5-14.5 seconds
INR	1.13	1.49	5.04	1.07	0.84-1.19
BUN	20	19	107	39	5-25 mg/dL
Creatinine	1.28	1.39	4.03	4.37	0.6-1.3 mg/dL
eGFR	56	51	13	12	ml/min/1.73sq.m
Hemoglobin	17	16.5	16.7	11.3	12.0-17.0 g/dL
WBC	7.89	8.74	16.2	6.13	4.31-10.16 thousands/uL

Table 1. (*continued*)

Test	Admission #1	Admission #2	Admission #3	Post-transplant	Reference range
Platelets	125	118	165	165	149-390 thousands/uL
Sodium	134	132	131	139	136-145 mmol/L
Potassium	3.7	3.7	5.3	4.4	3.5-5.3 mmol/L
Chloride	100	100	103	103	100-108 mmol/L
Calcium	8.9	9.2	9.2	10.1	8.3-10.1 mmol/L
Ammonia	31	24	53	N/A	11 - 35 umol/L
ASMA		40			0-19 U
AMA		< 20			0-20 U
ANA	Negative				Negative
IMMUNOGLOBULIN A IMMUNOGLOBULIN G IMMUNOGLOBULIN M		101 1123 93			50-500 mg/dL 650-2000 mg/dL 40-270 mg/dL
Acetaminophen	< 2				10-20 ug/ml
Urine drug screen	Negative				Negative
Ceruloplasmin		22.2			16-31 mg/dL
A1 antitrypsin		131			90-200 mg/dL
Viral hepatitis panel	Negative				Negative
HIV 1/2 AB-AG	Negative				Negative

AST: Aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; INR: international normalized ratio; PT: prothrombin time; BUN: blood urea nitrogen; eGFR: estimated glomerular filtration rate; WBC: white blood count; ASMA: Anti-smooth muscle antibody; AMA: anti-mitochondrial antibody; ANA: antinuclear antibody.

S3120

Peritoneal Tuberculosis in a Healthy Man With No Risk Factors for Tuberculosis

Kimberline Chew, MD, Clara Tow, MD.
Montefiore Medical Center, Bronx, NY.

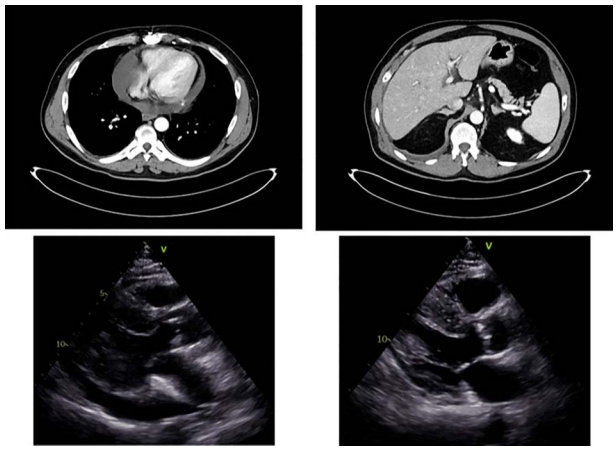
Introduction: Peritoneal tuberculosis (TB) only accounts for 4.7% of all cases of extrapulmonary TB and is associated with significant mortality (50-60%). The diagnosis of peritoneal TB is difficult given its rarity in developed countries, insidious onset and variable presentations. We present a case of a patient with no risk factors for TB who was found to have peritoneal TB.
Case Description/Methods: A middle-aged male with history of fevers of unknown origin for one year was sent to the hospital by his primary care doctor for abnormal liver tests. He reported a 100 lbs weight loss over one year. He had no known exposure to TB, was born in and never travelled outside of the United States, never incarcerated or lived in a shelter, and works as a bus driver. Ultrasound revealed new cirrhotic-looking liver with ascites. CT imaging showed left upper lobe micronodules, and mediastinal, lower thoracic and abdominopelvic lymphadenopathy. Diagnostic paracentesis yielded WBC of 3,300 cells/uL, lymphocytic predominance (82%) and serum ascitic albumin gradient (SAAG) of 0.8 g/dL. Ascitic culture was negative, and ascitic cytology was negative for malignant cells. Ceftriaxone was started for presumed spontaneous bacterial peritonitis as met criteria despite inconsistent fluid studies. He continued to have fevers up to 102.7°F during the hospital stay. Given the persistent fevers, ascitic fluid lymphocytic predominance and lymphadenopathy, malignancy was highest on the differential. Given that lung micronodules were present, serum QuantiFERON was also ordered, which came back positive. Repeat paracentesis revealed ascitic adenosine deaminase activity (ADA) elevated at 114.4 U/L. He was started on quadruple therapy. Given initial high suspicion for malignancy, a bone marrow biopsy was performed which showed overall normocellular marrow with no evidence of lymphoma. A few days after starting therapy, fever was no longer noted and he remained afebrile post-discharge.
Discussion: This describes a unique case of peritoneal TB in a patient with no known risk factors. Reaching the final diagnosis for this case was challenging because TB was not the highest on the differential, although liver cirrhosis does have an increased risk of developing peritoneal TB. Given its high mortality, early diagnosis and initiation of treatment are essential. This highlights that it is important for clinicians to consider peritoneal TB in all patients with lymphocytic ascites with a SAAG of <1.1 g/dL, regardless of risk factors.

S3121

Oysters Are Dangerous for Your Liver

Haider Abbas Naqvi, MD, Ashik Pokharel, MBBS, Christopher Haas, MD, PhD.
MedStar Health, Baltimore, MD.

Introduction: Hepatitis A is generally a benign, self-limited viral infection transmitted by the fecal-oral route. Since 2016, there have been over 33,000 reported infections in the United States. Outbreaks have been known to occur in the setting of seafood consumption; oysters have been identified as a main culprit. A few reports have noted extraintestinal manifestations of Hepatitis A, with only a single report noting pericarditis. No cases of pericardial effusion with or without tamponade have been reported.
Case Description/Methods: A 58-year-old male with coronary disease s/p bypass grafting, diverticulitis s/p colostomy with reversal, and diabetes mellitus type 2 presented with several days of constant, crampy left lower quadrant abdominal pain. He had otherwise been in usual health with no new exposures except for consuming a bushel of oysters a couple of days prior to symptom onset. On presentation he was hemodynamically stable. Initial labs demonstrated ALT [1480 U/L], AST [776 U/L], alkaline phosphatase [212 U/L], and D-dimer [5.70 mcg/mL], with a normal lipase and a positive Hepatitis A IgM. CT chest/abdomen/pelvis with IV contrast revealed no pulmonary embolism, a small to moderate pericardial effusion, and edema around the gallbladder without signs of acute cholecystitis. Hospital course complicated by cardiac tamponade, necessitating emergent pericardiocentesis and removal of 600 cc of transudative, dark-brown fluid and negative for ANA, RF, and anti-Scl7, malignant cells, and cultures. Subsequently, requiring pericardial window placement. He was discharged home with a pericardial drain and outpatient echocardiogram demonstrated resolution of the pericardial effusion and in clinic the drain was removed (Figure 1).
Discussion: Acute Hepatitis A infection may present on a spectrum from asymptomatic presentation to acute liver failure. Though primarily presenting with gastrointestinal symptomatology with viral prodrome, extraintestinal manifestations such as autoimmune hemolytic anemia, aplastic anemia, reactive arthritis, Guillain-Barré syndrome, myocarditis and pericardial effusions, have been rarely reported. Acute infection will have positive Hepatitis A IgM in conjunction with elevated transaminases, specifically ALT. The extraintestinal manifestations of Hepatitis viral infections are typically diagnoses of exclusion. Hepatitis induced pericardial effusion will have yellowish orange fluid and the presence of bilirubin on analysis. In the absence of liver failure, Hepatitis A is managed supportively.



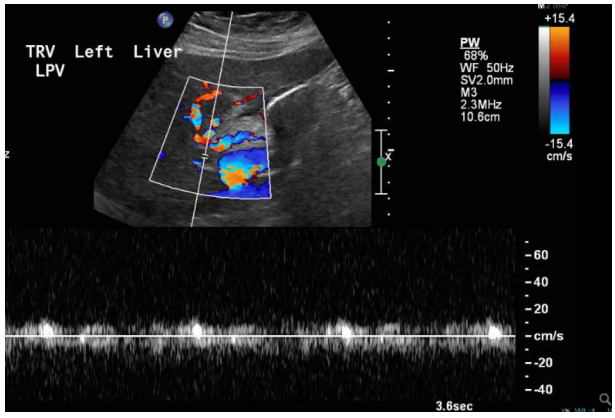
[3121] **Figure 1.** Presence of moderate pericardial effusion and normal liver morphology on CT scans. Pericardial Effusion and the development of tamponade. Resolution of pericardial effusion on follow-up echocardiogram.

S3122

Portal Vein Thrombosis Post Laparoscopic Sleeve Gastrectomy

Mujtaba Mohamed, MD[1], Ali Wakil, MD[2].
[1]Marshall University Joan C. Edwards School of Medicine, Huntington, WV; [2]Rutgers New Jersey Medical School, Newark, NJ.

Introduction: Portal vein thrombosis (PVT) is a clot formation in one of the branches of the portal vein. It is a well-known complication in patients with cirrhosis or post-abdominal surgery. Data on PVT post laparoscopic bariatric surgeries are scarce. Here we present a case of PVT post laparoscopic sleeve gastrectomy.
Case Description/Methods: A 43- years-old female with a known past medical history of hypertension, Asthma, and obesity underwent a robotic-assisted laparoscopic sleeve gastrectomy. She presented to the Emergency Department (ED) complaining of Abdominal pain 2 days after the sleeve gastrectomy operation. The patient reported doing well postoperatively till 2 days when she developed sudden epigastric abdominal pain and generalized abdominal discomfort, associated with nausea and anorexia. On arrival at the ED, her vital signs were stable and liver enzymes were normal. She had RUQ tenderness on the physical exam without guarding or rebound. The patient then had a Computed tomography (CT) of the abdomen and pelvis with IV contrast which showed occlusive left portal vein thrombosis extending to branches. She was started on oral direct Anticoagulant (DOAC) with Rivaroxaban. She had complete hypercoagulable work up with hematology which was unremarkable (Figure 1).
Discussion: PVT is a well-described complication of cirrhosis. in non-cirrhotic patients, the etiology of PVT can be due to systemic or local factors. A hypercoagulable state such as Factor V Leiden, antithrombin III deficiency, protein C and S deficiency, or Methylenetetrahydrofolate reductase (MTHFR) mutation is associated with higher thrombosis risk. Local factors include direct manipulation of portosystemic vessels such as during splenectomy with resultant endothelial damage and increase vascular thrombogenicity. A gastric sleeve is less likely to cause PVT since it only involves the short gastric vein. However, with the use of laparoscopic surgical techniques, there has been an increase in reported PVT. It is suspected that an increase in intra-abdominal pressure during carbon dioxide insufflation causes a pro-thrombotic state by decreasing venous and portal blood flow. The diagnosis of PVT post-operatively requires a high level of suspicion and can be made with abdominal imaging such as Ultrasound with color doppler or CT with IV contrast. The treatment is usually anticoagulation with the goal of recanalization of the portal vein on repeat imaging to prevent the development of non-cirrhotic portal hypertension in these patients.



[3122] **Figure 1.** Color doppler showing left portal vein thrombosis.

S3123

Piling on More Problems: A Rare Case of Pilewort Drug-Induced Liver Injury (DILI)

Peter Bhandari, MD, Isabella Bergagnini, DO, Luis Barraza, MD, Hillel Tobias, MD, PhD.
Northwell Health, Lenox Hill Hospital, New York, NY.

Introduction: Herbal supplements (HS) are being widely utilized for the self-treatment of various medical conditions. Despite their growing popularity, HS utilization has resulted in drug induced liver injury (DILI) as well as acute liver failure (ALF). Lesser Celandine, also known as pilewort, is a topical and ingestible HS native to Northern Africa, used for the treatment of hemorrhoids. Here, we present a case of severe DILI secondary to this rarely reported agent.
Case Description/Methods: A 34-year-old female with hemorrhoids and fibroids presented after noticing yellowing of her eyes for one week. She denied associated fevers, nausea, vomiting, abdominal pain, toxic habits, personal or familial history of autoimmune illnesses or malignancy. She traveled to Ghana 10 months prior, at which time she self-initiated treatment with the HS known as pilewort for her hemorrhoids. Physical exam demonstrated enlarged abdominal girth, scleral icterus, and was negative for asterixis. Laboratory studies (Table 1) revealed severe hepatocellular injury and hyperbilirubinemia. Autoimmune and viral hepatitis serologies were negative. CT abdomen and pelvis revealed no hepatosplenomegaly, no biliary ductal dilation, and an enlarged leiomyomatous uterus. Non-acetaminophen N-

Acetylcysteine protocol was initiated with observed improvement in her liver enzymes. She subsequently underwent a liver biopsy, which exhibited confluent pericentral necroinflammation, apoptotic hepatocytes, and no evidence of steatosis or fibrosis, consistent with DILI. The patient ultimately left against medical advice and was lost to follow up.
Discussion: DILI comprises up to 50% of ALF cases in the United States, with a higher incidence in the Eastern world secondary to increased use of HS. Identification remains a challenge, as presentation is highly variable. Diagnosis is based on exclusion with workup largely dependent on pattern of liver injury. As in our case, in those with hepatocellular injury, ischemia, viral, autoimmune and toxic etiologies must be ruled out. A comprehensive medication reconciliation is essential, including a detailed inquiry on the use of herbal medicines, especially in cases of unexplained liver injury. Pilewort DILI is reported once in the literature since first described in 1904. We present this case to raise awareness of the hepatotoxic properties of pilewort.

Table 1. Laboratory Investigations

	On Admission	Reference Range
Hemoglobin g/dL	10.6 g/dL	11.5-15.5 g/dL
Alkaline Phosphatase units/L	92 U/L	40-120 U/L
AST units/L	1096 U/L	10-40 U/L
ALT units/L	807 U/L	10-45 U/L
Total Bilirubin mg/dL	4.9 mg/dL	0.2-1.2 mg/dL
Direct Bilirubin mg/dL	3.0 mg/dL	0-0.3 mg/dL
MELD-Na	18	
R Factor	26.3	
Hepatitis A IgM	Negative	
Hepatitis B Surface Ab	Positive	
Hepatitis B Surface Ag	Negative	
Hepatitis C Ab	Negative	
CMV IgM	Negative	
Alpha-1 Antitrypsin mg/dL	164 mg/dL	90-200 mg/dL
Ceruloplasmin mg/dL	25 mg/dL	16-45 mg/dL
Alcohol, Blood	Not Detectable	
Acetaminophen	Not Detectable	
Ethyl Glucuronide	Not Detectable	
Urine Toxicology	Negative	
Anti-nuclear Antibody	Negative	
Anti-mitochondrial Antibody	Negative	
Smooth Muscle Antibody	1:20	
Liver Kidney Microsomal Antibody	Negative	
HSV-1 IgG Ab	Positive	
HIV-1/2 Ag/Ab Combo	Negative	
Quantitative IgA	165 mg/dL	84-499 mg/dL
Quantitative IgM	220 mg/dL	35-242 mg/dL
Quantitative IgG	2252 mg/dL	610-1660 mg/dL

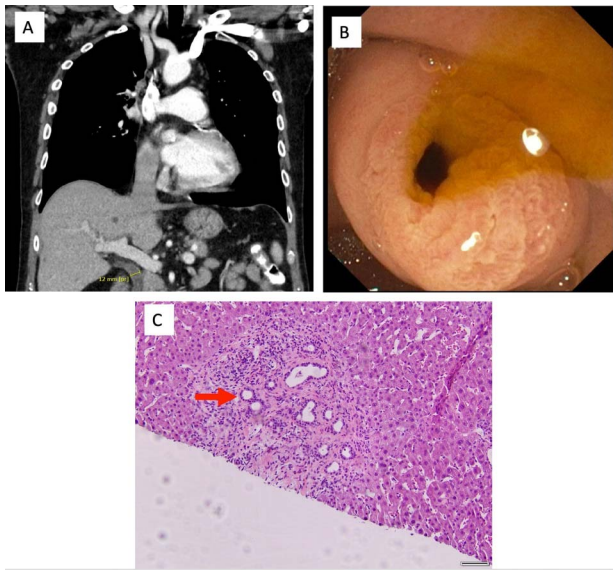
S3124 WITHDRAWN

S3125

Overlapping Hepatotoxicity and Colitis Associated With Immune Checkpoint Inhibitors

Alexander Malik, MD[1], Muhammad Nadeem Yousaf, MD[2], Sami Samiullah, MD[1], Tahan Veysel, MD[2], Yuchu Sun, MD[1], Amin Mahdi, MD[1], Omer Basar, MD[2].
[1]Summa Health System, Akron, OH; [2]University of Missouri, Columbia, MO.

Introduction: Immunotherapy with immune checkpoint inhibitors (ICIs) has become the standard of care for many solid-organ and hematologic malignancies. Gastrointestinal toxicities are common side effects that typically occur 6 weeks after starting ICI therapy. Overlapping hepatotoxicity and colitis associated with ICIs is rare and may be overlooked, resulting in high mortality.
Case Description/Methods: A 73-year-old man with metastatic squamous cell carcinoma (SCC) of the head and neck presented with intermittent, diffuse abdominal discomfort, bloating and painless non-bloody diarrhea (up to 6 episodes daily) triggered by meals. Symptom onset was 6 weeks after starting pembrolizumab for head and neck SCC. Pembrolizumab therapy was held, and supportive therapy with proton pump inhibitors, antacids and antidiarrheals provided partial symptom relief. Laboratory work up showed elevated AST 132 U/L, ALT 220 U/L, ALP 851 U/L and normal total bilirubin. ANA was elevated with titer 1:80, with normal total IgG (699.5 mg/dL) and negative serologies for anti-smooth muscle antibody, CMV, EBV, and HSV. Abdominal CT with contrast showed a dilated common bile duct (CBD) of 11 mm and a new curvilinear filling defect in the distal CBD, without choledocholithiasis. MRCP revealed mildly dilated intrahepatic biliary ducts, CBD dilation (13 mm), and an ovoid 8mm soft tissue nodule in the periampullary duodenum. Upper endoscopy was nondiagnostic. At this time, the patient's symptoms improved, but there was high suspicion of grade-2 ICI-associated hepatitis (ICIH). Percutaneous liver biopsy showed interlobular bile duct injury with intraepithelial lymphocytes and neutrophils and a neutrophilic ductular reaction consistent with ICIH. Serial lab work demonstrated improvement of liver enzymes, with AST 77 U/L, ALT 69 U/L and ALP 667 U/L at 60 days after initial labs. Diarrhea resumed, up to 6 episodes per day, consistent with the Common Terminology Criteria for Adverse Events diagnosis of ICI-related colitis (ICIC). Initial supportive therapy failed to improve diarrhea; thus, a prednisone taper was started, resolving the colitis. He declined the option to resume immunotherapy and transitioned to comfort care (Figure 1).
Discussion: Overlapping ICIH and ICIC is rare and may have discordance between symptomatology and lab findings. Early recognition of overlapping ICIH and ICIC, supportive treatment, permanent cessation of the inciting ICI and switching it with alternative immunosuppressive therapy may improve patient outcomes and quality of life.



[3125] **Figure 1.** Panel A - CT chest/abdomen with contrast (coronal view) showing common bile duct dilation of 1.2 cm (yellow calipers) status post cholecystectomy. Panel B. Upper endoscopy showing prominent ampulla with free-flowing bile. Panel C. Histology of percutaneous, ultrasound-guided liver biopsy with H&E staining (10×) shows preserved lobular hepatic architecture with mixed inflammatory infiltrate including lymphocytes, histiocytes, and scattered eosinophils expanding the portal tracts.

Table 1. Immune Checkpoint Inhibitor Associated Hepatitis Grades, adapted from the American Society of Clinical Oncology, 2021

Grade	Criteria
1	Asymptomatic (AST or ALT > ULN to 3.0 X ULN and/or total bilirubin > ULN to 1.5 X ULN)
2	Asymptomatic (AST or ALT > 3.0 to ≤ 5 X ULN and/or total bilirubin > 1.5 to ≤ 3 X ULN)
3	AST or ALT 5-20 X ULN and/or total bilirubin 3-10 X ULN, OR symptomatic liver dysfunction; fibrosis by biopsy; compensated cirrhosis; and reactivation of chronic hepatitis
4	AST or ALT > 20 X ULN and/or total bilirubin > 10 X ULN OR decompensated liver function (e.g., ascites, coagulopathy, encephalopathy, and coma)

Abbreviations: AST, aspartate transferase; ALT, alanine transaminase; ULN, upper limit of normal.

S3126

Peritoneal Mesothelioma as an Unexpected Cause of Recurrent Abdominal Ascites

Nida Anwaar, MD[1], Jason Chang, MD[2], Muhammad Masood, MD[1].
[1]Danbury Hospital, Danbury, CT; [2]Stanford University Medical Center, Palo Alto, CA.

Introduction: Peritoneal mesothelioma is an aggressive neoplasm that arises from the lining mesothelial cells of the peritoneum. The disease is very rare with an incidence of 1 case per 4–5 million of the population. Peritoneal mesothelioma accounts for approximately 20–30% of all mesothelioma type cancers, and as with all forms of mesothelioma, asbestos exposure is a strong risk factor associated with the disease.
Case Description/Methods: A 68-year-old male presented with progressively worsening abdominal distension and generalized lethargy for a few months. His past medical history includes untreated rheumatoid arthritis and a 40-pack-year history of tobacco smoking with recent cessation. He was a veteran in the army reserve but never saw combat. He had undergone therapeutic paracentesis removing 5 L of clear yellow fluid a week prior, but developed recurrence of abdominal distention within a few days thus came to the emergency room for evaluation. A CT scan of abdomen and pelvis with contrast revealed tense ascites throughout the abdomen, in addition to hepatic nodules and a large area of soft tissue attenuation suspicious for diffuse carcinomatosis. A diagnostic paracentesis performed in the ED was negative for malignant cells. He was admitted to the hospital for recurrent ascites requiring workups for possible malignancy. Soon after admission he underwent a large-volume paracentesis which removed 5.1 L of yellow, foamy fluid with no evidence of bacterial infection. A CT-guided biopsy of an omental mass revealed malignant mesothelioma, epithelioid type. Per assessment by Medical Oncology patient was determined to be a poor candidate for cytoreductive surgery (CRS) or HIPEC therapy, but palliative chemotherapy was considered a viable option. He was subsequently discharged from the hospital with a peritoneal drainage catheter to be used thrice-weekly for rapid re-accumulation of ascites.
Discussion: There is currently no consensus as to the optimal treatment for peritoneal mesothelioma. For patients who are not candidates for CRS/HIPEC, systemic chemotherapy with a pemetrexed-containing regimen is usually the preferred option. Immunotherapy has shown activity in pleural mesothelioma, but data is limited on its use in mesothelioma of extrapleural sites and there are no consensus-based guidelines regarding immunotherapy for peritoneal mesothelioma. In conclusion, peritoneal mesothelioma is a rare cancer with a poor prognosis, and further studies are needed to determine the best treatment option.

S3127

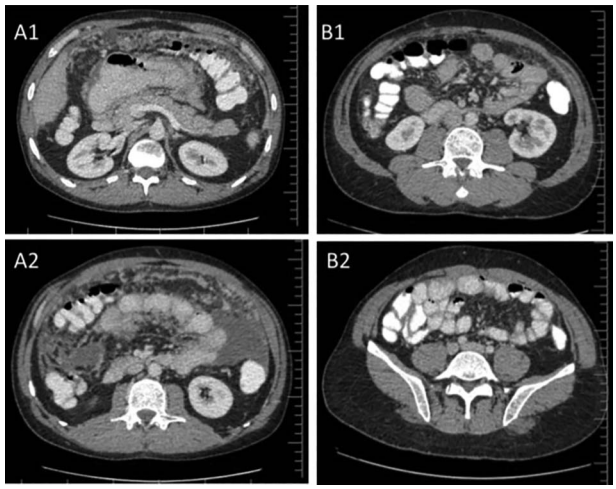
Peritoneal Tuberculosis in an Immunocompetent Patient With Findings of Chronic Liver Disease and Peritoneal Carcinomatosis

Nazli Begum Ozturk, MD[1], Naile Dolek, MD[2], Raim Iliaz, MD[2].
[1]Istanbul University, Istanbul School of Medicine, Rochester, MN; [2]Atlas University School of Medicine, Istanbul, Istanbul, Turkey.

Introduction: Peritoneal tuberculosis (PT) accounts for 1-2% of all tuberculosis cases. Diagnosis of PT can be challenging due to non-specific symptoms, insidious onset, and variable imaging findings. We present a case of PT presenting with ascites and imaging studies suggestive of chronic liver disease (CLD), and peritoneal carcinomatosis.
Case Description/Methods: A 48-year-old man presented to our clinic with complaints of increased abdominal girth, weight loss, and night sweats for the past 3 months. Physical exam revealed soft and distended abdomen with shifting dullness. Laboratory tests showed a mild anemia of Hb at 13.0 g/dL (13.1-17.2) and ESR at 34 mm/h (0-15). Abdominal ultrasound (US) revealed the presence of free abdominal fluid, and diffuse heterogenous granular liver parenchyma supporting the diagnosis of CLD. His liver tests were within normal ranges. Ascitic fluid analysis revealed serum ascites albumin gradient (SAAG) of 0.2 g/dL, WBCs of 1.70×10^3 cells/dL (91.8% lymphocytes). Ascitic fluid cytology was negative for malignant cells. Ascitic fluid acid-fast bacilli (AFB) test and mycobacterial culture were negative. Adenosine deaminase (ADA) level in ascitic fluid was 108.5 U/L (0-40). Interferon-gamma release assay (IGRA) test was positive. Contrast-enhanced computed tomography (CT) scan revealed normal liver parenchyma, diffuse thickening and nodularity of the peritoneum, and thickening of omentum with omental cake appearance. CT scan was suggestive of

peritoneal carcinomatosis. EGD and colonoscopy were unremarkable. With consistent clinical history, SAAG < 1.1, ascitic fluid with lymphocytic predominance, high ADA levels, and positive IGRA, a diagnosis of PT was made. Anti-tuberculosis treatment was initiated, and the patient's clinical symptoms have improved. CT scan at the end of treatment showed the resolution of ascites and peritoneal findings (Figure 1).

Discussion: Ascites is the most common presenting symptom of PT. High suspicion of PT is important in the setting of ascites of unknown origin. Despite the US findings were suggestive of CLD in this patient, CT scan revealed normal liver findings. Differing PT from peritoneal carcinomatosis might be challenging due to overlapping findings on imaging. Ascitic fluid analysis of SAAG < 1.1, high ADA levels, positive IGRA test should lead towards the diagnosis of PT. AFB and mycobacterial culture of the ascitic fluid may often have low diagnostic yield in PT. Early diagnosis and treatment are important to prevent complications.



[3127] **Figure 1.** A1, A2, Before treatment; axial contrast-enhanced computed tomography scan showing moderate ascites, and diffuse thickening and nodularity of peritoneum. Imaging is suggestive of peritoneal carcinomatosis. B1, B2, After treatment; resolution of ascites and peritoneal findings.

S3128

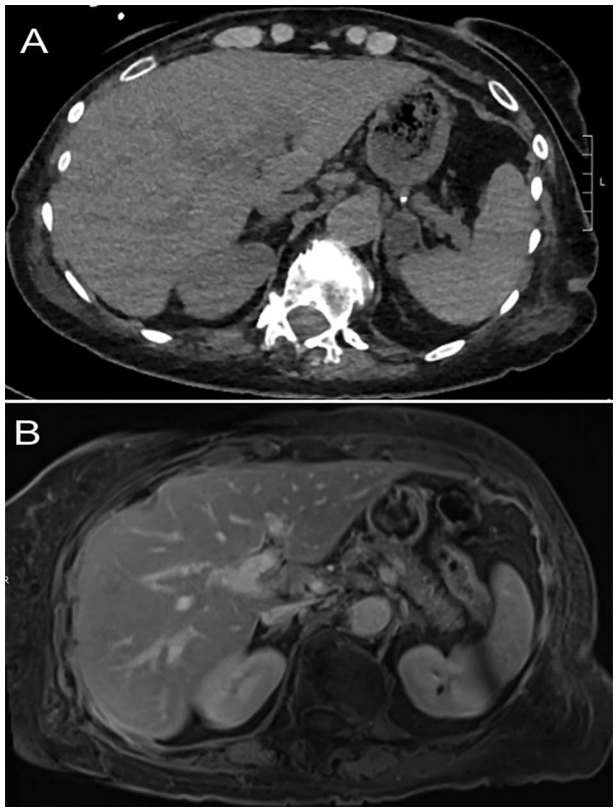
Positive Smooth Muscle Antibodies in a Patient With Pembrolizumab-Induced Liver Injury

Julianna Tantum, DO, Nadia Al Haddad, MD, Romy Chamoun, MD, Michael Choe, MD.
Lankenau Medical Center, Wynnewood, PA.

Introduction: Pembrolizumab is an anti-programmed cell death (PD-1) receptor inhibitor that is commonly used for the treatment of small-cell lung cancer (SCLC). This checkpoint inhibitor has been associated with numerous immune-related side effects, including colitis, vasculitis, myocarditis, and hepatitis. We report a case of pembrolizumab-induced liver injury in a SCLC patient that was successfully treated with steroids.

Case Description/Methods: This is a 74-year-old female with a history of SCLC who presented to the hospital with abnormal liver function tests (LFTs) on outpatient labs. She had a 2-month history of jaundice, fatigue, lower extremity weakness, and generalized malaise. Her last pembrolizumab infusion was 10 days prior. On admission, she was hemodynamically stable. Labs were notable for AST 1,211 IU/L, ALT 674 IU/L, alkaline phosphatase 1,216 IU/L, total bilirubin 13.8 mg/dL, direct bilirubin 8.8 mg/dL, total CK 4,474 U/L. Serum ethanol and acetaminophen levels were negative. Imaging was unremarkable (Image 1A). She was treated with N-acetylcysteine, ceftriaxone & metronidazole, and methylprednisolone 60 mg IV daily. Acute hepatitis panel, hepatitis C antibody, ANA, LKM-1 antibody, IGG subclass, and anti-mitochondrial antibody were negative. Smooth muscle antibody (SM-Ab) was positive with titer 1:40. Four days after admission, her labs worsened. MRCP was unremarkable (Image 1B). Methylprednisolone was increased to 100 mg daily with improvement in her LFTs after 3 days. She was changed to prednisone 80 mg daily and was discharged on a steroid taper with normalization of her LFTs 1 month later. Due to the temporal relationship with her infusion, her SM-Ab positivity was not representative of autoimmune hepatitis but rather of drug induced liver injury.

Discussion: Immune-mediated hepatitis is one of the most common and serious side effects of pembrolizumab. Severe elevations of LFTs, such as in our patient, are uncommon, occurring in about 1% of patients. It is postulated that anti-PD-1 antibodies remain attached to lymphocytes weeks after the last infusion, which can cause hepatitis after discontinuation or long after initiation of therapy. Most cases resolve with treatment, including immunosuppressants and corticosteroids, but immune-mediated hepatitis related deaths have been reported. After resolution, most patients can restart therapy with close monitoring but often require concomitant immunosuppressant therapy.



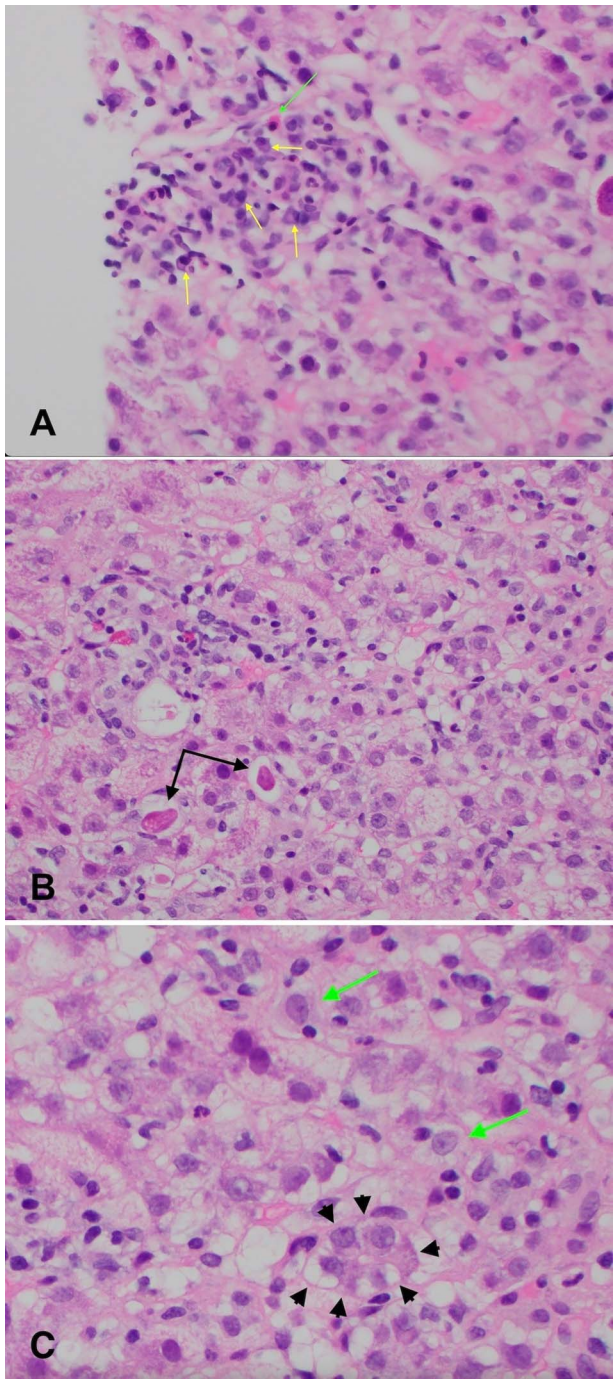
[3128] **Figure 1.** A, Computed Tomography (CT) abdomen and pelvis with intravenous contrast showing a collapsed but thickened gallbladder with trace pericholecystic fluid, no biliary ductal dilation, mild periportal edema. 1B, Magnetic resonance cholangiopancreatography (MRCP) showing no intrahepatic or common bile ductal dilation or stricture, no choledocholithiasis, normal liver size, and periportal edema.

S3129

Pregnancy-Induced Type 2 Autoimmune Hepatitis

Brandon H. Busch, MD, Michael Eiswerth, DO, Freeha Khan, MD, Matthew Cave, MD.
University of Louisville, Louisville, KY.

Introduction: Pregnancy has been established as a risk factor for the generation of autoimmune disease. The pathophysiology linking pregnancy and autoimmune disease is not well-established but hypothesized to be a result of hormonal modulation and fetal microchimerism. We report a case of Type 2 Autoimmune Hepatitis (AIH) that is elicited by pregnancy as confirmed by the presence of high titer anti-LKM (Liver-Kidney Microsomal) antibodies and classic histopathologic findings. To our knowledge, this is the first reported case of Type 2 Autoimmune Hepatitis directly triggered by pregnancy.
Case Description/Methods: A 19-year-old female with a history of recent uncomplicated pregnancy with successful delivery 3 months prior presented to the hospital with complaint of 1 week of right upper quadrant pain and 2 days of itching and yellowing of her skin. Exam showed scleral icterus and jaundice while abdomen was negative for tenderness or palpable mass. Labs revealed elevated AST at 670 U/L, ALT of 1107, ALP of 329 U/L, total bilirubin of 6.7 mg/dL, conjugated bilirubin of 4.8 mg/dL, unconjugated bilirubin of 1.9 mg/dL, and an INR of 1.3. These labs were all within normal limits 3 months prior to admission. Further workup revealed positive anti-liver-kidney (anti-LKM) antibodies with a titer of >1:2560. Imaging was normal. Liver biopsy revealed interface hepatitis, plasma cell infiltration, apoptotic hepatocytes, emperipolesis and rosette formation consistent with autoimmune hepatitis (Figure 1).
Discussion: The mechanism for initiation of autoimmune disease is of great interest and is unclear in the setting of pregnancy. It has been hypothesized that exchange of fetal and maternal cells occurs, known as fetal microchimerism, which may be a trigger for generation of autoimmune disease in pregnancy. This mechanism is proposed to be through immune sensitization by exposure to HLA-susceptibility alleles or through a graft-vs-host type of mechanism. Another proposed mechanism suggests that hormonal fluctuations mediate a shift between Th1 and Th2-mediated immunity during pregnancy. In the 3rd trimester and continuing into the postpartum period, there is a decrease in hCG which results in decreased expression of T-regulatory populations and resultant increase in inflammation. Prolactin increases which results in increased production of proinflammatory TNF-α, INF-γ, and IL-2. We believe this patient had a defect in allogeneic immune tolerance during pregnancy with resultant initial flare of AIH in the post-partum period.



[3129] **Figure 1.** A, H&E stain at 400× magnification. Portal tract with lymphocytic infiltrate into surrounding hepatocytes consistent with interface hepatitis. Plasma cells present and indicated by yellow arrows. A lone eosinophil indicated by the green arrow. B, H&E stain at 400× magnification. Acidophil bodies present (indicated by black arrows) which represent apoptotic hepatocytes, found in a background of lobular inflammation. C, H&E stain at 400× magnification. Emperipolesis is present (indicated by green arrows) and is characterized by infiltration of lymphocytes into the cytoplasm of a hepatocyte. Rosette formation observed (outlined by black arrowheads) which represents small groups of hepatocytes arranged around a lumen and thought to be indicative of hepatocellular regeneration. Emperipolesis and rosette formation are thought to be typical of autoimmune hepatitis.

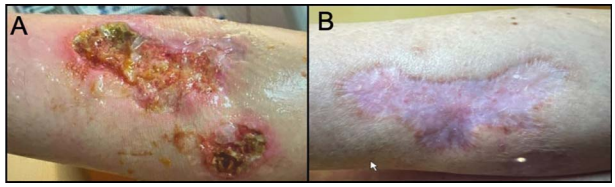
S3130

Recognizing Deadly Skin Lesions in a Post Liver Transplant Patient: A Survival Story

Aldanah Althwanay, MD, Sassan Sakiani, MD.
University of Maryland Medical Center, Baltimore, MD.

Introduction: Calciphylaxis is a syndrome where calcium accumulates in vessels of the fat and skin, leading to necrosis, infections, and potentially death. The one year mortality is more than 50%. There are two types: uremic and non-uremic (NUC). The latter is rare and has poorly understood associations, such as Caucasian race, female sex, connective tissue disease, and medications such as warfarin and steroids. In the literature thus far, there are only 3 reported cases of NUC post orthotopic liver transplantation (OLT) with varied prognosis. Etiology is largely unclear, but high doses of steroids have been associated with this. Further studying of possible correlation is vital to improve outcomes in OLT population.

Case Description/Methods: A 48-year-old woman with decompensated alcoholic cirrhosis was transferred for liver transplant evaluation after presenting with jaundice, ascites, and a MELD score of 41. She underwent OLT and was discharged on prednisone 20 mg, tacrolimus 3 mg BID, and mycophenolic acid 360 mg. There was no history of calcium, vitamin D supplementation or warfarin use. Post-operative course was complicated by rapid liver enzymes elevation (AST 1267 units/L, ALT 704 units/L) following initially down-trending values (AST 100 units/L, ALT 153 units/L) in the week following transplant. A liver biopsy was performed and showed mild-moderate acute cellular rejection (Banff 5). Prednisone was increased from 20 mg to 60 mg daily, with subsequent improvement in liver enzymes. A month later, she developed ulcers on her legs. She had a wedge biopsy, demonstrating multiple calcified small vessels in the subcutis and fat necrosis, indicative of calciphylaxis. Her kidney functions, calcium, phosphorus, vitamin D, and parathyroid hormone levels were within normal range (Table 1). Similarly, auto-immune workup, protein C and protein S screen were unremarkable. Her prednisone was tapered off, sodium thiosulfate infusions were started, and wound care follow-ups were arranged; resulting in marked improvement in the lesions (Figure 1).
Discussion: NUC is a rare fatal condition associated with several risk factors. In this case, the cause of NUC was uncertain, triggering a thorough literature review for clinical correlation with OLT population. Etiology is largely unclear and prognosis varied, but there was some association with steroids. Given the timing of the increase in steroid dose and the appearance of the lesions, steroids were thought to be the cause, and where tapered off subsequently resulting in improvement.



[3130] **Figure 1.** A, Subcutaneous nodules with ulceration and sloughing of overlying skin involving the posterior calve. B, Healed lesions with residual scarring after 3 months of treatment with sodium thiosulfate, wound care, and hyperbaric oxygen.

Table 1. Calcium Phosphate Metabolism "Calciphylaxis" Lab Values

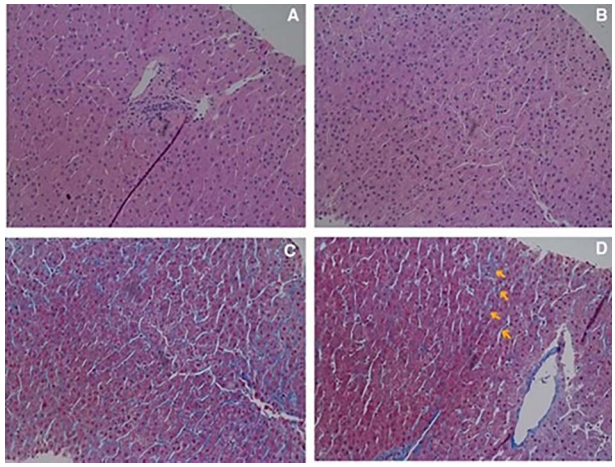
Lab test	Lab value	Reference range
Creatinine	1.05 mg/dL	0.56-1.00 mg/dL
eGFR	63 mL/min	>59 mL/min/1.73
Blood Urea Nitrogen	20 mg/dL	8.0 - 25.0 mg/dL
Calcium	9.6 mg/dL	8.4 - 10.1 mg/dL
Phosphorus	4.2 mg/dL	3.0 - 4.3 mg/dL
Vitamin D	25 ng/mL	20 - 80 ng/mL
Parathyroid hormone level	21 pg/mL	15 - 65 pg/mL

S3131

Rare Case of Liver Fibrosis and Hepatic Manifestations in HNF1B

John H. Kinney, Ahmed Sherif, MD, Wesam Frandah, MD, Tejas Joshi, MD.
Marshall University Joan C. Edwards School of Medicine, Huntington, WV.

Introduction: Mature Onset Diabetes of the Young (MODY) is a rare form of DM caused by various mutations. Hepatocyte Nuclear Factor 1-beta (HNF1B) defects are a rare cause of MODY that can result renal cysts and abnormalities of the uterine tract and pancreas. MODY increases the risk of complications of diabetes, such as cirrhosis. Diabetic cirrhosis is often associated with steatosis of the liver. The prevalence of NAFLD in Type 2 diabetics is thought to be 30-50%. Rarely do we see severe liver fibrosis without steatosis. Diabetic hepatosclerosis is thought to be secondary to microangiopathic injury. Such cases present with normal serum aminotransferase levels (ATL) and elevated alkaline phosphatase (AP) levels, likely a result of decreased sinusoidal volume. NAFLD patients present with elevated ATL and normal AP, due to steatohepatitis. We present a case of persistently elevated AP with normal ATL and hepatic fibrosis with biopsy findings of NASH.
Case Description/Methods: A 40-year-old F with a BMI of 20 and PMH of HNF1B mediated MODY, anatomical defects of the pancreas and uterus, and 5 year elevation in AP. Fibroscan confirmed stage 2 fibrosis of the liver and no steatosis. Liver biopsy confirmed Fibroscan results, and showed nodular regenerative hyperplasia (NRH), perisinusoidal fibrosis, and diabetic hepatosclerosis (Figure 1). CT scan of the abdomen showed normal liver and spleen and absent body and tail of pancreas.
Discussion: This patient is of particular interest due to their fibrotic liver damage in the presence of diabetes and in the absence of any fatty liver disease. Hepatosclerosis induced liver damage is a rare cause of liver fibrosis in diabetics. NRH is a rare condition that is associated with autoimmune disease, immunodeficiency, hematologic factors, infection, neoplasms, and drug-related cases (2). To our knowledge this is the first published case of HNF1B mediated liver fibrosis and findings of NRH in this patient population. It is important to monitor these patients in regard to progression towards cirrhosis as well as monitoring for presence of non-cirrhotic portal hypertension and its complications seen in patients with NRH.



[3131] **Figure 1.** Diabetic Hepatosclerosis in a Woman with Type 3 MODY.

Table 1. Patient Lab Data

AP	Aspartate Aminotransferase	Alanine Aminotransferase	Total Bilirubin	Gamma Glutamyl Transpeptidase	Platelet Count	HbA1c	C-Reactive Protein
589 U/L	48 U/L	75 U/L	0.8 mg/dL	662 U/L	325,000 platelets /mcL	10.0%	< 0.2 mg/L

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S3132

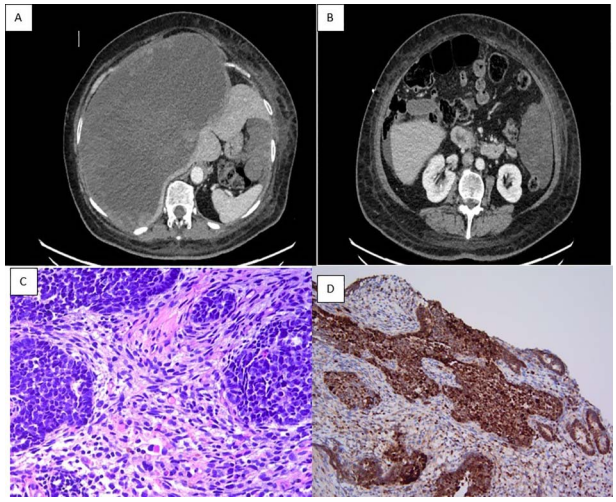
Primary Malignant Mixed Mullerian Tumor: An Uncommon Liver Cyst

Zunirah Ahmed, MD, Rose Anton, MD, Joseph Anton, David Victor, MD.
Houston Methodist Hospital, Houston, TX.

Introduction: Carcinosarcoma, or malignant mixed Mullerian tumor (MMMT), is a biphasic malignant neoplasm consisting of epithelial and non-epithelial components.These tumors typically rise from the female genital tract. Primary extragenital MMMTs are exceedingly rare.

Case Description/Methods: 71 yrs old woman presented with 3-month history of diffuse abdominal pain associated with abdominal distension and lower extremity edema. Other history was unremarkable. Physical examination showed a distended abdomen with liver edge palpable 4 cm below the coastal margin and edema in lower extremities. Laboratory data showed WBC 14 k/uL, ALT 50 U/L, AST 86 U/L, AP 388 U/L, and total bilirubin 0.3 mg/dL. CT abdomen and pelvis demonstrated a multiloculated cystic mass in the liver measuring 22 cm with septation, cystic lesion in the left peritoneum adjacent to the ascending colon measuring 15 cm, and a peritoneal nodule near the umbilicus measuring 3.5 cm (Figure 1A, B).CA 125 was 481 U/mL, AFP 42 ng/mL, CA 19-9 14 U/mL, and CEA < 1.2 ng/mL. She underwent drain placement into the liver cyst. Fluid cultures and cytology showed no infection or malignancy. Laparoscopic abdominal exploration was performed which demonstrated omental caking and multiple peritoneal nodules. Omental nodule biopsies showed carcinosarcoma composed of high-grade epithelial and mesenchymal components, with areas of rhabdomyosarcomatous differentiation (1C). Immunohistochemical staining favored Mullerian origin (1D). CT chest, colonoscopy, and transvaginal ultrasound were unremarkable. Diagnosis of primary peritoneal carcinosarcoma was made and carboplatin and paclitaxel were started. Her hospital course was complicated with extensive left lower extremity DVT. She was not deemed a candidate for cytoreductive surgery and opted for hospice.

Discussion: Primary peritoneal carcinosarcomas mostly occur in the pelvic peritoneum, followed by serosal surface of the colon, retroperitoneum, and omentum. Extragenital carcinosarcomas are very rare. Carcinosarcomas of extragenital sites are thought to arise from Müllerian duct remnants, secondary Müllerian system, or pre-existing foci of endometriosis, all of which are derivatives of the coelomic epithelium. Due to a common embryonic origin of the ovary and peritoneum, they have much histologic similarity. Most cases occur in women above the age of 40. Complete cytoreduction surgery and systemic chemotherapy is the mainstay of treatment. These tumors have poor prognosis with average survival rate between 11-17 months.



[3132] **Figure 1.** (A and B) CT abdomen and pelvis with multiloculated cystic mass in the liver measuring 22 cm with septation and cystic lesion in the left peritoneum adjacent to the ascending colon measuring 15 cm (C) islands of malignant epithelial cells are separated by a malignant cellular stroma with rhabdomyosarcomatous differentiation (H&E, 100×) (D) Pax8 immunostain is positive in the epithelial components (100×).

S3133

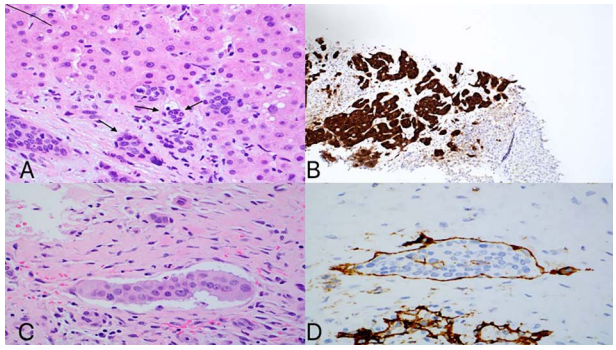
Pseudo-Cirrhosis With Portal Hypertension Secondary to Metastatic p16+ Vaginal Squamous Cell Carcinoma

Sarah Schimming, DO[1], David N. Oakland, BS[2], Shravani Reddy, MD[1], Farhad Sahebjam, MD[1], Douglas Grider, MD[1].
[1]Virginia Tech Carilion School of Medicine, Roanoke, VA; [2]Virginia Tech Carilion, Roanoke, VA.

Introduction: The liver is a common site of metastasis. Adenocarcinoma is the most common type of hepatic metastasis (75%) followed by small cell carcinoma (5.9%) and neuroendocrine carcinoma (4.6%). Squamous cell carcinoma (SCC) is rarely metastatic to the liver, representing only 1.4% of all cases. Presented is the first reported case of metastatic p16+ vaginal SCC to the liver with biliary and sinusoidal involvement clinically mimicking portal hypertension caused by cirrhosis.

Case Description/Methods: A 68-year-old female underwent an outpatient colonoscopy for rectal bleeding and was found to have a rectal mass involving the posterior vaginal wall. Biopsies from the rectal and vaginal lesions showed invasive SCC with venous invasion (Figures 1C and D). The carcinoma was p16+ representing a high-risk human papilloma virus induced SCC. Computed tomography of the chest, abdomen and pelvis showed the vaginal mass and a nodular liver suggestive of cirrhosis. In preparation for palliative chemotherapy, blood work was obtained and showed elevated liver enzymes: aspartate aminotransferase 127 U/L, alanine aminotransferase 65 U/L, alkaline phosphatase 1133 U/L and bilirubin 4.8 mg/dL. Concern for biliary obstruction prompted inpatient evaluation. Abdominal ultrasound and subsequent magnetic resonance cholangiopancreaticogram (MRCP) demonstrated cirrhosis and ascites without biliary obstruction. Serologic work up was unremarkable for viral, autoimmune or metabolic etiologies of liver disease. A non-targeted, trans-jugular liver biopsy showed diffuse infiltration of SCC with biliary and sinusoidal involvement (Figures 1A and B). The hepatic venous pressure gradient was 12 mmHg which confirmed portal hypertension in the setting of infiltrative metastasis. Given the extent of disease, the patient opted for palliative radiation with transition to hospice care.

Discussion: This is a rare case of infiltrative hepatic metastasis with biliary and sinusoidal involvement due to vaginal SCC. Sinusoidal infiltration of the liver by malignant cells is rare and can be seen in cases of lymphoma, breast adenocarcinoma and neuroendocrine tumors. Sinusoidal infiltration may lead to portal hypertension that mimics cirrhosis. This case serves as an important reminder that not all cases of portal hypertension are from liver cirrhosis. When a thorough work up is unrevealing, malignant infiltration in the appropriate clinical setting should be considered.



[3133] **Figure 1.** A, Metastatic squamous cell carcinoma involving the sinusoidal and biliary spaces (note arrows) (40×). B, P16 positive metastatic squamous cell carcinoma to the liver identical to the vaginal primary (10×). C, Vaginal biopsy with venous invasion of squamous cell carcinoma (H&E 40×). D, CD31 marking endothelial cells of vein with intraluminal vaginal squamous cell carcinoma (40×).

S3134

Pyruvate Kinase Deficiency and Recurrent Splanchnic Venous Thrombosis: A Case Report

Austin Mueller, BS[1], Syed-Mohammed Jafri, MD[2].
[1]Wayne State University School of Medicine, Detroit, MI; [2]Henry Ford Health System, Detroit, MI.

Introduction: Pyruvate Kinase Deficiency (PKD) is a rare autosomal recessive enzymatic disorder resulting in red blood cell ATP deficiency. The faulty glycolytic pathway commonly manifests as nonspherocytic hemolytic anemia and iron overload. Splenectomy typically increases hemoglobin and may reduce transfusion frequency. However, the risk of venous thrombotic events post-splenectomy is 10% in patients with PKD. Currently, there is little information available on patients with PKD and recurrent thrombosis post-splenectomy.
Case Description/Methods: We present a case of a 25 y.o. male with PKD S/P splenectomy (2001) experiencing RUQ abdominal pain in 2015. MRI revealed iron overload and a thrombus at the junction of the superior mesenteric vein (SMV) and the portal vein. Scleral icterus was present and the liver was non-enlarged with normal contour. Labs showed mildly elevated liver enzymes (AST 39, ALT 71, Total Bilirubin 4.4). Patient was placed on enoxaparin; however, enoxaparin was stopped due to an upper GI bleed. Nine months later, the patient presented with a recurrent SMV thrombus with edematous small and large bowels. Hypercoagulation studies were negative. Patient was given IV pantoprazole and octreotide. IV heparin was initiated but stopped due to acute hematemesis. The patient was bridged to coumadin and checks INR weekly.
Discussion: To our knowledge, only two PKD case reports have detailed recurrent thrombotic events post-splenectomy. Patient A received a splenectomy at age 20 with two episodes of portal vein thrombosis at 6 days and 2 years post-splenectomy. Patient B received a splenectomy at age 1 with two episodes of pulmonary thromboembolism at 29 and 36 years post-splenectomy. This case is unique since our patient experienced two recurrent episodes of venous thrombosis involving the SMV. Our patient received a splenectomy at age 5 with thrombotic events at 13 and 14 years post-splenectomy. Although the etiology remains unclear, chronic anticoagulation and screening for thrombocytosis is essential to improving patient prognosis.

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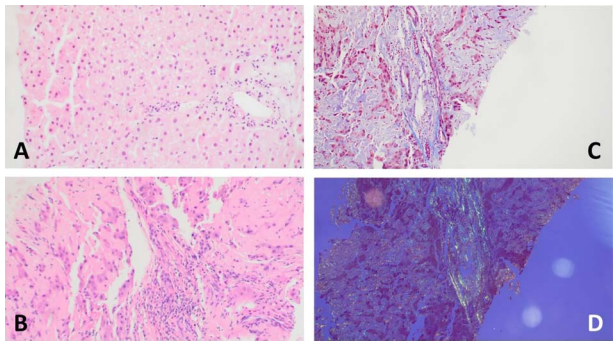
S3135

Primary Hepatic Amyloidosis as a Mimic of the Rapid Development of Decompensated Cirrhosis

Debra Yen, MD[1], Divya Sharma, MD[1], Marshall Weesner, MD[2], Michael Schoech, MD[1].
[1]University of Cincinnati Medical Center, Cincinnati, OH; [2]Cincinnati VA Medical Center, Cincinnati, OH.

Introduction: Primary AL amyloidosis is a systemic disease characterized by the deposition of insoluble fibrils from immunoglobulin light chains. Hepatic involvement is common, and portal hypertension, jaundice, and liver failure have been described. We present a case of primary hepatic amyloidosis that mimicked the rapid development of decompensated cirrhosis.
Case Description/Methods: A 63-year-old male with a history of remotely treated hepatitis C presented with new onset ascites over one month. Laboratory values were notable for AST 201, ALT 64, TBili 1.3, ALP 582, albumin 2.5, platelet count 105, INR 1.5. Paracentesis was performed with 4 liters removed. Serum ascites albumin gradient (SAAG) was >1.1, protein <2.0. Other features of his presentation included acute kidney injury with creatinine 3.1 and nephrotic range proteinuria. Of note, he had a liver biopsy 5 years ago with only portal fibrosis. Serologic evaluation for causes of chronic liver disease was negative. Further testing revealed monoclonal free kappa light chains on urine immunofixation and an elevated kappa/lambda ratio of 8.35. Both liver and kidney biopsies displayed amyloid confirmed by Congo red stain, and he was diagnosed with AL amyloidosis. However, a week later he expired from ventricular fibrillation cardiac arrest. (Figure 1).
Discussion: This case highlights the diagnostic dilemma of a patient without known liver disease who rapidly developed signs of portal hypertension and systemic features of other end-organ involvement (ie, proteinuria, cardiomyopathy), demonstrating the importance of including amyloidosis in the differential. In this case, the patient had a recent liver biopsy that was unremarkable. On presentation, however, he seemed to have progressed to decompensated cirrhosis in a short period of time, with ascites presumed to be from portal hypertension given the elevated SAAG. Ultimately, his entire presentation was attributable to primary hepatic amyloidosis. While clinical manifestations of hepatic amyloidosis are uncommon, with hepatomegaly and elevated alkaline phosphatase as the most frequent findings, sinusoidal portal hypertension may occur and manifest as ascites, splenomegaly, and bleeding esophageal varices. The portal hypertension is thought to be related to the decreased vascular space of hepatic sinusoids from massive perisinusoidal amyloid deposits. Diagnosis involves biopsy of other organs and/or the liver, in which bleeding is a risk. Prognosis is poor for hepatic amyloidosis, with median survival of 9 months.



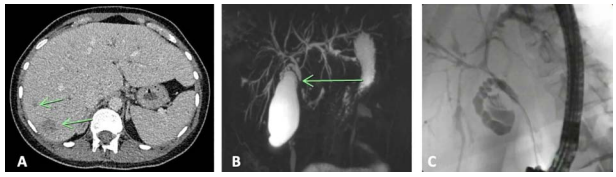
[3135] **Figure 1.** A, H&E stain 20× (2017): Normal hepatic parenchymal architecture. No significant fibrosis, inflammation or amyloid deposition. B, H&E stain 10× (2022): Deposition of abundant amorphous, acellular, eosinophilic amyloid deposits within portal vessels and hepatic sinusoids leading to hepatocyte atrophy. C. Trichrome stain 20× (2022): gray blue amyloid deposits within sinusoidal spaces and within portal tracts. Bright blue collagen deposition is minimal. D, Congo red stain 10× (2022): Apple-green birefringence under polarized light within sinusoids and portal tracts.

S3136

Primary Sclerosing Cholangitis Confused for Von Meyenburg Complex

Dayana Nasr, MD[1], Vishnu Kumar, MD[1], Pujitha Kudaravalli, MBBS[1], Alyssa Ionno, MD[1], Janane Nasr, MD[2], Ganesh Aswath, MD[1].
[1]SUNY Upstate University Hospital, Syracuse, NY; [2]Lebanese American University, Beirut, Beyrouth, Lebanon.

Introduction: Bile duct hamartomas (BHs), also called von Meyenburg complex (VMC) are benign biliary malformations that are caused by disorganization of the small intrahepatic ducts. Patients are usually symptomatic with jaundice being the most predominant symptom. Abdominal pain and fever are also common. Due to similar findings on CT scan, primary sclerosing cholangitis (PSC) can be mistaken for bile duct hamartomas.
Case Description/Methods: A 30-year-old female presented to the hospital for RUQ abdominal pain, fever, nausea, and vomiting. Liver enzymes were elevated in a cholestatic pattern with ALT of 87 U/L, AST of 71 U/L, ALP of 922 U/L, total bilirubin of 2.1 mg/dl and direct bilirubin of 1.2 mg/dl. CT abdomen with contrast showed numerous nodules suspicious of biliary hamartomas and intrahepatic biliary dilation (Figure 1A). MRI abdomen showed cutoff of the proximal common bile duct (CBD) of unknown etiology but possibly due to stricture, extrinsic compression or intraductal lesion (Figure 1B). ERCP was performed and revealed a single localized biliary stricture in the common hepatic duct in addition to segmental dilation and narrowing of the right and left intrahepatic branches (Figure 1C). No biliary hamartomas were seen. In fact, these findings were highly suggestive of primary sclerosing cholangitis despite negative serology. One plastic stent was placed into the CBD. The patient was later discharged on ursodiol 300 mg 3 times daily. Repeat liver enzymes 2 weeks after discharge showed improvement in liver enzymes and patient continued to deny GI symptoms.
Discussion: The prevalence of BHs varies from 1% in children to 5.6% in adults based on autopsy results while that of PSC is 16.2 cases per 100,000 person-years. They can both present with similar symptoms such as jaundice, abdominal pain, and nausea, although pruritis is also common in PSC. There haven't been any reported cases where these 2 entities have been confused prior to this one. However, when comparing CT scan findings one can understand why they might be misread. PSC is characterized by a soft-tissue concentric smooth thickening of the extrahepatic biliary duct resulting in mild segmental and often peripheral intrahepatic biliary ducts. Nodules can also be seen as in our case. BHs on plain CT appear as multiple hypodense lesions with irregular and round shape. To achieve the final diagnosis, visualization of the bile ducts by MRCP and ERCP can be performed. In addition, liver biopsies can be obtained to confirm the diagnosis of BHs.



[3136] **Figure 1.** A, Axial CT abdomen of the liver showing multiple non-enhancing hypodense lesions suspicious for hamartomas (B) MRCP 3D recon images showing a dilated proximal CBD with distal abrupt loss suggestive of obstruction (C) ERCP showing a single localized biliary stricture in the common hepatic duct likely secondary to primary sclerosing cholangitis.

S3137

Post-COVID-19 Cholestatic Hepatitis

Mithil Gowda Suresh, MD[1], Curuchi Anand, MD[1], Aakriti Soni, MD[1], Anuroop Yekula, MD[1], Safia Mohamed, MBBS[2].
[1]St. Vincent Hospital, Worcester, MA; [2]Landmark Medical Center, Woonsocket, RI.

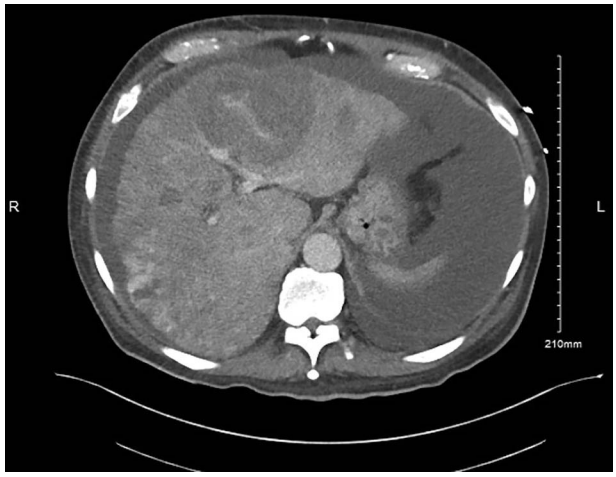
Introduction: Coronavirus Disease 2019 (COVID-19) caused by SARS-COV-2 has led to a global crisis of unprecedented reach and proportion. In addition to the pulmonary consequences, the incidence of liver injury is increasingly being reported. Understanding the hepatotropism of SARS-COV-2 is of paramount importance as it can lead to a severe COVID-19 course. This case details a unique presentation of cholestatic hepatitis likely a sequela of a severe SARS-COV-2 infection in our patient.
Case Description/Methods: A 49-year-old lady with a history of seizure disorder, migraines, hypertension, and recent six-week ICU admission for COVID illness requiring mechanical ventilation (ECMO), presented to our gastroenterology clinic for a post-discharge follow-up. During admission, she consistently had an abnormal elevation of liver enzymes (Peak ALP 2000, AST, ALT 300, Bilirubin 2.5). MRCP was normal and a liver biopsy showed marked intralobular bile duct injury showing severe cytologic atypia, periductal edema, periductal and intraductal neutrophilic, and lymphocytic inflammation and marked ductular reaction, focal mild hepatocellular cholestasis, Kupffer cell hyperplasia. She had no prior history of chronic liver disease and LFT was normal at baseline. Post discharge LFT showed alkaline phosphatase of 1514 with total bilirubin 1.4. Infective (Hepatitis A, B, C panel), Inflammatory/Autoimmune (ANA, ASMA, AMA, P-ANC, Anti-SLA/LP, Anti ds-DNA, ALKM-1, and ALKM-3) workup was negative. Repeat imaging with USG and MRI was unremarkable. She received a course of prednisone after discharge with no improvement in LFT, it was tapered off. She is currently asymptomatic 6 months after acute illness but continues to have persistently elevated liver enzymes with a gradual downtrend with the latest being ALP 490, AST 86, ALT 95, and T. Bilirubin 0.8.
Discussion: We describe a unique case of post-COVID-19 cholestatic hepatitis with persistently elevated liver biochemistry with unclear etiology despite extensive workup. It could be a confluence of Secondary Cholangitis in Critically Ill Patients (SSC-CIP) and direct hepatic injury from COVID-19. Further data is needed to understand the patterns of liver injury due to COVID and the development of possible treatment for this newly described condition associated with the virus.

S3138

Primary Hepatic Angiosarcoma: A Rare Cause of Decompensated Pseudo-Cirrhosis and Acute Liver Failure

Garvit Chhabra, MD[1], Priyanshu Nainm, MBBS[2], Anuja Abhyankar, MD[1], Navroop Nagra, MD[1], Endashaw Omer, MD, MPH[3].
[1]University of Louisville, Louisville, KY; [2]Medical College of Georgia, Augusta, GA; [3]University of Louisville Hospital, Louisville, KY.

Introduction: Primary hepatic angiosarcoma (PHA) is a rare aggressive endothelial cell tumor which is seen in patients in their 60s and 70s with 3:1 male predominance. Industrial exposure to vinyl chloride, radium, chronic arsenic ingestion, anabolic steroid use, and iatrogenic exposure to thorotrast radiocontrast are some of the known etiologies for the development of PHA. Whereas, in most cases, attributable risk factors are rarely identified.
Case Description/Methods: A 68-year-old male patient with NIDDM presented with chest pain and abdominal distension. Examination revealed marked ascites, abdominal tenderness, and bilateral leg edema. Labs were significant for thrombocytopenia and cholestatic pattern of liver enzymes with AST 94, ALT 47, ALP 413, T bilirubin 5.7. Paracentesis was negative for SBP or malignant cytology and was indicative of portal hypertensive ascites. Tumor marker workup was positive for CA 19-9 106.1, whereas negative for CEA 1.82, and AFP 2.7. CT A/P showed multifocal large heterogeneous enhancing lesions in the liver which were biopsied showing diffuse proliferation of abnormal vascular endothelial cells staining positive for CD34 and a diagnosis of PHA was made. With multifocal lesions, the patient was started on paclitaxel chemotherapy, which was eventually stopped with worsening liver function. The hospital course was complicated by hepatic encephalopathy and coagulopathy. The patient and the family later chose to transition to palliative therapy for comfort (Figure 1).
Discussion: PHA is a rare and aggressive tumor with poor outcomes and an average survival rate of less than a year. Early diagnosis is challenging as it presents with nonspecific abdominal symptoms. With progression, PHA can present as decompensated pseudocirrhosis due to compression of liver parenchyma leading to portal hypertension. Contrast-enhanced US and CT can help in diagnosis by showing lesions characteristic of central non-enhancement and peripheral irregular enhancement in the arterial and portal phase, and complete wash-out in the late phase. A definitive diagnosis of PHA is established via histopathological analysis with immunohistochemistry staining but can make diagnosis more challenging due to the potential of associated bleeding. Surgical excision with negative surgical margins is the standard treatment for PHA which is localized and resectable. Multifocal and metastatic PHA is a radio-resistant tumor with a paucity of treatment options, however, TACE and/or salvage chemotherapy can be potentially attempted.



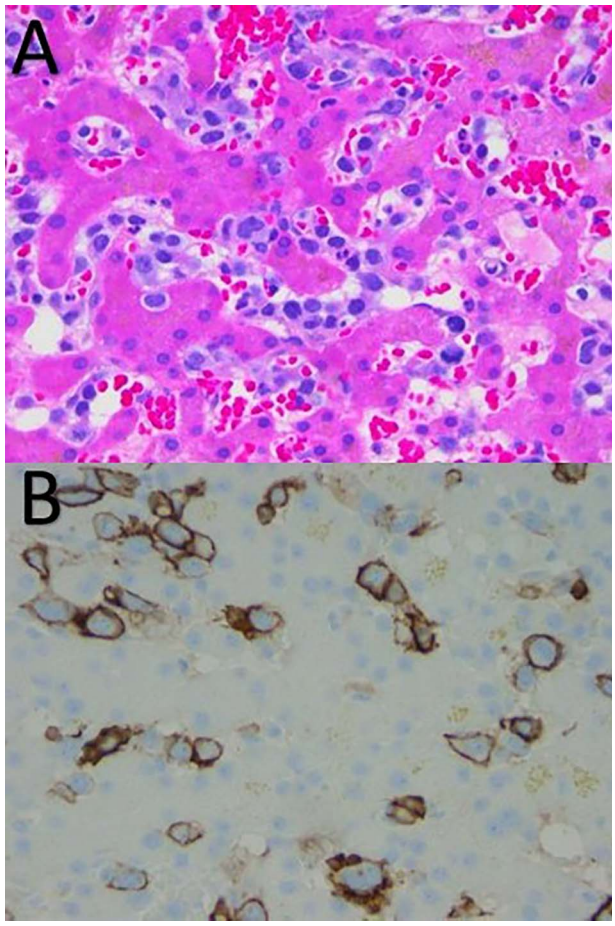
[3138] **Figure 1.** CT Abdomen pelvis showing multiple mass lesions occupying most of liver parenchyma.

S3139

Primary Hepatic Diffuse Large B Cell Lymphoma Presenting as a Picture of Acute Alcoholic Hepatitis

Sreenidhi Chintalapani, MD[1], Bryce Chavez, MD[1], John H. Grider, DO[2].
[1]HCA Healthcare Eastern Idaho Regional Medical Center, Idaho Falls, ID; [2]Eastern Idaho Regional Medical Center, Idaho Falls, ID.

Introduction: Diffuse large B cell lymphoma (DLBCL) is a prevalent subcategory of Non-Hodgkin lymphoma (NHL) comprising around 25% of all NHL occurrences. Generally, 60% of cases will present with Stage III/IV advanced disease vs 40% with localized disease. While DLBCL can arise from any tissue, 40% of cases will come from extranodal extramedullary tissue; most commonly the stomach or GI. Interestingly, primary hepatic lymphoma is exceedingly rare with a reported incidence of 0.4% of all extranodal NHL and 0.016% for all NHL. The presentation can be vague and a mimicker of other disease with symptoms such as abdominal pain, bloating, nausea, vomiting, and B symptoms.
Case Description/Methods: A 59-year-old female with no significant medical history presented for dizziness, fatigue, fever, shortness of breath, weight loss, night sweats, chills × 1 month. She drinks 18 beers/week and is employed at a bar. Initially found to have thrombocytopenia, transaminitis (AST > ALT), hepatosplenomegaly, hypotension, normocytic anemia, latent hepatitis B, and a pulmonary embolism. Her hepatitis was thought to be secondary to alcoholic hepatitis. Her MELD was 12 and Maddrey of -0.6. GI was consulted, further workup showed a past EBV and CMV infection. Quantiferon, HIV, mono, autoimmune and rheumatologic workup were negative. Ultimately a liver biopsy was positive for DLBCL. She was vaccinated for encapsulated organisms, treated for her Hepatitis B, and initiated on R-EPOCH regimen. She was transitioned to RCHOP and ultimately had no evidence of FDG presence on PET Scan and complete response to therapy. She was advised to have routine surveillance per NCCN guidelines (Figure 1).
Discussion: Primary hepatic DLBCL is rare and not often diagnosed when a patient presents with acute hepatitis and known risk factors of alcohol use. However, our patient had multiple B-symptoms concerning for lymphoma. The only way to definitively diagnose DLBCL is by liver biopsy with the prevailing management of EPOCH chemotherapy. Early diagnosis and initiation of treatment is crucial due to its poor prognosis and advanced stage presentation. For this reason, a broad differential diagnosis must be considered when evaluating patients with alcoholic hepatitis. This research was supported (in whole or in part) by HCA Healthcare and/or an HCA Healthcare affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA Healthcare or any of its affiliated entities.



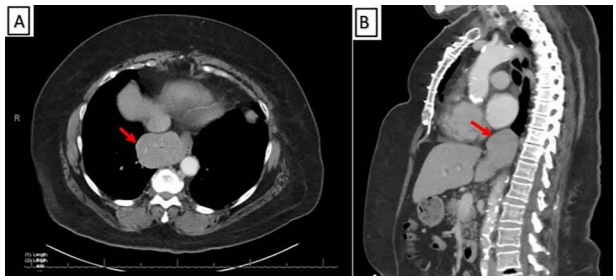
[3139] **Figure 1.** A, Markedly atypical lymphoid infiltrate involving both the hepatic sinusoids and portal tracts. The lymphocytes are large with vesicular chromatin, prominent nucleoli, and fairly abundant mitotic activity B, Immunohistochemical stains are performed. The atypical lymphocytes react with CD20, PAX5, and coexpress BCL-2 (90%) and C-MYC (60%).

S3140

Posterior Mediastinal Mass: Unraveling a Non-Traumatic Herniation of the Caudate Lobe of the Liver

Justine M. Chinnappan, MD, Nageshwari Palanisamy, MD, Ekwevugbe Ogbon, MD, Jami Foreback, MD, Ghassan Bachuwa, MD, MS, MHSA.
Hurley Medical Center, Flint, MI.

Introduction: Posterior mediastinal mass is most likely due to neurogenic tumor, meningocele or thoracic spine lesions. Caudate lobe of the liver herniation presenting as posterior mediastinal mass is a rare occurrence. Diaphragmatic herniation (DH) of the caudate lobe presents in various way including dyspnea, dyspepsia or incidental finding on imaging. We present a case of diaphragmatic hernia of the caudate lobe of the liver presenting as a posterior mediastinal mass found during evaluation of dyspnea.
Case Description/Methods: A 75-year-old female presented to her physician with worsening shortness of breath from her baseline of 3 days duration. She had a history of sarcoidosis, COVID pneumonia over 1 year ago, COPD, diastolic heart failure, and hypertension. She was initially evaluated for COVID re-infection, which was negative and a CT of the chest with contrast to check for sarcoidosis flare revealed posterior mediastinal mass measuring 4.5 × 6.5 × 6.4 cm. Further work up with CT chest and abdomen with contrast revealed that the posterior mediastinal mass had similar attenuation as the liver and appears continuous with the caudate lobe of the liver. This was confirmed by NM scan of liver. Review of her records from an outside organization revealed similar finding on imaging a few years ago. Patient denied any history of trauma and laboratory work up revealed normal liver functions. After pulmonologist evaluation she was started on 2 L home oxygen following six-minute walk test, and also CPAP following a positive sleep study. Pulmonary function tests were performed and inhalers were continued. Given the chronicity of her symptoms and co-morbidities with stable caudate lobe herniation, conservative management was advised with surgery warranted if symptoms persist despite treatment (Figure 1).
Discussion: DH is typically found on the left side with stomach or intestine while the right side is usually guarded by the liver. Isolated herniation of part of the liver into the thoracic cavity is rarely reported and is mostly acute from traumatic or spontaneous rupture requiring immediate repair. Our patient was initially evaluated for the posterior mediastinal mass for concerns of tumor, followed by the finding of what was thought to be acute herniation of the caudate lobe of liver into the thoracic cavity. Review of records showed this to be a stable lesion, we suspect that the patient had congenital diaphragmatic defect. Chronic and stable liver herniation into thoracic cavity can be managed conservatively if uncomplicated.



[3140] **Figure 1.** (A) Computed Tomography of chest with contrast (axial view) showing a well-defined soft tissue density lesion in the posterior mediastinum (red arrow). (B) Computed Tomography of the chest, abdomen and pelvis with contrast (sagittal view) showing well defined soft tissue mass in the posterior mediastinum (red arrow) similar in attenuation to the adjacent liver and appears continuous with the caudate lobe of liver.

S3141

Rare Late Disseminated Histoplasmosis in a Liver Transplant Patient

Aditya Suresh, MD[1], Anuj Chhaparia, MD[2], Roshani Desai, MD[1].
[1]Saint Louis University School of Medicine, St. Louis, MO; [2]Saint Louis University, St. Louis, MO.

Introduction: Histoplasmosis capsulatum is a dimorphic fungus found throughout the world and in the United States is particularly endemic to the Ohio and Mississippi river valleys. It is often found in soil and associated with bat guano and bird droppings. Disseminated histoplasmosis infection is rare, but commonly associated with immunosuppressed states. However, disseminated histoplasmosis infection is particularly rare in patients who underwent orthotopic liver transplantation (OLT) and most commonly occurs within 1 to 2 years post transplantation.
Case Description/Methods: A 63-year-old male with history of OLT (21 years prior to presentation) from hepatitis C virus cirrhosis presents with complaints of fevers, chills, fatigue, & abdominal distention for three months. On presentation he was febrile but hemodynamically stable. Physical exam was notable for abdominal distention and scleral icterus. Laboratory data was notable for elevated alanine aminotransferase (71 U/L), aspartate aminotransaminase (98 U/L), total bilirubin (6.2 mg/dL), and alkaline phosphatase (300 U/L). A magnetic resonance cholangiopancreatography was performed which demonstrated cirrhotic changes in the graft liver and a 4.4 cm right adrenal mass and 3 cm left adrenal mass. A biopsy of the adrenal mass showed fungal forms consistent with histoplasma species, background necrosis, and acute inflammation. A liver biopsy showed granulomatous hepatitis with fungal forms consistent with histoplasma species and advanced bridging fibrosis. Further imaging demonstrated bilateral ground glass opacities in the lungs but no abnormalities elsewhere. The patient was treated with amphotericin B and transitioned to itraconazole with 12 months of therapy planned with surveillance of urine histoplasma antigen levels.
Discussion: In this case, we present a unique case of disseminated histoplasmosis with histoplasmosis hepatitis in a patient who underwent OLT greater than 20 years prior. While any immunosuppressed patient is at a higher risk of disseminated histoplasmosis, it remains relatively rare among patients who have undergone solid organ transplantation. This case highlights the need to remain vigilant against all opportunistic infections in the post-transplant patients at any time post transplantation and particularly those that are endemic to the patient's area of residence.

S3142

Rare Case of Drug-Induced Autoimmune Hepatitis

Saakshi Joshi, MD, Alexandra Davies, DO, Jaspreet Ghumman, DO.
McLaren Macomb, Macomb, MI.

Introduction: Our case represents rare hepatotoxic effects of Norvasq and Lipitior, more specifically Drug Induced Autoimmune Hepatitis with mixed hepatitis and cholestatic pattern and positive autoimmune hepatitis markers.
Case Description/Methods: 65 year old female, with history of hypertension, alcohol use, quit in October 2021 and sigmoid diverticulosis, presented to the office clinic with dark urine, unintentional 10lb weight loss as of 2 months ago, pale colored loose stools and new abnormal lab findings as seen in Figure 1. Patient's abdominal ultrasound showed cholelithiasis and hepatic hemangioma. Patient's home medications included Lipitor, which had a dose increase from 10 mg to 40 mg in August 2021. Additionally, the patient was started on Norvasc 5 mg in August 2021. Both medications were discontinued on 12/10/2021. Patient had resolution of liver enzymes after discontinuation of medications as seen in Figure 1.
Discussion: Drug-induced autoimmune hepatitis can have a wide array of clinical presentations and therefore is included as a differential for abnormal liver biochemical tests, acute hepatitis, cirrhosis, or acute liver failure. Symptoms can range from being asymptomatic to anorexia, fatigue, abdominal pain, itching, and weight loss. Clinical studies have shown adverse hepatic function with statins, representing 0.5 to 3% of patients taking them. More specifically, statins do cause hepatitis in a dose-dependent manner. Rarely, there are reports of severe symptoms associated which include the hepatocellular, cholestatic and autoimmune injury. Additionally, amlodipine's adverse drug reactions do not include hepatic injury; but post-marketing cases were reported showing amlodipine causing cholestatic hepatitis and hepatotoxicity. Similar to our case report, there was a resolution of liver enzymes after discontinuation of amlodipine. Our case is unique as the patient had drug-induced autoimmune hepatitis with a cholestatic pattern. It is important to consider Lipitor's dose increase as a result of hepatitis, cholestatic and autoimmune injury. Additionally, Norvasq causing cholestatic hepatitis and hepatotoxicity. This case demonstrates the rare hepatotoxic effects of amlodipine and Lipitor, further studies are needed to document the incidences.

Table 1.

	12/14/2021	12/23/2021		12/14/2021
AST	699	379	IgG	1885
ALT	657	519	IgM	298
ALP	1079	731	ANA	Positive
Total Bilirubin	7.3	2.1	Anti-Smooth Muscle Antibody	53
Direct Bilirubin	6.04	1.4	Anti-Mitochondrial Antibody	115

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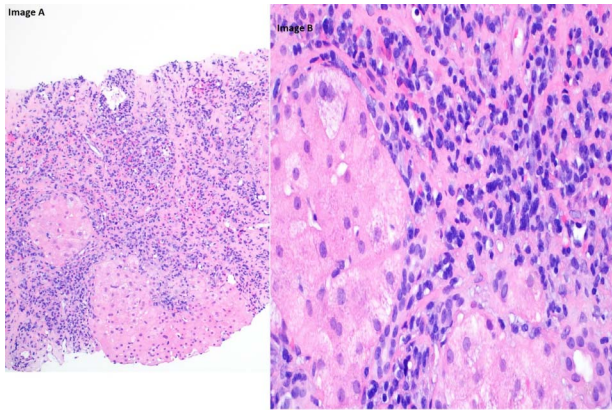
S3143

Rare Case of Plasma Cell Infiltration-Induced Cirrhosis

Zachary Wright, BA, Xiaoliang Wang, MD, PhD, Ahmed Sherif, MD, Phillip R. Jones, DO, Wesam Frandah, MD, Tejas Joshi, MD.
Joan C. Edwards School of Medicine, Marshall University, Huntington, WV.

Introduction: Multiple myeloma (MM) is a type of malignancy that arises from the unorganized replication of plasma cells within the bone marrow. The overproduction of these cells damages systems throughout the body leading to early asymptomatic symptoms that can rapidly progress to systemic signs of disease. Liver involvement with MM is less common and generally occurs through plasma cell infiltration, light chain/amyloid deposition, and biliary obstruction. Though it can occur, the probability of it presenting near the time of diagnosis of MM is very rare and associated with poorer outcomes.
Case Description/Methods: A 59-year-old male presented with generalized weakness, shortness of breath and weight gain. Physical exam showed sign of ascites and hepatomegaly. PMH was significant for esophageal cancer and a recent diagnosis of MM. No history of acute or chronic liver disease or cirrhosis risk factors. CT scan showed cardiomegaly, bilateral pleural effusions, pulmonary edema, and cirrhosis with abdominal ascites. Labs was unremarkable other than normocytic anemia. An echocardiogram showed mild reduced ejection fraction (47%) with speckle tracking suggestive of cardiac amyloidosis. Abdominal ultrasound confirmed appearance of cirrhosis, ascites, and right pleural effusion. Cirrhosis work up revealed negative hepatitis panel, normal alpha-1 antitrypsin, and normal ceruloplasmin. EGD was performed and ruled out extracellular vesicles (EV) but noted type I isolated gastric varices. Liver biopsy confirmed cirrhosis and showed portal tract damage with severe extensive plasma cell infiltration. Additionally, fragments of hepatic parenchymal showed disrupted lobular architecture by septal fibrosis and nodular formation. Immunohistochemistry was positive for CD3, CD20, CD5, BCL2, CD79a, and CD138 as well as increased lambda light chain compared to free kappa light chain. Despite extensive treatment including repeated thoracentesis and paracentesis, patient eventually developed in to decompensating liver cirrhosis (Figure 1).
Discussion: In previous studies, plasma cell infiltration of the liver was seen in about 40% of cases of MM, but rarely lead to cirrhosis. This case is unique in that the patient had liver failure caused by cirrhosis and that it was some of the presenting symptoms. Whether due to masked signs or aggressive onset, the outcome for this diagnosis is poor and more specific factors observing liver function could have provided a more favorable outcome.



[3143] **Figure 1.** Cirrhosis secondary to plasma cell infiltration.

Table 1.

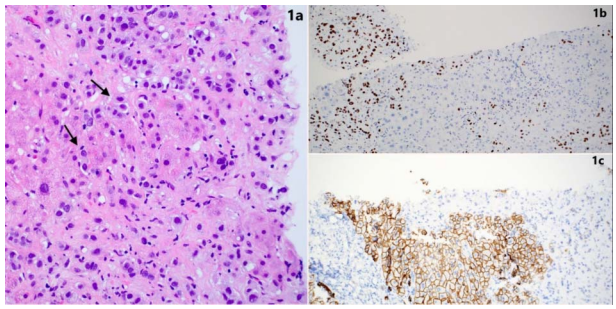
Total Protein	**5.0 g/dL**
Serum Ascites Albumin Gradient (SAAG)	1.9 g/dL
Left Hepatic Vein Pressure	25 Hg
Left Portal Vein Pressure	29 Hg
Portosystemic Pressure Gradient	5 Hg
EUS Guided Shear Wave Elastography of Liver	27 kPa

S3144

Pseudo-Cirrhosis Secondary to Metastatic Breast Carcinoma Causing Sinusoidal Obstruction

Adil S. Mir, MD[1], Diana Dougherty, MD[1], Kristin Newman, PA-C[2], Robert D. Moylan, MD, PhD[1], David Lebel, MD[1], Douglas Grider, MD[1].
[1]Virginia Tech Carilion School of Medicine, Roanoke, VA; [2]Carilion Clinic, Roanoke, VA.

Introduction: Pseudo-cirrhosis Secondary to Metastatic Breast Carcinoma Causing Sinusoidal Obstruction
Case Description/Methods: 70-year-old female with past history of colonic adenocarcinoma, hemicolectomy presented with acute abdominal pain and distension. Labs showed AST 387 IU/L, ALT 167 IU/L, alkaline phosphatase 546 IU/L, total bilirubin 2.5 and direct 1.1 mg/dl (no past history of liver disease, previously normal LFTs), negative acute viral hepatitis panel. Computed tomography (CT) abdomen and pelvis without contrast showed normal liver without focal lesions, mildly enlarged spleen and moderate ascites. Diagnostic paracentesis was negative for spontaneous bacterial peritonitis, with serum-to-ascites albumin gradient (SAAG) >1.1 and total protein 1.2 g/dl. Surgical cytology from the ascitic fluid was negative for metastatic disease. Magnetic resonance imaging (MRI) abdomen: unremarkable liver, gall bladder and biliary system. 2D echocardiogram showed no acute abnormalities. Abdominal veins duplex showed hepato-fugal flow in the main portal vein but normal flow in the hepatic vein and inferior vena cava. A diagnostic liver biopsy was obtained (porto-systemic gradient = 26 mmHg) Histopathology revealed the initial presentation of ductal breast carcinoma metastatic to the liver, poorly differentiated involving portal areas and filing the sinusoids (Figure 1a). The carcinoma was keratin CAM5.2, CK7, GATA3 (Figure 1b), mammaglobin, and BRST-2 positive, confirming metastatic breast carcinoma; the positive e-cadherin (Figure 1c) stain helps confirm ductal phenotype. Negative stains included CDX-2 and villin, helping to exclude a colorectal primary. Napsin-A and TTF-1 were negative, helping to exclude a pulmonary adenocarcinoma. Arginase 1 negative suggesting against a poorly differentiated hepatocellular carcinoma. Masson-trichrome/reticulin stains showed pseudo-cirrhotic pattern with many hepatocytes separated by fibrosis along with infiltration of large numbers of tumor cells. The carcinoma was noted to be Her2/Neu, quantitative estrogen and progesterone receptor staining negative (Triple negative).
Discussion: The possibility of microscopic tumoral sinusoidal obstruction should always be in the clinical and histopathologic differential diagnosis in patients presenting with acute hepatitis and portal hypertensive features, especially ascites.



[3144] **Figure 1.** (a) H&E, 20×, Arrows show carcinoma in the sinusoids causing obstruction (b) GATA3, 10× (c) E-cadherin, 10×.

S3145

Severe Transplantation-Mediated Alloimmune Thrombocytopenia After Liver Transplant Treated Successfully With Efgartigimod

R. Christopher Chase, MD, Andree H. Koop, MD, Denise M. Harnois, DO, Jennifer J. O'Brien, MD, PhD, Nicole M. Loo, MD.
Mayo Clinic, Jacksonville, FL.

Introduction: Transplantation-mediated alloimmune thrombocytopenia (TMAT) is a rare form of passenger lymphocyte syndrome in which lymphocytes from the donor organ produce antibodies against recipient platelets resulting in thrombocytopenia. We describe a novel treatment of TMAT with efgartigimod, a human IgG1 Fc antibody fragment, in a patient with severe, refractory thrombocytopenia following liver transplantation.
Case Description/Methods: A 70-year-old man with cirrhosis secondary to non-alcoholic steatohepatitis complicated by hepatocellular carcinoma underwent deceased donor orthotopic liver transplantation. He required emergent retransplantation on post-operative day 7 for acute hepatic artery thrombosis. Seven days after retransplantation, the patient developed severe thrombocytopenia with platelets $<1 \times 10^9$/L. He responded poorly to platelet transfusion, and two days later developed severe intra-abdominal bleeding requiring exploratory laparotomy. No active bleeding was found, and splenectomy was performed. Testing for heparin induced thrombocytopenia was negative, and the working diagnoses were post transfusion purpura and idiopathic thrombocytopenic purpura. Over the next 8 weeks, the patient had persistent, severe thrombocytopenia despite platelet transfusions and treatment with intravenous immunoglobulin, plasmapheresis, rituximab, romiplostim, and eltrombopag. Notably, two patients who received lung and kidney transplants from the same donor also developed severe thrombocytopenia. Ultimately, testing revealed the presence of serum antibodies to human platelet antigen (HPA) 1a. Genetic testing revealed the genotypes HPA 1a/1b and HPA 1b/1b in the patient and donor respectively, consistent with a mismatch and TMAT. The patient received weekly efgartigimod for 4 doses, and after his third infusion developed sustained improvement in platelets to $>100 \times 10^9$/L without need for transfusion.
Discussion: TMAT is a rare disorder after solid organ transplantation that can lead to severe thrombocytopenia with life threatening bleeding and even death. Although TMAT generally resolves a few months after transplantation as donor lymphocytes are cleared from the circulation, initial management is challenging. Efgartigimod is a novel treatment approved by the food and drug administration for myasthenia gravis that leads to degradation of IgG by binding to the neonatal Fc receptor and should be considered for severe TMAT.

S3146

Severe COVID-19-Associated Acute Liver Failure From Hemophagocytic Lymphohistiocytosis (HLH) and Herpes Simplex Virus-1 (HSV) Hepatitis

Shoma Bommena, MD, Moises Nevah Rubin, MD, Ma Ai Thanda Han, MD.
Banner University Medical Center, University of Arizona College of Medicine, Phoenix, AZ.

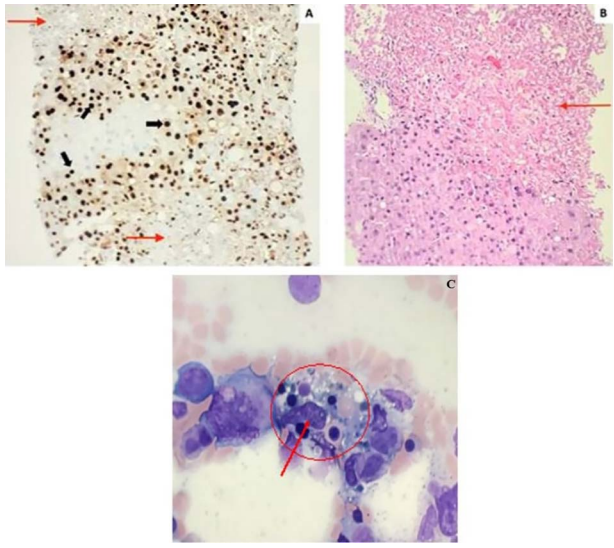
Introduction: Acute liver failure from HSV hepatitis and Hemophagocytic lymphohistiocytosis (HLH), a syndrome characterized by an uncontrolled inflammatory response with cytokine storm, are uncommon associations with COVID-19 infection. We report an unusual case of acute HSV-hepatitis and HLH in an otherwise healthy male with a severe COVID-19 infection.
Case Description/Methods: A 39-year-old male without significant medical history presented to the ER with progressive dyspnea after testing positive for COVID-19 a week before and was admitted for acute hypoxic respiratory failure. He was treated with Dexamethasone, Remdesivir, and Baricitinib. Liver enzyme elevation of Aspartate transaminase (AST) 57 and Alanine transaminase (ALT) 35 at the time of admission, was attributed to COVID-19. Thirteen days later, enzymes increased to AST 2457 and ALT 3119, with normal Total Bilirubin (TB) and INR. N-acetyl Cysteine was initiated, and Baricitinib was discontinued. He was transferred to a liver transplant center for impending liver failure. Serologies for HAV, HBV, HCV and the autoimmune panel were negative. Ultrasound abdomen did not show acute abnormalities. HLH was suspected based on hypofibrinogenemia, fasting hypertriglyceridemia, cytopenias, elevated ferritin (>100,000 ng/ml), and Interleukin-2 (Table 1). The diagnosis was confirmed with a Bone marrow biopsy showing hemophagocytosis. A diagnosis of primary HSV-1 hepatitis was made based on positive serum HSV-1 DNA PCR and histologic evidence of sub-massive hemorrhagic necrosis of the liver with diffuse nuclear positivity for HSV-1 in the hepatocytes (Figure 1). Anakinra and dexamethasone for HLH treatment and Acyclovir for HSV hepatitis were administered. He progressed to acute fulminant liver failure with shock and multiorgan failure and died three days after the confirmed diagnosis and treatment initiation.
Discussion: Elevated transaminases are commonly seen in patients with COVID 19, and clinicians often attribute them to COVID 19 infection or drug-induced liver injury. Occurrence of primary HSV infection or reactivation could happen in severe COVID 19 infection. Similarly, HLH could be part of the spectrum of Immune-mediated complications in patients with severe COVID-19. Our case has both HSV hepatitis and HLH leading to acute fulminant liver failure, which is detrimental. Clinicians should be aware of these conditions and consider them as differential diagnoses for elevated transaminases seen in COVID-19.

Table 1. Serum laboratory values

Laboratory parameters	Day of admission	Hospitalization day 13	Hospitalization day 21
Complete blood picture			
WBC	7.4 K/uL	6.9 K/uL	4.1 K/uL
Hemoglobin	15.3 g/dL	17.3 g/dL	7.2 g/dL
Platelets	163 K/uL	112 K/uL	64 K/uL
Prothrombin time (PT)	12.0 seconds	14.2 seconds	33.6 seconds
INR	1.0	1.2	2.9
Complete metabolic panel			
Sodium	139 mmol/L	123 mmol/L	133 mmol/L
Potassium	3.7 mmol/L	4.4 mmol/L	4.7 mmol/L
Chloride	100 mmol/L	86 mmol/L	97 mmol/L
C02	26 mmol/L	25 mmol/L	14 mmol/L
Anion Gap	13	12	22

Table 1. (*continued*)

Laboratory parameters	Day of admission	Hospitalization day 13	Hospitalization day 21
BUN	23	24	11
Creatinine	0.94 mg/dL	1.14 mg/dL	2.17 mg/dL
Albumin	4.1 g/dL	3.3 g/dL	2.8 g/dL
Bilirubin Total	0.5 mg/dL	0.7 mg/dL	7.1 mg/dL
AST	57 U/L	2457 U/L	15750 U/L
ALT	35 U/L	3119 U/L	9150 U/L
Alkaline Phosphatase	65 U/L	98 U/L	339 U/L
Others			
Ferritin			>100000 ng/mL
Triglycerides			317 mg/dL
Interleukin-2 (IL-2)			6461 mg/mL
Fibrinogen			41 mg/dL
EBV DNA, QN PCR (copies/ml)			18816 copies/ml
HSV 1 DNA, real-time PCR			Detected



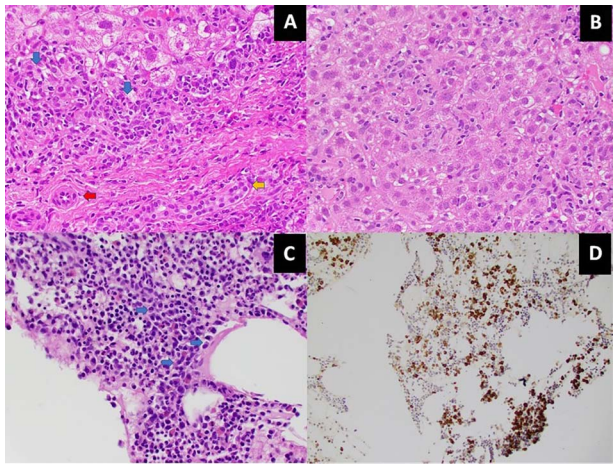
[3146] **Figure 1.** (Panel A) Liver biopsy with HSV immunohistochemical stain. Nearly all hepatocytes are infected by the HSV virus, as demonstrated by dark brown nuclear staining (black arrow). Red arrows indicate large areas of necrotic liver tissue. (Panel B) Hematoxylin and eosin stain showing large areas of liver necrosis (red arrow) and viral cytopathic effect in remaining viable hepatocytes. (Panel C) Bone marrow aspirate smear demonstrating an HLH histiocyte (outlined in red, nucleus indicated by red arrow) engulfing mature and precursor red blood cells and platelets.

S3147

Remission of Autoimmune Hepatitis Following Autologous Stem Cell Transplantation

Zarian Prenatt, DO, Brittney Shupp, DO, Lisa Stoll, MD, MPH, Hammad Liaquat, MD, Berhanu Geme, MD.
St. Luke's University Health Network, Bethlehem, PA.

Introduction: Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease that typically requires lifelong immunosuppression with medications like corticosteroids and azathioprine to prevent fibrosis. We present a patient with type 1 autoimmune hepatitis who achieved biochemical remission following autologous stem cell (SC) transplantation for IgA multiple myeloma.
Case Description/Methods: A 67-year-old female with a history of GERD, hypertension, type 2 diabetes, hypothyroidism, and CKD 3 presented in February 2016 with abdominal pain and jaundice. Labs were notable for an AST 1198 U/L, ALT 950 U/L, alkaline phosphatase 244 U/L and total bilirubin 8.09 mg/dL. A hepatitis panel was negative and drug-induced liver injury was ruled out. However, anti-nuclear antibody was found to be positive and anti-smooth muscle antibody IgG was elevated at 191 U. The diagnosis of type 1 AIH was later confirmed by liver biopsy (Image A and B), which revealed chronic active hepatitis with portal lymphoplasmacytic inflammation and stage 2 fibrosis according to the Ludwig-Batts classification. She was started on prednisone and azathioprine. Steroids were able to be withdrawn, but she remained on azathioprine and Ursodiol for multiple years due to recurrent exacerbations. In October 2018, the patient was diagnosed with multiple myeloma confirmed on bone marrow biopsy (Image C and D), which demonstrated IgA kappa restricted plasma cell myeloma. Prednisone, Ursodiol, and azathioprine were all discontinued, and the patient then underwent an autologous SC transplantation in May 2019. She has since been continued on maintenance ixazomib and low dose dexamethasone once weekly but has been monitored off her previous AIH therapies for over 2 years. Now two years following SC transplantation, she remains in biochemical remission and has achieved normalization of her liver enzymes. She currently has her LFT's monitored every 6 months.
Discussion: Treatment options for AIH remain limited to immunosuppressant medications like corticosteroids and azathioprine, which can lead to substantial adverse side effects. Although limited data exists and additional clinical investigations are required, many have postulated the effects of SC transplantation in patients with AIH given their promising results in animal studies. Therefore, our case encourages further studies investigating the use of stem cells as an alternative treatment for AIH or as a synergistic therapy in patients whose AIH remains uncontrolled.



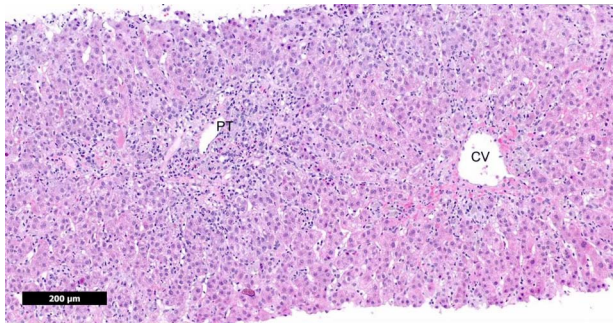
[3147] **Figure 1.** (A) H&E stain of the liver showing portal tract with artery (red arrow), bile duct (yellow arrow) and interface hepatitis (blue arrow) comprised of numerous plasma cells and scattered lymphocytes. (B) H&E stain of the liver showing lobular inflammation consisting of lymphocytes. (C) H&E stain of the bone marrow showing cellular marrow with numerous plasma cells (blue arrows). (D) Bone marrow, Kappa in situ hybridization reveals Kappa-light chain restricted plasma cell neoplasm.

S3148

Seronegative Autoimmune Hepatitis: A Rare Manifestation of COVID-19

Hwe Won E. Lee, DO, Julia Zhang, MD, Ronak Gandhi, DO, Alyeesha B. Wilhelm, MD, Sheharyar K. Merwat, MBBS, Heather L. Stevenson, MD, PhD, Julieanna Camarena, DO.
University of Texas Medical Branch, Galveston, TX.

Introduction: Hepatic dysfunction is seen in 14-53% of patients with COVID-19, particularly in those with significant comorbidities. There have been reports of the development of autoimmune hepatitis (AIH) following COVID-19 vaccination and/or infection. AIH is usually associated with circulating autoantibodies but 10-20% of AIH patients are initially seronegative. We present the first reported episode to our knowledge of a case of seronegative AIH in the setting of concurrent COVID-19 infection.
Case Description/Methods: A 39-year-old otherwise healthy female with a family history of mother with rheumatoid arthritis presented with 2 weeks of nausea, diarrhea, abdominal pain, and jaundice. She had significant transaminitis in the 1000 s and a conjugated hyperbilirubinemia. Despite previous vaccination and no respiratory symptoms, the patient tested positive for COVID-19. The patient denied taking hepatotoxic agents, and the workup was negative for acute viral hepatitis. The antinuclear, alpha-1-antitrypsin, ceruloplasmin, IgG, anti-smooth muscle, anti-mitochondrial, anti-neutrophil cytoplasmic, and anti-liver/kidney microsomal-1 antibodies were all unremarkable. Imaging was only remarkable for hepatic hemangiomas. Liver biopsy was ultimately performed showing active lymphoplasmacytic hepatitis with prominent regenerative changes and areas of confluent necrosis, consistent with autoimmune hepatitis. The patient's symptoms and transaminitis improved following initiation of steroids (Figure 1).
Discussion: We present a unique case of AIH as a manifestation of COVID-19 infection despite negative serology. Hepatic injury from COVID-19 is thought to be mediated by multitude of mechanisms including upregulation of ACE2 and DPP4 receptors, which are involved in RAS signaling pathways and regulation of the immune system. Further research is needed to better understand if there is a causal link between COVID-19 and hepatic autoimmune dysfunction and the underlying molecular mechanisms. Identifying autoimmune dysfunction following COVID-19 infection may allow us to recognize and treat these cases early on, leading to better hospitalization outcomes.



[3148] **Figure 1.** Liver biopsy showing marked interface activity and areas of confluent necrosis consistent with autoimmune hepatitis (AIH). A representative portal tract (PT) and central vein (CV) are illustrated.

S3149

Resolution of Autoimmune Hepatitis With Concurrent Ulcerative Colitis After Colectomy: A Case Report

Faris Hammad, MD[1], Abdul Rahman Al Armashi, MD[2], Anas Al Zubaidi, MD[3], Mohamed Homeida, MD[1], Apoorva Krishna Chandar, MD[2], Akram Alkrekshi, MD[4].
[1]St. Vincent Charity Medical Center, Cleveland, OH; [2]University Hospitals Case Western Reserve University, Cleveland, OH; [3]Johns Hopkins University, Baltimore, MD; [4]MetroHealth Medical Center/Case Western Reserve University, Cleveland, OH.

Introduction: UC is an idiopathic inflammatory disease results in diffuse inflammation and ulcers of the colonic mucosa. Hepatobiliary diseases are extraintestinal manifestations of UC. PSC is the most common condition; it shares a similar pathogenesis with IBD. However, some patients may develop AIH/overlapping syndrome, that may result from the chronic inflammation, alteration of the intestinal microbiota and mucosal barrier disruption resulting in toxins and bacterial translocation into the portal circulation. We aim to spot the light on a patient with severe UC complicated with AIH that resolved after colectomy.
Case Description/Methods: A 37-year-old Caucasian female was diagnosed with UC at age 29, she had severe UC, colonoscopy showed pancolitis with extensive mucosal ulcerations proximal to the splenic flexure. LFTs have been persistently high during follow up with elevation of AST and ALT 7-8 times the upper limit of normal. Serology showed positive ANA, ASMA, and elevated IgG. Liver biopsy showed portal mononuclear cell infiltrates, nondestructive cholangitis, and mild fibrosis consistent with AIH. Due to persistent symptoms despite medical therapy, and long-standing disease, patient had a proctocolectomy with one stage ileal pouch-anal anastomosis. During a follow up period of 24 months after surgery, LFT has consistently trended down to normal, and the titer of ANA dropped to $<$ 1:80.

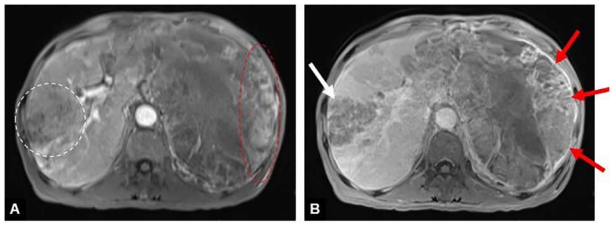
Discussion: Concurrent UC and AIH isn't commonly seen in practice. These patients have more tendency to develop the disease at younger age, severe disease, pancolitis, resistant to medical therapy, and higher risk of death or liver transplantation. Different pathogenesis behind this phenomenon have been suggested by researchers based on human or mice studies. Chronic inflammatory reaction, autoimmunity activation, and elevation of inflammatory markers could have resulted from the alteration of intestinal microbiota, disruption of the tight junctions proteins that increase the permeability of the colonic mucosa, resulting in toxins and bacterial translocation by the level of lipopolysaccharide to find their way to the liver through the portal circulation. Human studies showed that increased intestinal permeability correlated with the severity of the AIH. In our patient, we hypothesize that severity of the AIH has significantly improved after the patient had a colectomy in which the source of toxins has been eliminated. However, larger retrospective studies using the databases or prospective trials are suggested.

S3150

Refractory Hypoglycemia From Hepatocellular Carcinoma: How Low Can You Go?

Jaclyn E. Kagihara, MD[1], Giancarlo Colon Rosa, MD[2], Sunny Rhee, MD[3], Jessica Davis, MD, MSCR[4].
[1]George Washington University, Washington, DC; [2]George Washington University Hospital, Washington, DC; [3]Vancouver Clinic, Vancouver, WA; [4]Washington DC VA Medical Center, Washington, DC.

Introduction: Refractory hypoglycemia is a rare paraneoplastic manifestation of hepatocellular carcinoma (HCC) with poor prognosis. We present a patient with hypoglycemia as initial presentation of HCC.
Case Description/Methods: A 68-year-old male with decompensated Child-Pugh class B hepatitis C cirrhosis with sustained virologic response initially presented to an outside facility for syncope. He was diagnosed with 22 centimeter multifocal metastatic HCC to bone and lung (Figure 1). He suffered hypoglycemia-induced seizures requiring debulking therapy with transarterial bland embolization (TAE) and dexamethasone. Two months later he was hospitalized at our facility for altered mental status and found to have blood glucose (BG) 12 milligrams per deciliter. He received 50 grams intravenous dextrose and intramuscular glucagon with improvement but continued to have recurrent hypoglycemia despite oral intake. Laboratory testing was consistent with HCC-related hypoglycemia (Table 1). Endocrinology recommended dextrose 10% infusion, corticosteroids, and frequent meals. His BG remained labile with frequent morning hypoglycemia. Previous TAE limited further options for locoregional therapy (LRT). Sorafenib was considered for palliation, but the patient opted for comfort-directed care and died one month after admission.
Discussion: Two types of hypoglycemia are seen in HCC patients. Type A hypoglycemia is mild, occurs in rapidly-growing tumors, and mortality may occur within weeks. It is caused by the inability of a tumor-ridden liver to meet the body's glucose demand. Type B hypoglycemia is severe, occurs with slowly-growing tumors, and mortality may occur within a year. It is caused by defective processing of the insulin-like growth factor (IGF)-2 precursor by tumor cells, resulting in increased glucose uptake. Our patient's persistent and profound hypoglycemia made type B most likely, and his low insulin, c-peptide, IGF-1, normal IGF-2 and high IGF-2/IGF-1 ratio confirmed non-islet cell tumor hypoglycemia. Current literature suggests the most effective management of HCC-induced hypoglycemia is cytoreduction by surgery, chemotherapy and LRT. There is little data on effective pharmaceutical treatments. Steroids, frequent feeding, dextrose infusion, and growth hormone have been attempted with mixed results. Patients with cirrhosis and refractory hypoglycemia should be screened for HCC as this may be a presenting symptom as in our case. Tumor reduction appears the most durable method for hypoglycemia management.



[3150] **Figure 1.** MRI of the abdomen shows large hepatic mass with mosaic architecture replacing the left lobe. Foci of (A) arterial phase hyperenhancement (APHE) along its left periphery (red dotted oval) demonstrates corresponding washout in (B) delayed phase (red arrows). Smaller right hepatic mass also demonstrates APHE (white dotted circle) and washout (white arrow).

Table 1. Laboratory Data

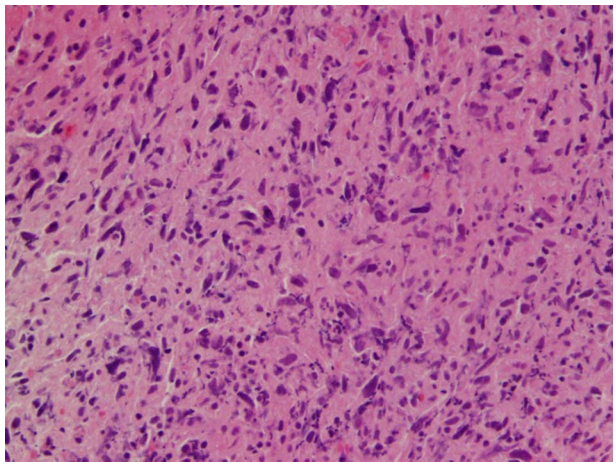
Serologies	Patient Values	Reference Range
Blood glucose (mg/dL)	12	70-121
C-peptide (ng/mL)	0.26	0.80-3.85
Insulin (μIU/mL)	< 0.1	2.0-19.6
Insulin-like growth factor 1 (IGF-1) (ng/mL)	12	41-279
Insulin-like growth factor 2 (IGF-2) (ng/mL)	392	267-616
IGF-2/IGF-1 ratio	32.7	< 10

S3151

Sarcomatoid Hepatocellular Carcinoma: A Dangerous, Spindled Subtype of Hepatocellular Carcinoma

Pranavteja Gutta, MD, Siva Santosh Kumar Gandu, MD.
Ochsner LSU Health Shreveport, Shreveport, LA.

Introduction: Spindle cell hepatocellular carcinoma (SpHCC) and sarcomatoid hepatocellular carcinoma (SHC) are rare and unique variants of the more common hepatocellular carcinoma (HCC). SpHCC and SHC rarely have been reported despite HCC being a leading cause of liver cancer. SHC is an aggressive, rapidly growing tumor with a highly unfavorable prognosis.
Case Description/Methods: A 50-year-old male with a past medical history of hypertension presented to the emergency department complaining of abdominal pain and nausea that started six weeks prior to admission. The patient additionally reported approximately ten pounds of weight loss over the past two weeks. A triple phase computer tomography scan showed a hepatic mass with numerous multiloculated, multiseptated, bi-lobar measuring up to 13 × 11 centimeters in size with compression/invasion of vasculature and necrotic lymph nodes. A fine needle aspiration and core liver biopsy was performed that showed pathology suggestive of spindle cell carcinoma/carcinosarcoma (Figure 1). The patient's case was discussed at tumor board where it was decided he was not a surgical candidate due to the elevated risk of bleeding. Carboplatin/gemcitabine regimen started with re-staging planned.
Discussion: The SHC subtype of HCCs is rarely diagnosed (0.79%) but most cases are found on autopsy studies (14.3%). The challenge in diagnosing these rare subtypes of HCC arises due to lab values of alpha-fetoprotein, bilirubin, and liver function tests being lower than seen in traditional HCC. As seen in our case study, at the time of diagnosis of SHC and SpHCC many of the cancers are found to have vascular invasion and invasion of nearby organs. When compared to HCC, SHC patients have larger tumors, frequent necrosis, invasion of adjacent organs, and more advanced tumors with higher incidence of lymph node involvement. Treatment modalities have not been established and no consensus has been formed about SHC. Patients with this aggressive subtype of HCC have lower survival rates and lower response to chemotherapy. Some studies have shown that SHC has the lowest survival rate among all subtypes of HCC with median survival of four to eight months. Unfortunately, the poor prognosis, adjacent invasion, and poor survival times despite chemotherapy show the need for better understanding of this subtype of HCC. A collaborative effort among different centers would allow for a better understanding and establish a more effective therapy for this extremely aggressive cancer.



[3151] **Figure 1.** Patient's pathology showing spindle cells.

S3152

Resolution of Cholea Movement Secondary to Non-Wilsonian Hepatolenticular Degeneration (NWHD) Post Orthotopic Liver Transplantation (OLT)

Kaveh Zivari, MD[1], Pearl Tan, MD[2], Saikiran Kilaru, MD[3], James Park, MD[1].
[1]Maimonides Medical Center, Brooklyn, NY; [2]New York University, Manhatan, NY; [3]New York University Langone Medical Center, New York, NY.

Introduction: Non-Wilsonian hepatolenticular degeneration (NWHD) is a heterogeneous neurological disorder occurring secondary to chronic acquired liver disease. Genetically determined familial NWHD is rare, poorly understood, and often mistaken for Wilson's disease (WD). We present a case of a 65-year-old woman with a family history of NWHD, found to have dystonia, parkinsonism, tremor, cerebellar ataxia, progression of behavioral abnormalities who presented with cognitive decline and progression of liver failure. The patient was evaluated for and later underwent Orthotopic liver transplant (OLT)
Case Description/Methods: 65-year-old woman with family history of early-onset cirrhosis with dystonia and dyskinesia in her father, sister, and daughter is transferred to our institution after she was noted to have accelerated progression of her neurological decline which started the year prior. The patient was not obese (BMI 27) and did not use any alcohol. Huntington's disease workup was negative. Workup for causes of cirrhosis did not yield any findings including multiple 24-hour urine copper collections, and no finding of Kayser Fleischer rings on ophthalmology exam. Multiple CT and MRI brain showed linear abnormal signal foci noted along a medial portion of the bilateral lentiform nucleus in anterior to posterior orientation. The patient was diagnosed with NWHD and underwent OLT. Pathology on explanted native liver showed focal steatohepatitis, Mallory-Denk bodies, and focal mixed inflammatory infiltrates. The copper stain was negative. Post liver transplant our patient's dystonia, parkinsonism, tremor, cerebellar ataxia, and behavioral abnormalities all resolved.
Discussion: Degeneration of basal ganglia leads to movement neurological disorders. There is an association between basal ganglia-related neurological disorders and cirrhosis of the liver in the absence of acquired liver disease such as Wilson's disease. NWHD is a distinct disease entity. Specific areas of the brain, such as the basal ganglia, are more likely to be injured from liver failure. The basal ganglia is involved in control of movement. If damage to this area is not from copper, this condition is the "non-Wilsonian" type. Non-Wilsonian hepatolenticular degeneration may represent a disorder of other poorly known toxic depositions. We demonstrate that with liver transplantation this damage can be reversible.

S3153

Secondary HLH: Why Didn't I Think of That First?

Jessica Corder, MD, Daniel Shields, DO, Brett Sadowski, MD.
Naval Medical Center Portsmouth, Portsmouth, VA.

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a rare disease hallmarked by widespread immune activation ultimately leading to multi-organ failure. HLH occurs in two subsets: primary, typically with onset in childhood, and secondary, that occurs in response to another disease process. Given its rarity and nonspecific protean manifestations, the diagnosis of secondary HLH hinges heavily on excluding other more common diagnoses, a high index of suspicion, and interpretation of lab data. Our case highlights the workup and therapeutic dilemma in diagnosing HLH in a previously healthy 52-year-old male.
Case Description/Methods: A 52-year-old healthy male presented to the emergency department with the concern of leg swelling and vague abdominal pain. Initial lab work up was significant for WBC 1.56, Hgb 9.2, platelets 94, conjugated hyperbilirubinemia, AST 154 and ALT 99. CT abdomen/pelvis showed hepatosplenomegaly, gallbladder wall thickening, and intrahepatic ductal dilation. Initial concerns included cholangitis which prompted broad spectrum antibiotics and ERCP with stent placement, though no stones, sludge, or purulent discharge were found. MRCP noted splenomegaly and dilation of the main portal vein suspicious for cirrhosis. Given the confusing clinical picture and doubt that cirrhosis was the unifying diagnosis, a liver biopsy was performed which showed significant hemophagocytosis. Given this finding, in addition to a ferritin >35,000 and IL2 soluble receptor of 16,000, a diagnosis of secondary HLH was made. Steroid treatment was deferred due to a possible undiagnosed smoldering infection and concern for possible relapse with induction vs obscuring the diagnosis of some underlying hematologic malignancy. He was transferred to a quaternary institution where he succumbed to his profound illness a few days post transfer.
Discussion: This case demonstrates the importance of keeping secondary HLH on the differential in the setting of liver injury. Cholangitis, cirrhosis, and acute liver failure were all considered given the lab and imaging findings, however the inability of the patient to improve after interventions created the need to explore alternative diagnosis. This prompted the consideration and ultimately led to the diagnosis of secondary HLH. The mainstay treatment of HLH involves treating the underlying causes, but also blocking the dysregulation of the immune system. As most protocols for HLH call for high dose steroids, caution was taken in this case given the concerns for an infection.

S3154

Severe Noncirrhotic Hyperammonemia: What Urea-lly Should Consider

Samantha Wu, MD, Carlo S. Basilio, MD, MPH.
Tulane University School of Medicine, New Orleans, LA.

Introduction: Hyperammonemia is often the result of liver pathology. When hyperammonemia is not the result of liver disease, an occult disorder of metabolism must be on the differential for unexplained hyperammonemia, such as a urea cycle disorder (UCD).
Case Description/Methods: A 57-year-old man with heart failure with recovered ejection fraction, atrial fibrillation, hypertension, and cervical neck fracture who had undergone a recent cervical spine corpectomy and fusion presented with worsening dysphagia and was found to have a cervical fluid collection for which he received a dexamethasone taper and a lumbar drain. He had no known underlying liver disease and denied significant alcohol history. He was later admitted to the intensive care unit (ICU) for unstable atrial fibrillation with rapid ventricular response (RVR). In the ICU, he became more somnolent and was unable to follow commands. Encephalopathy workup was significant for elevated BUN with normal creatinine, ammonia 772, and mildly elevated transaminases. His labs and imaging did not demonstrate cirrhosis or acute liver failure. Workup for inborn errors of metabolism showed mildly decreased citrulline. Urine and plasma amino acids were otherwise normal. His hyperammonemia resolved and his mentation improved with lactulose and rifaximin. UCD gene panel and further genetic workup is ongoing and to be continued outpatient.

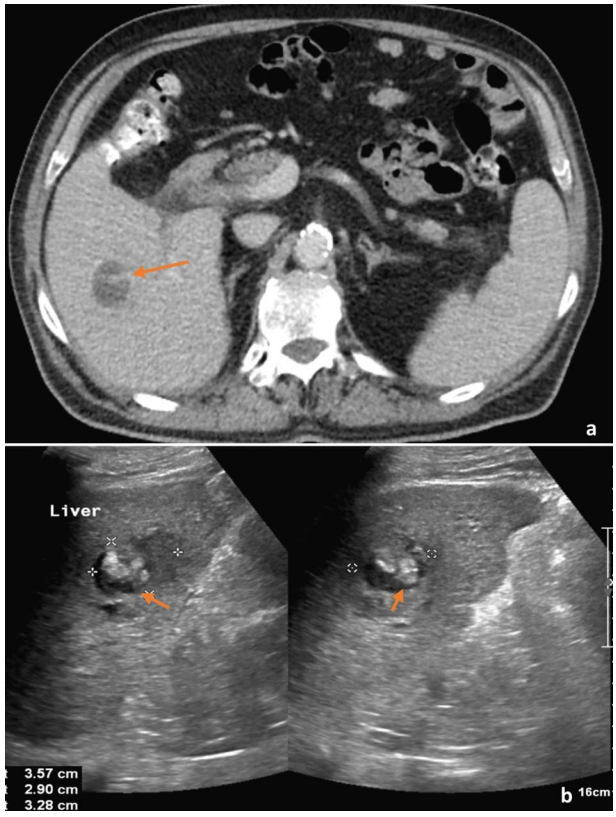
Discussion: Patients with elevations of ammonia present with encephalopathy, which may progress quickly to cerebral herniation. Survival requires immediate reduction of ammonia levels. Although the differential for hyperammonemia is broad, inborn errors of metabolism (IEM), like a UCD, should be considered when hyperammonemia is of unclear etiology. This patient's liver workup revealed no findings consistent with cirrhosis or liver failure. Potential causes for his hyperammonemia include ischemic liver disease secondary to atrial fibrillation with RVR or degradation of blood products. IEMs may also be unmasked by steroid therapy, which is plausible in this patient who was on a steroid taper prior to his hyperammonemic state. Although IEMs often have early age of onset, UCDs have multiple modes of inheritance and can present at later stage. Ornithine transcarbamylase deficiency is the most common UCD and can present with reduced plasma citrulline in both kids and adults. Treatment for a potential IEM begins prior to confirmation of an etiology. Geneticists should be consulted early on for evaluation and management.

S3155

Recurrent Liver Abscess in a Non-Toxic Patient

Ayham Khrais, DO[1], Ahmad Ali, MD[2], Catherine Choi, MD[2], Siddharth Verma, DO[2].
[1]Rutgers New Jersey Medical School, West Orange, NJ; [2]Rutgers New Jersey Medical School, Newark, NJ.

Introduction: Liver abscesses are caused by direct spread from peritonitis, biliary tract infection or via hematogenous seeding from a distant source. Most are polymicrobial, however Escherichia coli and Klebsiella pneumoniae are the most common offending pathogens. Patients usually present with pain, fever, and clinical signs of infection. We describe a case of spontaneous liver abscess in a non-toxic patient that recurred 10 years after a previous abscess.
Case Description/Methods: A 73-year-old-man with a history of type 2 diabetes mellitus, hypertension, CAD status post CABG and PCI 3 years ago, and abdominal aortic aneurysm status post endovascular aneurysm repair presented with 2 weeks of dark urine. After receiving his COVID-19 booster and influenza vaccinations, he developed flu-like symptoms with a self-resolving fever of 101.8°F. He had dark amber urine without dysuria or hematuria. Later, he experienced generalized weakness and decreased oral intake. Outpatient labs showed elevated liver function tests, and he was told to present to the ED. On arrival, he was afebrile with stable vitals. Physical exam was unremarkable. Laboratory evaluation showed a hemoglobin of 11.7 g/dL, sodium of 133 mEq/L, creatinine of 1.4 mg/dL, aspartate aminotransferase of 117 U/L, alanine aminotransferase of 212 U/L, alkaline phosphatase of 825 U/L, total bilirubin of 4.1 mg/dL, and direct bilirubin of 2.1 mg/dL. Triple-phase CT showed a 2.8 cm mass in the right liver lobe with linear enhancement. Ultrasound showed mixed echogenicity measuring 3.6 × 2.9 × 3.3 cm in segment 8 of the liver. On further evaluation, patient had an *E. coli* abscess diagnosed 10 years prior, managed with antibiotics and drainage. At that time, the abscess was within the right inferior liver lobe, similar to his current abscess. LFTs downtrended. Abscess was aspirated, with culture growing oxidase negative, gram-negative rods, likely *E. coli*. Patient started on ceftriaxone and metronidazole, to undergo colonoscopy as an outpatient and rule out colonic bacterial translocation.
Discussion: Pyogenic liver abscess can result in significant morbidity and mortality because of worsening infection and sepsis. Abscesses occur because of spread from adjacent infection or after recent surgeries. Recurrence is very rare. Here, we describe a very unusual case of a pyogenic liver abscess growing *E. coli* in a non-toxic patient, with the same location and causative organism as an abscess managed 10 years prior.



[3155] **Figure 1.** Triple phase CT scan of the abdomen and pelvis in the transverse plane (a) demonstrating a 2.8 cm mass (arrow) in the right lobe of the liver with a small area of linear enhancement. Abdominal ultrasound demonstrating an area of mixed echogenicity (arrows) with areas of increased and decreased sonographic texture measuring 3.6 × 2.9 × 3.3 cm in segment 8 of the liver (b). CT, computed tomography.

S3156 WITHDRAWN

S3157

Respiratory Syncytial Virus-Associated Hepatitis in Pregnancy

Alexander Malik, MD[1], Muhammad Nadeem Yousaf, MD[2], John Abdelnour, MD[3], Sami Samiullah, MD[1], Tahan Veysel, MD[2], Amin Mahdi, MD[1], Omer Basar, MD[2].
[1]Summa Health System, Akron, OH; [2]University of Missouri, Columbia, MO; [3]Metro Health System, Avon Lake, OH.

Introduction: Respiratory syncytial virus (RSV) predominantly affects children and typically manifests as an upper respiratory tract infection. Primary RSV infection in immunosuppressed adults may increase risks of disseminated infection manifesting as RSV hepatitis. RSV hepatitis may present with fever, abdominal pain, nausea, vomiting, jaundice, coagulopathy, and elevation of transaminases.
Case Description/Methods: A 29-year-old woman with 10 weeks of pregnancy, history of anemia and vitamin B12 deficiency was admitted to the hospital for fatigue, intractable nausea, vomiting and inability to tolerate oral intake. She denied respiratory symptoms. She was taking prenatal multivitamins but denied starting new medications or supplements. At presentation, her vital signs and clinical exam were unremarkable with uterine fundus palpable just above the pubic bone. Initial laboratory work up revealed elevated liver enzymes with AST 497 U/L, ALT 712 U/L, normal total bilirubin and ALP. Abdominal ultrasound (US) demonstrated cholelithiasis without evidence of cholecystitis or common bile duct dilation. Subsequent workup including an acute hepatitis panel; CMV, EBV serology; stool H. pylori testing; and autoimmune workup was negative. HSV IgM serology was indeterminant. A respiratory viral molecular panel by PCR was positive for RSV. Abdominal US with doppler showed normal hepatic and portal vessel blood flow and magnetic resonance cholangiopancreatography was negative for choledocholithiasis. She was treated supportively with intravenous fluids, antiemetics and close fetal monitoring. Her liver enzymes peaked on hospital day 4 with AST 863 U/L, ALT 1214 U/L, total bilirubin 1.1 mg/dL. By hospital day 5, her symptoms improved, and she was discharged. At 5 week follow up, AST and ALT improved to 62 U/L and 82 U/L, respectively. Her elective laparoscopic cholecystectomy was deferred until after delivery due to symptom resolution and absence of acute cholecystitis.
Discussion: Disseminated RSV a rare manifestation in immunocompromised individuals. Clinical presentation may be atypical, creating diagnostic challenges. Liver biopsy is rarely required to establish the diagnosis. RSV hepatitis is typically self-limited and can be treated with supportive care as antiviral agents have no proven efficacy. A high index of suspicion is required for early identification of RSV hepatitis as timely supportive care may prevent progression to acute liver failure.

Table 1. Laboratory Testing Done to Investigate Emesis Etiology Caption

Laboratory Test	Reference Range and Units	Results
Liver Function Tests		
Alanine aminotransferase (ALT)	0-34 U/L	990 (H)
Aspartate aminotransferase (AST)	15-46 U/L	750 (H)
Alkaline phosphatase (ALP)	38-126 U/L	89 (N)
Total bilirubin	0.2-1.3 mg/dL	1.4 (H)
Total Protein	6.3-8.2 g/dL	6.4 (N)
Albumin	3.5-5.0 g/dL	3.6 (N)
Coagulation Studies		
Prothrombin time (PT)	9.0-12.0	10.9 (N)
International normalized ratio (INR)	0.9-1.1	1.0 (N)
Viral Serologies		
Hepatitis A, IgM	Non-reactive	Non-reactive
Hepatitis B, core IgM	Non-reactive	Non-reactive
Hepatitis B, surface antigen	Non-reactive	Non-reactive
Hepatitis C antibody	Non-reactive	Non-reactive
Hepatitis E antibody	Non-reactive	Non-reactive
Human immunodeficiency virus 1 and 2 antibody/ antigen	Non-reactive	Non-reactive
Herpes simplex virus 1 and 2 IgM	< =0.89	0.96 (intermediate)
Cytomegalovirus quantitative PCR	Non-reactive	Not detected
Epstein Barr virus, IgM	Not detected	Not detected
Influenza A, antigen	Not detected	Not detected
Influenza B, antigen	Not detected	Not detected
Respiratory Syncytial Virus	Not detected	Detected
Autoimmune liver disease panel		
Liver-Kidney Microsome-1 Antibody IgG (Anti-LMK)	0.0 - 24.9 U	0.8 (N)
Antinuclear antibody (ANA) titer	< 1:80	< 1:80 (N)
Anti-smooth muscle antibody (ASMA)	0-19 Units	6 (N)
Antimitochondrial antibody (AMA)	0.0-24.9 Units	2.4 (N)
Miscellaneous		
Rapid plasma regain (RPR)	Negative	Negative
Total Creatinine Kinase (CK)	30-170 U/L	< 20 (L)
H. pylori antigen	Negative	Negative

- H: High; N: Normal; L: Low.

S3158

Supplement Gone Wrong: Drug-Induced Liver Injury Caused by Artemisinin

Joanne Lin, DO, Amitpaul Gill, MD, Marina Roytman, MD.
UCSF Fresno, Fresno, CA.

Introduction: Drug-induced liver injury (DILI) is defined as hepatic dysfunction caused by prescription medications, supplements, or xenobiotics after alternative causes have been excluded. As one of the leading causes of acute liver failure, DILI should be considered when patients present with hepatic dysfunction. We present a case of symptomatic DILI secondary to artemisinin use.
Case Description/Methods: A 78-year-old Chinese man with no medical history presented to the hepatology clinic with 10 weeks of jaundice, weakness, and pruritis. He started taking Artemisinin/ Bioperine 12 weeks ago to prevent COVID-19 but stopped 3 weeks ago. He denied abdominal pain, a family history of liver disease, substance/alcohol use, and taking other concomitant drugs. Physical examination revealed scleral icterus and no other signs of chronic liver disease. Laboratory studies showed total bilirubin 11 mg/dL, alkaline phosphatase 293 U/L, aspartate transaminase 170 U/L, and alanine transaminase 196 U/L with negative workup for hepatitis A, B, and C. CT abdomen and MRCP were unremarkable for liver or biliary pathology. Further serological workup was negative and follow-up labs revealed normalization of liver enzymes and bilirubin. Given the patient's improvement, liver biopsy was not pursued. The patient was instructed to avoid supplements unless prescribed by a physician.
Discussion: DILI is a global issue with an estimated annual incidence rate of 13.9 to 24.0 per 100,000 persons. Diagnosing DILI is important as it can cause acute liver injury and liver failure in certain cases. Since COVID-19 emerged, supplement use has increased given claims of boosting the immune system. Artemisinin is an herb used in traditional Chinese medicine with antimalarial activity investigated to be a possible COVID-19 treatment, but no current evidence exists to support it being effective against COVID-19[3]. Our patient's supplement also contained Bioperine, a black pepper extract, which is likely benign. Contrarily, artemisinin is a well-described cause of idiosyncratic acute liver injury and hepatotoxicity, causing self-limited mild to moderate transaminitis but also severe cases requiring emergent liver

transplantation. Our patient's unrevealing workup, his spontaneous improvement correlating with supplement discontinuation, and RUCAM score of 7 led to high suspicion of DILI secondary to artemisinin. Providers should always ask patients about supplement use and consider DILI when patients present with liver injury.

Table 1. The patient's laboratory values, including total bilirubin, alkaline phosphatase (ALP), aspartate transaminase (AST), and alanine transaminase (ALT) improved after stopping Artemisinin/Bioperine

	Total bilirubin (mg/dL)	ALP (U/L)	AST (U/L)	ALT (U/L)
Initial labs with PCP	11	293	170	196
Repeat labs with PCP	5.6	143	75	98
3 weeks after stopping supplement	2.4	85	34	41

S3159

Skin as Yellow as Turmeric: A Case Report on Turmeric-Induced Liver Injury

Bilal Ashraf, MD[1], Anastasia Tambovtseva, MD[1], Zainab Saeed, MD[1], Sara Saeed, MD[2], Thomas Brown, MD[1].
[1]UCF College of Medicine-HCA GME Consortium/HCA Florida Ocala Hospital, Ocala, FL; [2]Fatima Jinnah Medical University, Lahore, Punjab, Pakistan.

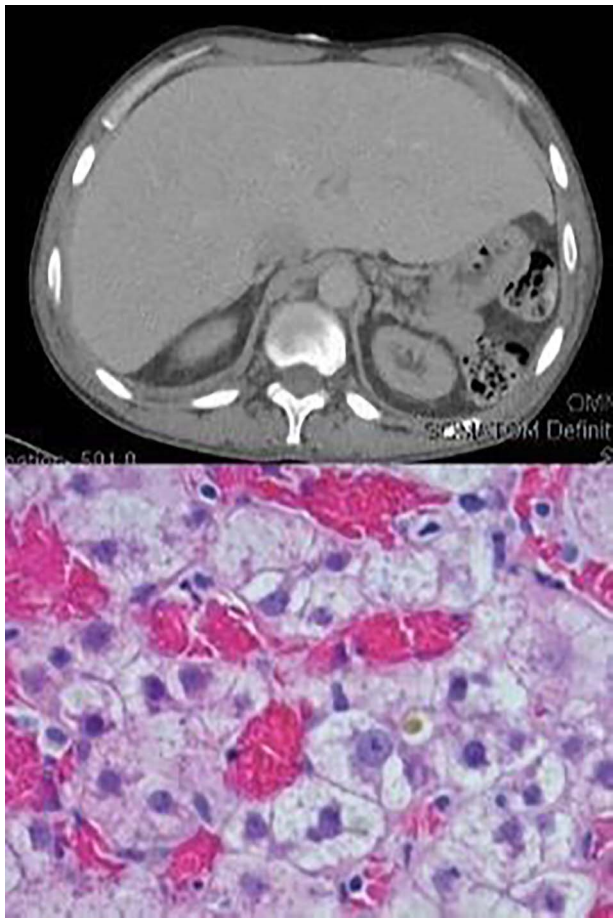
Introduction: Turmeric has been used for centuries, in food and as a medical remedy. It generally has a good safety profile, however over-the-counter and online available turmeric herbal and dietary supplement (HDS) have been recently implicated in Drug Induced Acute Liver Injury (DILI).
Case Description/Methods: A 63 year old woman was admitted for worsening jaundice for two weeks. On admission total bilirubin was 20.9mg/dL, direct bilirubin 12.1mg/dl, AST 1987 IU/L, ALT 2075 IU/L, Alkaline phosphatase (ALKP) 285 IU/L, INR 0.96, normal albumin and platelets levels. Meld-Na score was calculated to be 24. Ultrasound of liver and CT scan of abdomen did not show any hepatic or biliary abnormality. Viral hepatitis serology was negative. Auto-immune hepatitis was ruled out with negative anti-nuclear antibody (ANA), anti-smooth muscle (ASM) and anti-mitochondrial antibodies and there was no hypergammaglobulinemia. Patient denied drinking alcohol or using any other medication. She reported consuming daily Turmeric dietary supplements 6 weeks ago. Liver biopsy reveled minimal portal chronic inflammation, but no lobular inflammation/necrosis, fibrosis or plasma cell infiltration- suggesting in favor of drug induced liver injury and against autoimmune hepatitis (AIH). Turmeric was discontinued and liver enzymes gradually trended down in next few weeks.
Discussion: Turmeric has been used for centuries in food and recently is available over-the-counter and online as herbal dietary supplements (HDS). It is also used as herbal remedy for arthritis, digestive diseases etcetera for its anti-inflammatory and anti-oxidant properties. It is hypothesized that Turmeric supplements available online often contain piperine or nanoparticle delivery methods that increase its bioavailability resulting in DILI. It can occur from few weeks to months after initiation of drug. Some patient may develop drug induced autoimmune hepatitis (DI-AIH) with positive autoantibodies, however in this patient negative autoantibodies, imaging studies and liver biopsy, ruled out all other possible causes of liver injury. Roussel-Uclaf Causality Assessment Method (RUCAM) score can be used for causality assessment in DILI. RUCAM score was 9 in this case which means 'high probability' that Turmeric was the cause of DILI.

S3160

Sickle Hepatopathy: An Uncommon Sickle Cell Crisis

Anni Chowdhury, DO, Nikita Prasad, MD, Jaison John, MD, Shehzad Merwat, MD.
University of Texas Medical Branch, Galveston, TX.

Introduction: Intrahepatic vaso-occlusive syndrome, or sickle cell hepatopathy (SCH), is a rare complication of sickle cell disease. We present a case of acute hepatic sequestration in the setting of bacteremia.
Case Description/Methods: 32 male with history of sickle cell disease, auto-splenectomy and end stage renal disease presented with fatigue and diffuse myalgia. Physical exam was notable for scleral icterus, left knee swelling and erythema, and crackles in bilateral lungs. Initial labs showed leukocytosis (WBC 43,000/μL), profound anemia (Hgb 3.7 g/dL) and coagulopathy (INR 2.1). Liver enzymes were elevated at ALP 527 U/L, ALT 48 U/L, AST 209 U/L. Total bilirubin was 39 mg/dL with conjugated fraction 34.4 mg/dL, blood urea nitrogen 67mg/dL, creatinine of 4.34mg/dL, and ferritin >10,000. Blood cultures revealed Staphylococcus Aureus, likely due to left knee septic joint. US showed hepatomegaly with liver span of 24 cm. Clinical picture consistent with sepsis secondary to MSSA bacteremia in setting of sickle cell crisis and hepatopathy. Patient was admitted to ICU for hypoxic respiratory failure due to fluid overload. Clinical course was complicated by septic shock with multi-organ dysfunction including acute liver failure due to hepatic sequestration crisis, hemochromatosis, encephalopathy and disseminated intravascular coagulation. Patient required CRRT and supportive care including treatment of underlying infection, PRBC transfusions, and vitamin K administration to reverse coagulopathy (Figure).
Discussion: Hepatopathy occurs in 10% of homozygous sickle cell patients. It can be self resolving or lead to fulminant hepatic failure. Subsets of SCH include Acute Hepatic Crisis(AHC), Hepatic Sequestration, Intrahepatic Cholestasis(IC). Sickling in the sinusoids leading to hepatocellular necrosis, engorgement of Kupffer cells and bile stasis. Common presentation of all three conditions includes acute RUQ pain, nausea, fever, and elevated liver enzymes. IC is severe presentation of SCH with hypoxic injury leading to blockage and cholestasis. Presentation overlaps with AHC with addition of hyperbilirubinemia, severe jaundice, renal impairment, and encephalopathy. Total bilirubin is typically markedly elevated due to combination of hemolysis, cholestasis and renal dysfunction. Sequestration presents with significant rapid onset hepatomegaly, drop in hemoglobin with RUQ pain, which can ultimately lead to cardiac and pulmonary instability. Treatment is supportive with exchange transfusion, fresh plasma for coagulopathy.



[3160] **Figure 1.** Top: Contrast enhanced spiral CT of the chest, abdomen and pelvis showing markedly enlarged measuring 20 cm in craniocaudal dimension with lobulated contour and non visualized spleen Bottom: Liver biopsy showing dilated sinusoids with aggregates of sickled red blood cells. There is also presence of canalicular cholestasis.

S3161

Sickle Cell Hepatopathy: A Rare Complication of Sickle Cell Anemia

Jasleen Sandhu, MD, MS, Karolina Krawczyk, MD, MS, Natasha Von Roenn, MD.
Loyola University Medical Center, Maywood, IL.

Introduction: Sickle cell hepatopathy (SCH) describes the spectrum of hepatobiliary complications seen in sickle cell disease (SS). The etiology is multifaceted; repeated sickling causes liver vaso-occlusion and sinusoidal obstruction, which contributes to ischemic hepatic damage. SCH treatment is supportive; it includes supplemental oxygen, intravenous fluids and exchange transfusions. We present the diagnosis and management of a rare case of SCH.
Case Description/Methods: Our patient is a 30-year-old male with SS anemia complicated by avascular necrosis of the shoulder, infrequent pain crises and cholecystectomy, who presented to our ED with fevers >100.4F, abdominal pain, acholic stools, dark urine and jaundice. Initial workup showed transaminitis, cholestasis, with an alkaline phosphatase 189, ALT 104, AST 145 and a total bilirubin 46.5. Abdominal ultrasound and CT did not show any acute hepatic causes for these lab abnormalities. Fevers prompted an infectious workup; viral hepatitis markers and blood cultures were negative. Autoimmune processes were ruled out with normal ANA, ASMA and immunoglobulins. He was given supportive care via intravenous fluids. The fevers resolved, but his abdominal pain and jaundice persisted. His t-bili increased without any evidence of infection or renal failure. Hepatology was concerned that he had intrahepatic cholestasis as a result of his SS. They recommended an exchange transfusion and considered a liver transplant. Our patient had many antibodies in his blood, increasing the risk of complications from an exchange transfusion. We deferred a liver biopsy due to the known increased risk of bleeding in SS patients and a lack of impact on management. We continued supportive care, and his liver function improved.
Discussion: Acute sickle cell hepatopathy can range from an intra-hepatic sickle cell crisis, acute liver sequestration, and intrahepatic cholestasis; treatments include supportive care and exchange transfusions. Severe cases may require a liver transplant. We were initially concerned for acute intrahepatic cholestasis for our patient, however, his fevers did not persist and he did not have evidence of multi-organ system failure. There was also no evidence of worsening of sickling on a peripheral smear. If his lab abnormalities and symptoms worsened, we would consider doing exchange transfusions or a liver transplant. He improved with conservative measures, and this is an example of a rare complication of a serious disease.

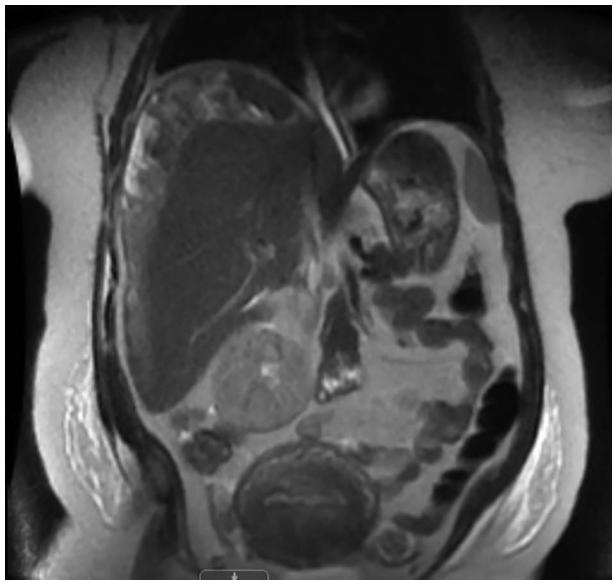
S3162

Subcapsular Hepatic Hematoma: A Rare Complication of Hemolysis, Elevated Liver Enzymes, and Low Platelet Count (HELLP) Syndrome in Pregnancy

Sarvani Surapaneni, MD, Katherine Wong, MD, Jing Wang, MD, Angy Hanna, MD, Anirudh R. Damughatla, DO.
Wayne State University/Detroit Medical Center, Detroit, MI.

Introduction: Hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome is seen in 0.1-1.0% of pregnant people overall, with the risk increased by preeclampsia. Common complications of HELLP include bleeding, disseminated intravascular coagulation (DIC), and abruptio placentae. Here we present a rare case of subcapsular liver hematoma that occurs in only 1% of HELLP patients.
Case Description/Methods: A 29-year-old woman, gravida 4, para 3 with a history of preeclampsia during prior pregnancies presents with acute onset epigastric and pelvic pain for one day at 27th-week gestation. The severe, intermittent epigastric pain radiates laterally down to the pelvis and is aggravated with laying down. The review of systems and vitals were normal. Mild tenderness in the right upper quadrant (RUQ) was noted. Labs showed high ALT 233 U/L, AST 378 U/L, low platelet 108,000/microL, and high LDH 519 IU/L. The rest of the labs were normal. She underwent an emergent C-section at admission for HELLP syndrome. An ultrasound showed two complex hypoechoic hepatic mass-like areas measuring 9.3cm and 3.5 cm in the setting of hepatomegaly and diffuse hepatic steatosis suggestive of focal fatty sparing or cavernous hemangioma. Biliary sludge without cholecystitis or cholelithiasis was also noted. An MRI with IV contrast with the liver mass protocol was obtained which showed hepatic

subcapsular hematoma of dimensions 24 cm x 9.2 cm x 3.9 cm at the level of the right hepatic lobe. The pain continued to worsen with a drop in hemoglobin from 12.8 gm/dl at admission to 7.1 gm/dl on day 4. IR was consulted for rupture concern and recommended CT angiography of abdomen and pelvis with and without arterial/venous phase that showed similar appearing subcapsular hematoma without an active bleed. Underwent image-guided hepatic angiography that showed large perihepatic hematoma along the right lateral abdominal wall without active contrast extravasation. Right hepatic artery embolization was done with a reduction in peripheral hepatic arterial blood flow by 25%. Later hemoglobin stabilized, liver enzymes improved, and the patient was discharged home 6 days later in a clinically stable state (Figure).
Discussion: Early diagnosis of subcapsular hematoma is challenging due to nonspecific signs and symptoms but crucial for improved outcomes. Physicians should evaluate for a subcapsular hematoma in patients with HELLP and RUQ pain or hypotension. Therapeutic options for ruptured hematomas include laparotomy and hepatic artery embolization.



[3162] **Figure 1.** Coronal section of MRI with IV contrast (Liver mass protocol) of abdomen and pelvis demonstrates subcapsular hepatic hematoma involving the hepatic dome and right hepatic lobe approximately measuring 24 cm in craniocaudal dimension.

S3163

Skull Metastasis in a Patient With HCC

Andres Garcia-Berrios, MD[1], Gabriela M. Negron-Ocasio, MD[1], Paola Laracuente Roman, MD[2], Tania Águila, MD[2], Jose Colon, MD[3].
[1]University of Puerto Rico Medical Sciences Campus, San Juan, Puerto Rico; [2]University of Puerto Rico School of Medicine Internal Medicine Program, San Juan, Puerto Rico; [3]University of Puerto Rico, Internal Medicine Program, San Juan, Puerto Rico.

Introduction: Hepatocellular carcinoma (HCC) rarely metastasizes to the skull. The prevalence of skull metastasis in HCC has been described to be 6.1%. Cranial nerve deficits are seen in more than a third of patients presenting with skull metastasis. We present a patient with a diagnosis of HCC with a concomitant soft tissue mass at the clivus concerning metastatic HCC.
Case Description/Methods: A 62-year-old male with a medical history of cirrhosis secondary to hepatitis C, arterial hypertension and type 2 diabetes mellitus who arrived to our institution due to 5-day history of progressive abdominal swelling and distension. In addition, he referred to having intense left-sided headache and visual disturbances, described as "double vision," three months before arrival. The physical exam showed an uncomfortable, acutely ill, with a temporal wasting patient. HEENT exam revealed the inability to abduct his left eye—suggestive of cranial nerve VI palsy—and tongue deviation to the right side. Also, the abdominal exam showed a soft and depressible abdomen, with flank dullness and mild abdominal tenderness to palpation. Imaging workup included a brain MRI which showed a soft tissue mass at the clivus extending superiorly to the suprasellar region invading the sphenoid sinuses. Additional abdominopelvic CT scan revealed a cirrhotic liver with associated portal hypertension, a large infiltrative right liver lobe LT-TIV observation due to hepatocellular carcinoma (HCC), and celiac lymph node findings concerning for metastatic disease. Management strategies include performing a diagnostic and therapeutic paracentesis which yielded results concerning portal hypertension. Neurosurgery service evaluated the patient and recommended radiotherapy without further neurosurgery management. The patient was eventually discharged home with multidisciplinary follow-up.
Discussion: Skull metastasis from HCC is uncommon; but it is notorious for affecting the patient's prognosis and quality of life. Therefore, it is important to make an early diagnosis and properly manage skull metastasis from HCC. This case exhibits the importance of considering the skull and brain metastases of a hepatocellular when evaluating a patient with HCC with neurological symptoms.

S3164

Successful Use of Ribavirin in an Immunocompetent Patient With Severe Hepatitis E

Rahul Grover, MD, Erica Chung, MD, Shalom Frager, MD, Samuel Sigal, MD.
Montefiore Medical Center, New York, NY.

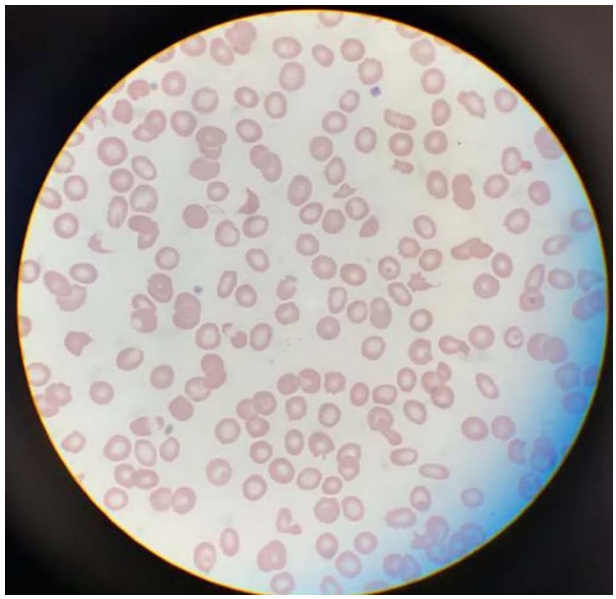
Introduction: Hepatitis E is usually a self-limited disease that does not require antiviral therapy. Limited use of Ribavirin has been described in immunocompromised patients with chronic hepatitis E virus (HEV) infection. We present a rare case of acute hepatitis E in an immunocompetent patient who was successfully treated with Ribavirin, highlighting its potential use in severe cases.
Case Description/Methods: A 23-year-old man presented with 5 days of abdominal pain and jaundice 1 month after returning from South Asia. History was negative for liver disease, unprotected sexual activity, or toxic ingestion. Liver chemistries were abnormal with AST 1684, ALT 3001, T bili 6.6, and ALP 188. Initial CBC, BMP, and coagulation studies were at baseline. Abdominal imaging was also unremarkable except for evidence of hepatic parenchymal changes. An extensive viral, toxicological, and autoimmune workup revealed acute HEV infection with a viral load of 1.8 million IU/ml. He was initially managed conservatively but required admission to the liver service by day 4 due to persistently abnormal liver studies and a worsening INR to 1.7. A trial of Ribavirin 200mg BID was initiated and the patient's transaminases and INR started to downtrend by day 5. By day 6, his symptoms improved and viral load decreased to 23,700 IU/mL. He was discharged on day 8 with a 12-week course of Ribavirin 400mg BID. Liver studies had completely resolved at follow-up 35 days after presentation.
Discussion: HEV is a common source of liver failure in regions with high endemicity but is typically self-limited in developed nations. No therapy has been approved for acute hepatitis E, however, ribavirin, a nucleoside inhibitor used for hepatitis C, has been administered effectively in immunocompromised patients with chronic HEV. Limited instances of it being used in healthy patients with acute hepatitis E have also been reported but no clear guidance exists on its use in such rare, severe cases. Although our patient was young and healthy, we chose to trial Ribavirin in light of worsening synthetic liver function and to forestall impending fulminant liver failure. Various regimens have been previously described, ranging from 600-1200mg/day, but we found that a dose of 400-800mg/day was sufficient to induce a 76-fold decrease in viral load within days of Ribavirin initiation. This case provides yet another example of Ribavirin's effectiveness in the treatment of severe hepatitis E and raises the need for further evaluation for use in such cases.

S3165

Spur Cell Anemia Mimicking DIC: A Marker of Advanced Cirrhosis

Mouhand F. Mohamed, MD, MSc[1], Kanhai Farrakhan, MD[1], Ari Pelcovits, MD[1], Pranavi Sanka, MD[1], Ronan Farrell, MD[2].
[1]Warren Alpert Medical School of Brown University, Providence, RI; [2]Brown Medicine, Providence, RI.

Introduction: Spur-cell anemia (SCA) is an uncommon, non-immune hemolytic anemia. When found in cirrhosis patients it is usually an indicator of advanced liver disease and carries a poor prognosis. Here we present a case of SCA and discuss its impact on the management and prognosis.
Case Description/Methods: A 58-year-old male with a history of decompensated alcoholic cirrhosis, diabetes, and hypertension was admitted for abdominal distention and melena. Diagnostic paracentesis ruled out spontaneous bacterial peritonitis. His initial labs showed hemoglobin 9.7 g/dL, platelets 123 × 109/L, total bilirubin 6.6 mg/dL, direct bilirubin, 3.0 mg/dL, and INR 3.4. The patient underwent an upper endoscopy showing nonbleeding grade II esophageal varices and portal hypertensive gastropathy. The patient was discharged home after stabilization. Over the next 8 months, the patient had multiple hospitalizations secondary to hepatic decompensation and continued to be anemic (Hgb down to 8.3 g/dl) with persistent hyperbilirubinemia, largely indirect (total bilirubin up to 10.3 mg/dl, indirect bilirubin, 3.1 mg/dl). Subsequent workup including hemolysis labs revealed haptoglobin < 8 mg/dl, fibrinogen 100 mg/dl, mildly elevated LDH (287 IU/L), and a negative direct antibody test (DAT). Reticulocyte index, however, was 1.2, indicative of hypoproliferative anemia. Due to chronicity and persistence of findings, DIC was deemed less likely. Multiple peripheral smears showed Spur cells, burr cells, and a few schistocytes (Figure 1). Based on this, SCA was the likely diagnosis and the patient was referred for liver transplant consideration.
Discussion: Anemia in cirrhosis patients is usually multifactorial. SCA is an uncommon form of acquired, non-immune hemolytic anemia that was first described in 1964. It is linked to advanced liver disease, primarily alcohol-related, and is associated with a poor prognosis. In a study by Vassiliadis et al., out of nine SCA patients, eight died at three months, and only one was alive at one year (one of two who underwent liver transplantation). Doll et al. reported a similarly high mortality rate (7/8 patients at eight months). SCA pathogenesis is not well elucidated; however, abnormal lipid metabolism with consequent development of spike-like projections in RBCs is thought to contribute to the formation of spur cells. The presence of SCA is an important prognostic indicator for cirrhotic patients that clinicians should be aware of.



[3165] **Figure 1.** Peripheral-blood smear showing red cells with irregular surface projections suggestive of acanthocytes, or spur cells.

Table 1. Summary of laboratory test results

Laboratory test	Latest Reference Range & Units	Result	Laboratory test	Latest Reference Range & Units	Result
Sodium	135 - 145 MEQ/L	137	Bilirubin, Direct	0.0 - 0.3 MG/DL	2.0
Potassium	3.6 - 5.1 MEQ/L	3.0	WBC	3.5 - 11.0 x10exp9/L	5.6
Creatinine, Ser	0.64 - 1.27 MG/DL	0.63	Hemoglobin	13.5 - 16.0 G/DL	8.3
AST (SGOT)	10 - 42 IU/L	161	MCV	80.0 - 98.0 fL	74.3
ALT	6 - 45 IU/L	60	Platelets	150 - 400 x10exp9/L	135
Alkaline Phosphatase	34 - 104 IU/L	174	Haptoglobin	14 - 258 MG/DL	< 8
LDH	100 - 220 IU/L	287	Fibrinogen Level	150 - 480 MG/DL	110
Bilirubin, Total	0.2 - 1.3 MG/DL	9.3	D Dimer Level	0 - 300 ng/mL	3,716
Direct Antiglobulin Test	Negative	Negative	INR	0.8 - 1.2	3.4

S3166

Syphilitic Hepatitis: Unmasking "The Great Masquerader"

Chidiebele Enunwa, MD[1], Maya Krasnow, MD[1], Lakshmi Katta, MD[1], Viniya Patidar, MD[1], Jason Cobb, MD[2].
[1]Emory Internal Medicine, Atlanta, GA; [2]Emory University School of Medicine, Atlanta, GA.

Introduction: Syphilis is a bacterial infection known as 'The Great Masquerader' for its ability to mimic other diseases. We present the case of a young man with abdominal pain and abnormal liver enzymes, initially thought to have a primary liver pathology, who was found to have secondary syphilis.
Case Description/Methods: A 22-year-old male with a past medical history of H. pylori presented after two weeks of right upper quadrant abdominal pain, pruritus, and lower extremity edema. Two months prior, he had an EGD with biopsy for nausea, vomiting, and diarrhea that confirmed H. pylori. Treatment was held for elevated LFTs. He was referred to hepatology where workup revealed elevated antimitochondrial (AMA) M2 antibody IgG (42 units), positive EBV/CMV IgG, and a negative viral hepatitis panel. Physical exam in the ED revealed a tender, non-distended abdomen and lower extremity

edema. Labs were significant for AST 87 U/L, ALT 91 U/L, ALP 858 U/L, and TBIL 1.0 mg/dL. MRCP showed nonspecific heterogeneous enhancement of the liver. Infectious workup was pursued when the patient reported recent unprotected sex with male partners, and revealed negative HIV screen, positive IgM/IgG antibodies to both CMV and EBV, positive treponemal antibody, and RPR titer of 1:128. After one dose of Penicillin, the patient had resolution of his symptoms and lab abnormalities.

Discussion: Hepatitis is a rare manifestation of syphilis, occurring in 0.2%-3% of patients with syphilis.[1] It is defined as abnormal liver enzyme levels with serological evidence of syphilis, exclusion of other causes of liver injury, and resolution of abnormal liver enzymes after treatment of syphilis.[2] Although liver involvement in syphilis can be observed at any stage of the disease, secondary syphilis is most common.[3] The clinical presentation of syphilitic hepatitis is nonspecific and usually involves rash and fatigue. Other symptoms are jaundice, fever, and abdominal pain. Lab tests usually show a marked elevation of serum ALP.[4] AMA antibodies, typically highly specific for PBC, can be positive due to molecular mimicry.[5] A diagnosis of syphilitic hepatitis is supported here by rapid resolution in symptoms and lab abnormalities after treatment with penicillin. Syphilis should be considered a cause of liver injury in patients with high-risk features, including unprotected sex with multiple partners. Early diagnosis and prompt treatment are important for preventing progression to late syphilis.

S3167

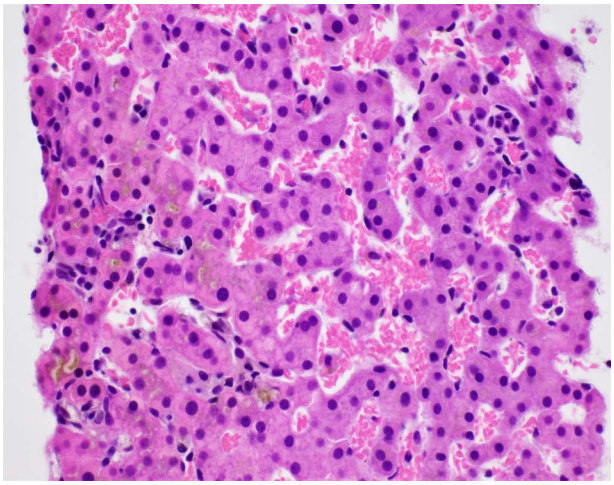
Sickle Cell Hepatopathy in a Young Patient With Markedly Elevated Bilirubin

Alexander Garcia, DO[1], Andrew Alabd, MD[2], Upasana Joneja, MD[2], Manav Bandlamudi, MD[2].
[1]Cooper Medical School at Rowan University, Cooper University Hospital, Camden, NJ; [2]Cooper University Hospital, Camden, NJ.

Introduction: The vaso-occlusive events that characterize a sickle cell pain crisis are known to affect multiple organ systems. Sickling in the hepatic sinusoids leads to ischemia, cholestasis, and sequestration, which collectively contribute to acute liver dysfunction often referred to as sickle cell hepatopathy (SCH). As one of the rarer complications of sickle cell disease (SCD) SCH may go under-recognized. Here we present a case of acute sickle cell hepatic crisis, a subset of SCH, with the aim of increasing the available literature on this important topic.

Case Description/Methods: A 20-year-old male with a history of sickle cell disease (SCD) presented for evaluation of right upper quadrant abdominal pain. Vitals signs were normal and physical examination was notable for jaundice and right upper quadrant abdominal tenderness. Labs revealed mildly elevated liver enzymes and elevated direct bilirubin of 34 mg/dL. Abdominal sonography revealed findings suggestive of hepatitis and common bile duct dilation, with suggestion of gallbladder wall thickening. MRCP showed mild intrahepatic and extrahepatic bile duct dilation, as well as splenic hemosiderosis. The common bile duct was 0.9 cm in diameter. Hepatitis A, B, C, as well as HIV, CMV, and herpes screening tests were nonreactive. An ultrasound-guided liver biopsy then revealed features of an acute sickle cell hepatic crisis, including distended sinusoids with erythrocyte sludging. With two days of symptomatic treatment alone, the patient had improvement in his direct bilirubin to 7 mg/dL and was discharged after complete resolution of his symptoms. (Figure)

Discussion: Acute sickle cell hepatic crisis (ASCHC), a subset of sickle cell hepatopathy (SCH), is often difficult to diagnose for a multitude of reasons including vague presenting symptoms and clinician unfamiliarity with the disease. While treatment guidelines are lacking with regards to the specific treatment for ASCHC, it is worth noting that in our case clinical and assay improvement was seen after several days of symptomatic treatment alone. Although not present in our case, ASCHC has the potential to be concurrent with a more severe form of the disease known as sickle cell intrahepatic cholestasis (SCIC), which if untreated can be fatal. Both ASCHC and SCIC can present with jaundice, as in our patient, but only SCIC progresses to organ failure. As more cases are described, the optimal treatment modalities for these rarer complications of sickle cell disease will only increase.



[3167] **Figure 1.** Liver parenchyma with dilated sinusoids, readily visualized sickled red blood cells (right side of the image) and canalicular cholestasis (brown pigment on the left side of the image).

S3168

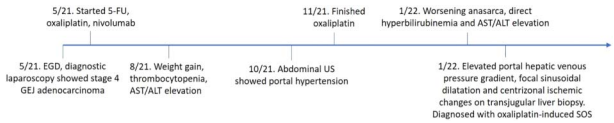
Sinusoidal Obstruction Syndrome: A Known and Often Missed Complication of Oxaliplatin Therapy

Cesar Moreno, MD, Anas Khouri, MD, Gabriel A. Bolanos Guzamn, MD, Rajab Idriss, MD.
University of South Alabama, Mobile, AL.

Introduction: Sinusoidal obstruction syndrome (SOS) is caused by sinusoidal endothelial injury due to hematopoietic cell transplantation, liver irradiation or exposure to chemotherapy such as oxaliplatin and may occur days to weeks following exposure. SOS can be a life-threatening complication and its presentation may include jaundice, volume overload or hepatic failure. We present the case of a 32-year-old male with gastroesophageal adenocarcinoma who underwent oxaliplatin-based chemotherapy and developed SOS.

Case Description/Methods: A 32-year-old male with no significant medical history presented with dysphagia and weight loss. Esophagogastroduodenoscopy showed a mass at the gastroesophageal junction. He underwent diagnostic laparoscopy which showed peritoneal metastasis. Tissue biopsy was consistent with stage 4 gastroesophageal junction adenocarcinoma. He was started on 5-fluorouracil (5-FU), oxaliplatin and nivolumab. The patient developed thrombocytopenia and moderate aminotransferase elevation after 6 cycles of chemotherapy. An abdominal ultrasound obtained after 10 of 12 cycles of chemotherapy showed portal hypertension. Subsequent abdominal imaging showed worsening ascites (serum ascites to albumin gradient > 1.1), anasarca and splenomegaly. Transjugular liver biopsy showed elevated portal hepatic venous pressure gradient, focal sinusoidal dilatation and centrizonal ischemic changes suggesting drug induced hepatopathy, and a diagnosis of oxaliplatin induced SOS was made. The patient was not a candidate for defibrotide and was treated conservatively. He continued to have worsening ascites with severe direct hyperbilirubinemia and moderately elevated aminotransferases without biliary obstruction (Figure).

Discussion: Oxaliplatin-based chemotherapies are known to cause the potentially life-threatening complication of SOS which is oftentimes missed. Our patient developed signs and symptoms suggestive of SOS by cycle 10 of oxaliplatin therapy. These changes included thrombocytopenia, elevated liver associated enzymes, volume overload and portal hypertension. He went on to complete 12 cycles of oxaliplatin with eventual worsening of his portal hypertension related symptoms and worsening of his hepatic function. This case highlights the importance of having a high index of suspicion for the development of SOS with plans to pursue patient-centered discussions regarding alternative chemotherapeutic regimens, instituting prophylactic therapies and timely SOS screening protocols.



[3168] **Figure 1.** Timeline of events leading up to SOS diagnosis.

S3169

Stauffer Syndrome and Post-Nephrectomy: Say Good-Bye to Acute Transaminitis!

Bassam Hossain, DO, MPH[1], Sumair Ahmad, MD, MPH[2].
[1]UHS WIlson Hospital, Vestal, NY; [2]Great Lakes Gastroenterology, Mentor, OH.

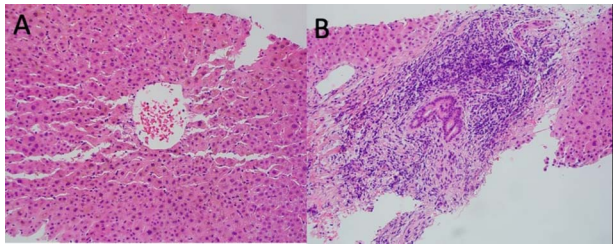
Introduction: Stauffer Syndrome (SS) is a paraneoplastic disorder associated with renal cell carcinoma (RCC). It is characterized by hepatic dysfunction in the absence of metastasis, and elevated alkaline phosphatase, aminotransferases and prolonged prothrombin time. We present a rare case of a patient who had a resolution of his transaminases status post nephrectomy.
Case Description/Methods: Patient is a 54 year old male with a past history remarkable for GERD, hypertension with recent inpatient admission for a newly diagnosed renal cell carcinoma status post partial nephrectomy. Patient was identified in having abnormal transaminases during the patient's hospital course preoperatively. Patient had elevated transaminases on admission and preoperatively that were 3 times as much when compared to his baseline, with peak values of, total bilirubin of 1.07 mg/dl, Aspartate Transaminase (AST) 88 U/L, Alanine transaminase (ALT) 146 U/L, Alkaline Phosphatase (Alk Phos) 154 U/L. Patient was diagnosed with having a left renal 7.7 x 6.8 x 6.0 cm, yellow tan hemorrhagic mass lesion that did not involve the renal capsule, which was resected during his partial nephrectomy procedure. Patient remained asymptomatic from a GI standpoint and was discharged with close outpatient GI follow-up for hepatic abnormalities. Patient's transaminases/LFTs have since normalized status post nephrectomy.
Discussion: Clinicians should be aware of Renal cell carcinoma ability to present as a broad spectrum of non renal manifestations and should have a high index of suspicion when encountered with unexplained liver abnormalities. Imaging studies should be performed to make an early diagnosis and increase the likelihood of operative success. Surgical treatment of underlying malignancy appears to resolve hepatic abnormalities.

S3170

Syphilitic Hepatitis With Cholestatic Liver Injury

Sanjeevani Tomar, MD[1], Tiberiu Moga, MD[1], Meghan Cloutier[2], Naren Nallapeta, MD[1], Elizabeth Gregory, MD[1], Emad Alqassim, MD, PhD[1], Yasmin Yusuf, MD[1], Thomas C. Mahl, MD[1].
[1]University at Buffalo, Buffalo, NY; [2]Jacobs School of Medicine and Biomedical Sciences, Buffalo, NY.

Introduction: The incidence of syphilis in the United States has increased, predominantly among men who have sex with men. Very few patients with early syphilis meet the criteria for syphilitic hepatitis, but many are HIV^+ and engage in high-risk sexual behavior.
Case Description/Methods: A 30-year-old man who identified as a MSM presented with yellowing of his eyes and hyperbilirubinemia, preceded by one month with a rash and photosensitivity and redness of his right eye. He denied any penile lesions, history of liver disease or alcohol use. A physical examination revealed multiple hyperpigmented macular lesions over his torso, arms, palms, and the soles of his feet and scleral icterus. A slit lamp examination indicated anterior uveitis in both eyes and chorioretinitis/placoid retinitis in the right eye. CBC and iron studies showed mild anemia of chronic disease (Hb 11.7 g/dL). BMP was within normal limits. LFTs were as mentioned in Table 1. Blood tests showed positive HBsAb and *T. pallidum* antibody with RPR 1:128. Serum markers for viral hepatitis and HIV were negative. CSF from a lumbar puncture had an elevated WBC count ($9/mm^3$). No biliary obstruction or masses were noted during on US and MRCP. Liver biopsy showed bland perivenular cholestasis (zone 3) (Fig. 1A) without necroinflammatory changes, mild to minimal portal inflammation (Fig. 1B), bile duct damage, and ductular reaction. The patient was treated with penicillin G for neurosyphilis, prednisolone and cyclopentolate for ocular syphilis, and ursodiol for cholestasis. Total bilirubin levels decreased to 1.7 mg/dL after 8 weeks of follow-up, and liver enzymes normalized.
Discussion: This patient met the criteria for syphilitic hepatitis (elevated liver enzymes, serologic evidence of syphilis, exclusion of other etiology, and improvement following appropriate therapy). He also had bilateral anterior uveitis and placoid retinitis of the right eye, suggesting ocular syphilis. Serum chemistries, testing for *T. pallidum* and other serologies (including positive anti-smooth muscle antibody) supported hepatic involvement, and CSF studies were consistent with neurosyphilis. Liver biopsy showed a bland cholestatic pattern without evidence of other causes of liver disease. The biopsy results are compatible with syphilitic hepatitis, though histologic features can vary and direct staining for spirochetes has limited sensitivity. These findings, together with his rapid improvement with penicillin therapy, confirmed a diagnosis of syphilitic hepatitis.



[3170] **Figure 1.** (A) Liver specimen (x40 magnification, hematoxylin and eosin stain) showing bland perivenular cholestasis (zone 3 with rare apoptotic hepatocytes. No necroinflammatory activity is present. (B) Liver specimen (x40 magnification, hematoxylin and eosin stain) showing periportal inflammation and bile duct damage with mild bile ductular reaction.

Table 1. Liver function test results

AST	41 IU/L ↑ (ULN < 38 IU/L)
ALT	53 IU/L ↑ (ULN < 42 IU/L)
ALP	210 IU/L ↑↑ (ULN < 129 IU/L)
Total bilirubin	11.6 mg/dL ↑↑ (ULN < 1.3 mg/dL)
Direct bilirubin	8.8 mg/dL ↑↑ (ULN < 0.4 mg/dL)
GGT	53 (ULN < 61 IU/L)
Albumin	3.8 g/dL
INR	1.0

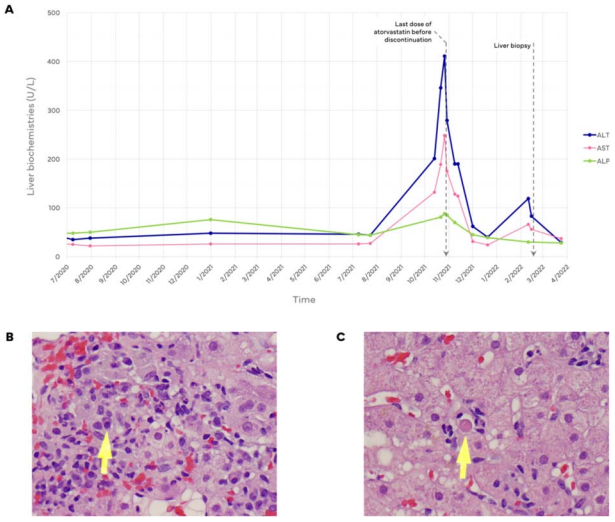
AST, aspartate transaminase; ALT, alanine transaminase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase; INR, international normalized ratio; UNL, upper limits of normal.

S3171

Statin-Related Autoimmune Hepatitis: A Case Study of a Rare Entity and Review of Current Knowledge

Antonio Pizuorno Machado, MD[1], Dongfeng Tan, MD[2], Rafaela Horrillo, PA-C[2], Hao Chi Zhang, MD[2].
[1]University of Texas Health Science Center, Houston, TX; [2]MD Anderson Cancer Center, Houston, TX.

Introduction: Statins are the most commonly prescribed drugs for the prevention and management of dyslipidemia globally. Although statins provide therapeutic benefits, they are associated with potential clinically-significant adverse effects involving muscle and the liver. While non-life-threatening statin-related drug-induced liver injury (DILI) can occur, a rare subset of cases comprise of a phenotype resembling autoimmune hepatitis (AIH). We report a case of DILI related to atorvastatin resembling AIH, which naturally resolved with cessation of the statin and without need for immunosuppressive therapy.
Case Description/Methods: A 74-year-old woman with a history of hypertension, hyperlipidemia, diabetes mellitus type II, and endometrial carcinoma presented for evaluation after being referred her oncologist for elevated liver enzymes. The patient had taken atorvastatin 40 mg/d for many years. The liver enzymes exhibited a hepatocellular pattern (Figure 1). Lab studies revealed positive ANA, ASMA, and AMA (Table 1). An ultrasound and CT of the abdomen were performed which reported no discrete hepatic changes. After the patient discontinued atorvastatin, the liver enzymes exhibited subsequent decrease (Figure 1). Liver biopsy was performed which reported liver parenchyma with portal and interface chronic inflammation, patchy minimal steatosis, and single cell necrosis of hepatocytes. The patient was diagnosed with AIH-like DILI induced by atorvastatin which was then permanently discontinued. Thirty-one days after discontinuation of statin therapy, liver enzymes improved without steroids.
Discussion: Statins are generally considered safe. The risk of severe statin-induced hepatotoxicity is reported as being 0.001%. True idiopathic AIH can be difficult to distinguish from drug-induced AIH due to overlap of clinical, laboratory, and histologic features. In the event of autoimmune-like DILI, liver enzymes normalize after the discontinuation of the offending agent. Our case shares similarities with only two other cases in the literature. In clinical practice, a case-by-case decision about statin re-challenge should be formulated in a multidisciplinary fashion to ensure the safety of prescribing a statin.



[3171] **Figure 1.** (A): Timeline of liver enzyme trends from July 2020 through March 2022. Atorvastatin was discontinued on October 29, 2021. The delayed onset of transaminase elevations while on statin therapy may in retrospect represent also an idiosyncratic form of drug-induced liver injury. Liver enzymes naturally improved after discontinuation of statin medication, without the need for systemic corticosteroid therapy. (B): Liver biopsy histology (H&E, 400x) - Moderately portal chronic inflammation consisting of mainly lymphocytes, admixed with eosinophils and occasional plasma cells (arrow: a plasma cell). (C): Liver biopsy histology (H&E, 400x) - Minimal steatosis and single cell necrosis (Councilman body) of hepatocytes (arrow: Councilman body). Abbreviations: ALT, alanine amino-transferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase.

Table 1. Laboratory results for patient within timeframe from 10/29/2021-3/24/2022

Variable	Reference Range, Adults*	Result
White blood cell count (per μL)	4,000–11,000	6.6
Hemoglobin (g/dL)	14.0–18.0	13.2
Mean corpuscular volume (fL)	82–98	92
Platelet count (per μL)	140,000–440,000	214
Sodium (mEq/L)	136–145	135
Blood urea nitrogen (mg/dL)	8–20	9
Creatinine (mg/dL)	0.70–1.30	0.81
Lactate dehydrogenase (U/L)	135–225	172
Total IgG (mg/dL)	610–1,616	1811
Total IgA (mg/dL)	61–356	Not obtained
Hepatitis A IgG antibody	Negative	Positive
Hepatitis B surface antigen	Non-reactive	Non-reactive
Hepatitis B surface antibody (mIU/mL)	< 5.0 (Negative)	21.6
Hepatitis B total core antibody	Non-reactive	Negative
Hepatitis C antibody	Non-reactive	Negative
Hepatitis E IgG antibody	Negative	Negative
Anti-nuclear antibody titer	< 1:80 (Negative)	1:640
Anti-smooth muscle antibody	Negative	63.4
Anti-liver/kidney microsomal-1 antibody	Negative	Negative
Anti-mitochondrial antibody	< 0.1 (Negative)	105
Ceruloplasmin (mg/dL)	19.0–31.0	27

Table 1. (*continued*)

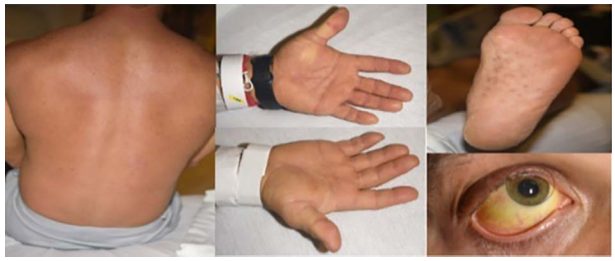
Variable	Reference Range, Adults*	Result
Ferritin (ng/mL)	30–400	284
Iron (μg/dL)	59–158	90
Total iron binding capacity (μg/dL)	250–450	342
HFE gene mutation analysis	—	Not obtained
Alpha-1 antitrypsin antibody (serum)	***	176
Triglycerides (mg/dL)	< 150	185
Total cholesterol (mg/dL)	< 200	152
LDL-cholesterol (mg/dL)	< 100	89
Anti-tissue transglutaminase IgA antibody (U/mL)	< 4.0	1.2
Anti-tissue transglutaminase IgG antibody (U/mL)	< 6.0	Not obtained
Celiac HLA DQ alleles	—	Negative

S3172

Syphilis: "The Great Imitator" Presenting as Acute Hepatitis

Ammar Ahmad, MD[1], Fadi Odish, MD[1], Sangeeta Agrawal, MD[2].
[1]Wright State University Boonshoft School of Medicine, Dayton, OH; [2]Dayton VA Medical Center, Dayton, OH.

Introduction: *Syphilis* is a multi-systemic disease caused by spirochete *Treponema pallidum*. Very rarely, it can affect the liver and cause hepatitis. Since most cases of hepatitis are caused by viral illnesses, syphilitic hepatitis can be missed. Here, we present a case of syphilitic hepatitis in a 35-year-old male.
Case Description/Methods: Patient was a 35-year-old male who presented to the hospital for jaundice and mild intermittent right upper quadrant abdominal pain. His medical history was only significant for alcohol abuse. His last drink was 4 weeks ago. He was sexually active with men. On exam, hepatomegaly, mild tenderness in the right upper quadrant, jaundice, and fine macular rash on both hands and feet were noted. Lab tests revealed an ALT of 965 U/L, AST of 404 U/L, ALP of 1056 U/L, total bilirubin of 9.5 mg/dL, direct bilirubin of 6.5 mg/dL, INR of 0.96, and albumin of 2.0 g/dL. Right upper quadrant ultrasound showed an enlarged liver but was negative for gallstones and hepatic vein thrombosis. MRI of the abdomen showed periportal edema consistent with hepatitis without any gallstones, masses, or common bile duct dilation. HIV viral load and Hepatitis C viral RNA were undetectable. Hepatitis A & B serologies were indicative of prior immunization. Hepatitis E serology and SARS-CoV-2 PCR were negative. Ferritin level was 177 ng/mL. Alpha-1-antitrypsin levels and ceruloplasmin levels were normal. Anti-Smooth muscle antibody titers were slightly elevated at 1:80 (Normal < 1:20). Anti-Mitochondrial antibody levels were also slightly elevated at 47.9 units (Normal < 25 units). RPR titer was 1:32 and fluorescent treponemal antibody test was reactive which confirmed the diagnosis of syphilis. Liver biopsy was then performed which showed presence of mixed inflammatory cells without any granulomas which is consistent with other cases of syphilitic hepatitis. Immunohistochemical stain was negative for treponemes. Patient was treated with penicillin and did have Jarisch-Herxheimer reaction. ALT, AST, ALP, and total bilirubin down trended after treatment. Repeat tests drawn exactly 1 month post treatment showed normal levels of ALT, AST, ALP, and total bilirubin (Figure).
Discussion: Liver damage can occur in syphilis and can easily be missed because of the non-specific nature of presenting symptoms. In our patient, the fine macular rash on both hands and feet along with history of sexual activity with men prompted us to test for syphilis which ultimately led to diagnosis and treatment in a timely manner.



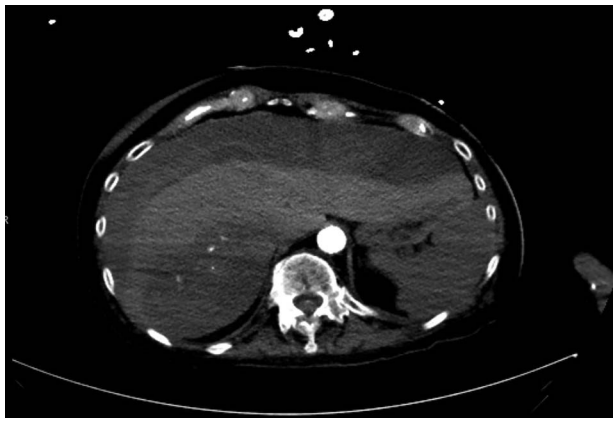
[3172] **Figure 1.** Maculopapular rash on hands, feet, back and scleral icterus.

S3173

Spontaneous Hepatic Subcapsular Hemorrhage in Malignancy: A Case Report

Kyle L. Marrache, DO, Stephanie Tzarnas, MD, Shravya Ginnaram, MD, Sarah Elrod, DO.
Abington-Jefferson Health, Abington, PA.

Introduction: Hepatic Subcapsular Hematoma (HSH) is an infrequent condition most associated with HELLP syndrome, as an uncommon complication of ERCP and as a result of coagulopathies or malignancies. In this case report we present a 72-year-old woman with metastatic cancer who died after a spontaneous rupture of a HSH.
Case Description/Methods: A 72-year-old woman with a history of metastatic lung adenocarcinoma presented to the hospital for lower extremity weakness. She was currently undergoing immunotherapy with pembrolizumab. On admission she no complaint and abdominal exam was normal. No imaging was done of the abdomen, but prior MRI and CT of the abdomen had noted small right lobe lesions within the liver. Previous ultrasound of the liver had also shown 3.5cm posterior right lobe mass. Initial labs showed a PT/INR of 14.6 and 1.3 respectively. Alkaline phosphatase was 237, AST was 36 and ALT was 28, and platelet of 290. Her stay was uneventful until day 7 when she started to develop thrombocytopenia with a platelet count of 24, PT/INR of 16.3/1.4 respectively, and fibrinogen of 68. The next day LDH was 2,500 and she started to develop a leukocytosis. On day 10 a rapid response was called for acute right upper quadrant pain. Initial evaluation showed sinus tachycardia but a soft and non-tender abdomen. She became less responsive, so she was taken to the CT scanner which showed a large subcapsular hemorrhage compressing both her liver and her aorta against her spine. (Fig. 1) She was intubated and taken to the MICU where she was started on pressor support and the massive transfusion protocol was activated. Surgery came and evaluated her but felt that given the size of her hemorrhage an emergent laparotomy would be fatal. She ultimately was changed to DNR/DNI by family and died.
Discussion: HSH ruptures are cited as complication of tumors, HELLP syndrome, and as a rare complication of ERCP. Initial symptoms almost always include abdominal pain but can also include anemia and shock. When shock is present in-hospital mortality rate can be as high as 23%. The mainstay of treatment in unstable patients is surgical resection or trans-arterial embolization. Even if surgical intervention is initially successful most patients die within a year due to either liver failure or hemorrhagic recurrence. In our patient her age and underlying cancer made surgical intervention almost certainly fatal. This case reinforces the current literature around HSH rupture and serves to demonstrate the severity of this condition.



[3173] **Figure 1.** Large spontaneous subcapsular hemorrhage with compression of spine.

S3174

Statin-Induced Liver Injury With Short Latency to Onset

Kimberline Chew, MD, Clara Tow, MD.
Montefiore Medical Center, Bronx, NY.

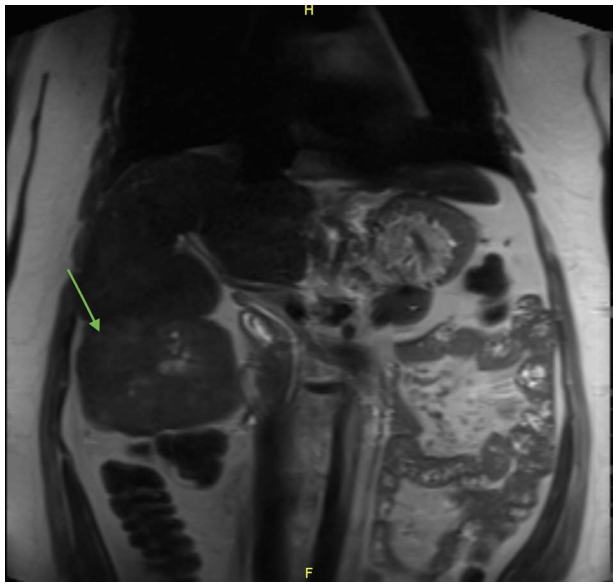
Introduction: Statins can cause an increase in liver enzymes, but these elevations resolve spontaneously in 70% of patients despite continued therapy. It is rare for it to cause significant, clinically apparent liver injury. The latency to the onset of liver injury had a median of 155 days, with most cases occurring around 3-4 months after initiation of statin. We describe a patient with statin-induced symptomatic liver injury within one month of statin initiation.
Case Description/Methods: A middle-aged woman with past medical history of diabetes mellitus type 2, hypertension, hyperlipidemia, and recent ST-elevation myocardial infarction (STEMI) presented with nausea, vomiting, pruritus, painless jaundice and loss of appetite for over one week. One month prior to her onset of symptoms, she was started on atorvastatin after her STEMI. Her baseline liver biochemistries were normal. Her initial labs on presentation were alkaline phosphatase 2,170, AST 456, ALT 502 and direct bilirubin 10.7, showing a predominantly cholestatic pattern but also with some hepatocellular injury. Atorvastatin was stopped on admission. CT imaging and ultrasound showed liver steatosis and cholelithiasis. No biliary dilatation was seen on MRCP. Other workup was negative, including viral markers and autoimmune studies. Liver biopsy showed sinusoidal dilation/congestion and lymphocytic predominant portal inflammation with eosinophils and plasma cells suggestive of drug-induced liver injury (DILI). She was started on N-acetylcysteine and ursodiol. Her hepatic function initially continued to worsen as noted by high total bilirubin and rising INR, with mildly worsening mental status. She also had acute kidney injury requiring initiation of hemodialysis. Decision was made for expedited liver transplant evaluation. While waiting for the evaluation, she showed gradual improvement in her liver biochemistries and symptoms.
Discussion: This case describes a patient who developed statin-induced liver injury within one month of atorvastatin initiation. Although statins can develop mild liver-enzyme elevations, it is rare for it to cause such clinically apparent DILI. Given the widespread use of statins, this case highlights the importance for physicians to closely monitor liver enzymes in patients after initiation of statins.

S3175

The Premalignant Shunt

Baha Aldeen Bani Fawwaz, MBBS, Aimen Farooq, MD, Sadaf Raoof, MD, Gurdeep Singh, DO, Manoucher Manoucheri, MD.
AdventHealth Orlando, Orlando, FL.

Introduction: Abernathy malformation is a rare vascular anomaly in which the splenic (SV) and the superior mesenteric veins (SMV) bypass the liver and drain directly into the IVC. It is classified into two types depending on whether the shunt is complete (type I) or partial (type II). Type I is further subclassified into 2 subtypes; in type Ia the SV and SMV drain separately into the IVC, while in type Ib the 2 veins conjugate to form a trunk before draining into IVC. Type I Abernathy syndrome is mostly diagnosed in females and is associated FNH, adenoma and HCC. Here, we report a case of Abernathy syndrome type Ib that was diagnosed in a male patient who was found to have adenomas, FNH and HCC.
Case Description/Methods: A 37-year-old male patient with history of Beals syndrome was referred to the hepatology clinic for evaluation of incidentally discovered hepatic masses on CT scan. Further imaging with MRI abdomen showed that one lesion has grown in size with worrisome appearance, a new lesion, other lesions unchanged and type Ib Abernathy malformation. The patient underwent liver biopsy which was positive for HCC without any significant parenchymal fibrosis. The patient denied any active complaints,denied any contributory family or social histories.Basic laboratory studies were remarkable for mild to moderate elevations in LFTs.Chronic liver disease work-up was unremarkable.Staging work up did not reveal any metastasis.Patient underwent liver resection (LR) and pathology showed HCC arising from adenoma.The patient tolerated the procedure well and will continue to follow up with hepatology clinic (Figure).
Discussion: Although it is unknown how rare Abernathy malformation is, Franchi-Abella et al reported 413 patients in literature between 1979 and 2017. It is estimated that around half of patients with type Ib malformation will ultimately develop one or more liver tumors. It is believed that the absence of portal flow leads to a compensatory hyper-arterialization, this might lead to an increase in oxygen free radicals and tumorigenesis. Treatment options for early HCC include LR and liver transplantation. According to current guidelines, LR is the treatment of choice for non-cirrhotic HCC. In our case, biopsy was negative for cirrhosis; thus, it was decided to proceed with LR. Multiple connective tissue and genetic disorders have been reported to be associated with Abernathy malformation, this raises the question of whether there might be any connection between connective tissue disorders and Abernathy malformation.



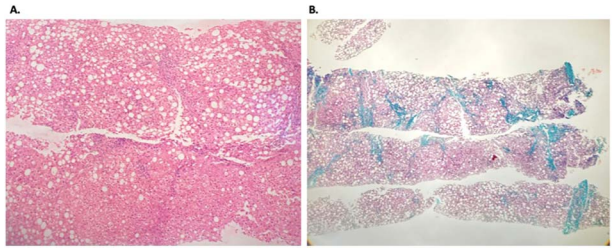
[3175] **Figure 1.** MRI abdomen showing hepatic segment 5/8 lesion has significantly increased in size compared to last CT and likely represents an adenoma with possibility of fat-containing hepatocellular carcinoma given its growth.

S3176

Tenofovir Disoproxil Fumarate-Related Metabolic Syndrome and Steatohepatitis in a Case of Chronic Hepatitis B

Nazli Begum Ozturk, MD[1], Mehmet Akyuz[2], Mine Gulluoglu, MD[3], Filiz Akyuz, MD[3].
[1]Istanbul University, Istanbul School of Medicine, Rochester, MN; [2]Yeditepe University School of Medicine, Istanbul, Istanbul, Turkey; [3]Istanbul University, Istanbul School of Medicine, Istanbul, Istanbul, Turkey.

Introduction: Tenofovir disoproxil fumarate (TDF) is a nucleotide analog used in the treatment of chronic hepatitis B virus infection (HBV). TDF is considered to have no direct hepatotoxicity. Herein we present a patient with HBV developing liver test abnormalities and biopsy-proven steatohepatitis (SH) after starting TDF, and the resolution of liver tests and SH after switching TDF to entecavir (ETV).
Case Description/Methods: A 48-year-old female patient was admitted to our clinic with positive HBsAg and HBV-DNA level of 21600 copies/mL. Her Anti-HBe was positive and Anti-HDV was negative. Abdominal ultrasonography revealed 2 cm hepatomegaly; her AST was 74 U/L (5-40 U/L) and ALT 125 U/L (10-40 U/L). Her BMI was within normal limits, she had no comorbidities and was not on any medication. Her lipid panel was within the normal range. She was prescribed 245 mg/day TDF, and was scheduled for regular clinical follow-up. Six months later, her AST was 113 U/L, ALT 148 U/L, and her HBV-DNA was negative. The patient was advised to follow-up closely. Eighteen months after the initiation of TDF, her AST increased to 211 U/L and ALT to 216 U/L. Her HbA1c was 6.1% and HOMA-IR was 4.0. MRI showed fat accumulation in the liver, and a liver needle biopsy was performed. Histological evaluation revealed grade 2 steatosis with moderate ballooning degeneration and stage 3/4 fibrosis (Figure 1). Autoimmune serologies including antinuclear antibody and anti-smooth muscle antibody were negative, and ceruloplasmin phenotype was normal. The causation of TDF was hypothesized. TDF was stopped, and ETV 0.5 mg/day was initiated. Her follow-up at month 3 revealed a decreased trend in transaminase levels. The patient lost to follow-up for 2 years, but she used ETV regularly. Her control HbA1c was 5.5% at this last visit and a control abdominal ultrasonography showed no fat accumulation.
Discussion: While it is known that TDF co-administration with other nucleoside analogs (e.g., didanosine, stavudine) can cause fatty infiltration of the liver, we present a rare case of metabolic syndrome and SH related with TDF monotherapy. Despite some reports suggesting tenofovir, specifically tenofovir alafenamide, might be associated with adverse metabolic changes in patients with HIV, our patient with HBV developed these changes while she was on TDF. We show that hepatic steatosis associated with TDF can be reversible with the medication change to ETV.



[3176] **Figure 1.** A: Hematoxylin-eosin staining shows 50% steatosis (grade 2), portal and lobular inflammation and moderate hepatocyte ballooning. B: Masson-trichrome staining shows portocentral and pericellular fibrosis (stage 3/4).

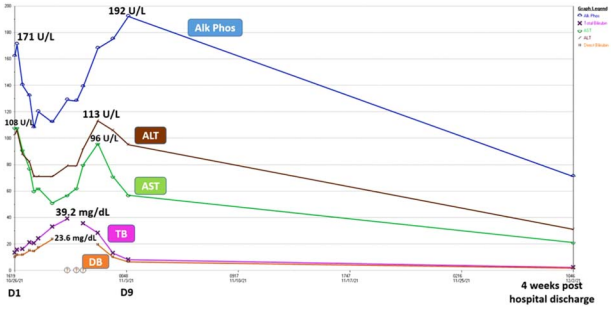
S3177

The (Icteric) Eye of the Storm: A Case of Weil's Syndrome

Baruh B. Mulat, Hilary Hertan, MD, FACG.
Montefiore Medical Center, Bronx, NY.

Introduction: Leptospirosis is one of the most prevalent zoonotic diseases affecting humans residing in tropical areas. We herein describe a case of a patient with leptospirosis acquired in the Bronx, NY.
Case Description/Methods: A 63 year-old man with no significant medical history was evaluated for muscle pain, fatigue, and diarrhea of 3 days duration. He was feverish to 102 F and icteric. Laboratory evaluation revealed hemoglobin of 7.5 g/dL, total white count of 18 k/uL, platelet count of 40 k/uL, INR 1.5, creatinine of 5 mg/dL, urea nitrogen 85 mg/dL, creatine kinase 4000 U/L, alkaline phosphatase 192 U/L, total bilirubin 39 mg/dL, direct bilirubin > 23 mg/dL, AST 96 U/L, ALT 113 U/L and albumin 2.5 g/dL. Initial laboratory evaluation was negative for common and atypical infections. Radiologic evaluation revealed unremarkable non-contrast MRCP and US abdomen. Upon further detailed history, the patient reported that his basement was recently flooded by a devastating hurricane. Despite negative parasite blood smear and IgM antibodies for leptospira, empiric treatment for leptospirosis with Ceftriaxone resulted in resolution of all symptoms and lab abnormalities. Repeated blood work on follow-up visits at 4 weeks was noted with positive IgM antibodies for leptospira (Figure).

Discussion: Leptospirosis is caused by a poorly staining gram-negative spirochetal bacteria. Transmission to humans occurs through mucous membranes, open skin wounds or after drinking, eating, swimming, or wading in water contaminated by stool or urine of infected rodents. Outbreaks have been documented in non tropical, urban areas affected by hurricanes, heavy rains or floods. In approximately 90% of cases, the disease is anicteric. Phase 1 includes viral-like illness, conjunctival suppuration followed by transient improvement. Phase 2 includes myalgia, nausea, vomiting, abdominal pain, hepatomegaly, increased serum bilirubin and serum aminotransferases. 10% of cases manifest as Weil's syndrome, characterized by jaundice in the first phase, followed by second phase manifesting with high fever, profound jaundice with severely elevated serum bilirubin levels (up to 30mg/dL, mostly direct) and elevated serum transaminases. Renal and liver involvement are predominant and can lead to severe morbidity and death. Blood cultures, urine cultures and specific anti Leptospira IgM serology can assist with diagnosis. Antibiotic treatments, primarily Penicillin, 3rd generation Cephalosporins and Doxycycline, can benefit if administered at an early stage.



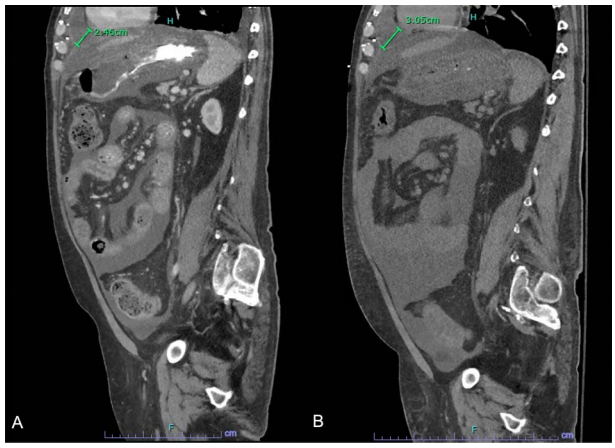
[3177] **Figure 1.** Laboratory evaluation during hospital stay and 4 weeks after discharge. Abbreviation: ALP, alkaline phosphatase; TB, total bilirubin; DB, direct bilirubin; AST, Aspartate transaminase; ALT, Alanine transaminase.

S3178

Tips for a Heartbreak: Hepatic Hydropericardium Improves After Transhepatic Portosystemic Shunt Placement

Brendan Andres, MD, Radhika Babaria, MD, Avin Aggarwal, MD.
University of Arizona, Tucson, AZ.

Introduction: Hepatic hydropericardium (HHP) has been observed as a complication of cirrhosis in patients with and without compensation. We present a case of HHP that improved after transhepatic portosystemic shunt (TIPS) placement in a patient with decompensated, non-alcoholic cirrhosis and constrictive cardiomyopathy (CCM) with a remote history of cardiac tamponade due to viral pericarditis.
Case Description/Methods: A 63-year-old-male with compensated non-alcoholic cirrhosis, obesity, chronic kidney disease, coronary artery disease, and CCM presented with abdominal distension and generalized anasarca despite high dose diuretics. Abdomen and pelvis computerized tomography (CT) revealed large volume ascites, moderate anasarca, varices, splenomegaly and a 11.3 x 3.8 x 4.2 cm substernal fluid collection (Figure 1A). Transthoracic echocardiogram to further evaluate the fluid collection showed a 70 % left ventricular ejection fraction and a 2.8 cm loculated pericardial effusion (PE) with tamponade physiology. Emergent pericardiocentesis and paracentesis were performed. During the bubble study of the pericardiocentesis, translocation from the pericardial to the peritoneal space suggestive of a fistula was noted. Fluid studies were transudative and sterile with equal protein and albumin levels of 1.7 and 1.2 g/dL respectively. The PE and ascites recurred (Figure 1B). CT chest again suggested a peritoneal-pericardial fistula. Two years prior, the patient had inflammatory pleural effusions and PE with cardiac tamponade due to viral pneumonia that completely resolved after drainage. Work up at that time revealed CCM on cardiac catheterization, and a pericardiectomy showing chronic inflammation and fibrosis was performed 4 months prior to the current admission. Due to refractory ascites and PE with tamponade, the patient underwent TIPS placement. Subsequently, the patient had significant improvement in volume status and was discharged on daily furosemide 40 mg and spironolactone 25 mg. Follow up imaging revealed resolution of ascites and a non-tamponading 1.7 cm loculated PE.
Discussion: Our case illustrates HHP can be diagnosed via pericardiocentesis with bubble study and improves after TIPS. We hypothesize the patient developed a fistula after pericarditis or cardiothoracic surgery that led to HHP in the setting of portal hypertension via this tract, ultimately improving with TIPS. The HHP likely did not fully resolve due to loculation from PE chronicity and prior inflammation.



[3178] **Figure 1.** A) Sagittal CT demonstrating PE prior to pericardiocentesis. B) Sagittal CT demonstrating recurrence of PE after pericardiocentesis.

S3179

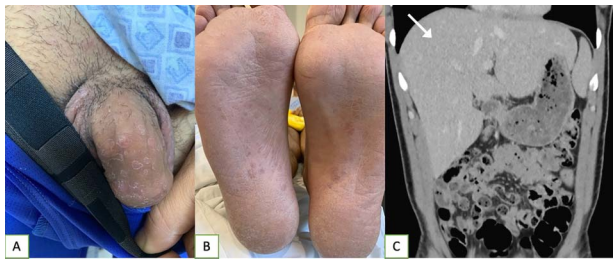
The Great Imitator Strikes Again: A Case of Syphilitic Hepatitis

Blaine Massey, DO, Kyaw Min Tun, DO, Zahra Dossaji, DO, Dimal Patel, MD.
Kirk Kerkorian School of Medicine at UNLV, Las Vegas, NV.

Introduction: Syphilis, a STI caused by Treponema pallidum, is often referred to as the "Great Imitator." The incidence of syphilis has been rising throughout the U.S. since 2001. Recent CDC data reveal 133,945 cases of all stages in 2020, a 52% increase from 2016. The exact incidence of SH in early syphilis is thought to be roughly 3%, but this is likely underestimated. We present the case of a HIV-negative male with secondary syphilis and syphilitic hepatitis (SH).
Case Description/Methods: A 33-year-old healthy male presented with malaise and painless rash for 3 weeks. Other symptoms included fevers, chills, night sweats, weight loss, decreased appetite, sore throat, and odynophagia. History was negative for abdominal pain, substance use, primary chancre, sexual activity with men. Exam revealed jaundice, pharyngitis without mucosal lesions, nontender cervical

lymphadenopathy, and a papulosquamous rash involving the trunk, penile shaft, and soles of feet (Fig 1). Initial labs showed hyperbilirubinemia, alkaline phosphatase in the 1000s, and moderately elevated transaminases (Table 1). HIV, acute hepatitis panel, anti-mitochondrial and smooth muscle antibodies were negative. Notably, syphilis antibody immunoassay was positive, with reflex RPR titer of 1:128. CT and ultrasound showed hepatomegaly (20.2cm) without focal liver lesions (Fig 1), and mild gallbladder wall thickening (4mm). Patient was treated with one dose of benzathine penicillin G 2.4M units. Symptoms resolved within 24 hours. A clinical diagnosis of SH was made, and liver biopsy was not performed. Abnormal liver enzymes seen while inpatient normalized over the course of 2 months.

Discussion: While syphilis rates continue to rise, we can expect to see SH more frequently. Disproportionate elevations of ALP and GGT are frequently seen, likely due to pericholangiolar inflammation. A disproportionate elevation in ALP can be an important clue present in most patients with SH, with relatively lesser elevations in transaminases and moderate hyperbilirubinemia. It is important to note that SH can mimic other causes of acute liver injury such as autoimmune hepatitis and primary biliary cholangitis. Proposed diagnostic criteria for SH include: abnormal liver enzymes, clinical presentation of secondary syphilis with positive syphilis serologies, absence of other causes of liver injury, and improvement in liver enzymes after antibiotics. SH should be high on the differential for patients presenting with acute liver injury and maculopapular rash.



[3179] **Figure 1.** A. Papulosquamous rash of the penile shaft B. Resolving hyperpigmented papulosquamous rash involving the soles of bilateral feet C. CT abdomen with contrast (coronal view) demonstrating hepatomegaly, liver (arrow) measuring 20.2 cm.

Table 1. Laboratory Testing

Test	Pre-Treatment	Post-Treatment (2 months)	Reference Range
Total bilirubin	5.6	0.8	0.3-1.2 mg/dL
Direct bilirubin	2.8	N/A	0.0-0.2 mg/dL
ALP	1050	246	32-110 IU/L
AST	195	31	15-41 IU/L
ALT	442	58	15-63 IU/L
GGT	555	192	7-50 IU/L
Albumin	3.7	4.3	3.3-4.8 g/dL
Platelets	495	345	150-400 K/mcL
INR/PT	1.0/12.9	N/A	0.9-1.2/12.2- 14.4 seconds
Syphilis Antibody EIA	Reactive	N/A	Nonreactive
RPR Titer	1:128	N/A	Negative
WBC	8.9	4.8	4.8-10.8 K/mcL
HIV	Nonreactive	Nonreactive	Nonreactive
Anti-mitochondrial (M2) IgG	< 20.0	N/A	Negative < 20
Anti-smooth muscle antibodies, total	Negative	N/A	Negative
Hep A antibody, total	Reactive	N/A	Nonreactive
Hep B surface antigen	Nonreactive	N/A	Nonreactive
Hep B core antibody	Nonreactive	N/A	Nonreactive
Hep B surface antibody	Reactive	N/A	Nonreactive
Hep C antibody	Nonreactive	N/A	Nonreactive

Abbreviations: ALP: alkaline phosphatase, AST: aspartate aminotransferase, ALT: alanine aminotransferase, GGT: gamma-glutamyl transferase, INR: international normalized ratio, PT: prothrombin time, EIA: enzyme immunoassay, RPR: rapid plasma reagin, WBC: white blood cell, HIV: human immunodeficiency virus.

S3180

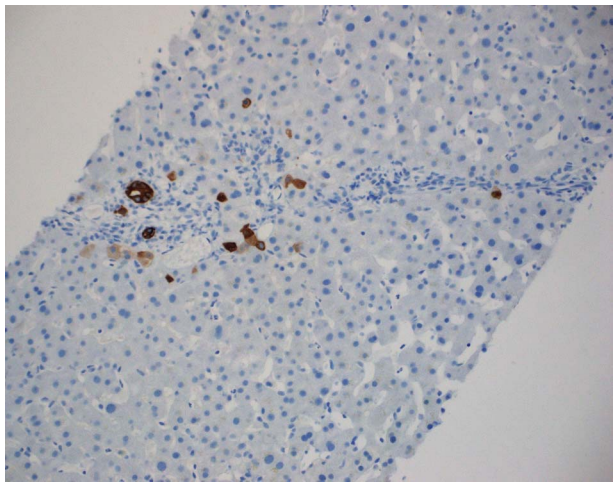
The Case of the Disappearing Ducts: Hodgkin's Lymphoma Presenting With Vanishing Bile Duct Syndrome

Judah Morgan, MD[1], Robert C. Weishar, BS[2], Charles Larcom, MD[1], Ryan Kwok, MD[1].
[1]Madigan Army Medical Center, Auburn, WA; [2]Uniformed Services University of the Health Sciences, Bethesda, MD.

Introduction: Classical Hodgkin's Lymphoma (cHL) can involve hepatic dysfunction that may arise through a variety of mechanisms. These include direct infiltration, viral reactivation, bile duct obstruction, hemophagocytic proliferation, drug induced injury, and paraneoplastic phenomenon. Vanishing Bile Duct Syndrome (VBDS) is a rare, acquired condition involving destruction and loss of intrahepatic bile ducts leading to cholestasis that has been associated with various etiologies including as a paraneoplastic phenomenon associated with cHL. This is a case of a 24-year-old with VBDS ultimately found to be the cause of profound cholestatic liver injury present at the time of a new diagnosis of cHL.

Case Description/Methods: The patient presented with abdominal pain, nausea, and jaundice for 1 week as well as night sweats and weight loss for about 6 months. Physical exam was notable for marked jaundice, scleral icterus, and diffuse lymphadenopathy. Labs showed a cholestatic pattern of liver injury with a severe direct hyperbilirubinemia and mildly elevated transaminases. A lymph node biopsy demonstrated cHL and an MRCP showed hepatomegaly without biliary dilation, obstruction, or other focal abnormalities. Laboratory evaluation for metabolic, autoimmune, viral and other infectious etiologies were negative. A subsequent liver biopsy showed inflammation of the portal tracts, no lymphoproliferative infiltration, and focal small bile duct drop out consistent with VBDS. The patient was treated with prednisone, colestipol, ursodiol, and diphenhydramine for symptom management and promptly initiated on chemotherapy (Figure).

Discussion: In cases such as this, a complete evaluation is essential. A liver biopsy is required for this diagnosis and can be done while some other tests are still pending if suspicion is high enough. Overall, there have been 29 cases of this syndrome associated with HL reported in the literature since 1993. It seems that 10 of these cases, a total of 34%, resolved after successful treatment and remission of cHL. Early and aggressive treatment of the underlying malignancy is recommended in these cases which highlights the importance of recognition of this rare syndrome. Although this case highlights a particular rare entity that may cause cholestatic liver injury in patients with cHL, it is paramount to conduct a thorough evaluation for other potential causes to include infiltrative, viral, obstructive, autoimmune, and drug-induced processes.



[3180] **Figure 1.** Liver Biopsy with IHC stain for CK7 showing disrupted small bile ducts and small duct proliferation.

S3181

TB or Not TB? That Is the Question: A Case of Granulomatous Hepatitis

Marcellus A. Edwards, MS, MD, Chibuzo Eze, MD.
Emory University College of Medicine, Atlanta, GA.

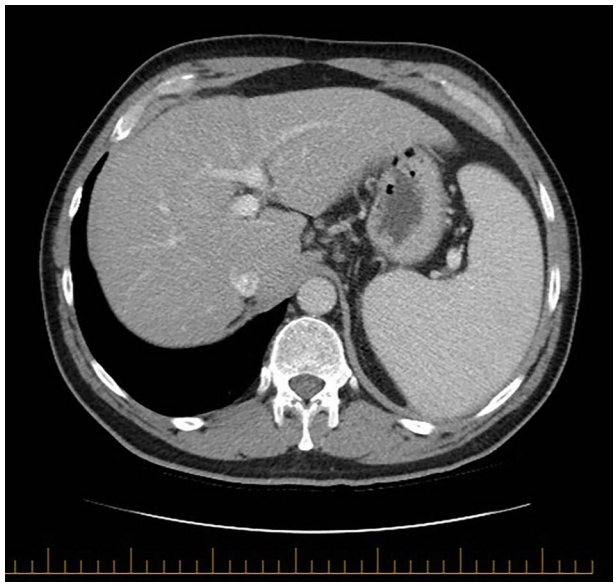
Introduction: Granulomatous hepatitis is characterized by the presence of granulomas within the liver parenchyma. This disease has multiple etiologies with the most common being primary biliary cholangitis and sarcoidosis in the US, and tuberculosis (TB) worldwide. Despite TB being a leading cause of granulomatous hepatitis globally, it is exceedingly rare to have presentation of this disease isolated in the liver without the presence of pulmonary or disseminated infection. Increased awareness of this presentation, especially in HIV/AIDs patients can lead to a decrease in unnecessary testing and procedures as well as, efficient resource allocation.
Case Description/Methods: A 53-year-old female with a history of HIV, not on antiretrovirals (ARVs), presented with fever for several days, and was found to be septic secondary to pneumonia. After resolution of her acute illness, she was restarted on ARVs, and was doing well until she developed fevers of unknown origin and transaminitis. Leading differential diagnosis included immune reconstitution inflammatory syndrome, drug-induced liver injury, and autoimmune hepatitis. Initial work-up revealed an elevated ANA and anti-smooth muscle antibody. However, given the low antibody titers and marginal improvement with steroids it was determined that an autoimmune etiology was unlikely. Additional workup led to a liver biopsy which was significant for necrotizing granulomas favored to be tuberculous over necrotizing sarcoidosis by pathology. Although the acid fast bacillus stain was negative, the patient was started on empiric therapy for TB given a high clinical suspicion, and she subsequently had a significant decrease in her transaminases.
Discussion: The diagnosis of granulomatous hepatitis presents a challenge given the multiple etiologies of the disease. This difficulty is confounded in the setting of uncontrolled HIV/AIDs, especially when the underlying causes is from TB without pulmonary or disseminated TB infection. Diagnosis requires liver biopsy, with the initial analysis including direct microscopic visualization; and AFB staining. The problem that arose in this case is that tissue stains have a relatively low sensitivity of ~72.7%, and ~88% with PCR. It is important for providers to acknowledge that these diagnostic tests are not infallible, and the clinical context and medical history surrounding the patient can be the most important factor in clinical decision making when these test fail to yield the expected results.

S3182

The Mystery Case of Chronic Thrombocytopenia in a Firefighter

Niala Moallem, MD[1], Garrett Fiscus, DO[2], Jacqueline A. Savage, MD[3].
[1]*University of Connecticut, Hartford, CT;* [2]*UCONN Health, Farmington, CT;* [3]*Hartford Hospital, Hartford, CT.*

Introduction: We present a case of chronic unexplained thrombocytopenia and incidental thromboembolism in a patient with newly diagnosed chronic obstructive pulmonary disease (COPD). He was admitted to the hospital for assessment of anticoagulant tolerance and workup for his thrombocytopenia which resulted in the diagnosis of alpha-1 antitrypsin (AAT) deficiency.
Case Description/Methods: A 57-year-old non-smoker male recently diagnosed with COPD was undergoing workup for chronic thrombocytopenia. A computed tomography (CT) chest revealed bilateral pulmonary emboli, mild hepatic steatosis, gastric varices, and splenomegaly. He was admitted to the hospital for initiation of anticoagulation under medical supervision to assess tolerance given his chronic thrombocytopenia. On arrival, vitals were normal and labs were significant for a chronic thrombocytopenia, elevated ALP and GGT levels. A lower extremity duplex revealed an extensive thrombus within the entire left femoral/popliteal veins. An abdominal ultrasound was negative for a portal vein thrombosis. Hepatitis panel, ceruloplasmin, lupus anticoagulant, and JAK-2 were normal. Anti-neutrophilic, anti-smooth muscle, anti-mitochondrial, and antiphospholipid antibodies were negative. AAT level was low at 33mg/dL and genotype ZZ with liver biopsy further confirming a diagnosis of AAT deficiency (Figure).
Discussion: The diagnosis of AAT deficiency is generally made after the identification of COPD or liver disease in a young adult or after the deficiency has been diagnosed in a family member. However, some patients may present with a secondary diagnosis as their presenting symptom which should prompt the identification of a unifying disease state. Our patient likely had thrombocytopenia secondary to his undiagnosed cirrhosis. Severe AAT deficiency in those homozygous for ZZ allele has been shown to cause an increased risk of thromboembolism compared to the general population. In the appropriate cohort of patients with the constellation of liver and/or lung disease and unprovoked thromboembolism, AAT deficiency should be part of the workup as the morbidity and prognosis are greatly changed with this diagnosis. Our patient's case was further complicated by thrombocytopenia which makes treatment of his thromboembolism more difficult. There have been no reported cases of AAT deficiency presenting with thrombocytopenia and concomitant thromboembolism. We propose the inclusion of AAT deficiency as part of the workup for such patients as the outcomes of the diagnosis are vast.



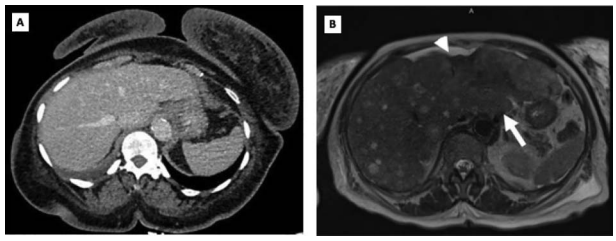
[3182] **Figure 1.** CT Abdomen, axial view: Decreased density of liver relative to the spleen is suggestive of hepatic steatosis. Splenomegaly present.

S3183

The Great Imitator: A Case of Pseudocirrhosis in Metastatic Breast Cancer

Amanda Eisinger, DO, Vishnu Kumar, MD, Pujitha Kudaravalli, MBBS, Kathleen L. Donovan, MD, Ganesh Aswath, MD.
SUNY Upstate Medical University, Syracuse, NY.

Introduction: Pseudocirrhosis (PC) is the radiographic appearance of cirrhosis without corresponding histologic findings, usually in patients with metastatic cancer. We present a case of PC in a patient with metastatic breast cancer, which mimicked decompensated cirrhosis.
Case Description/Methods: A 69-year-old female with stage IV ER+ breast cancer and metastatic disease to the liver and spine presented to the ED for intermittent RUQ pain with associated nausea and decreased oral intake for one week. She had no history of cirrhosis and no risk factors for the development of cirrhosis. Her lab work on presentation is shown in Table 1. Imaging including CT/MRCP showed numerous liver lesions consistent with known metastatic disease, which had decreased in size as compared to the previous study and perihepatic ascites but was negative for biliary pathology. PC was suspected because of nodular liver contour, asymmetric enlargement of the left and caudate lobes, and prominent fissures. Imaging one year prior did not demonstrate evidence of cirrhosis (Figure 1). Given her worsening liver enzymes, bilirubin, and thrombocytopenia, we suspected the patient had PC on top of underlying intrahepatic cholestasis from liver metastasis. A non-targeted liver biopsy was obtained, which showed poorly differentiated adenocarcinoma of the breast with surrounding normal liver parenchyma without any evidence of cirrhosis or advanced fibrosis.
Discussion: PC is a radiological diagnosis in which the morphological changes of the liver closely mimic cirrhosis; without the typical histopathological changes seen on biopsy. It most commonly occurs in patients with metastatic breast cancer, although it has also been reported in other malignancies. Portal hypertension is often seen in patients with PC. The prevalence of PC in patients with metastatic breast cancer is thought to be up to 50%. The pathophysiology leading to the formation of PC is not clearly understood. In chemotherapy-naive patients, it has been associated with a desmoplastic reaction. In patients who received chemotherapy, it has been attributed to tumor necrosis, development of nodular regenerative hyperplasia, or sinusoidal obstruction syndrome (SOS). Identifying this condition early on is crucial as it carries the same complications and clinical progression as cirrhosis. Further studies are required to help us understand this entity better.



[3183] **Figure 1.** A. CT one year prior to presentation demonstrating interval improvement in liver lesions with no evidence of cirrhosis B. Axial T2 image demonstrating extensive hepatic metastases, a nodular liver contour (arrowhead) and caudate lobe hypertrophy (arrow), findings consistent with pseudocirrhosis.

Table 1. Laboratory Results at Presentation

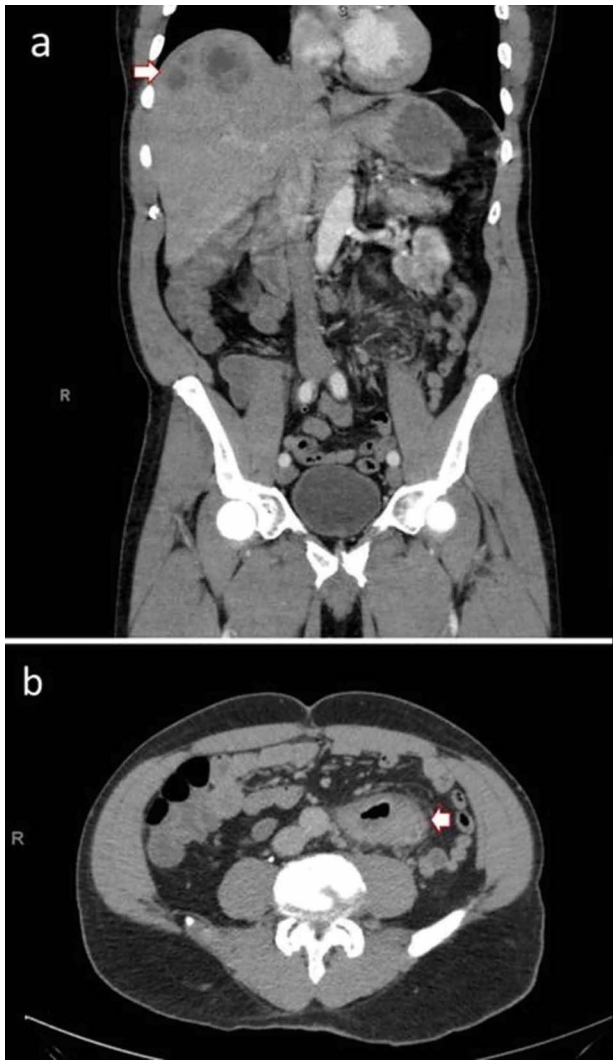
AST (U/L)	280
ALT (U/L)	98
Total Bilirubin (mg/dL)	18.6
Direct Bilirubin (mg/dL)	14.6
INR	1.3
Platelets (/uL)	190
Albumin (g/dL)	3.0
Creatinine (mg/dL)	2.15

S3184

The Ominous Hiccup: Pyogenic Liver Abscesses as a Complication of Acute Sigmoid Diverticulitis

Kwabena O. Adu-Gyamfi, MBChB, Praneeth Kudaravalli, MD, Angela Barnes, BSc, MD, John Erikson L. Yap, MD.
Medical College of Georgia - Augusta University, Augusta, GA.

Introduction: Pyogenic liver abscess (PLA) may occur as a rare and potentially life-threatening complication of acute diverticulitis. We present a case of a man who developed PLA as a complication of acute sigmoid diverticulitis. His major complaint was intractable hiccups and low-grade fever leading to his diagnosis. PLA or diverticulitis may present with atypical symptoms or may even be asymptomatic leading to delays in diagnosis. A high index of suspicion in the right clinical context is key to early diagnosis and timely intervention.
Case Description/Methods: A 66-year-old Caucasian man presented to the emergency department (ED) with a 10-day history of intractable hiccups and low-grade fever. He denied abdominal pain. Five days earlier, he was seen at another ED for same symptoms and was discharged on oral antibiotics for presumed pneumonia but with no clinical improvement. He was febrile to 38.7°C with mild tenderness in lower abdomen and leukocytosis (23.4x 10^9/L) was noted. Rest of examination and basic labs were normal. CT abdomen/pelvis with IV contrast revealed multiloculated hypodense lesions in multiple liver segments (Fig. 1a) measuring up to 5.6cm x 8.3 cm, consistent with liver abscesses as well as inflammatory changes around sigmoid colon (Fig 1b) suggesting acute uncomplicated diverticulitis. Treatment with Broad spectrum antibiotics and percutaneous drainage of largest liver collection led to resolution of all symptoms and he was discharged after 7 days. Both blood and abscess aspirate had no growth. Follow-up 5 months later, he remained asymptomatic. Repeat CT scan and colonoscopy was normal and only notable for diverticulosis.
Discussion: PLA as a complication of acute colonic diverticulitis is rare with unclear incidence. PLA is usually a polymicrobial infection, and the Streptococcus milleri group of bacteria is frequently involved. Both hematogenous and portal venous spread of colonic bacterial to the liver from disruption of gut mucosal barrier has been described. Typical symptoms include fever and abdominal pain. Our patient's major symptom was intractable hiccups which may be related to diaphragmatic irritation from a subcapsular PLA collection. Several days of outpatient antibiotic use as well as IV antibiotics for two days, prior to abscess aspiration likely contributed to negative cultures. With an ageing population and increase in diverticular diseases, increased provider awareness of this uncommon disease association can contribute to improved patient outcomes.



[3184] **Figure 1.** CT Abdomen/Pelvis with IV contrast (coronal view) showing hypodense lesions in right hepatic lobe consistent with liver abscesses (Fig 1a). Sagittal view CT showing proximal sigmoid colon diverticulosis with wall thickening and surrounding inflammatory changes consistent with diverticulitis (Fig 1b).

S3185

The Great Imitator Spares No Organ: A Case of Syphilitic Hepatitis in an Immunocompetent Patient

Paloma Khamly, DO[1], Devika Gandhi, MD[1], Muhammad B. Hammami, MD[2].
[1]*Loma Linda University, Redlands, CA;* [2]*VA Loma Linda Healthcare System, Rancho Cucamonga, CA.*

Introduction: Syphilis, infamously known as the "great imitator," is an infectious disease caused by the spirochete *Treponema pallidum* and can affect many organs. Though uncommon, syphilis can also affect the liver, often manifesting as elevated liver enzymes. Herein we describe the case of a 48-year-old immunocompetent male who was found to have a syphilis-induced acute liver injury.
Case Description/Methods: A 48-year-old male presented with a two-week history of dry cough, fever, anorexia, and sore throat. He initially presented to an outside hospital where he was diagnosed with community-acquired pneumonia and discharged on amoxicillin-clavulanate. Given poor improvement in symptoms, he presented to our emergency department several days later and noted additional symptoms of headache, blurry vision, and rash. On admission, liver enzymes were elevated (Table 1), and a mild maculopapular rash was noted on his trunk and extremities. Clinical findings and liver injury were attributed to a drug injury in the setting of recent amoxicillin-clavulanate use. However, the liver injury persisted, prompting additional evaluation. Acute and chronic liver serology tests were unrevealing except for a positive anti-mitochondrial antibody, 44 units, and anti-nuclear antibodies, titers of 1:160. Syphilis screen with rapid plasma reagin was reactive with a titer of 1:32. Given neurological symptoms, a lumbar puncture was performed and revealed a reactive fluorescent treponemal antibody test absorption test. The patient was diagnosed with secondary syphilis complicated by neurosyphilis and syphilitic hepatitis. The patient completed a two-week course of intravenous penicillin with complete resolution in symptoms. After initiation of penicillin, the patient's liver chemistries downtrended and normalized after treatment, confirming the diagnosis of syphilitic hepatitis.
Discussion: Syphilitic hepatitis is an uncommon manifestation of syphilis. Suspicion should be raised in patients with elevated liver chemistries, in particular cholestatic pattern, who also have manifestations such as maculopapular rash, low grade fever, arthralgias, headache, and changes in vision. This case highlights the importance of maintaining a broad differential and avoiding anchoring bias when approaching elevated liver chemistries.

Table 1. Liver Function Tests

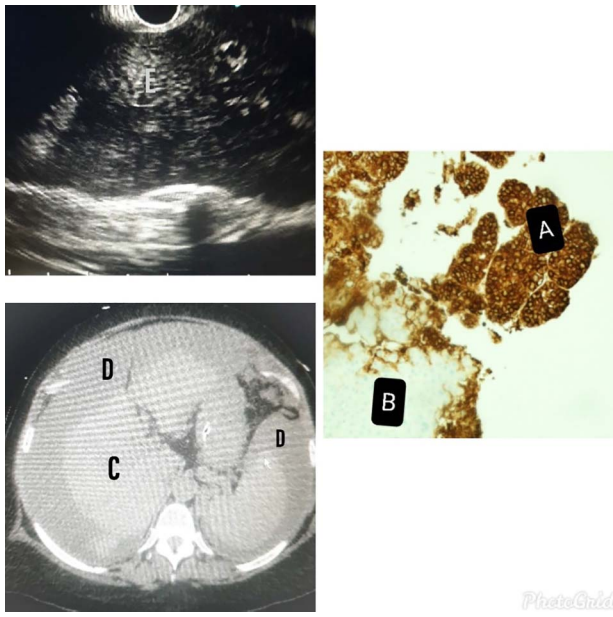
	Baseline	Outside Hospital	Presentation	Week 1	Week 3
Alkaline phosphatase (IU/L)	78	1126	1118	997	437
ALT (IU/L)	14	225	231	224	53
AST (IU/L)	19	130	154	139	37
Total bilirubin (mg/dL)	0.8	1.9	4.3	2.0	0.9

S3186

The Imaging Negative Hepatic Lesions: A Rare Case of Infiltrative Hepatocellular Carcinoma

Onyinye Ugonabo, MD, Joseph Simmons, MD, Saba Altarawneh, MD, Phillip R. Jones, DO, Tejas Joshi, MD, Wesam Frandah, MD.
Marshall University Joan C. Edwards School of Medicine, Huntington, WV.

Introduction: Hepatocellular Carcinoma (HCC) is the 6th most common cancer and the 4th leading cause of cancer-related death worldwide. The infiltrative subtype is the most difficult to diagnose with imaging because of its inherently ill-defined micronodules involving a segment or entire hepatic parenchyma without a distinguishable mass. Prognosis is poor and estimated at a 5-year survival rate of < 20%. Diagnosis of HCC requires a sensitive imaging test. In situations where imaging is not able to identify a mass, EUS-FNA has been shown to have a higher detection rate.
Case Description/Methods: A 61-year-old female with a past medical history of HCV cirrhosis with sustained virology response (SVR), presented with abdominal pain and worsening lower extremity edema. Remarkable examination findings include a distended abdomen and bilateral pitting lower extremity edema. Significant work up showed platelet of 109k/cmm (150-440), AST of 159 unit/l (15-37), total bilirubin of 1.2mg/dl (0.2-1.0), AFP of >20,000ng/ml (0.5-8.0), peritoneal fluid cytology was negative. A right upper quadrant ultrasound, CT, and MRI showed no hepatic mass, Fig CD. With markedly elevated AFP and a high suspicion for HCC, the patient had EGD-EUS guided fine-needle aspiration showing multiple infiltrative hepatic lesions, Fig E. The biopsy report showed malignant cells positive for AFP with cells reactive for glypican-3 and negative for Hep-Par1 (Fig A), supporting the diagnosis of HCC. The patient was referred to oncology and a month later, she died.
Discussion: SVR is associated with decreased risk of HCC, however, with a cirrhotic liver, patients still have an absolute risk of developing HCC. Initial diagnosis of HCC can be obtained non-invasively using abdominal ultrasound, CT, MRI, and EUS-FNA. Abdominal ultrasound is the best imaging modality recommended for HCC surveillance because it is readily available. However, its sensitivity for detecting early HCC is about 47%. AFP cut-off level of > 20ng/ml has a sensitivity of about 60% with low specificity. A level of > 400ng/ml is diagnostic for HCC with a specificity of almost 100%. Incremental changes in AFP are associated with an increased mortality rate. The median survival rate of infiltrative HCC with AFP of > 400 is estimated to be about 5 months. EUS is superior to CT in detecting small hepatic lesions, with a sensitivity of 100% compared to 71% of CT. We recommend that EUS be considered an integral modality while investigating the diagnosis of infiltrative HCC.



[3186] **Figure 1.** Fig A showing Hepatocellular carcinoma, 200x. Glypican-3 immunostain shows strong, diffuse staining in HCC (A). Background non-neoplastic liver is negative (B). Fig C is the cirrhotic liver with no identifiable mass. Fig D shows fluid around the liver and spleen. Fig E is the EUS showing multiple infiltrative hepatic lesions.

S3187

Toxic Hepatitis During Self-Medication With Conjugated Linoleic Acid

Rahul R. Thakkar, MD, Keerthana Pakanati, MD, Bryce Bushe, MD, Erik Rahimi, MD.
Baylor Scott & White Round Rock, Round Rock, TX.

Introduction: Despite the growing prevalence of herbal and dietary supplements in patient care, there is limited knowledge regarding their safety and efficacy due to the great variability in their use and subsequent lack of randomized controlled trials; furthermore, there is a general reluctance in discussing their use in the clinical setting, increasing the risk of adverse effects.[1] Conjugated linoleic acid (CLA) is a polyunsaturated omega-6 fatty acid found in ruminant milk fat and meat. A synthetic form of CLA, produced from certain vegetable oils, is found in dietary supplements that are often used for weight loss.[2] CLA-induced hepatotoxicity is a rare but adverse effect that warrants greater awareness, particularly given the widespread availability and popularity of CLA supplements. In current literature, this is only the fourth known case of CLA-induced hepatotoxicity, the second in the United States.[3-5]
Case Description/Methods: A 77-year-old female with a history of calculous cholecystitis s/p cholecystectomy presented to ED with a one-day history of epigastric pain with associated nausea and dry-heaving. She frustratedly noted similar episodes over the past few months despite her healthy lifestyle. In the ED, her vital signs were stable and her physical exam was benign. Serum lipase was normal but LFTs were elevated with total bilirubin 1.5, ALP 398, AST 1003, AST 409. CT imaging did not demonstrate biliary dilatation or filling defects. Gastroenterology was consulted for further evaluation of her elevated transaminases, the pattern of which was suggestive of viral hepatitis, drug-induced liver injury (DILI), or ischemic hepatitis. Investigation into these etiologies – including a viral hepatitis panel, serum acetaminophen level, anti-nuclear antibody titer, and a careful medication reconciliation – did not reveal any inciting factors. Fortunately, solely with supportive care, her symptoms gradually improved along with her LFTs. On further interview, the patient revealed that she had recently started consuming CLA-safflower oil to lose weight. She was instructed to stop its use and follow up closely with her PCP, during which her LFTs normalized.
Discussion: Synthetic conjugated linoleic acid – commonly used in dietary supplements advertised for weight loss – may rarely lead to toxic hepatitis. With a significantly increasing proportion of DILI secondary to herbal and dietary supplements, inquiring about their use may be imperative in stopping offending agents and preventing long-term liver injury.

S3188

The Tale of an Ancient Herb: A Stress Reliever or a Liver Stressor

Minh Tran, MD, Jaison John, MD, Kian Abdul-Baki, DO, Kumar Pallav, MD.
UTMB, Galveston, TX.

Introduction: Ashwagandha is found in several herbal products today. However, there is a paucity of literature regarding its safety. Our case serves to remind physicians about the potential liver toxicity of Ashwagandha and to illustrate how early recognition of drug-induced liver injury (DILI) and discontinuation of the offending substance can be lifesaving.
Case Description/Methods: A 58-year-old man with a remote history of hepatitis B and C presented with nausea, vomiting, and right upper quadrant abdominal pain of one day duration. He reported using an herbal supplement called "Primal Male," which contained Ashwagandha as a main ingredient, over the past two weeks. He was normotensive, tachycardic up to 120 beats per minute (bpm), and febrile. The initial laboratory studies were notable for elevated AST level of 954 U/L, ALT level of 860 U/L, and a normal alkaline phosphatase level of 101 U/L. He had a normal total bilirubin level and an international normalized ratio of 1.3. His complete blood count and renal function were unremarkable. His creatinine kinase (CK) level was also within the normal range. His drug test, alcohol level, and acetaminophen level were unremarkable. The viral panel did not suggest active viral infections. His autoimmune panel was negative. An abdominal CT scan did not reveal any acute abnormality. In the following days, his symptoms worsened, and he became more encephalopathic. His AST, ALT, total bilirubin, and INR levels continued to rise. He developed hypotension and received intravenous fluids and midodrine. He also completed a course of N-acetylcysteine treatment. On the fifth day of admission, he underwent a transjugular liver biopsy. Liver histology showed active lymphocytic hepatitis with moderate inflammation (primarily lymphocytes, abundant neutrophils, and occasionally eosinophils) around the portal tract. On the sixth day of hospitalization, he reported decreasing pain and was more hemodynamically stable. His liver enzymes also showed improvement. The cause of acute liver injury was suspected to be drug-induced liver injury from Ashwagandha use. He was counseled to avoid the use of the herbal supplement and other hepatotoxic products. One month later, his liver enzymes were in normal range.
Discussion: DILI is a less common form of liver injury but is a leading cause of acute liver failure in the United States. Ashwagandha has been reported in a few cases of DILI. It is important to have an early suspicion of the offending agent and avoid further substance exposure.

S3189

Too Much Turmeric? A Case of Drug-Induced Liver Injury

Juan Garcia Lopez, BA[1], Tajah Kee, BS[1], Zachary Marwil, MD[2].
[1]Endoscopy Center of Long Island/Northwell Health, Garden City, NY; [2]Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, Woodbury, NY.

Introduction: Drug induced liver injury (DILI), the most common reason for drug withdrawal, accounts for approximately 10% of all cases of acute hepatitis. Herbal and dietary supplements (HDS) have been indicated in approximately 15 to 20% of all reported DILI cases. Turmeric, a very popular HDS, is an herb derived from a Southeast Asian plant, Curcuma longa. It has been widely used for its anti-inflammatory properties to treat many conditions and is considered safe at appropriate doses
Case Description/Methods: A 65 year-old female was referred for jaundice associated with fatigue and pruritis. Her medical history was significant for hyperlipidemia and arthritis. Her medications included aspirin 81 mg, rosuvastatin 5 mg, niacinamide 500 mg, and various vitamins and supplements, including 500 mg per day of Turmeric. She denied using alcohol, IV drugs, or acetaminophen. Physical exam was unremarkable, except for diffuse jaundice and scleral icterus. Blood work revealed an AST of 2,460 U/L, ALT of 2,464 U/L, total bilirubin of 14.9 mg/dL, and alkaline phosphatase of 250 U/L. An acute viral hepatitis panel and CBC were within normal limits. Further labs were unremarkable, including negative serologies for autoimmune hepatitis. An MRI of her abdomen revealed cholelithiasis and periportal edema, with no evidence of cholecystitis, choledocholithiasis, or biliary ductal dilatation. At onset of symptoms, the patient had stopped all her medications and supplements. Within 3 months, the jaundice had resolved, and liver enzymes returned to normal. However, 2 months later the patient returned with diffuse, yet milder jaundice. Labs again noted markedly elevated aminotransferases and total bilirubin. Other liver disease labs, including autoimmune serologies, remained negative. She reported that while she remained off her statin, she had restarted Turmeric 3 weeks prior. A liver biopsy revealed pronounced interface chronic hepatitis with lymphocytes and plasma cells, extending into the lobule with Kupffer cell activation. The Turmeric was again discontinued. The jaundice subsequently resolved, and liver enzymes normalized within 6 weeks.
Discussion: This is a case of Turmeric induced liver injury with recurrence after re-challenge. While Turmeric is considered a safe HDS at appropriate doses, it must be considered as a primary cause of DILI and acute hepatitis. While most patients tolerate this HDS without incident, more research is needed to help identify individuals who are potentially at risk for adverse events.

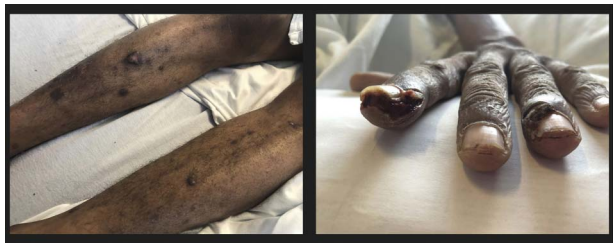
S3190

The Great Masquerader: A Case of Syphilitic Hepatitis

Sanya Goswami, MD, Mohammad Almeqdadi, MD, Qi Yu, MD.
NYC H+H Kings County/SUNY Downstate Medical Center, Brookyln, NY.

Introduction: Syphilis, colloquially termed "the Great Imitator," is a sexually transmitted infection caused by the spirochete treponema pallidum. Syphilis occurs in multiple stages. Tertiary syphilis occurs years after inoculation and presents with chronic granulomas, cardiovascular, and neurologic manifestations. Syphilitic hepatitis is a rare and often missed diagnosis in patients who have syphilis and who are often are co-infected with HIV or hepatitis B.
Case Description/Methods: The diagnosis is made by both clinical presentations and positive serological markers, characteristic transaminitis, with no other alternative cause of hepatobiliary injury identified. Here, we highlight a case of an undomiciled 40-year-old male with HIV (on Genvoya) and chronic hepatitis B, who presented with tachycardia to 107. Physical examination was remarkable for a diffusely spread rash with erythema on the bilateral lower extremities, a small purulent boil on the right shin, left index finger and dry, itchy, distinct raised purple lesions on the scalp, eyebrows, upper and lower extremities, back, and shoulders. Laboratory findings were remarkable for a severely elevated alkaline phosphatase, mild transaminitis, and elevated total bilirubin, with negative pertinent imaging including a right upper quadrant sonogram, CT scan of the abdomen, and MRCP. Extensive additional work-up revealed elevated VDRL titers. Both a skin and liver biopsy were completed. Immunohistochemical stain of shave biopsy of a verrucous plaque on the scalp vertex and an excoriated papule of the left neck highlighted numerous spirochetes. Liver biopsy revealed hepatocellular necrosis with moderate mixed inflammation involving the portal tract and periportal hepatocytes, with immunohistochemical marker for spirochetes negative for organisms. However, given the patient's clinical history of secondary syphilis involving the skin and lab abnormalities, it raised the differential diagnosis of syphilitic hepatitis (Figure).

Discussion: With our case report and extensive research of the literature, we hope to shed light on the importance of certain infections, even those in decline, on the differential. Additionally, contemplation of coexisting infections often helps to contribute to diagnoses not otherwise common. Because of how rarely seen syphilitic hepatitis is, it is often a missed diagnosis, but we are hopeful our case report helps physicians with patients with similar presentations, especially patients with otherwise unexplained high alkaline phosphatase levels.



[3190] **Figure 1.** Skin Findings.

S3191

TNF Inhibitor-Induced Liver Injury: Clinical Presentations and Outcomes

Patrick J. Tempera, DO[1], Julian Remouns, DO[1], Cassidy Alexandre, MD[2].
[1]Albany Medical Center, Albany, NY; [2]St. Peter's Hospital, Albany, NY.

Introduction: Inflammatory bowel disease (IBD) is commonly treated with a sub-set of medications that inhibit tumor necrosis factor (TNF), an integral component during the pro-inflammatory phase. However, like all medications, TNF inhibitors are not without adverse side effects, one of them being drug induced liver injury (DILI). Although commonly described in literature, clinical presentation is often not encountered or recognized. We describe the cases of two individuals with IBD and the development of DILI secondary to TNF inhibition.
Case Description/Methods: Our first case describes a 40-year-old female with ulcerative colitis who was started on adalimumab. Within 8 months of starting, aminotransferases were found to be elevated. Abdominal ultrasound showed hepatic steatosis, Fibroscan CAP 100, E Score 12kPa, and liver biopsy was consistent with autoimmune pattern liver injury with focal periportal fibrosis. Her only symptom was constipation. Adalimumab was discontinued and a short course of prednisone was started with improvement in aminotransferase elevations. Our second case is a 25-year-old female with Crohn's Disease who was started on infliximab. Within 6 months, aminotransferases were found to be elevated. Liver biopsy showed portal-based chronic hepatitis with mild activity suspicious for DILI. Autoimmune hepatitis workup including ANA (640), A-SMA (negative), AMA (negative), Anti-dsDNA (60-borderline), IgG (1713) was not convincing but autoimmune hepatitis due to anti-TNF activity could not be excluded. At this time, infliximab was discontinued, and the patient began to have an improvement in clinical presentation.
Discussion: All the TNF inhibitors currently marketed have been associated with DILI. However, the optimal management of liver injury related to TNF inhibitors is still a matter of debate. Some practitioners recommend the discontinuation of treatment in the case of elevated aminotransferase levels or the occurrence of jaundice. Others have recommended the continuation TNF inhibitor in the setting of similar clinical and laboratory findings with hopeful resolution of liver injury. Too often, the clinical signs or evidence of DILI is occult and can go unnoticed. Further research into best practice outcomes when IBD patients are taking such medications is needed. Additionally, better guidelines for the use and management of TNF inhibitors in IBD patients is imperative to minimize the risk of unchecked drug induced liver injury.

S3192

There Is No Such Thing as a Cute Liver: AFL vs HELLP in a Pregnant Patient

Masuma Syed, MD, Xin Zheng, DO, Mina Rakoski, MD.
Loma Linda University Medical Center, Loma Linda, CA.

Introduction: The etiology of liver disease in pregnancy can be challenging to diagnose as it may be related to the pregnancy or completely independent. Several disorders can cause elevated LFTs during pregnancy including the syndrome of hemolysis, elevated liver tests, and low platelets (HELLP) and acute fatty liver of pregnancy (AFL). There is often difficulty differentiating between HELLP and AFL, both of which are life-threatening complications of pregnancy. This vignette is an example of the complexity involved in differentiating between these pregnancy-associated liver diseases.
Case Description/Methods: A 34 yo G4P3104 female initially presented with nausea and vomiting. She was hypertensive with laboratory workup significant for elevated LFTs with total bilirubin 5.7, ALP 450, AST 776, ALT 683, along with AKI. She was initially suspected to have AFL at admission, however due to progressively worsening blood pressures and development of thrombocytopenia (platelets 13), HELLP syndrome was suspected so she was placed on a nicardipine drip and underwent an emergent C-section. Liver and renal function continued to decline postpartum so patient briefly required CRRT with eventual transition to HD. Due to rising INR and LFTs in the setting of worsened lethargy, she was diagnosed with acute liver failure and urgently listed for liver transplant status 1a. Comprehensive acute liver failure workup included negative AMA, ANA, ASMA, hepatitis serologies, alpha-1 antitrypsin level and phenotype and ceruloplasmin. After requiring mechanical ventilation for several days, patient was extubated due to improved GCS and transferred out of ICU. She remained listed for liver transplant, however, her LFTs started to improve within days of delivery. She was removed from the liver transplant list nine days after presentation.
Discussion: This case was a difficult diagnosis as her presenting symptoms of nausea and vomiting with elevated LFTs created a broad differential. Given the progressive hypertension and thrombocytopenia, she was diagnosed with HELLP syndrome. Despite an initially unclear diagnosis, the patient received the appropriate treatment – prompt delivery, as that remains the treatment for both diagnoses. After an initial decline in liver and kidney function immediately postpartum, symptoms rapidly improved with full resolution of elevated liver enzymes and coagulopathy. It is particularly important for patients to be aware of their diagnosis as these clinical disorders can reoccur in subsequent pregnancies.

S3193

The Diagnostic Challenge of Wilson's Disease in the Presence of Valproic Acid

Ana R. Vilela, MD, Alejandra Vargas, MD, Buyng Soo Yoo, MD, Alsiddig Elmahdi, MS.
Eastern Virginia Medical School, Norfolk, VA.

Introduction: Wilson's disease (WD) is an inherited disorder of copper metabolism caused by pathological copper accumulation in different organs, particularly the liver, brain, and cornea. Toxic copper accumulation contributes to a variety of clinical conditions that may mimic other diseases or, as was observed here, might have been mistaken for valproic acid side effects.
Case Description/Methods: A 23-year-old male with autism spectrum disorder, and generalized tonic-clonic seizures presented to the emergency department with lethargy and confusion. Physical examination revealed scleral icterus, epigastric tenderness, psychomotor retardation and fine postural tremor. Initial labs showed hemoglobin 7.3 g/dL, MCV 102.7 fl, platelet count 87 K/uL, leukocytes 2.4 K/uL, total bilirubin 4.0 mg/dL, direct bilirubin 1.7 mg/dL, AST 99 IU/L, ALT 57 IU/L, and valproic acid level was 64 (normal 50-100 mcg/ml). Valproic acid was discontinued, as adverse effects were believed to be associated with it. Patient returned with behavioral changes, insomnia, tremors and jaundice two weeks later. Labs revealed persistent pancytopenia, bilirubinemia, and transaminitis. Ammonia level was 169 and INR was 1.67. Computed tomography (CT) demonstrated splenomegaly and portal hypertension. Patient was admitted for decompensated cirrhosis and hepatic encephalopathy. Additional investigations were negative for viral and autoimmune hepatitis. Iron level and saturation level were normal. Serum ceruloplasmin level was reduced to 12.2 mg/dL (normal 18 - 50 mg/dL) and 24-hours urinary copper level was elevated to 37 μg/24 hrs (normal 3 - 35 ug/24 hr). In light of the raised suspicion for Wilson's disease, a slit lamp test was performed, and despite the absence of Kayser-Fleischer rings, a liver biopsy was performed. The latter revealed end-stage fibrosis with nodular formation, vacuolated nuclei, hepatic necrosis, and positive Prussian blue staining. After sending the samples for an exchangeable copper assay, the diagnosis of WD was confirmed.
Discussion: Wilson's disease is an inherited copper overload disorder, which can be involved with a wide range of signs, symptoms, and tissue damage making the diagnostic process often difficult. As in this case, valproic acid's side effects closely resemble the clinical manifestations of WD including neurological, hepatic, and hematologic symptoms. Nevertheless, maintaining a high suspicion is crucial to early diagnosis and treatment.

S3194

The Search for Chest Pain and Shortness of Breath: Drug-Induced Liver Injury

Patrick J. Tempera, DO[1], Joo Hye Park, MD[1], Lavinia Salama, PharmD[2], Asha Pandu, MD[1].
[1]Albany Medical Center, Albany, NY; [2]Albany College of Pharmacy and Health Sciences, Albany, NY.

Introduction: Patients with drug-induced liver injury (DILI) are often asymptomatic and have undetected increases in their liver function tests (LFTs). Symptoms of DILI, if present, are vague, mimic a host of diseases, and are attributable to other etiologies before DILI is considered. In our case, we present a series of overlapping and vague symptoms that ultimately were attributed to DILI.
Case Description/Methods: We describe the case of a 43-year-old male who presented to his cardiologist office with complaints of chest pain followed by shortness of breath (SOB). The patient has a past history for coronary artery disease and two kidney transplants on tacrolimus. Due to the patient's cardiac history, cardiac catheterization was performed which did not show any signs of re-stenosis or obstructive disease. He underwent evaluation for possible pulmonary cause as well including pulmonary function testing and diagnostic imaging which were normal. Laboratories drawn showed elevated liver enzymes (ALT 154, AST 56) and gamma-glutamyl transferase (GGT) of 382 with a normal total bilirubin. Abdominal ultrasound showed no evidence of biliary dilation, choledocholithiasis, or structural abnormality. On further review, patient had recently started a second immunosuppressant, mycophenolate, around the time he started to experience these unexplained symptoms. Additionally, on thorough physical exam, it was noted that the patient's subjectively stated chest pain was isolated to the epigastrium on palpation. Liver enzymes were continually monitored with eventual return to normal ranges with resolution of reported pain and SOB without having to hold any immunosuppressant agents.
Discussion: This case illustrates the unusual clinical presentation of hepatic injury secondary to an unusual cause of DILI. It was surmised that the mild transient elevation in liver enzymes and GGT was due to the initiation of mycophenolate. Mycophenolate is known to cause liver injury in rare cases and in the setting of inflamed liver tissue irritating Glisson's capsule, abdominal tenderness can develop. It is important to perform an encompassing physical examination to correlate patients' subjective complaints with objective findings. For patients taking hepatotoxic medications, widening one's differential diagnosis to include DILI can ensure minimalization of liver damage.

S3195

The Liver and Cryoglobulinemia: A Relationship Beyond Hepatitis C

Lakshmi P. Pappoppula, MD[1], Khalid Ahmed, MD[1], Abdul Ahad Ehsan Sheikh, MD[1], Beshir Saeed, MD[1], Roop Sunil Reddy, MD[2].
[1]The Wright Center for GME, Scranton, PA; [2]Geisinger Community Medical Center, Scranton, PA.

Introduction: Cryoglobulinemia is a small and medium vessel vasculitis with varied clinical manifestations. The most common etiology is hepatitis C virus. Other hepatotropic viruses, including hepatitis A, have been rarely associated with very few cases reported. Here we present a unique case of cryoglobulinemia associated with hepatitis A.
Case Description/Methods: A 58-year-old female presented to the Emergency Department (ED) with bloating, abdominal pain, and fullness for about a week. She has a past medical history of diabetes mellitus type II, hypothyroidism, and hypertension. She was afebrile and hemodynamically stable in the ED with Initial lab workup showing AST/ALT of 5228/4792, total bilirubin of 7.9 with a direct bilirubin of 6.1. Further workup showed positivity for hepatitis A, including IgM and positive type II cryoglobulins. The rest of the autoimmune workup was negative for rheumatoid factor, ANA, SS/Ro, SS/La, anti-smooth muscle antibody, and anti-mitochondrial antibody. She also had acute renal failure requiring hemodialysis. Management included supportive therapy with significant improvement in her symptoms.
Discussion: Cryoglobulinemia is a condition where abnormal immunoglobulins precipitate in serum at temperatures below 37C. They can deposit in blood vessels and cause obstruction or vasculitis, with involvement of various organs. Type I cryoglobulinemia is usually associated with hematologic malignancies whereas mixed cryoglobulinemia (type II, III) is associated with hepatitis C and rarely seen with hepatitis A. Very few cases showing an interrelation between hepatitis A and cryoglobulinemia have been reported in the literature making this a rare association. Hence, more studies need to be done to investigate extrahepatic features of hepatitis A.

S3196

Wilson Disease in a U.S. Navy Pilot: Delayed Diagnosis With Drastic Career Implications

Zachary Johnston, MD[1], Ryan Kwok, MD[2].
[1]Walter Reed National Military Medical Center, Bethesda, MD; [2]Madigan Army Medical Center, Auburn, WA.

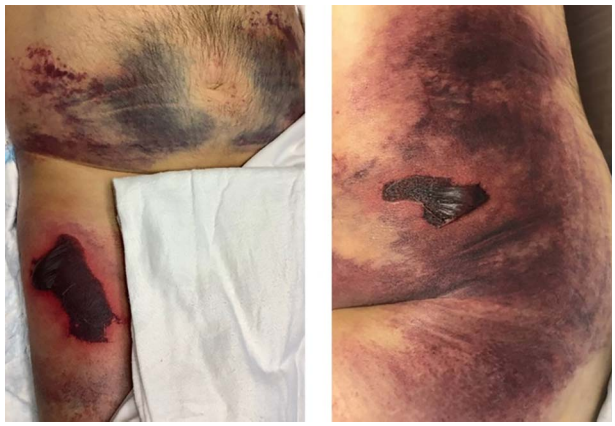
Introduction: Wilson disease is an autosomal recessive genetic disorder of the intracellular copper transport ATP7B leading to impaired copper excretion and subsequent accumulation in the liver, brain and cornea. Index symptoms vary widely and include neurologic and psychiatric abnormalities. The wide array of subtle symptoms often lead to significant delays in diagnosis and thus prognostic implications for patients diagnosed with this rare disease. In the Armed Forces, the diagnosis and clinical sequelae can have drastic career implications especially when neurologic sequelae are resistant to treatment.
Case Description/Methods: A 28 year old US Navy pilot male presented to behavioral health clinic with complaint of depressed mood. He was diagnosed with major depressive disorder which was resistant to traditional treatment for over a year. He developed fasciculations of his neck and arms which was associated with initiation of an SSRI. When he had difficulty landing an aircraft due these progressive symptoms, he was deemed incapable of flight. He later required admission for suicidal ideations. He had several concerning incidents occur including driving over a median and through several red lights. Neurology was consulted and examination showed hyperkinetic movements of his extremities with head and voice tremors. Lab work revealed ALT 52 and AST 48 and later, a ceruloplasmin of 6.7. A slit lamp exam was performed revealing Kayser-Fleischer rings. MRI brain showed abnormal T2 signal within the basal ganglia and midbrain consistent with copper deposition. MRI liver was consistent with cirrhosis. A liver biopsy confirmed the diagnosis of Wilson disease with cirrhosis. He was started on trientene and zinc for chelation and his mood markedly improved. However, his neurologic abnormalities have persisted despite ongoing therapy for over 4 years, permanently ending his piloting career.
Discussion: Our case highlights the importance of careful clinical consideration in patients presenting with psychiatric complaints. We aim to highlight this patient's response to therapy, and lack thereof, as the range of patient response can be quite broad. The majority of patient's have significant or even total resolution of symptoms. Our patient had significant improvement in depression after chelation therapy but his neurologic symptoms did not recover. Regrettably, the patient's Navy pilot career was brought to an early close given the severity of his symptoms and lack of response and thus, inability to perform as an aviator.

S3197

Warfarin-Induced Skin Necrosis Following Routine Endoscopy in a Patient With Cirrhosis

Franklyn Wallace, MD, Jennifer Kleinman Sween, MD.
Mayo Clinic Rochester, Rochester, MN.

Introduction: Warfarin-induced skin necrosis (WISN) is rare, and most cases are caused by inherited deficiencies of protein C. Acquired protein C deficiency has been associated with advanced liver disease, ulcerative colitis (UC), and malignancies. Due to its rarity, clinicians must have a high index of suspicion for WISN to avoid delays in diagnosis or treatment.
Case Description/Methods: A 67-year-old male with UC, cirrhosis due to primary biliary cholangitis, and prior pulmonary embolism on warfarin underwent surveillance EGD and colonoscopy. Warfarin was held for five days prior to the procedures without bridging. EGD was unremarkable and colonoscopy revealed active UC. He resumed warfarin therapy the next day. Two days later, he developed significant tenderness and bruising on his abdomen, flank, and outer thighs. After evaluation by multiple providers, his pain was attributed to central sensitization, and he was prescribed gabapentin. Over the next several days, he developed extensive painful retiform purpura and expanding areas of superficial necrosis (Figure 1). He was admitted to the hospital for multidisciplinary evaluation. Extensive workup assessed potential causes, such as WISN, sepsis, vasculitis, disseminated intravascular coagulation (DIC), thrombotic thrombocytopenic purpura (TTP), or cryoglobulinemia. Given the characteristic appearance and temporal association with restarting warfarin, WISN was thought to be the most likely etiology. Protein C activity was low at 22% (normal 70-150%). Genetic testing for inherited protein C deficiency was negative, confirming an acquired deficiency. For management of WISN, warfarin was discontinued, heparin was initiated, and protein C was repleted with fresh frozen plasma (FFP). He was discharged on warfarin after bridging with heparin. He later had recurrence of his necrotic rash, and an excisional biopsy confirmed WISN. Warfarin was then discontinued in favor of lifelong enoxaparin.
Discussion: Our case highlights a rare but devastating complication of warfarin use. In WISN, protein C is depleted prior to other vitamin K-dependent factors, leading to a transient hypercoagulable state, microvascular occlusion, and tissue necrosis. Despite our patient's risk factors (liver disease and active UC) and characteristic presentation, diagnosis of WISN was delayed. Prompt recognition and treatment (discontinuation of warfarin, IV heparin, vitamin K and FFP) is crucial for tissue preservation and avoidance of morbid surgical interventions.



[3197] **Figure 1.** Images of the patient's abdomen and right thigh (left) and left hip (right). The skin is extensively involved with painful retiform purpura and areas of superficial necrosis with associated hemorrhagic bullae.

S3198

Was It Always in the Genes? ABCB4 and Its Variable Presentations

Andre Khazak, DO[1], Gres Karim, MD[1], Carolina Villarroel, MD[1], Silpa Yarra, MD[1], Ilan Weisberg, MD, MSc[2].
[1]Mount Sinai Beth Israel, New York, NY; [2]New York-Presbyterian Brooklyn Methodist Hospital, Brooklyn, NY.

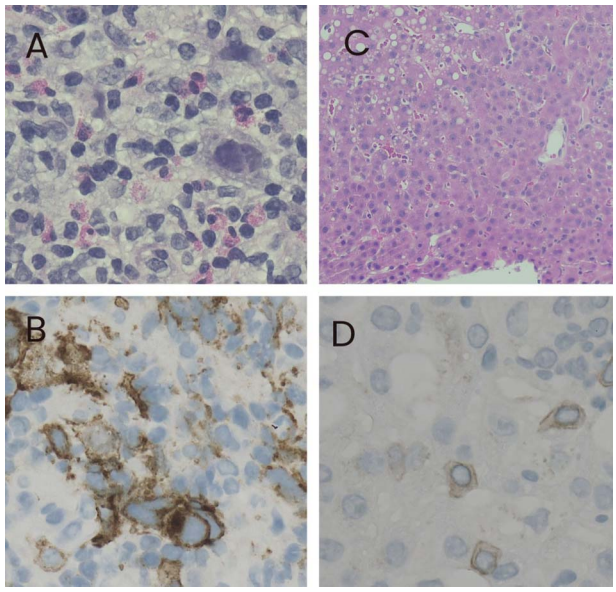
Introduction: Progressive familial intrahepatic cholestasis (PFIC), low phospholipid-associated cholestasis (LPAC), and autosomal dominant intrahepatic cholestasis of pregnancy (ICP) are rare genetic disorders that should be considered in patients with elevated alkaline phosphatase and unrevealing primary work ups. We present a case of a 36-year-old female with a history of ICP who presented with elevated ALP, eventually found to have a nonspecific variant in the ABCB4 gene, concerning for Type 3 PFIC.
Case Description/Methods: A 36-year-old female with a history of ICP treated with Ursodiol, current oral contraceptive use (OCP), and remote cholecystectomy presented to a gastroenterologist for asymptomatic ALP elevation. Initial labs revealed ALP 279 U/L, bile acids 9.9 μmol/L, and gamma-glutamyl transferase (GGT) 283U/L. Magnetic resonance cholangiopancreatography (MRCP) was unrevealing. Given no signs of hepatic dysfunction, portal hypertension, or steatosis on fibroscan, liver biopsy was obtained, which was unremarkable. Due to persistent elevation in ALP without a clear etiology and history of ICP, genetic testing was pursued. Genetic testing revealed a heterozygous variant of uncertain significance in the ABCB4 gene that is associated with PFIC type 3. OCPs were discontinued due to an association of ICP and OCP induced cholestasis. Ursodiol was restarted given high rates of recurrent cholelithiasis in patients with PFIC.
Discussion: Type 3 PFIC, LPAC, and ICP are rare genetic disorders linked to unique mutations in the ABCB4 gene responsible for ALP elevation. The ABCB4 gene may manifest later in life as cholestasis, jaundice, or pruritus. Mutations in the ABCB4 gene can cause a reduction in biliary phospholipid concentration, leading to sludge, cholesterol stones, and microlithiasis. Type 3 PFIC has a unique presentation associated with elevated GGT, unseen in type 1 or 2. LPAC should be considered in patients presenting with cholelithiasis before the age of 40, recurrent cholithialsis after cholecystectomy, ICP, and family history of cholelithiasis. The variant of uncertain significance in our patient's ABCB4 gene appeared to be a unifying diagnostic factor given her history of ICP, cholelithaisis at age 18 years requiring cholecystectomy, and persistent new elevation in ALP. She ultimately met criteria for Type 3 PFIC, LPAC and ICP. Given the rapidly evolving nature of medical genetics, reclassification and evaluation of genetic mutations requires follow up.

S3199

Vanishing Bile Duct Syndrome as a Presentation of Classical Hodgkin's Lymphoma

Ifrah Fatima, MD, Mariam Haji, MD, Wael T. Mohamed, MD, Valerica Mateescu, MD, Sheshadri Madhusudhana, MD, Anuj Shrestha, MD.
University of Missouri-Kansas City, Kansas City, MO.

Introduction: Vanishing bile duct syndrome (VBDS) is an uncommon, acquired form of cholestatic liver disease characterized by hepatic ductopenia and cholestasis. We present a case of VBDS as the initial manifestation of classical Hodgkin lymphoma (CHL), with normalization of liver enzymes after treatment with standard-dose chemotherapy- Doxorubicin, Bleomycin, Vinblastine, and Dacarbazine (ABVD).
Case Description/Methods: A 26-year-old female presented with nausea, weight loss, right-sided neck mass, pruritus, and yellowing of eyes and skin. She was on Celecoxib and Prednisone 40mg (5 days), for back pain associated with scoliosis. On physical exam, she had jaundice and cervical lymphadenopathy. Labs were significant for a total bilirubin of 22 mg/dL and elevated aspartate transaminase (AST) 91 U/L, alanine transaminase (ALT) 108 U/L, alkaline phosphatase 662 U/L, INR 1.3. Ultrasound abdomen showed mild hepatosplenomegaly but no bile duct dilatation. Hepatitis panel, EBV, and HIV were negative. CA 19-9 and AFP were normal. MRCP ruled out obstructive process/mass. Liver biopsy revealed prominent cholestasis and Kupffer cell hyperplasia with focal perivenular hepatocyte dropout and loss of bile ducts, greater than 50%- consistent with idiopathic cholestasis with ductopenia, also known as VBDS. PET/CT of neck showed extensive supra-diaphragmatic lymphadenopathy, large nodal mass in the neck, and superior mediastinum with associated mass effect. Biopsy of right cervical lymph node showed CHL, nodular sclerosis type. Immunohistochemical staining was positive for CD30, CD15, and PAX5 (weak) supporting the diagnosis. She was started on Gemcitabine, Cyclophosphamide, and Dexamethasone as liver-sparing agents. However, liver enzymes remained elevated. She was started on standard chemotherapy for CHL – ABVD along with Rituximab (ABVD-R) with dose-adjusted vinblastine for cycles 1-2. She then completed cycles 3 to 6 of ABVD. Post C4, PET/CT showed a complete metabolic response. Liver enzymes normalized progressively with chemotherapy (Figure).
Discussion: Idiopathic cholestasis with ductopenia can be seen as a paraneoplastic phenomenon in CHL even in the absence of direct hepatic involvement by lymphoma. The primary goal of treatment for hepatic injury was treatment of CHL as able with her liver dysfunction. Our case illustrates the use of standard ABVD even with elevated LFTs in the setting of CHL and VBDS. Repeat liver biopsy was not done as the patient remains in complete remission at oncology follow up.



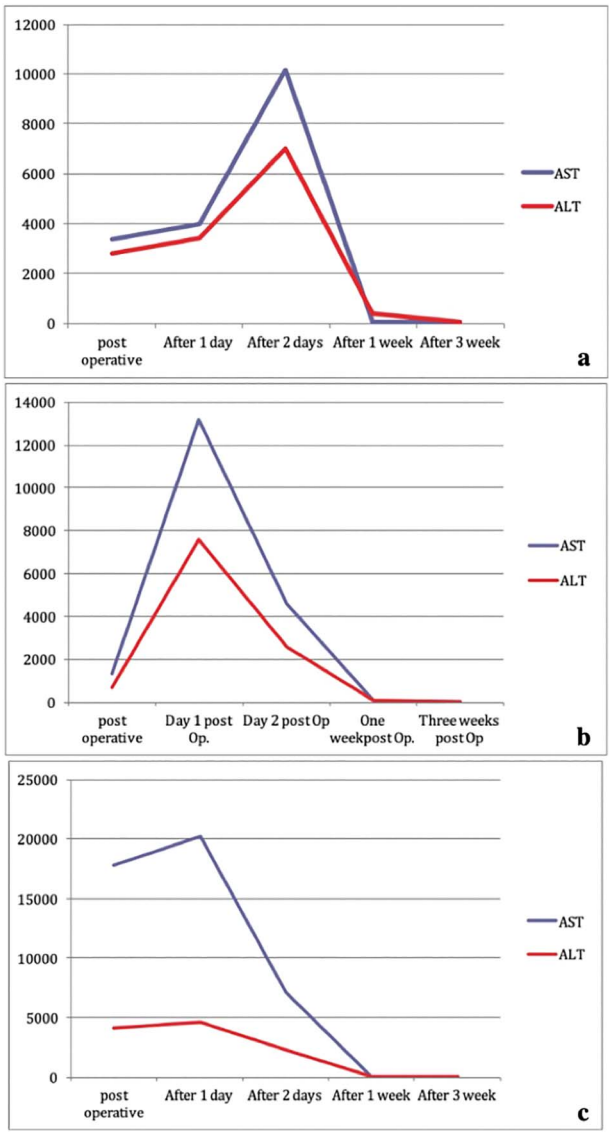
[3199] **Figure 1.** A: Lymph node biopsy, Hematoxylin & Eosin (H&E) stain, 200X B: Lymph node biopsy, CD30 immunohistochemical (IHC) stain, 100X (highlights CD30+ Reed-Sternberg and Hodgkin cells) C: Liver biopsy, H&E stain, 100X (shows bile duct loss) D: Liver biopsy, CK7 IHC, 200X (shows vanishing bile ducts, only isolated cells CK7+ with no intact bile ducts).

S3200

Unexpected Outcome of Ischemia-Reperfusion Injury Following Liver Transplantation: A Case Series

Antoine Boustany, MD, MPH[1], Mohammad AbuAssi, MD[2], Aiman Obed, MD, PhD[2], Abdalla Bashir, MD[2], Anwar Jarrad, MBBS[2].
[1]Cleveland Clinic Foundation, Cleveland, OH; [2]Jordan Hospital, Amman, 'Amman, Jordan.

Introduction: Severe ischemia-reperfusion injury is a potential deadly complication after liver transplantation that is characterized by a significant elevation of liver enzymes. It is usually complicated by severe organ dysfunction, rejection, and re-transplantation. In this case series, we report three cases of severe ischemia-reperfusion injury following liver transplantation with unexpected clinical course.
Case Description/Methods: Three cases of patients with autoimmune hepatitis, alcoholic cirrhosis, and idiopathic liver cirrhosis were selected. All of them underwent liver transplantation. Few hours after the surgery, their liver enzymes started to rise to the thousands raising concern for ischemia-reperfusion injury (Image 1). Patients remained clinically stable with reduced but preserved urine output and state of consciousness. Doppler-ultrasound of the graft revealed patent hepatic artery, hepatic vein, and portal vein. Therefore, the patients received supportive care. After few days of observation, liver enzymes started to decrease unexpectedly reaching a normal level after a week. The patients gradually improved and their liver function remained stable upon follow-up after discharge from the hospital.
Discussion: Severe ischemia-reperfusion injury is a potential deadly complication of liver transplantation. Liver function test abnormalities following liver transplantation are a hallmark of the clinical presentation. Upon diagnosis, the outcome is usually poor without re-transplantation. In this study, we presented three cases of patients with increased LFTs following liver transplantation, who improved with supportive measures alone. Although LFTs are commonly accepted as indicators of liver injury post-transplant, these cases highlight the importance of taking into account other parameters such as the clinical picture and radiological findings before determining the outcome of the ischemia-reperfusion injury following liver transplant; therefore, avoiding unnecessary and invasive measures.



[3200] **Figure 1.** Post-transplant AST and ALT levels in patient with autoimmune hepatitis (a), alcoholic cirrhosis (b), and liver cirrhosis of unknown origin (c).

LIVER

S3201

Zieve's Syndrome: A Case of Hemolytic Anemia in the Setting of Alcoholic Liver Disease

Jesus Salas Noain, MD, Parth Desai, DO, Shefali Amin, DO, MSEd, Andrew E. Lee, MD, Oluwaseun Shogbesan, MD, John Altomare, MD.
Tower Health - Reading Hospital, Reading, PA.

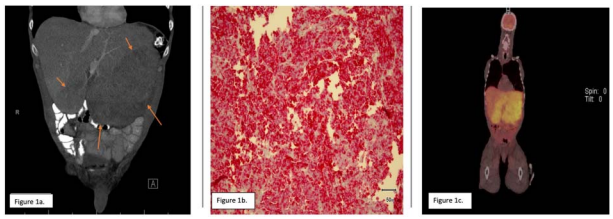
Introduction: Zieve's syndrome is a rare condition which typically manifests with hemolytic anemia, hyperlipidemia, jaundice in the setting of alcohol-induced liver injury. This is distinct from alcoholic hepatitis which may present simultaneously or develop later.
Case Description/Methods: 42-year-old man with a history of alcohol abuse and compensated cirrhosis presented to the emergency department for evaluation of 7 days of fatigue and jaundice in the setting of heavy alcohol use. He was noted to have a normocytic anemia with hemoglobin of 5.6 g/dl, INR of 3.0, total bilirubin of 26.2 mg/dl, AST of 65, and ALT of 23 IU/L. Lipid profile was normal. US liver and Doppler US revealed heterogenous liver with biphasic flow through majority of the veins consistent with portal hypertension. Patient denied recent use of new medications or herbal supplements. Prophylactic antibiotics for suspected gastrointestinal bleeding were started. He underwent EGD which revealed trace esophageal varices and portal hypertensive gastropathy without active bleeding. A peripheral blood smear showed spur cells, but no spherocytes or schistocytes consistent with Zieve's syndrome. Direct and indirect Coombs tests were negative, low haptoglobin levels, and elevated serum LDH. Blood cultures were all negative. In view of evidence of hemolysis and lack of infectious source a diagnosis of Zieve's syndrome was made and patient treated with supportive care and blood transfusions as needed. Unfortunately, the patient was placed in comfort measures after developing refractory grade IV hepatic encephalopathy and hepato-renal syndrome despite supportive measures.
Discussion: Hyperlipidemia in Zieve's syndrome results from cholestasis and hemolysis induced by alcohol liver injury, but was not present in our case. It is postulated that vitamin E deficiency secondary to alcohol use can result in pyruvate kinase instability which affects RBC metabolism resulting in hemolytic anemia. It is important to recognize that Zieve's can mimic or present concurrently with alcoholic hepatitis as both conditions present with anemia, jaundice, and alcohol use. An important distinction is the presence of hemolytic anemia in the former. Management is supportive as well as abstinence from alcohol.

S3202

What Lies Beneath the Skin? A Rare Cause of Acute Liver Failure

Miguel J. Anzalota Del Toro, MD, MPH, RIcardo L. Lopez Valle, MD, Jose Martin Ortiz, MD, FACG.
VA Caribbean Health Care System, Guaynabo, Puerto Rico.

Introduction: Malignant melanoma of the liver is a rare cause of acute liver failure despite the liver being the most common affected organ by metastatic melanomas. Liver metastasis may occur months to years after the primary lesion is removed. Severe coagulopathy, as may be present in acute liver failure, might be a challenge while performing liver biopsy. Herein a case of a male with abnormal liver chemistries and a liver mass diagnosed as metastatic melanoma.
Case Description/Methods: This is the case of a 65-year-old male with history of a right mastoid melanoma s/p excision who was lost to follow-up with dermatology for several years and who presented to ER with complaints of abdominal pain, left ocular pain with decreased vision, and bone aches. Patient also reported anorexia and 30 pounds weight loss. Initial labs showed abnormal liver chemistries. Abdominal CT scan showed a large infiltrating mass occupying most of the left liver lobe, with encasing vessels, and thrombosis of the left hepatic vein. A head CT showed large soft tissue masses in the left intraconal compartment causing mass effect upon the optic nerve and eye globe with associated proptosis. Metastatic disease was suspected. Given these findings, IR service was consulted for liver biopsy, but he first required treatment for his coagulopathy. After INR reached acceptable parameters, a percutaneous liver biopsy was done. On site prep was performed by cytotechnologist and pathologist, who deemed the sample appropriate for diagnosis and with features of a melanoma. Thus, patient was diagnosed with a metastatic melanoma of the liver leading to acute liver failure. Unfortunately, the patient had rapid deterioration and progression of his disease, making him a poor candidate for palliative immuno-/radiotherapy, and was placed under hospice care (Figure).
Discussion: Diffuse liver infiltration by a melanoma is an extremely rare cause of acute liver failure, often presenting aggressively, leading to a poor prognosis and a high mortality. Recurrence of malignancy after a long period of time is often reported despite patient's having previously achieved remission of their primary cancer. For this reason, metastatic liver cancer should be considered in patients with history of another primary malignancy. Hepatic resection has been proposed as therapeutic and potentially curative procedure. Closer follow up and early detection with liver biopsy would have been helpful in establishing an early diagnosis, leading to favorable treatment options and survival.



[3202] **Figure 1.** a. Large infiltrating mass of liver measuring 20 cm transversely by 20 cm longitudinally by 12 cm AP.

S3203

What the Eye Cannot See: A Case of Persistent Altered Mental Status in a Patient With Cholestatic Liver Disease

Jason Nasser, MD, William Carey, MD.
Cleveland Clinic Foundation, Cleveland, OH.

Introduction: Altered mental status (AMS) is a common symptom in patients with liver disease with a wide list of differential diagnoses. Knowledge of etiologies of AMS unique to patients with hepatic dysfunction is vital in order to help recognize, diagnose, and treat the underlying cause in a timely manner.
Case Description/Methods: A 46-year-old man with a history of recent COVID infection was transferred to our hospital for further evaluation of acute liver injury and AMS. On arrival, his labs were notable for AST of 408 U/L, ALT of 620 U/L, ALP of 5942 U/L, TB of 11.0 mg/dL, and an INR of 1.1. His work-up included an MRCP that showed segmental biliary ductal dilation with associated restricted diffusion and peribiliary enhancement concerning for sclerosing cholangitis. ERCP revealed a 3cm biliary cast that was removed and noted diffuse rarefaction of ducts throughout the entire biliary tree. A liver biopsy revealed centrizonal cholestasis with portal-based bile ductular reaction and mild bile duct injury. Despite adequate treatment of suspected infection and hepatic encephalopathy, his AMS persisted. His basic metabolic panel (BMP) was notable for Na of 143 mEq/L. A send-out lipid panel that was obtained to work-up his dyslipidemia revealed a total cholesterol of 1018 mg/dL, triglycerides of 420mg/dL, and the presence of lipoprotein X. A venous blood gas (VBG) was obtained showing a Na of 157 mEq/L and serum osmolality was 322 mmol/kg, confirming true hypernatremia. He was slowly treated with hypotonic solutions with significant improvement in his mentation. On follow-up one year later, he has persistent cholestasis and is currently being considered for liver transplant.
Discussion: The final diagnosis was COVID-related ischemic cholangitis and disappearing bile ducts with persistent cholangiopathy, presenting with severe cholestasis, accumulation of lipoprotein X, and pseudonormonatremia. When faced with severe cholestatic liver disease, clinicians should keep in mind the possibility of accumulation of lipoprotein X and its association with hyperviscosity and spurious electrolyte abnormalities. Clinicians should rely on obtaining blood gas analyses for accurate electrolyte measurement in such cholestatic patients as blood gas analyses utilize direct ion-sensitive electrodes (ISE) to measure electrolytes, whereas routine basic metabolic panels utilize indirect ISE that are liable to spurious results in the presence of hyperlipoproteinemia/lipoprotein X.

S3204

Turmeric-Induced Liver Injury: A Rare Case of DILI

Prisca Pungwe, MD, Leah Kuntz, BS, Brandilyn K. Monene, MD, Lauren L. Comer, MD, Keziah M. Thomas, MD, Teminioluwa A. Ajayi, MPH, MD, Milena Gould Suaez, MD, FACG.
Baylor College of Medicine, Houston, TX.

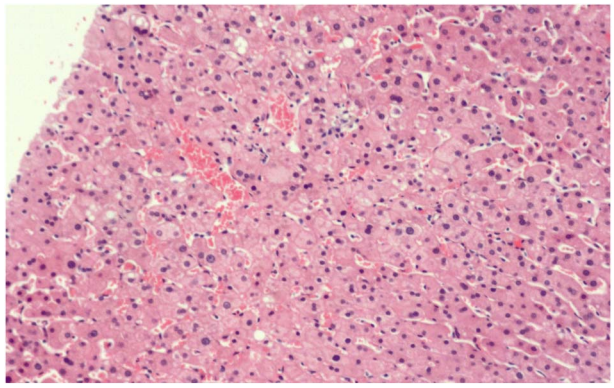
Introduction: Turmeric, a plant prevalent in South Asia, is often marketed to be safe, with many benefits including antioxidant and anti-inflammatory effects. Hepatotoxicity, a rare adverse effect, has only been documented in a few case reports. Here we present a case of turmeric Drug Induced Liver Injury (DILI) with normalization of liver function tests after its cessation.
Case Description/Methods: A 62-year-old female with a history of hypertension presented with nausea and generalized abdominal pain for five days. She denied taking medications other than hydrochlorothiazide. Further interview noted that she initiated turmeric tea over the preceding three weeks. On physical exam, she had scleral icterus and right upper quadrant tenderness. Laboratory workup showed AST 1510 U/L, ALT 1889 U/L, total bilirubin 13.9 mg/dL, direct bilirubin 8.1 mg/dL, ALP 134 U/L, LDH 542 U/L, with an ALT/LDH ratio of 3.49. INR was normal. Ultrasound revealed findings consistent with acute hepatitis. Ferritin, ceruloplasmin, and acetaminophen levels were normal. Alpha-1-antitrypsin, anti-mitochondrial, and anti-smooth muscle antibodies were unremarkable. Viral serologies were normal. The patient improved with turmeric cessation, with complete resolution of abnormal liver enzymes on follow-up.
Discussion: Turmeric, previously considered safe, has now been reported to be associated with DILI in a few case reports. Our case adds to the growing body of evidence supporting turmeric induced DILI. Although we could not uncover the exact dose, we do establish a high probability of adverse effects from turmeric using the validated Roussel Uclaf Causality Method (RUCAM) scoring system. Our patient scored 9, with a score >8 representing a high probability. Turmeric induced DILI is thought to be both dose dependent and associated with formulations that contain supplements or nanoparticles that increase turmeric's bioavailability. Lombardi et al. and Sohal et al. for example both report several cases of acute hepatitis associated with turmeric preparations developed with piperine (black pepper), which has been shown to increase the absorption of turmeric by 2000%. In our case, the patient's turmeric tea also included black pepper, which likely explains the hepatotoxicity. This case underscores the importance of supplement history, with an emphasis on turmeric, when evaluating for potential causes of DILI.

S3205

Voxelotor (Oxbryta) Induced Liver Injury

Ifrah Fatima, MD, Quinton Palmer, MD, Tahar Mahmoudi, MD.
University of Missouri-Kansas City, Kansas City, MO.

Introduction: Voxelotor is a HbS polymerization inhibitor approved by FDA in 2019 for treatment of sickle cell disease. Drug-induced liver injury (DILI) is one of the most common causes of acute liver injury and accounts for approximately 10% of all acute hepatitis. We present a rare case of DILI associated with Voxelotor at the dose of 1500mg which has not been previously reported.
Case Description/Methods: A 37-year-old male with a past medical history of HbSC disease was on Hydroxyurea 1.5g for 2 years which was discontinued due to pancytopenia. He was started on Voxelotor 1500mg daily with improvement in sickle cell symptoms, indirect bilirubin, hemoglobin, and reticulocyte count. He tolerated Voxelotor well. He had elevated alanine transaminase (ALT) 47 U/L after 4 months of therapy. Aspartate transaminase (AST) and ALT were uptrending at monthly follow-ups. Liver enzymes peaked at 6 months with AST 90 U/L and ALT 95 U/L. Alkaline phosphatase and total bilirubin were normal. Voxelotor 1500 mg daily was held for a week with improvement in liver enzymes- AST 35 U/L and ALT 45 U/L. A thorough review of history and physical examination was performed. Patient was asymptomatic. BMI was 23.8 kg/m2 and he drank alcohol socially. He was not on any other hepatotoxic, over-the-counter, or herbal medications. Acute hepatitis panel was negative. The R factor was 3.6, indicating a mixed hepatocellular-cholestatic pattern. Chronic liver disease workup including ceruloplasmin, A1AT, IgG, LKMA, AMA, ASMA were negative. Fibroscan showed no fibrosis or steatosis (F0S0). US gallbladder noted cholelithiasis with normal bile ducts. Liver biopsy showed mild sinusoidal congestion, normal architecture, no steatosis, hepatitis, or necrosis (Fig 1). Given the benefits of Voxelotor for sickle cell disease and negative workup, it was restarted at a lower dose of 1g. We recommended monitoring liver enzymes every 3-6 months and repeating Fibroscan in 1 year.
Discussion: DILI can be diagnosed based on correlation with exposure and improvement with cessation of drug. Only 2.2% of patients were reported to have elevated AST with a dose of 900mg in the Voxelotor randomized clinical trial. However, liver injury with Voxelotor at the dose of 1500mg was not reported. We described the first reported case of DILI with Voxelotor 1500 mg daily. Providers should be aware of this potential DILI and consider the risk and benefits of withdrawing treatment.



[3205] **Figure 1.** Liver biopsy showing mild sinusoidal congestion.

S3206

Turmeric-Associated Liver Injury: The Yellow in Your Food Causing the Yellow in Your Skin

Gaurav Mohan, MBBS, Farhan Khalid, MD, Charmee Vyas, MBBS, Swara Afiniwala, MD.
Monmouth Medical Center, Long Branch, NJ.

Introduction: Turmeric is a herbal medication that has gained significant popularity recently. Turmeric is used for its flavor, color, and purported antiinflammatory, antioxidant, antineoplastic, and antimicrobial properties. Turmeric supplements are generally self-medicated and patients can avail of them over the counter or through online portals. Turmeric typically has low bioavailability orally and is hence sometimes enhanced with nanoparticle delivery methods or piperine (black pepper) to increase absorption. Studies have linked these formulations to a higher incidence of hepatotoxicity.
Case Description/Methods: A 55-year-old female with a past medical-only significant for alcohol use presented to the emergency room with jaundice. She stated that she drank about 2 glasses of wine daily for the past 30 years and five days before her admission she drank around 5 drinks during St' Patrick's Day. Vital signs were stable and scleral icterus was noted. Lab investigations showed an alanine transaminase (ALT) of 2143U/L, aspartate transaminase (AST) of 2025U/L, alkaline phosphatase (ALP) of 590U/L, total bilirubin 8.1mg/dL, direct bilirubin 6mg/dL, and INR of 1.2. On further questioning, she revealed that she had been taking turmeric supplements daily for the past month for wrist arthralgias. She was consuming Qunol turmeric 1500mg once daily. Ultrasound of the abdomen was normal. She was given 19,200mg of NAC over 24 hours and all her medications were held. Over the course of the next five days her ALT came down to 1,730U/L and her AST came down to 1,517U/L without any additional therapy and she was asymptomatic. An extensive workup including hepatitis viruses, cytomegalovirus, smooth muscle antibody, and ceruloplasmin was sent which were all negative. She was discharged with close outpatient monitoring and her liver function tests normalized over two months (Figure).
Discussion: What makes our case unique is the use of NAC and quick discharge. There are few studies that have explored the role of NAC in the management of acute liver failure for non-acetaminophen-induced causes. The data is mixed with one Cochrane analysis showing no benefit in terms of death and need for a liver transplant. Another study shows that there may be a survival benefit in terms of post-transplant survival, transplant-free survival, and overall survival while decreasing the overall length of hospital stay. Through our case report, we hope to encourage clinicians to keep this etiology in mind when diagnosing acute liver enzyme elevations.

[3206] **Figure 1.** Turmeric.

S3207

Vanishing Tacrolimus Syndrome

Willie M. Johnson, MD[1], Thomas Leventhal, MD[2].
[1]University of Minnesota, Roseville, MN; [2]University of Minnesota, Minneapolis, MN.

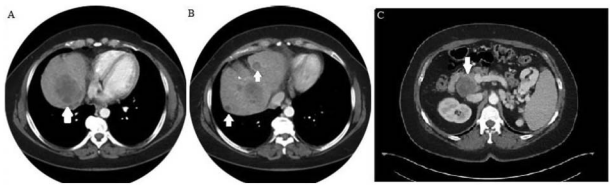
Introduction: Cellular rejection is common and therapeutic IS levels are needed to minimize this risk. This case stresses the need to fully explore, and potentially reexplore common causes of subtherapeutic IS levels.
Case Description/Methods: A 38-year-old man with history of alcohol related cirrhosis and liver transplant in 2019. followed by 2 episodes of acute rejection, presented with significantly abnormal liver tests. Tacrolimus levels were undetectable and liver biopsy showed moderate rejection. After a course of IV steroid, he was started on prednisone, sirolimus, and increased doses of tacrolimus. Tacrolimus levels remained undetectable despite escalating doses, and addition of fluconazole– used for cytochrome (CYP) inhibition. Liver tests worsened after initial improvements, and repeat biopsy showed changes of chronic rejection. He was treated with a 5-day course of anti-thymocyte globulin, steroids, and maintained on increased immunosuppression (IS). Drug levels for tacrolimus, sirolimus, and mycophenolate were undetectable. He reported medication adherence, and undetectable tacrolimus levels were thought to be due to CYP metabolism. With this concern, he was started on azathioprine and belatacept infusions for IS. He was readmitted with abdominal pain and chronic 10-15 stools daily. Physical exam was notable for abdominal pain, no jaundice or asterixis. Labs showed WBC 5.7, INR 1.03, total bilirubin 11.9, AST 784, ALT 714, Alkaline phosphatase 538, albumin 3.5, negative serology for EBV, CMV, and HSV, and tacrolimus level < 1.0. Lab testing for diarrhea was negative for viral and bacterial causes. With concern for malabsorption, IV tacrolimus was started. EGD and colonoscopy with biopsies were negative for malabsorptive pathology. He was started on pancreatic enzymes for concerns of pancreatic insufficiency, and fecal elastase obtained after was elevated. After 24 hours of IV tacrolimus, serum level was 22.7, and continued to rise even after reduction of dosing suggesting malabsorption as cause of subtherapeutic IS.
Discussion: Therapeutic IS levels are vital for transplant success, with an understanding that this needs effective absorption and metabolism. Our patient failed to have therapeutic levels since shortly after his transplant– despite previous negative lab and endoscopic evaluations for malabsorption. We used tactics focused on abnormal metabolism, however, resultant drug levels after IV tacrolimus shows the importance of considering malabsorption as barriers to achieving therapeutic IS.

S3208

Unanchoring Bias: A Case of Multifocal Hepatocellular Carcinoma and a "Pancreatic" Lesion

Prateek S. Harne, MBBS, MD, Ans Albustamy, MD, Arturo Suplee Rivera, MD, Murthy Badiga, MD, FACG, Asif Zamir, MD, FACG.
University of Texas Rio Grande Valley at Doctors Hospital at Renaissance, Edinburg, TX.

Introduction: Hepatocellular carcinoma (HCC) is the 4th leading cause of cancer-related mortality worldwide. About 20-40% of patients with HCC present in advanced stage at the time of diagnosis. Jaundice occurs in 1-12% of cases with HCC (icteric-type or cholestatic type HCC) attributed to tumor infiltration of liver parenchyma, liver failure, advanced cirrhosis and less commonly, obstruction by direct invasion of biliary tree and extrinsic compression by lymph node metastasis. We present a case of HCC that presented with painless jaundice with liver mass, satellite lesions and lymph node metastasis near the pancreatic head mimicking a primary pancreatic malignancy.
Case Description/Methods: A 50-year-old female patient with history of cholecystectomy presented with painless jaundice for ten days with generalized body weakness. Vital signs were stable. Physical examination revealed conjunctival icterus, jaundiced skin, soft, non-tender abdomen. Labs revealed white count 7100, AST 232, ALT 124, ALP 346, total bilirubin 42.4, direct bilirubin 26, AFP 339, CA 19-9 117. CT abdomen with contrast revealed scattered multiple hypodense lesions in liver; largest measuring 5.9 x 4.8cm, cirrhotic morphology and splenomegaly (Image A). Intra and extrahepatic biliary duct dilation was noted with CBD diameter of 16mm. At the level of pancreatic head, a 2cm soft tissue mass attenuating the CBD (Image B) was noted. These images were concerning for primary pancreatic malignancy with metastasis to liver. An ERCP was attempted but CBD could not be cannulated due to extrinsic compression by the mass and patient underwent radiology-guided internal-external percutaneous transhepatic cholangiogram and biliary drainage with CT-guided biopsy of the largest liver lesion which revealed moderately differentiated hepatocellular carcinoma. Patient was evaluated by Oncology and Surgery, but due to the tumor burden and performance status, opted for comfort measures.
Discussion: Metastasis of HCC to pancreatic tissue is rare and sporadically reported. Peripancreatic lymphadenopathy should be considered in patients with hepatic and 'pancreatic' masses as in our case, where the mass near the pancreas was a peripancreatic lymph node compressing the CBD, causing obstruction. Such unusual cases can be easily misdiagnosed as primary pancreatic cancer with liver metastasis instead. If doubt persists, biopsy should be used for definitive diagnosis, as misdiagnosis can preclude potentially curative resection.



[3208] **Figure 1.** A,B) CT scan showing heterogenous, multiple liver lesions and C) Mass lesion at the level of the head of pancreas attenuating the CBD.

S3209

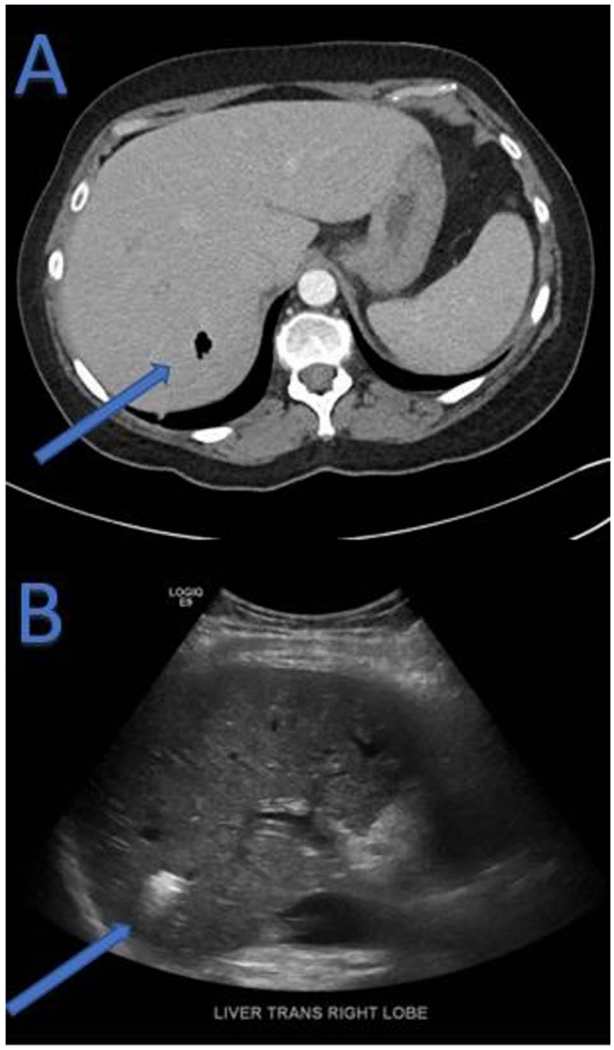
Unusual Cause of Liver Abscess: *Clostridium perfringens*

Tim Brotherton, MD, Soumojit Ghosh, MD, Jay Shah, MD, Laith Numan, MD, Michelle Baliss, DO, Zarir Ahmed, DO, Antonio Cheesman, MD.
Saint Louis University, St. Louis, MO.

Introduction: *Clostridium perfringens* is a gas-forming, spore-forming, gram-positive bacillus that is typically found in soil or fresh water sources. Bacteremia due to *C. perfringens* is rare. Our case is a 52-year-old woman with *C. perfringens* bacteremia resulting in gas-forming liver abscess.
Case Description/Methods: Our patient was a 52-year-old female with history of acute myelogenous leukemia who presented to the hospital with neutropenic fever. She was currently on consolidation chemotherapy with high dose cytarabine and her last treatment was four weeks prior to admission. Vitals signs show temperature of 103.0 Fahrenheit. She has pancytopenia with hemoglobin 4.0 g/dL, white blood cell count of 0.6 X 10^3/uL, and platelets are 7 X 10^3/uL. Her hepatic function testing on admission was abnormal with peak values of ALT 407 U/L, AST 474 U/L and total bilirubin 4.1 mg/dL (2.7 mg/dL was direct). Alkaline phosphatase was normal. Blood cultures were positive for *C. perfringens*. Computed tomography (CT) was obtained and showed gas within the right hepatic lobe (Figure 1A). An

abdominal ultrasound showed a hyperechoic focus in the right hepatic lobe corresponding to the findings on CT (Figure 1B). Ultimately, the patient was treated with two weeks of cefepime and metronidazole for her *C. perfringens* bacteremia with hepatic focus. Her clinical course improved with antimicrobial therapy. Hepatic function tests were within normal limits by the time of discharge.

Discussion: *C. perfringens* causes cytotoxic infection due to its alpha toxin, a lecithinase which breaks down cell membranes leading to cell lysis. Thus, our patient's severe acute anemia is explained in part by hemolysis due to clostridial infection. This organism is an uncommon cause of gas-forming liver abscess. A prior review of 119 cases of patients with gas-forming pyogenic liver abscess found only 8 to be infected with clostridia species. Malignancy and immunosuppression are both risk factors for *C. perfringens* infection and septicemia, both of which are present in our patient and thus made her more susceptible to clostridial infection. The etiology of her infection was potentially a bacterial translocation from the gastrointestinal tract. Mortality rate in patients with sepsis due to *C. perfringens* has been previously estimated at 70-100%. Thus, prompt recognition of this clinical syndrome is paramount so that early treatment can be initiated. Early appropriate antimicrobial therapy was essential to this patient's good outcome.



[3209] **Figure 1.** A) A CT scan of the abdomen showing a focus of gas in the right hepatic lobe (arrow). B) An ultrasound of the right upper quadrant showing focus of gas in the right hepatic lobe (arrow).

S3210

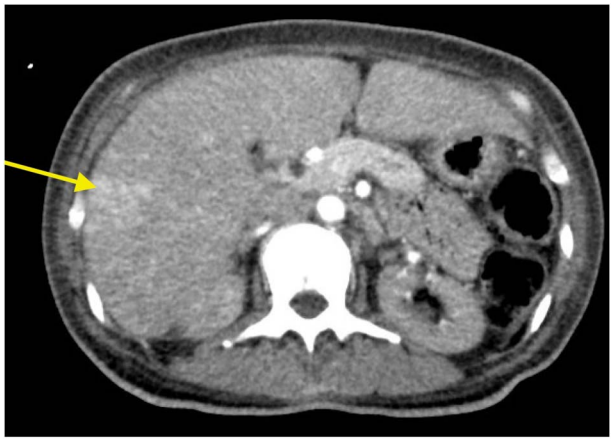
Unraveling Extrapulmonary Sarcoidosis in a Patient With Unexplained Cholestatic Liver Injury

VIctoria Green, MD, Ernesto Rodriguez, MD, Kiranjot Kaur, MS, Catherine Cadang, MD, Irene Guzman, DO, Justin Lewis, MD, Amitpaul Gill, MD, Marina Roytman, MD.
UCSF Fresno, Fresno, CA.

Introduction: Sarcoidosis is an autoimmune condition characterized by non-caseating granulomas most commonly in the lungs and hilar lymph nodes. We report a case of sarcoidosis presenting as hepatomegaly and cholestatic liver injury.

Case Description/Methods: A 46-year-old female with end stage renal disease, latent tuberculosis (TB) and daily use of herbal teas, who had multiple hospitalizations over a 6-month period with similar presentation, presented with right upper quadrant pain and jaundice. Examination revealed pulsatile abdomen and jugular venous distention. During each admission, she had elevated alkaline phosphatase and total bilirubin to a peak of 2003 U/L and 37.4 mg/dL respectively, with otherwise normal liver enzymes. Infectious workup and tumor markers were negative. On her second admission, she had rapid improvement in her lab abnormalities and symptoms after initiating steroids for treatment of Bell's palsy, otherwise, on other admissions she had slow improvement with ursodeoxycholic acid. Initial ultrasound abdomen, magnetic resonance cholangiopancreatography, and triphasic computed tomography (CT) liver showed hepatomegaly, otherwise unremarkable. She had two liver biopsies which revealed sinusoidal congestion and dilation, hepatocellular and sinusoidal fibrosis consistent with chronic venous outflow obstruction and few non-necrotizing granulomas. Repeated transthoracic echocardiogram eventually showed mild tricuspid and pulmonary insufficiency. Right heart catheterization showed mild pulmonary hypertension and hemodynamics not suggestive of a cardiac cause of liver disease. In a last attempt to locate tissue for biopsy, a CT chest, abdomen and pelvis was obtained and showed airspace opacities within the left lower lobe likely related to inflammatory process and an area of enhancement at the junction of segment 5 and 6 of the liver that could relate to hepatic sarcoid. She was started on Prednisone 40mg daily for presumed hepatic sarcoid and Rifampin 600mg daily for latent TB. She will undergo bronchoscopy with biopsy and renal biopsy to further establish a unifying diagnosis (Figure).

Discussion: After an extensive workup was found to be negative, sarcoidosis became the top differential given non-caseating granulomas on liver biopsy, nerve palsy, renal disease, mild pulmonary hypertension and response to steroids. Lack of pulmonary involvement is an uncommon presentation in sarcoidosis, occurring in 5-9% of cases overall. Hepatic sarcoidosis occurs in 11-80% of cases and is mostly asymptomatic.



[3210] **Figure 1.** Hepatomegaly with heterogeneous enhancement. There is an area of increased enhancement at the junction of segment 5 and 6 (arrow) of the liver which is nonspecific, but could relate to hepatic sarcoidosis given clinical picture.

S3211

Weight Loss, Ascites, and Diarrhea as the Presenting Symptoms for Pseudo-Pseudo Meigs Syndrome

Reem Q. Al Shabeeb, MD, Robert Chao, MD, Michael P. Keith, MD.
Inova Fairfax Medical Campus, Falls Church, VA.

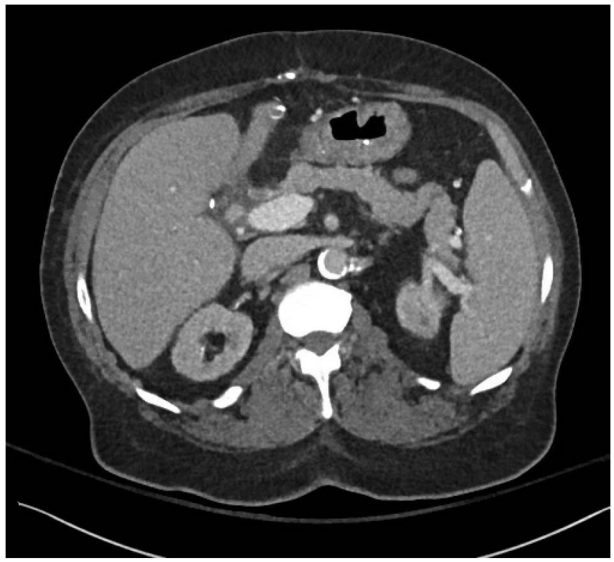
Introduction: Pseudo-pseudo Meigs syndrome (PPMS) is a rare presentation of systemic lupus erythematous (SLE) characterized by ascites, pleural effusion, and elevated CA-125 level.
Case Description/Methods: A 30-year-old woman with SLE and history of small bowel obstruction presented with diarrhea, weight loss, anasarca, and alopecia. She was initially tachycardic and hypertensive. Her exam revealed profound anasarca with ascites and bilateral pleural effusions. Labs were notable for Cr=5.65 mg/dL (baseline 0.7 mg/dL), spot protein/creatinine=3.9, ferritin=554.6 ng/ml, ESR=145 mm/Hr, CRP=6 mm/Hr, C3=49 mg/dL, C4=19 mg/dL, unremarkable FSH and LH, +ANA, dsDNA 356 U/ml, and +pANCA/MPO. Outpatient workup showed elevated CA-125 to 260.8 U/mL but ovarian malignancy was ruled out by MRI and CT abdomen/pelvis. Transvaginal ultrasound was normal. Inflammatory bowel disease was ruled out by EGD and colonoscopy during a previous hospitalization. Renal ultrasound showed bilateral hydronephrosis. The patient underwent a paracentesis (SAAG 1.3), and thoracentesis that showed total serum protein 4.2 g/dl, pleural fluid LDH 67 U/L, no serum LDH or pleural protein available. Kidney biopsy demonstrated class IV / V lupus nephritis. She was treated with a three-day course of pulse dose methylprednisolone followed by prednisone, hydroxychloroquine, and furosemide infusion. Her symptoms mostly resolved and CA-125 trended down to 27 U/mL.
Discussion: PPMS is a rare condition with few cases reported in the literature. This case illustrates the challenges of evaluating abdominal symptoms and ascites in patients with SLE, and the importance of a kidney biopsy in the evaluation of nephrotic range proteinuria. Due to infrequency and concern for malignancy due to elevated CA-125 level, patients with this syndrome undergo extensive workup. CA-125 has been shown to be elevated due to inflammatory markers enhancing its expression in peritoneal mesothelial cells. Prior publications have shown the correlation of CA-125 with extent of serositis in PPMS. Additionally, our patient has many other clinical features reported in PPMS patients including elevated ferritin, alopecia, and positive pANCA. CT scan of abdomen and pelvis is essential in the workup of this syndrome to rule out ovarian malignancy. Moreover, imaging may exclude mesenteric vasculitis, an uncommon feature of SLE which can be seen as targetoid lesions of the bowel on CT scan. Steroids are the treatment for PPMS.

S3212

Very Late Hepatic Artery Thrombosis After Orthotopic Liver Transplantation

Aditya Suresh, MD[1], Anuj Chhaparia, MD[2], Roshani Desai, MD[1].
[1]Saint Louis University School of Medicine, St. Louis, MO; [2]Saint Louis University, St. Louis, MO.

Introduction: Hepatic artery thrombosis (HAT) is a serious complication after orthotopic liver transplantation (OLT) and is the most common vascular complication. It is often categorized into early HAT which is defined as occurring < 30 days after liver transplantation & late HAT defined as occurring > 30 days post transplantation. The etiology of HAT is often related to surgical factors, such as vessel kinking, anastomotic stenosis, and intimal dissection, but can factors such as hypercoagulability, elderly donors, and rejection episodes can contribute. Symptoms can often be vague with common complaints being abdominal pain, fever, nausea, & emesis.
Case Description/Methods: A 68-year-old male with OLT (20 years prior to presentation) for primary sclerosing cholangitis (PSC), ulcerative colitis presented with 1 month history of nausea, emesis, and weakness. He was afebrile & hemodynamically stable. On physical exam, abdomen was soft, nontender, nondistended, and patient was jaundice. Laboratory data was notable for a two-month elevation of alkaline phosphatase, ALT, AST, direct bilirubin. An ultrasound of the liver with doppler evaluation was obtained that demonstrated an absence of flow in the left and right hepatic arteries within the liver. A computed tomography angiography (CTA) of the abdomen was obtained that confirmed these findings (Figure 1). Due to the history of PSC, a magnetic resonance cholangiopancreatography was obtained that showed no biliary dilation or evidence of recurrent PSC. A liver biopsy demonstrated paucicellular ductopenia with cholestasis consistent with chronic arterial insufficiency and no significant fibrosis. Transplant surgery and interventional radiology were consulted for potential recanalization. However, due to prolonged elevations in liver enzymes >4 weeks, both teams determined that recanalization would likely provide no benefit yet held greater risk.
Discussion: This case highlights an uncommon, very late presentation of HAT. In the literature, late HAT has been described in the order of months, not years as it was in this case. It is usually associated with a less fulminant presentation and a milder course, in comparison to cases of HAT that present earlier in the post-transplant course. This case highlights the importance of considering HAT as a diagnosis in a patient with history of OLT who presents with abdominal pain, nausea, and emesis.



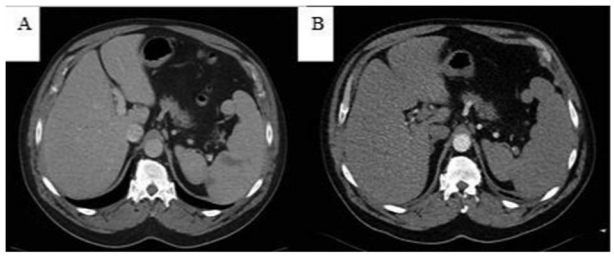
[3212] **Figure 1.** Focal filling defect in the hepatic artery seen in this venous phase CTA.

S3213

Transient Splenic Infarct in a Patient With Epstein Barr Virus (EBV) Infection: A Rare Presentation

Brian Sowka, DO, Padmavathi Mali, MD.
Gundersen Health System, La Crosse, WI.

Introduction: We report a case of transient splenic infarction as a rare complication of EBV infection. There have been few case reports of splenic infarcts as a complication of EBV infection in the literature. This case is unique as it presented with a transient splenic infarct which resolved in few days
Case Description/Methods: A 60-year-old male with a history of hypertension presented to the gastroenterology clinic in consultation for transaminitis. He presented to the hospital a week prior with three weeks of chills, myalgias, arthralgias, and abdominal pain. Laboratory evaluation during that admission showed elevated transaminases AST 118 (0-40 U/L), ALT 111 (0-40U/L), and Alkaline Phosphatase 223 U/L (40-129 U/L), total bilirubin was 1.0, LDH was elevated at 512, and mild pancytopenia. A CT scan of the abdomen showed faint wedge-shaped splenic hypodensities concerning for infarctions. He was started on enoxaparin which was later transitioned to aspirin. Transthoracic echocardiogram showed no evidence of endocarditis and blood cultures were negative. Pancytopenia improved but transaminases increased to AST 463, ALT 482, ALP 482, and Bilirubin rose to 1.4. A CT of the chest was performed to exclude pulmonary embolism which showed resolution of the splenic infarcts. He was discharged home and a follow-up workup for elevated liver tests showed a negative viral hepatitis panel, negative autoimmune work-up, and EBV antibody to Viral Capsid IgM and IgG returned elevated at 121 and 53 respectively. EBV antibody to Early Antigen was also elevated at 127. Over the next few weeks, his clinical symptoms and transaminases improved (Figure).
Discussion: There are few prior reports of splenic infarction as an uncommon complication of infectious mononucleosis related to EBV infection. This patient had a splenic infarct which resolved in a few days with no other etiology which was identified. The mechanism related to thrombosis and infarct formation is thought secondary to the pro-inflammatory state causing platelet adhesion in EBV infection. In our patient, this effect was transient likely secondary to perfusion abnormalities and he recovered well. This case reinforces the importance of ruling out EBV infection in patients with elevated liver tests and splenic infarct.



[3213] **Figure 1.** A – Axial CT demonstrating multiple visible hypodensities in the spleen B – Axial CT for pulmonary embolism with resolution of splenic infarcts.

S3214

Two Cases of *Klebsiella pneumoniae* Liver Abscess With Metastatic Infection

Justin J. Wagner, DO[1], Michael Parise, OMS-4[2], Nikolas St. Cyr, MD[1], John Trillo, MD[1], Sreekari Kedarisetti, MD[1].
[1]Coney Island Hospital, Brooklyn, NY; [2]Touro College of Osteopathic Medicine, Manhattan, NY.

Introduction: Klebsiella pneumoniae (K. pneumoniae) is a gram-negative organism that is a known cause of pyogenic liver abscess (PLA). When K. pneumoniae causes PLA in the absence of hepatobiliary disease it is defined as invasive liver abscess syndrome (ILAS), and is commonly seen in individuals from Southeast Asia who have diabetes mellitus, cholelithiasis, or steatosis. ILAS can sometimes include metastatic spread that most commonly results in endophthalmitis or meningitis. Although K. pneumonia PLA is an endemic disease in Southeast Asia, it is now seen throughout the world and should elicit greater awareness as one of the common causes of PLA in the United States.
Case Description/Methods: We present two cases of ILAS with metastatic spread causing endophthalmitis in male patients. The first case highlights known epidemiological and medical risk factors of ILAS with a 62-year-old from Southeast Asia who was diagnosed with poorly controlled type 2 diabetes mellitus (DM). The second case highlights an incidence of ILAS without any epidemiological or medical risk factors in a 36-year-old from the United States with no medical history. Both patients required interventional radiology for abscess drainage and intravenous ceftriaxone with subsequent clinical improvement and outpatient follow-ups in primary care and ophthalmology (Figure).
Discussion: Liver abscesses most commonly occur with hepatobiliary disease, ILAS from K. pneumoniae is a potentially life-threatening infection and associated with significant morbidity and mortality that can occur without any hepatobiliary disease. ILAS was once commonly thought to occur typically in males and isolated in Southeast Asia, but are now seen in all sexes across the world and becoming increasingly common in the US. DM tends to be associated with K. pneumonia PLA, and the disease process also tends to be more invasive with metastatic spread to other organs, thereby leading to sepsis. Endophthalmitis

is the most common infectious sequelae and is specifically associated with more virulent strains of K. pneumonia. ILAS should be part of the differential diagnosis of liver mass in the setting of sepsis, and due to K. pneumoniae metastasizing to other organ systems, early drainage and antibiotic administration is essential to decrease morbidity and mortality. A higher index of suspicion should be held for Southeast Asian diabetic males, the incidence of ILAS in all demographics is increasing, which should elicit greater awareness of risk factors, pathogenesis, and management.

Figure 1:

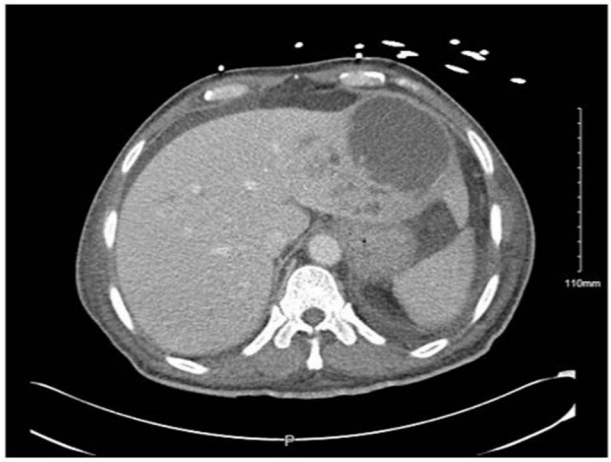
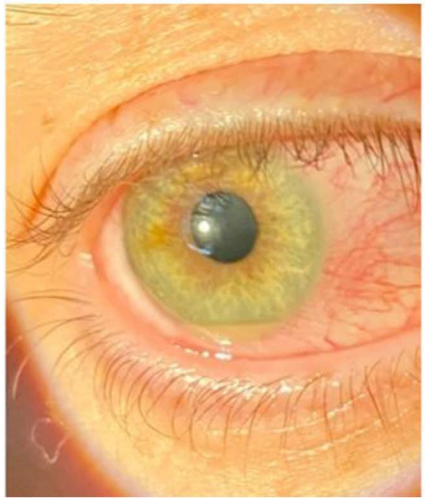


Figure 2:



[3214] **Figure 1.** Contrast-enhanced computed tomography scan of the abdomen in axial view demonstrating complex lesion in the left hepatic lobe (Case 1). Figure 2: Inflammation of the episclera indicative of episcleritis (Case 2).

S3215

Treatment of Invasive Liver Abscess Syndrome With Limited Percutaneous Drainage and Antibiotics

Levon Tantoushian, DO, Kory Cummings, DO, Thomas Ilustrisimo, DO, Mark McKenzie, MD, Paul Kalekas, DO.
Valley Hospital Medical Center, Las Vegas, NV.

Introduction: Klebsiella pneumoniae primary liver abscess (KLA) is characterized by bacteremia, liver abscesses, and metastatic infections. It is prevalent in Asian countries but has been rarely documented in the U.S. with approximately 20 documented cases as of 2007 [1]. KLA often originates as a single liver mass, but complications include multiple liver lesions, endophthalmitis, meningitis, or distant abscesses. First line therapy involves abscess drainage. We present a case of KLA in a patient with multiple liver abscesses which were unable to be entirely drained due to location and quantity. This case highlights the ability to treat KLA with a combination of abscess drainage and systemic antibiotics to remove inaccessible abscesses, preventing complications, and surgical resection.
Case Description/Methods: A 47-year-old Thai male who immigrated 8 years prior presented with right upper quadrant pain, nausea, vomiting, and diarrhea. He was febrile, tachcyardic and found to have a mild transaminitis and elevated procalcitonin. CT of the abdomen and pelvis showed a 4.1 x 3.3 cm mass in the anterior right liver lobe and a 2.9 x 3.2 cm mass in the medial right liver lobe. MRI of the abdomen revealed 3 additional subcentimeter foci in the left posterior liver. Blood cultures grew pansensitive *Klebsiella pneumoniae*. He underwent CT guided drain placement in the largest right liver abscess due to location and size of remaining liver masses. Abscess culture confirmed diagnosis of KLA. Intravenous antibiotics with metronidazole and ceftriaxone were completed for 2 weeks, followed by transition to oral amoxicillin-clavulanic acid for an additional 2 weeks. Repeat CT abdomen was obtained one week following treatment, revealing moderately diminished right lobe lesions. Patient's symptoms improved and transaminitis resolved. Abscess drains were removed, patient was discharged with oral antibiotics and instructed to follow-up in continuity clinic for repeat imaging.
Discussion: Invasive liver abscess syndrome is a phenomenon which typically affects the Southeast Asian population, but is extremely rare within the United States. Complications and mortality arise due to metastatic disease, fulminant sepsis, and lifelong deficits. Our case presented a patient with no risk factors who developed multiple liver abscesses, of which only 1 was accessible via percutaneous drainage. With our therapy, the abscesses were treated appropriately, avoiding further invasive source control and metastatic complications.

S3216

When to Start Anticoagulation? A Case of Portal Vein Thrombosis in a Post-Partum Cirrhosis Patient With Variceal Bleeding

Ke Ma, MD, Nathan Davis, DO, Yogesh Govil, MD.
Einstein Healthcare Network, Philadelphia, PA.

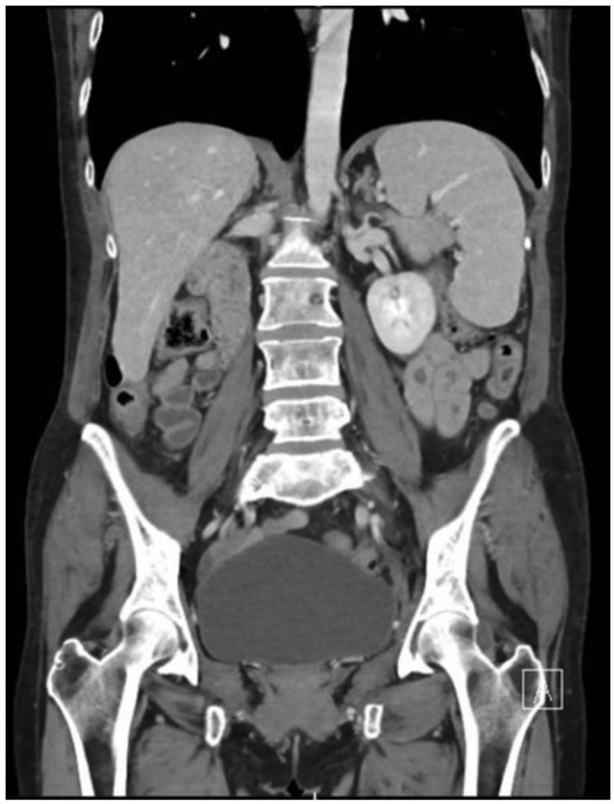
Introduction: While portal vein thrombosis (PVT) is a relatively rare disease in the general population, the prevalence of PVT in cirrhosis increases in parallel with the severity of cirrhosis. The pathogenesis of PVT in cirrhosis is thought to be multifactorial resulting mainly from alterations in the different components of Virchow's triad.
Case Description/Methods: Here we report a case of a 36-year-old woman with history of alcoholic cirrhosis who presented 2 weeks status post c-section complaining of acute onset abdominal pain, bloating, and nausea with two episodes of hematemesis. Her initial vital signs were unremarkable and hemoglobin was 7.4 gm/dL. Her model for end-stage liver disease (MELD) score was 16. She underwent upper endoscopy (EGD) with successful banding of 3 columns of large varices which showed stigmata of recent bleeding. Patient also underwent diagnostic and therapeutic paracentesis which did not show signs of spontaneous bacterial peritonitis (SBP). However, she did have history of wound dehiscence of her cesarean incision with persistent leakage of serous fluid despite prior negative pressure wound therapy. Therefore, CT abdomen/pelvis was obtained which demonstrated nonocclusive thrombus in the main portal vein extending into both the right and left branches. Due to finding of PVT, MRI was also obtained which showed no evidence of malignancy. As her hemoglobin remained stable (>7 gm/dL) during hospitalization, she was started on therapeutic low molecular weight heparin (LMWH). Unfortunately, patient was re-admitted five days later for hematemesis and drop in her hemoglobin to 5.6 gm/dL. She received blood products, and repeat EGD showed cratered and superficial esophageal ulcers at the site of prior variceal treatment. Ultimately LMWH was held and resumed after hemoglobin stabilized.
Discussion: This case demonstrates the conundrum of if/when to treat PVT in patients with cirrhosis. Current guidelines recommend anticoagulation as the mainstay of PVT treatment. However, this is can be complicated by other sequelae of decompensated cirrhosis such as variceal bleeding. Consequences of non-treatment include further increase in resistance to portal blood thus worsening portal hypertension. In this case, patient's post-partum status also favors anticoagulation as other society guidelines have recommended anticoagulation with LMWH for postpartum patients with vascular disease of the liver. Therefore, individualized treatment algorithms should be developed and used to guide management.

S3217

Non-Cirrhotic Portal Hypertension in a Patient With Rheumatoid Arthritis

Rebecca Yao, MD, MPH, Daniela Guerrero Vinsard, MD, Seth Sweetser, MD.
Mayo Clinic, Rochester, MN.

Introduction: Gastrointestinal bleeding in rheumatoid arthritis (RA) most commonly occurs from gastroduodenal ulceration in the context of chronic NSAID and prednisone use. We present an unexpected case of variceal bleeding in a patient with RA that was found to be secondary to non-cirrhotic portal hypertension, with liver biopsy revealing nodular regenerative hyperplasia.
Case Description/Methods: A 59-year-old woman with chronic neutropenia and longstanding seropositive RA on prednisone presented with a 3-week history of cough, fevers, hematemesis, and melena. On presentation, she was febrile to 39.4 C and tachycardic with heart rates up to 130 bpm. Physical examination revealed right-sided abdominal tenderness, subcutaneous rheumatoid nodules on her upper extremities, and several spider nevi on the upper chest. Initial laboratory findings were remarkable for a white blood cell count of 16.8x109/L (baseline 2-3x109/L), hemoglobin 4.7 g/dL (baseline 8-9 g/dL), BUN 22 mg/dL, serum albumin 1.5 g/dL, ESR 160 mm/h, and CRP 53 mg/L. She was found to have pneumococcal sepsis, with CT imaging additionally revealing mild splenomegaly measuring 14 cm in length. Evaluation of her hematemesis with upper endoscopy unexpectantly revealed bleeding esophageal varices that were banded. Transjugular liver biopsy revealed nodular regenerative hyperplasia (NRH).In the context of her chronic neutropenia, splenomegaly, and rheumatoid arthritis with further findings of NRH, the patient was diagnosed with Felty Syndrome (Figure).
Discussion: Felty Syndrome is a rare extra-articular manifestation of longstanding, joint-deforming, seropositive rheumatoid arthritis defined by the triad of RA, neutropenia, and splenomegaly. A strong association exists between Felty Syndrome and NRH. NRH leads to a form of presinusoidal non-cirrhotic portal hypertension, which may lead to variceal formation and bleeding. This case demonstrates the importance of maintaining a wide differential for upper GI bleeding in patients with RA and highlights the hepatic and gastrointestinal associations in Felty Syndrome.



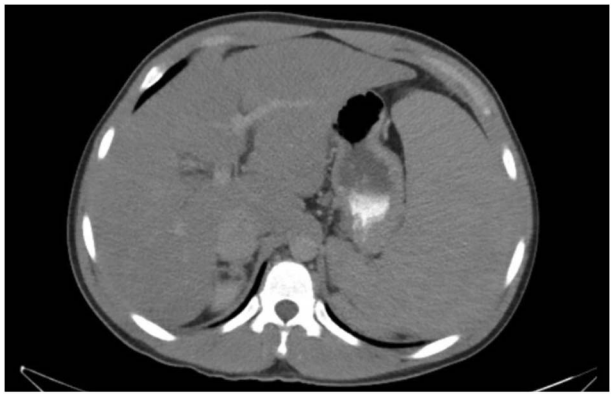
[3217] **Figure 1.** CT abdomen and pelvis demonstrating mild splenomegaly.

S3218

Two Concurrent Genetic Disorders Leading to Liver Cirrhosis

Subhash Garikipati, MD[1], Dhruvkumar Patel, MBBS[2], Hrishikesh Samant, MD[3].
[1]Louisiana State University, Shreveport, LA; [2]LSU Health Sciences Center, Shreveport, LA; [3]Ochsner Medical Center, New Orleans, LA.

Introduction: Cirrhosis in younger adults is often caused by autoimmune hepatitis, Wilson's disease, and primary sclerosing cholangitis, but genetic-metabolic disorders must also be included in the differential. Lysosomal acid lipase deficiency (LAL-D) is a rare lysosomal storage disorder that can range from mild and presenting in early adulthood to severe with mortality within one year of life. The adult variant of LAL-D can sometimes be overlooked since symptoms are nonspecific, but it is important to recognize as enzyme replacement therapy can be life-saving.
Case Description/Methods: A 20-year-old male with no medical history presented with incidental transaminitis (AST 99U/L, ALT 99 U/L), thrombocytopenia (62,000/ul) and leukocytopenia (2200/ul) without any clinical symptoms. A bone marrow biopsy showed normal cellularity for the age group without evidence of clonal proliferation. Abdominal ultrasound revealed features suggestive of portal hypertension and fibroscan showed steatosis grade S3/fibrosis score F4. Next, a liver biopsy showed glycogenic hepatopathy with periportal and focal bridging fibrosis. Furthermore, an EGD showed possible villous blunting but biopsy ruled out celiac disease and there was no evidence of esophageal varices/portal gastropathy. Both pathologies did reveal clear foamy appearing cytoplasm that contain Period acid-Schiff-diastase stain positive. In addition, enzyme levels of liposomal acid lipase were 0.0 nmol/h/ml (normal $>$ 21.0) and confirmatory LIPA gene sequencing was done and the patient was found to have liposomal acid lipase deficiency. He was also found to be heterozygous for alpha-1 antitrypsin (A1AT) deficiency of the M and Z isoforms with a level of 87 mg/dl. Treatment included sebelipase alfa 1 mg/kg infusions every 2 weeks (Figure).
Discussion: LAL-D is a rare, autosomal recessive lysosomal storage disease that involves a deficiency of LAL which is involved in the degradation of triglycerides and cholesterols, thereby causing accumulation of these substances in the cells. This deficiency is caused by a mutation in the LIPA gene. Our patient not only had the lysosomal storage disease causing liver injury but was possibly exacerbated by the addition of heterozygous A1AT deficiency which causes modest deficiency of the enzyme. These patients should be monitored for premature atherosclerotic vascular disease and hepatocellular carcinoma. Administering recombinant human LAL enzyme, sebelipase alfa, improves life expectancy.



[3218] **Figure 1.** Diffuse hypodense liver suggestive of fatty liver disease and massive splenomegaly.

S3219

Acute Hepatitis C After Penile Stem Cell Injection

Sahil Zaveri, MD, Ariana R. Tagliaferri, MD, Gabriel Melki, MD, Polina Aron, MD, Patrick Michael, MD.
St. Joseph's University Medical Center, Paterson, NJ.

Introduction: Hepatitis C (HCV) is clinically quiescent after transmission; however, some may be symptomatic. Cases that present within a 6-month window from the time of exposure are considered to be acute. Acute HCV can be difficult to distinguish from chronic infection as serum antibodies can take up to 12 weeks to form; however, HCV RNA is the most accurate way to detect acute HCV. Cases are likely underdiagnosed due to the clinical quiescence of the disease in its acute state. HCV is commonly transmitted via blood-borne pathogens and usually manifests as a chronic infection. We present a unique case of acute HCV from a penile stem cell injection.
Case Description/Methods: A 58-yo M, no PMH presented with scleral icterus after traveling to the Dominican Republic 3-weeks prior, where he underwent a penile stem cell injection for erectile dysfunction and subsequently experienced nausea, nonbilious emesis, watery diarrhea, chills and general malaise lasting 14 days prior to presentation. VS were remarkable for hypertension (183/104 mm/Hg) and on examination he was jaundiced with diffuse abdominal tenderness without peritonitis. All other risk factors for hepatitis were negative. Labs were significant for mild leukocytosis (8.6 K/uL), abnormal LFT's (ALT 1046 U/L, AST 570 U/L, ALP 163 U/L), conjugated hyperbilirubinemia (total bilirubin 20.8 mg/dL, direct bilirubin 14.3 mg/dL), and elevated prothrombin time (16.3 seconds). He was admitted for suspected hepatitis and was initially treated with NAC-infusion until further testing. PCR testing was negative for EBV, CMV, and HSV 1/2. His autoimmune markers were negative. Hepatitis A total Ab's and anti-Hepatitis C were positive, with HCV RNA qualitative of 8050 IU/mL. The HCV PCR revealed genotype 1a. A portal vein U/S was unremarkable, and a CT abdomen and pelvis with IV contrast revealed non-specific, small hypodensities in the liver lobes. In the GI clinic 2 weeks later, his jaundice had resolved, bilirubin levels had normalized, and the patient was entirely asymptomatic. Anti-HCV treatment was initiated.
Discussion: Although previous cases have reported the reactivation of chronic HCV after hematopoietic stem cell transplantation, it is uncommon for HCV to present acutely, especially in an immuno-competent patient. Despite literature of reactivated HCV, to our knowledge this is the first case of an acute HCV infection after a penile stem cell injection. It is essential that clinicians detect HCV in early stages to prevent long-term complications.

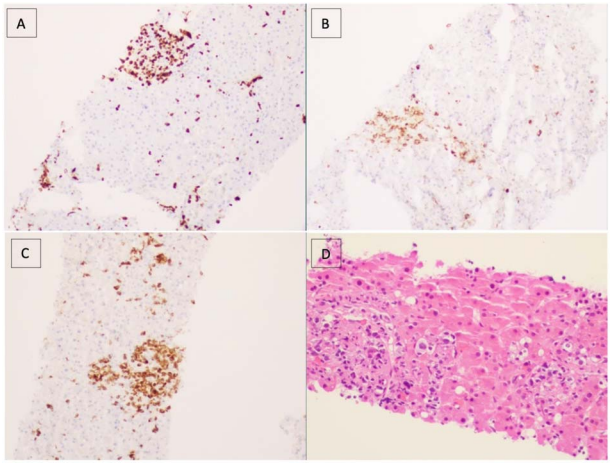
S3220

An Unusual Case of Jaundice

Lyndie R. Wilkins Parker, DO, Idrees Suliman, MD, Spogmai R. Khan, MD, Abdul Nadir, MD.
Mountain Vista Medical Center, Mesa, AZ.

Introduction: Peripheral T- Cell Non-Hodgkin's lymphoma is the most common subtype of hematologic malignancies, which rarely presents as jaundice. Here in, a case of obstructive jaundice attributable to recurrent Peripheral T Cell Lymphoma (PTCL) is described.
Case Description/Methods: A 75-year-old Caucasian man presented to the Emergency Department with confusion. Fifteen months earlier he was diagnosed with PTCL on axillary lymph node biopsy and treatment with Cyclophosphamide, Etoposide, Prednisolone, and Vincristine (CEOP) was initiated with improvement in his symptoms, primarily itching. Eight months later, he was found to have a cardiac ejection fraction of 20% and a relapse of PTCL was documented. Romidepsin was initiated three months prior to the current hospital admission, but was stopped within three weeks due to the relapse of PTCL, documented on bone marrow biopsy. His liver tests were normal two months prior to admission. On current admission, liver tests showed alkaline phosphatase of 400 IU, AST of 148 IU, ALT 142 IU, and total bilirubin of 3.4 mg/dL. A week later, bilirubin increased to 20.1 IU, being predominantly direct. CT scan and ultrasound of the abdomen, as well as nuclear medicine biliary scan, did not indicate extrahepatic biliary obstruction. A liver biopsy showed diffuse infiltration of the liver parenchyma with abnormal lymphocytes which were stained with CD3+ and CD5+ markers, but did not stain with CD7, documenting relapse of his PTCL in the liver. Diffuse intrahepatic cholestasis was also documented (Image). Two days after the liver biopsy, supportive treatment was withdrawn; and he expired.

Discussion: While the bile duct may be compressed by lymphomatous mass' anywhere along its path; more commonly at the hepatic hilum (by porta hepatis lymph nodes) and the distal common bile duct (by peripancreatic nodes), canalicular obstruction and resultant intrahepatic cholestasis from relapse in the liver has rarely been reported. This case highlights the importance of performing a liver biopsy to document the diagnosis and provide closure for the family. In this particular situation, the family decided to withdraw care, after a liver biopsy confirmed relapse of PTCL.



[3220] **Figure 1.** Random Liver Needle Biopsy Staining- Ki-67(A), CD30(B), CD3(C), and H&E(D).

S3221

Adult Onset Progressive Familial Intrahepatic Cholestasis: A Rare Presentation

Aldanah Althwanay, MD, Dania Hudhud, MD, Sassan Sakiani, MD.
University of Maryland Medical Center, Baltimore, MD.

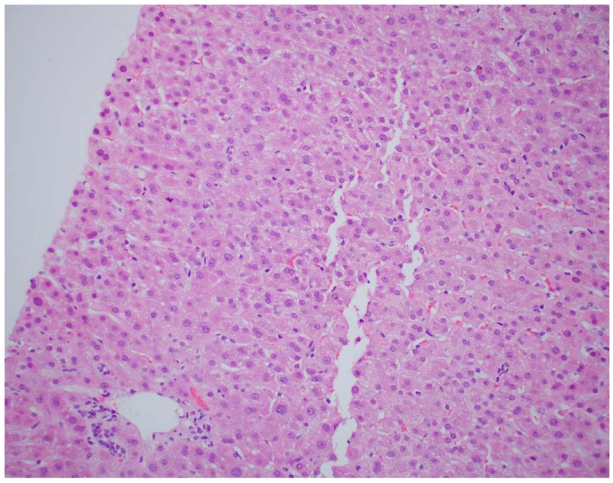
Introduction: Progressive familial intrahepatic cholestasis (PFIC) is a heterogeneous group of autosomal recessive childhood genetic disorders that disrupt intrahepatic biliary secretion. PFIC accounts for 10 to 15% of children's cholestatic diseases and liver transplantation. Although all patients will require liver transplantation at a certain point, early diagnosis and intervention can delay complications and transplantation.
Case Description/Methods: A 41-year-old male presented with cryptogenic cirrhosis and a MELD score of 35 for transplantation evaluation. He had a childhood history of abnormal liver enzymes with a mixed pattern and cholecystectomy due to recurrent gallstones. Prior liver biopsy showed autoimmune features; however, there was no significant response to the conventional therapy with prednisone and azathioprine. However, a re-evaluation of his liver biopsy favored biliary disease: subsequent MRCP with normal bile ducts. Given the insidious onset of his cirrhosis without an identifiable etiology and the suggestion of biliary cause, PFIC was considered during pre-transplantation workup. Genetic testing for ABCB4 confirmed PFIC type 3; subsequently, he underwent liver transplantation with an unremarkable post-operative course.
Discussion: This case highlights the impact of exploring a diagnosis of PFIC in adults referred for liver transplantation with long-standing cholestatic liver disease and negative initial workup. While currently, liver transplantation is the primary treatment option for end-stage liver disease from PFIC, advances in gene therapy have led to potentially curative treatments. As such, improved provider awareness and having a lower threshold for diagnostic testing may lead to an earlier diagnosis which is critical for optimal management, delaying the onset of end-stage liver disease, and offering to screen for family members.

S3222

Drug-Induced Liver Injury by Novel Dietary Supplement

Matthew Kobeszko, MD, MBA, MS, Rehana Begum, MD.
Advocate Aurora Health, Milwaukee, WI.

Introduction: Drug-induced liver injury (DILI) secondary to herbal and dietary supplements present with unique diagnostic challenges with potentially devastating clinical outcomes if left untreated. We present a case of biopsy confirmed drug induced liver injury secondary to ningxia wolfberry puree. To our knowledge, this is the first case report in literature.
Case Description/Methods: A 60-year-old female with no prior medical history presented to an outpatient clinic for evaluation of jaundice, pruritis, and a cutaneous rash. Patient was not previously taking any prescription medications and the only recent change was the addition of a new supplement containing ningxia wolfberry puree 6 weeks prior. Liver enzymes were elevated with total bilirubin 7.5, direct bilirubin 5.8, ALT 161, AST 381, and alkaline phosphatase 304. Patient underwent a chronic liver disease work up that was negative including viral, autoimmune, and metabolic etiologies. MRCP demonstrated no common bile duct dilation or biliary obstruction. Liver biopsy demonstrated intracanalicular cholestasis and lobular inflammation (image 1). Liver enzymes continued to up-trend at which point she was started on prednisone with a subsequent taper. Liver enzymes reached normalization over the course of 5 weeks. A 3-month follow up after treatment and supplement discontinuation demonstrated continued normal liver enzymes.
Discussion: The growth of over-the-counter supplement use has begun to present diagnostic challenges of identifying accurate diagnosis and management. While prescription medications undergo scrutinous review, most dietary and herbal supplements are classified as a food product. Therefore, the Food and Drug Administration does not require provisional review for safety. The worldwide incidence of DILI reported as 19 per 100,000 with 16 percent attributed to dietary supplements. In addition, DILI secondary to supplements accounts for 11% of acute liver failure cases overall, with a mortality rate of 8% and 2% requiring a transplantation. Even with prompt diagnosis and management, studies have demonstrated 14% of individuals will have continued liver test abnormalities beyond 6 months. While discontinuation of the offending agent is typically sufficient, more severe cases require further treatment with prednisone or n-acetyl cysteine. While many supplements are advertised as having antioxidant and immunomodulatory activity, many of these may contain ingredients that have strong biological effects with unclear acute and chronic risks.



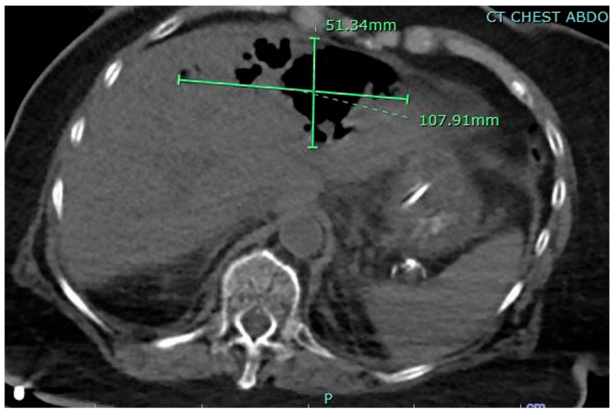
[3222] **Figure 1.** Liver Biopsy.

S3223

***Clostridium perfringens* Liver Abscess With Massive Intravascular Hemolysis**

Muhammad Farooq, MD.
East Carolina University Brody School of Medicine, Greenville, NC.

Introduction: Pyogenic liver abscess caused by Clostridium Perfringens is a rare, but a rapidly fatal infection with a reported mortality of 70 -100 %, therefore making prompt diagnosis and early aggressive management critical.
Case Description/Methods: A 75 year old female with intellectual impairment, diabetes mellitus type 2 and hypertension presented with a one day history of fatigue, poor oral intake and one episode of emesis. There was no reported fevers, chills, night sweats, abdominal pain, diarrhea, exposure to sick contacts or recent travel. On presentation the patient was febrile (101.4 F), hypotensive (65/49 mm Hg) and tachycardic (123 beats per minute). She appeared diffusely icteric, fatigued, had prominent bilateral scleral icterus and conjunctival pallor. Abdominal exam did not reveal any distension, tenderness, guarding or rigidity. Multiple lab draws returned hemolyzed with the exception of serum lactate which was initially 8.0 mmol/L. Arterial blood gas returned with a pH of 7.21 and PCO2 of 29. A non-contrast CT scan of the abdomen revealed air lucency in the liver. The patient was intubated due to worsening lethargy and hypoxia. She was also started on vasopressor support and broad spectrum antibiotics (Vancomycin, Cefepime and Flagyl). Immediate CT-guided aspiration of the abscess with drain placement was performed and the patient was transfused with multiple units of packed red blood cells in the setting of persistent hemolysis with worsening hypotension. Repeat blood draw showed worsening lactic acidosis (lactate of 19 mmol/L and pH of 6.96) despite receiving multiple bicarbonate injections and an infusion of sodium bicarbonate. The patient was deemed too unstable for renal replacement therapy and went on to develop rapid refractory shock and acidosis followed by cardiac arrest. She did not receive cardiopulmonary resuscitation as per the family's wishes and eventually passed away. Blood and abscess cultures later retuned positive for Clostridium Perfringens (Figure).
Discussion: Amongst prior reported cases of C. Perfringens liver abscess, 30 % had Diabetes Mellitus, 20% had advanced cancer and 15 % had cirrhosis including two who were liver transplant recipients on immunosuppressive therapy. This indicates a possible immune deficient state being a causative factor. The main toxin of the bacteria is a lecthinase (α toxin) that splits lecithin of red cell membrane leading to massive intravascular hemolysis. Early surgical intervention has been shown to be the most effective mode of treatment.



[3223] **Figure 1.** Liver abscess.

S3224

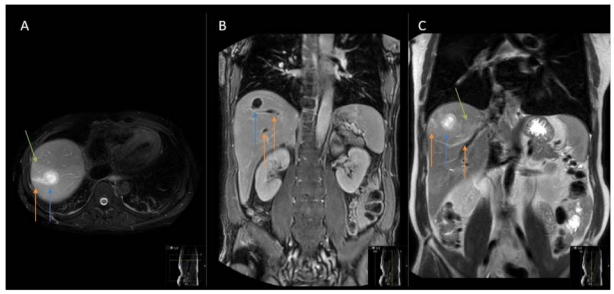
Pylephlebitis Presumed to Be Secondary to a Perforated Diverticulum

Suraj Pai, MD[1], Michael W. Song, MD[1], Fadi Hawa, MD[1], Nikhilesh Mazumder, MD, MPH[2].
[1]*University of Michigan Medical Center, Ann Arbor, MI;* [2]*University of Michigan, Ann Arbor, MI.*

Introduction: Pylephlebitis is acute thrombosis of the portal system in the setting of intraabdominal infection. Diagnosis is challenging since symptoms may be nonspecific, but early identification and management with broad spectrum antibiotics is essential. We present a case of pylephlebitis presumed to be secondary to a perforated diverticulum.
Case Description/Methods: A 63-year-old African American male with colonic diverticulosis presented with recurrent fevers and chills of two weeks duration. He immigrated from Benin 8 years ago but denied recent travel or sick contacts. On presentation, patient was febrile to 103.1F with tachycardia (107 beats/minute). Physical examination was normal. Labs revealed leukocytosis (15.9 K/uL) and mildly elevated liver enzymes with hyperbilirubinemia (1.4 mg/dL). Abdominal imaging revealed two hepatic abscesses with a multifocal occlusive thrombus in the right hepatic vein and a nonocclusive thrombus in the right portal vein. He underwent image guided aspiration of the abscesses and was started on intravenous (IV) antibiotics and heparin infusion. Fluid cultures grew *Fusobacterium nucleatum* and blood cultures had no

growth. Due to concerns for colonic bacterial translocation, colonoscopy was performed which showed diffuse diverticulosis throughout the colon without evidence of diverticulitis. Upon clinical improvement, patient was discharged on a 4-week course of ceftriaxone/metronidazole with a 12-week course of apixaban. Repeat abdominal ultrasound a month after discharge showed interval improvement of hepatic abscesses and resolution of portal vein thrombus. (Figure)

Discussion: Pylephlebitis is a rare condition but frequently associated with intra-abdominal infections, mainly diverticulitis which is the likely source in this patient. Inflammatory bowel disease and recent abdominal surgery have also been described as sources. Diagnosis is established via imaging confirming portal vein thrombosis in setting of an intraabdominal infection. Gram-negative organisms are most commonly isolated. The mainstay of therapy is broad spectrum antibiotics for 4-6 weeks. The role of anticoagulation (AC) is controversial as robust evidence is lacking. However, it has been proposed that AC prevents bowel ischemia and infarction secondary to thrombus extension. Despite the risk of sepsis and bowel infarction, morbidity and mortality rates have improved in recent years as imaging modalities have advanced and antibiotic options have diversified. AC should be considered on case-by-case basis.



[3224] **Figure 1.** A) 2.8 cm rim-enhancing abscess with rim-sign/double target sign (blue arrow) seen in axial view of segment 8; B) Associated T2 hyperintense, T1 hypointense linear filling defect consistent with thrombus along course of right hepatic vein branches (orange arrow) adjacent to abscess; C) Associated perfusional changes/wedge-shaped edema and T2 hyperintensity in liver parenchyma (green arrow).

S3225

Autoimmune Hepatitis Presenting Without Autoantibodies: A Diagnostic Challenge

Sherin Samuel, DO, Kevin Groudan, MD.
UMass Chan Medical School - Baystate, Springfield, MA.

Introduction: Autoimmune hepatitis (AIH) refers to chronic inflammatory liver disease characterized by loss of tolerance to hepatocyte antigens. Approximately 70 to 80% of cases present with detectable autoantibodies, such as antinuclear antibody (ANA), anti-smooth muscle antibody (ASMA), and anti-liver-kidney microsomal-1 antibodies (anti-LKM-1). We report a unique case of seronegative AIH in a patient whose above mentioned antibodies were negative, but liver biopsy results were consistent with a diagnosis of AIH.

Case Description/Methods: A 54-year-old female with no significant past medical history presented with weeks of fever, night sweats, fatigue, and weight loss. She was vitally stable. Labs were significant for an aspartate aminotransferase (AST) of 362, alanine aminotransferase (ALT) of 448, alkaline phosphatase of 496, and normal total bilirubin. Imaging was significant for a normal right upper quadrant ultrasound and a computed tomography abdomen that was unremarkable for liver pathology. Further lab work-up included negative viral hepatitis serologies and negative ANA, ASMA, and anti-LKM-1. Due to concern for liver toxicity, patient's home tylenol and duloxetine were held without significant improvement in her transaminitis or symptoms. Liver biopsy was performed and showed marked lobular inflammation of hepatic architecture with predominantly intrasinusoidal pattern and portal tracts demonstrating interface hepatitis, consistent with a diagnosis of AIH. Patient was started on budesonide with resolution of her transaminitis and symptomatic improvement.

Discussion: Seronegative AIH follows a similar clinical course as seropositive AIH. When symptomatic, patients can present with abdominal pain, weight loss, and fatigue as well as laboratory workup with elevated liver enzymes. Atypical autoantibodies that may assist in the diagnosis of AIH when ANA, ASMA, and anti-LKM-1 are negative include atypical perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA) and anti-soluble liver antigen/liver pancreas antibody (anti-SLA/LP). Clinicians with a high degree of suspicion for AIH should be aware that it can present without autoantibodies because when absent, appropriate diagnosis and treatment can be delayed.

S3226

Hepatic Sarcoidosis Manifesting as Asymptomatic Elevation in Liver Enzymes

Chinmay Trivedi, MBBS, Amer Jarri, MD, Mahabuba Akhter, MD.
Hackensack Meridian Health - Palisades Medical Center, North Bergen, NJ.

Introduction: Sarcoidosis is a systemic disorder of unknown etiology characterized by the formation of non-caseating granulomas and can affect any organ in the human body. The most frequently involved organs are lungs and hilar lymph nodes; however, involvement of the liver has been described in prior literature. The spectrum of hepatobiliary involvement can range from asymptomatic hepatic granulomas, and minimally deranged LFTs to symptomatic disease complicated with cholestasis, portal HTN, and cirrhosis. We report a case of asymptomatic LFT elevation in a patient with a prior history of sarcoidosis.

Case Description/Methods: An 82-year-old African American woman with a past medical history of coronary artery disease, hyperlipidemia, hypertension, Asthma, and Sarcoidosis presented with asymptomatic elevated LFTs discovered by her PCP. The patient denied any complaints from the GI or any other organ system standpoint. Prior blood transfusions, needle sharing, IV drug use, excessive alcohol use, herbal/alternative medicine use, recent changes in medications, or suicidal ideation were ruled out by meticulous history taking. Initial AST, ALT, ALP, GGT, T. bilirubin, and CK measured 783, 532, 1041, 443, 1.6, and 5,699, respectively. (Table 1) Imaging ruled out gallstone and intra-abdominal pathologies, prompting further investigation of possible infiltrative/infectious/autoimmune/neoplastic etiology. Subsequent laboratory findings ruled out thyroid abnormalities, viral or toxic hepatitis, hemochromatosis, Wilson's Disease, autoimmune hepatitis, and primary biliary cholangitis. The liver biopsy showed granulomatous hepatitis and noncaseating (non-necrotizing) epithelioid granuloma favoring sarcoid deposition (Figure 1).

Discussion: This case highlights the importance of considering hepatic sarcoidosis as a potential cause of LFT elevation, especially in a patient with a prior history of sarcoidosis. In such patients, after common causes of LFT elevations have been ruled out based on the patient's history, imaging, blood tests, and screening for autoimmune diseases, liver biopsy should be considered earlier in the management course to reach a timely diagnosis. This is especially important as not all cases of hepatic sarcoidosis require treatment, so considerations for treatment vs. closer follow-up could be decided as soon as the liver biopsy results are available. This would significantly decrease the burden on healthcare resources by avoiding unnecessary testing and aiding in timely and accurate management.

US GUIDED BIOPSY LIVER

Final Diagnosis

A. Liver, needle biopsy:
Liver needle biopsy specimen showing noncaseating (nonnecrotizing) epithelioid granuloma in one portal tract. Portal tracts, showing mild acute and chronic inflammation and ductular reaction with focal bile duct damage. Focal mild lobular inflammation and mild sinusoidal dilatation are also seen.
Trichrome stain shows portal, perivenular and perisinusoidal fibrosis. Reticulin stain highlights areas of fibrosis and otherwise normal reticulin network. Iron stain is negative for iron deposition. PASD stain shows focal nonspecific increase in intracytoplasmic granules.
GMS stain is negative for fungal organisms. AFB stain is negative for mycobacterial infection.
Findings are compatible with granulomatous hepatitis. Differential diagnosis includes sarcoidosis (favored), infectious process and primary biliary cholangitis (less likely).
Please correlate with clinical, radiologic and serologic findings.

[3226] **Figure 1.** Liver Biopsy Results.

Table 1. **LFT trend**

Test name	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day of discharge
AST	783	673	566	469	429	355	267	237
ALT	532	441	395	335	330	284	238	224
ALP	1041	920	898	822	780	715	631	653
Total Bilirubin	1.6	1.5	1.3	1.0	0.9	0.9	0.9	1.0
CK	5699	4154	3225	2721	2378	1402	725	437

S3227

A Case of Chylous Ascites From Flood Syndrome

Joyce Roy, DO[1], Natalie Vidal, MD[1], Kunal Elete, DO[1], Julia Janecki, MSPH[2], Fatima Charara, MS, DO[3], Menalee Hapuarachchi, MD[1], Long Hoang, DO[2].
[1]Medical City Fort Worth, Fort Worth, TX; [2]University of North Texas Health Science Center, Fort Worth, TX; [3]Henry Ford Macomb Hospital, Clinton Township, MI.

Introduction: Cirrhotic patients with ascites have a 20% risk of umbilical hernia development during the course of their disease due to elevated intra-abdominal pressures. In rare cases, patients with large volume ascites may develop a spontaneous umbilical hernia rupture leading to a sudden rush of ascitic fluid through a skin lesion, a complication known as Flood syndrome. The development of Flood syndrome is often provoked by local trauma or sudden rise in intra-abdominal pressure such as coughing, straining, vomiting, heavy lifting, or large volume ascites. Complications of a ruptured umbilical hernia include bowel incarceration, cellulitis, peritonitis, evisceration of the small bowel, and eventually sepsis. In this case report, we present the unique case of a patient with decompensated liver cirrhosis complicated by Flood syndrome.
Case Description/Methods: We present the case of a 56 year old male with a past medical history of decompensated cirrhosis secondary to alcoholism/chronic Hepatitis C infection complicated by ascites and hepatic encephalopathy, and chronic kidney disease of unknown stage, who presented to the ED with spontaneous leakage of fluid from his umbilical hernia. Emergent paracentesis expelled a total of 12 L milky fluid in a stream from his umbilicus and the patient was medically managed with IV albumin. Peritoneal fluid analysis revealed glucose of 144, WBC of 152, fluid protein of 1.4, and elevated triglycerides of 843, suggesting chylous ascites. A fluid serum ascites albumin gradient (SAAG) of 3 and fluid protein of 1.4 suggested the etiology was primarily cirrhosis complicated by portal hypertension. Following paracentesis, his ascites did not recur. His kidney function declined and he developed hepatorenal syndrome, deeming him a poor candidate for surgical intervention of his umbilical hernia (Figure).
Discussion: Flood syndrome is a rare complication of refractory ascites and liver cirrhosis, with a significant morbidity and mortality rate of 30%. Rupture prevention is dependent on the optimal management of underlying ascites with conventional strategies that includes diuretics, regular paracentesis, avoidance of alcohol/non-steroidal inflammatory drugs along with dietary salt and fluid restriction. Due to the complexity of syndrome, surgical treatment is not a well-established procedure and is associated with a mortality rate of up to 30%, especially in patients undergoing emergent hernia repair.

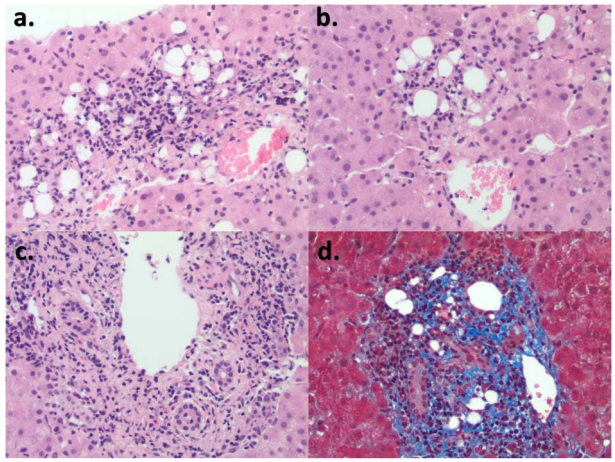
[3227] **Figure 1.** Chylous Ascities.

S3228

A Case of Immediate Hepatitis C Infection Leading to Cholestatic Hepatitis With Significant Viremia After Liver Transplantation

Parth M. Patel, MD[1], Hamna Fahad, MD[2], Hassan Zreik, MD[1], Zarqa Yasin, MD[1], Mehak Bhatia, MD[1], Syed-Mohammed Jafri, MD[2].
[1]Henry Ford Jackson, Jackson, MI; [2]Henry Ford Health System, Detroit, MI.

Introduction: With the success of direct active antiviral therapy (DAA), organs from Hepatitis-C (HCV) positive donors can be used for transplant in HCV-negative (HCV-) recipients with excellent graft survival and post-transplant course. This has expanded the donor pool to meet the accelerating need of liver transplantation in the United States. In trials involving HCV- recipients and HCV+ donors, up to 100% of the recipients achieved sustained virologic response using DAA therapy. However, the risk of HCV infection and subsequent hepatitis remained in certain patients.
Case Description/Methods: We present a case of a 55-year-old male with a medical history of atrial fibrillation and hepatic cirrhosis secondary to primary biliary cholangitis, complicated by hepatocellular carcinoma. Pre-operative HCV antibody (ab) and RNA tests were negative for the recipient. He underwent successful orthotopic liver transplantation from a deceased donor that was HCV+. The post-transplantation course was complicated by undifferentiated shock, requiring vasopressors postoperative day (POD) 1, acute renal failure requiring dialysis POD 14, and persistent ascites complicated by multi-organism peritonitis. On POD 5, the patient had worsening hyperbilirubinemia, with a peak total bilirubin of 25.6 mg/dL on POD 12. A liver biopsy suggested mild acute cellular rejection but not fibrosing cholestatic hepatitis (FCH), which was inconsistent with the degree of hyperbilirubinemia. Histology revealed peri-portal lipogranulomas that are sometimes seen with early HCV-induced liver injury. The HCV RNA load was 25,863,636 IU/mL POD 7. The patient was started on Sofosbuvir / Velpatasvir for the treatment of HCV on POD 9. There was an improvement in HCV viral load (3,097 IU/mL POD 16), hyperbilirubinemia, and well shock. (Figure)
Discussion: Acute HCV infection is rare and usually asymptomatic in immunocompetent patients; hence evidence regarding histologic appearance is limited. In post-transplant patients, HCV activation can mimic acute cellular rejection on pathology. It is recommended to start DAA within the first 7-14 days after clinical stability is achieved. Since the genotype of HCV from the donor is not routinely evaluated, it is recommended to use a pan-genotypic regimen (Glecaprevir/Pibrentasvir or Sofosbuvir/Velpatasvi). FCH is an important differential. Our patient did not have hepatitis C prior to the transplant, characteristic histopathologic findings, or a time course of more than 1 month after transplant as seen in FCH.



[3228] **Figure 1.** a & b. Some lipogranulomas are shown in portal tracts (left). In addition, hepatocyte focal necrosis evidenced by hepatocyte dropout and replaced with mononuclear cells and macrophages mixed with lipogranulomas are identified in the lobule (right). No dense lymphoid aggregate was identified, a typical feature for HCV. An unusual finding in acute cellular rejection, points to the recurrent HCV infection. c. Mild to focal moderate mixed inflammatory cell infiltrate seen in most portal tracts/areas, including neutrophils, mononuclear, eosinophils, and plasma cells, associated with scattered interface hepatitis. Focal endotheliitis is evidenced by inflammatory cells undermining the endothelial cells. Interlobular bile duct injury is focal and mild. Feature of Mild acute cellular rejection. d. Trichrome stain shows no delicate periportal strands of "chicken wire" like pericellular fibrosis; an early fibrosing cholestatic hepatitis is still under consideration.

S3229

A Case of Cholestatic Drug-Induced Liver Injury After Ashwagandha Root Supplementation

Connor Wayman, MD, Timothy Halterman, MD, FACG.
University of Nevada, Reno, NV.

Introduction: Ashwagandha root is an ancient Indian Ayurvedic herbal medicine sometimes referred to as "Indian Winter cherry" or "Indian Ginseng," that is growing in popularity for its purported benefits of weight loss, muscle building and stamina benefits, and cognition promoting effects. In vitro studies suggest possible benefits of ashwagandha to include muscle growth, exercise stamina, weight loss, hepatocellular carcinoma targeting, and others, but no large scale studies have been done to test for safety or adverse effects. Rare case reports of drug induced liver injury (DILI) related to ashwagandha have been documented.
Case Description/Methods: 19-year-old male presented to our hospital with severe pruritus and new onset jaundice after starting Ashwagandha. Two weeks after starting he developed progressive pruritus without any noted rash. Five days after the onset of pruritis he noted jaundice with nausea and dark urine, no abdominal pain. Initial labs showed a total bilirubin 7.6, AP 149, ALT 144 and AST 74. He had imaging including MRI and CT scan which were unremarkable. He was discharged with prn hydroxyzine for itching and told his DILI would resolve with time. A month since discontinuing the ashwagandha supplements his pruritus and jaundice worsened and he arrived to our hospital for evaluation. He was treated with cholestyramine, ursodiol and antihistamines. His bilirubin ultimately nadired at 30. Was also started on low dose naltrexone, and discharged. Returned for short hospital stay with severe pruritis. His t. bili remained similar compared to prior at 29, with normal INR. He was started on low dose rifampin given persistent pruritus, with mild improvement, and ultimately discharged with close follow-up.
Discussion: Ashwagandha root is a rare cause of cholestatic liver injury. The Drug Induced Liver Injury Network (DILIN) were able to identify five incidences of drug induced liver injury related to ashwagandha. The clinical course of our patient closely matches the six total cases described in the literature so far. Latency period of 2 weeks to the development of jaundice. He then had a prolonged course of cholestatic liver injury after discontinuing supplements that lasted several months. Ashwagandha-containing herbal medications can result in severe cholestatic liver injury, without the development of chronic DILI or progression to acute liver failure. This case report further re-enforces the importance of careful monitoring and awareness of our patients' supplemental health products.

Table 1. Bilirubin and symptoms trend

Days since Symptom onset	6 days	36 days	42 days	44 days	46 days	49 days	52 days	56 days	67 days
Total Bilirubin Level	7.6	25	30	29	31.3	21.2	18	16.6	-
Symptoms	Jaundice/ pruritis	Worsening jaundice/pruritis/ pale stools	Persistent pruritis	AMS vs. panic attack, persistent pruritis	Mild improvement pruritis	-	Pruritis improving	-	Pruritis resolved, visible jaundice improving

S3230

A Case of Acute Hepatitis E Infection Associated With Deer Meat in the U.S.

Kesiena J. Akpoiegbe, MD, MS, Joan Culpepper-Morgan, MD, FACG, Peter Santogade, MD, FACG.
Harlem Hospital, Bronx, NY.

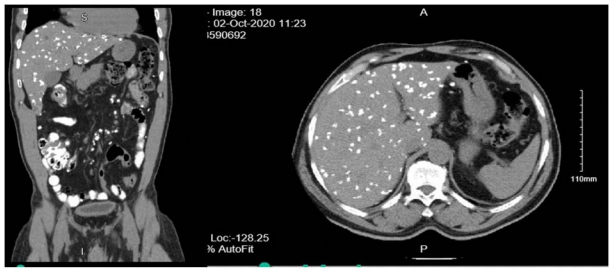
Introduction: The seroprevalence rate of hepatitis E virus (HEV)infection in the United States (U.S.) is only 6% (95% CI 5.1-6.9%). Most cases of HEV have been reported in travelers from endemic countries. Fecal oral transmission is the usual route and associated with genotypes 1 and 2. Pregnant women and the immunocompromised are especially vulnerable. Zoonotic cases of HEV acquired from the consumption of swine liver or other organ meats, are more common outside the U.S. and associated with HEV 3 and 4. Multiple potential animal reservoirs have been identified, but few cases of zoonotic infection have been confirmed within the U.S.
Case Description/Methods: A 53-year-old female with past medical history significant for depression and hyperlipidemia presented with a one-week history of worsening epigastric pain, nausea and vomiting, fatigue, and intermittent fevers after butchering several deer. She smoked 10 cigarettes/day regularly, and only drank occasionally. She had no known allergies. Her home medications included aspirin, citalopram, atorvastatin, and melatonin. She was alert and oriented with a labile affect. She had mild scleral icterus. Her abdomen was soft with epigastric tenderness. There was no hepatomegaly, spider nevi, telangiectasia, or caput medusa. Her laboratory tests revealed a mixed but mostly hepatocellular injury pattern with an ALT 2365 U/L (0-35 U/L), AST 1107 U/L (0-35 U/L), alkaline phosphatase 262 U/L (36-92 U/L), and total bilirubin 2.6 mg/dl (0.3-1.2 mg/dl). Hepatitis B, C, and A serologies were negative. Anti-smooth muscle antibody, liver-kidney-microsomal-1 antibody, anti-mitochondria antibody, and hemochromatosis screen were negative. Abdominal ultrasound did not show any biliary or pancreatic abnormalities. Further testing for Hepatitis E, IgM and IgG were positive. A diagnosis of acute HEV infection was made. She was managed conservatively with intravenous fluids and analgesics without antiviral therapy. She denied symptoms and her hepatic panel returned to normal at one month's follow up.
Discussion: This case confirms deer as a vector for the transmission of HEV to humans in the U.S. We note the proximity of Wisconsin to Canada. While the seroprevalence of HEV among deer in Canada has been estimated to range from 3.2-8.8%, the HEV RNA itself has not been detected in the animals. A prior report of multiple HEV infections from the consumption of raw deer meat has been reported in Japan.

S3231

***Armillifer armillatus*, Travel Token From Toga: Innumerable Asymptomatic Liver Calcifications**

Tracey O'Brien, MD[1], Saphwat Eskaros, MD[1], Anastasia Novikov, MD[2].
[1]Queens Hospital Center, Jamaica, NY; [2]Elmhurst Hospital Center, Elmhurst, NY.

Introduction: Armillifer armillatus found in tropical Africa, typical hosts are pythons,rodents presumed to be intermediate hosts. Accidental infection reported in humans stemming from consumption of infected snakes sold as the bushmeat. Most infections are asymptomatic, but there exist rare reports of debilitating and rarely fatal infections.
Case Description/Methods: 61 yo M with HTN, h/o malaria,chronic back pain presented for primary care visit. DX lumbar spine obtained and showed multiple calcifications,followed by CT abdomen pelvis with following findings: extensive subcentimeter calcifications throughout liver and mesentery. Laboratory data did not show LFT abnormality. CBC with elevated eosinophil count ranging 4.1-6.2%. Upon evaluation with EGD/colonoscopy, seven 5 to 10 mm submucosal polypoid lesions in the rectum, transverse colon,hepatic flexure, and cecum. One of the polypoid lesion was removed with a hot snare. Path showed completely calcified nodule in the submucosa. PAS stain failed to show any parasites, pathology, however, favoring infection such as parasite associated. Standard ova and parasite panel is negative for all organisms tested in the panel. (Figure)
Discussion: The first documentation of human infection by Armifiller armilatus dates back to 1847,several cases were since documented with noted male predominance possibly attributed to more adventurous lifestyle and greater prevalence of certain activities. Most cases of Armillifer armillatus infestations are asymptomatic with only few with mild symptoms such as dry cough attributed to pneumonitis in a setting of parasitic infection. In patients with severe infestation, there could be associated pericarditis, pleuritis, acute abdominal conditions (intestinal obstruction,viscus perforation).These acute abdominal conditions have resulted in unnecessary laparotomy before true diagnosis was made. Close surveillance using abdominal ultrasound scan, liver function test and fecal occult blood test are advised in view of the radiologically confirmed severe hepatic affectation. Diagnosis usually is made incidentally as in our case when radiographic study obtained for unrelated reasons with findings of extensive crescentic calcific lesions in the lungs, peritoneum and liver. Some patients may have deranged LFTs. This is usually seen in those with severe liver affectation as noted by some authors. Our patient had normal liver function test parameters despite severe liver involvement. Liver biopsy is required as complementary investigation tool.



[3231] **Figure 1.** innumerable subcentimeter calcified liver inclusions.

S3232

***JAK2* Positive Budd-Chiari Syndrome and Risk in Liver Transplant**

Myra Quiroga, MD, Samiha Karim, MD, Kevin Douglass, MD, Thomas Werth, MD.
Medical University of South Carolina, Charleston, SC.

Introduction: Patients that present with Budd-Chiari Syndrome (BCS) may have underlying myeloproliferative neoplasms (MPN). Long-term prognosis is taken into consideration when evaluating for transplant. Here we present a complex patient with BCS secondary to MPN.
Case Description/Methods: A 63-year-old male with new onset cirrhosis complicated by ascites and hepatic encephalopathy was hospitalized with three days of abdominal pain, distention, and diarrhea. He denied any new medications. He had no known personal or family history of liver or autoimmune diseases. He denied alcohol use. Initial laboratory evaluation included MELD-Na 15, undetectable acetaminophen, negative viral hepatitis serologies, and paracentesis was negative for infection with SAAG of 1.9. CT showed cirrhotic morphology, splenomegaly, and ascites. Ultrasound with doppler and CT venogram showed findings consistent with Budd-Chiari Syndrome (BCS). He was started on therapeutic heparin. The patient decompensated further with high MELD-Na and renal failure ultimately requiring renal replacement therapy and pressors. He was found to have a positive JAK2 V617F mutation. With leukocytosis and JAK2 mutation, bone marrow biopsy was obtained showing MPN consistent with myelofibrosis (MF). He was listed for transplant as MF was not deemed an absolute contraindication. Unfortunately, he decompensated further with variceal bleed and expired prior to transplant.
Discussion: Considerations for our patient prior to transplant included the risk of leukemic transformation in setting of immunosuppression as well as increased thrombotic risk in the peri/post-operative setting. One study evaluated the prognosis of liver transplant with BCS and MPN and effects of immunosuppression. There was no difference in survival in BCS patients with or without MPN. Progression of MPN was not noted after transplant. The major concern was, however, was his thrombotic risk. One study identified portal vein thrombosis as an independent risk factor for graft loss in transplant recipients, independent of other factors. Listing the patient for transplant was controversial for these reasons. Hydroxyurea was initiated to mitigate these risks and bridge to transplant in this otherwise healthy, young, and high functioning patient. This case highlights the rapid decline of a complex patient with BCS secondary to myeloproliferative disease and the current data available to guide decision making in the transplant evaluation process.

S3233

A Case of Acute Co-Infection With Hepatitis B Virus and Herpes Simplex Virus in an Immunocompetent Patient

Madeline K. Cleary, DO, Muhammad Farhan Ashraf, MBBS, Umer Ejaz Malik, MD, Asra Batool, MBBS.
Albany Medical Center, Albany, NY.

Introduction: While viral hepatitis is associated with several viruses, herpes simplex virus (HSV) hepatitis is a rare cause that carries a high mortality rate. Co-infection with multiple viruses can occasionally be seen in immunocompromised patients. To the best of our knowledge, there have been no cases reported in literature of acute co-infection with hepatitis B virus (HBV) and HSV occurring in a patient without human immunodeficiency virus (HIV) or other immunocompromised states. We present a case of an immunocompetent patient found to have acute hepatitis in the setting of co-infection with HBV and HSV.
Case Description/Methods: A 49-year-old female with history of IV drug abuse presented with two weeks of malaise, dark urine, pruritus, and jaundice. She reported abstinence from alcohol and illicit drug use for over four years and had no known history of liver disease. Physical examination was remarkable for scleral icterus and epigastric tenderness. Laboratory workup found AST of 1147 IU/L, ALT of 924 IU/L, total bilirubin of 7.0 mg/dL, and direct bilirubin of 4.2 mg/dL. Imaging showed hepatosplenomegaly with periportal edema and severe adenopathy at the porta hepatis, gastrohepatic ligament and periaortic lymph nodes. Hepatic viral panel was positive for hepatitis B surface antigen, hepatitis B core antibody-IgM, herpes simplex virus I and II IgM, and negative for hepatitis B surface antibody. This was consistent with acute hepatitis due to co-infection of hepatitis B and herpes simplex viruses. Further testing revealed HBV viral load of 6140000 IU/mL. Her HIV, anti-smooth muscle, and antinuclear antibodies were negative. During her hospitalization, AST peaked at 1857 IU/L, ALT peaked at 1225 IU/L, and bilirubin peaked at 42.9 mg/dL before trending down. She was started on treatment with Acyclovir and Tenofovir. Laboratory results four months later revealed normalization of liver enzymes with AST and ALT of 19 IU/L and total bilirubin of 0.5 mg/dL.
Discussion: Viral co-infection causing chronic illness is typically seen in patients with HIV or other immunocompromised conditions, although acute co-infections are still uncommon in this patient population. We report the first case of two hepatitis-causing viruses, HBV and HSV, acutely infecting a patient without a history of HIV. We recommend that HSV be considered as a differential diagnosis in patients presenting with acute hepatitis and should be included in a complete viral panel, as it can remain undetected and may cause fulminant hepatic failure.

S3234

A Cancer Encasing the Porta Hepatis: An Aggressive Intra-Abdominal B Cell Lymphoma Presenting as a Post-Obstructive Jaundice With Reactivation of Hepatitis C Virus

Amit Sah, MD, Farrell Sheehan, MD, Lesley-Ann G. McCook, MD, Larnelle Simms, MD, Kayode Olowe, MD, Akiva Marcus, MD.
University of Miami/JFK Medical Center, Atlantis, FL.

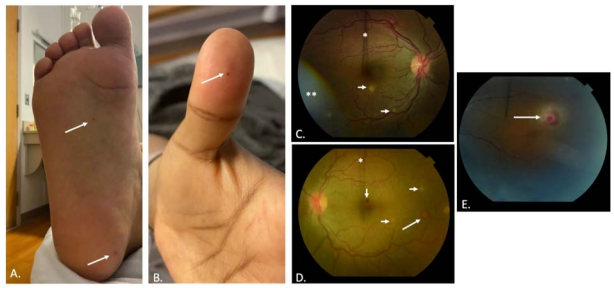
Introduction: Hepatitis C virus (HCV) is a lymphotropic virus that promotes HCV-mediated B-cell proliferation and transformation to Non-Hodgkin Lymphoma (NHL). Here we present a case of reactivation of HCV presenting as an aggressive intra-abdominal B cell lymphoma.
Case Description/Methods: A 68-years-old-male with history of eradicated HCV with recombinant interferon-alfa and resolved HBV infection presented with painless jaundice and pruritus. CT abdomen revealed a 9.6 x 11.5 x 9.5 cm heterogeneous mass causing CBD and intrahepatic biliary duct dilation. Labs revealed reactivation of HCV infection with worsening of liver function tests with total bilirubin of >40mg/dl. FNA biopsy confirmed mature B-cell lymphoma. Given persistent worsening jaundice despite biliary stent, trial of high dose steroid was initiated to improve lymphoma burden in order to get a window to initiate chemotherapy. After tumor board discussion, he received radiation therapy for tumor shrinkage for this aggressive lymphoma. Prior to initiation of planned R-CHOP chemotherapy, he developed severe sepsis with hepatic abscess. Patient and family opted for hospice care and comfort care was initiated.
Discussion: Chronic HCV is a fast-growing epidemic in the U.S.A which carries an enormous threat to general population and healthcare. HCV becomes chronic in >70% of infected patients with risk for cirrhosis, hepatocellular carcinoma and lymphoma. HCV can cause NHL via lymphocyte proliferation from viral antigenic stimulation, HCV replication inside B-cell and mutation via B-cell damage. Among patient who achieve sustained viral response (SVR), reactivation occurs in 1%, 11% and 15% among low-risk monoinfected HCV, high-risk monoinfected HCV and HIV/HCV coinfected group respectively. Treatment of HCV with resolution of NHL has been reported and there is some literature with sequential chemotherapy followed by antiviral therapy (AVT) showing promising results in HCV-NHL cases. Concurrent use of AVT and chemotherapy is primarily discouraged due to hematological toxicity. More evidence and studies need to be explored for such aggressive HCV-NHL management. Patient with SVR should have at least an annual testing and periodic follow-up to access for HCV reactivation or reinfection. Hepatitis C education regarding risk factors reduction and transmission prevention should be encouraged at every healthcare level and in general community and only then we can achieve WHO aim of eradication by 2030.

S3235

***Staphylococcus Aureus* Endocarditis Complicated by Endophthalmitis in a Young Patient with Cirrhosis**

Stavros A. Doumas, MD, MSc, Albert C. Shu, MD, Bren Davis, MD, Saraa Khan, MD, Veronica Nguyen, MD, Amol S. Rangnekar, MD.
Medstar Georgetown University Hospital, Washington, DC.

Introduction: The incidence of various infections in the setting of cirrhosis remains to be defined, and it is unclear whether liver dysfunction predisposes to specific infections. Reports in the literature suggest that liver dysfunction might be a significant risk factor for bacterial endocarditis. In this light, we present a case of a young patient with acute bacterial endocarditis complicated by endophthalmitis in the setting of AIH-induced cirrhosis.
Case Description/Methods: A 31-year-old female with a history of autoimmune hepatitis (AIH) complicated by cirrhosis presented with one day of vomiting, headaches, and fevers. The patient recently developed mild pancytopenia secondary to azathioprine and was switched to tacrolimus. Her admission Model for End-Stage Liver disease (MELD-Na) score was 21. On the first day of hospitalization, the patient endorsed blurry vision with associated eye pain and photophobia. Admission blood cultures grew methicillin-resistant Staphylococcus Aureus (MRSA). Careful examination of the extremities demonstrated characteristic Janeway lesions. An echocardiogram was significant for a thickened mitral valve with associated trace mitral regurgitation. Dilated eye examination revealed bilateral Roth spots, intraretinal hemorrhages/infiltrates, and findings consistent with endophthalmitis. The patient was initiated on intravenous vancomycin and received a left intravitreal vancomycin injection. Her condition gradually improved over two weeks, and she was discharged on a 6-week course of IV vancomycin and close ophthalmological follow-up. She completed her antibiotic course with daptomycin as she developed vancomycin-induced drug rash and acute kidney injury. Post-antibiotic fundoscopic examination demonstrated complete resolution of her eye infection and associated retinal findings. (Figure)
Discussion: We present a case of acute MRSA endocarditis complicated by endophthalmitis in a patient with cirrhosis secondary to AIH while on immunosuppression. This case underlines that cirrhosis, especially in the setting of immunosuppression, can predispose to significant and uncommon infections in the absence of traditional risk factors. The acuity of the patient's presentation and the propensity to develop severe and rare complications (endophthalmitis) indicates that these patients might have worse clinical outcomes. Those observations support previous reports in the literature. Clinicians should have a low index of suspicion to consider infectious endocarditis in cirrhotic patients.



[3235] **Figure 1.** A. Right foot with characteristic Janeway Lesions (white arrow). B. Left palm with a single prominent Janeway lesion (white arrow). C. Color fundus photographic of the right eye with an eyelash (*) obscuring the view and light artifact (**). The vitreous clear and macula are flat but a few scattered septic emboli (white arrows) are evident along with tortuous vessels. D. Color fundus photographic of left eye with an eyelash obscuring the view. There is evidence of vitritis. The macula is flat with a small associated hemorrhage (perpendicular white arrow). Vessels are tortuous. There is a prominent Roth spot (long white arrow) along the inferior arcade with a few scattered septic emboli (horizontal short white arrows). E. Magnified color fundus photographic of left eye demonstrating a single prominent Roth spot (white arrow).

S3236

***Klebsiella Pneumoniae* Invasive Syndrome: Two Cases Occurring in Northern America**

Arpine Petrosyan, MD, Jennifer Yoon, MD, Kamal Khorfan, MD, Marina Roytman, MD.
UCSF Fresno, Fresno, CA.

Introduction: Klebsiella pneumoniae invasive syndrome(KPIS) is a rare infectious disease involving a primary liver abscess with metastatic infection and is rarely reported in North America. We present two cases of KPIS with a liver abscess and concomitant bacteremia occurring in California.
Case Description/Methods: Case 1A 46 year old male with history of beta thalassemia and splenectomy presented with two days of abdominal pain and fever. He was febrile to 39.5°C, heart rate 111, and blood pressure 90/60. Labs demonstrated WBC of 32.3 x 103 uL, bilirubin 5.9mg/dL, ALT of 27 U/L, AST of 30 U/L. Computerized tomography showed a cystic mass in the liver measuring 2x2.7x2.6cm. Patient was admitted to the intensive care unit with septic shock and treated with IV antibiotics. He underwent CT guided drainage of the abscess. Both drainage and blood cultures grew pan-sensitive klebsiella pneumoniae. He improved clinically and was discharged on a four week regimen of oral antibiotics. He was seen two weeks later with no abdominal pain and imaging showing improved hepatic abscesses. Case 2 A 47 year old male presented with three days of abdominal pain. Vitals were within normal limits apart from sinus tachycardia. Labs notable for WBC 13.9 x 103uL, ALP 194 U/L, total bilirubin 1 mg/dL, ALT 91 U/L, AST 51 U/L. CT of the abdomen showed a 3x4.3x5.1cm area of decreased attenuation within the hepatic parenchyma, suspicious on initial read for fatty infiltration. He was treated with IV antibiotics but required ICU transfer for septic shock and delirium tremens. Blood cultures grew pan-sensitive Klebsiella pneumoniae. A repeat CT on hospital day 7 showed a 15 x 9.6 cm multi-septated hypodense lesion in the right hepatic lobe. CT- guided drainage was done with cultures growing Klebsiella pneumoniae. He improved clinically and was discharged home with two weeks of antibiotics. Imaging one month following discharge showed significantly near complete resolution of the abscess (Figure).

Discussion: KPIS is rare condition involving formation of a liver abscess with associated metastatic findings such as bacteremia and meningitis (1). The condition, when present, is normally witnessed in Southeast Asia and rarely reported in North America (2). Our two cases highlight the difficulty in diagnosing and treating this condition, often requiring prolonged antibiotics. It is important to have a high index of suspicion to direct appropriate diagnostic work up, typically imaging and drainage if indicated, in order to appropriately guide management.

Figure 1.

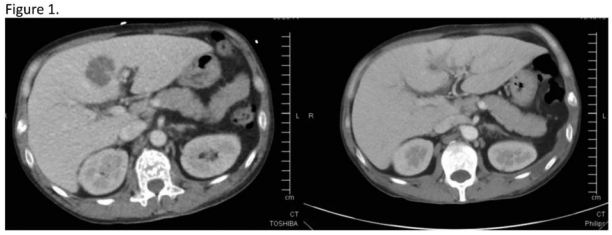


Figure 2.

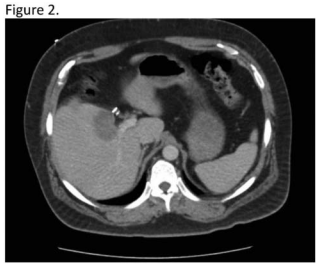
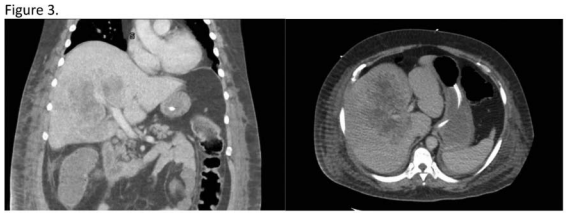


Figure 3.



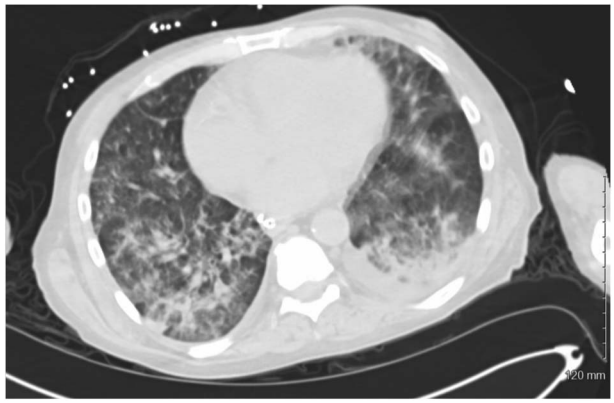
[3236] **Figure 1.** Case 1. Initial CT of the abdomen with IV contrast demonstrating the ill-defined cystic mass in the right lobe of the liver measuring 2x2.7x2.6cm (left). Repeat CT following two weeks after discharge showing decrease in size of liver abscess(Right) Figure 2. Case 2. Initial CT on admission showing area of decreased attenuation within the hepatic parenchyma measuring 3x4.3x5.1cm, stated to be possible fatty infiltration. Figure 3. Case 2. Repeat CT showing 15 x 9.6 cm multi-septated hypodense lesion in the right hepatic lobe (same image in coronal and axial views).

S3237

***Pneumocystis jirovecii* Infection in Cirrhosis: A Case Report and Review of the Literature**

Michael Meyers, MD[1], Andrew Jameson, MD[2], Curtis R. Weaver, MD[1].
[1]Mercy Health Grand Rapids/Michigan State University Internal Medicine Residency Program, Grand Rapids, MI; [2]Trinity Health Saint Mary's, Grand Rapids, MI.

Introduction: *Pneumocystis jirovecii* is a fungus that causes serious pneumonia in immunocompromised individuals most classically associated with AIDS or prolonged steroid use. Immune dysfunction is a well- nown complication of cirrhosis; however, Pneumocystis pneumonia (PCP) is not typically associated with cirrhosis as a common risk factor. We present a case of PCP in a patient with cirrhosis and review the existing cases in the literature.
Case Description/Methods: A 59-year-old male with past medical history of alcoholic cirrhosis (MELD 30, Child Pugh Class C) presented with sepsis secondary to spontaneous bacterial peritonitis. He reported he was 6 months sober. He initially improved on ceftriaxone alone; he did not receive steroids. On hospital day five he developed dyspnea and hypoxia. CT-angiogram revealed extensive ground glass opacities. Despite broad spectrum antimicrobials, his condition deteriorated resulting in invasive ventilation. Bronchoalveolar lavage was significant for *Pneumocystis jiroveci* by polymerase chain reaction. HIV testing was negative. Due to renal impairment, therapy was initiated with clindamycin and primaquine before he expired (Figure).
Discussion: This case was unique in that cirrhosis was the only identified risk factor for immunosuppression which predisposed to PCP. PCP is rare outside of risk factors including AIDS, prolonged systemic steroids and other immunosuppressive therapies, organ transplantation, hematologic malignancy, and solid tumors. There were 14 similar cases in the literature. Of the 15 total cases, 66% were male; the median age was 54. Eleven were in the setting of alcoholic hepatitis, and 9 of these received steroids. However, none of the steroid exposures were significant enough to be a classic risk factor for PCP. Common comorbidities included COPD and lymphopenia. CMV pneumonia was seen in 33% of cases. Esophageal candidiasis and Histoplasma capsulatum pneumonia were also recorded. Fatality in 87% of cases suggests that PCP in cirrhosis is associated with a poor prognosis. There are multiple possible mechanisms why these patients developed PCP. Systemic inflammation related to cirrhosis may dysregulate key inflammatory mediators required for immune response to Pneumocystis. Also, Low CD4 counts related to portal hypertension and splenic sequestration may play a role, although an effective immune response also requires interplay of B cells and natural killer cells. This case and review are important reminders of the severity of immunocompromise in cirrhosis.



[3237] **Figure 1.** CT-Angiogram with diffuse ground glass opacities.

Table 1.

Author	Date	Age/ sex	Prior steroid exposure (duration if applicable)	Etiology of cirrhosis	Additional conditions	Treatment for PCP	Death (y/n)
Ikawa	2001	40M	Y (10 days and 22 days with a 7 day interim in between)	alcohol abuse	cytomegalovirus pneumonia, esophageal candidiasis, lymphopenia	none	Y
Faria	2007	44F	present in 6 of 7 cases (median duration: 16 days)*	alcohol abuse	cytomegalovirus isolated in 3 of 7 cases*	trimethoprim/ sulfamethoxazole (TMP/ SMX)	Y
		49M		alcohol abuse		TMP/SMX	Y
		50M		alcohol abuse		TMP/SMX	Y
		53M		alcohol abuse		TMP/SMX	Y
		56F		alcohol abuse		TMP/SMX	Y
		58F		alcohol abuse		TMP/SMX	Y
		61M		alcohol abuse		TMP/SMX	Y
Dodi	2010	54F	Y (9 days)	alcohol abuse	cytomegalovirus pneumonia, hepatorenal syndrome	TMP/SMX	Y
Yee	2017	52M	N	hepatitis c	hepatitis c, lymphopenia	primaquine, clindamycin, corticosteroids, TMP/SMX	N
Hadfield	2019	63M	N	alcohol abuse	COPD	TMP/SMX	Y
Akhter	2020	67F	N	hepatitis c	COPD	steroids, antibiotics (unspecified)	N
Dugan	2020	64M	N	NASH	COPD, lymphopenia, histoplasma capsulatum pneumonia	TMP/SMX	Y
Chung	2020	43M	Y (26 days)	alcohol abuse	n/a	TMP/SMX	Y
Meyers	2022	59M	N	alcohol abuse	COPD, lymphopenia	clindamycin, primaquine	Y

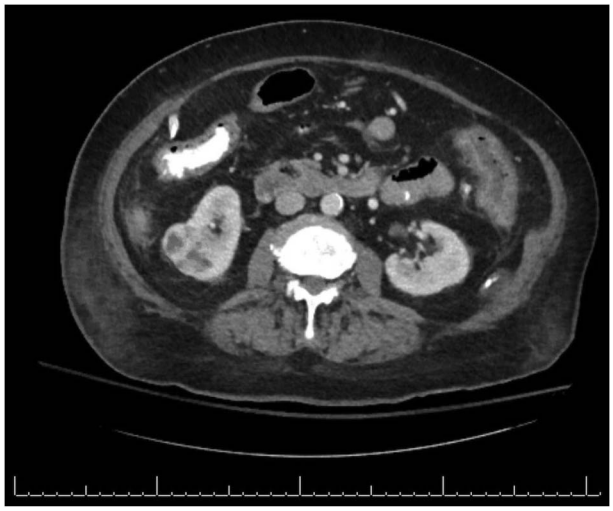
*Not specified which cases had the exposures and/or conditions.

S3238

A Case of Concurrent Cholecystitis and Stauffer Syndrome

Mark Michael, MD, Patrick J. Tempera, DO, Umer Ejaz Malik, MD, Seth Richter, MD.
Albany Medical Center, Albany, NY.

Introduction: Stauffer syndrome (SS) is a paraneoplastic syndrome seen in some malignancies, most commonly 4-15% of primary renal cell carcinomas, resulting in nephrogenic hepatic dysfunction. SS commonly results in elevated alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, and prothrombin time (PT) as well as decreased albumin in the absence of liver metastasis. The etiology of SS is unknown, although three have been suggested: elevated interleukin-6, elevated colony stimulating factor or, least likely, autoimmune. We describe a case of a woman presenting with right upper quadrant (RUQ) pain who was found to have indirect predominant hyperbilirubinemia and acute cholecystitis and renal mass on ultrasound.
Case Description/Methods: An 81-year-old female presented to an outside hospital for RUQ and epigastric pain that worsened after fatty meals. Imaging showed acute cholecystitis as well as a right renal mass. Physical exam at the time was remarkable only for diffuse tenderness to abdominal palpation, most notably in the RUQ. There was no scleral icterus or costovertebral angle tenderness on exam. Labs on presentation can be seen in Table 1. Given the elevated indirect bilirubin, gastroenterology was consulted and suggested Gilbert syndrome (GS) or SS given their concern for renal cell carcinoma (RCC). The next day the patient underwent a laparoscopic cholecystectomy notable for acute gangrenous cholecystitis with cholelithiasis, and had rapid normalization of bilirubin over 48 hours. The patient followed with outpatient urology for renal biopsy, which was positive for clear cell RCC. Follow up imaging revealed scattered pulmonary nodules up to 1.1cm, with concern for metastasis. Surgery was deemed inappropriate and the patient was conservatively managed, given her wishes to prioritize quality of life and not undergo chemotherapy. (Figure)
Discussion: This case illustrates an interesting clinical picture where a patient with confirmed cholecystitis also has a new renal mass, with elevated indirect bilirubin and PT, consistent with SS. The diagnosis of SS is made clinically, with hopeful resolution of abnormal lab values upon RCC resection. In this case, surgery was not indicated, hemolysis was ruled out with stable hemoglobin and normal peripheral smear, and the diagnoses of acute cholecystitis and RCC causing SS was made. In addition to hemolysis, GS, and Criggler-Najjar when working up indirect hyperbilirubinemia, it is imperative to include SS on the differential diagnosis.



[3238] **Figure 1.** CT abdomen with right renal mass.

Table 1. Patient labs on presentation and 2 days post-cholecystectomy

Lab	Admission	Post-Op Day 2	Normal
WBC Count (10^3 cells/uL)	17	14	4.1-9.3
Platelet (10^3/uL)	246	248	130-350
Hemoglobin (g/dL)	10.6	9.8	11-14.7
Albumin (g/dL)	3.3	2.7	3.5-5.2
Total Bilirubin (mg/dL)	2.8	0.9	0.1-1.2
Indirect Bilirubin (mg/dL)	2.4		0-0.9
Alkaline Phosphatase (IU/L)	96	102	30-115
AST (IU/L)	23	20	5-45
ALT (IU/L)	12	16	5-60
Lipase (U/L)	12		11-82
Prothrombin Time (s)	14.3		9.4-12.9
INR	1.2		0.83-1.14

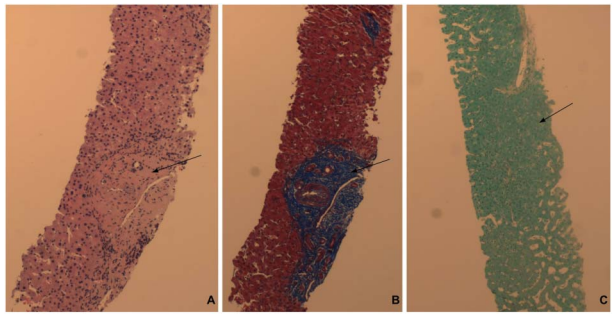
AST = aspartate transaminase, ALT = alanine transaminase.

S3239

A Case of Idiopathic Non-Cirrhotic Portal Hypertension (INCPH) in a Patient With *Klebsiella pneumoniae* Pyogenic Liver Abscess (PLA)

Yassine Kilani, MD[1], Dwayvania Miller, MD[1], Ebehiwele Ebhohon, MD, MPH[1], Mubarak Yusuf, MD[1], Mohammad Aldiabat, MD[1], Albert Yao, MS[2], Henry Ackerman, MS[2], Anjana Pillai, MD[3].
[1]Lincoln Medical and Mental Health Center, New York, NY; [2]New York Medical College, New York, NY; [3]Morristown Medical Center, New Jersey, NJ.

Introduction: Pyogenic Liver Abscess (PLA) remains rare in the United States (US). Higher incidences have been reported in Asian countries. Recent data shows that Klebsiella pneumoniae is becoming the leading bacteria in PLA internationally. Causes of bacterial seeding in the liver include biliary tract pathology, portal vein bacteriemia, systemic bacteremia, penetrating wounds or liver surgery. We describe a case of cryptogenic Klebsiella pneumoniae PLA, associated with an incidental finding of idiopathic non-cirrhotic portal hypertension (INCPH).
Case Description/Methods: A 55 year old Hispanic female, with no past medical history, presented with a 7 day history of nocturnal fever, dry cough, mild back pain, and jaundice. On admission, the patient was febrile with tachycardia and jaundice. Abdomen was soft and non tender with a negative Murphy's sign. Laboratory workup revealed a normocytic anemia, leukocytosis with neutrophilia, and evidence of cholestasis (table 1). Blood cultures were drawn. Imaging with liver ultrasound (USG), Computer Tomography (CT) and Magnetic Resonance Imaging (MRI) showed a heterogeneous cystic mass in the left hepatic lobe, a heterogeneous hepatic parenchyma and a large stone in the fundus of the gallbladder without evidence of cholecystitis, along with signs of portal hypertension. CT guided drainage showed a purulent fluid. The patient was treated with antibiotics. Klebsiella pneumoniae was isolated in both blood and pus cultures. Further workup for etiology of liver disease was inconclusive. A CT-guided liver biopsy was performed and showed evidence of non-cirrhotic portal fibrosis (NCPF) (image 1). The diagnosis of INCPH was made.
Discussion: The incidental discovery of portal hypertension in patients with underlying liver disease should prompt a full diagnostic workup to rule out liver cirrhosis and towards finding etiology for liver disease. Diagnosis of INCPH requires clinical signs of portal hypertension (ascites, esophageal &/or gastric varices, splenomegaly/ hypersplenism, porto-venous collaterals), along with patent portal veins on imaging (doppler US or CT), exclusion of cirrhosis on liver biopsy, and exclusion of liver diseases that might cause either cirrhosis (Hepatitis B &/or C, non alcoholic steatohepatitis, hemochromatosis, Wilson's disease, primary biliary cirrhosis) or INCPH (Congenital liver fibrosis, sarcoidosis, schistosomiasis). The management of INCPH, regardless of the cause, involves reducing the portal pressure to prevent variceal bleeding and death.



[3239] **Figure 1.** 1 - Normal liver parenchyma with a few chronic inflammatory cells within portal tracts (Image A, hematoxylin-eosin stain, see arrow), with mild portal fibrosis on trichrome stain (Image B, see arrow), and normal reticulin pattern of hepatic plates on reticulin stain (Image C, see arrow) indicating the absence of liver fibrosis (original magnification X100).

Table 1. Initial laboratory workup

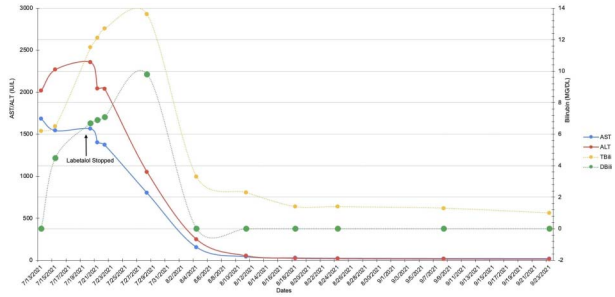
Laboratory test	Results (Normal ranges)
Complete Blood Count (CBC)	Hemoglobin 10.6 g/dL (12.0 - 16.0 g/dL) MCV 89.4 fL (80.0 - 99.9 fL) Leukocytes 12910 cells/uL (4.80 - 10.8 cells/uL) Neutrophils 86.8 % (44.0 - 77.0 %)
Liver Function Tests (LFT)	AST 35 (< = 32 U/L) ALT 49 (< = 33 U/L) ALP 301 (35 - 105 U/L) Total bilirubin 3.04 (0.20 - 1.20 mg/dL) Direct bilirubin 1.90 (0.00 - 0.30 mg/dL) Albumin 3.3 g/dL (3.5- 5.2 g/dL)
Basic metabolic panel	BUN 10.0 mg/dL (6.0 - 23.0 mg/dL) Creatinine 0.70 (0.50 - 0.90 mg/dL) Sodium 126 mmol/L (136 - 145 mmol/L) Potassium 3.7 mmol/L (3.5 - 5.1 mmol/L)
Lipid panel	Total cholesterol 91 mg/dL (< = 200 mg/dL) HDL cholesterol 19 mg/dL (>= 50 mg/dL) LDL cholesterol 53 mg/dL (< = 129 mg/dL) Triglycerides 94 mg/dL (< = 150 mg/dL)
Iron studies	Iron 26 ug/dL (30-160 ug/dL) TIBC 171 ug/dL (220-430 ug/dL) Ferritin 243 ng/dL (15/150 ng/mL)
Bacterial cultures	Blood culture: Growth Klebsiella pneumoniae Abscess fluid culture: Growth Klebsiella pneumoniae
Viral hepatitis panel	Hepatitis B surface antigen (HBsAg) non reactive Hepatitis B surface antibody (HBsAb) non reactive Hepatitis C virus (HCV) antibody non reactive
Liver parasites serology	Schistosoma antibodies IgG negative Entamoeba histolytica serology negative Echinococcus antibodies IgG negative
Autoimmune workup	Antinuclear antibodies negative Anti-smooth muscle antibodies weakly positive 1:20 Anti-LKM antibodies negative < 20.1 Antimitochondrial antibodies negative < 1:20 Anti neutrophil cytoplasmic antibodies negative
Tumor markers	Alpha fetoprotein (AFP) negative Carcinogenic Antigen 19-9 (CA-19-9) negative

S3240

A Case of Labetalol-Induced Liver Injury

Naveena Sunkara, MD[1], George M. Hanna, MD[2], Simran Gupta, MD[1], Nabil Toubia, MD[3].
[1]Brown University, Providence, RI; [2]Warren Alpert Medical School of Brown University, Providence, RI; [3]Roger Williams Medical Center, Providence, RI.

Introduction: Drug induced liver injury (DILI) is a rare but important cause of hepatotoxicity. Presentation varies widely, from mild elevations of serum aminotransferases to fulminant hepatic failure. Here, we present a case of DILI secondary to labetalol use, a rarely cited cause.
Case Description/Methods: A 36-year-old woman with a history of hypertension presented to the emergency room with 10 days of progressive painless jaundice. She switched from hydrochlorothiazide to labetalol 9 weeks prior to presentation. Vitals were within normal limits and her exam was remarkable for scleral icterus and jaundice of the skin. Laboratory investigation revealed AST 2,042 IU/L, ALT 1,402 IU/L, total bilirubin 12.1 mg/dL, direct bilirubin 6.9 mg/dL, ANA titer 1:160 and IgG 2,078 g/L. CT abdomen pelvis was unremarkable. Given clinical stability, the patient was discharged with instruction to discontinue labetalol use indefinitely. Her labs and clinical status were closely followed by outpatient GI. Her symptoms and lab abnormalities resolved within two months without further intervention (Figure 1).
Discussion: Labetalol has been associated with mild-to-moderate elevations of serum aminotransferase levels in up to 8% of patients, a rate far higher than with other beta-blockers. DILI is a diagnosis of exclusion and may pose a diagnostic challenge, especially in the setting of drugs that are not commonly implicated. Our case is consistent with other reports that describe typical symptom onset between 5 to 90 days after initial exposure to a drug. Our patient first noted symptoms 53 days after labetalol initiation and resolution of symptoms and normalization of labs 66 days after cessation. Additional associated symptoms reported with labetalol mediated DILI include jaundice, abdominal pain, pruritus, nausea and dark urine. Autoantibody formation is rare in labetolol mediated toxicity, however, it is unclear in this case whether autoantibody formation was drug mediated or found incidentally. Our patient improved with drug cessation alone and did not require steroid therapy. Providers must take care to keep a broad differential for liver injury in the absence of a typical history, as a missed diagnosis of DILI without prompt intervention may progress to fulminant liver failure.



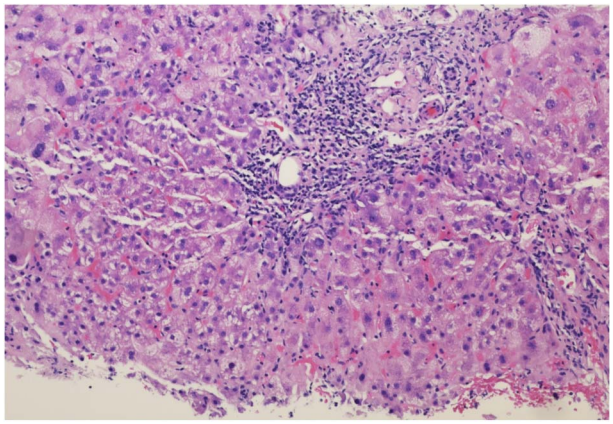
[3240] **Figure 1.** Trend of AST, ALT, Direct and Total Bilirubin after labetalol cessation.

S3241

A Case of Autoimmune Hepatitis in the Setting of the Janssen COVID-19 Vaccine

Ahamed Khalyfa, DO, Shil Punatar, DO, Mohammad Arfeen, DO, Tilemahos Spyratos, DO.
Franciscan Health, Olympia Fields, IL.

Introduction: Autoimmune hepatitis (AIH) is an inflammatory autoimmune condition that can result in chronic liver disease, it can be caused by the interaction of hereditary, epigenetic, immune, and environmental triggers. Vaccine related AIH is an uncommon phenomenon that has been reported in the presence of other vaccinations.. We present a unique case of AIH in the setting of the Janssen SARS-CoV-2 vaccine (JJ).
Case Description/Methods: A 45-year-old female with a history ofHTN, DM, GERD, and obesity presented from her primary care physician's office with abnormal liver chemistries in a hepatocellular pattern. Her hepatic profile showed AST 1119, ALT 988, AP 130, and total bilirubin of 1.5 (R-factor 22.8). Given the magnitude and rapidity of the elevated chemistries a broad serologic workup was initiated. She was negative for hepatitis A, B, and C, as well as HSV 1/2, EBV, CMV, HIV, VZV, and adenovirus. Her ferritin and iron studies did not suggest iron overload. Her ceruloplasmin and alpha 1 antitrypsin levels were within acceptable ranges. Her ANA was negative; her smooth muscle antibody was positive (1:320), IgG was elevated to 3,451 mg/dL. Her urine toxin screen was negative. She had chronically been on HCTZ and took occasionally seamoss supplements. She denied using tylenol or any other over the counter or herbal supplements. In the past month she had received a single dose of the Janssen SARS CoV-2 vaccination (Johnson and Johnson). Her liver chemistries prior were AST 17, ALT 14, AP 52, and total bilirubin of 0.3. She subsequently underwent a liver biopsy which showed moderate (grade 3) lobular inflammatory activity, mild (grade 2) periportal activity, and mild (stage 2) fibrosis with an abundance of plasma cells consistent with autoimmune hepatitis. Treatment was initiated with oral prednisone with a resulting improvement in her liver chemistries (Figure).
Discussion: Autoimmune hepatitis in the setting of recent administration COVID 19 vaccine administration has been rarely reported. We present a unique case of COVID vaccine induced autoimmune hepatitis from a non-mRNA based vaccine. It has been suggested that possible mechanisms may include molecular mimicry or bystander activation of dormant autoreactive T-helper cells for both tissue-specific and non-tissue-specific reactions. We aim to shed light to this interesting paradigm and to spark the scientific discourse to further delineate potential mechanisms leading to this uncommon complication.



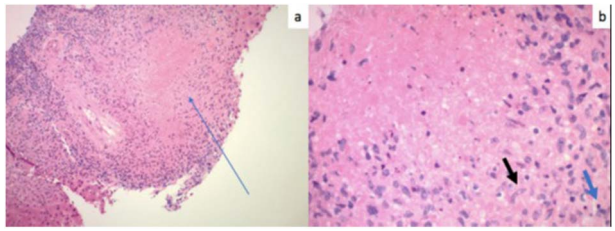
[3241] **Figure 1.** Liver biopsy which showed moderate (grade 3) lobular inflammatory activity, mild (grade 2) periportal activity, and mild (stage 2) fibrosis with an abundance of plasma cells consistent with autoimmune hepatitis.

S3242

A Case of Isolated Hepatic Tuberculosis in an Immunocompromised Patient

Irtiqa Fazili, MD, Cindy Ye, MD, Emad Qayed, MD.
Emory University School of Medicine, Atlanta, GA.

Introduction: Primary hepatic tuberculosis is a rare clinical entity with non-specific clinical and imaging features that can mimic other liver diseases, presenting a diagnostic challenge. We present a case of a patient with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) who developed fevers of unknown origin and elevated liver enzymes. Liver biopsy was key to diagnosis as it revealed necrotizing granulomas consistent with tuberculous hepatitis.
Case Description/Methods: The patient is a 54-year-old female with uncontrolled HIV/AIDS (CD4 6/1%) who presented with fever, encephalopathy, and abdominal pain. She was febrile to 38, tachycardic to 103, blood pressure 144/115, 96% on room air. She was ill-appearing, altered, nonverbal, with epigastric and lower abdominal tenderness on exam. Complete metabolic panel, complete blood count, lipase, and lactic acid were all within normal limits on presentation. CT of the abdomen and pelvis was unrevealing. On admission, she was found to have community acquired pneumonia, a urinary tract infection, a pulmonary embolism, and HIV-associated neurocognitive disorder. She was started on antiretrovirals and improved until she developed fevers of unknown origin and newly elevated liver enzymes (aspartate transaminase 400s, alanine aminotransferase 300s, alkaline phosphatase 1200s, total bilirubin 12.4, direct bilirubin 7.1) over the course of her prolonged hospitalization. A hepatic workup was largely unrevealing for etiology. Hepatitis panel, drug panel, cytomegalovirus, and Epstein-Barr virus were all negative. Autoimmune workup was weakly positive for anti-nuclear and anti-smooth muscle antibodies, but these were felt to be clinically insignificant due to low titers. The patient was also trialed on intravenous steroids with little improvement, further arguing against the diagnosis of autoimmune hepatitis. Liver biopsy revealed lobular necrotizing granulomas with scattered histiocytes and lymphocytes, consistent with tuberculous granulomatous hepatitis. She started tuberculosis therapy and tolerated it well.
Discussion: Primary hepatic tuberculosis with no clinical extrahepatic manifestations is a rare presentation and sporadically reported in the literature. This clinical case highlights the diagnostic difficulty of hepatic tuberculosis, which should be considered in immunocompromised patients with elevated liver enzymes and unrevealing initial workup. Liver biopsy is essential for diagnosis.



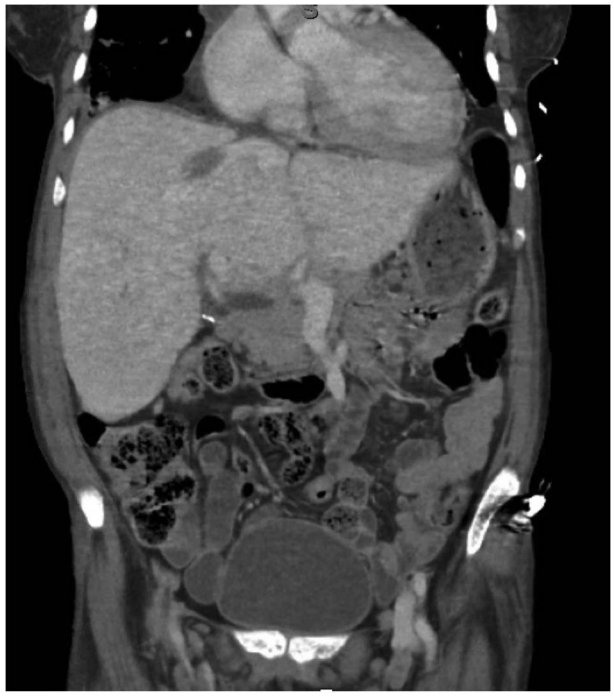
[3242] **Figure 1.** a) Lobular necrotizing granuloma; b) Center of necrotizing granuloma with histiocytes (black arrow) and lymphocytes (blue arrow).

S3243

A Budding Dilemma: A Rare Case of Sickle Cell Disease Causing Incidental Budd-Chiari Syndrome

Grace E. Kim, MD, Mary Rinella, MD.
The University of Chicago Medical Center, Chicago, IL.

Introduction: Budd-Chiari syndrome (BCS), resulting from obstruction of hepatic venous outflow, is quite rare (reported incidence varies 0.1-10 per million). The most common underlying etiologies in BCS are myeloproliferative disease, e.g. polycythemia vera and essential thrombocytosis, and less commonly, inherited thrombophilia e.g. Factor V Leiden deficiency. BCS associated with sickle cell disease (SSD) is extremely rare with case rare reports in the setting of pronounced symptoms. Here we describe a patient with SSD and hepatomegaly found to have incidental BCS.
Case Description/Methods: A 42-year-old African American female with SSD with prior acute chest syndrome, cerebrovascular accident, cocaine and heroin use, and fungal endocarditis initially presented after a motor vehicle accident. A full body computed tomography (CT) identified a segmental occlusive thrombus of the middle hepatic vein (Figure 1). She was also noted to have a large mediastinal hematoma compatible with a contained rupture of the superior vena cava (SVC). Her AST was 110 U/L (reference range 8 – 37 U/L), ALT 46 U/L (8 – 35 U/L), alkaline phosphatase of 245 U/L (30 – 120 U/L), and total bilirubin of 1.6 mg/dL (0.1 – 1.0 mg/dL). Upon further chart review, she was noted to have elevated AST, ALT, and alkaline phosphatase of 82 U/L, 36 U/L, and 184 U/L, respectively a month prior to the presentation. Given the subacute nature of BCS with no signs of acute hepatic dysfunction, the decision was made to manage conservatively, without anticoagulation, in light of the in the setting of the mediastinal hematoma, with a plan for outpatient follow-up.
Discussion: Typically, patients with BCS present with abdominal pain or signs of decompensated cirrhosis. They are managed in a step-wise fashion with anticoagulation and treatment of underlying etiology and escalated to endovascular approach or liver transplant. Our case was unique in that the thrombus was only incidentally found in the setting of trauma and that the patient likely had the thrombus prior to presentation, possibly related to sickling in the sinusoids, explaining her abnormal liver chemistries 1 month prior. While hepatic complications of sickle cell disease, such as acute hepatopathy, cholestasis and iron overload are well reported, hepatic outflow obstruction, as demonstrated here, is extremely rare. This highlights the importance of considering Budd-Chiari in the differential when evaluating the patient with SSD and abnormal liver chemistries, even in the absence of symptoms.



[3243] **Figure 1.** Computed tomography (CT) showing a segmental occlusive thrombus of the middle hepatic vein.

S3244

A Case of EBV Hepatitis Complicated by Hemolytic Anemia

Munraj Singh, MD, Scott R. Piechocki, MD, MS.
Louisiana State University Health Sciences Center, New Orleans, LA.

Introduction: Epstein-Barr virus (EBV) infection is highly prevalent in the first 2 decades of life and approximately 90% of the US population is seropositive for the infection before the age of 25. EBV infection usually manifests as the clinical syndrome of infectious mononucleosis, which presents as headaches, malaise and low-grade fevers with symptom resolution in 1-2 weeks. Other manifestations of EBV infections include evidence of mild hepatocellular damage and rarely hemolytic anemia. However, jaundice, symptomatic hepatitis and hospital admission is uncommon, specifically in the absence of infectious mononucleosis symptoms.

Case Description/Methods: A 23-year-old woman with no past medical history presented with one week history of generalized malaise, right upper quadrant abdominal pain, jaundice and dark urine. Initial laboratory studies were notable for WBC 16.3 K/uL, Hb 8.8 gm/dL, Haptoglobin < 30 mg/dL, LDH 845 U/L, Reticulocyte count 11.8 %, INR 1.0, AST 127 U/L, ALT 118 U/L, ALP 176 U/L, and Total Bilirubin 9.3 mg/dL (Direct 4.3 mg/dL, Indirect 3.5 mg/dL). CT scan of the abdomen and pelvis showed hepatomegaly with a normal biliary tree. The EBV viral capsid antigen (VCA) IgM was positive at >160 u/ML. Patient underwent a liver biopsy on hospital day 3 which showed sinusoidal patterns of inflammation and an in-situ hybridization study confirmed the diagnosis of EBV hepatitis. She was started on Solumedrol 1 mg/kg with improvement of symptoms and resolution of hepatic and hemolytic anemia lab abnormalities.
Discussion: Hepatic involvement due to Epstein-Barr virus infection can be common but is typically subclinical or mild in presentation with only 5% of patients presenting with jaundice. The pathological manifestations can be extensive, as patients can also present with hemolytic anemia, specifically cold agglutinin autoimmune hemolytic anemia. The pathogenesis is believed to be due to EBV IgM antibodies cross reacting with RBC antigens. The pathogenesis of cholestasis in EBV hepatitis involves direct damage of hepatic cells by autoantibody free radical activation and the inflammation of bile ducts. The majority of cases are self-resolving; however, antivirals such as ganciclovir in conjunction with corticosteroids can provide benefit in severe cases. Due to the high global prevalence of Epstein-Barr virus, healthcare professionals should be aware of the diagnosis, management and complications of hepatic manifestations.

Table 1. Liver Injury and Cholestasis Lab Trend From Hospital Day (HD) 1-7

	HD 1	HD 2	HD 3	HD 4	HD 5	HD 6	HD 7
AST(U/L)	127	133	159	142	95	60	35
ALT (U/L)	118	134	170	213	186	173	142
Alk Phos (U/L)	176	136	131	131	115	106	94
T. Bilirubin (mg/dL)	9.3	6.4	3.6	2.3	1.6	1.4	1.3

S3245

"Hard-to-Treat HCV"—An Approach to Retreatment With Extended Duration: A Case Report

Yara Dababneh, MD[1], Tabassum Chowdhury, BSc[2], Shiva Bongu[2], Vivek Mendiratta, MD[1], Syed-Mohammed Jafri, MD[1], Stuart Gordon, MD[1].
[1]Henry Ford Hospital, Detroit, MI; [2]Wayne State University, Detroit, MI.

Introduction: Treatment of patients with chronic hepatitis C virus (HCV) who fail to respond to Direct Acting Antivirals (DAA) remains challenging. We present a case of DAA treatment resistant chronic HCV who responded to a prolonged 1-year long course of antiviral therapy.
Case Description/Methods: A 71-year-old man with HCV genotype 1a and cirrhosis failed to respond to multiple courses of DAAs with subsequent complex NS3 protease domain and NS5B resistant associated substitutions (RAS). Among his previous salvage courses included glecaprevir-pibrentasivir-sofosbuvir-ribavirin (SOF/G/P) for 24 weeks (with relapse) followed by 24 weeks of sofosbuvir-velpatasvir-voxilaprevir-ribavirin (SOF/VEL/VOX) for 24 weeks (with relapse). We then elected to initiate a protracted course of antiviral therapy SOF/daclatasvir followed by SOF/velpatasvir (SOF/VEL), SOF/G/P, and SOF/VEL for a total duration of 56 weeks. Low doses of pegylated interferon (IFN) were added and low doses of ribavirin (RBV) as tolerated throughout the course of therapy.
Discussion: A small number of HCV patients still have DAA treatment failures or breakthroughs, which is considered a dilemma as these patients can remain chronically infected with HCV. According to the AASLD guidelines the use of the quad regimen of SOF/G/P and ribavirin for 24 weeks is a successful salvage regimen, which our patient failed. Given that our patient had failed multiple courses of currently recommended therapies for re-treatment after DAA failure, there was an absence of evidence-based options to guide treatment. Our goal, based on clinical judgment, was to achieve long-term viral suppression even if this required maintaining patient on treatment indefinitely. The patient also took IFN and RBV intermittently for the entire duration of treatment. Although there is evidence that adjuvant RBV increases the rate of Sustained Virologic Response (SVR) after DAA treatment failure, the potential beneficial role of IFN can only be speculated upon. It is also possible that some refractory cases of HCV may simply require a considerably longer span of treatment to successfully eliminate the virus. Our patient despite a history of failing to achieve SVR, was finally able to achieve SVR12 after roughly 13 months of continuous DAAs coupled with RBV and IFN as tolerated. It is possible, therefore, that extending treatment length beyond current recommendations may be necessary to successfully induce a durable response among patients with multiple RASs and previous treatment failures.

S3246

***Streptococcus Anginosus* Causing Simultaneous Infections in an Elderly Male: Empyema and Infrahepatic Abscess: Case Report and Literature Review**

Ahmed Hmidat, MD, Saba Altarawneh, MD, Joseph Simmons, MD, Hamza Mlatoum, MD, Fuad Zeid, MD.
Marshall University Joan C. Edwards School of Medicine, Huntington, WV.

Introduction: Streptococcus Anginosus is a gram-positive bacterium of the Streptococcus Viridians group. Usually presents as normal flora in the oral cavity and the gastrointestinal tract, S. Anginosus can cause pyogenic infections in multiple organs in the body especially in the immunocompromised host. We present a case of S. Anginosus multiple sites infection in an immunocompetent host that was successfully treated.
Case Description/Methods: A 75-year-old male with history of diabetes, coronary artery disease, and heart failure with reduced ejection fraction presented to ER with worsening lower abdominal pain associated with poor appetite and decreased oral intake over 3 weeks. He had cholecystectomy about a year prior to presentation. He was clinically stable on examination with mild right upper quadrant tenderness without rebound. CT abdomen with intravenous contrast showed 10*7 cm low-density fluid collection with mild rim enhancement inferior to the right hepatic lobe. He underwent CT-guided drainage of the infrahepatic collection with removal of 50 cc of purulent material and placement of an intraabdominal drain. Also, a loculated right pleural effusion was noted on CT, and 350 cc of purulent pleural fluid was drained, with analysis compatible with empyema. Both fluid cultures grew S. anginosus. Empirical antibiotics were changed to ceftriaxone, but patient had recurrence of right pleural effusion and required repeat thoracentesis. He gradually improved and completed 6 weeks of Ceftriaxone.
Discussion: S. anginosus is a normal flora that can cause infection in any organ. Most of the patients reported to have S. anginosus infection were immunosuppressed by cancer, long term steroid use, diabetes or splenectomy. It is less common for this organism to cause concurrent multiple organs infections. Published cases of simultaneous S. anginosus infection of multiple organs were limited and usually ended in poor prognosis like death or turned out to be the same infection with an anatomic connection between the organs. Our patient had the infrahepatic collection diagnosed first before the right sided pleural effusion enlarged. When the infrahepatic collection was drained, the pleural effusion size did not go down, suggesting lack of connection between both collections. S. anginosus is commonly sensitive to antibiotics. Thus, it is commonly treated with Ceftriaxone. Penicillin-intermediate or resistant S. anginosus is rare comprising < 2% of infections and does not usually lead to treatment failure.

S3247

A Case of Liver Failure Secondary to Chronic Intestinal Failure and Augmentin Use

Anita Mahadevan, BA[1], Bianca Islam, MD, PhD[2], Naemat Sandhu[2].
[1]Case Western Reserve University School of Medicine, Cleveland, OH; [2]University Hospitals Cleveland Medical Center, Cleveland, OH.

Introduction: We present a case of acute on chronic liver failure in a patient on chronic Augmentin and total parenteral nutrition (TPN). We highlight the mechanisms and key findings of liver injury associated with intestinal failure and Augmentin, which are relevant for evaluating the risks and benefits of such therapies.
Case Description/Methods: A 49-year-old woman with a history of cervical cancer treated with chemoradiation complicated by vaginal stenosis with reconstructive surgery complicated by short gut syndrome with chronic TPN dependency and chronic pelvic infections on Augmentin suppression therapy presented with hyperbilirubinemia and acute renal failure. Initial laboratory results include bilirubin 29.4 (predominantly direct), mildly elevated liver enzymes, normal alkaline phosphatase, INR 1.6, and creatinine 3.64. Her TPN and Augmentin were held. Evaluation for autoimmune markers and acute viral hepatitis serologies were negative. Genetic testing showed heterozygous C282Y mutation. Urinary copper was high with low serum ceruloplasmin, however ophthalmology exam was not concerning for Wilson's disease. Abdominopelvic non-contrast CT showed new abdominopelvic ascites. Liver biopsy revealed cholestatic hepatitis and cirrhosis. Her presentation was likely secondary to acute Augmentin hepatotoxicity in the setting of chronic intestinal-failure associated liver disease (IFALD). The patient ultimately expired due to septic shock.
Discussion: Augmentin is a known cause of DILI, with clavulanic acid established as the causative agent. Onset occurs days to months following use and liver histology reveals cholestasis, bile duct inflammation and commonly granulomas and eosinophils, which is consistent with current thinking that the DILI is an immunoallergic response. IFALD has a multifactorial disease process. Chronic cholestasis has been

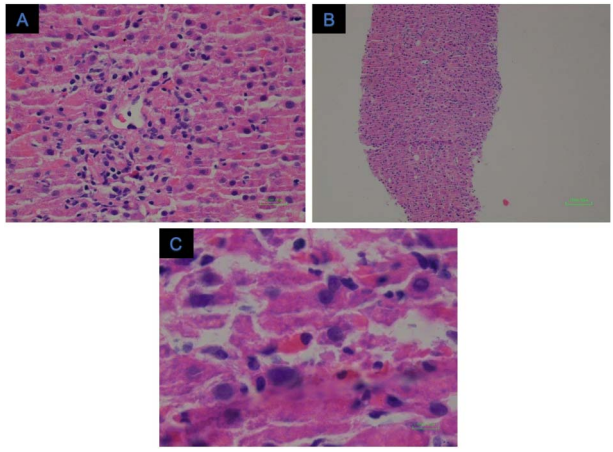
shown to occur in TPN-dependent patients, due to lack of enteral intake stimulating bile flow and hepatocyte phytosterol accumulation interfering with bile acid secretion in patients utilizing soybean-oil lipid emulsions. Steatosis has been linked to TPN-induced depletion of carnitine and choline, which results in disrupted hepatocyte lipid transport and storage. Liver histology reveals cholestasis, portal inflammation, macrosteatosis, and ductular proliferation. TPN formulas with fish-oil and olive-oil lipid emulsions are emerging as less hepatotoxic. Ursodiol may reduce cholestasis and liver injury. Death is often due to liver disease, sepsis, and primary disease.

S3248

A Case of Drug-Induced Liver Injury Secondary to Clobenzorex Use

Ushan A. Ranasinghe, MS, MD[1], Syed I. Hussaini, DO[1], Michael Gianarakis, BSc[2], Eduardo Villa, MD[1].
[1]Swedish Hospital, Chicago, IL; [2]St. George's University of London, Chicago, IL.

Introduction: Drug-induced liver injury (DILI) is a cause of both acute and chronic liver disease and is often a challenging condition for clinicians. It is a diagnosis of exclusion that requires a detailed history and workup to ensure other potential causes have been ruled out before affirming the diagnosis. There are numerous offending agents with varied presentations, often idiosyncratic in nature, with the additional challenge being a lack of specific testing or biomarkers to aid diagnosis.
Case Description/Methods: We present a case of a 25-year-old male who immigrated from Mexico 4 months ago, with no reported past medical history, who presented to the emergency department for a two-day history of postprandial non-radiating right upper quadrant and epigastric pain. Associated symptoms include subjective fevers, chills, decreased appetite, nausea, one episode of blood-tinged emesis, night sweats and dyspnea. The patient worked as a driver in Mexico and was taking two different formulations of 30mg Clobenzorex, an amphetamine pro-drug, daily for two years to help him stay awake for work. These medications were available over the counter. He had not taken it since he moved to the US. He denies the use of illicit drugs, contact with any inmates or Tuberculosis patients. At the time of his presentation, he works in a wooden floor-making company. Labs revealed a markedly elevated total bilirubin of 4.4 mg/dL, INR 1.1, AST 1325 IU/L, ALT 3151 IU/L, LDH 745 IU/L and a mildly elevated ALP 220 IU/L. Acute hepatitis and autoimmune panels were not reactive. Serum acetaminophen level was also within normal limits. Ultrasound of the abdomen and MRCP noted gallbladder wall thickening but no biliary dilatation or pathology. The patient subsequently underwent a core-needle liver biopsy that displayed lobular infiltrates of inflammatory cells of predominantly lymphocytes (Figure 1). Given the absence of positive viral serology, a diagnosis of drug-induced hepatitis was concluded.
Discussion: This case report documents a unique instance of drug-induced liver injury secondary to Clobenzorex use. This case highlights the importance of a detailed social history and should encourage clinicians not to neglect to ask about over-the-counter medications to help elucidate potential causes of liver injury. This is an important consideration, especially in the subset of patients from countries outside the United States who have access to a variety of OTC medications that are potentially hepatotoxic.



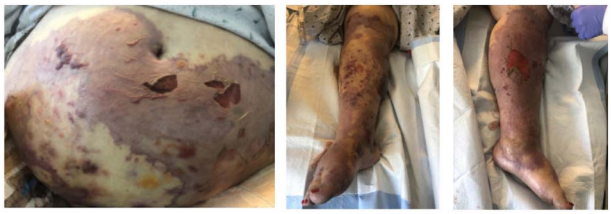
[3248] **Figure 1.** Core needle liver biopsy showing lobular infiltrates of inflammatory cells consisting of predominantly lymphocytes.

S3249

A Deadly Case of *Vibrio vulnificus* Bacteremia After Shellfish Consumption in a Patient With Cirrhosis

Madison Force, MD[1], Justin Bilello, MD[1], Louis Kishfy, MD[1], Daniel Garrido[2], David A. Sass, MD[1].
[1]Thomas Jefferson University Hospital, Philadelphia, PA; [2]MD, Philadelphia, PA.

Introduction: Infection with *Vibrio vulnificus* is the leading cause of death related to seafood consumption in the United States. The consumption of raw or undercooked seafood can cause bacteremia and sepsis with fatality rates >50%. Approximately 80% of patients who die from *V. vulnificus* infections have underlying liver disease.[1]
Case Description/Methods: The patient is a 51-year-old female with a history of decompensated alcoholic cirrhosis complicated by ascites and esophageal varices with a baseline MELD score of 17. She presented with one day of altered mental status, increased abdominal distension and lower extremity edema, skin bruising, and fever. On hospital presentation, she was afebrile, tachycardic, and normotensive. Labs revealed a Na 122 mmol/L, Cr 1.5 mg/dL, WBC 9,000 /mL, Total Bilirubin 6.8 mg/dL, INR 13, pH 7.17, lactate 13 mmol/L. Shortly after arrival, the patient had a cardiac arrest and return of spontaneous circulation was achieved after 1 round of chest compressions. She was admitted to the ICU on three vasopressors, bicarbonate, pantoprazole, and octreotide infusions, and broad-spectrum antibiotics with vancomycin and piperacillin/tazobactam. After two days she was transferred to our hospital where her exam revealed hemorrhagic bullae over abdomen and extremities (Figure 1). Her admission blood cultures eventually grew *V. vulnificus*. Upon questioning, the patient had eaten oysters and sushi three days prior to admission. Antibiotics were transitioned to doxycycline and cefepime though the patient had progressive shock and died four days after admission.
Discussion: This case demonstrates the severity and rapid progression of *V. vulnificus* infection in patients with underlying liver disease. The prognosis is linked to time to treatment. Studies have shown that patients who have treatment delayed for 72 hours have a mortality of 100%.[2] *V. vulnificus* is not covered by typical broad-spectrum antibiotics and delay in onset of treatment by enough time for a blood culture to speciate dramatically increases mortality. *V. vulnificus* is important to keep on the differential in any patient with gram-negative sepsis, skin wounds, and a history of liver disease. Questions about raw seafood and swimming in sea water should be routinely asked to guide clinicians to an early diagnosis. This case demonstrates a classic presentation of an uncommon infection and suggests that the clinician should consider empiric treatment for a *V. vulnificus* if there is a high index of suspicion.



[3249] **Figure 1.** Abdominal and lower extremity necrotizing skin wounds in a patient with V. vulnificus bacteremia.

S3250

A Hare Unusual: *Francisella tularensis* Peritonitis Following Orthotopic Liver Transplantation

Jacob Ciricillo, MD, Alexandra N. Willauer, MD, Michael Schoech, MD.
University of Cincinnati Medical Center, Cincinnati, OH.

Introduction: *Francisella tularensis* (FT) causes the rare zoonotic disease tularemia. FT is transmitted following arthropod bite, inhalation, or ingestion of contaminated water or soil. Only a couple hundred cases are reported annually by the CDC, with few in transplant recipients. In immunocompromised hosts (ICH), the clinical manifestations may be atypical and there is limited literature on the management of FT in this population.
Case Description/Methods: A 59-year-old male who was 79 days post orthotopic liver transplant (OLT) secondary to non-alcoholic steatohepatitis cirrhosis presents with acute onset generalized weakness, abdominal pain, and diarrhea. Immunosuppression included mycophenolate mofetil and tacrolimus. He was febrile to 103.2F on admission. Physical examination revealed diffuse abdominal tenderness and distension due to ascites. Laboratory tests were notable for WBC count of 8.2x10^3/μL with 96% neutrophils and a normal hepatic panel. Imaging displayed diffuse distention of the small bowel with pneumatosis. Given the broad infectious differential in an ICH, he was empirically started on cefepime, vancomycin, metronidazole, acyclovir, and micafungin. Diagnostic paracentesis revealed peritonitis with 2,850 polymorphonuclear leukocytes and gram stain with gram-negative coccobacillus. Work-up including lumbar puncture, blood cultures, and fungal and viral serologies was negative. His fever persisted and repeat ascitic fluid was unchanged at 72 hours despite empiric treatment. Ascitic fluid culture later speciated as FT confirmed with microagglutination and direct fluorescence antibody staining. When asked about exposures, the patient revealed he was a farmer and had recently runover rabbits with his tractor, requiring handling and sanitization of contaminated equipment. With transition to ciprofloxacin 500mg twice daily for 21 days, both fevers and ascites resolved.
Discussion: To our knowledge, this is the first case of FT peritonitis reported post-OLT and the third case of tularemia post-liver transplant. Our patient likely inhaled bacteria from contaminated soil or after handling infected rabbits and experienced subsequent gut translocation. Treatment with gentamicin or streptomycin is recommended in severe FT cases and doxycycline or ciprofloxacin for mild disease. Given atypical presentation in immunosuppressed hosts, we recommend consideration of tularemia as a cause of unexplained persistent fever, ascites, or lymphadenopathy post-transplant in patients with risk of FT exposure.

S3251

A Peculiar Case of Fatal Concomitant Acute Pancreatitis and Fulminant Hepatitis Due to Disseminated Varicella Zoster Infection

Dariush Shahsavari, MD[1], Praneeth Kudaravalli, MD[2], Kwabena O. Adu-Gyamfi, MBChB[1], John Erikson L. Yap, MD[3], Viveksandeep Chandrasekar, MBBS[3], Zain A. Sobani, MD[4].
[1]Medical College of Georgia - Augusta University, Augusta, GA; [2]Augusta University Medical Center, Augusta, GA; [3]Augusta University Medical College of Georiga, Augusta, GA; [4]Augusta University/Medical College of Georgia, Augusta, GA.

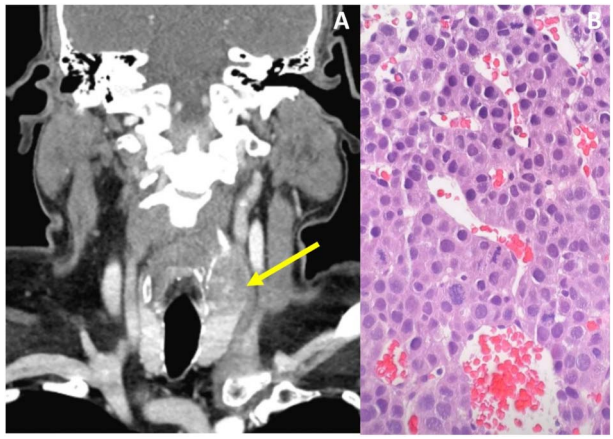
Introduction: Disseminated Varicella Zoster virus (DVZV) can be associated with elevated liver enzymes. DVZV-associated acute pancreatitis is rarely reported. Fulminant hepatitis due to DVZV is even less frequently reported and is usually deadly. We present a case of concomitant acute pancreatitis and fulminant liver failure due to DVZV in an HIV/AIDS patient.
Case Description/Methods: A 44-year-old Caucasian male with a history of asthma and traumatic splenectomy was admitted for persistent epigastric pain radiating to the back and vesicular skin rash. No history of smoking, alcohol abuse, and illicit drug use. His current medications are his asthma inhalers and fluconazole treatment for fungal infection which was started 5 days prior. His vitals were within normal limits except for tachycardia of 105 beats/min. His symptoms worsened and he started having non-bloody vomiting and oral intolerance. His vesicular rash progressed and now involved the left ear, entire abdomen, back, and extremities including palms with no pain or itching. His labs were significant for elevated lipase 273 U/L, AST 281 U/L, ALT 228 U/L, Alkaline phosphatase 216 U/L, Total bilirubin 1.2 mg/dL, with negative viral hepatitis panel, HSV, and syphilis serology. Acute and chronic liver disease work-up were all negative. HIV was reactive (CD4 count of 14). Abdominal CT showed severe edematous pancreatitis. Ultrasound and MRI did not reveal any obstructions or ductal dilatation. Patient was treated with IV fluids and symptom control for acute pancreatitis. His symptoms continued to worsen, and his mental status deteriorated. The rash became more diffuse spreading to the entire body. Skin shave biopsy showed VZV as well as positive VZV serology. Patient was started on IV acyclovir and was planned for a liver biopsy as AST and ALT above 1000 U/L with a total bilirubin of 11.9 mg/dL. Patient ultimately suffered a cardiac arrest and died a week after admission.
Discussion: DVZV infection has been reported mainly in solid organ transplants and hematologic malignancies receiving chemotherapy. Our patient had a history of splenectomy and previously undiagnosed HIV/AIDS not on treatment. There have been a few DVZV-associated acute pancreatitis cases reported, and fulminant hepatitis is even less frequent and has been often deadly. In conclusion, concomitant pancreatitis and fulminant hepatitis can occur in DVZV infections and need to be considered in the differential diagnosis especially in patients presenting with vesicular skin lesions.

S3252

A Pain in the Neck: A Rare Site for Hepatocellular Carcinoma

Esteban Grovas-Cordovi, MD, Zeyn Mirza, MD, Carla Blanco, MD, Alejandro Martino, MD, Kathia E. Rosado-Orozco, MD, Jose Martin Ortiz, MD, FACG.
VA Caribbean Health Care System, San Juan, Puerto Rico.

Introduction: Hepatocellular carcinoma (HCC) with metastasis to thyroid is very rare with few cases reported in the literature. The clinical presentation of HCC ranges from asymptomatic to hepatic encephalopathy, ascites, palpable mass in the upper abdomen, with paraneoplastic syndromes or with extrahepatic metastasis. Thyroid metastasis is uncommon as the initial presentation of HCC.
Case Description/Methods: Case of a 62 year-old man with history of compensated liver cirrhosis, likely due to alcohol. Patient was a retired worker from heavy metal processing industry. He complained of worsening right shoulder pain for several months and a growing left neck mass. Imaging studies were done and shoulder radiograph revealed the presence of a large lytic lesion at the proximal humerus. Thyroid ultrasound and neck computed tomography revealed a large complex heterogeneous hyper vascular nodularity projecting at the left aspect of the neck, concerning for malignancy. A neck biopsy was performed as thyroid malignancy was suspected. The morphology (polygonal cells in a vaguely trabecular pattern) in combination with arginase1 positivity and lack of CK7 and CK20 was more consistent with HCC. Due to the rarity of the case, bone lesion was biopsied and showed same result as the neck lesion. Further studies revealed extensive metastatic disease and presence of several liver lesions, markedly elevated Alpha-Feto protein above 60 000.00ng/dL, thyroglobulin at 6.2 and Antithryroglobuin antibody at < 1 all in favor of HCC. Patient was started on Bevacizumab with Atezolizumab and radiotherapy for bone lesions. (Figure)
Discussion: HCC is the most common primary tumor of the liver, mostly affecting patients that are diagnosed with cirrhosis, it constitutes the fifth most common cause of cancer in the world. The most common sites for HCC metastasis are the lungs, abdominal lymph nodes, adrenal glands, or bones, making distant lymph node metastases rare. Given the rarity of this case and atypical presentation of HCC, the diagnosis was delayed since the patient never developed the usual symptoms of cirrhosis despite his advanced disease stage. This case stresses the importance that there should be a low index of suspicion in patients with known risk factors for HCC and that special attention should be given to the clinical history despite such an unusual presentation and atypical disease course.



[3252] **Figure 1.** A. Coronal image of contrast-enhanced CT neck soft tissues partially showing a large complex hypervascular mass (yellow arrow) in the left aspect of the neck, involving the superior pole of the left thyroid lobe. B. Bone biopsy showing neoplastic cells with few mitoses arranged in cords, surrounded by an endothelial lining.

S3253

A Case of Recurrent Ascites Secondary to Ventriculoperitoneal Shunt in a Patient With Idiopathic Intracranial Hypertension

Reem Q. Al Shabeeb, MD[1], Dipam Shah, MD[1], Nitin K. Sardana, MD[2].
[1]Inova Fairfax Medical Campus, Falls Church, VA; [2]Gastroenterology Associates of Northern Virginia, Fairfax, VA.

Introduction: Idiopathic Intracranial Hypertension (IIH) is a rare diagnosis affecting approximately 0.9–3/100,000 adults, mostly females of childbearing age with obesity. Surgical intervention is usually indicated in cases that prove refractory to conservative measures (such as weight loss and acetazolamide) or in cases of progressive papilledema and visual deficit. Ventriculoperitoneal shunt (VPS) is the preferred internalized cerebrospinal fluid diversion method. Here, we present a case of VPS causing ascites in a woman with IIH.
Case Description/Methods: A 33-year-old female smoker with obesity, hypertension, and IIH with VPS (Medtronic strata valve, set to 1.0., placed six years ago for headache and visual problems) presents to the ED with increased abdominal distention and discomfort along with shortness of breath. She underwent a paracentesis two weeks prior for new ascites. Paracentesis showed no malignant cells. She had no family history of gastrointestinal pathology, no risk factors for viral hepatitis, and negative hepatitis serologies. She started noticing re-accumulation of fluid that did not improve with prescribed furosemide. She had no other symptoms including fevers, chills, or neurological symptoms. Physical exam showed ascites with a normal neurologic exam. Liver function tests were normal. CT abdomen and pelvis showed an unremarkable liver and VPS catheter without kinking. Pelvic ultrasound was unrevealing except for an ovarian cyst. Repeat paracentesis showed SAAG (Serum Ascites Albumin Gradient) 0.8, WBC 650 cells/ul, and only 10% polymorphonuclear cells (PMN). Culture of peritoneal fluid was positive for *Cutibacterium acnes*. The patient completed an antibiotics course and underwent a removal of the distal (peritoneal) catheter of the VPS that led to resolution of her ascites.
Discussion: We present a case of recurrent ascites resolution following VPS removal. Cerebrospinal fluid (CSF) ascites is one of the rarest intra-abdominal complications of VPS. Only 29 cases have been described in the literature and only six of them were in adults. In such cases, VPS are removed and Ventriculo-pleural or Ventriculo-atrial shunts are placed. While pathophysiology behind IIH is hypothesized to be due to inability of CSF to be reabsorbed by arachnoid granulations and extracranial lymphatics, there is no satisfactory explanation in the literature regarding the pathophysiology of CSF ascites due to VPS. It is suspected that our patient's infected VPS caused device malfunction leading to ascites.

S3254

A CCase of Seronegative Autoimmune Hepatitis During Pregnancy

Sandra Gomez-Paz, MD, Kevin Yeroushalmi, MD, Jiten Desai, MD, Rajmohan Rammohan, MD, Deepthi Kagolanu, MD, Kaleem Rizvon, Nausheer Khan, MD, Paul Mustacchia, MD, MBA.
Nassau University Medical Center, East Meadow, NY.

Introduction: Autoimmune hepatitis (AIH) is a rare disease that usually affects women at reproductive age and is associated with an increased risk of adverse maternal and fetal outcomes. Diagnosis of AIH during pregnancy, particularly in absence of autoantibodies, poses a challenge for clinicians. We present the case of a patient with new-onset seronegative AIH during pregnancy.
Case Description/Methods: A 29-year-old Hispanic female, G2P101 at 19 weeks presents complaining of yellowing of her skin and eyes for 1 week, associated with nocturnal pruritus. Physical exam showed generalized jaundice and scleral icterus. Laboratory investigations showed significantly elevated AST (452 U/L), ALT (165 U/L), alkaline phosphatase (182 U/L), hyperbilirubinemia, elevated bile acids, and elevated INR. Hepatitis panel and autoimmune work up, were negative. Abdominal ultrasound was normal. MRCP showed nonspecific hepatic parenchyma heterogeneity on T2 signal suggestive of infiltrative process. A diagnosis of intrahepatic cholestasis of pregnancy (IHCP) with atypical presentation was made, and patient was started on ursodeoxycholic acid, which improved her symptoms. At day 15, she developed lower abdominal and vaginal bleeding, caused by placental abruption. The fetus was deemed non-viable and an emergent C-section was performed. Patient was eventually discharged with outpatient GI follow up. At follow up, recurrent rise in liver function tests were noted, and repeat autoimmune work up showed elevated IgG (3600). Liver biopsy was performed, which showed active chronic inflammation with lymphoplasmacytic infiltrates, moderate interface hepatitis with ballooning degeneration and emperipolesis, and moderate portal fibrosis with bridging fibrosis, suggestive of autoimmune hepatitis. Patient was subsequently started on steroid therapy, with appropriate response as symptoms progressively resolved and liver function tests normalized.
Discussion: AIH is a rare condition that can manifest with antepartum and postpartum flares, having negative consequences to fetal and maternal outcomes, hence the importance of controlling AIH activity throughout pregnancy and in the postpartum period. Liver biopsy should be considered in cases of antibody negative AIH, however there is paucity of data regarding its safety in pregnancy. We present the rare case of an initially seronegative AIH flare during pregnancy, possibly attributed to the potential physiologic effects of pregnancy in the regulation of immune system mechanisms.

S3255

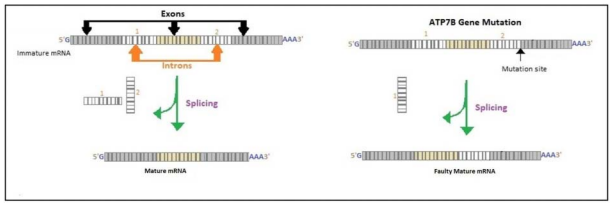
A Novel Mutation in ATP7B Gene: A Rare Manifestation of Wilson's Disease With Liver Failure

Rehmat ullah Awan, MD[1], Shazia Rashid, MD[2], Ambreen Nabeel, MBBS[1], Hrishikesh Samant, MD[3].
[1]Ochsner Rush Health System, Meridian, MS; [2]LSUHSC, Shreveport, LA; [3]Ochsner Health System, New Orleans, LA.

Introduction: Wilson's disease (WD) is a rare genetic condition characterized by excess copper build. Due to the presence of several genotypic and phenotypic variances; diagnosis can be challenging. We report a case of acute liver failure due to a novel ATP7B gene mutation, which has never been reported before.
Case Description/Methods: A 16-year-old previously healthy male presented to the hospital with abdominal pain, nausea and vomiting for 3 days. Family history was significant for a sister who passed away due to "hepatitis", he denied using alcohol or Tylenol; of note, parents immigrated from Honduras. On examination he was hemodynamically stable, jaundiced with marked scleral icterus, furthermore he was also lethargic and somnolent. His labs showed acute liver failure and acute kidney injury. Further workup included genetic testing with, alpha 1-antitrypsin, copper, ceruloplasmin and iron studies. Serum toxicology was negative. Infectious and autoimmune work-up was performed including ANA, anti-smooth muscle antibody; EBV, HSV, CMV, Hepatitis panel, VZV serologies; total IgG and subclasses, IgA, IgM, soluble IL-2, and *Treponema Cruzi*, which all came back negative other than low ceruloplasmin 6.0 mg/dL, and high 24hr urine copper excretion. Patient was diagnosed with decompensated liver failure due to Wilson's disease and thus was listed for liver transplant at MELD-Na of 41. He later underwent orthotopic liver transplant. Of note, his detailed genetic studies with whole exome sequencing (WES) identified,

two likely pathogenic ATP7B variants (c.3446G >C & c.2355G >A) (see Table-1). Postoperatively, he improved considerably. Moreover, patient and his entire family were also seen by genetic counselor once he was discharged home.

Discussion: Wilson's disease is an autosomal recessive disease involving ATP7B gene, responsible for copper metabolism. Around 1019 mutations for ATP7B gene have been reported in literature. In our patient, p.(G1149A) variant was inherited from his father and has been reported before, however p.(K785=) variant has been unreported. This mutation alters the last nucleotide of the exon and causes aberrant splicing (see Figure-1). The fact that our patient exclusively had liver failure without any other organ system involvement in the setting of a novel mutation makes it a unique case for gastroenterologists for timely diagnosis and management of WD. Early treatment with copper chelators and zinc salts can not only halt the disease progression but may also prevent end organ damage.



[3255] **Figure 1.** A diagrammatic representation of mutated splicing site. Splicing is a process whereby introns (non-coding part) of mRNA are removed allowing for joining of exons (coding part of mRNA) resulting in mature mRNA which is then used for protein synthesis. The p.(K785=) mutation in ATP7B gene destroys the splicing donor site affecting downstream protein production leading to a dysfunctional copper metabolism.

Table 1. Causative Variant(s) in Disease Genes Associated with Reported Phenotype

Gene	Disease	Mode of Inheritance	Variant	Zygosity	Inherited Form	Classification
ATP7B	ATP7B-related Wilson disease	Autosomal Recessive	c.3446 G >C p.(G1149A)	Heterozygous	Father	Likely Pathogenic Variant
ATP7B	ATP7B-related Wilson disease	Autosomal Recessive	c.2355 G >A p.(K785=)	Heterozygous	Unknown	Likely Pathogenic Variant

S3256

A Case of Syphilitic Hepatitis

Simran Gupta, MD, Rei Mitsuyama, MD, Rami Kantor, MD, Martha Sanchez, MD.
Brown University, Providence, RI.

Introduction: Syphilis is a sexually transmitted infection that is recently increasing nationwide. While it can affect many organ systems, liver involvement is uncommon. We present a case of syphilitic hepatitis in an individual with HIV presenting with abdominal pain and LFT elevations.

Case Description/Methods: A 60-year-old male with well-controlled HIV on HAART (CD4 699, undetectable VL) presented to his PCP with right upper quadrant and epigastric pain. Workup revealed elevated LFTs and he was admitted for further workup. The patient reported 2-3 weeks of abdominal pain exacerbated by eating as well as headaches, dry mouth, and nausea. He recalled a penile lesion that had healed before presentation and denied other symptoms including jaundice, fevers, and chills. On exam, he was noted to have macules and papules on his palms and chest. LFTs demonstrated AST 97, ALT 284, ALP 870, GGT 1062, and total/direct bilirubin 1.6/0.8. Other labs revealed treponema pallidum antibodies >8 (prior unavailable), a reactive RPR (titer 1:64; non-reactive 6 months prior), and lyme antibodies >8. Additionally, HAV IgM Ab, HBV SAg, HBV Core IgM Ab were non-reactive, RUQ US with incidental liver hemangioma and MRCP unrevealing. A diagnosis of syphilitic hepatitis was made and the patient was treated with penicillin G 2.4 million units weekly for 3 doses, due to unknown latency status. Within 2 months, LFTs normalized and symptoms resolved.

Discussion: Syphilis has variable presentations depending on stage (primary, secondary, tertiary) and the involved organ systems. Secondary syphilis is a systemic illness typically presenting with a characteristic rash that is disseminated and/or on the palms and soles. Syphilitic hepatitis is uncommon but when seen, presents as part of secondary syphilis with abdominal pain accompanied by disproportionate elevations in ALP and GGT rather than AST and ALT, as seen here. Definitive diagnosis is made by liver biopsy. However, combination of positive syphilis serology, lack of other identifiable hepatic insult, and the resolution of symptoms and lab abnormalities following treatment, strongly support syphilitic hepatitis as the primary diagnosis. This case should increase recognition of hepatitis as a possible manifestation of syphilis and allow proper treatment without unnecessary expensive and invasive work-up. Prompt recognition and treatment are particularly important in this case, given an increased risk for neurologic and ophthalmologic syphilis complications in individuals with HIV.

S3257

A Fatal Case of Herpes Simplex Hepatitis in Pregnancy

Dylan Lopez, MD, Bhavik Hirapara, DO, Kinnari Modi, DO, Divya Naik, DO.
Methodist Dallas Medical Center, Dallas, TX.

Introduction: Herpes Simplex Virus (HSV) hepatitis is a rare but potentially fatal presentation of HSV that can affect immunocompromised individuals such as pregnant women, particularly in the 3rd trimester. It can lead to liver failure with mortality up to 75%. Early recognition with the initiation of antivirals and supportive therapy has been shown to reduce mortality, however, insult to the liver can lead to liver failure complicated with encephalopathy and ascites. We present a case of HSV hepatitis in pregnancy which despite supportive therapy and acyclovir, resulted in demise.

Case Description/Methods: A 24 year old pregnant female at 23 weeks gestation presented to a facility with complaints of fevers and fatigue. She was discharged and treated symptomatically. 2 weeks later she developed encephalopathy, profound liver failure, and thrombocytopenia, with induction of labor and loss of the infant. She was intubated and developed shock requiring pressors. She was placed on Molecular Adsorbent Recirculating System (MARS) dialysis for support. Cultures were collected, including HSV panel, which was positive for HSV-1. Acyclovir was immediately started, and patient continued to be on supportive therapy. She improved on MARS and acyclovir, with improvement of her liver enzymes and viral load, and later extubated. Patient appeared to return to baseline, however days later, patient developed jaundice, ascites significant for spontaneous bacterial peritonitis, and encephalopathy refractory to lactulose and rifaximin. Despite acyclovir, broad-spectrum antibiotics, and prior MARS therapy, her liver enzymes increased and patient had to be reintubated, where she had multiorgan failure and expired to disseminated HSV.

Discussion: This case demonstrates a rare occurrence of HSV hepatitis in a pregnant woman. Recognition of HSV occurred once patient developed acute liver failure and encephalopathy, however, based on her history patient likely presented with HSV symptoms, such as fevers, and fatigue that remained untreated. Despite supportive therapy such as MARs and Acyclovir, patient expired of liver failure from HSV. This case shows the importance of considering HSV in the pregnant population, for if it were recognized and treated earlier, patient may not have reached fulminant liver failure resulting in death. In conclusion, HSV hepatitis and initiation of early antiviral therapy should be considered in all pregnant patients with concern for evolving hepatic failure.

S3258

A Case of Syphilitic Hepatitis (SH): An Elusive Diagnosis

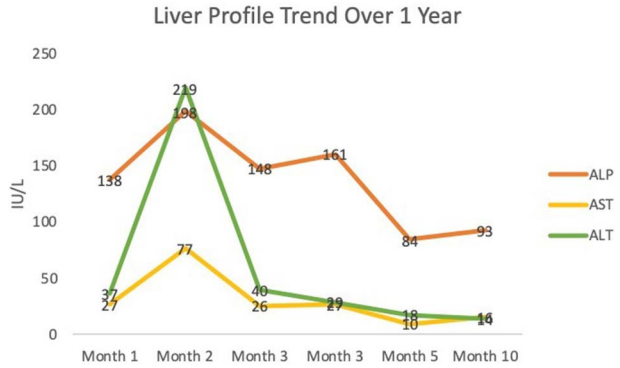
Abhishek A. Chouthai, MD[1], Alexander Garcia, DO[2], Ishita Dhawan, MD[2], Christina Capanescu, MD[2].
[1]Cooper Medical School at Rowan University, Cooper University Hospital, New Brunswick, NJ; [2]Cooper Medical School at Rowan University, Cooper University Hospital, Camden, NJ.

Introduction: Syphilis commonly known as the great imitator, is caused by a systemic bacterial infection by Treponema palladium.[1] In 2020 alone, the CDC reported 133,945 cases of syphilis, 41,655 of them from the most infectious stages of the disease: primary and secondary.[2] Given its array of clinical manifestations, syphilis can often be difficult to diagnose as reflected by its rising incidence in the US. While

clinicians are well adept at identifying the most common clinical, physical exam, and laboratory findings of syphilis infection, some of the rarer abnormalities such as liver involvement can often go unrecognized. While cutaneous involvement of syphilis is found in 90% of cases, abnormal liver enzymes, notably alkaline phosphatase (ALP), is found in 10% of diagnoses.[3,4] Liver disease, namely syphilitic hepatitis (SH), in the setting of active syphilis infection has rarely been reported due to its elusive diagnosis. Here we present a unique case of SH.

Case Description/Methods: A 46-year-old male with a chronic macular rash presented with elevated ALT, ALP, and GGT. Physical exam was remarkable for an improving macular rash for which prior biopsy was nonspecific. Repeat liver profile revealed continually elevated ALT and ALP (Figure 1) with normal total/direct bilirubin. Moreover, AMA, ESR, and CRP were found to be elevated. In this clinical context the patient was started on a steroid course for presumed primary biliary cirrhosis (PBC). Continually elevated ALP prompted ultrasound elastography of the liver which revealed hepatomegaly without evidence of steatosis or cirrhosis. Several months later, the patient developed uveitis with a positive RPR titer; therapeutic Penicillin G was initiated. On follow up, the patient was found to have complete resolution of his elevated transaminases (Figure 1). Given cholestatic liver enzyme abnormalities, positive treponemal serology, and resolution with antimicrobial therapy, diagnosis of SH was made.[5]

Discussion: Our case represents the importance of maintaining a broad differential diagnosis in hepatic enzyme abnormalities as well as a review of patient risk factors for both common and uncommon causes of hepatitis. Although patient met diagnostic criteria mimicking PBC, AMA likely was elevated in the setting of active syphilis infection and thereby highlights the importance of reviewing risk factors including polysubstance abuse, IVDU, and unprotected sexual activity which may expedite identification and management of a less common presentation of syphilis.



[3258] **Figure 1.** Patient's liver profile trend over 1 year. Notably total and direct bilirubin were normal through the patient's clinical course. Month 5 indicates post Penicillin G administration at time of positive RPR (1:256). Patient's GGT was 279 and AMA 88. [Alkaline Phosphatase (ALP), Aspartate Aminotransferase (AST), Alanine Aminotransferase (ALT)].

S3259

A Case of Propofol-Induced Acute Liver Failure

Madeline K. Cleary, DO[1], Ahmad Abulawi, MBBS[2], Patrick J. Tempera, DO[1], Ahmad Al-Dwairy, MD[2], Sonia Samuel, DO[1], Andrew Zaky, MD[1], Omar Tageldin, MD[1], Amit Chopra, MD[1].
[1]Albany Medical Center, Albany, NY; [2]Medstar Georgetown/Washington Hospital Center, Washington, DC.

Introduction: Propofol is a parenteral anesthetic routinely used for procedures given its beneficial pharmacokinetic profile of rapid onset of action and reversal. Propofol drug toxicity is rare but has been associated with idiosyncratic acute liver injury in a small number of cases. To the best of our knowledge, we present the second case of propofol-induced acute liver failure (ALF) reported in literature.

Case Description/Methods: An 88-year-old male with concern for gastrointestinal bleed underwent an upper endoscopy with propofol used as the anesthetic agent. The patient tolerated the endoscopy well with no hemodynamic compromise during or afterwards. No source of bleeding was identified so the plan was to proceed with a colonoscopy the next day. Shortly after the procedure, he was noted to have altered mental status. Evaluation with routine laboratory testing revealed elevated AST of 3248 IU/L and ALT of 2411 IU/L, both increased from 35 IU/L and 24 IU/L, respectively. He was also found to have high anion gap metabolic acidosis and bicarbonate level less than 9 mEq/L. His altered mentation continued to worsen, and he became hemodynamically unstable requiring transfer to the intensive care unit. He had no history of liver disease or excessive alcohol use, no risk factors for viral hepatitis, and took no medications known to cause liver injury. Work-up was unremarkable for acute and chronic liver disease. Propofol was ultimately surmised as the cause of ALF. Unfortunately, the patient's mental status progressively worsened despite hemodynamic stabilization and normalization of liver enzymes. His family ultimately chose to transition to comfort measures and the patient passed soon after.

Discussion: The incidence of drug-induced liver injury is increasing in developed countries and is a major etiology of ALF, accounting for greater than 50% of cases. Drug toxicity secondary to an idiosyncratic reaction is unpredictable and occurs infrequently in certain individuals. While propofol is generally considered a safe agent widely used for sedation, propofol-induced acute liver injury is a rare outcome. To the best of our knowledge, it is reported to have caused five cases of acute hepatitis and one other case of ALF. We report the second case of ALF due to propofol with a lethal outcome. We recommend close monitoring of patients treated with propofol for signs of hepatitis and that ALF due to a drug insult should be considered after common causes have been excluded.

S3260

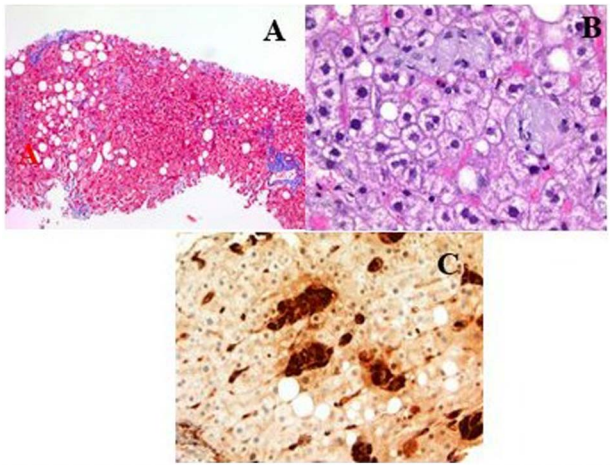
A Case of Smoldering Multiple Myeloma in a Patient With Gaucher's Disease Diagnosed on Liver Biopsy

Sadaf Afraz, DO[1], Kanwarpreet Tandon, MD[1], Ashraf Almomani, MD[2], Adalberto Gonzalez, MD[1], Asad ur Rahman, MD[1], Pablo Bejarano, MD[1], David Grossman, MD[1], Fernando Castro-Pavia, MD[1].
[1]Cleveland Clinic Florida, Weston, FL; [2]Cleveland Clinic Foundation, Cleveland, OH.

Introduction: We describe a rare case of Gaucher Disease (GD) diagnosed on liver biopsy in a patient with thrombocytopenia and hepatic steatosis with subsequent findings of smoldering multiple myeloma (SMM).

Case Description/Methods: 59-year-old male with history of chronic thrombocytopenia presented to the clinic for mild elevation in liver enzymes. At the time of evaluation, patient was asymptomatic and denied any history or risk factors for liver disease. Labs were notable for ALT 53, AST 43, Total bilirubin 0.5, alkaline phosphatase 111, INR 1, platelet 73, and ferritin 1157. Serology for hemochromatosis, autoimmune hepatitis, and viral hepatitis were negative. A CT scan of abdomen revealed hepatic steatosis and splenomegaly. Transient elastography was consistent with stage F3-F4 fibrosis. Subsequent liver biopsy showed features of steatohepatitis and sinusoidal cells with pale-gray striated cytoplasm characteristic of Gaucher's cells. No evidence of iron overload and cirrhosis on trichome stain. The degree of thrombocytopenia and splenomegaly were out of proportion to the liver disease, prompting further hematological evaluation. Serum protein electrophoresis showed 1300 mg/dL IgA kappa monoclonal protein spike. Bone marrow biopsy revealed hypercellular marrow (50%), Gaucher cells, and plasma cell neoplasm (>20%) consistent with Type I GD with smoldering multiple myeloma given absence of end organ damage. He was treated conservatively with close monitoring for disease progression. (Figure)

Discussion: GD is a rare autosomal recessive disorder characterized by deficiency of the lysosomal enzyme glucocerebrosidase that leads to accumulation of glycophospholipids in the cells of the reticuloendothelial system. It is a multisystem progressive disorder with involvement of bone marrow, liver and spleen. Type 1 GD is the most common non-neuropathic variant in adults with prevalence of 1: 1000. Predominant clinical features include thrombocytopenia, hepatosplenomegaly with variable patterns of liver involvement ranging from sub-clinical to decompensated cirrhosis. GD is often associated with laboratory abnormalities including hyperferritinemia or hypergammaglobulinemia that may lead to misdiagnosis with other chronic liver conditions. Additionally, there is an increased risk of multiple myeloma and other gammopathies in type 1 GD. Lack of awareness of early clinical course and its associated conditions can lead to delay in diagnosis and treatment.



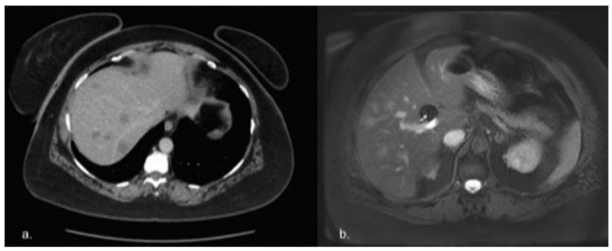
[3260] **Figure 1.** (A) There is mild pericellular fibrosis, but no periportal or bridging fibrosis. Mild steatosis supportive of a component of NASH accompany the Gaucher's cells is seen (Trichrome, 100X). (B) Sinusoidal cells are enlarged and show pale-gray striated cytoplasm representing Gaucher's cells whereas the hepatocytes are ballooned. Inflammatory infiltrates are absent(hematoxylin and eosin, 200X). (C) The Gaucher's cell are positive for the histiocytic marker CD68 (Immunohistochemistry, 200X).

S3261

A Case of Rheumatoid Nodules in the Liver

Ankur P. Patel, MD, Sarah Bohac, BS, Andrew Caruso, MD.
Baylor College of Medicine, Houston, TX.

Introduction: Rheumatoid arthritis (RA) has a prevalence of 0.5-1%. Extra-articular manifestations, including rheumatoid nodules (RNs), can be present in up to 40% of individuals. Rarely, RNs can manifest in the gastrointestinal system. We present a case of a 52-year old female with RA who presented with hepatic nodules and fever.
Case Description/Methods: A 52-year-old woman with seronegative RA on sulfasalazine 1000mg BID presented with three weeks of fevers ranging from 101 – 103 F. She had been on adalimumab until one year ago, which was stopped after she developed a psoriatic rash. Six months ago she diagnosed with an incidental left upper lobe pulmonary nodule but IR biopsy was non-diagnostic. On admission, the patient was afebrile with stable vital signs, and the following labs: WBC 25, CRP 6.44, ESR 70, and ALP 179, with normal ALT, AST, and bilirubin. A chest, abdomen and pelvis contrast CT scan showed reduced size of the previous lung nodule but multiple new hypodensities within the liver, with the largest lesion 2cm in diameter (Figure 1a). Liver MRI confirmed numerous hepatic lesions (Figure 1b). After admission, she remained afebrile and her WBC resolved without antibiotics. Infectious, malignancy, and autoimmune workup were negative. The patient underwent a colonoscopy with no concerning signs for malignancy. She then underwent an IR liver biopsy and pathology revealed multiple necrotizing granulomas with plasma cells, most concerning for RNs. Rheumatology and hepatology were consulted and planned to increase her sulfasalazine and get repeat abdominal imaging at her next visit.
Discussion: Although extra-articular disease is evident in RA, RNs in the liver are rare. A postmortem analysis of a patient from 1986 identified multiple nodules up to 5mm. In 2019, a 62 year-old female had abdominal pain and was found to have a 5cm RN in the liver. A 61 year-old male in 2020 had a 4cm liver mass resected with histology revealing a necrotizing granulomatous inflammation suggestive of a RN. Additionally, prior studies have shown that cells in RNs produce similar proinflammatory cytokines as those in synovial membranes. This could suggest cytokine release due to RNs as a cause of our patient's fevers. Although she had well controlled RA, she was also previously treated with TNF inhibitors which have been noted to cause RNs in the lungs. This may have led to her pulmonary and hepatic nodules. Overall, our case adds to the data highlighting uncommon gastrointestinal manifestations of RA.



[3261] **Figure 1.** Figures 1a and 1b. CT and MRI Studies Revealing Multiple Hepatic Hypodensities.

S3262

A Fatal Case of Acute Liver Failure Secondary to Autoimmune Hepatitis

Syed Hamaad Rahman, DO[1], Baha Aldeen Bani Fawwaz, MBBS[2], Nihal Ijaz Khan, MBBS[3], Abdul Arham, MBBS[4], Abu Hurairah, MD[2].
[1]Methodist Dallas Medical Center, Dallas, TX; [2]AdventHealth Orlando, Orlando, FL; [3]Allama Iqbal Medical College, Sarnia, ON, Canada; [4]Allama Iqbal Medical College, Mississauga, ON, Canada.

Introduction: Autoimmune hepatitis (AIH) refers to an inflammatory liver condition characterized by the circulation of autoantibodies that cause damage to the liver. AIH can present in a number of ways, varying from completely asymptomatic to severe hepatic dysfunction, making it a disease which is difficult to diagnose. Although autoimmune hepatitis is a common cause of chronic liver disease, in some rare cases it can also be the cause of acute liver failure (ALF).
Case Description/Methods: A 66-year-old male with no history of liver disease presented for progressively rising liver enzymes, jaundice, and hyperbilirubinemia. Upon arrival he complained of upper abdominal pain, fatigue, bloating, constipation, dizziness, pruritus, and loss of appetite. He noted dark colored urine, light colored stools, and 10 lbs of weight loss over the past weeks. Physical examination demonstrated icteric sclera, jaundiced skin, and mild encephalopathy. After ruling out drug-induced, viral, and alcoholic causes of acute liver failure, autoimmune hepatitis was suspected as a possible etiology. Testing revealed that the patient was positive for anti-smooth muscle antibody with a titer of 1:80. A transjugular liver biopsy with histopathological analysis revealed acute hepatitis with extensive liver cell necrosis (80%) and several plasma cells, highly indicative of autoimmune hepatitis. He was started on high dose prednisone and transplant evaluation was promptly initiated due the rapid deterioration of his liver function. On the 10th day of the patient's hospital stay, his condition rapidly declined when he became hypotensive, hypoxic, tachycardic, and tachypneic. Laboratory results at that time were consistent with severe acidosis. He was admitted to the intensive care unit where he was intubated, started on continuous renal replacement therapy and multiple vasopressors. Despite aggressive treatment, the patient expired on the 11th day of hospitalization.

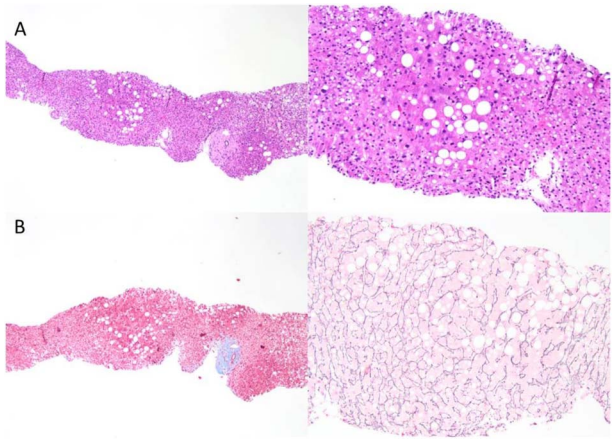
Discussion: Despite being mainly thought of as a chronic condition, AIH can manifest acutely as demonstrated by our case. In one study, of 142 cases of acute liver failure that were originally labeled as "acute liver failure of indeterminate etiology", 34 of them were found to be caused by AIH upon further investigation. This case demonstrates the importance of considering autoimmune hepatitis as a possible etiology of acute liver failure. Overlooking autoimmune hepatitis as a cause of acute liver failure may cause delays in treatment and obtaining a liver transplant.

S3263

A Case of Oxaliplatin-Induced Nodular Regenerative Hyperplasia of the Liver

Jennifer Park, MD, Bianca N. Islam, MD, PhD, Naemat Sandhu.
University Hospitals Cleveland Medical Center, Cleveland, OH.

Introduction: Nodular regenerative hyperplasia(NRH) is a form of noncirrhotic portal HTN most commonly seen in patients with multiple, recurrent vascular and infectious complications. We present an uncommon case of oxaliplatin induced NRH in a patient with rectal adenocarcinoma.
Case Description/Methods: A 64-year-old female was diagnosed with rectal adenocarcinoma T4 N2 stage. She underwent neoadjuvant chemotherapy with Capecitabine/Oxaliplatin and pelvic radiation. Her course was complicated by acute bowel obstruction requiring placement of a colonic stent which became dislodged, leading to acute bowel perforation and an emergency exploratory laparotomy. Intraoperative findings included 4 Liters of cloudy ascitic fluid, no evidence of peritoneal metastases and a normal appearing liver. She was treated with IV antibiotics, diversion ileostomy, and aggressive fluid replacement. On closer review of imaging, there was evidence of ascites 2 months prior. Fluid cytology was negative for malignant cells and SAAG was greater than 1.1 but felt to be unreliable given recent aggressive albumin therapy. Ultrasound of liver showed coarse architecture but normal portal venous flow velocities and patent hepatic veins. There was no history of alcohol abuse and hepatitis viral serologies were negative. A definitive major abdominal surgery for her rectal cancer with possible need for pelvic exenteration was being considered by the surgical team. Ascertaining the presence of portal hypertension, establishing underlying liver pathology if present, and determining etiology of ascites were all felt to be vital. Patient underwent a transjugular liver biopsy with measurement of free and wedge hepatic vein pressures. Her hepatic vein pressure gradient was 10mmHg, consistent with portal hypertension. Liver biopsy showed no fibrosis but was notable for sinusoidal congestion and nodular regenerative hyperplasia like pattern. 6 months later, patient underwent extensive abdominal surgery with curative intent. Wedge liver biopsy at time of the surgery showed liver pathology with only reactive changes. She is not currently on any chemotherapy and is in surveillance. (Figure)
Discussion: Nodular regenerative hyperplasia(NRH) can present with the insidious or unexpected onset of signs or symptoms of portal HTN (weakness, ascites, esophageal varices) in a patient with little evidence of chronic liver disease. In patients receiving chemotherapy and presenting with noncirrhotic hypertension, oxaliplatin induced NRH should remain on the differential.



[3263] **Figure 1.** A) Nodules with hyperplastic hepatocytes in the center and adjacent areas of plate atrophy. B) Reticulin stain highlights the changes.

S3264

A Hepatitis C (HCV) Infection Treatment Dilemma

Tara Koehler, PharmD, MPH[1], Kristin Harper, PA-C[1], Pooja Mude, DO[2], Praneeth Kudaravalli, MD[1], John Erikson L. Yap, MD[2].
[1]Augusta University Medical Center, Augusta, GA; [2]Augusta University Medical College of Georiga, Augusta, GA.

Introduction: HIV/HCV coinfection increases liver-related morbidity and mortality. This special population should be prioritized for treatment. Currently available direct-acting antivirals (DAAs) are safe and effective, but there are many drug interactions (DIs) to consider. Carbamazepine (CBZ) is contraindicated with all available DAAs. Patients that require treatment for HCV but cannot stop CBZ do not have a clear treatment option. Patients may be transitioned to newer antiepileptics that do not have the same contraindications, but some patients are unwilling or unable to change medications.
Case Description/Methods: Patient is a 59-year-old man with well-controlled HIV on HAART, well-controlled trigeminal neuralgia on CBZ, anemia, and non-cirrhotic (FibroScan 12 kPa), treatment naïve, chronic HCV genotype 1a. Due to DIs between DAAs and CBZ, multiple attempts were made to discontinue CBZ but were unsuccessful. Neurosurgery offered surgical gamma knife management, but up to 40% of patients still require CBZ post-procedure. An initial plan for daclatasvir (DAC)-sofosbuvir (SOF)-ribavirin (RBV) was proposed, but DAC is no longer available in the US. Patient was followed conservatively but developed thrombocytopenia and progression of fibrosis (FibroScan 14.7kPa). Given worsening liver condition, shared decision-making was performed, and patient completed 16 weeks of Glecaprevir (GLE)/pibrentasvir (PIB) + SOF 400mg with 4 weeks of ezetimibe 10mg stopping when the viral load was undetectable. Patient was instructed to start the ezetimibe with the GLE/PIB + SOF but was hesitant to take it due to adverse reaction to statins in the past. At 4 weeks of treatment, HCV viral load was still detectable. Patient was re-educated on the purpose of the ezetimibe and reassured it was not a statin. He took it from week 4-8 of treatment. At week 8, HCV viral load was undetectable. Patient achieved SVR 12 and showed improvement of his FibroScan score to 9.6 kPa.
Discussion: GLE/PIB is pangenotypic, has a high barrier to resistance, and has been proven safe and effective in combination with SOF + RBV in MAGELLAN 3 for 16 weeks duration. Patient has history of anemia, making RBV undesirable. Ezetimibe blocks the NPC1L receptor which potentially inhibits HCV entry into the hepatocyte. HCV relies on cell-to-cell transmission, so we chose to add ezetimibe 10mg daily until HCV not detected. There is a need for larger studies to determine the applicability of this case study to other patients.

S3265

A Case of Portal Vein Thrombosis Following COVID Vaccination

Fariba Rana, BS, Shannon Todd, PA-C, MHS, MPAS, Deepak Venkat, MD, Ashina Singh, MD, Syed-Mohammed Jafri, MD.
Henry Ford Health System, Detroit, MI.

Introduction: We present a case of portal vein thrombosis in a patient following COVID vaccination.
Case Description/Methods: A 44-year-old female with a past medical history of type 2 diabetes mellitus, GERD, tobacco use, asthma, chronic migraines without aura, and COVID infection presents to the emergency department with a one-week history of abdominal pain, back pain, and black stools. She is hemodynamically stable and afebrile. Her labs are significant for elevated AST and ALT at 78 and 200 respectively. CT abdomen/pelvis with IV contrast is obtained and reveals hepatic infarcts with right portal vein thrombosis. She is subsequently started on a heparin drip for treatment. Esophagogastroduodenoscopy reveals gastritis and a 5 mm ulcer in the antrum which is not actively bleeding. Antiphospholipid antibody panel and activated protein C resistance lab results are unremarkable. On further history, the patient reports that she received her first dose of the Moderna COVID vaccine about three weeks prior to presentation. Per gastroenterology, she is able to go home on apixaban 5 mg twice a day for

anticoagulation. Follow-up CT abdomen/pelvis three weeks after admission displays significantly improved enhancement pattern throughout the liver with minimal residual heterogeneous enhancement. The patient reports improvement in her abdominal pain during follow-up appointments, and her liver enzymes are trending back to normal levels. She follows up outpatient with hematology/oncology, however her hypercoagulable lab work-up continues to be unremarkable.
Discussion: This case demonstrates that portal vein thrombosis may occur following COVID vaccination. The timeline of her developing portal vein thrombosis soon after receiving the COVID vaccination may be suggestive of the vaccine precipitating her condition. This patient did have an additional risk factor of tobacco use. This can be a challenging situation for many clinicians to navigate, as COVID remains a significant threat to patients' health. Patients with hypercoagulable risk factors may benefit from close monitoring for abdominal pain or other symptoms surrounding COVID vaccination.

S3266

A Comparison of Two Cases of Acute Graft versus Host Disease Following Liver Transplant

Cody J. Timmermann, MD, Nimy John, MD, Camila C. Simoes, MD, Ragesh B. Thandassery, MD, DM, Matthew G. Deneke, MD, Mary Rude, MD.
University of Arkansas for Medical Sciences, Little Rock, AR.

Introduction: Acute graft versus host disease (aGVHD) is a rare complication of orthotopic liver transplant (OLT) with estimated 75-85% mortality. GVHD is a common complication of an allogeneic bone-marrow transplant.Incidence of aGVHD in OLT is about 0.1% and presents with rash, fever, diarrhea, and pancytopenia. Diagnosis is difficult given the non-specific symptoms, overlapping infections,drug reactions in patients on immunosuppression. Mortality is associated with bone marrow failure. Early diagnosis and treatment are vital to survival. This report aims to juxtapose two cases of aGVHD after OLT. Chart review was conducted to obtain the history and progression of two patients with post-liver transplant GVHD.
Case Description/Methods: First case is a Hispanic patient who underwent OLT for decompensated HCV cirrhosis. The patient presented 3 months postop with diffuse rash. He was admitted two weeks later with worsening rash, fever, flu-like symptoms, and diarrhea. GVHD was diagnosed by skin biopsy and treated with steroids and ruxolitinib (JAK1/2 inhibitor). The patient improved, but ultimately expired six months later due to bone marrow failure and infectious complications (invasive fungal sinusitis, VRE bacteremia, clostridium difficile infection, multifocal pneumonia). Second case is a Caucasian patient who underwent OLT for decompensated NASH cirrhosis. The patient was admitted 3 weeks postop with fever, cough, sore throat and headache. Diarrhea and rash developed shortly after and extensive infectious workup was unremarkable. aGVHD was diagnosed by skin biopsy and treated with steroids and ruxolitinib. The patient's clinical course was complicated by pseudomonas bacteremia, abdominal surgical wound infection, ecthyma gangrenosum of his right thigh requiring surgical debridements and IV antibiotics. Despite many complications, he is living to this date (over 2.5 years from the diagnosis of GVHD) and doing well. (Figure)
Discussion: Both cases of aGVHD occurred at the same center less than one month apart, but one was fatal. Both patients received MMF as part of their immunosuppression (Per studies, MMF reduces the risks of fatal GVHD). Only the second patient (who survived) received basiliximab for induction (Studies show that risks of fatal GVHD increase in association with basiliximab induction). The principal difference between the two cases is the time of symptom onset and time to treatment initiation. The patient who survived had earlier symptom onset, a rapid diagnosis, and early-onset treatment.

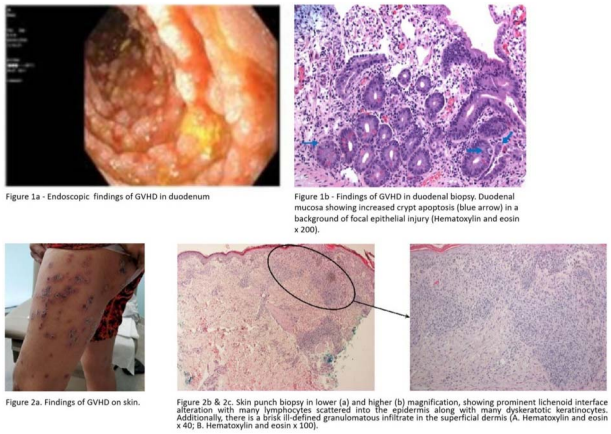


Figure 1a - Endoscopic findings of GVHD in duodenum

Figure 1b - Findings of GVHD in duodenal biopsy. Duodenal mucosa showing increased crypt apoptosis (blue arrow) in a background of focal epithelial injury (Hematoxylin and eosin x 200).

Figure 2a. Findings of GVHD on skin.

Figure 2b & 2c. Skin punch biopsy in lower (a) and higher (b) magnification, showing prominent lichenoid interface alteration with many lymphocytes scattered into the epidermis along with many dyskeratotic keratinocytes. Additionally, there is a brisk ill-defined granulomatous infiltrate in the superficial dermis (A. Hematoxylin and eosin x 40; B. Hematoxylin and eosin x 100).

[3266] **Figure 1.** GVHD Findings.

S3267

A Case of Spontaneous Resolution of Malignant Peritoneal Mesothelioma

Spogmai S. Khan, MD[1], Abdul Nadir, MD[2], Wilkins Lyndie, DO[1], Idress Suliman, MD[1].
[1]Midwestern University, Mesa, AZ; [2]Mountain Vista Medical Center, Mesa, AZ.

Introduction: Malignant peritoneal mesothelioma (MPM) is an aggressive tumor of the peritoneum. Few cases have been reported in the literature describing spontaneous regression possibly related to immune reconstitution. Here in, we report a unique case of peritoneal mesothelioma that regressed over time.
Case Description/Methods: A case of 44 y/o female with a history of severe psoriasis (on Humira) and NASH, coccidioidomycosis in 2018 (on ketoconazole) presented with jaundice and abdominal distention in September 2020. She was diagnosed with cirrhosis secondary to NASH, and DILI with a MELD score of 30. Ketoconazole and Humira were held. She was medically managed with furosemide and spironolactone. Her MELD score improved to 18. In January 2021, an orthotopic liver transplant (OLTx) was attempted but due to incidental finding of extensive peritoneal mesothelioma, this was aborted. MPM was confirmed on biopsy with cytokeratin 7+, calretinin+, WT1+, and p53 reactivity. The patient was not considered a candidate for cytoreductive surgery/hyperthermic intraperitoneal chemotherapy. She had multiple psoriatic flares which were managed with prednisone. A PET scan done in June 2022 revealed mild FDG uptake in sub-centimeter bilateral axillary and pelvic LN, cirrhotic liver, and splenomegaly. The patient denied the use of alternative medicine, except for the Atkins diet. She is now being considered for OLTx
Discussion: We present a unique case of spontaneous regression of MPM and its association with psoriatic flare. Previous case reports suggest a co-relation of immune-mediated response with spontaneous regression.[1] The immunologically relevant cytokines IF-alpha, IF-gama, and TNF-alpha can directly inhibit the growth of mesothelioma.[2] These cytokines are elevated in psoriasis, which this case highlights. Trials with immunomodulatory agents e.g. CTLA-4 inhibitor have shown promising outcomes.[3] However, little information is available about regression without any chemo/radiation/biologic therapy and the role of the innate immune response leading to tumor regression.

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S3268

A Case of Pyogenic Liver Abscesses and Pylephlebitis With Sigmoidal Diverticulitis

Aaron Fein, DO[1], Fatai Akemokwe, MD[1], Mansi Parekh, MD[1], Donald R. Campbell, MD[1], Kristen E. Fletcher, MD[2].
[1]University of Kentucky, Lexington, KY; [2]Lexington VA Health Care & University of Kentucky College of Medicine, Lexington, KY.

Introduction: Pyogenic liver abscesses (PLA) are suppurative infections in the hepatic parenchyma with high mortality. Risk factors include diabetes mellitus, underlying hepatic/biliary disease, and gastrointestinal malignancy. It is hypothesized that diverticular disease leads to disruption of the colonic mucosa, facilitating bacterial translocation into the portal venous system. We present a patient with acute sigmoidal diverticulitis complicated by pyogenic liver abscesses and septic portal vein thrombosis.
Case Description/Methods: A 71-year-old male with type 2 diabetes presented with acute onset fevers/chills, abdominal pain, and jaundice. Labs showed a leukocytosis of 19,000 IU/L and a cholestatic liver injury pattern with hyperbilirubinemia. CT abdomen and pelvis revealed right portal vein thrombosis, a right hepatic hypodense lesion, and concurrent sigmoidal diverticulitis. He was started empirically on piperacillin-tazobactam and metronidazole. *E. histolytica* serology and AFP were negative. MRCP demonstrated right hepatic duct diminution and numerous hyperintense foci in the right liver lobe. He underwent ECRP with multiple stent placements and liver biopsy, revealing acute cholangitis. Blood cultures grew *Streptococcus intermedius*. At discharge, he was prescribed amoxicillin/clavulanic acid for four weeks. At follow up two weeks later, imaging showed intrahepatic abscess resolution and interval improvement in portal vein thrombosis.
Discussion: Many case reports illustrate a relationship between pyogenic liver abscesses and sigmoidal diverticulitis. Although *Escherichia coli* and *Klebsiella* spp. are the predominate pathogens in PLA, the *Streptococcus milleri* group (including *S. intermedius*) are well documented as culprits. As seen in our patient, pylephlebitis is a known consequence of diverticulitis and treatment is aimed at the primary infection; anticoagulation remains controversial. In patients with acute diverticulitis with features of acute cholangitis, we suggest a heightened consideration of both pyogenic liver abscess and pylephlebitis

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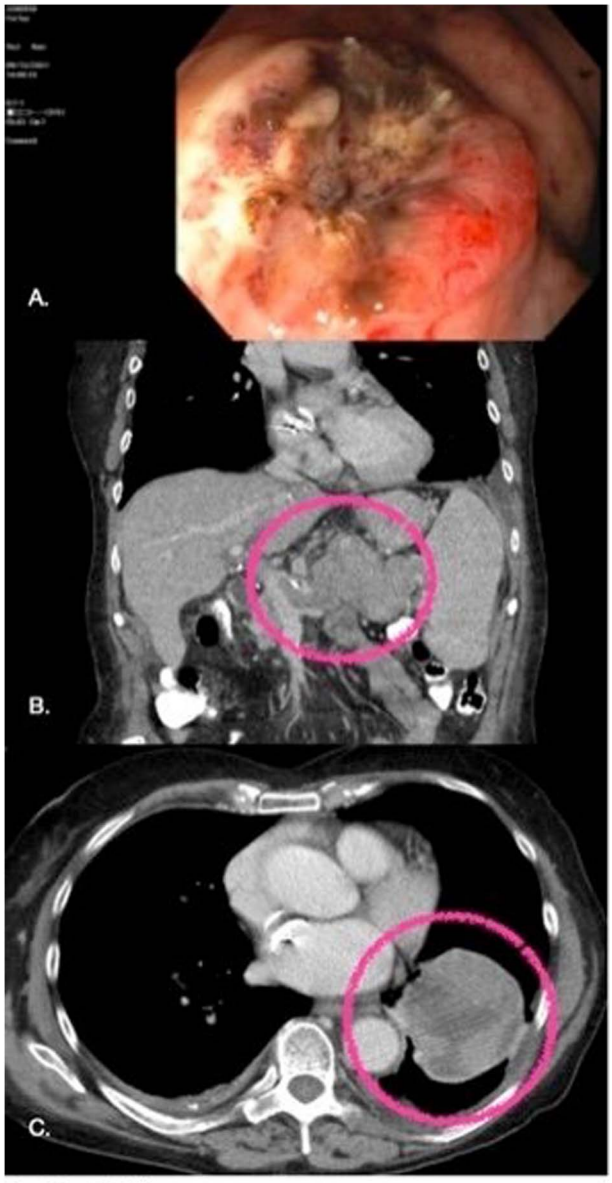
S3269

A Rare Case of HCC Recurrence as a Bleeding Gastric Ulcer

Alex Blachowicz, MD, Steven Hendler, MD, Marcel Ghanim, MD.
Loyola University Medical Center, Maywood, IL.

Introduction: Hepatocellular Carcinoma (HCC) is a potential complication of cirrhosis. Liver transplantation is often viewed as curative of HCC when fulfilling the Milan criteria prior to surgery. However, recurrence rates of HCC post-transplant remain high. Here, we present a case of post-transplant HCC recurrence presenting as a bleeding gastric ulcer.
Case Description/Methods: A 72-year-old Caucasian female presented with coffee-ground emesis. Three years prior, she had orthotopic liver transplantation due to hepatitis C virus (HCV) related cirrhosis, previously treated with Vosevi/ribavarin with sustained virologic response and HCC with evidence of moderate differentiation and focus of vascular invasion on explanted liver. Upper endoscopy revealed a cratered gastric ulcer with heaped edges. Biopsies from the ulcer edges showed poorly differentiated adenocarcinoma with signet ring features. Immunohistochemical testing showed tumor cells positive for CK8/18, Glutamine Synthetase, and polyclonal CEA with canalicular pattern; features consistent with high grade HCC. CT scan revealed a necrotic mass replacing most of the pancreatic body and tail, a lung mass and multiple vertebral compression fractures. Fine needle biopsy of pancreatic mass was consistent with metastatic carcinoma compatible with HCC. (Figure)
Discussion: Tumor recurrence post-liver transplantation is noted in 15-20% of cases after 5 years. There is no clear consensus for Standard post-transplantation monitoring. A multi-center study has proposed a protocol using the RETREAT (Risk Estimation of Tumor Recurrence Post-Transplantation) scoring system for determining screening of HCC recurrence. This includes: AFP levels prior to transplant (max score of 3 for AFP >1000), vascular invasion (max score of 2 for any evidence of invasion), and sum of the diameters of HCC lesions (max score of 3 for sum >10). In our patient, with microvascular invasion, a score of 2 would standardize her to screening every 6 months for 2 years which was consistent with the monitoring she had. The median time to HCC tumor recurrence is within 12-16 months post-transplantation, with over 75% occurring in first 2 years. Initial presentation of HCC recurrence as a direct gastrointestinal invasion is exceeding rare. There have been few case reports describing similar presentations as in our case, however, none have occurred after liver transplantation. Given its rarity, differential diagnosis of gastric ulcers in patients with history of HCC should include metastatic disease.



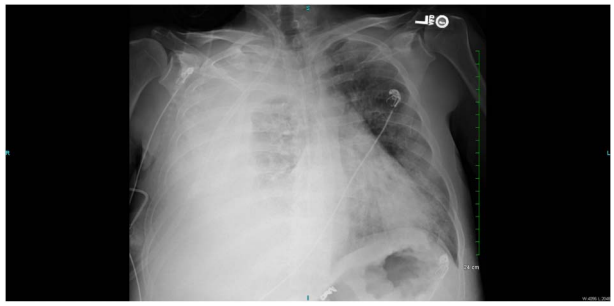
[3269] **Figure 1.** A. Gastric Ulcer B. Pancreatic Mass C. Lung Mass.

S3270

A Rare Case of Disseminated Cryptococcosis Presenting as a Pleural Effusion in a Patient With Cirrhosis

William G. Jones, MD[1], Elizabeth Craig, DO[2], Charles Duckworth, MD[3].
[1]HCA Memorial Health University Medical Center, Savannah, GA; [2]University of South Alabama, Mobile, AL; [3]Mercer School of Medicine, Savannah, GA.

Introduction: Cryptococcus neoformans is an encapsulated yeast classically know as an opportunistic infection notorious for affecting patients with acquired immunodeficiency syndrome (AIDS) and other immunosuppressed patients. Over the last few decades, there have been very few cases of pleural effusions as the initial presentation of disseminated cryptococcosis, particularly with cirrhosis as the only risk factor for an immunocompromised state. Here we present and discuss this unique case.
Case Description/Methods: A 48 y/o male with a past medical history of alcoholic cirrhosis presented to the ED with shortness of breath. His past medical history is significant for multiple hospitalizations for decompensated hepatic cirrhosis, ascites, pleural effusions, portal hypertension, gastropathy, and s/p TIPS. MELD-Na score of 31. He presents with a 3-day history of worsening shortness of breath. Chest x-ray on admission showed a right sided pleural effusion concerning for hepatic hydrothorax and pleural fluid eventually grew yeast. Pleural fluid and blood cultures later grew cryptococcus neoformans. Respiratory culture His hospital course was complicated by worsening respiratory failure requiring a short period of intubation, acute kidney injury secondary to hepatorenal syndrome and amphotericin B toxicity, and eventually his demise. (Figure)
Discussion: C. neoformans is an encapsulated yeast typically known for infecting immunocompromised hosts, classically patients with AIDS or patient's receiving immunosuppression therapy [1]. The pathophysiology normally involves inhalation causing primary lung lesion then dissemination, usually to the meninges and brain. While AIDS and patients receiving immunosuppression therapy are predisposing factors for disseminated cryptococcosis, cirrhosis appears to be an increasingly more common risk factor. Interestingly, this patient presented initially with a large pleural effusion that later grew C. neoformans with eventual disseminated cryptococcosis with fungemia. In a 2015 case report by Wang et al, they reported only 5 documented cases of pleural effusion as the initial clinical presentation of disseminated cryptococcosis in the English language literature [2]. The 5 patients studied all had an obvious cause of immunosuppression, making our patient with cirrhosis an even more unique case.



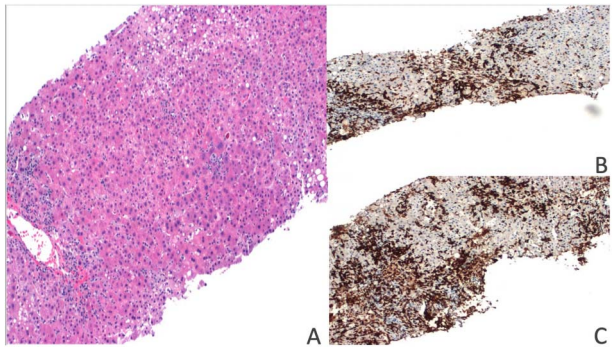
[3270] **Figure 1.** AP chest X-ray from admission showing right-sided pleural effusion with associated passive atelectasis and near complete opacification of the right hemithorax.

S3271

A Rare Case of Pembrolizumab-Induced Hepatitis

Hunza Chaudhry, MD, Joanne Lin, DO, Aalam Sohal, MD, Alakh Gulati, MD, Marina Roytman, MD.
UCSF-Fresno, Fresno, CA.

Introduction: The recent use of immune checkpoint inhibitors (ICI) has contributed to major breakthroughs in cancer therapy. Pembrolizumab is a monoclonal antibody used for many solid tumors. We report a case of a patient with gastric adenocarcinoma who developed drug-induced liver injury (DILI) from pembrolizumab.
Case Description/Methods: A 64-year-old woman with a history of latent tuberculosis on isoniazid and gastric adenocarcinoma undergoing pembrolizumab immunotherapy presented for elevated liver enzymes noticed during an outpatient visit. The patient was asymptomatic, but laboratory values revealed AST (aspartate transaminase) 227 U/L, ALT (alanine transaminase) 494 U/L, alkaline phosphatase 217 U/L, and total bilirubin 1.4 mg/dL. Complete blood count (CBC) was unremarkable. She had received seven doses of pembrolizumab, with the last dose being four weeks before admission. Computed tomography (CT) of the abdomen and magnetic resonance cholangiopancreatography (MRCP) were unremarkable. Considering a RUCAM (Roussel Uclaf Causality Assessment Method) score of 5 and a possibility for DILI, liver biopsy was pursued. Biopsy results revealed small clusters of plasma cells (CD3+/CD8+) and numerous apoptotic hepatocytes with evidence of confluent centrizonal necrosis (Figure 1). Given the immunohistochemical staining pattern, a diagnosis of immune checkpoint inhibitor hepatitis was considered more likely. Pembrolizumab was discontinued, methylprednisolone and ursodiol were initiated. Patient's liver enzymes improved with discontinuation of pembrolizumab and transition of isoniazid to another anti-tubercular agent. Ultimately, pembrolizumab was resumed outpatient with close monitoring.
Discussion: Pembrolizumab is a highly selective, humanized monoclonal antibody that inhibits lymphocytes' PD-1 receptors, allowing an immune response against cancer cells. The risk of liver injury is higher when combined with other hepatotoxic drugs, as demonstrated in our patient. Treatment consists of suspected medication cessation and immunosuppressants. Following cessation of ICI's, liver enzymes usually normalize within weeks, as seen in our patient. Although not required for diagnosis, a liver biopsy can help rule out other etiologies. As ICIs become more widespread in the fight against malignancies, recognition of their adverse effects can lead to early diagnosis, intervention, and initiation of life-saving treatment.



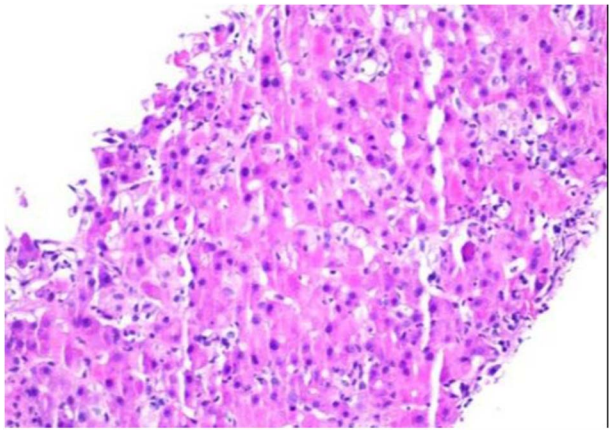
[3271] **Figure 1.** A. H&E stain showing apoptotic hepatocytes and centrizonal vein with perivenular inflammation and hepatocellular dropout. B&C. Immunohistochemical staining positive for CD8 and CD3 lymphocytes.

S3272

A Potential New Use for Tocilizumab: A Case of Refractory Checkpoint Inhibitor Hepatitis

Laine A. Lyles, DO[1], Reagan Farmer[2].
[1]Prisma Health-Midlands/ University of South Carolina, Lexington, SC; [2]University of South Carolina School of Medicine, Columbia, SC.

Introduction: Immune checkpoint inhibitors (CPI) are becoming increasingly common treatment options for several types of cancers. While their use in cancer treatment is very promising, these medications are not without side effects. Specifically, these drugs are commonly associated with hepatic adverse events, including auto-immune hepatitis (AIH). In fact, in one study, AIH was found in approximately 5% of people treated with a CPI. While we know that CPI hepatitis is a potential outcome of treatment with CPIs, we have yet to establish the best treatment options for this adverse event. Based on expert opinion, the recommended course of treatment involves high dose steroids and immunomodulators if needed. There is not, however, much literature on management of CPI hepatitis that is refractory to the typical treatments, though use of tocilizumab has shown some success previously in treatment of immune-related adverse events secondary to CPIs .
Case Description/Methods: A 35 y.o. female with recurrent right renal cell carcinoma, status-post resection with nephrectomy, on palliative treatment with nivolumab/ipilimumab, presented to the hospital for further evaluation of elevated liver function tests (LFT) and an abdominal MRI concerning for hepatitis. At time of admission, she was on her 3rd week of steroids, started with concern for drug-induced autoimmune hepatitis due to her checkpoint inhibitor (CPI) treatment. At admission, she was started on IV steroids and mycophenolic acid. LFTs and bilirubin continued to rise despite several days of this treatment and she was given a dose of rituximab. Liver biopsy was obtained which was consistent with CPI induced hepatitis. LFTs and bilirubin continued to rise and tocilizumab was given. 4 days after that treatment, AST and ALT were nearly half their pretreatment values and bilirubin began downtrending. At this point, the patient was stable enough for discharge with very close follow up (Figure).
Discussion: Studies from 2019 reveal that 43.68% of all people with cancer in the United States are eligible for CPI therapy. This number translates to 7.9 million patients who are potentially eligible for CPI therapy. With the incidence of severe CPI hepatitis suspected to be up to 20% of all people taking CPIs, this equates to 1.58 million people of whom could be affected. More studies need to be done to address treatment for refractory auto-immune CPI hepatitis, and to better understand the underlying pathogenesis in this rapidly growing population.



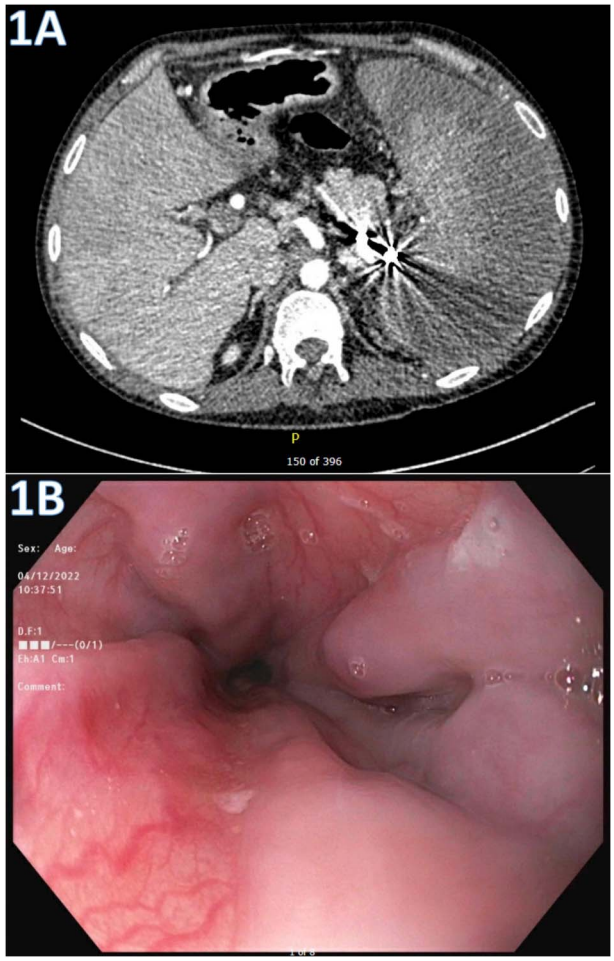
[3272] **Figure 1.** Liver biopsy with significant lobular hepatitis, loss of zone 3 hepatocytes, acidophil bodies, and Kupffer cell hyperplasia consistant with checkpoint inhibitor auto-immune hepatitis.

S3273

A Rare Case of Antiphospholipid Syndrome Presenting With Variceal Bleeding and Ascites Secondary to Portal Vein Thrombosis Without Liver Cirrhosis

Ahmed Eltelbany, MD, MPH, Rama Nanah, MD, Osama Hamid, MD, MRCPI, Sushrut Trakroo, MD, Khaled Alsabbagh Alchirazi, MD, M Housam Nanah, MD, Omar Massoud, MD.
Cleveland Clinic, Cleveland, OH.

Introduction: Portal vein thrombosis (PVT) mostly occurs with cirrhosis of the liver. However, it can also rarely occur without an associated liver disease. We present a case of PVT complicated by ascites and esophageal varices in the setting of antiphospholipid syndrome (APLS).
Case Description/Methods: A 31-year-old male with a history of ulcerative colitis and anti-phospholipid syndrome presented to the hospital after experiencing abdominal distension and upper gastrointestinal bleeding. Initial lab results revealed a hemoglobin of 6.4 g/dL. An abdominal CT showed heterogenous enhancement of liver parenchyma and extensive chronic portal venous system thrombosis with collaterals and splenomegaly with multiple infarcts (Figure 1A). Esophagogastroduodenoscopy (EGD) revealed non-bleeding grade I and small esophageal varices and type 1 gastroesophageal varies (Figure 1B). Patient was transfused packed red blood cells and underwent paracentesis with 4 liters drained. Liver ultrasound revealed mildly heterogenous liver with slightly increased echogenicity and chronically thrombosed left portal vein branch. Given the unclear etiology of PVT, a trans-jugular liver biopsy was completed and revealed mild steatosis and normal portal pressure of 8mmHg. Hospitalization was also complicated by splenic rupture and underwent embolization.
Discussion: PVT in the absence of cirrhosis is rare. In this case, the etiology of PVT is thought to be secondary to the hypercoagulable state in APLS. Ascites is a common symptom in patients with portal hypertension secondary to cirrhosis, however our patient was found to have ascites secondary to PVT in the absence of cirrhosis. Furthermore, PVT in APLS has been very rarely reported and may thus lead to PVT underdiagnosis or misdiagnosis at first. The most common presentation of PVT is variceal bleeding followed by pancytopenia due to hypersplenism while symptomatic portal hypertension is often indicative of the late stage of the PVT. Management is controversial and typically depends on the acuity of the thrombi. Anticoagulation therapy (AC) is generally warranted and may lead to significantly high rates of thrombus re-canalization. However, a significant cause of mortality in PVT is caused by variceal bleeding. It is important to recognize portal vein thrombosis as an alternative etiology of ascites in patients without evidence of cirrhosis and carefully time AC for improved outcomes.



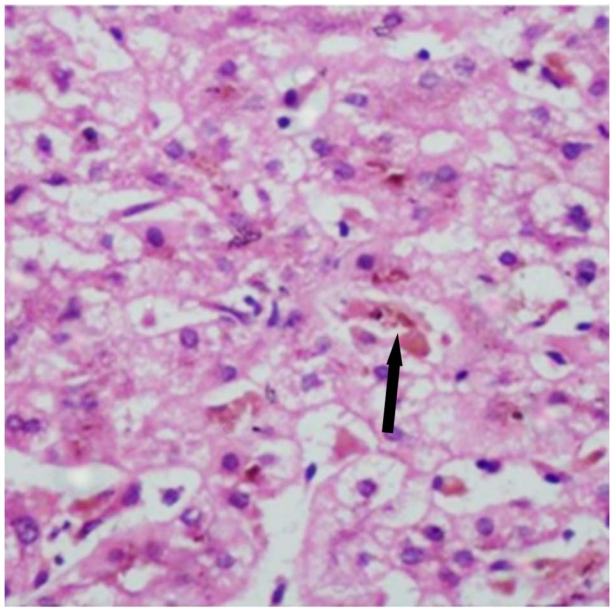
[3273] **Figure 1.** A) Transverse CT abdomen demonstrating large portal vein thrombosis B) Endoscopy image of the of the esophagus with varices.

S3274

A Rare Case of Benign Recurrent Intrahepatic Cholestasis (BRIC) Presenting as an Unexplained Cholestatic Jaundice

Dhruvkumar Patel, MBBS[1], Mukundkumar Patel, MBBS, MD[2], Maitri Patel, MBBS[3], Ram Parihar, MD[4], Aditya Vyas, MD[1].
[1]LSU Health Sciences Center, Shreveport, LA; [2]Dr. Kiran C. Patel Medical College, Bharuch, Gujarat, India; [3]GCS Medical College, Hospital and Research Centre, Ahmedabad, Gujarat, India; [4]Gastroplus Institute, Ahmedabad, Gujarat, India.

Introduction: Benign Recurrent Intrahepatic Cholestasis (BRIC) is a very rare autosomal recessive disorder characterized by episodes of recurrent cholestatic jaundice followed by complete resolution. BRIC is associated with a mutation in both alleles of ATP8B1 (BRIC 1) or ABCB11 (BRIC2). It causes cholestasis by impairing the function of the bile salt export pump. Recognition of BRIC is important as it can lead to delayed or no diagnosis, also it is underrecognized and challenging diagnosis.
Case Description/Methods: A 19-year-old South Asian male presented with nausea, vomiting, deep jaundice and pruritus of 10 days duration. Family history was negative for jaundice. He was not consuming alcohol or any drugs. Past medical records revealed three past episodes of undiagnosed cholestatic jaundice lasting for three to four weeks with complete recovery at the age of 8, 11 and 14 years respectively. Physical examination revealed stable vitals, deep icterus, cutaneous scratch marks and mild tender hepatomegaly. No stigmata of acute or chronic liver disease were identified. PT was prolonged with an INR of 1.8. AST and ALT were initially raised on admission which gradually trended down while serum total bilirubin, direct bilirubin and alkaline phosphatase continued to raise over 8 weeks. GGT remained within normal limits (Table 1). Viral markers for hepatitis A, B, C, E; HIV 1&2; EBV and CMV were negative. Workups for autoimmune hepatitis and Wilson's disease were negative. Ultrasound of the abdomen and MRCP were unremarkable. A liver biopsy was performed which showed intrahepatic and canalicular cholestasis predominantly involving zone 3 suggestive of BRIC (Figure A). He was treated with intravenous fluids, antiemetics, cholestyramine and antihistamines for symptomatic relief. Because of severe pruritus, Rifampicin was added later on for 14 days resulting in significant improvement. Liver function tests returned to baseline after 3 months. The patient was followed up for 6 months and remained asymptomatic.
Discussion: Till date very few cases of BRIC have been reported, which may be due to the rarity of the condition compounded by under-recognition by clinicians. Despite being a genetic disease, most cases are sporadic. Diagnosis in our case was based on the past episodes of jaundice, present clinical features, laboratory parameters and liver biopsy findings as proposed by Luketic and Shiffman for the diagnosis of BRIC. This is a self-limiting disease with no residual damage and treatment is symptomatic.



[3274] **Figure 1.** A. Liver Histology (H&E × 400): Intrahepatic cholestasis (black arrow) with preserved liver architecture.

Table 1. Laboratory results

	T. Bilirubin (mg/dl)	D. Bilirubin (mg/dl)	ALP (U/L)	GGT (U/L)	ALT (U/L)	AST (U/L)
Day – 1	8.2	6.1	388	15.2	415	205
Day – 7	12.4	9.8	421	15.4	354	106
Day – 14	14.6	12.2	510	14.8	156	88
Day – 21	18.0	16.5	615	14.5	80	60
Day – 31	21.5	18.3	830	12	48	32
Day – 45	24.32	22.45	950	11.3	38	22
Day – 60	26.82	24.13	1152	10.1	36	20
Day – 75	11.24	9.12	353	9.2	32	18
Day – 90	1.6	0.8	86	9	28	15

S3275

A Peculiar Case of Limb-Girdle Muscular Dystrophy Masquerading as Elevated Transaminases

Willie C. Gilbert, DO[1], Aaron Fieker, DO[2], Anand Shah, MD[3].
[1]Northside Hospital Gwinnett, Lawrenceville, GA; [2]Oklahoma State University Gastroenterology & Hepatology Fellowship, Tulsa, OK; [3]Emory University School of Medicine, Decatur, GA.

Introduction: A disproportional elevation in aspartate transaminase (AST) and alanine transaminase (ALT) is considered a marker of hepatocellular injury. Guidance on the work up of chronically elevated liver chemistry suggests an algorithmic approach to testing of common causes of liver injury based on the degree of elevation and even mentions consideration of nonhepatic etiologies such as skeletal muscle damage. We present a unique case of asymptomatic elevation in liver chemistries as the herald finding in a patient with limb-girdle muscular dystrophy (LGMD).
Case Description/Methods: Our patient is a 19-year-old adopted male who was referred to outpatient gastroenterology clinic for asymptomatic elevation in transaminases. AST was 133 IU/L and ALT was 223 IU/L. Total bilirubin and alkaline phosphatase were normal. He denied alcohol use, prescribed medications, herbal supplements, or weightlifting supplements. BMI was normal. Workup for viral hepatitis, hemochromatosis, autoimmune hepatitis, Wilson's disease, alpha-1 antitrypsin, celiac disease, and thyroid disease were normal. Liver biopsy revealed reticulin collapse with complete regeneration without inflammation. Subsequent follow-up over years demonstrated persistently elevated but stable elevations. At the age of 24, he began to endorse proximal lower extremity muscle pain and weakness. Creatine kinase (CK) was elevated at 11,937 U/L; aldolase was elevated at 31.4 U/L; myositis workup was normal. Right thigh biopsy revealed a myopathic process. Genetic testing confirmed mutation in the CAPN3 gene consistent with calpainopathy, a subtype of LGMD.
Discussion: LGMD is a rare, genetic disorder resulting in loss of ambulation within 20 years after disease onset. Concurrent liver disease is rare in LGMD, but elevations in transaminases can be found because of severe elevations in CK. Our patient was found to have calpainopathy, a subtype associated with a recessive mutation in the CAPN3 gene. Our case demonstrates that beyond the recommended algorithmic evaluation of abnormal liver chemistries, inconclusive evaluation of abnormal liver chemistries in children and young adults should include a CK level to further investigate nonhepatic causes, specifically a myopathic process. Although there is no cure for LGMD, interventions focused on muscle conditioning are more beneficial with early detection to mitigate the devastating effects of this disorder.

S3276

A Rare Case of Non-Small Cell Lung Cancer Causing False Positive Hepatitis B Surface Antigen

Jason J. John, MD[1], Wojciech Blonski, MD, PhD[2].
[1]University of South Florida Health, Tampa, FL; [2]James A. Haley VA Hospital, Tampa, FL.

Introduction: Hepatitis B surface antigen (HBsAg) is a protein on the surface of HBV which can be a distinctive serological marker of acute or chronic hepatitis B infection. We present a case of 74-year-old male with a false positive HBsAg test.
Case Description/Methods: A 74-year-old African American male with medical comorbidities of stage IV non-small cell lung cancer (NSCLC) and HTN presented to the hospital for an acute kidney injury with proteinuria. Hepatitis B virus (HBV) panel was obtained and revealed positive HBsAg. HBsAg was previously negative two years prior. Patient had multiple risk factors for hepatitis B infection including prior use of IV cocaine and multiple sexual partners. The patient denied any recent vaccinations. Liver function tests were normal. Physical examination did not reveal chronic liver disease manifestations. HBs

Antibody (HBsAb) was positive with a quantitative value of 0.53. Hepatitis B core immunoglobulin M (HBc IgM), HBcAb total, HBeAg, HBeAb were all negative with undetectable serum HBV DNA. The HBsAg was not confirmed as a true positive after repeat testing with an antibody neutralizing procedure involving a human antibody to HbsAg.
Discussion: Accurate interpretation of laboratory tests is imperative in diagnosing hepatitis B. There was high suspicion that patient had false positive HBsAg. The patient's serological profile did not fit acute or chronic hepatitis B. The patient had no evidence of acute hepatitis B with negative HBc IgM, HBV DNA and no evidence of chronic hepatitis B with HBcAb total being negative. Furthermore, a positive HBsAb is not seen in acute and chronic hepatitis B. Transient HBsAg has been seen in patients for up to 2 weeks after HBV vaccination, however, our patient had not been vaccinated recently. The most common cause is the presence of heterophile antibodies. These naturally occurring human antibodies can bind to a variety of chemical structures including animal antibodies seen in immunochemistry assays. They can be neutralized by specific inactivating binders. Other viral infections such as Epstein-Barr Virus (EBV), transfusions, or systemic disease can cause false positives. A likely suspect was the patient's NSCLC causing a paraneoplastic syndrome which would be the first instance of such in the literature. There have been cases of parathyroid adenoma and basal cell carcinoma causing false positivity. Patients with false-positive HBsAg need further workup to prevent misdiagnosis and potentially harmful management.

S3277

A Rare Case of Glycogen Storage Disease Presenting as Liver Failure in an Adult

Ankita Prasad, MD[1], Dina Alnabwani, MD[1], Pramil Cheriyath, MD, MS[1], Varun Vankeshwaram, MD[2], Sheilabi Seeburun, MD[2].
[1]Ocean University Medical Center, Brick, NJ; [2]Hackensack Meridian Ocean Medical Center, Brick, NJ.

Introduction: Glycogen storage disorder (GSD)III results from the absence of enzymes in converting glycogen to glucose, which results in the accumulation of glycogen in tissues. It usually presents in childhood with hypoglycemia and hepatomegaly. It is an autosomal recessive disease, accounting for 2.3 in 100,000 children in the United States annually.
Case Description/Methods: The patient is a 22-year-old previously healthy man with a one-month history of jaundice, fatigue, weight loss, and pain in the abdomen, no history of medical or surgical illness, blood transfusions, or taking herbal supplements or medications. On examination, he had jaundice, bruises on his upper and lower limbs, a palpable splenic tip, and no hepatomegaly or ascites. His liver function tests showed bilirubin of 92 mol/L with normal enzymes, albumin of 23 g/L, and PT/INR 22/1.8. The random blood sugar of 5.0 mmol/L; pancytopenia, neutropenia, thrombocytopenia, and anemia with raised LDH; negative autoantibody screens; negative for infection; normal serum copper; ultrasound showed splenomegaly; a liver biopsy revealed significant glycogen load in hepatocytes on special stains; He was then referred for further management of GSD with cirrhosis
Discussion: GSD III presents in infancy with hypoglycemia and hepatomegaly. It is associated with full cheeks, hypertriglyceridemia, hypoglycemia, immunodeficiency, intellectual disability, short stature, and myopathy. This condition is diagnosed by histology and genetic testing. It can cause cirrhosis with liver failure and lesions with malignant transformations. Treatment aims to prevent hypoglycemia through carbohydrate meals and night feeds with cornstarch.

S3278

A Rare Case of Gabapentin-Induced Hepatotoxicity

Herman Suga, DO[1], David J. Truscello, DO[1], Zainab Shahid, DO[1], Maulik Shah, DO[3], Aarati Malliah, MD[4].
[1]Jefferson Health New Jersey, Turnersville, NJ; [2]Virtua Voorhees, Voorhees, NJ; [3]Jefferson Health New Jersey, Cherry Hill, NJ; [4]Jefferson Health, Woodbury, NJ.

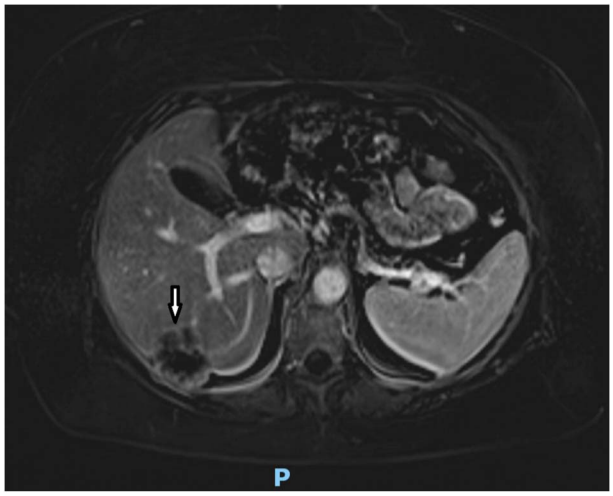
Introduction: Gabapentin is an anti-convulsant that is also used off-label to treat neuropathic pain. It is not metabolized by the liver, and there have been few reports of hepatotoxity associated with it. We present a rare case of gabapentin-induced hepatotoxicity occurring in a young male.
Case Description/Methods: A 41-year-old male with an extensive past medical history including type 1 diabetes, end stage renal disease on hemodialysis, TIA, hypertension, and hyperlipidemia was admitted for severe hyperkalemia with K 8.2 and peaked T waves on EKG, and hyperglycemia with glucose 561. He was admitted to the intensive care unit for urgent dialysis. Lab work revealed abnormal liver function tests, with ALP 1,232 IU/L, AST 291 IU/L, and ALT 188 IU/L, and normal total bilirubin of .8 mg/dL. Prior records revealed completely normal liver function tests 6 months prior to presentation. Ultrasound of the RUQ revealed small volume of ascites but was otherwise unremarkable with no abnormality of hepatic parenchyma or biliary ducts noted. Patient reported having a below-knee amputation 3 months prior to presentation, after which he started taking gabapentin 300 mg three times per day. He denied any other medication changes. He reported taking acetaminophen occasionally, and acetaminophen level was < 10 mcg/mL. A full hepatitis panel was negative. An autoimmune workup was planned including Anti-LKM, ASMA, AMA but patient insisted on leaving prior to those labs being drawn. Gabapentin was held and patient's liver function tests improved, with ALP 1058 IU/L, AST 158 IU/L, and ALT 149 IU/L, and remained stable. Patient discontinued gabapentin and was advised to follow up outpatient, unfortunately he was lost to follow-up.
Discussion: Although the exact mechanism of gabapentin is unknown, it is structurally related to GABA and high-affinity binding sites for gabapentin which modulate the release of excitatory neurotransmitters which participate in epileptogenesis have been found throughout the brain. Gabapentin is not hepatically metabolized, and therefore was not studied in patients with hepatic impairment during the FDA approval process. There have been very few cases of gabapentin induced liver injury. In our patient there was a clear temporal association of liver injury occurring after our patient began gabapentin therapy, and slight improvement after a few days of discontinuation. Although further studies on this topic are needed, gabapentin should be considered as a cause of drug induced liver injury.

S3279

A Rare Case of Late Metastatic Disease From Granulosa Cell Tumor as a Solitary Hepatic Mass

James Pitcher, MD[1], Andrew Mims, MD[1], Vick DiCarlo, MD[1], Laxmi Parsa, MD[1], Christina Birsan, MD[2].
[1]University of Tennessee Health Science Center, Chattanooga, TN; [2]Erlanger Medical Center, Chattanooga, TN.

Introduction: Granulosa Cell Tumor (GCT) is a rare type of sex cord-stromal neoplasm that often has an indolent course. These tumors make up 2-5% of all ovarian malignancies. When they do metastasize, it is often within the lower abdomen and pelvis. Recurrence of the disease often occurs within 10 years of the diagnosis, but there is potential for recurrent disease beyond this timeframe. Because of the risk of late recurrence with GCT, continued surveillance is crucial beyond surgical resection of the primary tumor. Inhibin has been shown to be a valid marker in both management of primary disease and surveillance for late recurrence. The overall prognosis for GCT is generally favorable given that the disease is detected early. Surgical intervention is mainstay of treatment for these tumors, although systemic chemotherapy is considered in nonresectable or advanced cases.
Case Description/Methods: A 53-year-old female with a past medical history of bilateral GCT of the ovaries status post bilateral salpingo-oophorectomy (2009), gastroesophageal reflux disease, and Hepatitis C presented to the emergency department with lower abdominal pain and occasional hematochezia in 2021. She was discharged with referrals for further evaluation. During her work up, she underwent a CT scan that revealed a 4.3 x 3.4 cm exophytic mass in the right lobe of the liver. Further evaluation with MRI showed that the mass was irregular and exophytic with minimally increased size, now measuring 4.6 x 3.7 cm. A CT-guided biopsy showed that the tumor was positive for inhibin, suggestive of metastatic GCT. The solitary hepatic metastasis was treated with microwave ablation. (Figure)
Discussion: GCTs are a rare subtype of sex-cord stromal tumor that are typically primary ovarian neoplasms and often have an indolent course and favorable prognosis, although there is evidence that metastatic disease can occur at distant sites and greater than 10 years after diagnosis and surgical resection of the primary tumor. This case describes the recurrence of disease as a solitary hepatic mass. Diagnostic confirmation was made by CT-guided biopsy that found the presence of inhibin, a protein known to be produced by GCT. This case highlights the importance of long-term surveillance in patients with a history of GCT beyond surgical resection and undetectable disease activity.



[3279] **Figure 1.** MRI of the abdomen showing metastatic granulosa cell tumor (arrow).

S3280

A Rare Case of Isolated ALECT-2 Hepatic Amyloidosis

Benjamin Schwartz, MD, Jessica Strzepka, MD, Mary Biglin, MD, Nikunj Shah, MD.
Rush University Medical Center, Chicago, IL.

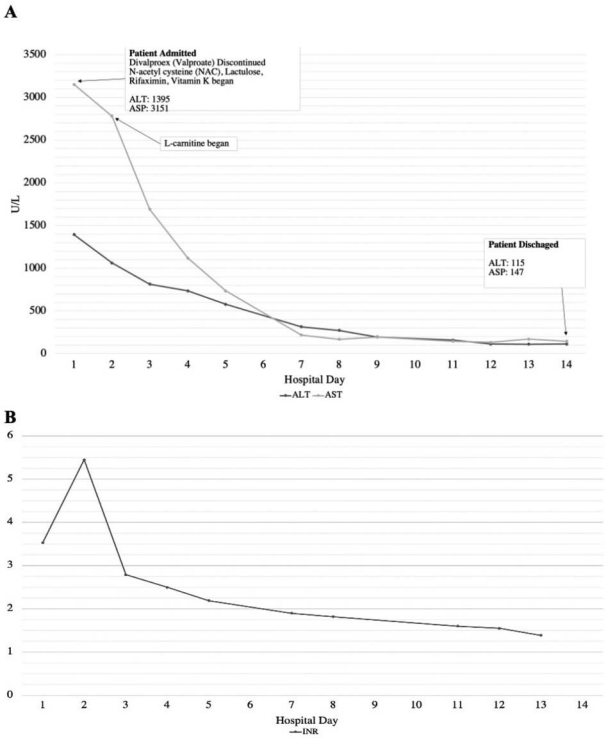
Introduction: Amyloidosis is a rare disease that is defined by deposition of extracellular fibrils from immunoglobulin light chains which results in organ dysfunction. Many patients with primary systemic amyloid have hepatic involvement. Hepatic amyloid deposition is characterized by mild hepatomegaly and occasional elevated liver enzymes. Ultrasound may reveal a heterogenous echogenicity of the liver and Computed Tomography (CT) may reveal decreased parenchymal attenuation. Biopsy is the gold standard for diagnosis, seen as extracellular amorphous material, and Congo red staining will reveal apple-green birefringence under polarized light. Whereas amyloidosis is most often systemic, there are rare cases of isolated hepatic amyloid deposition. Leukocyte cell-derived chemotaxin 2 (ALECT-2) is a novel amyloid subtype, previously thought to be found in renal amyloid with a predominance in Hispanic patients but has recently been reported in the liver. We present a rare case of isolated hepatic amyloidosis without systemic involvement.
Case Description/Methods: A 53-year-old Hispanic female with history of diabetes mellitus, hyperlipidemia, and nonalcoholic fatty liver disease presented to hepatology clinic for liver enzyme elevation. Lab studies were significant for AST 36 U/L, ALT 67 U/L, Alkaline Phosphatase, total bilirubin 2.7 mg/dL, positive anti-smooth muscle antibody (22 U), immunoglobulin G 1049 mg/dL, normal anti-nuclear antibody screen and normal anti-mitochondrial antibody. CT showed post cholecystectomy changes, a calcified granuloma in the right hepatic lobe, but otherwise normal liver. Liver biopsy showed focal amyloid deposition, mild steatosis, mild lobular activity, and rare ballooning hepatocytes without fibrosis. Positron emission tomography-CT, bone marrow biopsy, urine and serum protein electrophoresis were unremarkable. Further amyloid testing showed leukocyte chemotactic factor-2 (ALECT-2) amyloidosis. A referral for second opinion and experimental treatment options was recommended, however the patient declined. The patient has had stable lab values over the last two years with observation.
Discussion: This is a rare case of isolated hepatic amyloidosis, highlighting the need for maintaining a broad differential diagnosis in a patient presenting with elevated liver enzymes. Given the rare nature of this condition, it is important to demonstrate this patient's presentation and two-year outcomes with observation, given lack of available treatment regimens.

S3281

A Rare Case of Drug-Induced Liver Injury (DILI) Due to Valproate Hepatocellular Injury

Hope Baldwin, BS[1], Syed-Mohammed Jafri, MD[2], Ahmed Elbanna, DO[2].
[1]Wayne State University School of Medicine, Detroit, MI; [2]Henry Ford Health System, Detroit, MI.

Introduction: Valproate is a widely-used antiepileptic drug which works by increasing gamma-aminobutyric acid (GABA) levels in the brain. We present a unique form of drug-induced liver injury (DILI) in a patient prescribed valproate for trigeminal neuralgia pain.
Case Description/Methods: A 45-year old African American female with a history of hypertension and trigeminal neuralgia was prescribed divalproex for trigeminal neuralgia pain after trials of duloxetine, gabapentin, baclofen, oxcarbazepine, carbamazepine, topiramate, amitriptyline, and tizanidine failed to provide relief. Six weeks later she presented with a two week history of worsening nausea, diarrhea, fever, facial rash, production of dark phlegm, and grade 1 encephalopathy. Lab results showed acute liver failure (ALF) with total bilirubin 15.6 mg/dL, ALP 180 U/L, AST 937 U/L, ALT 943 U/L, and INR 2.8. Exam findings included scleral icterus, hepatomegaly, small volume ascites, and some small liver cysts. A diagnosis of DILI was made, with likely drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome. Divalproex was discontinued and the patient was admitted for two weeks of stabilizing treatment including N-acetylcysteine, lactulose, rifaximin, Vitamin K, and L-carnitine transfusion (Figure 1). The patient was evaluated for liver transplant, but it was ultimately not indicated given her subsequent improvement in liver function. An acute kidney injury developed but was resolved before patient discharge. Two weeks later the patient presented with signs of acute kidney injury. Upon readmission, complications including hepatic encephalopathy, macrocytic anemia, spontaneous bacterial peritonitis, and acute respiratory failure developed over the course of several weeks. All stabilizing treatment efforts were made but the patient ultimately developed septic shock and died.
Discussion: DILI is the most common cause of ALF in the United States. Valproate-induced hepatotoxicity as a result of mitochondrial injury from impaired beta-oxidation and decreased tissue carnitine levels is one rare effect of use. Such hepatocellular injury typically shows very elevated AST and ALT levels with a smaller elevation in ALP. Early diagnosis of DILI is crucial to promptly discontinue use of the causative agent and begin optimal treatment. In the case of valproate, it is recommended that patients receive intravenous (IV) carnitine until symptoms improve. Liver transplant evaluation is crucial for ALF patients given the high morality risk.



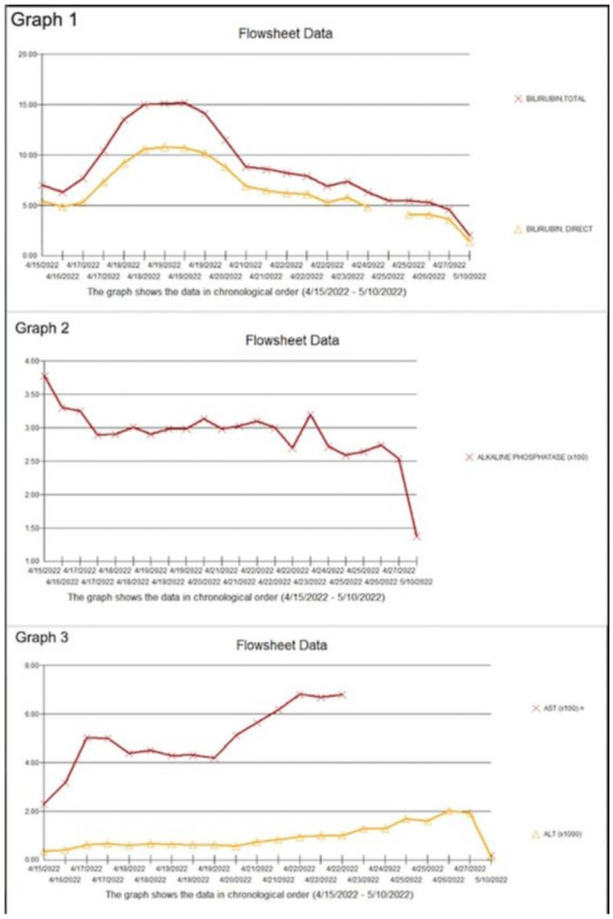
[3281] **Figure 1.** 1A. Patient's AST and ALT values over the course of her hospital admission. Figure 1B. Patient's INR values over the course of her hospital admission.

S3282

A Rare Case of Acute Cholestatic Hepatitis Due to Acute Hepatitis E Infection

Jasparit Minhas, MD, Vikash Kumar, MD, Srilaxmi Gujjula, MD, Praneeth Bandaru, MD, Vijay Gayam, MD, Suut Gokturk, MD, Jamil Shah, MD, Denzil Etienne, MD, Madhavi Reddy, MD.
The Brooklyn Hospital Center, Brooklyn, NY.

Introduction: Hepatitis E virus (HEV) is a non-enveloped, single stranded, ribonucleic acid (RNA) virus that belongs to Herpeviridae family. It is a common cause of acute hepatitis and transmission can occur through blood transfusions, contaminated water or food, and mother to child transmission. Symptoms can present as fever, anorexia, abdominal pain, nausea, vomiting, diarrhea and jaundice. Labs usually reveal elevated bilirubin, aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Here we present a patient with acute hepatitis initially found to have mixed pattern of liver enzymes which eventually progressed to hepatocellular pattern, and confirmed to have acute cholestatic hepatitis compatible with clinical history of HEV on liver biopsy.
Case Description/Methods: 40-year-old healthy male presented with a two week history of pruritus, dark urine, and light stools. Patient reported eating undercooked eggs and started experiencing right upper quadrant (RUQ) discomfort which progressed to flu-like illness. He tried over the counter treatment with no improvement. Labs initially showed a mixed pattern of liver enzymes which progressed to the hepatocellular pattern. Comprehensive acute and chronic liver disease workup was done (shown in table 1) and it was remarkable for elevated immunoglobulin G titers and positive Hepatitis E immunoglobulin G. RUQ ultrasound did not reveal any evidence of biliary obstruction. Magnetic resonance cholangiopancreatography/Magnetic resonance imaging (MRCP/MRI) abdomen was negative for hepatobiliary disease or acute cholangitis. Liver biopsy results confirmed acute cholestatic hepatitis related to HEV infection and ruled out autoimmune hepatitis. Patient adhered to lifestyle modifications resulting in symptomatic improvement. The patient was educated on hygienic precautions and avoidance of hepatotoxic medications and drugs as well as uncooked meals. (Figure)
Discussion: HEV is a concern to public health and is the cause of acute viral hepatitis worldwide with a high risk of progressing to chronic infection in immunocompromised patients. HEV usually spontaneously resolves in the young and immunocompetent population, however some patients may develop acute hepatic failure, cholestatic hepatitis, or chronic HEV. Acute HEV is managed with supportive care whereas chronic HEV involves antiviral therapy and reduction in immunosuppressive therapy (in immunocompromised patients).



[3282] **Figure 1.** Trend of liver enzymes.

Table 1. Acute Infectious and Drugs Test Results, Autoimmune and Metabolic Test Results

Acute Infectious and Drugs Test Results:	
Hepatitis A IgG	Reactive
Hepatitis A IgM	Negative
Hepatitis B surface antigen	Not reactive
Hepatitis B core antibody	Not reactive
Hepatitis B core antigen	Not reactive
Hepatitis B viral load	Negative
Hepatitis C antibody	Reactive
Hepatitis C viral load	Negative
Hepatitis E Immunoglobulin G	**Detected**
Human Immunodeficiency Virus antigen/antibody	Negative
Epstein-Barr Virus PCR	Negative
Cytomegalovirus Immunoglobulin G	1.90
Cytomegalovirus Immunoglobulin M	Negative
Herpes Simplex Virus 1 Antibody Immunoglobulin G	Not elevated
Herpes Simplex Virus 1 and 2 PCR	Negative
Gonorrhea	Negative
Chlamydia	Negative
Urine toxicology (10 drug panel)	Negative
Serum acetaminophen levels	< 1.0
Serum salicylate levels	< 5.0
Autoimmune and Metabolic Test Results:	
Antinuclear antibody	Negative
Anti-smooth muscle antibody	Negative
Antimitochondrial antibody	Negative

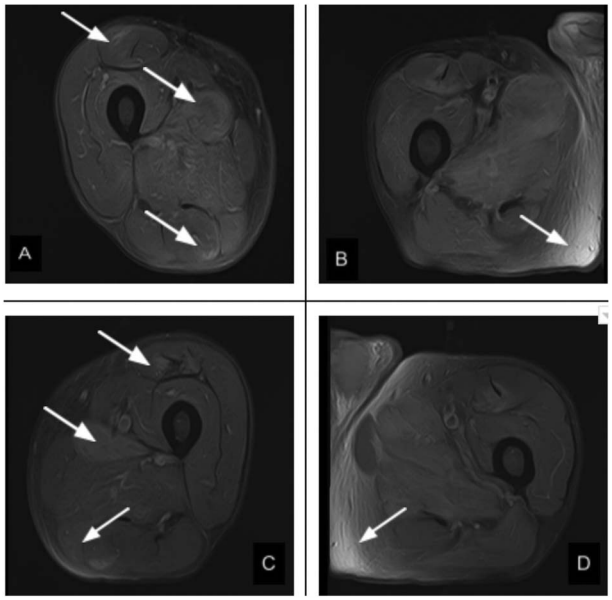
Table 1. (*continued*)	
Tissue transglutaminase antibody	Negative
Immunoglobulin A titers	Within normal limits
Ceruloplasmin	Within normal limits
Soluble liver antigen antibody	Negative
Anti-liver-kidney microsomal type 1 antibody	Negative
Immunoglobulin G titers	**2196 (elevated)**
Alpha 1 antitrypsin	Within normal limits
Hemochromatosis gene (HFE) panel	Negative

S3283

A Tough Pill to Swallow: A Case Series of Statin-Induced Necrotizing Autoimmune Myopathy Manifesting as Dysphagia and Transaminitis

Hunza Chaudhry, MD, Joanne Lin, DO, Rameen Atefi, DO, Jeffrey Hagino, DO, Candice Reyes, MD, Marina Roytman, MD.
UCSF-Fresno, Fresno, CA.

Introduction: Statins are commonly used to prevent cardiovascular disease. However, a small subset of patients may develop autoimmune myopathy, a form of inflammatory myositis characterized by proximal muscle weakness and in some cases, dysphagia. We present two cases highlighting statin-induced necrotizing autoimmune myopathy (SINAM), a rare entity affecting roughly 2 cases per 1 million people.
Case Description/Methods: Two Hispanic men, aged 60 and 71, presented with dysphagia and transaminitis. They reported gradual onset of proximal muscle weakness and weight loss and were taking atorvastatin 20mg and 80mg, respectively. Case 1: Labs revealed ALP 58 IU/L, AST 238 U/L, ALT 407 U/L, CK 7228 U/L, normal CRP, ESR, and HMG CoA reductase (HMGCR) antibodies 257 U/mL. EGD was unrevealing. MRI of the pelvis/femur showed patchy, symmetric intramuscular edema in leg muscles (Figure 1). Muscle biopsy revealed myonecrosis consistent with immune-mediated necrotizing myopathy. Case 2: Labs revealed ALP 79 IU/L, AST 571 U/L, ALT 373 U/L, CK 8392 U/L, CRP 19.2 mg/L, ESR 67 mm/hr, and HMGCR antibodies 367 U/mL. Muscle biopsy was not pursued. Both cases revealed negative viral hepatitis, ANA, myositis panel, smooth muscle Ab, LKM-1 Ab, total IgG, ferritin, anti-mitochondrial Ab, and alpha-1 antitrypsin. Both patients were started on high-dose steroids. Case 1 patient had minimal improvement and was subsequently started on mycophenolate and rituximab. He remains under close monitoring. Case 2 patient had persistent severe weakness and dysphagia, therefore intravenous immunoglobulin (IVIG) and mycophenolate were added. However, dysphagia persists despite a consistent decrease in muscle enzymes. He remains hospitalized and is pending PEG tube placement. (Figure)
Discussion: SINAM is a rare variant of idiopathic inflammatory myopathy characterized by proximal muscle weakness and myofiber necrosis after statin exposure. Diagnosis is made with positive antibodies to HMGCR and may avoid the need for muscle biopsy in the appropriate clinical context. Cessation of statin and initiation of glucocorticoids are first-line treatments. However, many cases are refractory to steroid monotherapy. Other therapies such as IVIG and immunosuppressants serve a role in refractory disease. Alternative lipid-lowering agents should be used, and statins must not be rechallenged. These cases emphasize that muscle weakness, dysphagia and transaminitis should be evaluated diligently as early recognition can lead to initiation of life-saving treatment.



[3283] **Figure 1.** A-D. MRI pelvis/femur showing bilateral patchy and symmetric intramuscular edema in leg muscles.

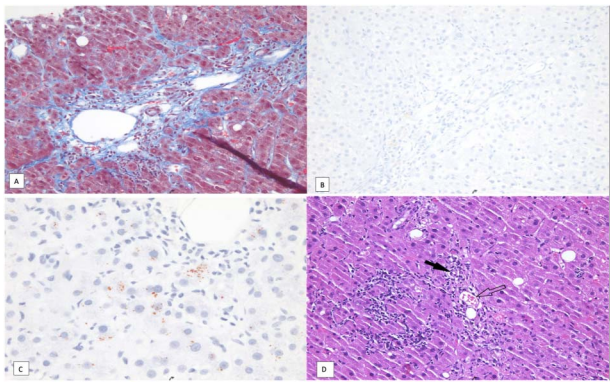
S3284

A Rare Case of Vanishing Bile Duct Syndrome in a Patient With Sarcoidosis

Annie Shergill, MD.
Larkin Community Hospital, Fresno, CA.

Introduction: Vanishing Bile Duct Syndrome (VBDS) is an uncommon, acquired, chronic cholestatic liver disease mostly caused by drug induced liver injury, autoimmune disorders, malignancy and infectious causes. It presents with symptoms of cholestasis such as jaundice, pruritus and abnormal liver enzymes. Sarcoidosis is not known to commonly cause VBDS. We present a unique case of VBDS as a hepatic manifestation of sarcoidosis.
Case Description/Methods: A 39-year-old male with no significant past medical history presented with recurrent epistaxis, pruritus and was discovered to have a right nasal mass. Maxillofacial computed tomography (CT) revealed a nodular lesion in the nose with some bony erosions and multiple nodules in the parotid gland. Chest CT showed mediastinal lymphadenopathy. Biopsy of nasal mass showed focally necrotizing granulomatous inflammation without evidence of vasculitis consistent with sarcoidosis. Biopsy cultures were negative. He also had a history of getting recent tattoos which were found to be partially scarred and nodular. He did not report any significant alcohol use. Hepatitis B and C serologies were negative. Routine bloodwork revealed elevated liver enzymes: Total Bilirubin 1.0 mg/dl (direct 0.4), AST 270 units/L, ALT 316 units/L, Alk Phos 421 units/L, and GGT of 1425 units/L. Abdominal and pelvic CT showed hepatic morphology consistent with cirrhosis. Chronic liver disease workup included IgG level of 1680 mg/dL, but normal IgG4 level of 59.1 mg/dL, negative ANA, negative AMA and an ACE level of 65 mcg/L. Liver biopsy results showed significant ductopenia (approximately 75% of sampled portal tracts),

mild portal, focal interface chronic inflammation and mild periportal fibrosis with delicate non-bridging septae, confirming a diagnosis of VBDS. The patient was treated with ursodexycholic acid and mycophenolate with significant improvement in his symptoms and liver enzymes.
Discussion: VBDS has rarely been associated with sarcoidosis. The most common hepatic manifestation in sarcoidosis is hepatic granulomas. However, in this case the liver biopsy did not show any sarcoid lesions but instead showed severe ductopenia consistent with VBDS. VBDS is characterized by bile duct paucity secondary to biliary apoptosis triggered by oxidative stress, drug induced injury and down regulation of B cell lymphoma-2 proteins. Hepatic involvement in sarcoidosis should be evaluated with a liver biopsy to elucidate causes such as VBDS as it changes the therapeutic management for the patient.



[3284] **Figure 1.** 1A: Trichrome stain of a portal tract shows delicate periportal fibrosis. The tract contains several large lumens representing the portal vein branch and a smaller hepatic artery branch. No bile duct is present. (Trichrome x20). Figure 1B: Cytokeratin 19 stain shows no specific uptake in a portal tract running diagonally in the center of the photograph. Bile ducts strongly express this cytokeratin and its absence confirms bile duct loss in this tract (CK19x20). Figure 1C: Copper stain shows frequent deposition of coarse granular material representing copper in periportal hepatocytes. Chronic compromise of biliary flow leads to build-up of copper at this location and reflect a physiologic effect of duct loss in this biopsy (rhodanine x40). Figure 1D: A small portal tract is in the center of the field. Hepatic artery (solid arrow) and portal vein (open arrow) branches are present in the absence of bile duct. Mild inflammation is seen in the portal tract and adjacent hepatic parenchyma (H&Ex20).

S3285

A Rare Case of Pyogenic Liver Abscess Caused by *Raoultella ornithinolytica*

Apaar Dadlani, MBBS[1], Dylan Flaherty, DO[1], James w. Snyder, PhD[1], Endashaw Omer, MD, MPH[2].
[1]University of Louisville, Louisville, KY; [2]University of Louisville Hospital, Louisville, KY.

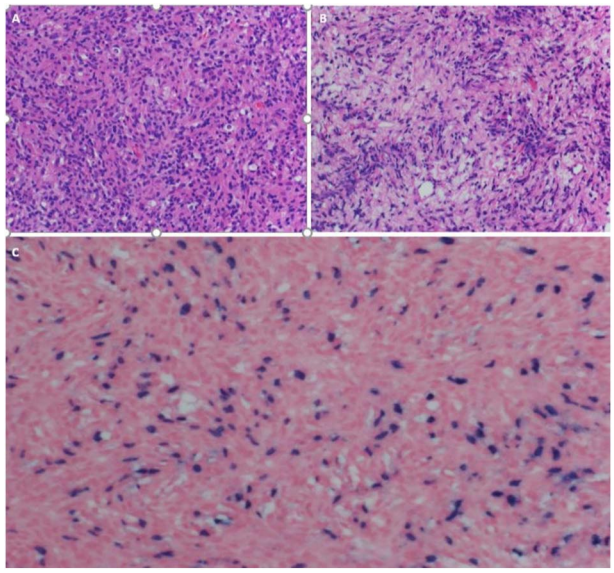
Introduction: Patients with a history of splenectomy have a higher prevalence of infections with encapsulated organisms due to decreased phagocytic activity and humoral response. One such rare organism is Raoultella ornithinolytica, a Gram-negative bacillus commonly found in the aquatic environment. Here we present a rare case of this bacterium causing liver abscess.
Case Description/Methods: A 72-year-old female with a history of hypertension, hyperlipidemia, and splenectomy (secondary to abdominal trauma) presented after being found down. She complained of a recent history of diarrhea 2 days ago. Blood pressure was 80/60 mmHg and temperature was 101 degrees F at presentation. She was found to have elevated liver enzymes and Klebsiella oxytoca bacteremia (found on Verigene multiplex PCR testing) and was started on broad-spectrum antibiotics. CT of the abdomen in the emergency room revealed a 3x2x5 cm hypodense lesion in the left lobe of the liver. She underwent ultrasound-guided removal of 35 cc dark brown colored fluid, which was positive by culture for Raoultella ornithinolytica. Her antibiotics were narrowed to cefazolin, and the infection was thought to be related to recent diarrhea (of unknown etiology) resulting in intestinal mucosal damage leading to translocation of the bacterium across the intestinal wall into the liver. Of note, on high specificity microbiological testing using matrix assisted desorption-time of flight (MALDI-Tof) technology, the organism recovered from the blood was confirmed to be Raoultella ornithinolytica.
Discussion: Incidence of Raoultella ornithinolytica infection has been on the rise in the past decade, and our patient is the second reported case of Raoultella ornithinolytica liver abscess. In humans, it usually causes skin flushing, vomiting, diarrhea, and headache. While the first case, as reported by Surani et al in 2020, had a long-standing liver cyst that was thought to be infected with Raoultella ornithinolytica, our patient did not have any nidus in the liver that could harbor this bacterium. However, she had a history of splenectomy, possibly putting her at higher risk of infection with encapsulated bacteria. In addition, Raoultella ornithinolytica is closely related to Klebsiella oxytoca, but is not recognized in the multiplex PCR database which accounts for the discrepancy in initial identification as seen in our case. Our case adds to the growing literature regarding Raoultella ornithinolytica as an increasing virulent pathogen in humans.

S3286

A Rare Epstein-Barr Virus-Associated Tumor of the Liver and Spleen

Shoma Bommena, MD[1], Rohit Nathan, DO[2].
[1]Banner University Medical Center, University of Arizona College of Medicine, Phoenix, AZ; [2]University of Arizona College of Medicine, Phoenix, AZ.

Introduction: Inflammatory Pseudotumor-like follicular dendritic cell tumor (IPT-like FDC) is a rare histological subtype of follicle dendritic cell tumors, with unique clinical and pathological characteristics characterized by an intense lymphoplasmacytic infiltrate mixed with a neoplastic spindle, and consistent association with Epstein-Barr virus (EBV). We describe a rare case of splenic ITP-like FDC with hepatic metastasis.
Case Description/Methods: A 67-year-old female with unremarkable medical history presented with a one-day history of periumbilical abdominal pain and was found to have incidental findings of hypodense hepatic and splenic lesions on abdominal CT. Subsequent MRI abdomen showed numerous new peripherally enhancing hypodense lesions throughout the liver and a slightly enlarged 8.0 x 5.5 cm heterogeneously enhancing splenic lesion, seen in scans dated ten years back. These radiological findings, combined with histological evidence of an atypical EBV-associated lymphohistiocytic proliferation obtained from a biopsy of the hypoechoic liver mass (Figure), helped diagnose IPT-like FDC that started in the spleen and later metastasized to the liver. Surgical resection with Right hepatectomy, wedge resection of the left-sided lesion, and splenectomy was recommended to reduce the tumor burden. Unfortunately, the patient did not follow up and pursue surgical options due to the pandemic. Repeat imaging two years later showed multiple enhancing liver lesions and enlarged splenic lesions. Given the progression of the disease, surgical resection was not an option, and the medical oncology team initiated systemic treatment with Gemcitabine and Docetaxel. Imaging after four months showed a slightly decreased size of metastatic hepatic disease and dominant splenic mass (Figure).
Discussion: IPT-like FDC accounts for less than 1% of all primary splenic and hepatic tumors, with female predominance. It should be differentiated from classic FDC, IPT, and Inflammatory myofibroblastic tumors by examining morphological features combined with immunohistochemistry, including FDC markers: CD21, CD35, and CD23, clustering, CNA.42, CXCL-13, and D2.40 (podoplanin). Detection of EBV-encoded RNA by in situ hybridization supports the diagnosis. Given the indolent clinical course with low malignant potential, Surgical resection is the mainstay of treatment for localized lesions, with systemic chemotherapy and radiation therapy used in nonoperative cases, but efficacy is unclear.



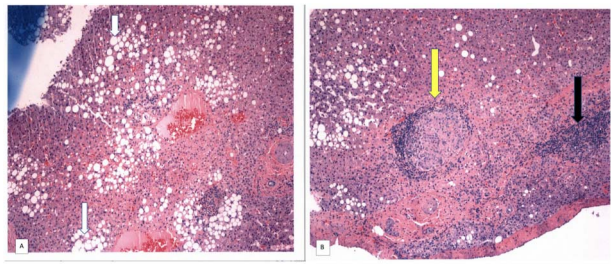
[3286] **Figure 1.** Figure: Dense lymphohistiocytic and spindle cell infiltrate in the liver (hematoxylin-eosin stain, panel A & B). EBER (EBV encoded RNA) in situ hybridization shows positive staining of many neoplastic cells (panel C).

S3287

A Rare Case of Primary Biliary Cholangitis and Hepatic Sarcoidosis

Annie Shergill, MD[1], Abhishek Gulati, MD[2].
[1]Larkin Community Hospital, Fresno, CA; [2]Clovis Community Medical Center, Clovis, CA.

Introduction: Primary biliary cholangitis (PBC) is a disease characterized by immune-mediated destruction of intrahepatic bile ducts resulting in cholestatic liver disease. Sarcoidosis is a multisystem, chronic granulomatous disease of unknown etiology with hepatic involvement seen in majority of the patients. Very rarely, hepatic sarcoidosis can present as diffuse intrahepatic biliary inflammation, destruction and cholestatic liver disease, thereby resembling PBC. We present a rare case of hepatic sarcoidosis with PBC and non-alcoholic fatty liver disease (NAFLD).
Case Description/Methods: A 47 year old female with history of asymptomatic pulmonary sarcoidosis, type 2 diabetes mellitus, morbid obesity underwent liver biopsy for evaluation of fatty liver and abnormal liver enzymes (AST 41 U/L, ALT 55 U/L, alkaline phosphatase 238 U/L, Total bilirubin 0.5 mg/dL). Biopsy showed steatohepatitis with non-necrotizing granulomas consistent with sarcoidosis. Patient was asymptomatic and not on treatment for sarcoidosis or any other hepatotoxic medications. Social history was pertinent for no significant alcohol intake. Patient continued to have persistently abnormal liver enzymes and was further evaluated with a comprehensive chronic liver disease work up. This came back positive for Anti-mitochondrial antibody (AMA), raising suspicion for underlying PBC. Patient underwent repeat liver biopsy which showed similar findings of steatohepatitis and non-necrotizing granulomas along with focally identified biliary inflammation suggestive of underlying chronic biliary disease such as PBC given positive serologic testing for AMA. Iron and copper stains were negative. Patient was subsequently started on therapy with Ursodiol. (Figure)
Discussion: Hepatic sarcoidosis most commonly manifests as non-necrotizing granulomas as seen on liver biopsy. However, in this case the liver biopsy showed evidence of three pathological processes- PBC, sarcoidosis and NAFLD. Patients with hepatic sarcoidosis very rarely develop an underlying co-existing cholestatic liver disease. The postulated underlying mechanism of chronic cholestasis in sarcoidosis involves confluent granulomas causing granulomatous cholangitis and ductopenia. It is prudent to identify this co-existing pathological process in order to initiate appropriate treatment and prevent further hepatic damage. Therefore, chronic cholestasis in hepatic sarcoidosis should prompt extensive evaluation for an alternative cause of chronic liver disease.



[3287] **Figure 1.** 1A: Moderate large-droplet steatosis (white arrow) with mild to moderate lobular inflammation is seen. Figure 1B: Scattered non-necrotizing granulomas (yellow arrow) with focal portal inflammation (black arrow) is seen.

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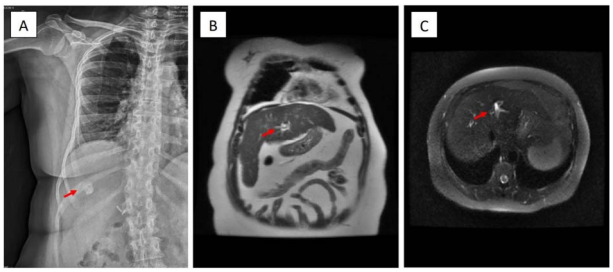
A Rare Complication After Transarterial Chemoembolization (TACE): Hepatic Dystrophic Calcification

Vikash Kumar, MD, Srilaxmi Gujjula, MD, Praneeth Bandaru, MD, Vijay Gayam, MD, Suut Gokturk, MD, Denzil Etienne, MD, Madhavi Reddy, MD.
The Brooklyn Hospital Center, Brooklyn, NY.

Introduction: Transarterial chemoembolization (TACE) with radiofrequency ablation (RFA) therapy are considered therapeutic options. It is mostly used in hepatocellular carcinoma (HCC), colorectal, or neuroendocrine tumors. Although it is considered a safe procedure, TACE presents rare complications. We present a case of a 58-year-old female with right sided chest pain who was found to have hepatic calcification seen on chest x-ray after TACE procedure.
Case Description/Methods: A 58-year-old female with a past medical history of compensated cirrhosis due to hepatitis C virus (HCV) in sustained virologic response (SVR) presented to clinic for follow-up. Alpha-fetoprotein level was elevated to 464. Magnetic resonance imaging (MRI) of abdomen was significant for HCC (Li RADS 5), with no signs of metastatic disease seen on computed tomography (CT) chest and pelvis. The patient was treated with TACE with RFA for early stage of HCC. Six months later, the patient reported chronic intermittent right sided chest and right sided abdominal pain. Physical examination was unremarkable. X-ray chest revealed one-centimeter calcific density projecting over the right upper quadrant of the abdomen (Figure 1A). Repeat MRI of the abdomen showed a stable necrotic

treatment cavity and a non-enhancing nodular focus of fat along the posterior aspect of the cavity (Figure 1B, C) without evidence of new HCC. Case was reviewed with a radiologist who deemed the calcification seen on chest x-ray is due to liver directed therapy. Patient was managed conservatively with pain medications.

Discussion: Patients who undergo TACE commonly (about 60%-80% of the time) experience a postembolization syndrome of abdominal pain, fever, and nausea hours to days after the procedure. However, persistent abdominal or chest pain should be evaluated with further imaging. There is a rare possibility of necrosis in the tumor itself even without therapy leading to similar calcification. However, these calcifications are more common after liver directed therapy. Our case demonstrates that a hepatic calcified density can occur as a complication of TACE rather than a de novo tumor in patients with history of HCC and liver directed therapy. This is due to tumor cell necrosis followed by dystrophic calcification which is better seen on x-rays and CT when compared to MRI examination. This case demonstrates a distinctive differential diagnosis of calcified hepatic densities in patients who have previously undergone TACE procedure for HCC.



[3288] **Figure 1.** A: One centimeter calcific density projecting over the right upper quadrant of the abdomen B, C: Repeat MRI of abdomen showed a stable necrotic treatment cavity and a non-enhancing nodular focus of fat along the posterior aspect of the cavity without evidence of HCC.

S3289 WITHDRAWN

S3290

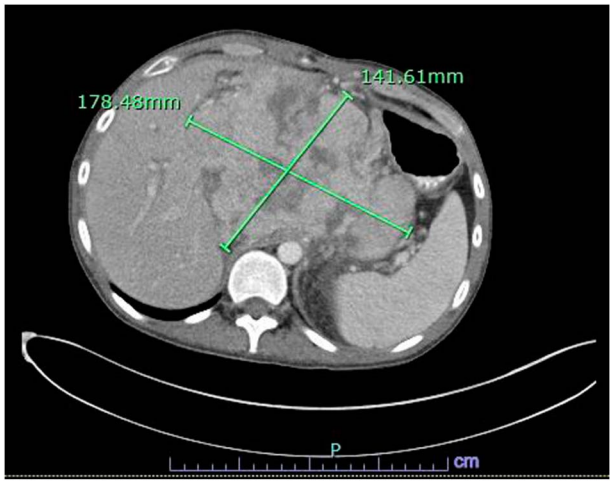
A Rare Diagnosis of Hepatic Hemangioendothelioma Neoplasm of the Liver

Muhammad Farooq, MD.
East Carolina University Brody School of Medicine, Greenville, NC.

Introduction: Hepatic Epithelioid Hemangioendothelioma (HEH) is a rare malignant vascular soft tissue sarcoma with only about 200 cases reported in the current literature.It is often discovered incidentally and is often misdiagnosed, leading to poor prognosis.

Case Description/Methods: A 31-year-old male with no prior medical history presented to the emergency room (ER) with change in mental status, a few months history of generalized abdominal discomfort and weight loss of 100 pounds over two years. He had a history of prior heavy alcohol use but was abstinent for 4 years. On arrival, he had stable vital signs and labs were significant for BUN of 39 mg/dL, creatinine 4.5 mg/dL, AST 121 U/L, ALT 135 U/L, Alkaline Phosphatase of 166 U/L, total bilirubin of 0.8 mg/dL, LDH of 467 U/L, WBC count of 11.0 k/uL with predominant neutrophils, INR of 2.1, PT of 26.7s and serum ammonia level of 244 μ/dL. Ethanol level was < 10. Urine analysis showed pyuria and bacteriuria. Urine toxicology screen was positive for cannabinoids. Acute hepatitis panel was negative. Tylenol and Salicylate levels were undetectable. CT head and MRI of the brain did not reveal any acute intracranial process. CT Abdomen and Pelvis with contrast showed left sided renal stone, multiple right hepatic lobe masses measuring up to 4.4 cm, a heterogeneously enhancing mass invading/replacing the pancreas and left hepatic lobe measuring 17.8cm x 14.1 cm with marked mass effect on patent portal vein along with bulky retroperitoneal, periportal and mesenteric lymphadenopathy. CT chest revealed a 2.7 cm and 5 mm nodule in the left lung concerning for metastatic process.The patient was treated for urinary infection and ureteral stone and received lactulose with return of renal function and mental status to baseline. Subsequent CT guided biopsy of liver mass revealed a diagnosis of hepatic hemangioendothelioma. The patient was started on combination chemotherapy with Atezolizumab and Bevacizumab but follow up CT scan showed worsening disease. He was subsequently started on second line chemotherapy but became progressively more confused and required hospital admission for sepsis at which point the family decided to transition to comfort measures only and the patient eventually passed away (Figure).

Discussion: HEH is a rare entity with an unclear etiology. Liver transplantation for localized disease remains the only viable treatment option. Different chemotherapy regimens have been suggested for metastatic HEH but there is no general consensus for a standard regimen.



[3290] **Figure 1.** Liver mass.

S3291

A Unique Case of Drug Induced Liver Injury Related to Fluoxetine

Lauren A. Lyssy, DO, Ryan Mui, DO, PhD, Matthew Griffin, DO.
McLaren Greater Lansing, Lansing, MI.

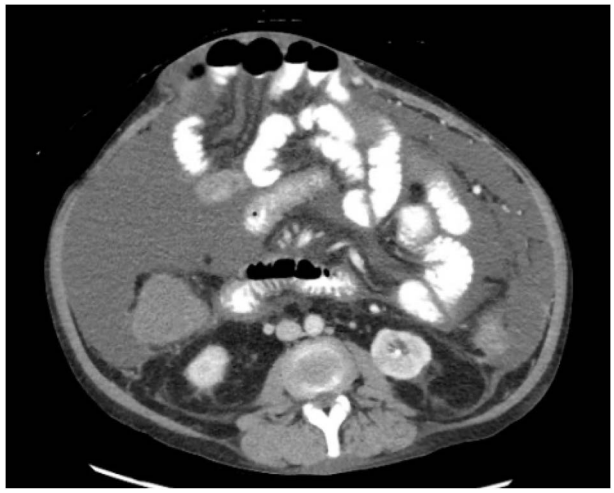
Introduction: Fluoxetine is a commonly prescribed antidepressant with generalized gastrointestinal side effects that can include nausea and diarrhea. While fluoxetine is metabolized in the liver, fewer than 1% of patients develop a mild and self-limited transaminitis. Here, we present a unique case of clinically apparent drug-induced liver injury (DILI) soon after fluoxetine initiation.
Case Description/Methods: A 28-year-old male with schizophrenia and bipolar disorder was admitted to the hospital due to a 3-day history of worsening right upper quadrant (RUQ) abdominal pain with a 2-day history of yellowing of the skin and eyes. A thorough history revealed that he was on chronic citalopram and olanzapine therapy but was started fluoxetine (20 mg daily) 5-days prior. Additionally, he denied use of alcohol, recreational drugs, and herbal supplements, and denied a history of known liver disease. On admission, the patient was alert and oriented x4 with jaundice, scleral icterus, and asterixis. Laboratory work demonstrated WBC 15,000, platelets 235,000, ammonia 131, AST 3219, ALT 6574, ALP 181, total bilirubin 22.9, and INR 1.97. Acetaminophen and salicylate levels were unremarkable. Fluoxetine was held immediately, and the patient was empirically given N-acetylcysteine. RUQ ultrasound was unremarkable. MRCP demonstrated fatty infiltration of the liver without nodularity or biliary dilation. A thorough workup including viral hepatitis (A, B, C, E), EBV, CMV, HIV, hemochromatosis, Wilson's disease, and autoimmune hepatitis was unremarkable. A liver biopsy was performed, and pathology was consistent with mixed-pattern DILI. With the removal of fluoxetine, the patient's liver function tests and clinical symptoms continued to improve, and on hospital day 10 he was discharged in stable condition.
Discussion: Transaminitis from fluoxetine is typically mild, asymptomatic, and self-limited. Our case highlights a rare instance where fluoxetine precipitated DILI. It is unclear whether polypharmacy with his other psychiatric medications, or underlying hepatic steatosis, were predisposing factors to developing fluoxetine-associated DILI. Higher awareness of fluoxetine-associated hepatotoxicity is needed and caution should be used when considering current medications and underlying liver pathology when starting this medication.

S3292

A Rare Complication of End-Stage Liver Disease: Flood Syndrome

Bianca Varda, MD, Jake Jasurda, DO, Steve Scaglione, MD.
Loyola University Medical Center, Maywood, IL.

Introduction: Flood syndrome describes a condition seen in the setting of end-stage liver disease in which an umbilical hernia ruptures leading to a rush of fluid through the defect. It is a rare complication of cirrhosis and has a high mortality rate. Herein, we present two cases of flood syndrome in patients with advanced liver disease.
Case Description/Methods: *Case 1*: A 37-year-old female with a history of factor Leiden deficiency, MTHFR deficiency, and decompensates alcoholic cirrhosis presented with 1 day of continued fluid leakage from an umbilical wound. She was afebrile, normotensive yet tachycardic to 114 bpm. Abdominal exam showed a positive fluid wave shift and small umbilical hernia with ascitic fluid in the stoma bag. Labs showed a total bilirubin of 11.6 mg/dl, AST of 61 U/L, ALT of 36 U/L, and ALP of 79 U/L. She was treated with antibiotics and underwent hernia repair. She later required large volume paracentesis before the fluid leakage stopped and she was discharged home. *Case 2*: A 48-year-old female with alcoholic cirrhosis and a history of incisional abdominal hernia secondary to laparotomy presented with abdominal distension, pain and leakage of fluid through the hernia site. Exam showed a midline abdominal protuberance with an ulcerated hernia. CT confirmed the abdominal wall hernia with sigmoid colon in the hernia sac and ascitic fluid and air (Figure 1). She was treated with antibiotics and the defect was closed by general surgery prior to discharge.
Discussion: Flood syndrome is a rare complication seen in cirrhotic patients with ascites defined as a rush of fluid through rupture of an umbilical hernia. It is associated with serious complications and a high mortality rate of up to 30%. As such, the most important step in management of ascites is prevention. Sodium and fluid restrictions and diuretics may be used to reduce hypervolemia in these patients. Paracentesis is often necessary to aid in fluid removal in those with recurrent ascites; however, the procedure can also increase the risk of developing flood syndrome. Ultimately, patients may require surgery to repair the defect.



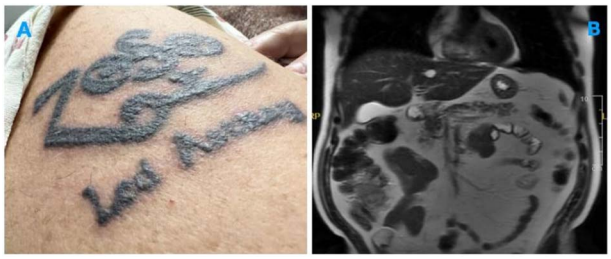
[3292] **Figure 1.** CT demonstrating an abdominal wall hernia with sigmoid colon in the hernia sac and ascitic fluid and air.

S3293

A Rare Case of Sarcoidosis Involving Multiple Tattoos and Organ Systems in an Elderly White Male

Garry Lachhar, MD, Elnaz Mahbub, DO, Elaina Suridis, DO, Cassidy Guida, DO, Sabrina Mohsin, DO, Andrew Melek, MS, Saloni Sachar, MS, Jorge Maldonado, MS, Nishok Srinivasan, MS, Sejal Kotecha, MS, Israel Umoh, MS, Heidi Roppelt, MD, Ahsan Khan, MD.
Stony Brook Southampton Hospital, Southampton, NY.

Introduction: Sarcoidosis is a multisystem inflammatory disease of unknown etiology with a wide range of clinical presentations, most commonly seen in African American women of reproductive age. We present a unique case of sarcoidosis with tattoo and liver involvement in an older white male, a very rare occurrence with about a .04% incident rate.
Case Description/Methods: A 65-year-old male presented to the emergency department with dry cough, dyspnea, progressive malaise, night sweats, and insomnia for 3 months. The patient reported having a sebaceous cyst on his left upper extremity around a tattoo which he had for 40 years. The patient had no family history of rheumatologic disease and denied any history of smoking, vaping or toxic exposures. Lab results showed significantly elevated liver enzymes and c-reactive protein, and an evaluation of his presumed cyst noted that his entire tattoo was raised, erythematous and warm (Figure 1A). The 15 year old tattoo on his contralateral shoulder was similar, and neither tattoo had a history of trauma. Computed tomography of the chest revealed mediastinal and hilar adenopathy and ruled out pulmonary embolism and masses. Of note, angiotensin converting enzyme (ACE) level was elevated at 37. The patient was diagnosed with extrapulmonary sarcoidosis with liver involvement (Figure 1B).
Discussion: Sarcoidosis may affect any organ in the body; pulmonary involvement is most common, followed by involvement of the skin, eyes and lymph nodes. Liver involvement occurs in 20% of cases. The first association between a tattooed skin granulomatous complication and generalized sarcoidosis was reported in 1952, and since then, multiple cases have been described as occurring in association with hilar adenopathy and pulmonary sarcoidosis. In the majority of reported cases, the tattoo reactions subsequently led to the diagnosis of systemic sarcoidosis. The patient's elevated serum ACE had diagnostic value for sarcoidosis. The etiology of tattoo sarcoidosis is still unknown, but it may arise from chronic antigenic stimulation in predisposed patients. Further investigation is warranted with a larger number of patients and long-term follow-up to better understand this phenomenon and improve diagnostic accuracy and management. Awareness of tattoo involvement as a sign of generalized sarcoidosis can aid in a timely diagnosis and prompt treatment. A high index of suspicion is required even in non-classical demographics.



[3293] **Figure 1.** Extrapulmonary Sarcoidosis manifestations. Figure 1A: Tattoo. Figure 1B: Liver MRI.

S3294

Acute Hepatitis E Infection With False Positive Cross-Reactivity to Epstein-Barr Virus

Justin J. Wagner, DO, Ishani Patel, MD, Meron Debesai, MD, Nikolas St. Cyr, MD, Michael Bernstein, MD.
Coney Island Hospital, Brooklyn, NY.

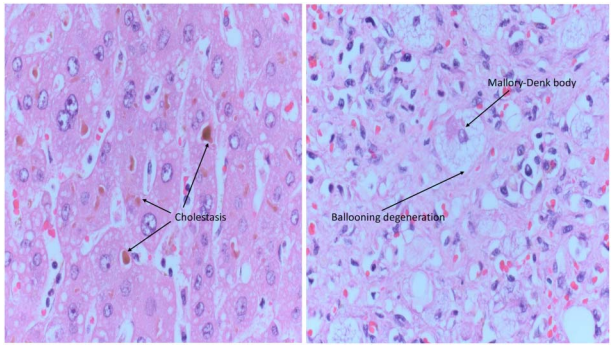
Introduction: Hepatitis E virus (HEV) can cause acute or chronic viral hepatitis and is a common cause of acute viral hepatitis worldwide and is an important public health concern. A high level of HEV, hepatitis A virus (HAV), cytomegalovirus (CMV), and Epstein-Barr virus (EBV) cross reactivity can occur, which indicates that serology can sometimes be unreliable in the diagnosis of acute viral hepatitis.
Case Description/Methods: A 28-year-old Pakistani male presented with right upper quadrant abdominal pain, general malaise, and deranged liver function tests. Work-up for his abnormal liver tests was unremarkable except for detection of Hepatitis E antibody, Hepatitis E IgM antibody, and Epstein-Barr Virus IgM antibody with polymerase chain reaction (PCR) analysis revealing no detection of Epstein-Barr Virus DNA. The patient was diagnosed with acute hepatitis E with a false-positive EBV infection due to serological cross-reactivity. Supportive care was continued, liver function tests trended down, and the patient clinically improved and was discharged with outpatient follow-up.
Discussion: The incubation time for HEV infection can last up to six weeks and HEV RNA, anti-HEV IgM, and anti-HEV IgG antibodies can be detected at the time of diagnosis. Anti-HEV IgM antibodies have a short window of positivity at three to four months, whereas HEV RNA can be detected in the blood within three weeks with viral shedding lasting up to six weeks in the stool. The enzyme immunoassay is the most widely used serological method for the identification of anti-HEV IgG and IgM antibodies, but the identification of anti-HEV IgM and rising titers of anti-HEV IgG antibodies are inadequate for diagnosis due to the lack of specificity for these antibodies. Confounding the diagnosis of HEV is the incidence of HEV, HAV, CMV, and EBV cross reactivity, which is posited to be due to polyclonal B-cell stimulation. The diagnosis of HEV infection in the clinical setting relies on the performance of assays of anti-HEV IgM, and subsequently the awareness of factors influencing diagnostic accuracy is crucial regarding potential treatment options. This case suggests that both anti-HEV IgM and EBV IgM should be interpreted with caution in acute HEV infection and that confirmatory testing with PCR analysis should be conducted to evaluate for false-positive serological cross-reactivity. We conclude that the diagnosis of viral hepatitis should be based on characteristic symptoms, elevated liver enzymes, serology, and confirmatory PCR testing.

S3295

Acute Liver Failure in an Incidentally COVID-19 Positive Patient With No Respiratory Symptoms

Ritwick Mynam, BS, Saatchi Kuwelker, MD, Ariadna Perez Sanchez, MD, Allison Harrington, MD, Jacob Ritter, MD, Ricardo De la Cruz, MD.
University of Texas Health Science Center, San Antonio, TX.

Introduction: Given that COVID-19 uses angiotensin converting enzyme 2 as its primary viral entry receptor, which is present on multiple organs including the liver, it has the capacity to cause severe systemic disease. Several studies have documented elevations in liver associated enzymes (LAE), in patients with COVID-19 ranging from 2 to 50 % above baseline. Currently, it is not entirely clear whether LAE elevations can be used for prognostication purposes. Here, we present a case of COVID-19 induced fulminant liver failure in an otherwise asymptomatic non-hypoxemic patient.
Case Description/Methods: A 50 year-old male with no significant past medical history who had recently recovered from a mild COVID-19 infection ten days prior presents with symptoms of fatigue, abdominal pain, nausea and vomiting for three days. Physical exam revealed diffuse abdominal tenderness and scleral icterus. Patient denied recent travel, alcohol use, contact with animals, multiple sexual partners, or drug/medication use. Lab findings were significant for elevation of aspartate transaminase, alanine transaminase, total bilirubin, and INR of 4985 IU, 9895 IU, 11 mg/dl and >9 respectively. CT abdomen revealed periportal edema with perihepatic free fluid. In conjunction with marked hepatocellular transaminitis, these findings were concerning for acute hepatitis. However, viral panels including Hepatitis A, B, C, D, E, EBV, CMV and HSV were negative. Drug screens, autoimmune panel, ceruloplasmin level and fungal panels were also negative. Over the next 3 days patient developed lactic acidosis, shock, hepatic encephalopathy, and liver and kidney failure that necessitated ICU admission. Deemed to be too unstable for a liver transplant, despite aggressive supportive measures, the patient died on the 5th day of hospital stay. A post-mortem evaluation of hepatic tissue revealed extensive cholestasis, mild mixed steatohepatitis, Mallory bodies, periportal fibrosis, ballooning hepatocytes, and lymphocytic infiltrates throughout the parenchyma (Figure)
Discussion: Although COVID-19 infections presenting with hypoxemia have shown to present with elevated LAEs in the past, very few cases of fulminant liver failure in an otherwise non-hypoxemic patient have been documented. In the setting of no other explanation for acute liver failure, it could be hypothesized that COVID-19 induced viral hepatitis or hypercoagulability leading to a Budd Chiari-like syndrome could have potentiated liver failure in this patient.



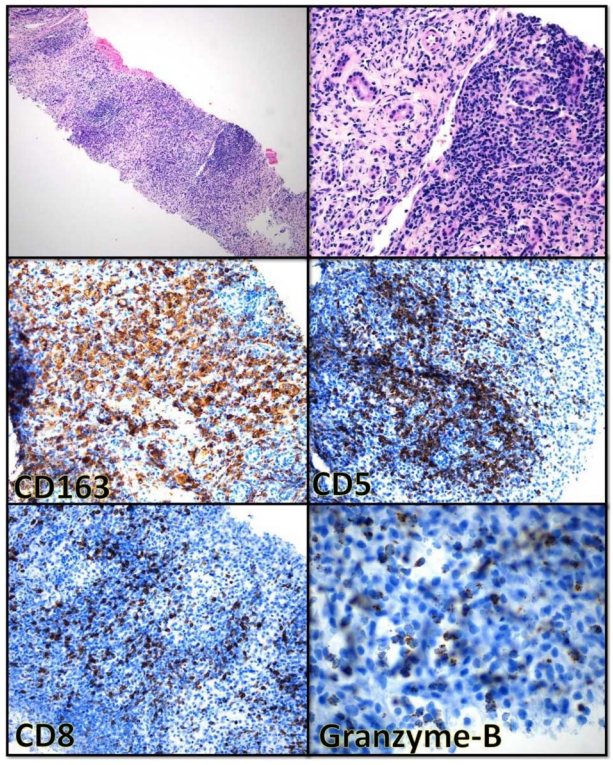
[3295] **Figure 1.** 1a (left): Liver biopsy at the time of autopsy showing cholestasis and mild mixed steatohepatitis Figure1b (right): Liver biopsy at the time of autopsy showing ballooning degeneration and Mallory Denck bodies.

S3296

Acute on Chronic Liver Disease Due to Hepatic Infiltration of Chronic Lymphoproliferative Disorder of Natural Killer Cells

Estefania M. Flores, MD, Ibrahim Abukhiran, MBBS, Antonio Sanchez, MD.
University of Iowa Hospitals and Clinics, Iowa City, IA.

Introduction: We report a case of acute on chronic liver disease with a predominantly cholestatic pattern which was diagnosed as hepatic infiltration of chronic lymphoproliferative disorder of natural killer cells (CLPD-NK) after extensive workup and an initial false-negative liver biopsy.
Case Description/Methods: A 54-year-old woman with alcohol use disorder, celiac disease, and Hashimoto thyroiditis initially presented with 8-week history of night sweats, fever and jaundice. She reported taking daily naproxen for a few weeks. The physical exam was remarkable for jaundice. Labs showed bilirubin of 2.5, AST 869, ALT 543, alkaline phosphatase 966 and INR with 0.9. Right upper quadrant ultrasound showed no ductal dilation with a non-specific hypoechoic area in the superior right lobe and nodular appearance of the liver. Autoimmune workup revealed positive anti-smooth muscle antibody in a low titer but was otherwise negative. A liver biopsy showed massive hepatic necrosis with marked pan-acinar lymphocytic inflammation. The leading cause of her liver injury was thought to be drug-induced due to naproxen in a background of chronic liver disease. Her liver disease progressed, developing ascites, encephalopathy and pruritus refractory to treatment. Nine months after initial presentation she presented with a hemoglobin of 7, MCV 104, LDH 590, Haptoglobin < 10, total bilirubin 2.9, and ferritin of 4289. WBC count was noted to have an absolute increase in lymphocytes, bone marrow biopsy showed CLPD-NK. A second liver biopsy was performed showing diffuse lymphohistiocytic infiltrate and flow cytometry revealed atypical lymphocytes/NK cell population. The patient was started on chemotherapy which led to clinical and biochemical improvement of her liver disease. (Figure)
Discussion: CLPD-NK is a rare and heterogeneous disorder. Unlike other NK cell disorders, CLPD-NKs is not associated with EBV infection. This disorder follows an indolent course, making it difficult to detect it in the initial stages. Hepatic abnormalities in patients with lymphoproliferative disorders can occur from direct infiltration by abnormal cells, bile duct obstruction by lymphadenopathy, paraneoplastic syndrome, hemophagocytic syndrome, and reactivation of viral hepatitis. Our patient is to our knowledge the first case reported of hepatic infiltration from CLPD-NK. Treatment is indicated in symptomatic patients. Hence, early diagnosis is essential so patients can receive timely administration of immunosuppressive therapy.



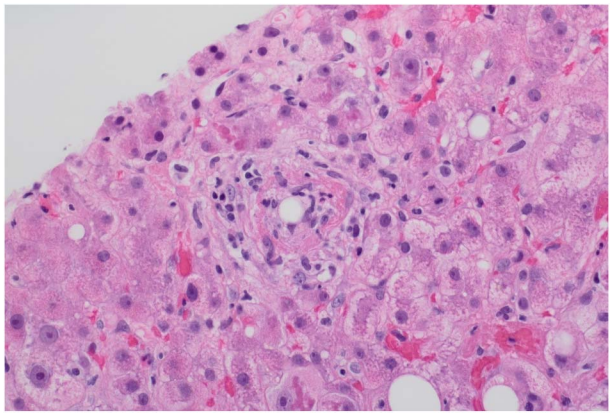
[3296] **Figure 1.** Liver with diffuse lymphohistiocytic infiltrate. Sections show an atypical lymphohistiocytic infiltrate of the liver and composed of small to medium-sized cells that stain CD2+, CD3+, CD4 and CD8 with down-regulation, CD3, CD5+, CD7+, CD20 focally+, CD68+, CD163+, and Granzyme B +.

S3297

Acute Q Fever Diagnosis in a Patient Presenting With Alcohol Associated Hepatitis and Positive Autoantibodies

Charlie W. Fox, MD, Juan F. Gallegos-Orozco, MD, Jordon K. March, MD.
University of Utah, Salt Lake City, UT.

Introduction: Q fever is caused by infection with zoonotic organism Coxiella burnetti (CB). It is often characterized by fever, headaches, myalgias and malaise. It commonly affects the liver leading to elevated aminotransferases. There are case reports of autoantibody positivity in cases of Q fever, namely smooth muscle antibodies and Anti-neutrophilic antibodies. There are no reports discussing the presentation or management of both alcohol associated hepatitis (AAH) and acute Q fever in a patient with positive liver autoantibodies. In this case we report a patient presenting with AAH and positive autoantibodies who was diagnosed with acute Q fever.
Case Description/Methods: 59-year-old male presents with jaundice, nausea, vomiting and abdominal pain. He drank ten beers daily for more than ten years and quit two weeks prior to presentation. Admission labs shown in Table 1. Unremarkable abdominal ultrasound. He was diagnosed with severe AAH, MELD 25, DF 59.5, started on prednisolone. Liver biopsy demonstrated active and chronic steatohepatitis with significant hepatocyte ballooning and Mallory-Denk bodies with stage 3 fibrosis. There were also fibrin ring granulomas typical of CB infection. CB titers were typical of acute infection. He started doxycycline 100mg BID for 14 days. At follow up labs were improved and symptoms had resolved. (Figure)
Discussion: Presentation was consistent with AAH for which he was started on corticosteroids. However, elevated auto-antibodies raised concern for AIH so biopsy was performed. The findings confirmed that alcohol was the main etiology with active steatohepatitis. However, the presence of fibrin ring granulomas was a surprise and was another possible etiology for his acute liver injury. Testing for CB confirmed acute Q fever. Prednisolone was stopped and he started antibiotics. Q fever diagnosis was intriguing because he did not present with classic symptoms. His elevated autoantibodies are interesting as there have been previous cases reporting an association between Q fever and AIH. We did not find evidence of this on biopsy and he improved with antibiotics alone. This case reminds us to maintain a broad differential diagnosis in patients with acute liver injury because there may be more than one process contributing. It reinforces the value of liver biopsy in clarifying the diagnosis, especially when there are positive autoimmune markers. Furthermore, it highlights knowledge gaps regarding the role of autoimmune markers in both infectious and primary autoimmune liver disease.



[3297] **Figure 1.** Fibrin Ring Granuloma on Liver Biopsy.

Table 1.

	Total Bilirubin	Alkaline Phosphatase	AST	ALT	F-actin Ab IgG (ref< 19)	Mitochondrial Ab IgG (ref< 20)	Serum IgG	INR	CB IgG phase II titer	CB IgM phase II titer
Admission	19	159	142	100	63	73.6	1408	1.8	1:131,072	1:4,096
Follow up	2	319	52	60	-	-	-	1.2	-	-

S3298

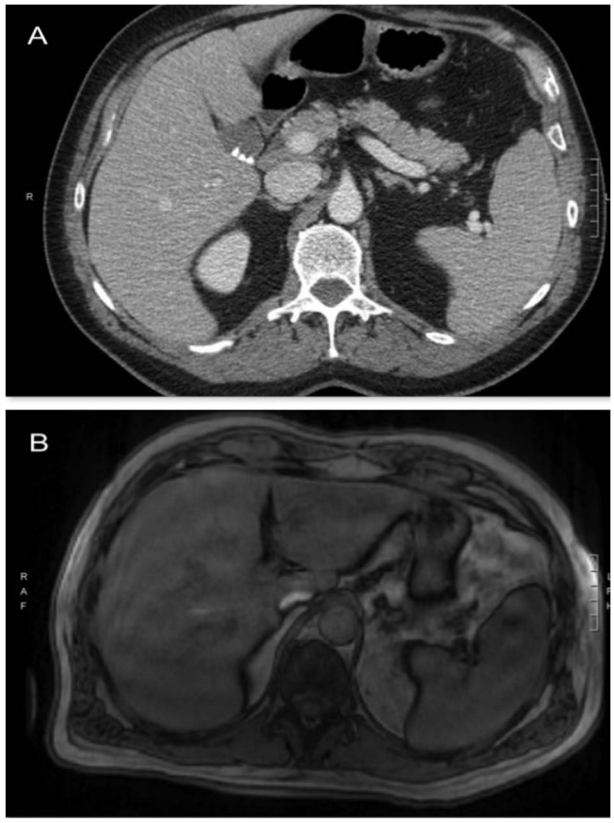
Abnormal Liver Function Tests in an Immunocompetent Patient With Severe Babesiosis

Julianna Tantum, DO[1], Andrew Bensinger, DO[1], Patricia Wong, MD[2].
[1]Lankenau Medical Center, Wynnewood, PA; [2]Lankenau Medical Center, Philadelphia, PA.

Introduction: Babesiosis is a tick-borne disease that is caused by the Babesia parasite. Babesia is a rare cause of liver function test (LFT) abnormalities. This case outlines the importance of keeping tick-borne and parasitic infections on the differential for abnormal LFTs, especially in patients who live in the Northeastern United States.

Case Description/Methods: A 61-year-old male presented to the emergency department for fevers, abdominal pain, nausea, decreased oral intake, and 10-pound weight loss. On arrival, he was febrile to 103.1°F and blood pressures 80-90s. His exam was notable for a soft, non-tender abdomen without hepatosplenomegaly or ascites and skin without rashes or jaundice. Labs showed AST 163 IU/L, ALT 203 U/L, alkaline phosphatase (AP) 184 IU/L, total bilirubin(TB) 3.7 mg/dL, direct bilirubin(DB) 1.7mg/dL, WBC 3.5 K/uL, hemoglobin 11.8 g/dL, platelets 35 x 10^9/L. CT abdomen and pelvis with IV contrast and MRI abdomen with gadolinium showed an enlarged fatty, mildly lobulated liver, cholelithiasis, distended gallbladder, no biliary ductal dilation and mild splenomegaly (Image 1A &1B). A peripheral blood smear was obtained since he lived near woods and fed foxes, showing Babesia with 4% parasitemia. He was started on atovaquone and azithromycin. Lyme Disease was positive; Doxycycline was added. An undetectable haptoglobin and LDH of 679 were consistent with hemolysis. LFTs peaked at AST 436 IU/L, ALT 316 IU/L, AP 235 IU/L, TB 5.8 mg/dL, DB 3.8 mg/dL on hospital day 5. Viral hepatitis serologies, ANA, ASMA, and ceruloplasmin were unrevealing. His symptoms and labs improved. Five days after discharge no parasites were seen on blood smear. LFTs normalized six weeks later.

Discussion: Babesiosis is a zoonotic infection that is transmitted by Ixodes ticks. Symptoms depend on patient age and immunocompetence, ranging from asymptomatic infection to severe disease including hemolytic anemia, DIC, renal failure, sepsis, and pulmonary edema. Gastrointestinal symptoms of nausea, vomiting, abdominal pain, and anorexia are common. Mild LFT abnormalities may occur but severe elevations are uncommon. Acute liver failure has been reported. This case highlights the importance of a broad differential when evaluating patients with elevated LFTs. While fevers and malaise may suggest underlying infection, patients with babesiosis often do not recall a tick bite or develop a rash. Obtaining a careful history for tick borne exposures during summer months in the Northeast is important, as outlined in this case.



[3298] **Figure 1.** 1A. Computed Tomography (CT) of the abdomen and pelvis with IV contrast showed evidence of hepatosplenomegaly and cholelithiasis. 1B. Magnetic Resonance Imaging (MRI) of the abdomen and pelvis with gadolinium showed cholelithiasis, no evidence of ductal dilation, and splenomegaly.

S3299

Acute Liver Injury as Initial Presentation of Adult Onset Still's Disease

Yara Dababneh, MD[1], Susheela Sreedhar, MD[2], Ankita Nagirimadugu, MD[1], Vivek Mendiratta, MD[1], Dilip Moonka, MD[1], Donard Haggins, MD[1].
[1]Henry Ford Hospital, Detroit, MI; [2]Wayne State University, Detroit, MI.

Introduction: We present a case of acute liver injury as a first presentation of Adult-Onset Still's Disease (AOSD).
Case Description/Methods: A 20-year-old man presented with fever and nausea. On admission liver function tests (LFTs) were notable for ALT 81 IU/L, AST 42 IU/L, ALP 58 IU/L and total bilirubin 6.7 mg/dL. Infectious work up was notable for positive EBV IgM that was treated appropriately. He presented again with worsening headache, fever, dark urine and petechial rash on both lower extremities. LFTs were still elevated. Patient was symptomatically managed and the elevation in LFTs was attributed to infectious etiology. Patient was discharged home with close follow up. He presented again, nine days after initial symptoms onset with worsening symptoms, jaundice and new left hip pain with lower extremity weakness. Patient had taken one pill of Metronidazole from India but denied any other drugs. LFTs were ALT 214 IU/L, AST 215 IU/L, ALP 215 IU/L and total bilirubin 6.6 mg/dL. Patient's liver enzymes continued to rise despite discontinuing hepatotoxic medications, with ALT 186 IU/L, AST 146 IU/L, ALP 219 IU/L, total bilirubin 10.7 mg/dL and direct bilirubin 6.4 mg/dL. Therefore, a liver biopsy was ultimately performed. Histology showed a portal inflammatory process, with acute eosinophil spill over to the liver, without hepatic necrosis. Rheumatology team was consulted due to arthralgia. The patient met the Yamaguchi criteria for AOSD. He was started on prednisone 40 mg daily, with subjective and objective improvement with down trending LFTs. Outpatient follow-up with both rheumatology and hepatology continued to show significant improvement and down-trending LFTs. He continues to be on a prolonged steroid taper over 12 weeks.
Discussion: In our case the patient met the Yamaguchi criteria which involves major and minor criteria with transaminitis included. In this case, the main challenge was to find a suitable diagnosis that fits all the symptoms. A previous case report suggested that Drug Induced Liver Injury (DILI) might be the inciting factor in AOSD activation, which might be proposed in our case as well. It was reported that liver involvement in AOSD can be attributed to its association with Macrophage Activation Syndrome (MAS). Treatment mainly focuses on treating AOSD itself with systemic steroid therapy. In conclusion, acute liver injury is being increasingly reported in literature and this case supports acute liver injury in association with AOSD which can be aggravated by DILI or MAS.

S3300

Acetaminophen Toxicity Amplified: Drug-Induced Liver Injury in a Malnourished Patient

Nicholas Kunce, MD, Azfar S. Syed, DO, MBA, Dawn Torres, MD.
Walter Reed National Military Medical Center, Bethesda, MD.

Introduction: Acetaminophen toxicity is a leading cause of acute liver injury and failure in the U.S. Excess acetaminophen results in formation of N-acetyl-p-benzoquinoneimine (NAPQI) which causes irreversible oxidative hepatocyte injury and cell necrosis. Treatment with N-acetylcysteine (NAC) replenishes glutathione stores which inactivate NAPQI. In a healthy adult, acetaminophen doses less than 4 g per 24 hours are considered safe. Toxicity from therapeutic doses of acetaminophen, in the setting of malnourishment, has been rarely reported and is presented here.
Case Description/Methods: A 37-year-old female with a history of gastroparesis and chronic malnutrition was admitted for elevated liver enzymes on routine blood work. She reported symptoms of fatigue, nausea, and poor oral intake from suspected gastroparesis. She also admitted to taking 1300 mg of acetaminophen intermittently during the past week for myalgias. Exam revealed normal mentation without evidence of hepatic encephalopathy (HE), cachexia with a BMI of 15.2, and mild epigastric and left upper quadrant tenderness. Laboratory data (36 hours after last ingestion of acetaminophen) revealed an ALT of 1180 u/L, AST of 1950 u/L, ALP of 121 u/L, total bilirubin of 2.7 mg/dL, direct bilirubin of 2.5 mg/dL and INR of 1.6. Her acetaminophen level was 15.8 mcg/mL. A liver ultrasound was unremarkable. NAC was subsequently administered as per protocol 21 hour infusion. She was ruled out for other etiologies of acute liver injury. She never developed HE and her liver enzymes, along with INR, normalized at discharge.
Discussion: This case highlights the lower threshold for toxicity of acetaminophen in a malnourished patient. In previous case reports, acetaminophen doses less than 2 g per 24 hours are listed as being safe in a nutritionally deficient patient. Our case questions this assertion given our patient ingested 65% of this amount. Evidence in the relatively limited pool of literature suggests that malnourished patients generally have low stores of glutathione, thereby limiting the metabolic clearance of acetaminophen ingested even at normal dosages, amplifying total acetaminophen levels, and leading to increased NAPQI. Prospective

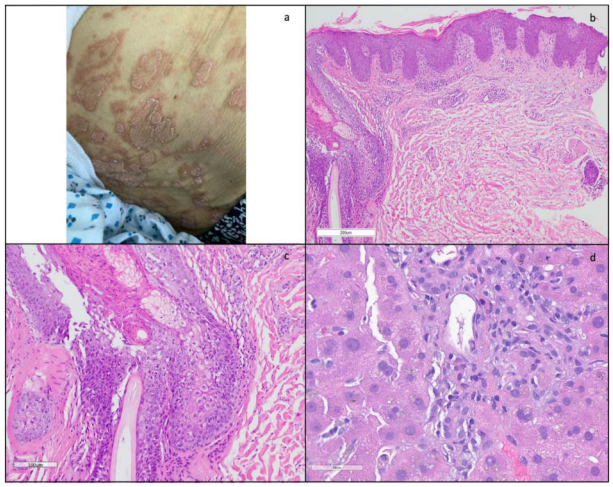
study is limited to rat models and single case reports. Fortunately, our patient was promptly treated with NAC and did not progress to liver failure. This case highlights the narrow therapeutic window of acetaminophen in malnourished patients, one that warrants close observation by clinicians.

S3301

Abnormal Liver Enzymes in Thymoma-Associated Multiorgan Autoimmunity

Bianca Di Cocco, MD, Hnin Ingyin, MD, Michael J. Mintz, MD, Cynthia Magro, MD, David Wan, MD.
New York-Presbyterian/Weill Cornell Medical Center, New York, NY.

Introduction: Abnormal liver function tests (LFTs) are common in hospitalized patients, particularly in those treated with antibiotics. When due to medications, LFT abnormalities are typically transient and resolve with drug discontinuation. However, for patients with known thymomas and a new rash, rising LFTs can be a sign of thymoma-associated multiorgan autoimmunity (TAMA). We present a case where abnormal LFTs were initially attributed to an antibiotic, but were revealed to be TAMA based on skin and liver histopathological findings.
Case Description/Methods: A 74-year-old man with myasthenia gravis and a malignant thymoma presented with shortness of breath. He had recently been hospitalized for methicillin-resistant *Staphylococcus aureus* aspiration pneumonia treated with trimethoprim-sulfamethoxazole (TMP-SMX). On admission, he had newly elevated LFTs with a total bilirubin of 1.7 mg/dL and alanine aminotransferase (ALT) of 259 U/L. He also had a widespread papulosquamous rash (Figure 1a). The LFT abnormalities were initially attributed to TMP-SMX, but continued to rise despite drug discontinuation. Total bilirubin reached 28.4 mg/dL and ALT 698 U/L. A skin biopsy revealed psoriasiform epidermal hyperplasia accompanied by a lymphocyte-mediated epithelial injury targeting the epidermis, hair follicle and eccrine apparatus. Superficial and follicular-based dyskeratosis unaccompanied by lymphocyte satellitosis was observed. The skin biopsy was consistent with TAMA (Figure 1b-c). A liver biopsy was also performed which demonstrated lymphocyte-mediated injury to the interlobular bile ducts (Figure 1d), consistent with either drug-induced liver injury or as a further manifestation of TAMA. Following multidisciplinary discussion, the patient received antithyroglobulin (ATG) and dexamethasone for TAMA.
Discussion: TAMA is a rare graft-versus-host-like disease associated with thymomas. While skin is the most commonly affected organ, it can also involve the hepatobiliary and gastrointestinal systems. The pathophysiology is not fully elucidated, but is potentially due to the absence of the autoimmune regulator in thymomas that eliminates autoreactive T-cells, or due to decreased amounts of FoxP3-positive regulator T-cells. Treatment is aimed at addressing both the underlying thymoma with either surgical resection or ATG, and the autoimmune response with either a corticosteroid or immunosuppressant. However, given the condition's rarity, there are no large studies to support these treatments and prognosis is poor.



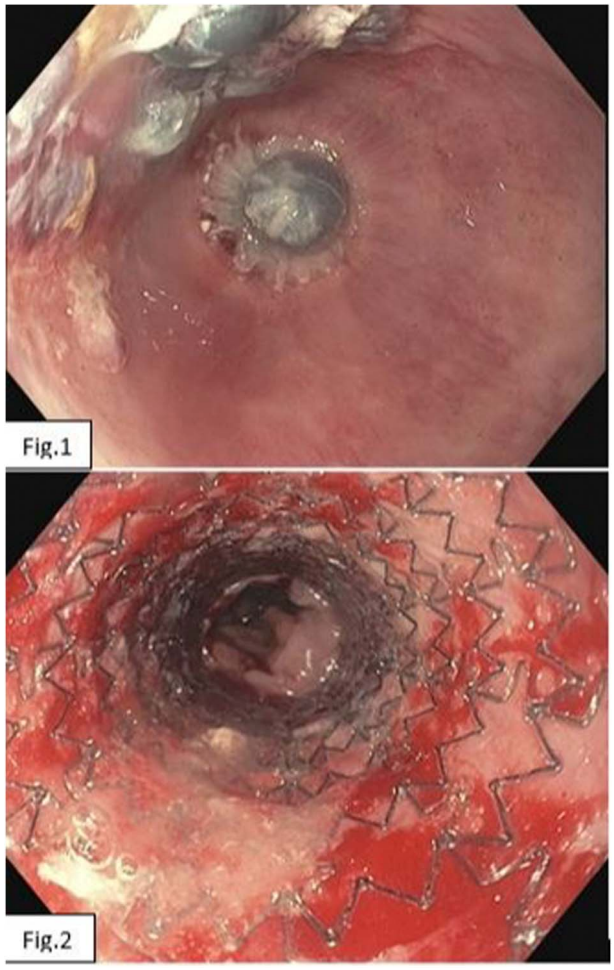
[3301] **Figure 1.** A. Papulosquamous rash on patient's back. B. Skin biopsy with psoriasiform hyperplasia and interface dermatitis. C. Skin biopsy at higher power demonstrating follicular plugging by keratin and superficial clustered dyskeratosis without lymphocyte satellitosis. D. Liver biopsy with interlobular bile ducts surrounded and infiltrated by lymphocytes, monocytes, and a few neutrophils, reflective of a cellular type IV reaction targeting the bile duct epithelium with resultant cholestasis.

S3302

Acute Esophageal Obstruction: An Uncommon Complication of Variceal Ligation Treated With Band Removal and Stenting

Suaka Kagbo-Kue, MBBS[1], Blanca Lizaola-Mayo, MD[1], Jaime Bering, MD[2], David Chascsa, MD[1].
[1]Mayo Clinic, Phoenix, AZ; [2]Mayo Clinic, Scottsdale, AZ.

Introduction: Esophageal varices (EV) are a complication of portal hypertension. Over time, EV enlarge and may spontaneously rupture, leading to bleeding and possibly death with a high mortality rate of up to 20%. Esophageal variceal ligation (EVL) is used for prophylaxis, as well as treatment of acute variceal bleeding with the goal of eradicating varices. EVL is an effective therapy with rare but serious complications including bleeding, ulcers, strictures, and rarely acute obstruction. Amongst the few reported cases of esophageal obstruction, treatment has varied but mostly involved conservative management or band removal. We present a case of post EVL acute esophageal obstruction that was uniquely treated with band removal and esophageal stenting.
Case Description/Methods: A 79yo female with nonalcoholic steatohepatitis cirrhosis and a MELD-Na of 12, complicated by portal hypertension and EV status post previous banding one year prior came to the emergency room two days after esophagogastroduodenoscopy (EGD) for routine EV surveillance with placement of two bands resulting in complete eradication. She presented with one episode of hematemesis and dysphagia to liquids, and regurgitation; denied odynophagia and chest pain. Hematologic parameters were at baseline. EGD revealed near-complete occlusion of the esophagus from banding at 40cm from the incisors (fig1). One band was dislodged with the gastroscope, which permitted the obstructed area to be traversed after downsizing the gastroscope. No active bleeding was evident. To prevent recurrent bleeding given hematemesis at presentation, the area of obstruction was stented with a 16mm x 100mm fully covered stent (fig2), proximal and distal margins at 30cm and 40cm respectively. She was discharged home after 24 hours able to tolerate soft foods. The stent was kept for seven days. At follow-up EGD for stent removal, there was slight luminal narrowing treated with graded balloon dilation from 18mm to 20mm. She continued to do well and nine months later had EGD surveillance with banding of grade II varices only.
Discussion: Acute esophageal obstruction, though rare, is a complication of variceal banding that should be considered in patients with dysphagia after banding. This is a case that required innovative thinking and priority to treatment options that provide hemostasis while relieving obstruction in a cirrhotic patient with a high risk of bleeding.



[3302] **Figure 1.** 1. Near complete occlusion of esophagus Fig 2. After placement of esophageal stent.

S3303

Acute Intermittent Porphyria

Alexandra Scanameo, MS, MS[1], Shefali Amin, DO, MSEd[2], Andrew E. Lee, MD[2], John F. Altomare, MD[3].
[1]Drexel University College of Medicine, Philadelphia, PA; [2]Reading Tower Health, Reading, PA; [3]Reading Hospital/Tower Health/Digestive Disease Associates, Wyomissing, PA.

Introduction: An 18 year old female with no past medical history presented with an episode of severe abdominal pain, associated with nausea and vomiting. She had intermittent episodes for the past 2 years, and attacks usually occurred 1 week before her menses. She was previously seen by her PCP, who suspected she had celiac disease and was placed on a gluten-free diet without improvement. A few months ago, she was admitted the hospital for similar complaints, and underwent both an upper endoscopy as well as a colonoscopy, both of which were unremarkable. She was discharged from the hospital at that time with a presumptive diagnosis of IBS. On this admission, she was hypertensive. Additional review of systems included intermittent tingling at her fingertips, which she noticed more frequently. Labs were notable for a sodium of 126 mmol/L. Imaging, including a CT of her abdomen/pelvis as well as an MR Enterography, were unremarkable. The young patient continued to have pain despite opiate analgesics, which prompted the search for more rare etiologies of abdominal pain.
Case Description/Methods: Her total porphyrins were 20.1mcg/L, and random urine porphyrins were extremely elevated. Her Porphobilinogen Deaminase (PBGD) level was diminished, and was found to have a PBGD mutation. She was diagnosed with Acute Intermittent Porphyria. She was treated with hemin infusions with mild improvement in her symptoms, and oral glucose loading at onset of symptoms. However, her symptoms eventually came back, and she was initiated on Givosiran.
Discussion: Acute intermittent porphyria is an autosomal dominant condition with low penetrance of a mutation in the porphobilinogen deaminase (PBGD) gene, which leads to an accumulation of heme intermediates. Around 90% of carriers are asymptomatic, and thus, the prevalence is difficult to ascertain. Abdominal pain is often the initial presenting symptom, and is characteristically severe & unremitting. It can be associated with other GI symptoms, including nausea, vomiting & diarrhea. Neurologic symptoms, including peripheral neuropathy and muscle weakness can occur. Psychologic symptoms can also be present, including irritability, anxiety, paranoia & altered consciousness. During an acute AIP attack, hyponatremia may also occur, and may lead to seizures. The primary management of the disease revolves around prevention of attacks, including avoiding implicating medications, hormonal contraception, low-carb diets & alcohol. Pharmacologic therapy, including Givosiran, can also be used.

S3304

Acute Occlusive Mesenteric Ischemia From Venous and Aortic Thrombosis in a Patient with Hyperhomocysteinemia and Liver Cirrhosis

Camille K. Pascasio, MD, Tim Lawrence Lin, MD, Rejine Divinagracia, MD, Ian Homer Cua, MD.
St. Luke's Medical Center Global City, Taguig City, National Capital Region, Philippines.

Introduction: Portal vein thrombosis (PVT) is a severe complication of liver cirrhosis and one of its major consequences include intestinal ischemia. Hyperhomocysteinemia (HHcy) has been suggested to be an independent risk factor for deep venous thrombosis, but limited data are available on the prevalence of HHcy in patients with PVT complicating liver cirrhosis. Several clinical studies have also demonstrated the role of HHcy in arterial and venous thrombosis.
Case Description/Methods: We report a case of a 57 year old male with Liver Cirrhosis from Alcoholic Liver Disease, who presented at our ED with a two week history of dull periumbilical pain which worsened on the day of consult. Plain abdominal CT scan showed nonspecific small bowel enteritis. He was placed on general liquid diet and started on Ciprofloxacin and Metronidazole intravenously. However, on the third hospital day, he complained of sudden severe abdominal pain accompanied by abdominal distention, guarding, and direct and rebound tenderness on all quadrants. He had hypotension and on

laboratory workup, had decreasing hemoglobin levels with elevated WBC count and lactate. Contrast-enhanced abdominal CT scan revealed portal and superior mesenteric vein thrombosis, bowel wall ischemia along the segment of the jejenum in the left hemiabdomen, and infrarenal abdominal aorta and ascending aorta thrombi. Emergency exploratory laparotomy was done with segmental jejunoileal resection and primary end-to-end anastomosis with intraoperative findings of gangrenous small bowels measuring 100 cm in length, with the rest of the proximal small bowels noted to be dilated and edematous. The patient was transferred to the intensive care unit post-operatively for closer monitoring; Heparin drip was started 24 hours post-surgery. Histopathology results showed extensive transmural infarction, hemorrhage, and necrosis on small bowel segments; organizing thrombi were seen on mesenteric vessels. The patient was worked-up for other causes of hypercoagulable states which showed elevated Homocysteine levels at 20.6 (NV: 5-12).

Discussion: It is likely that HHcy may increase the risk of patients with liver cirrhosis to develop arterial and venous thrombosis considering the pivotal role the liver plays in the metabolism of sulphur amino acids and Hcy-related vitamin storage. Therefore, identification of this high-risk group may be important to plan prevention management, such as vitamin supplementation, other Hcy-lowering strategies, or long-term anticoagulation.

S3305

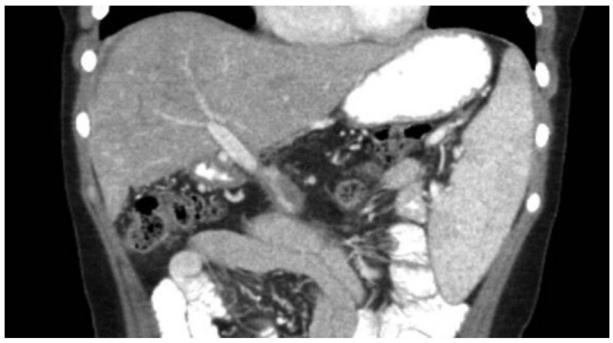
A Unique Case of Extra Hepatic Portal Venous Obstruction Managed by Meso-Rex Shunt

Alex Blachowicz, MD, Kajali Mishra, MD, Jonah Rubin, MD.
Loyola University Medical Center, Maywood, IL.

Introduction: Extra-hepatic portal venous obstruction (EHPVO) can cause non-cirrhotic portal hypertension leading to sequelae including bleeding esophageal varices, portal gastropathy and cavernous transformation of the portal vein. We present a unique case of early-adulthood EHPVO presenting as a variceal bleed managed surgically with Meso-Rex bypass.

Case Description/Methods: An 18-year-old previously healthy female presented with multiple episodes of hematemesis and was found to have bleeding esophageal varices which were treated with band ligation. Her exam was significant for lack of stigmata of chronic liver disease. Labs were significant for platelets of 59,000, INR of 1.9 and a total bilirubin of 1.4. Her work up for acute and chronic liver disease was unremarkable. Cross sectional imaging showed an extrahepatic portal vein thrombosis with extension to superior mesenteric vein (SMV), formation of portal venous collaterals and splenomegaly. Work up for a hypercoagulable disorder was negative. Transjugular liver biopsy was performed with a free hepatic vein pressure of 14 mmHg and a hepatic wedge pressure of 15 mmHg and a portal venous gradient of 1 mmHg consistent with pre-hepatic portal hypertension. Liver pathology was significant for lack of fibrosis. She underwent serial endoscopies with continued band ligation and was started on warfarin due to extension of the thrombus into the SMV to prevent ischemia. For long term management the patient underwent Meso-Rex bypass shunt procedure with a jejunal branch used as inflow. Her coumadin was transitioned to clopidogrel which was stopped after 6 months. She has had no further bleeding episodes. (Figure)

Discussion: Variceal bleed secondary to EHPVO is challenging to diagnose and manage. EHPVO is most commonly seen in children under fourteen years old and rarely presents in adulthood. According to the WHO the prevalence is $<$ 5 per 10,000 population with a majority of cases without an etiology found. There have been reported cases of patients with hypercoagulable states, umbilical catheters or neonatal septic shock. The data behind anticoagulation without a known prothrombotic state is inconclusive, but should be considered if there is concern for development of mesenteric ischemia. Meso-Rex bypass is more definitive management because it is a physiologic repair of the underlying EHPVO with the efficacy and safety of shunting inversely proportional to age. This case highlights a rare case which highlights the need for surgical management.



[3305] **Figure 1.** Coronal Cross-Section of Meso-Rex Shunt.

S3306

Acute Cholestatic Hepatitis Due to Rhinovirus-Induced Hemophagocytic Lymphohistiocytosis: A Case Report

Rosanna Sanchez, BA, Mohammad Abdallah, MBBS, Deena Kapadia, MD.
Rutgers New Jersey Medical School, Clifton, NJ.

Introduction: Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening syndrome in which an overactive immune response causes inflammation and tissue destruction. HLH may occur sporadically with triggers including infections, malignancy or rheumatologic disorders. Diagnosing HLH involves both clinical suspicion, as patients present acutely ill with a range of manifestations such as hepatitis, and diagnostic criteria required to facilitate the diagnosis. We report a case of rhinovirus-induced HLH that presented as acute cholestatic hepatitis.

Case Description/Methods: A 56-year-old male presented to our center with acute liver injury. Two weeks prior to admission, he developed an upper respiratory infection. His condition deteriorated five days prior to admission as he experienced weakness, jaundice, dark urine, and pale stools. Physical examination revealed scleral icterus and jaundice. He was found to have transaminitis with AST/ALT of 850/789 U/L, a total bilirubin of 22.7 mg/dL, a normocytic anemia, elevated INR, elevated ferritin of 36018 ng/dL, and hypertriglyceridemia. Nasal swab was positive for rhinovirus PCR. Cross sectional imaging revealed hepatomegaly and gallbladder wall thickening. His work-up for liver-specific causes was unremarkable. Endoscopic ultrasound showed no clinically significant portal hypertension or cirrhosis, and a liver biopsy revealed cholestatic hepatitis and increased iron deposition. Genetic testing for hemochromatosis was negative. A bone marrow biopsy revealed hemophagocytosis. A soluble IL-2 receptor alpha test was unsuccessful due to laboratory error. Despite only meeting three diagnostic criteria for HLH (hyperferritinemia, hypertriglyceridemia, and hemophagocytosis on the bone marrow biopsy), due to highly suggestive features and worsening clinical status, the patient was started on empiric HLH-directed therapy with steroids and IVIG which resulted in a dramatic improvement in his liver function studies, ferritin down to 6518 ng/dL, and resolution of the coagulopathy. The patient was discharged on steroids and clinically improved with near normalization of transaminases.

Discussion: Diagnosing HLH can be challenging given the range of symptoms and nonspecific diagnostic criteria. However, it is crucial to recognize HLH as a potential cause of acute hepatitis presenting with elevated liver function tests, ferritin, triglycerides, and coagulation abnormalities as delaying treatment while waiting for laboratory and pathology to return can prove to be fatal.

S3307

'Yogi Herbal Tea' Consumption Causing a Cholestatic Drug-induced Liver Injury

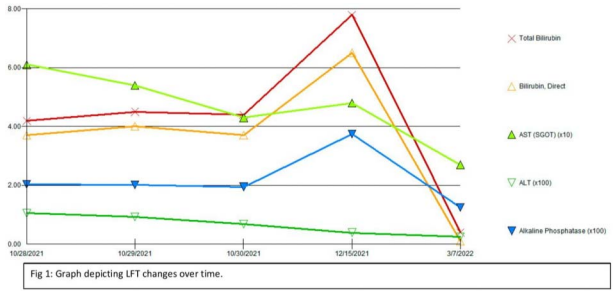
Khushboo V. Bhatia, MBBS, Ruma Rajbhandari, MD, MPH.
Mount Auburn Hospital, Cambridge, MA.

Introduction: We present a case of cholestatic drug-induced liver injury (DILI) with total bilirubin peaking at 24 from the consumption of an herbal tea in a middle-aged woman.

Case Description/Methods: A 55-year-old woman presented with generalized pruritus over 10 days, starting on her palms and spreading all over her body, accompanied by dark brown urine, dyspepsia, early satiety, and poor appetite, causing her to lose 8 pounds. Abdominal pain, hematochezia, melena, diarrhea, nausea, vomiting, fever, rash and, high-risk sexual behaviors were denied. She associated her symptoms with eating raw buckwheat honey and denied use of supplements, medications, tobacco, alcohol, or illicit drugs. Past medical history was notable for obesity, gastric band surgery, thyroid nodules, endometrioma, and menopause. Family history was significant for pancreatic cancer in paternal aunt, and colon cancer in maternal grandparent. Physical exam was within normal limits. Total bilirubin was 4.2 with direct

bilirubin 3.7, ALT 105, AST 61, alkaline phosphatase 203, and GGT 139. CBC, BMP, INR, lipase, Hepatitis A, B, and C, ANA, AMA, ASMA, iron panel, EBV, CMV, CA 19-9, ESR, and CRP were normal. Abdominal ultrasound showed multiple stones with no evidence of intra- or extrahepatic biliary ductal dilatation. Abdominal CT and MRCP confirmed the same. Bilirubin continued to rise peaking at 24.1 roughly 12 days after presentation, alkaline phosphatase increased to 362 while ALT and AST were stable at 47 and 50. It was later revealed the patient had been drinking "Yogi" herbal tea for the last year. Strict cessation of the herbal tea was recommended along with ursodiol for pruritus. Liver biopsy demonstrated panlobular cholestasis with feather hepatocyte changes and bile accumulation in canaliculi, mild periportal and lobular inflammation. At 5-month follow-up, the patient had complete resolution of symptoms and liver chemistries. (Figure)

Discussion: Our patient consumed "Yogi tea", an American-produced tea that consists of multiple herbs, including skullcap root, gardenia fruit, and rhubarb root that have been linked to hepatotoxicity, which can range from mild hepatitis to liver failure necessitating transplantation. We also found a case report of acute fulminant liver failure linked to the same herbal tea brand. Therefore, herbal tea use should be monitored by healthcare providers, and any negative effects reported so that regulatory actions may be taken.



[3307] **Figure 1.** Patient's serial LFTs depicted over time.

S3308

Acute Liver Injury Secondary to Rickettsial Infection

Abigail Braun, MD, Alexander Hayden, MD, Arun Kannappan, MD.
University of Colorado Anschutz Medical Center, Aurora, CO.

Introduction: The differential diagnosis of acute liver injury (ALI) is broad and includes toxins, inflammatory, vascular, and infectious etiologies. Most infectious ALI cases in the US stem from viral hepatitis, though rarer causes exist. In this case, workup of ALI revealed rickettsial-induced acute liver injury, which is a rare presentation of rickettsial disease.

Case Description/Methods: A man with no past medical history presented to the Emergency Department with one week of fevers, myalgias, vomiting, and malaise. He was febrile, tachycardic, hypotensive, and jaundiced. No rash was identified. He lived in Colorado and had recently traveled to Southern California. Laboratory workup revealed platelets of 70,000 , AST 232 U/L, ALT 287 U/L, alkaline phosphatase 216 U/L, and total bilirubin 7.6 mg/dl. His INR was elevated to 1.2. Urine toxicology screens were negative. Abdominal imaging revealed hepatosplenomegaly. He was started on broad spectrum antibiotics due to concern for severe sepsis. Hepatitis serologies, EBV, CMV, VZV, HSV, and HIV were negative. Testing for autoimmune etiologies, Wilson's disease, and alpha-1 antitrypsin were also negative. MRCP had no ductal obstruction and liver biopsy was inconclusive. Additional studies were notable for elevated Rocky Mountain Spotted Fever and typhus antibody titers concerning for rickettsial infection. He was given doxycycline and rapidly improved with normalization of liver enzymes. Repeat convalescent serologies remained elevated for both Rickettsia species and were inconclusive as to which organism was the causative pathogen. CDC confirmatory testing was sent but the sample was unable to be processed due to the amount of time between test collection and arrival at the laboratory.

Discussion: Rickettsial diseases classically cause rash, headache, fever, thrombocytopenia, and hyponatremia. While mild transaminase elevations accompany infection, there are few cases of rickettsial species causing acute liver injury. This case highlights the need for clinical suspicion of rickettsial pathogens causing severe liver dysfunction when standard evaluation is negative, even in instances where rash and travel exposure are absent. When rickettsial infection is suspected, antibiotics are often begun while awaiting laboratory testing. However, serologic testing for rickettsial pathogens is often inconclusive and timely PCR based evaluation through the CDC is required for a definitive diagnosis.

S3309

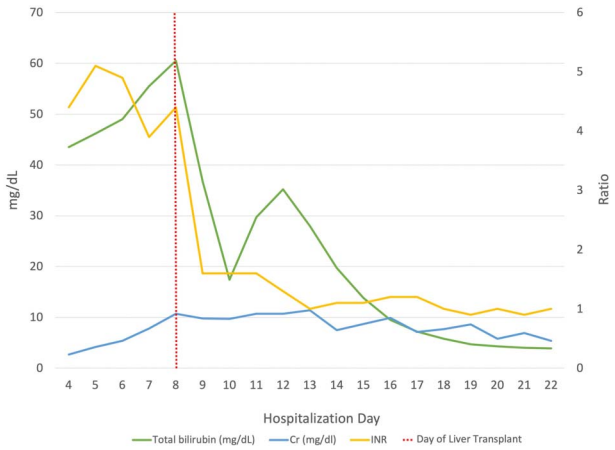
Acute on Chronic Liver Failure From Wilson's Disease After Initial Therapy with Ammonium Tetrathiomolybdate Followed by Zinc Monotherapy for 35 Years: A Case Report

Shuji Mitsuhashi, MD[1], Ritu Nahar, MD[1], Akash Patel, DO[2], Katherine Keck, BA[3], Nicholas Zirn, MD[2], Dina Halegoua-DeMarzio, MD[1].
[1]Thomas Jefferson University Hospital, Philadelphia, PA; [2]Thomas Jefferson University, Philadelphia, PA.

Introduction: Wilson's disease (WD) is an inherited disease of toxic accumulation of Cu mainly affecting the brain and liver. We describe a case of acute on chronic liver failure caused by WD despite being stable on Zn maintenance therapy for 35 years after initial treatment with ammonium (NH4) tetrathiomolybdate.

Case Description/Methods: A 58-year-old Caucasian male with WD presented to a local hospital with acute onset of jaundice and dark urine. He is a minimal alcohol drinker. He had no exposures to hepatotoxicity and no prior evidence of liver fibrosis or decompensation. He was diagnosed WD at age 18 with neurologic symptoms and was treated in a clinical trial with NH4 tetrathiomolybdate at the time of diagnosis followed by Zn monotherapy. His Zn dosage was monitored by his PCP based on its level. On admission, his vitals were stable. Exam revealed jaundice, scleral icterus, and abdominal distension. No sign of encephalopathy or asterixis. Labs were notable for INR 2.4, ALP/AST/ALT 86/148/47 IU/L, Tbili 12.6 mg/dL, Cr 0.9 mg/dL, elevated Cu, and normal ceruloplasmin. CT noted cirrhotic liver morphology. On Day 4, he was transferred to our hospital for an urgent liver transplant (LT) evaluation. On arrival, his condition rapidly progressed with active hemolysis and elevated Tbili (peaked at 60.1) and Cr (Figure 1). Other precipitating causes including viral and autoimmune hepatitis were ruled out. On Day 5, he was sent to ICU for dialysis and was listed for transplant as Status 1A. On Day 8, he successfully underwent deceased donor LT. Liver explant showed cholestatic hepatitis on chronic hepatitis with cirrhosis consistent with WD. On Day 22, he was discharged home.

Discussion: NH4 tetrathiomolybdate is proposed to be a more efficacious treatment option for neurologic predominant WD but it is not yet commercially available. Zn monotherapy is shown to be comparable to penicillamine in preventing neurologic and hepatic decompensation by inhibiting Cu uptake by intestinal mucosa. Close monitoring is paramount for patients on Zn due to risk of treatment failure. AASLD and EASL recommend monitoring liver function test, serum Cu, ceruloplasmin and physical exam twice yearly, and urine Cu yearly. However, parameters for treatment failure of Zn need to be clearly defined to consider alternative treatment before disease progression occurs. LT is the only effective option for WD patients with decompensated liver disease unresponsive to medical therapy. One-year survival following LT ranges from 79-87%.



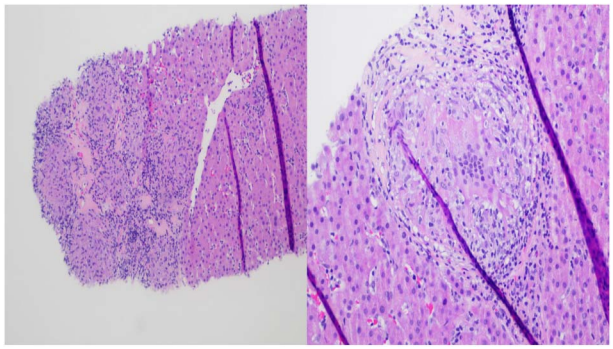
[3309] **Figure 1.** Trend of patient's total bilirubin, creatinine, and INR during hospitalization.

S3310

Abdominal Sarcoidosis Including Hepatic and Portal Lymphadenopathy as a Primary Manifestation in a Case of Systemic Sarcoidosis

Saatchi Kuwelker, MD, Farah H. Ladak, MD, Eugenia Tsai, MD.
UT Health San Antonio, San Antonio, TX.

Introduction: Sarcoidosis is a rare systemic inflammatory disease characterized by non-caseating granulomas. Most patients are asymptomatic but hepatic sarcoid can progress to portal hypertension which can be cirrhotic or non-cirrhotic. Definitive diagnosis is made by liver biopsy. This is a case of hepatic sarcoidosis presenting with elevation in alkaline phosphatase (ALP) which improved with treatment.
Case Description/Methods: A 50 year-old man presented with three months of worsening dyspnea on exertion and dysphagia to liquids and solids. On arrival, the patient was tachycardic, tachypneic and hypoxemic. Laboratory values revealed an elevated ALP, alanine transaminase (ALT) and aspartate transaminase (AST) of 458 U/L, 113 U/L, 110 U/L, respectively. Computed tomography of the chest showed a small apical pneumothorax and reticular opacities with bronchiectasis. Abdominal ultrasound showed hepatic steatosis. Magnetic resonance cholangiopancreatography of the abdomen revealed enlarged portal lymph nodes. Work-up for fungal, autoimmune and infectious etiologies was negative except for an anti-mitochondrial antibody of 29 units and anti-smooth muscle antibody of 30 units. Liver biopsy revealed granulomatous portal and lobular inflammation, portal lymphocytic inflammation with ductitis, focal interface hepatitis and focal bridging fibrosis (Figure 1). Lung biopsy showed non-necrotizing granulomas that initially was thought to be due to atypical mycobacteria and not sarcoidosis. Therefore, the patient was not immediately started on steroids. Ursodiol was initiated to help with cholestasis. Biopsy of portal lymph nodes confirmed non-caseating granulomas. The patient was then diagnosed with systemic sarcoidosis with hepatic and pulmonary involvement. He was started on steroids in the outpatient setting in conjunction with ursodiol. The AST, ALT, and ALP improved to 50 U/L, 70 U/L and 161 U/L, respectively two months after initiation of therapy.
Discussion: Sarcoidosis should be among one of the differentials in patients presenting with predominant ALP elevation in the absence of bilirubin elevation and concomitant lung manifestations. A liver biopsy is essential for diagnosing hepatic sarcoidosis and to rule out other similar pathologies like primary biliary cholangitis and primary sclerosing cholangitis. Ursodiol in conjunction with steroids can help improve liver enzyme elevations seen in hepatic sarcoidosis.



[3310] **Figure 1.** 1a. Shows a large lobular focus of non-caseating granulomatous inflammation with interdigitating hyaline fibrosis in 200x magnification. The right side of the image shows hepatic parenchyma without steatosis and a small focus of lymphocytic inflammation. Figure 1b. Shows a higher power image of a portal tract expanded by a poorly formed granuloma with a multinucleated giant cell in 400x magnification. The remaining portal tract shows a lymphohistiocytic infiltrate with infiltration and damage of the bile duct by lymphocytes.

S3311

Acute Liver Injury as Presenting Feature of Hemophagocytic Lymphohistiocytosis in a Patient With Post-COVID 19 and EBV Infection

Afshin A. Khan, MD, MPH, Kaitlin Payne, MD, Talal Adhami, MD.
Cleveland Clinic Foundation, Cleveland, OH.

Introduction: Viral infections such as Epstein Barr Virus (EBV) have been associated with secondary hemophagocytic lymphohistiocytosis (HLH) with some recent studies alluding to an association between severe COVID-19 and HLH. Herein, we report a case of a patient with an earlier mild case of COVID, followed by EBV infection, that precipitated HLH.
Case Description/Methods: A 36-year-old man with a history of mild COVID-19 infection a month prior presented to an outside hospital with nausea, fatigue, and jaundice. Patient had total bilirubin (tibili) 7.1, ALT 2714, AST 1273 and an elevated EBV viral capsid antigen. Liver US was negative. CT abdomen showed significant portal hepatitis and peri-pancreatic head lymphadenopathy. Lymph node biopsy was unrevealing. Patient was diagnosed with mononucleosis and discharged. Patient presented to multiple institutions and discharged with mononucleosis associated liver injury. With worsening jaundice, patient

ultimately presented to Cleveland Clinic with tbili 23.8, conjugated bilirubin 19.8, ALT 1400, AST 1145, Alkaline phosphatase 461, with repeat EBV titers negative. CT revealed upper abdominal lymphadenopathy in peri-portal and periceliac regions. MRCP confirmed lymphadenopathy, borderline splenomegaly with no biliary dilation. Acute liver injury workup was negative. Liver biopsy showed marked lobular hepatitis, areas of dropout with hemorrhage and increased cellularity within the sinusoids and evidence of hemophagocytosis. The portal areas were expanded by inflammatory cell infiltrates including plasma cells, lymphocytes, histiocytes, and granulocytes. Patient was diagnosed with HLH, using the criteria in Table 1. H-score was 184 points with 70-80% probability of HLH. Patient was discharged with close follow up with hepatology and hematology for treatment initiation. (Figure)

Discussion: HLH carries a grim prognosis and requires earlier detection and treatment. Acute phase COVID-19 has been associated with HLH, though post-COVID 19 cases associated with HLH are rare. It is thought that post-COVID 19 causes immune dysregulation and activation of macrophages which can cause HLH. Though asymptomatic, our patient subsequently tested positive for EBV, which was cleared by the time repeat testing was done. This raises the question for concurrent or subsequent infections as precipitating factors for HLH. Our case report highlights the importance of exploring HLH in patient where no obvious signs of acute liver injury are present, particularly in patients with COVID-19 infection.

Table 1. Comparison of HLH- 2004 diagnostic guidelines an H-score in our patient

HLH-2004 Criteria*		H-Score Criteria	
Fever	**Present**	**Temperature, °F (°C)**	<101.1 (<38.4) **101.1–102.9 (38.4-39.4) (+33)** >102.9 (>39.4) (+49)
Splenomegaly	**Present**	**Organomegaly**	No **Hepatomegaly or splenomegaly (+33)** Hepatomegaly and splenomegaly (+38)
Cytopenias (affecting ≥2 of 3 lineages in the peripheral blood) • **Hemoglobin <90 g/L** • **Platelets <100 x 10^9/L** • **Neutrophils <1.0 x 10^9/L**	Absent	**Number of cytopenias Defined as hemoglobin ≤9.2 g/dL (≤5.71 mmol/L) and/or WBC ≤5,000/mm^3 and/or platelets ≤110,000/mm^3**	**1 lineage** 2 lineages (+24) 3 lineages (+34)
Hypertriglyceridemia and/or hypofibrinogenemia: • **Fasting triglycerides ≥ 3.0 mmol/L (i.e. ≥265 mg/dl)** • **Fibrinogen ≤1.5 g/L**	Absent (triglycerides 217 mg/dl; fibrinogen 2.27 g/L)	**Triglyceride, mg/dL (mmol/L)**	<132.7 (<1.5) **132.7-354 (1.5–4) (+44)** >354 (>4) (+64)
		Fibrinogen, mg/dL (g/L)	>250 (>2.5) **≤250 (≤2.5) (+30)**
Hemophagocytosis in bone marrow/spleen/lymph nodes	**Present in liver biopsy**	**Hemophagocytosis features on bone marrow aspirate**	**No** Yes (+35)
Ferritin ≥ 500 µg/L	**Present (5,511.0)**	**Ferritin, ng/mL (or µg/L)**	<2,000 **2,000–6,000 (+35)** >6,000 (+50)
Low or absent NK-cell activity	Absent (low normal)	**AST, U/L**	<30 **≥30 (+19)**
Soluble CD25 (i.e., soluble IL-2 receptor) ≥ 2,400 U/ml	**Present (5803.6)**	**Known underlying immunosuppression**	**No** Yes (+18)

* The diagnosis of HLH can be established if one of either 1 or 2 below is fulfilled 1) A molecular diagnosis consistent with HLH, 2) Diagnostic criteria for HLH fulfilled (5 out of 8 criteria below)

[3311] **Figure 1.** Comparison of HLH- 2004 diagnostic guidelines an H-score in our patient.

Table 1. Comparison of HLH- 2004 diagnostic guidelines an H-score in our patient

HLH-2004 Criteria*		H-Score Criteria	
Fever	Present	Temperature, °F (°C)	< 101.1 (< 38.4) 101.1–102.9 (38.4-39.4) (+33) >102.9 (>39.4) (+49)
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Cytopenias (affecting ≥2 of 3 lineages in the peripheral blood) < ![if !supportLists] >• < ![endif] >Hemoglobin < 90 g/L < ![if !supportLists] >• < ![endif] >Platelets < 100 x 10^9/L < ![if !supportLists] >• < ![endif] >Neutrophils < 1.0 x 10^9/L	Absent	Number of cytopenias Defined as hemoglobin ≤9.2 g/dL (≤5.71 mmol/L) and/or WBC ≤5,000/mm^3 and/or platelets ≤110,000/mm^3	1 lineage 2 lineages (+24) 3 lineages (+34)
Hypertriglyceridemia and/or hypofibrinogenemia: < ![if !supportLists] >• < ![endif] >Fasting triglycerides ≥ 3.0 mmol/L (i.e. ≥265 mg/dl)	Absent (triglycerides 217 mg/dl; fibrinogen 2.27 g/L)	Triglyceride, mg/dL (mmol/L)	< 132.7 (< 1.5) 132.7-354 (1.5–4) (+44) >354 (>4) (+64)
< ![if !supportLists] >• < ![endif] >Fibrinogen ≤1.5 g/L		Fibrinogen, mg/dL (g/L)	>250 (>2.5) ≤250 (≤2.5) (+30)
Hemophagocytosis in bone marrow/spleen/lymph nodes	Present in liver biopsy	Hemophagocytosis features on bone marrow aspirate	No Yes (+35)
Ferritin ≥ 500 µg/L	Present (5,511.0)	Ferritin, ng/mL (or µg/L)	< 2,000 2,000–6,000 (+35) >6,000 (+50)
Low or absent NK-cell activity	Absent (low normal)	AST, U/L	< 30 ≥30 (+19)
Soluble CD25 (i.e., soluble IL-2 receptor) ≥ 2,400 U/ml	Present (5803.6)	Known underlying immunosuppression	No Yes (+18)

*The diagnosis of HLH can be established if one of either 1 or 2 below is fulfilled (1) A molecular diagnosis consistent with HLH, (2) Diagnostic criteria for HLH fulfilled (5 out of 8 criteria below).

S3312

Acute Decompensation of Late-Onset Glutaric Acidemia Type II in the Setting of Multifactorial Cirrhosis

Tamara Koritarov, DO, MSc[1], Homaira Rasool, DO[2].
[1]Brown University, Providence, RI; [2]Kent Hospital/Brown University, Warwick, RI.

Introduction: Glutaric acidemia type 2 (GA2), or multiple acyl CoA dehydrogenase deficiency (MADD), is a disorder of fatty acid oxidation, amino acid and choline metabolism. GA2 is inherited in an autosomal recessive manner by changes in the ETFA, ETFB, or ETFDH genes. The classic and most severe form of GA2 is neonatal onset. Our patient has symptoms of late onset disease, including fatigue, vomiting, episodic hyperammonemia, fatty infiltration of the liver, and an absence of congenital malformations. In acute decompensations, triggered by catabolism and/or intercurrent illness, individuals with GA2 may develop metabolic acidosis, rhabdomyolysis, elevation of transaminases, and hyperammonemia.
Case Description/Methods: This case describes a 36-year-old gentleman with late onset glutaric acidemia type II, multifactorial cirrhosis from non-alcoholic steatohepatitis in the setting of a metabolic disorder, class III obesity, alcoholic associated liver disease, portal hypertension, splenic embolization, CHF, presented to the ED for confusion following progressive nausea and vomiting over five days, and was found to have elevated ammonia level at 165 mcmol/L. His vital signs were tachycardia of 133 bpm, hypertension of 221/133, he was afebrile in no respiratory distress. Physical exam was notable for a drowsy obese male oriented to self and place, diaphoretic, tachycardic, no JVD, no crackles, non-tender abdomen with normoactive bowel sounds, and asterixis. Initial lab studies were significant for platelets of 131k, potassium of 2.4 mmol/L, total bilirubin 3.7 mg/dL, AST 355 ALT 130 ALP 143 IU/L, and no detectable ethanol, acetaminophen or salicylates. Multiple factors can influence his ammonia level, including underlying cirrhosis and glutaric acidemia. His metabolic crisis was suspected due to a catabolic state from intake of high-protein diet, and was managed with 10% Dextrose IV fluids, levocarnitine, riboflavin, lactulose, low-protein low-fat diet with improvement in encephalopathy. Hypokalemia from decreased absorption and lactulose use resolved with potassium supplements and addition of spironolactone.
Discussion: Metabolic disorders are rare but important to consider in patients that present with metabolic crisis. They can decompensate after periods of dehydration, exercise, alcohol ingestion or illnesses and develop hyperammonemia, transaminitis, metabolic acidosis. These individuals have a lifelong risk of intermittent episodes of metabolic crisis and prompt management is imperative to recovery.

S3313

Acute Sickle Intrahepatic Cholestasis From Sickle Cell SC Disease Causing Acute on Chronic Liver Failure

Vivek Lingiah, MD, Mumtaz Niazi, MD.
Rutgers New Jersey Medical School, Newark, NJ.

Introduction: Acute on Chronic Liver Failure (ACLF) is a disorder that results in grave systemic consequences. The dysregulated inflammatory response caused by an acute liver injury often results in multi-organ dysfunction. And while alcoholic/viral hepatitis or infections are common causes of ACLF, hematologic disorders are unusual. We present a case of ACLF due to sickle cell intrahepatic cholestasis from SC disease.
Case Description/Methods: A 61-year-old man with history of HTN, sickle-cell trait, alcoholic cirrhosis presented for altered mental status. Initial labs showed TBili 30.7, DBili >20.0, AST/ALT 308/123, ALP 532 and INR 1.6. Phosphatidylethanol level negative. No new history of medication/herbal use, new sexual contacts, or drug use. Workup negative for hepatitis A/B/C/E. ANA negative, ASMA 26.7, IgG 2742. MRI Abdomen showed no liver mass with patent hepatic vasculature/biliary tree. LDH 531, Haptoglobin < 10, reticulocyte count 11.2, and Coombs test was positive. Hb electrophoresis showed 44.4% Hb C and 49.5% Hb S, consistent with SC disease. Hb was 8.5 (baseline), with no known crises since youth. Liver biopsy findings were consistent with sickle cell hepatopathy (sinusoidal dilation and congestion of sickled cells, ballooning degeneration of hepatocytes, and marked intracanalicular cholestasis). Patient had exchange blood transfusion (EBT) with improvement in Hb to 11.8 and decrease in retic count, LDH. Patient however continued to deteriorate, with increasing aminotransferases into thousands, worsening mental status, AKI, and coagulopathy. Patient made hospice care and eventually expired.
Discussion: Hemoglobin SC (HbSC) disease is a less common form of sickle cell disease (SCD). Patients with HbSC disease have milder symptoms than SCD but more severe than sickle cell trait. Acute sickle hepatic crisis occurs in 10% of patients with SCD. Acute sickle intrahepatic cholestasis is the most severe form of sickle hepatopathy, associated with increased mortality. It is usually seen in SCD, but rarely in HbSC. Clinical presentation is more severe, with significant hyperbilirubinemia >15 mg/dL and aminotransferase elevations >1000 IU/L, reflecting ischemic injury. ALF/ACLF can evolve rapidly with multi-organ failure. EBT is considered in these situations. Non-responders have poor prognosis. Clinicians should be aware of the liver complications that can arise in patients with sickle cell-related disorders, including HbSC, particularly in patients with chronic liver disease.

S3314

Acute Epstein-Barr Virus Infection-Induced Hemophagocytic Lymphohistiocytosis as a Rare Cause of Acute Liver Failure

Andreas W. Bub, MD[1], Jonathan Kandiah, MD[2], Sherose Chaudry, MD[1], Henry Beecher, MS, MD[3].
[1]Stamford Hospital, Stamford, CT; [2]Norwalk Hospital, Norwalk, CT; [3]Gastroenterology/Hepatology Associates, LLC, Stamford, CT.

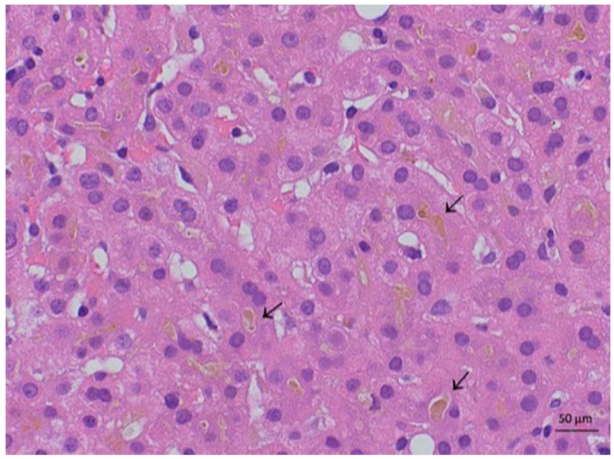
Introduction: Hemophagocytic lymphohistiocytosis (HLH) is caused by an unregulated immunoinflammatory response leading to acute organ failure and even death. It has multiple etiologies such as genetics, autoimmune disorders, malignancy, and viral infections like Epstein-Barr Virus (EBV). Findings include fever, cytopenia in two cell lines, hepatosplenomegaly, hypertriglyceridemia, hemophagocytosis in bone marrow or liver biopsy, low natural killer T-cell (NK) activity, hyperferritinemia, and elevated soluble IL-2 receptor (sIL2r). Diagnosis is made when 5 of the above criteria are met. Identification is key as survival is low even if treated. We present a patient who developed acute liver failure (ALF) from EBV induced HLH.
Case Description/Methods: A 57-year old woman with selective IgA-deficiency (s-IgA-d) presented with malaise, jaundice, and right upper quadrant tenderness. Blood-work revealed leukopenia, thrombocytopenia, cholestasis, and elevated C- reactive protein. Abdominal ultrasound showed a dilated common bile duct without cholelithiasis or choledocholithiasis and MRCP showed hepatosplenomegaly and numerous hepatic cysts. Viral serologies were checked and supportive treatment was started. She reported improvement in symptoms and was discharged but returned 4 days later with fever, vomiting, and encephalopathy. Blood-work revealed anemia, elevated inflammatory markers, decreased synthetic liver function, and acute EBV infection. A diagnosis of ALF secondary to EBV was made. HLH was considered so sIL2r was checked and corticosteroids were started. Levels of sIL2r ultimately came back elevated, confirming the diagnosis. The patient improved and was discharged without complications.
Discussion: HLH is a devastating condition with multiple etiologies and our patient's s-IgA-d may be relevant. IgA levels were significantly lower in pediatric patients who developed HLH from EBV compared to those who developed Infectious Mononucleosis. The function of serum IgA is not fully known but it inhibits macrophages through an IgA-specific receptor. Lack of inhibition and abnormal signaling from atypical lymphocytes produced by EBV offers a potential mechanism. S-IgA-d is the most common immunodeficiency with a prevalence of 1 in 500. Most are asymptomatic though there is a higher prevalence of autoimmune and allergic disorders in these patients. More research is required as HLH remains a complex syndrome that must be identified before catastrophic tissue destruction occurs.

S3315

Acute Cholestatic Hepatitis Following m-RNABNT162b2 SARS-CoV-2 Vaccination: Could It Be Genetic?

Dana Toy, MD, Melanie A. Hundt, MD, Andrew Stolz, MD.
University of Southern California Keck School of Medicine, Los Angeles, CA.

Introduction: Given the morbidity and mortality of COVID-19, development of vaccines targeting SARs-CoV-2 was essential. Despite their overall safety and efficacy, atypical complications have been reported. We report a case of post-vaccination acute cholestatic hepatitis, consistent with drug-induced liver injury (DILI).
Case Description/Methods: A 61-year-old man with no past medical history presented with 2 weeks of jaundice and pruritus, 1 month after his second dose of the m-RNABNT162b2 SARS-CoV-2 vaccine. He denied use of any medications, supplements, tobacco, alcohol, drugs, or family history of liver disease. Vital signs and physical exam were unremarkable. Admission labs included ALP 265 U/L, AST 50 U/L, ALT 68 U/L, total bilirubin 14.9 mg/dL, and direct bilirubin 12 mg/dL. Ultrasound revealed normal liver surface without ductal dilatation, confirmed by MRCP. Serologies (HIV, HAV IgM, HBV core IgM, HBV surface antigen, HCV antibody), autoimmune markers (ANA, anti-mitochondrial, anti-actin, anti-liver-kidney-microsomal), and respiratory viral panel (SARS-CoV-2, influenza A/B, RSV) were negative. Hemoglobin, platelets, INR, albumin, gamma-glutamyl transpeptidase (GGT), and immunoglobulins were normal. Percutaneous liver biopsy revealed cholestasis with mild portal lymphocytic infiltrates and lobular inflammation (Figure 1), without portal fibrosis, PAS+ globules, or iron deposition. Molecular genetic testing (Next Generation Sequencing, Prevention Genetics® 77 Gene Cholestasis Panel) was performed. No genetic variants were identified in 77 genes associated with cholestasis, though single copy variants of several autosomal recessive genes were detected. Symptoms resolved with supportive care. Labs one month later were notable for ALP 175 U/L, (AST hemolyzed), ALT 68 U/L, and total bilirubin 3.2 mg/dL.
Discussion: Prior reports have described cholestasis due to immune-mediated hepatitis after COVID mRNA vaccination[1], but these features were absent in this case. Given the patient's normal GGT, we hypothesized that increased cytokine release led to cholestatic injury in an individual genetically predisposed to cholestasis by genetic variation in ATP8B1 or ABCB11. Future studies comparing genetic variants in patients with liver injury may provide insight into mechanisms of idiosyncratic DILI following mRNA vaccination against SARS-CoV-2.



[3315] **Figure 1.** Liver biopsy showing cholestasis in a patient who developed jaundice after COVID mRNA vaccination.

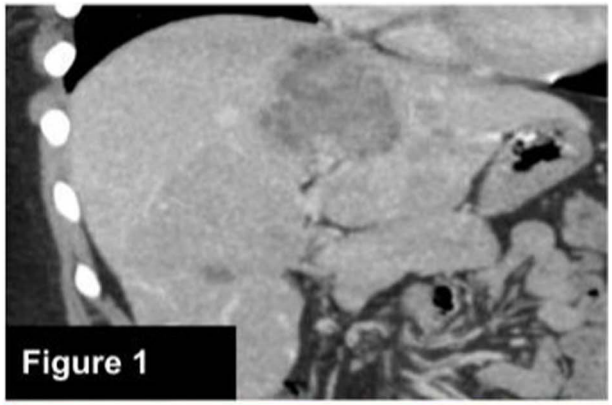
REFERENCE
1. Bril F et al. Autoimmune hepatitis developing after coronavirus disease 2019 (COVID-19) vaccine: Casuality or casualty? J Hepatol. 2021 Jul; 75 (1): 222-224

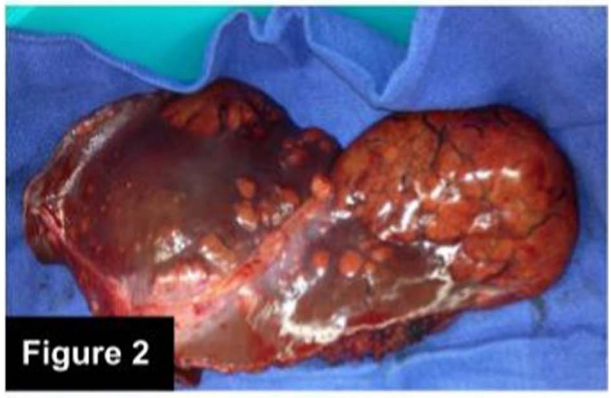
S3316

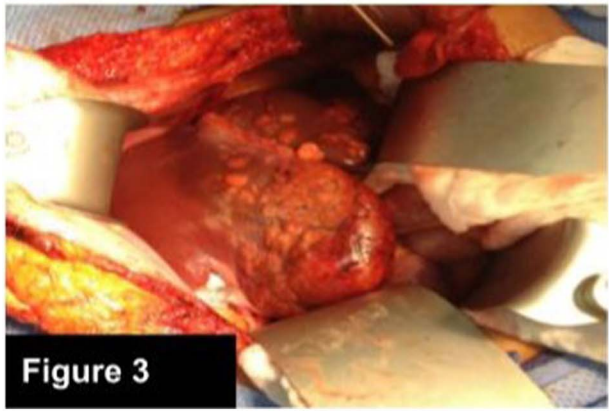
Aden-oh-my!-tosis: A Case of Severe Symptomatic Hepatocellular Adenomatosis

Miranda Anderson, DO, Allison Bush, MD, Mark Damiano, MD, John Lee, MD.
Walter Reed National Military Medical Center, Bethesda, MD.

Introduction: Hepatocellular adenomas (HCA) are benign solid liver lesions often found incidentally on imaging. HCA arise in normal livers, often in women aged 40-50. Large HCA carry risk of malignant transformation and spontaneous rupture. Hepatocellular adenomatosis is a rare disorder of 10+ HCA. We present a case of severe and symptomatic hepatocellular adenomatosis successfully treated with extensive hepatectomy.
Case Description/Methods: A 39-year-old woman presented to the emergency department multiple times with severe, intermittent right upper abdominal pain that radiated to her back. Lab tests revealed Alkaline Phosphatase 114 (H) U/L, Alanine Aminotransferase 37, Aspartate Aminotransferase 44 U/L. Liver ultrasound demonstrated a lobular liver contour with diffuse heterogeneous echotexture. CT showed multiple liver masses (Figure 1). Subsequent MRI demonstrated numerous hyperintense lesions, the largest was 4.9 x 8 x 5.8 cm exophytic lesion. AFP, CA 19-9, colonoscopy, pelvic ultrasound were normal. Pathology demonstrated a HCA. The patient underwent left hepatic and caudate lobe resection and intraoperative radiofrequency ablation of her right lobe lesion (Figures 2 and 3). There were multiple smaller HCA near the portal vessels that were untreated as they were thought to be low risk. Her symptoms and liver enzymes improved.
Discussion: Hepatocellular adenomatosis is a rare liver disorder that is often asymptomatic and incidentally found but carries risk of malignant transformation and bleeding. There are four subtypes of HCA: hepatocyte-nuclear-factor-1 alpha mutated, beta-catenin-mutated type (higher risk), inflammatory type, and unclassified type. The size and number of lesions is associated with morbidity and warrants consideration for early intervention. Resection is recommended for all HCA over 5cm or symptomatic lesions. For other HCA and patients with hepatocellular adenomatosis, biopsy may be considered to determine the subtype to aid in future prognostication. This case highlights a rare case of symptomatic, severe hepatocellular adenomatosis successfully treated with extensive hepatectomy.







[3316] **Figure 1.** CT Liver with HCA. Figure 2: gross liver specimen post hepatectomy. Figure 3: intraoperative view of liver with visible adenomatosis.

S3317

Acute Hepatitis C Infection Accounts for Suspected Isoniazid Hepatitis in a Patient With Controlled HIV Infection on Antiretroviral Therapy

Paloma Khamly, DO[1], Devika Gandhi, MD[2], Cindy X. Cai, MD, PhD[3], Shobha Castelino-Prabhu, MD[3], Chitra Damodaran, MD[3].
[1]Loma Linda University, Redlands, CA; [2]Loma Linda University, Loma Linda, CA; [3]VA Loma Linda Healthcare System, Loma Linda University School of Medicine, University of California Riverside School of Medicine, Loma Linda, CA.

Introduction: Acute hepatitis C (HCV) infection is rising in intravenous drug users/non-monogamy. Isoniazid (INH), commonly used for tuberculosis, may cause idiosyncratic drug-induced liver injury (DILI). Acute HCV infection was reported in 1.5% of suspected DILI. We here report a case of acute HCV infection for suspicious INH hepatitis in a patient with controlled human immunodeficiency virus (HIV) infection on antiretroviral therapy (ART).
Case Description/Methods: A 54 years-old Caucasian male was admitted for several days of fatigue/nausea/abdominal pain with elevated ALT/AST at 179/93. He has a history of controlled HIV infection on dolutegravir/tenofovir/emtricitabine, non-monogamy (men having sex with men), marijuana/herbal use, and latent tuberculosis (with a normal liver test/negative HCV antibody) treated with INH for 3 months. Serology for acute hepatitis A, B, C infection, other work-ups, and abdominal ultrasound/CT were negative. INH rather than ART/herbals was suspected as the possible culprit for hepatotoxicity and was continued due to ALT $<$ 5 times upper normal limit. Patient was discharged after a negative esophagogastroduodenoscopy and symptomatic improvement. He presented again 2 days later with ALT/AST of 287/153 then to 984/388 on the 9th day after INH cessation. Total bilirubin and INR had remained normal with minimally elevated ALP. HCV RNA was detected at 16.5 million IU/ml. A liver biopsy was performed for rising liver enzymes and excluding autoimmune hepatitis. Moderate portal/lobular inflammation with abundant hepatocyte apoptosis and mild intra-canalicular cholestasis without autoimmune hepatitis was reported, suggesting possible DILI with viral hepatitis per pathologist. However, with a strong competing diagnosis of acute HCV hepatitis, persistently rising ALT/AST despite cessation of INH, and only possible causality score for INH-DILI, the suspected INH hepatitis was adjudicated as acute HCV infection. Sofosbuvir-velpatasvir treatment was initiated with rapid normalization of ALT/AST and decreasing HCV RNA.
Discussion: This is the first reported case of acute HCV infection for suspected INH hepatitis in a patient with controlled HIV infection on ART. The case emphasizes the importance of early HCV RNA testing for diagnosing/excluding acute HCV infection in suspicious DILI due to delayed seroconversion of HCV antibody. Prompt treatment with highly potent antiviral agents leads to eradication of HCV with potential public health benefit.

S3318

Acute Hepatitis Secondary to Syphilis

Matthew Kubina, BS[1], Mohamad Mouchli, MD[2], Rami Musallam, MD[3], Dalbir Sandhu, MD[4].
[1]Northeast Ohio Medical University, Rootstown, OH; [2]Cleveland Clinic Akron General, Akron, OH; [3]St. Vincent Charity Medical Center, Cleveland, OH; [4]Cleveland Clinic Foundation, Cleveland, OH.

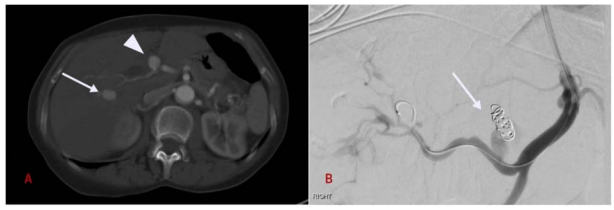
Introduction: Hepatitis is acute inflammation of the liver, and it is most often caused by viral infections (Hepatitis A, B, C), as well as by alcohol, toxins, and other causes. This can be acute or chronic in nature, and will often present with symptoms including fatigue, nausea and vomiting, and jaundice. Elevated liver enzymes will aid in diagnosis. However, when some of the most common causes have been ruled out, additional evaluation is performed into less common causes. We present a case of acute hepatitis secondary to syphilis.
Case Description/Methods: The patient is a 38-year-old male who presented with increasing fatigue and abnormal liver enzymes. He also noted lower extremity swelling as well as the presence of a new rash all over his body. He denied any nausea, vomiting, fevers, abdominal pain, constipation or diarrhea. He denied any recent medication changes, and denies smoking, alcohol use, and illicit drug use. Family history was unremarkable. There was no history of blood transfusion. The patient reports being sexually active with multiple female partners in the past. A CMP was significant for an ALP of 451, AST 155, and ALT 240. Bilirubin was normal. A CBC showed a hemoglobin of 11.7 with an MCV of 76.8. His most recent CBC and LFTs from four months prior were normal. Of note, Hep B/C, HIV, HSV, GC, and Chlamydia all recently tested negative. Serologic studies for ANA, ASMA, ceruloplasmin, alpha-1 antitrypsin, and iron panel all were normal. The patient states that he tested positive for syphilis during plasma donation. A red, macular rash was present on the palms of his hands, soles, and lower legs. Treatment with IM Penicillin G was initiated. Following treatment, his LFTs were trended, which revealed ALP of 438, AST 54, and ALT 114. The patient was advised to follow-up with his PCP to assess for symptom resolution.
Discussion: Syphilis is an infection that can present with a variety of different symptoms. Though syphilis and hepatitis has been rarely associated in the past, it is a rare cause, and delayed diagnosis can result in significant patient morbidity. Many patients will note a characteristic painless, genital lesion. However, this can go unnoticed or underreported by the patient as it self-resolves. While it is incredibly sensitive to penicillin, prompt diagnosis can be challenging, and late stages of syphilis can result in irreversible complications.

S3319

An Unusual Presentation of Segmental Artery Mediolysis

Rahul Karna, MD, Dylan Kaye, BA, Balaji Jagdish, DO, Alexandra Johnston, DO, Divya Venkat, MD.
Allegheny Health Network, Pittsburgh, PA.

Introduction: Segmental Arterial Mediolysis (SAM) is a rare non-atherosclerotic and non-inflammatory disease that often mimics vasculitis. It most commonly affects celiac artery and superior mesenteric artery. Here we describe an unusual presentation of SAM in an elderly woman.
Case Description/Methods: A 65-year-old female with history of smoking presented with sudden onset dull epigastric pain, radiating to the back, nausea, vomiting and decreased oral intake for three days. Review of system was negative for other symptoms. Vital signs upon presentation were blood pressure 140/87 mm Hg, heart rate 92 beats per minute, respiratory rate 16 per minute and saturating 96% on room air. Physical examination revealed epigastric tenderness without rebound tenderness or guarding. Initial labs showed WBC 15,200/mL, CRP 9.4 mg/L and ESR 92 mm/hour. LFTs revealed AST 2173 U/L, ALT 2013 U/L, total bilirubin 0.6 mg/dl, albumin 3.7 and INR 1.1 Further labs ruled out infectious etiology for her presentation. Rheumatologic work up was negative for ANA, ANCA or abnormalities in complement proteins. Computed tomographic angiography (CTA) of the abdomen demonstrated multiple large aneurysms involving bilateral hepatic arteries with diffuse irregularity and ectasia of the hepatic arterial system and blood products surrounding the left hepatic artery aneurysm. (Figure A) She subsequently underwent coil embolization of left hepatic artery. (Figure B) The procedure was complicated by complete occlusion of left hepatic artery and spontaneous thrombosis of right hepatic artery upon catheterization. CTA abdomen after procedure showed reconstitution of flow within hepatic arterial supply via collaterals. Post procedure investigations revealed down trending inflammatory markers and transaminitis with levels returning to baseline within a week.
Discussion: To the best of our knowledge, this is the first reported case with multiple pseudoaneurysms involving both hepatic arteries. Although, in our case, histologic confirmation was not done, clinical presentation, laboratory findings and radiologic pattern, led to the diagnosis of SAM. Early detection is the key in the prognosis of SAM, as clinical course tends to be unpredictable and complications of vascular injury including stenosis, dissection, aneurysm or rupture may occur.



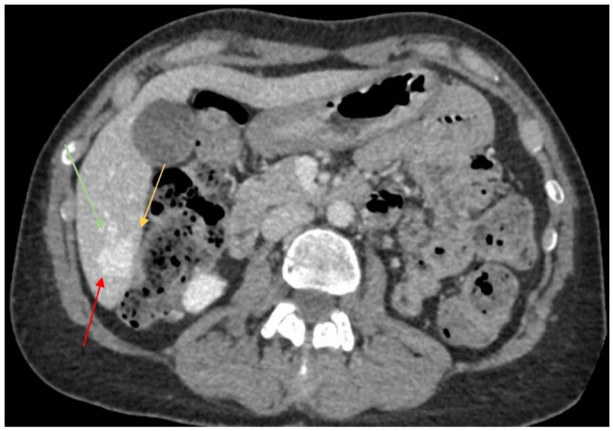
[3319] **Figure 1.** A) Computed tomographic angiography (CTA) of the abdomen demonstrated left hepatic artery aneurysm (arrowhead) and right hepatic artery aneurysm (arrow). B) Angiogram showing coil embolization of left hepatic artery (arrow).

S3320

An Intrahepatic Portosystemic Shunt Masquerading as a Tumor

Dayana Nasr, MD[1], Pujitha Kudaravalli, MBBS[1], Vishnu Kumar, MD[1], Alyssa Ionno, MD[1], Janane Nasr, MD[2], Ganesh Aswath, MD[1], Savio John, MD[1].
[1]SUNY Upstate University Hospital, Syracuse, NY; [2]Lebanese American University, Beirut, Beyrouth, Lebanon.

Introduction: Portosystemic shunts can be congenital or acquired and are divided into extrahepatic and intrahepatic shunts. Intrahepatic portosystemic venous shunts (IPSVSs) are abnormal intrahepatic connections that occur between branches of the portal vein (PV) and hepatic veins (HV) or IVC. Patients with IPSVSs can be symptomatic or asymptomatic as most of the cases are detected incidentally on imaging. However, they can potentially lead to long term complications by allowing bypass of mesenteric venous return into systemic circulation without going through the liver. We herein describe the case of a 58-year-old woman who was incidentally found to have an IPSVS on imaging for which she was recommended periodic surveillance imaging.
Case Description/Methods: A 58-year-old woman was undergoing workup for weight loss and was noted to have a 2.3 x 2.0 cm hyperdense nodular lesion in the posterior segment of the right hepatic lobe suspicious for hemangioma or a hypervascular neoplasm on CT abdomen pelvis. She was referred to the hepatobiliary clinic and a CTA abdomen pelvis was then performed and showed a lobulated 2.2 x 2.1 x 2.9 cm lesion in segment 6 consistent with an intrahepatic portosystemic shunt (figure 1). The patient was otherwise asymptomatic and therefore was advised to continue with periodic imaging surveillance.
Discussion: IPSVSs are divided into 4 types depending on their morphology. In type 1, a single large vessel connects the PV or its right branch to the IVC. Type 2 involves a localized peripheral shunt within one hepatic segment that has one or more communications between peripheral branches of the PV and HV. In type 3, an aneurysmal connection exists between PV and HV. And finally, multiple communications between PV and HV in multiple lobes are present in type 4. In patients without a history of liver disease or trauma, the origin of the IPSVS is presumed to be congenital. CT scan, MRI and even color Doppler sonograms should provide visualizations of the feeding portal vein and draining hepatic vein. For symptomatic IPSVSs, endovascular closure is the standard of care. However, no consensus exists for asymptomatic cases and surveillance is an appropriate option in asymptomatic patients.



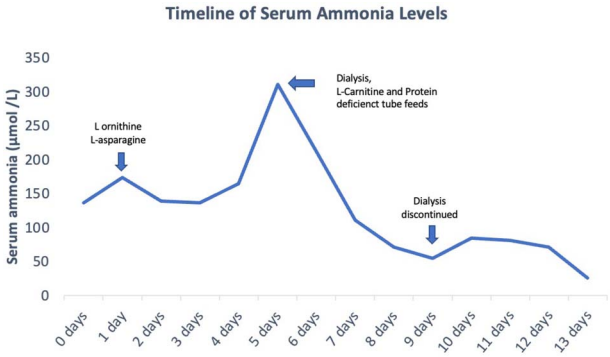
[3320] **Figure 1.** Axial CTA abdomen venous phase showing aneurysmal communication (red arrow) between right portal vein (yellow arrow) and middle hepatic vein (green arrow) in segment 6 of the liver.

S3321

All That Is Ammonia Is Not Cirrhosis: A Case of Hyperammonemic Encephalopathy and Anchoring Bias

Nimish Thakral, MD, Naba Saeed, MD, Hammad Qureshi, MD.
University of Kentucky, Lexington, KY.

Introduction: Anchoring bias is a cognitive bias that results from relying too heavily on the first piece of information that one is provided about a topic. Physicians must make a concerted attempt not to fall prey to anchoring bias as this can lead to a delay in diagnosis and often misdiagnosis. We describe a case of anchoring bias secondary to the diagnosis of hyperammonemic encephalopathy (HAE).
Case Description/Methods: A 20-year-old male with a history of Cerebral Palsy, Cognitive Dysfunction, and Epilepsy, was transferred to our institution secondary to refractory seizures and PEA arrest. Further history entailed gradually worsening mentation with increasing somnolence 3 weeks prior to initial presentation. Initial evaluation revealed elevated venous ammonia of 174 μmol/L. The Hepatology service was consulted for the management of refractory Hepatic Encephalopathy and presumed cirrhosis after the failure of response to lactulose and rifaximin. The addition of L-Ornithine-L-Asparagine had a paradoxical effect with serum ammonia rising to 311μmol/L. Subsequent MRI abdomen and MRCP showed smooth hepatomegaly with no splenomegaly or vascular shunts. Testing for etiologies of chronic liver disease including Autoimmune Hepatitis, Acute and Chronic viral hepatitis, Cholestatic liver disease, Wilson's disease, and Alpha-1 antitrypsin deficiency was negative. Plasma amino acids showed elevated Glutamine and Ornithine. Urinary amino acids showed elevated orotic acid. These findings were consistent with Ornithine Aminotransferase deficiency (OAT D). Dialysis was initiated for the rapid reduction of hyperammonemia. The patient responded well to low protein tube feeds and L-Carnitine supplementation with a sustained ammonia reduction after dialysis discontinuation. (Figure)
Discussion: HAE occurs most commonly in cirrhosis. Other causes include medications, vascular shunts, small intestinal bacterial overgrowth, and inborn errors of metabolism. Our patient likely had OAT D that was unmasked by the recent change in tube feeds. HAE was likely precipitated secondary to the change in feeds and cessation of home bowel regimen due to a self-limited diarrhea episode. OAT D is a rare cause of hyperammonemia which usually presents in the neonatal period but can sometimes be diagnosed in adulthood. In this case, the diagnosis was delayed by anchoring on cirrhosis as a cause for HAE. A high index of suspicion is required to make an accurate diagnosis leading to prompt intervention, thus avoiding delay in care and associated morbidity.



[3321] **Figure 1.** Timeline of Serum Ammonia Levels.

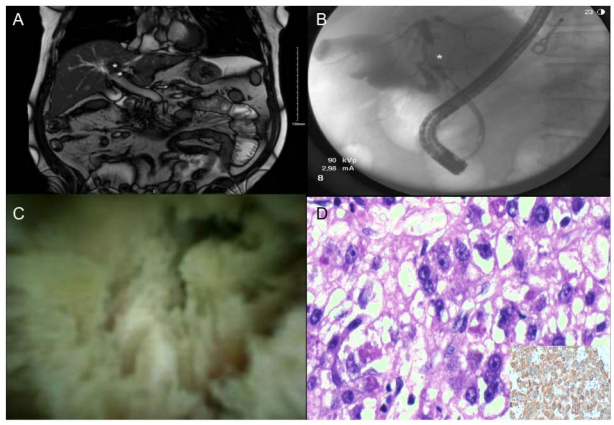
S3322

An Uncommon Cause of Obstructive Jaundice: Icteric Type Hepatoma

Jiten P. Kothadia, MD[1], Joshua French, MD[2], Richa Jain, MD[1], Thomas W. Faust, MD, MBE[1], Satheesh P. Nair, MD, FACG[1].
[1]*James D. Eason Transplant Institute, Methodist University Hospital, University of Tennessee Health Science Center, Memphis, TN;* [2]*Gastro One, Germantown, TN.*

Introduction: Hepatocellular carcinoma (HCC) presenting with obstructive jaundice as the initial symptom is rare, with incidence ranging from 1-12% of HCC cases. We report a case of a 73-year-old male patient with cirrhosis secondary to hepatitis C who presented with obstructive jaundice as an initial symptom of HCC.
Case Description/Methods: A 73-year-old male with history of hepatitis C (HCV) related cirrhosis presented to the hospital with a one-week history jaundice. Admission labs were notable for ALP 442 unit/L, ALT 127 unit/L, AST 146 unit/L, and total bilirubin of 10.2 mg/dL (0.2–1). A viral hepatitis serologies were negative. AFP was normal. CT scan of the abdomen showed some intrahepatic ductal dilation and a 3cm mass-like lesion in the inferior liver. A subsequent MRI abdomen with contrast showed a 3cm mass causing biliary obstruction with extension into hepatic segment VIII concerning a hilar cholangiocarcinoma (Fig 1A). Gastroenterology was consulted, and an ERCP was done. On cholangiogram, there was a significant stricture of the common hepatic duct approximately 1 cm above the cystic duct with intrahepatic ductal dilation (Fig B). Biliary brushing was obtained from the stricture, followed by plastic stent placement. Post-procedure, his bilirubin improved as expected. His biliary brushing returned as benign ductal epithelial. Given the high suspicion of malignancy, ERCP was repeated. On cholangioscopy was performed, demonstrating the stricture area with concerning features for malignancy, including an abnormal, villious, and ragged type appearance. (Fig C and D). The biliary aspirate, repeat brushings, and direct tissue biopsies were obtained via cholangioscopy for histopathological analysis and plastic stent was replaced. The final pathology result showed hepatocellular carcinoma (Figure E). Given a history of cirrhosis and evidence of portal hypertension on imaging, the patient was deemed not a surgical candidate.

He was started on treatment with atezolizumab and bevacizumab in November 2021 with a favorable outcome. His most recent scan showed a stable treatment site without any recurrence of extrahepatic spread. His case was discussed in our multidisciplinary transplant evaluation committee and he is considered for a liver transplant.
Discussion: Although rare, HCC should be considered a differential in cirrhotic patients presenting with obstructive jaundice. ERCP with Bile duct brushing cytology is extremely valuable in diagnosing HCC with an invasion of the biliary tract.



[3322] **Figure 1.** A: MRI image with mass (marked with *) Fig B: Cholangiogram with common hepatic duct stricture (marked with *) Fig C: Cholangioscopy view of the abnormal appearing tissue in the stricture Figure D: H&E x400: The carcinoma cells are large, polygonal with abundant cytoplasm and prominent central nucleoli. Some of the cells contain Mallory-Denk bodies (*) characteristic of steatohepatitic type hepatocellular carcinoma. Inset shows strong and diffuse immunoreactivity for Hep Par 1 antibody, which is a hepatocellular marker.

S3323

An Interesting Case of Meloxicam-Induced Autoimmune Hepatitis

Zainab Farooqui, DO, Alexander Savino, DO, Cristian Rios Perez, MD, Loretta Jophlin, MD, PhD.
University of Louisville, Louisville, KY.

Introduction: Autoimmune hepatitis (AIH) is an immune-mediated inflammatory liver disease with a highly variable clinical presentation ranging from mildly abnormal liver tests to fulminant liver failure. Symptoms can vary and are not limited to nausea, vomiting, and anorexia. It is difficult to discern an inciting culprit for AIH, but it may be triggered by infections, environmental factors, and medications. The most common medications reported to induce AIH are nitrofurantoin and minocycline. Non-steroidal anti-inflammatory drug (NSAID)- induced AIH is less frequently reported. In this case report, we describe a rare presentation of drug-induced AIH from the NSAID meloxicam.
Case Description/Methods: A 33-year-old female with history of obesity and rheumatoid arthritis presented for symptoms of abdominal pain, fatigue, nausea, and non-bloody emesis for one month. She denied alcohol or drug abuse, liver disease, and recent herbal supplements. Medications included meloxicam for one year prior and tizanidine. Physical exam was unremarkable. Labs were remarkable for severe transaminitis (table 1), elevated immunoglobulin G (IgG), positive ANA. Liver biopsy revealed periportal and pericentral lymphoplasmacytic inflammatory infiltrate associated with interface hepatitis and marked hepatocyte dropout. Meloxicam was discontinued and the patient received IV N-acetylcysteine and prednisone taper. Follow up in clinic showed significant improvement of patient's labs, symptoms, and resolution of her IgG autoantibody.
Discussion: Early diagnosis of DI-AIH, as well as distinguishing it from other forms of acute liver injury, is important because DI-AIH is responsive to immunosuppressive therapy, and early initiation of treatment can obviate the need for liver transplantation. Some distinguishing factors of DI-AIH include resolution of transaminitis after discontinuation of the offending agent, and lower duration of treatment required without relapse. To our knowledge, there are very few case reports describing meloxicam induced-AIH. However, a retrospective cohort did report that most DI-AIH cases were due to nitrofurantoin (67%), followed by NSAIDS (17%). NSAIDs are more frequently associated with this disease process than previously considered and should be on the differential when posed with acute liver injury. This would allow early identification and management of affected patients, and may prevent chronic liver injury progression and the need for liver transplantation.

Table 1. Timeline of Liver Function Tests

Event	Duration of Meloxicam	AST	ALT	INR	Total Bilirubin	Alkaline Phosphatase
Prior to initiation of meloxicam	0 months	11	15	-	0.3	47
Onset of symptoms	40 weeks after initiation	44	83	-	0.6	37
Hospital Admission	2 weeks after discontinuation	772	962	1.63	2.9	68
Hospital Discharge	3 weeks after discontinuation	29	234	1.48	1.6	53
Clinic Follow-up	7.5 weeks after discontinuation	21	49	-	0.3	47
Clinic Follow-up	16.5 weeks after discontinuation	41	79	-	0.6	34

S3324

An Unusual Case of Chronic Rejection Following Liver Transplant Due to Autoantibody Formation During Pregnancy

Raisa S. Zahir, BS[1], Sharon M. Skorupski, BS[2], Hamna Fahad, MD[3], Syed-Mohammed Jafri, MD[3].
[1]Wayne State University School of Medicine & Henry Ford Health System, Novi, MI; [2]Henry Ford Hospital, Detroit, MI.

Introduction: We present a unique case of chronic rejection due to HLA-antibody formation during pregnancy.
Case Description/Methods: A 51-year-old female with a history of autoimmune hepatitis received a live donor liver transplant (LDLT) with her husband as the donor in January 2008. HLA crossmatch testing was performed prior to transplant with a negative result. Patient was well overall for 8 years following liver transplant. Moderate elevation of liver enzymes during this period lead to liver biopsies consistent with autoimmune hepatitis but no evidence of acute rejection. Patient became pregnant 8 years after LDLT. She developed moderate-chronic elevation of liver enzymes during pregnancy with the most prominent peaks being 731 IU/L alkaline phosphatase (N: 40-140 IU/L), 200 IU/L aspartate aminotransferase (N: < 35 IU/L), and 160 IU/L alanine aminotransferase (N: < 52 IU/L). In December 2016, the patient had a liver biopsy displaying microvesicular steatosis, endotheliitis, and bile duct damage representative of acute cellular rejection. The patient was placed on quadruple immunosuppression therapy: everolimus, tacrolimus, prednisone, and mycophenolate mofetil. HLA antibody testing was performed at that time to check for donor specific antibodies (DSA). DSA was identified against HLA DQ6, suggesting sensitization to her husband's HLA antigens that occurred during pregnancy/delivery. Despite extensive treatment, chronic rejection of the liver transplant prevailed with bile duct damage progressing to ductopenia. Additional treatments for her condition included intravenous immunoglobulin (IVIG) therapy every 28 days, with photopheresis and plasmapheresis to reduce leukocytes and immunoglobulins

attacking the transplanted liver respectively. However, even with multi-modal therapy, the patient's condition worsened indicated by a rising MELD (model of end-stage liver disease) score and bilirubin concentration to 8 mg/dL.
Discussion: We present a unique case of acute (antibody-mediated) rejection progressing to chronic rejection in a LDLT patient following pregnancy due to sensitization to donor HLA DQ6. Consistent post-transplant HLA antibody testing should be a consideration for LDLT patients for early detection and treatment of DSA before memory B-cell production allows rejection to become chronic. Testing is especially important for monitoring female patients with LDLTs from spouse due to risk of blood exposure during pregnancy.

S3325

An SOS for SOS: A Case of Orthotopic Liver Transplantation in the Management of Hepatic Sinusoidal Obstruction Syndrome

Sandy Chan, MD[1], Cecillia Wong, MD[2], Matthew Petersile, MD[2], Hau Nguyen, MD[2], John Malinowski, MD[2], Curtis Barry, MD[2].
[1]Hofstra Northwell, Worcester, MA; [2]UMass Memorial Medical Center, Worcester, MA.

Introduction: Hepatic sinusoidal obstruction syndrome (SOS), previously known as veno-occlusive disease, is a rare syndrome that typically occurs after hematopoietic cell transplantation and is caused by injury to sinusoidal endothelial cells, which activates an inflammatory response with involvement of coagulation and fibrinolytic pathways, ultimately causing liver necrosis. Risk factors for SOS include aggressive myeloablative regimens. Treatment includes defibrotide and supportive management for severe disease. Transjugular intrahepatic portosystemic shunt placement and liver transplant are rarely indicated for patients in the setting of failed medical therapy with only case reports described in the literature. We describe a case of a patient with SOS who underwent liver transplantation in order to add to the medical literature and help inform future care teams.
Case Description/Methods: A 46-year-old male with medical history significant for acute myeloid leukemia status post allogenic stem cell transplant presented with abdominal pain, diarrhea, and nausea and vomiting. Labs were notable for transaminases in the thousands with transjugular biopsy showing peliosis hepatitis, prominent centrilobular sinusoidal dilation, and focal fibrous obliteration of small venules, consistent with SOS. Hospitalization was also complicated by hypoxic respiratory failure requiring intubation for airway protection secondary to encephalopathy and renal failure. Patient was started on defibrotide but liver function continued to worsen. He ultimately underwent orthotopic liver transplantation but remained persistently hypotensive despite multiple vasopressors with evidence of shock liver. Patient passed away despite maximal medical therapies and orthotopic liver transplantation.
Discussion: Prognosis for patients with severe SOS is poor with patients developing irreversible liver disease and subsequent multiorgan failure despite medical therapies. Liver transplant is rarely done in attempts to stabilize patients with SOS. Our case describes a patient with acute myeloid leukemia status post allogenic stem cell transplant who presented with abdominal pain and was found to have fulminant liver failure from SOS. Patient passed away despite maximal medical therapies and orthotopic liver transplantation. Further research is warranted to assess whether OLT should be considered in patients who present with severe SOS.

S3326

An Unusual Suspect in a Case of Severe Hyperammonemia

James Yang, MD[1], Rohit Agrawal, MD[1], Saul E. Turcios Escobar, MD[1], Michael Stone, MD[1], Adam Mikolajczyk, MD[2].
[1]University of Illinois, Chicago, IL; [2]University of Illinois Medical Center, Chicago, IL.

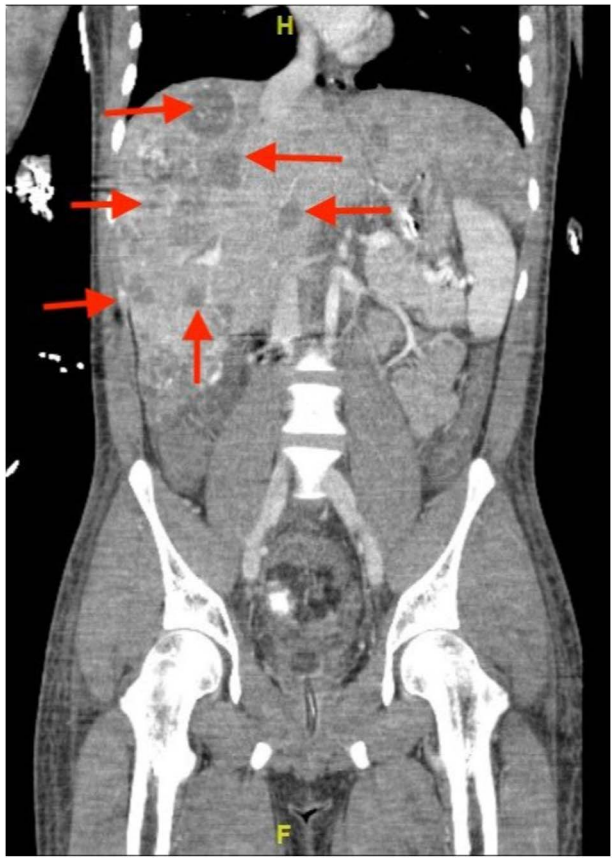
Introduction: Severe hyperammonemia is a life-threatening condition that can cause catastrophic neurologic complications from cerebral edema. Liver dysfunction is the most common etiology, but urease-producing infections are an uncommon cause. We present a rare case of severe hyperammonemia from disseminated *Cryptococcus neoformans*.
Case Description/Methods: A 69-year-old male with compensated cryptogenic cirrhosis who received a kidney transplant 1 year prior presented with confusion and a 30 kg weight loss. Examination revealed a scaphoid abdomen, a pericardial knock, and asterixis. Laboratory tests revealed stable kidney function (BUN 37 mg/dL, creatinine 2.3 mg/dL), mild elevation in alkaline phosphastase (204 U/L), normal albumin (3.8 g/dL) and prothrombin time (14.2 seconds), and an elevated venous ammonia level (466 mcg/dL). Serum carnitine, protein electrophoeresis, urine culture, and serum/urine amino acid analyses were normal. Magnetic resonance imaging revealed no large portosytemic shunts. Echocardiogram revealed a septal bounce in diastole consistent with constrictive physiology. The patient was started on lactulose, rifaximin, and antimicrobial therapy with improvement in his mentation. However, on the third hospital day, the patient became obtunded. Arterial ammonia level was 758 mcg/dL. Continuous renal replacement therapy was initiated, but shortly thereafter he developed anisocoria and cardiac arrest. CT head revealed marked cerebral edema without herniation, and comfort care was then pursued. A subsequent autopsy revealed disseminated *Cryptococcus neoformans* infection involving the lungs, pericardium, pancreas, liver, and adrenal glands.
Discussion: Hyperammonemia results from 1) diminished metabolism to urea in the liver due to hepatocellular dysfunction, an enzymatic defect, or portosystemic shunting; 2) increased production; and/or 3) decreased excretion in kidneys or muscles. Certain organisms (e.g. *Proteus*) express urease, which catalyzes the hydrolysis of urea to carbon dioxide and ammonia, and can cause increased production of ammonia. *Cryptococcus neoformans* is a less recognized urease-producing organism and was the major source of ammonia production in our patient (coupled with impaired metabolism and excretion from cirrhosis and sarcopenia/kidney disease, respectively). Gastroenterologists need to be aware of *Cryptococcus neoformans* as an etiology of hyperammonemia, particularly in immunosuppressed patients unresponsive to standard therapy.

S3327

Angiosarcoma Presenting as Hemorrhagic Shock: A Rare Diagnosis of Liver Lesions in a Young Patient

Shivani Trivedi, DO[1], Vikas Sethi, DO[2], Kritos Vasiloudes, MD[3], Amit Toor, MD[3], Gilad Shapira, DO[3], Salah Al-andary, MD[3], Joseph Namey, DO[1].
[1]HCA Healthcare/USF Morsani College of Medicine GME, Largo Medical Center, Largo, FL; [2]HCA Healthcare, Clearwater, FL; [3]HCA Healthcare, Largo, FL.

Introduction: Angiosarcoma is an aggressive tumor and rare in young healthy patients. Angiosarcoma is associated with exposure to arsenic, vinyl chloride, and oral contraceptives[1]. This is an unusual case of angiosarcoma in a young patient presenting with an acute abdomen from hemorrhagic shock. Angiosarcoma has poor prognosis and even removal and transplantation of the Liver is no sufficient [1]. It is important to include angiosarcoma in the differential for patients presenting with acute abdomen and explore aggressive medical therapy along with surgical resection.
Case Description/Methods: A 19-year-old male with no past medical history presented with sudden severe diffuse abdominal pain, difficulty breathing, nausea, vomiting, and diarrhea. FAST exam showed large volume of free fluid in the abdomen. Initial labs showed elevated liver associated enzymes, Hgb 5.4, lactic acidosis, fibrinogen 171. CT of abdomen and pelvis showed hepatomegaly with multiple small hypervascular lesions in liver [Figure 1]. Massive transfusion protocol was initiated, and he subsequently required emergent exploratory laparotomy with 3L of old-appearing blood drained. Intraoperatively, the liver was found to be multinodular, woody, enlarged, and cirrhotic with fresh heme. Final surgical pathology showed angiosarcoma with atypia over epithelioid hemangioendothelioma. Patient continued losing blood and required a hepatic angiogram with embolization. He continued to worsen, developing acute liver failure. Patient continued to decompensate and eventually required palliative care. (Figure)
Discussion: Angiosarcoma often presents in elderly patients with nonspecific symptoms such as abdominal distention, abdominal discomfort, weight loss, and fatigue[2,3,4]. Differentiating liver tumors such as hepatoma, adenoma, or vascular malformations on imaging is challenging and surgical resection is essential. Average survival of patients with untreated liver angiosarcoma is approximately 6 months and it increases by 2 years with treatment[2,3,4]. The standard treatment for liver angiosarcoma is surgical resection. Liver transplant is not indicated because of the high recurrence rate and poor prognosis[2,3,4]. There are no standardized treatments for patients presenting with hemorrhagic shock besides embolization and resection. More treatments need to be explored for aggressive treatment for hepatic angiosarcoma, as this case highlights a healthy 19-year-old patient dying one month after initial diagnosis with limited options for therapeutic intervention.



[3327] **Figure 1.** CT Abdomen and Pelvis with multiple hypervascular lesions.

S3328

An Atypical Etiology of Chronic Abdominal Pain: Peritoneal Tuberculosis

Hannah L. Zuercher, BS, Lucy Guerra, MD, MPH.
University of South Florida Morsani College of Medicine, Tampa, FL.

Introduction: Peritoneal tuberculosis (TB) is a rare presentation of extra-pulmonary TB, comprising only 5% of extra-pulmonary TB cases. Patients oftentimes present with non-specific complaints and without typical *Mycobacterium tuberculosis* risk factors. Additionally, diagnostic testing lacks high sensitivity and specificity with further invasive methods frequently necessitated. We present an unusual case of peritoneal TB presenting as non-localized abdominal pain, facilitating insight into this uncommon disease.
Case Description/Methods: A 53-year-old female with a past medical history of hypothyroidism presented with joint pain diagnosed as seronegative rheumatoid arthritis and subsequently began treatment with methotrexate and adalimumab for one year. Within the following year, she experienced chronic right lower quadrant (RLQ) abdominal pain, fevers, night sweats, fatigue, and ascites. Extensive work-up revealed a positive QuantiFERON gold TB test. A diagnostic laparoscopy with peritoneal biopsy successively demonstrated elevated CA-125 and large-volume ascites. *Mycobacterium tuberculosis* PCR and Grocott methenamine silver staining were negative. One of two acid-fast bacillus (AFB) smear and cultures yielded a positive result. Our patient was treated for peritoneal TB. She continued to have several years of chronic RLQ abdominal pain several months after RIPE treatment completion, undergoing a cholecystectomy and three adhesion lysis procedures with mild symptomatic improvement and subsequent worsening thereafter. An operative note highlighted the large amount of adhesions from the patient's liver to her diaphragm and anterior abdominal wall, appearing similar to Fitz-Hugh-Curtis syndrome. She continues to have chronic abdominal pain and is followed closely by her treatment team.
Discussion: Our patient was on immunosuppressive medications and had a history of travel to Mexico, a high TB burden country. Notably, AFB smear and culture has a poor sensitivity for peritoneal TB, oftentimes necessitating laparoscopic peritoneal biopsy for further diagnosis. Our patient was empirically treated for peritoneal TB before AFB culture yielded positive results, given her B symptoms and positive QuantiFERON test. She also had an elevated CA-125 value, which is associated with peritoneal TB and may be used in patients with a negative AFB stain. Our patient experienced recurrent adhesions, an unfortunate and persistent consequence of the inflammatory nature of peritoneal TB.

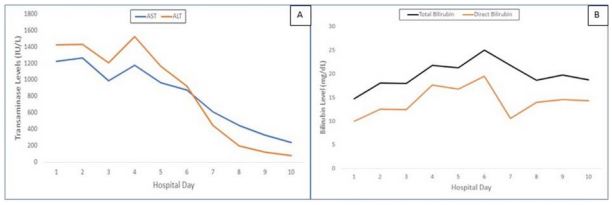
S3329

An Unusual Presentation of Seronegative Autoimmune Hepatitis

Gagan Verma, DO, MPH, Adam Meyer, MD, Joseph Mizrahi, MD, Edward Sun, MD, MBA.
Stony Brook University Hospital, Stony Brook, NY.

Introduction: Seronegative autoimmune hepatitis presenting as acute liver failure is uncommon.
Case Description/Methods: A 74-year-old woman with past medical history of GERD and moderate Alzheimer's dementia was noted to have transaminitis on routine outpatient labs (ALT 272, AST 183), which did not resolve over four months. Her only medications were calcium and famotidine. She took no supplements or substances. She presented to the hospital after recent repeat chemistries showed worsening transaminitis. Upon admission, she appeared fatigued and jaundiced. Family noted she was unable to care for herself as usual. Initial labs revealed: total bilirubin 14.8 mg/dL, direct bilirubin 10.0 mg/dL, INR 1.7, AST / ALT 1428 and 1224 IU/L, Alk Phos 168 IU/L , Albumin 3.4 g/dL. Toxicology, immunologic serologies, including anti-smooth muscle antibodies (ASMA), and infectious testing were unremarkable. Immunoglobulin G was elevated (2700). CT of the abdomen and pelvis noted a small lesion in the pancreas and pancreatic and periportal adenopathy, and an enlarged gallbladder with sludge. She was admitted to medical oncology with a presumed diagnosis of pancreatic cancer. She could not complete MRI and underwent endoscopic ultrasound (EUS) on hospital day 4, which revealed normal pancreas and bile ducts. EUS-guided biopsy of the liver was done. On hospital day 6, she developed acute liver failure and methylprednisolone and N-acetylcysteine were started. Due to dementia, she was ineligible for liver transplant. On hospital day 8, pathology confirmed autoimmune hepatitis with bridging fibrosis. By hospital day 10, liver function improved (Figure 1 A-B); however, she developed fever and, despite broad-spectrum antibiotics, suffered from septic shock and expired on day 11.

Discussion: Fulminant-onset autoimmune hepatitis (AIH) with hepatic encephalopathy is rare, occurring in < 6% of patients with AIH. The frequency of seronegative autoimmune hepatitis in acute and severe AIH is further estimated to be ≤7%. This case illustrates the challenge in diagnosis, particularly when confounding factors are present. Our patient suffered from dementia and aphasia, which masked developing hepatic encephalopathy, and pancreatic cancer was initially suspected as the cause of her painless jaundice, which delayed steroid treatment. A high index of suspicion for AIH is needed in patients with liver failure of uncertain etiology, as early treatment with steroids may improve the clinical outcome.



[3329] **Figure 1.** (A-B).

S3330

An Extremely Rare Case of Wilson's Disease Related Cirrhosis in a Patient With Neurofibromatosis Requiring Liver Transplant

Deena Kapadia, MD[1], Mohammad Abdallah, MBBS[1], Luisa Recinos, MD[2].
[1]Rutgers New Jersey Medical School, Newark, NJ; [2]Saint Peter's University Hospital, Somerset, NJ.

Introduction: Wilson's Disease (WD) is a rare cause of end stage liver disease with a prevalence of approximately one case in 30,000 live births in most populations. Similarly, neurofibromatosis type 1 (NF1) is a rare neurocutaneous disorder with an incidence of approximately 1:2600 to 1:3000 individuals. This is an extremely rare case of WD in a patient with neurofibromatosis requiring liver transplant.
Case Description/Methods: A 34 year-old man presented to the ED for bilateral lower extremity swelling, fatigue, and jaundice for one month. His past medical history was significant for NF1 and abnormal liver enzymes. Labs showed total bilirubin 15.5, Alkaline Phosphatase 228, ALT 575, AST 297; MELD-Na score was 27. MRCP was negative for choledocholithiasis. Additional workup was significant for ceruloplasmin 10 (low), 24-hour urine copper 782 ug/24 hours (high); ferritin 2,000. Hemochromatosis gene testing was negative. AFP 38, CEA 8.3, IgG 2457, Alpha-1-Antitrypsin 197. Serologies for viruses and testing for autoimmune conditions were negative. Liver biopsy histology showed severe cholestatic hepatitis, Stage 3-4 cirrhosis, increased iron deposition, and mild interface activity. He received an orthotropic liver transplant. A liver biopsy was significant for elevated copper concentration, strongly suggestive of WD. Histology revealed minimally active cirrhosis, and severe intracanalicular and intracytoplasmic cholestasis. WD genetic sequencing demonstrated one pathogenic variant (c.2731-2A >G; Heterozygous) and one variant of uncertain significance (c.1171T >C; Heterozygous) in the ATP7B gene.
Discussion: WD is a genetic disorder of copper metabolism causing impaired biliary copper excretion, which leads to accumulation of copper in several organs, most notably the liver, eventually leading to cirrhosis. WD results from an autosomal recessive mutation in hepatocyte copper-transporting ATPase. The hallmarks of NF1, the most common type of neurofibromatosis, are the multiple café-au-lait macules and associated cutaneous neurofibromas. It results from a mutation in NF1 tumor suppressor gene. It is autosomal dominant with 100% penetrance rate but with variable expressivity. WD and NF result from two uniquely different mutations on different chromosomes and inheritance patterns. In our patient, it remains a mystery if those two diseases occurred as a result from extremely rare odds of inheritance or from a non-familial incident leading to both mutations sporadically.

S3331

An Unusual Case of Syphilis Presenting as Acute Liver Injury

Sadaf Afraz, DO[1], Lewjain Sakr, MD[1], Amy Van, MD[1], Niraj Bhalani, MD[2], Kanwarpreet Tandon, MD[1], Raymond Lee, MD[1], Kevin Perry, MD[1], Nikhil Kapila, MD[1].
[1]Cleveland Clinic Florida, Weston, FL; [2]Larkin Hospital, Miami, FL.

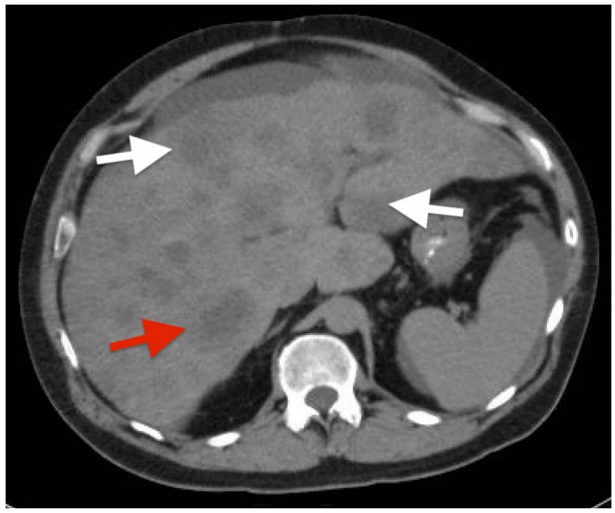
Introduction: Syphilis is a rare cause of hepatitis with a reported incidence of 0.25%–38%. Given the resurgence of syphilis in the past decade, awareness of syphilitic hepatitis (SH) remains critical in early diagnosis and treatment. We present a case of secondary syphilis presenting with elevated liver enzymes diagnosed initially as DILI on liver biopsy.
Case Description/Methods: A 45-year-old male presented to the hospital for two-week history of generalized abdominal pain associated with nonpruritic truncal rash. Pertinent medication history included use of testosterone and anastrozole for 5 years, and recent use of the herbal supplement ashwagandha. Of note, patient had taken HIV prophylaxis medications for potential exposure prior to presentation. Physical exam was notable for epigastric tenderness and erythematous blanching maculopapular dispersed rash throughout the chest, abdomen, back, and extremities. Initial workup revealed elevated ALP 385, AST 100, ALT 247, and total bilirubin 2.9. Abdominal CT showed hepatosplenomegaly and liver doppler ultrasound demonstrated patent vasculature. Viral hepatitis panel, HIV, ceruloplasmin, alpha-1-antitrypsin and autoimmune serologies were negative except for mildly positive ANA of 1:160. Given his medication history, rash and cholestatic pattern of liver injury, drug-induced liver injury (DILI) was suspected and liver biopsy was performed. Liver biopsy demonstrated moderate mixed portal inflammatory infiltrates and mild portal fibrosis with no bridging fibrosis or cirrhosis. Iron and PAS stains were negative. He was initially diagnosed with DILI and treatment was initiated with steroids. During the hospitalization, his rash progressed to involve both hands. A syphilis screen was positive with reflex RPR revealing titer of 1: 256. Subsequent treponemal stain of the liver biopsy showed spirochetes in the connective tissue and blood vessels in large size portal tracts consistent with SH. A single dose of Benzathine penicillin 2.4 million units for secondary syphilis was administered with subsequent resolution of the rash and normalization of liver enzymes on follow-up.
Discussion: The clinical presentation, liver abnormalities and histopathological testing of SH are often nonspecific. A high index of clinical suspicion along with targeted screening can lead to prompt diagnosis. Treatment with penicillin leads to rapid improvement in liver enzymes and prevents progression to fulminant liver failure, a rare complication.

S3332

An Unusual Case of Aggressive Prostate Cancer With Liver Metastasis in a Patient With Ulcerative Colitis and Total Colectomy

Reshad Salam, MD, Iana Gueorguieva, MD, Bradley J. Warren, MD.
Ascension Providence Hospital, Southfield, MI.

Introduction: Prostate cancer is a relatively slow-growing with 96.8% 5-year survival rate. Less than 7% of patients are found to have aggressive cancer with metastasis to distant sites, such as: bone, lymph nodes, liver and thorax. The incidence of prostate cancer in the 45-54 year age group is merely 7.2% and median age at diagnosis is 67 years. We present a rare case of a 51-year old patient found to have liver metastasis of prostate origin, which was initially thought to be of gastrointestinal origin given elevated Carcinoembryonic Antigen (CEA) and CA 19-9.
Case Description/Methods: A 51-year-old male patient with history of ulcerative colitis status post total colectomy thirty years prior, presented with progressive abdominal distension and unintentional twenty pound weight loss over a two month period. Physical exam revealed a mild jaundice, non-tender distended abdomen with palpable hepato-splenomegaly. Routine laboratory revealed normal complete blood count, with liver function elevation: total bilirubin 3.8, direct bilirubin 3, ALP 722, ALT 112, and AST 395. Computed tomography (CT) scan of the abdomen and pelvis without contrast showed findings concerning for metastatic disease with innumerable hepatic mass lesions, and lymphadenopathy. Cancer markers obtained showed normal AFP, however elevated Chromogranin A, CEA and CA 19-9, at 111 ng/mL, 741 ng/mL and 892 ng/mL, respectively. While initially a gastrointestinal origin of cancer was suspected, a liver biopsy was performed which revealed a poorly differentiated adenocarcinoma of prostate origin with positive staining for NKX3.1 and elevated PSA. (Figure)
Discussion: Prostate cancer is the second most frequent malignancy in men worldwide and second leading cause of male-cancer related death in the United States, yet has favorable survival rates. The most common tumor marker for prostate carcinoma is serum PSA, however there is no current guideline recommending periodic PSA measurements. Elevated levels of CEA and CA 19-9 in the setting of metastatic prostate cancer have rarely been reported in literature, with merely eight known other cases. Given patient's underlying history of ulcerative colitis, suspicion for cholangiocarcinoma would have been high on differential and in this case liver biopsy would be contraindicated. Our case highlights a rare presentation of liver metastasis and highlights importance of keeping broad differential. Importantly, liver metastatic burden is of concern and limiting factor when choosing immunotherapy or chemotherapy.



[3332] **Figure 1.** Computed tomography (CT) image of the abdomen, showing innumerable hypodense hepatic mass lesions (white arrows), with the largest in the right hepatic lobe measuring up to 5.4 x 5.0 cm indicated by red arrow.

S3333

Aloe Vera: A Boon or Bane

Uneza Khawaja, MD, Nida Anwaar, MD, Muhammad Masood, MD.
Danbury Hospital, Danbury, CT.

Introduction: Aloe Vera is derived from a cactus-like plant, a member of the Lily family that grows best in arid climates. Aloe Vera products are derived from the leaf and contain over 75 identified substances including anthraquinones, vitamins (A, C, E), enzymes, minerals, sugars, fatty acids, amino acids, salicylic acid and hormones. Aloe has been widely used in phytomedicine and is described as a herb which has anti-inflammatory, anti-proliferative, anti-aging effects.
Case Description/Methods: 54-year-old male with a history of Hashimoto's thyroiditis, Gilbert's syndrome, came to ER with RUQ abdominal pain. He used to drink 4-5 glasses of wine everyday and quit 3 months ago. Also reported dark urine and a 4 pound weight loss over a month. He has been taking Aloe Vera supplements for gut motility for 4 months. Had scleral icterus and hepatosplenomegaly on exam. Labs showed elevated total bilirubin of 8.1 with direct hyperbilirubinemia(5.3) , ALP of 163, ALT 2790, AST 1892. Work up ruled out acetaminophen toxicity, viral and autoimmune hepatitis. Abdominal US showed hepatic cyst and small amount of sludge in the gallbladder without biliary ductal dilation. Liver biopsy showed an acute hepatitis pattern of injury with no fibrosis/cirrhosis. Aloe Vera supplements were discontinued and LFTs started trending down and repeat values after 10 days showed total bilirubin of 3.4, ALP 133, ALT 1308, AST 506. LFTs have almost normalized after 1 month.
Discussion: Cases of Aloe Vera-induced hepatotoxicity have been reported since 2005 but its pharmacokinetics and toxicity are poorly described in literature. It has been reported in multiple countries with the most in Switzerland. Injury typically arises between 3 and 24 weeks after starting oral Aloe Vera. Typical pattern of injury is hepatocellular and the clinical course resembles acute viral hepatitis. Injury is rarely severe and fatal cases have not been reported. Most cases of hepatotoxicity from Aloe Vera have been self-limiting upon discontinuing it. Few cases have been severe or prolonged, but no instances leading to liver transplantation, chronic hepatitis, or vanishing bile duct syndrome. Rechallenge has led to recurrence of injury in at least one published case report and should be avoided. Our case was one of the examples of rare cases of acute toxin induced liver injury secondary to herbal products. Though Aloe Vera has its benefits, patients should be discouraged from using OTC supplements containing this product as the data on the safety is scarce.

S3334

An Atypical Presentation of Propylthiouracil-Associated Fulminant Hepatitis in a Patient With Thyroid Storm

Jabari Munroe, MD, Bakhtawer Siraj, MD, Sumedha Singh, MD, Richard Kalman, MD.
Einstein Medical Center, Philadelphia, PA.

Introduction: Propylthiouracil (PTU) is an antithyroid agent used in thyrotoxicosis which is known to have hepatotoxic complications typically within weeks to months of administration. We present a case in which PTU liver injury was observed to occur within 48 hours of administration in a patient with underlying liver disease.
Case Description/Methods: A 59 y.o. F with diet-controlled DM presented with palpitations and diarrhea. She was hypertensive and tachycardic in the ED; physical exam was unremarkable. Her laboratory workup was significant for TSH $<$ 0.01 mcIU/mL with FT4 3.2 ng/dL, TSI positive at 353%. Liver function tests (LFTs) showed ALP 277, Tbili 2.4, DBili 1.5, ALT 76, and AST 98. An echocardiogram showed an EF of 35%. The patient was diagnosed with thyroid storm and started on PTU (received 800mg over 24hrs) and propranolol. On day 2, her LFTs showed ALT 450 and AST 890, and PTU was stopped. The next day, she became encephalopathic and developed acute liver failure with an INR of 4.1. General surgery was consulted for thyroidectomy. However, she was deemed unsuitable for surgery given her increased pre-operative risk. She was started on a NAC drip for DILI. Other etiologies for fulminant liver failure were ruled out including Hep A, B, and C, autoimmune hepatitis, and portal vein thrombosis. Liver biopsy showed acute steatohepatitis with necrosis, hepatocanalicular cholestasis, and periportal and pericellular fibrosis. After stopping PTU, patient's LFTs normalized. On day 14, after potassium iodide preparation, the patient successfully underwent total thyroidectomy. Microscopic evaluation of thyroid tissue revealed multinodular hyperplasia. 6 weeks post-discharge she is asymptomatic with normal LFTs, TFTs and EF.
Discussion: PTU is a thionamide used to manage hyperthyroidism. It causes DILI in approximately 1 in 10,000 people. Onset is often within 2-12 weeks of initiation. In our patient, fulminant hepatic failure developed within 48 hours of initiation. The findings of her core needle liver biopsy noted above indicated a DILI superimposed on baseline liver disease. This baseline liver disease may have been related to thyroid disease, NASH or congestive hepatopathy which may have predisposed her to an exaggerated response to PTU. Fortunately, our patient recovered and had a successful total thyroidectomy. This case illustrates that PTU-associated DILI may present atypically and with short latency. Close monitoring of LFTs and early surgical evaluation may prevent fulminant liver failure.

S3335

An Unusual Presentation of DILI: Ketamine-Induced Liver Injury

Christopher Chhoun, DO[1], Neethi Dasu, DO[1], Kirti Dasu, BA[2], Yaser Khalid, DO[3], Drew Chiesa, DO[4].
[1]Jefferson Health NJ, Voorhees, NJ; [2]Drexel Graduate School of Biomedical Sciences and Professional Studies, Philadelphia, PA; [3]Wright Center for GME/Geisinger Health System, Scranton, PA; [4]Jefferson Health New Jersey, Turnersville, NJ.

Introduction: Drug induced liver injury (DILI) is common and it can be secondary to various classes of medication, such as over the counter drugs, antibiotics and herbal and dietary supplements. Antibiotics are the class of drugs that are most frequently associated with DILI. We present a unique cause of DILI caused by long-standing ketamine abuse in a 36-year-old patient.
Case Description/Methods: A 36-year-old female with a past medical history of migraines presented for evaluation of elevated liver enzymes. On hospital admission, she was found to have a significantly elevated serum alkaline phosphatase (AP) level of 1506 U/L and GgT of 1015 IU/L. Furthermore, the patient's AP had shown persistent elevations for approximately two years. Her daily AP ranged from 957-1506 U/L. Further workup was negative for viral hepatitis, autoimmune disease, hemochromatosis or other toxins (tylenol/alcohol). Abdominal ultrasound was unremarkable except for a common bile duct diameter of 3 mm. After further investigation, the patient admitted to ketamine abuse which had been ongoing for 5 years prior to admission. She admitted to using intranasal ketamine over 100 times daily, which she now discontinued. However, the patient's AP remained elevated and a liver biopsy revealed prominent lymphocytic cholangitis with acute and chronic pericholangitis, bile ductular proliferation, and bridging fibrosis stage 3/4 consistent with DILI. Although the patient no longer uses Ketamine for months, her AP remains elevated.
Discussion: The majority of DILI is benign and resolves after withdrawal of the offending agent. However, it is also the number one cause of acute liver failure in the United States. The clinical presentation of cholestatic DILI can be variable with an asymptomatic elevation in AP, ranging from a hepatocellular pattern to rarely having a cholestatic pattern, which depends on the offending drug. Intravenous ketamine-induced DILI is more widely reported in the literature unlike our case of intranasal ketamine-induced DILI. As Ketamine becomes more frequently used in the treatment of chronic pain, our case highlights the need for physicians to remain vigilant in recognizing the potentially adverse effects of powerful anesthetics. We present an extremely rare case of intranasal ketamine abuse leading to DILI.

S3336

An Unusual Cause of Encephalopathy

Shalini Tripathi, MD[1], Akhilesh Khuttan, MD[2].
[1]CarolinaEast Medical Center, New Bern, NC; [2]University of Iowa, Coralville, IA.

Introduction: Hepatic encephalopathy, while common among cirrhosis patients, can occur without cirrhosis. In adults, 90% cases of hyperammonemia are due to cirrhosis. Ammonia is central to its pathophysiology produced by bacterial degradation in the gut from protein, urea, and glutamine. Ammonia acts as a neurotoxin which is metabolized by hepatocytes to urea for excretion via kidney and colon. Type B hepatic encephalopathy is due to portosystemic shunting without parenchymal liver disease.
Case Description/Methods: A 69-year-old female with history of hepatoma with resection and chemoembolization in 1997, mucosa-associated lymphoid tissue, perforated diverticulitis status post colostomy and reversal in 2015 was in her usual state of health a day prior to admission when she presented with lethargy after episodes of nausea and vomiting. She was arousable and not able to follow commands. Her CT (computed tomography) of the brain with no acute changes followed by CT of abdomen showing dilated small intestine extending into ventral hernia with concern for obstruction. Blood tests revealed ammonia 141 umol/L, total bilirubin 4.1 mg/dL, direct bilirubin 1.5 mg/dL, international normalized ratio 1.2 and platelet 130 k/ul. Alkaline phosphatase, alanine and aspartate transaminases were within normal limits. Obstruction resolved with conservative management and she was started on rectal and oral lactulose with rifaximin via nasogastric tube. Her mentation improved significantly although the cause of her hepatic encephalopathy remained unclear. Then she underwent magnetic resonance imaging of the abdomen with contrast showing portal to hepatic vein shunt with stable postoperative changes. Shunt closure option was discussed and patient opted outpatient follow up.
Discussion: Prompt diagnosis and treatment of hepatic encephalopathy is important to prevent cerebral edema. In patients with encephalopathy and elevated ammonia in absence of liver parenchymal disease, work-up for shunt and urea cycle enzyme deficiency should be done. In the absence of cirrhosis most portosystemic shunts are congenital or traumatic. This case brings a rare cause due to remote liver surgery and chemoembolization. The treatment of portosystemic shunts is evolving, balloon-occlusion retrograde transvenous obliteration is one such technique. In the absence of varices, bland embolization with ectopic coils and vascular plugs can be utilized. Endovascular treatment carries an improvement of hepatic encephalopathy by 50 to 80% at 18 to 24 months.

S3337

An Unusual Case of Drug-Induced Liver Injury Secondary to Nitrofurantoin Use

Vikash Kumar, MD[1], Stephen Wonnacott, MS[2], Dhir Gala, BS[2], Anmol Mohan, MBBS[3], Mili Shah, BS[2], Jasparit Minhas, MD[1].
[1]The Brooklyn Hospital Center, Brooklyn, NY; [2]American University of the Caribbean School of Medicine, Brooklyn, NY; [3]Karachi Medical & Dental College, Karachi, Sindh, Pakistan.

Introduction: Nitrofurantoin is a commonly prescribed antibiotic for uncomplicated urinary tract infections (UTI). One of nitrofurantoin's rare, however significant, adverse effects is idiosyncratic drug-induced liver injury (DILI).
Case Description/Methods: A 24-year-old obese female with a past medical history of GERD presented to the emergency department complaining of epigastric pain with nausea and vomiting for the past few hours. The patient was recently diagnosed with a UTI and took nitrofurantoin (50 mg every 6 hours) for the past 3 days. Blood tests on admission were significant for direct bilirubin of 0.7 mg/dL, total bilirubin of 1.9 mg/dL, AST of >717 U/L, and ALT of 476 U/L. The patient had liver function tests (LFTs) a week ago, which were normal. Toxicology labs were negative for acetylsalicylic acid, acetaminophen, and alcohol. Viral workups, including Hepatitis tests, were negative for hepatitis A, B, C, and E and showed immunity for hepatitis A with IgG positive/IgM negative. Autoimmune workup including antinuclear antibodies (ANA), Anti-smooth muscle, anti-mitochondrial, and anti-LK- antibodies were negative. Ultrasound showed stones within the gallbladder. Nitrofurantoin was immediately discontinued, and after 3 days in the hospital, symptoms subsided. Repeat LFTs showed enzyme values trending towards normal (Table 1). The patient was educated to follow up with PCP for repeat LFTs and chances of possible relapse with similar medications on discharge.
Discussion: DILI can be a rare and fatal adverse effect of nitrofurantoin use. There is a higher risk of developing DILI from taking nitrofurantoin for female patients with increased age, reduced renal function, or increased duration of treatment with the drug. DILI generally presents in two forms with nitrofurantoin, acute and chronic. Acute DILI can present with fever, rash, eosinophilia, hepatomegaly, jaundice, abdominal pain, nausea, malaise, pulmonary signs, anorexia, and elevated liver enzymes. Most cases of acute DILI due to nitrofurantoin will resolve on their own after discontinuation of the drug but should be monitored in case chronic liver injury develops. Severe cases may require treatment with steroids, N-acetylcysteine, or transplantation. Our patient presented with signs of liver damage after starting a course of nitrofurantoin for a lower urinary tract infection. Once treatment was discontinued, the patient's condition improved, consistent with DILI.

Table 1. Patients lab results post discontinuation of Nitrofurantoin on day 1

Parameters	Day 1	Day 2	Day 3	Normal Values
Platelets	245,000	250,000	225,000	150,000-400,000/mm^3
Total Bilirubin	1.9	1.7	1.6	0.1-1.0 mg/dL
Direct Bilirubin	0.7	0.8	0.6	0.0-0.3 mg/dL
AST	> 717	406	135	8-20 U/L
ALT	476	520	331	8-20 U/L
Alkaline Phosphatase	120	129	104	20-70 U/L
Glucose	90	123	86	Fasting: 70-110 mg/dL 2-h postprandial: < 120 mg/dL
Albumin	4.5	4.1	3.7	3.5-5.5 g/dL
INR	1.0	1.2	1.2	0.8-1.2 secs
BUN	11	5	6	7-18 mg/dL

Table 1. (*continued*)

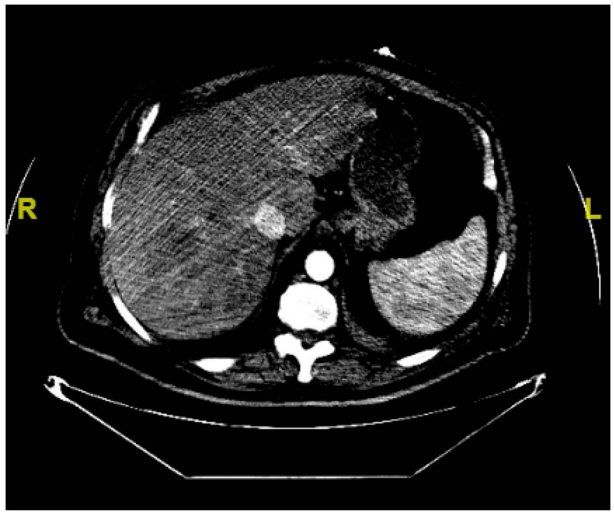
Parameters	Day 1	Day 2	Day 3	Normal Values
Creatinine	0.8	0.8	0.7	0.6-1.2 mg/dL
Sodium	135	138	137	136-145 mEq/L
Potassium	5.8	3.7	3.8	3.5-5.0 mEq/L
Chloride	103	106	106	95-105 mEq/L
ASA	< 5.0			5.0-20.0 mg/dL
Acetaminophen	< 1			5.0-20.0 mg/dL
Alcohol	< 0.01			
Gammaglobulin	wnl			

S3338

An Unusual Case of a Large Cell Neuroendocrine Carcinoma and Acute Liver Failure

Deep Patel, DO, Wadia Hamed, MD, Ian Lancaster, MD, Cyrus Tamboli, DO.
Largo Medical Center, Largo, FL.

Introduction: Large cell neuroendocrine carcinomas (LCNECs) are characterized as large cells with areas of necrosis, neuroendocrine traits (palisading, trabeculae, etc.), ample cytoplasm and high mitotic rates. LCNECs are a rare, aggressive form of neuroendocrine tumor known to have high rates of metastasis. Our case highlights acute liver failure with rapid progression to death due to metastatic spread of LCNEC.
Case Description/Methods: 71-year-old male with a history of prostate cancer requiring prostatectomy, hypertension, hyperlipidemia, and tobacco use, presented for shortness of breath and abdominal pain for 3 days. He was a poor historian and was unable to provide much history. Of note, the patient reported that he had imaging performed in spring 2021 to further evaluate his degenerative disc disease and was told there was concern for a cancerous process. Initial laboratory studies showed an elevated AST and ALT (over 300), and thrombocytopenia, but were otherwise unremarkable. Further imaging was performed, given the patient's history of reported malignancy. Initial chest X-ray was negative for acute findings and an abdominal ultrasound revealed a markedly abnormal appearance of the liver, suggestive of possible hepatic metastatic disease. Computed tomography scan of the chest/abdomen/pelvis showed a left hilar mass measuring 5.5 x 6cm with regional lymphadenopathy. The patient underwent a biopsy of the liver, and was later noted to have worsening liver enzymes, platelets, INR, and renal function, along with a lactic acid. He was transferred to the intensive care unit for further monitoring. Bowel ischemia and portal vein thrombosis were ruled out, and the patient became continually more acidotic, and had a pulseless electrical activity (PEA) arrest. He was revived and intubated, and placed on pressor support and continuous renal replacement therapy. His family decided to change his code status to "do not resuscitate" and the patient was made comfortable. Final results of the liver biopsy were obtained 2 days later, and showed findings consistent with high grade large cell neuroendocrine carcinoma. Immunohistochemical staining was positive for CD56, synaptophysin, CK7 and CK5/6. (Figure)
Discussion: Liver metastasis has been documented throughout the literature for other carcinomas, however very few in regards to LCNEC. Acute liver failure in such a short span of time, as in our case, highlights the mortality associated with LCNECs, and the difficulty in diagnosing the condition early in the disease course.



[3338] **Figure 1.** CT abdomen/pelvis showing diffuse heterogeneity of the liver.

S3339

An Atypical Source of Thrombocytopenia

Tyson Broadbent, MD, John S. Kelley, MD, Lancaster Weld, DO.
Baylor Scott and White, Temple, TX.

Introduction: We report a 29-year-old pregnant female with a past medical history notable for poorly controlled ulcerative colitis, recurrent c.diff, recent stent placement for choledocholithiasis with retained stent who was found to have a rare neuroendocrine adenocarcinoma with enteroblastic morphology following extensive workup for thrombocytosis .
Case Description/Methods: Patient had cholecystectomy due to ruq pain six months prior to admission.ERCP was performed with findings indicating choledocholithiasis for which she underwent stent placement. Due to lapse in insurance stent removal was not performed. Patient began to experience increased abdominal pain and and ct revealed a new soft tissue density involving the porta hepatis and gastrohepatic regions concerning for soft tissue mass, at which time she additionally discovered she was pregnant. OSH elected to not remove stent or biopsy mass at time due to pregnancy. She continued to experience abdominal pain with new radiation to back. Patient was initially transferred for evaluation of thrombocytosis platelet count of 1.2 million with multiple potential etiologies. Patient's ulcerative colitis was not treated so underwent flexible sigmoidoscopy which demonstrated friable tissue in active ulcerative colitis flare. Other etiologies that were evaluated included retained biliary stent, patient was also found to be C diff positive without toxin, additionally was found to be Campylobacter positive for which she underwent antibiotic treatment. Following initiation of ulcerative colitis therapy and exchange of biliary stent patient had some improvement in right upper quadrant pain but continued to have thrombocytosis. Patient underwent biopsy of abdominal lymphadenopathy which showed signs concerning for hepatocellular carcinoma. Due to concerns probability of hepatocellular carcinoma patient underwent repeat biopsy which demonstrated findings concerning for neuroendocrine tumor with yolk sac features.

Discussion: Patient was followed by multiple services including heme Onc, Gastroenterology, Maternal-Fetal Medicine who initiated treatment with chemotherapy while the patient was still pregnant, and ultimatelyly she had successful delivery of child. patient ultimately ending up passing away following progression of disease. This case helps to demonstrate the need for broad differential and the ability to reexamine assumed diagnosis in addition to constraints associated with socioeconomic factors impacting care.

S3340

An Immunocompetent Adult With Adenovirus Hepatitis

Jarin Prasa, DO[1], Syed S. Karim, DO[2], Michael Bernstein, MD[2].
[1]Staten Island University Hospital, Staten Island, NY; [2]Coney Island Hospital, Brooklyn, NY.

Introduction: In the US there has been a recent outbreak of adenovirus hepatitis in the pediatric population. However, to our knowledge, there has been only one reported case of adenovirus hepatitis in an immunocompetent adult. We have identified another such case.
Case Description/Methods: A 25 year old female with no medical history presented with abdominal pain, nausea, vomiting, diarrhea, and subjective fevers for two weeks and was found to have transaminitis 25-30x the upper limit of normal, which were: AST 791, ALT 542, ALP 92, and total bilirubin of 2.9. The patient reported no prior history of liver disease. She denied alcohol, tobacco, illicit drugs, or herbal medications, but did report taking acetaminophen 1500 mg daily for two weeks. Serum acetaminophen levels were normal and serum and urine toxicology were negative. US with doppler was unremarkable, CT showed cholelithiasis, MRCP showed a normal common bile duct without obstructive calculus. Autoimmune causes of hepatitis, ceruloplasmin and alpha-1 antitrypsin were all unremarkable. HAV, HBV, HCV, HDV, HEV, CMV, HSV, VZV, EBV, HIV, and COVID19 were all negative. Ultimately, the serology for adenovirus was positive. After a week of supportive treatment, the patient's labs trended down and symptoms resolved.
Discussion: Adenovirus is confirmed by a rise in antibody titer or by virus detection. Coagulative necrosis in histopathology is a finding in liver biopsies if they are pursued in unexplained cases of liver injury. Ultimately, adenovirus hepatitis can be diagnosed once all common causes of hepatitis have been excluded. In the current outbreak, only children have been getting adenovirus hepatitis. In adults, a high prevalence of neutralizing antibodies contributes to immunity, and therefore only in immunocompromised states, do adults get such an infection. Supportive care with IV fluids, electrolyte correction, and antiemetics usually is enough with eventual symptomatic and laboratory improvement as it was for our patient. Studies have shown that extensive disease can be treated with antiviral drugs, cidofovir, and ribavirin. Our patient's history of acetaminophen use is a confounder, however, her normal serum level and her symptoms suggestive of an infectious cause made acetaminophen less of a culprit. We hypothesize that our patient's use of acetaminophen when she was initially exposed to the virus is what made her susceptible to developing adenovirus hepatitis and we hope this case adds insight for clinicians dealing with future adult cases.