S2472

Gone in a Flash: Influenza-Induced Vanishing Bile Duct Syndrome
Michael Epstein, MD1, Matthew A. Heckworth, MD2, Bruce R. Yousem, MD, FACG2.
1University of Louisville School of Medicine, Louisville, KY.

INTRODUCTION: Vanishing bile duct syndrome (VBDS) is a group of acquired disorders that cause the destruction and disappearance of intrahepatic bile ducts and cholangitis. Influenza has been reported to rarely cause adverse events of cholestasis due to ductopenia characteristic of VBDS.

CASE DESCRIPTION/METHODS: Patient is a 51-year-old male with stage III melanoma treated with pembrolizumab, a programmed death 1 (PD-1) checkpoint inhibitor. The patient’s clinical course was complicated by pembrolizumab-induced colitis, and Influenza therapy was initiated. He only received two doses before developing worsening body diaphoresis greater than four times a day and dark urine. Vital signs on admission were within normal limits. His physical exam revealed jaundiced skin and right upper quadrant abdominal tenderness. Labs demonstrated a mixed pattern cholestasis with AST of 1480, ALT 627, total bilirubin 16, with an INR of 1.0. Liver biopsy on admission was consistent with a drug-induced liver injury or autoimmune hepatitis. An RGD was performed concerning for eosinophilic hepatitis but pathology reported demonstrated rare (< 1/100FP) intrahepatic eosinophils. Flexible sigmoidoscopy revealed CMV colitis by immunostaining biopsies from the rectosigmoid colon. MRCP showed no evidence of biliary obstruction or extra-hepatic ductal disease. However, repeat liver biopsy demonstrated extralobular and intralobular degeneration of bile ducts and acute cholestasis consistent with VBDS. No evidence of CMV by immunostaining was noted on this repeat liver biopsy.

DISCUSSION: Influenza can induce hepatotoxicity by causing cholestatic liver disease. This cholestasis has been typically described as self-limiting but can manifest with a prolonged clinical course of greater than six months. This is clinically distinct from that of (VBDS), which can progress to established liver cirrhosis VBDS is primarily a diagnosis of exclusion and although it is not commonly caused by influenza, it has been recently reported. Diagnosing VBDS is challenging, and as our patient showed, requires multiple liver biopsies, MRCP, and extensive infectious and immunological workup to rule out alternate causes. The mechanism of influenza induced cholestatic hepatitis has been described in recent literature, however the pathophysiology of this condition remains unclear, further studies into this condition are important and will likely include both genetic and environmental factors.

S2473

Obscure Chemical Causing Fulminant Hepatic Failure
Adderly Toribio, MD1, Seetha Lakshmanan, MD1, Alan Epstein, MD2.
1Roger Williams Medical Center, Providence, RI.

INTRODUCTION: Fulminant hepatic failure (FHF) is characterized by severe acute liver failure with impaired synthetic function and encephalopathy in patients without prior liver disease. FHF can result in coagulopathy, increased risk of infection, metabolic derangements including acute renal failure, electrolyte abnormalities, and pancreatitis. Toxins and viral hepatitits are the most common causes of FHF. Here we present a case of FHF due to poisoning by an unknown chemical.

CASE DESCRIPTION/METHODS: A 45-year-old female with no prior liver disease presented to the ED for sudden onset of burning sensation along her chest, abdomen, and extremities, associated with shivering and watery diarrhea for 1 day. The patient suspects someone spraying a white chemical into her house prior to her developing these symptoms. Labs on admission revealed ALT 7045/UL, AST 2480/UL with normal ALP, CBC, and renal function. The next day she became encephalopathic and liver enzymes worsened to AST 5048/UL and ALT 13800/UL. The patient’s GGT was 1499/UL, ammonia 285/µL, INR 1.7, total bilirubin 5.8mg/dL with a direct bilirubin of 4.0mg/dL. A complete infectious, antibody and common drug toxicity workup was negative. Both Ultrasound Abdomen with Doppler and CT Abdomen was normal. The patient was treated with intravenous thiosulfate and a 24-hour N-Acetylcysteine transfusion regimen. Though there were some improvements clinically, her liver enzymes were still very high mandating transfer to a tertiary facility for possible liver transplantation. However, the patient continued to improve in the next couple of weeks with only supportive management and was discharged home with normal liver enzymes after almost a month of hospitalization. Official authorities continue to investigate the cause of poisoning although toxicity experts suggest a possibility of tetrachloroethylene, white phosphorous, or nitroaromatic exposure.

DISCUSSION: Common toxins associated with FHF include ecstasy, acetaminophen, anti-tuberculous drugs, NSAIDs, or Amanita mushroom poisonous. N-acetylcysteine can significantly improve the prognosis of acetaminophen intoxication however it has also been used in other conditions as a last resort like in our patient which showed a possible benefit. In FHF, the probability of spontaneous hepatic recovery can be predicted by the severity of encephalopathy, patient’s age, and the cause of FHF. This determines the urgency for liver transplantation which is the only proven therapy to improve survival in these patients.

S2474

A Case of Cholangioblastic Variant of Intrahepatic Cholangiocarcinoma
Hamsh C. Sairanesh, MD1, Samreed Al Yassin, MD2.
1Virginia Commonwealth University Health System, Lorton, VA; 2Virginia Commonwealth University Health System, Richmond, VA.

INTRODUCTION: Hepatic adenocarcinoma expressing inhibinβ can be misdiagnosed as neuroendocrine tumors. We describe a rare case of Cholangioblastic variant of intrahepatic cholangiocarcinoma.

CASE DESCRIPTION/METHODS: A 24 year old female patient who presented with worsening chronic abdominal pain and jaundice was recently noted to have asymptomatic increase in alkaline phosphatase. She denied any prior history of jaundice and presented her ED to be urgently to surgery. She was evaluated almost 4 years prior to presentation for similar symptoms and was told that she had liver mass (1.8 cm on liver Ultrasonography) at the time that need further evaluation. Unfortunately, evaluation was never completed. Patient denied a family history of gastrointestinal hepatic malignancy. Physical examination was pertinent for hepatomegaly as well as tenderness over right upper quadrant. Laboratory workup was pertinent for normocytic anemia along with elevated levels of ALT, AST, ALP and normal Bilirubin level. Computed Tomography of the abdomen/pelvis showed a large (18 cm in greatest dimension) enhancing mass with central hypodensity in the right hepatic lobe along with multiple additional heterogeneous masses at the inferior margin of right hepatic lobe. Magnetic resonance imaging showed large T2 hyperintense/T1 hypointense right hepatic mass (21.9 cm in greatest dimension) extending to medial segment of liver lobe of liver, showing diffusion restriction, arterial enhancement and relative hyperenhancement on delayed images, concerning for large hepatocellular carcinoma. Staging imaging of chest did not show any evidence of extrahepatic disease. Case was discussed in Multidisciplinary committee and plans for patient to undergo right extended hepatectomy. Patient underwent right extended hepatectomy. Resected tissue showed evidence of usual adenocarcinoma, with positive margins and vascular invasion. Staining was positive for CK7, CK5/6, Inhibin, synaptophysin and CK19. Tumor was negative for melan A, S100, desmin, actin, TTF1, CEA and CD 99. Tumor was deemed to be morphologically similar to previously described cholangioblastic variant of intrahepatic cholangiocarcinoma. Although Cholangiocarcinoma frequently occurs in males over age of 65, especially in the setting of advanced liver disease and biliary infection, Cholangioblastic variant of intrahepatic cholangiocarcinoma was first described in a case series of 3 young women. These tumors may mimic well-differentiated neuroendocrine tumors. Two of the 3 patients had recurrence disease causing death.

S2475

The Buck Stops Here: A Case of Deer Antler Extract-Induced Liver Injury
Gladya Meija, MD1, Mohammad T. Sultan, DO2, Faisal Siddiqui, DO1, Nyan Latt, MD1.
1Orincon Medical Center, New Orleans, LA; 2Oakview Clinic Foundation, New Orleans, LA.

INTRODUCTION: Liver injury secondary to prescription or over the counter drugs and supplements is an ongoing public health concern, especially with the growing marketplace of unregulated supplements. Drug induced liver injury (DILI) has been reported as the most common reason for regulatory actions instituted by the US FDA against medications and supplements. Deer antlers are the only mammalian organs that can entirely grow back once lost and their extract contains insulin-like growth factor. Many athletes admit using deer antler extract as a performance-enhancing supplement, but its safety is unknown. The following is the first reported case of a deer antler supplement induced liver injury.

CASE DESCRIPTION/METHODS: A 38 YO male presented with fatigue and elevated liver chemistry tests: ALT 360, ALT 1297, ALP 78 (U/L) and bilirubin 2.9 mg/dl. He was not on any medications but did note recent use of a supplement called “Braced Up” consisting of deer antler velvet extracts, ginseng, caffeine, taurine and Himalayan rock salt. He denied any concurrent supplement, herb, drug, or alcohol use, and noted improved symptoms following stopping the use of supplement 5 days prior to presentation. The patient underwent a thorough work-up which ruled out infectious causes such as viral hepatitis, CMV, HSV, EBV, biliary obstruction, genetic diseases and autoimmune hepatitis. Liver biopsy showed moderate acute lobular cholestasis. Patient was followed for total of 150 days after the liver injury. Figure 1 shows the peaks of ALP, T-bil, AST and ALT throughout the follow-up period. ALP, T-Bil and AST peaked around day 25 after the injury, while ALT and AST peaked at the first day and decreased to normal level at day 10. Another peak in ALT and AST was noted at around day 30. At day 50, all the labs started to return to the normal level. DILI resolved spontaneously without any treatment.

DISCUSSION: The updated RUCAM Causality Assessment was used to evaluate the probability of DILI secondary to the deer antler velvet blend. The R ratio was consistent with hepatocellular type injury. It showed a probable correlation with a 7-point score, which along with the patient’s clinical course confirms the diagnosis of DILI secondary to the supplement. This report represents the first case of a deer antler-based supplement leading to hepatotoxicity, highlighting the need for public awareness regarding the hepatotoxicity of deer antler extract containing supplements.