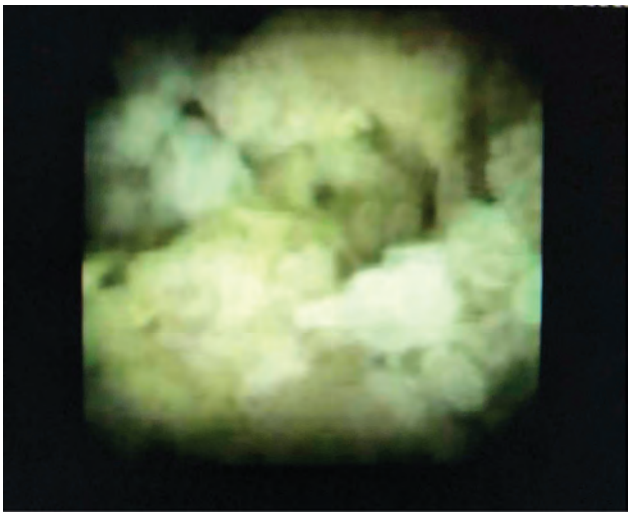

[1332C] Cholangioscopy image.

have been reported, acute pancreatitis has never been reported.To our knowledge, this is the first of probable primary Cephalexin-induced acute pancreatitis. A 55 years old female patient with past medical history significant for multiple sclerosis (MS), basal cell carcinoma (BCC), history laparoscopic cholecystectomy due to gallstones had excision of BCC done in dermatology clinic and received a dose of 500 mg Cephalexin for prophylaxis. Three hours later, she presented to emergency department (ED) for sudden onset of upper abdominal pain radiating to back. The pain was associated with severe nausea and poor appetite. She denied history of drinking alcohol, trauma, insect or scorpion bite and previous history of pancreatitis. Her medication list included Fingolimod (Gilenya) and Oxcabazepine both of which she was taking for years without any side effects. Abdominal examination revealed epigastric tenderness, with no rebound or palpable masses. Initial laboratory workup revealed lipase of 889 Units/L (6 times upper normal limit). However, CT abdomen in the ED was negative for peripancreatic fat stranding, fluid collection, and pancreatic focal lesions. Based on abdominal pain and elevated lipase three-time upper normal limit, the patient was diagnosed with acute pancreatitis. She was admitted and started on aggressive intravenous hydration. Further work up showed normal liver enzymes and serum triglyceride of 68 mg/dl. IgG subclasses were normal. Ultrasound showed unremarkable liver, absent gallbladder, and extra hepatic duct measured 10 mm. MRCP was obtained, which showed mild extrahepatic duct dilatation up to 11 mm without any dominant stricture or stone. Cephalexin was discontinued. Four days later her appetite improved and pain resolved and she was discharged home on low fat diet. In the absence of other causes of acute pancreatitis, cephalosporins such as Cephalexin should be considered as a potential etiologic factor of acute pancreatitis in patients who present with abdominal pain and elevated serum lipase levels.

1334

Portal Cavernoma Cholangiopathy Secondary to Polycythemia Vera: Case Report and Echoendoscopic Findings

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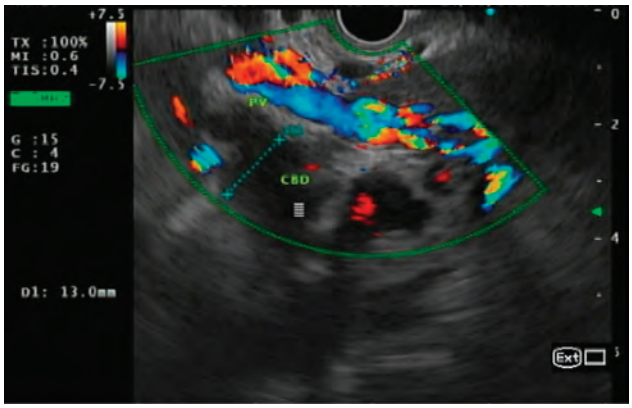
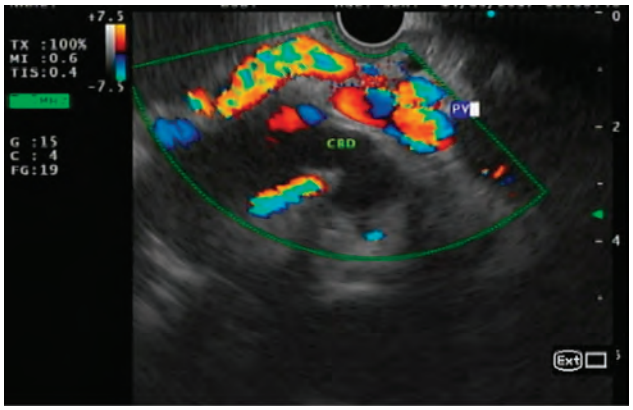
Portal cholangiopathy describes the abnormalities arising anywhere in the wall of biliary tree as a result of extrahepatic portal hypertension. This includes extra and intrahepatic bile duct narrowing and dilatation, and choledochal varices both favoring cholestasis and stone formation. The pathogen-



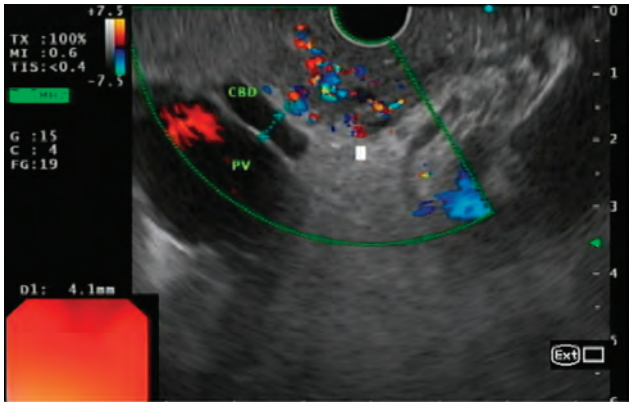
[1334A] EUS image showing dilatation of CBD (13mm) at the level of the hepatic hilum and the portal vein.

Study	Year	Age	Gender	Indication	Dose	Latency	Lipase	Amylase	Imaging	Other Findings	Improvement after holding Ceftriaxone
Ruggiero et al.	2010	2	Male	Fever, vomiting, diarrhea	700 mg daily	2 days	465 U/L	125 U/L	US: no GB stones	None	3 days
Sasaki et al.	2009	35	Female	Diverticulitis	2 g daily	12 days	Elevated	Elevated	CT for AP	Sludge in GB and CBD	Enzymes normalized 5 days after holding ceftriaxone
Famularo et al.	1999	74	Female	Root abcess	2 g daily	10 days	NR	NR	CT: AP and cholecystitis		
Maranan et al.	1998	13	Male	Right frontal subdural empyema secondary to sinusitis	2 g Q12H	5 weeks	3528 IU/L	1133 IU/L	US: Cholelithiasis	Pathological exam showed GB material of 100% ceftriaxone	1 month after cholecystectomy
Zimmermann et all	1993	71	Female	Gram negative sepsis	2 g daily	10 days	2400	413	CT: Enlarged Pancreas, no stones	Renal failure	2 days

[1333] Characteristics findings of published case reports of Cephalosporin induced pancreatitis.



[1334B] EUS image at the level of hepatic hilum showing portal vein thrombosis with para and peri-choledochal varices.



[1334C] EUS image showing the distal portion of CBD with a normal diameter (4mm) and intrapancreatic venous collaterals.

esis implies a reversible component, the compression of collaterals, and a fixed component, consequence of ischemia at the time of portal vein thrombosis. This can be associated with hematological and coagulation disorders such as myelodysplastic syndromes or protein C deficiency. Endoscopic ultrasound (EUS) is considered the modality of choice for evaluating common bile duct obstruction, however its role in evaluating collateral vascular pathways in and around the common bile duct is poorly defined. We present a case of a 67-year-old woman with polycythemia vera diagnosed 19 years before, that culminated in thrombosis of portal, superior mesenteric and splenic veins. She had a previous history of cholecystectomy for gallstones. During the last decade, she developed various episodes of esophageal variceal hemorrhage. A right quadrant biliary-type pain associated with persistent elevation of alkaline phosphatase was also reported. She underwent Magnetic Resonance Cholangiopancreatography (MRCP) that showed exuberant cavernomatous portal vein transformation by previous portal thrombosis and significant stenosis at the confluence of the right and left hepatic ducts, with upstream ectasia. Due to edema and unspecific alterations at the hepatic hilum, it was not possible to exclude suspicious lesions. EUS was then performed, which showed a serpiginous trajectory of the common bile duct (CBD) from the hepatic hilum to its supra-pancreatic portion. Large para and peri-choledochal varices were observed, as well as intrapancreatic venous collaterals. This extensive network of neo vessels was responsible for the compression of the biliary tree and its upstream dilatation. EUS excluded other etiologies for CBD dilatation. This case illustrates EUS potential in the algorithm of management of portal colangiopathy, particular in symptomatic cases. Its imaging resolution (associated with Doppler) can trace the origin, caliber, entry and course of varices outside and throughout the CBD. The careful study of collateral vessels prior to therapeutic interventions (as ERCP) can help to plan the procedure and manage anticipated complications such as hemobilia.

1335

Splenic Artery Pseudoaneurysm (SAP): A Case Report

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A 58 year old female presented to our group for work up of abdominal pain and pancreatic mass revealed on a previously obtained abdominal CT scan. She had past medical history significant for type 2 diabetes, arthritis, and hypertension. She had no history of abdominal surgeries, and specifically denied history of alcohol use. Physical exam revealed a soft abdomen and no hepatosplenomegaly. Her laboratory studies including a CA 19-9 were unremarkable. EGD with endoscopic ultrasound (EUS) was performed and a 16 mm x 17 mm round, well-defined, hypoechoic exophytic lesion in the posterior pancreatic head (not involving the PD) was noted. This lesion was abutting the major veins at the portal confluence, however there was no evidence of invasion into the vessels. Elastographic EUS showed a predominantly solid mass. Based on the EUS, the differential diagnosis included neuroendocrine tumor and rare lymphoepithelial keratinized cystic lesion. Pathology from FNA biopsy reported scant pancreatic tissue insufficient for a definitive diagnosis. Ultrasonography of the abdomen showed a solid hypodense heterogeneous mass. A CT with contrast confirmed the presence of a 2.0 x 2.2 x 1.7 cm rounded space occupying mass lesion in the pancreatic head. Finally an MRI with contrast showed that the mass was medial to the portal confluence abutting the posterior aspect of the pancreatic head. The findings were suggestive of a splenic artery pseudoaneurysm that might be clotted off. Discussion:- Splenic artery pseudoaneurysm (SAP) is an uncommon finding, with only 157 cases being reported in English-language literature. 77.3% of cases were found in males. Mayo clinic reported 10 cases over 18 years (1980 to 1998). The most common causes of SAP include chronic pancreatitis (46%), abdominal trauma (29%), iatrogenic and postoperative complications (3%), and peptic ulcer disease (2%). Following pancreatitis, SAP can be caused by the leakage of pancreatic enzymes, which eventually leads to necrotizing arteritis and subsequent vessel wall destruction. Pancreatic pseudocyts can occur in conjunction with SAP 41% of cases. The most common symptoms associated with SAP are hematochezia or melena at 26%, hematemesis at 16%, abdominal pain at 29% as well as hemosuccus pancreaticus at 20%. Diagnosis is most commonly made with angiography (52%) followed by CT scan (36%). Since the risk of rupture is high (37%), urgent repair of SAP's is paramount regardless of the size of the SAP.

1336

UnMASking the Diagnosis: Idiopathic Non-Cirrhotic Portal Hypertension Diagnosed in the Setting of Macrophage Activation Syndrome Complicated by Severe Pancreatitis

2017 Presidential Poster Award

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Systemic Lupus Erythematosus (SLE) is a common rheumatologic condition with known GI involvement, most notably autoimmune hepatitis and lupus colitis. Acute pancreatitis (AP) is a rare complication of SLE and is typically associated with disease activity. Macrophage activation syndrome (MAS) is an unusual, hyper-inflammatory response to a rheumatologic stimulus characterized by hyperferritinemia, pancytopenia, thermal dysregulation and multiorgan dysfunction. MAS, more commonly seen in children, has been reported to complicate both adult onset SLE and AP. We present a case of necrotizing AP secondary to an SLE flare ultimately complicated by MAS in an adult patient and successfully treated with Anakinra. A 29 year-old female with SLE and non-adherence to medical therapy was admitted with severe necrotizing AP evidenced by epigastric pain, nausea, oral intolerance, lipase >3000 U/L, and CT scan demonstrating necrosis and inflammation in the pancreatic head. After admission, she developed tachycardia, hypothermia, lactate elevation of 17.2, and hypercarbic respiratory failure with pulmonary edema requiring intubation and mechanical ventilation with aggressive supportive care. Laboratory evaluation demonstrated evidence of DIC with pancytopenia, elevated ANA/ds DNA, and a ferritin of 1332 ng/mL. She was started on methylprednisolone for treatment of a SLE flare with clinical improvement leading to extubation. Following extubation, her ferritin level doubled in association with clinical deterioration raising suspicion for MAS. She was then started on Anakinra resulting in brisk clinical response and down trending ferritin. AP occurs in 1-8% of SLE patients and is proposed to be the result of vascular damage. MAS complicates approximately 5.5% of childhood SLE compared to 0.7% of adult SLE but is more commonly the result of AP in combination with SLE compared to SLE alone and is associated with higher mortality rates. Our case uniquely illustrates a rare etiology of AP and the relationship between systemic diseases and the GI system in a critically ill patient. Treatment of MAS is aimed at treating the underlying SLE; however, our patient required the addition of a chemotherapeutic agent. Anakinra is a novel agent which has been used to treat refractory childhood MAS and was successful in treating this adult patient. To the best of our knowledge, this is the only reported case of refractory MAS due to SLE induced AP necessitating treatment with Anakinra.

1337

Gallbladder Adenocarcinoma as the First Manifestation of Germline BRCA1 Mutation

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Gallbladder adenocarcinoma (GBAC) is an uncommon but highly fatal malignancy. It follows an aggressive course and complete remission is rare. Germline BRCA1 mutations have been linked with an increased risk of several abdominal malignancies, most prominently pancreatic cancer and more recently hepatobiliary malignancies. A 47-year-old female with history of gastroesophageal reflux disease presented to the clinic with a 2-month history of abdominal discomfort. Family history was unknown as she had no contact with her family. Exam was notable for scleral icterus and right upper quadrant abdominal tenderness. Labs were notable for conjugated hyperbilirubinemia. Computed tomography of the abdomen revealed a 6x5-centimeter hypodense left hepatic lobe mass and regional lymphadenopathy (figure 1). CA 19-9 was elevated at 214 U/mL. Endoscopic retrograde cholangiopancreatography with papillotomy was performed and aspiration biopsy of the peripancreatic lymph nodes revealed adenocarcinoma. She then underwent exploratory laparotomy with Whipple's procedure and biopsy of the hepatic lesion revealed GBAC (figure 2). Adjuvant chemotherapy with gemcitabine and cisplatin was not tolerated due to side effects. Three years later, she was diagnosed with right-sided breast cancer for which she underwent surgical resection. Given the personal history of two malignancies diagnosed under the age of 50 and the limited family history, genetic testing was pursued and patient tested positive for BRCA1 mutation (c.5177_5180delGAAA (p.Arg1726LysfsX3)). She later underwent prophylactic hysterectomy and bilateral salpingo-oophorectomy. Association of hepatobiliary tumors with BRCA1 mutations has been described in several population studies. However, GBAC in the setting of BRCA1 mutation has been previously described in only one case report. Unique to our patient with germline BRCA1 mutation is the diagnosis of GBAC as the patient's first malignancy. Therefore, germline BRCA1 mutations need to be considered in patients with GBAC and atypical features such young age and absence of risk factors. Referral to a geneticist is appropriate in such cases as this can reduce future risk of other malignancies by genetic counseling, enhanced screening and prophylactic surgeries.