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 sub-classes 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-
A 58 year old female presented to our group for work up of abdominal pain and pancreatic mass revealed at ERCP. 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 is paramount regardless of the size of the SAP.

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 atypical course with young age and absence of risk factors. Referral to a geneticist is appropriate in such cases.

The most common symptoms associated with SAP are hematochezia or melena at 26%, hematemesis 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 is paramount regardless of the size of the SAP.

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. Her past medical history significant for type 2 diabetes, arthritis, and hypertension. She had no history of abdominal surgeries, and specifically denied his 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, hyperechoic exophytic lesion in the posterior pancreatic head (not involving the PD) was noted. This lesion was abutting the major vena at the level of splenic hilum, however there was no evidence of invasion into the vessels. Elasticographic EUS showed a predominantly solid mass. Based on the EUS, the differential diagnosis included neuroendocrine tumor and rare lymphoepithelial hilar lymphoma from FNAB biopsy reported as pancreatic cystic lesion 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 2.1 x 1.7 cm rounded space occupying mass lesion in the pancreatic head. Finally an MRE 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 pancreatic masses (49%), abdominal trauma (38%), iatrogenic and postoperative complications (3%), and peptic ulcer disease (2%). Following pancreaticitis, SAP can be caused by the leakage of pancreatic enzymes, which eventually leads to necrotizing arterial and subsequent vessel wall destruction. Pancreatic pseudoaneurysm can occur in conjunction with SAP in 41% of cases. The most common symptoms associated with SAP are hepatomegaly or melena at 26%, hematemesis at 16%, abdominal pain at 29% as well as hemorrhagic pancreatitis 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 is paramount regardless of the size of the SAP.