DISCUSSION: Younger patients, patients receiving a prolonged or larger dose and patients with impaired gallbladder emptying are at a greater risk for developing sludge secondary to ceftriaxone use. This is often asymptomatic with discontinuation of the drug usually resulting in resolution of this phenomenon. This case highlights the fact that ceftriaxone induced biliary sludge may rarely result in potentially serious adverse events including acute cholecystitis and pancreatitis.

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Duodenobiliary Fistula: A Rare Complication of Peptic Ulcer Disease
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INTRODUCTION: Spontaneous biliary enteric fistulas are usually late complications of cholelithiasis or choledocholithiasis. The incidence of biliary enteric fistulas in this population ranges from 0.3 – 0.9%. Biliary enteric fistula formation is usually occurs from a gallstone ulcer perforating into the normal duodenum. However in rare circumstances (3.5-10% of cases) fistula’s can form from a duodenal ulcer penetrating into the biliary tree, as is the case with biliary enteric fistula’s due to peptic ulcer disease (PUD). These fistula’s often present with abdominal pain, malaise and/or hematemesis. We discuss a rare case of a duodenobiliary fistula as a result of long standing peptic ulcer disease.

CASE DESCRIPTION/METHODS: A 57-year-old male with long standing abdominal pain who recently immigrated to the United States from the Democratic Republic of Congo presented to the ED with acute on chronic epigastric abdominal pain. He denied previous surgery. Notably, he had tested positive for H. pylori and was started on antibiotics several months prior. Despite treatment, he continued to have symptoms. He was admitted with hypokalemia and renal failure. Persistent symptoms prompted a computed tomography (CT) of his abdomen and pelvis. Findings from the CT demonstrated a mass effect on the distal common bile duct with material within the common bile duct and intrahepatic bile ducts with a fistulous connection to the second portion of the duodenum. Endoscopic evaluation demonstrated esophagitis, gastritis and severe pyloric stenosis likely due to long-standing PUD. The standard endoscope was unable to traverse this stenosis, but was successfully traversed with an ultrathin scope. The ampulla was briefly visualized and was grossly normal. There was no definitive fistulous tract identified. The patient was placed upon high dose proton pump inhibitor therapy. A repeat CT of the abdomen and pelvis was done 6 weeks later and was unchanged. The patient was referred to surgical oncology for definitive management.

DISCUSSION: Historically, biliary enteric fistulas were often diagnosed during surgical interventions. Currently, CT can often diagnose these fistulas by detecting minimal pneumobilia. Endoscopic evaluation may or may not demonstrate a biliary enteric fistula due to duodenal narrowing or edema. Antibiotic therapy usually allows clinical improvement and fistula closure for PUD associated fistulas. Surgery is reserved for cases refractory to medical care, poorly controlled symptoms, hemorrhage or biliary obstruction.

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Abrupt Onset of Acute Pancreatitis Following the Ingestion of Eluxadoline
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INTRODUCTION: Acute pancreatitis is a common condition in the US, with high mortality and burden on the health care system. In the US, it is one of the main causes of gastrointestinal related admissions with more than 291,000 hospitalization per year. The most common causes are alcohol use (30%) and gallstones (40%). Less frequently, drug induced. Interestingly, drug induced pancreatitis has excellent prognosis and low mortality. The mechanisms of drug induced pancreatitis require the diagnosis of drug induced pancreatitis requires first the diagnosis of acute pancreatitis based on symptoms, biomarkers or imaging. Common causes, such as alcohol use and gallstones, must be ruled out. A medication review should be conducted to look for potential culprits. Any medication of suspect should be discontinued and resolution of pancreatitis should be appreciated. Eluxadoline is used in treatment of IBS-D. It reduces abdominal pain and lessens diarrhea frequency, but has been noted to be associated with acute pancreatitis in 82% of patients who has previously underwent cholecystectomy. It is thought to be due to spams in the sphincter of oddi, leading to pancreatic duct hypertension. The FDA has suggested to avoid the use of this medication in those cases. Careful attention should be made to the potential adverse events, especially acute pancreatitis, in those patients on Eluxadoline.

CASE DESCRIPTION/METHODS: A 78-year-old woman with surgical history of cholecystectomy presented to the emergency room with a sudden onset of sharp epigastric pain radiating to the back, that was associated with vomiting, 60 minutes after taking Eluxadoline for the first time. She had a history of hypertension, diabetic mellitus, and irritable bowel syndrome with diarrhea (IBS-D) for which Eluxadoline was prescribed. On exam, she was hemodynamically stable and had epigastric tenderness without rebound. Laboratory tests remarkable for elevated lipase, 2754 U/L. Based on the Revised Atlanta criteria, patient had mild pancreatitis. A computed tomography of the abdomen and pelvis was unremarkable without biliary pathology. She was admitted for acute pancreatitis presumed secondary to Eluxadoline ingestion. The patient was treated conservatively and Eluxadoline was discontinued. Five hours later, her lipase was 68 U/L and symptoms resolved. She was advised to discontinue Eluxadole and discharged home.

DISCUSSION: The diagnosis of drug induced pancreatitis requires first the diagnosis of acute pancreatitis based on symptoms, biomarkers or imaging. Common causes, such as alcohol use and gallstones, must be ruled out. A medication review should be conducted to look for potential culprits. Any medication of suspect should be discontinued and resolution of pancreatitis should be appreciated. Eluxadoline is used in treatment of IBS-D. It reduces abdominal pain and lessens diarrhea frequency, but has been noted to be associated with acute pancreatitis in 82% of patients who has previously underwent cholecystectomy. It is thought to be due to spams in the sphincter of oddi, leading to pancreatic duct hypertension. The FDA has suggested to avoid the use of this medication in those cases. Careful attention should be made to the potential adverse events, especially acute pancreatitis, in those patients on Eluxadoline.

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Liver Abscesses as a Complication of Side-to-Side Choledochoduodenostomy
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INTRODUCTION: Side-to-side Choledochoduodenostomy (CDD) is a low morbidity surgical procedure commonly performed in the pre-Endoscopic Retrograde Cholangiopancreatography (ERCP) era to provide permanent relief and drainage of the common bile duct (CBD) in patients with retained or recurrent CBD stones, ampullary stenosis or stricture of the CBD. In side-to-side CDD, the distal bile duct becomes a poorly drained reservoir or “sump” as bile is unable to be drained from the area. Bile, stones, or debris may accumulate and lead to inflammation and bacterial growth. In many cases the inflammation in the sump can cause biliary obstruction, recurrent cholangitis, or pancreatitis, incurring the name “sump syndrome”. Although it is rare complication, sump syndrome carries a high mortality. In addition, hepatic abscesses have been reported as a complication.

CASE DESCRIPTION/METHODS: A 63-year-old man, with a surgical history of cholecystectomy 15 years ago, presented with acute epigastric pain and severe sepsis. Laboratory studies suggested a biliary source of infection. A computed tomography showed multiple liver abscesses and peritonitis. Urgent percutaneous biliary drainage with CBD stent was performed. Cholangiogram demonstrated side-to-side CDD with residual debris in the sump. ERCP revealed choledolithiasis. Sphincterotomy and balloon extraction was performed to remove residual stones and debris. Distal biliary structure was found and biliary stent was placed. The presentation of biliary disease complicated by liver abscess secondary to retrograde intrahepatic flow and history of side-to-side CDD with typical ERCP findings, established the diagnosis of sump syndrome.
DISCUSSION: Hepatic abscesses have an incidence of 3.6 per 100,000 in the US. The majority of these cases are secondary to biliary tree disease, with pyogenic abscesses being the most common. There is limited data regarding abscesses as a late complication of sump syndrome, with few cases reported in the literature. This complication may be due to an obstruction between proximal CBD to duodenum, which leads to regurgitation of bile into intrahepatic bile ducts. Although the incidence of those abscesses is low, mortality remains as high as 12%. It is important to recognize the underlying cause of hepatic abscesses, as timely drainage and antibiotic therapy are essential to prevent significant morbidity. Furthermore, the restoration of normal bile flow from CBD via an ERCP is needed to eliminate any culprit source of infection and reduce recurrence.

INTRODUCTION: EBV infection is mostly subclinical and resolves spontaneously without complications. GI involvement manifests mainly with asymptomatic liver enzyme elevation. We report a case of acute pancreatitis complicating EBV infection.

CASE DESCRIPTION/METHODS: An 18-year-old woman presented with a 12-day history of moderate unremitting upper abdominal pain, nausea, and anorexia. She had a temperature of 36.8°C, bilateral non-tender cervical lymphadenopathy, mild pharyngeal injection, and moderate epigastric tenderness, but no splenomegaly or jaundice. She didn’t take any medications and denied alcohol/illicit drug use. Leukocyte count was 14.5 cells/μL (15% neutrophils, 9% monocytes, 44% reactive lymphocytes), hemoglobin 12.7 g/dL, amylase 327 IU/L, lipase 2016 U/L, alkaline phosphatase 246 IU/L, aspartate aminotransferase 210 IU/L, alanine aminotransferase 283 IU/L, total/direct bilirubin 2/1.6 mg/dL. Calcium, blood glucose, and triglycerides were normal. Ultrasound showed mildly enlarged liver with coarse echotexture, contracted gallbladder, normal common bile duct, and mildly enlarged right upper quadrant lymph nodes, but no intrahepatic biliary ductal dilation. Computed tomography showed acute pancreatitis and mild splenomegaly. Anti-HAV IgM; HBsAg and anti-HBV core IgM; anti-HCV; HIV1 p24 antigen; anti-HIV1 and anti-HIV2 antibodies (AB); and CMV (quantitative PCR) were negative. EBV heterophile AB was positive, EBV IgG viral capsid AB 78.6 U/mL, EBV IgM viral capsid AB 160 U/mL, and EBV IgG early disease AB 70 U/mL. She was treated conservatively and discharged on the fourth day of hospitalization.

DISCUSSION: Clinical pancreatitis is rarely associated with EBV infection; only 17 cases have been previously reported. In our case, most patients were adolescents/young adults, have clear EBV infection picture, and have mild to moderate pancreatitis course with spontaneous recovery. Although acyclovir was tried, it isn’t clear if it’s required. Our patient was unique in that her hospital stay was 4 days in comparison to a mean of 10 days in other reported cases. EBV infection should be considered in the differential diagnosis of mild to moderate pancreatitis. It is not clear if subclinical pancreatitis in the setting of EBV infection is more common and why only few patients develop clinical pancreatitis.

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