A rare cause of biliary obstruction and pancreatitis

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The prevalence of duodenal diverticula varies considerably in the literature, between 3-24% [1,2]. They are acquired extraluminal outpouchings of the mucosal wall through the muscularis propria and their incidence increases with age. While most duodenal diverticula do not cause major symptoms, they are increasingly recognized as causes of pancreaticobiliary disease. Food bezoar in a juxtapapillary duodenal diverticulum (JPDD) causing pancreaticobiliary obstruction is rare, with the first of such case reported in 1977 [3]. We present a case of food bezoar in JPDD causing pancreatitis, severe pancreaticobiliary duct obstruction and dilation--mimicking as neoplasm-- that was successfully diagnosed and treated endoscopically.

A 60-year-old female presented with two months of right upper quadrant abdominal (RUQ) pain radiating to the back with associated jaundice, nausea, vomiting, pruritus and clay colored stools. Physical exam was significant for jaundice, epigastric and RUQ tenderness without guarding. Vital signs were normal and afebrile. Laboratory testing revealed leukocyte count of 8,800 mm$^3$, lipase of 1085 U/L, alanine aminotransferase 164 IU/L, total/direct bilirubin of 10.8/8.9 mg/dL and alkaline phosphatase of 523 IU/L. Abdominal ultrasound showed common bile duct (CBD) dilation to 17 mm and was without choledocholithiasis. CT of abdomen showed a 3.8 cm pancreatic head mass causing significant biliary ductal dilatation as well as pancreatic ductal dilatation (Fig. 1). Esophagogastroduodenoscopy (EGD) revealed large periampullary diverticula with impacted bezoar that was disimpacted with flushes and Roth Net (Fig. 2). Endoscopic ultrasound performed post disimpaction demonstrated aforementioned ductal dilatation on CT and diverticula, but without pancreatic head mass, choledocholithiasis, or pancreatic duct stones. The patient’s symptoms and obstructive liver associated enzymes improved to normal levels over the next two months. Patient did not have recurrence of symptoms and laboratory findings also did not show evidence of hepatobiliary obstruction at one year follow up.

Diagnosis of JPDD can be suggested by upper gastrointestinal barium examinations visualized as collections of gas and barium in round or oval sack-like protrusions that usually arise from the medial aspect of the periampullary duodenum [4]. Direct visualization of the extraluminal outpouchings of the duodenum mucosal wall with EGD provides definitive diagnosis.

There is no consensus about the optimal treatment modality. Surgical procedures are available and are considered in diverticula with complications, but postoperative complications are not uncommon and carry a considerable mortality rate [5,6]. Complications of untreated duodenal diverticula, in addition to pancreatitis, biliary obstruction, cholangitis; can also include ulceration, hemorrhage, diverticulitis, and perforation with fistula and abscess formation [5]. Studies of endoscopic treatments of symptomatic duodenal diverticula are lacking. In our patient, the obstruction and symptoms were successfully relieved through endoscopic removal of the impacted material. There were no immediate complications observed in this case. Although anecdotal, this case suggests endoscopy as a safe and effective alternative treatment modality to impacted JPDD. Furthermore, because the clinical and radiological findings associated with an impacted JPDD can be very similar to pancreatic neoplasm, cysts or abscess; therefore, it is important to consider impacted juxtapapillary duodenal diverticulum in the differential diagnosis in patients presenting with obstructive jaundice and pancreatic head mass.

References


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