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Case Report

Periampullary Duodenal Diverticulitis – Another Cause for Acute Pancreatitis

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Abstract

Small bowel diverticula are uncommon and usually asymptomatic. Rare complications include abdominal pain, small bowel diverticulitis, small bowel perforation, pancreaticobiliary obstruction, and related sequelae (i.e., ascending cholangitis, pancreatitis). We present a case of a 42-year-old female with peri-ampullary duodenal diverticulitis complicated by pancreaticobiliary obstruction successfully managed conservatively. Small bowel diverticula are often found in the second portion of the duodenum adjacent to the ampulla of Vater and are usually asymptomatic but can predispose to non-pancreaticobiliary or pancreaticobiliary complications. This case highlights the success of conservative management with antibiotics and endoscopic lavage upon prompt recognition and treatment avoiding invasive measures.

Keywords: Diverticulum; Duodenum; Diverticulitis; Pancreatitis; Pancreaticobiliary

Introduction

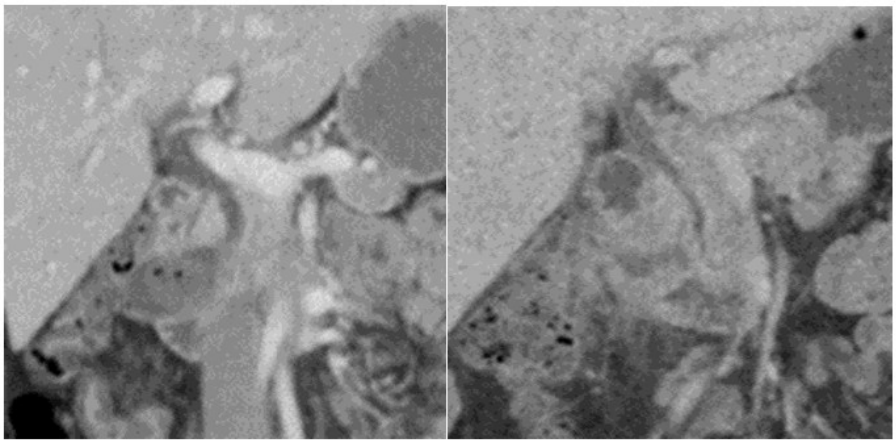
Small bowel diverticula are uncommon occurring in 0.3-1.3% of post-mortem cases with the duodenum being the most likely site, and are usually asymptomatic [1,2]. However, although rare, complications include abdominal pain, small bowel diverticulitis, small bowel perforation, pancreaticobiliary obstruction, and related sequela (i.e. ascending cholangitis, pancreatitis) [2]. When complications arise, clinical suspicion and early diagnosis are crucial as these patients can often suffer from a delay in diagnosis and treatment. Here, we present a case of a 42-year-old female with peri-ampullary duodenal diverticulitis complicated by pancreaticobiliary obstruction successfully managed conservatively.

Case Presentation

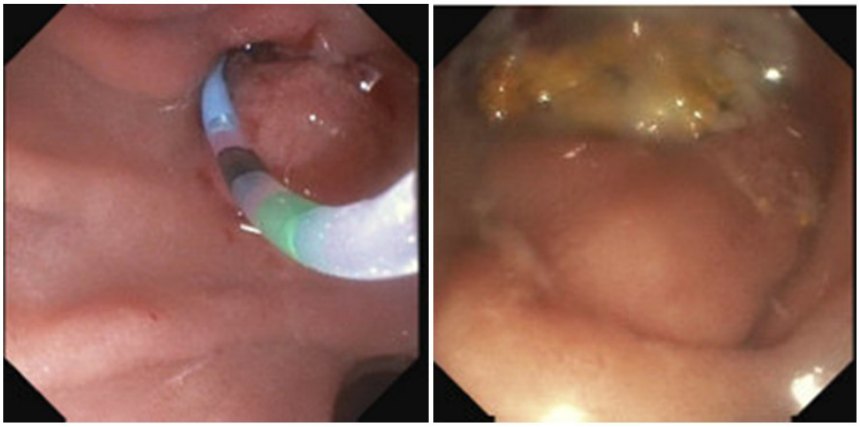
A 42-year-old female with a past medical history of hypertension, hyperlipidemia, migraines, pleomorphic adenoma of the right salivary gland, herpes labialis, and prior laparoscopic cholecystectomy presented with acute onset of nausea and bilious vomiting ongoing for 4 days accompanied by severe epigastric abdominal pain. She complained of associated poor oral intake, loose stools, fevers, and chills. On initial presentation, she was noted to be tachycardic but vital signs were otherwise unremarkable. The patient had no prior history of heavy alcohol, tobacco, or illicit drug use. On physical examination, the patient had diffuse tenderness to abdominal palpation with normal active bowel sounds. Labs were significant for leukocytosis, hypokalemia, and elevations to serum liver chemistries: alkaline phosphatase 159, ALT 318, AST 347. Hepatitis panel was negative and lipase was within normal limits. Computerized tomography

(CT) abdomen/pelvis (AP) showed a prominent duodenal diverticulum in the second portion of the duodenum, measuring 3.8 x 2.3 x 2.5 cm, and containing particulate material. The extrahepatic common bile duct was mildly prominent at 8 mm, and the diverticulum was noted to exert some mass effect on the adjacent ampulla of Vater, although the degree of dilatation was also within normal limits given prior cholecystectomy. Given these findings, gastroenterology was consulted. Magnetic resonance cholangiopancreatography (MRCP) showed redemonstration of a relatively large duodenal diverticulum emanating from the second portion of the duodenum along with generalized body wall edema and small bilateral pleural effusions. Endoscopy with ultrasound excluded pancreatic pathology and bile duct stones and confirmed a duodenal diverticulum impacted with solid debris. Endoscopic ultrasound (EUS) revealed edematous bulging of the medial duodenal wall suspicious for diverticulum with impacted solid debris.

Partial disimpaction with the tip of a sphincterotome revealed purulent drainage which was aggressively lavaged with sterile saline (Figures 1 and 2). Common bile and cystic ducts were dilated to 9mm and 6mm, respectively as seen on EUS. Multiple biopsies were taken from the margins to differentiate duodenal diverticulitis from a cavitary mass lesion. Endoscopic retrograde cholangiopancreatography (ERCP) was initially planned but was deferred due to features of duodenal diverticulitis. Post-procedure, the patient was treated with intravenous (IV) Levofloxacin and Metronidazole for one week. Day one post-procedure, the patient developed sharp epigastric pain radiating to the back. Vital signs remained stable; however, lipase was elevated to 1689, with elevated serum liver chemistries: alkaline phosphatase 313, ALT 212, AST 225. The patient was treated symptomatically for acute pancreatitis with IV fluids, pain control, and diet restriction. A repeat CT-AP was obtained showing new thickening of the wall of the diverticulum, with mild mucosal enhancement and adjacent inflammatory change, suggestive of mild inflammation/diverticulitis. Given these findings, endoscopy with lavage was repeated after 3 days showing significant improvement (Figure 3 and 4). A repeat CT scan at one week showed collapse of the diverticulum and improvement in inflammatory changes (Figure 5). The patient was discharged with oral antibiotics. At 6 month follow-up, she was asymptomatic with normal liver enzymes.



Figures 1 and 2: EUS.



Figures 3 and 4: EGD with lavage.

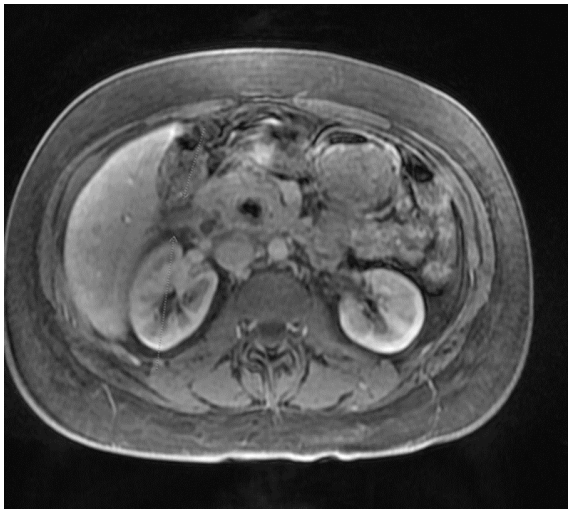


Figure 5: CT-abdomen/pelvis with contrast.

Discussion

Small bowel diverticula are of two types, congenital or acquired/false diverticula [3,4]. Congenital diverticula are exclusively composed of Meckel's diverticulum while false diverticula are lesions consisting of mucosa, submucosa, and serosa without a tunica muscularis [3,4]. Duodenal diverticula are most often found in the second portion of the duodenum adjacent to the ampulla of Vater [4]. These diverticula are pseudo-diverticula consisting of outpouchings of mucosa that lack a muscularis layer [5]. Amongst the different types of duodenal diverticula, periampullary duodenal diverticula are the most common [5]. Although the majority of periampullary diverticula are asymptomatic, occasionally non-pancreaticobiliary or pancreaticobiliary complications can occur [5]. Non-pancreaticobiliary complications are rare and may include diverticulitis, hemorrhage, perforation, or fistula formation [5]. Pancreaticobiliary complications can present as recurrent gallbladder or bile duct stones, obstructive jaundice, cholangitis, or acute pancreatitis [5]. Duodenal diverticulitis can mimic a wide range of intra-abdominal pathologies. Occasionally, an impacted diverticulum can be infected causing diverticulitis such as the case presented, and at times, even perforation [6].

The causation of periampullary diverticulitis and associated pancreatitis does not have a clearly defined relationship. Pancreatitis may be due to the mechanical pressure from diverticula and diverticular inflammation causing narrowing at the duodenal papilla [7].

It has been proposed that in such cases, there is dysfunction in the sphincter of Oddi, which in turn causes reflux of pancreatic fluid and intestinal content [8, 9]. In addition, biliary and pancreatic

complications may occur as a complication of diverticula stasis [10]. Another theory argues that diverticula cause spasm of the sphincter and increases biliary tract pressure [5].

CT is the imaging modality of choice for diagnosing duodenal diverticula [11]. Diagnosis can also be reached via barium studies, ultrasound, and MRI [11]. MRCP can be used to exclude associated biliary and pancreatic complications [11]. Treatment options for patients presenting with duodenal diverticula ~~is~~ are guided by the clinical presentation. Asymptomatic patients do not require treatment. Patients presenting with symptoms of diverticulitis should be treated with broad-spectrum antibiotics as initial therapy [6]. If patients fail or have equivocal results with initial therapy, debridement, irrigation with normal saline, and stent placement can be achieved endoscopically for symptomatic relief [6]. If conservative therapy fails or there is concern for perforation, surgery with diverticulectomy should be considered [6].

We speculate duodenal diverticulitis to be the cause of pancreatic inflammation in our case. However, we recognize that the patient underwent instrumentation via ERCP which may have also contributed to the development of pancreatitis.

Conclusions

As such, it is critical to have clinical suspicion when concerned about this condition to make the diagnosis in a timely manner and avoid delays in treatment. Our case highlights the role of conservative management with antibiotics and endoscopic lavage in the treatment of duodenal diverticula and stresses the importance of prompt recognition of this diagnosis, which may help avoid invasive measures.

Disclosures

The authors have nothing to disclose.

Conflicts of Interest/Financial Support

There were no competing conflicts of interest including financial, consultant, institutional or otherwise. In addition, no funding was involved in the completion of this case report.

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