Small Bowel Obstruction Secondary to Acute Pancreatitis

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Abstract

While an uncommon occurrence, it is possible for patients diagnosed with acute pancreatitis to develop colonic ileus, obstruction, or perforation. By extension, it is also possible to develop a small bowel obstruction following an episode of acute pancreatitis. Here, we present the case of a 44-year-old male, who after repeated attacks of acute pancreatitis, came to the emergency department with continuous, non-bloody vomiting. This patient also complained of both left upper quadrant and epigastric pain, and was subsequently diagnosed with a small bowel obstruction involving the proximal jejunum.

Keywords: Bowel obstruction; Colon obstruction; Ileus; Pancreatitis

Introduction

In rare event acute pancreatitis leads to colonic obstructions, and it most often does so in the locations of the splenic flexure and transverse colon [1]. Small bowel obstructions following an episode of acute pancreatitis are even less documented in the literature, and tend to be the result of retroperitoneal inflammation. The small bowel is susceptible to the inflammation because of its proximity to the anterior surface of the pancreas. An enzyme-rich extravasation product, released in response to the inflammatory process, travels to the colon and small bowels, mechanically obstructing bowel pathways [2].

Case Report

A 44-year-old man with medical history of alcohol dependence with recurrent attacks of acute pancreatitis came to the emergency department complaining of persistent non-bloody, greenish vomiting associated with epigastric and left upper quadrant pain for 3 days. Patient admitted to drinking alcohol daily for about 20 years. Patient had pertinent history of an episode of alcohol-induced acute pancreatitis 10 days prior to this index admission with amylase and lipase levels of 91 and 189 U/L, respectively. Patient was hemodynamically stable at the time of admission. Physical exam was consistent with epigastric and left upper quadrant tenderness without guarding or rigidity. Laboratory studies revealed a hematocrit of 43%, leukocyte count of 10.3 × 10^9/L (71% neutrophils), lipase of 163 U/L, amylase of 90 U/L and creatinine of 2.7 mg/dL (baseline of 0.5 mg/dL). Ultrasound (US) of abdomen did not reveal gallstones but showed mild hepatomegaly and hepatic steatosis. Computerized tomography (CT) scan of abdomen revealed changes in pancreas consistent with acute pancreatitis along with distended stomach and duodenum raising concern for obstruction at the proximal jejunum (Figs. 1 and 2) from surrounding edema/inflammation. Patient was admitted with an impression of alcohol-induced severe acute pancreatitis with acute kidney injury and small bowel obstruction.

Figure 1. CT of abdomen coronal section showing severely dilated duodenum (white arrow) and peri-pancreatic fat stranding (yellow arrow).

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The management is directed towards treating the underlying pathophysiological mechanisms and management when compared to colonic complications.

Other obstructive manifestations of acute pancreatitis include colonic stenosis which may present as a “pseudocarcinoma” with classic “apple-core” appearance on imaging a few months following an episode of acute pancreatitis [1, 13]. This complication has been most frequently described at the splenic flexure and, like mechanical obstruction, may be the result of the intimate anatomic relationship shared between the pancreatic tail and splenic flexure. The pancreatic tail lies in the phrenocolic and phrenocolic ligament, which is contiguous with the splenic flexure of the colon. This shared communication facilitates direct extension of inflammatory enzyme-rich material to the splenic flexure with progressive strictureting of the colon segment [14, 15]. Additionally, the splenic flexure is a watershed area often supplied by a poorly developed marginal artery making this area more vulnerable to ischemic insult [1].

Similar anatomic relationship exists between the anterior surface of the pancreas and transverse colon where the two layers of the transverse mesocolon cover the head and body of the pancreas. Thus, enzyme-rich inflammatory extravasation can easily access the transverse colon leading to local complications (including mechanical obstruction). Furthermore, the peritoneal reflection from the anterior surface of the pancreas facilitates communication to the small bowel mesentery making the small bowel vulnerable to inflammatory complications [2, 16].

Finally, colonic paralytic ileus is a relatively more common and less severe complication of acute pancreatitis than true mechanical obstruction. The etiology of ileus is not entirely understood but may arise from a viscerally mediated reflex within the superior mesenteric plexus secondary to retroperitoneal inflammation and/or transient colonic ischemia [1].

Colonic obstruction secondary to acute pancreatitis usually resolves with conservative management unless it is associated with retroperitoneal collections or necrotizing pancreatitis, in which case prompt surgical intervention is warranted [17, 18]. There is paucity of literature on mechanical small bowel complications from acute pancreatitis but likely has similar pathophysiologic mechanisms and management when compared to colonic complications.

Conclusion

Clinicians must be keenly aware of mechanical small bowel complications of acute pancreatitis which albeit rare, are potentially deadly and therefore should be managed aggressively. The management is directed towards treating the underlying pancreatitis and surgical management is usually reserved for

Discussion

Bowel complications of acute pancreatitis such as paralytic ileus, ischemic necrosis, perforation and mechanical obstruction are relatively infrequent [3, 4]. Mechanical bowel obstruction as a result of acute pancreatitis has been described in the literature and is more likely to occur in the splenic flexure and transverse colon. This is believed to stem from either: 1) severe inflammation of the body and tail of the pancreas causing extrinsic compression; 2) retroperitoneal extravasation of pancreatic enzymes causing pericolic fibrosis; 3) thrombosis of mesenteric arteries (often associated with hypercoagulability during severe inflammatory states); or 4) infarction/ischemic necrosis of watershed areas second-
unstable patients with refractory bowel obstruction.

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**Conflicts of Interest**

The authors do not have any conflicts of interest or financial relationships to disclose.

**Author Contributions**

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