A CASE REPORT

Spontaneous Pancreaticoduodenal Fistula: An Uncommon Case of Severe GI Bleeding

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Spontaneous upper gastrointestinal (GI) tract fistulas are an uncommon occurrence that may present with fever, nausea, and vomiting. We describe a spontaneous pancreaticoduodenal fistula presenting with large-volume hematemesis requiring transfusions and emergency esophagogastroduodenoscopy (EGD) in a patient with acute on chronic pancreatitis in the setting of alcoholism and cirrhosis. Computed tomography (CT) imaging and endoscopic retrograde cholangiopancreatography (ERCP) demonstrated pseudocyst decompression and vascular compromise secondary to the formation of the fistula. Conservative management, rather than surgical ligation of the fistula, allowed full recovery.

INTRODUCTION

Pancreatic fistulas are a well-described phenomenon, particularly following surgical procedures involving disruption of the pancreatic parenchyma. However, spontaneous pancreatic fistulas are rare, occurring most commonly in the setting of pseudocyst formation secondary to necrotizing pancreatitis in patients with alcoholism. When spontaneous fistulization occurs, pain is typically partially relieved, though upper gastrointestinal (GI) fistula can be associated with new onset vomiting, diarrhea, and fever.

Typical management of upper GI fistula formation is conservative, with medical therapy resulting in closure of approximately 80% of fistulas. Octreotide therapy has been shown to reduce fistula output resulting in accelerated closure of the aberrant connection in some cases. When intervention is required, endoscopic retrograde cholangiopancreatography (ERCP) can be used to stent the sphincter of Oddi providing an alternative, low-resistance pathway and facilitating fistula closure. Surgical ligation of the fistula is an acceptable intervention in refractory cases. In addition to fistula formation, a number of vascular adverse events have been observed in the setting of chronic pancreatitis, with an incidence of 7-10% and mortality as high as 34-52%. Direct vascular injuries, while uncommon, can result in rapid blood loss and clinical decline. Pseudoaneurysm and arterial rupture can occur secondary to the leakage of exocrine pancreatic secretions, which weaken the vessel wall. This vascular compromise may result in any combination of hematoma or intraperitoneal, retroperitoneal, or intraluminal bleeding. Treatment involves establishing vascular control via endovascular, endoscopic, or surgical modalities, and the treatment option depends on hemodynamic stability, coagulation status, and vascular accessibility.
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A 53 year-old man with a history of acute on chronic pancreatitis, cirrhosis, alcoholism, gallstones, and a 40 pack-year smoking history presented with acute abdominal pain in the epigastric region radiating to the back. While he experienced chronic epigastric pain that varied in intensity, he noted that this pain was substantially more severe than usual. He described four episodes of large-volume hematemesis and one episode of bright red blood per rectum occurring within 12 hours preceding presentation to the ED. He had no history of prior abdominal surgery. Physical exam findings included pallor and fatigue.

At admission, vital signs showed temperature of 36.9 °C, blood pressure of 99/68, heart rate of 117, respiratory rate of 18 br/min, and oxygen saturation of 100%. He was found to have prominent leukocytosis (18.4 k/cumm) and a hemoglobin of 9.7 g/dL, decreased from 14.8 g/dL two months earlier. Lipase levels were elevated at 139 units/L. BUN was elevated at 36 mg/dL. Analgesics, intravenous (IV) octreotide, and an IV proton pump inhibitor (PPI) were administered.

CT scan demonstrated interval decompression of a primary cystic lesion (Figure 1), now 2.4 cm in diameter (3.5 cm two months prior), in the head of the pancreas exerting a mass effect on the second portion of the duodenum (Figure 2). The clinical impression was that of a pancreatic pseudocyst.

Due to continued anemia (Hgb 6.8 g/dL), the patient received two units of packed red blood cells (RBCs) and emergent esophagogastroduodenoscopy (EGD).

The EGD revealed no evidence of esophageal or gastric varices, peptic ulcers or blood in the stomach. A large fistula was noted in the first portion of the duodenum (Figure 3). An ulcerated lesion involving a compromised blood vessel was observed on the border of the fistula, and two clips were successfully applied. The remainder of the duodenum was normal. An additional unit of packed RBCs was required to stabilize the patient. Subsequent physical examinations following the procedure demonstrated severe epigastric pain that slowly abated over the course of hospitalization. No additional hematemesis or frank hematochezia was documented, and the patient’s hemoglobin stabilized at 9.4 g/dL. Repeat abdominal CT four days later demonstrated no significant change in the pseudocyst diameter. Once the patient’s diet was advanced, he was discharged after resolution of his acute pain.
Discussion and Conclusion

This case represents both an unusual pathology and presentation of pancreatoenteric fistula, a rare disease occurring in less than 5% of patients with acute pancreatitis. Spontaneous fistulization into the duodenum has been reported by several authors, though symptoms have more commonly included pain, fever, nausea, and vomiting rather than bright red hematemesis. Colonic fistulization resulting in hematemesis has also been reported, and its proclivity for severe hemorrhage documented, though these cases are often associated with hematochezia and melena rather than large-volume, bright red hematemesis. A broad differential diagnosis should be considered when evaluating patients with inflammatory abdominal disease. EGD may have therapeutic value in some instances, notably for the debridement of necrotic tissue and stent placement in other instances of pancreatoenteric fistulas. Most importantly, the case raises a dilemma regarding management, as this patient presented with a large-volume bleeding and hemodynamic instability analogous to the vascular compromise typically seen in colonic fistulization. Management of duodenal fistulas is traditionally conservative, while colonic fistulas often require surgical intervention. After application of Resolution clips to achieve hemostasis, conservative management was employed with careful monitoring of symptoms and hemoglobin levels. This facilitated symptomatic relief via continued drainage of the pancreatic fluid collection and allowed for the avoidance of an invasive procedure in a patient with multiple surgical risk factors.

In summary, this patient presented with an episode of pain consistent with acute on chronic pancreatitis in the setting of gallstones, liver disease, alcoholism, and tobacco use. However, this pain was complicated by the presence of large-volume hematemesis. A combination of CT and EGD images strongly suggested fistula formation, which not only resulted in pseudocyst drainage, but, in addition, also resulted in significant upper gastrointestinal hemorrhage due to vascular involvement. This case reaffirms the versatility of EGD in diagnostic and therapeutic interventions for pancreatitis, and demonstrates how conservative management of pancreaticoduodenal fistulas may be appropriate even in the setting of significant vascular compromise.

References