

Gastroduodenal Artery Psuedoaneurysm Bleed through a Spontaneous Pancreaticoduodenal Fistula as a Result of Acute Necrotizing Pancreatitis

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ABSTRACT

Spontaneous pancreaticoduodenal fistulization and arterial psuedoaneurysm formation are both complications of acute pancreatitis. We present a 27-year-old man with hematemesis who was found to be bleeding from a gastroduodenal artery psuedoaneurysm through a spontaneous pancreaticoduodenal fistula as a result of severe alcohol-related necrotizing pancreatitis. This is the first reported case in the literature to describe this occurrence.

INTRODUCTION

Acute necrotizing pancreatitis is a severe form of pancreatitis associated with significant morbidity and mortality. This form of the disease can lead to serious complications in 10–30% of patients, including exocrine insufficiency, multiorgan failure, fistulae formation, bleeding, and death.¹

CASE REPORT

A 27-year-old man with history of heavy alcohol intake (50 g ethanol/day) was transferred to our tertiary center with acute pancreatitis after presenting to a peripheral center with severe abdominal pain and vomiting. A computed tomography scan with contrast of his abdomen at presentation showed evidence of acute necrotizing pancreatitis with extensive portal vein thrombosis (Figure 1). Initial blood parameters demonstrated pancreatitis with an elevated lipase of 8,080 U/L. He was also noted to have a lactic acidosis (pH 7.07 and lactate 14.1 mmol/L), as well as an acute kidney injury (creatinine 141 μ mol/L). He was managed conservatively with aggressive hydration, administration of therapeutic heparin for his portal vein thrombosis, and enteral nutrition.

One week after presentation, the patient became febrile and tachycardic. Subsequent computed tomography imaging revealed a peri-pancreatic collection with extension along the mesentery (Figure 2). The collection had gas within it, suggestive of a fistula between the head of the pancreas and the first part of the duodenum. Ultrasound-guided drainage yielded fluid that cultured *Klebsiella oxytoca* and *Fusobacterium moriferum*, for which he was commenced on appropriate antimicrobial therapy. A 14F pigtail drain was inserted due to persistent collection and unresolved sepsis.

Three weeks into his admission, he had several episodes of large volume hematemesis with blood clots and associated hypotension and tachycardia. He also had blood in his external drain tubing. Laboratory tests demonstrated a drop in his hemoglobin from 12.5 g/dL to 8.2 g/dL. The patient was resuscitated with intravenous fluids and packed

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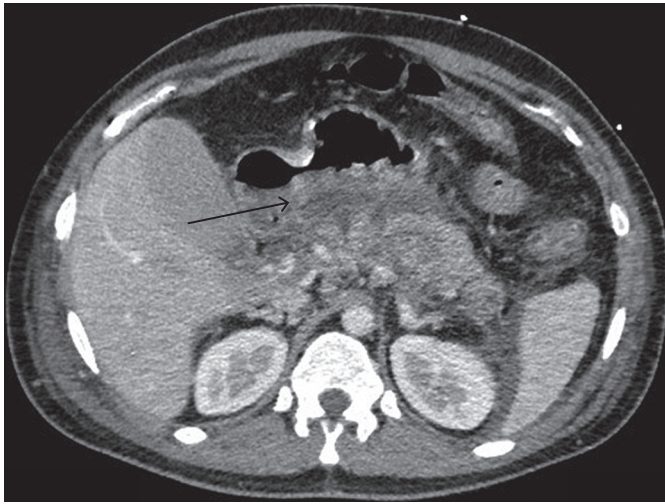


Figure 1. Computed tomography (CT) scan at initial presentation demonstrating acute necrotizing pancreatitis.

red blood cell transfusion. He was commenced on a proton pump inhibitor and octreotide infusion, given the risk of gastroesophagoduodenal varices as a result of his portal vein thrombosis.

Emergency esophagogastroduodenoscopy (EGD) which revealed a large adherent blood clot extending from the superior medial wall of the D1/2 junction. Adrenaline was injected at the clot base, and the clot was removed. On removal of the clot, there was evidence of a spontaneous pancreaticoduodenal fistula, with the flow of necrotic pancreatic contents into the duodenum (Figure 3). An endoclip was deployed at the site of fistulization to guide further interventional therapy.

On review of the patient's imaging, the gastroduodenal artery was noted to enter and be distorted by the patient's necrotic pancreatic collection. Angiography demonstrated a gastric artery pseudoaneurysm with spasm suggesting recent bleed, and this was successfully treated with coil embolization (Figure 4). The pancreaticoduodenal fistula was also subsequently confirmed with a fistulogram (Figure 5). The patient had no further episodes of hematemesis or bleeding through his pancreatic drain after embolization. His pancreatic collection and fistula improved slowly over several months, with external drainage and a prolonged course of intravenous antibiotic therapy and enteral nutrition.

DISCUSSION

Acute pancreatitis, an inflammatory condition of the pancreas, is a major cause of hospital admission with an increasing incidence worldwide.^{1,2} In the majority of cases, it has a mild, self-limiting course, but severe necrotizing pancreatitis is associated with significant morbidity and mortality.³



Figure 2. CT scan 1 week after initial presentation demonstrating necrotizing pancreatitis with associated peripancreatic collection with multiple air locules within the collection, suggestive of a pancreaticoduodenal fistula.

Gallstones and alcohol use account for a majority of cases of acute pancreatitis.¹

A small proportion of patients with acute necrotizing pancreatitis may develop vascular complications including venous thrombosis, arterial pseudoaneurysm, and varices.⁴ Initial imaging in our patient demonstrated an extensive portal vein thrombosis for which he was commenced on anticoagulation therapy. Angiography subsequently identified a gastroduodenal artery pseudoaneurysm as the source of his bleeding. The incidence of pseudoaneurysms associated with acute pancreatitis is low at 1.3–10%.⁵ They are thought to form as a result of arterial wall auto-digestion by proteolytic enzymes, which cause weakening and ballooning of the vessel lumen.⁶ The splenic artery is the most commonly affected vessel (up to 50% of cases), followed by the gastroduodenal artery (20% of cases) and the pancreaticoduodenal artery (10% of cases).⁵ Other vessels that can be involved include the superior mesenteric and proper hepatic arteries.

The most common therapeutic option for pancreatic pseudoaneurysms is the placement of endovascular coils.⁷ Several other

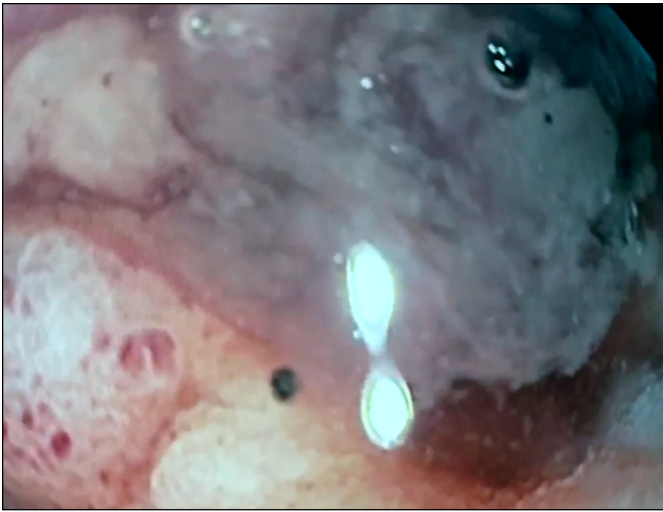


Figure 3. Endoscopy demonstrating pancreatic debris flowing into the duodenum after clot removal, confirming pancreaticoduodenal fistulization.

interventional techniques that have been used with reasonable success include the deployment of covered stents, detachable balloons, and gel foam. Endoscopic ultrasound (EUS)-guided coiling is an emerging therapeutic option that can be used for bleeding involving duodenal and peri-pancreatic blood vessels. A group in India successfully used this technique by placing 3 coils, using a 19G needle, to control massive hematemesis from actively bleeding collaterals associated with a pancreatic neuroendocrine tumour.⁸ This could potentially have been used in our case;

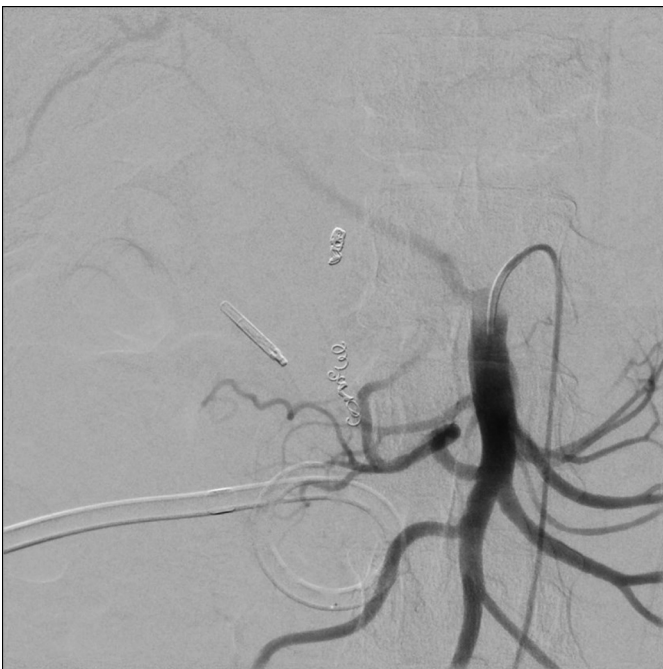


Figure 4. Digital subtraction angiography demonstrating successful coil embolization of the gastroduodenal artery pseudoaneurysm.

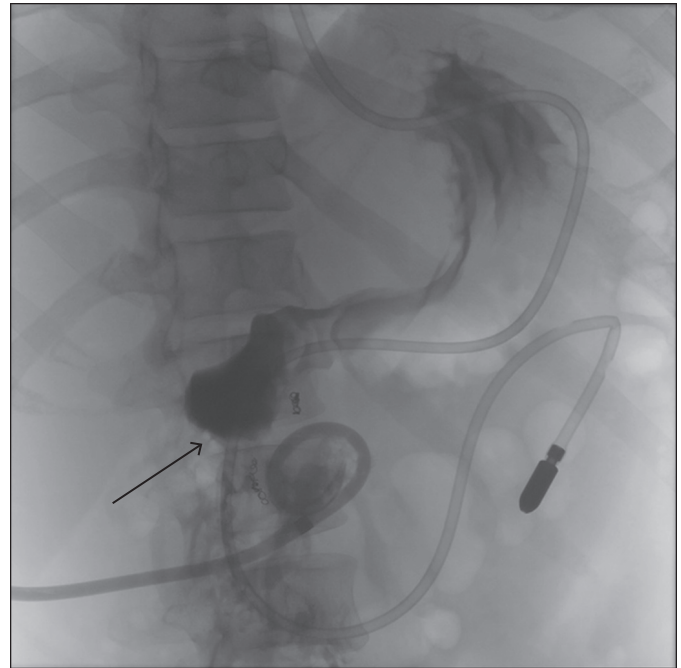


Figure 5. Fistulogram performed by injecting contrast into indwelling drain within the inferior aspect of pancreatic collection. Contrast flow into stomach confirmed fistulous communication.

however, expertise in this technique was not available at our center. The close proximity of the gastrointestinal (GI) tract to vascular structures makes EUS-guided coiling and other EUS-guided vascular interventions a promising set of therapeutic option in the future.⁹

In our case, EGD demonstrated a pancreaticoduodenal fistula. The inflammatory process of acute pancreatitis may lead internal fistulization to the GI tract.¹⁰ Spontaneous pancreaticoduodenal fistulas are not uncommon; depending on the study population, the incidence varies from 3% to 47%.¹¹⁻¹⁴ Duodenal and colonic fistulas occur more frequently compared to other sites due to their anatomic proximity. The majority of fistulas in the upper GI tract close spontaneously and can be managed conservatively compared to colonic fistulas, which require a higher rate of intervention.¹⁵ Entero-pancreatic fistulas in this setting can predispose to GI bleeding. Our patient bled from his gastroduodenal artery pseudoaneurysm through his spontaneous fistula. There was clot overlying the visualized fistulous tract, but no active bleeding was present at the time of EGD.

Pancreaticoduodenal fistula and arterial pseudoaneurysm should be considered in any patient with acute necrotizing pancreatitis who presents with hematemesis. Early diagnosis and aggressive management with a combination of endoscopic and radiologic interventions and nutritional support are key to minimizing morbidity and mortality in these patients.

DISCLOSURES

Author contributions: All authors contributed equally to the manuscript. S. Picardo is the article guarantor.

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Informed consent was obtained for this case report.

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