

# An Atypical Presentation of Hemosuccus Pancreaticus Manifesting as Hematochezia

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## ABSTRACT

Hemosuccus pancreaticus is a rare cause of upper gastrointestinal bleeding that is often associated with chronic pancreatitis. The bleeding usually manifests as melena because the source originates superior to the ligament of Treitz. We present a patient who was admitted for acute-on-chronic pancreatitis and ultimately developed hematochezia. Endoscopy revealed active oozing at the minor duodenal papilla. Computed tomography angiography identified active contrast extravasation at the gastroduodenal artery, and it was managed successfully with angioembolization. Our case emphasizes clinicians to consider hemosuccus pancreaticus as an alternative differential in a patient with a history of chronic pancreatitis manifesting with hematochezia.

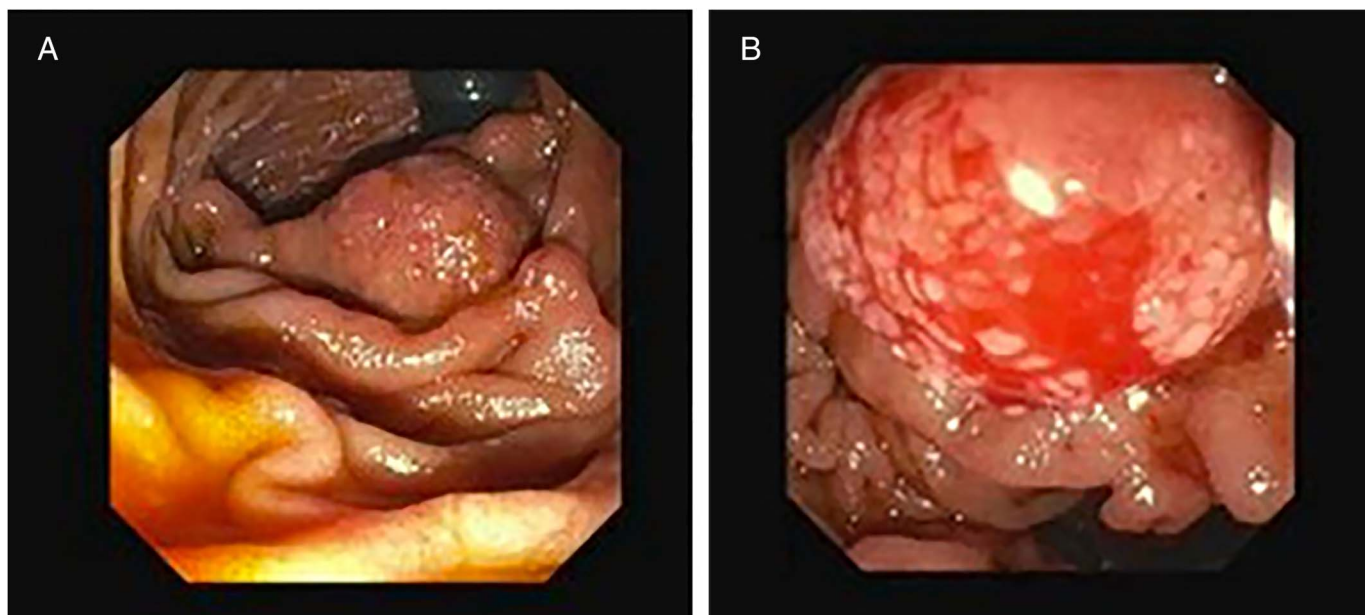
## INTRODUCTION

Hemosuccus pancreaticus (HP) is an uncommon cause of upper gastrointestinal (GI) bleeding typically seen in patients with alcohol-related chronic pancreatitis.<sup>1</sup> Other potential causes of chronic pancreatitis include, but are not limited to, tobacco use, cystic fibrosis, systemic lupus erythematosus, and pancreatic malignancy. The bleeding in HP originates from the papilla of Vater and flows into the duodenum through the pancreatic duct, which allows it to be visualized by esophagogastroduodenoscopy (EGD). In clinical practice, the bleeding can present as silver stools because of the mixture of blood from the pseudoaneurysm and pancreatic enzymes. The pathophysiology of the hemorrhage is most likely because of chronic inflammatory changes to the pancreas from alcohol excess. This ultimately necroses the peripancreatic vessels, resulting in the formation of a pseudoaneurysm.<sup>1</sup> The most common vessels that hemorrhage in HP include the splenic, gastroduodenal (GDA), pancreaticoduodenal, gastric, and hepatic arteries.<sup>2</sup> It remains a rare cause of upper GI hemorrhage with an estimated incidence of 1 in 1,500 cases.<sup>3</sup> Achieving hemostasis by therapeutic EGD is high risk and challenging given an increased likelihood of bleeding. Angioembolization offers the safest route to intervene on these patients and is successful in 79%–100% of cases, often with immediate resolution of clinical signs of bleeding.<sup>4</sup>

## CASE REPORT

A 79-year-old African American woman with a history of cirrhosis secondary to hepatitis C, chronic pancreatitis, Billroth I gastrectomy, renal cancer, peripheral vascular disease, coronary artery disease, and heart failure was admitted for epigastric abdominal pain. She was noted to be hypertensive at 161/100, but vital signs were otherwise stable. Laboratory evaluation showed white blood cell—12.0 k/ $\mu$ L, hemoglobin B—10.6 g/dL, creatinine—6.1 mg/dL (baseline 1.5–2 mg/dL), blood urea nitrogen—45 mg/dL, glomerular filtration rate—7 mL/min, and lipase—474 U/L. With diagnoses of acute-on-chronic pancreatitis and prerenal acute kidney injury, she was resuscitated with crystalloids according to the pancreatic protocol and started on a pain regimen. Gastroenterology was consulted for ongoing dysphagia. An EGD revealed mucosal changes suspicious for eosinophilic esophagitis, Billroth type I gastrectomy, and mild esophageal stenosis. An antrectomy with GDA anastomosis was previously performed in this patient because of recurrent peptic ulcer disease. Biopsies from the stomach and esophagus were negative for *Helicobacter pylori*, malignancy, and eosinophilic esophagitis.

One week after the endoscopy, the patient developed multiple episodes of bright red blood per rectum. Her hemoglobin dropped to 6.9, which was below her baseline of 9 g/dL. Colonoscopy performed because of suspected lower GI bleeding only revealed polyps and



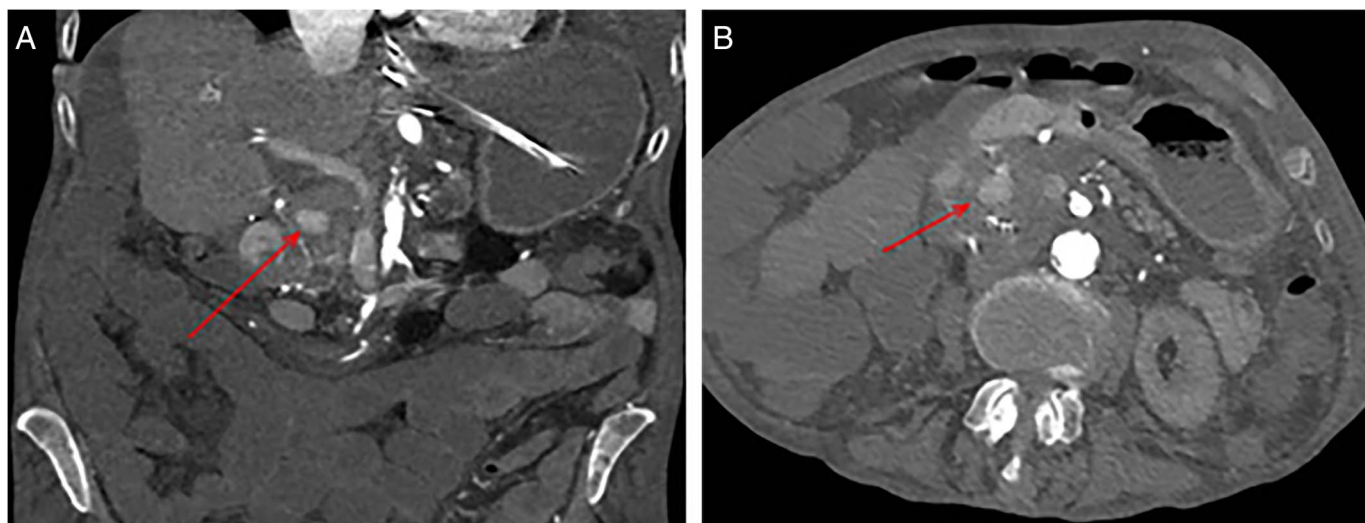
**Figure 1.** (A) Esophagogastroduodenoscopy (EGD) shows a deformity at the minor duodenal papilla in the second portion of the duodenum. (B) EGD with a side-viewing duodenoscope shows active bleeding from the minor duodenal papilla in the second portion of the duodenum.

no source of bleeding. A follow-up EGD revealed a deformity at the minor duodenal papilla with mild blood oozing on minimal contact (Figure 1). Computed tomography angiography identified active extravasation of contrast adjacent to the duodenum in the expected region of the GDA into the pancreatic pseudocyst (Figure 2). These findings were favored to represent a pseudoaneurysm. Visceral angiogram identified active extravasation from the superior pancreaticoduodenal artery, occlusion of the superior mesenteric artery (SMA), and collateral flow to SMA through the celiac artery via the GDA artery (Figure 3). Superselective embolization of the superior pancreaticoduodenal artery was

performed using a Tornado Embolization Coil (Cook Medical, Bloomington, IN) and Gelfoam into the aneurysm without compromising blood supply to the SMA through GDA (Figure 4). The patient did not have any further bleeding episodes, and her hemoglobin remained at baseline. She was discharged to an extended care facility.

## DISCUSSION

HP can be notoriously hard to manage because the bleeding can range from intermittent occult to a massive hemorrhage constituting a surgical emergency. The bleeding often manifests as



**Figure 2.** (A) Contrast-enhanced abdominal/pelvic computed tomography coronal plane image (arrow) demonstrating active extravasation into the pseudocyst near the gastroduodenal (GDA) artery. (B) Contrast-enhanced abdominal/pelvic computed tomography axial plane image (arrow) demonstrating active extravasation of contrast in the pancreatic pseudocyst in the region of the GDA artery.



**Figure 3.** Celiac artery angiography with active extravasation of contrast from the superior pancreaticoduodenal artery (arrow). Occlusion of the superior mesenteric artery and collateral flow from the celiac artery through the gastroduodenal artery was also noted.

melena or hematemesis, which are expected clinical signs of an upper GI hemorrhage. In our case, we have an atypical presentation of hematochezia as the sign of HP. Most cases of hematochezia stem from lower GI sources including diverticular disease, hemorrhoids, colon cancer, and inflammatory bowel disease.<sup>5</sup> However, clinical practitioners must be vigilant to avoid missing this potentially fatal diagnosis in patients with a documented history of chronic pancreatitis.

Embolization is regarded as the safest treatment modality for HP; however, careful attention must be paid to identify collateral vessels before provoking a thrombus. In our case, the patient's SMA was severely occluded and, thus, negated nonselective GDA artery embolization because of the risk of bowel ischemia. In cases where the splenic artery is the hemorrhaging vessel secondary to HP, proximal splenic artery embolization with coils in a sandwich technique is indicated. This is because of the risk of splenic infarction with Gelfoam or distant embolization.<sup>6</sup> Different techniques such as stent graft placement are used for managing the pseudoaneurysm at the celiac trunk, common hepatic artery, and SMA. Alternative potential treatment options such as an endoscopic ultrasound-guided thrombin injection and percutaneous cyanoacrylate glue injection need to be investigated further before being declared standards of care.<sup>7</sup>

HP can be an easily missed diagnosis in a patient manifesting with hematochezia. Minimally invasive interventional radiology techniques, such as coil angioembolization, are recommended for hemodynamically stable patients whose hemorrhaging vessel has sufficient collateral circulation. Medical practitioners must be



**Figure 4.** Successful coil embolization of the superior pancreaticoduodenal artery with preservation of collateral flow to the superior mesenteric artery through the gastroduodenal (GDA) artery (arrow). Occlusion of superior mesenteric artery and collateral flow through the celiac artery via the GDA artery was also noted.

vigilant when assessing atypical presentations of upper GI bleeding in patients with chronic pancreatitis.

## DISCLOSURES

**Author contributions:** All authors were involved in the care of the patient. A. Sagalov wrote the first draft of the manuscript, wrote the final draft of the manuscript, and is the article guarantor. Y. Turk edited the first draft of the manuscript. H. Siddiqui and S. Nida edited the second draft of the manuscript. H. Rathinamanickam edited the third draft of the manuscript. P. Sharma provided images and captions of the computed tomography angiography and the visceral angiogram and edited the first draft of the manuscript. A. Swied performed diagnostic EGD on the pt., provided pictures and captions of EGD images, edited the first draft of manuscript, and approved the final draft of the manuscript.

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