

Acute ischemic pancreatitis: A rare complication of empirical gastroduodenal artery embolization

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Abstract

Empirical embolization of the gastroduodenal artery (GDA) is accepted as a safe and effective treatment option for endoscopy-refractory nonvariceal upper gastrointestinal bleeding (UGIB) in patients with high surgical risk. Nontarget embolization is a recognized complication of transarterial embolization, however, symptomatic pancreatic injury is extremely rare. We report a patient who developed acute ischemic pancreatitis immediately after embolization of the GDA, which was confirmed intraoperatively. Interventionists as well as referring clinicians need to be aware of this rare but life threatening complication.

Key words: Acute pancreatitis; empirical embolization; gastroduodenal artery; nonvariceal bleed; upper gastrointestinal bleed

Introduction

Empirical gastroduodenal artery (GDA) embolization is well-established as a relatively safe treatment in high surgical risk patients for intractable upper gastrointestinal bleeding (UGIB) following failed attempts at endoscopic hemostasis.^[1-3] The technical and clinical success rates are high with low rates of complications (9%), most of which are minor.^[3] It has been shown to be safe and similar in efficacy compared to targeted endovascular embolization.^[3-7] Although uncommon, the well-recognized major complications of GDA embolization include duodenal ischemia and hepatic infarction from nontarget embolization.^[2,5,8,9] We report a very rare but fatal complication of acute pancreatitis, post GDA embolization.

Case Report

A 90-year-old female presented to the emergency department with five episodes of hematemesis. Other relevant clinical history included low back pain, for which she was recently taking nonsteroidal anti-inflammatory medications and hypertension. Her vitals were stable and physical examination was unremarkable. Urgent esophagogastroduodenoscopy (OGD) revealed a Mallory Weiss tear in the esophagus and a Forrest 1a ulcer on the anterior wall of the first to second part of the duodenum [Figure 1A]. Three hemoclips were deployed to the duodenal ulcer resulting in hemostasis [Figure 1B], and the patient was also commenced on intravenous proton pump inhibitor. Three days later, the patient developed hemodynamic instability with a drop in hemoglobin from

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9.0 to 6.5 g/dL. A second OGD showed slow active bleeding from a vessel at the site of intact clips which was treated with additional hemoclips and adrenaline injection resulting in hemostasis. Twelve days later, the patient developed a new episode of melena and repeat OGD showed friable tissue and active bleeding at the site of previous duodenal ulcer with intact clips. Additionally, three hemoclips were deployed. A computed tomography (CT) mesenteric angiogram was performed which showed no evidence of bleeding from the gastrointestinal tract or signs of pancreatitis [Figure 2]. In view of the refractory UGI bleeding despite several attempts at endoscopic hemostasis, the patient was referred to Interventional Radiology to empirically embolize the GDA.

Super selective angiograms of the GDA, superior (SPDA) and inferior pancreaticoduodenal arteries (IPDA) were performed using a 2.7F Progreat™ (Terumo, JAPAN) microcatheter inserted coaxially through the 4F C2 catheter. No active contrast extravasation or pseudoaneurysm was demonstrated at the known site of duodenal ulcer or elsewhere.

Embolization of the GDA was performed using three 6 mm and three 8 mm Nester™ (COOK, USA) 018 microcoils, commencing distally from the right gastroepiploic artery and including the origin of the SPDA. No gel foam or particles were used for the embolization. Completion angiograms showed no contrast opacification of the GDA by antegrade or retrograde flow [Figure 3].

By the end of the procedure, the patient developed sudden onset of severe epigastric pain and hematemesis. She, however, remained hemodynamically stable. An urgent OGD showed fresh blood in the esophagus and a large blood clot in the gastric antrum extending to the pylorus, as well as in the duodenum. Of note, her serum amylase and lipase showed an increase from 56 U/L and 87 U/L previously on admission to 716 U/L and >600 U/L, respectively (reference value for amylase 38–149 U/L; for lipase 8–55 U/L). Liver enzymes were within normal range.

Exploratory laparotomy with pyloromyoduodenotomy, under running of the duodenal ulcer, followed by

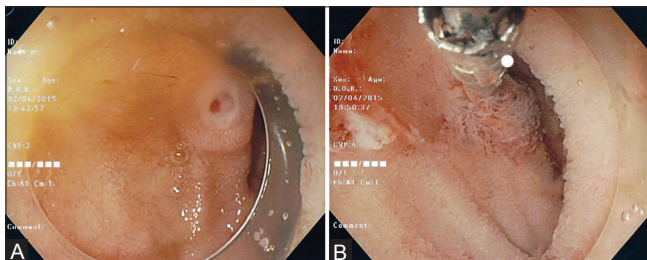


Figure 1 (A and B): Endoscopic images of the duodenal ulcer and haemostasis using hemoclips. (A) OGD image of the Forrest 1a duodenal ulcer. (B) Endoscopic deployment hemoclips

gastrojejunal bypass was performed on the same day. Intraoperatively, the pancreas was edematous with generalized bruising and fat saponification on the surface, predominantly involving the head and uncinate process. The findings were consistent with hemorrhagic pancreatitis. Her subsequent recovery in surgical intensive care unit, followed by high dependency unit, during the course of the next 9 days was complicated by pseudomonas septicemia and atrial fibrillation. A CT scan performed 1 week later showed findings consistent with acute focal pancreatitis complicated by loculated peripancreatic collection extending into the lesser sac [Figure 4A and B]. She failed to recover from this acute episode and died from multiorgan failure.

Discussion

Transarterial embolization (TAE) is a widely accepted treatment option for persistent UGIB after failed endoscopic hemostasis, especially in patients with high surgical risk. It has been recommended as an alternative to surgery in several guidelines.^[8,10] A recent systemic review by Beggs *et al.* comparing TAE and surgery showed that there was no statistical difference in 30-day mortality rates and secondary outcomes except for rebleeding rates, which were less in patients undergoing surgery. This is despite patients in the TAE group having more comorbidities such as ischemic heart disease and coagulopathy.^[9] The clinical success rates of TAE for UGIB have been shown to be more than 60%, independent of the demonstration of active bleeding during angiography.^[10]

In addition, empirical GDA embolization has been reported as a safe technique with low rates of complications. Major ischemic complications range from 0–16% in past studies.^[7] These usually present acutely as gastrointestinal necrosis or later, with ischemic duodenal stenosis.^[9] Previous

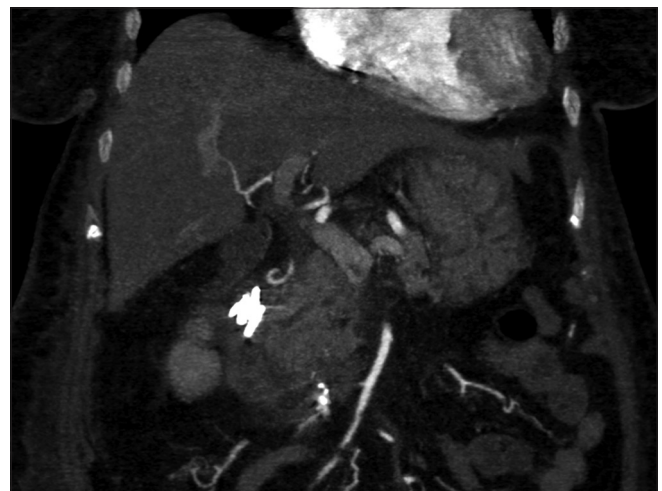


Figure 2: Coronal reformat of the CT mesenteric angiogram. Arterial phase image show no evidence of contrast extravasation within the duodenum which contains multiple metallic clips



Figure 3: Selective angiogram of the common hepatic artery post embolization shows no GDA opacification

foregut surgery resulting in altered anatomy and use of liquid (cyanoacrylate) or particulate embolics (polyvinyl alcohol), which can penetrate into the distal branches are identified as risk factors for intestinal ischemic complications.^[3,7]

In general, ischemic complications are not seen with pancreas from TAE due to its rich vascular supply.^[5,7] The pancreatic head receives blood supply from the SPDA, which is a branch of the GDA as well as the IPDA, which originates from the superior mesenteric artery. The body and tail of the pancreas are supplied by the dorsal pancreatic and caudal pancreatic arteries, both of which are branches of the splenic artery.^[11]

A literature search yielded four cases of acute pancreatitis reported after TAE for refractory UGIB.^[11-14] Coils ± gelfoam were used as the embolic agents in all four cases with embolization of “front and back door” supply as per conventional principles. The patients presented with symptoms of pancreatitis of 12 hours to 70 days duration (mean = 29 days) post GDA embolization. This is in contrast to our case where the patient became symptomatic immediately after embolization, which has not been previously reported. Poor collateral supply owing to background ischemic heart disease, diabetes mellitus, and chronic renal disease being reported as the predisposing factors for ischaemic pancreatitis in one of these cases.^[13] Previous upper GI tract surgery/altered anatomy has also been reported as a risk factor for developing ischemic sequelae post embolization, as mentioned earlier.^[3,7]

In our patient, the GDA as well as the proximal segments of the SPDA and right gastroepiploic artery were embolized using coils as the sole embolic agent to prevent retrograde filling. The IPDA was not embolized. The CT or conventional mesenteric angiography did not reveal any

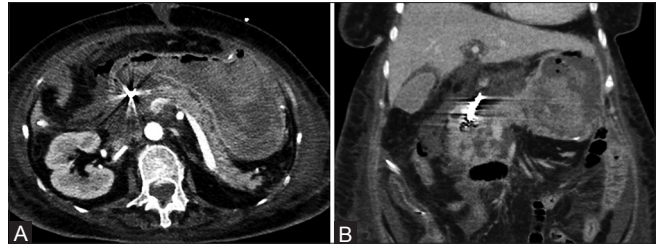


Figure 4 (A and B): CT axial (A) and coronal (B) images 1 week post embolization showing acute pancreatic changes. Images are impaired by streak artifacts from the metallic coils

significant stenosis of the coeliac axis, superior mesenteric artery, or their major branches to suggest poor collateral supply.

In reviewing the other possible causes of pancreatitis, our patient was not found to be on medications that are commonly known to cause pancreatitis. Of note, she had no history of alcohol intake. Moreover, no gallstone disease or obstruction of the pancreatic duct was identified on the initial CT scans.

Severe hemorrhage has been implicated as a complication of acute pancreatitis.^[15] However, our patient did not present with symptoms of acute pancreatitis, and the biochemical markers for acute pancreatitis were normal on admission. These make her bleeding episodes less likely to be a result of acute pancreatitis. Instead, our patient’s age and nature of the ulcer may be the cause of recurrent bleeding. Studies have shown that 10–30% of patients with bleeding peptic ulcer have recurrent bleeding after initial endoscopic therapy.^[16] Risk factors for rebleeding include actively bleeding ulcer (Forest 1a and 1b), location of ulcer (lesser curvature of stomach and posterior wall of duodenum), ulcer larger than 2 cm and age greater than 65.^[17] Our patient was 90 years old and had a Forest 1a ulcer at presentation, both of which are risk factors for rebleeding. This may explain the refractoriness of her bleeding episodes to endoscopic hemostasis.

Ultimately, the combination of the acute presentation immediately after embolization, the concordant rise of biochemical markers for pancreatitis and the preferential involvement of the head and uncinata process of the pancreas with relative sparing of the rest of the organ makes acute ischemia as the likely cause of focal pancreatitis in our case.

Conclusion

Although empiric GDA embolization is relatively safe, it is essential for interventionists and referring clinicians to be aware of this rare but life threatening complication as most of the candidates referred for this therapy are elderly with vascular compromise due to the acute blood loss and other pre-existing comorbidities.

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Conflicts of interest

There are no conflicts of interest.

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