

Ruptured Gastroduodenal Artery Pseudo Aneurysm in Case of Pancreatitis

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Abstract

Case Report

Pseudo-aneurysms rarely occur in gastroduodenal artery. It is most commonly seen in splenic artery. Gastroduodenal artery pseudo aneurysm has high mortality rate in case of rupture. Upper gastrointestinal bleeding due to visceral artery pseudoaneurysm is a rare cause. Clinical suspicion and advanced imaging tools should be employed in a timely fashion to make a diagnosis before this ominous event. Hereby I present a case of GDA pseudoaneurysm masked with pancreatitis, and plan of management when patient arrive in emergency room.

Keywords: Hematemesis, Gastroduodenal Artery, Pancreatitis, Pseudoaneurysm, Rupture.

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INTRODUCTION

The gastroduodenal artery (GDA) is a blood vessel that supplies blood to the duodenum and the head of the pancreas. An aneurysm is an abnormal bulge or weakening in the wall of a blood vessel. GDA aneurysms can be either true aneurysms (involving all layers of the vessel wall) or pseudoaneurysms (involving only the outer layers of the vessel wall). Pancreatitis, especially chronic pancreatitis, is a major risk factor for developing GDA pseudoaneurysms. During pancreatitis, the pancreatic enzymes can damage the GDA wall, leading to weakening and causing the formation of a pseudoaneurysm. Other causes include atherosclerosis, trauma, and infections. GDA aneurysms can be asymptomatic. Common symptoms include abdominal pain and gastrointestinal bleeding, which can manifest as hematemesis (vomiting blood) or melena (black, tarry stools). Rupture aneurysm can lead to life-threatening conditions. CT angiography is the preferred imaging modality for diagnosis. Angiography can also be used to visualize the aneurysm and assess its characteristics. Endovascular intervention (embolization) is often the first-line treatment, involving blocking off the aneurysm with coils or other materials. Surgery may be necessary in cases of failed embolization, rebleeding, or when the aneurysm is not amenable to endovascular treatment. Regardless of the treatment chosen, close monitoring for complications like rebleeding is crucial.

CASE STUDY

A 40-year-old male with a history of alcohol abuse for the last 15-17 years, recurrent alcoholic pancreatitis (last episode 3 weeks ago), and presented to the emergency department with complaints of abdominal pain and hematemesis. Pain was in the epigastric region, dull in nature, 7/10 in intensity, radiating to the back, associated with nausea but with no relation to food intake or bowel movement since 3 weeks. Yellowish discoloration of sclera and urine since 3 weeks. No significant family history of pancreatitis, pancreatic cancer, or other malignancies was present. He was discharged from another hospital 1 week ago after an episode of acute interstitial edematous pancreatitis secondary to alcohol intake, which improved with intravenous fluids, pain control, and bowel rest. Physical examination was positive for mild epigastric tenderness without guarding, rigidity, or distension. Vitals on admission were blood pressure of 110/80 mm HG, heart rate of 90 beats/min, and oxygen saturation of 98% on room air. Laboratory examination revealed a white blood cell count of 10,000, hemoglobin of 5.20 g/dL, blood urea nitrogen of 23mg/dL, creatinine of 1.2mg/dL, international normalized ratio of 1.1, calcium of 8.4 mg/dL, lipase of 318 U/L, total bilirubin of 8.6mg/dl, direct bilirubin of 5.4 mg/dl, SGOT of 92 U/L, SGPT of 54 U/L.

The computed tomography (CT) of the abdomen angiography showed there is evidence 5.5 x 5.4 x 4.9 cm well-defined thin-walled (maximum thickness 2.6 mm) collection noted in the pancreatic head and uncinate process with hyperdense (Avg HU +35) contents noted within likely suggestive of hemorrhagic contents (fig:01,02). A saccular outpouching measuring 3.9 x 3.9 x 4.2 cm (AP x ML x S1) with neck 3.7 mm is noted arising from gastroduodenal artery, It is directed anteroinferiorly and noted within the above defined collection suggestive of pseudoaneurysm formation and rupture. No active blush noted within the collection. The collection is abutting the SMV medially with focal loss of intervening fat planes. It is causing compression of distal CBD with resultant moderate dilatation of the entire CBD (max diameter

10.6 mm), CHD (max diameter 11.2 mm) with mild central and peripheral Communicating IHBRD, and dilatation of cystic duct with over distension of GB. A suspicious communication is noted between the collection and the MPD via a defect of 1.5mm super o-medially. There was mild peripancreatic fat stranding noted near the head region (fig:01,02). Multiple intraparenchymal calcification foci were noted in the head and uncinate process. After transfusing two packed red cells and two fresh frozen plasma, the patient shifted to an interventional radiology room for digital subtraction angiography and gastroduodenal artery coiling (fig:03). The procedure was uneventful, and the patient was observed for 24 hours for rebleeding. The patient was vitally stable, complaints of hematemesis had resolved, and oral intake was started after 48hr.



Fig. 01: Contrast extravasation on CT abdomen angiography in axial section



Fig. 02: CT abdomen angiography in coronal section



Fig. 03: Extravasation of contrast on DSA

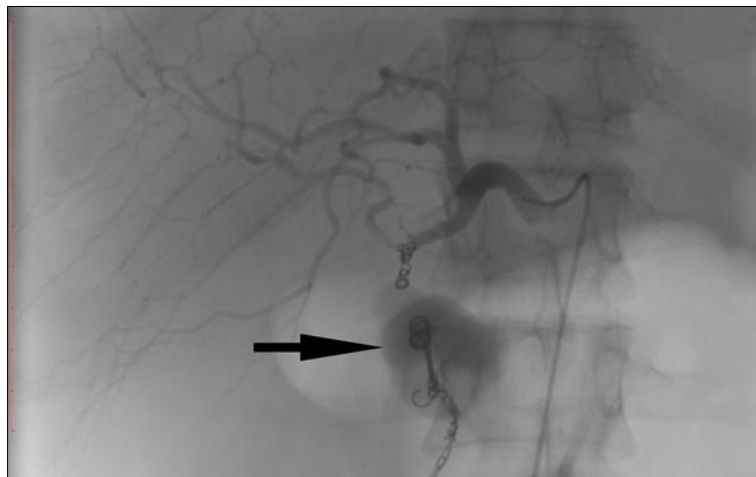


Fig. 04: Post coiling image of GDA on DSA

DISCUSSION

Gastrooduodenal artery aneurysm is rare, but the most common causes are pancreatitis and trauma. Other causes can be post-surgical, including pancreatic and biliary surgery and also peptic ulcer disease, alcohol abuse, vasculitis or connective tissue disorder. In postoperative studies, mortality rates in the surgical group were generally higher (50–100 %) than those in the embolization group (13–50 %). However, this might be expected given that patients requiring early surgical intervention are usually more haemodynamically unstable than those who can wait for angiography [1]. The arteries from which the pseudoaneurysm arises can be classified into three groups [1].

1. type I arises from a minor artery but must be at least 5 mm away from its junction of origin from a type II or III artery
2. type II arises from a major artery that may be sacrificed without physiological consequences (e.g., splenic and gastroduodenal arteries)
3. type III arises from a major artery that cannot be sacrificed without significant consequences

for the patient (e.g., the SMA or the hepatic artery proper)

Pseudoaneurysms can be further subclassified into:

- a. Type A where there is no communication with the GIT
- b. Type B where there is communication with the GIT

Visceral artery aneurysms are divided into true or pseudoaneurysm [1]. A true aneurysm involves all layers of the vessel wall and is created due to partial digestion of the arterial wall, destroying the elastic tissue of tunica media by the inflammatory process, whereas pseudoaneurysms are false aneurysms which result from injury to one or more vessel wall layers. Pancreatic pseudoaneurysms are formed by the erosion of the pancreatic or peripancreatic artery into a pseudocyst. Gastrooduodenal pseudoaneurysm most commonly presents as gastrointestinal bleeding secondary to rupture (52%) or as abdominal pain (46%); it is asymptomatic in 7.5% and rarely presents as retroperitoneal bleeding, intraperitoneal bleed, or bleeding into the pancreatic duct or common bile duct, which can cause obstructive jaundice. Treatment modalities include either

embolization (coils, covered stent, percutaneous or transcatheter thrombin injection) or surgery [1-3]. Pseudoaneurysm have been reported in different visceral arteries; splenic artery being the most common (46%), followed by the renal artery (22%), the hepatic artery (16.25%), and least commonly the pancreaticoduodenal artery (1.3%) [4]. Pseudoaneurysms of the gastroduodenal artery are extremely rare [5].

CONCLUSION

GDA aneurysm, especially pseudoaneurysm, is a rare but serious complication of pancreatitis. Early diagnosis and prompt treatment are crucial for preventing potentially fatal complications.

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