Clinical Case Report

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Case report and systematic literature review of a novel etiology of sinistral portal hypertension presenting with UGI bleeding

Left gastric artery pseudoaneurysm compressing the splenic vein treated by embolization of the pseudoaneurysm

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Abstract

Introduction: A novel case is reported of upper gastrointestinal (UGI) bleeding from sinistral portal hypertension, caused by a left gastric artery (LGA) pseudoaneurysm (PA) compressing the splenic vein (SV) that was successfully treated with PA embolization.

Case report: A 41-year-old man with previous medical history of recurrent, alcoholic pancreatitis presented with several episodes of hematemesis and abdominal pain for 48 hours. Physical examination revealed a soft abdomen, with no abdominal bruit, no pulsatile abdominal mass, and no stigmata of chronic liver disease. The hemoglobin declined acutely from 12.3 to 9.3 g/dL. Biochemical parameters of liver function and routine coagulation profile were entirely within normal limits. Abdominal CT revealed a 5-cm-wide peripancreatic mass compressing the stomach and constricting the SV. Esophagogastroduodenoscopy showed blood oozing from portal hypertensive gastropathy, small nonbleeding gastric cardial and fundal varices, gastric compression from the extrinsic mass, and no esophageal varices. MRCP and angiography showed that the mass was vascular, arose from the LGA, compressed the mid SV without SV thrombosis, and caused sinistral portal hypertension. At angiography, the PA was angioembolized and occluded. The patient has been asymptomatic with no further bleeding and a stable hemoglobin level during 8 weeks of follow-up.

Discussion: Literature review of the 14 reported cases of LGA PA revealed that this report of acute UGI bleeding from sinistral portal hypertension from a LGA PA constricting the SV is novel; one previously reported patient had severe anemia without acute UGI bleeding associated with sinistral portal hypertension from a LGA PA.

Conclusion: A patient presented with UGI bleeding from sinistral portal hypertension from a LGA PA compressing the SV that was treated by angiographic obliteration of the PA which relieved the SV compression and arrested the UGI bleeding. Primary therapy for this syndrome should be addressed to obliterate the PA and not the secondarily constricted SV.

Abbreviations: CT = computerized tomography, EGD = esophagogastroduodenoscopy, GI = gastrointestinal, LGA = left gastric artery, MRCP = magnetic resonance cholangio-pancreatography, PA = pseudoaneurysm, SRH = stigmata of recent hemorrhage, SV = splenic vein, SVT = splenic vein thrombosis, UGI = upper gastrointestinal.

Keywords: hematemesis, left gastric artery, pancreatitis, portal hypertensive gastropathy, pseudoaneurysm, sinistral portal hypertension, splenic vein thrombosis, therapeutic angiography

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KEY POINTS

Previously reported:

- GI bleeding from gastric varices or portal hypertensive gastropathy can occur from sinistral (left-sided) portal hypertension, typically secondary to splenic vein injury. Reported etiologies include splenic vein thrombosis, embolism, or compression by various enlarged, adjacent lesions.

What is new here:

- A novel case is reported of UGI bleeding from sinistral portal hypertension from a left gastric artery pseudoaneurysm compressing the splenic vein. In this case the UGI bleeding at EGD was from portal hypertensive gastropathy rather than gastric varices, as determined by SRH.
- Angiographic embolization of the left gastric artery pseudoaneurysm relieved the splenic vein compression, reversed the sinistral portal hypertension, and arrested the UGI bleeding.

1. Introduction

Although numerous etiologies of gross gastrointestinal (GI) bleeding from sinistral portal hypertension have been reported (Table 1[1–34]), systematic literature review revealed no case of gross GI bleeding from sinistral (left-sided) portal hypertension from splenic vein (SV) compression by a left gastric artery (LGA) pseudoaneurysm (PA). A patient is reported who presented with this novel syndrome from this PA. This syndrome is important to diagnose because therapy for this syndrome should be directed at the PA to eliminate SV compression and reverse the sinistral portal hypertension rather than the secondarily affected SV; this case illustrates this principle by the novel report of angioembolization of this PA to achieve hemostasis.

2. Methods

The literature was systematically reviewed using PubMed with the following medical subject headings (MeSH) or keywords: {"left gastric artery"} and {pseudoaneurysm} OR {"sinistral" or "left"} and {"portal hypertension"} and [{"gastrointestinal bleeding"} or {"gastrointestinal hemorrhage"}]. Two authors independently reviewed the literature and decided by consensus which articles to incorporate in this study. Two articles written in French were professionally translated into English.[35,36]Table 1 includes only cases of bleeding from PAs of the LGA or its branches and excludes PAs of other arteries arising from the celiac trunk.[37,38] Two cases of bleeding from "gastric arteries" in a study of 104 arterial complications of pancreatitis were excluded because the specific bleeding "gastric" artery and case details were not reported.[39] This case report received exemption/approval by the IRB at William Beaumont Hospital, Royal Oak, on July 21, 2016.

3. Case report

A 41-year-old man with a history of alcoholism for 10 years and sober for the last 3 years, recurrent alcoholic pancreatitis, and no known liver disease, presented with several episodes of hematemesis and abdominal pain for 2 days. Esophagogastroduodenoscopy (EGD), performed 3 years earlier for abdominal pain, had revealed no esophageal varices, gastric varices, portal hypertensive gastropathy, or other GI lesions. Physical examination revealed a blood pressure = 100/60 mm Hg, pulse = 60 beats/min, no jaundice, no stigmata of chronic liver disease, a soft abdomen with mild epigastric tenderness but no rebound tenderness, no abdominal bruit, and no pulsatile abdominal mass. Rectal examination revealed gross melena. Laboratory tests revealed hemoglobin = 12.5 g/dL, platelets = 301,000/mL, INR (international normalized ratio) = 1.0, blood urea nitrogen = 20 mg/dL, and creatinine = 1.1 mg/dL. Serum aspartate aminotransferase = 21 IU/L, alanine aminotransferase = 16 IU/L, total bilirubin = 0.6 mg/dL, alkaline phosphatase = 64 IU/L, albumin = 4.4 gm/dL, and lipase = 32 U/dL. The hemoglobin declined acutely to 9.3 g/dL. Abdomino-pelvic computerized tomography (CT), with intravenous contrast, revealed a 5-cm wide, irregular, pancreatic/peripancreatic mass, compressing both the lesser curvature of the stomach and the SV (Fig. 1A, B), a normal portal vein, and normal liver parenchyma. The SV compression was pathophysiologically significant as indicated by proximal SV dilatation. EGD revealed in the proximal stomach a fine, reticular, pale-white, polygonal, mucosal, network in a snakeskin pattern, and characteristic of portal hypertensive gastropathy that was actively oozing; extensive coffee-ground, blood clots in the stomach; small gastric cardial and fundal varices without stigmata of recent hemorrhage (SRH); and no esophageal varices (Fig. 2). The extrinsic mass produced a large, round bulge extending into the lumen of the proximal gastric body along the lesser curvature (Fig. 2). Magnetic resonance cholangio-pancreatography (MRCP) revealed a 5-cm wide, enhancing, vascular mass likely arising from the LGA and located between the gastric lesser curvature and distal pancreatic body; compressing the stomach; compressing the middle SV; and resulting in large collateral veins draining the SV into the superior mesenteric vein (Fig. 3A, B). Abdominal ultrasound with Doppler studies demonstrated large, turbulent arterial flow into this vascular mass, suggesting a large PA (Fig. 4). Visceral arteriogram showed a 5.3×2.2-cm-wide PA supplied by an LGA branch (Fig. 5A), which was embolized and occluded with microcoils (Fig. 5B). Eight weeks later, the patient had a stable hemoglobin level with no further GI bleeding. Abdomino-pelvic CT angiography demonstrated the PA had markedly decreased in diameter, contained numerous microcoils, and had no blood flow (Fig. 1C).

4. Discussion

Comprehensive literature review revealed 14 cases of LGA PA, including the currently reported case (Table 2[35,36,40–50]). Twelve patients were male and 1 was female (sex not reported in 1 patient). Twelve patients were adults and 1 was an infant, with a mean age of 46.9 ± 20.4 years old (age not reported in 1 patient). PA etiologies included: recurrent/chronic pancreatitis – 9, blunt abdominal trauma – 2, gastric ulcer penetrating into LGA – 1, recent laparoscopic cholecystectomy – 1, and alcoholic cirrhosis – 1. Nine patients had alcoholic pancreatitis and 1 had alcoholic cirrhosis. Pancreatitis is the most common cause of PAs of this branch or other branches of the celiac axis.[40,51,52] Pancreatitis causes leakage of pancreatic enzymes that injure the intima and media, the 2 innermost vascular layers, which then merge with pancreatic pseudocysts to create a false cavity communicating with the arterial lumen.[43,53]

Clinical presentation in the 14 patients included gross upper gastrointestinal (UGI) bleeding – 9, severe anemia without

Table 1

Literature review of reported etiologies of sinistral portal hypertension presenting with gross GI bleeding*.

Underlying pathology causing sinistral portal hypertension	Mechanism	Bleeding manifestation from sinistral portal hypertension	Reference no
Benign pancreatic lesions			
Acute pancreatitis	Splenic vein thrombosis	Isolated gastric varices	[1]
Pancreatic pseudocyst	Splenic vein thrombosis	Isolated gastric varices	[2,3]
Pancreatic pseudotumor (focal pancreatitis in the tail)	Splenic vein compression	Gastroesophageal varices	[4]
Pancreatic cancers			
Pancreatic adenocarcinoma	Splenic vein thrombosis or compression	Isolated gastric varices	[5,6]
Acinar cell pancreatic cancer	Splenic vein thrombosis	Isolated gastric varices	[7]
Neuroendocrine pancreatic tumor	Splenic vein thrombosis	Gastric varices	[8]
Islet cell carcinoma	Splenic vein infiltration	Gastric varices	[9,10]
	Splenic vein compression	Isolated gastric varices	[11,12]
Acinar cell pancreatic cancer	Splenic vein compression and thrombosis	Gastric varices	[13]
Other pancreatic tumors			
MCN of pancreas	Splenic vein compression	Isolated gastric varices	[14]
Solid pseudopapillary neoplasm of pancreas	Splenic vein compression and occlusion	Isolated gastric varices	[15]
Serous cystadenoma of pancreas	Compression of splenic vein	Isolated gastric varices	[16]
Tumors of spleen or other organs			
Hodgkin lymphoma	Splenic vein infiltration	Portal hypertensive gastropathy	[17]
Splenic lymphoma	Splenic vein occlusion	Isolated gastric varices	[18]
Postsurgical changes			
Iatrogenic, from segmental splenic vein resection 8 years earlier	Splenic vein compression	Isolated gastric varices	[19]
Liver transplantation	Ligation of portal tributaries of porto-systemic shunts, proximal splenic vein thrombosis	Variceal bleeding	[20,21]
Vascular lesions/disorders			
Isolated splenic vein thrombosis	Splenic vein thrombosis	Isolated gastric varices	[22]
Splenic artery pseudoaneurysms/aneurysm (s)	Splenic vein compression and thrombosis	Isolated gastric varices	[23]
Left gastric artery pseudoaneurysm	Splenic vein compression	Portal hypertensive gastropathy (based on stigmata of recent hemorrhage [SRH])	Current report
Other disorders			
Umbilical vein catheterization	Splenic vein thrombosis	Isolated gastric varices and gastroesophageal varices	[24]
Wandering spleen	Splenic vein occlusion, torsion of splenic vein	Isolated gastric varices	[25,26]
Perirenal abscess	Isolated splenic vein thrombosis	Isolated gastric varices	[27]
Pregnancy	Splenic vein compression/stenosis	Isolated gastric varices	[28]
Splenic hydatid cysts	Splenic vein compression	Isolated gastric varices	[29]
Gastrectomy	Isolated splenic vein thrombosis	Isolated gastric varices	[30]
Retroperitoneal fibrosis	Isolated splenic vein thrombosis	Gastroesophageal varices	[31]
Myeloproliferative disorders	Splenic vein thrombosis or hepatoportal sclerosis	Isolated gastric varices or gastroesophageal varices	[32]
Tuberculous lymphadenitis	Splenic vein compression	Isolated gastric varices	[33]

GI = gastrointestinal, MCN = mucinous cystic neoplasm.
* Generally only 1 case cited per etiology.

gross GI bleeding – 2, intrapancreatic (retroperitoneal) hemorrhage – 1, intrahepatic (intraperitoneal) hemorrhage – 1, and abdominal pain – 2 ([Table 2]; 1 patient had 2 presentations). Etiologies of gross UGI bleeding in the 9 patients included: PA penetrating through gastric wall into gastric lumen – 5, peptic ulcer eroding into PA in gastric wall – 2, hemosucuss pancreaticus from PA eroding into pancreatic duct – 1, and gastric oozing from portal hypertensive gastropathy with sinistral portal hypertension – 1. PAs commonly present with upper or lower GI bleeding due to the underlying vascular injury.[42]

Abdominal CT findings of enhancement of a pancreatic pseudocyst or of an adjacent vascular structure are highly suspicious for PAs. However, endoscopic ultrasound (EUS) is superior to CT for diagnosing small PAs.[43,54,55] PAs usually appear on transabdominal ultrasonography as anechoic, frequently pulsatile lesions, and often contain a cyst within a larger anechoic mass. Color and pulse Doppler sonography further enhance test sensitivity and specificity by demonstrating turbulent blood flow ("to and fro" sign) within the PA. Angiography is the gold standard diagnostic test: it identifies the affected artery, determines local vascular anatomy, and permits interventional therapy.[55] This case represents the first case of LGA PA diagnosed by MRCP. Abdominal CT with intravenous contrast showed a pancreatic/peripancreatic mass, initially suspicious for pancreatic adenocarcinoma, but MRCP demonstrated that the mass was vascular, and likely originated from the LGA.

Only 2 of the 14 patients had sinistral portal hypertension. In sinistral portal hypertension, defined as partial portal hypertension without cirrhosis from venous obstruction proximal to the

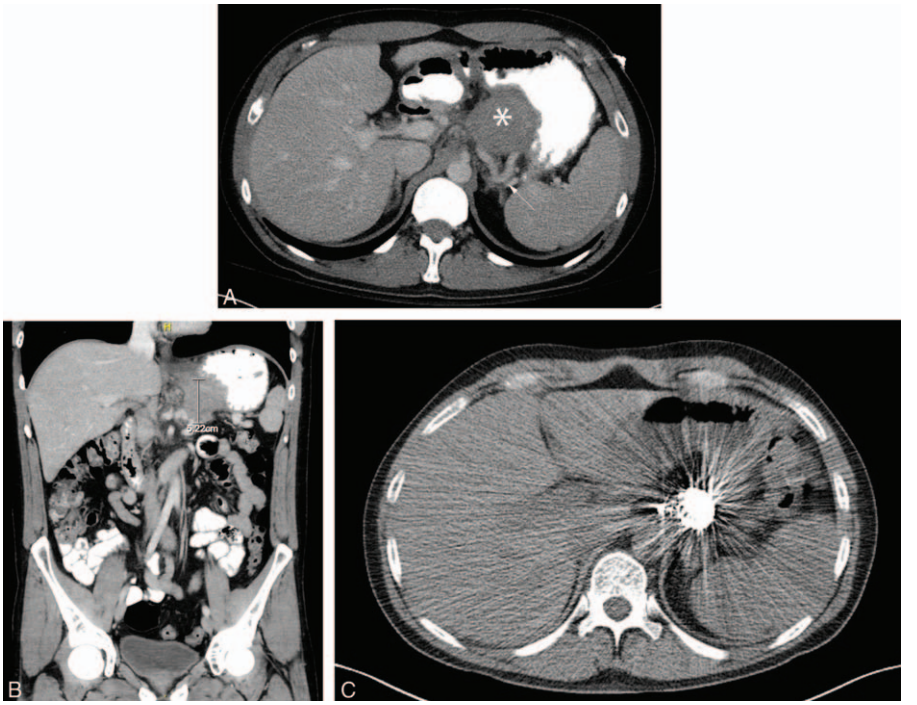

Figure 1. (A) Axial view of abdominal CT, with IV contrast, shows a 5.2 cm pancreatic/peripancreatic mass (labeled*) compressing the stomach (which has a radiopaque penumbra due to orally ingested contrast) along the lesser curve, and shows that this mass causes a kink (arrow) that partially obstructs the splenic vein and causes sinistral portal hypertension. (B) Coronal view of abdominal CT, with IV contrast, shows a 5.22 cm pancreatic/peripancreatic mass extending superiorly and compressing the stomach, with a radiopaque penumbra due to orally ingested contrast along the lesser curve, and extending inferiorly to the pancreas. (C) CTA 8 weeks after angiographic embolization of left gastric artery demonstrates the pseudoaneurysm has markedly decreased in size, contains metallic coils (producing a metal streak artifact), and has no blood flow. Occlusion of the pseudoaneurysm reverses the splenic vein compression and relieves the sinistral portal hypertension responsible for bleeding from portal hypertensive gastropathy. CT = computerized tomography, CTA = computerized tomographic angiography, IV = intravenous.

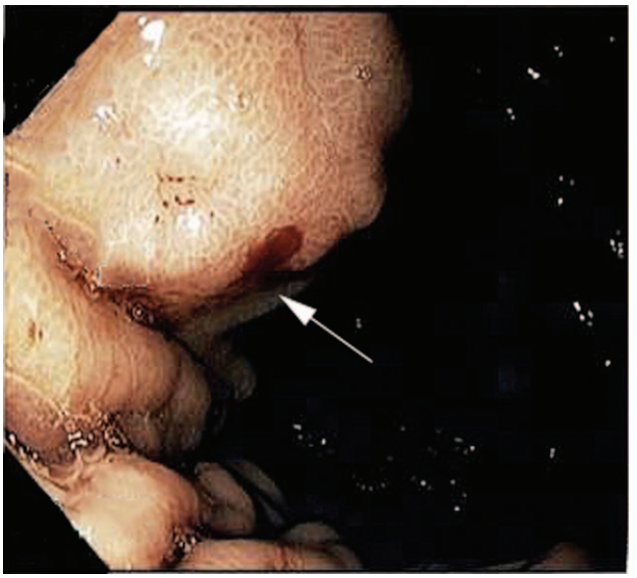

Figure 2. Endoscopic retroflexion at esophagogastroduodenoscopy shows a prominent, pale white, reticular, network surrounding individual pink polygons, a characteristic finding of portal hypertensive gastropathy, in the proximal gastric body. This mucosa bulges into the gastric lumen due to extrinsic compression by the pseudoaneurysm. This lesion is actively oozing, as evidenced by stigmata of recent hemorrhage: an intensely erythematous, oozing, lesion on the dependent side of the bulge (arrow) within the area of portal gastropathy. The proximal stomach along the greater curvature is dark because of the presence of dark blood clots within the lumen.

portal vein (typically occurring at the SV), patients commonly bleed from gastric varices or uncommonly from portal hypertensive gastropathy, but do not bleed from esophageal varices because the portal hypertension only affects the stomach. One prior patient had sinistral portal hypertension from LGA PA compression of the SV; but this patient presented with anemia (hemoglobin = 7.1 g/dL) without gross bleeding and had gastric varices identified by EGD which presumably caused the anemia.[46] The currently reported patient is the first presenting with gross bleeding from sinistral portal hypertension from SV compression by an LGA PA. This patient had hematemesis from portal hypertensive gastropathy (based on endoscopic SRH), without liver disease, from sinistral portal hypertension.

Among the 9 patients with acute UGI bleeding, angioembolization achieved hemostasis in 5 of 7 cases, surgery was successful in 2 of 3 cases, and dual mode intraoperative angiography and surgery was successful in 1 case. In the current case, hemostasis was achieved by angioembolizing the LGA to relieve SV compression and reverse the sinistral portal hypertension. Angioembolization provides definitive therapy in about 78% of all bleeding visceral PAs, and provides partially successful therapy to stabilize a patient before definitive surgical therapy in many of the remaining cases.[55] Rebleeding is the most common complication of angioembolization.[53] Alternative therapies include endovascular stents or vascular surgery which is reserved for patients who are appropriate surgical candidates, and who have either failed angioembolization or have other surgical indications, such as pancreatic

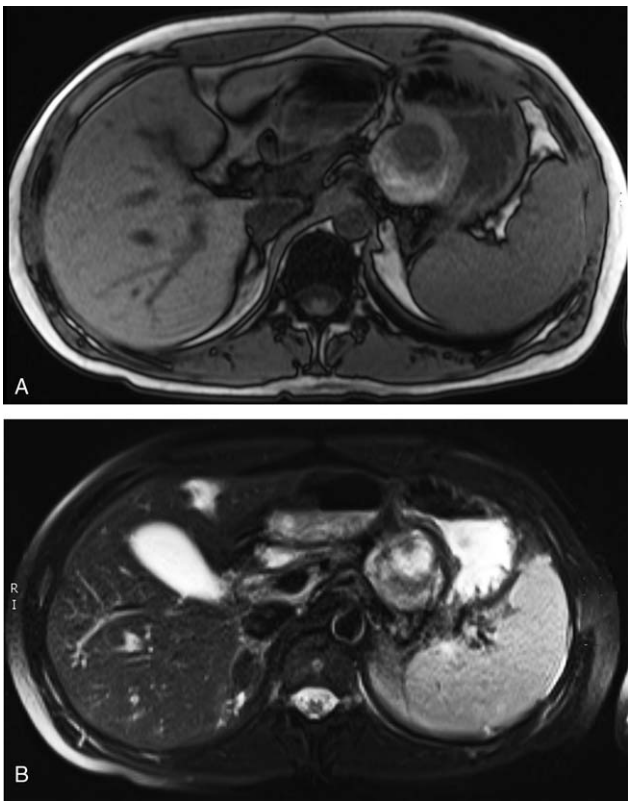

Figure 3. (A, B) Abdominal MRCP shows a 5-cm-wide mass compressing the gastric mid-body, along the lesser curve. The mass is radiolucent before IV contrast administration (A), but markedly enhances after contrast administration (B), a temporal pattern consistent with a vascular mass. The stomach has a moderately radiopaque penumbra from ingested oral contrast. IV = intravenous, MRCP = magnetic resonance cholangio-pancreatography.

pseudocyst, pancreatic abscess, or gastric outlet obstruction.[40,55] It is hoped that prompt angioembolization may reduce the previously reported mortality of bleeding from visceral PAs of 60%.[43]

Splenic vein thrombosis (SVT) also causes GI bleeding associated with pancreatitis from formation of gastric varices due to sinistral portal hypertension.[56] SVT should be suspected in patients with pancreatic disease, especially pancreatitis; splenomegaly without generalized portal hypertension or cirrhosis; and isolated gastric varices.[57] Notably, this work illustrates different therapies depending upon the etiology: sinistral portal hypertension caused by LGA PA compressing the SV should be treated by obliterating the PA, whereas sinistral portal hypertension caused by SVT should be treated by splenic artery embolization, SV recanalization, or splenectomy.[56] The pathophysiology of UGI bleeding for sinistral portal hypertension from LGA is illustrated in Fig. 6.

Study limitations include: this represents only 1 case report, the methodology is retrospective, bleeding from leakage from the PA into the gastric lumen or from the small gastric varices cannot be excluded as possible etiologies of the UGI bleeding, and bleeding from portal hypertensive gastropathy, rather than gastric varices, is based primarily on endoscopic SRH. Bleeding from sinistral portal hypertension is usually from gastric varices, but in this reported case was apparently from portal hypertensive gastropathy, as demonstrated by endoscopic SRH. The observed gastric varices were small and lacked SRH, and the bleeding was mild, more consistent with portal hypertensive gastropathy than gastric varices.[58] A prospective clinical series is unlikely because this syndrome is rare.

In conclusion, novel findings in this case include: gross gastric bleeding from sinistral portal hypertension secondary to an LGA PA compressing the SV, and hemostasis achieved by occluding the PA to relieve SV compression.

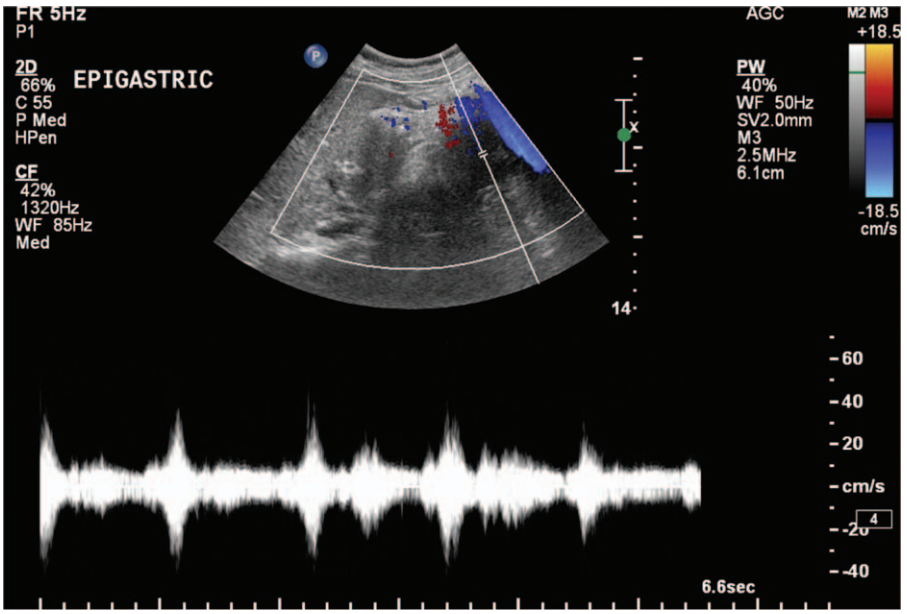

Figure 4. Abdominal ultrasound with Doppler studies reveals a strong arterial waveform in tracing on bottom of screen, indicating a vascular, arterial mass. Blue and red Doppler signals within the mass demonstrate turbulent (to-and-fro) blood flow.

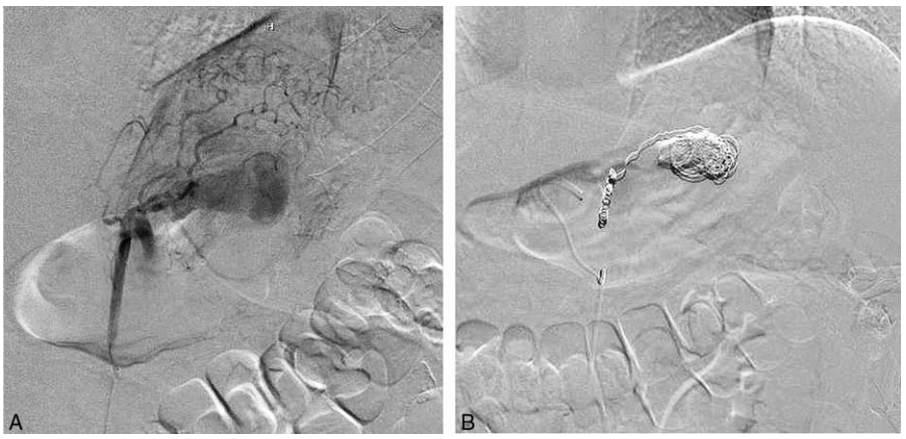

Figure 5. Arteriogram during arterial phase after selective injection of contrast into left gastric artery shows pseudoaneurysm filled with injected contrast (A, before embolization); and shows no blood flow (no contrast) within pseudoaneurysm after angiographic coil embolization (B, note coils in pseudoaneurysm and left gastric artery).

Table 2

Fourteen reported cases of left gastric artery pseudoaneurysm including the current case: clinical presentation, diagnostic evaluation, pathophysiology, therapy, and outcome.

Clinical presentation: age in years, sex, symptoms, signs, and laboratory findings	Tests including EGD, radiologic imaging, and surgical findings	Causality	Sinistral portal hypertension	Therapy	Outcome	Reference no
Presenting with GI bleeding						
41-year-old M with recurrent alcoholic pancreatitis and no known liver disease presented with hematemesis, melena, and abdominal pain for 2 days. BP = 100/60 mm Hg. Hgb declined from 12.5 to 9.3 g/dL.	Abdominal CT with IV contrast: 5-cm-wide peripancreatic mass compressing splenic vein and gastric lesser curve. EGD: Active oozing from portal hypertensive gastropathy. Bulge into proximal stomach lumen. MRCP: vascular mass derived from LGA. Visceral angiography: 5.3 × 2.2 LGA branch PA.	GI bleeding from portal hypertensive gastropathy (sinistral portal hypertension) from splenic vein compression by LGA aneurysm caused by recurrent pancreatitis.	Yes	Angiographic microcoil embolization and occlusion of LGA	No further bleeding during hospitalization and during 8 weeks afterwards	Current report
40-year-old M with chronic alcoholic pancreatitis presented with melena and abdominal pain. Pancreatic duct stent placed 1 month earlier. Pulse = 145 beats/min. Epigastric tenderness. Hgb = 7.1 g/dL, lipase = 466 IU/L.	EGD: protruding mass in upper gastric body with central red spot. Abdominal CT: contrast leaking into oval structure between stomach and pancreatic tail. Celiac angiography: bleeding from 2.0 × 1.3 cm PA of LGA branch that has ruptured into stomach.	Bleeding from PA rupture through stomach wall into gastric lumen. PA secondary to chronic alcoholic pancreatitis	No	Angiographic microcoil embolization and occlusion	No further bleeding	[40]

(*continued*)

Table 2

(continued).

Clinical presentation: age in years, sex, symptoms, signs, and laboratory findings	Tests including EGD, radiologic imaging, and surgical findings	Causality	Sinistral portal hypertension	Therapy	Outcome	Reference no
53-year-old M with Child C alcoholic cirrhosis presented with acute hematemesis.	EGD: Large extrinsic mass and ulceration of posterior lesser curvature of stomach. Abdominal CT: 4-cm enhancing mass between gastric fundus and pancreatic tail. EUS: 3.7 × 2.5 cm "cystic" lesion with large pulsating artery at margin of lesion (suspected ruptured PA). CT-angiography: PA of left gastric artery branch.	Bleeding from LGA PA reaching to gastric mucosa with overlying gastric mucosal ulcer.		Angiographic microcoil embolization and occlusion	No further bleeding during hospitalization	[41]
37-year-old alcoholic F with chronic alcoholic pancreatitis presented with hematemesis. Pulse = 111 beats/min, blood pressure = 85/51 mm Hg. Hgb = 6.6 g/dL.	EGD: pulsating bulge in proximal gastric body with a central red spot from PA. Abdominal CT: 3.0 × 4.5 × 2.8 cm partly thrombosed PA between pancreatic body and posterior curvature of stomach. Celiac angiography: PA of terminal branch of LGA.	Bleeding from PA, secondary to chronic alcoholic pancreatitis, rupturing into stomach.	No	Angioembolization	Successful	
65-year-old M smoker with COPD and chronic alcoholic pancreatitis presented with severe UGI bleeding. Pulse = 155/min, blood pressure = 70/50 mm Hg. Epigastric pulsatile mass. Hgb = 7.2 g/dL.	Abdominal CT: 7-cm-wide mass along gastric lesser curve consistent with PA of LGA.	Apparently bleeding from PA (secondary to chronic alcoholic pancreatitis) rupturing into gastric lumen via gastric wall.		Massively bleeding LGA PA first controlled by inflating balloon in left femoral artery. Laparotomy: cyst deroofed and sutured. Occluded LGA PA	Successful. No further bleeding during hospitalization. Discharged 5 days after surgery	[35]
55-year-old M with chronic alcoholism for 10 years, with hematemesis and melena for 1 month.	EGD: blood oozing from prominent gastric folds below cardia on posterior gastric wall. CT: thickened posterior gastric wall, chronic pancreatitis, and small pancreatic pseudocyst. EUS and Doppler: chronic pancreatitis, 1.5-cm-wide pancreatic tail PA. CT with IV contrast: left gastric artery PA, splenic vein occluded. Angiography: left gastric artery PA.	GI bleeding from PA likely eroding towards gastric lumen (fistula from aneurysm to gastric lumen). PA due to chronic pancreatitis.	Unlikely (no gastric varices at EGD)	Angioembolization failed (recurrent bleeding). Laparotomy with sleeve gastrectomy, splenectomy, and ligation of left gastric artery	Died from septic shock after laparotomy	[43]
50-year-old M with chronic alcoholic pancreatitis presented	EGD: blood in stomach. Blood oozing from ampulla of Vater.	Bleeding from LGA PA between neck of	Unlikely	Unable to perform superselective cannulation for	Discharged after 10 days with no further bleeding.	[44]

(*continued*)

Table 2
(continued).

Clinical presentation: age in years, sex, symptoms, signs, and laboratory findings	Tests including EGD, radiologic imaging, and surgical findings	Causality	Sinistral portal hypertension	Therapy	Outcome	Reference no
with epigastric pain and melena for 2 days. Blood pressure = 100/60 mm Hg, pulse = 110/min, periumbilical tenderness. Hgb = 7.0 g/dL. NG tube revealed gross blood.	Abdominal CT with IV contrast: 3-cm-wide PA between lesser curve of stomach and pancreatic neck. Visceral angiography: LGA PA likely actively bleeding. Surgical laparotomy: 3-cm-wide pseudocyst between neck of pancreas and gastric lesser curve. LGA PA revealed by deroofing the pseudocyst.	pancreas and lesser curve of stomach.		angiographic embolization. Surgery: PA closed off and excised	Asymptomatic during following 10 months while abstinent from alcohol	
"Upper gastrointestinal bleeding" from gastric ulcer.	EGD: GI bleeding from ulcerated gastric mass at lesser curve of stomach. Abdominal CT: homogeneously enhancing mass at lesser curve of stomach consistent with PA. CT angiography: LGA PA.	UGI bleeding from rupture of LGA PA due to penetrating gastric ulcer.	No	Surgery: ligated LGA PA and excised gastric ulcer. No angiography available at hospital	Survived	[45]
81-year-old M with chronic alcoholic pancreatitis for 15 years presented with melena and weakness. BP = 80/60 mm Hg, pulse = 100/min. Hgb = 2.6 g/dL. Transfused 12 units packed erythrocytes.	Abdominal USD: pancreatic duct dilated to 3.1 cm. EGD: fresh blood in duodenum, Acute bleeding from papilla (pancreatic duct). Abdominal CT: fluid-filled cavity in pancreatic head and nearby cyst that enhances with IV contrast (suspected arterial PA).	GI bleeding from pancreatic duct (hemosuccus pancreaticus) due to left gastric artery PA secondary to chronic alcoholic pancreatitis.	Not evident	Angiographic embolization of left gastric artery branch that occluded flow to PA	Discharged after 7 days with no further GI bleeding. Asymptomatic during 6 months of follow-up	[36]
Anemia without overt GI bleeding						
72-year-old M with history of resolved pancreatic pseudocysts from alcoholic pancreatitis presented with anemia, Hgb = 7.1 g/dL. No overt GI bleeding.	EGD: nonbleeding gastric varices. Colonoscopy: 1 benign polyp. Abdominal CT with contrast: heterogeneous mass in pancreatic tail extending to gastric body. EUS: 5 × 2-cm mass with large internal flow. Abdominal MRI: 4-cm-wide PA at distal body of pancreas. Angiography: LGA	GI bleeding most likely from gastric varices (sinistral portal hypertension?)	Yes	Angioembolization	Successful	[46]

(*continued*)

Table 2

(continued).

Clinical presentation: age in years, sex, symptoms, signs, and laboratory findings	Tests including EGD, radiologic imaging, and surgical findings	Causality	Sinistral portal hypertension	Therapy	Outcome	Reference no
	arising directly from aorta and containing a PA.					
1.6-year-old emaciated infant, M with abdominal pain, and abdominal distension from blunt abdominal trauma. Epigastric abdominal mass. Hgb = 6.5 g/dL. Normal liver function tests. No overt GI bleeding.	Abdominal CT: PA and hematoma in left lobe of liver. Doppler ultrasound: flow in lesion consistent with PA. Abdominal MRI: PA between stomach and left lobe of liver.	Bleeding from blunt trauma to abdomen that caused rupture of anomalous LGA with PA in branch supplying left lobe of liver.	Not described	LGA angioembolization with mcrocoils	Successful	[47]
	Visceral angiography: left hepatic artery arising from anomalous left gastric artery with PA of left gastric artery branch.					
Intraabdominal or intrapancreatic hemorrhage						
45-year-old M with left upper quadrant pain which complicated pancreatitis.	Abdominal CT (noncontrast): 9.5 × 6.0-cm hemorrhagic mass in pancreatic body. Abdominal CT with IV contrast: 2.4-cm-wide ruptured PA of LGA within a large pseudocyst. Angiography: leakage from LGA PA.	Bleeding from ruptured LGA PA in pancreas. PA due to pancreatitis. No suspected sinistral portal hypertension.	No	Angiographic embolization of LGA PA within pancreas	NA	[48]
24-year-old M with massive intraabdominal bleeding 2 weeks after distal pancreatectomy and splenectomy for pancreatic transection from blunt abdominal trauma.	Abdominal CT with IV contrast: hematoma inferior to liver.	Bleeding from PA of LGA branch after undergoing distal pancreatectomy for pancreatic transection from blunt abdominal trauma.	No	Angiographic embolization of LGA branch PA	Successfully occluded flow. No further bleeding during hospitalization	[49]
	Digital subtraction angiogram: PA of descending branch of LGA.					
Abdominal pain						
45-year-old M with epigastric pain and nausea 5 weeks after laparoscopic cholecystectomy for recurrent gallstone pancreatitis. Had abdominal tenderness without guarding.	Abdominal CT (no IV contrast): 38 mm wide spherical structure apparently within wall of proximal body of stomach. Streaky peripancreatic fat from acute pancreatitis. Abdominal CT with IV contrast: central enhancement of lesion. Visceral angiography: PA of cephalic branch of LGA.	Bleeding from PA of LGA branch (within gastric wall). PA consequent to acute pancreatitis.	No	Angiographic embolization of LGA branch PA	Successful	[50]

CT = computerized tomography, EGD = esophagogastroduodenoscopy, F = female, GI = gastrointestinal, Hgb = hemoglobin, IV = intravenous, LGA = left gastric artery, M = male, NA = not available, PA = pseudoaneurysm, UGI = upper gastrointestinal.

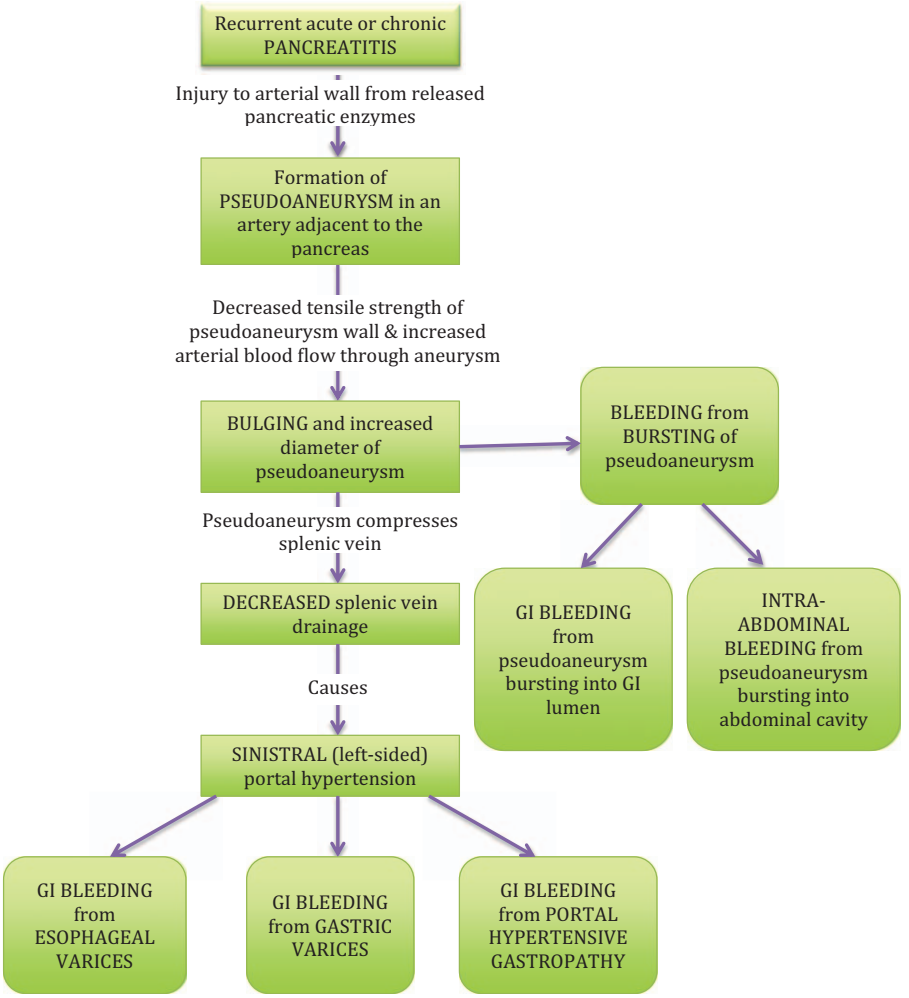

Figure 6. Flow diagram illustrates the pathophysiology of bleeding from sinistral portal hypertension from splenic vein compression by an adjacent left gastric artery (LGA) pseudoaneurysm from chronic or recurrent acute pancreatitis.

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