Inferior Pancreaticoduodenal Artery Aneurysms in Association with Celiac Stenosis/Occlusion

Rekha Kallamadi, M.D.,[1,3] Marc A. deMoya, M.D.,[2,3] and Sanjeeva P. Kalva, M.D.[1,3]

ABSTRACT

Inferior pancreaticoduodenal artery aneurysms in association with celiac stenosis or occlusion are well described in the literature. These aneurysms are true aneurysms and develop as a result of increased flow through the pancreaticoduodenal arcades in the presence of hemodynamically significant stenosis of the celiac axis or common hepatic artery. Aneurysms may be multiple and rarely associated with aneurysms in other collateral pathways—such as the dorsal pancreatic artery or the arc of Buhler. These aneurysms may be incidentally detected or patients may present with abdominal pain or shock secondary to rupture of the aneurysms. Treatment options include surgical resection and transcatheter embolization; current literature favors the latter option. Treatment of celiac axis stenosis may be recommended in addition to treating the aneurysms; however, no formal guidelines exist on this recommendation.

KEYWORDS: Inferior pancreaticoduodenal artery aneurysm, celiac axis stenosis, embolization

Objectives: Upon completion of this article, the reader should be able to (1) explain the incidence, etiopathogenesis, and clinical presentation of inferior pancreaticoduodenal artery aneurysms associated with celiac stenosis/occlusion; (2) assess the current literature on the diagnosis and management of these aneurysms; and (3) state current accepted guidelines for the diagnosis and management of these aneurysms.
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Aneurysms of the pancreaticoduodenal arcades were described as early as late 18th century.[1] These are often associated with pancreatitis,[2] Whipple's procedure,[3] trauma,[4] cholecystitis,[5] and rarely, vasculitis.[6] Such aneurysms are often pseudoaneurysms secondary to inflammation, infection, or trauma. True aneurysms of the pancreaticoduodenal arcades are rare.[7] These may be congenital[8] or acquired secondary to fibromuscular dysplasia[9] or atherosclerosis.[10] True aneurysms of the pancreaticoduodenal arcades in the presence of celiac

[1]Department of Radiology (Division of Cardiovascular Imaging and Intervention); [2]Department of Surgery (Division of Trauma, Emergency Surgery, Surgical Critical Care), Massachusetts General Hospital; [3]Harvard Medical School, Boston, Massachusetts.

Address for correspondence and reprint requests: Sanjeeva P. Kalva, M.D., Department of Radiology (Division of Cardiovascular Imaging and Intervention), 55 Fruit Street, GRB-290, Boston, MA 02114 (e-mail: skalva@partners.org).

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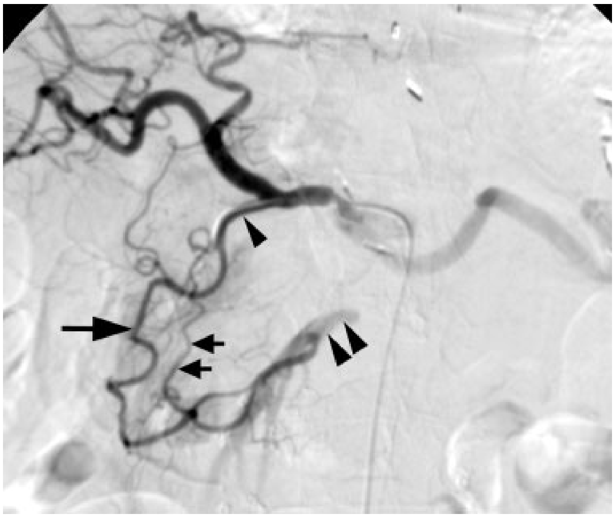

Figure 1 Normal anatomy of the pancreaticoduodenal arcades. Selective arteriogram from the gastroduodenal artery (single arrowhead) shows the anterior (black arrow) and posterior (two black arrows) pancreaticoduodenal arcades, which opacify the superior mesenteric artery (two arrowheads).

Figure 2 Normal anatomy of the dorsal pancreatic artery. Selective arteriogram after accidental catheterization of the dorsal pancreatic artery. The dorsal pancreatic artery (small black arrow) arises from the common hepatic artery (white vertical arrow). The right branches (black arrowheads) of the dorsal pancreatic artery communicate with the pancreaticoduodenal arcades (big black arrow). The vertical branch (white arrowhead) connects to the superior mesenteric artery (SMA) (white horizontal arrow) and forms the longitudinal collateral pathway between the celiac axis and the SMA.

artery stenosis or occlusion were first described by Sutton,[11] and later supported by further observations of Kadir et al.[12] In such cases, celiac axis stenosis may be secondary to atherosclerosis[10] or due to compression by the median arcuate ligament.[13–20] Additionally, a few reports described occurrence of these aneurysms in the presence of common hepatic artery occlusion.[21] The pathophysiology of these aneurysms is poorly explained, but appears to be related to increased flow in these vessels in the presence of celiac or common hepatic artery stenosis/occlusion.[12]

ANATOMY OF THE PANCREATICODUODENAL ARCADES

The superior and inferior pancreaticoduodenal arteries arise from the gastroduodenal artery and the superior mesenteric artery, respectively.[22] The superior pancreaticoduodenal artery divides into anterior and posterior branches, which communicate with corresponding branches of the inferior pancreaticoduodenal artery. The inferior pancreaticoduodenal artery may arise directly from the superior mesenteric artery or from the first jejunal branch and divides into anterior and posterior branches. The anterior and posterior branches of these vessels form the "pancreaticoduodenal arcades" (Fig. 1). The posterior arcade is more cephalad, and runs posterior to the head of the pancreas. Often, multiple arcades may exist.[22] The arcades communicate with the transverse branch of the dorsal pancreatic artery (Fig. 2).[22] In addition, a few unnamed pancreatic arteries communicate with the arcades. All these vessels form a network of collateral pathways between the celiac artery and superior mesenteric artery. As such, aneurysms may occur in any of these vessels in the presence of celiac axis stenosis/occlusion.

In addition to the pancreaticoduodenal arteries, other forms of collateralization between the celiac axis and the superior mesenteric artery include the arc of Buhler (an inconsistent, embryonic vessel that directly communicates the proximal celiac axis to the proximal segment of the superior mesenteric artery (Fig. 3),[23] the dorsal pancreatic artery,[22] and the arcs of Barkow (the collateral pathway within the omentum, between the epiploic arteries of the splenic artery and superior mesenteric artery).[24] These collaterals may also enlarge and form aneurysms in the presence of celiac axis stenosis or occlusion.[25]

PATHOPHYSIOLOGY OF INFERIOR PANCREATICODUODENAL ARTERY ANEURYSMS

Current evidence supports the theory that increased flow through the small, fragile pancreaticoduodenal arteries

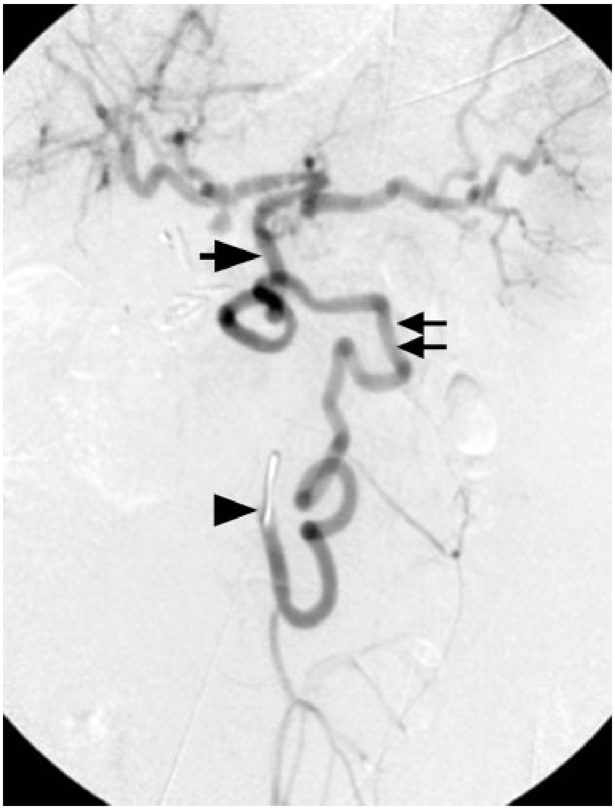

Figure 3 Arc of Buhler. Selective arteriogram from the inferior mesenteric artery (arrowhead) in a 75-year-old man presenting with mesenteric ischemia demonstrates a large arc of Riolan (two arrows), that communicates with the occluded superior mesenteric artery (SMA). The arc of Buhler (arrow) runs vertically from the SMA reconstituting the celiac axis.

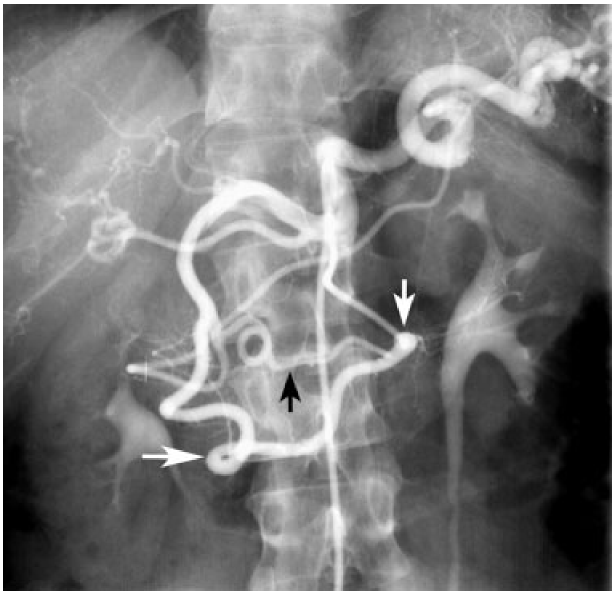

Figure 4 Preferential enlargement of the anterior pancreaticoduodenal arcade in a patient with celiac axis occlusion. Selective arteriogram from inferior pancreaticoduodenal artery (vertical white arrow) demonstrates enlarged anterior (horizontal white arrow) pancreaticoduodenal arcade and a small posterior (black arrow) arcade.

in the presence of celiac axis stenosis/occlusion may be responsible for the formation of the aneurysms. First, the vessels enlarge to accommodate the increased flow (Figs. 4, 5). In a few cases, the persistent increased flow and high intraarterial pressure lead to weakening of the vessel wall and the formation of a true aneurysm.[25] The aneurysms may be multiple and may occur in the pancreaticoduodenal arcades or in other collateral vessels such as the Arc of Buhler or dorsal pancreatic artery. The aneurysms may be asymptomatic or may present with symptoms related to extrinsic compression of the bowel or biliary ducts. The rupture of these aneurysms may lead to the acute clinical presentation of hemorrhagic shock.

CLINICAL PRESENTATION

The most common clinical presentation of an unruptured aneurysm is vague abdominal pain (Table 1).[4,7,8,13–20,25–37] This may be related to extrinsic compression on the duodenum or pancreatic and biliary ducts. Jaundice as a presenting clinical symptom is rare.[38] Patients may present with gastrointestinal bleeding,[13,29] probably secondary to rupture of the aneurysms into the duodenum and/or pancreatic duct. A few patients may present with acute abdominal pain and shock due to rupture of the aneurysm. With the advent of computed tomography (CT), many asymptomatic aneurysms are diagnosed at an early stage.[8,10,15,26,34–37]

RISK OF RUPTURE

The risk of rupture appears to be unrelated to the size of the aneurysm (Table 1). In the published reports the size of ruptured aneurysms ranged from 0.6 cm to 2 cm (Table 1). The size of unruptured aneurysms ranged from 0.7 cm to 6 cm (Table 1). There appears to be no relation between the risk of rupture and multiplicity of the aneurysms (Table 1). Similarly, no association has been found between age of the patient or cause of the stenosis and risk of aneurysm rupture. The median age of patients who presented with unruptured aneurysm was 52 years whereas the median age of patients with ruptured aneurysm was 57 years (Table 1).

DIAGNOSIS

Contrast-enhanced multidetector CT is very useful in diagnosing symptomatic and asymptomatic aneurysms

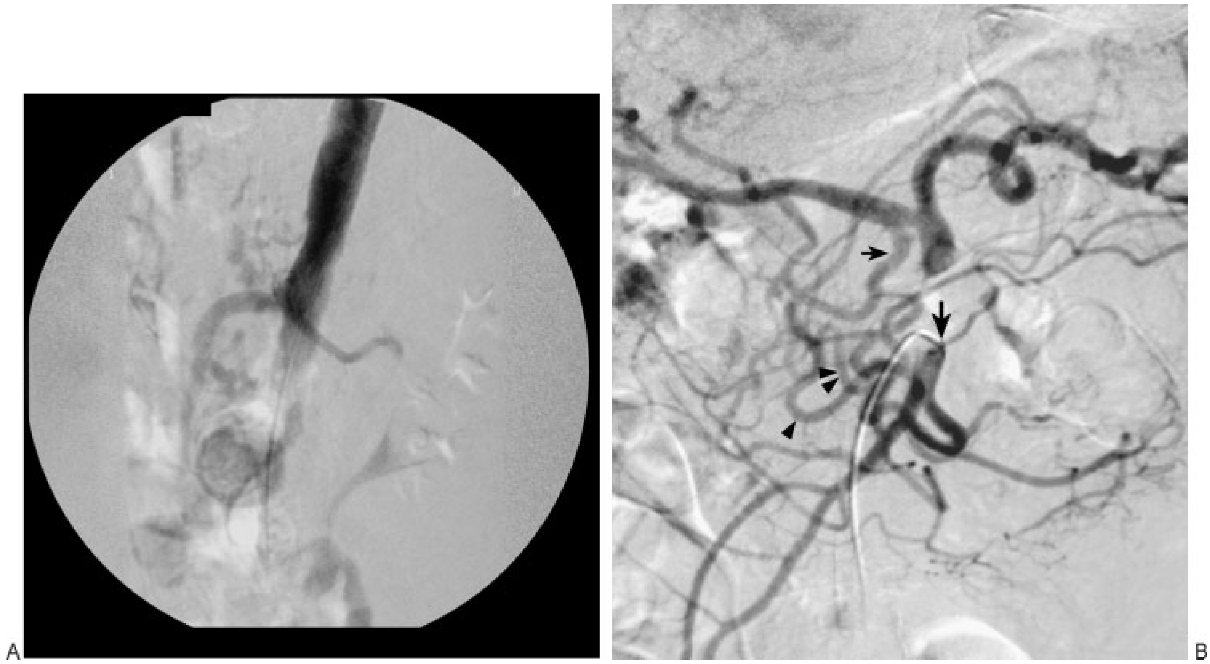

Figure 5 Enlarged dorsal pancreatic artery and pancreaticoduodenal arcades in a patient with celiac axis occlusion. (A) A lateral abdominal aortogram shows occluded celiac axis (large arrow) and patent superior mesenteric artery (SMA) (small arrow). (B) Selective arteriogram from the SMA (vertical arrow) demonstrates mildly enlarged anterior (single arrowhead) and posterior (two arrowheads) pancreaticoduodenal arcades and dorsal pancreatic artery (horizontal arrow). Multiple vascular loops are the result of enlargement of multiple collateral vessels.

(Figs. 6, 7). Special attention must be paid to slice thickness (preferably 0.625 mm or less), contrast material administration, and three-dimensional reconstruction algorithm.[39] Large aneurysms may be visible on ultrasound examination with the aid of color flow imaging. However, the aneurysm and its parent arterial supply are best assessed with contrast-enhanced CT.

Conventional catheter angiography remains the gold standard for diagnosing these aneurysms (Fig. 8). Though aortography is useful in diagnosing large aneurysms, selective catheter injections through the superior mesenteric artery, inferior pancreaticoduodenal artery, celiac artery, and gastroduodenal artery are highly recommended for diagnosing small aneurysms (Fig. 8). These may also help assess aneurysms in other collateral pathways and provide a roadmap for further superselective angiography and embolization.

TREATMENT

Many options exist for therapy of these aneurysms. Surgical resection, endoaneurysmorrhaphy, and ligation are surgical options.[8,14,18,25,26,28] They may be combined with treatment of celiac axis stenosis. Given the location of these aneurysms, pancreaticoduodenectomy may be necessary to remove the aneurismal arcade.

An endovascular approach to the treatment of these aneurysms has many advantages. It is less invasive than surgery and can be safely applied with minimal complications. It better defines the aneurysm and the parent vessel, and allows one to selectively embolize the aneurysm, sparing other vessels (Fig. 9). This may be combined with revascularization of the celiac stenosis.

Endovascular coil embolization has been shown to have excellent long-term outcomes in the treatment of these aneurysms. Neither recurrence of the aneurysms nor recurrent bleeding have been reported after successful occlusion of the aneurysms.[4,16,19,29,30] In addition to coils, other embolic agents (such as thrombin, *N*-acetyl cyanoacrylate, and Onyx® [Ev3 Endovascular, Inc., Plymouth, MN])[4] have been used successfully for embolization. However, tortuosity of the inferior pancreaticoduodenal artery may present difficulties to selectively embolize the aneurysms. In such cases, CT-guided percutaneous approach to directly access the aneurysm may be helpful.[40] The aneurysm may be embolized with coils or *N*-acetyl cyanoacrylate.

Treatment of celiac artery stenosis in addition to the aneurysm therapy remains controversial. Though it appears logical that relieving the celiac stenosis would decrease recurrence of aneurysms in the pancreaticoduodenal arcades, no recurrences have been reported in the

Table 1 Summary of Recent Publications on Inferior Pancreaticoduodenal Artery Aneurysms Associated with Celiac Axis Stenosis/Occlusion

SN	Author	Year	No. of Patients	Age	Sex	Clinical Presentation	Ruptured/ Unruptured	Celiac Stenosis/ Occlusion	No. of Aneurysms	Size	Treatment	Follow-up
1	Hildebrand et al[26]	2007	3	66	F	Asymptomatic	UR	Y	1	2.8 cm	Surgery	NA
				53	F	Gallstones	UR	Y	1	2.9 cm	Surgical resection	NA
				48	F	Epigastric pain	UR	Y	1	2.7 cm	Surgical resection	NA
2	Ikeda et al[20]	2007	3	62	F	Asymptomatic	UR	Y MALC	1	20 mm × 18 mm	TAE	6 months
				46	F	Asymptomatic	UR	Y MALC	1	11 mm × 10 mm	TAE	6 months
				48	M	Asymptomatic	UR	Y	1	12 mm × 13 mm	TAE	6 months
3	Bageacu et al[27]	2006	1	55	NA	NA	R	Y	1	15 mm	TAE	89 months
			1	43	NA	NA	R	Y	1	20 mm	TAE	78 months
			1	51	NA	NA	UR	Y	1	18 mm	TAE	27 months
4	Sugiyama & Takehara[17]	2006	5	57	F	Abdominal pain	NA	Y MALC	1	8 mm	TAE	NA
				66	M	Abdominal pain	NA	Y MALC	1	15 mm	TAE	NA
				65	M	Asymptomatic	UR	Y MALC	1	20 mm	Observation	NA
				73	M	Asymptomatic	UR	Y MALC	1	7 mm	Observation	NA
				61	M	Abdominal pain	NA	Y MALC	1	5 mm	TAE	NA
5	Tori et al[10]	2006	1	37	M	Asymptomatic	UR	Y AS	Multiple	2 cm	Minimally invasive surgery	28 months
6	Bratby et al[4]	2006	1	37	M	Abdominal pain	UR	Y	1	4 × 6 cm	TAE with Onyx®	3 months
7	Ignashov et al[18]	2005	2	NA	NA	Operative Rx of celiac stenosis	UR	Y MALC	1	NA	TAE	NA
				NA	NA	Operative Rx of celiac stenosis	UR	Y MALC	1	NA	Surgical resection	NA
8	Bellosta et al[28]	2005	3	72	F	Dyspepsia, epigastric discomfort	UR	Y	1	8 mm	Endoaneurysmectomy	2 years
				34	F	Intermittent epigastric pain	UR	Y	1	28 mm	Surgical resection	6 days
				74	F	Asymptomatic	UR	Y	1	30 mm	Surgical resection	9 days
9	Calkins et al[29]	2004	1	63	F	Retroperitoneal hemorrhage	R	Y	Multiple	NA	TAE	3 months
10	Shibata et al[30]	2004	2	64	M	Abdominal fullness	UR	Y	1	3.6 cm	TAE	30 months
				72	F	Abdominal pain, mild liver dysfunction	UR	Y	1	5.5 cm	TAE	18 months
11	Guijt et al[31]	2004	2	63	F	Abdominal pain radiating to back	R	Y		NA	TAE	2 months
				67	F	Abdominal pain	R	Y	1	NA	TAE	1 month
12	Kobayashi et al[25]	2004	1	57	F	Abdominal pain	R	Y	1	1 cm	TAE	2 years
13	Tien et al[32]	2004	2	66	M	Abdominal pain	R	Y	1	1.5 cm	Surgical resection	3 years
				46	F	Abdominal pain	R	Y	1	2 cm	Surgery + celiac stent	2 years
14	Ducasse et al[15]	2004	1	54	M	Asymptomatic	R	Y MALC	2	6 mm, NA	TAE	3–4 months
15	Akatsu et al[16]	2004	1	49	F	Abdominal pain	R	Y MALC	1	1 cm	TAE	2 years
16	Peterson et al[33]	2003	1	55	M	NA	NA	Y	1	2.2 × 2.1 cm	TAE	NA
17	Ogino et al[13]	2002	1	54	M	Shock	R	Y MALC	1	2 cm	TAE	3 years
18	Tarazov et al[19]	2001	1	52	F	Epigastric and upper right quadrant pain	UR	Y MALC	1	22 mm	TAE	8 months
19	Imamura et al[7]	1998	1	43	F	Asymptomatic	UR	Y AS	NA	NA	Surgical resection	NA
20	Suzuki et al[14]	1998	1	43	M	Asymptomatic	UR	Y MALC	2	30 mm, 15 mm	Ligation and resection	32 months
21	Uher et al[34]	1996	4	52	M	Intermittent right flank pain	UR	Y	1	3 cm	Surgical resection	NA
				69	M	Acute upper abdominal pain	R	Y	1	1.5 cm	TAE	2 months
				50	M	Evaluation of aneurysm of SMA	UR	Y	1	2.5 cm	No treatment	2 years
				48	M	Recurrent episodes of GI bleeding	UR	Y	1	1 cm	Conservative therapy	Died 1 year

Table 1 (*Continued*)

SN	Author	Year	No. of Patients	Age	Sex	Clinical Presentation	Ruptured/ Unruptured	Celiac Stenosis/ Occlusion	No. of Aneurysms	Size	Treatment	Follow-up
22	Lossing et al[35]	1995	1	77	F	Pain lower abdomen radiating to back	R	Y	1	1.5 x 1.3 cm	TAE	2 years
23	Chiang et al[36]	1994	1	59	M	Asymptomatic	UR	Y	2	2.5 × 4 cm, 1.5 × 1.3 cm	Surgical resection	4 months
24	Chiou et al[8]	1993	1	59	F	Intermittent epigastric pain	UR	Y	1	3 cm	Surgical resection	NA
25	Grech et al[37]	1988	1	50	F	Asymptomatic	UR	Y	1	23 mm	NA	NA

AS, atherosclerosis; F, female; GI, gastrointestinal; M, male; MALC, median arcuate ligament compression; NA, not available; R, ruptured; RX, prescription; SMA, superior mesenteric artery; TAE, transarterial embolization; UR, unruptured; Y, yes.

literature after successful embolization of the aneurysms alone.[4,16,19,29,30] However, many authors now recommend treatment of celiac stenosis immediately after treating the aneurysm.[32] It may be important to address the underlying cause of celiac stenosis. Celiac stenosis secondary to median arcuate ligament compression is treated with surgical resection of the ligament.[14] Atherosclerotic celiac stenosis/occlusion may be amenable to endovascular therapy with angioplasty and stenting. Surgical revascularization procedures aim at creating a bypass conduit to the hepatic artery through the renal artery or the aorta.[41]

CONCLUSION

Inferior pancreaticoduodenal artery aneurysms in association with celiac axis stenosis are rare. These are true aneurysms related to increased flow. Treatment is usually transcatheter embolization of the aneurysms. Revascularization of celiac axis stenosis may be considered.

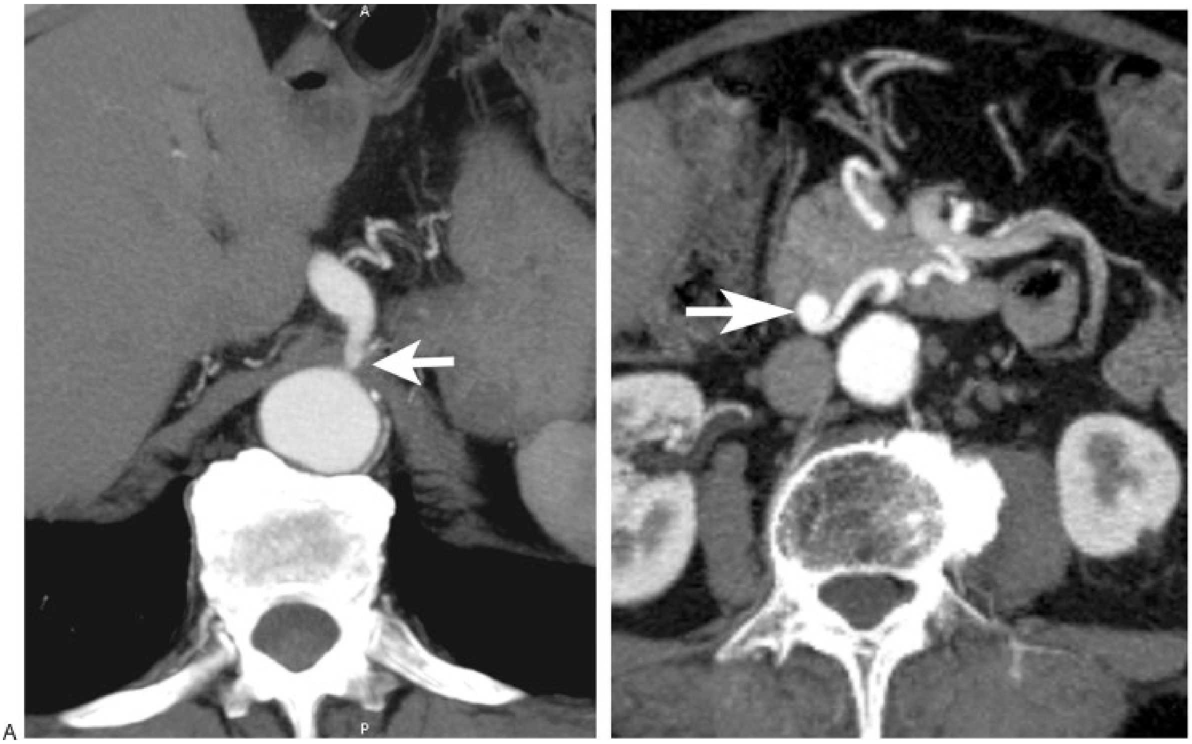

Figure 6 Incidentally detected small inferior pancreaticoduodenal artery aneurysm in a 50-year-old man. (A) Axial subvolume (4.75 mm thick) maximum intensity projection (MIP) of a computed tomography (CT) angiography dataset shows a high-grade stenosis (arrow) at the origin of the celiac axis. (B) Axial subvolume (4.75 mm thick) MIP of a CT angiography dataset shows a small aneurysm (arrow) within the head of the pancreas.

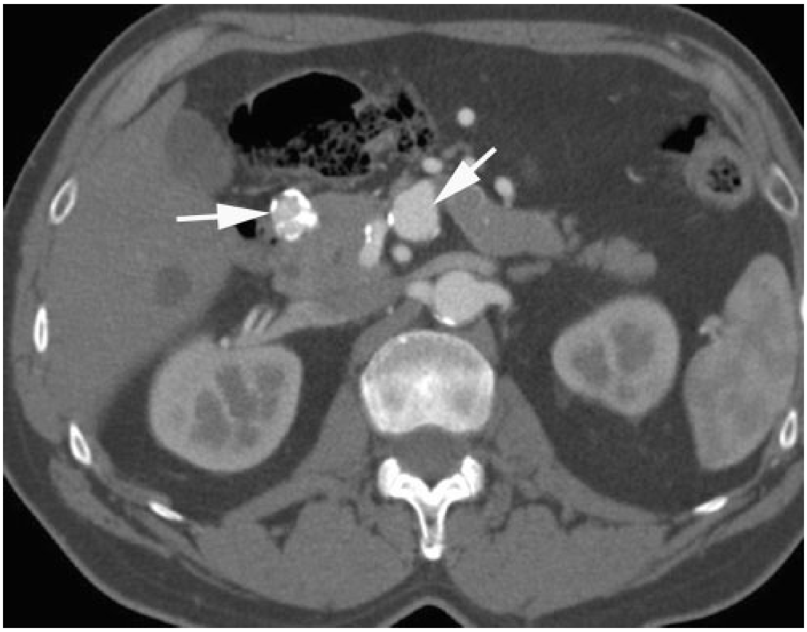

Figure 7 Multiple inferior pancreaticoduodenal artery aneurysms on a computed tomography (CT) scan in an asymptomatic 65-year-old man. Axial contrast-enhanced CT scan shows two aneurysms (arrows) in the head of the pancreas. Celiac axis stenosis was also present (not shown).

Figure 8 Large Inferior pancreaticoduodenal artery aneurysm in a 60-year-old male patient who presented with abdominal pain. Selective arteriogram from the superior mesenteric artery (SMA) (arrowhead) shows a large inferior pancreaticoduodenal artery aneurysm (arrow). The enlarged pancreaticoduodenal arcades that reconstitute the celiac axis are also seen.

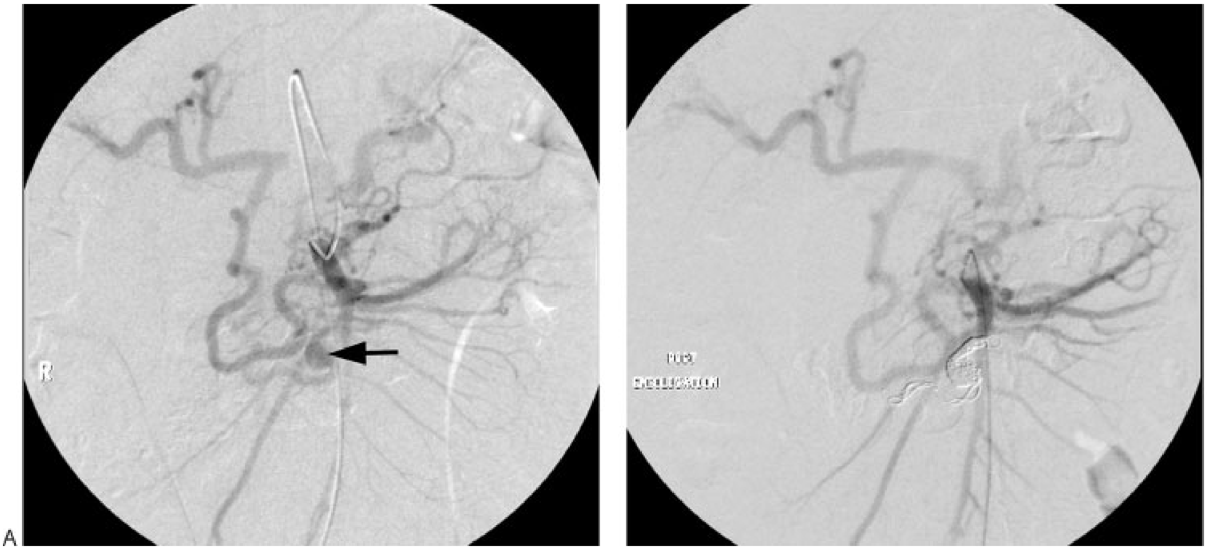

Figure 9 Transcatheter embolization of an inferior pancreaticoduodenal artery aneurysm. This 50-year-old man presented with acute abdominal pain and shock. Superior mesenteric arteriography (A) demonstrates an inferior pancreaticoduodenal artery aneurysm (arrow) in the anterior branch, which was successfully embolized with coils (B).

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