Splanchnic Artery Aneurysms

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Autopsy studies suggest that splanchnic artery aneurysms may be more frequent than abdominal aortic aneurysms. These aneurysms are important to recognize because up to 25% may be complicated by rupture, and the mortality rate after rupture is between 25% and 70%. However, little is known about the natural history and clinical presentation of splanchnic artery aneurysms. Splenic artery aneurysms are the most common of the splanchnic artery aneurysms; multiple aneurysms are present in approximately one third of patients. Hepatic artery pseudoaneurysms are more common than true aneurysms because of increasing numbers of hepatobiliary interventional procedures. The diagnosis of splanchnic artery aneurysm should be considered in any patient with abdominal pain, a pulsatile mass, or an abdominal bruit with or without associated bleeding. However, most aneurysms are asymptomatic and are detected incidentally on imaging studies. Treatment, which can be either surgical or interventional radiology-based, should be considered in all patients with symptoms related to the aneurysms, if the aneurysm is more than 2 cm in diameter, if the patient is pregnant, or if there is demonstrated growth of the aneurysm.


CT = computed tomography; MRI = magnetic resonance imaging; US = ultrasonography

Although abdominal aortic aneurysms are repaired more often than splanchnic artery aneurysms, autopsy studies suggest that splanchnic artery aneurysms may be more frequent than abdominal aortic aneurysms. The prevalence of these aneurysms has been estimated, on the basis of autopsy reports, to be up to 10%1 and that of abdominal aortic aneurysms, 0.5%2. Splanchnic artery aneurysms are important to recognize because up to 25% may be complicated by rupture, and the mortality rate after rupture is between 25% and 70%. Most series have comprised fewer than 30 cases and have had limited follow-up. Little is known about the natural history and clinical presentation of splanchnic artery aneurysms. Moreover, because the diagnosis is not often considered in patients with abdominal pain, the treatment of symptomatic aneurysms is delayed. The goal of this review is to define the causes, characteristics, clinical manifestations, and complications of splanchnic artery aneurysms and to outline the diagnostic and therapeutic options currently available. Because of the limited data available on optimal management, the recommendations in this review are based on the authors’ practice.

GENERAL CONSIDERATIONS: FEATURES OF SPLANCHNIC ARTERY ANEURYSMS

ANATOMY OF THE SPLANCHNIC ARTERIAL CIRCULATION

The splanchnic circulation includes the celiac, superior mesenteric, and inferior mesenteric arteries, which arise from the abdominal aorta (Figure 1). The most proximal is the celiac artery, which has 3 branches: the left gastric, splenic, and common hepatic arteries. The left gastric artery supplies the fundus and proximal lesser curvature of the stomach. The splenic artery arises from the celiac artery distal to the origin of the left gastric artery. It is associated closely with the pancreas and supplies blood to the spleen, pancreas, and stomach through the short gastric and left gastroepiploic arteries. The common hepatic artery divides into the gastroduodenal and proper hepatic arteries. The gastroduodenal artery has multiple branches, including the anterior and posterior pancreaticoduodenal arcades and the right gastroepiploic artery. The gastroduodenal artery is an important source of large-vessel collateral circulation when the celiac or superior mesenteric artery is occluded proximally. The right gastric artery arises from the common hepatic or left hepatic artery and supplies the distal lesser curvature of the stomach. The proper hepatic artery usually divides into the right and left hepatic arteries, which supply the liver. The right hepatic artery may arise from the superior mesenteric artery and the left hepatic artery from the left gastric artery.

The superior mesenteric artery originates from the anterior surface of the aorta 1 to 2 cm distal to the celiac trunk. The superior mesenteric artery passes posteriorly to the pancreas and anteriorly to the third part of the duodenum. It supplies the head and uncinate process of the pancreas through the inferior pancreaticoduodenal artery, the small intestine through jejunal and ileal branches, and the ascending and transverse colon through the ileocolic, right colic, and middle colic branches. The jejunal and ileal branches form anastomotic arcades in the mesentery and supply the small intestine by the vasa rectae. The inferior mesenteric artery arises from the anterior abdominal aorta 5 to 6 cm distal to the superior mesenteric artery and supplies the splenic flexure and entire descend-
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The inferior mesenteric artery has a variable number of sigmoid branches and terminates caudally as the paired superior hemorrhoidal arteries.

**EPIDEMIOLOGY**
A true aneurysm is a permanent, localized dilatation (>1.5 times the expected diameter) of an artery that involves all 3 layers of the vessel wall. A pseudoaneurysm ("false aneurysm") is a localized arterial disruption of the intimal and medial layers; it is lined by adventitia or perivascular tissue and caused by blunt or penetrating trauma. Fusiform aneurysms involve the entire circumference, and saccular aneurysms involve only a portion of the vessel wall. Most patients present in the sixth decade of life. Splenic artery aneurysms occur predominantly in multiparous women, but a male preponderance has been noted for hepatic and gastroduodenal artery aneurysms. Both sexes are affected equally with celiac and superior mesenteric artery aneurysms. Multiple aneurysms are present in approximately one third of patients.

**CLINICAL MANIFESTATIONS**
Most splanchic artery aneurysms are asymptomatic and detected incidentally on imaging studies. The common causes and complications of splanchic artery aneurysms are listed in Table 1. Symptomatic aneurysms present with abdominal pain or bleeding, which may be intra-abdominal or gastrointestinal. A bruit may be heard on auscultation, but an abdominal mass is rarely palpable because the aneurysms are small.

**COMPLICATIONS**
The complications of splanchic artery aneurysms are listed in Table 1. Up to 22% of the aneurysms present with intraperitoneal rupture or gastrointestinal hemorrhage. The reported mortality rate after intraperitoneal rupture is 21% for hepatic, 36% for splenic, and up to 100% for celiac artery aneurysms. Erosion of an aneurysm into the gastrointestinal lumen can present as sporadic gastrointestinal bleeding, the so-called herald bleed. Rupture into a mesenteric vein results in a mesenteric arteriovenous fistula. In this situation, the portal venous circulation develops sys-
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IMAGING STUDIES

Splanchnic artery aneurysms, if sufficiently calcified, may be found incidentally on plain radiographs of the abdomen. The diagnosis can be confirmed with computed tomography (CT), magnetic resonance imaging (MRI), ultrasonography (US), or angiography (Figure 2). The widespread use of US and CT has led to increased detection of asymptomatic aneurysms. Ultrasonography has a low sensitivity for small aneurysms because identification may be compromised by overlying gas and obesity. The use of CT after blunt abdominal trauma or an interventional biliary tract procedure has resulted in increased identification of false hepatic artery aneurysms. Moreover, CT is useful for detecting small aneurysms and assessing anatomical details. The need for intravenous contrast limits its use in patients with renal insufficiency or severe contrast allergy. Results of MRI are similar to those of CT, and like CT, MRI allows 2- and 3-dimensional imaging of the aneurysm and vessels.

The definitive diagnosis of small splanchnic artery aneurysms is made with contrast angiography, but it also can be made with high-quality CT and MRI. Angiography localizes and defines the size of the aneurysm and detects other aneurysms as well as vasculitides. It offers the advantage of therapeutic intervention. Therefore, angiography is usually performed when radiologic or surgical therapy is planned.

Rarely, some splanchnic artery aneurysms appear as an extrinsic gastric impression on upper gastrointestinal endoscopy (Figure 2, C). Endoscopic US can reliably differentiate aneurysms from other extrinsic lesions such as pancreatic pseudocysts (Figure 2, D).

TREATMENT

Treatment depends on the presentation, location, and size of the aneurysm. Generally, treatment is considered even for asymptomatic patients if the diameter of the aneurysm is larger than 2 cm. Elective surgical repair is safe and effective. Patients who present with a ruptured aneurysm require rapid resuscitation and surgical or radiologic intervention. Often, emergency surgical treatment is limited to ligation of the aneurysm without arterial reconstruction. In most cases, the rich mesenteric collateral circulation prevents ischemic damage to splanchnic organs supplied by the artery distal to the ligation.

Percutaneous transcatheter embolization with metallic coils has a success rate of 85%. Embolization may be preferred for aneurysms difficult to manage surgically and for high-risk surgical patients. Complications include migration of the coils (leading to organ infarction), abscess formation, and, rarely, aneurysm rupture. The aneurysm may undergo recanalization after successful embolization. Endovascular stent-graft placement is a promising treatment modality, but long-term results are unknown.

SPECIFIC CONSIDERATIONS

Splenic Artery Aneurysms

Etiology and Pathogenesis. Seventy-two percent of aneurysms of the splenic artery are true aneurysms. They

| TABLE 1. Etiology and Complications of Splanchnic Artery Aneurysms |
|-------------------------------|-------------------------------|
| Etiology                      | Complications                |
| True aneurysms                | Intrapерitoneal rupture       |
| Common causes                 | Hemoperitoneum                |
| Arteriosclerosis              | Hypovolemic shock             |
| Fibromuscular dysplasia       | Intrahepatic subcapsular rupture |
| Cystic medial necrosis        | Retroperitoneal hemorrhage    |
| Portal hypertension           | Gastrointestinal hemorrhage   |
| Uncommon causes               | Hemobilia                    |
| Autoimmune/collagen vascular diseases | Hemosuccus pancreaticus      |
| Polymyositis nodosa           | Herald bleed                  |
| Systemic lupus erythematosus  | Arteriovenous fistula formation |
| Takayasu arteritis            | Portal hypertension           |
| Ehlers-Danlos syndrome        | Aneurysms                    |
| Marfan syndrome               | Myotic aneurysms              |
| Neurofibromatosis             | Syphilis                     |
| Hypertension                  | Infective endocarditis        |
| Congenital                    | Tuberculosis                 |
| α1-Antitrypsin deficiency     |                              |

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Splanchnic artery aneurysms are usually saccular and occur at a bifurcation in the splenic hilum. Twenty percent of patients have multiple aneurysms. The more common causes are arteriosclerosis and portal hypertension; pancreatitis results in pseudoaneurysm. Less common causes include idiopathic dissection, septic emboli, essential hypertension, polyarteritis nodosa, systemic lupus erythematosus, Ehlers-Danlos syndrome, fibromuscular dysplasia, and neurofibromatosis. Pseudoaneurysms of the splenic artery are most often caused by chronic pancreatitis or by trauma. The incidence of splenic artery aneurysms is higher in multiparous women (average, 4.5 pregnancies) and in patients with splenomegaly or those who have undergone orthotopic liver transplantation. The increased prevalence in multiparous women may be related to increased splenic blood flow and the effects of estrogen on the elastic tissue of the tunica media. Dilatation of the splenic artery resulting from increased blood flow during pregnancy likely predisposes to aneurysm formation. Similarly, increased splenic blood flow is considered the cause of splenic artery aneurysms in portal hypertension and after liver transplantation.

The role of arteriosclerosis is unclear. Localized arteriosclerotic changes in aneurysms without involvement of adjacent vessels have been demonstrated. Moreover, the presence of arteriosclerosis in some but not all aneurysms of patients with multiple aneurysms suggests that arteriosclerosis is a secondary event rather than the cause of most splenic artery aneurysms.

Clinical Manifestations. Most splenic artery aneurysms are smaller than 2 cm. They may be detected on plain radiographs as curvilinear calcifications in the left upper quadrant. The differential diagnosis includes tortuous splenic artery, renal artery aneurysm, calcified lymph nodes, and calcific cysts of the spleen or adrenal gland. Symptomatic patients manifest with left upper quadrant or epigastric pain that radiates to the left shoulder. Rupture of the aneurysm, which may manifest as hypovolemic...
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Management. Ruptured aneurysms of the splenic artery usually are treated with splenectomy. A symptomatic aneurysm or an aneurysm of any diameter in a pregnant woman or a woman of childbearing age is considered by some to be an absolute indication for elective repair. Most would agree that an aneurysm larger than 2 cm is an indication for surgery; an aneurysm between 1 and 2 cm in diameter should be monitored closely with imaging studies every 6 months. Mortality after emergency surgery is as high as 40%, compared with negligible mortality after elective repair. Transcatheter embolization may be performed for all splenic artery aneurysms, except those located at the splenic hilum. Aneurysms of the proximal splenic artery can be treated with simple ligation, but those involving the hilum require splenectomy. Aneurysmectomy with end-to-end anastomosis is recommended for mid splenic aneurysms in a tortuous and redundant artery. Surgical repair is preferred for all symptomatic aneurysms because of the greater likelihood of success. In the presence of portal hypertension, transcatheter embolization or stent-graft placement may be preferred because the extensive collateral circulation that develops as a result of portal hypertension makes surgery more difficult. Although conservative management of splenic artery pseudoaneurysms has produced excellent results according to some reports, we recommend intervention in all cases irrespective of size or symptoms. Transcatheter embolization of the pseudoaneurysm is gaining popularity; however, failure does occur, especially when the pseudoaneurysm is associated with a pseudocyst of the pancreas. Splenectomy, with or without distal pancreatec-

tomy is the current standard of treatment, with no reports of failure.

HEPATIC ARTERY ANEURYSMS

Etiology. Almost 50% of hepatic artery aneurysms are pseudoaneurysms. This reflects the increased use of interventional procedures of the biliary tract and CT after blunt abdominal trauma. True aneurysms occur 4 times more frequently in the extrahepatic arteries, usually involve the common hepatic artery, and are associated mainly with arteriosclerosis and acquired medial degeneration. Mycotic aneurysms are rare (<5% of hepatic artery aneurysms). Other causes include polyarteritis nodosa, pancreatitis, liver transplantation, neurofibromatosis, Wegener granulomatosis, and tuberculosis.

Clinical Manifestations. More than 50% of patients present with right upper quadrant abdominal pain that radiates to the back. Almost 20% to 30% of hepatic artery aneurysms may rupture into the peritoneal cavity and manifest as abdominal pain with hypovolemic shock. Risk factors for rupture of true aneurysms include multiple hepatic artery aneurysms and a nonatherosclerotic etiology of the aneurysm. Gastrointestinal hemorrhage may occur from erosion of the aneurysm into the stomach or duodenum. Erosion into the biliary tract occurs in nearly 50% of patients with rupture of a hepatic artery aneurysm, with one third of patients presenting with the classic triad of biliary colic, hemobilia, and obstructive jaundice. Obstructive jaundice due to extrinsic compression of the biliary duct by the aneurysm and liver abscesses are uncommon complications. The diagnosis of hepatic artery aneurysm should be considered in patients with biliary colic, especially those with associated gastrointestinal bleeding (Figure 3).

Management. The risk of rupture in relationship to the size of an aneurysm of the hepatic artery is unknown. When 22 patients with a mean hepatic artery diameter of 2.3 cm (range, 1.5-5 cm) were followed up for a mean of 68.4 months (range, 1-372 months), no complications were identified. Therefore, we recommend intervention only when the aneurysm is symptomatic or when risk factors for rupture (multiple aneurysms and a nonatherosclerotic etiology) are present. To prevent hepatic infarction, ligation of the affected artery or embolization of the aneurysm is best performed only if the portal vein is patent. Asymptomatic common hepatic artery aneurysms can be treated with embolization or by ligation of the aneurysm without reconstruction. However, vascular reconstruction is required for the treatment of proper hepatic artery aneurysms to prevent hepatic ischemia resulting from interruption of collateral circulation through the gastroduodenal and right gastric arteries. Embolization of the hepatic artery (Fig-
Celiac Artery Aneurysms

Etiology. The common causes of celiac artery aneurysms are arteriosclerosis and medial degeneration. Trauma, dissection, and Takayasu arteritis are other causes. Syphilitic aneurysms are now uncommon. Peripheral artery aneurysms are seen in 18% to 67% of patients with celiac artery aneurysms.36

Clinical Manifestations. Celiac artery aneurysms are manifested initially by epigastric pain or upper gastrointestinal hemorrhage. Worsening abdominal pain usually indicates a rapidly expanding aneurysm or rupture. Dysphagia may occur from esophageal compression.37 In earlier reports, nearly 80% of celiac artery aneurysms had ruptured by the time the patient presented, and a large number were detected at autopsy.7 However, recent series have reported a lifetime risk of rupture of about 6%.36 Aneurysm size, calcification, and thrombus formation are not risk factors for rupture.36

Management. Celiac artery aneurysms can be treated with celiac ligation, followed by aortohepatic bypass or direct aortic reimplantation.20 In patients undergoing revascularization, prosthetic grafts have a lower risk of occlusion than saphenous vein grafts.36 If the aneurysm ruptures, intervention may include ligation or percutaneous transcatheter embolization.

Superior Mesenteric Artery Aneurysms

Etiology. Septic emboli account for about one third of superior mesenteric artery aneurysms, with nonhemolytic streptococci, staphylococci, and gram-negative bacteria being the organisms commonly implicated. Other causes include arteriosclerosis, polyarteritis nodosa, pancreatitis, biliary tract disease, neurofibromatosis, and trauma. Pseudoaneurysms arising from arterial dissection most frequently involve the superior mesenteric artery.38
Clinical Manifestations. More than 90% of superior mesenteric artery aneurysms are symptomatic, with associated abdominal pain and gastrointestinal bleeding. Acute mesenteric ischemia may result from thromboembolism of the artery.\textsuperscript{18} Although superior mesenteric artery aneurysms are rare, up to 50% of patients present with rupture, with a mortality rate of 30%.\textsuperscript{39,40} \textbeta-Adrenergic blockers may have a protective effect against rupture.\textsuperscript{40}

Management. Because of the high rate of complications, intervention is recommended for all patients at low surgical risk. Ligation of an aneurysm of a branch of a mesenteric artery should be accompanied by resection of any ischemic segment of bowel.\textsuperscript{40} Transcatheter embolization is safe in hemodynamically stable patients with a ruptured aneurysm. An endovascular stent-graft can be used,\textsuperscript{41} but it increases the risk of mesenteric ischemia. The use of \textbeta-adrenergic blockers can be considered for asymptomatic patients who are reluctant to undergo intervention procedures.

Pancreatoduodenal and Gastro-duodenal Artery Aneurysms

Etiology. Pancreatoduodenal and gastroduodenal artery pseudoaneurysms usually result from pancreatitis\textsuperscript{42} but may develop after pancreatoduodenectomy.\textsuperscript{8} True aneurysms are caused by arteriosclerosis, polyarteritis nodosa, or Takayasu arteritis and may occur with occlusion of the celiac artery.\textsuperscript{43}

Clinical Manifestations. Abdominal pain, indistinguishable from pain due to pancreatitis or pancreatic pseudocyst, is the most common presentation. After pancreatoduodenectomy, “herald” or “sentinel” bleeding from a biliary drain may indicate a pseudoaneurysm.\textsuperscript{8}

Erosion of an aneurysm of the pancreaticoduodenal artery into the pancreatic duct can manifest as hemosuccus pancreaticus, which is the presence of bleeding into the pancreatic duct. A sudden increase in the size of a pancreatic pseudocyst or a pulsatile pseudocyst indicates a communication between an aneurysm and pseudocyst and warrants prompt treatment.

Management. When a pancreaticoduodenal or gastroduodenal artery aneurysm occurs close to a pseudocyst, the pseudocyst should be drained after the aneurysm is either ligated or embolized. A ruptured aneurysm is managed by resection, with or without vascular reconstruction, or by transcatheter embolization. For aneurysms associated with occlusion of the celiac axis, the median arcuate ligament may need to be resected.\textsuperscript{44} Pancreatoduodenal artery aneurysms can be managed with stent-graft placement.\textsuperscript{45} There is no established cutoff for treatment of pancreatoduodenal aneurysms; many experts consider treating all such aneurysms with either surgical ligation or endovascular exclusion. When the celiac artery is occluded, hepatic artery revascularization may be required.

CONCLUSION

Splanchnic artery aneurysms, although uncommon, are important to recognize because of the risk for rupture and associated mortality. Improvements in imaging technology of CT, MRI, and angiography have led to increased detection of asymptomatic aneurysms. Because of the heterogeneity of presentation, management must be individualized according to the artery involved. Elective intervention is required for all asymptomatic aneurysms and for most aneurysms larger than 2 cm in diameter to reduce the risk of rupture and hence mortality.

REFERENCES

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