Visceral Artery Aneurysms: Decision Making and Treatment Options in the New Era of Minimally Invasive and Endovascular Surgery

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Abstract

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The abdominal viscera blood supply is derived from anterior branches of the abdominal aorta. Visceral artery aneurysms (VAAs) include aneurysms of the following arteries and their branches: the celiac artery, the hepatic artery, the splenic artery, the superior mesenteric artery, the inferior mesenteric artery, the pancreaticoduodenal artery, and the gastroduodenal artery. Overall VAAs comprise < 2% of all types of arterial aneurysms. Asymptomatic VAAs are now being encountered more frequently due to the widespread use of advanced diagnostic abdominal imaging. The incidental finding of a VAA frequently leaves clinicians with a dilemma as to the best course of management. The focus of this review is on current treatment options and management guidelines for both symptomatic and asymptomatic VAAs.

The autopsy incidence of a visceral artery aneurysm (VAA) is reported to be 0.01 to 0.2%. Twenty two percent of VAAs present as a clinical emergency and death may result in 8.5% of cases.¹ Given their relative rarity, the precise natural history of the various VAAs remains unclear.^{2,3} Recent studies have suggested that the majority remains stable over time with only a minority of aneurysms showing significant growth, which is known to be associated with an increased risk of rupture.^{4,5} However, certain VAAs, nonatherosclerotic, inflammatory, mycotic, or false aneurysms, have been noted to have a higher risk of rupture and in these aneurysms size may not be an accurate predictor of rupture.

Many VAAs are asymptomatic and are usually found incidentally when a patient undergoes abdominal diagnostic imaging for unrelated reasons. A plain abdominal film may reveal a calcified VAA. Visceral artery aneurysm-related symptoms are frequently nonspecific and are related to the size and location of the aneurysm. Hence, despite the presence of symptoms, there

is often a delay in diagnosis.⁶ While the majority of asymptomatic VAAs can be safely observed, rupture remains the most feared complication with rupture rates ranging from 2.3 to 18% with mortality rates ranging from 20 to 100%.⁷ Interestingly a few case series from large institutions have shown a relatively benign course for well-calcified VAAs, despite their size being >2cm.^{7,8} It appears that the variability in rupture rates is influenced by several factors including the location, size, type, and etiology of the aneurysm. These same parameters also govern the choices of management which include serial observations with imaging studies, an open surgical procedure such as ligation, aneurysmectomy or interposition grafting or a minimally invasive endovascular intervention such as embolization or covered stent placement.⁸ A surgical or endovascular intervention obviously removes the risk of rupture, with its associated mortality, but is not indicated in all cases and hence the risk of the specific procedure must be weighed against the known risk of rupture of a VAA.

Multiple underlying etiologies have been associated with VAA formation with the most common being atherosclerotic degeneration followed by medial degeneration. Other etiological entities associated with VAAs include trauma, pregnancy, portal hypertension, connective tissue disorders, vasculitis, congenital disorders (von Recklinghausen's disease, Ehlers-Danlos type IV, Marfan's syndrome), and infection. Pseudoaneurysms related to blunt, penetrating and iatrogenic abdominal trauma, inflammation, infection, and vasculitis are now frequently encountered.^{2,3} Poststenotic dilatation may be seen distal to a hemodynamically significant stenosis.

Celiac Artery Aneurysms

Celiac artery aneurysms (CAAs) are rare, accounting for only 4% of all VAAs. A concomitant aortic aneurysm is found in 20% of cases and other VAAs are seen in up to 40% of cases. Males and females are equally affected, with a mean age at the time of diagnosis of 56 years.⁹

Historically, many CAAs had an infectious etiology, with syphilis being the causative agent in up to 30% of cases.¹⁰ Nowadays the most common underlying etiology is atherosclerotic degeneration resulting in a saccular aneurysm. A CAA may also be seen in a patient with a median arcuate ligament syndrome as a result of extrinsic compression causing post-stenotic dilation.¹¹

Patients with CAAs are mostly asymptomatic or present with vague abdominal or back pain symptoms that can mimic pancreatitis. The majority are incidentally discovered on computed tomography (CT), magnetic resonance imaging, or ultrasonography (US) examinations.

The natural history of CAAs is not well delineated. Literature reports suggest an estimated rupture risk of up to 20%. Rupture typically results in intraperitoneal bleeding or occasionally gastrointestinal bleeding and carries a mortality upwards of 50%.¹²

Intervention is indicated for all symptomatic CAAs and noncalcified aneurysms with a diameter > 2 cm. The treatment approach depends on the anatomy and location of the aneurysm, urgency of intervention (ruptured versus elective repair) as well as the patient's underlying co-morbidities. Open surgical repair of a CAA remains the gold standard. The celiac artery can be exposed using a direct anterior approach to access the lesser sac, allowing for dissection of the diaphragmatic crura to expose the infradiaphragmatic aorta and the celiac artery and its branches. Alternatively, for large aneurysms, medial visceral rotation or even a thoracoabdominal approach may be required to achieve adequate celiac artery exposure proximal and distal to the aneurysm for control. If the aneurysm does not involve the origin of the artery, it can be resected and the artery reanastomosed. If this is not possible, it can be replaced with either a vein or polytetrafluoroethylene interposition graft. Otherwise, the aneurysm can be ligated proximally and distally, and revascularization achieved with a bypass graft from the supraceliac aorta to the distal celiac artery or to the hepatic or splenic arteries. Aneurysm ligation without revascularization is typically reserved for cases of aneurysm rupture with hemodynamic instability or when

revascularization is technically difficult. In such instances, collateral blood flow from the superior mesenteric artery (SMA) must be sufficient to prevent severe hepatic or foregut ischemia. Endovascular treatment of CAAs is not often performed given the proximity and possible occlusion of vital vessels such as the hepatic, splenic, and left gastric arteries, as well as the inferior phrenic arteries in some cases. When feasible, endovascular options include exclusion with a covered stent or embolization. Unfortunately, endovascular options are rarely feasible especially if the aneurysm involves the origin of the artery as there is no seal zone for a stent graft and also no place to place coils proximal to the aneurysm (►Fig. 1). Embolization of the aneurysm's inflow and outflow achieves a similar effect to simple ligation but should only be performed in patients without liver dysfunction and with an intact collateral circulation from the SMA and gastroduodenal artery (GDA). Ideally a covered or multilayer stent graft is used when there are adequate proximal and distal seal zones and the risk of coverage of any vital arterial branches is minimal.

Data on treatment outcomes for CAAs are scarce and are limited to published case reports and small series of patients. Patency rates for open and endovascular celiac artery revascularization can be inferred from the published data on chronic mesenteric ischemia and juxtarenal aneurysm repair and appear to be comparable between both approaches, although open repair remains superior in the long term with fewer reinterventions compared with endovascular procedures.^{13,14} Both open surgical and endovascular procedures can result in serious post procedural complications including gastric ischemia, gangrenous cholecystitis, liver abscess, bleeding, and wound infection, although the morbidity associated with open repair remains higher.

Fig. 1 Diagnostic angiogram showing a celiac artery aneurysm extending from the ostium to the bifurcation.

Splenic Artery Aneurysms

Splenic artery aneurysms (SAAs) are the most common of all VAAs accounting for 60 to 80%. The majority are located in the mid to distal third of the splenic artery and are usually small saccular and asymptomatic. These aneurysms have a 4:1 female to male predominance and are often found in multiparous women. It is posited that hormonal changes during pregnancy may be associated with arterial wall weakening, although no clear mechanism is understood. Additionally, increased flow rates through the vessel during pregnancy may be a factor. Portal hypertension may also play a role in the development of SAAs. However, most true SAAs are degenerative in nature. Pseudoaneurysms develop secondary to pancreatitis or trauma.

The natural history of SAAs is not well defined. Rupture risk is increased with pregnancy, portal hypertension, liver transplantation, and concomitant vasculitis.¹⁵ Size is a poor predictor of rupture. Some reports indicate a rupture rate of 2% with an associated mortality rate of 36%.¹⁶ Rupture during pregnancy can be catastrophic especially when occurring in the third trimester with maternal and fetal mortality rates as high as 70 and 90%, respectively. While all symptomatic aneurysms require repair, the current literature supports intervention, irrespective of size or symptoms, in patients who are pregnant or may become pregnant, require liver transplantation, have fibromuscular dysplasia, polyarteritis nodosa, or when an infected aneurysm is suspected.¹⁷

Open surgical approaches for a SAA depend on the location of the aneurysm. To gain access to the splenic artery, the transverse colon is taken down by incising between it and the greater omentum. When the lesser sac is entered, the splenic artery is readily accessible after the inferior edge of the pancreas is mobilized. Ligation of the splenic artery just proximal and distal to the aneurysm is usually well tolerated as the short gastric and gastroepiploic arteries provide adequate collateral circulation. However, often the aneurysm can be resected and flow restored via an end-to-end anastomosis which removes any concern about splenic infarction. In their series of 11 patients with SAAs, Małczak et al reported successful aneurysmectomy with spleen preservation in seven patients with one patient undergoing partial splenectomy and

two patients receiving intraoperative splenectomies.¹⁸ Distal SAAs and those intimately associated with the splenic parenchyma are treated by splenectomy and occasionally distal pancreatectomy may be required for an embedded aneurysm.¹⁹ More recently, laparoscopic aneurysmectomy with or without spleen preservation has been reported with excellent results. Such an approach performed by surgeons proficient in advanced laparoscopy involves identification and control of the splenic artery proximal and distal to the aneurysm. The aneurysm can then be excluded or excised depending on its location and the ease of dissection.²⁰ An infected SAA is treated with intravenous antibacterial therapy and surgical excision with concomitant splenectomy is recommended as definitive treatment.

Endovascular approaches include stent graft exclusion of the aneurysm or occlusion of the aneurysm with coil embolization of the splenic artery proximally and distally to the aneurysm²¹ (►Fig. 2). Several small series of patients with endovascular management of SAAs showed a high success rate.^{22,23} Postembolization syndrome, characterized by fever and abdominal pain, ileum and pancreatic inflammation, was present in 35% of patients. All cases were self-limiting with resolution within 3 to 5 days.²⁴

Hepatic Artery Aneurysms

Hepatic artery aneurysms (HAAs) are the second most common VAAs accounting for 20% with 80% being extrahepatic solitary aneurysms involving the common hepatic artery or its branches. HAAs are found more frequently in men and there has been an increased incidence of reporting in the past two decades most likely due to the emergence of more percutaneous biliary procedures, liver transplantation, and the acceptance of nonoperative management for many blunt abdominal trauma cases.

Atherosclerotic degenerative disease is the underlying cause of the majority of HAAs. Other less common etiologies include fibromuscular dysplasia, polyarteritis nodosa, infection, and inflammation associated with pancreatitis or cholecystitis. Pseudoaneurysms occur as a result of blunt or sharp abdominal trauma or secondary to iatrogenic trauma from biopsies or other types of hepatic or biliary interventions.

Fig. 2 (A) Angiogram showing distal splenic artery aneurysm. (B) Exclusion of the aneurysm with sandwich double coils embolization.

Traumatic pseudoaneurysms account for almost half of the reported HAAs involving the intrahepatic arterial branches.²⁵

The majority of HAAs are asymptomatic and are usually discovered incidentally following abdominal magnetic resonance imaging or CT imaging or during the performance of percutaneous diagnostic and therapeutic biliary tract procedures. Diagnosis is commonly made in the sixth decade of life with concomitant VAAs seen in up to 31% of patients.²⁶ When symptomatic, patients can present with vague right upper quadrant or epigastric pain or rarely obstructive jaundice as seen with large expanding intrahepatic aneurysms.²⁷ Although data on this subject are sparse, rupture rates of extrahepatic HAAs are estimated to be around 20%. Rupture of an intrahepatic pseudoaneurysm appears to be more common and is reported to occur in more than half of the cases. Nonatherosclerotic HAAs have a higher risk of rupture and the rupture mortality is reported to be 21 to 35%.²⁸ Depending on the location of the ruptured aneurysm, bleeding can occur into the gastrointestinal or hepatobiliary tract. When the presentation is jaundice, biliary colic and upper gastrointestinal hemorrhage, it is known as Quincke's triad. This is the mode of presentation in one-third of symptomatic patients.

Treatment is indicated for all symptomatic HAAs. Asymptomatic aneurysms are indicated for repair if they are thought to be nonatherosclerotic aneurysms due to vasculitis or fibromuscular dysplasia or if there are multiple aneurysms. Atherosclerotic aneurysms should be treated if they enlarge or become symptomatic. Like other VAAs treatment depends on the anatomy and location of the aneurysm as well as the patient's clinical status and comorbidities. Surgical exposure of these vessels is generally accomplished through a generous bilateral subcostal incision. The hepatic artery is exposed by accessing the lesser sac, dissecting the diaphragmatic crura to expose the infradiaphragmatic aorta and then the celiac artery trunk and its branches. The hepatic artery can then be isolated and clamped proximally and distally to the aneurysm prior to opening the aneurysm sac. Alternatively, when the aneurysm is too large or inflamed to achieve safe proximal control of the hepatic artery, supraceliac aortic clamping is required. Alternatively in this situation, a hybrid approach can also be utilized in which endovascular proximal control with balloon occlusion is performed prior to accessing the HAA to minimize blood loss.²⁹ Open surgical options include ligation and exclusion of the aneurysm provided that there are adequate collaterals. Conversely, in the absence of sufficient collaterals, aneurysmectomy with reconstruction using a bypass or interposition graft can be performed. The pancreaticoduodenal artery (PDA) and GDA provide collateral flow when the common hepatic artery is ligated. Revascularization should be performed to prevent hepatic ischemia especially in HAAs distal to the origin of the GDA. Ligation without revascularization may be contemplated for an HAA proximal to the GDA since hepatic perfusion is maintained through collateral flow from the GDA and right gastric arteries. Simple ligation, when utilized to control bleeding from a ruptured intrahepatic aneurysm, may result in liver necrosis requiring partial hepatic resection of the associated necrotic territory. In

very rare instances, partial hepatectomy or liver transplantation may be required.³⁰ Endovascular options can be used in high-risk patients or when the aneurysm is not easily accessible via an open approach. An example of the latter includes percutaneous embolization using microcatheters for intrahepatic artery HAAs. Embolization of a common hepatic artery aneurysm can also be performed but only if the portal vein is patent and can provide sufficient flow to avoid fulminant hepatic necrosis. Exclusion of an HAA with a covered stent is a better option as it preserves flow but requires that there are adequate proximal and distal sealing zones. Kinking of the stent graft may be problematic leading to stent graft thrombosis and liver ischemia. For hepatic artery proper aneurysms, embolization of the GDA may also be required to avoid recanalization of the aneurysm. Direct thrombin injection under CT or US guidance has been used successfully for small peripheral intrahepatic aneurysms.

As with other VAAs, treatment outcomes of HAAs are limited to published case reports and small case series. The reported 30-day mortality for both elective and urgent open and endovascular procedures ranges between 6 and 14%. Major complications occur in ~24% of cases and include graft or stent occlusion, arterial dissection, liver failure, abscess formation, cholecystitis, and hemorrhage.^{17,31} For open reconstructions, primary patency rates are reported to be 86% at 5 years.³²

Superior Mesenteric Artery Aneurysms

Superior mesenteric artery aneurysms (SMAAs) have a similar prevalence to CAAs, with rates ranging from 3 to 7%.^{33,34} They usually occur within the first 5 cm of the takeoff of the SMA from the aorta and may be fusiform or saccular.

Historically, as with CAAs, the majority of SMAAs was attributed to infectious etiology, most commonly syphilis.³⁵ In the current era, however, most are associated with atherosclerotic vascular wall degeneration. Other etiologies include vasculitis, dysplasia, dissection, inflammation, and infection. Mycotic aneurysms are usually secondary to bacterial endocarditis. A small minority of SMAAs occur as a result of poststenotic dilation secondary to increased velocities distal to a proximal SMA stenosis. A careful distinction regarding SMAAs and aneurysms of the pancreaticoduodenal arcade must be made, as the latter are often the result of pancreatitis and have a worse prognosis when left untreated. Patients frequently present with colicky upper abdominal pain, nausea, and vomiting. The mural contents of the aneurysm may embolize to smaller branches of the splanchnic circulation resulting in bowel ischemia. As many as 50% of SMAAs may present with rupture.

As with aneurysms of the celiac axis, treatment is indicated for symptomatic aneurysms and asymptomatic aneurysms with a diameter > 2cm. Treatment options include an open surgical procedure such as aneurysmectomy, aneurysmorrhaphy, simple ligation with or without arterial reconstruction, or an endovascular intervention with coil embolization or stent graft exclusion. With an open surgical approach, the type of surgery depends on the etiology and mode of presentation. The exposure of the SMA requires the transverse colon to be

Fig. 3 Saccular aneurysm of the superior mesenteric artery demonstrated on angiography.

retracted superiorly. When this is done, the base of the mesentery can be grasped between the fingers and the pulsating aneurysm will be felt facilitating the exposure of the artery proximal and distal to the aneurysm. The need for further revascularization following simple ligation of the SMA depends on the patency of the PDA and the presence of a robust GDA. The bowel should be carefully evaluated for ischemia at the end of surgery. If revascularization was not done and if there is any doubt about its viability, an immediate revascularization procedure should be done. An endovascular approach with a stent graft precisely placed to exclude the aneurysm may be preferred if adequate proximal and distal landing zones are present (→**Fig. 3**). Although long-term follow-up data are lacking, several published case series show endovascular therapy for SMAAs to be safe and effective provided that the aneurysm is sufficiently distal to the origin of the artery so that there is an adequate proximal seal zone or room to place coils safely in the artery.^{3,31}

Inferior Mesenteric Artery Aneurysms

These are very rare and are usually asymptomatic. They make up ~1% of VAAs. The etiology appears to be related to increased blood flow through the inferior mesenteric artery (IMA) which is an artery unadapted to high flow. Increased flow occurs in the IMA when there is significant disease with occlusion or severe stenosis of the celiac and SMAs. An ostial stenosis of the IMA is also usually present.

Revascularization of the diseased celiac and SMAs is usually required for satisfactory treatment to avoid small or large bowel ischemia. After flow is improved through the celiac and SM arteries, the IMA aneurysm can then be resected and the IMA reimplanted into the aorta or revascularized with a short interposition graft from the aorta to the distal IMA.

Pancreaticoduodenal and Gastroduodenal Artery Aneurysms

Both PDA and GDA aneurysms are rare comprising 6% of VAAs. Pseudoaneurysms of these arteries are more frequent than true aneurysms and usually occur secondary to pancreatitis and pancreatic surgery. Atherosclerotic degeneration is the etiology of most true aneurysms of these arteries. Increased flow through the PDA and GDA is also theorized to be a predisposing factor to the formation of some aneurysms due to a compensatory increased flow in these vessels associated with stenosis or occlusion of the celiac artery and/or SMA.³⁶

Symptomatic aneurysms present with epigastric pain radiating to the back. Reports on these rare aneurysms are limited to published case reports that suggest rupture can occur at a smaller diameter compared with other VAAs and 90%. The mean size at time of rupture was reported to be 9 mm.³⁷ The rupture rates are estimated to be around 100% for an aneurysm of the PDA and 80% for an aneurysm of the GDA.³⁰ More than half of patients with a ruptured aneurysm present with gastrointestinal bleeding or bleeding into the pancreatic and biliary ducts.³²

As with other VAAs, the choice of therapeutic approach varies depending on the presenting symptom, the location and type of aneurysm, the patient's condition, and the presence or absence of collaterals. Given the size and location of these aneurysms, open repair is technically challenging. Moreover, in the presence of adequate collateral flow, open reconstruction is most often not needed. As such, endovascular repair either with coil embolization or stent graft placement is the optimal treatment choice and appears to be associated with a lower morbidity when compared with open repair. Endovascular access to the PDA and GDA can be achieved using either a femoral or a brachial artery approach. The CA or SMA is then selected and a mesenteric angiogram is performed to delineate the anatomy of the aneurysm. Coil embolization can then be performed using either microcoils or vascular plugs. Prior to completion, a careful search should be made to ensure that all collateral blood supply to the aneurysm has been occluded. Interval follow-up with angiography may be needed to ensure aneurysm occlusion. Stent graft placement is only considered when flow through the artery is necessary due to inadequate collateral blood flow and is only performed when there are adequate proximal and distal seal zones. Open repair is typically reserved for hemodynamically unstable patients or those with a failed endovascular repair. Direct percutaneous thrombin injection under CT or US guidance is another option for the management of failed endovascular treatments.

Conclusion

VAAs are rare, and most are discovered incidentally. Although data on their natural history is limited, VAAs appear to carry a significant rupture risk especially for large or expanding aneurysms and those associated with an underlying inflammatory process or trauma. Ruptured VAAs have a high mortality rate. Open, endovascular, or hybrid approaches can be utilized depending on the anatomic location of the aneurysm,

clinical presentation, presence of collateral circulation, and the patient's comorbidities. Improved and refined endovascular technologies have expanded the role of endovascular procedures for the management of VAAs. Minimally invasive procedures are now a dependable option for the management of many VAAs. Longer term follow-up is anticipated to confirm the preference of endovascular procedures compared with open surgical reconstructions in appropriately selected cases. In-depth review of imaging studies to assess the vascular anatomy and end-organ perfusion will define the ideal treatment method.

Conflict of Interest

None declared.

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