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VASC ENDOVASCULAR SURG 2011 45: 681

DOI: 10.1177/1538574411418129

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Visceral Artery Aneurysms

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Vascular and Endovascular Surgery
45(8) 681-687
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DOI: 10.1177/1538574411418129
http://ves.sagepub.com


Abstract

Visceral artery aneurysms (VAAs) are a rare condition, in case of a rupture they have a high mortality rate up to 70%. Visceral artery aneurysms are seen more often these days with the more widespread use of computed tomography and angiography. There are various options for treating VAAs; open surgical repair, endovascular treatment, and laparoscopic surgery. We report 5 cases of visceral aneurysms, all treated differently—ligation, aneurysmectomy (with splenectomy), emergency and elective coil embolization, and conservatively. We will further give a review of the literature on etiology, diagnosis, and treatment options.

Keywords

visceral, aneurysm, rupture

Introduction

Visceral artery aneurysms (VAAs) are a rare condition, in case of a rupture, which occurs in about 25% of the cases, they have a high mortality rate up to 70%.¹ Most VAAs are of degenerative origin.² Atherosclerosis seems to play a secondary role in the development of these lesions and does not always represent the first pathological mechanism.³ Treatment options for VAA vary and depend on the clinical presentation of the patient and the location of the aneurysm, as well as patients' comorbidities.⁴ Options for treating VAAs include, open surgical repair (ligation, aneurysmectomy, venous grafting, or synthetic grafting), endovascular treatment (embolization and covered stent placement), and most recently laparoscopic surgery (ligation).⁵ We present 4 cases of ruptured visceral aneurysms and 1 case that was treated electively. We will further give a review of the literature on etiology, diagnosis, and treatment options.

Case Reports

Case 1

A 51-year-old female patient was admitted to the hospital with progressive shortness of breath. She had severe chronic obstructive pulmonary disease ([COPD] class Global initiative for chronic Obstructive Lung Disease [GOLD] III) and was treated with intravenous steroids. Three days after admission the patient suddenly collapsed and complained of epigastric pain. Shortly after this, the patient developed a cardiac arrest and had to be resuscitated. A subsequent computed tomography (CT) revealed a 4-cm ruptured aneurysm of the splenic artery (Figure 1). An emergency laparotomy was performed and a hematoma was evacuated. The splenic artery aneurysm was identified and an aneurysmectomy with splenectomy (Figure 2)

was performed. During surgery, there was massive blood loss. At the intensive care unit (ICU) the patient was unstable, hypothermic, and developed bleeding diathesis. A few hours after surgery, the patient died. Pathology of the specimen showed atherosclerosis and medial degeneration.

Case 2

A 52-year-old male patient with mental illness was admitted to the hospital after a sudden collapse. History taken was complicated because of the mental illness. On physical examination, he had a soft, nontender abdomen, with an abdominal bruit. On the day of admission, he produced dark stool and was admitted for further analysis of his suspected melena. A gastroscopy was planned. Although blood was administered, he collapsed again 1 day after admission. A CT showed a ruptured hepatic aneurysm and a splenic aneurysm (Figure 3). An endovascular procedure (coiling) was attempted but was not successful because the catheter could not be positioned in the hepatic artery. At laparotomy, a hematoma was evacuated and the aneurysm of the proper hepatic artery was excluded by ligation on both sides. An interposition graft was not deemed necessary because the liver was not ischemic. Furthermore the splenic aneurysm was treated with an aneurysmectomy and splenectomy. In the postoperative period

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Figure 1. Ruptured splenic artery aneurysm..



Figure 3. Ruptured splenic artery aneurysm and hepatic artery aneurysm.



Figure 2. Splenectomy specimen with opened splenic artery aneurysm.

at the ICU, the patient developed a myocardial infarction and a cerebrovascular accident, from which he did not recover. Six days after surgery, he died. Autopsy showed extensive medial degeneration and no atherosclerosis.

Case 3

A 75-year-old female patient with a history of COPD (GOLD II) and atrial fibrillation for which she received oral anticoagulants. She was admitted to the hospital with abdominal pain on her left-hand side. During her admission, the hemoglobin slightly decreased, without hemodynamic instability. A CT was performed, which showed a hematoma in her left

retroperitoneum and an aneurysm of the splenic artery. An endovascular coiling of the splenic artery was performed successfully (Figure 4). After this procedure, the abdominal pain on her left-hand side slowly decreased and she was discharged after a few days. At follow-up, the abdominal pain disappeared and a CT showed a completely thrombosed aneurysm.

Case 4

An 85-year-old male patient presented at the emergency department with acute abdominal pain and hypotension. The patient used oral anticoagulants (phenprocoumon) for atrial fibrillation. Because an abdominal aneurysm was suspected, a CT was performed. A hematoma in the hepatoduodenal ligament was found, as well as an aneurysm of the common hepatic artery (Figure 5). The patient was treated with vitamin K to normalize his international normalized ratio (INR) level. An emergency coil embolization was planned, but during this procedure the common hepatic artery could not be cannulated due to an occlusion. On angiogram, there were no signs of active bleeding (Figure 6). The patient was further treated conservatively. The abdominal pain slowly decreased, and he was discharged after a few days of observation.

Case 5

A 50-year-old woman was analyzed for abdominal pain microscopic hematuria. A CT showed unexpectedly a small aneurysm of the splenic artery (Figure 7). She was referred to the vascular surgeon by the urologist. History taken revealed that she was physically abused in the past, and therefore this aneurysm is

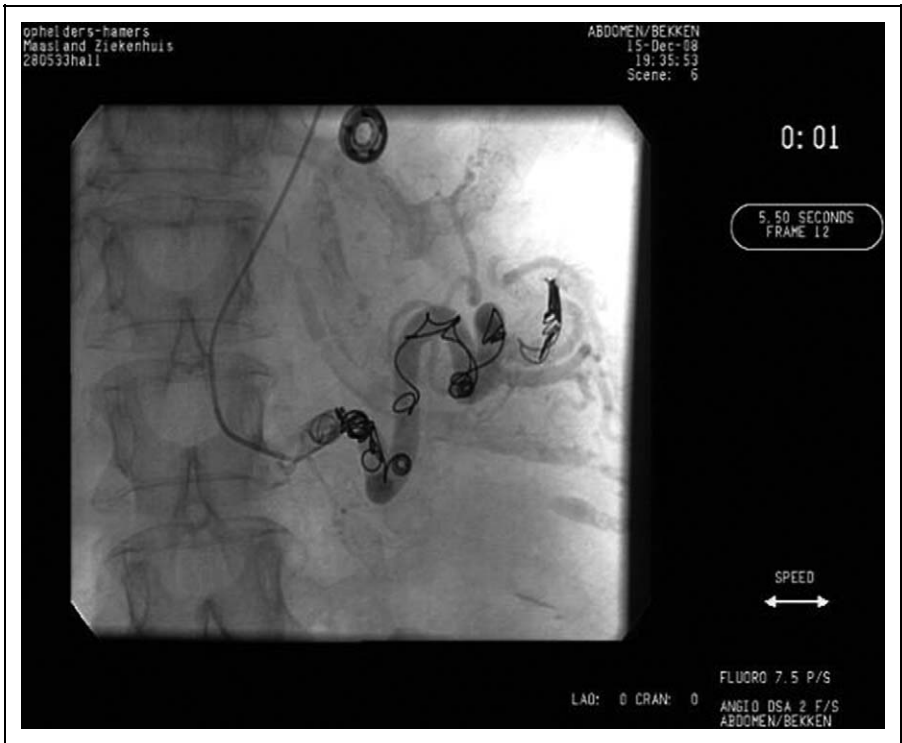


Figure 4. Endovascular coiling of splenic artery aneurysm.

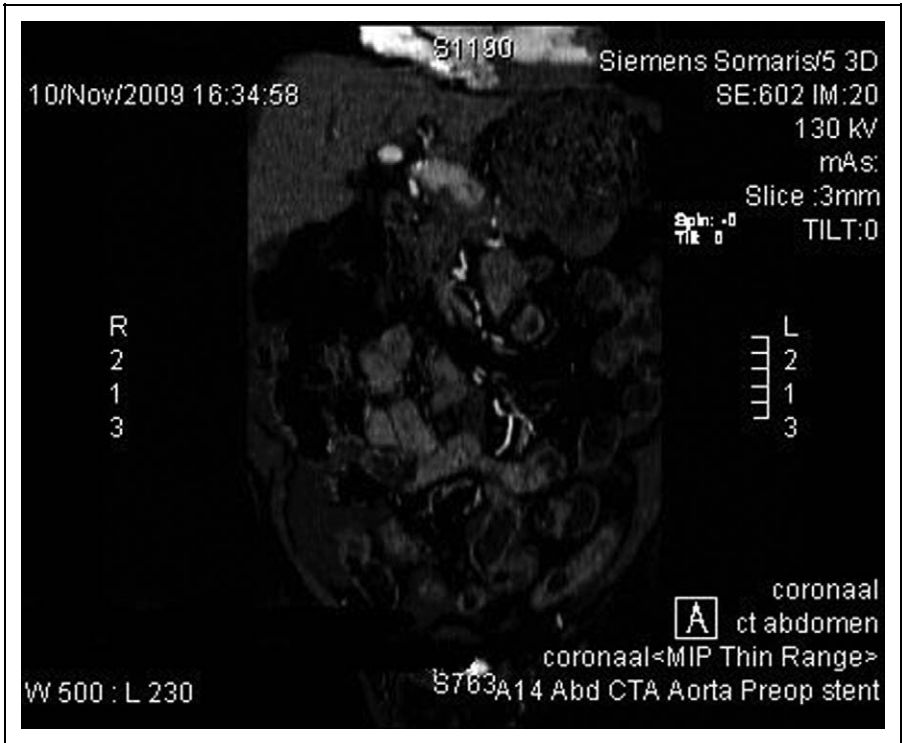


Figure 5. Hematoma in the hepatoduodenal ligament and aneurysm of the common hepatic artery.

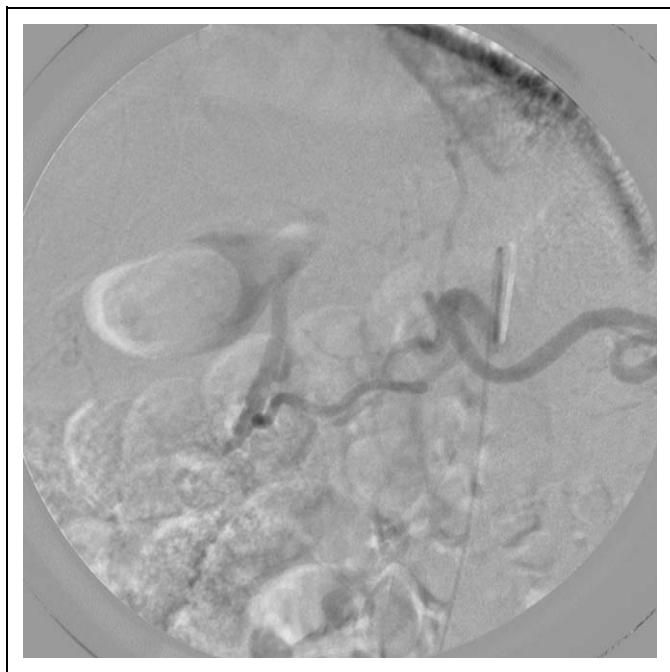


Figure 6. Angiogram of hepatic artery aneurysm, with no signs of active bleeding.



Figure 7. Small aneurysm of the splenic artery.

believed to be a traumatic aneurysm. The patient was treated electively by coil embolization of the aneurysm (Figure 8).

Discussion

Incidence

Visceral artery aneurysms are a rare condition, the incidence in postmortem studies varies from 0.01% to 0.2%. With the more

widespread use of CT and angiography, many aneurysms are detected asymptotically today.⁴ In case of rupture, which occurs in about 25% of the cases, the mortality rates¹ vary from 25% to 70%.

The most common VAA is the splenic artery (60%), followed by the hepatic artery (20%). Less common are the celiac trunk and its other branches and the superior mesenteric artery (Table 1).

Multiple aneurysms are described in patients with hepatic aneurysms. Other aneurysms are reported in the visceral and nonvisceral circulation in 31% and 42%, respectively.⁶ Abdominal aortic aneurysms are seen in about 20% in association with VAAs.⁵

Renal artery aneurysms are usually described separately, as they show a slightly different natural history, and they are strongly associated with hypertension.¹

Rupture is most frequently seen in the hepatic artery (80%) in contrast to the splenic artery where rupture is rarely seen. Splenic artery aneurysm rupture occurs in female patients 4 times more than in male patients. A ruptured splenic artery aneurysm during pregnancy is associated with high mortality rates for the mother and the unborn child.^{4,5} The frequency of deliveries seems to be of importance since a higher rupture rate has been reported in patients with multiple deliveries than those who have had none. It is thought that the various changes during pregnancy, secondary to hormone and hemodynamic alterations, such as great splenic AV shunting, seem to be the causing factors of this disease.⁷

Etiology

VAAs include both true aneurysms and pseudoaneurysms. In true aneurysms, all 3 layers of the arterial wall are intact, pseudoaneurysms lack a complete arterial wall. Atherosclerosis seems to play a secondary role in the development of these lesions and does not always represent the first pathological mechanism.³ Some VAAs are of degenerative origin, demonstrating deficiency of the arterial media wall with reduced smooth muscle cells and reduction or fragmentation of fibers.²

Prior to the era of antibiotics, most were considered to be mycotic and/or inflammatory and frequently associated with endocarditis. Atherosclerosis accounts for an estimated 32% of the cases. Medial degeneration (24%), abdominal and surgical trauma (22%), and infectious or inflammatory diseases (10%) are other well-known causes⁸ (Table 2).

Defects in the congenital arterial wall can also cause VAAs, including Marfan syndrome, Ehlers-Danlos syndrome, hereditary hemorrhagic telangiectasia, Osler-Weber-Rendu disease, mucocutaneous lymph node syndrome (Kawasaki), and fibromuscular dysplasia.

Systemic diseases with potential for diffuse vascular abnormality and multiple aneurysms like juvenile rheumatoid arthritis (Still disease), systemic lupus erythematosus, Hashimoto thyroiditis, and polyarteritis nodosa are other rare causes. Gestation and portal hypertension have also been shown to be



Figure 8. Endovascular coiling of splenic artery aneurysm.

Table 1. Incidence and rupture rate in VAA's

	Incidence	Rupture rate
Splenic artery	60%	20%
Hepatic artery	20%	80%
Superior mesenteric artery	5%	38%
Celiac trunk	4%	80%
Gastric and gastroepiploic arteries	3%	90%
Intestinal arteries	2%	
Pancreatic duodenal arteries	1%	75%
Jejunal, ilial, and colonic arteries	3%	30%
Gastroduodenal and inferior mesenteric arteries	<2%	

Table 2. Etiology visceral artery aneurysms (VAA's)

Atherosclerosis	32%
Medial degeneration	24%
Abdominal and surgical trauma	22%
Infectious and inflammatory diseases	10%
Congenital diseases (Marfan, Ehlers-Danlos, hereditary hemorrhagic telangiectasia, Osler-Weber-Rendu disease, Kawasaki, and fibromuscular dysplasia)	
Systemic diseases (juvenile rheumatoid arthritis (Still disease), systemic lupus erythematosus, Hashimoto thyroiditis, and polyarteritis nodosa)	

important factors in not only splenic aneurysms but also hepatic aneurysms.⁹

Symptoms

The majority of patients with visceral aneurysms are asymptomatic prior to rupture. The first presentation of a hepatic artery aneurysm is rupture in 80% of the cases.⁸ Of the patients who present with a rupture of a hepatic artery aneurysm, abdominal pain is present in 55%, and gastrointestinal hemorrhage or

hemobilia occur in up to 46%. Quinke triade described as the classic triade of epigastric pain, hemobilia, and obstructive jaundice is only present in up to one third of the patients. Physical examination is usually normal, although large aneurysms may be associated with a pulsatile mass or an abdominal bruit.¹⁰

Of those patients who present with a ruptured splenic aneurysm, the majority demonstrates a frank rupture, characterized by abdominal or chest pain followed by hemodynamic instability 6 to 96 hours later. This delay onset of rapid blood loss is

thought to be secondary to the “double rupture phenomenon,” which results from initial tamponade of the bleeding within the lesser omental sac and delayed onset of intraperitoneal hemorrhage.¹¹

Superior mesenteric artery aneurysms can present with intermittent abdominal pain. These lesions often contain mural thrombosis, which can cause symptoms by chronic embolization or may lead to thrombosis, resulting in acute mesenteric ischemia.

Patients with gastric artery aneurysms and gastroepiploic artery aneurysms often present with gastrointestinal bleeding. A CT scan can reveal these aneurysms.

Patients with pancreatic artery aneurysms and gastroduodenal artery aneurysms, often associated with pancreatitis, have symptoms of abdominal pain and shock.

Aneurysms of the mesenteric branch and inferior mesenteric artery are rare. These patients may present with abdominal pain, gastrointestinal hemorrhage, or shock. Most of these aneurysms are discovered during exploratory laparotomy for gastrointestinal bleeding or intraperitoneal hemorrhage.⁴

Treatment Options

Treatment options for VAA depend on the presentation of the patient and location of the aneurysm, as well as patients' comorbidities and risk factors.⁴ Various options for treating VAAs include open surgical repair (ligation, aneurysmectomy, venous grafting, and synthetic grafting), endovascular treatment (embolization and covered stent placement), and most recently laparoscopic surgery (ligation).⁵

In 1809, Wilson first described a hepatic artery aneurysm. The first successful operative repair, ligation of the common hepatic artery was performed by Kehr in 1903, followed by the first successful revascularization⁹ by Paul in 1951.

In emergency conditions, ligation of the aneurysm without reconstruction is the most widely used technique, especially if collateral circulation is adequate. This approach has also been proposed for treatment of aneurysms involving the superior mesenteric artery. There is the risk of small intestine ischemia reported in up to 40% in small series. For ruptured aneurysms involving small arteries such as the pancreaticoduodenal artery, gastroduodenal artery, and gastroepiploic artery, simple ligation is the preferred technique because of difficulty in performing reconstruction and the low risk of organ ischemia. Ligation can also be used for emergency treatment of aneurysms involving the celiac trunk and common hepatic artery with a low risk of hepatic ischemia, provided that ligation is preformed distal to a functional gastroduodenal artery.⁵ Splenectomy was the most common operation in the past for patients with splenic artery aneurysm, however several studies have highlighted the importance of splenic preservation to prevent infectious complications in the perioperative period. Splenic artery aneurysms are often saccular and localized in the proximal or middle third of the artery; for this reason aneurysmectomy with end-to-end anastomosis can be safely performed with the preservation of the spleen.³

Endovascular techniques, including transcatheter embolization and covered stent placement, can be used to treat most VAAs regardless of the clinical presentation, etiology, or location of the aneurysm. Endovascular treatment is also indicated for ruptured VAA in hemodynamically stable patients, especially when the aneurysm is poorly accessible for surgical treatment or when operative risks are high. The main limitations to endovascular treatment are availability of facilities for emergency treatment and the risk of failure due to difficulty in achieving selective catheterization.⁵ True- and pseudoaneurysms require different transcatheter coil embolization techniques. Pseudoaneurysms should be treated by proximal and distal coil embolization, but true aneurysms may be treated only by coil packing.¹² Although experience with endovascular treatment repair has been modest, data suggest that these techniques offer the potential to intervene with very low mortality. The associated morbidity in this group approaches 25%, transient postembolization syndrome or incomplete aneurysm exclusion are most often present. The latter is of danger since the aneurysm is not excluded and therefore not prevented from rupture.¹¹

Visceral artery aneurysms are seen more often these days with the more widespread use of CT and angiography. Asymptomatic aneurysms should be treated aggressively, the natural course seems progression to rupture.⁸ Treatment of asymptomatic VAAs larger than 2 cm and aneurysms in women of childbearing age is recommended.^{1,3,5,6,11}

Conclusion

Visceral artery aneurysms are a rare condition, and in case of a rupture, which occurs in about 25%, they have a high mortality. Treatment options for VAA depend on the presentation of the patient and location of the aneurysm, as well as patients' comorbidities and risk factors. Various options for treating VAAs include open surgical repair, endovascular treatment, and laparoscopic surgery.

Visceral artery aneurysms are seen more often these days with the more widespread use of CT and angiography. Asymptomatic aneurysms should be treated aggressively, since they tend to rupture. Treatment of asymptomatic VAAs larger than 2 cm and aneurysms in women of childbearing age is recommended.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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