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Visceral aneurysms: Old paradigms, new insights?

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A B S T R A C T

True visceral artery aneurysms (VAAs) are a rare entity with an incidence of 0.01–2%. The risk of rupture varies amongst the different types of VAAs and is higher for pseudo aneurysms compared with true aneurysms. Size, growth, symptoms, underlying disease, pregnancy and liver transplantation have all been associated with increased risk of rupture. Mortality rates after rupture are around 25%. The splenic artery is most commonly affected and the etiology is predominantly atherosclerosis. Open repair can be done by simple ligation or reconstruction of the artery, while endovascular options include embolization or using a stent graft. Location, collateral circulation and medical condition of the patient should all be taken into account when an intervention is planned. We compared types of treatment and searched for risk factors for rupture but unfortunately, the level of evidence found in the literature is low. Therefore, deciding when and how to treat a patient with a VAA based on the current literature, remains challenging for clinicians.

Introduction

VAAs are defined as aneurysms of the celiac (CA), superior (SMA) or inferior mesenteric (IMA) arteries and their branches. They are a rare entity with a reported incidence of 0.01%–2% [1]. The incidence of 0.78% in nearly 3600 abdominal arteriographic studies may better reflect their true frequency in the general population, although postmortem studies have found an incidence of splenic artery aneurysms (SAA) of 10.4% [2,3]. The first description of a VAA was done by Beaussiers in 1770 when he found a splenic artery aneurysm on autopsy. Both this case, and a second case reported by Parker in 1844, were omitted from the literature for many years and mistakenly given to Crisp. In 1871 Quincke first described the “classic” triad of jaundice, biliary colic and upper gastrointestinal hemorrhage caused by a hepatic artery aneurysm rupture. Kehr performed the first successful surgical procedure in 1903 when he ligated a hepatic artery aneurysm.

Depending on the location of the aneurysm, different symptoms can be expected. However, clinical symptoms and signs are frequently unspecific and since VAAs are rare, they are not often suspected, leading to a delay in diagnosis. The mortality rate of a ruptured VAA is around 25%, although various rates have been reported in the literature and differ between location [4–6]. Risk of rupture is also related to size, growth rate and underlying disease [4,7]. With the increasing use of diagnostic tools like ultrasonography, computed tomography angiography (CTA) and magnetic resonance imaging (MRI), the incidence incidental finding of asymptomatic VAAs has increased.

Treatment of VAAs can be done by either open or endovascular repair. Discriminating between VAAs that can be monitored and those that require an intervention remains a challenge as no randomized controlled trials (RCTs) have been performed in this area. This chapter provides an overview of the currently available literature on VAAs. We will separately describe them by anatomic location and distinguish true VAAs from visceral artery pseudo aneurysms (VAPAs).

Epidemiology, etiology and natural behavior

In VAAs, all three layers of the arterial wall are intact, while VAPAs are actually contained ruptures lined by the adventitia or perivascular tissues. VAAs are a focal dilatation of the artery with a diameter more than 1.5 times the normal diameter of the vessel.

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They are located in the CA, SMA, IMA or their branches. Renal artery aneurysms are usually not considered VAAs as they have a slightly different etiology. The splenic artery is most commonly affected (60%). Second is the hepatic artery (20%), followed by the SMA (5%) and the CA (4%, see Fig. 1) [5,6,8,9]. Other possible locations are the IMA, gastric, gastroepiploic, intestinal, pancreatic, gastroduodenal (GD) and pancreaticoduodenal (PD) arteries which together account for 11% of the locations. Table 1 summarizes the most common location per artery [10,11]. VAPAs mostly occur in the hepatic artery (39%), the CA or its branches (39%) [12].

VAAs are rare with an incidence of 0.01–2% [1]. With the increasing use of diagnostic tools for complaints unrelated to the VAA, the finding of asymptomatic VAAs has increased and the growing number of interventions in the arterial bed and the biliary tract has increased the number of VAPAs. Multiple etiologies account for the development of VAAs [5,8]. The most common pathway is through atherosclerosis (32%), followed by medial degeneration (24%) and abdominal trauma (22%). Hyperflow conditions (e.g., pregnancy, portal hypertension), connective tissue disorders (e.g., Marfan, Ehlers–Danlos, fibromuscular dysplasia), vasculitis (e.g., polyarteritis nodosa, Takayasu, Kawasaki), neurofibromatosis and the antiphospholipid syndrome have all been associated with the formation of VAAs (see Table 2). VAPAs are the result of iatrogenic injury, infection or abdominal trauma.

Recently a paper by Corey et al. was published describing the natural behavior of VAAs while under surveillance [7]. During a study period of 20 years, 176 VAAs with a mean size of 16.28 mm (8–41 mm), were monitored instead of immediately treated. Of these, 91.3% remained stable over time (mean of 36.1 months, ranging from 2 to 155 months) without any change in size. There were no ruptures in this group of patients and only 5.8% required an intervention during follow-up. None of the hepatic, jejunal or IMA aneurysms grew in time. The only aneurysms showing some growth were located in the CA, SA, PDA, GDA and SMA.

Table 1
Location per type of aneurysm.

Type of VAA	Most common location per artery
Splenic artery	Distal third of the artery Bifurcation distal to short gastrics Splenic hilum
Hepatic artery	Extrahepatic (80%): - Common hepatic artery 60% - Right hepatic artery 30% - Left hepatic artery 10% Intrahepatic (20%)
SMA	Proximal 5 cm
CA	Distal to site of chronic vascular compression

VAA = visceral artery aneurysm, SMA = superior mesenteric artery, CA = celiac artery.

Comparing intact and ruptured VAAs

Shukla et al. [6] studied 261 patients of which 181 were repaired. In 77 patients, the VAA had ruptured (rVAA), in 104 it was intact (iVAA). The percentage of men was 63.2% in de rVAA group compared with 42.0% in the iVAA group ($p = 0.005$). Of the patients with a rVAA, 29.7% were in hemodynamic shock. The most common cause of iVAA was degenerative disease (31.7%), for rVAA this was inflammatory/pancreatitis (33.8%). VAPAs were more common in rVAAs (81.8%) compared with iVAAs (35.3%, $p < 0.001$). The perioperative complication rate was higher for rVAAs (13.7% vs 1%, $p = 0.003$). Mortality for rVAAs at 30 days was 13%, at 1 year 32.5% and at 3 years 36.4%, all significantly higher compared with iVAAs (0%, 4.1% and 8.3% respectively). The 30-day mortality for rVAAs was highest in splenic artery aneurysms (27.7%) and the complication rate in rVAAs was highest for SMA (62.5%) and hepatic aneurysms (41.1%).

Pitton et al. [5] described 233 patients with 253 VAAs. Fifteen percent of the patients presented with a rupture, all of them had symptoms. Of the rVAAs, 76.3% were VAPAs. Only 3.1% of the VAAs ruptured. There was no significant difference between the diameter of rVAAs and iVAAs (14.8 vs 16.3 mm). The greatest diameters were found in splenic artery aneurysms. After treatment, the 30-day mortality was 6.7% in rVAAs compared with no mortality in iVAAs.

A univariate regression model examining risk factors associated with rupturing was presented in the study by Corey et al. [7] An odds ratio of 1.1 ($p = 0.004$) was found for aneurysm size, of 11.2 ($p = 0.0002$) for PDA and GDA aneurysms and of 32.5 ($p = 0.01$) for Ehlers–Danlos. There was no difference in 5-year survival between patients that underwent surveillance compared with those that underwent early repair.

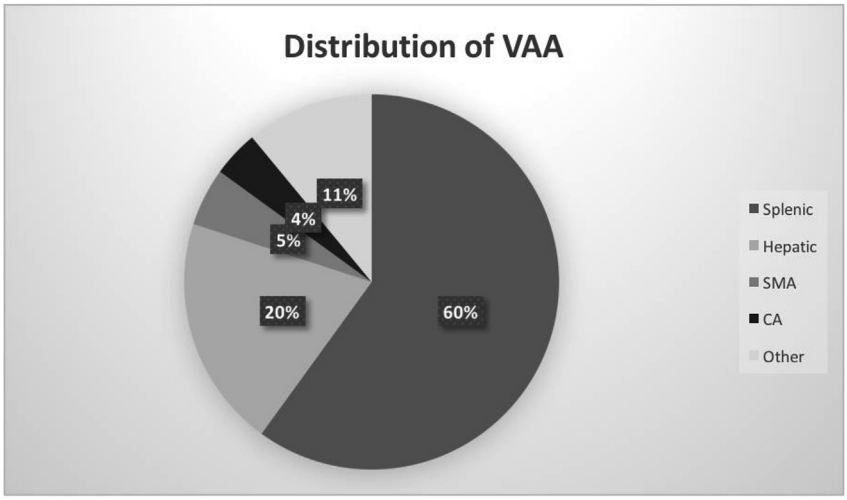

Fig. 1. Distribution of visceral artery aneurysms by arterial bed. VAA = visceral artery aneurysm, SMA = superior mesenteric artery, CA = celiac artery.