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MANAGEMENT OF VISCERAL ARTERY ANEURISMS: A PICTORIAL ESSAY

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Abstract

Visceral artery aneurysms are an uncommon vascular disease. Despite their low incidence of 0.01–2%, aneurysms of visceral arteries represent an important disease because of their high risk of rupture and mortality rate of 20% to 75%, due to life-threatening hemorrhage.

The aim of this article is to discuss visceral artery aneurysms management from diagnosis to treatment, with a particular focus on endovascular techniques and follow-up protocols.

Keywords: *Visceral artery aneurysms, diagnosis, treatment, endovascular techniques*

Introduction

Visceral artery aneurysms (VAAs) are an uncommon vascular disease. Despite their low incidence of 0.01–2% [1], aneurysms of visceral arteries (VAAs) represent an important disease because of their high risk of rupture and mortality rate of 20% to 75%, due to life-threatening haemorrhage [2,3].

3000 cases have been reported approximately in the literature, with an incidence of 1% within the general population and 0.1% to 10% in autopsy series [4]. More recent reports include large numbers of asymptomatic patients in whom aneurysms are discovered incidentally [5].

VAAs are usually classified into true and false aneurysms.

True aneurysms may occur as a result of underlying arterial pathologies such as atherosclerosis, fibromuscular dysplasia, and arteritis. False aneurysms, or pseudoaneurysms, are ruptures of the artery contained by the adventitia or by the perivascular soft tissues. False aneurysms may occur following an inflammation, infection, or trauma [6].

The most common sites are the splenic artery (SAAs) in 60% of cases, the renal artery (RAAs) in 22%, the hepatic artery (HAAs) in 20%, the superior mesenteric artery (SMAAs) in 5.5%, the celiac artery in 4%, the gastric and gastroepiploic artery in 4%, the gastroduodenal artery and pancreatic branches in 6%, the jejunal and ileocolic arteries in 3%, and the inferior mesenteric artery in less than 1% [6,7].

The incidental diagnosis of visceral artery aneurysms is usually made with Color-Doppler ultrasound (ECD), which can be used to evaluate the flow within the vessels as well as the origin of the sac, its size and the presence or absence of thrombi [8].

Catheter-based angiography used to be the gold standard exam to diagnose vascular disease.

3D imaging with CT and CT-angiography, that allows visualizing the aorta and its branches, may detect visceral artery aneurysms with greater frequency in symptomatic and asymptomatic patients [9].

3D CE-MRA represents another non-invasive diagnostic imaging technique [10].

Treatments depend on site, type and size of the aneurysm as well as patient's medical condition [11]. Several publications recommend treatment for VAAs >2 cm in diameter. However, clinical experience suggests that the risk of rupture depends not only on the aneurysm diameter, but also on its site [12].

In the past, surgery was the treatment of choice for VAAs; but the surgical approach is complex and associated with elevated mortality and morbidity. Over the last few years, endovascular treatment has been reported as a safe and effective alternative [1].

In more recent decades, endovascular treatments include the exclusion of the aneurysm with embolization agents, usually released through the most widely used embolization coils, placement of coated stents or inert particles or combination of these techniques [13]. The choice of endovascular treatment is influenced by aneurysm site and shape.

In case of saccular aneurysms, the embolization is performed by the aneurysm's neck catheterization, which is carried out by using coils and/or cyanoacrylate or thrombin [13].

Conversely, endovascular exclusion of fusiform aneurysms requires the placement of coils into the efferent branches first and then into the feeding artery so that the organ perfusion can be maintained by collateral vessels, even if only partially [1].

The artery anatomy and the aneurysm site influence treatment with stent grafts, which require an appropriate artery diameter to work properly [1].

The follow-up protocols after embolization have not been well defined yet. There is not an optimal CT-angiography method to evaluate treatments involving embolic agents use such as micro coils and glues [14]. CT evaluation of complete aneurysm occlusion is indeed complicated by coil and glue artefacts [15]. CE-MRA achieves a moderate to high diagnostic performance to detect neck recanalization, hemodynamic status and organ infarction secondary to coil embolization [13].

CT-angiography, on the other hand, is reliable to evaluate integrity and patency of stent grafts, evolution of the aneurysm, and chronic ischemic organ injuries.

Contrast-enhanced US (CEUS) is also used as a follow-up exam after embolization or coated stent placement [16].

The aim of this article is to discuss VAAs management from diagnosis to treatment, with a particular focus on endovascular techniques and follow-up protocols in SAAs, RAAs, HAAs, SMAAs.

Splenic Artery Aneurysms (SAA)

Splenic artery aneurysms (SAA) are the most common, accounting for 60% of all cases of VAAs [7] and are also the third most frequent peripheral artery aneurysms following the aorta and the iliac artery ones [13, 17]. Most of these aneurysms are small (2 to 4 cm in diameter), saccular and sited in the intermediate or distal segment of the splenic artery in 80% of cases. Giant aneurysms (more than 10 cm in diameter) are rare [7, 13].

In the past, SAAs usually presented with rupture or were incidentally discovered at autopsy. Over the last few decades, along with the increased use of non-invasive cross-sectional imaging, these lesions have been detected more and more frequently in asymptomatic patients too. SAAs seem to be more frequent in women, during pregnancy, especially in multiparae and in those affected by portal hypertension [13]. The correlation between SAAs and pregnant women is likely due to hormonal effects of estrogen and progesterone [18].

There is not yet consensus around the indication of treatment, but a lot of authors agree on the following indications.

True aneurysms should be treated when symptomatic. If asymptomatic, they can be treated when their largest diameter is greater than 2 cm or when complications occur [19]. Same indications should be followed in case of pregnancy, women of childbearing age, children, patient with portal hypertension listed for liver transplant.

False aneurysms should be nearly always treated, as symptomatic in 90% of cases and because of their parietal fragility. Patient age and comorbidity influence treatment choice too [7, 20].

Treatment of SAAs relies on surgery traditionally, depending on VAAs site firstly, patient age, surgical risks and clinical status. Surgical options include

ligation or aneurysm resection, when located in the proximal or middle portion of splenic artery, splenectomy, if sited near to the splenic hilum [19].

More recently, several endovascular techniques have offered an important alternative to surgery. The “packing technique” is the most used and well indicated for the narrow-necked aneurysms. This technique allows maintaining the splenic artery and its collateral vessels patent by filling the aneurysms with coils or other embolic agents.

Another technique is called the “sandwich method” involving the release of microcoils distally and proximally to the aneurysm neck. This one is more indicated for fusiform or wide-necked aneurysms and pseudoaneurysms [21].

Stent graft repair is usually performed in case of wide-necked aneurysms and pseudoaneurysm due to pancreatitis [22]. This method offers the great advantage of maintaining patency of the main splenic artery as well as its collateral vessels, which is important in case of portal hypertension. Its use is limited by the excessive tortuosity of the artery [6].

Hepatic Artery Aneurysms (HAAs)

Hepatic artery aneurysms (HAA) are the second most common type of VAAs after SAAs and are more common in men than women [22]. Aetiology was thought to be associated with mycotic infections and bacterial endocarditis, but atherosclerosis, connective tissue diseases and fibromuscular dysplasia are often seen as underlying disorders. HAAs are usually solitary and observed more into the extrahepatic than the intrahepatic arteries [22]. Extrahepatic aneurysms are associated with degenerative or dysplastic diseases, whereas the intrahepatic ones occur as a result of trauma, percutaneous biliary procedures, infection or vasculitis [2].

Risk factors for rupture are poorly defined due to its rarity. They include multiple HAAs and non-atherosclerotic aetiology [6-22]. The majority of HAAs are discovered incidentally but they are often symptomatic causing back or abdominal pain. When rupture occurs they present with gastrointestinal haemorrhage or haemobilia and obstructive jaundice [23]. Treatment is recommended in symptomatic and ruptured HAAs, and in some asymptomatic ones

whenever multiple or non-atherosclerotic. There is no agreement yet around size criteria for intervention in asymptomatic atherosclerotic HAAs [22]. Treatment techniques are similar to those mentioned for SAAs.

Superior Mesenteric Aneurysms (SMAAs)

Superior mesenteric artery aneurysms (SMAAs) are rare, accounting for 6% of all VAAs [7]. Aetiologies include atherosclerosis, collagen vascular disorders, cystic medial dysplasia, polyarteritis nodosa and infections [24]. Due to the increased use of cross-sectional imaging, SMAAs have been detected more often than they used to. If symptomatic, they present with abdominal pain or bleeding; in 38% of cases rupture occurs at presentation [25].

Management of SMAAs depends on patient's condition (hemodynamic status and surgical risk in particularly) and aneurysms anatomical features. As reported for SAAs, surgical options include aneurysmectomy, ligation and rarely bowel resection. Revascularization is recommended when symptoms or imaging evaluation are suggestive of mesenteric ischemia. Endovascular treatment is indicated in patients with anatomically conducive lesions such as narrow-necked aneurysms and those sited distally to the origin of the SMA.

Renal Artery Aneurysms (RAAs)

Renal artery aneurysms are uncommon, having a prevalence of 1% [25] of all aneurysms and 22% of all VAAs [6]. Women are more often affected. Hypertension and fibromuscular dysplasia are common associated disorders in true RAAs. Pseudoaneurysms are again secondary to trauma or infection [26]. Clinical presentation includes hypertension, haematuria, abdominal pain, decreased renal function and rarely rupture. In these patients, hypertension may be related to other associated conditions such as renal artery

stenosis, ischemia secondary to renal artery kinking, turbulence in the aneurysm and distal embolization from the aneurysm [26]. Rupture is rare but its incidence seems to increase in larger aneurysms and pregnancy. The rupture mortality rate is higher in pregnancy too (50% maternal mortality and 80% fetal mortality) [27-28].

Indications for treatment RAAs include symptomatic aneurysms, aneurysm of >2 cm in diameter, childbearing women [6], ruptured aneurysms, pseudoaneurysms and those associated with arteriovenous fistulas or dissection [29, 30].

Endovascular treatment options depend on the aneurysm site within the renal artery.

When the aneurism involves a peripheral intrarenal artery, embolization through coils or other embolic agents is recommended. If, instead, the main renal artery, its hilum or first-order branches are affected, aneurysm exclusion with self-expanding or balloon expandable stent graft is the treatment of choice. This technique preserves the main artery preventing a possible kidney dysfunction. In some cases aneurysms involving the hilum are challenging to treat so their treatment depends on neck features [6].

Conclusions

As reported in the literature, the increased use of cross-sectional imaging techniques has led to an increasing diagnosis of VAAs, especially in asymptomatic patients. CT-angiography, MRA and the US play an important role to identify the aneurysm features in order to plan its management, from the endovascular treatment to the most appropriate follow-up protocols [30, 31].

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Figure 1. SAA (A) Arteriogram shows a saccular aneurysm that originates from the proximal portion of splenic artery. (B) Axial oblique maximum-intensity-projection (MIP) image shows a stent graft that was placed to exclude the aneurysm from the circulation and to maintain the patency of the splenic artery. (C) Coronal oblique volume-rendered image shows clearly splenic artery stent. (D) Axial contrast enhancement ultrasonography (CEUS) shows the flow within the stent graft without perfusion of the sac

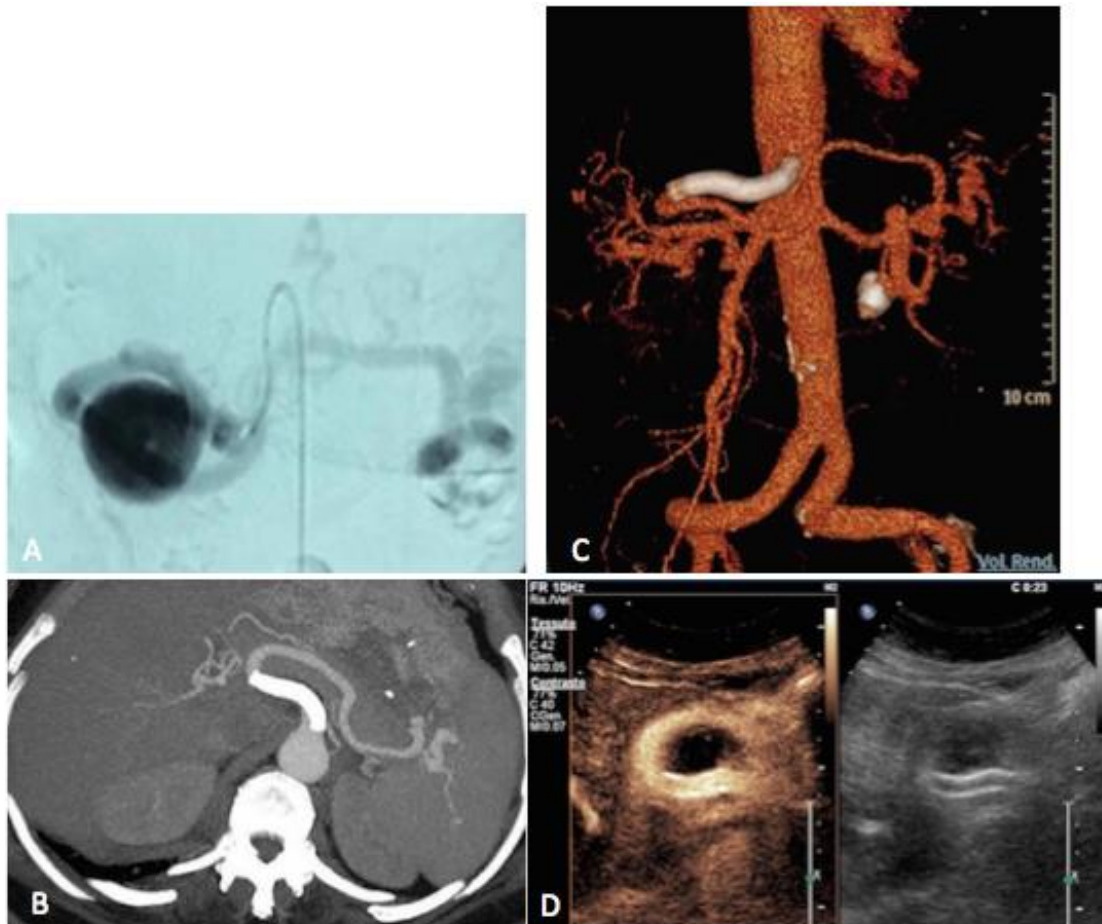


Figure 2. PHA (A) Axial contrast-enhanced CT arteriogram shows aneurysm of common hepatic artery. (B) Coronal maximum-intensity-projection (MIP) image showing aneurysm with wide neck. (C) Coronal oblique volume-rendered image shows again hepatic artery aneurysm. (D) Arteriogram shows coils that were placed into the sac, after embolization of gastroduodenal artery, and a covered stent placed to exclude the aneurysm from the circulation and maintaining patency of the artery. (E) CE-MRI shows complete aneurysm's exclusion by coils without metallic artifacts.

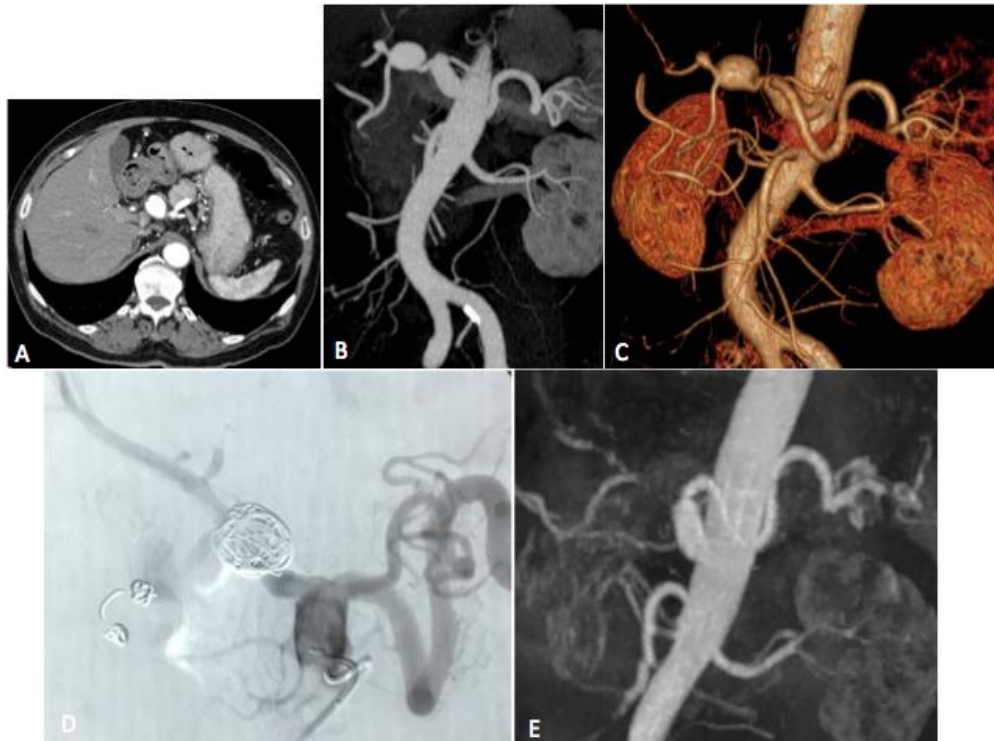


Figure 3. SMA (A) The saccular aneurysm located in the mesenteric origin of duodenal pancreatic artery, which was treated by using coils (B).

