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Endovascular Management of Visceral Arterial Aneurysms

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Visceral artery aneurysms are rare entities involving the celiac, superior mesenteric or inferior mesenteric arteries and their branches. While the natural history of these aneurysms is not well known as many are found incidentally, a definite risk of rupture and subsequent mortality has been demonstrated. There are several endovascular methods that an operator may choose to treat visceral artery aneurysms, and selection of the appropriate technique depends on the type and size of aneurysm and the anatomy of the affected artery. It is the aim of this paper to describe the indications, technical considerations and endovascular methods of treatment of visceral artery aneurysms and pseudoaneurysms. The following techniques of angiographic intervention are presented and discussed: isolation, covered stents, coil packing, liquid embolic agents and percutaneous thrombin injection. Where appropriate, individual aneurysm and artery specific treatment considerations are emphasized. To guide and assist practice, a suggested treatment algorithm is presented.

KEYWORDS Aneurysm, Pseudoaneurysm, Visceral, Embolization

Introduction

Visceral artery aneurysms (VAAs) are rare entities involving the celiac, superior mesenteric, or inferior mesenteric arteries and their branches, with a prevalence of 0.1%-2%.[1] A true aneurysm affects all 3 vessel walls, with multiple etiologies ranging from collagen vascular diseases to vasculitides (Table 1).[2-6] The splenic artery is most commonly affected, followed by the hepatic artery; however, any visceral artery may be involved. Although the natural history of these aneurysms is not well known, as many are found incidentally on studies performed for other reasons, a definite risk of rupture has been demonstrated.[1,6-9] Depending on size and location, mortality from rupture ranges from 25%-100%.[1,2] Treatment recommendations are based on the specific artery affected. Visceral artery pseudoaneurysms (VAPAs), or false aneurysms, only involve the outermost vessel wall and are secondary to infectious, inflammatory, or iatrogenic causes and have an increased propensity to progress to rupture as compared with true aneurysms. Commonly, they are found in the setting of pancreatitis or postbiliary intervention. In contrast to true aneurysms, pseudoaneurysms are often symptomatic, with a study reporting 92% requiring urgent intervention because of hemorrhage.[1] Because of the increased risk of rupture, it is recommended that all pseudoaneurysms be repaired.

There are several endovascular methods that an operator may choose, and the selection of the appropriate technique depends on the type and size of the aneurysm and the anatomy of the affected artery. Techniques include isolation, covered stents, coil packing, liquid embolic agents, and percutaneous thrombin injection. It is the aim of this article to describe the indications, technical considerations, and endovascular methods of treatment of VAAs and VAPAs.

Indications and Artery-Specific Considerations

As there have been no prospective studies, indications to treat VAAs and VAPAs are variable, with differing recommendations found in the literature. Universally, it is agreed that pseudoaneurysms of any size should be treated. There are few large studies that evaluate aneurysms by their parent artery; however, from those that do exist, a loose consensus has been reached (Table 2).[2,4,5,10-12] Particularly, repair is recommended for all VAAs that demonstrate interval growth or are symptomatic. Pseudoaneurysms are

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Table 1 Pathogenesis of VAA and VAPA

True aneurysms
Atherosclerosis (32%)
Medial degeneration or dysplasia (24%)
Abdominal trauma (22%)
Infection or inflammation (10%)
Hypertension
Connective tissue disorders
Marfan syndrome, Ehlers-Danolos syndrome, Osler-Weber-Rendu disease, systemic lupus erythematosus, Behçet syndrome, fibromuscular dysplasia, and alpha1-antitrypsin deficiency
Hyperflow
Portal hypertension, pregnancy, and median arcuate ligament syndrome
Vasculitis
Polyarteritis nodosa, Takayasu arteritis, Kawasaki disease, and Wegener granulomatosis
Neurofibromatosis
Hereditary hemorrhagic telangiectasia
Long-term amphetamine use
Pseudoaneurysms
Iatrogenic
Surgery, endoscopy, and interventional procedures
Trauma
Infection or inflammation

often found in the setting of multiple comorbidities, and these patients are particularly likely to benefit from an endovascular approach.

Celiac Artery

Celiac artery aneurysms are the fourth most common VAAs, accounting for 4% of all VAAs.[12,13] Association with other aneurysms is very common, with a study reporting a rate of 66%.[14] The risk of rupture has been demonstrated at 10%-20%, with 72% being asymptomatic at presentation.[14] Mortality at rupture has been reported as high as 100%.[1] These aneurysms are most likely due to atherosclerosis; however, the celiac trunk itself can be affected by median arcuate ligament syndrome with poststenotic dilatation owing to altered flow mechanics with retrograde flow through the pancreatic arcades.[13] Symptoms can include abdominal pain and mimic pancreatitis.[14]

Endovascular repair is most suitable in patients who are at high surgical risk with good, nondiseased collateral circulation, although studies of aortic aneurysm repair have shown that the proximal celiac artery may be embolized safely.[12,14,15]

Table 2 General Guidelines for Intervention

True aneurysms
Symptomatic
Women of childbearing age
Patients who may require a liver transplant
Nonatherosclerotic etiology (ie, connective tissue disease)
Interval growth >0.5 cm/y
Multiple hepatic VAA
>2 cm Hepatic, splenic, or celiac VAAs
Any size rare VAA (SMA and branches and IMA aneurysms)
Pseudoaneurysms
All

Splenic Artery

Splenic artery aneurysms are the most common true aneurysms, comprising 50%-75% of all VAAs. They are found in conjunction with other splanchnic aneurysms in 3% of cases and with other nonvisceral aneurysms in 14%.[3] Most are saccular and located in the middle-to-distal splenic artery.[10] The rate of rupture is low and has been estimated at 3%-20%.[2,3,16] True aneurysms have been found more commonly in women and in association with multiparty and portal hypertension, whereas pseudoaneurysms are more commonly seen in the setting of pancreatitis.[3,10]

Endovascular repair of splenic artery aneurysms must take into account the tortuosity of the parent artery. For example, stent graft placement is difficult in a distal tortuous vessel; however, it may be appropriate for proximal splenic aneurysms. With a rich collateral flow between the celiac axis and the superior mesenteric artery (SMA) via the pancreaticoduodenal arteries, as well as collateral flow to the spleen via the short gastric arteries, embolization can be undertaken with less risk for ischemia. As such, splenic artery aneurysms are good candidates for coil embolization.

Hepatic Artery

Hepatic artery aneurysms are the second most common true aneurysms comprising 20% of all visceral aneurysms.[4] They are found in conjunction with other VAAs in 31% of cases and nonvisceral aneurysms in 42%; however, they are most often solitary. In contrast to splenic artery aneurysms, true aneurysms have been found to be more common in men. An increased risk of rupture was found in cases of multiple aneurysms or with aneurysms of nonatherosclerotic etiology, such as those with fibromuscular dysplasia or polyarteritis nodosa.[4] Rupture rates as high as 80% have been described, with a mortality rate of 20%.[2] Hepatic artery aneurysms may rupture into the biliary tree, resulting in Quincke's triad of jaundice, biliary colic, and gastrointestinal hemorrhage. Overall, extrahepatic aneurysms are more common. In particular, the incidence of hepatic pseudoaneurysms is rising because of the increasing use of hepatic interventions such as percutaneous transhepatic cholangiography and transarterial chemoembolization.

Endovascular repair of intrahepatic aneurysms is considered first line because of the complicated nature of open repair. However, special attention must be given to limit end-organ ischemia when aneurysms of the proper hepatic artery are addressed, as there is not appropriate collateral flow to compensate in the event of complete occlusion.