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REVIEW ARTICLE

Splenic Artery Aneurysms

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Abstract: Splenic artery aneurysms (SAAs) are a rare arterial disease (1) and defined as a diameter of more than 50% of expected (0.9 - 1 cm) splenic artery diameter. They account for 60-70% of visceral artery aneurysms making them the most common of this group (4). They are often difficult to diagnose due to their vague or non-existing symptoms but present a high risk of rupture in certain patient groups making this a clinically important differential diagnosis.

Keywords: Splenic artery aneurysm; Splenic artery pseudoaneurysm; Splenic artery

SAA may be true or false (pseudo-SAA). True aneurysms account for 60% of SAAs and affect women four times more than men and are seen in the 5th and 6th decades of life[1,3,4]. True SAA risk factors are hypertension, atherosclerosis, smoking, cirrhosis, portal hypertension, liver transplantation, female sex and multiparity[1-4]. Less common risk factors include splenomegaly, collagen vascular disease, inflammatory conditions (SLE, polyarteritis nodosa), and anomalous splenic artery origin. Pseudo-SAAs occur more often in men and are most commonly due to acute or chronic pancreatitis, pancreatic pseudocyst and trauma[1,4].

SAAs are frequently missed or have a delayed diagnosis due to nonspecific or lack of clinical symptoms. An estimated 80% are asymptomatic and therefore incidentally diagnosed. Non-specific symptoms include epigastric or left upper quadrant pain, nausea, vomiting or anorexia[1,4]. Life-threatening complications of SAA are spontaneous rupture, fistulisation (stomach, duodenum, colon, pancreatic duct, pancreatic pseudocyst), and formation of an arterio-venous fistula[4]. Some 2-10% of patients will present with spontaneous rupture with a mortality of 10-40% (4). These patients may present with sudden on-set sharp left upper quadrant or epigastric pain, left shoulder tip pain, and haemodynamic instability[1,4]. 95% of ruptures occur in pregnancy (especially in the 3rd trimester) with a maternal and foetal mortality rate of 75% and 90%. Pregnancy itself presents a 24% risk of spontaneous SAA rupture[1,3,4]. Other high–risk groups are clinically symptomatic SAAs, diameter >2cm, increase in diameter, portal hypertension, liver transplantation and surgical treatments affecting portal system pressure[1,3,4]. There is some evidence of an inverse relationship between the amount of calcification and size of the aneurysm and therefore the risk of rupture[1]. Pseudo-SAAs have a significantly higher risk of rupture at 37% with a spontaneous rupture mortality rate of almost 100%[1,4].

The radiological diagnosis of an SAA may be made with abdominal ultrasound as an anechoic mass with or without peripheral calcification or on a CT angiography. MRI/MRA will better show smaller aneurysms with the added benefit of no radiation exposure. Digital Subtraction Angiography (DSA) is the gold-standard and identifies the exact location of the SAA as well as dynamic evidence of bleeding[1,4].

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Although it is invasive, DSA carries the added benefit of endovascular treatment in the acute setting[1,4].

It is generally agreed that incidentally found SAAs >2 cm or enlarging in high-risk groups (pregnant or likely to become pregnant women, liver transplant, portal hypertension), and in symptomatic patients regardless of size require treatment (1, 4). No consensus currently exists for the management of asymptomatic <2cm SAAs. Risk factor modification for peripheral arterial disease with lifestyle changes, smoking cessation, antiplatelets, antihypertensives and statins has been offered and seems logical but the evidence is poor[4]. One of the largest case series (128 patients) suggests that small (<2cm), heavily calcified and asymptomatic SAAs could be serially imaged or even discharged as they have a negligible risk of rupture[2]. It is widely agreed that splenic pseudoaneurysms should always be treated urgently as the risk of rupture is nearly 100%[1] and endovascular surgery is now the treatment of choice. Surveillance should be offered lifelong post liver transplantation but there is no consensus for follow-up of previously treated patients or conservatively managed patients with some having 6 monthly or yearly imaging[1].

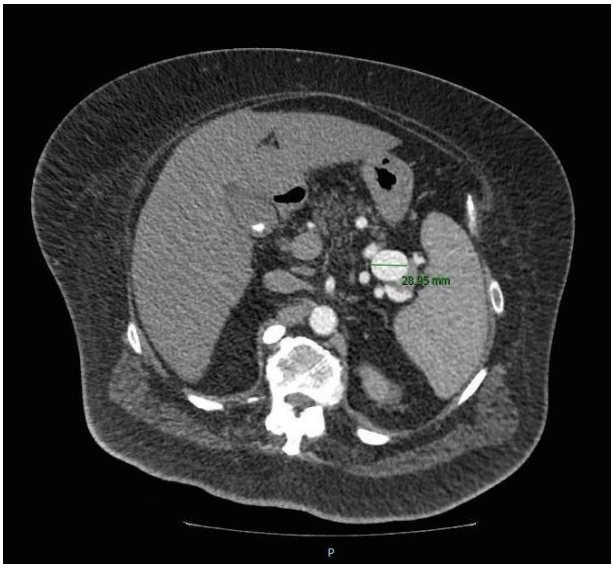

Figure 1. A CT angiogram showing a 2.8 cm splenic artery aneurysm.

There are a variety of treatment options and selection will depend on patient and anatomical factors as well as local expertise. Laparoscopic and endovascular (embolisation, coiling or stenting) surgery have largely replaced open surgery (ligation or reconstruction)[1], however there still remains limited long-term data for endovascular treatments. Open surgery is now largely relegated to ruptures with haemodynamic instability[1].

SAAs are a clinically important differential diagnosis to consider in certain patient groups. Their management remains controversial and may require the combined expertise of the vascular, radiology and upper GI surgery services. When incidentally found, they should be referred for evaluation and vascular MDT discussion. When presenting with complications, surgical input should be sought and ultimate treatment will depend on the exact nature of the complication.

Conflicts of interest

VS reports no conflicts of interest

SB reports no conflicts of interest

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