ABSTRACT

Visceral artery aneurysms (VAAs) are an increasingly encountered clinical problem. Although their incidence is low at about 0.2% in the general population, they are increasingly found on cross-sectional imaging. Therefore, it is justifiable to have a protocol for dealing with such lesions.

KEYWORDS: visceral; artery; aneurysm; surveillance; intervention

Incidence

Visceral artery aneurysms (VAAs) are an increasingly encountered clinical problem. Although their incidence is low at about 0.2% in the general population, they are increasingly found on cross-sectional imaging. Therefore, it is justifiable to have a protocol for dealing with such lesions. There have been no randomised controlled studies comparing expectant versus interventional management; although such aneurysms have been the result of recent review articles [1]. Indeed, it is not clear from RCT data what constitutes a dangerous size of VAA.

Aetiology

Broadly speaking, VAAs can be of two types. Firstly, false aneurysms result as a consequence of blunt or penetrating injury, intra-abdominal inflammation (usually splenic artery secondary to pancreatitis) or iatrogenic trauma. The commonest iatrogenic injury is to branches of the hepatic artery from percutaneous or endoscopic biliary procedures. Because of the unstable nature of these lesions, intervention is recommended on all false visceral aneurysms as soon as patient comorbidities allow [2].

Secondly, VAAs may be degenerative. Rarely this may be from a proven connective tissue disorder; more frequently they occur with arteriosclerosis. Interestingly, there is an association between VAA and median arcuate ligament syndrome [3].

Location

The splenic artery is the most common site of both true and false aneurysms of all the visceral vessels, accounting for up to 60% of VAA incidence [1]. These can be on the splenic artery proper or juxtacapsular. Aneurysms of the celiac and branches, SMA and renal arteries have roughly equal incidence. Aneurysms of lumbar, inferior mesenteric or marginal arteries are vanishingly rare.

Risk of rupture

The incidence of rupture is unknown because of a lack of long-term surveillance data; however most recent case series suggest that the risk of rupture or thrombosis is low in sub-2cm VAAs [3,4]. There exists no consensus on the type, frequency or duration of surveillance of these small aneurysms. Small juxtahilar splenic aneurysms and renal artery aneurysms may be non-invasively followed up by ultrasound. Aneurysms in other sites may require CT surveillance and it may be prudent to undertake this every two years; again there is no consensus.

Most studies [1,3,5] suggest intervention on aneurysms above 2cm, lesions displaying rapid expansion, patients with abdominal symptoms or in those with a suspicion of aneurysm thrombosis. The exact rupture rate is unclear but is between 10-20%
lifetime risk. It makes a 2cm VAA more likely to rupture than a 5.5cm abdominal aortic aneurysm [6].

**Special situations**

Certain physiological states place VAAs at higher risk of rupture. Hormonal changes and hyperdynamic current state in pregnancy place splenic artery aneurysms at particularly high risk of rupture [7]. Rupture of such aneurysms is associated with maternal and fetal mortality rates of 70% and 90% respectively. Similarly, patients with portal hypertension are more likely to both develop and suffer a rupture of VAAs; the decision to intervene must be balanced with the patients decreased life expectancy.

**Endovascular repair**

Endovascular repair is an attractive proposition in many VAAs. The ability to administer therapy under local anaesthetic and with the avoidance of major incisions is very favourable [6]. However, endovascular repair required planning, expertise and continued to follow up. Indeed, there is a documented risk of aneurysmal reperfusion of around 10% in elective endovascular intervention [5] that may require further open or endovascular procedures. The rate or morbidity and mortality of elective endovascular repair are low at 1-2% [5] although unsurprisingly this increases in the emergency setting.

Endovascular repair may require covered stenting or stent-assisted coiling (well described in the interventional neuroradiology literature). These techniques are used when the parent artery of the VAA must have preserved flow, for example in the SMA. If the parent artery is non-essential (e.g., splenic) then simpler methods such as coil embolisation can be employed. Specific therapy depends on expertise, aneurysm morphology and proximity to major branches [3].

**Open repair**

Open repair remains a viable alternative in the endovascular era. It is particularly valuable when VAAs are close to or involving the branch points of major visceral arteries, or where endovascular access vessels are diseased.

**Summary**

In reasonably healthy patients, it is advisable to keep degenerative asymptomatic sub-2cm VAAs under surveillance. False or mycotic aneurysms should almost always be repaired. VAAs over 2cm should prompt discussion with the vascular MDT to (or “intending to”) treatment planning. Splenic artery aneurysms in premenopausal women should be considered for intervention. There has been no randomised study comparing the results of open or endovascular intervention and it is likely that both methods will be complimentary depending on specific circumstances.

**Authors’ Statements**

**Competing Interests**

The authors declare no conflict of interest.

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