Abdominal aortic aneurysms: screening, epidemiology and open surgical repair

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Abstract

Abdominal Aortic Aneurysm (AAA) is a dilatation of the infra-renal abdominal aorta to greater than 3 cm. Population screening is offered to men in their 65th year in the UK. Patients with small AAAs (<5.5 cm) are entered into surveillance programs and should have cardiovascular risk factors managed aggressively. An AAA with ≥5.5 cm diameter should be considered for surgical repair to prevent rupture. Open surgical repair has proven to be a durable treatment for AAA and while less often performed than endovascular aneurysm repair (EVAR) it remains a common approach in the surgical management of AAA. While associated with higher short-term risks than EVAR, the long-term outcomes are similar and many younger patients have a preference for open repair as routine follow-up is not required.

Keywords AAA; aneurysms; aorta; open surgical repair; risk factors; screening

Introduction

An aneurysm is a focal, permanent dilatation of an artery or vessel to more than 50% of its normal diameter. The natural history of aneurysms is asymptomatic growth followed by rupture, which is catastrophic in many cases. For the infra-renal abdominal aorta, an absolute diameter of 3 cm is the usual threshold at which a diagnosis of abdominal aortic aneurysm (AAA) is made. AAA affects approximately 5% of men[1] and 0.74% of women[2] over the age of 65, and is responsible for approximately 4000 deaths per year in the United Kingdom (UK).[3] Men aged 65 are invited for AAA screening in the UK and the prevalence of AAA in this age group is just over 1.1.[4] Approximately 8700 operations are carried out each year for AAA,[5] 5500 of which are planned and 3200 are emergency. Approximately 30% of the planned and 70% of the emergencies are open AAA repairs. This article will review the options and evidence for open repair of AAA. Endovascular repair is discussed on pages 00-00 of this issue.

Epidemiology and risk factors for AAA

Large-scale cross-sectional studies of AAA screening with ultrasound scans have demonstrated that the prevalence of an infra-renal aortic diameter greater than 3 cm is approximately 5% in men over the age of 65 years,[6] though this may be decreasing. The prevalence of AAA in women is lower than in men but there have been no comparable large-scale studies in the female population. Existing evidence suggests that the prevalence of AAA in women is about five times lower than in men. Other non-modifiable risk factors for AAA include increasing age (AAA is very rare before the age of 55 years) and positive family history for the disease. Having a sibling affected by AAA increases the risk by approximately sevenfold.

The strongest risk factor for AAA is cigarette smoking, the impact of which by far surpasses genetic and all other modifiable risk factors. Prospective observational studies have demonstrated that current cigarette smoking can increase the risk of AAA development by as much as eightfold compared to those that have never smoked, and that duration of smoking also has a linear correlation with AAA development.[7,8] Smoking is also related to faster AAA growth.[9] Indeed, it is likely that public health measures aimed at smoking cessation may, in part, explain the reduction in AAA prevalence seen in the past 10–15 years.[10]

Other risk factors for development of AAA include hypercholesterolaemia and other atherosclerotic diseases. Hypertension is also considered to be a risk factor, although this remains unproven with randomized evidence. Interestingly, diabetes appears to protect against both the development and progression of AAA. The mechanism for this unexpected observation is unclear at present but could provide clues to developing novel pharmacological treatments for AAA.

Clinical presentation

Most AAAs are asymptomatic until rupture. Occasionally, large AAAs may compress surrounding structures such as the ureters, inferior vena cava or duodenum, leading to development of symptoms, but this is unusual. It is more common for AAAs to be discovered incidentally, as part of the diagnostic work-up for unrelated conditions or to be found as part of the national screening programme. Approximately 0.65%–2% of AAA are classified as inflammatory or mycotic. These can present with abdominal or back pain, weight loss, fevers and elevated inflammatory markers.

An asymptomatic AAA may be detected as a pulsatile abdominal mass through routine physical examination; however, the sensitivity of this is poor and is affected by the examiner's experience and the patient's body habitus. Patients with AAA may also present with ischaemic symptoms in the lower limbs secondary to acute thrombosis or embolization to the peripheral circulation, but again this is not common. Very rarely AAA can present with haemorrhagic and thrombotic complications due to disseminated intravascular coagulation.

AAA rupture classically presents as a triad of abdominal pain radiating to the back, haemorrhagic shock and a palpable pulsatile abdominal mass. However, it is important to recognize that not all cases of AAA will have all of these features. It is not uncommon for patients with ruptured AAA to be diagnosed as having ureteric colic, musculoskeletal back pain or other diagnoses commonly presenting to the emergency department. There should therefore be a high clinical suspicion of ruptured

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AAA in older patients presenting to the emergency department with unexplained abdominal, back or loin pain.

Screening for AAA

The aim of treatment for AAA is to prevent death due to AAA rupture, which has a mortality exceeding 80%. A major challenge is the fact that AAA are usually asymptomatic until rupture occurs. This has led to the development of ultrasound screening for AAA. Ultrasound is safe, inexpensive and has both a high specificity and sensitivity.[11] Randomized controlled trials in the UK, Australia and Denmark have been performed to assess the clinical and economic effectiveness of AAA screening in men. The largest study is the UK-based Multicentre Aneurysm Screening Study (MASS) that randomized 68,700 males aged between 65 and 79 years to receive an invitation to ultrasound screening or not.[1] The risk of aneurysm-related death was reduced by 42% in the group invited for ultrasound screening after 4 years of follow-up. On the basis of this evidence, the NHS AAA Screening Programme (NAAASP) now invites men for AAA screening in the year of their 65th birthday. In the year 2015/16, almost 230,000 men were screened through the programme in England, representing an 80% uptake of screening invitations.[4] Recent data has also shown that attendance for screening also reduces all-cause mortality.[12] Women are not currently offered screening due to reduced prevalence of the condition in women. Approximately 35% of deaths due to AAA occur in women, however, and the risk of AAA rupture is four times higher in women than in men for an equivalent aneurysm diameter.[9] This suggests that AAA in women may be a more significant disease than previously thought. Current research aiming to assess the clinical and cost effectiveness of screening women for AAA is ongoing.[13]

Intervention to prevent rupture of AAA

Surgery is the main therapeutic option to repair an AAA and prevent rupture. When considering surgery, the risk of aneurysm rupture should therefore be weighed against the risk of the surgery. Aneurysm size is the major determinant of rupture risk (Table 1).[14] Post-mortem studies have demonstrated that the majority of deaths secondary to AAA rupture occurred in AAA greater than 6 cm. In the 1990s there was, therefore a degree of equipoise in the best strategy for management of patients with smaller AAA and the UK Small Aneurysm Trial (UKSAT) provided pivotal evidence in this regard.[15] This study randomized 1090 individuals with AAA measuring 4.0 cm–5.5 cm to receive either surgical repair or ultrasound surveillance and surgery was offered to AAA exceeding 5.5 cm or who became symptomatic. After 5 years of follow-up there was no survival benefit with early surgical intervention and surveillance of AAA sized 4.0–5.5 cm was found to be a safe strategy with low rupture rate. Therefore in the UK, the current recommendation for small asymptomatic AAA (<5.5 cm) is ultrasound-based surveillance.

The vast majority of AAAs identified by screening are small.[16] There are currently no medical therapies that have been proven to alter the history of small AAAs. Observational studies have reported an association between statin use and slower AAA growth but this has been inconsistent and there are no high-quality randomized trials.[17] There is also observational evidence that statin use reduces risk of rupture. Trials of calcium channel blockers, beta-blockers, ACE inhibitors, pemirolast and doxycycline have not shown any significant effect on AAA growth. A trial of ticagrelor is currently ongoing and a recent observational study has suggested a potential role for metformin in limiting AAA growth.[18] While evidence from these trials does not support the use of these agents to prevent AAA growth, there is evidence that patients with small AAA are at high risk of other cardiovascular disorders and these agents may therefore be commonly used in patients with AAA to manage cardiovascular risk.[19]

Annual rupture rate of AAA by AAA diameter

AAA size (cm)	Annual rupture rate (%)
<3	0
3–3.9	0.4
4–4.9	1.1
5–5.9	3.3
6–6.9	9.4
7–7.9	24

Table 1

Open surgery for AAA

Open surgery to repair AAA is nearly always performed under general anaesthesia. Routine use of epidural anaesthesia reduces the respiratory and gastrointestinal complications associated with other forms of postoperative analgesia. The approach to the aorta is usually transperitoneal via a longitudinal or transverse laparotomy but some surgeons favour a retroperitoneal approach via a left flank incision. The intraperitoneal contents are mobilized to the right side of the abdomen and the infra-renal aorta exposed by incision of the posterior peritoneum. The duodenum is mobilized off the aortic neck and the left renal vein marks the upper extent of the dissection in most cases. A clamp site in the proximal infra-renal aortic neck is exposed and prepared. Approximately two-thirds of infra-renal AAA can be repaired with a tube graft but the presence of iliac aneurysmal and/or occlusive disease necessitates the use bifurcated grafts extending to the iliac or femoral arteries. A longitudinal aortotomy is made after proximal and distal cross-clamping, and back bleeding vessels (e.g., lumbar, median sacral and inferior mesenteric arteries) are carefully oversewn after removal of the thrombus. A suitably sized synthetic graft is selected and sutured into the aneurysm with polypropylene sutures using an inlay technique (Figure 1). Following haemostasis the aneurysm sac and posterior peritoneum are closed over the graft to reduce the chances of graft infection and/or aorto-enteric fistula.

Open repair is a major surgical procedure. The 2017 National Vascular Registry (NVR) report identified that 13,058 elective open infra-renal AAA repairs were performed in the UK in the years 2014–2016.[5] The overall in-hospital mortality rate was 3.1%.[5] Randomized controlled trials have reported a 30-day mortality rate of just over 4.5% for open AAA repair,[20] with a long-term overall mortality of 8.9 deaths per 100 person-years.[21]

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