BMJ Best Practice

Abdominal aortic aneurysm

The right clinical information, right where it's needed

Last updated: Feb 28, 2018

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Summary

- Patients are usually asymptomatic and their abdominal aortic aneurysm is detected incidentally. In the minority of patients who experience symptoms, abdominal, back, and groin pain are typical.

- Ultrasound remains the definitive test for initial diagnosis and screening. Imaging with computed tomography angiography or magnetic resonance angiography is used for anatomical mapping to assist with operative planning.

- For AAA detected as an incidental finding, surveillance is preferred to repair until the theoretical risk of rupture exceeds the estimated risk of operative mortality. Repair is indicated in patients with large asymptomatic AAA.

- Mortality during elective surgical repair is higher for women than men for both open repair (7.0% vs. 5.2%) and endovascular aneurysm repair (EVAR) (2.1% vs. 1.3%).

- Complications of treatment include acute kidney injury, limb ischaemia, spinal cord ischaemia, anastomotic pseudoaneurysm, graft infection, graft limb occlusion, and distal embolisation. Endoleak is a complication of EVAR.

BASICS

Definition

Abdominal aortic aneurysm (AAA) is a permanent pathological dilation of the aorta with a diameter >1.5 times the expected anteroposterior (AP) diameter of that segment, given the patient's sex and body size.[1] [2]The most commonly adopted threshold is a diameter of 3 cm or more.[3] More than 90% of aneurysms originate below the renal arteries.[4]
[Fig-1]

Epidemiology

Epidemiology varies by region, age, and sex, with large screening studies offering the closest approximation. A screening study of US veterans aged 50 to 79 years found the highest prevalence of AAA (AAA >3 cm) among white male smokers (5.9%).[10] Prevalence among men is 4 to 6 times higher than in women.[1] [11] Current prevalence in the UK National Health Service AAA screening programme is 1.34% (AAA >2.9 cm) among men invited for screening in their 65th year.[12]

A systematic review of studies reporting screen-detected AAA in women found that the pooled prevalence of AAA increased with age (>1% for women >70 years of age) and with smoking status (>1% for female ever-smokers; >2% for current smokers).[13] The prevalence of aneurysms among men increases by about 6% per decade of life.[9] In 2014 there were 9863 deaths attributed to AAA in the US.[14]

In the UK, deaths from AAA declined sharply from 1997 to 2009, with mortality in men aged >65 years falling from 65.9 to 44.6 per 100,000 population.[15] Admissions for ruptured AAA fell from 18.6 to 13.5 per 100,000 in all age groups. The reduction in mortality and admissions has been attributed to the fall in smoking prevalence and the rise in elective AAA repair in older age groups.[15]

Aetiology

Traditionally, arterial aneurysms were thought to arise from atherosclerotic disease, and certainly intimal atherosclerosis reliably accompanies AAA.[16] Subsequent data suggest that altered tissue metalloproteinases may diminish the integrity of the arterial wall.[6]

Smoking remains the most important risk factor.[1] [9] [11] [17] [18]

A systematic review and meta-analysis found that diabetes protects against the growth and enlargement of AAA.[19] However, the protective mechanism is yet to be determined. Operative and long-term survival was lower among AAA repair patients with diabetes than those without, suggesting an increased cardiovascular burden.[19]

Pathophysiology

Histologically there is obliteration of collagen and elastin in the media and adventitia, smooth muscle cell loss with resulting tapering of the medial wall, infiltration of lymphocytes and macrophages, and neovascularisation.[16] There are 4 mechanisms relevant to AAA development:[20]

- Proteolytic degradation of aortic wall connective tissue: matrix metalloproteinases (MMPs) and other proteases are derived from macrophages and aortic smooth muscle cells and secreted into

the extracellular matrix. Disproportionate proteolytic enzyme activity in the aortic wall may promote deterioration of structural matrix proteins (e.g., elastin and collagen).[4] Increased expression of collagenases MMP-1 and -13 and elastases MMP-2, -9, and -12 has been demonstrated in human AAAs.[21] [22] [23] [24]

- Inflammation and immune responses: an extensive transmural infiltration by macrophages and lymphocytes is present on aneurysm histology and these cells may release a cascade of cytokines that subsequently activate many proteases.[16] Additionally, deposition of immunoglobulin G into the aortic wall supports the hypothesis that AAA formation may be an autoimmune response. There is currently interest in the role of reactive oxygen species and antioxidants in AAA formation.[21] [25] [26] [27] [28]
- Biomechanical wall stress: elastin levels and the elastin-collagen ratio decrease progressively distal down the aorta. Diminished elastin is associated with aortic dilation, and collagen degradation predisposes to rupture.[11] Additionally, data support increased MMP-9 expression and activity, disordered flow and an increase in wall tension, and relative tissue hypoxia in the distal aorta (i.e., infrarenal).[21] [29] [30]
- Molecular genetics: AAA exhibits significant heritability (the proportion of phenotype due to genotype). A Swedish Twin Registry study found that the twin of a monozygotic twin with AAA had a risk of AAA approximately 70 times greater than that of the twin of a monozygotic twin without AAA.[31] While there is no single genetic defect or polymorphism responsible, familial clustering suggests a polygenic aetiology. Genome-wide approaches have identified 10 risk loci for AAA, 4 of which are not shared with other cardiovascular diseases.[32]

Classification

Types of AAA

Specific types of AAA are:[5] [6]

- Congenital: while medial degeneration occurs naturally with age, it is accelerated in patients with bicuspid aortic valves and Marfan syndrome.
- Infectious: infection of the aortic wall (mycotic aneurysm) is a rare aetiology. *Staphylococcus* and *Salmonella* are the most common pathogens. *Chlamydia pneumoniae* has been postulated as an infectious aetiology for conventional aneurysms. Tertiary syphilis may manifest as aortic aneurysm, but this is an exceptionally rare presentation.[7]
- Inflammatory: the aetiology of inflammatory AAAs remains controversial. This variant is characterised by an abnormal accumulation of macrophages and cytokines in diseased tissue. Pathologically there is perianeurysmal fibrosis, thickened walls, and dense adhesions.

Screening

Screening 65-year-old men for AAA significantly reduces AAA-specific mortality.[71]

Current US Preventive Services Task Force (USPSTF) recommendations include:[3]

- One-time screening for AAA with ultrasonography in men aged 65 to 75 years who have ever smoked
- Selective screening (based on an assessment of risk factors, including older age and a first-degree relative with an AAA) for AAA in men aged 65 to 75 years who have never smoked.

The USPSTF concludes that there is currently insufficient evidence to assess the balance of benefits and harms of screening for AAA in women aged 65 to 75 years who have ever smoked.[3] In women who have never smoked, the USPSTF recommends against routine screening for AAA.

Routine screening for AAA among all men aged 65 and over has been available in the UK since 2013, and in England since 2009.[72] Aortic diameter at the screening appointment determines the subsequent management pathway:[73]

Aortic diameter (anterior-posterior, inner to inner)	Re-scanning interval
<3 cm	Discharged from programme
3-4.4 cm	Annual surveillance programme
4.5-5.4 cm	3-monthly surveillance programme
>5.5 cm	Referral to vascular surgeon*

Re-scanning intervals associated with aortic diameters
NHS population screening: care pathways, Public Health England

*European guidelines recommend review by a vascular surgeon within 2 weeks of an AAA >5.5 cm being detected, to prevent interval rupture.[74]

Meta-analysis of AAA surveillance studies suggest that longer surveillance intervals than those currently employed in the UK AAA screening programme could be safely implemented, thereby reducing cost.[75]

Case history

Case history #1

A 65-year-old man presents to his local aneurysm surveillance team for a screening ultrasound scan. He has been feeling well and in his usual state of good health. His medical history is notable for mild hypertension and he has a 100-pack-year tobacco history. On ultrasound an infrarenal AAA is identified.

Case history #2

A 55-year-old man with a history of hypertension (well controlled with medication) and tobacco use presents to his primary care physician with a 2-day history of constant and gnawing hypogastric pain. The pain has been steadily worsening in intensity. He says the pain radiates to his lower back and both groins at times. While he cannot identify any aggravating factors (such as movement), he feels the pain improves with his knees flexed. There is a palpable pulsatile mass just left of midline below the umbilicus. He is immediately referred for definitive management, but during transfer becomes hypotensive and unresponsive.

Other presentations

The triad of abdominal pain, weight loss, and elevated erythrocyte sedimentation rate suggests inflammatory AAA.[6] A tender, palpable pulsatile mass on examination and elevated C-reactive protein may also be present. Abdominal or back pain with fever is suggestive of mycotic or infectious AAA. Typically there is history of arterial trauma, intravenous drug abuse, local or concurrent infection, bacterial endocarditis, or impaired immunity. Osteomyelitis of the thoracic or lumbar spine may develop. Anaemia, leukocytosis, and positive blood cultures are common.[8] Diagnosis may be aided by complications of unruptured aneurysms, including distal embolisation, acute thrombosis, or symptoms caused by ureterohydronephrosis.[9]

Step-by-step diagnostic approach

Patients most commonly lack any symptoms and their aneurysm is noted on physical examination or imaging studies performed for other reasons.

History

In the minority of patients who experience symptoms, abdominal, back, and groin pain are typical. Medical history is directed towards risk factors:

- Development (i.e., hyperlipidaemia, connective tissue disorder, COPD, and hypertension)[1] [4] [9] [11] [17] [49] [50] [52]
- Expansion (i.e., previous cardiac or renal transplant, previous stroke, advanced age [>70 years], and severe cardiac disease)[53] [54]
- Rupture (i.e., female sex, previous cardiac or renal transplant, hypertension).[9] [45] [46] [51] [53] [55]

A history of cigarette smoking increases a patient's risk of AAA development, expansion, and rupture.[9] [17] [18] [33] [53] In men who have never smoked, important risk factors for AAA include older age and a first-degree relative with an AAA.[3]

A history of previous abdominal surgery or previous endovascular aortic aneurysm repair can be elicited as well as family history of AAA.

Physical examination

The abdomen can be palpated for a pulsatile abdominal mass and abdominal tenderness. Physical examination should include an assessment for peripheral artery aneurysm (femoral and popliteal).[56]

Aneurysm palpation on clinical examination has only been shown to be sensitive in thin patients and those with AAA >5 cm, with an overall sensitivity and specificity of 68% and 75%, respectively.[1] [57]

Ruptured aneurysm presents with the triad of abdominal and/or back pain, pulsatile abdominal mass, and hypotension.

The presence of fever may increase suspicion for infectious AAA in the appropriate clinical setting.

Key tests

Ultrasonography is the initial method of choice for AAA detection (sensitivity and specificity of 92% to 99% and nearly 100%, respectively).[1] [2] [57] [58] Once the diagnosis is made, further imaging with computed tomography angiography (CTA) or magnetic resonance angiography (MRA) is used for anatomical mapping to assist with operative planning (open or endovascular).[59]

Elevated erythrocyte sedimentation rate and C-reactive protein support a diagnosis of possible inflammatory AAA. Leukocytosis and a relative anaemia on full blood count with positive blood cultures are indicative of infectious AAA.

Predictors of rupture risk include AAA expansion rate, increase in intraluminal thrombus thickness, wall stiffness, wall tension, and peak AAA wall stress.[56] [60]

Risk factors

Strong

cigarette smoking

- This is the risk factor most strongly associated.[1] [9] [11] [17] [18]
- Active cigarette smoking is independently associated with histological high-grade tissue inflammation.[33]
- The duration of smoking is significantly associated with an increased risk in a linear dose-response relationship. Each year of smoking increases the relative risk by 4%.[18]

hereditary/family history

- Studies support a familial aggregation of and genetic predisposition to AAA.[1] [9] [34] [35] [36] [37] [38] [39] [40] [41]
- In a large population-based study, a positive first-degree family history for AAA was more common among cases than among controls (8.4% vs. 4.6%, P = 0.0001).[42] The risk of AAA associated with

family history was approximately doubled compared with no family history (odds ratio [OR] 1.9, 95% CI 1.6 to 2.2).

increased age

- Prevalence increases with age.[1] [11]
- Most frequently diagnosed in men >55 years of age, and rupture rarely occurs before 65 years of age.
- AAA is discovered approximately 10 years later in women.[9] [43] [44]

male sex (prevalence)

- AAAs are 4 to 6 times more prevalent in men than women.[1] [9] [11]

female sex (rupture)

- Risk of rupture is greater in women than in men.[45] [46]

congenital/connective tissue disorders

- Aortic degeneration is accelerated in patients with bicuspid aortic valves, Marfan syndrome, and during pregnancy.[47] [48] [49]
- Marfan syndrome specifically is associated with cystic medial necrosis of the aorta secondary to an autosomal dominant anomaly in fibrillin type 1, a structural protein that directs and orients elastin in the developing aorta.[47] [48] As a result, the mature aorta demonstrates abnormal elastic properties, progressive stiffening, and dilation.[49]

Weak

hyperlipidaemia

- Lipoproteins are elevated in patients with AAA independent of cardiovascular risk factors and extent of atherosclerosis.[4] [9]
- AAA patients have significantly lower levels of apolipoprotein AI and HDL cholesterol than matched controls with aorto-iliac occlusive disease.[1] [4]
- High serum total cholesterol is a relatively weak risk factor for AAA, whereas high HDL cholesterol was strongly associated with a low risk of AAA.[11] [44]

COPD

- This is attributed to tobacco-induced elastin degradation.[4] [9]
- Studies suggest that the association between reduced respiratory function and AAA may be due to the activation of inflammation and haemostasis in response to injury.[50]

atherosclerosis (i.e., coronary artery disease [CAD], peripheral arterial occlusive disease)

- CAD is an independent associated risk factor.[1] [17]

hypertension

- Hypertension is a weak independent risk factor.[1] [9] [11]
- There is a relation between systolic BP and AAA in women and an association with ever-use antihypertensive medication and AAA risk for both sexes.[11] [44]

increased height

- Increased height is an independent associated risk factor, although after adjustment for age and sex the association was no longer significant.[1] [17] [51]

central obesity

- One study of more than 12,000 men demonstrated an independent association between central obesity and AAA.[44]

non-diabetic

- A systematic review and meta-analysis found that diabetes protects against the growth and enlargement of AAA.[19] However, the protective mechanism is yet to be determined. Operative and long-term survival was lower among AAA repair patients with diabetes than those without, suggesting an increased cardiovascular burden.[19]

History & examination factors

Key diagnostic factors

presence of risk factors (common)

- Key risk factors include cigarette smoking, family history, increased age, male sex for prevalence and female sex for rupture, and congenital/connective tissue disorders.

palpable pulsatile abdominal mass (uncommon)

- Aneurysm palpation on clinical examination has been shown to be sensitive only in thin patients and those with AAA >5 cm (sensitivity and specificity of 68% and 75%, respectively).[1] [57]

Other diagnostic factors

abdominal, back, or groin pain (uncommon)

- Patients are usually asymptomatic and their aneurysm is detected incidentally.

hypotension (uncommon)

- Patients with ruptured aneurysm present with the triad of abdominal and/or back pain, pulsatile abdominal mass, and hypotension.

Diagnostic tests

1st test to order

Test	Result
abdominal ultrasound • Definitive test (sensitivity and specificity of 92% to 99% and nearly 100%, respectively).[1] [2] [57] [58] • The ultrasound is performed perpendicular to the aortic axis as oblique views may overestimate the true aortic diameter.[2] • Intra-observer correlation may be better near the aortic bifurcation than in the proximal infrarenal aorta.[61] • Unfortunately, ultrasound offers little utility in imaging aneurysms close to the origins of, or proximal to, the renal arteries.[62] [63]	**abdominal aortic dilation of >1.5 times the expected anterior-posterior diameter of that segment, given the patient's sex and body size; the most commonly adopted threshold is a diameter of 3 cm or more 24957320 LeFevre ML; US Preventive Services Task Force. Screening for abdominal aortic aneurysm: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med. 2014 Aug 19;161(4):281-90. http://annals.org/aim/fullarticle/1883339/screening-abdominal-aortic-aneurysm-u-s-preventive-services-task-force**

Other tests to consider

Test	Result
ESR/CRP • Suggests inflammatory AAA.	**elevated**
FBC • Leukocytosis and a relative anaemia on FBC with positive blood cultures are indicative of infectious AAA.	**leukocytosis, anaemia**
blood cultures • Leukocytosis and a relative anaemia on FBC with positive blood cultures are indicative of infectious AAA.	**positive**

Test	Result
computed tomography angiography (CTA)/CT • May demonstrate retroperitoneal haematoma, discontinuity of the aortic wall, or extravasation of contrast into the peritoneal cavity, which are all signs of rupture.[9] [64] • Also useful in diagnosing aortic aneurysms close to the origins of, or proximal to, the renal arteries.[63] [64] • The first choice for intraoperative planning; 0.5 mm slices are desirable and 3D reconstruction is essential for accurate planning.	**abdominal aortic dilation of >1.5 times the expected anterior-posterior diameter of that segment, given the patient's sex and body size; the most commonly adopted threshold is a diameter of 3 cm or more 24957320 LeFevre ML; US Preventive Services Task Force. Screening for abdominal aortic aneurysm: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med. 2014 Aug 19;161(4):281-90. http://annals.org/aim/fullarticle/1883339/screening-abdominal-aortic-aneurysm-u-s-preventive-services-task-force**
magnetic resonance angiography (MRA)/MRI • Preoperative study of choice for operative strategy if a patient has an iodinated contrast allergy.	**abdominal aortic dilation of >1.5 times the expected anterior-posterior diameter of that segment, given the patient's sex and body size; the most commonly adopted threshold is a diameter of 3 cm or more 24957320 LeFevre ML; US Preventive Services Task Force. Screening for abdominal aortic aneurysm: U.S. Preventive Services Task Force recommendation statement. Ann Intern Med. 2014 Aug 19;161(4):281-90. http://annals.org/aim/fullarticle/1883339/screening-abdominal-aortic-aneurysm-u-s-preventive-services-task-force**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Diverticulitis	• Obstipation; abdominal pain is more common and typically localises to the left lower quadrant. • No pulsatile abdominal mass on clinical examination. Instead, abdominal or perirectal 'fullness' may be appreciated. Fever is possible.[65]	• Stool guaiac testing may be trace positive. • Leukocytosis may be present. • CT scan will demonstrate a normal-calibre aorta and possibly diverticula, inflammation of the pericolic fat or other tissues, bowel-wall thickness >4 mm, or a peridiverticular abscess.[65]
Ureteric colic	• Severe abdominal pain that starts in the flank and radiates anteriorly to the groin. • Associated with nausea, emesis, haematuria, dysuria, and urinary frequency or urgency.[66] • Men >55 years of age presenting with ureteric colic should be considered to have a leaking/ruptured AAA until proven otherwise.	• Urinalysis positive for blood and may demonstrate crystals and/or evidence of infection. • Ultrasound and CT scan will demonstrate a normal-calibre aorta and possibly ureteral or renal stones.[66]
Irritable bowel syndrome (IBS)	• Intermittent abdominal discomfort with flares lasting 2 to 4 days. • Associated symptoms may include bloating, stool frequency, and abnormal defecation. • Women aged 20 to 40 years are affected more often than men. • General examination is usually normal, although some patients may appear anxious. There may be poorly localised abdominal tenderness to palpation.[67]	• Imaging modalities are often inconclusive, but will demonstrate a normal-calibre aorta.

Condition	Differentiating signs / symptoms	Differentiating tests
Inflammatory bowel disease	• Abdominal pain is often 'crampy' and left-sided. Patients typically suffer from diarrhoea (bloody and non-bloody), urgency of defecation, and tenesmus. • Extra-intestinal manifestations are common in Crohn's disease. • Abdominal examination may demonstrate abnormal bowel sounds, detection of an abdominal mass, and pain on palpation. Mucocutaneous lesions may be visible. Perianal fistulae, fissures, or abscesses may be present on rectal examination.[68]	• Anaemia is common. • Ultrasound or CT scan will demonstrate a normal-calibre aorta. • Endoscopic evaluation with biopsy shows typical lesions of ulcerative colitis or Crohn's disease.[68]
Appendicitis	• Pain is typically periumbilical with localisation to the right lower quadrant. • Associated nausea, emesis, and anorexia are common. • Patients are classically febrile with tenderness in the right lower quadrant or rebound tenderness on abdominal examination.	• Leukocytosis and sterile pyuria on urinalysis is common. • Ultrasound or CT scan will demonstrate a normal-calibre aorta with an inflamed appendix or evidence of perforation.
Ovarian torsion	• Women suffer sudden, continuous, non-specific pain in the lower abdomen; nausea and emesis are common. Patients may demonstrate fever on clinical examination, and an adnexal mass may be palpable.[69]	• Leukocytosis may be present. Ultrasound will demonstrate a normal-calibre aorta and possibly reduced or absent adnexal vascular flow.[69]
Gastrointestinal (GI) haemorrhage	• Patients presenting with haemorrhagic shock may mimic aortic rupture. A history of previous GI bleed, haematemesis, melaena, or bright red blood per rectum is common. • Historical risk factors for GI malignancy or peptic ulcer disease may be elicited. • On rectal examination gross blood may be visible, or coffee ground haematemesis may be returned with nasogastric tube placement.	• Stool is likely to be guaiac positive. • Endoscopic evaluation may demonstrate the luminal bleeding source along with mucosal ulcerations, polyps, or tumour. • Ultrasound or CT scan will demonstrate a normal-calibre aorta.

Condition	Differentiating signs / symptoms	Differentiating tests
Splanchnic artery aneurysms/acute occlusion	• Acute embolic or thrombotic occlusion of the splanchnic vessels results in a marked disparity between acute excruciating mid-abdominal pain and a paucity of early physical findings. • Patients typically suffer unremitting, intense mid-abdominal pain with nausea and vomiting that might be accompanied by explosive diarrhoea. • Most splanchnic artery aneurysms are asymptomatic until rupture.[70]	• Leukocytosis, haemoconcentration, and systemic acidosis are common with acute splanchnic vessel occlusion. Elevated levels of serum amylase, inorganic phosphorus, creatinine phosphokinase, and alkaline phosphatase may accompany frank bowel infarction. • Angiography is diagnostic and potentially therapeutic in the case of vascular occlusion. • Ultrasound and CT scan will demonstrate a normal-calibre aorta and will diagnose any splanchnic artery aneurysms.[70]

Step-by-step treatment approach

Patients presenting with a ruptured aneurysm require urgent repair.

For patients with symptomatic aortic aneurysms, repair is indicated regardless of diameter.[76]

For asymptomatic AAA detected as an incidental finding, surveillance is preferred to repair until the theoretical risk of rupture exceeds the estimated risk of operative mortality. Generally, repair is indicated in patients with large asymptomatic AAA (e.g., with a diameter exceeding 5.5 cm in men or 5.0 cm in women in the US, although treatment decisions based on greater size may differ in other countries, e.g., UK).[1] [77] [78] [79] [80] [81]

Repair of asymptomatic, symptomatic, and ruptured aneurysms can be accomplished using either an endovascular or open surgical technique; the selection of surgical technique should take account of patient preference, patient age, sex, perioperative risk factors, and anatomical factors.

Ruptured AAA

Patients with the triad of abdominal and/or back pain, pulsatile abdominal mass, and hypotension warrant immediate resuscitation and surgical evaluation as repair offers the only potential cure.[56] [82] However, most patients with rupture will not survive to reach theatre.
[Fig-3]

In patients with confirmed ruptured AAA, 3-year mortality was lower among those randomised to endovascular aneurysm repair (EVAR) than to an open repair strategy (48% vs. 56%; hazard ratio [HR] 0.57, 95% CI 0.36 to 0.90).[83] The difference between treatment groups was no longer evident after 7 years of follow-up (HR 0.92, 95% CI 0.75 to 1.13). Re-intervention rates were not significantly different between the randomised groups at 3 years (HR 1.02, 95% CI 0.79 to 1.32).[83] There is some evidence to suggest that an endovascular strategy for repair of ruptured AAA may reduce mortality more effectively in women than in men.[83] [84] [85]

Supportive treatment of ruptured AAA

Standard resuscitation measures are initiated immediately. These include:

- Airway management (supplemental oxygen or endotracheal intubation and assisted ventilation if the patient is unconscious).

[VIDEO: Tracheal intubation animated demonstration]

[VIDEO: Bag-valve-mask ventilation animated demonstration]

- Intravenous access (central venous catheter).
- Arterial catheter; urinary catheter.
- Hypotensive resuscitation: aggressive fluid replacement may cause dilutional and hypothermic coagulopathy and secondary clot disruption from increased blood flow, increased perfusion pressure, and decreased blood viscosity, thereby exacerbating bleeding.[89] [90] [91] [92] Infusing more than 3.5 litres of fluid preoperatively may increase the relative risk of death.[89] A target systolic BP of 50 to 70 mmHg and withholding fluids is advocated preoperatively.[90] [91] [92]
- Blood product (packed red cells, platelets, and fresh frozen plasma) availability and transfusion for resuscitation, severe anaemia, and coagulopathy.

- Notifying anaesthetic, ICU, and operating teams.

Data from one multi-centre randomised clinical trial support the use of CT scans in cases of ruptured AAA.[93] It did not result in significant delays, and allowed appropriate preoperative planning.

Symptomatic but not ruptured AAA

In patients with symptomatic aortic aneurysm, repair is indicated regardless of diameter.[76] [94] The development of new or worsening pain may herald aneurysm expansion and impending rupture. Symptomatic, non-ruptured aneurysm is, therefore, best treated urgently.[56] Under some circumstances, intervention may be delayed for several hours to optimise conditions to ensure successful repair; these patients should be closely monitored in the ICU.[56]

EVAR is increasingly used in the management of patients with symptomatic AAA.[95] [96] In observational studies, short-term all-cause mortality rates did not differ between endovascular and open repair of symptomatic AAA.[95] [96] [97]

Data from the 2011-2013 American College of Surgeons National Surgical Quality Improvement Program suggest that 30-day mortality among patients with symptomatic AAA was approximately double that of patients with asymptomatic AAA following EVAR (symptomatic 3.8% vs. asymptomatic 1.4%, P = 0.001) or open repair (symptomatic 7.7% vs. asymptomatic 4.3%, P = 0.08).[98] Smaller patient numbers probably contribute to the non-statistically significant finding reported for open repair.

Incidental finding of small asymptomatic AAA

For AAA detected as an incidental finding, surveillance is preferred to repair until the theoretical risk of rupture exceeds the estimated risk of operative mortality.[3] [94] Early surgery for the treatment of smaller AAAs does not reduce all-cause or AAA-specific mortality.[3] [99] One systematic review (4 trials, 3314 participants) found high-quality evidence to demonstrate that immediate repair of small AAA (4 cm to 5.5 cm) did not improve long-term survival compared with surveillance (adjusted HR 0.88, 95% CI 0.75 to 1.02, mean follow-up 10 years).[99] The lack of benefit attributable to immediate surgery was consistent regardless of patient age, diameter of small aneurysm, and whether repair was endovascular or open.[99]

Surgical referral of smaller AAA is usually reserved for rapid growth, or once the threshold diameter for aneurysm repair is reached on repeated ultrasonography.[3]

Medical goals for asymptomatic small aneurysms include:

1. Surveillance:

- American College of Cardiology Foundation/American Heart Association guidelines recommend that infra-/juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter by ultrasound/CT should be monitored every 6 to 12 months.[76] AAAs <4.0 cm require ultrasonography every 2 to 3 years.[76]
- The UK National Health Service recommends that annual screening intervals are employed for 3.0 to 4.4 cm AAAs and 3-month intervals for 4.5 to 5.4 cm AAAs.[100]
- One systematic review and meta-analysis of individual patient data concluded that surveillance intervals of 2 years for 3.0 to 4.4 cm AAA, and 6 months for 4.5 to 5.4 cm AAA, are safe and cost-effective.[75]
- Analysis of AAA growth and rupture rates indicated that in order to maintain an AAA rupture risk <1%, an 8.5-year surveillance interval is required for men with baseline AAA diameter of 3.0 cm.[75] The corresponding estimated surveillance interval for men with an initial aneurysm

diameter of 5.0 cm was 17 months. Despite having similar small aneurysm growth rates, rupture rates were 4 times higher in women than in men.[75] Surveillance programmes and criteria for considering surgery need to be tailored for women with opportunistically detected AAA.

2. Control of modifiable risk factors for expansion and rupture:

- Smoking cessation - nicotine-replacement therapy, nortriptyline, and bupropion, or counselling.[1] [9] [11] [17] [18] [101] [102] [103]
- Short-term treatment with beta-blockers does not appear to reduce the rate of AAA expansion.[3] Trials in which patients with small AAAs were randomised to propranolol with the intention of reducing the rate of aneurysm expansion failed to demonstrate significant protective effects.[104] Propranolol was poorly tolerated in these studies.[104]

3. Aggressive management of other cardiovascular disease:

- Modifiable cardiovascular risk factors such as hypertension and hyperlipidaemia should be treated. Statins should be started at least 1 month before surgery to reduce cardiovascular morbidity, and continued indefinitely.[74]

Incidental finding of large asymptomatic AAA

Generally, repair is indicated in patients with large asymptomatic AAA (e.g., with a diameter exceeding 5.5 cm in men or 5.0 cm in women in the US, although treatment decisions based on greater size may differ in other countries, e.g., UK). Repair of aneurysms ≥5.5 cm offers a survival advantage.[1] [78] [79] [80] [81]

Decisions regarding repair should be individualised, taking account of patient preference, patient age, sex, perioperative risk factors, and anatomical risk factors. Care should be taken to evaluate patient quality of life, and careful counselling undertaken regarding the risks of surgery (e.g., informing patients of their Vascular Quality Initiative perioperative mortality risk score) and subsequent quality of life. EVAR should be considered in patients who are unfit for open surgery.[56] [77] [78] [79] [80] [99] 1[B]Evidence

Data suggest that in patients with large AAAs (ranging from 5 to 5.5 cm) undergoing elective repair, EVAR is equivalent to open repair in terms of overall survival, although the rate of secondary interventions is higher for EVAR.[108] EVAR reduces AAA-related mortality (but not longer-term overall survival) in patients with large AAA (≥5.5 cm) who are unsuitable for open repair.[109] 2[A]Evidence Post-repair, larger AAAs appear to be associated with worse late survival than smaller aneurysms (pooled HR 1.14 per 1-cm increase in AAA diameter, 95% CI 1.09 to 1.18; 12- to 91.2-month follow-up).[111] The association is more pronounced with EVAR than with open repair.

Elective repair in asymptomatic patients allows for preoperative assessment, cardiac risk stratification, and medical optimisation of other comorbidities. Coronary artery disease remains the leading cause of early and late mortality after AAA repair.

Endovascular aneurysm repair (EVAR)

EVAR involves the transfemoral endoluminal delivery of a covered stent graft into the aorta, thus sealing off the aneurysm wall from systemic pressures, preventing rupture, and allowing for sac shrinkage. The endograft can be deployed percutaneously through low-profile devices, or after exposing the femoral arteries surgically. A Cochrane review found no difference between the techniques after short follow-up (6

months), except that the percutaneous approach may be faster.[112] Long-term data for the durability of low-profile devices are lacking.
[Fig-4]

[Fig-5]

Assessment of suitability for EVAR should be through the use of 0.5 mm-slice CT angiography. It is essential that the operator is familiar with the specific instructions for use of the endograft to be used.

Lifelong, annual surveillance with ultrasonography or CT is recommended following EVAR of AAAs.[56] [74]

Open repair

Open repair may be transperitoneal or retroperitoneal. With proximal and distal aortic control obtained, the aneurysm is opened, back-bleeding branch arteries are ligated, and a prosthetic graft is sutured from normal proximal aorta to normal distal aorta (or iliac segments). Once flow is restored to the bilateral iliac arteries, the aneurysm sac is closed over the graft.[113] A retroperitoneal approach should be considered for patients in which aneurysmal disease extends to the juxtarenal and/or visceral aortic segment, or in the presence of an inflammatory aneurysm, horseshoe kidney, or hostile abdomen.[56] [114] [115]

Straight tube grafts are recommended for repair in the absence of significant disease of the iliac arteries.[56] The proximal aortic anastomosis should be performed as close to the renal arteries as possible.[56] It is recommended that all portions of an aortic graft should be excluded from direct contact with the intestinal contents of the peritoneal cavity.[56] Re-implantation of a patent inferior mesenteric artery (IMA) should be considered under circumstances that suggest an increased risk of colonic ischaemia (i.e., associated coeliac or superior mesenteric artery occlusive disease, an enlarged meandering mesenteric artery, a history of prior colon resection, inability to preserve hypogastric perfusion, substantial blood loss or intraoperative hypotension, poor IMA back-bleeding when graft open, poor Doppler flow in colonic vessels, or should the colon appear ischaemic).[56] [116]

Choice of elective repair

EVAR accounts for more than 70% of all AAA repairs in the US.[117] In the UK, 69% of elective infrarenal AAAs and 89% of complex AAAs were treated with EVAR during 2014-15.[118] However, not all patients are suitable candidates for EVAR. Guidelines therefore recommend an individualised approach to surgical choice.[56] [74] [76] [119] [120] Factors that will influence the decision include: anatomical determinants (e.g., aneurysm diameter, neck length, neck diameter); life expectancy, sex, comorbidities; and perioperative risk.

EVAR may be preferred in patients who:

- Have high perioperative risk, and
- Have anatomy that is congruent with the relevant stent-graft manufacturer's eligibility criteria as determined in the instructions for use, and
- Are able to satisfy the mandatory surveillance regimen following surgery.

Patients with lower perioperative risk and favourable anatomy may also be candidates for EVAR.[121] [122] Consideration should be given to safety and durability of repair (need for re-intervention).

Open repair may be preferred in patients who:

- Have lower perioperative risk, and
- Are relatively younger.

American College of Cardiology/American Heart Association Practice Guidelines (compilation of 2005 and 2011 guideline recommendations) recommend open aneurysm repair for patients who cannot comply with the long-term surveillance required after EVAR.[76] Open repair is also indicated in patients who may otherwise be considered for EVAR, but who have unfavourable anatomy.[123]

Elective repair outcomes

Data regarding the comparative safety and efficacy of EVAR and open repair differ depending on the outcome of interest. Evidence to date suggests that:

- Short-term postoperative mortality (≤30 days) is lower for endovascular than open repair
- Operative, perioperative, and postoperative mortality (≤30 days) is greater among women than men for both open repair and EVAR
- Aneurysm-related mortality (≥3 years post repair) is higher after EVAR than open repair
- Overall rates of re-intervention are more common following EVAR than open repair.

Six-month postoperative mortality appears to be lower among patients with AAA who undergo EVAR than those who have open surgery, but this is primarily attributable to lower 30-day operative mortality.[124] Pooled analysis of data from 4 high-quality randomised trials (that enrolled patients with AAA diameter >5 cm) found that short-term mortality (30-day or in-hospital mortality) was significantly lower among those randomised to EVAR than among those who underwent open repair (1.4% vs. 4.2%, odds ratio [OR] 0.33, 95% CI 0.20 to 0.55; P <0.0001).[125] However, the early benefit of EVAR is diminished with follow-up.[124] [125] Longer-term mortality (>4 years) did not differ between patients randomised to EVAR or to open surgery (37.3% vs. 37.8%, OR 0.98, 95% CI 0.83 to 1.15; P = 0.78). Open repair was significantly associated with lower all-cause mortality than EVAR, after more than 8 years of follow-up, in the UK endovascular aneurysm repair trial 1 (46% vs. 53%, P = 0.048).[126]

Perioperative and short-term mortality is higher among women than among men. During elective AAA repair, operative mortality among women exceeds that of men for both open (7.0% vs. 5.2%) and endovascular approaches (2.1% vs. 1.3%).[127] In the UK, women undergoing elective AAA repair were found to have increased short-term mortality compared with men for open repair (30-day mortality: OR 1.39; 95% CI 1.25 to 1.56) and EVAR (30-day mortality: OR 1.57; 95% CI 1.23 to 2.00), despite having fewer preoperative cardiovascular risk factors.[128] Female sex was an independent risk factor for all-cause mortality among women who had an open repair at 1 year (crude cumulative all-cause mortality 15.9% vs. 12.1%, P <0.001) and at 5 years (22.2% vs. 19.6%, P <0.001).[128] Long-term all-cause survival did not differ significantly between women and men in the EVAR group (P = 0.356).

One meta-analysis of individual-patient data from 4 trials found that, after 3 years, aneurysm-related mortality was significantly higher in the EVAR group than in those with open repair (pooled hazard ratio [HR] 5.16, 95% CI 1.49 to 17.89; P = 0.010).[124] Data from the UK endovascular aneurysm repair trial 1 indicate that, after >8 years of follow-up, aneurysm-related mortality is greater among EVAR patients than open repair patients (5% vs. 1%, P = 0.0064).[126] Aneurysm rupture was more common in patients after EVAR than open repair (5.4% vs. 1.4%, P <0.001) in a large cohort study with 8 years of follow-up.[129] Meta-analysis found no significant difference between EVAR and open surgery in the incidence of myocardial death (OR 1.14, 95% CI 0.86 to 1.52; P = 0.36), fatal stroke (OR 0.81, 95% CI 0.42 to 1.55; P = 0.52), or non-fatal stroke (OR 0.81, 95% CI 0.50 to 1.31; P = 0.39).[125] Patients with moderate renal dysfunction or cardiovascular disease do not appear to derive an early survival benefit (to 6 months) from

EVAR, while those with peripheral arterial disease may benefit from open repair.[124] In another meta-analysis, long-term survival following elective AAA repair (EVAR or open) was worst among patients with end-stage renal disease (HR 3.15, 95% CI 2.45 to 4.04) and COPD requiring supplementary oxygen (HR 3.05, 95% CI 1.93 to 4.80).[130]

Overall rates of re-intervention were higher with EVAR than with open surgery; however, rates have been reported heterogeneously in clinical trials.[124] [125] In pooled analysis of individual-patient data, re-intervention was reported in 65.8% of EVAR patients with type I endoleak (79 of 120) and 22.8% of EVAR patients with type II endoleak (99 of 435) over 5 years of follow-up.[124] Observational data suggest that interventions related to the management of aneurysm or its complications are more common following EVAR than open surgery (18.8% vs. 3.7%, P <0.001) over 8 years of follow-up.[129]

Low-quality evidence from 4 small RCTs suggests that elective open repair performed retroperitoneally can reduce blood loss and hospital stay compared with a transperitoneal approach.[131] However, there was no difference in mortality between retroperitoneal and transperitoneal elective open AAA repair (very low-quality evidence). Moreover, the retroperitoneal approach may increase the risk of haematoma, chronic wound pain, and abdominal wall hernia compared with transperitoneal.[131]

Adverse effects

Complications of EVAR may include endoleak, graft occlusion, and graft migration with aortic neck expansion.[119] A systematic review reported aortic neck dilation in 24.6% of EVARs (9439 men included), which led to higher rates of type I endoleak, graft migration, and re-intervention.[132]

As an adjunct to EVAR, bilateral hypogastric artery occlusion may be acceptable in certain anatomical situations for patients at high risk for open surgical repair. Buttock claudication and erectile dysfunction may occur in up to 40% of patients after unilateral embolisation; these symptoms may persist in 11% to 13% of patients following bilateral occlusion.[56] [133] [134] Internal iliac artery revascularisation techniques, involving specialised iliac branch devices, have high technical success rates and are associated with low morbidity (e.g., buttock claudication rate of 4.1%).[135]

In patients with synchronous intra-abdominal malignancy, EVAR reduces mortality and delay between the treatment of the 2 pathologies despite a significant risk for thrombotic events.[136] [137]

Complications of open repair include cardiac and pulmonary events, mesenteric ischaemia, renal failure, bleeding, wound and graft infection, spinal cord ischaemia/paraplegia, embolisation/limb ischaemia, and late graft complications (i.e., aorto-enteric fistula and aortic pseudoaneurysm).[1] [138]

Perioperative management

Treatment of co-existing cardiac disease:

- Cardiovascular risk prevention should be prioritised in patients in AAA screening programmes.[139]
- Non-invasive stress testing should be considered for patients with a history of ≥3 clinical risk factors (i.e., coronary artery disease, congestive heart failure, stroke, diabetes mellitus, chronic renal insufficiency) and an unknown or poor functional capacity (MET <4) if it will change management.
- Coronary revascularisation is indicated for those patients who present with acute ST elevation myocardial infarction, unstable angina, or stable angina with left main coronary artery or 3-vessel disease, as well as those patients with 2-vessel disease that includes the proximal left anterior descending artery and either ischaemia on non-invasive testing or an ejection fraction <0.5.[56]

Regarding blood transfusion:[56]

- Preoperative autologous blood donation may be beneficial for patients undergoing open aneurysm repair.
- Cell salvage or an ultrafiltration device is recommended if large blood loss is anticipated or the risk of disease transmission from banked blood is considered high.
- Blood transfusion is recommended if the intraoperative haemoglobin level is <10 g/dL in the presence of ongoing blood loss. Consider use of fresh frozen plasma and platelets in a ratio with packed blood cells of 1:1:1.

Pulmonary artery catheters should not be used routinely in aortic surgery, unless there is a high risk for a major haemodynamic disturbance.[56] Central venous access is recommended for all patients undergoing open aneurysm repair.[56] DVT prophylaxis consisting of intermittent pneumatic compression and early ambulation are recommended for all patients undergoing open repair or EVAR.[56] [140]

Avoiding hypothermia during open repair and EVAR can reduce hospital length of stay, length of stay in the ITU, and rates of organ dysfunction.[141]

Preoperative cardiovascular risk reduction:

- Addressing modifiable cardiovascular risk factors preoperatively improves long-term survival after AAA repair.[142]
- Preoperative exercise training reduced post-surgical cardiac complications in a small randomised controlled trial of patients undergoing open or endovascular AAA repair.[143]
- Perioperative statin use reduces cardiovascular events during non-cardiac surgery.[144] Statins should be started at least 1 month before surgery to reduce cardiovascular mortality, and continued indefinitely.[74]
- In the absence of contraindications, patients should receive low-dose aspirin and this should be continued during the perioperative period.[74]
- Hypertension should be controlled to reduce cardiovascular morbidity.[74]
- Preoperative beta-blockade may be reasonable in patients at high risk of myocardial ischaemia (ischaemic heart disease or myocardial ischaemia present on stress testing), if the therapy can be commenced more than 1 month before surgery.[74] [145] Large trials where beta-blockade was started a few days before surgery have indicated no benefit, or even harm, from perioperative beta-blockade.[146] [147] [148]
- A large, multi-centre study of patients undergoing non-cardiac surgery found that clonidine did not reduce the rate of death or non-fatal myocardial infarction.[149] Alpha-2 agonists are not, therefore, recommended for non-cardiac surgery patients.[144]

Antibiotic cover:

- Antibiotic therapy is indicated for patients undergoing elective and urgent repair of ruptured AAA to cover gram-positive and gram-negative organisms (i.e., *Staphylococcus aureus* , *Staphylococcus epidermidis* , and enteric gram-negative bacilli) and prevent graft infection.
- Broad-spectrum antibiotic coverage is tailored to patient clinical presentation and cultures, and in accordance with local protocols.

Endovascular aneurysm repair (EVAR) leak

Endoleak is persistent blood flow outside the graft and within the aneurysm sac.[150] [151] It is not a complication following open repair.

Postoperative surveillance can detect major endoleaks and aneurysm sac expansion.

Risk of endoleak following EVAR is 24%.[150] There are 5 types of endoleak.

Type I:

- Leak at the attachment site (proximal/distal end of the endograft or iliac occluder); usually immediate, but delayed leaks may occur.
  [Fig-6]
- Every effort should be made to repair type I endoleak before completing the procedure (e.g., balloon moulding of the proximal seal zone, placement of a proximal cuff, endostaples). Persistent type IA endoleak may necessitate conversion to open repair.[56]
  [Fig-7]

  [Fig-8]

Type II:

- Patent branch leak.
  [Fig-9]
- Spontaneous resolution may occur, although persistence may result in sac growth.[152]
- If a type II endoleak or other abnormality of concern is observed on contrast-enhanced CT imaging at 1 month after EVAR, postoperative imaging at 6 months is recommended.[56]
- Treatment remains controversial and is advocated either if persistent at 6 to 12 months or when aneurysm sac size increases such that proximal and/or distal sealing zones may be compromised.[153] [154] [155] [156]
- Treatment of choice is transarterial coil embolisation, although laparoscopic ligation of collateral branches, direct percutaneous translumbar puncture of the sac, translumbar embolisation, and transcatheter transcaval embolisation have been reported.[151] [153] [154] [155] [157] [158] [159] [160]

Type III:

- Graft defect with leak through fabric tears, graft disconnection, or disintegration of the fabric.[150] [151]
- Repair is indicated upon discovery (endovascular stent graft extension).[56] [154]

Type IV:

- Leak from graft wall porosity.[150] [151]
- These leaks are uncommon with newer stent grafts and are self-limiting.[56] [154]

Type V (endotension):

- Endotension is increased intrasac pressure after EVAR without visualised endoleak on delayed contrast CT scans.

- Endotension is less common with the newer-generation grafts.[56]
- There is no standardised method to measure endotension or consensus on indicated therapy in the absence of aneurysm enlargement; however, treatment of endotension to prevent aneurysm rupture is suggested in selected patients with continued aneurysm expansion.[56] [151]

[VIDEO: Bag-valve-mask ventilation animated demonstration]

[VIDEO: Central venous catheter insertion animated demonstration]

[VIDEO: Female urethral catheterisation animated demonstration]

[VIDEO: Male urethral catheterisation animated demonstration]

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Acute (summary)

Patient group	Tx line	Treatment
ruptured AAA	1st	standard resuscitation measures
	plus	urgent surgical repair
	plus	perioperative antibiotic therapy
symptomatic, but not ruptured AAA	1st	urgent surgical repair
	plus	preoperative cardiovascular risk reduction
	plus	perioperative antibiotic therapy

Ongoing (summary)

Patient group	Tx line	Treatment
incidental finding: small asymptomatic AAA	1st	surveillance
	plus	aggressive cardiovascular risk management
incidental finding: large asymptomatic AAA	1st	elective surgical repair
	plus	preoperative cardiovascular risk reduction

Ongoing		(summary)
	plus	perioperative antibiotic therapy
endovascular repair leak requiring treatment	1st	corrective procedure
	plus	preoperative cardiovascular risk reduction
	plus	perioperative antibiotic therapy

Treatment options

Acute

Patient group	Tx line	Treatment
ruptured AAA	**1st**	**standard resuscitation measures**

» The airway is managed with supplemental oxygen or endotracheal intubation and assisted ventilation if the patient is unconscious.

[VIDEO: Tracheal intubation animated demonstration]

[VIDEO: Bag-valve-mask ventilation animated demonstration]

» A central venous catheter is inserted.

» Monitoring requires insertion of an arterial catheter and urinary catheter.

» A target systolic BP of 50 to 70 mmHg and withholding fluids is advocated preoperatively.[90] [91] [92]

» Aggressive fluid replacement may cause dilutional and hypothermic coagulopathy and secondary clot disruption from increased blood flow, increased perfusion pressure, and decreased blood viscosity, thereby exacerbating bleeding.[89] [90] [91] [92] Infusing more than 3.5 L of fluid preoperatively may increase the relative risk of death.[89]

plus **urgent surgical repair**

» Aorto-iliac anatomy permitting, endovascular aneurysm repair (EVAR) is preferred.[83]

» In patients with confirmed ruptured AAA, 3-year mortality was lower among those randomised to EVAR than to an open repair strategy (48% vs. 56%; hazard ratio [HR] 0.57, 95% CI 0.36 to 0.90).[83] The difference between treatment groups was no longer evident after 7 years of follow-up (HR 0.92, 95% CI 0.75 to 1.13). Re-intervention rates were not significantly different between the randomised groups at 3 years (HR 1.02, 95% CI 0.79 to 1.32).[83]

» There is some evidence to suggest that an endovascular strategy for repair of ruptured AAA

Acute

Patient group	Tx line	Treatment
		may reduce mortality more effectively in women than in men.[83] [84] [85]
	plus	**perioperative antibiotic therapy**
		» Antibiotic therapy is indicated for patients undergoing emergency repair of ruptured AAA to cover gram-positive and gram-negative organisms and prevent graft infection.
		» Broad-spectrum antibiotic coverage is tailored to patient clinical presentation and cultures, and in accordance with local protocols.
symptomatic, but not ruptured AAA	1st	**urgent surgical repair**
		» In patients with symptomatic aortic aneurysm, repair is indicated regardless of diameter.[76] [94] The development of new or worsening pain may herald aneurysm expansion and impending rupture. Symptomatic, non-ruptured aneurysm is, therefore, best treated urgently.[56] Under some circumstances, intervention may be delayed for several hours to optimise conditions to ensure successful repair; these patients should be closely monitored in the ICU.[56]
		» Endovascular aneurysm repair (EVAR) is increasingly used in the management of patients with symptomatic AAA.[95] [96] In observational studies, short-term all-cause mortality rates did not differ between endovascular and open repair of symptomatic AAA.[95] [96] [97]
	plus	**preoperative cardiovascular risk reduction**
		» In the absence of contraindications, patients should receive low-dose aspirin and this should be continued during the perioperative period.[74]
		» Hypertension should be controlled to reduce cardiovascular mortality.[74]
	plus	**perioperative antibiotic therapy**
		» Perioperative antibiotic therapy is given. Broad-spectrum antibiotic coverage is necessary, in accordance with local protocols.

Ongoing

Patient group	Tx line	Treatment
incidental finding: small asymptomatic AAA	**1st**	**surveillance** » For AAA detected as an incidental finding, surveillance is preferred to repair until the theoretical risk of rupture exceeds the estimated risk of operative mortality.[3] [94] » Early open surgery for the treatment of smaller AAAs does not reduce all-cause or AAA-specific mortality.[3] [99] One systematic review (4 trials, 3314 participants) found high-quality evidence to demonstrate that immediate repair of small AAA (4 cm to 5.5 cm) did not improve long-term survival compared with surveillance (adjusted HR 0.88, 95% CI 0.75 to 1.02, mean follow-up 10 years).[99] The lack of benefit attributable to immediate surgery was consistent regardless of patient age, diameter of small aneurysm, and whether repair was endovascular or open.[99] » Surgical referral of smaller AAA is usually reserved for rapid growth, or once the threshold diameter for aneurysm repair is reached on repeated ultrasonography.[3]
	plus	**aggressive cardiovascular risk management** » Patients should be encouraged to stop smoking and offered drug therapy to assist with this if needed. » Cardiovascular risk prevention should be prioritised in patients in AAA screening programmes.[139] » Addressing modifiable risk factors preoperatively improves long-term survival after AAA repair.[142] » Preoperative exercise training reduced post-surgical cardiac complications in a small randomised controlled trial of patients undergoing open or endovascular AAA repair.[143] » In the absence of contraindications, patients should receive low-dose aspirin and this should be continued during the perioperative period.[74] » Hypertension should be controlled to reduce cardiovascular mortality.[74]

Ongoing

Patient group	Tx line	Treatment
		» Perioperative statin use reduces cardiovascular events during non-cardiac surgery.[144] Statins should be started at least 1 month before surgery to reduce cardiovascular mortality, and continued indefinitely.[74] » Perioperative beta-blockade may be reasonable in patients at high risk of myocardial ischaemia (ischaemic heart disease or myocardial ischaemia present on stress testing), if the therapy can be commenced more than 1 month before surgery.[74] [145]
incidental finding: large asymptomatic AAA	**1st**	**elective surgical repair** » Generally, repair is indicated in patients with large asymptomatic AAA (e.g., with a diameter exceeding 5.5 cm in men or 5.0 cm in women in the US, although treatment decisions based on greater size may differ in other countries, e.g., UK). Repair of aneurysms ≥5.5 cm offers a survival advantage.[1] [78] [79] [80] [81] » Decisions regarding repair should be individualised, taking account of patient preference, patient age, sex, perioperative risk factors, and anatomical risk factors. Care should be taken to evaluate patient quality of life, and careful counselling undertaken regarding the risks of surgery and subsequent quality of life. Endovascular aneurysm repair (EVAR) should be considered in patients who are unfit for open surgery.[56] [77] [78] [79] [80] [99] 1[B]Evidence » Data suggest that in patients with large AAAs (≥5.5 cm) undergoing elective repair, EVAR is equivalent to open repair in terms of overall survival, although the rate of secondary interventions is higher for EVAR.[108] EVAR also reduces AAA-related mortality (but not longer-term overall survival) in patients with large AAA (≥5.5 cm) who are unsuitable for open repair.[109] 2[A]Evidence » Post repair, larger AAAs appear to be associated with worse late survival than smaller aneurysms (pooled HR 1.14 per 1-cm increase in AAA diameter, 95% CI 1.09 to 1.18; 12- to 91.2-month follow-up).[111] The association is more pronounced with EVAR than with open repair.
	plus	**preoperative cardiovascular risk reduction**

Ongoing

Patient group	Tx line	Treatment
		» Addressing modifiable risk factors preoperatively improves long-term survival after AAA repair.[142] » Preoperative exercise training reduced post-surgical cardiac complications in a small randomised controlled trial of patients undergoing open or endovascular AAA repair.[143] » In the absence of contraindications, patients should receive low-dose aspirin and this should be continued during the perioperative period.[74] » Hypertension should be controlled to reduce cardiovascular mortality.[74] » Perioperative statin use reduces cardiovascular events during non-cardiac surgery.[144] Statins should be started at least 1 month before surgery to reduce cardiovascular mortality, and continued indefinitely.[74] » Perioperative beta-blockade may be reasonable in patients at high risk of myocardial ischaemia (ischaemic heart disease or myocardial ischaemia present on stress testing), if the therapy can be commenced more than 1 month before surgery.[74] [145]
	plus	**perioperative antibiotic therapy** » Perioperative antibiotic therapy is given. Broad-spectrum antibiotic coverage is necessary, in accordance with local protocols.
endovascular repair leak requiring treatment	**1st**	**corrective procedure** » Endoleak is persistent blood flow outside the graft and within the aneurysm sac.[150] [151] It is not a complication following open repair. » Postoperative surveillance can detect major endoleaks and aneurysm sac expansion. Risk of endoleak following endovascular aneurysm repair (EVAR) is 24%.[150] » Type I: clinically significant; every effort should be made to repair type I endoleak before completing the procedure (e.g., balloon moulding of the proximal seal zone, placement of a proximal cuff, endostaples). Persistent type IA endoleak may necessitate conversion to open repair.[56]

Ongoing

Patient group	Tx line	Treatment
		[Fig-7] [Fig-8] » Type II: treatment remains controversial and is advocated either if persistent at 6 to 12 months or when aneurysm sac size increases such that proximal and/or distal sealing zones may be compromised.[153] [154] [155] [156] Treatment of choice is transarterial coil embolisation, although laparoscopic ligation of collateral branches, direct percutaneous translumbar puncture of the sac, translumbar embolisation, and transcatheter transcaval embolisation have been reported.[151] [153] [154] [155] [157] [158] [159] [160] » Type III: repair is indicated upon discovery (endovascular stent graft extension).[56] [154] » Type IV: these leaks are uncommon with newer stent grafts and are self-limiting, requiring no treatment.[56] [154] » Type V (endotension): less common with the newer-generation grafts;[56] there is no standardised method to measure endotension or consensus on indicated therapy in the absence of aneurysm enlargement; however, treatment of endotension to prevent aneurysm rupture is suggested in selected patients with continued aneurysm expansion.[56] [151]
	plus	**preoperative cardiovascular risk reduction** » In the absence of contraindications, patients should receive low-dose aspirin and this should be continued during the perioperative period.[74] » Hypertension should be controlled to reduce cardiovascular mortality.[74] » Perioperative statin use reduces cardiovascular events during non-cardiac surgery.[144] Statins should be started at least 1 month before surgery to reduce cardiovascular mortality, and continued indefinitely.[74] » Perioperative beta-blockade may be reasonable in patients at high risk of myocardial ischaemia (ischaemic heart disease or myocardial ischaemia present on stress testing), if the therapy can be commenced more than 1 month before surgery.[74] [145]

Ongoing

Patient group	Tx line	Treatment
	plus	**perioperative antibiotic therapy**
		» Perioperative antibiotic therapy is given. Broad-spectrum antibiotic coverage is necessary, in accordance with local protocols.

Emerging

Doxycycline

Doxycycline is a non-specific inhibitor of matrix metalloproteinases (MMPs). MMPs promote degradation of collagen and elastin and are integral to aneurysm formation.[1] [21] One clinical trial found that prolonged administration of doxycycline for 6 months is safe and well tolerated by patients with small asymptomatic AAAs and is associated with a gradual reduction in plasma MMP-9 levels.[1] [170] Another small randomised study reported no growth of aortic aneurysms in doxycycline-treated patients at 6 and 12 months.[171] [172] N-TA^3CT is a phase II randomised, double-blind, placebo-controlled study to determine whether doxycycline can reduce the rate of increase of small (3.5 to 5.0 cm among men and 3.5 to 4.5 cm among women) AAAs in patients ≥55 years of age.[173] The primary outcome is AAA maximum transverse diameter determined by CT scans at 2-year follow-up (with allowance for baseline diameter). The expected completion date of the N-TA^3CT study is September 2019. Currently, there is insufficient evidence to recommend use of doxycycline.[174]

Ticagrelor

A phase II randomised placebo-controlled trial is investigating whether treatment with ticagrelor (a P2Y12 receptor antagonist) inhibits growth of small AAA in aspirin-naive individuals (aged 50 to 85 years) with confirmed AAA between 35 and 49 mm.[175] The primary outcome of this study is mean reduction in AAA volume growth rate (%) measured with magnetic resonance imaging (MRI) at 12 months. Estimated study completion date is July 2017.

Endovascular aneurysm sealing

Endovascular aneurysm sealing (EVAS) has been developed to address the issues of endoleak and stent graft failure-related complications associated with endovascular aneurysm repair (EVAR). It involves inserting expandable stents, each with an attached bag (endobag), into the aneurysm via both femoral arteries simultaneously. A polymer is injected into the endobag to fill the aneurysm sac, thereby excluding it from the circulation. EVAS is intended to stabilise the stent graft position and reduce the rate of endoleaks and repeat interventions.[176] There is a paucity of good-quality evidence regarding the use of EVAS; however, an industry-sponsored trial and registry report study demonstrated high technical success rates, low perioperative mortality, and high rates of freedom from adverse events at 1 month.[177] [178] Endoleak rates were low, but they are more difficult to detect after EVAS and the treatment options are more complex than current EVAR endoleak treatments.[177] [178] Device migration was reported in a retrospective case series of EVAS patients (17% grafts had migrated caudally).[179] This was without clinical sequelae, but the long-term stability and reliability of the EVAS grafts requires further study and close observation.

Fenestrated EVAR (FEVAR)

FEVAR is a viable alternative to open repair for juxta- and suprarenal AAA, or for those with AAA where a short or diseased neck precludes conventional repair. However, there is no level 1 evidence for FEVAR.[180] The largest published single-centre series demonstrated mortality of 0.8% and renal occlusion rate of 4.3%.[181]

FOLLOW UP

Recommendations

Monitoring

In the US, guidelines recommend that infra-/juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter with ultrasonography/CT should be monitored every 6 to 12 months.[76] AAAs <4.0 cm require ultrasonography every 2 to 3 years.[76]

In the UK, annual intervals are employed for small AAAs (3.0 cm-4.4 cm), and 3-month intervals for 4.5- to 5.4-cm AAAs.[100]

A systematic review and meta-analysis of individual patient data concluded that surveillance intervals of 2 years for 3.0- to 4.4-cm AAAs, and 6 months for 4.5- to 5.4-cm AAAs, are safe and cost-effective.[75]

Analysis of AAA growth and rupture rates indicated that, in order to maintain an AAA rupture risk <1%, an 8.5-year surveillance interval is required for men with baseline AAA diameter of 3.0 cm.[75] The corresponding estimated surveillance interval for men with an initial aneurysm diameter of 5.0 cm was 17 months. Despite having similar growth rates of small aneurysms, rupture rates were 4 times greater in women than in men.[75] Surveillance programmes and criteria for considering surgery need to be tailored for women with opportunistically detected AAA.

Post repair

The US Society for Vascular Surgery recommends follow-up non-contrast CT imaging at 5-year intervals after open repair or endovascular aneurysm repair (EVAR).[56]

Post EVAR, baseline surveillance is recommended at 1 month with contrast-enhanced CT and colour duplex ultrasound imaging.[56] If neither endoleak nor AAA enlargement is documented, imaging should be repeated at 12 months using either contrast-enhanced CT or colour duplex ultrasound imaging.[56]

The European Society for Vascular Surgery recommends that all patients should have computed tomography angiography (CTA) and plain radiographs 30 days after the initial repair.[74] If there is endoleak, CTA at 6 months and 12 months with plain radiographs is warranted. Patients with no early endoleak and good component overlap may omit the 6-month CTA, but should have CTA and plain radiographs at 12 months. If there is no endoleak and a stable/shrinking AAA, annual duplex ultrasonography is recommended with plain radiographs.[74]

Antibiotic prophylaxis of graft infection is required prior to bronchoscopy, gastrointestinal or genitourinary endoscopy, and any dental procedure that may lead to bleeding.[56]

Generalised sepsis, groin drainage, pseudoaneurysm formation, or ill-defined pain after open repair or EVAR should prompt evaluation of graft infection.[56] Gastrointestinal bleeding after open repair or EVAR should prompt evaluation of an aorto-enteric fistula.[56]

Patient instructions

Patients should be educated on the importance of smoking cessation (including counselling and pharmacotherapy as needed), and of blood pressure and cholesterol control.

Complications

Complications	Timeframe	Likelihood
abdominal compartment syndrome	**short term**	**high**

Complications	Timeframe	Likelihood
Rates of abdominal compartment syndrome (ACS) may approach 55% after open repair of ruptured AAA.[74] A small retrospective review reported ACS in 34% of patients following open repair of ruptured AAA and 21% after endovascular aneurysm repair (EVAR).[183] A nationwide population-based study (Swedish Vascular Registry) found that after ruptured AAA repair, ACS developed in 6.8% following open repair compared with 6.9% after EVAR.[184]		
ileus, intestinal obstruction, and ischaemic colitis	**short term**	**high**
Ileus has been reported in 11% of patients, with intestinal obstruction and colitis each occurring in 1% of patients undergoing open repair.[182] [185] Ischaemic colitis requiring colectomy is rare.		
acute kidney injury	**short term**	**high**
Following EVAR and open repair of AAA, there is a significant incidence of acute kidney injury (AKI).[186] [187] [188] In open repair, this seems to be transient. However, following EVAR the causes are multifactorial, and decline in renal function is significantly greater (especially with suprarenal fixation) than in open surgery.[189] AKI following EVAR is associated with medium-term increased morbidity and mortality.[186]		
post-implantation syndrome	**short term**	**medium**
May last for up to 10 days following EVAR.[74] Fever, malaise, and back pain, which may be due to cytokine release, are typical.		
amputation due to limb ischaemia	**short term**	**low**
Rates of amputation due to limb ischaemia were very low in a large series of patients who underwent open repair.[182]		
spinal cord ischaemia	**short term**	**low**
Spinal cord ischaemia is rare after EVAR, with an incidence in the EUROSTAR collaborators registry of 0.21%.[190] In a retrospective analysis of emergency endovascular treatment for ruptured AAA, 4 of 35 patients (11.5%) developed spinal cord ischaemia postoperatively.[191] Delayed spinal cord ischaemia (developing 2 days after EVAR) has been reported.[192]		
impaired sexual function	**long term**	**high**
Damage to the autonomic nerves present at the aorto-iliac bifurcation during dissection, as well as reduction in pelvic blood supply, can result in impotence and retrograde ejaculation. In one trial, 10% of men developed new impotence in the first year after open repair.[193]		
anastomotic pseudoaneurysm	**long term**	**high**
One case series reported para-anastomotic aneurysms in 10% of patients after aortic bypass grafting.[194] The rate of femoral anastomotic pseudoaneurysm may be as high as 20% at 10 years after aortobifemoral reconstruction for AAA.[195]		
aortic neck dilation	**long term**	**high**
Aortic neck dilation occurred in 24.6% of EVAR patients during 15 months to 9 years of follow-up.[132] A composite clinical event of endoleak, migration, and re-intervention was significantly more common in this group than in those patients without aortic neck dilation.		

FOLLOW UP

Complications	Timeframe	Likelihood
graft infection	**long term**	**medium**
Can result from infection during implantation, or haematogenous seeding following dental procedures or endoscopic procedures with biopsy. Incidence is low: a retrospective cohort study found that the 2-year rate of graft infection was 0.19% following open repair versus 0.16% with EVAR.[196]		
ureteric obstruction	**long term**	**low**
Ureteric obstruction is related to encasement of the ureters in an inflammatory perianeurysmal fibrosis of unresolved aetiology rather than secondary to aneurysm compression.[197] Most often, ureteral compression is associated with inflammatory aortic aneurysm. Extensive retroperitoneal adhesions may result in ureteral obstruction in 18% of patients. The inferior vena cava may become involved as well.[198]		
functional gastric outlet obstruction	**long term**	**low**
Duodenal obstruction is a consequence of compression of the duodenum in its fixed retroperitoneal course between the aneurysmal aorta and the superior mesenteric artery.[197]		
graft limb occlusion	**long term**	**low**
The incidence of graft limb occlusion up to 10 years after open AAA repair has been reported as being between 2.6% and 3.0%.[199] [200] The risk of graft occlusion is greater with EVAR, with a reported incidence of up to 7.2% in follow-up studies.[201] Kinking is a risk factor for graft limb occlusion following EVAR.[202]		
endoleak	**variable**	**high**
Risk of endoleak following EVAR is 24%.[150] Type II endoleaks are the most common. Repair may be indicated upon discovery of postoperative type I endoleak. Endoleak is not a complication following open repair.		
distal embolisation	**variable**	**low**
Incidence is 3% to 29%, most commonly affecting the digits (blue toe syndrome). There is a 5% incidence of distal embolisation resulting in limb-threatening ischaemia, digital ischaemia, and calf myonecrosis.[203]		

Prognosis

The natural course involves slow and steady growth with ultimate progression to rupture. Most patients with rupture will not survive to reach the operating theatre. Given the morbidity and mortality associated with surgical intervention, repair is typically deferred until the theoretical risk of rupture exceeds the estimated risk of operative mortality. The majority of patients undergoing open repair remain without significant graft-related complications during the remainder of their lives (0.4% to 2.3% incidence of late graft-related complications).[1] [182] Five-year survival rates after intact aneurysm repair average 60% to 75%. Those undergoing endovascular aneurysm repair (EVAR) are more likely to have a delayed complication and require re-intervention.

Diagnostic guidelines

Europe

Abdominal aortic aneurysm screening: how it works

Published by: Public Health England **Last published:** 2015

Summary: Includes information about the NHS AAA screening programme, and the tests and processes involved.

2014 ESC guidelines on the diagnosis and treatment of aortic diseases

Published by: European Society of Cardiology **Last published:** 2014

Summary: Includes a critical evaluation of diagnostic imaging and screening for AAA.

North America

The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm

Published by: Society for Vascular Surgery **Last published:** 2018

Summary: Addresses patient evaluation and risk of rupture in AAA.

ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery

Published by: American College of Cardiology; American Heart Association **Last published:** 2014

Summary: Recommendations regarding preoperative risk assessment and cardiac testing in the adult patient undergoing non-cardiac surgery.

Screening for abdominal aortic aneurysm: US Preventive Services Task Force recommendation statement

Published by: US Preventive Services Task Force **Last published:** 2014

Summary: Describes risk factors and assessment, and includes recommendations regarding screening.

Treatment guidelines

Europe

2014 ESC guidelines on the diagnosis and treatment of aortic diseases

Published by: European Society of Cardiology **Last published:** 2014

Summary: Includes recommendations for the management of asymptomatic and symptomatic AAA.

Europe

Management of abdominal aortic aneurysms: clinical practice guidelines of the European Society for Vascular Surgery

Published by: European Society for Vascular Surgery **Last published:** 2011

Summary: Evidence-based recommendations regarding the clinical care of preoperative, perioperative, and postoperative AAA patients.

Endovascular stent-grafts for the treatment of abdominal aortic aneurysms

Published by: National Institute for Health and Care Excellence **Last published:** 2009

Summary: Appraises the use of grafts in the treatment of AAA.

Laparoscopic repair of abdominal aortic aneurysm

Published by: National Institute for Health and Care Excellence **Last published:** 2007

Summary: Recommendations regarding the laparoscopic repair of AAA, including who should carry out the procedure, and the information given to patients and their Trust.

North America

The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm

Published by: Society for Vascular Surgery **Last published:** 2018

Summary: Reviews peri- and intra-operative strategies, follow-up, and treatment of complications.

ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery

Published by: American College of Cardiology; American Heart Association **Last published:** 2014

Summary: Guideline addressing pharmacotherapeutic and anaesthetic considerations for non-cardiac surgery patients.

Screening for abdominal aortic aneurysm: US Preventive Services Task Force recommendation statement

Published by: US Preventive Services Task Force **Last published:** 2014

Summary: Identifies patients who are candidates for open surgical repair or endovascular aneurysm repair (EVAR), and those who should be managed conservatively via surveillance.

Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations)

Published by: American College of Cardiology Foundation; American Heart Association **Last published:** 2013

Summary: Recommendations for the screening and management of patients with aneurysms of the abdominal aorta.

North America

Guidelines for the treatment of abdominal aortic aneurysms

Published by: American Association for Vascular Surgery; Society for Vascular Surgery **Last published:** 2003

Summary: Decisions relating to elective repair of AAA require careful assessment of factors that influence rupture risk, operative mortality, and life expectancy.

Evidence scores

1. Rupture: there is medium-quality evidence that the risk of rupture was 20% for aneurysms measuring larger than 5.0 cm to 6.0 cm in diameter, and 2 prospective randomised trials comparing early intervention versus expectant observation for infrarenal AAAs measuring 4.0 cm to 5.4 cm in diameter were conducted in the UK and the US Department of Veterans Affairs during the past decade.[105] [106] [107]
Evidence level B: Randomized controlled trials (RCTs) of <200 participants, methodologically flawed RCTs of >200 participants, methodologically flawed systematic reviews (SRs) or good quality observational (cohort) studies.

2. Reduction in AAA-related mortality in patients unsuitable for open repair: there is good-quality evidence from a large randomised controlled trial that, in patients with large abdominal aortic aneurysms 5.5 cm or greater physically ineligible for open repair, endovascular repair (EVAR) was associated with a significantly lower AAA-related mortality compared with no repair, although no reduction in overall mortality was seen after 8 years. However, the rate of graft-related complications and secondary interventions following EVAR was high.[110]
Evidence level A: Systematic reviews (SRs) or randomized controlled trials (RCTs) of >200 participants.

Key articles

- Chaikof EL, Dalman RL, Eskandari MK, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg. 2018 Jan;67(1):2-77.e2. Full text Abstract
- Moll FL, Powell JT, Fraedrich G, et al; European Society for Vascular Surgery. Management of abdominal aortic aneurysms: clinical practice guidelines of the European Society for Vascular Surgery. Eur J Vasc Endovasc Surg. 2011 Jan;41 Suppl 1:S1-58. Full text Abstract
- Anderson JL, Halperin JL, Albert NM, et al. Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013 Apr 2;127(13):1425-43. Full text Abstract
- Brewster DC, Cronenwett JL, Hallett JW Jr, et al. Guidelines for the treatment of abdominal aortic aneurysms: report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. J Vasc Surg. 2003 May;37(5):1106-17. Full text Abstract
- IMPROVE Trial Investigators. Comparative clinical effectiveness and cost effectiveness of endovascular strategy v open repair for ruptured abdominal aortic aneurysm: three year results of the IMPROVE randomised trial. BMJ. 2017 Nov 14;359:j4859. Full text Abstract
- Greenhalgh RM, Brown LC, Powell JT, et al; United Kingdom EVAR Trial Investigators. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med. 2010 May 20;362(20):1863-71. Abstract
- Greenhalgh RM, Brown LC, Powell JT, et al; United Kingdom EVAR Trial Investigators. Endovascular repair of aortic aneurysm in patients physically ineligible for open repair. N Engl J Med. 2010 May 20;362(20):1872-80. Abstract
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Images

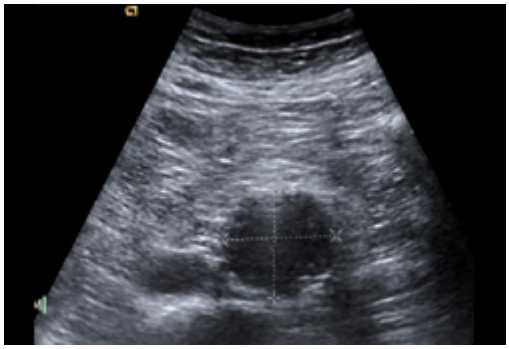

Figure 1: Ultrasound of a 3.8 cm x 4.2 cm AAA

University of Michigan, specifically the cases of Dr Upchurch reflecting the Departments of Vascular Surgery and Radiology

Aortic diameter (anterior-posterior, inner to inner)	Re-scanning interval
<3 cm	Discharged from programme
3-4.4 cm	Annual surveillance programme
4.5-5.4 cm	3-monthly surveillance programme
>5.5 cm	Referral to vascular surgeon*

Figure 2: Re-scanning intervals associated with aortic diameters

NHS population screening: care pathways, Public Health England

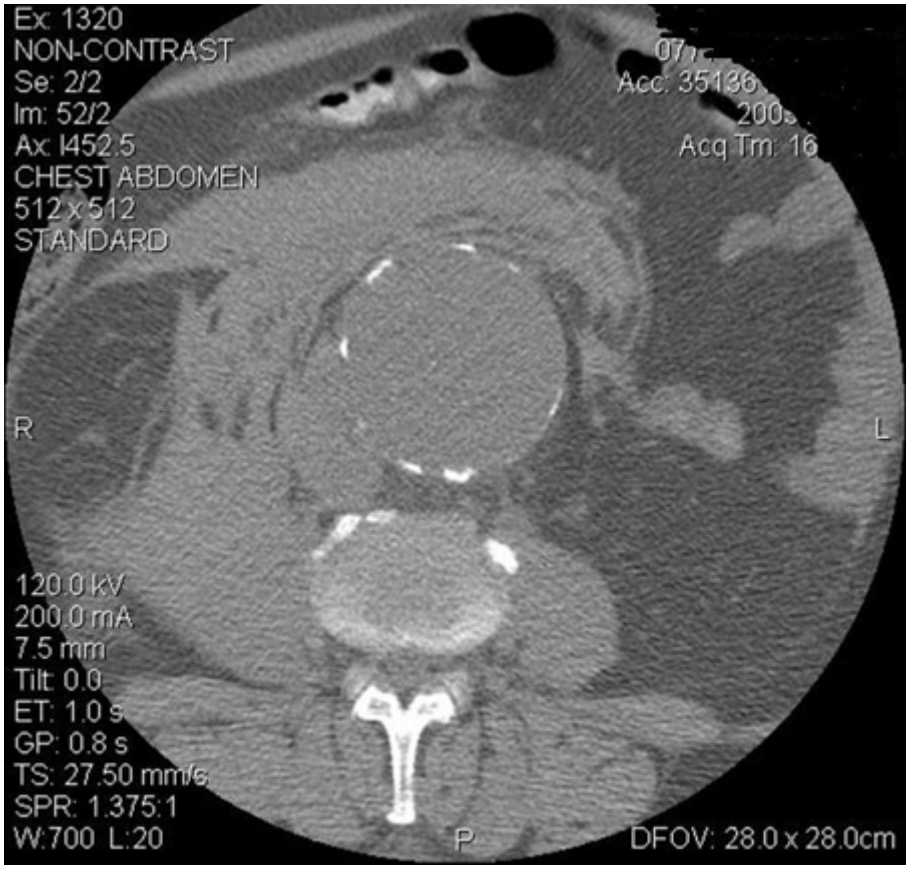

Figure 3: CT scan of a ruptured AAA

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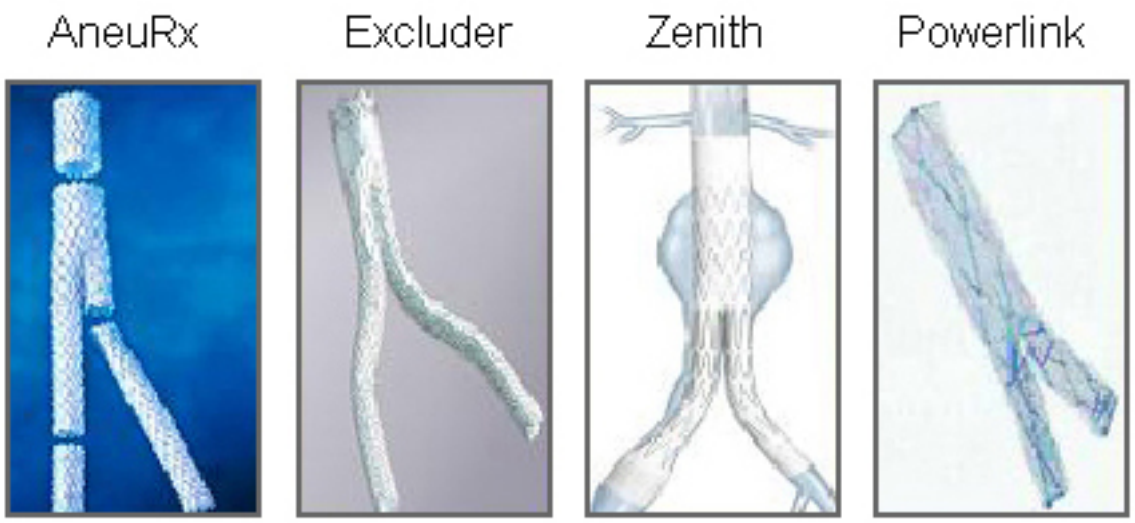

Figure 4: Various endovascular stent grafts used for endovascular aneurysm repair (EVAR)

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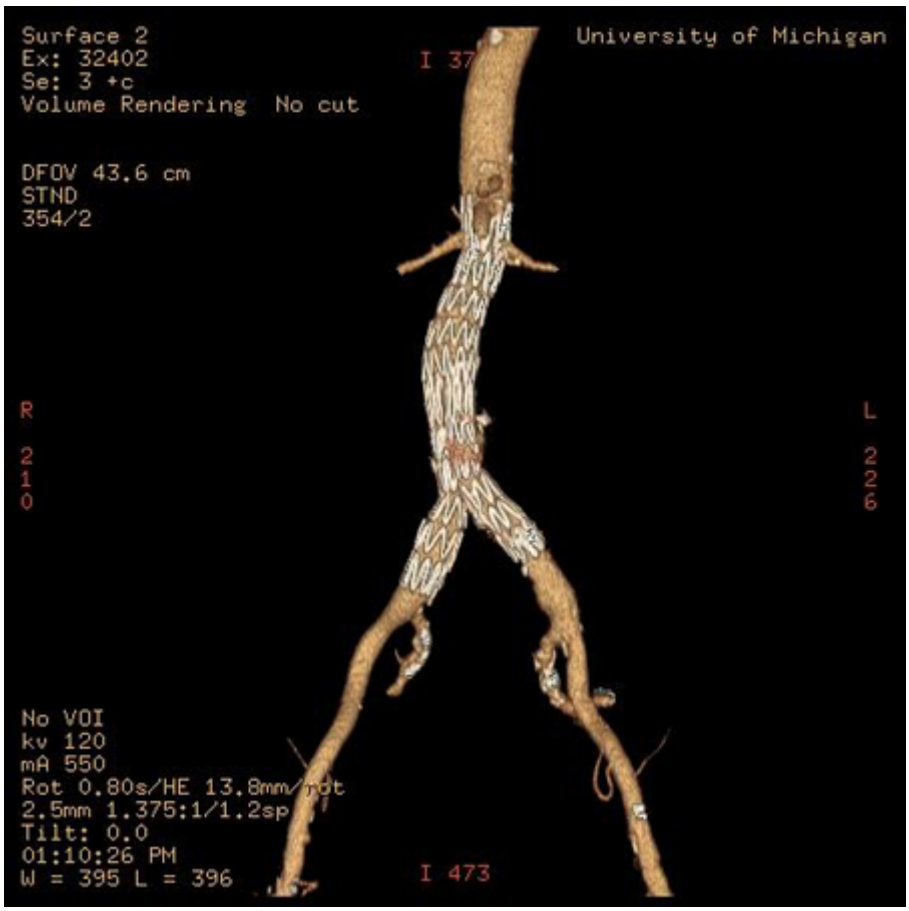

Figure 5: Endovascular aneurysm repair (EVAR)

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Figure 6: Type I endoleak at the distal left iliac anastomosis (leak encircled)

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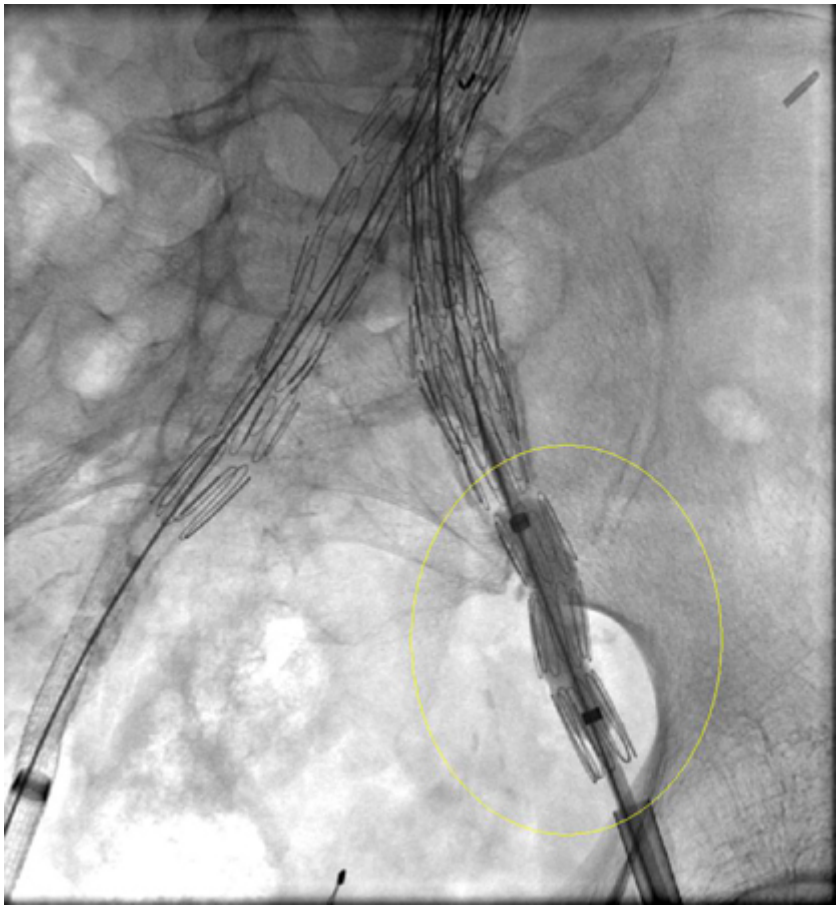

Figure 7: Extension stent graft deployed for the same type I endoleak (encircled)

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Figure 8: Resolution of the type I endoleak resolved after extension deployed

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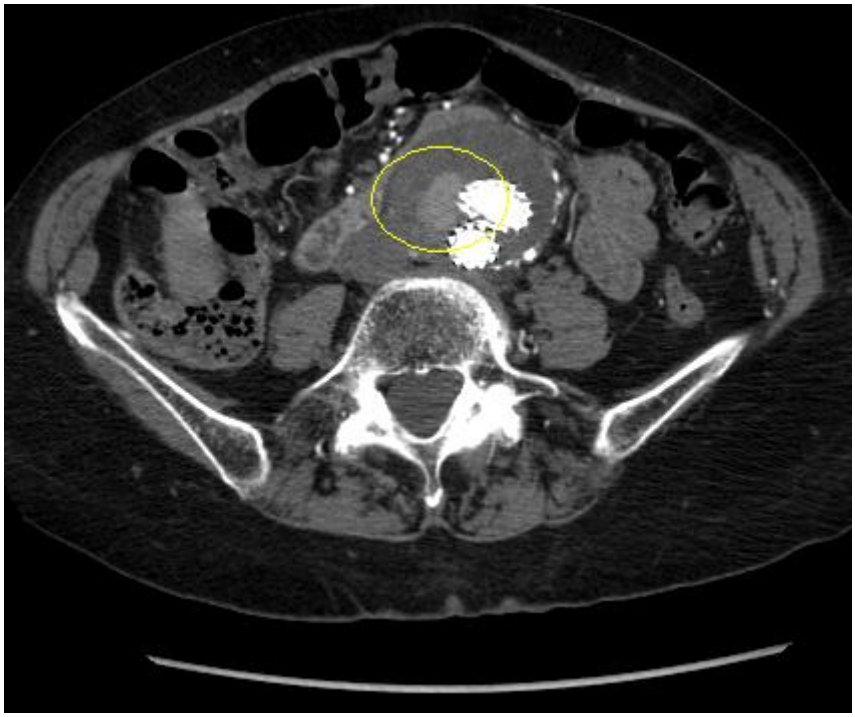

Figure 9: Type II endoleak (encircled) discovered on follow-up CT

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DISCLOSURES: MJB has grant funding from the British Heart Foundation and the National Institute for Health Research.

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// Acknowledgements:

Professor Matt Bown and Mr Andrew Duncan would like to gratefully acknowledge Dr Maureen K. Sheehan, Dr Dawn M. Barnes, and Dr Gilbert R. Upchurch, previous contributors to this monograph.
DISCLOSURES: MKS, DMB, and GRU declare that they have no competing interests.

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