Abdominal aortic aneurysm

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Rupture of an abdominal aortic aneurysm (AAA) causes 1–2% of all male deaths over 65 years of age in western countries.[1] The incidence is increasing and this can only partly be explained by an ageing population. Surgery improves survival of rupture, but most individuals with a ruptured AAA die before reaching the operating theatre. Of the remaining 25%, subjected to an emergency operation, about a half dies during or after the operation due to myocardial infarction, haemorrhage, or multiple organ dysfunction. The overall mortality of AAA rupture is therefore still around 80–90%.[2]

Although the life expectancy of individuals with symptomless AAA is decreased by the chance of rupture, elective surgical correction can improve survival. The mortality of elective AAA repair has diminished to 4–8%.[3] Since AAA is seldom symptomatic before rupture, investigation is necessary for timely diagnosis and operative correction.

A consensus definition of aneurysm was published by the Society of Vascular Surgery and the International Society for Cardiovascular Surgery in 1991:[4] a permanent localised dilation of an artery having at least a 50% increase in diameter compared with the expected normal diameter of the artery, or of the diameter of the segment proximal to the dilation. Based on a maximum diameter of 2·1 cm of the infrarenal aorta in healthy individuals, an AAA is present when the diameter exceeds 3·0 cm.

Prevalence and natural history

The AAA screening studies are difficult to compare because of differences in design, the composition of the population, and the definition of AAA. It is estimated that AAA is fourfold as common and tenfold more fatal in men than in women, who are affected when 10 years older than their male counterparts. Thus most screening studies are restricted to men. The reported prevalence of AAA in men over 65 years varies from 4·3 to 8·8%.[5-6]

Estimation of AAA incidence based on mortality statistics and hospital registrations is also influenced by variations in the populations concerned. The incidences of AAA and AAA-related mortality vary a lot. Nevertheless, over the years, the incidence of AAA is increasing. This is partly explained by ageing of the population; however, the prevalence standardised for age and sex is also rising.[7,8]

Necropsies show that a third of AAA are ruptured and can therefore be considered as the cause of death. The 5-year survival rate without AAA repair is around 50%.[9]

Aneurysm size is the most important factor related to likelihood of rupture. Although rupture may occur with an aortic diameter of less than 4 cm, the risk is small. With an AAA diameter under 5 cm, the yearly chance of rupture is about 0·5%.[10] For AAA of 5–6 cm, annual chance of rupture is 5%, increasing exponentially for larger aneurysms.[9,10]

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The risk of rupture increases when growth exceeds the expected expansion rate.[11] The normal rate of expansion increases with AAA diameter following an exponential curve with a median growth of 0·5 cm per year. An increased risk of rupture has been observed in patients with hypertension, chronic obstructive pulmonary disease, and familial AAA. The chance of rupture is also enhanced in cigarette smokers and in the absence of peripheral obstructive vascular disease. With few exceptions, AAA is diagnosed in patients over 55 years of age and rupture seldom occurs before age 65. Although uncommon, aneurysmal disease presenting before the sixth decade of life is reported to be symptomatic in a higher proportion of cases and associated with more proximal aortic involvement than AAA in older patients.[12]

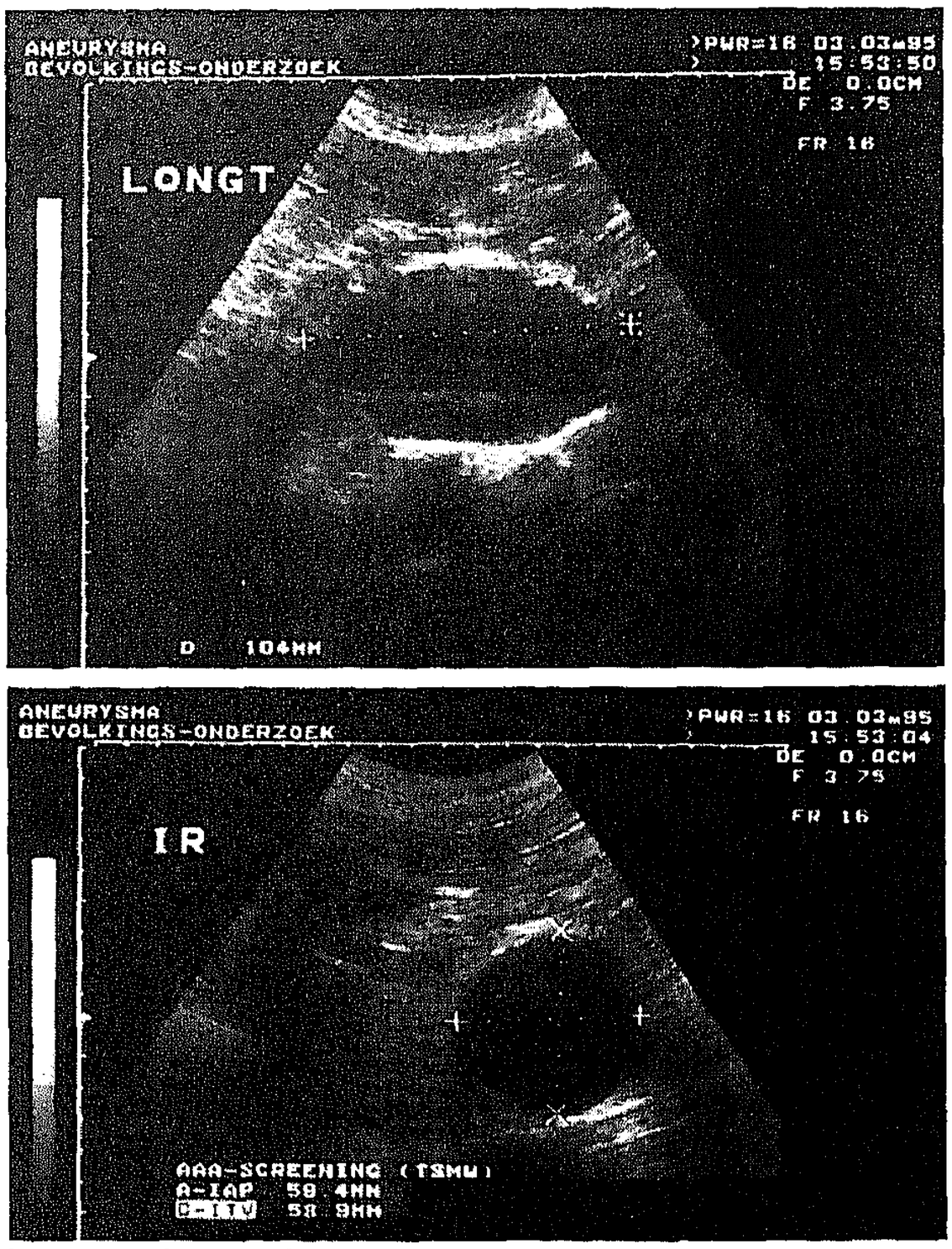
Pathogenesis and risk indicators

With vascular smooth-muscle cells, the key structural proteins elastin and collagen are arranged in concentric lamellae to withstand the haemodynamic stresses imposed on the aortic wall.[13] The architecture of these lamellae allows for a stretching of elastin fibres with increasing load. As the vessel wall continues to stretch, collagen fibres uncoil and are progressively recruited as load-bearing elements. Under normal conditions, elastin is the principal load-bearing element, with collagen acting as a strong, almost indistensible "safety net". Destruction of the architecture results in aneurysmal dilation. The pulse pressure in the aorta increases from proximal to distal, as the aorta branches and tapers, and because the abdominal aorta is stiffer than its more proximal part corresponding to a reduced number of elastic lamellae distally. This may explain the predominance of aneurysms in the abdominal aorta. Hypertension enhances the growth rate of aneurysms and is associated with an increased prevalence of AAA,[14] which indicates that an increased load on the aortic wall may be involved in pathogenesis.

Elastin is not synthesised in the adult aorta. With a half-life of 70 years the amount of elastin in the aortic wall decreases with age. The age-related alterations in the vessel wall affect the mechanical properties of the aorta. This explains why AAA is primarily a disease of old age. Aneurysmal dilation, and eventually rupture, can only occur with failure of the collagen safety-net. Collagen production continues throughout life and is even increased in the aneurysmal wall. Besides enhanced collagen synthesis, collagenolytic activity is increased in AAA. Several hereditary connective-tissue disorders (ie, Ehlers-Danlos and Marfan's syndromes) are associated with aneurysm formation at an early age. A positive family history in a first-degree relative is a risk factor for AAA.[15,16] The familial clustering of AAA indicates a genetic basis. Research has been directed at several candidate genes coding for matrix components (such as procollagen type III) and also for connective tissue proteases (such as type IV collagenase or matrix metalloprotease-2), and antiproteases (such as alpha-1-antitrypsin or tissue inhibitors of metalloproteases). Although several defects have been identified, the genetic basis of aneurysm formation remains unresolved.

The historical association of AAA with atherosclerosis has now expanded into a multifactorial causation for the disease.[17] It is unclear why atherosclerosis, normally causing narrowing of the arterial lumen, should in some cases result in dilation. There are epidemiological differences between patients with obstructive vascular and aneurysmal disease. The male preponderance in aneurysmal disease is stronger and diabetes mellitus is found less frequently. There are also differences in serum lipoprotein profiles. Paradoxically, patients with peripheral obstructive vascular disease carry an increased risk for AAA, indicating that many of the risk factors for dilating and stenosing arterial disease are shared. Histological examination of the aneurysm wall reveals a chronic adventitial and medial inflammatory infiltrate of varying intensity.[18] This distinguishes AAA from the purely atherosclerotic aorta, in which inflammatory cells are mainly associated with the plaque. An intense inflammatory-cell infiltrate that often extends beyond the aortic wall into surrounding tissues is present in the inflammatory aneurysm, which is no longer regarded as a separate entity. It is unclear whether the inflammatory response is primary or secondary. The inflammatory response subsides both in the aortic wall and in the retroperitoneum after repair of the inflammatory AAA.

Little is known about the role of micro-organisms in the pathogenesis of AAA. Seroepidemiological studies have, for instance, associated *Chlamydia pneumoniae* infection with several manifestations of atherosclerosis, such as coronary heart disease. This pathogen, which has been isolated from various atheromatous inflammatory lesions, was also detected in the AAA wall.[19]



Ultrasound scan of AAA
Longitudinal (LONGT) and transverse infrarenal (IR) scan of screen-detected asymptomatic AAA with diameter of 5·8 cm. Large thrombus surrounding small central lumen lines aneurysmal wall.

Detection

Physical examination for the diagnosis of AAA is associated with a large proportion of false-negative and false-positive findings, resulting in poor predictive value.[20,21] Therefore imaging techniques are required for the detection of AAA. Plain abdominal radiographs only reveal aneurysms with some wall calcification. As a consequence, more than a quarter of the results are false-negative. By contrast, ultrasonography (figure) is suited for visualising the abdominal aorta and in the detection of an increase in diameter.[20] The technique is reliable and reproducible with a sensitivity of 95% and a specificity of almost 100%. Other advanced techniques, such as computed tomography and magnetic resonance scanning are also reliable, but are not as accessible as ultrasonography and do not improve the diagnosis of AAA. Alone or with contrast angiography, these techniques can be useful in delineating anatomical details in preparation for vascular reconstruction. The less invasive magnetic-resonance angiography has been reported to provide accurate information on AAA anatomy and the presence of inflammatory change.

Treatment

For small aneurysms the use of β-blockers is advocated to slow expansion. However, evidence in support of this regimen is lacking.[22] Other doctors merely restrict follow-up to watchful waiting with periodic ultrasonography. In view of the rising chance of rupture, many surgeons have a policy of proceeding to repair when the AAA diameter has reached 5 or 5·5 cm or when growth of more than 1 cm per year is recorded. The conventional treatment involves insertion of vascular prosthesis within the lumen of the aneurysm, firmly sutured to the non-dilated proximal and distal vascular segments. In most cases a tube graft is placed, but with extension of the aneurysm into the iliac arteries a bifurcated graft is used. The aneurysm itself is not resected and the aneurysmal sac is wrapped around the vascular prosthesis to prevent contact with the intestine. The infrarenal AAA can either be approached transperitoneally through a midline laparotomy or retroperitoneally through various incisions.[23] Aneurysms that extend proximally beyond the renal arteries, especially those with involvement of the superior mesenteric and coeliac arteries, are best approached through a left thoracolaparotomy.

In preparation for these major operations, which are generally performed in elderly patients, careful cardiac and pulmonary evaluation is required. This may include dipyridamole-thallium scintigraphy or dobutamine-stress echocardiography.[24,25] Invasive haemodynamic monitoring is warranted perioperatively and the use of an autotransfusion device can be helpful. Nonetheless, much of the 4–8% mortality of elective AAA repair is caused by cardiac complications. Intraoperative events may result in excessive blood loss and rarely in lower extremity, renal, or colonic ischaemia. Ischaemic spinal involvement is rarely encountered in AAA repair due to the low level of aortic cross-clamping. Although bacteria can often be cultured from the aneurysm contents at surgery, life-threatening prosthetic infections occur in only 1–2% of cases with use of single-dose antibiotic prophylaxis.[26] Long-term follow-up after AAA repair appears to be unnecessary and life-style does not need to be restricted.

An interesting development is the introduction of less-invasive endovascular AAA repair.[27] A vascular prosthesis is introduced through femoral access and attached under radiographic guidance to the non-dilated vascular wall on

both sides of the aneurysm with the use of endovascular stents. Although controlled trials have not been done, experience is building and it is evident that the use of this technique reduces the surgical trauma of AAA repair. Promising results have been obtained with straight and bifurcated stent grafts in selected patients, but long-term follow-up is lacking. Some early failures due to perigraft leakage and stent fractures have been reported, indicating that further technical development is desired.

Intervention, prognosis, and secondary prevention

The life expectancy after elective and ruptured AAA repair has been compared with matched controls without AAA. Little or no differences were observed in length and quality of life, even in patients over 80 years of age. Patients with AAA have an increased mortality due to atherosclerotic manifestations, such as coronary disease, cerebrovascular disease, renal insufficiency, and thoracic aneurysms. This results in a reduction of life expectancy by 2 years: AAA rupture may shorten life by a mean of 9 years.[28]

A regional population-based study[29] of secondary prevention of AAA rupture in the UK randomised nearly 16 000 men and women aged 65-80, with one-half receiving an invitation from their family physician for ultrasonographic investigation of the abdominal aorta. The other half was not invited. Of the invitees 68% were screened. Both groups were followed up over 5 years. The prevalence of AAA in the screened population was 7·6% in men and 1·3% in women. According to a previously approved protocol, individuals with an AAA were followed up with or without elective aneurysm repair. During the study, AAA ruptured in 20 men in the control group and in 9 men invited for screening. As a result of this screening programme, the incidence of AAA rupture in men was reduced by 55% over the 5 years.

Conclusion

In view of the rising incidence of AAA and the related mortality from rupture, it is desirable to reduce the significance of this disorder. The causes that are known do not provide leads for primary prevention. However, mortality from rupture can be reduced by timely intervention. Elective surgical repair in secondary prevention has developed into a safe and effective intervention. New endovascular techniques may well prove to be an asset in AAA treatment.

There are a number of risks indicators that are asociated with AAA. However, these factors are present in a minority of individuals with AAA. Only age and sex can be used to delineate the population at risk. The group with the highest AAA-related mortality risk is men aged 60 or older. AAA can be reliably detected simply and cheaply with ultrasound of the abdominal aorta. The scheduling of elective AAA repair depends on life expectancy and chance of rupture, for which the aneurysm diameter is the only indicator.

A possible health-care strategy is an ultrasonographic AAA screening programme of men aged 60 or older. It is a prerequisite that most of the population at risk is actually examined. Promising results have been obtained in programmes in which general practitioners act as key persons, involved in approaching individuals at risk and in the care of those with detected AAA. Screening is best organised locally. Although no false-positive results are to be expected on ultrasound screening, some anxiety will inevitably be caused, especially in the group with a small AAA for which surgical repair is not immediately indicated. Quality-of-life assessments are necessary to estimate the importance of this side-effect. A cost-benefit analysis is required, in which the financial burden of the treatment of ruptured AAA is balanced against the direct and indirect costs and yield of secondary prevention.[30,31]

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