REVIEW ARTICLE:
ABDOMINAL AORTIC ANEURYSM: A COMPREHENSIVE REVIEW

By

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INTRODUCTION

An arterial aneurysm is defined as a focal dilation of a blood vessel with respect to the original artery. An abdominal aortic aneurysm (AAA) is defined as an aortic diameter at least one and one-half times the normal diameter at the level of the renal arteries, which is approximately 2.0 cm. Thus, generally, a segment of abdominal aorta with a diameter of greater than 3.0 cm is considered an aortic aneurysm (**Hirsch et al., 2006**).

Approximately 80% of aortic aneurysms occur between the renal arteries and the aortic bifurcation. Aortic aneurysms constitute the 14th leading cause of death in the United States (**Silverberg et al., 1990**). Each year in the United States, AAA rupture causes 4500 deaths, with an additional 1400 deaths resulting from the 45,000 repair procedures performed to prevent rupture (**McPhee et al., 2007**).

RISK FACTORS

The risk factors associated with AAA include age, sex, ethnicity and smoking, among others.

The risk of AAAs increases dramatically after 60 years of age. Clinically relevant aneurysms (more than 4 cm in diameter) are present in approximately 1% of men between 55 and 64 years of age, and the prevalence increases by 2% to 4% per decade thereafter (**Singh et al., 2001 and Powell &Greenhalgh, 2003**). AAAs are four to six times more common in men than in women (**Scott et al., 1995 and Lederle et al., 2001**). In addition, AAAs develop in women approximately 10 years later than in men (**McFarlane., 1991**). In one study, AAAs were found to occur more frequently in white people than in black people (**Lederle et al., 2000**).

Smoking has been found to be a major risk factor for aneurysm formation (**Hirsch et al., 2006**). A study found smoking to be the risk factor most strongly associated with AAA (**Lederle et al., 2000**). The association with smoking was directly related to the number of years of smoking, and the association decreased with the number of years after cessation of smoking (**Johnson et al., 1997**).

AAAs are more common in patients with atherosclerosis, with a prevalence of

approximately 5% in patients with coronary artery disease, and approximately 10% in those with arteriosclerosis obliterans (**Cabellon et al., 1983** and **Bengtsson et al., 1989**). Hypertension has also been found to be associated with AAA (**Johnson et al., 1997**). A positive family history is another potential factor that significantly increases the risk of AAA (**Fleming et al., 2005**). A family history of surgical intervention for an AAA in a first-degree relative may increase the risk fourfold (**Salo et al., 1999**). AAA has been found to be less common in patients with diabetes (**Lederle et al., 2000**).

RISK OF RUPTURE

The likelihood that an aneurysm will rupture is influenced by a number of factors including aneurysm size, expansion rate and sex.

Aneurysm size is one of the strongest predictors of the risk of rupture, with risk increasing markedly at aneurysm diameters greater than 5.5 cm. The five-year overall cumulative rupture rate of incidentally diagnosed aneurysms in population-based samples is 25% to 40% for aneurysms larger than 5.0 cm, compared with 1% to 7% for aneurysms 4.0 cm to 5.0 cm in diameter (**Nevitt et al., 1989; Lederle, Johansson et al., 1990, and Johnson et al., 2002**). A statement from the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgeryestimated the annual rupture risk according to AAA diameter to be the following (**Brewster et al., 2003**):

AAA Diameter	Risk Percentage
Less than 4.0 cm in diameter	0%
4.0 cm to 4.9 cm in diameter	0.5% to 5%
5.0 cm to 5.9 cm in diameter	3% to 15%
6.0 cm to 6.9 cm in diameter	10% to 20%
7.0 cm to 7.9 cm in diameter	20% to 40%
8.0 cm in diameter or greater	30% to 50%

The expansion rate may also be an important determinant of the risk of rupture (**Bengtsson et al., 1993 and Gadowski et al., 1994**). A small AAA that expands 0.5 cm or more over six months of follow-up is considered to be at high risk for rupture (**Hirsch et al., 2006**). Growth tends to be more rapid in smokers, and less rapid in patients with diabetes mellitus or peripheral vascular disease (**Brady et al., 2004**).

In addition to aneurysm size and expansion rate, other factors that increase the risks of rupture are continued smoking, uncontrolled hypertension and increased wall stress (**Brewster et al., 2003**).

CLINICAL PRESENTATION

The majority of AAAs are asymptomatic and are most often detected as an incidental finding on ultrasonography (USG), abdominal computed tomography (CT), or magnetic resonance imaging performed for other purposes. Most AAAs are silent until they rupture, although some are identified during evaluation for abdominal symptoms. Aneurysms producing symptoms, especially pain and tenderness on palpation, are at increased risk for rupture.

AAAs can also present with complications due to thrombosis, embolization, or, rarely, as clinically overt disseminated intravascular coagulation causing hemorrhagic and thrombotic complications (**Fisher et al., 1983 and Aboulafia & Aboulafia, 1996**).

Acute AAA rupture is one of the most dramatic emergencies in medicine. In the United States, ruptured AAAs are estimated to cause 4% to 5% of sudden deaths (**Schermerhorn, 2009**). Patients with ruptured AAAs classically present with shooting abdominal or back pain and a pulsatile abdominal mass. Aneurysm rupture typically causes severe hypotension. Only approximately, 50% of patients with ruptured AAAs reach the hospital alive, of those who reach the hospital, up to 50% do not survive repair (**Harris et al., 1991**).

Approximately, 5% of AAAs were classified as inflammatory aneurysms which present with abdominal or back pain, tenderness on palpation, weight loss, and an elevated erythrocyte sedimentation rate (**Hirsch et al., 2006**).

DIAGNOSIS

The diagnosis of an AAA should ideally be made before the development of clinical symptoms to prevent rupture. Approximately, 30% of asymptomatic AAAs are discovered as a pulsatile abdominal mass on routine physical examination. Physical examination may reveal a pulsatile expansile mass at or above the umbilicus. The vascular examination should include abdominal auscultation because the presence of a bruit may indicate aortic or visceral arterial atherosclerotic disease, or rarely an aorto-caval fistula (machinery murmur). Large aneurysms in thin people are easy to detect. The accuracy of the clinical examination is tremendously reduced by obese body habitus and small

aneurysm size. However, the physical examination has considerably variable interobserver sensitivity for detection of AAAs. The sensitivity of physical examination for the identification of an AAA ranges from 22% to 96%, and even an experienced physician may miss palpating an AAA in the presence of obesity or abdominal distention (**Chervu et al., 1995**).

An asymptomatic AAA is often discovered incidentally because of the performance of abdominal USG, CT, or magnetic resonance imaging for other purposes. An AAA may also be found with plain X-rays showing some calcification in the wall of the aneurysm. However, they are not reliable, because some aneurysms do not have sufficient calcification to be detected.Abdominal USG (*Figure 1*) is considered the screening modality of choice for AAAs because of its high sensitivity of 95% to 100% and a specificity of nearly 100%, as well as its safety and relatively low cost. USG has excellent test characteristics for diagnosing and following an AAA (**LaRoy et al., 1989**).
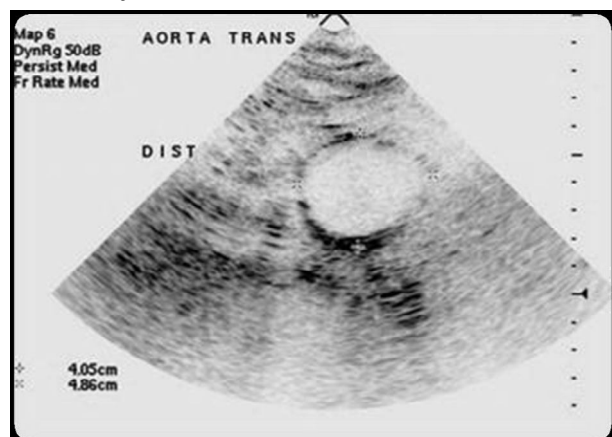

Figure (1): Ultrasonogram from patient with (AAA). This aneurysm was best visualized on transverse or axial image. Patient underwent conventional AAA repair (**LaRoy et al., 1989**).

Thrombus or echodense calcifications in or adjacent to the aortic wall may also be seen and both are quite common. Disadvantages of abdominal USG are that it is operator dependent and, in 1% to 2% of cases, overlying bowel gas or obesity hinders proper imaging of the abdominal aorta (**Scott et al., 1991**).

CT scanning evaluates the abdomen in more detail in patients with a specific abdominal complaint. It also assesses the shape of the aneurysm with more comprehensive anatomical details of the mesenteric and iliac arteries, and also provides better imaging of suprarenal aneurysms. Although USG is generally preferred, multislice CT angiography can be used for serial monitoring of aneurysm size *(Figure 2)*.

Figure (2): CT demonstrates abdominal aortic aneurysm (AAA).Aneurysm was noted during workup for back pain, and CT was ordered after AAA was identified on radiography. No evidence of rupture is seen (**Isselbacher, 2005**).

CT angiography is also essential in tailoring stent grafts in cases for which endovascular treatment is indicated. Disadvantages of CT scanning compared with USG include increased cost, requirement for contrast, exposure to radiation with repeated scans, and limitation of accuracy in localizing the aneurysm neck in some cases compared with contrast angiography (**Isselbacher, 2005**).

Magnetic resonance angiography is probably more accurate than CT, but is more expensive and not universally available (**Petersen et al.**, **1995**).

SCREENING

The decision to screen for AAA is difficult to make because it would expose many previously undiagnosed small aneurysms that are unlikely to rupture, resulting in needless disease labeling (**Melton et al., 1984**). This results in unnecessary psychological distress to patients, which manifests as lower quality of life scores (**Lindholtet al., 2000**), and transiently mildly higher anxiety scores and lower self-rated perception of health (**Ashton et al., 2002**). Only aneurysms of a certain size are considered for surgery, with smaller aneurysms subject to watchful waiting. Various studies analyzing the effectiveness of population-based screening for AAAs with abdominal USG in people older than 65 years of age concluded that screening for AAA significantly reduces the risk of AAA-related mortality by approximately 50% in men (**Fleming et al., 2005**). Another study indicated a significant reduction in AAA-related mortality in men 65 to 74 years of age but not in men 75 to 83 years of age(**Norman et al., 2004**). However,

no significant benefit to AAA-related mortality or all-cause mortality was achieved in a study that involved population-based screening in women (**Scott et al., 1995**). A model study involved only men between 65 to 74 years of age with a history of smoking reported an 89% anticipated reduction in AAA-related mortality(**Fleming et al., 2005**).

Various guidelines have been issued regarding screening for AAA. The United States Preventive Services Task Force (USPSTF) makes the following recommendations (**US Preventive Services Task Force Screening for abdominal aortic aneurysm, 2005**):

● Men between 65 to 75 years of age who have ever smoked should be screened once for AAAs by abdominal USG. The USPSTF found little benefit to repeat screening in men who have a negative USG and who are older than 75 years of age.

● The USPSTF does not make any recommendation for men 65 to 75 years of age who have never smoked.

● The USPSTF recommends against screening women for AAA.

The American College of Cardiology/ American Heart Association (ACC/AHA) guidelines issued in 2005 regarding screening of patients for AAA. They recommended that men 60 years of age or older are either siblings or offspring of patients with AAAs should undergo a physical examination and USG screening for the detection of AAA. As well, the guideline recommended that men 65 to 75 years of age who have ever smoked should undergo a physical examination

and one-time ultrasound screening for detection of AAAs(**Hirsch et al., 2006**).

The **Canadian Society for Vascular Surgery** recommended screening for men 65 to 75 years of age who are potential candidates for surgery, and not to screen women older than 65 years of age on a population basis, but to individualize screening for women with multiple risk factors, i.e. smoking, cerebrovascular disease and family history (**Mastracci and Cinà, 2007**).

MANAGEMENT

Management options for patients with an asymptomatic AAA include observation with follow-up, medical therapy, surgery and endovascular stenting.

Medical therapy: Medical therapy may be helpful in patients with small- to medium-sized aneurysms that are not surgically treated (Isselbacher, 2005).

Cessation of smoking: Smoking has been found to be a major risk factor for aneurysm formation, growth and rupture (Lederle et al., 2000 and Powell & Greenhalgh, 2003). One study estimated that continued smoking increases the rate of aneurysm growth by 20% to 25% (Powell and Greenhalgh, 2003). The guidelines issued by the ACC/AHA in 2005 recommended that smoking cessation should be advocated to all individuals with AAA or a positive family history of AAA, and offered cessation interventions (Hirsch et al., 2006).

Beta-blockers: Although the data regarding therapeutic benefit of beta-blockers in management of AAA are limited, beta-blockers have been shown to significantly reduce the expansion rate of AAA when monitored by serial USG examination

(Gadowski et al., 1994). The 2005 ACC/ AHA guidelines recommended beta-blocker therapy in patients with an AAA who do not undergo surgery. Because of the possible attenuation of aneurysm expansion, beta-blockers are also a preferred drug for patients with hypertension or angina with care taken in patients with atrio-ventricular blocks, bradycardia, chronic obstructive pulmonary disease and peripheral vascular disease **(Hirsch et al., 2006).**

Antibiotic therapy: Interest in antibiotic therapy in the management of AAA is based on evidences of chronic inflammation in AAA, inhibition of proteases and inflammation by antibiotics, and possible involvement of *Chlamydia pneumoniae* in the pathogenesis of AAA. A study evaluating the role of antibiotics in the management of AAA found a reduction in the mean annual expansion rate of the aneurysms among patients receiving an antibiotic (roxithromycin) compared with those receiving placebo therapy (Vammen et al., 2001). Also, long-term use of antibiotics has been associated with an increased risk for breast cancer. With uncertain benefits and known harms, more reassuring data are needed before this approach can be recommended **(Veliceret al., 2004).**

Risk factor reduction: The beneficial role of treating cardiovascular risk factors, such as hypertension and dyslipidemia, in aneurysm formation, growth or rupture are not clear. However, these approaches may prolong survival by their effect on cardiac and cerebrovascular disease. Long-term statin use has been found to reduce all-cause mortality in patients who underwent previous successful surgical repair of an

AAA **(Kertaiet al., 2004)**. The 2005 ACC/AHA guidelines recommended that patients with AAAs should have their blood pressure and lipids controlled as recommended for patients with atherosclerotic disease (Hirsch et al., 2006).A retrospective study concluded that statins may also be of therapeutic benefit in patients who are treated medically, reducing mortality and possibly slowing growth of the aneurysm **(Kertaiet al., 2004).**

Surgery or waiting: The decision to perform elective surgery to prevent aneurysm rupture is a tricky one. Appropriate patient selection and timing for repair of the aneurysm is based on identifying individuals at the greatest risk of aneurysm rupture. Patients undergoing surgical management have immediate perioperative risks, which must be thoughtfully weighed against the low likelihood of rupture before death from other causes **(Welch et al., 1996).** Once rupture occurs, emergency repair is indicated, but mortality is extremely high. The case fatality rate when emergency surgery is performed for ruptured aortic aneurysm, in patients who survive long enough to reach the hospital, is 50% compared with just 1% to 5% (depending on comorbidities and type of repair) when elective repair is performed **(Schermerhorn, 2009).**

For aneurysms between 4 cm and 5.5 cm, a few studies concluded that the likelihood of eventual surgical requirement is 60% to 65% at five years, and 70% to 75% at the end of eight years **(United Kingdom Small Aneurysm Trial Participants, 2002)**. A review indicated that there was no significant

difference in all-cause mortality between open repair and imaging surveillance at the end of five to eight years in these patients (**Lederleet al., 2007**). The 2005 ACC/AHA guidelines recommended surgical repair of AAAs 5.5 cm in diameter or greater in asymptomatic patients. Patients with symptomatic aneurysms and whose aneurysms increase in diameter by 0.5 cm or greater in six months should also undergo repair, regardless of aneurysm diameter (**Hirsch et al., 2006**).

However, for asymptomatic patients with aneurysms between 4.0 cm and 5.5 cm in diameter, the frequency of surveillance can be challenging to evaluate. For patients not treated surgically, regular imaging surveillance is necessary. The 2005 ACC/AHA guidelines recommended that aneurysms 3.0 cm to 4.0 cm in diameter should be monitored by USG every two to three years, and those with a diameter ranging from 4.0 cm to 5.4 cm should be monitored by USG or CT every six to 12 months (**Hirsch et al., 2006**).

Surgical repair versus endovascular repair: The options for repair include surgical repair (including the transabdominal route or the retroperitoneal route)or endovascular repair, which involves insertion of an endograft into the lumen that effectively excludes the aneurysm from blood flow, minimizing the risk of rupture (**Mitchell et al., 1995**).

Endovascular repair of an AAA is a less invasive and less expensive alternative to open surgical repair. The short-term technical success rate for endovascular aneurysm repair ranges from 83% to more than 95% (**Elkouri et al., 2003**). Thirty-day mortality after elective surgical repair

in major randomized trials ranges from 2.7% to 5.8% (**Lederle et al., 2009**), and is influenced by the volume of procedures performed at the hospital and expertise of the surgeon (**Dimick et al., 2003**). The short-term morbidity and mortality rates of endovascular therapy were found to be better than those of open surgical repair in many trials (**Greenhalgh et al., 2004**). A review (**Lederle et al., 2007**) concluded that the 30-day all-cause mortality was significantly lower with endovascular repair compared with surgical repair (1.6% versus 4.8%). For patients who are at high risk for surgery, the short-term mortality rate is significantly lower with endovascular repair (**Teufelsbauer et al., 2001**).

Other benefits of endovascular repair are reduced hospital stay, shorter recovery time and return to baseline functional capacity, and less blood loss. However, studies have failed to show long-term benefit of the endovascular approach over surgical repair at the end of one to two years (**Lederle, 2004**). Possible explanations for the apparently greater risk of late mortality with endovascular repair in these trials include chance, precipitation of death with open repair in high-risk patients who are more likely to die in the first year with endovascular repair, and failure to prevent aneurysm rupture with endovascular repair (**Lederle, 2005**).

The 2005 ACC/AHA guidelines recommended that open surgical repair should be performed in patients at low or average risk of operative complications. They also suggested endovascular repair in patients who are at high risk of complications from open surgical repair and recommended consideration of

endovascular repair in patients who are not at high surgical risk, although evidence of benefit is not well established in this group of patients (**Hirsch et al., 2006**). The size of the AAA following endovascular repair should be followed with multislice CT angiography. Abdominal USG has shown mixed success rates for the detection of complications and is, therefore, not recommended for routine follow-up (**Powell and Greenhalgh 2003**).

COMPLICATIONS

Patients with AAAs are likely to have underlying cardiovascular and pulmonary disease. Studies revealed that the most common nontechnical complications of AAA repair are related to the preoperative cardiac and pulmonary status of the patient. Patients with pre-existing coronary artery disease and chronic obstructive pulmonary disease have significantly increased morbidity after elective surgical repair and, therefore, a careful preoperative assessment is mandatory in these patients to minimize perioperative complications (**Mitchell et al., 1995**).

Complications that have been reported with endograft use include vascular injury during deployment (sometimes leading to aneurysm rupture), inadequate fixation or sealing of the graft to the vessel wall, stent frame fractures and separations, and breakdown of the graft material (**Hallett et al., 1997**). Other long-term disadvantages of endovascular repair include complications such as endoleaks, graft migration/kinking, spontaneous thrombosis, risk of rupture and need for re-intervention (**Blankensteijn et al., 2005**).

As for the risks associated with open repair, cardiac complications, in the form of either myocardial infarction or arrhythmias, remain the most common morbidity, with an incidence between 2% and 6%. Another significant complication is renal failure or transient renal insufficiency as a result of perioperative hypotension, embolization, inadvertent injury to the ureter, preoperative contrast-induced nephropathy or suprarenal aortic clamping. Although the incidence of renal failure is less than 2% in elective aneurysm repair, it can occur in more than 20% of patients after repair of a ruptured AAA (**Humphreys et al., 2000**).

Ischemic colitis is a devastating potential complication after open repair. The likelihood of such a complication is highest in those with a previous colon resection who underwent repair of a ruptured AAA due to the loss of collateral blood supply to the recto-sigmoid colon. It is estimated that 5% of patients who undergo elective aneurysm repair will develop partial-thickness ischemic colitis without significant clinical sequelae. However, if the partial-thickness ischemia progresses to full-thickness gangrene and peritonitis, mortality can be as high as 90% (**Hausegger et al., 2001**).

The incidence of prosthetic graft infection ranges between 1% and 4% after open repair. It is more common in those who undergo repair of a ruptured AAA. If the prosthetic graft is not fully covered by the aneurysm sac or retro-peritoneum, intestinal adhesion with subsequent bowel erosion may occur, resulting in an aorto-enteric fistula. The predominant sign of such a complication is massive hematemesis, and it typically occurs years after

the operation. Despite these potential complications, however, the majority of patients who undergo successful elective open repair have an uneventful recovery(**Hausegger et al., 2001**).

CONCLUSION

AAAs are mostly asymptomatic and found incidentally. The incidence of AAA is higher in Caucasian men, individuals older than 60 years of age and smokers. Diagnosis is usually reached using imaging modalities. Aneurysm rupture is a medical emergency and risk of aneurysm rupture increases with increasing diameter, rapid expansion, symptomatic aneurysm and history of smoking. Surgical intervention is recommended for all symptomatic aneurysms and asymptomatic aneurysms greater than 5.5 cm in diameter. Regular surveillance through imaging studies should be conducted in asymptomatic aneurysms 3 cm to 5.5 cm in size. Medical management with beta-blockers, cessation of smoking and management of risk factors, such as dyslipidemia and hypertension, may be helpful in patients with small- to medium-sized aneurysms that are not treated surgically.

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تمدد الشريان الأبهري البطني: دراسة شاملة

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تمدد الأوعية الدموية في الشرايين يتم تعريفه علي أنه تمدد موضعي للوعاء الدموي نسبة للشريان الأصلي. وتمدد الشريان الأورطي البطني يعرف بزيادة قطر الشريان الأبهر مرة ونصف على الأقل عند مستوى الشرايين الكلوية، وهو ما يقرب من 2,0 سم. وهكذا، وبشكل عام، أي جزء من الشريان الأورطي البطني يبلغ قطره أكبر من 3,0 سم يشخص علي أنه تمدد الأوعية الدموية الأبهري.

ما يقرب من 80% من تمدد الشريان الأبهري يحدث بين الشرايين الكلوية والتشعب الأبهري. ويشكل تمدد الشريان الأورطى السبب الرابع عشر للوفاة في الولايات المتحدة الأمريكية. وتمزق الشريان الأورطي البطني المتمدد يسبب حوالي 4500 حالة وفاة في الولايات المتحدة الأمريكية سنويا، بالإضافة إلي 1400 حالة وفاة ناجمة عن إجراءات إصلاح لـ45000 حالة في محاولة لمنع تمزق الشريان الأورطي البطني المتمدد.