Abdominal aortic aneurysm: A comprehensive review

Sourabh Aggarwal MBBS[1], Arman Qamar MBBS[2], Vishal Sharma MD[1], Alka Sharma MD[1]

S Aggarwal, A Qamar, V Sharma, A Sharma. Abdominal aortic aneurysm: A comprehensive review. Exp Clin Cardiol 2011;16(1): 11-15.

An arterial aneurysm is defined as a focal dilation of a blood vessel with respect to the original artery. The risk of abdominal aortic aneurysms (AAAs) increases dramatically in the presence of the following factors: age older than 60 years, smoking, hypertension and Caucasian ethnicity. The likelihood that an aneurysm will rupture is influenced by the aneurysm size, expansion rate, continued smoking and persistent hypertension. The majority of AAAs are asymptomatic and are detected as an incidental finding on ultrasonography, abdominal computed tomography or magnetic resonance imaging performed for other purposes. It can also present with abdominal pain or complications such as thrombosis, embolization and rupture. Approximately 30% of asymptomatic AAAs are discovered as a

pulsatile abdominal mass on routine physical examination. Abdominal ultrasonography is considered the screening modality of choice for detecting AAAs because of its high sensitivity and specificity, as well as its safety and relatively lower cost. The decision to screen for AAAs is challenging. The United States Preventive Services Task Force recommended that men between the age of 65 to 75 years who have ever smoked should be screened at least once for AAAs by abdominal ultrasonography. Management options for patients with an asymptomatic AAA include reduction of risk factors such as smoking, hypertension and dyslipidemia; medical therapy with beta-blockers; watchful waiting; endovascular stenting; and surgical repair depending on the size and expansion rate of the aneurysm and underlying comorbidities.

Key Words: *Abdominal aortic aneurysm; Complications; Diagnosis; Risk factor; Treatment*

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 (1,2). 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 (3). 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 (4).

RISK FACTORS

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

Age, sex and ethnicity

The risk of AAAs increases dramatically after 60 years of age (5,6). 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 (5,6). AAAs are four to six times more common in men than in women (7,8). In addition, AAAs develop in women approximately 10 years later than in men (9). In one study (10), AAAs were found to occur more frequently in white people than in black people.

Smoking

Smoking has been found to be a major risk factor for aneurysm formation (1). A study (10) found smoking to be the risk factor most strongly associated with AAA. 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 (11).

Other predisposing factors

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 (12,13). Hypertension has also been found to be associated with AAA (11). A positive family history is another potential factor that significantly increases the risk of AAA (14). A family history of surgical intervention for an AAA in a first-degree relative may increase the risk fourfold (15). AAA has been found to be less common in patients with diabetes (10).

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

Aneurysm size is one of the strongest predictors of the risk of rupture, with risk increasing markedly at aneurysm diameters of 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 (16-19). A statement from the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery (20) estimated the annual rupture risk according to AAA diameter to be the following:

- 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%

Expansion rate
The expansion rate may also be an important determinant of the risk of rupture (21,22). 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 (1). Growth tends to be more rapid in smokers, and less rapid in patients with diabetes mellitus or peripheral vascular disease (23).

Other factors
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 (20).

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 (24,25).

Ruptured aneurysm
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 (26). 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 (27).

Inflammatory aneurysm
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 (1).

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 aortocaval 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 (28). 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 (29).

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 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 (30,31). USG has excellent test characteristics for diagnosing and following an AAA. 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 (32).

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 (33). Although USG is generally preferred, multislice CT angiography can be used for serial monitoring of aneurysm size. 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 (33). Magnetic resonance angiography is probably more accurate than CT, but is more expensive and not universally available (34).

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 labelling (35). This results in unnecessary psychological distress to patients, which manifests as lower quality of life scores (36), and transiently mildly higher anxiety scores and lower self-rated perception of health (37). 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 (14) concluded that screening for AAA significantly reduces the risk of AAA-related mortality by approximately 50% in men. Another study (38) 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. However, no significant benefit to AAA-related mortality or all-cause mortality was achieved in a study that involved population-based screening in women (7). A model study (14) that involved only men between 65 to 74 years of age with a history of smoking reported an 89% anticipated reduction in AAA-related mortality.

Various guidelines have been issued regarding screening for AAA. The United States Preventive Services Task Force (USPSTF) makes the following recommendations (31):

- 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 (1) regarding screening of patients for AAA recommended that men 60 years of age or older who 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.

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 (smoking, cerebrovascular disease and family history) (39).

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 (33).

Cessation of smoking

Smoking has been found to be a major risk factor for aneurysm formation, growth and rupture (6,10). One study (6) estimated that continued smoking increases the rate of aneurysm growth by 20% to 25%. The guidelines issued by the ACC/AHA in 2005 (1) recommended that smoking cessation should be advocated to all individuals with AAA or a positive family history of AAA, and offered cessation interventions.

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 (21). The 2005 ACC/AHA guidelines (1) 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 atrioventricular blocks, bradycardia, chronic obstructive pulmonary disease and peripheral vascular disease.

Antibiotic therapy

Interest in antibiotic therapy in the management of AAA is based on evidence 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 (40) 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. Also, long-term use of antibiotics has been associated with an increased risk for breast cancer (41). With uncertain benefits and known harms, more reassuring data are needed before this approach can be recommended.

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 (42). The 2005 ACC/AHA guidelines (1) recommended that patients with AAAs should have their blood pressure and lipids controlled as recommended for patients with atherosclerotic disease. A retrospective study (43) concluded that statins may also be of therapeutic benefit in patients who are treated medically, reducing mortality and possibly slowing growth of the aneurysm.

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 (44). 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 (26).

For aneurysms between 4 cm and 5.5 cm, a few studies (19,45) 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. A review (46) 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. The 2005 ACC/AHA guidelines (1) 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.

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 (1) 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.

Surgical repair versus endovascular repair

The options for repair include surgical repair (including the transabdominal route or the retroperitoneal route) (47) 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.

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% (48-50). Thirty-day mortality after elective surgical repair in major randomized trials ranges from 2.7% to 5.8% (45,51,52) and is influenced by the volume of procedures performed at the hospital and expertise of the surgeon (53,54). The short-term morbidity and mortality rates of endovascular therapy were found to be better than those of open surgical repair in many trials (55,56). A review (46) 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 (57).

Other benefits of endovascular repair are reduced hospital stay, shorter recovery time and return to baseline functional capacity, and less blood loss. However, studies (58) have failed to show long-term benefit of the endovascular approach over surgical repair at the end of one to two years. 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 (59).

The 2005 ACC/AHA guidelines (1) 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. 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 (6).

COMPLICATIONS

Patients with AAAs are likely to have underlying cardiovascular and pulmonary disease. Studies (47) revealed that the most common non-technical 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.

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 (6,60). 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 (61,62).

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% (63). 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 neph-ropathy 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 (63).

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 rectosigmoid colon. It is estimated that 5% of patients who undergo elective aneurysm repair will develop partial-thickness ischemic colitis without significant clinical sequelae (64). However, if the partial-thickness ischemia progresses to full-thickness gangrene and peritonitis, mortality can be as high as 90% (64).

The incidence of prosthetic graft infection ranges between 1% and 4% after open repair (64). 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 retroperitoneum, intestinal adhesion with subsequent bowel erosion may occur, resulting in an aortoen-teric 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.

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 dyslipi-demia and hypertension, may be helpful in patients with small- to medium-sized aneurysms that are not treated surgically.

REFERENCES

1. Hirsch AT, Haskal ZJ, Hertzer NR, et al. ACC/AHA 2005 Practice Guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): A collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease): Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; Trans-Atlantic Inter-Society Consensus; and Vascular Disease Foundation. Circulation 2006;113:e463-e654.
2. Ouriel K, Green RM, Donayre C, et al. An evaluation of new methods of expressing aortic aneurysm size: Relationship to rupture. J Vasc Surg 1992;15:12-8.
3. Silverberg E, Boring CC, Squires TS. Cancer statistics, 1990. CA Cancer J Clin 1990;40:9-26.
4. McPhee JT, Hill JS, Eslami MH. The impact of gender on presentation, therapy, and mortality of abdominal aortic aneurysm in the United States, 2001-2004. J Vasc Surg 2007;45:891-9.
5. Singh K, Bonaa KH, Jacobsen BK, et al. Prevalence of and risk factors for abdominal aortic aneurysms in a population-based study: The Tromso Study. Am J Epidemiol 2001;154:236-44.
6. Powell JT, Greenhalgh RM. Clinical practice. Small abdominal aortic aneurysms. N Engl J Med 2003;348:1895-901.
7. Scott RA, Wilson NM, Ashton HA, et al. Influence of screening on the incidence of ruptured abdominal aortic aneurysm: 5-year results of a randomized controlled study. Br J Surg 1995;82:1066-70.
8. Lederle FA, Johnson GR, Wilson SE. Aneurysm Detection and Management Veterans Affairs Cooperative Study. Abdominal aortic aneurysm in women. J Vasc Surg 2001;34:122-6.
9. McFarlane MJ. The epidemiologic necropsy for abdominal aortic aneurysm. JAMA 1991;265:2085-8.
10. Lederle FA, Johnson GR, Wilson SE, et al. The aneurysm detection and management study screening program: Validation cohort and final results. Aneurysm Detection and Management Veterans Affairs Cooperative Study Investigators. Arch Intern Med 2000;160:1425-30.

11. Lederle FA, Johnson GR, Wilson SE, et al. Prevalence and associations of abdominal aortic aneurysm detected through screening. Ann Intern Med 1997;126:441-9.
12. Bengtsson H, Ekberg O, Aspelin P, et al. Ultrasound screening of the abdominal aorta in patients with intermittent claudication. Eur J Vasc Surg 1989;3:497-502.
13. Cabellon S Jr, Moncrief CL, Pierre DR, et al. Incidence of abdominal aortic aneurysms in patients with atheromatous arterial disease. Am J Surg 1983;146:575-6.
14. Fleming C, Whitlock EP, Beil TL, et al. Screening for abdominal aortic aneurysm: A best-evidence systematic review for the US Preventive Services Task Force. Ann Intern Med 2005;142:203-11.
15. Salo JA, Soisalon-Soininen S, Bondestam S, et al. Familial occurrence of abdominal aortic aneurysm. Ann Intern Med 1999;130:637-42.
16. Nevitt MP, Ballard DJ, Hallett JW Jr. Prognosis of abdominal aortic aneurysms. A population-based study. N Engl J Med 1989;321:1009-14.
17. Lederle FA, Johnson GR, Wilson SE, et al. Rupture rate of large abdominal aortic aneurysms in patients refusing or unfit for elective repair. JAMA 2002;287:2968-72.
18. Johansson G, Nydahl S, Olofsson P, et al. Survival in patients with abdominal aortic aneurysms. Comparison between operative and nonoperative management. Eur J Vasc Surg 1990;4:497-502.
19. Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. The UK Small Aneurysm Trial Participants. Lancet 1998;352:1649-55.
20. 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;37:1106-17.
21. Gadowski GR, Pilcher DB, Ricci MA. Abdominal aortic aneurysm expansion rate: Effect of size and beta-adrenergic blockade. J Vasc Surg 1994;19:727-31.
22. Bengtsson H, Bergqvist D, Ekberg O, Ranstam J. Expansion pattern and risk of rupture of abdominal aortic aneurysms that were not operated on. Eur J Surg 1993;159:461-7.

23. Brady AR, Thompson SG, Fowkes FG, Greenhalgh RM, Powell JT; UK Small Aneurysm Trial Participants. Abdominal aortic aneurysm expansion: Risk factors and time intervals for surveillance. Circulation 2004;110:16-21.

24. Aboulafia DM, Aboulafia ED. Aortic aneurysm-induced disseminated intravascular coagulation. Ann Vasc Surg 1996;10:396-405.

25. Fisher DF Jr, Yawn DH, Crawford ES. Preoperative disseminated intravascular coagulation associated with aortic aneurysms. A prospective study of 76 cases. Arch Surg 1983;118:1252-5.

26. Schermerhorn M. A 66-year-old man with an abdominal aortic aneurysm: Review of screening and treatment. JAMA 2009;302:2015-22.

27. Harris LM, Faggioli GL, Fiedler R, Curl GR, Ricotta JJ. Ruptured abdominal aortic aneurysms: Factors affecting mortality rates. J Vasc Surg 1991;14:812-8.

28. Chervu A, Clagett GP, Valentine RJ, Myers SI, Rossi PJ. Role of physical examination in detection of abdominal aortic aneurysms. Surgery 1995;117:454-7.

29. US Preventive Services Task Force. Guide to Clinical Preventive Services, 2nd edn. Baltimore: Williams & Wilkins, 1996:67.

30. LaRoy LL, Cormier PJ, Matalon TA, Patel SK, Turner DA, Silver B. Imaging of abdominal aortic aneurysms. AJR Am J Roentgenol 1989;152:785-92.

31. US Preventive Services Task Force. Screening for abdominal aortic aneurysm: Recommendation statement. Ann Intern Med 2005;142:198-202.

32. Scott RA, Ashton HA, Kay DN. Abdominal aortic aneurysm in 4237 screened patients: Prevalence, development and management over 6 years. Br J Surg 1991;78:1122-5.

33. Isselbacher EM. Thoracic and abdominal aortic aneurysms. Circulation 2005;111:816-28.

34. Petersen MJ, Cambria RP, Kaufman JA, et al. Magnetic resonance angiography in the preoperative evaluation of abdominal aortic aneurysms. J Vasc Surg 1995;21:891-8.

35. Melton LJ III, Bickerstaff LK, Hollier LH, et al. Changing incidence of abdominal aortic aneurysms: A population based study. Am J Epidemiol 1984;120:379-86.

36. Lindholt JS, Vammen S, Fasting H, Henneberg EW. Psychological consequences of screening for abdominal aortic aneurysm and conservative treatment of small abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2000;20:79-83.

37. Ashton HA, Buxton MJ, Day NE, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: A randomised controlled trial. Lancet 2002;360:1531-9.

38. Norman PE, Jamrozik K, Lawrence-Brown MM, et al. Population based randomised controlled trial on impact of screening on mortality from abdominal aortic aneurysm. BMJ 2004;329:1259.

39. Mastracci TM, Cinà CS; Canadian Society for Vascular Surgery. Screening for abdominal aortic aneurysm in Canada: Review and position statement of the Canadian Society for Vascular Surgery. J Vasc Surg 2007;45:1268-76.

40. Vammen S, Lindholt JS, Ostergaard L, Fasting H, Henneberg EW. Randomized double-blind controlled trial of roxithromycin for prevention of abdominal aortic aneurysm expansion. Br J Surg 2001;88:1066-72.

41. Velicer CM, Heckbert SR, Lampe JW, et al. Antibiotic use in relation to the risk of breast cancer. JAMA 2004;291:827-35.

42. Kertai MD, Boersma E, Westerhout CM, et al. Association between long-term statin use and mortality after successful abdominal aortic aneurysm surgery. Am J Med 2004;116:96-103.

43. Sukhija R, Aronow WS, Sandhu R, Kakar P, Babu S. Mortality and size of abdominal aortic aneurysm at long-term follow-up of patients not treated surgically and treated with and without statins. Am J Cardiol 2006;97:279-80.

44. Welch HG, Albertsen PC, Nease RF, Bubolz TA, Wasson JH. Estimating treatment benefits for the elderly: The effect of competing risks. Ann Intern Med 1996;124:577-84.

45. United Kingdom Small Aneurysm Trial Participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1445-52.

46. Lederle FA, Kane RL, MacDonald R, Wilt TJ. Systematic review: Repair of unruptured abdominal aortic aneurysm. Ann Intern Med 2007;146:735-41.

47. Mitchell MB, Rutherford RB, Krupski WC. Infrarenal aortic aneurysms. In: Rutherford RB, ed. Vascular Surgery. Philadelphia: WB Saunders, 1995:1032.

48. Blum U, Voshage G, Lammer J, et al. Endoluminal stent-grafts for infrarenal abdominal aortic aneurysms. N Engl J Med 1997;336:13-20.

49. Criado FJ, Wilson EP, Fairman RM, Abul-Khoudoud O, Wellons E. Update on the Talent aortic stent-graft: A preliminary report from United States phase I and II trials. J Vasc Surg 2001;33:S146-S149.

50. Elkouri S, Gloviczki P, McKusick MA, et al. Endovascular repair of abdominal aortic aneurysms: Initial experience with 100 consecutive patients. Mayo Clin Proc 2003;78:1234-42.

51. Lederle FA, Wilson SE, Johnson GR, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1437-44.

52. Lederle FA, Freischlag JA, Kyriakides TC, et al. Outcomes following endovascular vs open repair of abdominal aortic aneurysm: A randomized trial. JAMA 2009;302:1535-42.

53. Birkmeyer JD, Siewers AE, Finlayson EV, et al. Hospital volume and surgical mortality in the United States. N Engl J Med 2002;346:1128-37.

54. Dimick JB, Cowan JA Jr, Stanley JC, Henke PK, Pronovost PJ, Upchurch GR Jr. Surgeon specialty and provider volumes are related to outcome of intact abdominal aortic aneurysm repair in the United States. J Vasc Surg 2003;38:739-44.

55. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG; EVAR trial participants. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: Randomised controlled trial. Lancet 2004;364:843-8.

56. Prinssen M, Verhoeven EL, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med 2004;351:1607-18.

57. Teufelsbauer H, Prusa AM, Wolff K, et al. Endovascular stent grafting versus open surgical operation in patients with infrarenal aortic aneurysms: A propensity score-adjusted analysis. Circulation 2002;106:782-7.

58. Lederle FA. Abdominal aortic aneurysm – open versus endovascular repair. N Engl J Med 2004;351:1677-9.

59. Lederle FA. Endovascular repair of abdominal aortic aneurysm – round two. N Engl J Med 2005;352:2443-5.

60. Hallett JW Jr, Marshall DM, Petterson TM, et al. Graft-related complications after abdominal aortic aneurysm repair: Reassurance from a 36 year population based experience. J Vasc Surg 1997;25:277-84.

61. Laheij RJ, Buth J, Harris PL, Moll FL, Stelter WJ, Verhoeven EL. Need for secondary interventions after endovascular repair of abdominal aortic aneurysms. Intermediate-term follow-up results of a European collaborative registry (EUROSTAR). Br J Surg 2000;87:1666-73.

62. Blankensteijn JD, de Jong SE, Prinssen M, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med 2005;352:2398-405.

63. Humphreys WV, Byrne J, James W. Elective abdominal aortic aneurysm operations – the results of a single surgeon series of 243 consecutive operations from a district general hospital. Ann R Coll Surg Engl 2000;82:64-8.

64. Hausegger KA, Schedlbauer P, Deutschmann HA, Tiesenhausen K. Complications in endoluminal repair of abdominal aortic aneurysms. Eur J Radiol 2001;39:22-33.