REVIEW ARTICLE

Aortic Aneurysms: Clinical Guidelines for Primary Care Physicians

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Aortic aneurysms (AA) are permanent, localized abnormal dilation of the wall of the aorta, the largest artery in the body, occurring as a result of medial degeneration of the arterial wall, generally, as a result of increased aortic hypertension or genetic predisposition. Risk factors for AAs are similar to those of other cardiovascular diseases. Tobacco use is strongly associated with aneurysm formation and dilation, and patients diagnosed with AA should be advised to stop smoking. An abdominal aorta with a diameter greater than 3.00 cm is generally considered aneurysmal. By convention, a thoracic aorta with a diameter greater than 4.50 cm is generally considered aneurysmal. No specific laboratory tests exist to diagnose an AA, and testing should be ordered supplementary to imaging studies. Dedicated imaging studies offer definitive identification or exclusion of potential AAs, but the imaging modality used is largely dependent upon patient-related factors. Patients with small aneurysms may be candidates for medical management, however, any patient with an aortic diameter greater than 5.00 - 5.50 cm should be referred for immediate surgical consultation. With the majority of AAs asymptomatic prior to rupture, it is important that primary care physicians understand how to properly evaluate and diagnose patients at risk for developing an AA as well as the short and long-term management of patients diagnosed with an AA.

INTRODUCTION

An aortic aneurysm (AA) is a permanent, localized abnormal dilation of more than 50% of the normal diameter of the wall of the aorta, the largest artery in the body.[1,2] Aortic aneurysms develop as a result of medial degeneration of the arterial wall, and proteolytic degradation of the associated elastic tissues.[3] Degeneration of the arterial architecture coupled with hypertensive hemodynamic stress results in aneurysmal dilation.[3,4] While the etiology of an AA is not fully understood, risk factors for an AA are similar to those of other cardiovascular diseases.[2] Risk factors for the development of an AA include age greater than 65 years, male sex, trauma, hypertension, hypercholesterolemia, atherosclerosis, tobacco use, and a familial history of vascular aneurysm.[3,5,6,7] In the United States, AA, and dissection (I71, ICD-10, excluding aortic ectasia, syphilitic aortic aneurysm, traumatic aortic aneurysm) were the primary causes of 201,985 deaths in the United States between 1999 and 2014 (50,010 between 2010 and 2014); cases were predominantly male (59%), white (91.6%), and age older than 65 years (80%).[8] Patients with an AA are at increased risk of dissection, pulmonary/arterial embolism, stroke, and myocardial infarction.[9] With the majority of AAs asymptomatic prior to rupture, it is important that primary care physicians understand how to properly evaluate and diagnose patients at risk for developing an AA as well as the short and long-term management of patients diagnosed with an AA.

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PREVENTION

Primary prevention or risk factor reduction for AAs is aimed at counseling the patient on health-related behaviors, such as improved diet and smoking cessation. The Dietary Approaches to Stop Hypertension (DASH), a diet rich in fruits and vegetables with reduced saturated and total fat, is a recognized dietary recommendation effective in reducing blood pressure, and with higher adherence being associated with cardiovascular risk factor reduction.[10] Further, adherence to a Mediterranean diet, a diet rich in fruits and vegetables that is supplemented with olive oil or nuts, is also a well-established protective factor against cardiovascular disease.[11] Dietary sodium restriction is strongly advocated for the prevention and treatment of hypertension, however, despite the abundance of studies on its efficacy, current evidence suggests a J-shaped association between sodium intake and cardiovascular disease.[12] Tobacco use is a significant risk factor for AA with one trial demonstrating population attributable risk at or above 47%, and another estimating that smoking accounted for 75% of all abdominal aortic aneurysms (AAA) greater than 4.0 cm in diameter; therefore, effective strategies for smoking cessation should be advocated.[13,14]

SCREENING

Guidelines

The United States Preventive Services Task Force (USPSTF) recommends one-time screening for AAA with ultrasonography in men ages 65 to 75 years who have ever smoked, and that clinicians selectively offer to screen for AAA in men ages 65 to 75 years who

have never smoked. Currently, the USPSTF concludes that current evidence is insufficient to assess risks and benefits of screening women ages 65 to 75 who have ever smoked, and does not recommend screening women who have never smoked.[6] Recurrent screening in men age 75 or older does not appear advantageous.[15] Although the USPSTF does not provide recommendations for one-time screening for thoracic aortic aneurysm (TAA) the application of AAA guidelines may be beneficial. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines issued in 2005 recommend that men who are 65 to 75 years of age who have ever smoked should undergo a physical examination and one-time ultrasound screening for detection of AAAs, further, men 60 years of age or older who are either the siblings or offspring of patients with AAAs should also undergo physical examination and ultrasound screening for detection of aortic aneurysms.[16] The European Society of Cardiology recommends screening in all men greater than 65 years and considered in women greater than 65 years and tobacco smoking.[17]

Reimbursement

Although coverages may vary by insurer, physicians submitting claims for reimbursement for an ultrasound of the abdominal aorta will use CPT code G0389 when ordered during an initial preventive physical examination (IPPE). Eligible beneficiaries are those who: have received a referral for an ultrasound screening as a result of an IPPE, have not been previously furnished a covered AAA screening ultrasound examination under the Medicare program; and/or has a familial history of AAA or is a man age 65 to 75 who has smoked at least 100 cigarettes in his lifetime. If ordered during a visit that is not an IPPE, physicians will use CPT code 76775. Physicians submitting claims for payment by insurers will use ICD-10 code Z13.6, Z82.49 and Z87.891. Specific coverage for the screening of TAAs has not been established, and will vary by the insurer (See Summary: Table 1).

TABLE 1: Summary of CPT and ICD-10 codes of reimbursement

CODE	DESCRIPTION OF CODE
76000	Ultrasound, abdominal, real time with image documentation; complete
76705	Ultrasound, abdominal, real time with image documentation; limited (eg, single organ, quadrant, follow-up)
76770	Ultrasound, retroperitoneal (eg, renal, aorta, notes), real time with image documentation; complete
76775	Ultrasound, retroperitoneal, (eg, renal, aorta, nodes), real time with image documentation; limited
G0389	Ultrasound B-scan and/or real time image documentation; for abdominal aortic aneurysm (AAA) screening
Z13.6	Encounter for screening for cardiovascular disorders
Z82.49	Family history of ischemic heart disease and other diseases of the circulatory system
Z87.891	Personal history of nicotine dependence

DIAGNOSIS

Physical Examination

Detection of an AAA is difficult given that the majority are asymptomatic, and diagnoses made are often an incidental finding on radiographic studies. Physical examination is limited and associated with a large population of false-negative and false-positive findings.[3,4,9] Abdominal palpation has only moderate sensitivity for detecting AAAs with one study demonstrating 68% sensitivity and 75% specificity.[3,18] Abdominal palpation sensitivity is positively associated with AAAs of increasing diameter.[18] The most common finding in asymptomatic AAAs large enough to warrant intervention is a palpable pulsatile mass extending from the xiphoid to the umbilicus.[3,9] Abdominal palpation sensitivity is often affected by obesity, abdominal distention, and small aneurysm size.[3] Somatic findings correlated with AAAs can be manifested as ecchymotic, erythemic, and edematous tissue abnormalities.

Clinical features of symptomatic AAA include the presence of a Grey-Turner's sign — the extravasation of blood into the subcutaneous tissues producing flank ecchymosis, as consequence of an extensive retroperitoneal hematoma from ruptured AAA.[19] Further, Grey-Turner's sign may be coupled with periumbilical ecchymosis (Cullen's sign), ecchymosis of the proximal thigh (Fox's sign), and discoloration of the scrotum (Bryant's sign).[20,21] Abdominal pain is also common, however, abdominal pain may be associated with acute gastritis, bowel obstruction, ischemic bowel, mild pyelonephritis, emergent pancreatitis, and generalized musculoskeletal pain, and should be considered during the process of forming a differential. In hemodynamically unstable patients, abdominal pain may be symptomatic of appendicitis, diverticulitis, cholelithiasis, perforated peptic ulcer, myocardial infarction, or pulmonary embolism rather than an AAA. Acute limb ischemia can also be present with AAAs as a consequence of a distal thromboembolism originating from atherothrombotic debris from the AAA.[22] Pain varies with diameter and location of the aneurysm, and whether or not the aneurysm has ruptured (free or contained).[21] Anterior intraperitoneal rupture presents as sudden severe abdominal or back pain and collapse. The subsequent hemorrhaging often results in exsanguination and death.[21] Posterior retroperitoneal rupture also often manifests as back pain with or without associated abdominal pain and hypotension, however, the rupture is often contained allowing time for treatment.[21] Vertebral erosion may be resultant of a chronic contained rupture of an AAA presenting as chronic back pain.23 Auscultation of the aortic and femoral arteries may reveal the presence of bruits although their absence does not exclude the presence of an AAA3[24] *(See summary: Table 2, page 12).*

Like AAAs, TAAs are typically asymptomatic with diagnoses made incidentally on radiographic or electrocardiographic studies and are not easily detectable until a catastrophic complication occurs (i.e., a dissection or rupture).[25] Other symptoms include vocal cord palsy presenting as hoarseness resultant of vagus, or recurrent laryngeal nerve compression.[26] TAA compression of the trachea may cause a significant deviation, wheezing, dyspnea, or tussis.[9] Hemoptysis may be a sign of aneurysmal erosion into the trachea.[27] Dysphagia or hematemesis may be caused by esophageal compression or aortoesophageal fistula.[28] Dilation of the aortic root can mimic symptoms of congestive heart failure (CHF) due to aortic insufficiency.[9] Chest pain may be associated with acute aortic dissection,

TABLE 2:

Table 2A: Summary of risk factors, symptoms, and differential diagnoses for abdominal aortic aneurysm	
Abdominal Aortic Aneurysm	
Risk Factors	Age greater than 65 years male sex, hypertension, hypercholesterolemia, atherosclerosis, tobacco use, and familial history of vascular anuerysm
Symptoms	Abdominal pain, palpable pulsatile mass, flank ecchymosis (Grey-Turner's sign), periumbilical ecchymosis (Cullen's sign), ecchymosis of the proximal thigh (Fox's sign), discoloration of the scrotum (Bryant's sign), and acute limb ischemia. Bruits in aortic of femoral vessels
Differential Diagnoses	Acute gastritis, bowel obstruction, ischemic bowel, mild pyelonephritis, emergent pancreatitis, and generalized musculo-skeletal pain. Appendicitis, diverticulitis, cholelithiasis, perforated peptic ulcer, myocardial infarction, and pulmonary embolism
Table 2B: Summary of risk factors, symptoms, and differential diagnoses for thoracic aortic aneurysm	
Thoracic Aortic Aneurysm	
Risk Factors	Age greater than 65 years male sex, hypertension, hypercholesterolemia, atherosclerosis, tobacco use, and familial history of vascular anuerysm
Symptoms	Chest pain, vocal cord palsy presenting as hoarseness, tracheal deviation, wheezing, dyspnea, tussis, hemoptysis, dysphagia or hematemesis. Aortic regurgitation or other pathological murmurs.
Differential Diagnoses	Congestive heart failure, acute dissection, acute pericarditis, infective endocarditis, myocardial infarction, pulmonary embolism, and superior vena cava syndrome

acute pericarditis, infective endocarditis, myocardial infarction, pulmonary embolism, and superior vena cava syndrome. Cardiac auscultation for aortic regurgitation and other pathological murmurs may be revealing. Arterial perfusion differentials in both upper and lower extremities, as well as cardiac tamponade, may be evident (See summary: Table 2A and 2B).

Laboratory Studies

No specific laboratory tests exist to diagnose an AA. Laboratory testing should be ordered supplementary to imaging studies and cardio-pulmonary status studies. Blood should be drawn for complete blood count (CBC), prothrombin time/partial thromboplastin time (PT/PTT) with the international normalized ratio (INR) to evaluate for infection or bleeding disorders. Arterial blood gases, liver and kidney function tests, lipid panel, and blood lactate levels may be used to assess respiratory and metabolic status. HbA1c, while not diagnostic for an AA, should be used to test for Type 2 Diabetes Mellitus. Interestingly, some studies have demonstrated an inverse relationship between HbA1c and aneurysm expansion.[29,30] While not conclusive, hematocrit may be lowered in patients with a ruptured aneurysm. Disseminated intravascular coagulopathy (DIC) is a rare complication of an AA. Therefore coagulation studies should be ordered.[31,32]

Imaging Studies

Normal values for the intra-luminal diameter of the aortic root and ascending aorta have been reported as between 3.00 - 3.36 cm for males and 2.90 - 3.11 cm for females at end-systole.[33,34] The suprarenal diameter of the aorta tapers as it descends, and has been reported to be between 2.45 - 2.40 cm and 2.39 - 2.43 cm mid-descending to the diaphragmatic aorta in females and males, respectively.[35] An intra-luminal diameter less than 3.00 cm is considered normal. It is important to note that these measurements are heavily confounded by some factors including age, sex, body size, the location of the measurement, method of measurement, and the robustness of the type of imaging used.[35] By convention, a thoracic aorta with an intra-luminal diameter greater than 4.50 cm is considered aneurysmal.[35,36] The USPSTF defines an abdominal aorta with a diameter greater than 3.00 cm to be aneurysmal.[6] Definitive identification or exclusion of AAs requires dedicated imaging studies. While multiple imaging modalities can be used to evaluate potential AAs, selection of the most appropriate modality may depend upon patient-related factors such as hemodynamic stability, renal insufficiency, and contrast allergies.[35] Transesophageal echocardiography (TEE) and ultrasonography (US) serve as imaging modalities used to screen AAAs and TAAs respectively. US is the primary technology used to screen patients for an AAA. It is a noninvasive and inexpensive modality and offers the benefit of not requiring the use of contrast agent over computed tomography (CT).[9,37] US is suboptimal in obese patients, patients with increased bowel gas, and has increased inter-observer variation.[38] TEE offers considerable advantages in the diagnosis of TAAs. TEE offers high sensitivity/specificity, short duration, and is readily available.[39] Given the proximity of the esophagus to the aorta, TEE permits high-quality images to be obtained without interference from the thoracic wall or lungs.[39] TEE, however, cannot reliably image the distal ascending aorta and the aortic arch and is highly dependent upon an experienced echocardiographer.[40] Although TEE is a relatively invasive procedure requiring esophageal intubation, it is considered to be a safe procedure.[40,41] Cardiovascular contraindications including induced vagal and sympathetic reflexes, non-sustained ventricular and supraventricular tachycardia, atrial fibrillation, 3rd-degree block, angina, and myocardial ischemia should be considered.[41] The ACC/AHA guidelines issued in 2010 recommend that low to intermediate risk patients receive a chest x-ray during screening, as it may either establish an alternative diagnosis or demonstrate findings that are suggestive of a TAA. Plain radiography in patients with AAA may suggest an aneurysm secondary to a paravertebral curvilinear calcification.[42] Plain radiographs in patients with a TAA may suggest an enlarged thoracic aorta secondary to a calcified aortic wall similar to that seen in the atherosclerotic disease. Indirect findings suggestive of TAA include a widened mediastinum, although mediastinal masses may mimic aortic aneurysms; tracheal deviation may also be evident.[43] Ultimately, plain radiography is inadequate to definitively conclude the presence of a TAA or an AAA due to magnification effects, and often poor visualization of the aorta. Magnetic resonance imaging (MRI), CT, and angiography should be used when more definitive imaging is required and should be considered only when weighed with the potential benefits and risks.

Surveillance

The natural history of AAs shows that as aneurysms increase in size, the rate of expansion increases, and the risk of rupture increases (See summary: Table 3A and 3B).[16,44,45,46] For patients found to have AAAs on initial screening, ACC/AHA guidelines issued in 2005 recommend regular surveillance every six months to three years, depending on aneurysm size (See summary: Table 3A and 3B).[16] While no specific recommendations exist for TAAs, one study recommended regular surveillance every one to two years, depending on aneurysm size (See summary: Table 4A and 4B).[47]

TABLE 3:

Table 3A: Summary of expansion rates and risk of rupture for abdominal aortic aneurysm	
Aneurysm Diameter	**Annual Expansion Rate**
3.0 to 3.9 cm	1 to 4 mm
4.0 to 6.0 cm	3 to 5 mm
> 6.0 cm	7 to 8 mm
Aneurysm Diameter	**Annual Risk of Rupture**
< 4.0 cm	< 0.5%
4.0 to 4.9 cm	0.5 to 15%
5.0 to 5.9 cm	3 to 15%
6.0 to 6.9 cm	10 to 20%
7.0 to 7.9 cm	20 to 40%
> 8.0 cm	30 to 50%
Table 3B: Summary of expansion rates and risk of rupture for thoracic aortic aneurysm	
Aneurysm Type	**Annual Expansion Rate**
Aortic arch	5.6 mm
Ascending aorta	0.2 to 2.8 mm
Descending aorta	1.9 to 3.4 mm
Aneurysm Diameter	**Annual Risk of Rupture**
< 4.0 cm	0%
4.0 to 4.9 cm	2%
5.0 to 5.9 cm	3%
> 6.0 cm	7%

MANAGEMENT

Medical

Medical management of small and preoperative AAs generally consists of strict blood pressure control. A goal systolic blood pressure between 100 and 120 mm Hg has been recommended.[48] The AHA strongly recommends "stringent control of hypertension, lipid profile optimization, smoking cessation, and other atherosclerosis risk-reduction measures should be instituted for patients with small aneurysms not requiring surgery, as well as for patients who are not considered surgical or stent graft candidates."[49] Prophylactic beta-adrenergic blocking agent (beta blocker) therapy, may be beneficial in reducing the rate of aortic dilation by reducing the force of myocardial contraction.[43] However, while several animal and clinical studies have indicated a significant effect of beta-blockers on aneurysm growth rate, recent studies have not clearly shown to reduce aneurysm expansion rates.[50] Angiotensin-converting enzyme inhibitors (ACE inhibitors) have been demonstrated to "stimulate and inhibit matrix metalloproteinases (MMPs), and the degradation of extracellular matrix in AAs,"[41] Patients receiving ACE inhibitor therapy were significantly less likely to present with a ruptured aneurysm compared with those who were not receiving therapy.[50,51,52] Statin therapy is likely to be effective in preventing the growth of an AA and reduce the likelihood of adverse events by reducing MMP expression.[50,51,52] Meta analysis of eleven observational comparative studies suggested a significant reduction in AAA growth in patients receiving statin therapy compared to no therapy.[50] Tobacco use is strongly associated with aneurysm formation and dilation, and patients diagnosed with AA should be advised to stop smoking, and be offered smoking cessation interventions.[3,5,6,7,50,51,52]

TABLE 4:

Table 4A: Summary of surveillance intervals for abdominal aortic aneurysm	
Aneurysm Diameter	**Surveillance Interval**
< 3.0 cm	No surveillance
3.0 to 3.9 cm	US every two to three years
4.0 cm to 5.4 cm	US to CT every six to twelve months
>5.4 cm	Referral for surgical consultation
Table 4B: Summary of surveillance intervals for thoracic aortic aneurysm	
Aneurysm Diameter	**Surveillance Interval**
< 3.0 cm	No surveillance
3.0 cm to 3.9 cm	Every two years
> 4.5 cm	Annually
> 5.0 cm	Referral for surgical consultation

Osteopathic Manipulative Medicine

The use of manipulative techniques is contraindicated in patients with an aortic aneurysm due to the increased risk of rupture. However, osteopathic principals can be applied during the physical examination to aid in the diagnosis of an AA. Viscerosomatic reflexes are somatic dysfunction that develops in response to visceral pathology. Irritation, such as with compression from an aneurysm, results in activation of general visceral afferent neurons in surrounding tissues that project to the spinal cord. Prolonged afferent stimulation can stimulate interneurons that synapse with anterior horn motor neurons producing palpable tissue texture changes. Given that an aortic aneurysm can develop anywhere along the length of the aorta, viscerosomatic reflex somatic dysfunction will vary widely.

Referral

Patients for which medical management is refractory, with AAAs that exceed 1 cm of expansion per year or TAAs that exceed 0.5 cm of expansion per year, with symptomatic AA, or with aortic diameters greater than 5.5 cm should be referred for immediate surgical consultation (See Summary: Table 5). If surgical intervention is warranted, there are two main approaches: open and endovascular aneurysm repair. Prophylactic surgical repair is the most effective management to prevent rupture/dissection with 30-day mortality risk at 11.7% in open TAA repair versus 2.1% in EVAR TAA repair.[53] Endovascular repair is less invasive, has decreased morbidity and mortality, and is preferred in patients who are at high risk of complications from open surgical repair.[16] The Immediate Management of the Patient with Ruptured Aneurysm: Open Versus Endovascular repair (IMPROVE) trial reported 30-day mortality risk at 37.4% in open AA repair versus 35.4% in EVAR AAA repair. A Dutch trial reported 30-day mortality risk at 25% and 21% for open AAA and EVAR AAA repair, respectively.[53] The UK EVAR trial suggested that EVAR was associated with a significantly lower operative mortality than open surgical repair, but that these differences were not seen long-term.[54] Conversely, the Open Versus Endovascular Repair (OVER) trial reported that EVAR led to increased long-term survival among younger patients, but not among older patients suggesting that EVAR might be the more appropriate treatment for younger patients.[55] However, despite the many benefits of EVAR, graft failure and the associated risk of persistent sac enlargement requires lifelong surveillance.[56,57] CT/CT-angiography is recommended at one month, six months, and one-year post-EVAR repair to identify an endoleak, or persistent blood blow in the aneurysm sac extrinsic to the endograft, or other aberrant pathologies.[56,57] Open repair, while more invasive, is advantageous given that it is suitable for aneurysm repair in all areas, while EVAR has been associated with increased complications from upper extremity ischemia with aneurysms of the aortic arch.[58] Postoperative surveillance is minimal following open repair, and follow up CT imaging is recommended at five-year intervals.[56,57]

Special Considerations

Genetic predisposition (e.g., Marfan syndrome, Loeys-Dietz, Ehlers-Danlos syndrome) may also contribute to the development of an AA.[9] Special consideration should be made for patients with genetic syndromes that predispose them to the development of TAAs. Annual imaging is recommended for patients with Marfan syndrome if the stability of the aortic diameter is documented.[49]

TABLE 5:

Summary of indications for referral

INDICATION
Medical management is refractory
AAA growth greater than 1 cm per year; TAA growth greater than 0.5 cm per year
Symptomatic AA
Aortic diameter greater than 5.5 cm

More frequent imaging should be considered in patients with an aortic diameter of 4.5 cm or greater or if the aortic diameter shows significant growth.[49] Patients with Loeys-Dietz should obtain complete aortic imaging at initial diagnosis, and at six months to establish if enlargement is occurring.[49] Annually, patients should obtain magnetic resonance imaging from the cerebrovascular circulation to the pelvis.[49] Administration of beta-blockers has been recommended in patients with Marfan's syndrome unless contraindicated.[48] One study examining patients with Marfan syndrome reported that propranolol-treated patients had a 73% lower rate of dilation and mortality than placebo, however, later randomized studies in patients with an AAA failed to report similar findings.[50] Losartan (Cozaar®, Hyzaar®) an angiotensin receptor blocker (ARB), appears to exert a protective effect in a mouse model of Marfan syndrome through modulation of TGF-beta activity. However, no protective effect has been demonstrated in patients with AAAs.[50,51,52]

SUMMARY

Aortic aneurysms occur as a result of medial degeneration of the arterial wall generally as a result of increased aortic hypertension or genetic predisposition. Dedicated imaging studies offer definitive identification or exclusion of potential AAs, but the imaging modality used is largely dependent upon patient-related factors. Medical management of small AAs includes prophylactic beta blocker therapy, ACE inhibitors, ARBs, and statin therapy. There is large variability between patients presenting with an AA, and, therefore, no definitive therapy has been recommended for all patients. Studies evaluating a mouse model of the Marfan syndrome have suggested that Losartan (Cozaar,® Hyzaar®) an angiotensin receptor blocker, is advantageous. Patients for which medical management is refractory, with AAAs that exceed 1 cm of expansion per year or TAAs that exceed 0.5 cm of expansion per year, with symptomatic AA, or with aortic diameters greater than 5.5 cm should be referred for immediate surgical consultation, and postoperative surveillance should be followed accordingly.

AUTHOR DISCLOSURES

No relevant financial affiliations.

REFERENCES

1. Piechota-polanczyk A, Jozkowicz A, Nowak W, et al. The Abdominal Aortic Aneurysm and Intraluminal Thrombus: Current Concepts of Development and Treatment. Front Cardiovasc Med. 2015;2:19.
2. Goldfinger JZ, Halperin JL, Marin ML, Stewart AS, Eagle KA, Fuster V. Thoracic Aortic Aneurysm and Dissection. J Am Coll Cardiol. 2014;64(16):1725-1739. doi:10.1016/j.jacc.2014.08.025
3. Keisler B, Carter C. Abdominal aortic aneurysm. Am Fam Physician. 2015;91(8):538-43.
4. Van der Vliet A. Abdominal aortic aneurysm, The Lancet , Volume 349, Issue 9055 , 863 - 866
5. Vardulaki KA, Walker NM, Day NE, et al. Quantifying the risks of hypertension, age, sex and smoking in patients with abdominal aortic aneurysm. Br J Surg. 2000;87(2):195-200. PMID: 10671927.
6. U.S. Preventive Services Task Force. Screening for abdominal aortic aneurysm: recommendation statement. Ann Intern Med. 2014;161(4):281-290.
7. Jahangir E, Lipworth L, Edwards TL, et al. Smoking, sex, risk factors and abdominal aortic aneurysms: a prospective study of 18 782 persons aged above 65 years in the Southern Community Cohort Study. J Epidemiol Community Health. 2015;69(5):481-8.
8. Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999-2014 on CDC WONDER Online Database, released 2015. Data are from the Multiple Cause of Death Files, 1999-2014, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program. Accessed at http://wonder.cdc.gov/mcd-icd10.html on Aug 12, 2016 11:40:38 AM
9. Isselbacher EM. Thoracic and Abdominal Aortic Aneurysms. Circulation. 2005;111:816-828, published online before print February 14, 2005. http://dx.doi.org/10.1161/01.CIR.0000154569.08857.7A
10. Struijk EA, May AM, Wezenbeek NLW, Fransen HP, Soedamah-Muthu SS, Geelen A, Boer JMA, van der Schouw YT, Bueno-de-Mesquita HB, Beulens JWJ (2014) Adherence to dietary guidelines and cardiovascular disease risk in the EPIC-NL cohort. Int J Cardiol 176:354–359. doi:10.1016/j.ijcard.2014.07.017
11. Georgousopoulou EN, Mellor DD, Naumovski N, et al. Mediterranean lifestyle and cardiovascular disease prevention. Cardiovasc Diagn Ther. 2017;7(Suppl 1):S39-S47.
12. Smyth, A., O'Donnell, M., Mente, A. et al. Curr Hypertens Rep (2015) 17: 47. doi:10.1007/s11906-015-0559-8.
13. Sode BF, Nordestgaard BG, Grønbæk M, Dahl M. Tobacco smoking and aortic aneurysm: two population-based studies. Int J Cardiol. 2013;167(5):2271-7.
14. 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(10):1425-30.
15. Stanley JC, Veith F, Wakefield TW. Current Therapy in Vascular and Endovascular Surgery. Elsevier Health Sciences; 2014.
16. 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.
17. Erbel R, Aboyans V, Boileau C, et al. Corrigendum to: 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases. Eur Heart J. 2015;36(41):2779.
18. Fink HA, Lederle FA, Roth CS, Bowles CA, Nelson DB, Haas MA. The Accuracy of Physical Examination to Detect Abdominal Aortic Aneurysm. Arch Intern Med. 2000;160(6):833-836. doi:10.1001/archinte.160.6.833.
19. Grey TG. Local discoloration of the abdominal wall as a sign of acute pancreatitis. British Journal of Surgery. 7 (27): 394–395. doi:10.1002/bjs.1800072711
20. Jim J, Thompson RW. Clinical features and diagnosis of abdominal aortic aneurysm. Available at: http://www.uptodate.com/contents/clinical-features-and-diagnosis-of-abdominal-aortic-aneurysm. Accessed September 14, 2016.
21. Assar AN, Zarins CK. Ruptured abdominal aortic aneurysm: a surgical emergency with many clinical presentations. Postgrad Med J. 2009;85(1003):268-73.
22. Nigro G, Giovannacci L, Engelberger S, Van den berg JC, Rosso R. The challenge of posttraumatic thrombus embolization from abdominal aortic aneurysm causing acute limb ischemia. J Vasc Surg. 2011;54(3):840-3.
23. Singla V, Virmani V, Modi M, Kalra N, Khandelwal N. Chronic rupture of abdominal aortic aneurysm with vertebral erosion: an uncommon but important cause of back pain. Ann Vasc Surg. 2014;28(8):1931.e1-4.
24. De Boer NJ, Knaap SFC, de Zoete A. Clinical detection of abdominal aortic aneurysm in a 74-year-old man in chiropractic practice. Journal of Chiropractic Medicine. 2010;9(1):38-41. doi:10.1016/j.jcm.2009.12.002.
25. Greenland P, Alpert JS, Beller GA, et al. 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2010;122(25):2748-64.
26. Rizvi MM, Singh RB, Jain A, Sarkar A. Asymptomatic aortic aneurysm causing right vocal cord palsy and hoarseness: A rare presentation. Anesth Essays Res. 2014;8(3):397-400.
27. Tsui P, Lee JH, Maclennan G, Capdeville M. Hemoptysis as an unusual presenting symptom of invasion of a descending thoracic aortic aneurysmal dissection by lung cancer. Tex Heart Inst J. 2002;29(2):136-9.
28. Prokakis C, Charoulis N, Tselikos D, Koletsis EN, Apostolakis E, Dougenis D. Primary aortoesophageal fistula due to thoracic aortic aneurysm: successful surgical treatment. Tex Heart Inst J. 2009;36(6):607-10.
29. Taimour S, Zarrouk M, Holst J, et al. Aortic diameter at age 65 in men with newly diagnosed type 2 diabetes. Scand Cardiovasc J. 2017;:1-5.
30. Kristensen KL, Dahl M, Rasmussen LM, Lindholt JS. Glycated Hemoglobin Is Associated With the Growth Rate of Abdominal Aortic Aneurysms: A Substudy From the VIVA (Viborg Vascular) Randomized Screening Trial. Arterioscler Thromb Vasc Biol. 2017;37(4):730-736.
31. Kordzadeh A, Parsa AD, Askari A, Maddison B, Panayiotopoulos YP. Presenting Baseline Coagulation of Infra Renal Ruptured Abdominal Aortic Aneurysm: A Systematic Review and Pooled Analysis. Eur J Vasc Endovasc Surg. 2016;51(5):682-9.
32. Fernandez-bustamante A, Jimeno A. Disseminated intravascular coagulopathy in aortic aneurysms. Eur J Intern Med. 2005;16(8):551-60.
33. Mao SS, Ahmadi N, Shah B, et al. Normal thoracic aorta diameter on cardiac computed tomography in healthy asymptomatic adults: impact of age and gender. Acad Radiol. 2008;15(7):827-34.
34. Lin FY, Devereux RB, Roman MJ, et al. Assessment of the thoracic aorta by multidetector computed tomography: age- and sex-specific reference values in adults without evident cardiovascular disease. J Cardiovasc Comput Tomogr. 2008;2(5):298-308.

35. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121(13):e266-369.
36. Makaroun MS, Dillavou ED, Kee ST, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. J Vasc Surg. 2005;41(1):1-9.
37. Chiu KW, Ling L, Tripathi V, Ahmed M, Shrivastava V. Ultrasound measurement for abdominal aortic aneurysm screening: a direct comparison of the three leading methods. Eur J Vasc Endovasc Surg. 2014;47(4):367-73.
38. Sparks AR, Johnson PL, Meyer MC. Imaging of abdominal aortic aneurysms. Am Fam Physician. 2002;65(8):1565-70.
39. Evangelista A, Avegliano G, Elorz C, González-Alujas T, García del Castillo H. Role of Transoesophageal Echocardiography in the Diagnosis of Acute Aortic Syndrome.
40. Ramaiah V, Kpodonu J, Rodriguez-Lopez JA. Endovascular and Hybrid Management of the Thoracic Aorta, A Case-based Approach. Wiley-Blackwell; 2008.
41. Mathur SK, Singh P. Transoesophageal echocardiography related complications. Indian J Anaesth. 2009;53(5):567-74.
42. Popescu BA, Savu O, Beladan CC, Rosça M, Lupescu I, Ginghin C. A paravertebral pulsatile mass. Circulation. 2012;126(24):e344-7.
43. Cronenwett JL, Johnston KW. Rutherford's Vascular Surgery, 2-Volume Set. Elsevier Health Sciences; 2010.
44. Brewster DC, Cronenwett JL, Hallett JW, 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(5):1106-17.
45. Oladokun D, Patterson BO, Sobocinski J, et al. Systematic Review of the Growth Rates and Influencing Factors in Thoracic Aortic Aneurysms. Eur J Vasc Endovasc Surg. 2016;51(5):674-81.
46. Davies RR, Goldstein LJ, Coady MA, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. Ann Thorac Surg. 2002;73(1):17-27.
47. Patterson BO, Sobocinski J, Karthikesalingam A, et al. Expansion rate of descending thoracic aortic aneurysms. Br J Surg. 2016;103(13):1823-1827.
48. Ramanath VS, Oh JK, Sundt TM, Eagle KA. Acute aortic syndromes and thoracic aortic aneurysm. Mayo Clin Proc. 2009;84(5):465-81.
49. Elefteriades JA, Farkas EA. Thoracic aortic aneurysm: clinically pertinent controversies and uncertainties. Journal of the American College of Cardiology, 55 (2010), pp. 841–857
50. Danyi P, Elefteriades JA, Jovin IS. Medical Therapy of Thoracic Aortic Aneurysms, Are We There Yet?Circulation. 2011;124:1469-1476, published online before print September 26, 2011.
51. Mohler ER, Woo YJ, Greene CL. Management of thoracic aortic aneurysm in adults. Available at: http://www.uptodate.com/contents/management-of-thoracic-aortic-aneurysm-in-adults. Accessed September 14, 2016.
52. Dalman RL, Mell M. Management of asymptomatic abdominal aortic aneurysm. Available at: http://www.uptodate.com/contents/management-of-asymptomatic-abdominal-aortic-aneurysm. Accessed September 14, 2016.
53. Reimerink JJ, Hoornweg LL, Vahl AC, et al. Endovascular repair versus open repair of ruptured abdominal aortic aneurysms: a multicenter randomized controlled trial. Ann Surg. 2013;258(2):248-56.
54. Greenhalgh RM, Brown LC, Powell JT, et al. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med. 2010;362(20):1863-71.
55. Lederle FA, Freischlag JA, Kyriakides TC, et al.; OVER Veterans Affairs Cooperative Study Group. Long-term comparison of endovascular and open repair of abdominal aortic aneurysm. N Engl J Med. 2012;367(21):1988-1997.
56. Ricotta JJ. Endoleak management and postoperative surveillance following endovascular repair of thoracic aortic aneurysms. J Vasc Surg. 2010;52(4 Suppl):91S-9S.
57. Chaikof EL, Brewster DC, Dalman RL, et al. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg. 2009;50(4 Suppl):S2-49.
58. Zamor KC, Eskandari MK, Rodriguez HE, Ho KJ, Morasch MD, Hoel AW. Outcomes of Thoracic Endovascular Aortic Repair and Subclavian Revascularization Techniques. J Am Coll Surg. 2015;221(1):93-100.