Training Course

Aortic aneurysm

Atul Mathur, Varun Mohan, Deepak Ameta, Gaurav Bhardwaj, Pradeep Haranahalli
Department of Interventional Cardiology, Fortis Escorts Heart Institute, New Delhi, India

DEFINITION

Aortic aneurysm refers to pathologic dilatation of aortic segment that has the tendency to expand and rupture. The extent of dilatation is debatable but one criterion is an increase in the diameter of at least 50% greater than that expected for the same aortic segment in unaffected individuals of same age and sex. Aortic aneurysms are described in terms of their size, location, morphology, and cause.

EPIDEMIOLOGY

Prevalence

The incidence of abdominal aortic aneurysms (AAAs) has increased during the past two decades, due in part to the aging of the population, the rise in the number of smokers, the introduction of screening programs, and improved diagnostic tools. The disorder is more common in men than in women, with prevalence rates estimated at 1.3–8.9% in men and 1.0–2.2% in women.[1] However, thoracic aortic aneurysms (TAAs) have an estimated incidence of at least 5-10 per 100,000 person-years.[2] According to location, TAAs are classified into aortic root or ascending aortic aneurysms, which are most common (≈60%), followed by aneurysms of the descending aorta (≈35%) and aortic arch (<10%).[2] Thoracoabdominal aortic aneurysm refers to descending thoracic aortic aneurysms that extend distally to involve the abdominal aorta.

Risk factors

Important risk factors for AAA are advanced age, male gender, and smoking. A positive family history for AAA, especially first-degree male relative, is also associated with four times increased risk of AAA.[3] Additionally, history of other vascular aneurysms, greater height, coronary artery disease, cerebrovascular disease, atherosclerosis, hypercholesterolemia, and hypertension have been found to have association with AAA, although data for some of these factors are inconsistent.[4] Genomic studies have demonstrated the association with variants on chromosome 9p21. The presence of rs7025486[A] in the DAB21P gene is associated with a 20% increased risk of developing AAA.[5] Black or Asian race and diabetes mellitus are negatively associated with AAA development.[4] Besides conventional risk factors, TAAs are also attributed to genetic, inflammatory, and infectious diseases.

PATHOPHYSIOLOGY

Aortic aneurysmal disease is recognized as a distinct degenerative process involving all layers of the vessel wall. The pathophysiology is characterized by four events: infiltration of the vessel wall by lymphocytes and macrophages; destruction of elastin and collagen in the media and adventitia by proteases, including matrix metalloproteinases; loss of smooth muscle cells (SMCs) with thinning of the media; and neovascularization.[6] Important contributor to TAAs are genetic trigger, some of which are associated with widespread syndromic features and others with thoracic aortic disease alone. These disorders are associated with abnormalities in the aortic media, vascular SMCs, or contractile proteins, and many lead to overactivation of signaling pathways and downstream mediators.[7] Such disorders include Marfan syndrome (MFS), Loeys–Dietz syndrome (LDS), vascular Ehlers–Danlos syndrome (vEDS), familial thoracic aortic aneurysm and dissection syndrome (FTAA/D), bicuspid

Address for Correspondence:
Dr. Gaurav Bhardwaj, Consultant, Department of Interventional Cardiology, Fortis Escorts Heart Institute, New Delhi, India.
Email: drgauravbhardwaj@gmail.com

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aortic valve (BAV) disease, Turner syndrome (TS), and the aortopathy associated with many congenital heart diseases.

NATURAL HISTORY

Aneurysm growth rates

The average growth rate of AAAs of sizes 30 to 55mm ranges 0.2-0.3 cm/year. Larger AAAs are associated with higher AAA growth rates. Genetically triggered TAAs behave differently from atherosclerotic aneurysms. TAAs are relatively indolent, with a growth rate of 0.1–0.2 cm/year and with marked individual variability.[2,8] Aneurysms of the descending aorta have a much greater growth rate (0.19 cm/year) than do those of the ascending aorta (0.07 cm/year). Also, BAV ascending aortic aneurysms have a higher growth rate (0.19 cm/year) than do aneurysms in patients with a tricuspid aortic valve (TAV) (0.13 cm/year).[9]

Aneurysm rupture rates

Larger initial aneurysm diameter is a significant and independent risk factor for AAA rupture.[10] The association between AAA diameter and 12-month risk of rupture is depicted in Table 1.[10]

Table 1: Twelve-month AAA rupture risk by diameter

AAA diameter	Rupture risk (%)
30–39	0
40–49	1
50–59	1–11
60–69	10–22
>70	30–33

Other factors that have been associated with an increased risk of AAA rupture across several studies include female gender, smoking, hypertension, AAA expansion rate, and peak AAA wall stress.

Individual studies have suggested an increased risk of AAA rupture for patients with rapid increase in intraluminal thrombus, increased AAA wall stiffness, increased wall tension, low forced expiratory volume in 1 second (FEV1) and for transplant patients.[11]

For TAAs, mean rate of rupture or dissection was 2% per year for aneurysms smaller than 5 cm in diameter, 3% per year for those 5.0–5.9 cm, and 7% per year for those 6.0 cm or larger. Sex and body surface area may also play an important role in predicting complications of aneurysms.[9,12] Some have proposed using aortic cross-sectional area and body height.[12] Aortic risk calculator uses height, weight, and aortic size to calculate a yearly risk of rupture or dissection.[13] Patients with an aortic size index (ASI) of less than 2.75 cm/m² had a complication rate of 4%, those with an ASI between 2.75 and 4.25 cm/m² had an event rate of approximately 8%, and those with an ASI higher than 4.25 cm/m²had an event rate of 20–25%.[9]

SCREENING

Ultrasonography is the primary method used for screening AAA and is highly sensitive (95%) and specific (100%).[14] Computed tomography (CT) scanning and magnetic resonance imaging (MRI) are expensive, incur risks (radiation exposure from CT and risks associated with intravenous contrast material), and should not be used for screening but rather reserved for preinterventional planning.

The incidence of new AAA after a single normal scan at 65 years is rare and, when present, rarely reaches a significant size. Screening should be considered at an earlier age for those at higher risk for AAA. In 2005, the U.S. Preventive Services Task Force recommended a one-time ultrasound screening for AAAs in men aged 65–75 years with a history of smoking.[15,16] The Society for Vascular Surgery recommends a one-time screening for AAAs in all men older than 65 years or as early as 55 years in men and women with a family history of AAAs.[16]

ANEURYSM GROWTH AND SURVEILLANCE

Small AAAs (3.0–5.4 cm in diameter), when identified, should be monitored for expansion. In accordance with Laplace's law, the larger the aneurysm, the higher is the rate of expansion. Current guidelines regarding the frequency of monitoring are as follows: for aneurysms with a diameter of 3.0–3.4 cm, every 3 years; 3.5–4.4 cm, yearly; and 4.5–5.4 cm, every 6 months.[17]

CLINICAL PRESENTATION

Unruptured aortic aneurysms

Unruptured aneurysms are generally asymptomatic in most patients. They are essentially diagnosed incidentally during clinical examination or during population screening.

Nonruptured aneurysms might exceptionally be diagnosed after complications, such as distal embolization and, even more rarely, acute thrombosis. Minor and less specific symptoms include chronic vague abdominal and back pain, which can result from direct pressure on structures or by distension of visceral organs. Recent onset of severe lumbar pain has been deemed to indicate impending rupture. Ureterohydronephrosis might also take place, especially if the aneurysm is inflammatory or involves the iliac bifurcation. Similarly, most patients with a TAA are asymptomatic, and the aneurysm is discovered incidentally on chest radiography, echocardiography, CT,

or MRI. Findings on physical examination such as aortic regurgitation may lead to further imaging and diagnosis of TAA. Symptoms of TAAs are usually related to a local mass effect, progressive aortic regurgitation, heart failure from aortic root dilation, or systemic embolization as a result of mural thrombus or atheroembolism.

Ruptured abdominal aortic aneurysms

Rupture of AAAs is heralded by the triad of sudden onset of pain in the mid-abdomen or flank (that may radiate into the scrotum), shock, and the presence of a pulsatile abdominal mass. However, the degree of shock varies according to the location and size of the rupture and the delay before the patient is examined. Rupture from the anterolateral wall into the peritoneal cavity (Figure 1) is usually dramatic and most often associated with death at the scene. Most patients with a rupture who reach the hospital alive have a rupture of the posterolateral wall of aneurysm into the retroperitoneal space; a small tear can temporarily seal the rupture and the initial blood loss might be small. This initial event is systematically followed within hours by a larger rupture. This biphasic evolution emphasizes the importance of the intermediate period after the initial event, which should be used for medical transfer and emergency repair.

Anecdotically, the first episode of rupture could be definitely contained and become a chronic pulsatile extra-aortic hematoma. Very rarely, the aneurysm might spontaneously rupture into the duodenum (Figure 1); an incidence rate at necropsy of 0.04–0.07% has been reported.[18] More often, aortoduodenal fistula can occur after previous repair, with an incidence rate of 0.5–2.3%.[19] Rupture into the vena cava can also take place with an apparent pattern of lower extremity edema erroneously attributed to cavoiliac thrombophlebitis. However, the development of high-output congestive heart failure and the perception of continuous abdominal noise is pathognomonic. The overall prevalence of aortocaval fistula is 3–6% of all ruptured aortic aneurysms.[20]

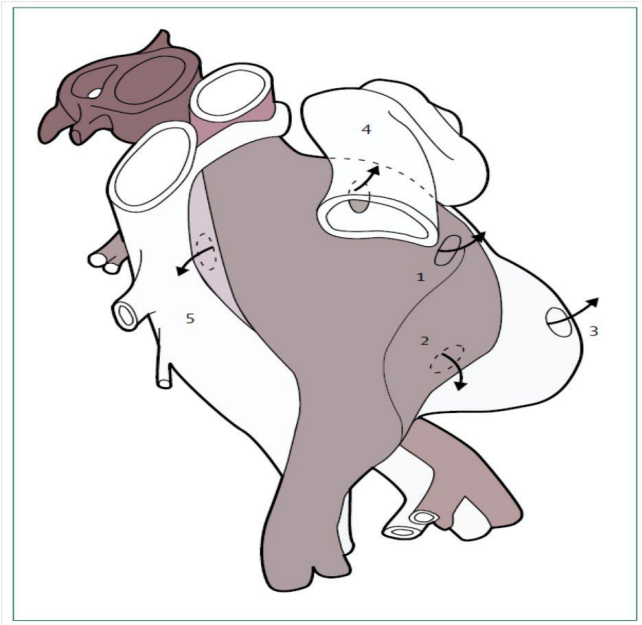

Figure 1: Different possible sites of rupture of an abdominal aortic aneurysm. 1: Anterolateral free rupture in the abdominal cavity. 2: Retroperitoneal rupture. 3: Rupture of retroperitoneal sac. 4: Rupture into the duodenum. 5: Rupture into the inferior vena cava.

Thoracic aortic rupture leads to sudden severe chest or back pain. Rupture into the pleural cavity (usually left) or into the mediastinum is associated with hypotension, rupture into the esophagus leads to hematemesis from an aortoesophageal fistula, and rupture into the bronchus or trachea results in hemoptysis. Infected TAAs are more commonly associated with fistulas. Acute aortic expansion, contained rupture, and pseudoaneurysm can cause severe chest or back pain. Thoracic aortic dissection is more common than rupture.

TREATMENT

Risk factor modification

Smoking cessation is associated with reduced rate of aneurysmal growth. The recognized association between either hypertension or hypercholesterolemia and the occurrence of abdominal aortic aneurysm suggests that control of these coexisting conditions with medications such as antihypertensive agents and statins may decrease the risk, although limited data are available to support this hypothesis. Patients with small aneurysms should be encouraged to exercise regularly because moderate physical activity does not adversely influence the risk for rupture and may even limit the rate of aneurysm growth.

Medical therapy

Several drugs have been evaluated for their potential to limit AAA. Betablockers, antibiotics, and anti-inflammatory agents have been examined in randomized trials, and angiotensin-converting-enzyme inhibitors, angiotensin-receptor blockers, statins, and antiplatelet agents have been examined in nonrandomized studies. Unfortunately, none of these drugs have been shown to provide a benefit.[21] Doxycycline inhibits matrix metalloproteinases, a finding that suggests that it might reduce the growth of aneurysms. However, in a placebo-controlled, randomized trial, doxycycline at a daily dose of 100 mg did not reduce the growth of small aneurysms over a follow-up period of 18 months.[22]

Indications for aneurysm repair

The management of aneurysm depends on the size or diameter of the aneurysm and is a balance between the

risk of aneurysm rupture and the operative mortality for aneurysm repair.

For very small aneurysms, 3.0–3.9 cm, the risk of rupture is negligible. Therefore, these aneurysms do not require surgical intervention and should be kept under ultrasound surveillance at regular intervals.

For aneurysms between 4 and 5.5 cm in diameter, rapid growth (>1 cm/year) and the development of symptoms referable to the aneurysm warrant intervention.

Aneurysm repair should be considered at a maximum aneurysm diameter of 5.2 cm in females and 5.5 cm in males. This is because females appear more likely to suffer AAA rupture at smaller aortic diameters than males.[23]

In general, surgical replacement of ascending aorta should be performed when the ascending aortic diameter reaches 5.5 cm and, in the setting of BAV aneurysm, MFS, and familial TAA syndromes, when it reaches 5 cm.[11,12] In adults with LDS, surgery is recommended when the aortic root measures 4.2 cm by transesophageal echocardiogram (TEE) or 4.4–4.6 cm by CT or MRI,[11] although some experts recommend surgery in patients with LDS once the aortic root is larger than 4 cm.[24,25] In TS, prophylactic surgery should be considered when the ascending aorta is 3.5 cm or larger or 2.5 cm/m^2 or larger.[26] Surgical timing also depends on the family history, sex, rate of aneurysm growth, body size, coexisting aortic valve disease, need for other heart surgery, comorbid conditions, and patient and physician preference.

INTERVENTIONS FOR ANEURYSM REPAIR

Endovascular aneurysm repair

Endovascular aneurysm repair (EVAR) is a minimally invasive procedure for the treatment of AAA based on the use of a stent graft, usually deployed inside the aneurysm through femoral access to exclude the AAA sac from the circulation. EVAR requires adequate aortic and iliac fixation sites for effective sealing and fixation.

Potential advantages of EVAR over open repair include reduced operative time, avoidance of general anesthesia, less trauma and postoperative pain, reduced hospital stay and less need for intensive care unit (ICU), reduced blood loss, and reduced postoperative mortality. Potential disadvantages include the risk of incomplete AAA sealing, with the development of continuous refilling of the aneurysm sac, either because the graft does not seal completely at the extremities (Type I endoleak), between segments (Type III endoleak), or because of backfilling of the aneurysm from other small vessels in the aneurysm wall (Type II endoleak). Morphological criteria for EVAR are shown in Table 2.

Graft model choice

Appropriately sized aortic endograft should be selected on the basis of patient anatomy: according to the instruction for use of abdominal endografts, generally, the device should be oversized 15–20% with respect to the aortic neck diameter to guarantee optimal seal.

Management of concomitant iliac aneurysms

Concomitant iliac aneurysms may be present in up to 40% of patients with EVAR. Coil embolization of hypogastric (internal iliac) artery, followed by endograft extension into the external iliac artery (EIA), is usually performed to prevent Type II endoleak. Hypogastric embolization is usually preferred over simple coverage of its ostium by the endograft to prevent the risk of Type II endoleak, but coils should be placed as proximal as possible to spare collateral circulation.

Approximately one-third of patients with hypogastric occlusion have symptoms of pelvic ischemia: buttock

Table 2: Morphological criteria for EVAR

Proximal aortic neck
Neck diameter >17 mm, < 32 mm
Angle between the suprarenal aorta and the juxtarenal aorta < 60°
Angle between the juxtarenal aorta and the long axis of the aneurysm sac <60–90°
Neck length >10 mm
Neck thrombus covering <50% of the proximal neck circumference
Neck dilated <3 mm within 10 mm of the most caudal renal artery
Focal neck enlargement <3 mm within 15 mm from the most caudal renal artery
Neck calcification <50% of the proximal neck circumference
Aortic bifurcation
Aortic bifurcation diameter >20 mm in case of a bifurcated graft
Iliac artery
Iliac luminal diameter > 7 mm
Angle between the long axis of the aneurysm and the iliac axis <60°
Iliac calcification: nonextensively circumferential
Iliac neck diameter <22 mm
Iliac neck length >15 mm

claudication is fortunately the most common, occurring in about 80% of symptomatic patients; impotence in about 10%; and colonic ischemia in 6–9% of all the pelvic ischemic complications.[27]

Perioperative mortality and morbidity

The 30-day mortality is approximately 1%, the hospital stay is on an average three days, and full recovery usually occurs over a period of days to weeks.

Open surgical treatment

Open repair requires an abdominal or a flank incision; vessels above and below the aneurysm are controlled, and the aneurysm sac is opened with interposition of a synthetic graft. The upper anastomosis is of the end-to-end type and the distal anastomosis is located on the aortic bifurcation, the iliac bifurcations, or the common femoral arteries depending on the extent of aneurysmal transformation and the patency of the external iliac arteries. Care is taken to preserve at least one internal iliac artery and to detect perioperatively a potential left colonic-ischemia. In sexually active male patients, the recommendation is not to dissect the lateral left aortic wall and the common left iliac artery. Whenever possible, iliac anastomosis should be the preferred choice instead of common femoral anastomosis because anastomosis in the groin is more prone to infection.

Graft configuration

There are several prosthetic grafts available for aortic replacement: knitted or woven Dacron, impregnated with collagen, albumin, or gelatin if needed, and polytetrafluoroethylene (PTFE). All materials show excellent patency and long-term results, so that the surgeon's preference and the costs determine the aortic graft choice. Prager *et al.* found a comparable long-term patency for PTFE and Dacron, but PTFE had a higher incidence of early graft failure and graft infection.[28]

Perioperative mortality and morbidity

Depending on the study design and patient selection, the perioperative 30-day mortality rate after open aortic aneurysm repair differs widely and ranges between 1% and 8%, with selected centers of excellence reporting a 1% mortality rate.

EVAR v/s open repair

Three major randomized trials have compared open repair with endovascular repair, each with a follow-up period of 7–10 years: the U.K. Endovascular Aneurysm Repair 1 (EVAR 1) trial,[29] the Dutch Randomized Endovascular Aneurysm Management (DREAM) trial,[30] and the Open versus Endovascular Repair (OVER) Veterans Affairs Cooperative Study.[31] The findings of all three trials were similar. Endovascular repair confers an initial survival benefit; however, this benefit disappears over a period of 1–3 years. Endovascular repair and open repair are associated with similar mortality over the long term (8–years).

Among patients who underwent endovascular repair in the three trials, approximately 20–30% required a secondary intervention during the next 6 years. Reintervention is often related to the development of endoleaks, which reperfuse the aneurysm and lead to continued aneurysm expansion. Conversion to open repair is necessary in 2–4% of patients. Late ruptures after endovascular repair were reported in each of the trials. The incidence was highest in the trial that began the earliest, the EVAR 1 trial (4.0% *vs.* 0.6 and 1.4% in the more recent trials).

Because of the potential for reperfusion and the associated risk of aneurysm rupture, patients who have undergone endovascular repair require long-term surveillance by means of CT or ultrasonography, which is recommended at 1 month and 12 months after the intervention and yearly thereafter.[32]

Patients who have undergone open surgery may also require other surgical interventions for complications related to the procedure, such as ventral hernia or adhesions. After open repair, CT monitoring for new or recurrent aneurysmal disease is recommended at 5-year intervals.[32]

Thoracic endovascular repair

Most of the data regarding endovascular repair of TAA/TAAA are essentially based on observational studies and suggest that thoracic endovascular repair (TEVAR) of TAAs is a safe alternative to open surgery and is associated with lower mortality and morbidity.[33] As a prerequisite to the success of TEVAR, the proximal and distal neck diameters should not exceed the largest diameter of the graft available, for a leak-proof seal. Also, the proximal and distal necks should be at least 20 mm long for an adequate landing zone. Impingement of the major arterial branches that might be occluded for adequate sealing have to be carefully assessed for risks associated with occlusion, with an option of surgical bypass or debranching to maintain perfusion while extending the sealing zone. In case any of these anatomic criteria is not met, an open approach is preferred to an endovascular approach. Many endovascular grafts are available now as depicted in Table 3.

With TEVAR use, there are considerably less known major side effects compared with open surgical intervention as shown in the TAG Study[34] and VALOR (Evaluation of the Medtronic Vascular Talent Thoracic Stent Graft System for the Treatment of Thoracic Aortic Aneurysms) Medical studies.[35]

Table 3: Endografts available for TAA repair

Product name	Company name	Stent material	Graft material
Relay Thoracic	Boston Scientific (Marlborough, MA)	Nitinol	Woven polyester with PTFE sutures
TX	Cook Medical (Bloomington, IN)	Stainless steel	Woven polyester
Gore TAG	Gore & Associates (Newark, DE)	Nitinol	ePTFE
Valiant Thoracic	Medtronic Inc (Minneapolis, MN)	Nitinol	Woven polyester

Vascular complications

These are mainly attributed to the large size of the sheath for the deployment of the device. In the presence of tortuous, calcified, and small-sized iliac arteries, use of arterial conduits can help bypass the hostile arterial anatomy and provide direct access, thereby lowering the access site complication rates.

Neurologic complications

These subsets of complications are mainly caused because of embolization during placement of the graft, leading to stroke or occlusion of the spinal arteries leading to spinal ischemia. There is a higher chance of neurologic involvement when the graft involves covering the hypogastric artery, left subclavian artery, and extensive thoracic aortic coverage.[36,37] Associated comorbid conditions such as renal insufficiency[36] and intraoperative hypotension (systolic blood pressure <80 mm Hg) also puts a patient at high risk of neurological complications.[38] Procedural techniques such as minimal manipulations, knowledge of important vascular anatomy and optimization of graft location can help minimize the risk of stroke during TEVAR.

Endoleaks

These are well-documented complications of TEVAR. The prevalence of complications are noted to vary from 26.0% in the VALOR Trial[38] to 3.6% in Gore TAG Trial.[35] The incidence is reported to be decreasing with the advent of second-generation devices.

Conflict of Interest

None declared.

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