Chapter

Aortic Aneurysm: Clinical Findings, Diagnostic and Treatment

Akshay Kumar, Purnadeo Persaud, Ana Francesca Leite, Nathaniel Aoyama, Ruan Matos, Dor Mordehay Avishay, Nimisha Shiwalkar, Vinit Garg, Juber Dastagir Shaikh, Roopvir Kaur and Asim Kichloo

Abstract

The aorta is the largest artery in the body and can have aneurysms, which are focal expansions of the vessel wall that can occur anywhere throughout the artery. These can be classified as thoracic, abdominal or thoracoabdominal aneurysms and can be caused by several etiologies, including degenerative, infectious, and genetic causes. Most aortic aneurysms are asymptomatic and are detected incidentally while looking for other primary diseases with a physical exam finding of a pulsatile mass, or with imaging such as ultrasound, computed tomography, x-rays, or magnetic resonance imaging. When symptoms are present, they are often nonspecific and occur due to inflammation, rapid expansion, compression/erosion of the aneurysm into surrounding structures, or rupture. Uncontrolled aortic aneurysms can lead to fatal outcomes, thus making proper management essential. Management can range from medical treatment to surgical repair based on location, size, rate of expansion, and presence of symptoms.

Keywords: Aortic aneurysm, thoracic aneurysm, abdominal aneurysm, diagnosis, treatment, risk factors, hypertension

1. Introduction

Aortic aneurysm is a heart disease that consists of the formation of a bulge in the largest artery in the human body: the aorta. For a better definition, the dilation must be permanent, localized and exceed at least 50% the normal diameter of the aorta. A dilation greater than 50% and that occurs in a diffuse way, that is, involving several arterial segments, differs as arteriomegaly. Likewise, ectasia is different from aneurysm when compared, since the dilation regarding ectasia shows less than 50% increase when compared to the original diameter of the artery [1].

AAAs tend to dilate progressively over time, while TAAs have a slower expansion when compared to AAAs [2, 3]. The expansion of AAAs can vary greatly from one to another, however, the larger the aneurysm, it tends to expand at a

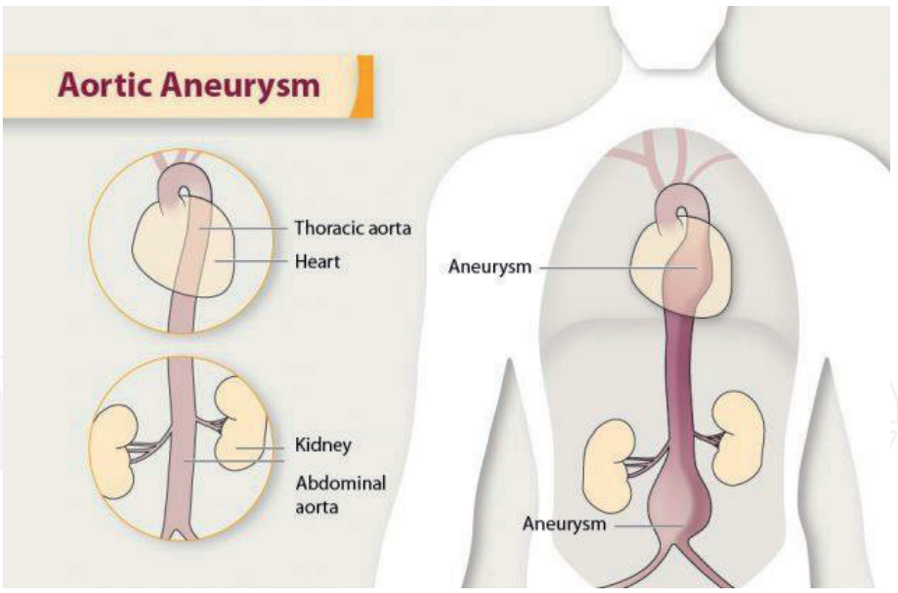

Figure 1.
Different sites of Aortic Aneurysms including Thoracic and Abdominal. (From Centers for Disease Control and Prevention (CDC).

higher rate than smaller aneurysms. In view of the lower growth rate, TAAs tend to be asymptomatic. When TAAs are symptomatic, they are associated with rapid growth, large size and high risk of rupture, which also leads to a higher risk of mortality (**Figure 1**) [4, 5].

1.1 Epidemiology

The epidemiology of aortic aneurysm has several important nuances when it comes to its division for obtaining data. The different types are divided into 8 categories by the Centers of Disease Control and Prevention in the United States of America (CDC), taking into account the location of the aneurysm (thoracic, abdominal, abdominal thoracic or unspecified site) and the presence of aneurysm rupture (ruptured or without mention of rupture).

Table 1 shows that the most lethal type of aortic aneurysm are abdominal aortic aneurysms, followed by aneurysms with an unspecified site, thoracic aneurysms and, finally, thoracoabdominal aneurysms. Also, through the data in the table, we can see that ruptured aneurysms have higher death numbers when compared to the absence of rupture.

171.1 (Thoracic aortic aneurysm, ruptured)	15,254
171.2 (Thoracic aortic aneurysm, without mention of rupture)	12,403
171.3 (Abdominal aortic aneurysm, ruptured)	74,390
171.4 (Abdominal aortic aneurysm, without mention of rupture)	36,233
171.5 (Thoracoabdominal aortic aneurysm, ruptured)	1,784
171.6 (Thoracoabdominal aortic aneurysm, without mention of rupture)	2,937
171.3 (Aortic aneurysm of unspecified site, ruptured)	23,809
171.9 (Aortic aneurysm of unspecified site, without mention of rupture)	13,654

Table 1.
Underlying Cause of Death, 1999–2019 (partial). Centers for Disease Control and Prevention (CDC) [6].

Analysing the epidemiology of AAAs separately, it is possible to extract several pertinent information, such as the predominance of males in the involvement of AAA as a result of screenings studies [7–10]. Abdominal aortic aneurysms have an annual incidence of 0.4% to 0.67% in western populations [11–14].

Deaths due to complications from TAAs, such as rupture and dissection, are usually attributed to other related causes such as acute myocardial infarction for those who are not conducted by post-mortem examinations. This situation is a consequence of TAA being a clinically silent disease [15]. The annual incidence of thoracic aortic aneurysm was estimated in two studies between 5.6 to 10.4 cases for 100,000 patient-years [16, 17].

1.2 Risk factors

There are different risk factors for the two main types of aneurysms. For AAAs, the main factors are atherosclerotic risk factors, advanced age, male sex, Caucasian race, family history, tobacco use, presence of other large vessel aneurysms, food and alcohol consumption. These factors have an influence on both the development of AAA and its expansion and rupture. Another important observation is that tobacco abuse seems to be the most relevant factor to its development. There are some factors that contribute to the reduction of the development of AAA, such as diabetes mellitus and moderate alcohol consumption [18].

For TAAs, there are some studies that show that the factors that predicted TAAs also were the same that predicted aortic atherosclerosis. These factors would be high blood pressure, high serum cholesterol and cigarette smoking [19].

1.3 Different types/causes

There are different types of arterial aneurysms. They are divided based on factors such as location, origin, histological aspects and clinicopathological manifestations [1]. The main study object in this chapter will be aortic aneurysms.

The main division that is used for aortic aneurysm is based on its location. Thus, its division is separated into abdominal, thoracic and thoracoabdominal aortic aneurysms. The first two are the most frequent ones [6].

The field of genetics and how it affects the development of this disease has brought new knowledge about its causes. TAAs may be associated with several syndromes, while AAAs are not [20]. The genetic predisposition of TAA development is well documented. Some of the main genetic influences are Marfan syndrome, Ehlers-Danlos syndrome, syndromic connective tissue disorders, Loeys-Dietz syndrome, as well as other syndromes and nonsyndromic disorders. These are being studied for better knowledge of TAA causes [21].

Apart from genetics, there are some causes that improve the chances of the development of thoracic aortic aneurysms. Some causes worth mentioning are syphilis, aortic arteritis, bicuspid aortic valve, aortic dissection, and trauma [22].

1.4 Pathogenesis

The origins of both of the main types of aortic aneurysm seem to be related to the origins of atherosclerosis in some cases, depending on the area affected [3, 23].

The main alteration TAAs have in the vascular walls are usually cystic medial degeneration. Histologically, there is the loss of smooth muscle cells and elastic fibre degeneration. These processes lead to arterial weakening and, as a result of it, the aortic dilatation occurs resulting in aneurysm formation [22].

In AAA, the extracellular matrix of the aorta is affected by the degradation of elastin and collagen, which are essential for wall integrity. This process weakens the aortic wall and contributes to the formation of the aneurysm [22].

2. Clinical findings

2.1 Thoracic aortic aneurysm (TAA)

2.1.1 Asymptomatic TAA

Aortic aneurysms are often clinically silent if there are no associated complications such as dissection, compression, or rupture. When complications are present, symptoms can mimic other diagnoses. Therefore, it is necessary to suspect a complicated aortic aneurysm in all patients with chest pain [24].

When a thoracic aortic aneurysm is asymptomatic, the diagnosis is usually made incidentally by imaging while searching for another medical condition, such as echocardiography (in aortic murmur), computed tomography (pulmonary nodule or pulmonary embolism), or in screening for the disease in question, in patients who are at high risk for the disease [25].

Regarding biomolecular markers, no specific one has yet been found that identifies the presence of thoracic aortic aneurysm [26–28]. Although some studies have correlated increased levels of D-dimer with the presence of thoracic aneurysm- due to the deposition of a thrombus in the dilatation, newer literature has shown that this product is nonspecific, and can rise in a series of thrombotic events, not only in thoracic aneurysms [29–31]. D-dimer has been used mainly in patients who show symptoms based on its negative predictive value. Moreover, matrix metalloproteinases, cytokines, acute phase reagents, lipoproteins, homocysteine and transforming growth factor beta were also studied, but none of them proved to be a useful predictor in the diagnosis of thoracic aneurysms [28].

Some of the clinical findings related to aortic aneurysm are intracranial aneurysm, inguinal hernia, abnormalities of the branched aortic arch, simple renal cyst and positive family history of aneurysm or aortic dissection. In this sense, patients with these conditions are considered patients with thoracic aortic disease [2].

2.1.2 Symptomatic TAA

The symptoms of thoracic aortic aneurysms are closely linked to rapid expansion, which predisposes dissection or rupture of the aorta. In order to reduce the chance of rupture, if it has not already happened, surgical management is indicated, even if the dimensions of the aneurysm are not extensive.

The most common symptom related to dissection/rupture of a thoracic aneurysm is sudden onset of chest, back and/or severe abdominal pain.

Especially in young patients, it is important that the doctor makes a detailed anamnesis, seeking to correlate the patient's clinical history with Marfan's syndrome, Loeys-Dietz syndrome, Ehlers-Danlos vascular syndrome [15], Turner syndrome, bicuspid aortic valve or other connective tissue disorders associated with thoracic aortic disease. As soon as the TAA expands and compresses the adjacent structures, the pain begins, which may be at the site of the rupture, or in adjacent regions, presenting with irradiated pain.

Depending on the location of the aneurysm, some specific symptoms can be found:

- Heart failure due to aortic regurgitation resulting from dilation of the aortic sinus and annular distortion, in the case of aneurysm in the aortic arch;

- Ischemia or myocardial infarction; due to compression of a coronary artery.

- Continuous murmur that can progress to heart failure, if the aneurysm is located in the sinus of Valsalva and there is a rupture on the right side of the heart;

- Dysphagia by esophageal compression (hoarseness of the left recurrent laryngeal nerve or compression of the left vagus nerve or hemidiaphragmatic paralysis by compression of the phrenic nerve), in the case of large aneurysms that affect the transverse and descending arch, [22];

- Symptoms such as wheezing, coughing, hemoptysis, dyspnea or pneumonitis, if the aneurysm compresses the tracheobronchial tree;

- Thromboembolism or superior vena cava syndrome (swelling of the neck, face or upper extremities), if the aneurysm causes occlusion of the superior vena cava;

- Acute neurological complaints if there is aneurysmal compression of the branches of the vessels of the aortic arch or arterial thromboembolism.

In order to cause symptoms, descending aortic aneurysms need to be much larger than ascending aortic aneurysms. Back pain may occur due to erosion of the aneurysm into the spine, or to visceral or extremity ischemia.

Rupture is the most serious complication, often in the left thorax or pericardium, with severe chest pain and hypotension or shock. If the rupture is in the descending aorta, aortoesophageal fistula and hematemesis may be present.

The association between systemic manifestations (fever, weight loss), increased leukocyte count, and increased D-dimer may indicate a major thoracoabdominal aneurysm [32, 33]. Another two important signs in clinical reasoning are the presence of anaemia, which reveals acute blood loss as the cause of the shock, and high levels of lactic acid due to ischemia.

Patients with thoracic aortic aneurysms that present with symptoms of chest pain are usually submitted to electrocardiography (ECG), which may be compatible with myocardial infarction, signs of myocardial hypertrophy due to long-standing hypertension or valve disease.

The main physical characteristics related to thoracic aortic aneurysms are: Marfan syndrome, Loeys-Dietz syndrome and familial TAA which is associated with livedo reticularis, iris flocculi, congenital mydriasis (ACTA2 mutation) and peripheral vascular malformation [15, 33, 34].

2.2 Abdominal aortic aneurysm (AAA)

2.2.1 Asymptomatic AAA

Most individuals with AAAs have no symptoms, but when they do, pain is the most common, and may or may not be associated with AAA rupture. Like TAA, asymptomatic AAA is discovered incidentally, on imaging studies that were aimed at investigating another cause, or in routine physical examination (especially in patients who complain of coronary, peripheral or cerebrovascular diseases, or during population screening) [35].

About 30 percent of asymptomatic abdominal aortic aneurysms can be suspected by the presence of a pulsatile abdominal mass, which can be palpated on routine physical examination [36]. In addition, asymptomatic AAA is also commonly detected as an incidental finding in imaging studies, and in most cases (2/3) it is not communicated to the patient's family [37].

Another manifestation that should lead to suspicion of AAA is arterial disease due to the presence of another peripheral aneurysm (iliac, femoral, popliteal).

The pulsatile abdominal mass that leads to AAA grows over time, and these dilations are typically asymptomatic, until the catastrophic rupture event [38], where abdominal pain, and even lower limb ischemia due to interrupted blood flow will be felt [39].

2.2.2 Symptomatic AAA

Non-ruptured aneurysms can 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 or distention of adjacent structures. The recent onset of severe low back pain was considered an indication of impending rupture. Ureterohydronephrosis can also occur, especially if the aneurysm is inflammatory or involves iliac bifurcation [38].

The set of symptoms that lead to the suspected diagnosis of a ruptured abdominal aortic aneurysm: sudden onset pain in the abdominal region or flank (which can radiate to the scrotum), shock and the presence of a pulsatile abdominal mass. Regarding shock, its degree will vary according to the location of the rupture, the size, and the time between the event and the diagnosis.

It is known that most patients affected with AAA are asymptomatic, however, when present, in 50% of symptomatic cases, the classic triad of severe severe pain, pulsatile abdominal mass, and hypotension is present [40]. Limb ischemia or systemic manifestations are also common in infected or inflamed aneurysms [41–43].

The pain varies according to the diameter and position of the aneurysm, whether intact or ruptured, and if ruptured, it may be contained or free. Despite being typically located in the abdomen, the pain can radiate to the groin or thigh [44].

Thrombus embolism or atherosclerotic debris from a ruptured aneurysm can lead to limb ischemia [45, 46], which in addition to the clinical manifestations of interrupted blood flow, can also cause pain. Such clinical manifestations will be apparent if vessels are more affected and arterial occlusion is not well compensated, and may present as sore and blue fingers (blue toe syndrome) or with a cold, pulseless and sore extremity.

Other possible manifestations of AAA are systemic symptoms that reflect the presence of an infected or inflammatory aneurysm or disseminated intravascular coagulation. The main symptoms to be mentioned are fever and malaise. In the case of inflammatory aneurysm, chronic abdominal pain, weight loss and changes in serum markers are suggestive symptoms [47–50].

With abdominal palpation, in some cases it is possible to feel a pulsating abdominal mass and in patients with AAA rupture, some degree of abdominal distension and tenderness will be present [40].

In the case of ruptured AAA, it is possible to find ecchymosis in the patient (Gray-Turner's sign, Cullen's sign, Fox's sign and Bryant's sign) [51]. Moreover, evidence of distal embolization or ischemia on vascular physical examination supports the diagnosis of AAA [52].

3. Diagnosis

Most people with aortic aneurysms are asymptomatic, leading to slow, unnoticed growth of the disease. In this sense, most diagnoses are made by routine exams. These tests are effective in reducing mortality, especially in high-risk populations. Therefore, imaging methods are used, with ultrasound being preferred, as it is not invasive, cheap and generates compelling information, such as the diameter of the vessels [53].

A diagnosis of aortic aneurysm requires an image of this artery to be confirmed. Thus, it is important for imaging tests, both initially and for probable complications, to be taken priority over tests such as ECG, for example [15]. Routine exams, such as chest x-rays on the other hand, often detect aneurysms in asymptomatic patients. Although ultrasound is a priority when a hypothesis of aortic aneurysm is raised, echocardiography, magnetic resonance imaging (MRI) or computed tomography (CT) are great tools for diagnosis, even when done for a condition unrelated to the aneurysm [54].

Expansion of the aneurysm can cause chest and lower back pain, as well as coughing, hoarseness or even difficulty breathing. Abdominal aortic aneurysms, for example, can be checked by physical examination of the abdomen, but the factors for the development of symptoms are not yet well defined, and factors such as obesity complicate this type of examination.

3.1 Screening

Thoracic aortic aneurysms (TAA) are among the top 15 causes of death in the United States. In addition, approximately 1 in 1000 Americans develop TAA per year, 95% of whom are asymptomatic. Data such as these demonstrate the importance of screening for the disease.

It is known that diseases as well as unhealthy behaviors (hypertension or smoking) can damage the heart and blood vessels. In addition, hereditary diseases such as Ehlers-Danlos syndrome and Marfan syndrome can increase the risk of aneurysm. Although the prevalence of abdominal aortic aneurysm (AAA) is high, AAA findings in screening are generally small in size [55]. These findings point to the importance of epidemiological studies and the impact they can have on the outcome of the diagnosis. In this sense, the incidence of AAA, for example, increases sharply in individuals over 60 years of age, as well as a reduction in smoking that can decrease the frequency of this injury [56].

In this sense, it is appropriate to survey the history of habits and diseases, not only for the individual patient, but also for aortic aneurysms in the entire population in question. Factors such as Caucasian race, smoking, men of advanced age and family history are important data to be analyzed for a blunt screening of great importance [3]. Screening, therefore, can change the outcome of the disease, since with clinical and epidemiological criteria, disease morbidity and mortality can be reduced.

The US Preventive Services Task Force conducted a review of the evidence on the effectiveness of single and repeated screening for AAA, as well as its harm and benefits. Ultrasonography, for example, was given as the primary method in primary care for the detection of AAA, due to its sensitivity (from 94–100%) and its specificity (from 98–100%), in addition to not being invasive and of easy execution [57]. In screening, there are some damages that can be observed. Although some data show that women who smoke or have a family history are at higher risk of AAA compared to women in the opposite group, there is insufficient evidence that screening has great benefit [57]; this is an example that even if it looks like a good strategy, it can still generate major complications: the concern about the

existence of overdiagnosis and excessive treatment is something of great impact. Overdiagnosis is a major problem in the screening and outcome of the disease-cure relationship of aortic aneurysms, including public health, as it can wrongly consider patients as mild to severe patients, which generates unnecessary or even harmful treatments [58]. In the U.S. Preventive Services Task Force study, it was reported that after the introduction of PSA exam, there was a considerable increase in new cases of prostate cancer. The expected drop in mortality did not occur, and this was justified by the numbers of overdiagnosis, which reached almost 50% of patients diagnosed in the study in that period [59]. For all these reasons, screening is understood as a tool of extreme need, but with the possibility of error and damage in the epidemiological study.

3.2 How to determine the etiology eg infectious vs. degenerative

Whether due to trauma or a pre-existing disease, aortic aneurysms occur due to the weakening of the vessel walls. In this sense, the failure is due to the biomechanics of the aortic wall [60]. Aortic aneurysms have as main factors for development of atherosclerosis, old age and male gender, as well as smoking and family history [61]. For hypertension, there was a 60% finding for patients with TAA [16]; besides that the existence of other aneurysms was also an important factor [62].

One can also mention the characteristics that inflammatory reactions in the vessels can bring. Especially, when considering aortitis, some inflammatory disorders can cause TAA, for example [63]. According to a prospective study by Pacini et al., among 788 patients referred for surgery for TAA, approximately 40% were due to aortitis, proven histologically. In this sense, these factors demonstrate an important causal relationship for the disease.

4. Treatment/management

4.1 Medical management

To this day, for a smaller aneurysm without indication for elective repair, there is no specific medication or other pharmaceutical therapy that is proven to reduce the rate of aneurysm growth or its risk of rupture. Even though Beta-blockers have been advocated to decrease aneurysm deterioration in the past, their effectiveness wasn't proven. Interestingly Doxycycline, an antibiotic with metalloproteinase inhibition action demonstrated decreased expansion rates in animals and humans. However, to this day it is not recommended for this purpose. Nevertheless, addressing future cardiovascular events in patients with aortic aneurysm is recommended. Hence, smoking cessation, blood pressure management, statins, beta-blockers and Angiotensin II receptor blockers are recommended. Alongside, serial noninvasive surveillance of aortic development [64, 65].

4.2 Surgical techniques

Surgical repair is the definitive treatment for aneurysms. It can be classified according to the approach: endovascular or open aortic, or according to the aortic segments: thoracic aorta, abdominal aorta, and aortic arch.

Open aortic repair is aimed to replace the dilated aortic segment with synthetic graft tube. This procedure is done under full anesthesia and it is classified as a high risk procedure. On the other hand endovascular repair aims to exclude the

dilated segment from the systemic circulation, by insertion of an endograft. This procedure can be done under local anesthesia and it carries relatively reduced risk in comparison to open repair.

During open surgical repair the aneurysmal sac is replaced with a polyethylene terephthalate graft. The procedure is carried out in a sterile environment under general anesthesia. First, abdominal or thoracic aorta is exposed, then a proximal clamp is placed: below the renal arteries in AAA or distal to the aortic arch in descending TAA, following a distal clamp. Later, the aneurysmal segment is dissected and replaced with a tube graft. Then depending on the aneurysm location visceral arteries are integrated to the tube graft. Finally, abdominal wall or thoracic closure is performed.

In contrast, in endovascular repair first, bilateral or unilateral access via the patient's common femoral artery is established, by a surgical incision or percutaneously. Then, vascular sheaths, through which guidewires, catheters, and the endograft are introduced into the femoral arteries. Then, retrogradely, the "main body" of the endograft is placed over the aneurysmal sac. Finally, the graft is dilated into place with a balloon, and wires, sheaths, and delivery systems are withdrawn. All along the procedure angiograms are used to evaluate graft location, landing zone and patency of visceral/renal arteries.

Alternatively, For aortic segments that involve vital arterial branches (e.g. renal arteries, celiac trunk, carotid artery and subclavian artery) a hybrid technique could be employed. During a hybrid repair, these vital branches are first passed surgically to a proximal or distal healthy aortic or iliac segment, and then an endograft is deployed [66, 67].

4.3 Indication for repair

Elective aortic aneurysm repair is indicated under any one of the following conditions [68, 69]:

- Rupture or dissection
- Acute dissection resulting in malperfusion or other life-altering complications
- Symptomatic states
  - Pain consistent with rupture and unexplained by other causes
  - Compression of adjacent organs
  - Documented enlargement ≥1 cm/year for AAA and descending TAA. Or > 0.5 cm/year for aortic arch aneurysm.
  - Diameter is greater than 5.5 cm for men, > 5 cm for women. Descending TAA with a diameter of >6 cm for men, >5.5 cm for women. While for Ascending TAA, surgical treatment is recommended for lower aortic diameter (5 cm for males).

Importantly, elective surgery, regardless of the modality and aneurysm location over the aorta carries lower mortality and morbidity in comparison to emergent surgery. Emergent surgery is done in life-threatening cases, such as aortic rupture or dissection, and should be avoided by encouraging patients to undergo elective aortic repair [70, 71].

4.4 Complications

Although different approaches of aortic aneurysm repair carry different risks, common complications are hemorrhage, myocardial infarction, renal failure, ileus, incisional hernia, ischemic colitis, embolization, and aneurysm rupture [65, 72]. Nevertheless, it appears that the endovascular repair method carries a lower short-term mortality rate than open repair [72, 73]. However, the biggest disadvantage of endovascular repair is the high risk of complications post-procedure that may need reintervention [74].

An endoleak is a complication that might appear in endovascular aortic repair, in which the graft fails to channel blood back to the systemic circulation. Endoleaks can subdivide into [67, 75]:

- Type I endoleak: an improper sealing between the proximal graft and aorta at the proximal or distal fixation zone.
- Type II endoleak: blood flow from collateral arteries flow to the aneurysmal sac.
- Type III endoleak: separation of stent-graft modular components leading to blood flow to the aneurysm sac.
- Type IV endoleak: blood flow through the pores of the stent-graft.
- Type V endoleak: blood flow into the aneurysmal sac from an unknown source.

Endovascular repair might also lead to access site complications such, perforation, hematoma, and fistula from vascular damage [72, 76].

Technical complications from the graft itself may also occur, with problems ranging from graft migration or kinking in the vessel, to graft infection. The incidence of endograft infection is less than 1 percent, with a mortality rate up to 50 percent [77].

Some studies have shown that ischemic complications are more common in endovascular compared to open repair of aortic aneurysms [78]. Causes of ischemia include occlusion due to endograft positioning, arterial thrombosis, embolism, or arterial dissection and can affect the distal limbs and body organs (intestines, kidneys, etc.).

Spinal ischemia is a rare complication in AAA endovascular repair, and is found in 0.21 percent of patients, but has an incidence of up to 12 percent in TAA repair [79]. This complication can manifest with findings such as paraplegia, pain or paralysis, typically developing within 12 hours after repair [80].

Although this complication is commonly seen in TAA, its mechanism continues to be ill-defined. In one study, perioperative hypotension (MAP <70 mmHg) was found to be a strong predictor of spinal cord ischemia in patients undergoing endovascular TAA repair [81]. Other contributing factors include long procedural duration, previous open infrarenal aortic repair, extent of aortic coverage by the device, endovascular leakage, or thrombosis of collateral blood flow. For spinal cord ischemia, spinal drainage is the most appropriate management, and is also used to reduce the risk of this ischemia [82].

5. Conclusion

Aortic aneurysms have a wide range of possible presentations and etiologies. Uncontrolled aortic aneurysms can lead to fatal outcomes. Therefore prevention, recommended screening, early diagnosis, regular monitoring, and prompt management are imperative to decrease mortality and complications.

DOI: http://dx.doi.org/10.5772/intechopen.97258

Author details

Akshay Kumar[1*], Purnadeo Persaud[2], Ana Francesca Leite[3], Nathaniel Aoyama[3], Ruan Matos[3], Dor Mordehay Avishay[4], Nimisha Shiwalkar[5], Vinit Garg[6], Juber Dastagir Shaikh[7], Roopvir Kaur[8] and Asim Kichloo[9]

1 Department of Cardiothoracic Surgery, University of Pittsburgh, Pittsburgh, PA, USA

2 Kansas City University, Kansas City, USA

3 University of State of Minas Gerais, Passos, Brazil

4 Department of Medicine, University of Milan, Milano, Italy

5 Department of Anaesthesiology, University of Texas Health Centre, Houston, TX, USA

6 Department of Anaesthesiology, Medanta Hospital, Gurugram, India

7 Department of Neurology, Mahatma Gandhi Hospital, Navi Mumbai, India

8 Department of Anesthesiology, Government Medical College, Amritsar, India

9 Department of Internal Medicine, Central Michigan University, Mt Pleasant, MI, USA

*Address all correspondence to: drakshay82@gmail.com

References

[1] JOHNSTON, K. Wayne et al. Suggested standards for reporting on arterial aneurysms. **Journal of vascular surgery**, v. 13, n. 3, p. 452-458, 1991.

[2] Black J, Burke C. Epidemiology, risk factors, pathogenesis, and natural history of thoracic aortic aneurysm and dissection. UpToDate. 2020

[3] Chung J. Epidemiology, risk factors, pathogenesis, and natural history of abdominal aortic aneurysm. UpToDate. 2021

[4] Chaikof EL, Dalman RL, Eskandari MK, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg 2018; 67:2.

[5] Moll FL, Powell JT, Fraedrich G, et al. Management of abdominal aortic aneurysms clinical practice guidelines of the European society for vascular surgery. Eur J Vasc Endovasc Surg 2011; 41 Suppl 1:S1.

[6] Centers for Disease Control and Prevention (CDC). **Aortic Aneurysm.** September 2020. Available at: https://www.cdc.gov/heartdisease/aortic_aneurysm.htm#:~:text=An%20aortic%20aneurysm%20is%20a,to%20leak%20in%20between%20them.

[7] 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.

[8] 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.

[9] Lindholt JS, Juul S, Fasting H, Henneberg EW. Screening for abdominal aortic aneurysms: single centre randomised controlled trial. BMJ 2005; 330:750.

[10] Ashton HA, Gao L, Kim LG, et al. Fifteen-year follow-up of a randomized clinical trial of ultrasonographic screening for abdominal aortic aneurysms. Br J Surg 2007; 94:696.

[11] Forsdahl SH, Singh K, Solberg S, Jacobsen BK. Risk factors for abdominal aortic aneurysms: a 7-year prospective study: the Tromsø Study, 1994-2001. Circulation 2009; 119:2202.

[12] Vardulaki KA, Prevost TC, Walker NM, et al. Incidence among men of asymptomatic abdominal aortic aneurysms: estimates from 500 screen detected cases. J Med Screen 1999; 6:50.

[13] Lederle FA, Johnson GR, Wilson SE, et al. Yield of repeated screening for abdominal aortic aneurysm after a 4-year interval. Aneurysm Detection and Management Veterans Affairs Cooperative Study Investigators. Arch Intern Med 2000; 160:1117.

[14] Wilmink AB, Hubbard CS, Day NE, Quick CR. The incidence of small abdominal aortic aneurysms and the change in normal infrarenal aortic diameter: implications for screening. Eur J Vasc Endovasc Surg 2001; 21:165.

[15] 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. J Am Coll Cardiol 2010; 55:e27.

[16] Bickerstaff LK, Pairolero PC, Hollier LH, et al. Thoracic aortic aneurysms: a population based study. Surgery 1982; 92:1103.

[17] Clouse WD, Hallett JW Jr, Schaff HV, et al. Improved prognosis of thoracic aortic aneurysms: a population-based study. JAMA 1998; 280:1926.

[18] Stackelberg O, Wolk A, Eliasson K, et al. Lifestyle and Risk of Screening-Detected Abdominal Aortic Aneurysm in Men. J Am Heart Assoc 2017; 6.

[19] Reed D, Reed C, Stemmermann G, Hayashi T. Are aortic aneurysms caused by atherosclerosis? Circulation 1992; 85:205.

[20] Pinard A, Jones GT, Milewicz DM. Genetics of Thoracic and Abdominal Aortic Diseases. Circ Res 2019; 124:588.

[21] Coady MA, Davies RR, Roberts M, et al. Familial patterns of thoracic aortic aneurysms. Arch Surg 1999; 134:361.

[22] Isselbacher EM. Thoracic and abdominal aortic aneurysms. Circulation 2005; 111:816.

[23] Elefteriades JA, Farkas EA. Thoracic aortic aneurysm clinically pertinent controversies and uncertainties. J Am Coll Cardiol 2010; 55:841.

[24] Elefteriades JA, Barrett PW, Kopf GS. Litigation in nontraumatic aortic diseases--a tempest in the malpractice maelstrom. Cardiology 2008; 109:263.

[25] Pressler V, McNamara JJ. Aneurysm of the thoracic aorta. Review of 260 cases. J Thorac Cardiovasc Surg 1985; 89:50.

[26] Wang Y, Barbacioru CC, Shiffman D, et al. Gene expression signature in peripheral blood detects thoracic aortic aneurysm. PLoS One 2007; 2:e1050.

[27] Trimarchi S, Sangiorgi G, Sang X, et al. In search of blood tests for thoracic aortic diseases. Ann Thorac Surg 2010; 90:1735.

[28] van Bogerijen GH, Tolenaar JL, Grassi V, et al. Biomarkers in TAA-the Holy Grail. Prog Cardiovasc Dis 2013; 56:109.

[29] Yuan SM, Shi YH, Wang JJ, et al. Elevated plasma D-dimer and hypersensitive C-reactive protein levels may indicate aortic disorders. Rev Bras Cir Cardiovasc 2011; 26:573.

[30] Diehm N, Baumgartner I. D-dimer measurement: a useful prognostic marker in surveillance of patients with abdominal aortic aneurysm? Eur Heart J 2011; 32:258.

[31] Golledge J, Muller R, Clancy P, et al. Evaluation of the diagnostic and prognostic value of plasma D-dimer for abdominal aortic aneurysm. Eur Heart J 2011; 32:354.

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

[33] 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.

[34] Braverman AC. Aortic involvement in patients with a bicuspid aortic valve. Heart. 2011 Mar;97(6):506-13. doi: 10.1136/hrt.2009.183871. PMID: 21339321.

[35] Wilmink TB, Quick Cr, Hubbard CS, Day NE. The influence of screening on

the incidence of ruptured abdominal aortic aneurysms. **J Vasc. Surg** 1999; 30: 203-08.

[36] Fink HA, Lederle FA, Roth CS, et al. The accuracy of physical examination to detect abdominal aortic aneurysm. Arch Intern Med 2000; 160:833.

[37] Van Walraven, C., Wong, J., Morant, K., Jennings, A., Jetty, P., & Forster, A. J. (2010). Incidence, follow-up, and outcomes of incidental abdominal aortic aneurysms. Journal of Vascular Surgery, 52(2), 282-289.e2. doi:10.1016/j.jvs.2010.03.006

[38] Sakalihasan, N., Limet, R., & Defawe, O. (2005). Abdominal aortic aneurysm. The Lancet, 365(9470), 1577-1589.doi:10.1016/s0140-6736(05)66459-8

[39] Farber, A., Wagner, W. H., Cossman, D. V., Cohen, J. L., Walsh, D. B., Fillinger, M. F., ... Levin, P. M. (2002). Isolated dissection of the abdominal aorta: Clinical presentation and therapeutic options. Journal of Vascular Surgery, 36(2), 205-210. doi:10.1067/mva.2002.125028

[40] Azhar B, Patel SR, Holt PJ, et al. Misdiagnosis of ruptured abdominal aortic aneurysm: systematic review and meta-analysis. J Endovasc Ther 2014; 21:568.

[41] Kiell CS, Ernst CB. Advances in management of abdominal aortic aneurysm. Adv Surg 1993; 26:73.

[42] Tsai YW, Blodgett JB, Wilson GS, et al. Ruptured abdominal aortic aneurysm. Pathognomonic triad. Vasc Surg 1973; 7:232.

[43] Rinckenbach S, Albertini JN, Thaveau F, et al. Prehospital treatment of infrarenal ruptured abdominal aortic aneurysms: a multicentric analysis. Ann Vasc Surg 2010; 24:308

[44] Assar AN, Zarins CK. Ruptured abdominal aortic aneurysm: a surgical emergency with many clinical presentations. Postgrad Med J 2009; 85:268.

[45] Nigro G, Giovannacci L, Engelberger S, et al. The challenge of posttraumatic thrombus embolization from abdominal aortic aneurysm causing acute limb ischemia. J Vasc Surg 2011; 54:840.

[46] Baxter BT, McGee GS, Flinn WR, et al. Distal embolization as a presenting symptom of aortic aneurysms. Am J Surg 1990; 160:197.

[47] Pennell RC, Hollier LH, Lie JT, et al. Inflammatory abdominal aortic aneurysms: a thirty-year review. J Vasc Surg 1985; 2:859.

[48] Rasmussen TE, Hallett JW Jr. Inflammatory aortic aneurysms. A clinical review with new perspectives in pathogenesis. Ann Surg 1997; 225:155.

[49] Cavallaro A, Sapienza P, di Marzo L, et al. [Inflammatory aneurysm of the abdominal aorta. Study of 355 patients with aortic aneurysm]. Recenti Prog Med 2001; 92:269.

[50] Hellmann DB, Grand DJ, Freischlag JA. Inflammatory abdominal aortic aneurysm. JAMA 2007; 297:395.

[51] David M, Pelberg J, Kuntz C. Grey Turner's sign. QJM 2013; 106:481.

[52] Diwan A, Sarkar R, Stanley JC, et al. Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysms. J Vasc Surg 2000; 31:863.

[53] Moxon JV, Parr A, Emeto TI, Walker P, Norman PE, Golledge J. Diagnosis and monitoring of abdominal aortic aneurysm: current status and future prospects. *Curr Probl Cardiol*. 2010;35(10):512-548. doi:10.1016/j.cpcardiol.2010.08.004

[54] Fatimi SH, Panni RZ, Ashfaq A. Incidental finding of a giant aortic root aneurysm and its repair. J Pak Med Assoc 2012; 62:617.

[55] Rylski B, Desjardins B, Moser W, et al. Gender-related changes in aortic geometry throughout life. Eur J Cardiothorac Surg 2014; 45:805.

[56] Safi HJ, Winnerkvist A, Miller CC 3rd, et al. Effect of extended cross-clamp time during thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 1998; 66:1204.

[57] Guirguis-Blake JM, Beil TL, Senger CA, Coppola EL. Triagem de cuidados primários para aneurisma de aorta abdominal: revisão sistemática atualizada para a força-tarefa de serviços preventivos dos EUA: Síntese de evidências n° 184 . Rockville, MD: Agency for Healthcare Research and Quality; 2019. Publicação AHRQ 19-05253-EF-1.

[58] Welch, HG; Schwartz, LM; Woloshin S. (2011). Overdiagnosed: Making people sick in the pursuit of health. Boston: Beacon Press.

[59] Fenton JJ, Weyrich MS, Durbin S, et al. Prostate-specific antigen–based screening for prostate cancer: A systematic evidence review for the U.S. Preventive Services Task Force. Agency for Healthcare Research and Quality 2018, Evidence Synthesis No. 154. AHRQ Publication No. 17-05229-EF-1.

[60] Vorp, David A. "Biomechanics of abdominal aortic aneurysm." Journal of biomechanics 40.9 (2007): 1887-1902.

[61] Creager, MA, Halperin, JL, Whittemore, AD. Aneurysmal disease of the aorta and its branches. In: Vascular Medicine, Loscalzo, J, Creager, MA, Dzau, VJ (Ed), Little, Brown, New York, 1996, p. 901

[62] Crawford ES, Cohen ES. Aortic aneurysm: a multifocal disease. Presidential address. Arch Surg 1982; 117:1393

[63] Nuenninghoff DM, Hunder GG, Christianson TJ, et al. Incidence and predictors of large artery complication (aortic aneurysm, aortic dissection, and/or large-artery stenosis) in patients with giant cell arteritis: a population-based study over 50 years. Arthritis Rheum 2003; 48:3522.

[64] Gowda RM, Khan IA. Thoracoabdominal aortic aneurysm: diagnosis and management. Curr Treat Options Cardiovasc Med. 2006;8(2):175-179. doi:10.1007/s11936-006-0010-1.

[65] David D. Yuh Luca A. Vricella John R. Doty. Johns Hopkins Textbook of Cardiothoracic Surgery second edition.

[66] Chiesa R, Civilini E, Melissano G, et al. Management of thoracoabdominal aortic aneurysms. HSR Proc Intensive Care Cardiovasc Anesth. 2009;1(1):45-53.

[67] Xydas S, Mihos CG, Williams RF, LaPietra A, Mawad M, Wittels SH, Santana O. Hybrid repair of aortic arch aneurysms: a comprehensive review. J Thorac Dis. 2017 Jun;9(Suppl 7):S629-S634

[68] Frederick JR, Woo YJ. Thoracoabdominal aortic aneurysm. Ann Cardiothorac Surg. 2012;1(3):277-285. doi:10.3978/j.issn.2225-319X.2012.09.01.

[69] ESC. Guidelines on the diagnosis and treatment of aortic diseases, 2014.

[70] Kawachi Y, Toshima Y, Nakashima A, Arinaga K, Komesu I. Emergency surgery results in life-threatening thoracic aortic disease. Jpn J Thorac Cardiovasc Surg. 2002 Apr;50(4):158-64. doi: 10.1007/BF02913197 Add to Citavi project by DOI. PMID: 11993197

[71] Timmers TK, van Herwaarden JA, de Borst GJ, Moll FL, Leenen LP. Long-term survival and quality of life after open abdominal aortic aneurysm repair. World J Surg. 2013 Dec;37(12):2957-64. doi: 10.1007/s00268-013-2206-3. PMID: 24132818

[72] Patel R, Sweeting MJ, Powell JT, Greenhalgh RM., EVAR trial investigators. Endovascular versus open repair of abdominal aortic aneurysm in 15-years' follow-up of the UK endovascular aneurysm repair trial 1 (EVAR trial 1): a randomised controlled trial. Lancet. 2016 Nov 12;388(10058):2366-2374.

[73] Conway KP, Byrne J, Townsend M, Lane IF. Prognosis of patients turned down for conventional abdominal aortic aneurysm repair in the endovascular and sonographic era: Szilagyi revisited? J Vasc Surg. 2001 Apr;33(4):752-7.

[74] Daye D, Walker TG. Complications of endovascular aneurysm repair of the thoracic and abdominal aorta: evaluation and management. *Cardiovasc Diagn Ther*. 2018;8(Suppl 1):S138-S156. doi:10.21037/cdt.2017.09.17

[75] Kim HO, Yim NY, Kim JK, Kang YJ, Lee BC. Endovascular Aneurysm Repair for Abdominal Aortic Aneurysm: A Comprehensive Review. Korean J Radiol. 2019 Aug;20(8):1247-1265.

[76] Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, Mastracci TM, Mell M, Murad MH, Nguyen LL, Oderich GS, Patel MS, Schermerhorn ML, Starnes BW. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg. 2018 Jan;67(1):2-77.e2

[77] Setacci C, Chisci E, Setacci F, et al. How To Diagnose and Manage Infected Endografts after Endovascular Aneurysm Repair. *Aorta (Stamford)*. 2014;2(6):255-264. Published 2014 Dec 1. doi:10.12945/j.aorta.2014.14-036

[78] Gupta PK, Ramanan B, Lynch TG, et al. Endovascular repair of abdominal aortic aneurysm does not improve early survival versus open repair in patients younger than 60 years. Eur J Vasc Endovasc Surg. 2012;43(5):506-12. http://dx.doi.org/10.1016/j.ejvs.2012.02.006. PMid:22386386

[79] Berg P, Kaufmann D, van Marrewijk CJ, et al. Spinal cord ischaemia after stent-graft treatment for infra-renal abdominal aortic aneurysms. Analysis of the Eurostar database. *Eur J Vasc Endovasc Surg* 2001; 22: 342-7.

[80] Awad H, Ramadan ME, El Sayed HF, Tolpin DA, Tili E, Collard CD. Spinal cord injury after thoracic endovascular aortic aneurysm repair. Lésion de la moelle épinière après réparation endovasculaire d'un anévrisme de l'aorte thoracique. *Can J Anaesth*. 2017;64(12):1218-1235. doi:10.1007/s12630-017-0974-1

[81] Awad, H., Ramadan, M. E., El Sayed, H. F., Tolpin, D. A., Tili, E., & Collard, C. D. (2017). *Spinal cord injury after thoracic endovascular aortic aneurysm repair. Canadian Journal of Anesthesia/Journal Canadien D'anesthésie, 64(12), 1218-1235.* doi:10.1007/s12630-017-0974-1

[82] Caton MT, Huff JS. Spinal Cord Ischemia. [Updated 2020 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539870/