We are IntechOpen, the world's leading publisher of Open Access books
Built by scientists, for scientists

5,200
Open access books available

128,000
International authors and editors

150M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS
BOOK CITATION INDEX
INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Aortic Aneurysm: A Surgical Point of View

Luciano Izzo, Daniela Messineo, Emiliano Venditti,
Virgilio Nicolanti, Sara Izzo and Paolo Izzo

Abstract

Aortic aneurysms are of different types as different ones are the types of treatment available to us. Following the advent of endovascular surgery, perioperative mortality has been significantly reduced, but open surgery remains the first choice under some occurrences. The purpose of this chapter is to try to clarify the dichotomy between open and endovascular aortic aneurysms in the several types of aortic aneurysms, highlighting the indications and complications to guide to the best therapeutic choice.

Keywords: EVAR, TEVAR, open, endovascular, AAA, TAA

1. Introduction

The aortic aneurysm is defined as a pathological condition characterized by permanent dilation of the aortic wall that most often occurs in the infra-renal region and the aortic arch. It generally presents asymptomatically, but progressive dilation can lead to the aorta rupture until the patient's death of the patient [1].

Based on autopsy studies, it is estimated that about 1–2% of the population has an aortic aneurysm, increasing to 10% in the older population [2]. In the United States, an aortic aneurysm rupture is in thirteenth place due to cause of death [2]. Abdominal aortic aneurysms (AAA) are the most common type, and most of them are atherosclerotic. The ascending tract aneurysms (ATAA) instead of the present as the most common etiology medial degeneration, a process normal in aging, but which accelerates pathologically if the person has other disorders, such as hypertension, bicuspid aortic valve, or genetic alterations [2].

2. Etiology

The etiology of aneurysm formation is not yet exact connoted. Aneurysmal disease main problem is the wear of the aortic wall, which progressively increases its dilation and the risk of rupture [3].

Biochemical studies have shown that there is a decrease in elastin in this pathology as opposed to a more significant increase in the share of collagen, increasing the ratio of collagen to elastin in the aneurysmal wall [4, 5].

Animal tests have highlighted that the aortic wall weakening is also due to the intervention of matrix metalloproteases, which act on the degradation of elastin and the destruction of smooth muscle cells [4].

Infections are an uncommon cause of aneurysmal pathology, with cases ranging from 0.65% to 1.3% of cases. Most are attributable to staphylococci, but streptococci, salmonella, and syphilis can also occur [6, 7].

Fungal infections are sporadic, but there are some documented cases [6].

Among the genetic disorders predisposing to this type of disease, we have Ehlers-Danlos, a hereditary connective tissue disorder, specifically type IV, which induces an autosomal dominant collagen type reduction III [8].

Another autosomal dominant genetic pathology of connective tissue is Marfan syndrome. People with this pathology present a mutation of the fibrillin gene, predisposing them to aortic aneurysms, dissection, mitral insufficiency, deformities of the chest wall, joint laxity, and increased arm and leg length [9].

Another one is the Loeys-Dietz syndrome, an autosomal dominant pathology characterized by arterial tortuosity and aneurysms, hypertelorism, and bifid uvula or cleft palate. To be suspected in cases of aortic dissection or aneurysmal rupture in young patients [10].

3. Natural course

Familial thoracic aortic aneurysms (TAA) grow faster, up to 2.1 mm/year. There are also various growth rates of syndromic TAA. In patients with Marfan syndrome, TAA growth rates are on average 0.5–1 mm/year, while TAA in patients with Loeys-Dietz syndrome (LDS) can grow even faster than 10 mm/year, causing death at an average age of 26 years [11–14].

Descending TAA (DTAA) grew more (at 3 mm/year) than ascending TAA (1 mm/year). Marfan syndrome patients with aortic valve disease and aortic dissection who underwent reparative surgery had TAA growth of 0.58 + 0.5 mm/year for the descending aorta. Factors influencing the growth of aortic diameters over time are hypertension, emergency procedures, and aortic dissection [15].

The dissection risk or rupture hazard increases rapidly when the aorta diameter is >60 mm for ATAA and > 70 mm for DTAA. Although dissection can occur in patients with a small aorta, the individual risk is very small [16].

AAA has a very long estimated asymptomatic growth period of approximately 1–6 mm/year. Several risk factors may contribute to its growth and progression such as genetic and environmental factors, among which smoking stands out as a determinant for rapid growth. The growth rate of the aneurysm increases in proportion to its size. Diameter has an exponential effect on the risk of rupture and women have a greater risk of rupture at diameters 10 mm smaller than men [17].

4. Clinic

An accurate anamnesis and objective examination must be performed. Most aneurysms are incidentalomas, occasional findings, highlighting chronicity, and growth rate. It investigates whether you are faced with a smoker if you have experienced weight loss, abdominal pain, fever, or intestinal bleeding over time. It is necessary to study the person from the renal point of view, as a situation of moderate or severe renal failure increases the morbidity and mortality of the subject. The patient's abdomen's objective examination is performed to see if a pulsatile

abdominal mass is appreciated. Also, a cardiological objective examination is performed for arrhythmias or valve defects [18].

For this pathology's diagnostic and therapeutic treatment, a division between thoracic aorta aneurysms and abdominal aneurysms is performed. This dichotomy is artificial, as there are thoracoabdominal aneurysms and the possibility of multiple lesions in both areas. The presence of an aortic aneurysm can be associated with aneurysms in other regions. During diagnostic tests, aneurysms of the iliac vessels can also be observed, but those of the popliteal vessels can escape instead. Some studies report that the prevalence of popliteal aneurysms on aortic aneurysm pathology maybe 14% [19].

People with TAA are often asymptomatic, and the diagnosis is often supported by images taken for other reasons or screening. In Marfan syndrome, aortic widening is generally maximum in Valsalva sinuses, responsible for anuloaortic ectasia [20].

Asymptomatic patient is also present in the abdominal aortic aneurysm. The sensitivity of the objective examination is low. People may present with atypical abdominal pain or lumbar pain, but it is not enough to alarm us for this pathology.

Acute abdominal pain with shock is often present in cases of aneurysmal rupture. Ultrasonography is an excellent tool for screening and surveillance, with no proven risks and low costs. It becomes useful during echocardiography to evaluate aortic necessary for the future investigated by other diagnostic tools [21].

The CT scan with intravenous contrast medium (IV contrast) is the diagnostic method for intervention planning. It also allows you to create a virtual angiogram with 3D reconstructions to study the organ ratios. It can also evaluate the coexistence of aneurysms or occlusive pathology at the level of the iliac or femoral artery sectors [22].

MRI with contrast medium, and without it, is the main examination in patients allergic to IV contrast or case of pregnancy of the patient and allows an accurate study of soft tissue, although it does not show calcifications within the aortic wall.

5. Intervention criteria

For the ATAA, the intervention is based on the diameter of the vessel and the patient's medical history, evaluating the risks and benefits of surgical intervention in the election. The surgery should be performed in patients with Marfan syndrome and a maximum diameter greater than or equal to 50 mm [23].

Diameters of 45 mm may be considered eligible for surgery in cases with multiple risk factors such as the family history of aortic dissection, increased diameter > 3 mm/year, severe aortic regurgitation, or desire for pregnancy [24].

Surgery should be considered in patients with a bicuspid aortic valve with a diameter greater than or equal to 55 mm. The limit may drop to 50 mm in patients with multiple risk factors, such as hypertension, aortic coarctation, familiarity, or a 3 mm/year increase in aneurysmal diameter. Without considering etiology, the surgery should be performed in patients with an aortic diameter greater than or equal to 55 mm.

The treatment is also indicated in patients with saccular aneurysms, pseudoaneurysms, symptomatic aneurysms and is also recommended for patients who need valve surgery, considering eligible those who have formations with diameters greater or equal to 45 mm [25, 26].

In aortic arch aneurysms, the surgery is performed for diameters greater than or equal to 55 mm or where the compressive symptomatology is very evident. The

decision must be accompanied by postoperative risk assessment, as replacement surgery is associated with increased mortality and stroke possibilities [27].

In descending aortic aneurysms, the use of stent-graft positioned by endovascular technique prevails. Endovascular surgery should be considered for aneurysms with a diameter greater than or equal to 55 mm. In cases where an open procedure must be performed, the indications are for diameters greater than or equal to 60 mm. Lower limits should be considered in patients with Marfan syndrome [28].

We recommend periodic ultrasound monitoring in AAA until it reaches diameters more significant than 55 mm, if it becomes symptomatic or if it increases its growth rate to values >10 mm/year [29].

6. Treatment

Until the advent of endovascular surgery, open surgery was the only possibility of treatment in cases of descending thoracic aortic aneurysm. Postoperative mortality from open surgery was 10% [30].

Over 40% of patients experience perioperative cardiopulmonary complications. According to some authors, perioperative mortality is about 3%, and the risk of complications like paraplegia increasing values between 0 and 4%. That is true in the open and the endovascular approach, and it may be due to spinal cord ischemia [31].

Since the use of stent-graft for endovascular surgery has been approved, this method has increasingly replaced open techniques. Post-operative mortality has decreased between 1.5% and 7%, with paraplegia risk between 1% and 3% [32, 33].

Open surgery should be considered in young patients with a low risk of perioperative complications.

Successful endovascular surgery depends on proximal implantation, distal to the left subclavian artery, and distal to the thoracic aorta or extending to the abdominal aorta above the celiac tripod. It is generally performed with one or more stent-graft tubes, and the choice of devices depends on the surgeon's anatomical characteristics and experience.

Open surgery remains an essential treatment for patients with altered anatomy, in young patients with few comorbidities, and in genetic collagen disorders. The operation involves replacing the aortic aneurysmal portion with a tube graft, performed by left thoracotomy [31].

During the clamping, perfusion is maintained by right or left cardiac by-pass, allowing for less postoperative morbidity and mortality [34].

With the left by-pass, the oxygenated blood is taken directly from the left pulmonary vein or left atrium and infused by the femoral artery's cannulation. The right by-pass allows greater hemodynamic control but requires higher levels of heparinization and therefore bleeding. The distal clamp is placed just below the proximal anastomosis, maintaining perfusion in almost the entire thoracic aorta as the proximal anastomosis is completed.

When distal aortic perfusion is not used, the repair is achieved with a clamp and seam technique, in which the aorta is clamped and repaired in sequence, with attention to speed to minimize distal ischemia time.

During the open operation, the number and location of the segmental arteries involved in the repair are predictive of the risk of spinal cord ischemia. In a wide range of open procedures, spinal cord ischemia was rare if the segmental arteries involved were < 8 but was 12.5% if >13. Before opening the aneurysm sac, the mental arteries are cut or clamped to avoid back bleeding and subsequent theft from the spinal cord [35].

Abdominal, thoracic aneurysms involve various extensions of the aorta, from the left subclavian artery to the aortic bifurcation, all with some degree of involvement of visceral segmentation. Currently, open surgery remains the therapy of choice in the United States. Successful surgery involves replacing the diseased aorta through a large left thoracoabdominal incision. When the entire visceral segment is involved, the renal and mesenteric arteries are reconstructed through a Carrel patch (typically a single patch for the right renal artery, the superior mesenteric artery, and the celiac trunk with a separate left renal by-pass), or a Coselli graft with a branch to each visceral vessel.

A branched graft is preferred for patients with congenital aortic disease, as these patients are at high risk of patch aneurysms. The need for exposure, dissection, and reconstruction of the visceral segment makes the repair of the thoracoabdominal aorta aneurysm technically complex and highly morbid. Postoperative mortality in high-volume centers has been reported as low as 8%. However, the National Surgical Quality Improvement Program and National In-patient Sample mortality was 10% and 22%, compared to 2% [36–38].

Half of the patients suffer from postoperative pulmonary, cardiac, and renal complications [36, 37].

Techniques have been developed to maintain distal perfusion and minimize renal and visceral ischemia during the reconstruction of the thoracoabdominal aorta aneurysm. In most cases, cardiopulmonary by-pass with femoral arterial cannulation is used to maintain distal perfusion. During the reconstruction of the visceral segment, renal ischemia is mitigated by direct perfusion of the renal arteries with cold crystalline solution or blood from the by-pass circuit. Visceral perfusion is also maintained with blood from the by-pass circuit. Besides, mild systemic hypothermia is often used to minimize the impact of ischemia [39].

There are no commercially available devices approved for total endovascular TAA repair in the United States. However, a hybrid type II TAA repair with thoracic aortic aneurysm repair (TEVAR) has been described, followed by open replacement of the remaining visceral segment and abdominal aorta involved. That can be performed either as a single-stage repair or as a 2-stage repair. While postoperative mortality is similar, there is evidence that a staged approach, either when performing a hybrid or open repair, is associated with a lower risk of spinal cord ischemia (SCI) [40, 41].

Aortic arch aneurysms are usually treated in conjunction with the treatment of an ascending or descending aneurysm. The arch is best exposed through a median sternotomy and most often is replaced with a branched graft with an individual branch for each vessel of the arch. Arch replacement is associated with significant early morbidity and mortality and involves substantial risk of neurological complications, both from cerebral ischemia and embolization. In most cases, arch repositioning is performed in hypothermic circulatory arrest. The cerebral blood flow can be maintained with selective cerebral perfusion prior to the degree of cerebral perfusion.

In the endovascular era, 2 techniques for hybrid aortic arch repair using a combination of open and TEVAR surgery have been developed. Eradication of the supraoptic arch involves by-passing all 3 branches of the arch from the ascending aorta, followed by the surgery TEVAR, including the arch. The proximal holding area is in the non-aneurysmal ascending aorta or proximal arch. The alternative approach is the endovascular modification of the elephant truck technique, used for patients with ascending and descending aneurysms. With this technique, the aortic arch is replaced with a branched graft that includes an extension of the distal tube graft. This is extended distally into the descending thoracic aorta beyond the distal anastomosis. The pathogenesis of the ascending aorta aneurysm is different from that of

descending aneurysms and abdominal aorta in many cases. It frequently occurs in patients with Marfan syndrome and other disorders of familial connective tissue or bicuspid aortic valves [42].

In addition, isolated ascending aortic aneurysms are rare, as most occur in conjunction with aortic root aneurysms and arch aneurysms. Therefore, ATAA repair is often performed in conjunction with additional procedures [43].

For aneurysms involving root aneurysms, surgery can be achieved by aortic root and/or ascending tract replacement and aortic valve replacement or reimplantation or composite aortic valve, root, and ascending aorta replacement with coronary artery reimplantation (Bentall procedure).

Recently, endovascular techniques have been introduced in the ascending aorta. The Zenith Ascend TAA Endovascular Graft (Cook Medical, Bloomington, IN) is a device dedicated to the ascending aorta that has been used for type A and ATAA dissection. However, its use remains experimental and is not approved by the Food and Drug Administration for commercial use in the United States [44].

Before the 1990s, open surgery was the only treatment option for abdominal aortic aneurysms and, while continuing to provide more remarkable survival than observation, was characterized by high morbidity and postoperative mortality [45].

However, the paradigm of intervention on AAA changed forever after the introduction of endovascular repair of AAA (EVAR), first described by Parodi et al. [46].

Since then, the use of endovascular techniques in treating aortic aneurysms has expanded considerably, and EVAR is now the primary treatment for AAA. Before the endovascular era, surgical mortality after AAA repair was ≈5%. After EVAR adoption, overall operating mortality from AAA repair dropped to 2.4% in 2008 [47].

The rapid adoption of EVAR has provided a AAA treatment option for many patients who are not candidates for open surgery. Instead, they remain patients with anatomical constraints that preclude the Use of EVAR. Young and otherwise, healthy patients may benefit from open surgery due to its longer duration. Also, patients with anatomical features that make it more likely to require revision, such as a highly angled neck, may benefit from an open approach. Finally, patients with connective tissue disorders should be treated with an open approach if possible, since long-term endovascular complications are inevitable in this population secondary to their disease progression. In addition to patients with substantial medical comorbidity, EVAR is preferred in patients for whom an open intervention would pose additional technical challenges, such as patients with a hostile abdomen. For patients who are eligible for both EVAR and Open, a shared decision-making process based on an in-depth discussion of each approach's risks and benefits is essential.

EVAR excludes an aneurysm from the bloodstream through the placement of a bifurcated stent, most introduced through the femoral arteries. The bag's exclusion depends on adequate proximal and distal sealing between the graft tissue and the vessel wall. For AAA under renal, the abdominal endovascular aortic aneurysm operation is performed with proximal fixation in the neck under renal and distal fixation in the common iliac arteries. The graft choice depends on anatomical criteria, the availability of the device, and the experience/preference of the surgeon. While each graft has been evaluated through prospective trials, the direct comparison between graft types is limited. In addition to neck length, angle, and diameter, other features that affect proximal hold are calcification of the neck, reverse tapering, and mural thrombus.

Some devices use suprarenal fixation to improve proximal tightness, where an uncovered stent extends over the renal arteries. Observational studies have shown

that suprarenal fixation is associated with a slightly higher risk of postoperative renal complications [48, 49]. When selecting a device that uses fixation above the renal arteries, this difference should be weighed against the potential improvement in proximal fixation.

Sub-renal devices are designed to fixate distally in the common iliac artery (CIA). However, aneurysmal CIAs presents a technical challenge as they prevent proper distal implantation. Surgeons have overcome this obstacle with 2 techniques: internal iliac artery embolization (IIA) and branched iliac devices.

Open AAA repair consists of replacing the aneurysmal segment with a synthetic graft. In most cases, a tubular graft from the renal neck to the bifurcation is enough. However, if the bifurcation or proximal CIA is diseased, a bifurcated graft can be used. The successful repair depends on the exposure of the abdominal aorta and proximal and distal vascular control.

For surgery, the abdominal aorta is exposed through a transperitoneal or retro-peritoneal approach.

A transperitoneal approach is performed with the patient in a supine position through a midline laparotomy. It provides rapid access to the distal abdominal aorta and bifurcation. The aneurysm's neck is exposed by packing the intestine into the right abdomen and dividing the Treitz ligament. A retroperitoneal approach is performed with the patient in the right lateral decubitus position through an incision on the left side that extends inferiorly parallel to the right abdomen. The peritoneal and retroperitoneal contents are moved anteromedially, exposing the entire abdominal aorta and the left iliac artery. It provides excellent visceral segment exposure; however, exposure of the right CIA bifurcation can be occasionally difficult. The choice of exposure depends on the patient's anatomy and the surgeon's preferences. Patients with hostile neck anatomy or short neck may benefit from a retroperitoneal approach and improved visceral segment exposure. Conversely, patients with right CIA aneurysms requiring a bifurcated graft may benefit from a transperitoneal approach.

With the aorta exposed, vascular control is achieved. During AAA repair, an aortic, renal cross-clamping position should be used whenever possible. Suprarenal clamping is associated with higher rates of acute renal injury (AKI) and higher overall complication rates [50].

Hostile neck anatomy may require an adrenal clamp position. In such cases, an attempt should be made to minimize renal ischemia time, and the clamp should be moved distally once the proximal anastomosis is completed. Distal control is achieved with bilateral CIA clamping or balloon occlusion if adequate distal control cannot be achieved through a cross-clamp.

Once access is obtained, the bag is opened, the thrombus is removed, and the aneurysmal segment is replaced with a synthetic graft [51].

7. Complications

Any manipulation of the aortic arch carries the risk of carotid artery embolization and subsequent stroke. This risk is magnified in patients with a very proximal implant area, mural thrombus in the arch, or history of previous stroke [52].

Stroke of the posterior circulation can also occur due to coverage of the left subclavian artery or embolization through the subclavian. Before TEVAR performing a carotid-subclavian by-pass can reduce this risk and benefit from improving the risk of paraplegia [53–55].

Paralysis due to spinal cord ischemia is a very feared risk in TEVAR. Risk factors include extensive coverage of the thoracic aorta, preventive AAA repair, and the left

subclavian artery coverage. Visceral ischemia can occur with intentional or unintentional coverage of the origin of the celiac artery. An intact pancreatic-duodenal arch may reduce this risk [56].

The large device diameter required for the TEVAR requires a relatively large sheath of 20 to 26F. It is essential to evaluate patient access through femoral and iliac vessels to preoperative CT to minimize access problems. Calcified, small, tortuous vessels are the most at risk. When an iliac rupture occurs, balloon occlusion can be used for temporary vascular control until the artery can be repaired. Reports suggest that 9.4 to 23.8% of patients require non-standard techniques for safe access [57–59].

Fever and leukocytosis may occur in the immediate postoperative period due to activation of the endothelium reacting to stent placement, to be considered in patients with elevated inflammatory markers and pleural effusions with an otherwise harmful infectious analysis [60–64].

Post-implantation graft migration occurs with an incidence from 1 to 2.8% [65]. Indeed, aortic tortuosity and graft oversizing are associated with migration risk [66].

Patients with traumatic aortic rupture may be particularly at risk of graft failure, possibly due to morphological differences in the unhealthy aorta [67]. Endoleak is relatively rare after TEVAR, with rates reported in the literature ranging from 3.9 to 15% [68].

The risk of death is about 4% in patients with uncomplicated aortic aneurysms. The most common etiology of death is myocardial infarction. For patients with a ruptured aortic aneurysm, mortality can reach 80%. The abdominal wall's retroperitoneal incision is associated with a weakening of the lateral abdominal wall muscles, resulting in swelling of up to 15% of patients. Trans peritoneal incision is associated with a 12–20% risk of ventral hernia formation [69, 70].

The para-anastomotic aneurysm risk is about 0.8% at 5 years, 6.2% at 10 years, and is close to 20–40% at 15 years after open surgery. Several factors influence the risk of postoperative renal failure. These include a longer renal ischemia time, the division of the left renal vein, the bilateral suprarenal aortic clamp position, and the use of additional renal artery procedures. In a juxta-renal aneurysm repair study, postoperative transient renal dysfunction occurred in 37% of patients, with the majority resolving by the end of hospital stay [71].

Colon ischemia can vary significantly in severity. It can be isolated to the mucosa of the colon, or it can be transmural. The incidence of colon ischemia is 1–3% after elective repair and up to 10% after the broken AAA emergency repair. A routine postoperative colonoscopy has been performed in centers where the incidence is even higher: 5–9% after elective repair and 15–60% after rupture. The etiology of colon ischemia may include non-obstructive ischemia due to shock or vasopressin medication, occlusion of the lower mesenteric artery and/or internal iliac arteries and/or atheroembolization. Risk factors associated with colon ischemia development have included the type of repair, rupture, duration of surgery, kidney disease, lung dysfunction, blood loss, femoral anastomosis, and loss of the hypogastric artery [72].

Sexual dysfunction is common in patients with peripheral arterial disease. As shown in the DREAM study, preoperative sexual dysfunction in the group of patients undergoing open surgery and, in the group, undergoing EVAR was 66% and 74%, respectively. The data analyzed after surgery show a greater level of sexual dysfunction in the open group and returned to normal after 1.5 months in the EVAR group and 3 months in the open group [73].

Postoperative sexual dysfunctions' incidence in the two treatments was estimated later, indicating 7.4% in open surgery and 4.7% in EVAR treatment,

equalizing sexual function at 1-year follow-up in the two groups according to the ACE analysis [74].

Graft infection complicates about 0.3% of all aortic operations. In endovascular and open aortic aneurysm operations, infection rates are low. Staphylococcus spp. is responsible for most infections followed by gram-negative bacteria. Infection tends to appear on average 3 years after surgery. Treatment includes removal of the graft and infected or necrotic tissue by extra-anatomic bypass or in situ repair [69, 75].

Advantages of extra-anatomic bypass include placement of a prosthetic graft through a noninfected field, excision in one or two settings, and easily accessible axillary and femoral arteries for relatively rapid surgery, but the risk of aortic stump rupture with fatal consequences is 33%. Extra-anatomic graft infection can be observed in up to 20% of patients and graft occlusion with limb loss in 27% of cases [76].

The extra-anatomic by-pass is not a viable option when the infrarenal aorta is insufficient to create an aortic stump. In this situation, an in-situ by-pass should be considered with the use of an antibiotic-impregnated graft, an aorta taken from a corpse, or an autologous venous by-pass. Normally rifampicin is used to impregnate the Dacron graft for antistaphylococcal and partially anti gram-negative action. Advantages of using Dacron include easy availability of materials and the absence of an aortic section. The procedure with neoaortoiliac system uses homologous grafts of femoropopliteal vein, which do not need anticoagulation therapy, to replace the injured wall and through the in-situ approach it is possible to avoid sectioning the aorta. Problems with this procedure include the need for a preintervention to obtain the vessel and the possibility that it may not meet the predetermined length [77].

An aortoenteric fistula (AEF) is a connection between the third or fourth portion of the duodenum and the aorta. Most aortoenteric fistulas occur after aortic surgery, making primary aortic fistulas an infrequent condition. The incidence of AEF has been reported up to 4% of aortic repairs. AEF causes aortic and graft infection and can cause bleeding. Symptomatology includes abdominal pain, fever, melena or hematemesis, and weight loss. Treatment includes control of excessive blood loss, peripheral organ perfusion and treatment of the infection.

The treatment option in open surgery involved the use of a bypass to allow resection of the infected tract and repair of the gastrointestinal tract injury. EVAR has also been studied in the setting of aortoenteric fistulas. In a revision of EVAR for AEF, persistent/recurrent infection or recurrent bleeding may occur in 44% of patients following the procedure and with a mortality rate of 29% due to septic complications in most cases [78].

Limb ischemia is a complication that can occur in up to 25% of cases of open surgery. It tends to occur more for grafts reaching the femoral arteries and in the female population [79].

The types of treatment are based on removal of the thrombus by endovascular secondary intervention or open. The most frequent complication in EVAR treatment involves endoleaks, which occur in up to 25% of cases in the postoperative period. There are different presentations of endoleaks. Type I represents incomplete adhesion between the aortic wall and the endovascular device, in turn dividing into two subtypes: Ia for the proximal complication and Ib for the distal one. When present, it must be treated as it is associated with a high risk of rupture due to increased pressure within the pouch [80, 81].

It can be treated with an open approach if resolution by endovascular surgery is not possible. The most common type of endoleak is type II, found in 10–20% of patients. The primary cause is a reversal of flow in the inferior mesenteric artery (IMA) or lumbar artery that carries blood within the aneurysmal sac. It undergoes spontaneous reslution in 50–60% of cases within 30 days [82].

Type II endoleaks were not associated with an increased risk of sac rupture, so they should not be treated. However, surgical treatment will be necessary in type II endoleaks if they are associated with increased pressure in the aneurysmal sac or if they do not spontaneously recede. They can be treated by retrograde trans arterial catheterization, direct puncture of the sac, or by laparoscopy.

Patients with type II endoleak had more significant complications (death, rupture, reintervention, or conversion to open surgery) and increased aneurysm sac enlargement. Type II endoleak was determined as a risk factor for the growth of the aneurysm diameter greater than 5 mm, especially if type II endoleak persisted for more than 6 months and was recurrent or associated with a type I and/or III endoleak [83].

A type III endoleak arises from a poor seal between the components or from a separation of the frank components. It is associated with the sacralization of the aneurysm and increased risk of rupture.

It must be treated when found. It can be treated either with a stent replacement for low leakage or with aortoiliac devices and a femoral-femoral by-pass for component separation.

A type IV endoleak refers to diffuse contrast redness seen occasionally immediately after implantation. It reflects the porosity of the graft material and is usually self-limiting and does not require treatment. Endoleak, also called type V endoleak, occurs when an aneurysm sac continues to be pressurized, and there is no exact source of endoleak. Management options include endograft realignment, open conversion with explantation and lumbar/IMA supervision if the culprit is found, or explantation with open surgery.

Aneurysm rupture after EVAR continues to be a significant driver of frequent postoperative follow-up in EVAR patients. The ACE study found a 2% incidence of aneurysm rupture after EVAR. Risk factors for aneurysm rupture included type Ia and III endoleak, graft migration, and graft bending [84].

Intermittent claudication after EVAR is usually related to the coverage of the hypogastric arteries by the stent. The ACE study found a 14% incidence of claudication after EVARs. In most patients, it usually resolves over time; however, in about 15% of patients, this complication persists [74, 85].

Author details

Luciano Izzo[1*], Daniela Messineo[2], Emiliano Venditti[2], Virgilio Nicolanti[1], Sara Izzo[3] and Paolo Izzo[1]

1 "Sapienza" University of Rome, Department of Surgery "Pietro Valdoni", Policlinico "Umberto I", Rome, Italy

2 Department of Radiological Sciences, Oncology and Pathology, Sapienza University, Rome, Italy

3 Human Neurosciences Department Neurosurgery Division "Sapienza" University, Italy

*Address all correspondence to: luciano.izzo@uniroma1.it

IntechOpen

References

[1] Davis FM, Daugherty A, Lu HS. Updates of Recent Aortic Aneurysm Research. *Arterioscler Thromb Vasc Biol*. 2019;39(3):e83-e90. doi:10.1161/ATVBAHA.119.312000

[2] Ladich E, Yahagi K, Romero ME, Virmani R. Vascular diseases: aortitis, aortic aneurysms, and vascular calcification. *Cardiovasc Pathol*. 2016;25(5):432-441. doi:10.1016/j.carpath.2016.07.002

[3] Golledge J, Muller J, Daugherty A, Norman P. Abdominal aortic aneurysm: pathogenesis and implications for management. *Arterioscler Thromb Vasc Biol*. 2006;26(12):2605-2613. doi:10.1161/01.ATV.0000245819.32762.cb

[4] van Vlijmen-van Keulen CJ, Pals G, Rauwerda JA. Familial abdominal aortic aneurysm: a systematic review of a genetic background. *Eur J Vasc Endovasc Surg*. 2002;24(2):105-116. doi:10.1053/ejvs.2002.1692

[5] Wills A, Thompson MM, Crowther M, Sayers RD, Bell PR. Pathogenesis of abdominal aortic aneurysms--cellular and biochemical mechanisms. *Eur J Vasc Endovasc Surg*. 1996;12(4):391-400. doi:10.1016/s1078-5884(96)80002-5

[6] Vallejo N, Picardo NE, Bourke P, et al. The changing management of primary mycotic aortic aneurysms. *J Vasc Surg*. 2011;54(2):334-340. doi:10.1016/j.jvs.2010.12.066

[7] Kyriakides C, Kan Y, Kerle M, Cheshire NJ, Mansfield AO, Wolfe JH. 11-year experience with anatomical and extra-anatomical repair of mycotic aortic aneurysms. *Eur J Vasc Endovasc Surg*. 2004;27(6):585-589. doi:10.1016/j.ejvs.2004.02.024

[8] Watanabe A, Shimada T. Vascular type of Ehlers-Danlos syndrome. *J Nippon Med Sch*. 2008;75(5):254-261. doi:10.1272/jnms.75.254

[9] Milewicz DM, Dietz HC, Miller DC. Treatment of aortic disease in patients with Marfan syndrome. *Circulation*. 2005;111(11):e150-e157. doi:10.1161/01.CIR.0000155243.70456.F4

[10] Loeys BL, Schwarze U, Holm T, et al. Aneurysm syndromes caused by mutations in the TGF-beta receptor. *N Engl J Med*. 2006;355(8):788-798. doi:10.1056/NEJMoa055695

[11] Blockmans D, de Ceuninck L, Vanderschueren S, Knockaert D, Mortelmans L, Bobbaers H. Repetitive 18F-fluorodeoxyglucose positron emission tomography in giant cell arteritis: a prospective study of 35 patients. *Arthritis Rheum*. 2006;55(1):131-137. doi:10.1002/art.21699

[12] Albornoz G, Coady MA, Roberts M, et al. Familial thoracic aortic aneurysms and dissections--incidence, modes of inheritance, and phenotypic patterns. *Ann Thorac Surg*. 2006;82(4):1400-1405. doi:10.1016/j.athoracsur.2006.04.098

[13] Kuzmik GA, Sang AX, Elefteriades JA. Natural history of thoracic aortic aneurysms. *J Vasc Surg*. 2012;56(2):565-571. doi:10.1016/j.jvs.2012.04.053

[14] Detaint D, Michelena HI, Nkomo VT, Vahanian A, Jondeau G, Sarano ME. Aortic dilatation patterns and rates in adults with bicuspid aortic valves: a comparative study with Marfan syndrome and degenerative aortopathy. *Heart*. 2014;100(2):126-134. doi:10.1136/heartjnl-2013-304920

[15] Kari FA, Russe MF, Peter P, et al. Late complications and distal growth rates of Marfan aortas after proximal aortic repair. *Eur J Cardiothorac Surg*.

2013;44(1):163-171. doi:10.1093/ejcts/ezs674

[16] Coady MA, Rizzo JA, Hammond GL, et al. What is the appropriate size criterion for resection of thoracic aortic aneurysms?. *J Thorac Cardiovasc Surg*. 1997;113(3):476-491. doi:10.1016/S0022-5223(97)70360-X

[17] Badger SA, Jones C, McClements J, Lau LL, Young IS, Patterson CC. Surveillance strategies according to the rate of growth of small abdominal aortic aneurysms. *Vasc Med*. 2011;16(6):415-421. doi:10.1177/1358863X11423971

[18] Calero A, Illig KA. Overview of aortic aneurysm management in the endovascular era. *Semin Vasc Surg*. 2016;29(1-2):3-17. doi:10.1053/j.semvascsurg.2016.07.003

[19] Gómez-Moreno S, Sabaté M, Jiménez-Quevedo P, et al. Iatrogenic dissection of the ascending aorta following heart catheterisation: incidence, management and outcome. *EuroIntervention*. 2006;2(2):197-202.

[20] Schaefer BM, Lewin MB, Stout KK, et al. The bicuspid aortic valve: an integrated phenotypic classification of leaflet morphology and aortic root shape. *Heart*. 2008;94(12):1634-1638. doi:10.1136/hrt.2007.132092

[21] Erbel R, Aboyans V, Boileau C, et al. Wytyczne ESC dotyczące rozpoznawania i leczenia chorób aorty w 2014 roku [2014 ESC Guidelines on the diagnosis and treatment of aortic diseases]. *Kardiol Pol*. 2014;72(12):1169-1252. doi:10.5603/KP.2014.0225

[22] Rutherford RB. Management of abdominal aortic aneurysms: which risk factors play a role in decision-making?. *Semin Vasc Surg*. 2008;21(3):124-131. doi:10.1053/j.semvascsurg.2008.05.005

[23] Jondeau G, Detaint D, Tubach F, et al. Aortic event rate in the Marfan population: a cohort study. *Circulation*. 2012;125(2):226-232. doi:10.1161/CIRCULATIONAHA.111.054676

[24] Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology (ESC); European Association for Cardio-Thoracic Surgery (EACTS), Vahanian A, et al. Guidelines on the management of valvular heart disease (version 2012). *Eur Heart J*. 2012;33(19):2451-2496. doi:10.1093/eurheartj/ehs109

[25] 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 [published correction appears in circulation. 2010 Jul 27;122(4):e410]. *Circulation*. 2010;121(13):e266-e369. doi:10.1161/CIR.0b013e3181d4739e

[26] Riambau V, Böckler D, Brunkwall J, et al. Editor's Choice - Management of Descending Thoracic Aorta Diseases: Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS). *Eur J Vasc Endovasc Surg*. 2017;53(1):4-52. doi:10.1016/j.ejvs.2016.06.005

[27] Chiesa R, Melissano G, Tshomba Y, et al. Ten years of endovascular aortic arch repair. *J Endovasc Ther*. 2010;17(1):1-11. doi:10.1583/09-2884.1

[28] Patterson B, Holt P, Nienaber C, Cambria R, Fairman R, Thompson M.

Aortic pathology determines midterm outcome after endovascular repair of the thoracic aorta: report from the Medtronic Thoracic Endovascular Registry (MOTHER) database. *Circulation*. 2013;127(1):24-32. doi:10.1161/CIRCULATIONAHA.112.110056

[29] Lederle FA, Wilson SE, Johnson GR, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Engl J Med*. 2002;346(19):1437-1444. doi:10.1056/NEJMoa012573

[30] Schermerhorn ML, Giles KA, Hamdan AD, Dalhberg SE, Hagberg R, Pomposelli F. Population-based outcomes of open descending thoracic aortic aneurysm repair. *J Vasc Surg*. 2008;48(4):821-827. doi:10.1016/j.jvs.2008.05.022

[31] Sadek M, Abjigitova D, Pellet Y, Rachakonda A, Panagopoulos G, Plestis K. Operative outcomes after open repair of descending thoracic aortic aneurysms in the era of endovascular surgery. *Ann Thorac Surg*. 2014;97(5):1562-1567. doi:10.1016/j.athoracsur.2014.01.046

[32] Wang GJ, Jackson BM, Foley PJ, et al. National trends in admissions, repair, and mortality for thoracic aortic aneurysm and type B dissection in the National Inpatient Sample. *J Vasc Surg*. 2018;67(6):1649-1658. doi:10.1016/j.jvs.2017.09.050

[33] Davies JE. Endovascular repair of descending thoracic aortic aneurysm: review of literature. *Semin Thorac Cardiovasc Surg*. 2009;21(4):341-346. doi:10.1053/j.semtcvs.2009.12.002

[34] Mohebali J, Carvalho S, Lancaster RT, et al. use of extracorporeal by-pass is associated with improved outcomes in open thoracic and thoracoabdominal aortic aneurysm

repair. *J Vasc Surg*. 2018;68(4):941-947. doi:10.1016/j.jvs.2017.12.072

[35] Zoli S, Roder F, Etz CD, et al. Predicting the risk of paraplegia after thoracic and thoracoabdominal aneurysm repair. *Ann Thorac Surg*. 2010;90(4):1237-1245. doi:10.1016/j.athoracsur.2010.04.091

[36] Bensley RP, Curran T, Hurks R, et al. Open repair of intact thoracoabdominal aortic aneurysms in the American College of Surgeons National Surgical Quality Improvement Program. *J Vasc Surg*. 2013;58(4):894-900. doi:10.1016/j.jvs.2013.03.037

[37] Cowan JA Jr, Dimick JB, Henke PK, Huber TS, Stanley JC, Upchurch GR Jr. Surgical treatment of intact thoracoabdominal aortic aneurysms in the United States: hospital and surgeon volume-related outcomes. *J Vasc Surg*. 2003;37(6):1169-1174. doi:10.1016/s0741-5214(03)00085-5

[38] Coselli JS, LeMaire SA, Preventza O, et al. Outcomes of 3309 thoracoabdominal aortic aneurysm repairs. *J Thorac Cardiovasc Surg*. 2016;151(5):1323-1337. doi:10.1016/j.jtcvs.2015.12.050

[39] Waked K, Schepens M. State-of the-art review on the renal and visceral protection during open thoracoabdominal aortic aneurysm repair. *J Vis Surg*. 2018;4:31. Published 2018 Feb 8. doi:10.21037/jovs.2018.01.12

[40] Hawkins RB, Mehaffey JH, Narahari AK, et al. Improved outcomes and value in staged hybrid extent II thoracoabdominal aortic aneurysm repair. *J Vasc Surg*. 2017;66(5):1357-1363. doi:10.1016/j.jvs.2017.03.420

[41] Etz CD, Zoli S, Mueller CS, et al. Staged repair significantly reduces paraplegia rate after extensive thoracoabdominal aortic aneurysm repair. *J Thorac Cardiovasc Surg*.

2010;139(6):1464-1472. doi:10.1016/j.
jtcvs.2010.02.037

[42] Isselbacher EM. Thoracic
and abdominal aortic aneurysms.
Circulation. 2005;111(6):816-
828. doi:10.1161/01.
CIR.0000154569.08857.7A

[43] Beller CJ, Farag M,
Wannaku S, et al. Gender-specific
differences in outcome of ascending
aortic aneurysm surgery. *PLoS One*.
2015;10(4):e0124461. Published
2015 Apr 22. doi:10.1371/journal.
pone.0124461

[44] Tsilimparis N, Debus ES, Oderich GS,
et al. International experience with
endovascular therapy of the ascending
aorta with a dedicated endograft.
J Vasc Surg. 2016;63(6):1476-1482.
doi:10.1016/j.jvs.2015.12.027

[45] Szilagyi DE, Smith RF,
DeRusso FJ, Elliott JP, Sherrin FW.
Contribution of abdominal aortic
aneurysmectomy to prolongation
of life. *Ann Surg*. 1966;164(4):678-
699. doi:10.1097/00000658-
196610000-00014

[46] Parodi JC, Palmaz JC, Barone HD.
Transfemoral intraluminal graft
im- plantation for abdominal aortic
aneurysms. *Ann Vasc Surg*. 1991;5:491-
499. doi: 10.1007/BF02015271

[47] Schermerhorn ML, Bensley RP,
Giles KA, et al. Changes in abdominal
aortic aneurysm rupture and short-term
mortality, 1995-2008: a retrospective
observational study. *Ann Surg*.
2012;256(4):651-658. doi:10.1097/
SLA.0b013e31826b4f91

[48] Zettervall SL, Soden PA,
Deery SE, et al. Comparison of Renal
Complications between Endografts with
Suprarenal and Infrarenal Fixation. *Eur
J Vasc Endovasc Surg*. 2017;54(1):5-11.
doi:10.1016/j.ejvs.2017.02.001

[49] Zettervall SL, Deery SE,
Soden PA, et al. Editor's Choice - Renal
complications after EVAR with
suprarenal versus infrarenal fixation
among all users and routine users. *Eur J
Vasc Endovasc Surg*. 2017;54(3):287-293.
doi:10.1016/j.ejvs.2017.05.012

[50] Chong T, Nguyen L, Owens CD,
Conte MS, Belkin M. Suprarenal aortic
cross-clamp position: a reappraisal of its
effects on outcomes for open abdominal
aortic aneurysm repair. *J Vasc Surg*.
2009;49(4):873-880. doi:10.1016/j.
jvs.2008.10.057

[51] Swerdlow NJ, Wu WW,
Schermerhorn ML. Open and
Endovascular Management of
Aortic Aneurysms. *Circ Res*.
2019;124(4):647-661. doi:10.1161/
CIRCRESAHA.118.313186

[52] Gutsche JT, Cheung AT,
McGarvey ML, et al. Risk factors for
perioperative stroke after thoracic
endovascular aortic repair. *Ann
Thorac Surg*. 2007;84(4):1195-1200.
doi:10.1016/j.athoracsur.2007.04.128

[53] Feezor RJ, Martin TD, Hess PJ,
et al. Risk factors for perioperative
stroke during thoracic endovascular
aortic repairs (TEVAR). *J Endovasc
Ther*. 2007;14(4):568-573.
doi:10.1177/152660280701400420

[54] Woo EY, Mcgarvey M, Jackson BM,
Bavaria JE, Fairman RM, Pochettino A.
Spinal cord ischemia may be reduced
via a novel technique of intercostal
artery revascularization during open
thoracoabdominal aneurysm repair.
J Vasc Surg. 2007;46(3):421-426.
doi:10.1016/j.jvs.2007.04.048

[55] Noor N, Sadat U, Hayes PD,
Thompson MM, Boyle JR. Management
of the left subclavian artery during
endovascular repair of the thoracic
aorta. *J Endovasc Ther*. 2008;15(2):168-
176. doi:10.1583/08-2406.1

[56] Vaddineni SK, Taylor SM, Patterson MA, Jordan WD Jr. Outcome after celiac artery coverage during endovascular thoracic aortic aneurysm repair: preliminary results. *J Vasc Surg*. 2007;45(3):467-471. doi:10.1016/j.jvs.2006.11.040

[57] Matsumura JS, Cambria RP, Dake MD, et al. International controlled clinical trial of thoracic endovascular aneurysm repair with the Zenith TX2 endovascular graft: 1-year results. *J Vasc Surg*. 2008;47(2):247-257. doi:10.1016/j.jvs.2007.10.032

[58] Najibi S, Terramani TT, Weiss VJ, et al. Endoluminal versus open treatment of descending thoracic aortic aneurysms. *J Vasc Surg*. 2002;36(4):732-737.

[59] Stone DH, Brewster DC, Kwolek CJ, et al. Stent-graft versus open-surgical repair of the thoracic aorta: mid-term results. *J Vasc Surg*. 2006;44(6):1188-1197. doi:10.1016/j.jvs.2006.08.005

[60] Gabriel EA, Locali RF, Romano CC, Duarte AJ, Palma JH, Buffolo E. Analysis of the inflammatory response in endovascular treatment of aortic aneurysms. *Eur J Cardiothorac Surg*. 2007;31(3):406-412. doi:10.1016/j.ejcts.2006.11.053

[61] Ishida M, Kato N, Hirano T, Shimono T, Shimpo H, Takeda K. Thoracic CT findings following endovascular stent-graft treatment for thoracic aortic aneurysm. *J Endovasc Ther*. 2007;14(3):333-341. doi:10.1583/06-1955.1

[62] Eggebrecht H, Mehta RH, Metozounve H, et al. Clinical implications of systemic inflammatory response syndrome following thoracic aortic stent-graft placement. *J Endovasc Ther*. 2008;15(2):135-143. doi:10.1583/07-2284.1

[63] Sakai T, Dake MD, Semba CP, et al. Descending thoracic aortic aneurysm:

thoracic CT findings after endovascular stent-graft placement. *Radiology*. 1999;212(1):169-174. doi:10.1148/radiology.212.1.r99jl37169

[64] Schoder M, Cartes-Zumelzu F, Grabenwöger M, et al. Elective endovascular stent-graft repair of atherosclerotic thoracic aortic aneurysms: clinical results and midterm follow-up. *AJR Am J Roentgenol*. 2003;180(3):709-715. doi:10.2214/ajr.180.3.1800709

[65] Makaroun MS, Dillavou ED, Wheatley GH, Cambria RP; Gore TAG Investigators. Five-year results of endovascular treatment with the Gore TAG device compared with open repair of thoracic aortic aneurysms. *J Vasc Surg*. 2008;47(5):912-918. doi:10.1016/j.jvs.2007.12.006

[66] Sternbergh WC 3rd, Money SR, Greenberg RK, Chuter TA; Zenith Investigators. Influence of endograft oversizing on device migration, endoleak, aneurysm shrinkage, and aortic neck dilation: results from the Zenith Multicenter Trial. *J Vasc Surg*. 2004;39(1):20-26. doi:10.1016/j.jvs.2003.09.022

[67] Tadros RO, Lipsitz EC, Chaer RA, Faries PL, Marin ML, Cho JS. A multicenter experience of the management of collapsed thoracic endografts. *J Vasc Surg*. 2011;53(5):1217-1222. doi:10.1016/j.jvs.2010.10.119

[68] Preventza O, Wheatley GH 3rd, Ramaiah VG, et al. Management of endoleaks associated with endovascular treatment of descending thoracic aortic diseases. *J Vasc Surg*. 2008;48(1):69-73. doi:10.1016/j.jvs.2008.02.032

[69] 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-S49. doi:10.1016/j.
jvs.2009.07.002

[70] Gruppo M, Mazzalai F, Lorenzetti R,
Piatto G, Toniato A, Ballotta E. Midline
abdominal wall incisional hernia
after aortic reconstructive surgery:
a prospective study. *Surgery*.
2012;151(6):882-888. doi:10.1016/j.
surg.2011.12.032

[71] Dubois L, Durant C,
Harrington DM, Forbes TL, Derose G,
Harris JR. Technical factors are strongest
predictors of postoperative renal
dysfunction after open transperitoneal
juxtarenal abdominal aortic aneurysm
repair. *J Vasc Surg*. 2013;57(3):648-654.
doi:10.1016/j.jvs.2012.09.043

[72] Becquemin JP, Majewski M,
Fermani N, et al. Colon ischemia
following abdominal aortic aneurysm
repair in the era of endovascular
abdominal aortic repair. *J Vasc Surg*.
2008;47(2):258-263. doi:10.1016/j.
jvs.2007.10.001

[73] Prinssen M, Buskens E,
Nolthenius RP, van Sterkenburg SM,
Teijink JA, Blankensteijn JD. Sexual
dysfunction after conventional
and endovascular AAA repair:
results of the DREAM trial. *J
Endovasc Ther*. 2004;11(6):613-620.
doi:10.1583/04-1280R.1

[74] Becquemin JP, Pillet JC, Lescalie F,
et al. A randomized controlled trial of
endovascular aneurysm repair versus
open surgery for abdominal aortic
aneurysms in low- to moderate-risk
patients. *J Vasc Surg*. 2011;53(5):1167-
1173.e1. doi:10.1016/j.jvs.2010.
10.124

[75] Laser A, Baker N, Rectenwald J,
Eliason JL, Criado-Pallares E, Upchurch
GR Jr. Graft infection after endovascular
abdominal aortic aneurysm repair. *J Vasc
Surg*. 2011;54(1):58-63. doi:10.1016/j.
jvs.2010.11.111

[76] Lew W, Moore W. Antibiotic-
impregnated grafts for aortic
reconstruction. *Semin Vasc Surg*.
2011;24(4):211-219. doi:10.1053/j.
semvascsurg.2011.10.015

[77] Chung J, Clagett GP. Neoaortoiliac
System (NAIS) procedure for the
treatment of the infected aortic graft.
Semin Vasc Surg. 2011;24(4):220-226.
doi:10.1053/j.semvascsurg.2011.10.012

[78] Setacci C, de Donato G,
Setacci F. Endografts for the treatment
of aortic infection. *Semin Vasc Surg*.
2011;24(4):242-249. doi:10.1053/j.
semvascsurg.2011.10.009

[79] EVAR trial participants.
Endovascular aneurysm repair versus
open repair in patients with abdominal
aortic aneurysm (EVAR trial 1):
randomised controlled trial. *Lancet*.
2005;365(9478):2179-2186. doi:10.1016/
S0140-6736(05)66627-5

[80] Arko FR 3rd, Murphy EH,
Boyes C, et al. Current status of
endovascular aneurysm repair: 20
years of learning. *Semin Vasc Surg*.
2012;25(3):131-135. doi:10.1053/j.
semvascsurg.2012.08.002

[81] Vallabhaneni SR, Harris PL.
Lessons learnt from the EUROSTAR
registry on endovascular repair of
abdominal aortic aneurysm repair. *Eur
J Radiol*. 2001;39(1):34-41. doi:10.1016/
s0720-048x(01)00340-0

[82] van Marrewijk C, Buth J,
Harris PL, Norgren L, Nevelsteen A,
Wyatt MG. Significance of endoleaks
after endovascular repair of abdominal
aortic aneurysms: The EUROSTAR
experience. *J Vasc Surg*. 2002;35(3):461-
473. doi:10.1067/mva.2002.118823

[83] El Batti S, Cochennec F, Roudot-
Thoraval F, Becquemin JP. Type II
endoleaks after endovascular repair
of abdominal aortic aneurysm are not
always a benign condition. *J Vasc Surg*.

2013;57(5):1291-1297. doi:10.1016/j. jvs.2012.10.118

[84] Harris PL, Vallabhaneni SR, Desgranges P, Becquemin JP, van Marrewijk C, Laheij RJ. Incidence and risk factors of late rupture, conversion, and death after endovascular repair of infrarenal aortic aneurysms: the EUROSTAR experience. European Collaborators on Stent/graft techniques for aortic aneurysm repair. *J Vasc Surg*. 2000;32(4):739-749. doi:10.1067/ mva.2000.109990

[85] Caporale A, Cosenza UM, Galati G, et al. Criteri prognostici associati negli aneurismi dell'aorta addominale rotti [Prognostic criteria in ruptured aneurysm of the abdominal aorta]. *G Chir*. 1999;20(10):393-396