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Complications of Endovascular Aortoiliac Aneurysm Repair – Narrative Review

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Abstract

Endovascular Abdominal Aortic Aneurysm Repair (EVAR) complications can be divided into early and late complications. Early complications can be systemic when there is dysfunction of one or more organs, device complications, vascular access, or a combination of these. The most common vascular access complications are surgical site infections, postoperative hematoma, femoral artery injury, pseudoaneurysm, and local lymphocele or seroma. The use of percutaneous arterial closure devices provides a feasible and less invasive option for EVAR, with satisfactory technical success rates. Moreover, local complications may occasionally cause or worsen systemic dysfunctions. The current early mortality rate from EVAR is 0.6% to 1.7%. In large registry studies, the major complications rate such as myocardial infarction, pneumonia, and acute renal injury can be as high as 7.0%, 9.3%, and 5.5%, respectively. Endoleaks are the most common complication during follow-up, reaching 30% in 5 years, although kinking and thrombosis of the iliac branches are also frequent. Undoubtedly, compared to open repair (OSR), the main disadvantage of the endovascular technique is the high rate of reinterventions that, in the majority, are done to treat endoleaks. Regarding late mortality, well-conducted randomized studies demonstrate an advantage of the endovascular technique in the first two years, mainly due to lower early mortality. However, there is a convergence in the survival curves after two years, and the initial advantage of the endovascular technique is lost in the very long term. Over time, there is a trend of more aneurysm-related deaths in patients undergoing the endovascular technique. This is one of the reasons that recently led some publications still to recommend OSR for those patients with good clinical conditions.

Keywords: Endovascular aneurysm repair; Aneurysm-related mortality; Complications; Abdominal aortic aneurysm; Device-related complications

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Introduction

The complications described in this review are essentially connected with endovascular aortic aneurysm repair, or endovascular infrarenal aortoiliac aneurysms repair, using conventional endograft. The repair of juxtarenal, pararenal, or thoracoabdominal aneurysms is not considered since this treatment requires more complex endovascular techniques, such as parallel stents or fenestrated branched stent-grafts. Therefore, this article aims to describe, as a form of a narrative review, the most common complications of Endovascular Aortic Aneurysm Repair (EVAR) and its different systemic and device-related issues in a structured manner.

Overall Mortality, Aneurysm-Related Mortality, and Systemic Complications

A systematic literature review with inclusion of the four main randomized controlled trials (RCT; EVAR-1 [1], DREAM [2], OVER [3], ACE [4]) has reported lower early and in-hospital mortality rates related to endovascular technique (1.4% *vs.* 4.2%; p<0.001) when compared to Open Surgical Repair (OSR) [5]. In EVAR trial 2, where high surgical risk patients were randomized into endovascular repair or "non-intervention", early mortality rate for EVAR was 9%, and no differences of overall mortality or aneurysm-related mortality occurred [6]. In a recent retrospective study, when analyzing the Vascular Quality Initiative/Society of Vascular Surgery database, patients who underwent EVAR, considered ineligible for open repair, showed a mortality rate of 1.7% in 30 days. This rate is much lower than EVAR trial 2; however, it is still significantly higher than those considered eligible (1.7 *vs.* 0.6%, respectively; p<0.001) [7].

The survival time at short-term follow-up (up to six months) still reveals an advantage of endovascular technique, especially due to better results in the first 30 days. Nevertheless, at mid-

term (six months to four years) and long-term (more than four years) follow-up, OSR and EVAR do not show differences regarding overall mortality, creating a survival curve convergence. Aneurysm-related deaths are also similar between the techniques [5,8], yet, a study using long-term follow-up data of EVAR trial-1 indicated a gradual increase in endovascular technique mortality rates. This increase started four years after EVAR, and reached a maximum after eight years of follow-up, making it the major cause of aneurysm-related deaths because of aneurysm sac rupture [9,10]. A pertinent remark concerning the results from those RCTs conducted must be made since the types of endograft used were mostly first and second-generation. Newer generation stent-grafts are those introduced after 2004, when major device changes were applied by manufacturers of the Excluder (W.L. Gore & Associates, Flagstaff, AZ, USA) and Anaconda (Vascutek, a Terumo Company, Inchinnan, Scotland) stent-grafts. First generation Endurant (Medtronic Cardiovascular, Santa Rosa, CA, USA) and Zenith (Cook Inc., Bloomington, IN, USA) stent grafts are also considered new-generation stent grafts. The modern version of stent-graft design is a bifurcated graft, most commonly using a modular system to allow for the most flexibility with regard to patient anatomy. Other advantages of the newer devices are full structural support with higher flexibility, better design leading to securer suturing of stents to the fabric to prevent excessive motion over time, more robust and low-porosity fabric and smaller delivery systems. Most current stent-graft have suprarenal stents to improve radial force and barbs to provide active fixation and, therefore, to inhibit downward migration. Long-term results with those new ones are yet to be revealed. Therefore, this data may not reflect the current situation correctly and must be evaluated cautiously. Recently, NICE Guideline recommendations have been a counterpoint to the endovascular technique indication since they recommend open surgical repair of AAA as the first alternative, provided no conditions such as hostile abdomen, horseshoe kidney, or high anesthetic-surgical risk exist. This guideline, however, recommends EVAR as the first alternative for repair of ruptured abdominal aortic aneurysm [11].

Lederle et al. [3] have reported post-EVAR myocardial infarction and stroke rates of 1.4% and 1.6%, respectively, in the first of follow-up year. This trial did not show significant differences compared to open repair [3]. On the other hand, lower rates of pulmonary complications in patients undergoing endovascular repair were seen [5].

The incidence of Acute Kidney Injury (AKI) after EVAR is reported to be between 1.1% and 18%, while some studies suggest lower rates for endovascular technique not confirmed by RCTs, which, as a rule, include selected and eligible individuals for both techniques [12-14]. The high variability of this outcome can be explained, in parts, not only by the study heterogeneity and patients' profile but also due to different diagnostic criteria used in each study.

Perioperative Complications

EVAR, compared to OSR, has, on average, shorter procedure time, reduced intraoperative blood loss, and shorter hospital length of stay [15-18]. A retrospective study, where data from Medicare (USA) beneficiaries subjected to AAA repair was used, reported lower complication rates after EVAR, in contrast to open repair, as well as lower rates of myocardial infarction (7.0% *vs.* 9.4%, p<0.001), pneumonia (9.3% *vs.* 17%, p<0.001), acute kidney dysfunction (5.5% *vs.* 11%, p<0.001), dialysis (0.4% *vs.* 0.6%, p<0.001), acute mesenteric ischemia (2.1% *vs.* 1.0%, p<0.001), bleeding-related reintervention (1.2% *vs.* 0.8%, p<0.001), embolectomy (1.7% *vs.* 1.3%, p<0.001), and tracheostomy (1.6% *vs.* 0.2%, p<0.001). Abdominal complications were also greater in the open repair group, including enterectomies (1.3% *vs.* 0.6%, p<0.001), and bowel obstruction or paralytic ileus (17% *vs.* 5.1%, p<0.001) [19]. Nonetheless, randomized trials did not show significant differences related to the incidence of greater complications between techniques [14,15].

Vascular access complications

Femoral artery access, whether open or percutaneous (Percutaneous Endovascular Aneurysm Repair - PEVAR), is the most used approach in endovascular repair of Abdominal Aortic Aneurysm (AAA). The most common complications are surgical site infections, postoperative bleeding or hematoma, femoral artery injury or occlusion, pseudoaneurysm, and local lymphocele or seroma. The use of percutaneous arterial closure devices provides a feasible and less invasive option for EVAR, with satisfactory technical success rates [16]. In a meta-analysis review involving the main randomized controlled trials, 530 inguinal accesses were studied. There were no significant differences in general surgical site complications between the two techniques, despite a significant reduction in mean surgical time of approximately 12 min and lower postoperative pain and inflammation incidence on percutaneous treated sites [16]. In this study, total surgical site complications rates were 11.8% and 7.6% (OR: 0.61; 0.34-1.11) for open and percutaneous techniques, respectively. Hematomas, postoperative bleeding, and arterial injuries were the most common complications [16]. Surgical site infection rates were 0.9% and 0% for open and percutaneous techniques, respectively. Evidence shows that women may have higher rates of surgical site complications, according to O'Donnell et al. [17]. Most complications can be avoided or minimized by meticulous surgical technique, smaller sheaths, especially in women, team training and careful patient selection for PEVAR, which includes choosing arterial access with less than 50% of circumferential calcification, and avoiding prior groin incision, hematoma or other local anatomic variation.

Device-related complications

Endoleak: Endoleaks can be classified as primary, originated at the time of surgical repair, or secondary, occurring during postoperative follow-up. The main factors that influence the occurrence of type I endoleak are the presence of short, conical, calcified, or tortuous necks [18-20]. In addition, endograft under or over-sizing, conflicting with manufacturer's recommendations, is also a common factor [21]. The primary type I endoleak may occur at the time of endograft implantation in up to 5.9% of cases. It may be settled by a balloon catheter, although its persistence must be preferably treated during the same surgical procedure, using a Palmaz balloon-expandable stent, cuff [22], or Endo-Anchors [23] in most cases. In the case of persistent type IB endoleak, the most appropriate conduct is the placement of distal extensions, often requiring coverage of the internal iliac artery ostium after coil embolization. Some evidence indicates that small and low-flow type I endoleaks can occur despite proper planning and release of the endograft, but it may spontaneously disappear, especially after reversing systemic heparinization. Thus, expectant management could be chosen in these cases without abstaining from usual checkups [24-26].

Type III endoleaks must also be treated immediately, in most cases with additional endograft extensions. Types II-IV endoleaks, in turn, adopt expectant management and rarely require any measures in the same surgical procedure. Immediate conversions to open

surgical repair due to persistent endoleaks must also be avoided. Fenestrated stent grafts and parallel stents technique are possible alternatives in persistent type I endoleaks. However, they are mostly used in subsequent reoperations [27,28].

Iliac branch thrombosis: Early iliac branch thrombosis may occur in 2% of cases due to technical problems, tortuous iliac arteries, kinking, dissection, nonsupported limbs, poor distal outflow, excessive oversizing (greater than 15%) or access injury, even during intraoperative time [29]. Other previously reported risk factors are calcification, common iliac artery stenosis, narrow distal aorta, small AAA, body mass index greater than 30 kg/m^2, use of the endograft conflicting with manufacturer's recommendations, first-generation devices, diameter mismatch between extension and native artery, device extension to the external iliac artery, among others [30]. Cochenec et al. [31] noticed that most cases occur at short-term follow-up, at a mean of 1.4 months after EVAR. In this series intraoperatively occlusion occurred in 6.1% of the cases [31]. Completion final angiography without iliac stiff wires should be performed to evaluate possible subtle limb narrowing. Cone beam computed tomography or intravascular ultrasound, when available, can improve graft infolding or kinking detection with more sensibility than digital subtraction angiography considering their three-dimensional assessment. Iliac pre-emptive bare metal stents can be deployed in the cases with high limb thrombosis risk or when a branch limb or outflow stenosis is diagnosed. Thrombectomy or thrombolysis usually is necessary to treat occluded limbs; however, it is crucial to properly address the underlying technical failure using adjunctive stent-grafts or bare metal stents. Ultimately, femorofemoral or axillofemoral bypass may be necessary to lower limb revascularization [29-31].

Inadvertent renal artery occlusion: It is a rare yet serious complication whose real incidence remains unclear. It is either caused by mispositioned endograft, leading to ostial occlusion of the renal artery, or a consequence of intraoperative embolization. In some cases, inadvertent renal artery occlusion may not be promptly identified during the surgical procedure. Parallax effect correction by properly positioning the C-arch during angiography is extremely important to reduce its risk. There is no consensus on settling this complication since authors have already reported different rescue maneuvers for renal artery occlusion, including endograft repositioning, parallel stents in renal arteries, open renal artery revascularization with splenic-renal or hepato-renal grafts, and conversion to open surgical repair [32-35].

Ischemic colitis: It is a rare complication (0.6%), with lower occurrence rates in the postoperative period after EVAR, when compared to open repair of AAA (3.6%), as reported by Behrendt et al. [36]. Ischemic colitis is more common in urgent and emergency surgeries, as they are one of the main risk factors for this complication. Other authors also revealed that, besides rupture, procedure time and previous kidney failure are independent risk factors [37]. The main form of investigation is through colonoscopy, even though diagnosis is often made during exploratory laparotomy.

Spinal cord injury: It is another unusual complication with an incidence of 0.21%, according to EUROSTAR study. In most cases, it is a severe consequence of internal iliac artery occlusion, correlated with coil embolization of internal iliac or lumbar arteries [38].

Post-implantation syndrome: post-implantation syndrome is a misunderstood condition that occurs in the immediate postoperative period after EVAR (first 24 h). It is a transient, self-limited inflammatory process characterized by fever, leukocytosis, and blood clotting disorders, and increased serum inflammatory markers. This condition has been associated with formation of new thrombi in the excluded aneurysm sac [39]. The incidence of post-implantation syndrome described in literature is highly varied, between 17 and 39% [40]. It may cause extended postoperative recovery, increasing hospital length of stay [41].

Device-Related Complications in Post-EVAR Follow-up

The major disadvantage of EVAR is its high rate of reinterventions, compared to OSR, which mainly reflects device failures during follow-up. Consequently, increased aneurysm-related mortality was verified after a certain number of years at long-term follow-up [9]. EVAR reintervention rate may reach nearly 30% during all follow-up periods, making it significantly higher than in OSR [14]. A database survey from Medicare beneficiaries also revealed higher EVAR aneurysm-related reintervention rates than OSR (19% *vs.* 3.7%, p<0.001). However, most cases, according to EVAR trial 1, occurred in minor surgical procedures (mainly endovascular), where the authors reported a reintervention-free rate of only 72% in contrast to 90% of open surgical repair (p<0.001) during eight years [19,42-50].

Conversion rates from EVAR to OSR are very low nowadays, ranging from 0.8% to 1.5% [51] approximately. There has been a progressive evolution over the years, with the improvement of the technique, devices, and surgical planning, as stated by Deery et al. [42] Another multicenter study showed a late conversion rate of 2.22%, with a progressive increase over follow-up, whose leading causes were endoleaks (80.2%) and endograft infection (15.5%) [43].

Endoleak

Endoleaks occur in approximately 30% of cases during all post-EVAR follow-up and are associated with a medium-sized aneurysmal diameter growth over time [44]. Lal et al. [44], who assessed the results and predictors of endoleaks related to RCT OVER, reported that out of 881 individuals, 53% of endoleaks (mostly type II) resolved spontaneously, and 32% required intervention. Besides, the rates found for types I, II, III, IV, and indeterminate endoleaks were 12%, 76%, 3%, 3%, and 6%, respectively. Still, the presence of endoleaks did not significantly impact the survival time curve in this study compared to those without endoleak [44].

The arterial-phase CT angiography and delayed-phase CT angiography are the most used imaging tests to diagnose endoleaks, while angio MRI technique has similar accuracy and even detects type II endoleaks more precisely [45]. Nevertheless, Doppler ultrasound has gained more visibility recently, showing good accuracy, especially for detection of types I and III endoleaks [46].

Type I: Type I endoleaks are caused by an inadequate seal between endograft and vessel wall, proximally (Ia) or distally (Ib), allowing direct aneurysm sac pressure. Delayed type I endoleaks, in turn, may be related to conformational changes in aneurysm sac, proximal neck and iliac arteries degeneration, severe angulation, stent-graft migration, or, more frequently, an association of all these factors. Aneurysms requiring endografts with a larger proximal diameter or conical iliac extensions are more likely to develop delayed type I endoleak [47,48]. Once diagnosed, type I endoleaks must be treated as soon as possible, preferably endovascularly, as they can

cause high rates of rupture and mortality if not treated in time. That is a class IB recommendation of 2019 Clinical Practice Guidelines on the management of abdominal aorto-iliac artery aneurysms and European Society of Vascular Surgery (ESVS) [24,49-52]. Type I endoleak treatment alternatives were previously described in the perioperative complication's topic. Open repair alternatives, such as cervical cerclage or conversions to open repairs, must only be chosen in refractory cases or when an endovascular option is unavailable [49].

Type II: Type II endoleaks result from filling aneurysm sac from side branch flow, such as lumbar arteries, inferior mesenteric artery, or internal iliac arteries. Most cases of type II endoleak have a benign course and disappear in post-EVAR follow-up, but they can also be persistent or delayed [44,53]. The risk of rupture arising from type II endoleak is low (less than 1%); however, there are no prospective studies concerning the natural evolution of this type of complication. Thus, this risk may be underrated, especially because those patients have been treated preventively. Physiologically, this type of endoleak behaves like an arteriovenous malformation with afferent and efferent branches. Type II endoleaks that are larger and more complex, with large inferior mesenteric or lumbar arteries, tend to show a more difficult spontaneous resolution [54,55]. Measures must be taken to assure diagnosis of concomitant types I and III endoleaks or any other which might resemble a type II endoleak.

The treatment is indicated for those endoleaks associated with aneurysm sac enlargement, whose diameter growth is equal to or greater than one centimeter. It is recommended by ESVS guidelines, despite not being a consensus [49]. Treatment options are transarterial, translumbar, or transcaval embolization, and between vessel wall and endograft, using one of several types of embolizing agents. Ideally, embolization of endoleak nidus and afferent and efferent arteries should be performed. In addition, direct ligation of vessels can be conducted *via* laparoscopic or open approaches. Ligation of vessels ostium *via* aneurysm sac opening or open conversion should only be chosen in refractory cases after failed endovascular attempts.

Type III: Type III endoleaks are characterized by direct aneurysm sac pressure caused by endograft disconnection or loss of tissue continuity, such as suture breakage or loosening. All factors that may lead to device migration or situations involving inadequate overlapping of iliac branches and additional proximal or distal extensions may cause this type of endoleak. Every type III endoleak must be treated as soon as possible, preferably with implantation of new extensions between disconnected parts or where there is endograft loss of continuity [29].

Type IV: Type IV endoleaks are portrayed as blood leak through pores of endograft tissue. It is mostly identified soon after device release on control angiography, mainly in older endograft. This type of endoleak resolves spontaneously after reversing heparinization or in the immediate postoperative period and does not require specific treatment.

Endotension: Endotension is defined as an aneurysm sac enlargement with no endoleak identified in imaging tests. Several pathophysiological mechanisms have already been suggested to explain this phenomenon, for example, the idea that there might be a direct transmission of pressure to the aneurysm through an increase in endograft material permeability [56]. Nonetheless, some cases may be leaks identified by imaging methods [57]. Treatment is recommended when the enlargement is greater than 1 cm in aneurysm diameter. It consists of endograft relining, percutaneous aneurysm sac embolization, or open surgical conversion [58,59].

Endograft migration

Endograft migration occurs when there is a displacement of at least 10 mm compared to device's original position or when the migration causes any sign or symptom that requires reintervention [60]. Migrations may also be cranial at iliac extension fixation site. It used to be a common complication during first generation of endografts, but its incidence declined due to improved fixation systems, such as barbs and free-flow proximal stents [61,62]. Progressive neck dilation (proximal or distal) may occur over time, regardless of the type of endograft fixation or repair (open or endovascular). Consequently, it may lead to type I endoleaks and a high risk of rupture [63]. Besides type I endoleaks, device migration may cause disconnections (type III endoleaks), as well as iliac branch stenosis or occlusion.

Iliac branch stenosis or occlusion

By conducting a systematic literature review, Coelho et al. [30] reported incidence of iliac branch occlusion, which varies between 0 and 10.6% in post-EVAR follow-up. The clinical picture mostly consists of intermittent claudication (53.9%), acute limb ischemia (32.4%), asymptomatic (6.6%), or chronic critical ischemia (6.5%) [30]. A kink was identified as the cause of patency loss in 42.8% of cases. Bare metal stents used preventively in situations where kinking was noticed intraoperatively have shown to be protective against later limb thrombosis. The most common treatment of limb thrombosis was a femorofemoral cross-over bypass (52.3%) [30]. Other maneuvers included surgical thrombectomy, stent placement (isolated or after thrombectomy), catheter-directed pharmacological or mechanical thrombolysis, and aortoiliac bypass. Evidence indicates that angiography may not be the most appropriate diagnostic method to identify suspected cases of iliac branches complications. In these cases, cone beam computed tomography, and intravascular ultrasound are the most accurate options [30].

Intermittent claudication of the buttock

Bosanquet et al. [64] performed a systematic review of the consequences of internal iliac artery occlusion during EVAR procedures, selecting sixty-one non-randomized trials with 2,671 patients altogether. The authors observed that internal iliac artery occlusion was required in 15% of EVARs, contributing to a 27.9% incidence of buttock claudication, although 48% of these cases resolved spontaneously in approximately 22 months. Buttock claudication occurred in 32.6% of cases where coils were used, 23.8% using plugs, and 12.9% where only one ostium coverage was performed. More proximal occlusions caused fewer claudication rates. Moreover, claudication was more severe and common with bilateral internal iliac artery occlusion. One internal iliac artery, at least, should be preserved whenever possible. When occlusion of both internal iliac is required, staged embolization of them separated by at least one week seems to reduce the risk of pelvic complications. Remarks must be made about low-quality research regarding this outcome, which may lead to biases when good-quality randomized trials are not available [64].

Erectile dysfunction

Patients with AAA have a high prevalence of sexual dysfunction, usually due to advanced age and comorbidities [65]. In a prospective single-center study, Majd et al. indicated that 43% of patients

Table 1: Vascular access and device-related complications and treatment options following endovascular aortic aneurysm repair.

Timing	Complication		Treatment
Early complications	Vascular access	Bleeding/hematoma	Local compression, Direct repair.
		Artery injury	Direct repair.
		Pseudoaneurysm	Direct repair, thrombin injection.
		Lymphocele/Seroma.	Drainage, direct repair.
		Infection	Debridement, antibiotics, local muscle rotation.
	Device-related	Endoleak	Balloon catheter, Palmaz stent, Endo Anchors, endograft extensions.
		Iliac branch thrombosis	Thrombectomy, thrombolysis, adjunctive endografts, bare metal stents, extra-anatomic bypass
		Inadvertent renal artery occlusion	Endograft repositioning, parallel stents, open revascularization, conversion.
		Ischemic colitis	Exploratory laparotomy.
		Spinal Cord Injury	Rare; preventing hypogastric embolization.
		Post-implantation syndrome	None; unknown.
Device-related late complications	Endoleak	Type I	Balloon catheter, Palmaz stent, Endo Anchors, endograft extensions, cervical cerclage, open conversion.
		Type II	Transarterial, translumbar, between wall-endograft or transcaval embolization; direct vessel or ostium ligation; open conversion.
		Type III	Endograft extension.
		Type IV	Rare nowadays; Cessation of heparinization.
		Endotension	Endograft relining, aneurysm sac embolization, open conversion.
	Endograft migration		Same as type I and III Endoleaks treatment.
	Iliac branch stenosis or occlusion		Thrombectomy, thrombolysis, adjunctive endografts, bare metal stents, extra-anatomic bypass.
	Intermittent claudication of the buttock		Hypogastric preservation/revascularization with open or iliac-bifurcated device
	Erectile dysfunction		Hypogastric preservation/revascularization with open or iliac-bifurcated device
	Infection		Explantation (extra-anatomic bypass, cryopreserved allografts or antibiotic-impregnated endografts); conservative/antibiotics
	Endograft material fatigue		Same as type I and III Endoleaks.

subjected to EVAR showed previous erectile dysfunction, and this incidence increased to 59% after the procedure, not differing from open surgical repair [66]. Bosanquet et al. also reported post-EVAR erectile dysfunction complaints as a complication resulting from internal iliac artery occlusion, with an incidence of 10.2% (10% related to unilateral internal iliac artery occlusion and 16.9% related to bilateral internal iliac artery occlusion, with no statistical difference). The use of coils also caused a higher incidence of erectile dysfunction when compared to ostium coverage only [64]. In order to minimize erectile dysfunction, strategies have been proposed after unilateral or bilateral internal iliac artery interruption as staging bilateral occlusion, avoiding occlusion of more distal internal iliac collateral branches and, thus preserving the femoral and external iliac arteries inflow. Adequate heparinization of the patient during the intervention has also been advised. Additionally, branched iliac stent grafts offer the possibility of maintaining perfusion to hypogastric arteries when common iliac distal landing zone is not possible. Finally, surgical bypass has been reported to be effective in preventing pelvic ischemia [67,68].

Infection

The incidence of aortic endograft infection found in literature is between 0.2% and 0.7% in small-sample studies. The most common infectious agents are gram-positive bacteria, particularly *Staphylococcus* sp., in approximately 50% of cases [69]. Three main occurrences were observed: One-third of cases are secondary of aortoenteric fistula (half with upper gastrointestinal bleeding); one-third show nonspecific signs of infection, such as weight loss and asthenia; one-third provoke frank sepsis [70]. Despite the initial impression that EVAR would lead to a lower incidence of infection when compared to open surgical repair, studies revealed that the incidence of this complication is similar in both techniques [1,42].

Conventional treatment is device explantation, wide local debridement, followed by extra-anatomic derivation. An aortic reconstruction can be performed in a second reoperation if necessary. Direct *in situ* reconstruction, using femoral vein, silver-coated grafts, or impregnated with antibiotics, or cryopreserved allografts are alternatives that showed acceptable results. However, they should be chosen in cases where there is no frank purulence or much debris [69,71]. Laser et al. [72] described nine cases of aortic endograft infection treated in tertiary care. Five of them required previous extra-anatomic derivation, and the other four were subjected to *in situ* aortic reconstructions with rifampicin-coated polyester grafts. Two patients from this group died before hospital discharge, and both had aortoenteric fistula [72]. Conservative treatment with antibiotics related to drainage and debridement of infected tissues, without device explantation, may be an option. Nevertheless, it should be used in palliative or ineligible cases or for those patients that decline major surgery [69,73].

Endograft material fatigue

Structural failures of the endograft, such as stent fracture, attachment hook separation, suture line rupture, and loss of tissue integrity were common, especially in first-generation devices [74-80]. Many mechanical problems in first-generation endograft were solved afterward [81]. Nonetheless, several authors reported that even more newly developed endograft, despite their developed material, are subject to these complications [82-84]. Not all identified structural failures require reintervention, but they must be repaired in case of significant migration or types I or III endoleak occurrence.

Table 1 summarizes all EVAR vascular access or device-related complications and its main treatment possibilities discussed in this article.

Conclusion

EVAR advent has, consensually, diminished early mortality and postoperative systemic AAA repair complications over the last decades. However, novel and challenging device-related problems have emerged and even increased the postoperative reinterventions compared to OSR. Contemporary vascular surgeons must be aware and prepared to prevent and manage it adequately, devoting special surveillance to the long-term device failure that tends to increase over several years.

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