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Review

Infection of Vascular Prostheses: A Comprehensive Review

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Abstract: Vascular graft or endograft infection (VGEI) is a complex disease that complicates vascular-surgery and endovascular-surgery procedures and determines high morbidity and mortality. This review article provides the most updated general evidence on the pathogenesis, prevention, diagnosis, and treatment of VGEI. Several microorganisms are involved in VGEI development, but the most frequent one, responsible for over 75% of infections, is Staphylococcus aureus. Specific clinical, surgical, radiologic, and laboratory criteria are pivotal for the diagnosis of VGEI. Surgery and antimicrobial therapy are cornerstones in treatment for most patients with VGEI. For patients unfit for surgery, alternative treatment is available to improve the clinical course of VGEI.

Keywords: VGEI; infection; vascular prosthesis; vascular graft; antibiotic

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1. Introduction

A vascular prosthesis is defined as a biological or artificial device meant to replace a segment of an arterial tree whose function is compromised by injury, occlusive disease, or aneurysmal degeneration. A prosthetic graft provides a substitute conduit for blood flow, allowing the diseased vessel segment to be repaired, excised, or bypassed [1–10].

Vascular prostheses include vascular grafts (VGs), generally implanted surgically, and vascular endografts (VEs) (or stent-grafts) implanted by endovascular procedure. VGs may be classified into biological grafts, which are composed of actual tissues, most often blood vessels (e.g., autologous grafts derived from the patient's own vessel); allografts (from human vessels); xenografts (generally of bovine origins); and synthetic grafts made from either poly-ethylene-terephthalate (PET, or Dacron), a textile material, or expanded-polytetrafluoroethylene (ePTFE), a non-textile material. In textile VGs, the basic polymer is first made into a yarn, which is then used to construct a graft using various methods of knitting or weaving. Non-textile VGs are manufactured using the techniques of the precipitation or the extrusion of the polymer from solutions or sheets of the material. When the available length of an autogenous tissue graft is inadequate for the required reconstruction, composite VGs can be used. Such VGs are constructed by combining segments of an artificial prosthetic material with autogenous material to form a substitute-vessel conduit. VGs are generally positioned during open surgery and sutured both proximally and distally to the healthy artery by end-to-end or end-to-side anastomosis (Figure 1). An ideal VG should be impermeable, thromboresistant, compliant, biocompatible, durable,

resistant to infection, easy to sterilize, easy to implant, readily available, and cost-effective. In particular, biocompatibility is necessary because significant tissue reaction may promote thrombosis, loss of graft integrity, and graft failure. Sterility and resistance to infection are necessary to decrease the incidence of infection. VEs are transluminally implanted vascular devices introduced into the vascular system via a remote artery using minimally invasive techniques that combine a prosthetic fabric with a vascular stent (Figure 1). Vascular stents are made by different alloys, with nitinol being the most used. The graft is anchored in place by a balloon-expandable or self-expanding metal frame that supports all or part of the graft and provides a tight seal proximal and distal to the diseased segment of the artery. Since it circumvents the need for laparotomy, the cross-clamping of the artery, and the obligatory blood loss associated with the opening of the aneurysm sac, the technique has been shown to reduce the morbidity and mortality associated with conventional surgery, and it expands the patient pool to include patients with severe medical- and co-morbidities who were previously denied treatment [11–22].

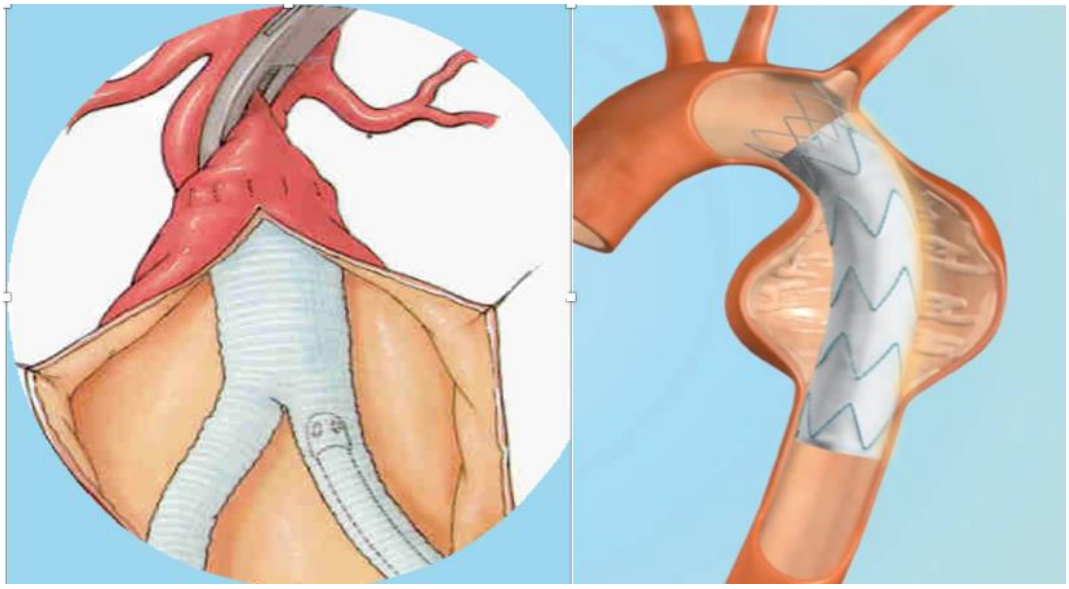

Figure 1. Vascular graft and vascular endograft. On the (**left**): vascular graft; on the (**right**): vascular endograft.

According to the location of implantation, grafts may be extra-cavitary (groins and lower limbs) and intra-cavitary (abdominal or thoracic aorta) [21,22].

Vascular prostheses may be burdened by a series of complications mainly due to fabrication or biomaterial failure such as dilation, rupture, thrombosis, allergic foreign-body reaction, and infection. In particular, a vascular graft or endograft infection (VGEI) is a clinically important complication that may occur following VG surgery or VE procedures accompanied by high morbidity and mortality rates [22–33]. The incidence of thoracic aortic VGEI is around 6% and, with mortality rates that relate to the clinical presentation, around 75%. The incidence of VGEI in the abdominal aorta is a rare complication, occurring in <1%, but one with a high mortality rate. VGEIs in peripheral-artery surgery are mainly represented by vascular graft infection (VGI) for open surgery with an incidence of up to 2.8% [22]. The percentage of infection in prosthetic arteriovenous hemodialysis grafts (AVHGs) is approximately 3.5% [34]. VGEIs are managed mainly through surgical removal, revascularization, and long-term antibiotic treatment. The explantation of an infected graft may determine important mortality rates (18–30%), while conservative management with long-term antibiotic therapy may result in a very high mortality rate, reaching about 100%, if the VGEI is not completely resolved [21].

VGEI, although not particularly frequent, is a multifaceted disease, and diagnosis may be challenging and even complicated for physicians, as they sometimes may be not

as timely as an optimal treatment requires. Considering the rarity of this disease, current literature is not specialized as it lacks randomized controlled trials and studies with high patient population. Furthermore, current clinical evidence is based on small case studies, which are very often retrospective in nature [35].

The primary aim of this article is to provide a comprehensive overview of VGEI, current diagnostics, and therapeutical options in the context of an effective and contemporary management of this disease. As its secondary aim, the article discusses the future perspectives on this topic.

To conduct this review, the libraries searched included Web of Science, Scopus, ScienceDirect, and Medline.

The following keywords were used in various combinations: "vascular prostheses", "vascular grafts", "vascular endografts", "infection", "diagnosis", "treatment", "management", "pathogenesis", and "prevention".

2. Pathogenesis

Several factors account for the onset of VGEIs, including factors related to patients, such as advanced age, male sex, overweight status or obesity, heart disease, immunocompromised status, diabetes, chronic kidney disease (CKD), chronic obstructive pulmonary disease (COPD), nasal colonization with *Staphylococcus aureus*, bacteremia at the time of graft placement, groin incision, skin wounds in the lower limbs, and prolonged hospitalization [36,37].

In particular, a prolonged hospital stay seemed to be responsible for skin colonization by more resistant organisms; diabetes seems to be directly and significantly related to the onset of infrainguinal surgical infections, particularly for CKD patients who are immunocompromised due to their uremic state and are more susceptible to *Staphylococcus aureus* colonization; COPD patients and heart-disease patients may experience bacteremia episodes that may reach implanted vascular prostheses; nasal carriers of Staphylococcus aureus have three to six times of the risk of developing VGEI compared with non-carriers; Obesity is an independent risk factor for VGEIL as it is associated with longer hospital stays, major operating times, and several cardiovascular and metabolic comorbidities [38–47].

Several infectious microorganisms are involved in VGEI development, but the most frequent type, responsible for over 75% of infections, is *Staphylococcus aureus* and, in particular, the most severe infections are those with *methicillin-resistant Staphylococcus aureus (MRSA)*. On the other hand, gram-negative bacteria infections, such as *Pseudomonas aeruginosa*, *Escherichia coli*, *Klebsiella*, *Enterobacter*, or *Proteus*, although less frequent, are associated with a more serious course, especially in open surgery, following which may result in the rupture of the anastomosis caused by the destruction of the arterial wall by the bacterial toxins. VGEI microbiology also changes according to graft location and to specific microbiology of each hospital, and it is also evolving over time with patterns similar to nosocomial infections. Co-infections with several bacteria, though rare, are also possible, especially when they are caused by skin wounds [37,48–57].

Several evidence have demonstrated two moments for the onset of VGIs following VG implantation. The first moment may occur in the early postoperative-recovery phase and is generally determined by contamination during the procedural phase (various sources of microbial contamination due inadequate sterile measures, unintentional contact with the patient's skin, especially of the groin, or with intra-abdominal organs) or by the direct spread of a superficial infection to the VG. Furthermore, the groin incision may directly contaminate the graft through infected lymphatic channels or glands that drain from distal infected tissues or ulcers. Moreover, this complication is more evident especially in obese subjects where the incisional wound is located within moist skin folds. The second moment may occur in the long-term period following surgery, up to one year after implantation, and is due to invasion of the graft by a novel bacteremic sprout or activation of a latent infection [58].

Bacteria are capable of initiating infection through biofilm creation. Bacterial biofilm development can be divided into 3 phases: attachment, maturation, and detachment. Initially, during attachment, specific proteins trigger the initial adhesion to host-matrix proteins such as fibrinogen and fibronectin. Subsequently, in the second phase (maturation), there is the formation of intercellular aggregation with final biofilm structuring. Finally, in the third phase, single cells or aggregates of cells disconnect from the biofilm, determining the sprout of infection. Biofilms assure antibiotic resistance substantially by preventing antibiotics from reaching bacteria that are embedded in the biofilm matrix and by decreasing their efficacy. Moreover, biofilms defend bacteria against the immune system, thus, protecting them from host defenses [36,58–69].

Furthermore, various bacteria can be specifically isolated according to the site of infection. In fact, in thoracic VEGIs, mainly gram-positive bacteria, like those found in infective endocarditis, can be found (*Staphylococcus aureus*, *Coagulase-negative Staphylococcus*, *Enterococcus*, and *Streptococcus*). On the other hand, Gram-negative bacteria and polymicrobial infections can be isolated in abdominal VGEIs [35].

Considering the time of appearance, vascular-prostheses infections are labeled under two categories, early-appearing VGEIs (occurring <4 months after implantation) or late-appearing VGEIs (occurring >4 months after implantation). The former is most often caused by more virulent organisms, such as Staphylococcus aureus, and the latter is commonly associated with less virulent bacteria such as *S. Epidermidis* and other coagulase-negative staphylococci, which can produce infections with mild symptoms [70].

3. Diagnosis

VGEIs can result in limb loss, systemic sepsis, and, sometimes, death, even in the setting of a correct diagnosis and treatment. Moreover, the clinical presentation can be subtle and is influenced by the anatomic location of the graft. An infection of an infrainguinal graft frequently appears as cellulitis, soft tissue infection, drainage tract, or psudoaneurysm. The clinical presentation of an extracavitary graft is usually not subtle. An intraabdominal graft may appear as systemic sepsis, or, alternatively, as an ileus or abdominal distension, with or without tenderness. Occasionally, an aortic graft infection may result in an aorto-digestive fistula, with herald bleeds as a first sign. Early VGEIs can manifest with fever, leukocytosis, and purulent drainage from the graft site. Late VGEIs appear as a healing complication, such as a seroma, a pseudoaneurysm, or even as a late graft thrombosis without apparent reason. In this case, systemic signs of illness such fever are not usually present. Therefore, the diagnosis of VGEI is complex, challenging, and centered on a multilevel and multidisciplinary approach. Clinical features and laboratory and imaging evaluation are pivotal for an effective diagnosis [22,30]. In 2016, the Management of Aortic Graft Infection Collaboration (MAGIC) introduced criteria to establish the diagnosis of a VGEI [71]. MAGIC criteria are a validated tool and offer good sensitivity and specificity in the context of VGEI diagnosis [72,73]. The MAGIC criteria include clinical/surgical, radiologic, and laboratory criteria for the diagnosis of VGEI [71,72]. Diagnostic criteria were also ranked as either "major" or "minor" within each category (Table 1).

According to these criteria, a VGEI may be confirmed in a patient with any isolated major criterion or minor criteria from two of the three categories (clinical/surgical, radiological, or laboratory). Furthermore, AGI is diagnosed in the presence of a single major criterion plus any other criterion (major or minor) from another category [71]. Moreover, any communication between a non-sterile site and a prosthesis demonstrates GI, such as: aorto-enteric fistula (AEnF), aortobronchial fistula (ABF), the displacement of a stent graft in a previous infected site (e.g., infected aneurysm), and exposed grafts in deep open wounds. The pathogenesis of AEnF, aorto-oesophageal (AEsF), and ABF is not clear. It is likely that bowel ischemia is due to an occlusion of the relative arteries, and mechanical damage is caused by the aneurysm. Fistula can occur because of traumatic forces caused by surgical injury, inadequate tunnelling, erosive processes by direct contact, or by the penetration of an oversized VE [73–83].

Table 1. Management of Aortic Graft Infection Collaboration (MAGIC) criteria [71].

Clinical/Surgical	Radiology	Laboratory
Major: - Pus (confirmed by microscopy) around graft or in aneurysm sac at surgery. - Open wound with exposed graft or communicating sinus. - Fistula development e.g., aorto-enteric or aorto-bornchial. - Graft insertion in an infected site, e.g., fistula, mycotic aneurysm, or infected pseudoaneurysm.	Major: - Perigraft fluid on CT scan $\geq$3 months after insertion. - Perigraft gas on CT scan $\geq$7 weeks after insertion. - Increase in perigraft gas volume demonstrated on serial imaging.	Major: - Organisms recovered from an explanted graft. - Organisms recovered from an intra-operative specimen. - Organisms recovered from a percutaneous, radiologically guided aspirate or peri-graft fluid.
Minor: - Localized clinical features of GI, e.g., erythema, warmth, swelling, purulent discharge, pain. - Fever $\geq$ 38 °C with GI as the most likely cause.	Minor: - Other, e.g., suspicious perigraft gas/fluid/soft tissue inflammation; aneurysm expansion; pseudoaneurysm formation; focal bowel wall thickening; discitis/osteomyelitis; suspicious metabolic activity on FDG-PET/CT; radiolabeled leukocyte uptake.	Minor: - Blood-culture positive and no apparent source except GI. - Abnormally elevated inflammatory markers with GI as most likely cause, e.g., ESR, CRP, white cell count.

Note: In case microbiological analysis identifies potential "contaminant" organisms (e.g., coagulase-negative staphylococci, propionibacteria, corynebacteria, and other skin commensals) a minimum of (I) two intraoperative specimens, (II) two blood cultures, or (III) one intraoperative specimen plus one blood culture must be positive with an indistinguishable microorganism in each sample based on antibiograms or other typing methods, e.g., pulsed-field electrophoresis. (CT = computed tomography; FDG = fluorodeoxyglucose; PET = positron emission tomography; ESR = erythrocyte sedimentation rate; CRP = C-reactive protein; GI = graft infection.)

Considering diagnostic imaging techniques as suggested in the MAGIC criteria, computed tomography (CT) scan is considered the first-choice technique. CT angiography (CTA) is the best acquisition protocol for VGEI as it can detect signs of infection or inflammation in the graft and perigraft tissues (Figure 2). CTA can detect perigraft fluid or gas (Figure 2) and some related complications such as psudoaneurysms, endoleaks, and fistulas to adjacent organs. The demonstration of contrast extravasion in adjacent organs such as the bronchi, esophagus, and bowel, is pathognomonic of VGEI. The use of Fluorodeoxyglucose (FDG) positron emission tomography/CT is suggested in the minor criteria of MAGIC, and it is a very useful nuclear-medicine technique that is able to detect infection and inflammation as FDG accumulates in neutrophils and monocytes/macrophages activated in VGEI processes. Also, it detects radiolabeled leukocyte uptake by white blood cell (WBC) scintigraphy based on the capacity of a radiolabeled granulocyte to reach the infection site and to accumulate in infectious tissues. WBC scintigraphy together with single-photon emission computed tomography (SPECT/CT) seems to increase diagnostic power [21,84–103].

The importance of MAGIC criteria, which are also recommended in the current guidelines [22], lies in the provided definition of classic and definite cases that can be managed with high sensitivity and specificity. Nevertheless, for some locations such as thoracic VGEI, there is evidence of reduced specificity, and this may lead to a certain overestimation of VGEI in this district. Moreover, it is not possible to scale the intensity of the criteria, and this may be limiting in certain cases. Therefore, the further assessment and validation of novel criteria (maybe with an intensity score) adapted for different districts are required in future studies [35,73].

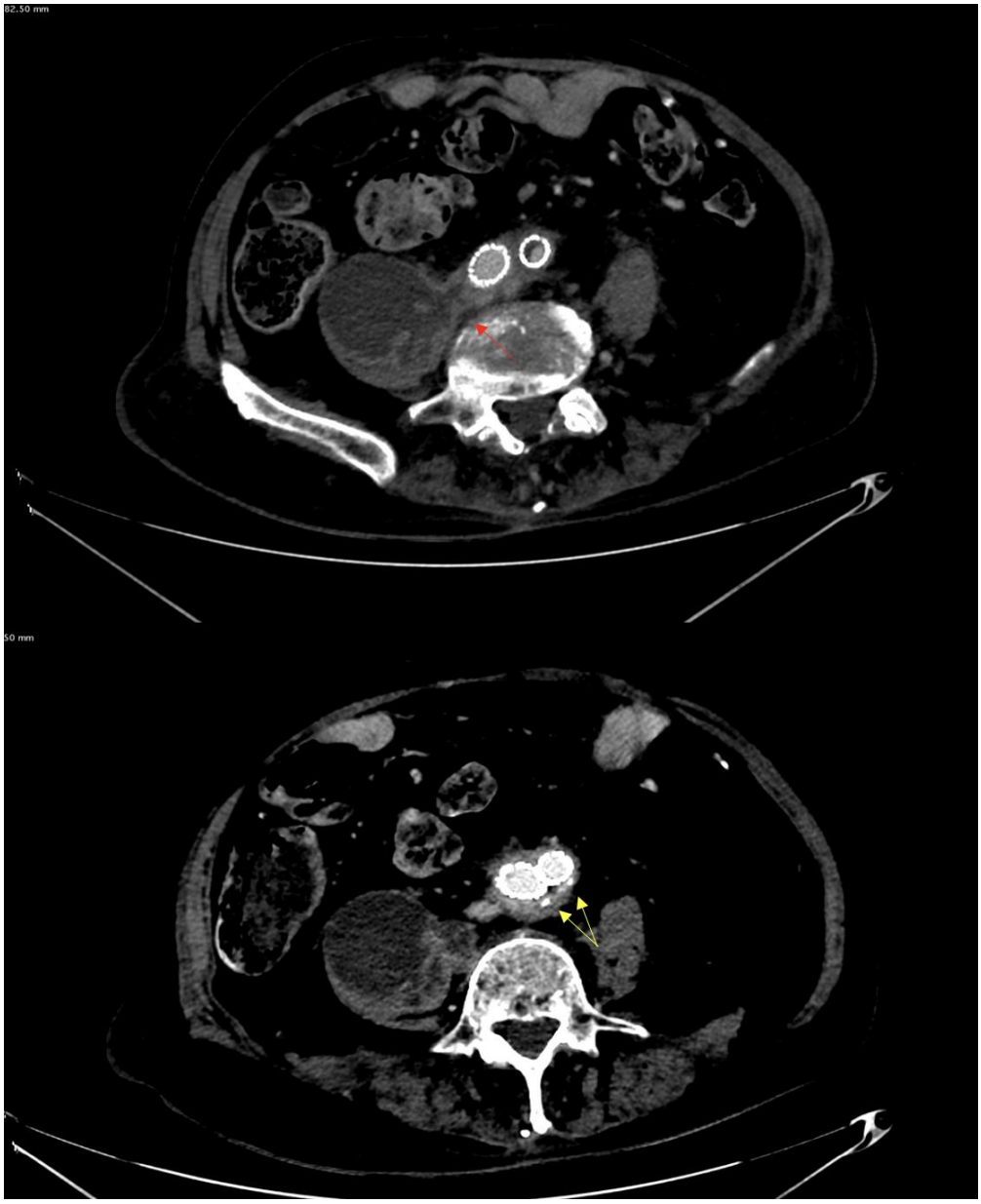

Figure 2. CTA scan of infected vascular endograft. (red arrow: psoas muscle abscess due to vascular endograft infection; yellow arrows: perigraft gas).

4. Specific Aspects and Clinical Presentation According to Location of VGEI

Clinical manifestations of thoracic aortic VGEI can range from a fever of unknown origin to serious sepsis, important bleeding, and, ultimately, shock. Generally, considering the depth position of thoracic aorta, there are no visible signs of infection. In the case of AEsF or ABF, hematemesis or hemoptysis may be the first manifestation. Bleeding may even be massive, especially in case of AEsF, and sometimes it is preceded by self-limiting hemorrhage [22]. Most patients (70%) with abdominal VGEI present with pain, fever, and leukocytosis; while 33% of patients present with weight loss, fatigue, or generalized weakness [22].

Considering peripheral arteries, the groin is frequently implicated in VGEI following aorto-iliac or infrainguinal reconstructive surgery. Signs generally include fever, pain, mass, skin blushing, or exposed vascular graft (Figure 3) [22].

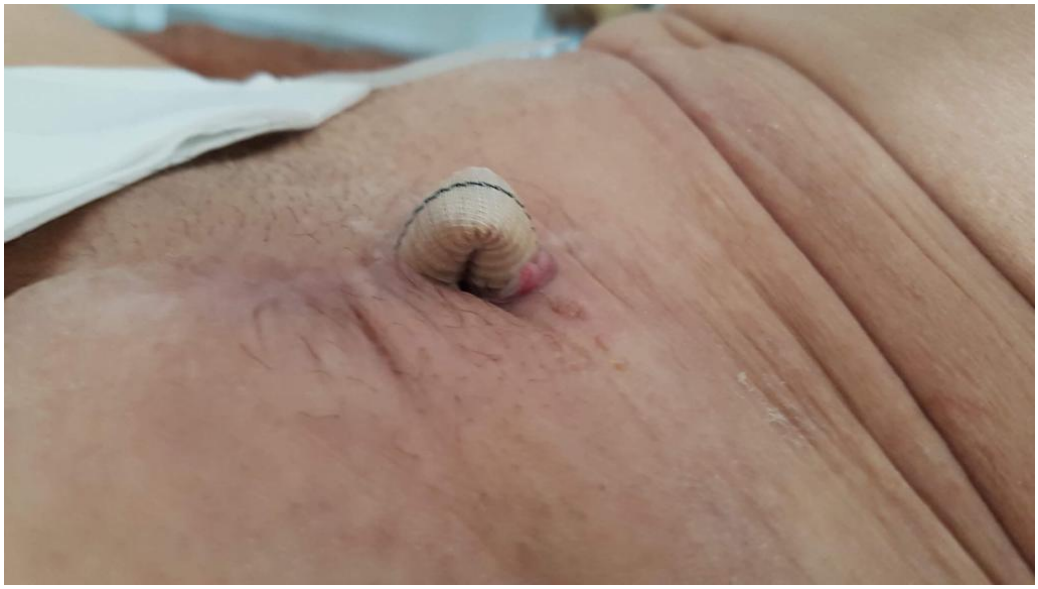

Figure 3. Exposed infected vascular graft.

For supra-aortic trunks (SAT), most infections are related to carotid patches, and the most common clinical manifestations are represented by abscess, neck mass, and hemorrhage. On the other hand, patients with endograft infection (EGI) generally present with fever, malaise, and pain. SAT EGI generally develop after stent-graft implantation for carotid blowout syndrome (CBS) [22,104].

For prosthetic AVHGs, the main manifestations of disease presentation include an exposed graft, purulent drainage, sepsis, erythema, hemorrhage, hematoma, and pain [34]. Table 2 resumes main clinical presentations of VGEI. In particular, systemic symptoms generally overlap. Local symptoms depend on the local district.

Table 2. Main clinical presentations of VGEI.

District	Clinical Presentation
Thoracic Aorta	- Unexplained fever - Severe sepsis - Massive bleeding - Shock - Hematemesis - Hemoptysis
Abdominal Aorta	- Fever - Pain - Leukocytosis - Weight loss - Fatigue - Generalized weakness
Peripheral Arteries	- Fever - Pain - Mass - Redness of the skin - Exposed graft
Supra-aortic trunks	- Fever - Malaise - Pain

Table 2. *Cont.*

District	Clinical Presentation
Prosthetic arteriovenous hemodialysis grafts	- Exposed graft - Purulent drainage - Sepsis - Erythema - Hemorrhage - Hematoma - Pain

5. Prevention of VGEI

The prevention of VGEI generally consist of adequate pre-operative patient preparation, heedful surgical and procedural management, antisepsis measures, prompting the administration of pre-operative antibiotic prophylaxis, and adequate wound care [34].

Before surgical procedures, it is important to treat any potential source of infection (i.e., dental problems, etc.) [22,58].

In the context of preoperative patient preparation, it seems reasonable to screen patients undergoing graft implantation for S. aureus nasal carriage and, if positive, provide peri-operative nasal eradication therapy because of the risk of potentially related severe VGEIs [22,105]. Adequate antimicrobial prophylaxis with broad-spectrum systemic antibiotics seems to significantly reduce the risk of early graft infection. Generally, the first or second generation of cephalosporin is used. It is also important that antimicrobial prophylaxis covers the most frequent bacteria, including MRSA, and for institutions with high rates of MRSA, daptomycin or vancomycin can be administered additionally [22,34]. Hair removal and appropriate aseptic care in the operating theater remain pivotal in peri-operative care [22].

Postoperative measures consist of speeding up the healing of surgical wounds, for example, the use of negative-pressure wound therapy (NPWT), whereby applying subatmospheric pressure decreases inflammatory exudates promoting granulation tissue, may be used to achieve fast wound closure, [106–110] and to consider antimicrobial prophylaxis before any dental procedure [22,111–115].

6. Treatment of VGEI

The best treatment for VGEI mainly depends on the location of the graft, the extent of the infection, and the type of microorganism. Generally, the management plan includes the removal of the graft, a careful debridement of the infected surrounding tissues, the restoration of circulation distal to the GI, and adequate antibiotic therapy [58]. Figures 4 and 5 show an infected VG and VE, respectively, during explantation procedures.

The clinical spectrum of graft infection permits physicians to individualize treatment that should be selected in view of eradicating related clinical manifestations and potential complications, also considering the patient's characteristics, vascular status, and comorbidities. Surgery is the keystone in the management of VGEI. In particular, the involvement of a suture line in the infectious process is an absolute indication for the removal of the entire infected graft, as an infected anastomosis inevitably leads to eventual rupture and hemorrhage. The infected graft must be completely excised with an aggressive debridement of the infected location. To restore blood flow, autologous venous material is considered adequate as a first-line option for arterial reconstruction. If the extraction of the infected vascular graft is not feasible due to important co-morbidities and/or a lack of revascularization options, surgical debridement and/or percutaneous-drainage irrigation and lifelong antimicrobial therapy must be performed, although a conservative treatment of VGEI is associated with high mortality [72,116–118]. Whenever possible and available, a cryopreserved arterial allograft may provide excellent results in all arterial districts [58,119–128]. In low-grade infections, rifampicin-bonded grafts may also be used. There are no wide studies on these materials, and, therefore, no definitive conclusions can be drawn. There is

still little evidence for silver-coated grafts and bovine-pericardium xenogenous grafts, and further studies are needed to confirm their efficacy in VGEIs [16,22,129–136]. Autologous femoral veins may also be used for an in situ reconstruction of the aorto-iliac segment in VGEIs [137–140]. Moreover, to avoid routing in an infected area, extra-anatomic reconstructions may be possible. This strategy of bypassing the infected area reduces the risk of reinfection and minimizes operative stress especially for high-risk patients unable to tolerate major surgical procedures, and the explantation of the infected graft may be programmed at a later stage. Prosthetic grafts may be used strategically since, although they have the advantages of a short operating time, the reinfection rate is high [22,141–145].

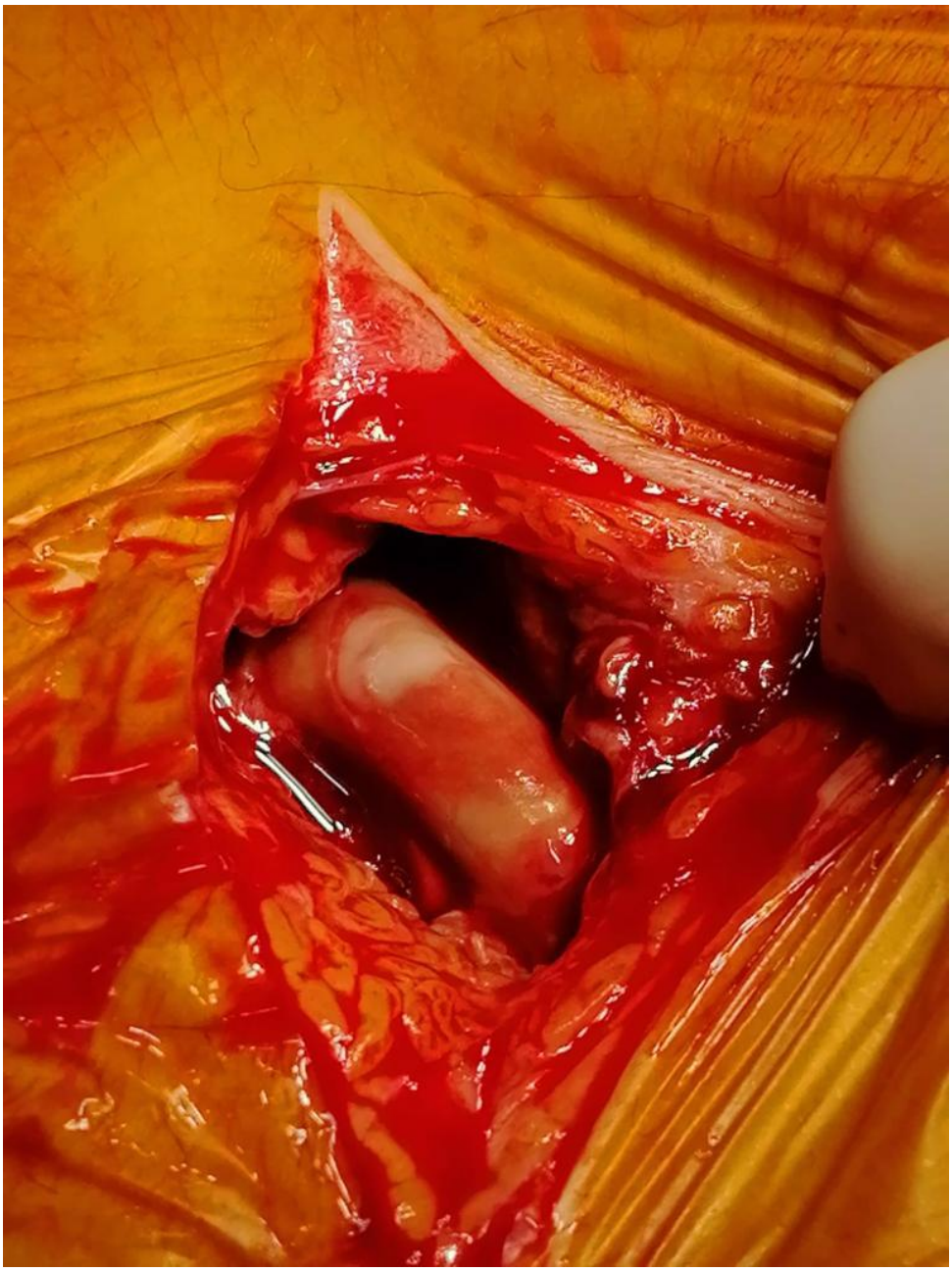

Figure 4. Explantation of an infected vascular graft at the groin.

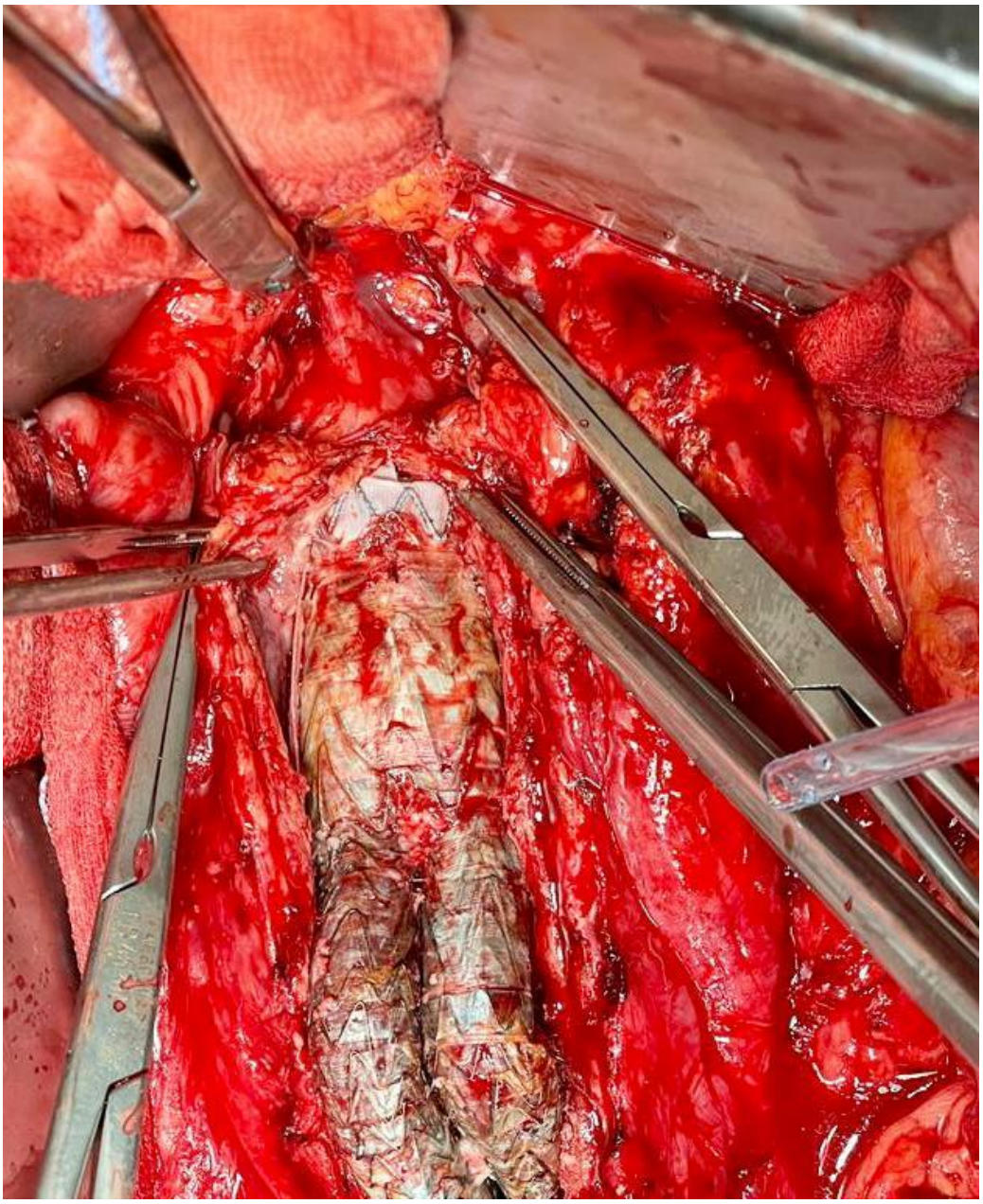

Figure 5. Explantation of an infected aortic and iliac endograft.

Sometimes, a bridge treatment is also considered, especially in cases of acute bleeding in unstable patients. This strategy permits a careful graft revision, including removal and novel reconstruction [22,146]. Following infected graft removal, and after eventual vascular reconstruction, large tissue defects may be treated with NPWT after appropriate debridement procedures [22].

Figure 6 shows a general evidence-based algorithm in a case of VGEI.

For the selection of antibiotics in the treatment of VGEI, it should be remembered that the use of empirical antibiotic treatments must be limited to suspected or known cases of VGEI when it does not seem feasible to wait for surgical microbiological-sample analysis, for example, in case of severe sepsis, septic shock, aneurysmal rupture, or anastomotic break.

Then, antibiotic adaptation should be started following the microbiological results from blood cultures or surgical samples. This serves to limit the selection pressure for resistant strains that may be caused by broad-spectrum antibiotic therapy [147].

Moreover, to achieve a better prognosis in VGEI patients, it is pivotal to take a multidisciplinary approach that includes several medical specialties, such as vascular surgery, radiology, microbiology, nuclear medicine, infectious disease, anesthesiology, and intensive care. It also important to tailor treatments for the different clinical situations that characterize VGEI [148].

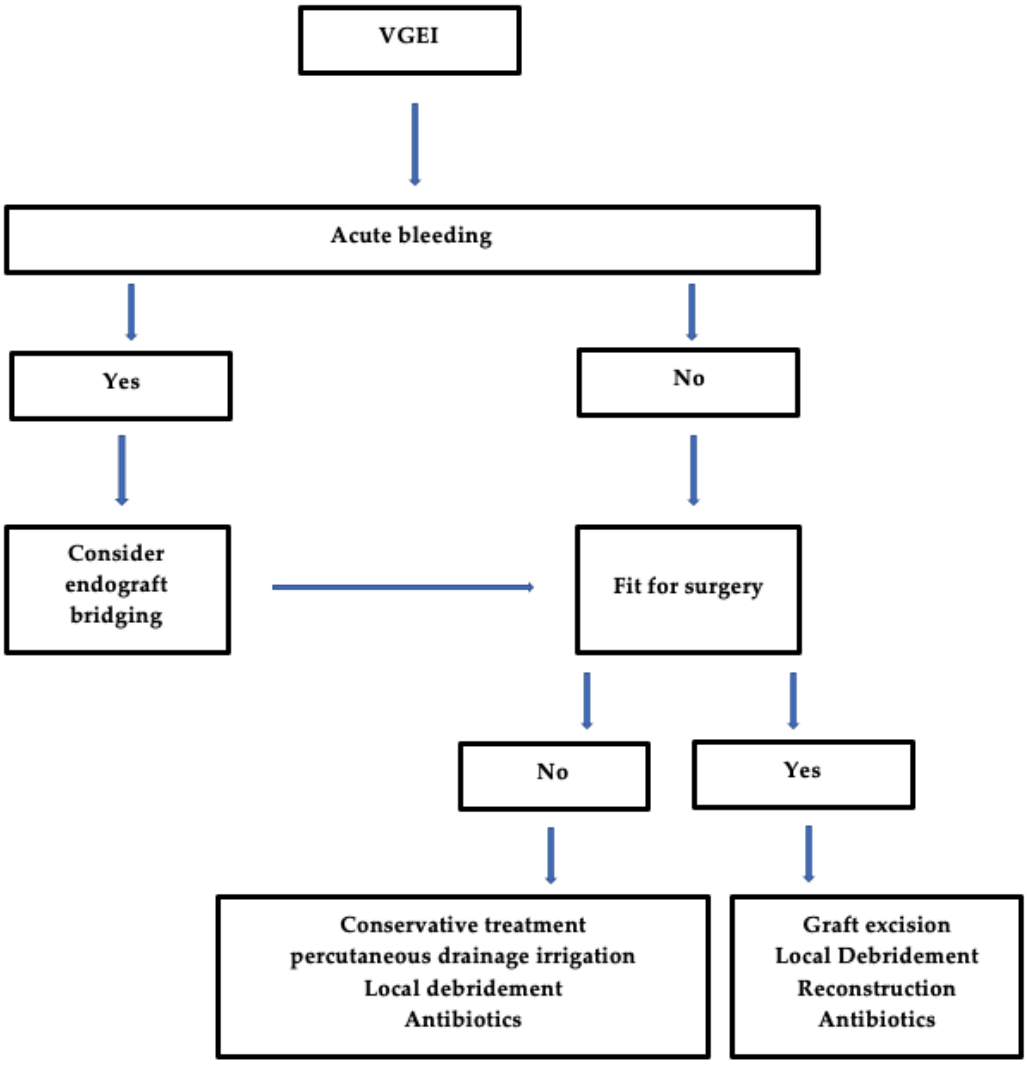

Figure 6. General Algorithm for the management of VGEI. VGEI = vascular graft or endograft infection.

7. Future Perspectives

Nanotechnology studies are ongoing with the aim to develop a new generation of grafts, tissue-engineered vascular grafts (TEVGs), in which synthetic materials can be bonded to endothelial cells, growth factors, and other active biomolecules to improve biocompatibility, thus, reducing graft-implant complications. Furthermore, using novel nanotechnology applications that involve nanofibers and nanoparticles, it will be possible to modify several features of the grafts' materials providing, for example, a greater antibiotic-loading capacity to prevent VGEI. In this context, 3D bioprinting, and even 4D bioprinting, may be an effective technology with the potential to produce patient-tailored grafts with major resistance to several biological assaults, thus, overcoming a series of complications [149–154]. Thus, the ideal engineered graft should have the biomechanical, morphologic, and cellular characteristics of a human vessel [155].

Another promising future material that may be used in preventing VGEI is graphene, a single layer of carbon atoms closely embedded in a hexagonal honeycomb lattice that

has bactericidal or bacteriostatic effects. This material, also known for its characteristics of safety and biocompatibility, seems particularly promising for future use in endovascular grafts [156–158].

More advanced technologies also contemplate the combination of biological and biodegradable artificial materials as scaffolds, in combination with the use of mesenchymal stem cells (MSCs) or induced pluripotent stem cells (iPSCs), thus, improving tissue engineering products for novel and resistant vascular substitutes, as endothelial cells derived from iPSCs (hiPSC-ECs) may have better functional characteristics [159–164].

8. Conclusions

VGEIs can further decrease the quality of life of vascular patients that experience, during the course of their vascular disease, several problems in their physical and social functioning. The prevention, the diagnosis, and the management of VGEIs are highly challenging, as VGEIs are burdened with significant rates of morbidity and mortality. The prompt interpretation of clinical manifestations with the correct use of imaging and laboratory strategy allow a timely diagnosis and appropriate decision-making to find effective therapeutic options. While great advances have been made over time, and specific guidelines for the management have been published, there is still a matter of debate on the optimal care for VGEI. Nevertheless, surgery and antimicrobial therapy are the cornerstones in the treatment of VGEI. For patients unfit for surgery, alternative tailored treatment is available.

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