

# Vascular graft infections

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## Summary

Vascular procedures are rarely complicated by infection, but if prosthetic vascular graft infection (PVGI) occurs, morbidity and mortality are high. Several patient-related, surgery-related and postoperative risk factors are reported, but they are not well validated. PVGI is due to bacterial colonisation of the wound and the underlying prosthetic graft, generally as a result of direct contamination during the operative procedure, mainly from the patient's skin or adjacent bowel. There is no consensus on diagnostic criteria or on the best management of PVGI. On the basis of reported clinical studies and our own experience, we advocate a surgical approach combining repeated radical local debridement, with graft preservation whenever possible or partial excision of the infected graft, depending on its condition, plus simultaneous negative-pressure wound therapy (NPWT). In addition, antimicrobial therapy is recommended, but there is no consensus on which classes of agent are adequate for the treatment of PVGI and whether certain infections may be treated by means of NPWT alone. Since staphylococci and Gram-negative rods are likely to be isolated, empirical treatment might include a penicillinase-resistant beta-lactam or a glycopeptide, plus an aminoglycoside, the latter for Gram-negative coverage and synergistic treatment of Gram-positive cocci. Additionally, empirical treatment might include rifampicin since it penetrates well into biofilms.

**Key words:** prosthetic vascular graft infection; negative pressure wound therapy; graft preservation; 18F-fluorodeoxyglucose positron emission tomography; computed tomographic angiography

## Introduction

Because of the growing prevalence of atherosclerosis and diabetes, vascular grafts are increasingly used, and about 1–6% of vascular procedures are complicated by infection [1]. Morbidity and mortality attributable to prosthetic vascular graft infection (PVGI) are high [2, 3], and are highly dependent on the location of the vascular graft, i.e., peripheral or aortic. Peripheral graft infections occur in about 4% of cases and potentially lead to limb loss and increased

mortality; the rate of infection of aortic grafts is lower (1–3%), but the attributable mortality is more than 20% [3–5]. According to the Swiss Federal Office of Statistics, 9/100,000 persons are hospitalised because of PVGI, and mean hospitalisation costs amount to 72,350 Swiss Francs per patient.

## Risk factors

Reported risk factors for the development of PVGI include patient-related factors, such as older age, male sex, high body mass index, heart failure, immunodeficiency, diabetes mellitus, renal failure, chronic obstructive pulmonary disease, blood stream infection at the time of graft placement, skin ulcers in the lower extremity and prolonged preoperative hospital stay [6–8]. Surgical risk factors include: arteriography injection site within the operative area; groin incision; prolonged, emergency, or “redo” vascular surgery; bowel injury; extensive lymphatic manipulation; and tissue injury during the dissection [4, 6, 7, 9]. Postoperative risk factors include wound infections, and local complications such as seroma or haematoma owing to pseudoaneurysm or wound-bed bleeding [6, 8]. Other potential risk factors pertain to the type of prosthetic graft. Although the incidence of infection in polyester and polytetrafluoroethylene (PTFE) grafts seems to be comparable, it has been hypothesised that polyester grafts are more resistant than PTFE grafts to eradication of infection [5].

## Prevention

Direct inoculation of bacteria from the patient's skin at the time of surgery or direct spread of bacteria due to local tissue breakdown may lead to surgical site infections. Most PVGI occurs in the groin as a consequence of a progressive surgical site infection. Factors that have been proved to be associated with PVGI are: (1) prolonged preoperative hospital stay; (2) remote infection before elective vascular surgery; (3) hair removal at the surgical incision site with a razor; (4) simultaneous gastrointestinal procedures during the insertion of an aortic graft; and (5) nasal carriage of *Staphylococcus aureus* (*S. aureus*). Several studies have highlighted the importance of preoperative antibiotic

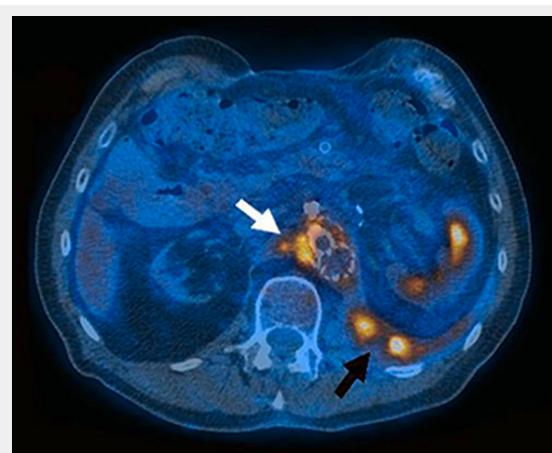
prophylaxis, normothermia, control of blood glucose in the setting of surgical procedures and patient surveillance for, and eradication of, nasal *S. aureus* [3].

### Pathogenesis

PVGI is due to bacterial colonisation of the wound and the underlying prosthetic graft, generally because of direct microbial contamination during the operative procedure by the patient's skin flora. Haematogenous spread of bacteria may occur during dental manipulation, and urological or endoscopic procedures. Other routes of infection are: (1) contiguous spread from the adjacent or overlying tissues; (2) bacterial colonisation of atherosclerotic plaques surrounding the prosthetic vascular graft; or (3) colonisation of a thrombus in the aneurysmal sac of the vessel wall [9]. Soft tissue oedema and injured skin structures may add to the risk of PVGI [4]. The implantation of a vascular prosthesis contributes to the risk of a vascular surgical site infection through biofilm formation, which may sustain bacterial colonisation, and protect microorganisms from host defences and antimicrobial therapy. Staphylococci (*S. aureus* and coagulase-negative staphylococci) account for 80% of PVGIs [10], but fungi (mainly *Candida*), other Gram-positive cocci (enterococci, streptococci), or Gram-negative rods (mainly *Escherichia coli*, *Pseudomonas aeruginosa* and *Klebsiella spp.*), are also reported to cause PVGI [11]. Staphylococci and *Pseudomonas spp.* are strong biofilm producers. PVGI is often polymicrobial, with involvement of anaerobes, and there are cases where cultures remain negative, often in the context of previous antimicrobial therapy [10, 12].

### Diagnostic procedures

The diagnosis of PVGI involves several elements, including microbiological and clinical findings and imaging studies, as well as inflammatory markers.



**Figure 1**

FDG-PET: Distinct, focal metabolic activity (SUV 11.8) adjacent to the Y- and renovisceral grafts (white arrow) with a spatial extension to an abscess in the left psoas region (black arrow).

### Microbiological diagnosis

Microorganisms retrieved from superficial or deep wounds may simply represent colonising flora. Therefore, it is essential to obtain bacterial cultures from the explanted grafts, if available, or from tissue surrounding the graft [12]. For adequate sampling, surgery is often mandatory. Molecular methods such as the 16s rRNA polymerase chain reaction (broad range PCR), and microbiological recovery techniques with broth cultures and sonication of the graft, are used for diagnosis of PVGI [13, 14]. To date, the relevance of cultures obtained from foams used for negative pressure wound therapy (NPWT) is still debated [15].

### Clinical diagnosis

The clinical presentation of PVGI is very variable and depends upon the location of the vascular graft (aortic or peripheral) and upon the timing of the infection after surgery (early or late).

*Early infections* develop less than 3 months after surgery. They often present with acute onset of fever, bacteraemia, pain, erythema, swelling, warmth, local bleeding, ulcer formation, graft occlusion or a pulsatile mass (from mycotic pseudoaneurysm) in the groin. *Late infections* develop more than 3 months after surgery. They may present with more subtle symptoms and signs such as back pain, fistula, graft occlusion, asymptomatic pseudoaneurysm formation and poor incorporation of the graft into the surrounding tissue. Systemic manifestations of infection are often absent (especially in diabetic patients) and blood cultures are often negative. Sometimes overlying soft tissue infections are difficult to distinguish from PVGI. There are two classification systems for vascular graft infections (the Szilagyi [16] and the Samson classifications [17]), which were developed for vascular groin infections in particular (table 1).

### Imaging techniques

The methods most commonly used to evaluate whether vascular grafts are infected include ultrasound, contrast enhanced computed tomographic angiography (CTA), 18F-fluorodeoxyglucose positron-emission tomography (FDG-PET) and fusion PET/computed tomography (CT). Perigraft fluid and inflammation can be rapidly identified with ultrasound, but CT is generally considered the diagnostic test of choice. CTA scans are assessed for the presence of ectopic gas, perigraft fluid (<20 Hounsfield units), perigraft soft tissue enhancement (>20 Hounsfield units), pseudoaneurysm formation, discontinuity of the aneurysmal sac and an increased amount of soft tissue (>5 mm) between the graft and the surrounding aneurysmal wall [18]. CT-guided puncture may yield periprosthetic fluid for Gram stain and culture to establish the microbiological diagnosis.

In advanced PVGI, the sensitivity of CTA is nearly 100% [19]. However, in the case of low-grade PVGI, the risk of false negative results is high [20]. Therefore, additional imaging modalities are needed. The use of FDG-PET to show the intensity (grade 0–4) and patterns (focal or diffuse) of FDG uptake for the evaluation of infected vascular grafts seems to be promising (figure 1). In a study of 33 consecutive patients with a suspected infected arterial prosthesis, FDG-PET showed a superior sensitivity (91% vs 64%), but a lower specificity (64% vs 84%) compared with

CTA. However, when focal uptake was taken as the positive criterion in FDG-uptake [21, 22], the specificity and positive predictive value of FDG-PET for the detection of graft infection improved to 95% [23]. A recently developed fusion technology of FDG-PET and CTA, in a single session, enables the precise localisation of any abnormal FDG uptake, and three studies have confirmed that PET/CTA is a reliable tool for the diagnosis of PVGI [24–27]. However, false-positive FDG uptake because of foreign body reaction has to be taken into account [23].

## Management

Up to now there are no clear guidelines for management of PVGI, mainly because of the variable clinical presentation and the lack of data from randomised controlled trials. Treatment modalities generally involve both surgical intervention and systemic antibiotics. Antimicrobial therapy alone without surgery is associated with a poor response and high mortality, and is therefore not recommended [2]. However, small case series have shown promising results for long-term suppressive antibiotic therapy in patients who are not fit for surgery because of co-morbidities or an unacceptably high perioperative risk [28–30].

### Surgical treatment

As well as aggressive debridement and the use of antibiotics, many earlier treatment approaches recommended graft excision and revascularisation through a non-infected field in a one-stage procedure. The drawbacks of extra-anatomic bypass revascularisation are long procedure time, low patency, high amputation rates and the risk of rupture along the suture line. These led to various graft-preserving treatment options such as partial graft excision and *in situ* reconstruction with cryopreserved homografts, fresh arterial allografts, autologous veins, or antibiotic- or silver-bonded prosthetic grafts [3, 31–33] with or without muscle flap coverage. The incidence of infection in arterial and vein grafts is lower than in prosthetic vascular grafts, but complications related to the harvest site should not be ignored [8]. The antibiotics most commonly used to coat vascular grafts are fusidic acid, quinupristin-dalfopristin, vancomycin, teicoplanin, gentamycin and rifampicin. Three randomised controlled trials evaluated the efficacy of rifampicin-bonded grafts in preventing early wound and/or graft infection with promising results [34–36]. However, genuine or acquired rifampicin resistance of microorganisms isolated from PVGI has to be taken into account [33]. Although all these alternative techniques proved to be superior to traditional methods in terms of rates of reinfection,

conduit failure and amputation rates [37], overall mortality was still high, underlining the need for novel surgical approaches.

Negative pressure wound therapy (NPWT) has recently provided new options for the treatment of infected vascular wounds [38], although treating a vascular infection by means of a local surgical therapy seems to be counterintuitive. NPWT has been increasingly used for the treatment of vascular surgical site infections either as a bridge to surgical closure or as a primary wound treatment modality [11, 39–41]. Additionally, a combined endovascular and surgical approach using radical debridement and NPWT on top of an endograft has been reported to lower mortality in the treatment of infected arterial ruptures [42].

### Antimicrobial therapy

There is no consensus on which classes of antimicrobial agents are adequate for empirical treatment of PVGI. The British Society for Antimicrobial Chemotherapy (BSAC) Steering Group on the treatment of hospital infections recommends combination treatment with cefuroxime and metronidazole [43], but since staphylococci and Gram-negative rods are likely to be isolated, empirical treatment might also include a penicillinase-resistant beta-lactam or a glycopeptide, plus an aminoglycoside for Gram-negative coverage and synergistic treatment in case of staphylococcal infection [44]. In a French study involving 37 patients admitted to the Intensive Care Unit for suspected or proven PVGI, combination therapy including aminoglycosides was associated with a lower 30-day mortality [45]. Empirical antifungal therapy is probably not necessary, but empirical combination treatment might include rifampicin because of its good penetration into biofilms. When the microorganisms involved are isolated, their susceptibility should be tested in order to optimise bacterial coverage and to narrow the antimicrobial spectrum.

For oral therapy, quinolones, trimethoprim-sulfamethoxazole, tetracyclines and rifampicin should preferably be used because of their high oral bioavailability. An increasing prevalence of multidrug-resistant organisms isolated from PVGI has been observed in recent years [4, 46, 47]. Whether multidrug-resistant organisms must be considered in the prophylaxis or empirical treatment of PVGI depends on the local patterns of resistance in individual hospitals. Details of the type and duration of antimicrobial therapy are unfortunately often missing in reports of studies of PVGI. A superficial infection might be treated with a 1-week course of oral antibiotics, whereas a deeper infection involving the subcutaneous tissue should presumably be treated longer [39, 48, 49]. The duration of treatment

**Table 1:** Classification systems for vascular graft infections.

	Szilagyi Classification [16]	Samson Classification [17]
Group 1	Infection involves only the dermis	Infection extends no deeper than the dermis
Group 2	Infection extends into the subcutaneous tissue but does not invade the arterial implant	Infection involves subcutaneous tissues but does not come into grossly observable direct contact with the graft
Group 3	The arterial implant proper is involved in the infection	Infection involves the body of the graft but not at an anastomotic site
Group 4		Infection surrounds an exposed anastomosis, but bacteraemia or anastomotic bleeding has not occurred
Group 5		Infection involves a graft-to-artery anastomosis and is associated with septicaemia and/ or bleeding at the time of presentation

for Szilagyí grade 3 or Samson grade 3–5 PVGI remains uncertain, and is highly dependent on the extent of infection, the location (peripheral or aortic), and the type of graft material (synthetic, biological or vein). Presumably, antimicrobial therapy should be prolonged, as is the case with prosthetic valve endocarditis [50]. After surgical debridement of the surrounding tissue (i.e., removal of necrosis, infected tissues and biofilm) and the infected graft (i.e., removal of biofilm), intravenous antimicrobial therapy for at least 4–6 weeks is warranted. Calligaro *et al.* and Legout *et al.* even suggest intravenous antibiotic treatment for 6 weeks followed by oral anti-infective treatment for a minimum of 6–12 months [3, 44]. In a small case series of nine Japanese patients with infected aortic grafts, surviving individuals received intravenous antimicrobial treatment followed by oral therapy for up to 3–6 months [51]. In a retrospective study of 68 infected autogenous vein grafts, the duration of intravenous treatment was 3 weeks without additional oral treatment [44]. However, in a recent study, 20% of patients with Szilagyí grade 3 PVGI treated with NPWT and graft preservation without administration of any antibiotic showed good long-term results [11]. Future studies should focus therefore not only on the various forms of surgical treatment but especially on the need for antibiotic treatment.

## Outcome

Few studies have compared the outcomes of different surgical strategies for treatment of vascular surgical site infection. Ohta *et al.* compared different graft-removal techniques with either extra-anatomic or *in situ* revascularisation, immediate vs staged timing of graft excision, and different graft materials (prosthetic, autogenous or allograft). The authors concluded that *in situ* techniques were superior to extra-anatomic reconstructions in preventing new graft failure and early mortality [51]. In another study of 85 patients, age >70 years and aortic graft infection were independent risk factors for in-patient mortality [44]. Graft preserving techniques are increasingly used [3], and clinical cure rates of up to 100% have been reported in small case series if the graft was patent, and if there was no evidence of systemic sepsis, local bleeding or formation of a pseudoaneurysm [51]. For the largest series (44 patients), Mayer *et al.* recently reported an excellent outcome with a graft preserving approach including NPWT as an adjunct [11]. All the patients survived 30 days. One-year mortality was 16% (7/44), and long-term mortality after a mean follow-up of 43 months was 41% (18/44).

## Conclusion

Prosthetic vascular graft infections are catastrophic events with a high morbidity and mortality. To date, there is no consensus about the best surgical treatment algorithm or

**Table 2:** Diagnosis and management of Prosthetic Vascular Graft Infection (PVGI).

Diagnostic or management modalities		Remarks
<b>Imaging</b>		
Extremities or neck	Ultrasound	Detection of pseudoaneurysms Can be performed at the bedside
Thorax, abdomen or pelvis	Combined FDG-PET/CTA	Precise localisation of abnormal FDG uptake Imaging-guided aspiration possible
	MRI	Good soft tissue resolution
<b>Microbiology</b>		
	Blood cultures	Identification of microorganism and antimicrobial resistance testing
	Tissue cultures	Identification of microorganism and antimicrobial resistance testing
	Broad range PCR	Identification of microorganism if cultures are negative >72 h
<b>Surgery</b>		
Graft and anastomosis intact	Graft preservation Debridement Negative pressure wound therapy	Plus antimicrobial therapy
Graft and anastomosis at risk for disruption	Surgical removal of parts at risk Local <i>in situ</i> reconstruction Debridement Negative pressure wound therapy	Plus antimicrobial therapy
<b>Antimicrobial therapy</b>		
	Empirical therapy	Systemic manifestations, SIRS or surgical intervention delayed (>24 h): Start empirical therapy immediately No systemic manifestations, no SIRS and prompt surgical intervention (<24 h): Hold antimicrobials until microbiological results are available
	Targeted therapy	In accordance with blood cultures, surgical deep tissue cultures or broad range PCR results
	Suppressive therapy	Long term suppressive antimicrobial therapy in the case of inoperable situation
Abbreviations: FDG-PET, 18F-fluorodeoxyglucose positron-emission tomography; CTA, computed tomographic angiography; MRI, Magnetic resonance imaging; Broad range PCR, 16s rRNA Gene Polymerase Chain Reaction; SIRS; Systemic inflammatory response syndrome		

antimicrobial management of PVGI. Recently, however, more targeted and conservative approaches (graft preserving techniques), especially when supported by NPWT, have shown excellent long-term results in nonrandomised case series. In order to address these uncertainties, we plan to establish a prospective, observational cohort study of a multidisciplinary approach to the management of PVGI at the University Hospital of Zurich (VASGRA-Cohort). Until further results are available, graft infection should be treated according to current knowledge; this includes repeated radical local debridement, graft preservation (if possible), NPWT and targeted antimicrobial therapy (table 2).

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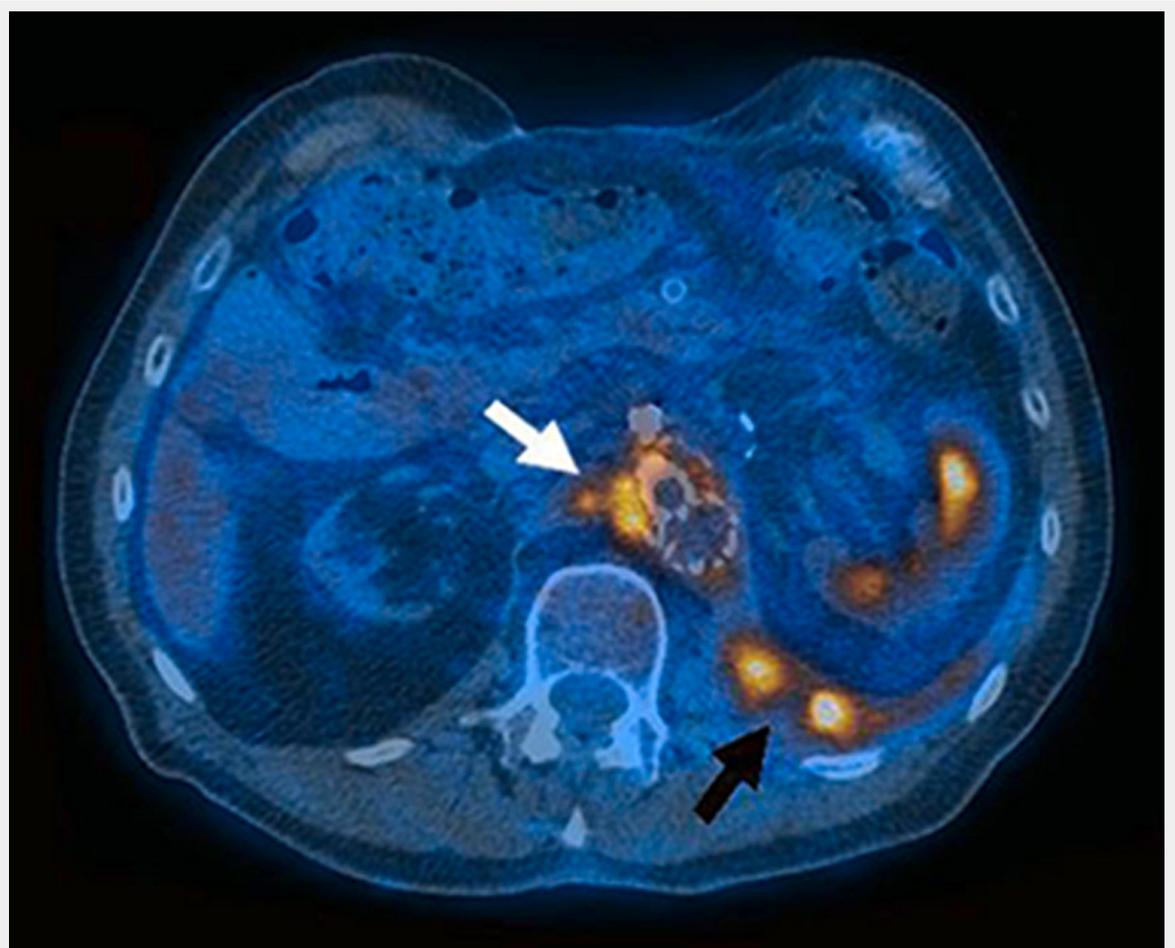
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Figures (large format)



**Figure 1**

Distinct, focal metabolic activity (SUV 11.8) adjacent to the Y- and renovisceral grafts (white arrow) with a spatial extension to an abscess in the left psoas region (black arrow). ■■ imaging technique?■■