Chapter from the book *Vascular Surgery - Principles and Practice*
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1. Introduction

Technical advances in Vascular Surgery have led to an increased use of prostheses (grafts, patches, stents, stent grafts etc.) and improved results for the patient. Despite routine antibiotic prophylaxis, infection, although rare, remains a serious complication, with catastrophic consequences. Vascular infections are divided into 3 groups according to Szilagyi (Table 1.), depending on the extent of the inflammation: the superficial, the deep and the mixed type.[1] Samson (Table 1.), as well as Karl and Storck (Table 1.), have modified the widely used classification system of Szilagyi.[1-3] While the superficial type is restricted to the skin and subcutaneous tissue, the deep infection involves the vessels or a prosthetic graft. The mixed type of vascular infection is the combination of the above types affects all the tissue layers and can produce trauma disruption. Vascular infections can be classified by appearance time into: a) early (<4 weeks after graft implantation) and b) late (>4 weeks). Samson’s and Karl’s modifications take into consideration further clinical parameters, which define the treatment (Table 2.). [2,3] When infection involves a graft anastomosis or the suture line of a patch, there is high risk of vessel rupture, septic hemorrhage or pseudoaneurysm formation. [4-6] Other serious complications are septic thrombosis, endocarditis, etc. [7] In severe cases, treatment can be problematic and mortality remains high, despite the use of antibiotics and surgical treatment. Keys to successful outcome include early and accurate diagnosis, identification of the infecting organism, and extent of graft infection, administration of culture-specific antibiotic therapy, and excision or replacement of the infected graft.

2. Epidemiology

The reported incidence of infection involving vascular prosthesis varies, occurring after 0.2% to 5% of vascular procedures. [4] The long-term incidence is possibly higher than that reported, since some graft infections (e.g. aortic graft infections) develop several years after implantation. [8]
### Table 1. Classification of vascular graft infections

<table>
<thead>
<tr>
<th>Groups</th>
<th>Szilagyi</th>
<th>Samson</th>
<th>Karl-Storck</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>infection involves only the dermis</td>
<td>infections extend no deeper than the dermis</td>
<td>Conservative treatment</td>
</tr>
<tr>
<td>II</td>
<td>infection extends into the subcutaneous tissue but does not invade the arterial implant</td>
<td>infections involve subcutaneous tissues but do not come into grossly observable direct contact with the graft</td>
<td>Partial graft infection without involvement of the anastomosis</td>
</tr>
<tr>
<td>III</td>
<td>the arterial implant proper is involved in the infection</td>
<td>infections involve the body of the graft but not at an anastomosis</td>
<td>involvement of the anastomosis and suture line</td>
</tr>
<tr>
<td>IV</td>
<td></td>
<td>infections surround an exposed anastomosis but bacteremia or anastomotic bleeding has not occurred</td>
<td>Wound disruption and complete exposure of the graft/patch</td>
</tr>
<tr>
<td>V</td>
<td></td>
<td>infections involve a graft-to-artery anastomosis and are associated with septicaemia and/or bleeding at the time of presentation</td>
<td>All the above groups with concomitant septic bleeding/pseudoaneurysm</td>
</tr>
<tr>
<td>VI</td>
<td></td>
<td></td>
<td>All the above groups with graft thrombosis or septic emboli</td>
</tr>
</tbody>
</table>

### Table 2. Therapeutic recommendations depending on the infection grade

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical findings</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Szilagyi I, Samson I</td>
<td>Infection involves only cutis</td>
<td>Conservative treatment</td>
</tr>
<tr>
<td>Szilagyi II, Samson II, Karl I</td>
<td>Cutis/subcutis infection without graft involvement</td>
<td>a) graft preservation combined with VAC</td>
</tr>
<tr>
<td>Szilagyi III, Samson III, Karl II</td>
<td>Deep graft infection without involvement of anastomosis or suture line</td>
<td>a) graft preservation combined with VAC</td>
</tr>
<tr>
<td>Szilagyi III, Samson IV, Karl III-IV</td>
<td>Deep graft infection with involvement of anastomosis or suture line</td>
<td>a) graft excision</td>
</tr>
<tr>
<td>Szilagyi III, Samson V, Karl V-VI</td>
<td>Deep graft infection associated with complications (bleeding, thrombosis, suture aneurysm)</td>
<td>b) graft preservation combined with VAC</td>
</tr>
<tr>
<td></td>
<td></td>
<td>graft excision</td>
</tr>
</tbody>
</table>
Incidence of vascular infections is influenced by patient’s general condition, the type of the procedure, the coexistence of other simultaneous inflammation sites, the type of prophylactic antibiotics given perioperatively and by prolonged operative time and hospital stay. [9-13] Infections are much more frequent in the groins (60% of cases), in grafts placed in a subcutaneous tunnel and after emergency cases (e.g. acute arterial ischemia). Infection can also develop after percutaneous stent angioplasty but in low rates (0.5%). [14,15]

Early graft infections usually affect extracavitary grafts, while majority of late infections involve cavitary (i.e., aortic) grafts. [16]

3. Pathogenesis

Exposure of vascular grafts to bacteria, irrespective of source, may result in colonization and subsequent infection. Microorganisms can result in clinical infection most commonly, perioperatively, during surgical implantation or through the surgical wound. The most common mechanisms of infection are: break of aseptic techniques in the operating room and contact of the graft with patient’s endogenous flora harboured in lymphatics rupturing intraoperatively, sweat glands or mucosas. Intraoperative injury of gastrointestinal or genitourinary tract, diseased arterial wall, healing problems of surgical wound and reoperations can result in graft infection. [4]

Bacterial contamination of the prosthesis via a hematogenous route is rare, though urinary tract infections, infected intravascular catheters, pneumonia or other remote tissue infections (e.g. infected foot ulcer) increase the risk of graft infection. Bacteremia can result in graft infection, years after the implantation, especially in elderly patients with altered immune status.

Moreover, erosion of a prosthetic graft through the skin or into the gastrointestinal or genitourinary tract can lead to an infection. GEE/GEF can develop due to pulsatile pressure transmitted via an aortic graft to the overlying adherent bowel, usually the third part of the duodenum. This can be prevented by coverage of the graft by adjacent omentum at the end of the procedure. A graft-cutaneous fistula by erosion through intact skin is most commonly the result of a low-grade infection caused by S. epidermidis.

Finally, grafts can get contaminated by a contiguous infectious process as a result of an adjacent infection (e.g. diverticulitis, infected lymphocele).

Predisposing factors for vascular infection are the use of prosthetic grafts, procedures in the groins, local or systemic septic conditions, while the predicting factors are patient’s immune status, graft’s characteristics, prolonged hospital stay, bacterial virulence or resistance to antibiotics. Additionally, reoperations, long or emergency procedures, faulty sterile surgical technique, postoperative complications (such as hematoma, graft thrombosis) and concomitant urological or biliary and colon operations contribute to increased rates of vascular infections. [17]
4. Bacteriology

Staphylococci (*Staphylococcus aureus* and coagulase negative staphylococci) account for more than 75% of vascular device-related infections. In fact, *S. aureus* is the most prevalent pathogen. Graft infections due to *S. epidermidis* or gram-negative bacteria have increased in frequency. Less frequently, microorganisms of the skin flora, such as streptococci and *Propionibacterium acnes*, are isolated.

Gram-negative bacteria such as *Pseudomonas, E. coli, Klebsiella, Enterobacter,* and *Proteus* species are particularly virulent, followed by high rates of anastomotic disruption. This can be explained by their ability to produce toxins, such as elastase and alkaline protease, which can decompose the arterial wall. [18,19]

MRSA (Methicillin-resistant *S. aureus*) vascular infections present with increased incidence. [20] Fungal infections are rare and develop usually in immunosuppressed patients.

Early infections are usually caused by especially virulent microorganisms, such as *S. aureus, Streptococcus faecalis, E. coli, Pneumococcus, Klebsiella and Proteus*. Late infections are the result of low-virulence microorganisms such as *S. Epidermidis*.

5. Clinical manifestations

Clinical manifestations vary according to the localization of the vessel that is involved. Graft infections in limbs (e.g. femoropopliteal graft) present with edema, cellulitis or with a pulsatile mass, in case anastomotic rupture and pseudoaneurysm formation. According to Szilagyi, vascular infections can be classified by relationship to postoperative wound infection. Graft contamination in the abdominal (Table 3.) or thoracic cavity, usually presents with systematic sepsis, aortoenteric, and aortobronchial or aortooesophageal fistula. Symptoms in early infections can be fever, leukocytosis and perigraft purulence.

Patients with aortic grafts and gastrointestinal bleeding should be investigated for GEE. [21,22] Bacteremia develops in advanced graft infections. Graft infection due to *S. epidermidis* typically presents months to years after graft implantation with anastomotic aneurysm, graft-cutaneous sinus tract or perigraft cavity with fluid. Vascular Surgeon should, also, look for other sources of infection, (e.g. feet or urinary infections).

6. Diagnosis

6.1. Laboratory testing

Early diagnosis is crucial for treatment and for prevention of septic complications that can threaten the affected limb or even patient’s life. It is based on physical examination and imaging modalities. Blood tests results are non-specific for vascular infection, with low diagnostic value. Elevated WBC count with left shifted differential, increased erythrocyte sedimentation rate or high levels of CRP can be found during the acute phase. Blood cultures are rarely positive (<5%) but such findings, in addition with high fever, are markers
of advanced infection and sepsis. In these cases, early hospital admission and treatment are essential. Laboratory tests should include cultures from other sites of infection and stool guaiac, in case GEE is suspected.

<table>
<thead>
<tr>
<th>Type of graft infection</th>
<th>Time from implantation</th>
<th>Microorganisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periprosthetic infection</td>
<td>Early (&lt; 4 mo)</td>
<td>Staphylococcus aureus, Streptococcus, Escherichia Coli, Pseudomonas</td>
</tr>
<tr>
<td></td>
<td>Late (&gt; 4 mo)</td>
<td>Staphylococcus epidermidis</td>
</tr>
<tr>
<td>Entero-paraprostatic</td>
<td>Late</td>
<td>Escherichia coli, Enterococcus, Bacteroides infection</td>
</tr>
<tr>
<td>Aorto-enteric fistula</td>
<td>Early</td>
<td>Escherichia coli, Staphylococcus aureus</td>
</tr>
<tr>
<td></td>
<td>Late</td>
<td>Escherichia coli, Klebsiella, Staphylococcus epidermidis</td>
</tr>
</tbody>
</table>

Table 3. Classification of aortic graft infection (Bandyk 1991)

6.2. Vascular imaging

Vascular imaging is of crucial significance in the diagnosis and treatment planning of vascular infections. Imaging modalities that are useful for diagnosis are ultrasonography, CT Angiography, MR Angiography, endoscopy or functional radionuclide imaging (indium 111-labelled leukocytes). The combination of anatomic and functional vascular imaging techniques shows high sensitivity (80% to 100%) and specificity (50% to 90%) in identification of infection.

Plain radiographs are of limited value, providing information only in the case of prosthesis misplacement or dislocation.

Color duplex scanning is a readily available imaging technique, reliable for diagnosis of perigraft fluid collection, which can be differentiated from anastomotic pseudoaneurysms, especially in extracavitary infections. Imaging of abdominal cavity or aortic grafts is not accurate in obese patients. Graft patency can be easily examined.

Contrast-enhanced CT is the preferred imaging technique for abdominal or thoracic aorta graft infections. Signs of abnormal fluid or gas collections around the prosthesis (beyond 2-3 months of implantation) or false aneurysm formation are suggestive of infection. Loss of normal retroperitoneal tissue planes or vertebral osteomyelitis in a patient with an aortic graft indicates a vascular infection. CT-guided aspiration is being increasingly used to differentiate perigraft abscesses from seromas.
MRA is an alternative modality to CTA, with equal specificity or sensitivity. It can also differentiate perigraft fluid from adjacent fibrosis. Gadolinium is less nephrotoxic in patients with renal insufficiency. However, it is contraindicated in patients with electrophysiological devices. The presence of metallic materials may cause artefacts that compromise image quality.

The use of arteriography is useful in the identification of anastomotic aneurysms or other graft complications (e.g. graft rupture) and for the evaluation of the vascular tree before revascularization planning. It should be a routine examination in hemodynamically stable patients with graft infection unless CT or MRI scans give the above type of information.

Functional White Blood Cell Scanning is indicated in special cases. 99mTc-labelled white blood cells, 111In or gallium scintigrams are most commonly used along with MRI and CT to define the extent of graft involvement. Positive predictive value of the functional imaging scans ranges between 80% to 90% in the detection of graft infection. False-positive results are not uncommon during the early postoperative period.

Endoscopy is very useful in cases of suspected secondary aortoenteric erosion or fistula and is an emergency procedure in patients with massive gastrointestinal bleeding where it can be performed in the operating theatre, with the patient prepared for operation. It is important is to visualize the third and fourth part of duodenum and rule out other sources of gastrointestinal bleeding. Though, an aortoduodenal fistula cannot be excluded by negative findings.

7. Operative findings

Operative exploration is sometimes mandatory for the final diagnosis, especially in unstable patients or in cases with a history of aortic grafting and gastrointestinal bleeding, where a GEF is suspected. Unfortunately only 50% of GEFs can be diagnosed by CT or MRI modalities. Operative exploration, graft excision and broth culture of the graft can lead to isolation of the responsible microorganisms and selection of proper antibiotic treatment.

8. Prevention

Prevention of graft contamination perioperatively is of great importance, given the high mortality and morbidity that follows a vascular infection. Antimicrobial prophylaxis should be administered within 60 min before incision and discontinued within 24 h after surgery. According to the published consensus of the Surgical Infection Prevention Guideline Writers Workgroup (SIPGWW), the recommended prophylactic antibiotics for cardiothoracic and vascular surgery include cefazolin and cefuroxime. [23] For intra-abdominal surgery coverage for anaerobes may be added (metronidazole). [24]

Culture-specific antibiotics should be administered to patients who have coexisting infections.

There are some principles that should be followed perioperatively, in order to prevent an infection:
• Patients should scrub the night before the operation
• Hair of the operative site should be removed by clippers and not by razors so as to prevent skin trauma
• Preoperative hospital stay should be minimized, if possible
• Remote infections must be controlled before elective grafting interventions
• Concomitant gastrointestinal procedures should be avoided, if a graft is planned to be used (cholecystectomy for asymptomatic cholelithiasis is possibly excepted)
• The use of iodine-impregnated plastic drapes is recommended, so as to prevent graft contamination
• Meticulous sterile technique is vital
• Careful hemostasis and closure of surgical incisions in multiple layers are recommended
• Irrigation of groin wounds with topical antibiotics before closure may decrease infection rates. [25]

9. Therapeutic management of vascular infection

9.1. General principles

Presentation of vascular infections varies and there is, usually, no standard treatment. Treatment should be individualized according to infection site, clinical presentation and the isolated microorganisms. For the extracavitary graft infections there are some recommendations, based on infection grade, simplifying the complexity of treatment. (Table 2) The main goal is eradication of the infection while preserving blood flow to the target organs or limbs.

Preparation of the patient is important, though takes time. In unstable patients due to septic or hypovolemic shock, no delay is justified. Blood or fluid resuscitation, antibiotic coverage and urgent surgical treatment are the only option. For the rest of the cases, where time is available, patient’s cardiac, pulmonary and renal function should be optimized. Diabetic patients must have their glucose levels controlled. Malnourished patients can improve by enteral or parenteral nutrition. When an abdominal operation is planned, colon should be mechanically, cleansed. A Duplex scan of the lower limb veins is recommended, especially in cases of in situ replacement with autologous graft. Preoperative antibiotic coverage of the patient is crucial.

Available options include graft excision with or without revascularization and graft preservation with local treatment. Graft excision can be followed by extra anatomic revascularization or in situ replacement of the graft.

9.2. Preservation of the graft

Preservation of the infected graft is indicated in few, selected cases, usually when infection involves autologous vein grafts or patches. [26-28] Patients must have no signs of sepsis and the graft should be patent with segmental contamination. Anastomoses must be spared.
Outcome is better with vein or PTFE than polyester grafts, with early than late infections (<4 months) and with extracavitary grafts. Infections caused by single Gram positive and not multiple Gram negative organisms (e.g. Pseudomonas) may be considered for graft preservation and local treatment.

Local treatment includes staged surgical debridement of infected tissues in healthy plane, mechanical irrigation of the wound (using povidone iodine solution and peroxide), on a regular basis, rotational muscle flap coverage, temporary use of antibiotic impregnated beads and VAC devices (vacuum assisted closure devices for wounds). Intravenous culture-based antibiotics are essential. Persistent infection or sepsis is an indication of treatment failure which happens in 30% of the patients. [27] In such cases, graft excision with or without revascularization should follow.

9.3. Graft excision

Graft excision without revascularization is rarely an option, mostly in patients where the indication for the initial procedure was claudication, or in cases where the infection has led to graft thrombosis but with no signs of critical ischemia. In patent infected grafts, the decision regarding the need for immediate revascularization is based on temporary graft occlusion. The presence of Doppler pedal pulsatile signal and systolic ankle pressure greater than 40 mmHg is a sign of sufficient preexisting collaterals. In cases of infected bypass grafts with end to side anastomosis, the graft can be removed and an autologous patch can be placed at the site of proximal anastomosis.

In the majority of the cases, graft excision should be accompanied by revascularization of the target organs or limbs, usually by means of extra-anatomic PTFE bypass, through uninfected tissues.

This technique is suitable, mainly for aortoiliac or aortobifemoral infected grafts, for patients with GEE/GEF or for more diffuse infections with signs of systemic sepsis. Graft excision can be accomplished through celiotomy or left-side retroperitoneal incision, so as to avoid contaminated areas. Preoperative stenting of the ureters is recommended in cases of extensive infection, for protection during dissection and easier identification. Supraceliac aortic clamping and control of iliac arteries (at healthy segments, distally to the infected part of the graft) may be necessary, though sometimes difficult due to perigraft inflammation. Some centers advocate the use of intraluminal occlusion balloons. Meticulous dissection of the adherent viscera’s or duodenum, especially in patients with GEE/GEF is important. Necrotic bowel segments must be excised and bowel continuity should be restored by end to end anastomosis. Complete removal and culture of the aortoiliofemoral graft must follow. Extensive debridement and irrigation (by use of cytotoxic agents) of perigraft contaminated or necrotic tissues are essential. Closure of the aortic stump is performed by double layers of interrupted monofilament sutures. Prosthetic pledgets should be avoided. Coverage of the aortic stump with omentum pedicles is believed to prevent stump blowout and its catastrophic consequences. The same technique can be applied for the ligation of iliac arteries, but flow must be maintained at least to one hypogastric artery, in order to avoid
pelvic or colon ischemia. Placement of closed suction drains can be placed in the retroperitoneal space. Reported mortality rates range between 11-22%, while limb loss 10-11%. [20,29] Stump blowout, which is a major complication, can happen up to 22% of the cases. [7]

Several authors suggest that staged management of infected aortic grafts, show lower morbidity and mortality rates. [29,30] Hemodynamically unstable patients, are an exception, and the vascular surgeon should focus on the site of hemorrhage (septic hemorrhage from anastomosis, GEE/GEF). In the rest of the cases, it is recommended, to perform the extra-anatomic bypass first, and graft excision can follow 1 to 2 days later.

Aortobifemoral graft infections, especially in the groins, constitute a challenge for the surgeon. Unilateral ex situ bypass to the profunda femoris or superficial femoral artery through uninfected planes is an option, while bypass to the popliteal artery results in low rates of patency (58% in 6 months). [31] In bilateral groin infections, graft excision followed by unilateral axillofemoral bypass and autogenous vein cross-femoral bypass is another solution.

In-situ graft replacement is an alternative solution, in selected cases. There should be no systemic signs of sepsis, any anastomotic bleeding or perigraft incorporation. Perigraft fluid cultures must be sterile unless bacteria of low virulence, such as S. Epidermis, are isolated. In fact, in patients with infection that involves the thoracic aorta or the visceral segment of abdominal aorta, in situ replacement may be the only option available. The most common grafts used in this technique are autologous grafts (e.g. superficial or deep veins of the limbs), antibiotic bonded prosthetic grafts or cryopreserved arterial allografts. (Figure 1.)

Great saphenous vein (GSV) or superficial veins from the upper limb can be used in cases where infection affects infrainguinal, upper extremity, visceral or cerebrovascular procedures. A preoperative Duplex vein mapping is essential for estimation of vein’s condition and diameter.

However, use of GSV in ilio-femoral or aorto-iliac reconstructions, results in low patency rates, due to diameter mismatch. [32] In these cases, superficial femoral vein harvesting has a strong indication. [33,34] Preoperative vein mapping is important. In cases of aortic reconstruction, with larger aortic diameter, “pantaloone technique” can be applied. (Figure 1.) Compared to graft excision and extra anatomic bypass, in situ graft replacement presents better patency and recurrent infection rates. [35] Superficial femoral vein can be used also, in aortofemoral graft infections localized in the groin caused by S. epidermidis. However its use in secondary GEE/GEF is not recommended. Deep veins are used non reversed, after valve excision.

Antibiotic bonded prosthetic grafts (PTFE or Dacron), can be used in segmental graft infections, where the isolated microorganism is of low virulence (e.g. S. Epidermidis) and the anastomoses are spared. [20] For example in segmental aortofemoral graft infections, with groin complications, especially in elderly patients, antibiotic bonded prosthetic grafts should be considered for replacement of one limb of the pre-existing graft.
An alternative option, especially in more diffuse infections, is the use of cryopreserved arterial allografts. While the survival and recurrent infection rates are comparable to other grafts, increased dilation (17%) and stenosis (20%) rates were noticed. [36]

Overall, outcomes following deep venous replacement are better than with the use of arterial allografts or implantation of a “new” prosthetic graft. When applied to low-grade aortic graft infections without GEE or GEF, this procedure is safe (4% in-hospital mortality), with a low (3%) incidence of long-term limb loss. In cases with GEE/GEF, mortality can reach 20%, similar to graft excision and ex-situ bypass.

10. Adjunctive treatments

10.1. Antibiotic-loaded beads

In vascular infections, where graft preservation and serial debridement of the wound is the selected treatment, implantation of antibiotic–loaded beads is an alternative adjunctive therapy. They are mainly used in extracavitary graft infections. Beads are usually loaded with vancomycin, daptomycin, tobramycin, or gentamicin based on initial culture results. Initial results are encouraging, with wound healing in 90% of the cases. [37]

10.2. Muscle flap coverage

Infected grafts that are treated locally must be surrounded by healthy, non-contaminated tissues. Coverage of the graft with a well vascularised, not infected muscle flap, contributes to wound healing. Sartorius muscle flap coverage is the most common technique used in
graft infections located in the groins. [38] This technique is mainly indicated, as an adjunct of graft preservation or in situ replacement therapies, especially in cases of recurrent infections or extensive tissue deficit after debridement. The muscle is divided from its proximal attachment to the iliac crest and sutured medially, so as to cover the infected graft. In a published series, recurrent infection rate after use of Sartorius flap was only 7%. [39]

Another similar technique is the rotational use of flaps of muscles that are mobilized from a separate healthy site. Their blood supply doesn’t come from the infected area. The gracilis, rectus abdominis, tensor fasciae latae or rectus femoris can be used, depending on site of infection. [40] Some authors consider this technique as a better option than the use of Sartorius muscle. [38]

10.3. Antibiotics

When the diagnosis of vascular infection is made, parenteral broad spectrum antibiotics should be given, until isolation of the infecting micro-organism is accomplished, through cultures. Additionally, if cultures reveal no pathogen or there are no available specimens for culture, empiric antimicrobial treatment should target skin-colonizing organisms and nosocomial pathogens as well.

Vancomycin is an indispensable agent in the initial empiric antimicrobial regimen, because of its excellent anti-Gram-positive spectrum. Teicoplanin has a similar antimicrobial spectrum to vancomycin but has not been tested in large prospective series for the treatment of vascular infections. [41-44]

Alternative antimicrobial agents are linezolid and quinupristin/dalfopristin, which provide coverage for methicillin-resistant staphylococci (MRSA and MRSE) and vancomycin-resistant enterococci (VRE). Their use should be reserved for infections due to pathogens resistant to vancomycin, or in patients who are allergic to vancomycin. [45,46]

Once cultures reveal the infecting pathogen, parenteral antibiotic treatment should be initiated, without any delay.

The duration of therapy is individualized but most authors recommend 4–6 weeks of treatment after the removal of the infected graft.

11. Management of specific graft site infection

11.1. Carotid infection

Depending on grading, carotid artery infections are reported up to 2% of cases. [47] Szylagyi III infections are found in a rate of less than 1%. [48,49] The majority of infections are postoperative wound contaminations, which seldom extent to the suture line. Wound dehiscence with septic haemorrhage is extremely rarely observed. There are reports that the use of prosthetic materials increases the infection rate. However, the management of such infections that may lead to catastrophic life-threatening septic complications is especially challenging. The standard treatment includes wound debridement and prosthetic graft
replacement with autologous material (e.g. saphenous vein). Recently the use of sternocleidomastoid muscle flap plasty for coverage of the infected area was described. More recently, carotid stent infections were reported in up to 0.4% of cases. [48] This complication may present primary or secondary to neck irradiation and trauma. [48,50,51] The treatment principles are similar to post-CEA infections. The use of vacuum assisted closure device emerges as a new trend with promising results. [52]

11.2. Infection of vascular access

Vascular access Infection is a major complication for haemodialysis patients. Clinical symptoms vary from simple local inflammation to systemic sepsis. In some cases, septic haemorrhage may develop, which is a life-threatening condition. (Figure 2.) Reported risk factors for this adverse event include immunodeficiency, low serum albumin level, female gender, adult polycystic kidney disease, diabetes mellitus, inadequate dialysis and the use of catheters or synthetic graft. [53] It is estimated that 30 to 50% of bacteraemia in haemodialysis patients is caused by vascular access infection. [53] There are reports that infection rates range from 0.5 to 3.5% for autogenous AVF, 5-8% for prosthetic graft accesses and 2-5.5 episodes of bacteremia per 1000 patient days for central venous catheters. [54,55]

Figure 2. Infection of brachio-cephalic fistula (Cimino) at the wrist with septic bleeding
Early postoperative graft infections usually affect the whole graft. The treatment of choice is excision of the entire graft. Late localised infection at the needles sites can be managed by segmental graft removal and bypass through uninfected planes. Sometimes, though, total excision is necessary.

The use of V.A.C. as an adjunctive treatment may be beneficial. Autogenous AV access infections often can be effectively treated with systemic antibiotics. In case of infected pseudoaneurysms or abscesses access ligation of the access or segmental bypass are mandatory.

Catheter infection presentation varies. Exit-site infections are treated with local antibiotics. In case of failure, parenteral antibiotics should be administered. Tunnel tract infections require intravenous antibiotics, and catheter exchange through a new tunnel and exit site. These patients require at least 3 weeks of culture-based antibiotic therapy and monitoring for recurrent infection. [56] Patients with systemic sepsis should have their catheter removed and a temporary catheter inserted. A new cuffed catheter may be placed if the patient remains afebrile for at least 48-72 hours.

11.3. Infection of thoracic aorta

The incidence of infections affecting thoracic or thoracoabdominal aortic grafts, ranges from 0.5% to 1.9%. Complications can be fatal, and mortality is high. Open surgical repair for primary or secondary thoracic aorta infections are associated with significant mortality and morbidity. Graft excision and extra-anatomic bypass are usually not applicable to infections involving ascending, transverse arch or descending aorta grafts. For most of these cases, in-situ replacement with the use of prosthetic grafts is the treatment of choice. The use of silver-bonded or antimicrobial-bonded synthetic grafts is possibly preferable. Surgical debridement and antibacterial irrigation of infected tissues are important. It is reported that coverage of the graft with pericardial fat, rotated muscles (e.g. pectoralis major, latissimus dorsi, rectus abdominis) or with a pedicle of greater omentum can prevent recurrent infections. [57] Antibiotic coverage is necessary. Mortality is reported to be 10-20% while reinfection rates 20%. [57,58] The only extra-anatomic repair, that may be recommended, is prosthetic grafting from the ascending to abdominal aorta, tunnelling through the diaphragm, with subsequent infected graft excision through a left thoracotomy. Limited surgical strategy involving extensive mediastinal debridement is reported in cases where infection is associated with sternal wound infection by low virulent pathogens. [59] Endovascular stent graft repair has been reported as an attractive and effective treatment option, but the persistence of infection is always a concern. Though in cases of severe local inflammation, with or without haemorrhage, this technique can serve as bridging therapy. [60]

Stent or stent-graft infections in the thoracic aorta are extremely rare. They are usually met in the literature as complications of systemic specific infections such as TBC or brucellosis. The general principles of treatment are similar to the thoracic graft infection. Some papers report secondary stent-graft infections after TEVAR due to aorto-oesophageal or aorto-bronchial fistulas.
11.4. Vascular infections in the groin

Infections after vascular reconstructions are most common in the groins. The main predisposing factors are surgical division of lymphatic channels, infected lymph glands, the superficial location of vascular grafts and the proximity of the surgical site to the perineum. A number of serious complications can arise such as fistula, septic hemorrhage, septic embolism and limb threatening ischemia. [61,62] Imaging of the infected area is essential for the diagnosis. (Figure 3.)

Figure 3. CT findings in a patient with Szilagyi III infection in the groin. (1=fistula, 2=perigraft inflammation/secretion, 3=graft)

There is a lot of controversy about the treatment of choice in groin infections, following vascular graft placement. It depends on the degree of graft involvement. If there is no graft infection (Szilagyi grade I or II), then wound debridement or drainage with culture-directed antibiotic administration is considered to be adequate. If, graft contamination is present
(Szilagyi grade III), then further treatment is controversial. In the majority of the cases, treatment includes excision of the graft, surgical debridement of the infected tissues followed by restoration of blood flow by in situ or extra-anatomic reconstruction.[63,64] Obturator or lateral femoral bypass are the most frequent extra-anatomic procedures for limb revascularization in vascular groin infections. [65-67] An 80% cumulative patency rate at 6 years has been reported. [68] However, many concerns have been associated to extra-anatomic bypass including lengthy procedure time, difficulty of extra-anatomic routing, high amputation rates. [69]. When in-situ reconstruction is selected, cryopreserved aortic homograft, autologous deep femoral vein, or rifampin-bonded prostheses can serve as grafts. Disadvantages associated with in situ reconstructions, include lengthier operative time in case of vein harvesting and contraindication in patients with previous deep vein thrombosis, high complication rates of cryopreserved allografts and lack of availability in emergent cases. In situ reconstructions are associated with higher stress than extra-anatomic bypasses, which is important in high risk patients.

Graft preservation is considered an option when the graft is patent, the entire length of the graft is not involved by the infection, the anastomosis is intact, there are no systemic signs of sepsis and the contaminating organism is not a virulent strain of bacteria, especially MRSA and Pseudomonas aeruginosa. [70,71]

The use of local muscle flaps to promote wound healing and vascular graft salvage has been well documented. [72-74]

VAC therapy has been reported as an adjunctive or definitive treatment for groin infections involving exposed grafts especially in high-risk surgical patients who are not candidates for graft replacement. VAC therapy along with aggressive debridement, antibiotic therapy and muscle flap coverage may be an effective alternative to current management strategies. Some authors recommend the use of V.A.C. even after graft replacement, in treatment of Szilagyi III infections. (Figure 4.)

The majority of current clinical evidence supporting the use of negative pressure therapy (VAC) on infected groin wounds following vascular reconstructions has been based on clinical experience and small cohort studies. However graft/patch salvage rates up to 97.2%, have been reported. [75].

11.5. Infection of femoral, popliteal, tibial grafts

Infection of infrainguinal grafts is quite rare but it can present with anastomotic disruption and septic hemorrhage or emboli. The preferred method of treatment is usually graft excision and revascularization with bypass grafting via adjacent or remote tunneling. In-situ revascularization is feasible in 80% of the cases. The use of autogenous vein grafts is preferred when they are available. Some authors advocate staged treatment. In this case, closure of the arteriotomies with monofilament suture and the administration of systemic and topical antibiotics follow the removal of the graft. Patients who had prosthetic grafts inserted for claudication or patients who do not develop limb-threatening ischemia after graft excision may not need revascularization.
Figure 4. Application of vacuum-assisted closure technique (VAC technique) in a patient with graft infection in groin (A=Szilagyi III infection with cutaneous fistula, B=marking of infected area with use of methylene blue, C=Wound debridement and replacement of infected graft with a silver-bonded synthetic graft, D=preparation of sartorius muscle, E=coverage of the graft with the muscle flap, D=Application of VAC system.
Graft preservation is reported as an alternative option, especially in high risk patients, unless there is sepsis or anastomotic bleeding. In such cases local treatment with surgical debridement, antibiotic administration and muscle flap coverage is applied. [19,30]

Treatment of peripheral grafts infection shows low mortality rates (0-9%) but increased amputation rates (33-67%) compared to treatment of aortic grafts infection. [11,76]

11.6. Endovascular stent-graft and stent infections

Infections involving endoluminal devices (stents or stent-grafts) are rare, although they present with increased frequency. The reported incidence after AAA repair is 0.2% to 1.2%. Infection of peripheral bare stents are extremely rare (<0.1%). They present clinically with sepsis, septic emboli, mycotic aneurysm or GEE/GEF. Periprocedural bacteremia from remote sites of infection or during secondary endovascular interventions is considered to be the cause of stent-graft contamination. [77] Perigraft inflammation or fluid is the main CT findings with diagnostic sensitivity of 85%. Treatment consists of antibiotics and graft excision followed by extra-anatomic bypass or in situ autogenous replacement. Mortality is high and ranges between 20-30%. [78,79] Endovascular treatment should be considered only as a bridging therapy. [80,81]

Author details

Kiriakos Ktenidis* and Argyrios Giannopoulos

1st Department of Surgery, Aristotle University of Thessaloniki, Greece

12. References


* Corresponding Author


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