Dreaded Late Complications: Infection, Blowout, Pseudoaneurysm, Fistula

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Introduction

Late complications are an unfortunate but inevitable part of vascular surgery. These complications arise due to a number of complex pathophysiologic conditions. Infection plagues vascular reconstructions, especially with the use of prosthetic graft materials. Additionally, pseudoaneurysm, anastomotic aneurysms, and frank anastomotic blowout can occur at any time following vascular reconstruction. Aortoenteric fistula formation is also a dreaded complication of aortic reconstruction. Finally, lymphocele development and its associated complications can cause disastrous complications following open vascular surgery. All of these dreaded late complications require careful consideration, diagnosis, understanding of the disease process, and treatment. This chapter will discuss the etiology, diagnosis, pathophysiology, and treatment of these aforementioned complications.

Infection (Surgical Site Infection, Prosthetic Graft Infection)

A vascular surgical site infection (SSI) can range from a simple superficial wound infection to a deep wound or devastating prosthetic graft infection. Graft infections significantly increase the risks of graft failure, limb loss, and mortality. While standard treatment is graft excision and replacement via extra-anatomic bypass, methods such as in situ reconstruction and graft preservation have become increasingly accepted alternatives. Given the variety of treatment options available, the approach to managing graft infections must be individualized to each patient for an optimal

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outcome. This section reviews interventions for the management and prevention of prosthetic graft infection.

Incidence

The incidence of surgical site infection after vascular procedures ranges from 5% to 10% [1]. Peripheral artery bypass procedures have the highest reported rate of infection ranging widely between 3% and 44% with the groin being the most common site of infection [2–4]. Vascular SSI can range from superficial cellulitis, to subcutaneous tissue infection, to involvement of the vascular graft itself. These levels of infection are graded I–III respectively according to the Szilagyi classification [5]. The most dreaded SSI is graft infection which occurs in up to 15% of vascular reconstructions with incidence differing based on location [6]. This incidence is highest in the presence of groin incisions for lower extremity bypass procedures, up to 10.6%, and lowest for aortoiliac grafts ranging from 0.6% to 5% [2, 7–9]. Endovascular device infections are rare with incidence ranging from 0.1% to 1.2% [9, 10].

Vascular procedures are at greater risk of infection than typical clean procedures given a set of unique factors after arterial reconstruction that impede wound healing. These include edema, subsequent superficial wound separation, underlying hematoma or seroma formation, disrupted lymphatics, and non-healing wounds that can all lead to bacterial invasion [1]. Independent predictors of SSI after vascular lower extremity reconstructions include obesity, antiplatelet medication, and previous vascular surgery, particularly for aneurysmal disease and implantation of prosthetic conduits, dialysis dependence, hypertension, intraoperative thrombosis, prolonged operative time, high peak intraoperative glucose, and surgery performed at a larger hospitals or major teaching centers [2, 11].

Pathophysiology

Graft infections can be caused by intraoperative contamination via direct contamination by skin and soft tissue, extension of intra-abdominal infection, communication with the gastrointestinal or genitourinary tract, and graft seeding during episodes of bacteremia.

At the time of surgery, contamination can occur from poor handling of the graft and contamination with skin flora, the source of bacteria in most SSIs [12]. In addition, lymphatics and sweat glands in the groin and plaque and thrombus within vessels can all harbor bacteria. Direct communication with the gastrointestinal tract, such as aortoenteric erosions or fistulas, is uncommon, occurring in 1–2% after open aortic reconstruction. However, they account for up to 25% of endograft infections at time of presentation and will be discussed in further detail in a subsequent section [13, 14]. Extensions from intra-abdominal infections such as diverticulitis or appendicitis are limited to isolated case reports. Bacteremia or hematogenous

spread of bacteria from distant sites of infection to grafts is rare, and it is often difficult to prove which infection came first. Transient bacteremia from colonoscopy or dental procedures may be a potential cause for late infection but evidence is also limited to case reports.

Microbiology

The most common isolated organisms in graft and endograft infections are gram-positive *Staphylococcus* species, including *S. aureus* and *S. epidermidis*, and the gram-negative organism *Pseudomonas aeruginosa* [1, 5, 15–17].

Early infections, occurring within the first 4–6 months, are most likely due to common gram-positive skin contaminants such as *S. aureus* and *Streptococcus* [11, 14, 17]. However, late graft infections are caused mostly by insidious, slow growing, low virulence organisms. The most commonly reported is *S. epidermidis*. These particular bacteria can be present in grafts for extended periods of time without overt evidence of gross infection or positive wound cultures due to production of a protective biofilm. Cultures of prosthetic graft infections may be negative in up to 40% of cases [9, 14–16]. Gram-negative bacteria are involved in approximately a quarter of vascular SSI and most commonly include *E. coli*, *Pseudomonas*, and *Proteus* species [18]. Gram-negative bacteremia in the presence of aortic graft infection should raise suspicion for aortoenteric erosion [1, 9].

Graft material does not appear to affect outcome of infection as much as the organism itself. In a study comparing inoculation of PTFE and vein grafts with low virulent *S. epidermidis* and high virulent *Pseudomonas*, Geary et al. found the virulent *P. aeruginosa* to cause anastomotic disruption in both graft types without discrimination [19].

Clinical Presentation

Diagnosis of infection begins with clinical presentation and physical examination, which varies depending on the graft location, organism, and timing of the infection. Early infections are defined as onset within the first 4–6 months postoperatively and are typically caused by more virulent, gram-negative bacteria [15]. Early peripheral infections tend to present with overt signs of high-grade infection such as drainage, dehiscence, or an abscess (Fig. 16.1), with or without systemic response [11], and have been associated with higher rates of bleeding from anastomotic disruption due to proteolytic characteristics of the more virulent bacteria [19]. Late, or delayed, infections typically present after the first year. Because they tend to be caused by more indolent organisms, they have a more insidious onset and may be more difficult to diagnose. Patients may lack systemic symptoms such as fever or sepsis, but rather may present with more nonspecific symptoms such as general malaise.

When there is concern for infection, wounds should be examined for surrounding cellulitis, drainage, and tenderness to palpation. These are signs of superficial

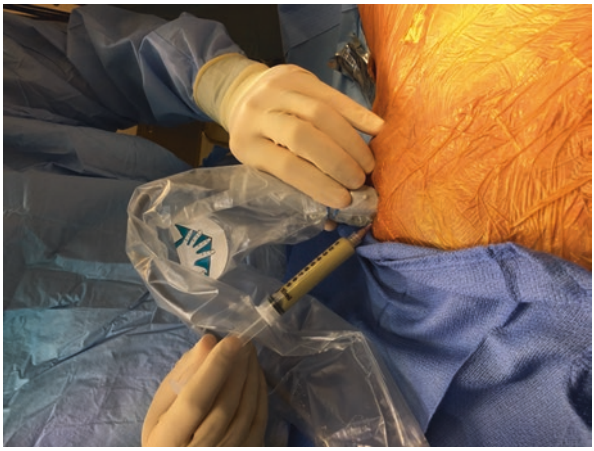

Fig. 16.1 Groin aspiration suggestive of underlying graft infection

wound infections but could also be signs of underlying graft infection. Pain and tenderness over a graft site with a sinus tract and drainage is the most common presentation for graft infection in the groin. Pseudoaneurysms and anastomotic bleeding should be assumed to be due to infection, as up to 60% of involved grafts are found to be culture positive [20]. Anastomotic pseudoaneurysm and blowout are discussed in detail in a subsequent section. Any exposed graft is considered infected.

Aortoiliac graft infections are not as clinically evident as lower extremity graft infections. Symptoms are less focal, and patients may complain of generalized malaise or dull abdominal or back pain. The most severe presentations consist of anastomotic bleeding, GI hemorrhage from aortoenteric erosion or fistula, and even septic emboli to the lower extremities [21]. Stent graft infections most commonly present with pain, fevers, and leukocytosis, with complaints of weight loss, fatigue, and generalized weakness in around 30% of cases [10, 17]. However, over a quarter have been found to present with aortic fistulas and endoleaks and 11% with rupture in a recent multi-institutional study [14]. A small number of aortoiliac graft infections are asymptomatic and found incidentally on routine follow-up imaging in a reported 5–10% of cases [10, 14].

Diagnosis

Diagnosis of prosthetic graft infection requires at least two of the following: (1) positive microbiological culture, (2) clinical or intraoperative signs of infection, (3) or radiologic evidence of graft infection with exclusion of other likely sources of infection [15].

Imaging can help establish the diagnosis and determine the extent of graft infection and involvement. Ultrasound is particularly useful in the extremity and often suggests infection with findings of perigraft fluid and pseudoaneurysms. Computed tomography (CT) is the most common initial study to evaluate for graft infection

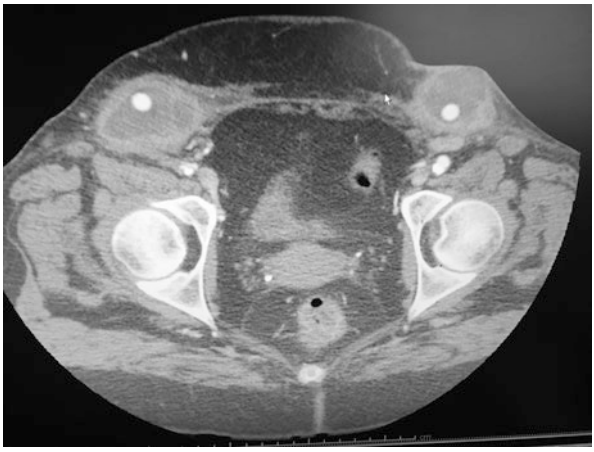

Fig. 16.2 CT scan showing infected femoral-femoral prosthetic bypass

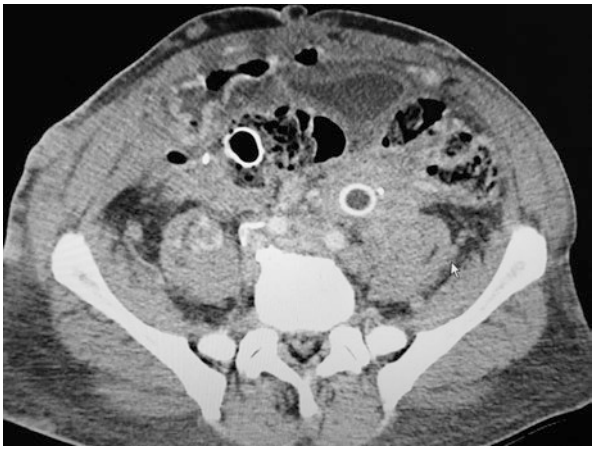

Fig. 16.3 CT scan showing graft limb within the colon lumen and surrounding inflammatory changes

with a reported sensitivity of 67–92% in published studies [7, 17]. Signs of graft infection on CT include perigraft fluid, gas, surrounding soft tissue stranding or inflammation, and pseudoaneurysm development (Figs. 16.2 and 16.3). However, one must keep in mind that perigraft air may persist for up to 2 months and fluid up to 3 months after surgery and thus should not be considered pathognomonic within this time frame [20]. Nuclear medicine studies can suggest the presence of infection, especially when there is clinical uncertainty. Leukocyte scintigraphy, or tagged white blood cell scan (TWBCS), uses a radioisotope to detect leukocytes involved in infection or inflammation and can identify 90% of graft infections. With a lower specificity of 82% there is some risk of false negatives, but fewer than CT scan alone [7]. 18-fluorine-fluorodeoxyglucose positron emission tomography (18F-FDG PET) uses a radioactive glucose isotope to detect the high glucose utilization of activated leukocytes in areas of infection and inflammation. Meta-analyses show focal uptake of glucose to have a sensitivity and specificity up to 97% and 89% [6,

7]. One concern with both leukocyte scintigraphy and PET is detection of inflammation which may or may not be associated with infection. Thus, performing these studies within the first 2–3 months after surgery may lead to false positives due to normal postoperative inflammation or uninvolved infections in the vicinity of the graft. Combining PET and TWBCS with CT, however, reportedly increases diagnostic accuracy by enabling differentiation between graft and soft tissue infection, making PET/CT and WBC SPECT/CT more favorable than CT imaging alone [6, 7]. The disadvantage is limited accessibility to these types of imaging in many institutions. MRI findings of graft infection include a high intensity signal surrounding the graft in T2-weighted imaging and have been described as being more accurate in detecting small fluid collections from staphylococcal epidermidis infections [20]. Angiography is not typically useful unless delineating unclear anatomy. When acute GI bleeding is involved or there is suspicion for aortoenteric fistula, endoscopy is recommended.

The confirmatory diagnostic study for graft infection is a direct culture from excised graft material, perigraft fluid, or biopsy of surrounding deep tissue. Superficial wound swabs are discouraged due to high risk of skin flora contamination. Intraoperative cultures during exploration and graft assessment are imperative, especially in cases of vague presentations or negative imaging studies with high clinical suspicion. Intraoperative findings of graft infection include the presence of perigraft fluid, gross purulence, and lack of graft incorporation. It is important to note that despite these findings, cultures can still be negative in the case of biofilm producing organisms such as *S. epidermidis*, which requires culture in chocolate agar media to reliably identify.

Laboratory values such as leukocytosis and increased inflammatory markers can raise suspicion for graft infection, but are not diagnostic and may be normal in occult cases. CRP, ESR, and procalcitonin are all studies used as adjunctive evidence of infection, although they are notoriously nonspecific. Blood cultures are commonly negative; however, when positive, they should prompt evaluation for cardiac valve vegetation.

Treatment

The primary principle for surgical infection treatment is source control and culture-based antibiotic therapy. With the exception of superficial skin infections, antibiotic therapy alone is largely inadequate. Deep wound infections and large lymphoceles at risk of infection require exploration, with cultures obtained from surrounding tissue, fluid, or graft material itself, followed by extensive lavage and debridement. For prosthetic graft infection, the gold standard treatment is complete graft or endograft excision. When graft removal is determined to be likely to result in limb or life-threatening ischemia, then reconstruction must be performed either by extra-anatomic bypass followed by graft excision or alternatively in situ reconstruction. In appropriately selected peripheral cases, wound exploration with graft preservation and wound sterilization is feasible. Overall, the approach to the management of

infected prosthetic grafts is largely patient specific and depends on the location, extent, and timing of infection, as well as the type of graft and organism involved.

When there is extensive contamination of the graft with gross purulence and revascularization is required, extra-anatomic bypass to avoid reconstruction in an infected field is generally recommended. In peripheral bypasses, commonly described routes include lateral bypass of the groin and obturator bypass. Aortoiliac routes include most commonly the axillobifemoral bypass to the common femoral artery. In the case of groin infection, alternative distal anastomosis sites such as the profunda femoris or superficial femoral arteries via a lateral approach are considered. Obturator canal aorta to femoral artery bypasses serve as an alternative extra-anatomic bypass with improved patency compared to axillofemoral bypass [22]. If immediate revascularization is required, reconstruction can be performed simultaneously or staged with bypass construction followed by excision of the infected graft in stable patients. This occurs ideally 2–3 days later but depends on the patient's condition and the urgency of graft removal [23]. In cases of hemorrhage or gastroenteric communication, simultaneous reconstruction is necessary. It is imperative that the wound bed is aggressively debrided and, for aortic stumps, that the closure be doubly oversewn and covered with an omental flap to prevent subsequent infection and rupture. Overall, extra-anatomic bypasses, particularly in aortic infections, have been shown to have significant morbidity with rates of aortic stump rupture up to 20%, reinfection up to 15%, and poor primary patency rates as low as 64% at 5 years [9, 17, 21, 23, 24]. For this reason, in situ reconstruction has become increasingly utilized and preferred when possible.

In situ replacement of infected grafts has superior outcomes with patency rates as high as 97% at 5 years and lower rates of limb loss and reinfection depending on the chosen conduit [8]. This technique is mostly recommended in complex reconstruction cases associated with minimal contamination and lower virulence organisms. If pursued, the graft must be totally excised, including anastomotic sites and the wound bed aggressively debrided. Partial graft excision can be considered when the infection is confined to a focal segment not involving the anastomoses, leaving uninvolved graft left behind; however, outcomes tend to be inferior [25]. The optimal conduit for in situ graft replacement is debated. Options include autologous vein, cryopreserved allografts, and antibiotic bonded prosthetic grafts. In peripheral reconstruction, including hemodialysis access, bioprosthetic conduits have excellent patency with low reinfection risk [26].

Autologous saphenous, femoropopliteal, and less commonly iliac veins are used for reconstruction. Vein conduits have the lowest rate of reinfection (1–2%) and late mortality (30–50%). They also have the highest primary patency, up to 91% in aortic reconstructions [8, 20]. This makes them ideal in stable patients with excessive gross contamination and more virulent organisms [21]. Use of femoral vein (Fig. 16.4) for NeoaortoIliac System (NAIS) reconstruction has been described by some as the standard of care for aortic graft infections and is the recommended reconstruction in stable patients [20, 27]. Venous reconstructions do however have disadvantages. Harvesting lengthens operative time, limiting use to stable patients. Rates of reintervention are high with reported rupture risk of 5%, and femoral vein

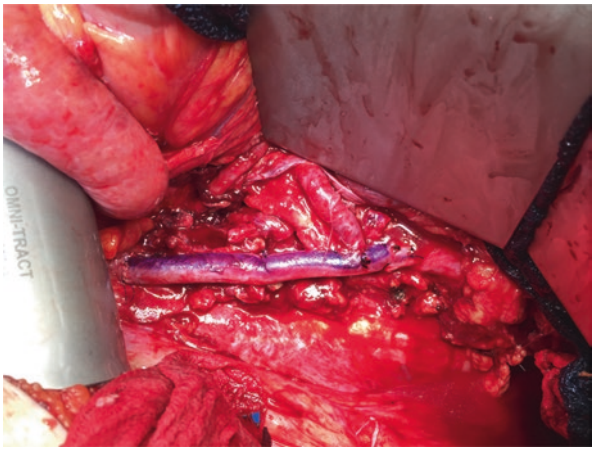

Fig. 16.4 Femoral vein conduit for infected aorta-femoral bypass reconstruction

harvesting includes risk of postoperative fasciotomy (12%) and chronic venous insufficiency (15%) [20, 21, 24].

Cryopreserved allografts have emerged as a frequently used option, with positive outcomes close to those using autologous vein grafts, specifically low reinfection (3–4%), similar rupture and mortality rates, and high primary patency of 93–97% at 5 years [8, 9, 25]. They are recommended in cases of minimal contamination and have the benefit of avoiding the morbidity of vein harvesting and decreasing operative time, each of which is advantageous in unstable patients. Disadvantages, however, include high rates of degeneration, cost, and limited availability [9, 25].

Antibiotic-bonded grafts have been studied with mixed results and most have been performed in animals. When studied in vivo, patency rates of rifampin bonded Dacron grafts rival both autologous and cryopreserved grafts with primary patency of 93% at 5 years and late mortality of 40–50% [21]. Reinfection rates have been reported to be as high as 4–11.5%, associated mostly with highly virulent and antibiotic-resistant organisms [8, 18, 21]. Reinfection risk is decreased when combined with tissue coverage of the graft [17]. Antibiotic-impregnated grafts have been described most successfully in elective cases with minimal to no gross contamination, and low virulence organisms [18, 21], especially in localized peripheral infections. Silver-coated grafts have also been described but reports are limited. While primary patency rates were 93% at 32 months with the benefit of not contributing to increasing antibiotic resistance, these grafts were associated with the highest reinfection rate up to 15.7% [8, 9]. Current guidelines only recommend the use of antibiotic or silver impregnated grafts in unstable patients needing immediate reconstruction [27].

Irrespective of conduit type, meta-analyses have shown overall graft failure and morbidity to be lower for in-situ graft replacement than extra-anatomic bypass [8]. The types of reconstruction for aortoiliac graft and endograft infections have not been shown to affect mortality [17]. Mortality is higher if grafts are not removed and treated non-operatively rather than surgically, with low survival rate of 33% versus 58%, respectively [16].

After graft removal, there is no standard recommendation for the duration of antibiotic treatment. Most studies describe continuation of tailored antibiotics for 4–6 weeks postoperatively. Some cases will require indefinite antibiotic suppression, especially with retained prosthetic material or in an immunosuppressed patient [14, 17, 24]. Antimycotic agents should be considered for patients with aortoenteric associated infection [9].

Finally, graft preservation with wound sterilization has been reported with success in appropriately selected cases. Graft preservation is most suitable for patent femoral or distal grafts with infection limited to the bed of the graft and not involving suture lines, in patients not presenting with sepsis or hemorrhage. Graft preservation should not be considered in the presence of high virulence organisms such as *Pseudomonas* [5, 11, 28]. Wound sterilization begins with thorough operative debridement and copious irrigation, with or without adjuncts such as betadine, hydrogen peroxide, or bacitracin. Sterilization with povidone-iodine-soaked dressing changes over exposed grafts was the original method described with successful graft preservation around 71% [28]. More recently, sterilization using antibiotic beads to deliver highly concentrated doses to a local wound has been reported. The technique consists of creating antibiotic beads intraoperatively by mixing polymethyl methacrylate powder with vancomycin and either tobramycin or gentamicin, rolling the mixture into small beads, linking them on a suture while solidifying, and once the thermal reaction has cooled they are inserted into the wound which is then sutured closed. Serial washouts and antibiotic bead exchanges are continued every 3–5 days until negative cultures are obtained, requiring a range of 1–3 explorations after initial bead placement [4, 11]. At the time of final closure, muscle flap coverage should be considered in the case of significant graft exposure or large soft tissue defects. Postoperative antibiotic course consists of at least 4–6 weeks of parenteral culture-specific antibiotics. Wound sterilization rates of 87–94% with reinfection rates of 11–12.5% are reported [4, 29]. Higher reinfection rates up to 20% were seen using therapy guided by the clinical appearance of the wound instead of culture results [4]. An association with late pseudoaneurysm formation seen in 4–5% of graft preservation patients highlights the need for long-term surveillance [4, 11].

Prevention

The ultimate treatment for infection is prevention. Giving prophylactic antibiotics prior to vascular arterial reconstruction reduces risk of wound infection by three quarters and early graft infection by two thirds [30]. Thus, prophylaxis against common gram-positive and gram-negative skin contaminants with first- or second-generation cephalosporin is recommended. Alternatively, clindamycin or vancomycin can be utilized in the case of a beta-lactam allergy. It is important to redose in the case of lengthy operations. Given that SSIs rates are doubled with operative time greater than 250 minutes, efficiency during procedures is essential [2].

Reference A
Skin preparation with chlorhexidine instead of povidone-iodine has been shown to reduce SSI in vascular surgical procedures. [Ref: Factors associated with surgical site infection after lower extremity bypass in the SVS VQI. Kalish JA, Farber A, Homa K, Trinidad M, Beck A, Davies M et al. J Vasc Surg 2014;60:11238–46}.

Intraoperatively, when handling graft material, one should avoid unnecessary contact with the skin. The use of iodine containing adhesive drapes to act as a microbial barrier has been shown to have no effect on SSI rate when compared to no drapes [12]. Optimizing patient factors such as maintaining blood sugar level below 180 mg/dL and avoiding hypothermia have shown association with lower SSI rates [2, 18]. Simultaneous gastrointestinal operations with aortic grafting should be avoided.

At closure, aortic grafts should be covered by reapproximating the posterior peritoneum, and if this is not possible, an omental flap is recommended. Groins should be closed in layers with soft tissue coverage to protect grafts from contact with skin. Placing vancomycin powder into groin wounds at the time of closure showed a small but statistically significant decrease of 7.9% in superficial infections alone, within the first 30 days [31]. Closed incision negative pressure therapy for the first 5–7 days after surgery has been suggested as an effective strategy to maintain approximation of skin edges, protect the wound from bacteria, and remove proinflammatory fluid and edema. Studies have reported reduced wound infections from 25–30% to 6–8.5% in high-risk femoral incisions [3, 32].

Postoperatively, antibiotics are not indicated for prevention. However, after a patient has undergone graft or endograft placement, prophylactic coverage is recommended when undergoing certain procedures such as dental work, colonoscopy, or cystoscopy [27].

Conclusion

Surgical site and graft infections are a frequent and dreaded complication of both open and increasingly endovascular vascular surgery. A diverse microbiological pathophysiology drives the clinical presentation and treatment algorithms. An understanding of diagnosis, microbiology, treatment, and prevention is paramount to performing safe and high-quality vascular surgery.

Late Anastomotic Complication: Pseudoaneurysm, Anastomotic Aneurysm, and Blowout

Late anastomotic failure presents in a variety of ways. The first form of anastomotic failure would be the formation of a true anastomotic aneurysm. This presents late as a dilation of the native artery at the site of the anastomosis and includes all walls of the vessel with containment of blood flow. The next, and more common entity, is the pseudoaneurysm or false aneurysm, with blood flow outside of the artery contained

within a pseudocapsule with continuous flow and arterial pressure within the pseudoaneurysm. Finally, frank rupture of the anastomosis or "blowout" can occur.

Early anastomotic failure typically occurs due to a technical problem or virulent infection. Following the initial postoperative period, the etiology of late failure is more complex. This section will focus on late anastomotic complication.

Pathophysiology

Anastomotic complications can occur due to several underlying pathophysiologic causes. The majority of episodes will involve native artery to prosthetic anastomoses, though these can all occur in relation to autogenous tissue anastomoses in rare circumstances [33]. Failure can occur at multiple sites in patients who undergo arterial reconstruction that have multiple anastomoses. Additionally, while anastomotic aneurysms and blowout can occur at any location, late manifestation is most frequently seen with aortoiliac and aortofemoral reconstruction and for reconstructions performed for occlusive rather than aneurysmal disease.

The anastomosis is initially dependent only on suture material for integrity. However, with time fibrous scarring will contribute to building integrity along with the suture material, and this must also be affected in order to have anastomotic failure [34]. Therefore, these late complications require failure of the suture line, the fibrous scar tissue, and/or prosthetic conduit at the anastomosis. This can occur due to primary suture failure, technical failure, arterial or prosthetic degeneration, or infection.

If arterial or autogenous graft dilation is the underlying cause, a true aneurysm involving all walls of the vessel can develop. Additionally, prosthetic conduit itself can dilate with time. Often this can be attributed to aneurysmal degeneration of the anastomosis proximal or distal to the conduit. This can be seen after aortic aneurysm repair with proximal degeneration involving the juxtarenal aorta or distally involving the iliac arteries when a tube graft was performed. This is also frequently seen at the femoral anastomosis in aortobifemoral reconstructions. Poor control of blood pressure, atherosclerotic disease progression, and excessively deep endarterectomy can attribute to full thickness dilation of the blood vessel creating a true aneurysm. Finally, compliance mismatch between graft and autologous tissue has been implicated as a cause of native artery true aneurysm formation [35].

Occurring more frequently than dilation and true aneurysm formation, a break in the vessel wall or anastomosis may allow blood to exit the lumen into the surrounding tissues to form a pseudoaneurysm. Suture line fracture, conduit, or native artery full thickness tears, infection, or technical errors such as poor graft sizing, excessive tension, or redundancy can contribute to anastomotic failure and manifest late as a pseudoaneurysm (Fig. 16.5). Graft failure or defect can also lead to this complication, though this is very rare. Pseudoaneurysms can erode through the skin causing external hemorrhage (Fig. 16.6) or into adjacent bowel which can contribute to aortoenteric fistula formation [36].

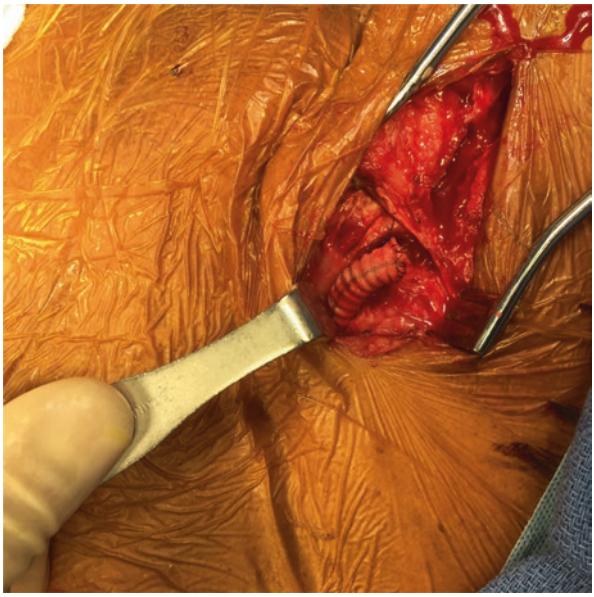

Fig. 16.5 Late graft anastomotic disruption

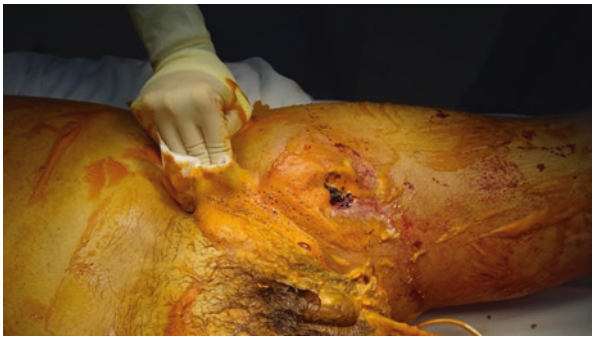

Fig. 16.6 Pseudoaneurysm of femoral artery eroding through the skin

The development of a late anastomotic failure is also associated with systemic disorders. Vasculitides and connective tissue disorders increase the risk of anastomotic failure [37]. Smoking is also a known risk factor for anastomotic failure. Poor control of hypertension can also contribute to this complication. Finally, the need for systemic anticoagulation and/or antiplatelet agents can contribute to persistent flow in pseudoaneurysms.

Diagnosis

Presentation of anastomotic complications varies based on the etiology of the underlying complication. In a series of 142 femoral anastomotic aneurysms, 64% presented with a painless pulsatile mass, 19% presented with acute limb ischemia,

8% presented with a painful mass, and 7% presented with hemorrhage [38]. Early presentation, within the first 6 weeks following intervention, is more likely to include pain, signs of infection, bleeding, and acute ischemia. Late presentations typically appear as painless pulsatile masses.

The most common site of late anastomotic complication is the femoral artery following reconstruction, with 3% of all patients developing aneurysms. The incidence is higher when patients are undergoing aortofemoral reconstruction, with 6–8% of patients developing anastomotic complications following aortobifemoral bypass [39].

A physical examination will often suggest the presence of a femoral aneurysm or pseudoaneurysm. Oftentimes, pseudoaneurysm development will be accompanied by pain, local tissue inflammation, and edema. Aortic anastomotic complication would more likely present as an incidental imaging finding, retroperitoneal rupture, or with development of an aortoenteric fistula [40]. Duplex ultrasonography can be suggestive of anastomotic complication and is particularly helpful when identifying a pseudoaneurysm with classic "to and fro" flow within an extravascular space. Cross-sectional imaging, most commonly CT scan, would definitively diagnose the lesion and allow evaluation of the entire reconstruction for asymptomatic involvement of other sites. Pseudoaneurysms at multiple sites (e.g., after aortobifemoral reconstruction) should raise the question of an underlying infection. If there is concern for infection at the site of anastomotic complication, some advocate for the use of PET CT scanning, though this will often not change the course of management. Additionally, some advocate for routine surveillance CT to assess for asymptomatic anastomotic aneurysms at 5 years post reconstruction. MRA can also be a useful adjunct in these patients when radiation exposure is a concern.

Treatment

Management of anastomotic complications is based on the underlying cause and location of the lesion.

Overt hemorrhage requires immediate operative intervention to control the source of bleeding, regardless of the presence of infection. Late blowout generally involves infection or graft failure. One should plan for massive transfusion and consider both open and endovascular treatment to address the cause of bleeding. Often endovascular exclusion can be used as temporizing measure prior to definitive, planned reconstruction.

All retroperitoneal aortic anastomotic aneurysms and pseudoaneurysms should be treated urgently when diagnosed to avoid rupture and erosion. In the presence of infection, excision and extra-anatomic bypass or in-line repair should be considered. However, when infection is unlikely, endovascular exclusion is becoming increasingly used as a safe and effective definitive treatment. Aortoenteric fistula treatment is discussed in a later section of this chapter.

All femoral anastomotic pseudoaneurysms should be considered for immediate treatment. Due to the high frequency of infection or graft failure as the cause of late

lesions, these generally require open revision. Some advocate for converting all end-to side aortofemoral anastomoses to an end-to-end configuration unless retrograde flow is required to perfuse the ipsilateral hypogastric artery. When infection is not suspected, primary repair is possible by repairing the site of graft or suture line failure. However, if infection is suspected, resection of the graft and extraanatomic bypass reconstruction should be considered. Conversion to an aortopopliteal or iliopopliteal bypass via the obturator canal is an option for infected femoral artery anastomoses [33]. If in-line reconstruction is performed, autologous tissue coverage is imperative. The use of muscle flap coverage has been extensively reported and is a very useful adjunct when local autologous tissue is insufficient for coverage [41].

True femoral anastomotic aneurysms should be fixed when they exceed 2 cm in greatest diameter [42]. Repair can often be performed with simple resection of the aneurysmal segment and interposition reconstruction. Again, conversion to an end-to-end configuration is often required when the aneurysmal segment of the native artery is involved and excised. A growing body of literature has suggested that endovascular exclusion has acceptable results, though long-term data is lacking.

Conclusion

Late anastomotic blowout, pseudoaneurysm and true aneurysm formation are complicated clinical problems. They require a high index of suspicion on symptom and examination findings. Additionally, infection must always be considered as the underlying cause. These unique clinical situations require urgent recognition and repair to prevent disastrous outcomes.

Aortoenteric Fistula

Aortoenteric fistula (AEF) occurs in both primary and secondary forms. Primary AEF is a rare entity which is associated with aneurysmal degeneration of the aorta. This chapter will focus on secondary AEF as a late complication of open and endovascular aortic intervention. This complication is uncommon, but carries a high mortality rate and requires prompt identification and treatment.

Incidence

Secondary AEF (SAEF) has a relatively low incidence. In a series of 307 patients by Hallett et al., 1.6% of patients developed a secondary AEF following aortic intervention [43]. In a selective review of patients presenting with SAEF, Pipinos et al. demonstrated 98% of patients had undergone reconstruction with prosthetic graft material [44]. Of these patients, they were nearly equally weighted between abdominal aortic aneurysm repair and aortobifemoral bypass performed for aortoiliac occlusive disease. These findings counter previous reports of increased occurrence

of SAEF following AAA repair. A proximal end-to-side configuration has been associated with increased risk of SAEF formation. Of note, there is minimal evidence to support increased occurrence of SAEF with a retroperitoneal exposure. These fistulas occur either at the anastomosis (graft enteric fistula) or along the graft material (graft enteric erosion). Additionally, aortoenteric fistula formation has been demonstrated following endovascular exclusion of AAAs [45]; however it remains rare, with an incidence of 0.01% following EVAR in the MAEFISTO study reviewing 3932 patients [46].

The most common presentation of secondary AEF is gastrointestinal hemorrhage. Classically, a small volume herald bleed will occur, though massive hemorrhage can certainly occur without herald bleeding. SAEFs can also present with chronic findings of weight loss, malaise, fevers, sepsis, and graft thrombosis. It is important to appreciate that many patients with SAEF will not present with clinical bleeding. The most frequent presentations of SAEF include one or more of the following clinical signs and symptoms: gastrointestinal bleeding (80%), sepsis (44%), abdominal pain (30%), back pain (15%), groin mass (12%), and abdominal pulsatile mass (6%) [47].

Even with treatment, morbidity and mortality with secondary AEF remains very high. Untreated SAEF are uniformly fatal and postoperative mortality of those who undergo all forms of repair still approaches 50%.

Pathophysiology

Aortoenteric fistula formation most frequently occurs between the duodenum and the proximal aortic graft [48] due to the close proximity of the bowel to the anastomosis on infrarenal aortic reconstructions (Fig. 16.7). The duodenum is a retroperitoneal structure at this location and minimal intervening tissue is present between the bowel and the graft. The most common site of secondary AEF formation is also

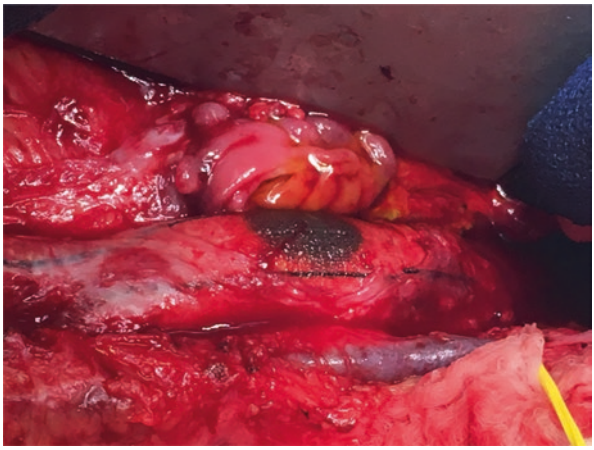

Fig. 16.7 Aortic graft erosion into duodenum

the duodenum (62%) followed by the jejunum and ileum (12%) and then colon (5%). Fistulous tracts have been reported to occur at any level of the graft, with approximately 4–6% occurring away from the anastomosis [49]. Careful approximation of tissue between the structures, use of an omental pedicle flap, and a retroperitoneal approach to the aorta for reconstruction are thought to decrease the risk of SAEF.

Secondary AEF occur due to several possible underlying mechanisms [50]. Infection of the aortic graft can contribute to local inflammation and tissue destruction. Virulent bacteria, especially *S. aureus*, are most often attributed to formation in animal models [51]. However, two thirds of operative cultures are generally polymicrobial, owing to the contamination with enteric contents. An additional component seen is the high frequency of *Candida* species growing in operative cultures [52]. This organism is thought to contribute to development of the fistulous connection. This is often difficult to distinguish however, as the infection may have been the underlying cause or the result of graft exposure to the enteric contents. The pulsatile nature of the aortic graft also is thought to contribute to graft erosion and fistula formation. This pulsatility produces constant friction as well as pressure and potential ischemia to the bowel tissue. Technical errors during the procedure may also contribute to SAEF development. Bowel injury, graft contamination, and inadequate separation of graft and bowel can all potentially occur during the operation.

SAEF following EVAR occur due to unique problems associated with endovascular aortic exclusion. One mechanism is sac enlargement due to persistent endoleak and direct erosion of the aneurysmal aorta into bowel. Infection can contribute to development of SAEF following EVAR as well as graft endotension and migration. Importantly, more than 30% of EVAR-related AEF were related to a defect in the aortic stents themselves such as fracture, erosion, or angulation of the stent [53].

Diagnosis

Diagnosis of SAEF is driven primarily by the presentation of the patient. Unfortunately, a high index of suspicion must be maintained in order to promptly make the correct diagnosis, and AEF should thus always be a consideration in patients with previous aortic intervention and GI bleeding. When a patient presents with hemodynamic instability and massive gastrointestinal hemorrhage, AEF will most often be diagnosed at the time of exploratory laparotomy. A subset of these patients will be diagnosed by aortography or endoscopy. If the patient is stable, they often first undergo endoscopy. This is insensitive for the diagnosis of AEF, with a sensitivity of only 50% [54]. Ideally, these patients should undergo CT angiography for full evaluation of the aortic graft and surrounding tissues. This imaging is also useful in operative planning for treatment of the condition. The sensitivity of CTA for diagnosis is still not ideal, with only 61% of patients being diagnosed correctly [55]. Findings associated with AEF include loss of fat planes around the aorta,

perigraft fluid and gas, tethering of adjacent bowel loops to the graft, and extravasation of contrast from the aorta into the involved segment of bowel. Angiography has not been found to be useful in stable patients, as active bleeding is rarely seen at the time of the procedure.

In patients who present with constitutional symptoms without gastrointestinal bleeding, additional tools often aid in diagnosis. CT angiography will often have the aforementioned findings around the aortic graft. PET scanning and tagged WBC scanning can be used as an adjunctive measure to diagnose AEF when aortic graft infection is present [56]. Finally, if communication exists between the colon and aortic graft, colonoscopy can be diagnostic.

Treatment

Treatment for secondary AEF should be catered to the presentation of the patient. Patients with massive hemorrhage, instability, or ongoing severe sepsis require specialized, emergent therapy to try to stave off an extremely high mortality condition. A stable, non-toxic patient presenting with a classic herald bleed has time for preoperative planning, diagnostics, and resuscitation prior to treatment. However, even they should be treated in an urgent manner, that is, during the index hospitalization.

Hemodynamically unstable patients must be urgently resuscitated, transfused, started on broad spectrum antibiotics, and brought to the operating room for emergent exploration. The preferred approach is midline laparotomy, and the first step is rapid proximal aortic clamping for control. Balloon control is also a reasonable option for obtaining proximal control given the potentially hostile nature of a reoperative abdomen. Distal control should similarly be obtained with iliac clamping or balloon occlusion. Some have advocated for the use of Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) for temporary control by inflating a compliant balloon to bridge the aortic defect, while the aorta is exposed and controlled [57]. The affected bowel should be controlled with bowel clamps, the fistula excised, and bowel closed primarily or resected with primary anastomosis. The aortic stump should be oversewn and covered with omentum, and the retroperitoneum should be drained widely. Traditionally, aortic reconstruction necessitates complete graft excision and extraanatomic bypass, often with axillobifemoral bypass. However, immediate in situ reconstruction can also be performed with Cryoartery, antibiotic-soaked Dacron, or creation of a Neo-Aortoiliac system with autogenous femoral vein (Fig. 16.8). There is not a superior conduit, and much of the conduit choice reflects individual surgeon and institutional practices [58]. Specific descriptions of these procedures are beyond the scope of this chapter.

Some have advocated for endograft exclusion of the AEF, typically used as a temporizing step or as a palliative option for patients would not physiologically tolerate excision and reconstruction [59]. However, long-term data is needed to fully evaluate the viability of endograft exclusion of SAEF as a destination therapy.

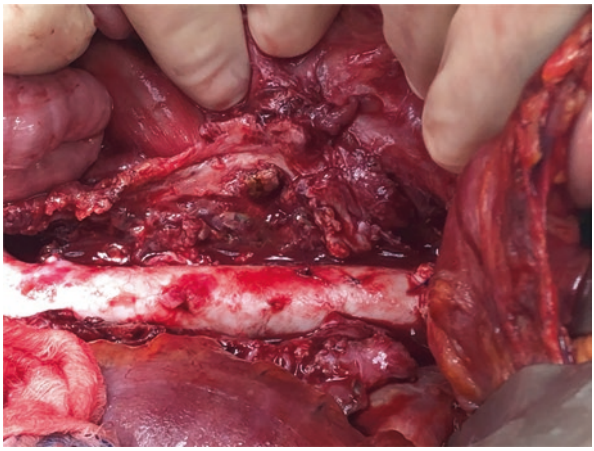

Fig. 16.8 Aortoenteric fistula repair with cadaveric aorta

Conclusions

Secondary AEF is a dreaded complication of open and endovascular aortic aneurysm repair as well as reconstructions for aortoiliac occlusive disease. Complex pathophysiologic mechanisms appear to lead to the development of these fistulas. While this diagnosis portends high morbidity and mortality, prompt diagnosis and treatment can improve outcomes.

Lymphocele/Lymphatic Fistula/Chylous Ascites

Lymphatic complications following vascular surgery are complex and irksome complications. Their presentation varies from early postoperative developments to late clinical findings. A spectrum of outcomes can be expected. Lymphoceles can be incidentally found and behave in benign fashion or may present as massive lymphatic leaks with infection which require operative intervention and can lead to significant morbidity and mortality. An understanding of the clinical findings and treatments are imperative for vascular surgeons.

Etiology

Damage to the lymphatic channels is a known complication of open surgical procedures, leading to lymphorrhea. If the lymph fluid is contained within the surrounding tissues, it is known as a lymphocele. If there is external communication, it is termed a lymphocutaneous fistula. This complication most frequently occurs during kidney transplantation, lymphadenectomies, and pelvic oncologic resections. For

vascular surgery, the most common site of lymphatic injury is the groin during open reconstruction of the femoral vessels, and in the retroperitoneum following aortoiliac reconstruction [60, 61] because of the dense concentration of lymphatic tissue within the femoral triangle and the retroperitoneum. Risks for lymphorrhea include failure to ligate lymphatic channels, reoperative surgical fields, infection, and placement prosthetic graft material [62]. These complications potentially delay wound healing, increase risk of infection, and increase fluid losses contributing to dehydration and increased length of stay. Additionally, the high triglyceride concentration within chyle can lead to nutritional deficiency, especially with high volume cutaneous loss or ascites. The change from lymph to chyle occurs in the retroperitoneal and intraabdominal lymphatic tissue when emulsified fats are added to the lymph fluid by the small intestine.

Incidence

The incidence of lymphoceles after groin arterial reconstruction is relatively common, with a large series demonstrating 4% [63]. In order for a lymphocele to develop, a persistent communication with a lymphatic channel is required. Lymphoceles develop pseudocapsules which contain the lymph fluid within a discrete space. The accumulation typically occurs in the first postoperative month, though lymphocele and lymphatic fistula can occur at any time following intervention [64]. Lymphocutaneous fistula is diagnosed when there is continuous drainage of clear to straw colored fluid from the incision site. These have occurred in fewer patients compared to contained lymphocele. Kalman et al. demonstrated a frequency of only 0.1% in a surgical series of 4000 patients undergoing femoral artery reconstruction [62]. Fistulas require more aggressive management when compared to lymphoceles due to the increased fluid losses, wound complications, and infection risk. Lymphatic complications can also occur with open aortoiliac surgery, demonstrated by both development of retroperitoneal lymphocele or chylous ascites [61]. This complication is noted to be rare and only sparingly reported in literature.

Diagnosis

Lymphocele is typically diagnosed with ultrasonography. If lymphocele develops further than 1 month following the operation, contrast-enhanced CT is helpful to distinguish the collection from pseudoaneurysm or abscess. Lymphocutaneous fistula is typically diagnosed with the aforementioned findings, though CT scan and ultrasound are useful adjuncts to rule out concomitant retroperitoneal involvement. The gold standard to diagnose a lymphocele, lymphatic fistula, or chylous ascites is lymphoscintigraphy [65]. This technique is also useful to distinguish contained collections from simple seromas during the early postoperative period.

Treatment

Treatment of lymphoceles should be considered in the presence of compressive symptoms, clinical signs of infection, or increase in size. Small lymphoceles are more appropriately observed as they often resolve spontaneously. When a lymphocele develops in proximity to prosthetic graft reconstruction, many advocate for prophylactic intervention to prevent graft infection. Some symptomatic lymphoceles will resolve with percutaneous drainage, often augmented by the use of sclerosing agents. Ethanol, povidone-iodine, tetracycline, doxycycline, bleomycin, talc, and fibrin glue have been used as sclerosing agents. There is a reported 50% recurrence rate for retroperitoneal lymphocele with drainage alone [66]. Wounds with refractory lymphorrhea should be explored with direct ligation of the lymphatic pedicle and resection of the pseudocapsule. Lymphocutaneous fistulas typically require intervention to aid in wound healing and avoid superinfection. However, conservative management with bed rest, local wound care, and empiric antibiotics has been advocated by some [67]. Increasing utilization of negative pressure wound vacuum closure has also been used to treat fistulae with excellent results, including a series by Haman et al. describing 100% closure of the fistulae [68]. Wound exploration with ligation of the damaged lymphatic channel is again advocated for lesions failing conservative therapy. Chylous ascites often requires treatment due to severe symptoms and nutritional losses and is mostly driven by dietary modifications. The use of total parenteral nutrition or restriction to a medium chain triglyceride only diet leads to decreased chyle production and resolution of chyle accumulation [69]. Percutaneous and operative interventions have been performed rarely for refractory cases of chylous ascites. In a series of patients who developed chylous ascites following open aortic surgery by Pabst et al., patients requiring operative management had high surgical success of lymphatic channel ligation; however the team recommends utilizing this approach only as a last resort [70]. In their series, chyle leak associated mortality following aortic intervention was reported at 11.5% regardless of treatment approach.

Conclusion

Lymphatic complications are diverse and complicated. These complications can present early in the postoperative course, but late presentation or superimposed infection can often lead to serious adverse patient morbidity. An understanding of the pathophysiology and clinical presentations drives prevention and treatment.

Conclusion

Dreaded late complications in vascular surgery encompass a broad clinical presentation and pathophysiology. Infection, anastomotic complications, aortoenteric fistula, and lymphatic complications can create devastating clinical morbidity and

mortality. Vascular surgeons should be aware of modern diagnostic modalities and treatments to address each of these late complications.

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Skin preparation with chlorhexidine instead of povidone-iodine has been shown to reduce SSI in vascular surgical procedures. [Ref: Factors associated with surgical site infection after lower extremity bypass in the SVS VQI. Kalish JA, Farber A, Homa K, Trinidad M, Beck A, Davies M et al. J Vasc Surg 2014;60:1 1238–46].