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Vascular surgical site infection: risk factors and preventive measures

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ABSTRACT

Surgical site infection (SSI) after arterial intervention is a common nosocomial vascular complication and an important cause of postoperative morbidity. Its prevention requires the vascular surgeon and the health care team to be cognizant of its epidemiology and patient-specific risk factors to apply effective measures to reduce the incidence. The majority of vascular SSIs are caused by Gram-positive bacteria with methicillin-resistant *Staphylococcus aureus* (MRSA) now a prevalent pathogen that is involved in more than one-third of cases. Nasal carriage of methicillin-sensitive *S. aureus* or MRSA strains, recent hospitalization, a failed arterial reconstruction, and the presence of a groin incision are major risk factors for developing a vascular SSI. Overall, the SSI rate after arterial intervention is higher than predicted by the Centers for Disease Control and Prevention's National Nosocomial Infections Surveillance Risk Category System, and ranges from 1% to 2% after open or endovascular aortic interventions, to as high as 10% to 20% after lower-limb bypass grafting procedures. Application of perioperative measures to reduce *S. aureus* nasal and skin colonization in conjunction with appropriate, bactericidal antibiotic prophylaxis, meticulous wound closure, and postoperative care to optimize patient host defense regulation mechanisms (eg, temperature, oxygenation, and blood sugar) can minimize SSI occurrence.

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1. Introduction

Vascular surgical site infection (SSI) occurs as a result of perioperative events leading to bacterial colonization of the wound and frequently the underlying prosthetic graft, when present. Patients undergoing arterial interventions are at increased risk for SSIs, with an overall incidence in the range of 5% to 10%, significantly higher than the 2% to 6% rate predicted by the Centers for Disease Control and Prevention's National Nosocomial Infections Surveillance System for "clean" procedures in risk index categories 1 and 2 [1,2]. A recent meta-analysis of 38 published articles of the outcomes after femoropopliteal bypass found a mean SSI rate of 7.8% [3]. The application of five Surgical Care Improvement Project guidelines, recommended in 2006, to prevent SSIs has not significantly reduced patient and hospital SSI rates when adjusted for procedure type [4]. At present, a preoperative patient-screening program for nasal *Staphylococcus* colonization coupled with subsequent skin de-colonization and perioperative antibiotic therapy are effective in reducing SSIs after vascular, orthopedic, and neurosurgery procedures [5–10].

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The increased likelihood of SSIs in the vascular surgery patient is due to both procedure- and patient-specific risk factors [11–16]. Implantation of a vascular prosthesis increases the SSI risk by producing a microenvironment conducive to bacterial attachment and biofilm formation that sustains bacterial colonization and protects encased organisms from host defenses and antimicrobial therapy. Injured skin and skin structures and soft-tissue edema, common sequelae after femoral and lower-limb arterial revascularizations, impede wound healing. Imprecise skin suture closure, dermal necrosis, underlying hematoma, and persistent serous wound drainage extend the time available for bacterial invasion from external sources, bacteremia, or via bacteria transport to the wound through lymphatic channels. The nonhealing surgical wound enables bacterial invasion, which, if not addressed promptly, can progress to an SSI, with its attendant increased morbidity and health care costs from an extended length of hospitalization, return to the operating room for wound debridement and closure procedures, home health care, and outpatient visits for wound management. Therefore, SSI prevention is a paramount concern for vascular surgeons, and reducing its incidence requires knowledge of its risk factors for development and use of effective patient-care strategies.

Infection involving a vascular wound can be superficial (cellulitis), deep incisional (involving the subcutaneous tissue/fascia), or involving other areas than the incision itself (organ/space), such as along the length of an implanted vascular prosthesis or as an intracavitary aortic graft infection. Although multiple grading systems for vascular SSIs exist (eg, Samson, Koenig and von Dongen, Bunt, and Karl-Stork), the most common grading system is that proposed by Szilagyi et al [9] in 1972, and it remains in use in the literature because of its ease of use. For autogenous arterial revascularizations, infections occurring within 30 days are classified as SSIs, but when a prosthetic graft or endovascular device is implanted, the incidence of SSI is calculated for 1 year, not only 30 days after the procedure. The diagnostic criteria for SSI should include signs and symptoms of infection (eg, pain, tenderness, erythema, and swelling), purulent drainage from the wound, and organisms isolated from aseptically obtained cultures of fluid or tissue (Table 1).

2. Changing epidemiology of vascular SSI

Although virtually any microorganism can produce an SSI or infect a vascular prosthesis, Gram-positive bacteria, especially *Staphylococcus aureus*, are the prevalent pathogens involved in approximately 80% of all cases [7–10]. As in other surgical disciplines, the microbiology of vascular SSIs has changed in the past decade, with an increased prevalence of antibiotic-resistant organisms, including staphylococcal strains. An audit of prosthetic arterial graft infections treated by our vascular surgery group demonstrated a four-fold increase in methicillin-resistant *S. aureus* (MRSA) infection from 10% in the 1990s to 40% since 2000. In a series of complex vascular SSIs treated by aggressive staged surgical debridement, antibiotic bead therapy, and selective sartorius muscle flap coverage, MRSA accounted for 20% of all infections and 50% of re-infections [7]. This trend has been verified from other vascular centers in the United States and Europe. A 2005 report from the University of Texas Galveston vascular group reported an SSI rate of 11% after lower-extremity bypass grafting, with *S. aureus* involved in 64% of cases, of which one-half were caused by MRSA [11]. In 2004, Taylor and Napolitano [12] reported an institutional incidence of MRSA SSI of 22%. They also noted an increased risk of amputation with MRSA, and an increased length of hospital stay (29.6 *v* 22.7 days) as compared with non-MRSA SSI. Today, MRSA should be suspected in any vascular patient presenting with an SSI, including patients with a nonhealing lower-limb amputation performed for ischemia. The evolution in the microbiology of SSIs, especially the increase in MRSA infection, has implications for both the initial treatment of SSIs and the antibiotic prophylaxis in vascular patients with

Table 1 – Centers for Disease Control and Prevention's criteria for superficial and deep incisional surgical site infection.

SSI Type	Criteria
Superficial	Purulent drainage from the surgical site Organisms identified from an aseptically obtained specimen from the incision or subcutaneous tissue by a culture- or nonculture-based microbiologic testing method performed for purposes of clinical diagnosis or treatment. Superficial incision that is deliberately opened by a surgeon or other designee and culture- or nonculture-based testing is not performed. AND the patient has at least one of the following signs or symptoms: pain or tenderness, localized swelling, erythema, or heat. A culture- or nonculture-based test that has a negative finding does not meet this criterion. Diagnosis of a superficial incisional SSI by the surgeon, attending physician or other designee in the medical record.
Deep incisional	Involves deep soft tissues of the incision (eg, fascial and muscle layers). AND the patient has at least one of the following: purulent drainage from the deep incision; a deep incision that spontaneously dehisces, or is deliberately opened or aspirated by a surgeon, or other designee and organism is identified by a culture-or nonculture-based microbiologic testing method which is performed for purposes of clinical diagnosis or treatment. AND the patient has at least one of the following signs or symptoms: fever (>38°C), localized pain or tenderness. A culture- or nonculture based test that has a negative finding does not meet this criterion. An abscess or other evidence of infection involving the deep incision that is detected on gross anatomical or histopathologic exam, or imaging test

SSI = surgical site infection.

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