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Surgical Site Infections in Orthopaedics: Epidemiology & Microbiology

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Abstract

Surgical site infections are one of the most common nosocomial infections. understanding epidemiology and microbiology of SSI will help in defining the problem and developing stratergies for prevention and management

Keywords: Surgical Site infection, Diagnosis,

Introduction

Surgical site infections (SSIs) are dreaded and devastating complications after any orthopedic procedure. They may necessitate removal of the prosthesis /implant resulting in loss of joint/ limb function, and even loss of limb or life. Consequently, patients with SSIs frequently need readmission, further surgical procedures and parenteral antibiotics for an extended duration. Functional outcomes after SSI are frequently suboptimal. Post-operative infection leads to a constrained relationship between the patient and the surgeon. It also increases the economic burden on the patient and the healthcare infrastructure[1]. Infact, studies have shown that SSI can extend the patient's hospitalization time by up to two weeks, double re-hospitalization rates, increase overall costs by more than 300%, besides causing important physical limitations that significantly reduce patients' quality of life after the surgery[2]. Periprosthetic joint infection (PJI) has a worse survivorship than cancer.

SSI's are one of the most common nosocomial infections besides pneumonia, urinary tract infections, and bloodstream infections. The incidence of SSI's following various orthopedic procedures ranges from 0.8 to 71%.[3-7] The risk of postoperative prosthetic joint infection following hip or knee arthroplasty is 0.5–2 %. For prosthetic shoulders and elbows, the risk is considerably higher at approximately 3-4 %. After the first 2 years, the risk of a hematogenous infection is 2.3 infections per 1000 prosthetic joint years[8,9]. PJI incidence has not changed in the last 4 decades.

The pathogenesis of SSI is multifactorial. Patients with poor immunity commonly get a SSI. The degree of bacterial colonization of the skin surface, the antisepsis methods used preoperatively, antibiotic prophylaxis, surgical technique, the healthcare environment, preoperative patient optimization, perioperative protocols, sterilization techniques, the operation theatre environment and preparation protocols – all have a role to play in the development of an SSI. The use of implants (foreign bodies)has frequently been implicated as the cause of infection in orthopedics[10]. In the presence of implants, even a small number of bacteria are sufficient to cause an infection. Besides, microorganisms such as staphylococci and gram negative bacilli frequently form a biofilm over the implant, dead bone and even tissue, which makes them difficult to eradicate.

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Definition

A surgical site infection is an infection that occurs after surgery in the part of the body where the surgery was done[11]. Surgical site infection (SSI) is defined as microbial contamination of the surgical wound within 30 days of an operation or within 1 year after surgery, if an implant is placed in a patient[12]. Based on the time of onset following the index surgery, surgical site infections can be classified as:[13,14]

a. Early infection (0–3 months)

Early infections are usually triggered by virulent pathogens such as S. aureus and gram negative rods (e coli, klebsiella, pseudomonas and others)

b. Delayed infection (3–24 months) = low grade

Delayed infections are generally acquired intraoperatively and involve low-virulence pathogens such as coagulase-negative staphylococci or Propionibacterium acnes

c. Late infection (> 24 months)

Late infections are almost always of hematogenous origin, most frequently as a result of bacteraemic infections of the skin (S. aureus), respiratory tract (pneumococci), intestine (Salmonella

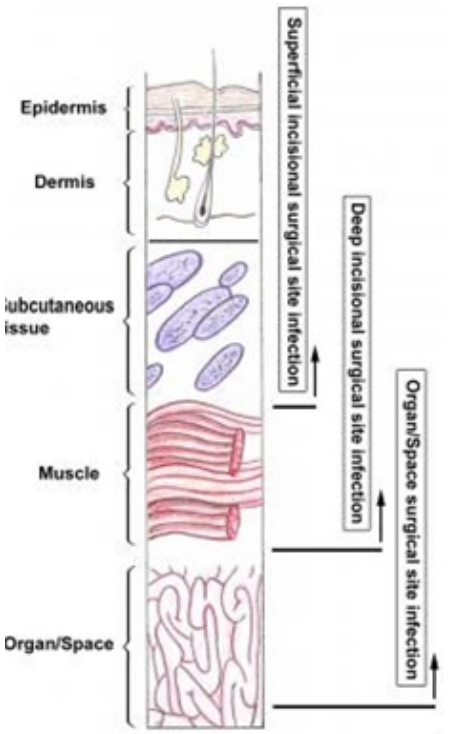

Figure 1: Types of SSI

spp.) or urinary tract (Escherichia coli).

SSIs are also classified (Figure 1)based upon the depth of infection[15].

- **Superficial incisional SSI** - Infection involves only skin and subcutaneous tissue of incision
- Deep incisional SSI - Infection involves deep tissues, such as fascial and muscle layers; this also includes infection involving both superficial and deep incision sites and organ/space SSI draining through incision
- Organ/space SSI - Infection involves any part of the anatomy in organs and spaces other than the incision, which was opened or manipulated during operation

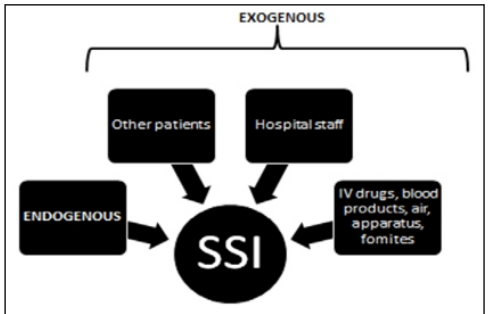

Figure 2: Sources of SSI

Microbiology

The source of an infective agent(Figure 2) can be:

1. endogenous from commensal microorganisms.
2. exogenous (Figure 3)which includes apparatus, fomites, caregivers - mostly through direct contact and less commonly via air or droplets[16].

The most common pathogens responsible for SSIs in orthopaedics include Staphylococcus aureus, Coagulase-negative staphylococci, gram negative rods (E. col, Klebsiella, Pseudomonas aeruginosa and others) and mixed flora. However, SSIs have also been associated with a plethora of other microbes such as Streptococci, Enterococci, Anaerobes, Fungi, Mycobacteria (tubercular and atypical) and Propionibacterium amongst others[17,18].

Das et al[19] reported 37 cases (12%) of SSI from amongst 308 post-operative patients who underwent orthopedic procedures. The most common infecting bacteria was Staphylococcus aureus (24.3%) and was followed by Escherichia coli (18.9%), Pseudomonas (18.9%), then Enterobacter spp. (8.1%) and Achromobacter (8.1%) and others in that order. Most of the Staphylococci were sensitive to linezolid and sensitivity to amoxicillin + clavulanic acid was also good. For Gram-negative infection (Enterobacteriacae) piperacillin + tazobactam, cefoperazone + sulbactam and amikacin were found highly sensitive. For Pseudomonas, again piperacillin + tazobactam was found out to be very effective.

Amardeep et al[20] reported from a prospective study on 248 patients with closed fractures who underwent clean and elective orthopedic implant surgeries. The surgical site infection was diagnosed in 11 (4.435%) patients within 3 months after surgery. Staphylococcus aureus was most common infective organism isolated in 54.54% cases. Gram negative enterobacteriae was the second common group. SSI was significantly associated with increasing age, diabetes mellitus, smoking and anemia. Various other authors have also seen similar results[21-23]. Singh et al[24] recorded gram-negative infections as major threat and isolated gram-negative organisms in 75.6% cases.

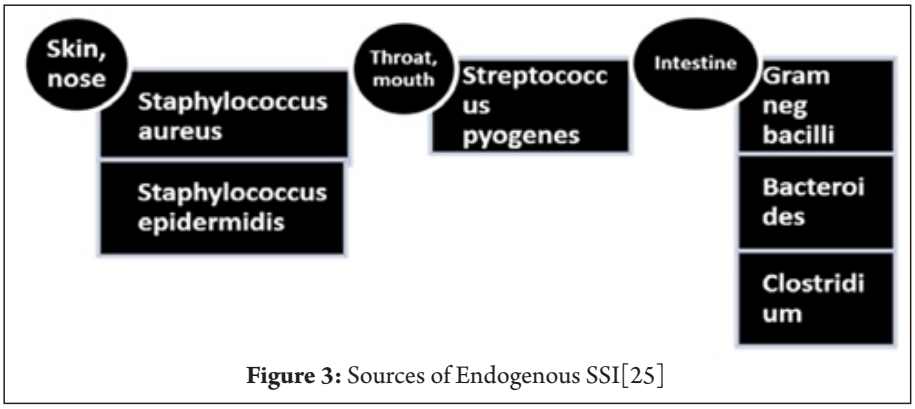

Figure 3: Sources of Endogenous SSI[25]

Staphylococcus Aureus

Staphylococcus aureus is a leading cause of orthopedic SSI. It is a common body commensal[16, 26-28]. They colonize skin surface of about one third of the general population[29]. Two strains of S aureus that cause orthopedic SSI are the methicillin sensitive SA and MRSA. Nasal carriage is strongly associated with SSIs in orthopedics and such patients are two to nine times more likely to develop SSI[30]. The prevalence of methicillin resistant strain (MRSA) is on the rise both in community and healthcare setting[26,27]. MRSA is associated with increased morbidity, mortality and hospital stay[31]

Nasal carriage of staph aureus has been shown in several studies to be the only

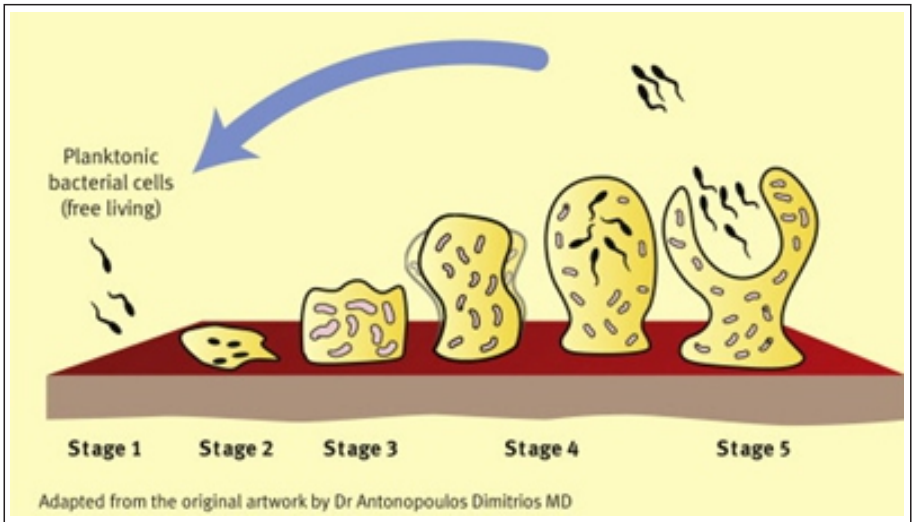

Figure 4: Formation of a Biofilm

independent risk factor in orthopedic SSI[32]. Nasal screening has shown to detect 66% of carriers and combined nasal and perineal swabs have improved detection rates up to 82%. Several studies have shown decolonization to decrease SSI rates[33,34]. The most commonly used protocol is topical intranasal mupirocin ointment twice daily and chlorhexidine body washes for 5 days immediately before surgery along with preoperative antibiotic prophylaxis; patients with MRSA additionally receive Vancomycin [30] Resistance to these antimicrobials should also be monitored. Dental procedures such as tooth extractions are associated with transient bacteremia which may result in direct hematogenous spread to the site of previous surgery, especially where there isa foreign material such as an implant[30]. Recovered bacterial species include viridians streptococci, beta hemolytic streptococci, non-pathogenic gonococci and gram positive anaerobes [35].

Commensals

Commensal organisms which are polymicrobial coexist on almost all healthy body surfaces exposed to the environment[36]. The body's innate and adaptive immunity normally prevents infection from these organisms. However, these defences are disrupted at surgical incision sites due to tissue injury and hematoma. Moreover, implanted medical devices provide a niche for such organisms. Progression to infection is determined by interplay between the host defence, microbial virulence and the presence of an attachment surface.

Biofilm infections

A recent analysis of the morbidity and mortality associated with biofilm infections has revealed that over 12 million people are affected, and that 400,000 people die as a result of these infections in the USA each year[37]. Orthopedic infections are archetypical biofilm infections.

Biofilms are slimy membranes that are often found on the surface of implants, dead bone and even tissue in patients with SSIs. Devitalized surfaces within the infected region are coated with host extracellular matrix. Free floating microbes (planktons) adhere to these surfaces. (Figure 4). Within the matrix these organisms have almost nil metabolic activity and are completely resistant to antibiotics. Multiple species co-exist within a closed spatially structured region that allows robust signaling and transfer of genetic material inducing new unique strains with enhanced diversity and survival characteristics. This entire genetic material distributed across the biofilm functions as a de facto genome larger than any one strain and is termed pangenome which prevents the host from developing an effective adaptive immunity[38-40]. This resistance to antimicrobial bactericidal action may be up to100 to 1000 times the levels that would easily kill the planktonic organisms[41].

The pathogenic organisms implicated in biofilms are extremely difficult to isolate. It is difficult to liberate the microbes from these biofilms and even if this is achieved, the isolated microbe may resemble the planktonic variety with vastly different characteristics. Moreover, conventional cultures are often unable to grow the sessile phenotypes especially the persisters thereby yielding false negative reports. Newer methods used for direct identification of microbes in biofilms using PCR, DNA array, RNA, FISH probes, ELISA, phase contrast microscopy, etc. are still investigational [42].

The acute symptoms and intermittent exacerbations of SSI are due to the rapid growth of planktonic organisms and the host responses to it. The infection is amenable to antimicrobial therapy at this stage. However, if abiotic or compromised tissue surfaces are present soon the sessile or biofilm phase ensues which can only be eradicated with surgical removal of devitalized tissue and implant[41].

Risk factors

There are numerous conditions which potentially increase the risk for developing a SSI [43-46]. Some of them, are

a. Diabetes mellitus,
b. Obesity
c. Immunosuppression- chronic kidney disease, systemic steroid therapy, chemotherapy for neoplasms
d. Intra articular steroid injections
e. Inflammatory arthritis
f. Prior history of infectious arthritis

Surgical and surgical site factors such as revision surgery, prolonged operative time, wound gaping, hematoma,

prolonged wound leakage also have a significant impact on the incidence of SSI.

Berbari et al.[8] defined four independent risk factors for PJI, namely,

a. Superficial surgical site infection (odds ratio [OR] 35.9; 95% confidence interval [CI] 8.3–154.6)

b.NNIS (National Nosocomial Infections Surveillance) surgical patient risk index score of 2 (OR 3.9; 95% CI 1.3–7.5%)

c. Presence of a malignancy (OR 3.1; 95% CI 1.3–7.2), and

d. A history of prior arthroplasty (OR 2.0; 95% CI 1.4–3.0%).

Conclusions

Surgical site infection is not an uncommon problem after orthopaedic procedures. It can be classified into early, delayed and late based on time of onset after the Index surgery. Late infections are almost always hematogenous while delayed infections are usually due to intraoperative contamination with low virulence organisms. The most common pathogens responsible for SSIs in orthopedics include staphylococcus aureus, coagulase negative staphylococci and gram negative rods such as Ecoli, Klebsiella and Pseudomonas. Orthopaedic SSIs are frequently accompanied by the formation of biofilms on implants/ deadbone or even dead tissue. Biofilms prevent penetration of antibiotics and therefore protect the microorganisms. Implants/ prosthesis that have developed biofilms usually need to explanted for control the infection.

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