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Review Article

Nasal carriage of staphylococcus aureus is a major risk factor for surgical-site infections in orthopedic surgery: A review

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ABSTRACT

Surgical site infection is the greatest enemy to the success of a surgeon which is a dreaded complication that can result in poor outcomes, increased morbidity, prolonged hospital stay, escalation of hospital expenditure and mainly constrained relationship between the patient and the surgeon, placing an immense economic burden on the patient and the healthcare infrastructure. The origin of SSI is multifactorial, where Bacteria may get access to the surgical site through both endogenous and exogenous routes, predominantly exogenous contact during the first operative exposure. Staphylococcus aureus is the leading cause of SSI. Implants give a niche for such organisms where biofilms offer a safe environment for their replication. Various modifiable risk factors are also there such as DM, Obesity, Malnutrition etc. The goal of this study was to find out whether nasal carriage of Staphylococcus aureus is a major risk factor for surgical site infection in orthopaedic surgery.

Keywords: Surgical site infection, Staphylococcus, Diabetes Mellitus

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INTRODUCTION

Surgical site infection is the greatest enemy to the success of a surgery. It is a dreaded complication following orthopaedic surgery that results in poor outcomes, increased morbidity, prolonged hospital stay, escalation of hospital expenditure following an otherwise excellent piece of craftsmanship [1]. Many times this may lead to a constrained relationship between the patient and the surgeon. Moreover, it causes an immense economic burden on the patient and the healthcare infrastructure. Recent WHO statistics show that for every 100 hospitalised patients at any given time, 7 in developed and 10 in developing countries acquire SSI[2]. These figures may be as high as 10-30% in centres dealing with critically ill patients. The pathogenesis of SSI is multifactorial. Bacteria can use the surgical site through both endogenous and exogenous routes. Strategies to cut SSI are preoperative patient optimisation, perioperative protocols, sterilisation and operation theatre environment and preparation protocols along with surgeon and operation theatre people preparation protocols, which based on an intricate understanding of the pathogenesis of SSI and the role of biofilms.

Pathogenesis of SSI

The source of the infective agent may be endogenous from commensal microorganisms or exogenous which includes apparatus, fomites, caregivers, etc. The route of infection is mostly through direct contact and less commonly via air or droplets [3]. Staphylococcus aureus is the leading cause of orthopaedic SSI. Methicillin-resistant strain (MRSA) is prevailed in both community and healthcare setting[4,5]. Gram-negative bacilli originate from solutions, fluids and invasive devices and viruses originate from blood and blood products. Fungal infections in people involve Aspergillus spp and Candida albicans. Microorganisms coexist on almost all healthy body surfaces exposed to the environment[6]. The body's innate and adaptive immunity prevent infection from these and these defenses are disrupted at surgical incision sites due to tissue injury and hematoma. Moreover implanted medical devices offer a comfort zone for such organisms. The interplay between the host defense, microbial virulence and exist an attachment surface find the progress of the infection. Once infection sets in, the antimicrobial therapy that being instituted controls the planktonic phase of these organisms which is the individually thriving, free-floating phase that induces acute illness[7]. Biofilms which are polymicrobial, sessile, community-based aggregations within a self-secreted matrix[8,9] exhibiting a radically altered phenotype of growth, gene expression, and protein production compared to taxonomically same planktonic organisms[10,11]. Planktonic organisms are a transient population which is susceptible to host defenses and antimicrobial since biofilms offer a safe environment for microbial replication[12,13] .

Abiotic and devitalized biotic surfaces which are coated in host extracellular matrix adhere the planktonic microbes. As replication of these microbes proceeds, an altered host humoral response and quorum sensing compound induced altered microbial gene expression cause biofilm production, host immune cell lysis and localized tissue destruction. With further expansion as the nutrients become limited in supply a phase of lowered metabolic activity ensues which cause tolerance to antimicrobial agents that act via synthesis of cell walls, nucleic acids or proteins.

Kim et al. reported that the patients themselves bring the microorganisms responsible for SSI[14], primarily Staphylococcus aureus found in their anterior nares. Lee et al. found in their nested, matched case-control study that the most common causative microorganism of orthopedic SSI is staph. aureus (MRSA), followed by coagulase-negative staphylococci, and E. Coli[15]. Berbari et al. revealed that staph. aureus was the most common microorganism causing PJI[16]. Whereas Phillips et al. found in their prospective survey that deep prosthetic infections are most commonly caused by coagulase-negative Staphylococcus, followed by staph. aureus as well as enterococci and streptococci. Staph. aureus was identified to affect 50% of the cases of SSI after hip arthroplasty [17]. Kalmeijer et al. also revealed that staph. aureus affected half of the cases of SSI after orthopaedic surgery; 36 % of all orthopaedic SSI cases were superficially incisional, and 71% of all orthopaedic SSI cases were deeply incisional[18].

Identification of pathogenic organism

The identification of pathogenic organisms implicated in biofilms is however extremely difficult to isolate. Biofilms are having extremely small foci of organisms causing a large area of surrounding inflammation and may be easily missed during biopsies. It is also difficult to liberate the microbes from these biofilms and even if done, it may actually resemble the planktonic variety with a lot of different characteristics. Moreover, conventional cultures are 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. The acute symptoms of SSI are due to the rapid growth of planktonic organisms and the host responses, 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.

Staphylococcus aureus

Two strains of S aureus that cause orthopaedic SSI are the methicillin-sensitive and MRSA. They may colonize on the skin surface. MRSA is associated with increased morbidity, mortality and hospital stay. Nasal carriage is the commonest site of colonization of S aureus and it is strongly associated with skin carriage and such patients are two to nine times more likely to develop SSI [19]. Several studies have shown it as the only independent risk factor in orthopaedic SSI[20]. Nasal screening has shown to detect 66% of carriers and joined nasal and perineal swabs have improved detection rates up to 82%. The most used protocol is topical intranasal mupirocin ointment twice daily and Chlorhexidine body washes for 5 days immediately before surgery along with preoperative antibiotic prophylaxis and patients with MRSA additionally receive Vancomycin.

Nasal carriage of S aureus is spotted as a risk factor for SSI approximately 4 decades ago in several excellent studies.

Prevention of orthopaedic SSI: Once the risk factors for SSI in orthpoaedics are identified, rapid prevention of such risk factors can be done [20]. Gheiti et al. added that the idea of improving the patients' health before surgical intervention is important to reduce the risk of SSI. They also emphasized screening for MRSA in orthopaedic patients, the necessity for smoking cessation, use of staples for wound closure instead of traditional suturing, blood transfusion only when indicated, and control of blood glucose level preoperatively and postoperatively [21].

Rutan et al. recommended monitoring of the blood glucose every two hours during the surgery if the surgery lasts for more than two hours, especially if the patients' preoperative blood glucose level was more than 110 mg/dL and to set up protocol for monitoring blood glucose level postoperatively for the patients having blood glucose level of more than 180 mg/dL [22]. Spahn suggested managing the anemia by iron and erythropoietin supplementation instead of allogenic blood transfusion in order to reduce postoperative complications such as SSI [23]. Daines et al. recommended to initiate the first dose of antibiotics within 60 minutes before making the surgical incision, discontinuing the prophylactic antimicrobial therapy within the first 24 hours postoperatively, limiting the number of healthcare personnel exiting and entering the operating room to decrease the level of airborne microbial contamination and replacing prolonged wound drainage with irrigation [24]. Namba et al. focused on optimizing patient body weight and controlling diabetes mellitus to decrease rates of deep SSI after total knee arthroplasty [25]. The preventive strategies for orthopaedic SSI are summarized in the table.

Preventive strategies for orthopaedic SSI

1. MRSA screening.
2. Administering intranasal mupirocin for nasal carriers of staph. aureus.
3. Using clindamycin and vancomycin fo patients with MRSA or for patients with β-lactam allergy.
4. Use of cefazolin or cefroxamine as antibiotic prophylaxis and adjusted according to obesity.
5. Smoking cessation.
6. Optimizing patient body weight.
7. Using chlrohexidine to prepare patient skin preoperatively at night before the surgery and in morning of the surgery.
8. Use of staples for wound closure.
9. Use of clippers instead of razors for hair removal.
10. Use of Chlorhexidine- alcohol rather than Povidone- iodine in cleansing surgical site.
11. Control of operating room traffic.
12. Irrigation of wound needing prolonged drainage.
13. Establishing protocols for controlling blood glucose levels preoperatively and postoperatively.
14. Optimal timing of administering and discontinuing prophylactic antibiotic therapy.
15. Blood transfusion only when indicated, treatment of anemia with iron and erythropoietin to replace blood transfusion as an option for anemia management.

Diagnosis of surgical site infections

The inflammation related to local procedural trauma causing pain and discomfort in the perioperative period may be confounding. The presence of clinical signs such as fever, erythema, warmth and incision site wound drainage may be important clues to the presence of infection. Chronic infection, on the other hand, is a diagnostic dilemma because the symptoms are typically indolent and less severe and in the presence of orthopaedic hardware differentiating infection from adverse local tissue reactions and mechanical or hardware failure can be challenging. Laboratory parameters such as leukocytosis and elevated C-reactive protein levels and erythrocyte sedimentation rates can be useful in the chronic setting, however, these parameters are nonspecific in the immediate perioperative period when they can be elevated due to inflammation.

Histologic and microbiologic analysis of soft-tissue specimens is the definitive standard for diagnosis of postoperative infection but sampling may sometimes be complex, invasive or contaminated yielding a low sensitivity and specificity rates. Image-guided aspiration of soft tissue collection or biopsy are options but biopsy carries the risk of artificial introduction of infection apart from yielding low positive rates. Imaging is the cornerstone of diagnosis of SSI and apart from confirming the diagnosis, it also provides details about the extent, severity and any associated complications. It provides an objective and longitudinal method of monitoring treatment. Plain radiography though easily accessible, findings lag behind the clinical disease. The initial loss of fat planes is followed by signs of osteomyelitis appearing 7 to 10 days later and signs of chronic osteomyelitis appearing still later. The effects of surgery such as cortical irregularity and the periosteal reaction can mimic osteomyelitis on radiographic findings.

Periprosthetic lucency on radiographs also can be due to aseptic loosening or infection. Arthrography with either ultrasonographic or fluoroscopic guidance can help evaluate joint infections. By taking two separate joint aspirates: a native sample if the fluid is present and a lavage sample using injected contrast material the incriminating organism can be detected. CT facilitates visualization of subtle erosive changes and periosteal reaction in acute osteomyelitis and bony sequester in chronic osteomyelitis that can be masked by postoperative changes and hardware on radiography [26]. Intravenous contrast material may help define focal sinus tracts and abscess which typically demonstrate thick, sometimes irregular rim enhancement although MRI better delineates soft tissue infections. Currently, MRI is the premier modality for diagnosing postoperative infection because of superior soft-tissue contrast and inherent ability to define the anatomic extent of osseous and soft tissue infection. In the postoperative setting, fat-saturated, fluid-sensitive sequences such as short tau hyper inversion recovery(STIR) are used to identify edema patterns within soft-tissue and osseous structures and to differentiate simple cellulitis from more complicated soft-tissue and/or osseous infection [27]. The absence of signal abnormality on STIR images almost excludes the presence of infection [28]. Gadolinium-enhanced, fat-suppressed TI-weighted sequences can distinguish between edematous and necrotic tissue and can identify the presence and extent of fluid collections and sinus tracts. Even when osteomyelitis can be diagnosed using radiography and/or CT, MRI may be vitally important in the detection of an intraosseous abscess, which often requires surgical debridement and not simple intravenous antibiotic therapy.

Diabetes mellitus and SSI

Hyperglycemia can adversely affect humoral and cell-mediated immunity impairing the neutrophil function. Glycated hemoglobin along with micro and macrovascular disease impairs tissue oxygen delivery, inhibition of fibroblast proliferation and collagen synthesis during wound healing. Perioperative hyperglycemia in non-diabetics (2 or more measurements of ≥200mg/dl) is also associated with incidence of SSI. The precise thresholds are not established but most guidelines recommend preprandial and postprandial levels of 90-130mg/dl and 180mg/dl respectively and hba1c levels less than 7% in elective surgeries [29]. Fasting blood sugars and urine for ketones should be checked on the day of surgery. In emergency setting, random blood sugars should be optimized to less than 200mg/dl.

CONCLUSION

SSI in orthopaedic surgery is a major complication that results in physical limitations and delayed recovery from orthopaedic surgical intervention. Therefore it is important to show risk factors and causative agents for orthopaedic SSI so that proper prevention strategies can be implemented. The incidence rates of orthopaedic SSI ranged from 0.7 % to 8%; even though it is a small percentage, it will reflect on hundreds of patients suffering from delayed healing, increased treatment costs and extended hospital stay. Orthopaedic SSI is caused by a number of microorganisms, the most prevalent of which is Staphylococcus aureus. Prevention of orthopaedic SSI requires evaluating patient's health status and establishing protocols and policy to manage the risk factors of orthopaedic SSI.

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