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Surgical site infection in orthopaedics

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Abstract
Surgical site infection is the greatest enemy to success of a surgeon eroding his pride and glory which is a dreaded complication and hence resulting in poor outcomes, increased morbidity, prolonged hospital stay, escalation of hospital expenditure and constrained relationship between the patient and the surgeon, placing an immense economic burden on the patient and the healthcare infrastructure. SSI is of multifactorial origin where Bacteria may access the surgical site through both endogenous and exogenous routes which is predominantly exogenous contacting during the initial operative exposure. *Staphylococcus aureus* is the leading cause. Implants provide a niche for such organisms where biofilms provide a safe environment for microbial replication. Various modifiable risk factors are there such as DM, Obesity, Malnutrition etc. Imaging not only confirms the diagnosis but also provides details about the extent, severity and any associated complications providing an objective and longitudinal method of monitoring treatment.

Keywords: Surgical site infection, Biofilms, Staphylococcus

Introduction
Surgical site infection is the greatest enemy to the success of a surgeon and erodes away his pride and glory. It is a dreaded complication following orthopaedic surgery resulting in poorer outcomes, increased morbidity, prolonged hospital stay, escalation of hospital expenditure following an otherwise excellent piece of craftsmanship [1]. Many a times this may lead to a constrained relationship between the patient and the surgeon. Moreover it places immense economic burden on the patient and the healthcare infrastructure. Recent WHO statistics reveal that for every 100 hospitalised patients at any given time, 7 in developed and 10 in developing countries will acquire healthcare associated infection [2]. These figures may be as high as 10-30% in centres dealing with critically ill patients [3]. Since no patient or surgery is immune to the risk of SSI the subject has received its due attention recently.

The pathogenesis of SSI is multifactorial. Bacteria may access the surgical site through both endogenous and exogenous routes. Patient related factors such as immunity and bacterial colonisation along with antisepsis, antibiotic prophylaxis, surgical technique and healthcare environment play an important role in predisposition to SSI. Strategies to reduce SSI include preoperative patient optimisation, perioperative protocols, sterilisation and operation theatre environment and preparation protocols along with surgeon and operation theatre personnel preparation protocols, all based on an intricate understanding of the pathogenesis of SSI and the role of biofilms.

Pathogenesis of SSI
The source of infective agent may be endogenous from commensal microorganisms or exogenous which includes apparatus, fomites, caregivers, etc. [Fig 1] [Fig 2]. The route of infection is mostly through direct contact and less commonly via air or droplets [3].

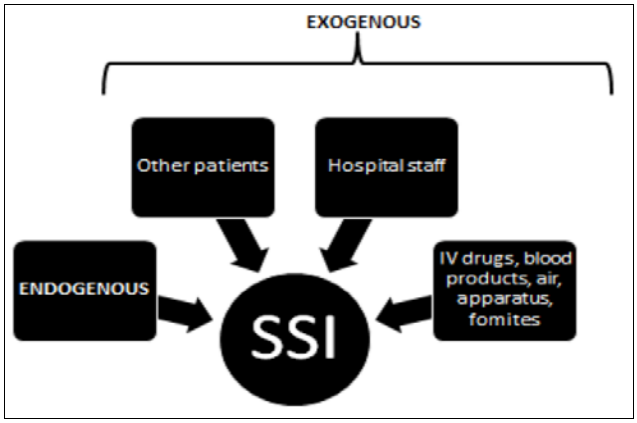

Fig 1

Staphylococcus aureus is the leading cause of orthopaedic SSI which is a common body commensal [3, 4, 5]. The prevalence of methicillin resistant strain (MRSA) importantly is on the rise both in community and healthcare setting [4, 5]. Gram negative bacilli originate from solutions, fluids and invasive devices and viruses from blood and blood products [3]. Fungal infections in immunocompromised individuals involves *aspergillus spp* and *Candida albicans* [3].

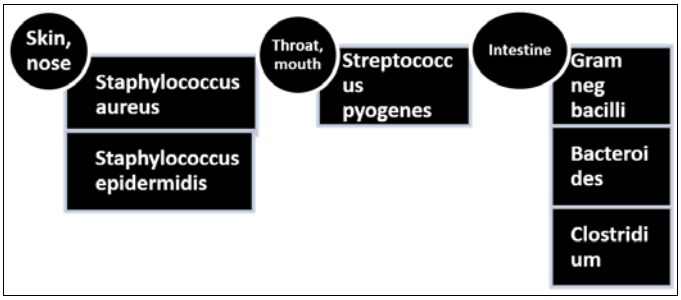

Fig 2: Endogenous infection

Commensal organisms which are polymicrobial coexist on almost all healthy body surfaces exposed to the environment [6]. The body's innate and adaptive immunity prevent infection from these circumstances 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. Once infection sets in, antimicrobial therapy being instituted controls the planktonic phase of these organisms- the individually thriving, free floating phase which induces acute illness [7].

Biofilms which are polymicrobial, sessile, community based aggregations within a self-secreted matrix [8, 9] exhibit radically altered phenotype regarding growth, gene expression and protein production compared to taxonomically identical planktonic organisms [10, 11].

Planktonic organisms are a transient population susceptible to host defences and antimicrobials whereas biofilms provide a safe environment for microbial replication [12, 13, 14] and microcolony growth via detachment and dispersion which is genetically programmed or under mechanical shear.

Abiotic and devitalised 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 localised 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. Such populations called persisters exhibit almost nil metabolic activity and are completely resistant to antimicrobials. Moreover in biofilms multiple species co-exist within a close spatially structured region that allow robust signalling 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 [7]. This resistance to antimicrobial bactericidal action may be upto100 to 1000 times the levels that would easily kill the planktonic organisms [15].

The identification of pathogenic organisms implicated in biofilms are however extremely difficult to isolate. Biofilms have extremely small foci of organisms causing a large area of surrounding inflammation and may be easily missed during biopsies. Also it is difficult to liberate the microbes from these biofilms and even if done may actually 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 [9]. The acute symptoms and intermittent exacerbations of SSI are due to the rapid growth of planktonic organisms and the host responses to it, which is amenable to antimicrobial therapy 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 devitalised tissue and implant [15].

Staphylococcus aureus

Two strains of S aureus that cause orthopaedic SSI are the methicillin sensitive and MRSA. They colonize skin surface of about one third of the general population [16]. MRSA is associated with increased morbidity, mortality and hospital stay [17]. Nasal carriage is the commonest site of colonisation of S aureus and is strongly associated with skin carriage and such patients are two to nine times more likely to develop SSI [18].

Several studies have shown it to be the only independent risk factor in orthopaedic SSI [19]. 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 decolonisation to decrease SSI rates [20, 21, 18]. 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 and patients with MRSA additionally receive Vancomycin [18]. Resistance to these antimicrobials should also be monitored.

Preoperative patient optimisation

Patient education

Patients should be preoperatively assessed for physiological factors such as age, recumbency as well as personal factors such as general recumbency. Hence patient education is a vital component of SSI prevention.

Modifiable patient risk factors

A. Diabetes mellitus

Hyperglycemia adversely affects humoral and cell mediated immunity specifically impairing neutrophil function. Glycated haemoglobin along with micro and macrovascular disease impairs tissue oxygen delivery, inhibition of fibroblast proliferation and collagen synthesis during wound healing [18]. Perioperative hyperglycemia in non-diabetics (2 or more measurements of ≥200mg/dl) [22] are also associated with increased 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 hba_{1c} levels less than 7% in elective surgeries [2, 23]. Fasting blood sugars and urine for ketones should be checked on the day of surgery [2]. In emergency setting random blood sugars should be optimised to less than 200mg/dl [2].

B. Obesity

Obesity may be one of the most common modifiable risk factor for SSI. It increases the technical challenge in surgery and results in larger incisions, more extensive tissue dissections, greater blood loss, and longer surgical times. Compounding these, such patients often receive insufficient doses of prophylactic antibiotics [18, 24].

C. Malnutrition

Inadequate nutritional reserves specially protein deficiencies, impairs collagen and proteoglycan synthesis thus tissue healing. Decreased lymphocyte count reduces the capacity of the immune system to combat infection thereby increasing the chances of SSI. Most significant predictors of protein deficiency include serum albumin <3.5g/dl, total lymphocyte count $<1500/mm^3$, and serum transferrin <200 mg/dl. The relatively short half-lives of albumin and transferrin make measurement of these protein levels particularly useful for detecting acute changes in nutritional status [18, 25, 26].

D. Rheumatoid Arthritis

Rheumatoid arthritis constitutes a greater risk in late infection as opposed to acute infection. It is a direct risk factor of disease specific impairment of wound healing and immune response to pathogenic organisms. The medications prescribed for rheumatoid arthritis indirectly acts as risk factor of SSI [18, 27, 28].

E. Corticosteroids

The use of corticosteroids is one of the most widely accepted risk factors for SSI. The level of risk appears to be directly proportional to the dosage [29]. They inhibit vascular permeability, leukocyte chemotaxis, adhesion, and phagocytises as well as impair cell mediated immune response. [18].

F. Methotrexate and Non Biologic DMARD

Most investigators conclude that nonbiologic DMARDS don't have substantial effect on SSI rates. The long half-lives of these drugs also make temporary discontinuation of surgery difficult and enhance the challenge of underlying disease flares and loss of disease controls [18]. An international panel of rheumatologists recommended against discontinuing methotrexate before surgery [30].

G. Anti-Tumour Necrosis Factor

Macrophage derived TNF alpha is a proinflammatory molecule that plays a critical role in combating infection, defending against intracellular pathogens and patients undergoing anti TNF treatment are predisposed to mycobacterial and other opportunistic infections. Moderate evidence suggests anti TNF drugs increase the risk of SSI from common bacteria [18].

H. Non rheumatoid Inflammatory Arthritides

Conditions other than rheumatoid arthritis such as psoriatic arthritis, ankylosing spondylitis also increase the risk of SSI. Medical treatment involves similar medications which are used in rheumatoid arthritis. Judicious preoperative use of these medications will reduce the rates of SSI [18].

I. Cigarette Smoking

Cigarette smoking appears to be the substantial modifiable risk factor of SSI. Nicotine mediated tissue hypo perfusion leads to unfavourable shift of Hb O2 dissociation curve which ultimately results in impaired tissue healing. It also inhibits collagen synthesis resulting inflammation and dysfunction of vascular endothelium. Nicotine, carbon monoxide nitric oxide are all implicated in impaired wound healing. Cessation of smoking results in decreased risk of SSI [31].

J. Urinary Tract Infection

Post-operative UTI can be a risk factor for periprosthetic [32] joint infection but its association with SSI is unclear. Concensus exists that patients should be asked about urgency or frequency of micturition. If such symptoms present urine analysis and urine culture should be obtained. In the absence of symptoms surgery can proceed if urine analysis doesn't suggest infection despite presence of bacteriuria. Surgery can also proceed in settings of symptoms with$<10^3$ CFU/mL. If CFU$>10^3$ in setting of urinary tract obstruction or symptomatic infection surgery should be postponed [18].

K. Anaemia

Anaemia is the 2nd most prevalent modifiable risk factor for SSI after obesity. Postoperative allogenic blood transfusion is a risk factor for SSI. Preop anaemia increases the risk for requiring postop blood transfusion. Although treatment of preop anaemia decreases this risk the optimal treatment method has not been established [18].

L. HIV

Conditions associated with immune deficiencies are risk factors for SSI. In patients with HIV infection CD count >400/mL and an unpredictable viral load is strongly associated with risk for patients undergoing total joint arthroplasty [33].

M. Timing of Dental Procedure

Dental procedures such as tooth extractions are associated with transient bacteraemia, and sepsis which can result in implant shedding and active infection [18]. Recovered bacterial species include viridians streptococci, beta haemolytic streptococci, non-pathogenic gonococci and gram positive anaerobes [34]. More ever dental procedures are associated with low grade bacteraemia or CFU $<10^4$ which is very less in magnitude achieving haematogenous seeding. However poor general health associated with potential risk factor for prosthetic joint infection in the hip and knee [18].

Diagnosis of surgical site infections

In the perioperative period inflammation related to local procedural trauma causing pain and discomfort may be confounding but the presence of clinical signs such as fever,

erythema, warmth and incision site wound drainage may be important clues to the presence of infection [35]. 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 [36]. 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 routinely be elevated because of inflammation [37].

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 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 [35].

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 clinical disease [35]. 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 periosteal reaction can mimic osteomyelitis on radiographic findings. Periprosthetic lucency on radiographs also can be due to aseptic loosening or infection. Arthrography with either ultrasono graphic or fluoroscopic guidance can help evaluate joint infections. By taking two separate joint aspirates: a native sample if 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 sequestrae in chronic osteomyelitis that can be masked by postoperative changes and hardware on radiography [35, 38]. 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 [39].

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 [39]. Absence of signal abnormality on STIR images virtually excludes the presence of infection [40]. 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 frequently requires surgical debridement rather than simple intravenous antibiotic therapy. Artifacts can be reduced with implants having lower ferrous content and afield strength of 1.5T or less [39].

Conclusion

Surgical site may lead to many complications. Adherence to proper pre-operative protocols may curtail this complication. Excellent surgery may lead to failure. However it is very much preventable by following strict surgical protocols.

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