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IP International Journal of Medical Microbiology and Tropical Diseases

Journal homepage: https://www.ijmmtd.org/

Review Article

Surgical site infections: A review

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ARTICLE INFO

Article history:
Received 05-07-2021
Accepted 06-08-2021
Available online 01-09-2021

Keywords:
Surgical site infections
Prevention
Microbiology

ABSTRACT

Infections that are found in the wound produced by any surgical process or procedure are termed surgical site infections in medical parlance. They can be caused by a number of virulent microorganisms, and can be prevented partly or fully, by proper precautionary measures and early diagnosis and treatment. We here present a brief, comprehensive review of the epidemiology, causative microbes and management of these infections.

1. Introduction

Surgical site infections (SSIs) are infections that occur at the site of incision for surgery and implants, that can occur upto 30 days after the surgical procedure is over.[1] It contributes to about 10-30% of all hospital acquired infections. It can inflict a huge financial load on both the patient and also the healthcare giver.[2] Scientific data indicates that the overall reported incidence of SSIs can be as much as 20%. It depends on many factors like nature or type of surgical procedure, the criteria of surveillance used, and the type of data obtained.[1] There is data to suggest that the true prevalence of SSIs that is currently unknown, is probably under-reported over the years.[2] Several reports state that the risk of surgical site infection is about 1-3% for an elective clean surgery.[3] Surgical site infections are in fact, the second most commonly encountered Hospital acquired infections (HAI) after asymptomatic bacteriuria.[3] It is also associated with a mortality of about 3%, and is a significant cause of morbidity as well.[4]

1.1. Type of surgeries associated

SSI is significantly more frequently found in prolonged surgeries (of more than 2 hours duration), and surgeries associated with placement of drains.[5] Aseptic surgical techniques have shown to diminish the incidence of SSIs. Such techniques include minimising blood loss, controlling fall in temperature, removing dead and necrosed tissues, using drains and sutures properly, removing dead space

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https://doi.org/10.18231/j.ijmmtd.2021.027

and properly taking meticulous care of the postoperative incision.[6] Rate of infection is found to be lesser in minimally invasive surgery as compared to open surgery.[6] Also, several studies suggest that putting drains through a separate incision far from the incision of operation can also mitigate the risk of infection to a great extent.[7] It seems that the risk of SSI also decreases when closed suction drains are used, instead of open drains.[8] Mild hypothermia also increases risk of SSI by causing vasoconstriction, and altering the proper functioning of phagocytic White blood corpuscles.[9]

1.2. Predisposing factors

Pre-existing health conditions like increased age (>55 years) and Diabetes mellitus lead to a significantly higher incidence of SSIs than others.[10] Other patient-related risk factors found to be associated significantly with higher occurrence of SSIs are: obesity, smoking and a low serum albumin concentration.[11] The major sources of infection, however, are the microorganisms present on the patient's own skin and, less commonly, the gut or female reproductive tract.[12]

1.3. Microbiology

Most cases of SSIs are due to bacteria. Common microbes causing SSI are *Staphylococcus aureus, Pseudomonas aeruginosa, Corynebacterium* spp. (diphtheroids), *Serratia marcescens*, and anaerobic bacteria like *Prevotella* spp. and Peptoniphilus spp.[12] Coagulase negative Staphylococci and Enterococcus spp. are also important causes of SSIs.[13] The microbial flora depends on the type and site of surgical procedure.[11] These organisms are part of the host's endogenous microflora, but may also come from the operative room environment and the flora of healthcare givers.[14] For instance, *Staphylococcus aureus* is most frequently found in SSIs occurring after breast cancer surgery.[15] *Staphylococcus aureus* is the most common pathogen behind most SSIs, accounting for about 30% of all the cases.[16] Entry into a hollow viscera by such procedures exposes the surrounding tissue to Gram negative bacterial pathogens like Escherichia coli, Gram-positive organisms such as Enterococcus spp., and, occasionally, anaerobes such as *Bacillus fragilis*.[17]

2. Pathogenesis of SSIs

There are many clinical variables that can lead to SSI as an outcome following surgery.

Pathogens that cause SSI are acquired either internally from the patient's own microbial flora present on skin. It can also come from an opened viscus or extraneously from the instruments used or the operating theatre environment. Microorganisms may also gain access to a wound after surgery, before the skin has been sealed. Less commonly, microbes from a far-away source of infection, mostly through haematogenous spread, can also cause SSI by adhering to a prosthesis or other implant or device that has been left in the site of operation. Prolonged operations increases the risk of exogenous contamination.[18] In clean surgeries which do not involve laparotomy or operation in genital tract, *Staphylococcus aureus* (MRSA) is the predominant microbe causing SSI ; it is associated with a poor clinical outcome. Other Gram positive bacteria like Coagulase negative Staphylococci, Streptococcus spp. and Enterococci are also incriminated but less commonly.[19–22] Surgeries which probe into hollow viscera like appendicectomy, colorectal, gastroduodenal, biliary tract and urologic operative procedures, exposes surrounding tissues to Gram negative bacilli like *Escherichia coli*, *Klebsiella* spp., *Proteus* spp., *Enterobacter* spp.,, Gram positive bacteria like *Enterococcus* spp., and anaerobes.[23–25] In surgeries involving the head and neck region, anaerobes such as Peptostreptococcus spp., *Propionibacterium* spp., *Prevotella* spp., *Veillonella* spp., *Bacteroides* spp., and *Clostridium* spp., are predominantly responsible for SSIs since these bacteria are present normally in the oropharynx as commensal flora. Hence they gain access to the surgical site quite easily.[26] SSIs can be monomicrobial or polymicrobial. Polymicrobial infections are usually found at surgeries involving the oropharyngeal, axilla, perineum and GIT region because of a mixed population of aerobic and anaerobic microorganisms. Yeasts of *Candida* species can also be the agents of polymicrobial SSIs.[27] Occurrence of SSI depends on the interplay of 4 factors as below:

2.1. Inoculum or load of bacteria

Procedures involving the sites which are heavily colonized with bacteria like the gut (10^3-10^4bacteria/ml of distal small bowel contents, 10^5-10^6 bacteria/ml in right colon, 10^{10}-10^{12} bacteria/gm of stool in rectum and sigmoid colon having about 600 different species of bacteria) and the female genital tract (10^6-10^7bacteria/ml) are at higher risk of developing SSIs since large inocula of bacteria can harbour the wound site during the course of operation.[27–30]

2.2. Virulence of bacteria

The more virulent the bacterial contaminating agent, the greater is its probability of causing infection. It also depends upon the type of exotoxins they release or the nature of LPS or endotoxins present in their cell walls. Bacteria like *Staphylococcus aureus*, *Clostridium perfringens* and *Streptococcus pyogenes* need only a small dose or inoculum to cause severe necrotizing infections at the surgical site. Aerobic gram negative bacteria (like *E.coli*) and anaerobic colonic bacteria like *Bacteroides fragilis* can have a synergistic relationship in vivo that leads to heightened

virulence when the two species are concomitantly present in critical inoculum counts at the surgical site.[31] Antibiotic resistance can ideally be considered as a virulence feature of bacterial contaminants as well; selected patients will hence have more colonization by resistant bugs inside the colonic lumen or at the level of skin. Patients who have prolonged preoperative hospitalization, recent hospitalization for other purposes, recent history of antibacterial agent consumption for the treating other infections, or those who are admitted in chronic care facilities will be colonized with more virulent microbes than any other patient having colon surgery. They can thus be expected to have more chances of developing SSI.

2.3. Microenvironment around surgical site

Factors like hemoglobin and presence of hematoma in the surgical incision site, foreign bodies or necrotic tissue from overuse of electrocautery procedure or wound trauma from too much traction pressure also increase rates of infection even from lower load of bacteria.[29,30] Finally, presence of dead space in the surgical incision serves as a dependent basin for the accumulation of serosanguinous fluid after the closure of the wound. This drainage basin later harbors bacterial contaminants in a watery environment that cannot be well tackled by host inflammatory or immune response.

2.4. Innate and acquired host defenses

The tissue response of the host is very important. There can be two components of the host response. Firstly there is the intrinsic, genetically programmed responsiveness that is poorly understood and possibly not affected by strategies for prevention.[28] Acquired derangement of the host immune and inflammatory response by many factors is also likely to be faced. These acquired factors may be chronic conditions like Diabetes mellitus, chronic kidney, chronic lung or chronic liver diseases. Also, there are acute conditions, namely hyperglycemia, hypoxemia, hypoalbuminemia, hypothermia or acute anemia that are associated with elevated rates of SSI. If wound contamination still continues or secondary infection occurs here, continuous activation of the complement system and other pathways can develop. It then provides a steady supply of chemotactic factors, leading to a greater influx of Polymorphonuclear Leucocytes into the wound. Monocyte takes up the role of a proinflammatory cells here with the release of many powerful cytokines. Serotonin is released from the mast cells, that causes vasodilation and enhanced vascular permeability. The combination of these two, that is intense vasodilation and increased blood vessel permeability then produces the classical clinical findings of inflammation, like rubor (redness), tumor (swelling), calor (heat), and dolor (pain).

2.5. Clinical features

Surgical site infections usually present 2-7 days after the procedure; however, with any prosthetic device they can also manifest later due to spread of bacteria from other sites. Physical examination may reveal localized tenderness, erythema, warmth and edema at the site of surgery. Purulent wound drainage may or may not be evident.[31] Pus-laden wound drainage occurs in about two-third of case of all SSIs after Instrumentation. Deep-seated infection present generally with constitutional symptoms. In rarer cases, patients might also suffer from server sepsis and end-organ failure. Deep infections often lack evident superficial features, making their diagnosis usually presumptive. An organ or space SSI may reveal a discharge of pus coming out from a drain put through the skin into the body space or organ. A collection of such purulent discharge is an enclosed area of pus and disintegrating tissue , surrounded by inflammation and epithelium, is called an abscess .

2.6. The common clinical features include

1. Localised pain (this is often different to the typical post-operative pain)
2. Wound dehiscence
3. Unexplained persistent fever.
4. Delayed healing of the surgical site
5. The tissue around the surgical site can be discoloured
6. A foul odor coming from the incision site
7. Incision is hot to touch

2.7. Laboratory diagnosis

2.7.1. Management (including treatment and prevention)

1. Complete and holistic assessment is needed for identifying the risk factors which can influence the surgical wound healing pre-operatively, intra-operatively and postoperatively. The pre-operative assessment can focus on the patient's general health condition and coexisting health conditions.[32]
2. Managing pre-operative risk factors**:** There are both intrinsic and extrinsic factors that heighten the risk of an SSI. In particular, there is accumulating evidence that a patient's age is a risk factor which is related to decreased potential for healing and also diminished immunity with aging.[32] Smoking is also a potent risk factor for SSIs; patients hence should be advocated to quit smoking.[33]
3. Managing intra-operative risk factors: The two primary intra-operative factors involved in prevention of SSIs are maintenance of patient's homeostasis and persistent practice of effective operating room (OR) safety procedures. This includes the maintenance of normal patient body temperature and blood glucose levels, and also blood oxygen saturation (SpO_2) of ≥95% all through the surgical procedure.[34,35] Finally,

it is advised that the dead space, within or below the skin, be reduced as much as is possible. Also, wound trauma be reduced by gentle tissue handling and limited use of electrocautery.[36,37]

4. Managing postoperative risk factors: Managing postoperative risk factors involves continuation of the intra-operative measures, which comprise maintenance of body temperature, adequate oxygenation, clean and intact wound dressing and well-controlled pain.[38]
5. Choosing an appropriate dressing or device to manage exudates and bacterial burden: Proper dressing and device should be employed to manage the amount and the nature of exudate. Antiseptic impregnated dressings can be actively bacteriostatic since microorganisms are then killed by the release of the antiseptic agent into the wound site or on contact with the antiseptic agent itself, if and when absorbed into the dressing.[39]
6. Considering adjunctive therapies: The treatment of open abdominal wounds (laparostomy) has improved a lot survival, and also has enabled a higher percentage of total abdominal wall closures. The role of hyperbaric oxygen therapy (HBOT) in open surgical wounds may also be of benefit. Consideration of HBOT must include costs to clients or families, like travelling costs and reimbursement fees for the cost of such therapy.[40]

3. Treatment

Surgical, enzymatic or ultrasound-guided debridement is also often helpful in treating SSI, along with regular antiseptic dressings.[41] Antiseptic impregnated dressings can be actively bacteriostatic when microbes are eliminated by the release of the antiseptic agent into the wound or on contact with the antiseptic when absorbed into the dressing material, as discussed previously.[41] Some dressings employ passive antimicrobial methods, when microorganisms bind to the dressing materials and are then physically removed from the wound on the change of dressing.[41] Cyanoacrylate-based microbial sealants can further decrease the rates of SSI occurrence, when incorporated into the antiseptic pre-surgical dressings.[42]

4. Conclusion

Surgical site infections can be quite challenging to detect and then treat. Prevention necessitates meticulous patient care and all required interventions. Clinicians and microbiologists should hence work hand in hand for the timely diagnosis and treatment of such infections. A uniform policy should be adopted in every tertiary healthcare facility for necessary guidance in this context.

5. Source of Funding

None.

6. Conflicts of interest

There are no conflicts of interest.

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Cite this article: Bhattacharyya S, Kumar M, Singh S, Sengupta A, Sarfraz A, Kumar A, Jaiswal NK, Kumar D, Kumar R. Surgical site infections: A review. *IP Int J Med Microbiol Trop Dis* 2021;7(3):124-128.