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SURGICAL SITE INFECTION IN ORTHOPEDIC SURGICAL WOUND

A Technical Note

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ABSTRACT

Surgical site infection (SSI) in orthopedic surgery is a serious complication and one of major complication in orthopedic surgery. methicillin-resistant strains of *S. aureus* (MRSA) is the most bacteria that causes SSI. There are three surgical stages that can have SSI, Preoperative, intraoperative and postoperative. Based on surgical wound there are four types of wound :clean, clean-contaminated, contaminated and dirty-infected. Classification of SSI are Superficial incisional SSI, Deep Incisional SSI and Organ/ Space SSI. Antibiotic prophylaxis is one of the most crucial in the prevention of SSI, choice of the antibiotic and respective pharmacokinetics.the antibiotic is chosen based on patient supposed colonization and the type of pathogens. The majority of orthopaedic SSIs are due to coagulase- negative staphylococci, mainly *S. aureus* and *S. epidermidis*, which are isolated in most cases. First and second generation cephalosporins are wide spectrum antibiotics acting mainly against aerobic gram-positive and gram-negative bacteria. Clindamycin and vancomycin can be use to patients with MRSA or patients with β-lactam allergy. There are many things that we can do to prevent SSI in future. This paper aims to review surgical site infection in orthopedic surgical wound

Keywords :*Surgical site infection,Risk factor, Orthopaedic Surgery*

INTRODUCTION

Surgical site infection (SSI) in orthopedic is a complication of surgery is one of major complication of surgery in orthopedic which increases morbidity and the cost of patient care in the hospital [1]. SSIs still a problem in surgery, although there are significant advances in surgical techniques, modern technologies in the operating room, and precautions such as perioperative intravenous antibiotics and skin antisepsis before surgery. Surgical site infections are increased risk of morbidity and mortality of patients and can have serious economic consequences.[2]

In indonesia,incidence of nosocomial infections from 10 education hospitals conducted active surveillance in 2010, In that study, it was reported that the incidence of nosocomial infections was quite high, around 6-16% with an average of 9.8%. Previous studies in various hospitals, both domestic and foreign hospitals, have identified several risk factors that can increase the prevalence of SSIs.[3]

There are many causes of SSI, which have been documented and reported as risk factors. A risk factor is any factor that is recognized as a contributor to increase of susceptibility to SSI. The risk factors of orthopedic SSI have been identified in the literature as preoperative risk factors, intraoperative risk factors, and postoperative risk factors [1,3]

The use of prophylactic antibiotics is one of the prevention of infection based on the surgical conditions used for patients who have not had the infection. In accuracy in prophylactic antibiotics can cause severe infections and prophylactic antibiotic use the inaccuracy is one of the factors of incidence infection of the operating area.[3] This paper aims to review surgical site infection in orthopedic surgical wound.

Epidemiology

The incidence of SSI globally varies from 0.9% of cumulative SSI rate in the USA, to 2.6% in Italy, to 2.8% in Australia, 2,1 % in Republic of Korea, to 6.1% in Low Middle Income Countries (LIMC) and 7,8 % in South East Asia[4].

In asrawal *et al* 2019 ,the incidence of SSI in orthopedic surgery at Fatmawati Hospital around 3.9% (30 SSI out of 770 patients were operated) which is much higher than the standard, it should be less than 2% for postoperative surgical site infections in Fatmawati hospital. [3]

In orthopedic prosedure there are reported that the incidence of SSI after hip arthroplasty procedures ranged from 2.23 % to 7.6%, whereas for adult spinal surgery the incidence of SSI ranged from 0.7% to 12% ,and for primary total knee arthroplasty incidence of deep SSI it was 0.72%. For spinal decompression and fusion surgeries, the incidence rate of SSIs was found to be 3.04%, 1.16% of these SSIs were deep incisional, and 1.88% were superficial incisional. the incidence rate of SSI following orthopedic spinal operations was 2%; 43% of the orthopedic SSI cases were classified as deep incisional, 17% were classified as organ space, and 39% were classified as superficial incisional. For patients who had SSI after hip arthroplasty, 74% of them had superficial incisional SSI, 16% of them had deep incisional, and 10% of them had a joint infection.[1]

Pathogenesis of SSI

Most SSIs are believed to be acquired during surgery. This is supported by the success of SSI prevention measures directed towards activities in the operating theatre and by reports demonstrating matching strains of pathogens from the surgeon's fingers and postoperative infection. Pathogens that cause wound infections endogenously derived from the patient's own normal flora in the skin or from a viscus open or exogenously from contact with operating room personnel or the environment. However, the period of greatest risk from infection remains the time between opening and closing the operating site. [12,13]

Prolonged operation that increases the length of time increases the risk of exogenous contamination. In the clean operation that does not open the abdomen or genital tract such as cardiothoracic surgery, neurosurgery, orthopedics, ophthalmic, and breast surgery, *Staphylococcus aureus* (MRSA) is a major pathogen that causes infection of the operating site and associated with a poor outcome. The emergence of a methicillin-resistant strain of *S. aureus* (MRSA) has increased morbidity and mortality from wound infection. Other gram-positive organisms such as coagulase-negative *staphylococci, enterococci* and *Streptococcus* species, more rarely involved.[12,13]

SSI can be monomicrobial or polymicrobial. Polymicrobial infections usually occur in ororopharyngeal, axilla, perineal and GIT surgeries due to mixed aerobic and anaerobic organisms. Yeast of *Candida* species can also be a part of polymicrobial SSI. The development of SSI depends on the interaction of four factors :

1. **Inoculum of bacteria** : procedures involving the sites which are heavily colonized with bacterias
2. **Virulence of bacteria** : the more virulent the bacterial contaminant, the greater the probability of infection
3. **Microenvironment around surgical site** : Presence of necrotic tissue, dead space, foreign bodies at the surgical site increases the probability of infection
4. **Innate and acquired host defences** : role of acute wound healing process play a role in wound healing. If wound contamination persists or secondary infection occurs, continuous activation of the complement system and other pathways. Sterile incisions will heal normally without the presence of PMN [12,13]

Pathophysiology of wound recovery

Wound healing is process to repair the damage. The main component in the wound healing process is collagen in addition to epithelial cells. Fibroblasts are cells that are responsible for collagen synthesis. The physiology of wound healing will naturally experience the following phases.[9,10]

1. **Inflammation phase**

When the tissue is injured,a cut blood vessel in the wound will cause bleeding, the body's first reaction is to try to stop the bleeding by activating the intrinsic and extrinsic coagulation factor , which leads to platelet aggregation and vasoconstriction clot formation, shrinkage of vessel ends blood loss (retraction) and haemostasis reactions. Haemostasis reactions will occur due to blood coming

out of the skin injured will come into contact with collagen and extracellular matrix, this will trigger the release of platelets or also known as platelets to express glycoproteins on the cell membrane so that the platelets can aggregate and stick to each other and form a mass (clotting).[9,10,11]

This hemostasis component will release and activate cytokines which include Epidermal Growth Factor (EGF), Insulin-like Growth Factor (IGF), Plateled-derived Growth Factor (PDGF) and Transforming Growth Factor beta (TGF-β) which play a role in the occurrence of neutrophil chemotaxis. ,macrophages, mast cells, endothelial cells and fibroblasts. This is called the inflammatory phase. In this phase, there is vasodilation and accumulation of polymorphonuclear (PMN) leucocytes. Platelet aggregates will release inflammatory mediators of Transforming Growth Factor beta 1 (TGF β-1) which are also released by macrophages. The presence of TGF β 1 will activate fibroblasts to synthesize collagen.[9,10,11]

2. Proliferation phase

The proliferative phase lasts from the 3rd to 14th posttraumatic day, characterized by an alternating provisional matrix dominated by platelets and macrophages gradually replaced by fibroblast cell migration and deposition of extracellular matrix synthesis. At the macroscopic level it is characterized by the presence of granulation tissue which is rich in new blood vessel networks, fibroblasts, and macrophages, granulocytes, endothelial cells and collagen which form extracellular and neovascular matrices that fill wound gaps and provide scaffold adhesion, migration, growth and cell differentiation. The purpose of this proliferation phase is to balance between scar tissue formation and tissue regeneration.[9,10,11]

There are three main processes in the proliferation phase, there are :

a. Neo angiogenesis

If there are tissue damage, the angiogenesis process plays a role in maintaining the continuity of the function of various affected tissues and organs. This happens through the formation of new blood vessels that replace damaged blood vessels. In angiogenesis, the formation of new blood vessels comes from capillaries that emerge from the blood vessels small all around [9,10,11]

In proliferation, angiogenesis is also known as neovascularization, which is the process of forming new blood vessels,is very important in the steps of wound healing. The tissue where new blood vessels form, usually looks red (erythematous) due to the formation of capillaries in that area. [9,10,11]During angiogenesis, endothelial cells produce and secrete cytokines. Several growth factors are involved in angiogenesis, including Vascular Endothelial Growth Factor (VEGF), angiopoetin, Fibroblast Growth Factor (FGF) and TGF-β. After adequate tissue formation, migration and proliferation of endothelial cells decreases, and excess cells die by apoptosis. [9,10,11]

b. Fibroblast

Fibroblasts produce an extracellular matrix which fills the wound cavity and provides the basis for keratinocyte migration. The extracellular matrix is the

most visible component in the scar in the skin. Macrophages produce growth factors such as PDGF, FGF and TGF- β which induce fibroblasts to proliferate, migrate, and form the extracellular matrix. With the help of matrix metalloproteinase (MMP-12), fibroblasts digest the fibrin matrix and replace it. with glycosaminoglycan (GAG). Over time, this extracellular matrix will be replaced by type III collagen which is also produced by fibroblasts. This collagen is composed of 33% glycine, 25% hydroxyproline, and the rest is water, glucose, and galactose. Hydroxyproline comes from proline residues which undergo a hydroxylation process by the enzyme prolyl hydroxylase with the help of vitamin C.

Hydroxyproline is only found in collagen, so it can be used as a measure of the amount of collagen. multiplying the result by 7.8. Furthermore, type III collagen will be replaced by collagen type I in the maturation phase. Factor Proangiogenic produced by macrophages such as vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF) -2, angiopoietin-1, and thrombospondin will stimulate endothelial cells to form neovascular through angiogenesis.[9,10,11]

c. Re-epithelization

Simultaneously, basal cells in the epithelium move from the wound edge to the wound area and cover the wound area. At the wound edges, a single layer of keratinocyte cells will proliferate later migrate from the basement membrane to the wound surface.

When migrating, keratinocytes become flat and long and also form long cytoplasmic protrusions. They will bind to collagen type I and migrate using integrin-specific receptors. Collagenase released by keratinocytes will dissociate cells from the dermis matrix and help the movement of the initial matrix. Keratinocytes that have migrated and differentiated into epithelial cells will migrate over the provisional matrix towards the middle of the wound, when these epithelial cells have met in the middle of the wound, cell migration will stop and basement membrane formation begins [9,10,11]

3. Remodelling phase (Maturation)

This phase is the last and longest phase in the wound healing process. There is a dynamic process in the form of collagen remodeling, wound contraction and scar maturation. Activity collagen synthesis and degradation resides within balance. This phase lasts from 3 weeks up to 2 years. The end of this healing is obtaineda mature scar that has 80% strength from normal skin. [9,10,11]

As soon as the wound cavity is filled with granulation tissue and the reepithelialization process is over, this phase begins immediately.

In this phase there is contraction of the wound and collagen remodeling. Wound contraction occurs due to the activity of fibroblasts which differentiate due to the influence of the TGF-β cytokine into myofibroblasts, namely fibroblasts containing intracellular actin microfilament components. Myofibroblasts will express a-SMA (a-Smooth Muscle Action) which will make the wound contract. The intracellular matrix will undergo maturation and hyaluronic acid and fibronectin will be degraded[9,10,11]

Approximately 80% of collagen in the skin is collagen type I and 20% collagen type III which allows the tensile strength of the skin to occur. Collagen fiber diameter will increase and collagen type III in this phase is gradually replaced by type I collagen with the help of matrix metalloproteinase (MMP) which is secreted by fibroblasts, macrophages & endothelial cells. While granulation tissue expresses collagen type 3 as much as 40%.[9,10,11]

In this phase, a balance occurs between the synthesis and degradation processes of collagen and the extracellular matrix. Excess collagen is degraded by collagenase enzymes and then absorbed. The rest will shrink according to the stress. The end result of this phase is scar tissue that is pale, thin, limp, and easy to move from the base. [9,10,11]

When the levels of collagen production and degradation reach an equilibrium, then the maturation phase of wound healing begins. This phase can last up to 1 year or more, depending on the size of the wound and the method of wound closure used. During the maturation process, type III collagen which plays a large role during the proliferation phase will gradually decrease its levels, replaced by stronger type I collagen. These collagen fibers will be arranged, stringed, and trimmed along the wound line.. [9,10,11]

Risk factor of surgical site Infection

In various studies and some case reports obtained some description of risk factors that may cause the occurrence of SSI. There are three main places the risk of SSI are: Preoperative, intra operative and postoperative.[1,17]

Table 1. Risk Factor

Surgical Stage	Risk Factor
Pre operative	Gender
	Age
	Obesity
	Use of prophylactic antibiotics
	Presence of anemia
	Malnutrition
	Length of preoperative stay
	ASA Score
	Tobacco smoking
	Presence of chronic disease
	Preoperative hyperglycemia
Intra Operative	Skin and surgical site preparation
	Surgical procedure time
	Type and location of operation
Post Operative	Length of postoperative stay
	Use of prophylactic antibiotics
	Use of drains
	Blood transfusion

Pre Operative

a. Gender

The male gender has been associated with increased risk of deep SSI, male patients had multiple risk factors such as tobacco smoking and HIV and so they will be more vulnerable to have SSI for such risk factors[1,14,15]

b. Age

Aging is related to changes in function and structure that make the skin and subcutis tissue more susceptible to infection. These changes cannot be stopped but their effects can be reduced by good surgical techniques and prophylactic antibiotics. SSI levels will increase in patients aged 65 years and over.[5]

c. Obesity

Obese patients have more adipose tissue that is minimally perfused, which in turn limits oxygen delivery. In addition, adipose tissue causes extra weight that imposes additional pressure on the incision, which may decrease its blood supply.[1]

d. Use of prophylactic antibiotics

The use of aminoglycosides as a prophylactic antibiotic and giving the antibiotic one hour or more before the incision as a factor increasing the risk of SSIs in orthopedic spinal operations.

They found the suboptimal timing of administering prophylactic antibiotic (i.e. antibiotic administration more than one hour before the incision) to increase the risk of SSI following orthopedic spinal operation. However, the use of cefazolin as antibiotic prophylaxis and increasing the antibiotic dose according to obesity have been associated with reduced risk of SSIs after orthopedic spinal operations.[1,14]

The recommendation that cefazolin or cefroxamine should be used as antibiotic prophylaxis and clindamycin and vancomycin for patients with MRSA or for patients with β-lactam allergy and to administer antibiotic within one hour before skin incision. [1,14]

e. Anemia

Perioperative anemia is asociated with higher transfusion rate and post operative infection and poor recovery. [14]

f. Length of preoperative stay

As the patient's hospital stay is prolonged, the patient will be more exposed to nosocomial microorganisms that may result in orthopedic SSI. Admission to hospital within the same day of surgery is associated with a low risk of SSI.[1]

g. Malnutrition

Tobacco smoking has been found to impair tissue oxygenation and cause hypoxia via vasoconstriction, thus impairing wound healing.[1,16,17]

h. ASA Score

American Society of Anaesthesiologists (ASA) score is also associated with post-surgical infection. ASA>2 is most likely associated with post surgical patient. increased the risk of SSI after spinal decompression and fusion surgeries by 1.45 times, and an ASA score of 4 or 5 increased the risk of SSI after spinal

decompression and fusion surgeries by 1.66 times.[16]

i. Tobacco smoking

Tobacco smoking has been found to impair tissue oxygenation and cause hypoxia via vasoconstriction, thus impairing wound healing[1,16,17]

j. Hyperglycemia

There is an association between hyperglycemia and exposure to surgical intervention, regardless of if the patient is diabetic or not, which in turn impairs the wound healing process and subsequently increases the risk of SSI. elevated preoperative blood glucose level of more than 125mg/dl as a factor increasing the risk of SSIs of orthopedic spinal operations by 5.3 times.[15]

1. Intra Operative

a. Operating room traffic

Personnel change in the surgical team during the operation, presence of visitors and family members in the operating room, and a high level of noise contribute significantly to SSIs. The increased number of people in the operation theatre can increase the risk of SSIs.[1]

b. Skin and surgical site preparation

Skin preparation for surgical intervention plays a role in the incidence of orthopedic SSIs, clippers should be used instead of shaving if hair removal is necessary in order to prevent microscopic cuts in the skin that act as foci for infection.there are recommendation the use clippers instead of razors for hair removal. [15]

c. Surgical procedure time

A long surgical procedure lasting more than three hours as a risk factor for developing SSIs as the percentage of patients who developed SSI was 24%. Long surgical procedure is increased surgical procedure time increases the susceptibility of wound exposure to bacterial contamination from the environment.[18]

d. Type and location of operation

Operations for seven or more intervertebral levels as a factor increasing the risk of SSIs of orthopedic spinal operations by 3.3 times. The surgeries for adult spinal deformities such as kyphosis and scoliosis have longer surgical times compared to other spinal surgeries and are, therefore, susceptible to a higher rate of infection postoperatively. [1,18]

2. Post Operative

a. Length of postoperative stay

Prolonged hospital stay postoperatively is a predisposing factor of PJI, the longer hospital stay can increase the risk of PJI [1,17,18]

b. Use of prophylactic antibiotics

Antibiotic prophylaxis with aminglycosides and the use of antibiotic solution to irrigate surgical wounds as factors that increase the risk of SSIs after orthopedic spinal operations by 2.7 times.[1]

c. Use of drains

Use of drains was also identified as a risk factor for SSI, as seen previously.Insertion of drains provides direct access for wound contamination by hospital microflora. Drains should therefore be used vigilantly and only when absolutely required.[19]

d. Blood transfusion

Blood transfusion has been found to enhance inflammation and to suppress immunity, and immunumodilation occurs in relation to the transfused blood, thereby increasing the risk of nosocomial infection, primarily surgical site infection[1]

Surgical wound Clasification (SWC)

Since the 1960s there has been an analysis of the risk factors that contribute to the incidence of SSI. In 1964 the National Research Council introduced four categories of operating site contamination degrees which were later popularized by the American College of Surgeons. [5,6,7]

This classification system has been widely used, and although it is less common these days, the system is still applied in a number of institutions for measures to increase the quality of hospitals, third-party payers, and collaborators to improve the quality. In the operating room, the surgical wounds are described as clean, clean-contaminated, contaminated, and dirty .(table 2) The SWC is based on rough analysis by the observer and is one of three components that have equal weight in the risk model. SSI has not been studied exclusively in the orthopedic population using this classification system. SWC has demonstrated efficacy for predicting SSI in visceral tissue; however, it also has been described as effective due to lower inter-observer reliability between health care providers and institutions[5,6]

Table 2.
Surgical Wound Classification Grades (I–IV)[7] CDC Surgical Wound Classification Definitions
Class I/Clean: An uninfected operative wound in which no inflammation is encountered, and the respiratory, alimentary, genital, or uninfected urinary tract is not entered. In addition, clean wounds are primarily closed and, if necessary, drained with closed drainage. Operative incisional wounds that follow no penetrating (blunt) trauma should be included in this category if they meet the criteria.
Class II/Clean-Contaminated: An operative wound in which the respiratory, alimentary, genital, or urinary tracts are entered under controlled conditions and without unusual contamination. Specifically, operations involving the biliary tract, appendix, vagina, and oropharynx are included in this category, provided no evidence of infection or major break in a sterile technique is encountered.
Class III/Contaminated: Open, fresh, accidental wounds. In addition, operations with major breaks in a sterile technique (eg, open cardiac massage) or gross spillage from the gastrointestinal tract, and incisions in which acute or no purulent inflammation is encountered are included in this category.
Class IV/Dirty-Infected: Old traumatic wounds with retained devitalized tissue and those that involve existing clinical infection or perforated viscera. This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation.

Classification of Surgical Site Infections

SSI is differentiated into incisional SSI and spatial or organ SSI for the purposes of surveillance classification. Incisional SSI is devided to superficial incisional SSI, which involves only the skin and subcutis tissue and deep incisional SSI that reaches deep soft tissues (eg fascia and muscle). Spatial / organ SSI involves anatomical parts (organs or spatium) other than

incisions that are open or manipulated during surgery.[2,8]

Classification of Surgical Site Infections, Summarized From the CDC/NHSN Surveillance Definitions for Specific Types of Infections [2,8]

Table 3.. **Classification of Surgical Site Infections**	
SSI	Criteria
Superficial incisional SS	Infection must occur within 30 day after any operative procedure and involve only the skin and subcutaneous tissue of incision.
	The patient must also have one of the following:
	(1) Purulent drainage from superficial incision
	(2) Organisms identified from an aseptically obtained Specimen
	(3) Superficial incision that is deliberately opened by a surgeon or other designee and culture or non culture-based testing is not performed, and at least one of the following signs or symptoms: pain or tenderness; localized swelling; erythema; or heat.
	(4) Diagnosis of a superficial incisional SSI by the surgeon or an attending physician or other designee
Deep incisional SSI	Infection must occur within 30 or 90 day after the operative procedure and involve deep soft tissues of the incision (fascial and muscle layers).
	The patient must also have at least one of the following:
	(1) Purulent drainage from the deep incision
	(2) A deep incision that spontaneously dehisces or is deliberately opened or aspirated by a surgeon, attending physician, or other designee, and the organism is identified by a culture or non culture based microbiologic testing method. The patient must also have one of the following: fever, localized pain, or tenderness.
	(3) An abscess or other evidence of infection involving the deep incision that is detected on gross anatomic or histopathologic examination or imaging test
Organ/space SSI	Infection occurs within 30 or 90 day after the operative procedure and involves any part of the body deeper than the fascial/muscle layers that is opened or manipulated during the operative procedure and the patient has one of the following:
	(1) Purulent drainage from a drain that is placed into the organ/space
	(2) Organisms are identified from an aseptically obtained fluid or tissue in the organ/space by a culture or non–culture-based microbiologic testing method.
	(3) An abscess or other evidence of infection involving the

	organ/space that is detected on gross anatomic or histopathologic examination or imaging test.

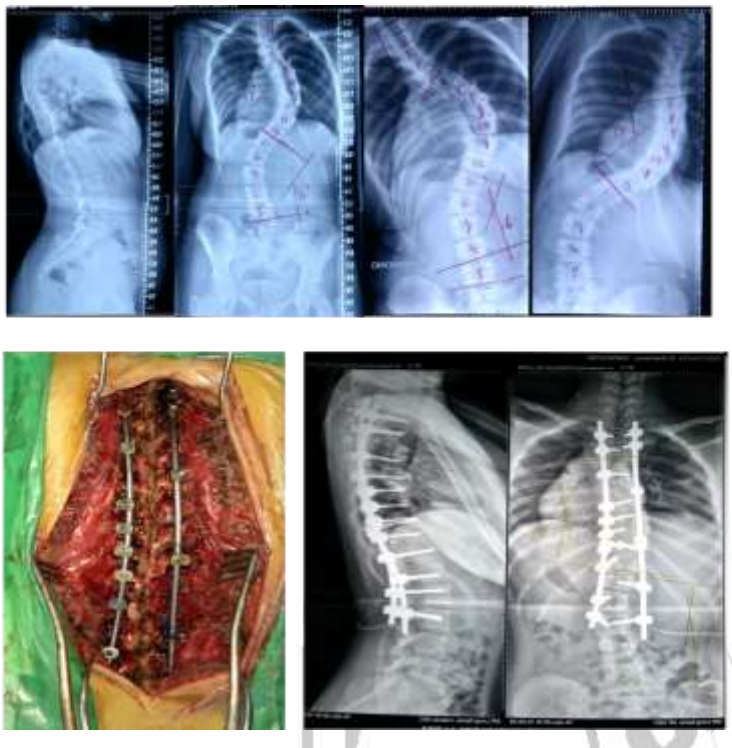

Figure 1.

A. Scoliosis Pre operative *X-ray*

B. Durante operation.

C. Post Operative

Figure 2. Deep infected wound after spinal surgery

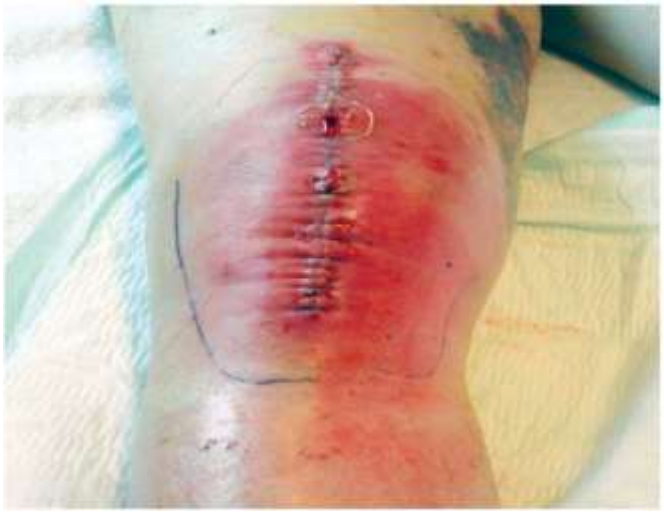

Figure 3. Superficial infected wound after knee arthoplasty [21]

Prevention Surgical Site infection

1. **MRSA Screening and Intranasal Mupirocin**

Infections caused by S. aureus represents a high percentage of all SSI. Over the last years, the increase of methicillin-resistant *S. aureus* (MRSA) infections has been observed also in orthopaedic surgery. Administering intranasal mupirocin for nasal carriers of *Staph. aureus* showed a reduction of MRSA SSI rates compared to placebo. [1,15,20]

2. **Preoperative Bathing**

Despite being an effective barrier against microbes, skin hosts many pathogens responsible for SSI. Actually, patient skin is considered the main source of microbial agents involved in orthopaedic infections. Skin colonization provides a reservoir from which bacteria can be introduced when the skin barrier is breached. Pathogens can reach the surgical site directly, during the intervention

Accurate personal hygiene of the operative staff and the patient is standard practice before any type of intervention [1,15,20]

3. **Hair Removal**

Hair removal from the location of the surgical incision has traditionally been a part of routine preoperative preparation of patients who underwent surgery with the aim of reducing the number of SSI. Hair has been linked to a lack of hygiene which can potentially lead to SSI. Hair removal can actually facilitate adequate exposure and marking of the skin before surgery, as well as suturing and applying adhesive dressings. [1,15,20]

In this case, the use of electric clippers instead of razors is highly recommended, as it cuts than shaving seems to reduce SSI possible to prevent microscopic trauma to the skin caused by the traditional razor sharp.[15,20]

4. **Glycemic control**

Hyperglycaemia, even if not related to diabetes, is associated to an increased risk of SSI. It is known that blood glucose levels rise during and after surgery due to surgical stress. Therefore, both diabetic and non-diabetic patients are at high risk for hyperglycaemia in the peri and postoperative time period, hence exposed to an increased risk of SSI.

WHO demonstrated that intensive protocols with stricter blood glucose target levels (≤ 150 mg/dL), compared to conventional protocol with higher target levels (≤ 220 mg/dL), are associated with a reduction in the number of SSI.[1,15,20]

5. **Perioperative Antibiotic Prophylaxis**

Antibiotic prophylaxis is crucial in the prevention of SSI. However, its value depends on proper administration, choice of the antibiotic and respective pharmacokinetics.the antibiotic is chosen based on patient supposed colonization and the type of pathogens commonly diffused in each surgical specialty. First and second generation cephalosporins are wide spectrum antibiotics acting mainly against aerobic gram-positive and gram-negative bacteria, with excellent bactericidal activity, good distribution in bony, synovial and muscle tissues, very low systemic toxicity and reasonable cost. [1,15,20]

The majority of orthopaedic SSIs are due to coagulase-negative staphylococci, mainly S. epidermidis, and S. aureus, which are isolated in most cases. The half-life of the antimicrobial agent to be selected should cover the time interval that is crucial for SSI (two hours after incision or contamination). First- and second-generation cephalosporins have many of these features and cephazolin, the most tested in clinical studies and using clindamycin and vancomycin for patients with MRSA or for patients with β-lactam allergy. [1,15,20]

The optimal timing to administer an antibiotic for prophylactic purpose is prior to incision. It is well demonstrated that the frst dose of frst- or second generation cephalosporins should be administered intravenously within 30-60 minutes before surgical incision. Timing depends on the specific antibiotic and its half-life; administration of vancomycin should begin within 120 minutes before incision because of the prolonged infusion times required for this molecule.[1,15,20]

6. **Surgical Site Skin Preparation**

Surgical skin preparation sites are usually performed on intact skin of patients in the operating room and includes not only the immediate site of the intended incision, but also a wider area of patient skin. This procedure is aimed at reducing the microbial load before incision of the skin barrier. The most frequently used antiseptic agents are Chlorhexidine (CHG) and Povidone Iodine (PI) in alcohol-based solutions, which have a wide spectrum of antimicrobial activity[1,15,20]

7. **Laminar Flow Ventilation Systems**

Ventilation system within the Operation Room (OR) is an extrinsic factor that can affect the SSI rate. Intraoperative wound contamination can happen directly, e.g., by contact with non-sterile devices, or indirectly by airborne microbial agents. While conventional ventilation systems pass air with a mixed or turbulent fow into the OR generating an irregular movement of aerosols and particles within the room, the goal of systems with laminar fow (LF) is to pass air unidirectionally to drive air, aerosols, and particles out of the room, thus potentially reducing SSI risk. [1,15,20]

8. **Control Traffic in OR**

Traffc in the OR, measured by number of people in the OR and number of door openings during surgery, is another extrinsic factor that may lead to an increased rate of SSI. The rationale behind limiting personnel and movement in the operating theatre is to reduce the shedding of pathogens from the skin of personnel and contamination of the air as a result of air entering from outside [1,15,20]

9. Incisional Wound Irrigation

Intraoperative wound irrigation is widely practiced at the end of surgery just before wound closure, to help reducing SSI risk. In addition to acting as a physical cleaner by removing debris, body fuids, and possible contamination, irrigation solution is believed to function as a local antibacterial agent when an antiseptic or antibiotic agent is added. [1,15,20]

10. Perioperative Oxygenation

The effect of perioperative oxygenation on the risk of SSI is well documented in the literature. This practice consists in providing patients with 80% fraction of inspired oxygen (FiO2) compared to the usual administration of 30% FiO2. Several trials have assessed the use of high FiO2 concentrations during the perioperative period and the potential association with lower rates of SSI. In fact, a high FiO2 would increase oxygen tension in blood, thus compensating a potentially not adequate perfusion of the surgical site. [1,15,20]

11. Maintaining Normal Body Temperature

Hypothermia is defned as a core temperature <36°C and is common during and after major surgical procedures lasting more than two hours. Anaesthetic induced impairment of thermoregulatory control, more than the exposure to a cold OR environment, is the main event leading to hypothermia.

Furthermore, cool intravenous and irrigation fuids directly cool patients. In fact, inadvertent hypothermia is considered to be an adverse effect of anaesthesia and is associated with adverse cardiac events. Hypothermia may increase blood loss and transfusion requirement, lengthen hospitalization, and predispose patients to the risk of SSI[1,15,20]

Conclusion

Surgical site infection (SSI) is a complication of surgery and one of major complication of surgery in orthopedic which increases morbidity and mortality to patient. Because of that, identify risk factors and causative microbial for orthopedic SSI are important to prevent infection of surgical site. Risk factor from SSI are identified and categorized to three different stages. Stages are preoperative, intraoperative and post operative. The most orthopedic SSIs are caused by a number of microorganisms infection such as *Staph. Aureus* (MRSA). Prevention of surgical site infection in orthopedics requires screening for MRSA infections with the use of intranasal mupirocin, the use of antibiotics with the first and second generation cephalosporins or clyndamisin and vancomysin if the patient alergic to β-lactam and improving protocols with various kinds of prevention against the risk of infection.

REFERENCES

1. Yahya W.Najjar,*et al*. Orthopedic Surgical Site Infection: Incidence, Predisposing factors, and Prevention. International Journal of Medical Science and Clinical Inventions 4(2): 2651-2661, 2017. DOI:10.18535/ijmsci/ v4i2.0
2. Ikemefuna O, *et al*. Surgical Wound Classification and Surgical Site Infections in the Orthopaedic Patient. JAAOS Glob Res Rev 2017;1:e022. DOI: 10.5435/ JAAOSGlobal-D-17-00022
3. Asrawal *et al*. Risk factors in surgical site infection on orthopedic surgery patients

at Fatmawati Hospital period July-October 2018. J Sains Farm Klin 6(2),104–112 (Agustus 2019). DOI : 10.25077/jsfk.6.2.104-112.2019

4. Ling ML, *et al.* APSIC guidelines for the prevention of surgical site infection. epidemiology of surgical site infection. 2019. 5-6
5. M.Alsen, Remson Sihombing. 2014. Infeksi Luka Operasi. MKS, Th. 46, No. 3, Juli 2014
6. Onyekwelu. I *et al*, Surgical Wound Classification and Surgical Site Infections in the Orthopaedic Patient. JAAOS Glob Res Rev 2017;1:e022.DOI: 10.5435/ JAAOSGlobal-D-17-00022
7. CDC, 2018, Surgical Site Infection (SSI) Event, Centers for Disease Control and Prevention, 9–9.
8. Badara GA, *et al*. Surgical Site Infection in Orthopedic Surgery at Dantec University Hospital Center. SM J Orthop. 2017; 3(4): 1062.
9. Cañedo-Dorantes, *et al*. Skin Acute Wound Healing: A Comprehensive Review. International Journal of Inflammation Volume 2019, Article ID 3706315, 15 pages https://doi.org/10.1155/2019/3706315
10. Sorg, Heiko. *Et al*. Skin Wound Healing: An Update on the Current Knowledge and Concepts. Eur Surg Res 2017;58:81–94. DOI: 10.1159/000454919
11. Primadina, Nova, *et al*. Proses Penyembuhan Luka Ditinjau Dari Aspek Mekanisme Seluler Dan Molekuler. Qanun Medika Vol. 3 No. 1 Januari 2019
12. Rajvir *et al*. Surgical Site Infections: Classification, Risk factors, Pathogenesis and Preventive Management. International Journal of Pharma Research and Health Sciences. Volume 2 (3), 2014, Page-203-214
13. I. Uckay *et al*. Prevention of surgical site infections in orthopaedic surgery and bone trauma: state-of-the-art update. Journal of Hospital Infection 84 (2013) 5e12. http://dx.doi.org/10.1016/j.jhin.2012.12.014
14. Amaradeep G *et al*. Surgical site infections in orthopedic implant surgery and its risk factors: A prospective study in teaching hospital. IJOS 2017; 3(3): 169-172. DOI: http://dx.doi.org/10.22271/ortho.2017.v3.i3c.28\
15. Tucci, G. *et al*. Prevention of surgical site infection in orthopaedic surgery: a synthesis of current recommendations. European Review for Medical and Pharmacological Sciences 2019; 23(2 Suppl.): 224-239
16. Tariq A, *et al*. (2017) A Systemic Review on Surgical Site Infections: Classifcation, Risk Factors, Treatment Complexities, Economical and Clinical Scenarios. J Bioequiv Availab 9: 336-340. doi: 10.4172/jbb.1000321
17. Florschutz, Anthony V. *Et al*. Surgical Site Infection Risk Factors and Risk Stratification. J Am Acad Orthop Surg. 2015 April ; 23(Suppl): S8–S11. doi:10.5435/JAAOS-D-14-00447
18. Mardanpour K, *et al*. Surgical site infections in orthopedic surgery: incidence and risk factors at an Iranian teaching hospital. Clin Trials Orthop Disord. 2017;2(4):132-137.
19. A. Pathak *et al*. Incidence and factors associated with surgical site infections in a teaching hospital in Ujjain, India. American Journal of Infection Control 42 (2014) e11-e15.

http://dx.doi.org/10.1016/j.ajic.2013.06.013

20. Kumar A. *et al*. Prevalence of surgical site infection in general surgery in a tertiary care centre in India. Int Surg J. 2017 Sep;4(9):3101-3106. DOI: http://dx.doi.org/10.18203/2349-2902.isj20173896

21. A. Stefánsdóttir. The infected knee arthroplast.*Thesis*. departement of orthopaedics clinical sciences Lund University Sweden;2010