Surgical Wound Infection: A General Overview

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INTRODUCTION

Infection may be defined as 'invasion and multiplication of microorganisms in body tissues, which may be clinically inapparent or result in local cellular injury because of competitive metabolism, toxins, intracellular replication, or antigen-antibody response'[1]. This series of events leads to progressive tissue destruction and eventual death of the host if left unchecked.

There is documentary evidence that the historical background of wound infection may be traced to as far back as 1st century AD when a Roman physician, Cornelius Celsus described the four principal signs of inflammation and used 'antiseptic' solutions. Claudius Galen (130-200 AD), another Roman physician had such an influence on the management of wounds that he is still thought of by many today as the 'father of surgery'. He and some of his followers instigated the 'laudable pus' theory, which incorrectly considered the development of pus in a wound as a positive part of the healing process [2]. This continued until the 16th century when Ambroise Pare "encouraged wounds to suppurate".

The 19th century witnessed the acceptance of the germ theory and introduction of antisepsis through Semmelweiss (1818-1865), Pasteur (1822-1895) and Lister (1827-1912). Mary Ayton [3], a Nursing officer, defined terminologies like wound contamination, wound colonization and wound infection, which are in current use. Vincent Falanga, in 1994 [4] identified the concept of 'critical colonisation' with fresh insights into chronic wound healing and non-healing wounds. These current terms are:

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- *Wound contamination* - the presence of bacteria within a wound without any host reaction [3]
- *Wound colonisation* - the presence of bacteria within the wound which do multi ply or initiate a host reaction [3]
- *Critical colonisation* - multiplication of bacteria causing a delay in wound heal ing, usually associated with an exacerbation of pain not previously reported but still withno overt host reaction [4, 5]
- *Wound infection* - the deposition and multiplication of bacteria in tissue with an associated host reaction [3].

In practice, it appears as if tissue viability health personnel use the term 'critical colonisation' to describe wounds that are considered to be moving from colonisation to local infection. However, the challenge is to ensure that most practitioners recognise this situation with confidence and for the bacterial bioburden to be reduced as soon as possible[6].

The presence of a microorganism within the margins of a wound does not indicate that wound infection is inevitable [7]. Some bacteria produce proteins that kill or inhibit other bacteria while in some other cases, bacteria produce a variety of metabolites that inhibit the multiplication of other micro-organisms [5]. This is called protective colonization.

The development of an infection will be influenced largely by the virulence of the organism and immunological status of the patient. Virulence describes both the pathogenicity and invasiveness of the relevant microorganism. When microorganisms are present to a degree of 10^5 per gram of tissue, an infection is likely to be present. Quantitatively, wounds harboring bacteria that exceed 10^5 colony-forming units per gram are considered infected wounds [8].

Histologically, a wound that is infected shows microorganisms in viable tissue. This produces tissue reactions evidenced in the classical features of inflammation like pain, heat, and swelling. Other features that may be exhibited are purulent discharge, unanticipated delay in healing, friable, bleeding granulation tissue despite gentle handling and the non-adhesive nature of wound management materials used. In addition, wound breakdown associated with wound pocketing/bridging at base of wound, i.e. when a wound that was assessed as healing starts to develop strips of granulation tissue in the base as opposed to a uniform spread of granulation tissue across the whole of the wound bed [6].

Skin and gut normally harbor certain bacteria and fungi in a commensal relationship with the host that serves to limit invasive, pathogenic microorganisms. When epithelial barrier is breached, the normal host response is a series of concerted, physiologic cascades that result in local inflammation [9]. Inflammation ultimately protects the host and initiates healing. However, if the initial injury is extensive, infection may develop as organisms that previously colonized skin and gut may now invade tissues. In addition, such an environment may allow secondary invaders to cause infection. These organisms or their liberated toxins may overwhelm the local protective environment with resultant systemic sepsis [10, 11]. The eventual clinical course depends, in part, on the organism involved, the host environment in which the offending organism is found, as well as the physical status of the host. This last factor may be the most critical.

Prior to the middle of the 19th century, when Ignaz Semmelweis and Joseph Lister became the pioneers of infection control by introducing antiseptic surgery, most wounds became infected [12]. In cases of deep or extensive infection this resulted in a mortality rate of 70-80% [13].

It was previously believed, at least until the late 80s, that the most important variable in determining postoperative wound infection rate was the type of operative procedure [14,15]. The traditional classification was as follows: clean (2%), clean contaminated (5%-15%), contaminated (15%-30%), and dirty or infected (30%) [10].

Patient factors that influence development of surgical site infection include the following:

(1) those undergoing abdominal operations,
(2) those whose operations last longer than 2 hours,
(3) those undergoing contaminated or dirty/ infected operations by traditional definition, and
(4) those having three or more different diagnoses.

Other systemic predisposing factors include weight loss greater than 10% of baseline, physiologic impairment of two or more organ systems [16], obesity [17], concurrent infection at a remote site, and immunocompromise [17, 18].

Local predisposing patient factors include preoperative shaving of the operative field, reduced vascular supply, disruption of lymphatic or venous drainage, and the presence of underlying inflammatory conditions like dermatitis [17, 19].

The *Surgical Wound Infection Task Force* published a consensus paper in 1992 on the definition and surveillance of surgical wound infections [20]. The term *surgical site* was used to replace *surgical wound* to clarify the specific anatomic location of deep infections after operations. This prevents confusion between the infection of a surgical incision and the infection of a traumatic wound. Surgical site infections (SSIs) are either incisional SSIs or organ SSIs. The former is further categorized by involvement of only the skin and subcutaneous tissue (superficial-incisional SSIs) or of deep soft tissue (fascia or muscle) or the incision (deep-incisional SSIs). Most SSIs are superficial. They however contribute greatly to the morbidity and mortality associated with surgery

Data from the *National Nosocomial Infections Surveillance System* reveals that the most common incisional SSI pathogens are *Staphylococcus aureus*, *Enterococcus* species, coagulase-negative *Staphylococcus*, Enterobacteriaceae, *Pseudomonas* species, and anaerobes [21]. The spectrum in the University College Hospital Ibadan appears to be similar. Specific pathogens vary from one hospital setting to another.

In individual patients, the type of operation performed and length of preoperative stay will be the strongest predictors of the species of organism isolated from a subsequent surgical site infection. In clean operations (no gastrointestinal, genitourinary, or respiratory tract violation), *Staphylococcus* species is the usual culprit; in contrast, a polymicrobial aerobic-anaerobic flora infection usually occurs in clean-contaminated cases, for example, elective colon resection [17, 18]. The longer the preoperative stay the greater the likelihood of infection from a more antibiotic-resistant organism. These points form the basis for the practice of prophylactic antibiotic administration to the surgical patient, and suggest the advantage of the current practice of Day Case Surgery.

The *US Centers for Disease Control* (CDC) definition states that only infections occurring within 30 days of surgery (or within a year in the case of implants) should be classified as SSIs. Wound infections have been subdivided according to the following clinically related subgroups [22]:

Aetiology: in a *primary infection*, the wound is the primary site of infection, whereas a *secondary infection* arises following a complication that is not directly related to the wound;

Time: an *early infection* presents within 30 days of a surgical procedure, whereas an infection is described as *intermediate* if it occurs between one and three months afterwards and *late* if it presents more than three months after surgery;

Severity : a wound infection is described as *minor* if there is discharge without cellulitis or deep tissue destruction, and *major* if the discharge of pus is associated with tissue breakdown, partial or total dehiscence of the deep fascial layers of the wound, or if systemic illness is present.

MANAGEMENT OF INCISIONAL SSI

Treatment of Organ SSI may be considered as an organ specific treatment in addition to the use of systemic antibiotic therapy. The general goal of surgery is to achieve healing down to the features of minimal or absent oedema, no serious discharge or infection, no separation of the wound edges; all with minimal scar formation [12]. Sometimes, surgical incisions are allowed to heal by delayed primary intention where debridement is performed and the wound left open. Wound edges are brought together at about 4-6 days, before granulation tissue is visible [23]. This method is often used after traumatic injury or dirty surgery.

It has been shown that a delay in wound closure of four to five days increases the tensile strength of the wound as well as resistance to infection. The overall rate of SSIs in traumatic war wounds using delayed principles was 3-4%, compared with more than 20% after primary closure [24]. In civilian practice, delayed healing has been used successfully mainly after laparotomy for infections such as may occur after typhoid perforation. Considerations however should be given in our environment to the difficulties involved in multiple theatre access.

Before the routine use of prophylactic antibiotics, infection rates were 1-2% or less for clean wounds, 6-9% for clean-contaminated wounds, 13-20% for contaminated wounds and about 40% for dirty wounds [25]. Infection rates have however reduced drastically in the most contaminated groups since introduction of routine antibiotics. There is considerable variation in each class according to the type of surgery being performed [26].

After sending a tissue or wound exudate sample for microbiologic assessment, an incisional SSI should be incised and drained. Necrotic tissue should be debrided and foreign bodies should be removed along with local wound care. Copious saline irrigation should be used to facilitate further mechanical debridement. This also achieves local dilution of the organism load. The majority of uncomplicated SSIs do not require any further therapy.

Antibiotic use is based on the clinical findings of both local and systemic involvement. The initial spectrum of coverage should include the organisms likely to be encountered from the type of operation recently performed. The coverage can be tailored when the pathogens are isolated and identified [9].

Most incisional SSIs are left open and allowed to heal by second intention. Topical antibiotics are safe on open wounds and promote wound healing possibly by providing a moist wound environ

ment[27]. The moist environment prevents desiccation and eschar formation, and encourages granulation tissue growth leading to increased reepithelialization rates. A moist environment is however best afforded by occlusive dressings (*eg*, hydrocolloids, hydrogels, foams, alginates). They should be used when available and should be stocked by Surgical Departments. Their main disadvantage is cost. They should not be applied to clinically infected wounds where a moist environment is afforded by wet-to-dry dressings, primarily in the form of a saline-soaked gauze layer covered by a dry gauze layer. This dressing requires frequent changes up to four times daily to maintain a moist wound environment. Succeeding days of this therapy will result in stabilization of the wound bacterial counts [9] so that by day 5 after operative drainage a secondary closure may be considered.

PREVENTIVE TECHNIQUES

Preventive techniques can be considered through modifications of the surgical procedure. These can be in relation to skin preparation, shaving and wound closure:

Skin Preparation: In analyses of contamination rates after cholecystectomy, the main source of wound contamination was found to be the skin of the patient [28]. Preoperative preparation of the skin should therefore be performed. It has been shown that chlorhexidine skin preparation decreases the bacterial count on skin by 80-90%, resulting in a decrease in preoperative wound contamination [29]. The effect on SSI incidence has, however, been more difficult to demonstrate and it is possible that prolonged washing releases organisms from deeper layers of the skin [12]. This problem may be reduced by the use of povidone iodine.

Povidone iodine is a polyvinyl pyrrolidone surfactant/iodine complex (PVP-I). that releases sustained low concentrations of free iodine whose exact mode of action is not known, but involves multiple cellular effects by binding to proteins, nucleotides and fatty acids. Iodine affects protein structure by oxidizing S-H bonds of cysteine and methionine, reacting with the phenolic groups of tyrosine and reacting with N-H groups in amino acids to block hydrogen bonding. It reacts with bases of nucleotides to prevent hydrogen bonding, and alters membrane structure by reacting with C=C bonds in fatty acids [30]. It has a broad spectrum of activity against bacteria, fungi, protozoa and viruses. Despite prolonged use of iodine, reports of resistance are limited to one [31]. Povidone iodine does not have to be used in sequential combination with other antiseptics. It should not be cleaned off with iodine and the effect should be allowed to last the operation.

Shaving: It is now recognised that shaving damages the skin and that the risk of infection increases with the length of time between shaving and surgery [32]. If shaving is essential, it should be performed as close to the time of surgery as possible.

Wound closure: The healing of closed surgical wounds depends on many factors, one of the most complex of which is the influence of technique and expertise [24]. In a report [33], the incidence of SSIs in relation to the different types of closure techniques used (under antibiotic cover) was as follows:

With opening and re-closure at once - 50%
Opening and re-closure after two days - 20%
Opening and re-closure after four days - 5%
Opening and re-closure after nine days - 10%

Once wounding has occurred, the surgeon has control over several factors concerning the wound itself that may reduce susceptibility to infection [12]. The duration of surgery is one factor that influences the wound infection rate. Procedures that take longer than two hours are associated with higher infection rates [34]. This may be due to the following factors: desiccation or maceration of the wound edges, an increase in the number of bacteria that accumulate within the wound, decreased temperature and hypovolaemia leading to peripheral vasoconstriction and therefore poorly perfused skin. Fewer bacteria are required to produce an infection in the presence of necrotic tissue, foreign bodies, haematomas, seromas and poor tissue perfusion [12].

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