A review study on isolation and prevalence of microbial communities on wound infection

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Abstract-Wound infection is frequently found. Infection of the wound happened due to entry of the bacteria through breached skin. These bacteria stop healing and produce sign and symptoms. From the beginning of the civilization scientist are fighting against infection. It is evident that wound infection is a challenging situation for the physicians. Multiple organisms can cause wound infection. For the treatment of infection alarge number of antibiotics are used. Both broad spectrum and narrow spectrum antibiotics are available nowadays. It is ideal to give proper antibiotic after culture and sensitivity of the wound swab, pus or infected tissue. Improper and irrational use of antibiotics and genetic and non-genetic drug resistant mechanisms of bacteria lead to drug resistance. Wound infection can be recognized by various sign symptoms. The inflammatory response is a protective mechanism that aims to neutralize and destroy any toxic agents at the site of an injury and restore tissue homeostasis. The classic signs of infection include: localized erythema, pain, heat, cellulitis and oedema. This study was aimed to assess bacterial isolates and their drug susceptibility patterns from inpatients and outpatients with pus and wound discharge. In this study we have tried to review various cross- sectional study. Wound swab samples were collected from each study participant and inoculated into appropriate media. Antimicrobial susceptibility tests were performed using disk diffusion technique following Kirby-Bauermethod.

Keywords: drug resistance, wound infection, microorganism, Wound swab, Kirby-Bauermethod.

INTRODUCTION

It is evident that wound infection is a challenging situation for the physician [1]. Wound infection is a common problem[1]. Infectionof the wound happened due to entry of the bacteria through breached skin. These bacteria stop healing and produce sign and symptoms. From the beginning of the civilization scientist are fighting against infection. Multiple organisms can cause wound infection[3]. For the treatment of infection a large number of antibiotics are used. Both broad spectrum and narrow spectrum antibiotics are available nowadays. Improper and irrational use of antibiotics and genetic and non-genetic drug resistant mechanisms of bacteria lead to drug resistance[4]. Drug resistant bacteria are the most important therapeutic challenge in the field of infectious diseases. Many of them are multi drug resistant. Among them MRSA and ESBL producing gram negative bacteria are of major concern.[2] Most wound infections can be classified into two major categories: skin and soft tissue infections, although they often overlap as a consequence of disease progression[5]. Infections of hospital-acquiredwounds are among the leading nosocomial causes of morbidity and increasing medical expense. It is ideal to give proper antibiotic after culture and sensitivity of the wound swab, pus or infected tissue. Unfortunately this practice is uncommon among the physicians especially of the developing countries. [3] A wound is a break in the integrity of the skin or tissues, which may be associated with disruption of the structure and function[6]. Another way, a wound may be defined as disruption of the normal continuity of bodily structures due to trauma, which may be penetrating or non-penetrating[7].

BACKGROUD STYUDIES

Wound infection is a major concern among healthcare practitioners, not only in terms of increased trauma to the patient but also in view of its burden on financial resources and the increasing requirement for cost-effective manage- ment within the healthcare system. Knowledge of the causative agents of wound infection has proved to be helpfulin the selection of empirical therapy, on infection control measures in health institution, and in

formulating rationales of antibiotic policy. [4] Hippocrates (Greek physician and surgeon, 460-377 BC), known as the father of medicine, used vinegar to irrigate open wounds and wrapped dressings around wounds to prevent further injury. His teachings remained unchallenged for centuries. Galen (Roman gladiatorial surgeon, 130-200 AD) was first to recognize that pus from wounds inflicted by the gladiators heralded healing (pus bonum et laudabile ["good and commendable pus"]. Unfortunately, this observation was misinterpreted, and the concept of pus preempting wound healing persevered well into the eighteenth century[5]. The link between pus formation and healing was emphasized so strongly that foreign material was introduced into wounds to promote pus formation and suppuration[8]. The concept of wound healing remained a mystery, as highlighted by the famous saying by Ambroise Pare (French military surgeon, 1510-1590), "I dressed the wound. God healed it" [6]. Koch, Professor of Hygiene and Microbiology, Berlin, 1843-1910, first recognized the cause of infective foci as secondary to microbial growth in his nineteenth century postulates. [7]. Lister recognized that antisepsis could prevent infection.In 1867, he placed carbolic acid into open fractures to sterilize the wound and prevent sepsis. In 1871, Lister began to use carbolic spray in the operating room to reduce contamination [8].

As late as the nineteenth century, aseptic surgery was not routine practice. Sterilization of instruments began in the 1880s as did the wearing of gowns, masks, and gloves. Penicillin was first used clinically in 1940by Howard Florey and with the use of antibiotics, a new era in the management of wound infections commenced. In developing countries, like Ethiopia, wound infections are major health problems [9]; large number of peopledie daily of preventable and curable wound infections [10]. These are serious problems in hospitals especially in surgicalpractices,

EPIDEMIOLOGY STUDY FOR INFECTION

In India , a study on wound infection four different types of organisms were identified. Highest percentage was *Escherichia coli* (55.9%), followed by *Pseudomonas* spp. (52.9%), *Proteus* spp. (38.2%) and *S. aureus* (17.6%). Of the 6 isolates of *S. aureus* 83.3% were MRSA[13]. In another study out of 171 cases of wounds of various etiologies examined and screened bacteriologically *S. aureus* was the most frequently isolated (39.9%) single organism and other organisms being *E. coli* (26.1%), *Pseudomonas aeruginosa* (15.4%), *Klebsiella* species (5.8%), *Streptococcus pyogenes* (4.9%), *Proteus* species (4.8%) and coliform organisms (3.1%); however, collectively the gram-negative organisms were the majority among the isolated organisms[11]. A study showed wound infection in the post-operative elective surgeries was 11.3% andthe microorganisms found were *Staphylococcus aureus* 70.5% and *Escherichia coli* 29.5% [12].

In another study showed wound infection rate 23.0% and isolated organisms were *P. aeruginosa* 29.4%, *S. aureus* 23.5%, *Acinatobacter baumani* 16.2%, *Escherichia coli* 11.8% and *A colcoaceticus* 8.8%[17]. A study has been reported that the surgical site infection rate was 3.03% in clean surgeries and 22.4% in clean-contaminated surgeries *Staphylococcus aureus* was the commonest isolate followed by *P. aeruginosa* and then *Escherichia coli*[18]. In a study showed that wound infection was 60% and the organisms isolated from wound were *Staphylococcus aureus* 50%, *E. coli* 11.7%, *Pseudomonas aeruginosa* 8.3%, *S. pyogenes* 8.3%, *Kl. pneumoniae* 6.7%, CoNS 6.7% and *Proteus* species 5%[20]. In a study with surgical wound infection a high preponderance of aerobic bacteria was observed. Among them the common pathogens were 28.2% *Staphylococcus aureus*, 25.2% *Pseudomonas aeruginosa,* 7.8% *E. coli, 7.1*% *Staphylococcus epidermidis* and 5.6% *E. faecalis*[21]. A study was done on post-operative wound infection showed infection rate 91% and the most common isolated organisms from postoperative wounds were *P. aeruginosa* 29.6%, *Escherichia coli* 20.3%, *Klebsiella* spp. 16.6%, *Staphylococcus aureus* 14.3%, *Proteus* species 6.3%, *Acinetobacter*spp. 3% and *Citrobacter* spp. 0.6% [13]. Another study showed the overall infection rate of SSIs was 8.29%; however, the infection rate in the wounds following dirty classes were 24.05% and following clean surgeries were 3.4% and common pathogens were *Staphylococcus aureus* (21.5%), *Escherichia coli* (21.5%), *Pseudomonas aeruginosa* (17.04%) and *Klebsiella pneumoniae* (14.7%)[14].

A study on "Pattern of aerobic bacteria with their

drugsusceptibility of surgical inpatients" was carried outin Mymensingh showed rate of wound infection 61.5%. The commonly isolated organisms were *Pseudomonas* spp., *Escherichia coli, Staphylococcus aureus, Klebsiella* spp. and others[14]. A study reported that 129 swabs & pus specimens from various types of surgical sites suspected to be infected on clinical ground were processed and the most common organisms were *Staphylococcus aureus* (50.32%) followed by *Pseudomonas aeruginosa* (16.3%), *Escherichia coli* (14.37%), *Klebsiella pneumoniae* (11.76%), miscellaneous gram negative rods (5.88%), and *Streptococcus pyogenes* (1.30%)[15].

In USA the overall incidence of SSI has been estimated to be 2.8% according to the U.S. Centers for Disease control and prevention[18]. Study on burn wound infection was done in Jordan showed rate of infection was 61.19% in third- degree burns and 38.80% in second degree burns[16]. *Pseudomonas* was the commonest bacterial cause ofinvasive burn wound infection followed by *Klebsiella* spp., *Staphylococcus aureus, Proteus* and *Escherichia coli*[10]. A study in Pakistan showed infection rate 9.3% and *S. aureus* was 24.4%, *Pseudomonas aeruginosa* 18.6%, *Klebsiella* spp. 13.9% and *E. coli* was in 11.6% cases[17].

Another study showed surgical site infections were 0.9% of patients undergoing clean surgery and in 3.6% of patients undergoing clean-contaminated surgery. In Romania 119 bacterial strains isolated from postoperative infected wounds. Regarding their frequency, the strains were isolated *E. coli* 68(57%) strains, [18] *Staphylococcus aureus* 37(31%)strains, *Pseudomonas* species 9(8%) strains and *Proteus* species 5(4%) strains[24]. In a study in Nigeria, 670 bacterial isolated from 29 patients were studied and the most common isolates were *Pseudomonas* spp. 29.9% and *S. aureus* 27.5%; in addition to that others were *Klebsiella* species 18.5%, *Proteus* species 15.1%, *Escherichia coli* 7%, *Streptococci 2%* and *Enterococci* 0.3%.[19]

Types of Wound infection : [20-27]

Wounds can be classified in various ways.

A.According to Rank and Wake field classification

1. Tidy wounds: They are wounds like surgical incisions and wounds caused by sharp objects.
2. Untidy wounds: They are due to: Crushing, tearing, avulsion, devitalized injury, vascular injury, multiple irregular wounds, burns etc.

B.Other classification

1. Clean incised wound
2. Lacerated wounds
3. Bruising and contusion
4. Haematoma
5. Puncture wounds and bites
6. Abrasion
7. Traction and avulsion injury
8. Crush injury
9. War wound
10. Penetrating wounds
11. Others

On the other hand surgeons and doctors have to face various surgical wounds daily. These wounds can be classified as below

- Clean (Class I): Uninfected operative wound; No acute inflammation; Closed primarily; Respiratory, gastrointestinal, biliary, and urinary tracts notentered; No break in aseptic technique Closed drainage used if necessary; Infective risk is <2%.
- Clean-contaminated (Class II): Elective entryinto respiratory, biliary, gastrointestinal, urinary tracts and with minimal spillage; No evidence of infection or major break in aseptic technique; Example: appendicectomy; Infective risk is <10%.
- Contaminated (Class III): Non-purulent inflammation present; Gross spillage from gastrointestinal tract; Penetrating traumatic wounds <4 hours; Major break in aseptic technique; Infective risk is about 20%.
- Dirty-infected (Class IV): Purulent inflammation present; Preoperative perforation of viscera; Penetratingtraumatic wounds >4 hours; Infective risk is about 40%.

Microbiological assessment on wound infection

Methicillin resistant *Staphylococcus aureus* (MRSA), popularly known as super bug, was first recognized at almost the same time that methicillin was marketed for clinical use in 1960. Subsequently large outbreak of MRSA occurred in Britain and Europe in the 1960s. In London in 1961, Jevons

reported one resistant strain in 5000 isolates. In the United Kingdom, isolates sent to the Central Public Health Laboratory increased from 3/5440 (0.06%)in 1960 to 293/7153 (4.1%) [24]. Screening in eight London teaching hospitals showed that 8.0% of isolates were methicillin-resistant. Reports of other isolates followed, including reports from Turkey and Poland even though methicillin or any other penicillinase-resistant penicillin was not yet used in these countries. The United States waslargely spared of the problem until the mid-1970s, when a number of large hospital wide outbreaks of MRSA infection occurred. The outbreaks reported in the United States in 1970s were confined primarily to large, tertiary-care teaching hospitals. [25] However in 1980s some community hospitals and rehabilitation or extended care facilities experienced an increasing prevalence of MRSA colonization or infections (Jorgensen *et al.*, 1971).In Zurich, the percentage of MRSA isolates increased from 9.7% in 1965 to 16.1% in 1967. Resistant strains were common in Denmark (46% of hospital strains in 1971). [26] In a Sydney hospital, isolates increased from 0.7% in 1965, to 5.7% in 1969 and to 18.5% by 1970. An increase in resistantstrains was also reported from France and from India. Although resistant strains had been isolated in the USA between 1960 and 1975, reports of outbreaks were rare. At the same time, other workers were reporting a decrease in the number of multiple-resistant methicillin-sensitive strains in theUSA and in England. This reduction was thought to be due to the more rational use of antibiotics and improved infection control. [27] However, the issue was complicated in the mid-1970s by the emergence of new strains of MRSA, often resistant to gentamicin,in the U.K., France, and some other countries; in the late 1970s, epidemics were reported in Ireland, Australia and the USA.

Table: List of Wound pathogens Causing Wound Infection

Gram-positive cocci	*Staphylococcus aureus; Enteroococcus faecalis; Beta Haemolytic Streptococci, (Streptococcus pyogenes)*
Gram-negative aerobic rods	*Pseudomonas aeruginosa*
Gram-negative facultative rods	*Escherichia coli, Enterobacter species, Klebsiella species Proteus species*
Anaerobes	*Bacteroides Clostridium*
Fungi	*Yeasts (Candida)*

Clinical Impact

Different terms are used for description of wound infection. Since 1985 the most commonly used terms have included wound contamination, wound colonization, wound infection and, more recently, critical colonization. These terms can be defined as:

- Wound contamination-the presence of bacteria within a wound without any host reaction[10]
- Wound colonization-the presence of bacteria within the wound which do multiply or initiate a host reaction[11]
- Critical colonization-multiplication of bacteria causing a delay in wound healing usually associated with an exacerbation of pain not previously reported but still with no overt host reaction[12].
- Wound infection-the deposition and multiplication of bacteria in tissue with an associated host reaction

Wound infection can be recognized by various sign symptoms. The inflammatory response is a protective mechanism that aims to neutralize and destroy any toxic agents at the site of an injury and restore tissue homeostasis. [28] The classic signs of infection include localized erythema, pain, heat, cellulitis and oedema and other criteria include abscess, discharge, delayed healing, discolouration of tissues within and at the wound margins, friable & bleeding granulation tissue[21]. Unexpected pain or tenderness, abnormal smell, wound breakdown wound pocketing are also seen in wound infection. The organisms that predominate ascausative agents of burn wound infection in any burn wound treatment facility change over time.Gram positive bacteria are initially prevalent and are then gradually superseded by gram negatives [29]. Burn injury is a major problem in many areas of theworld. Thermal injury destroys the physical skin barrier that normally prevents the invasion of micro-organisms. However, gram-positive in the depths of sweat glands and hair follicles may survive the heat of initial injury and unless topical antimicrobial agents are applied, these

bacteria heavily colonize the wounds within the first 48h post-injury[30].

CONCLUSION

In the conclusion it can be said that the clinical and microbiological aspect of wound infection are very wide range. The current findings showed that the rates of isolation of Gram-negative and Gram-positive were 56.6% and 43.4%, respectively. This was in agreement with studies done in Zaria, Nigeria, 55% and 44%, respectively [32]. The present findings show higher rates of isolation of Gram- negative wound pathogens from the same area. This high rateof Gram-negative and low rate of Gram-positive isolates from wound in the same area may be due to high number of cases included from inpatients in the present study compared to outpatients. This may probably contribute high number of Gram-negatives than Gram-positives. Multiple bacteria as well as other organism cause different wound infection. Proper wound management should be implemented to combat this problem. Alarmingly high rate of MDR to commonly used antibi- otics from wound infection were reported. Continuous surveillance is necessary to guide appropriate therapy for wound infection and rational use of antimicrobial agents should be sought to prevent the emergence of MDR pathogens.

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