Isolation and Identification of Bacterial Burn Wound Infection and Their Sensitivity to Antibiotics

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ABSTRACT The study aimed to determine the bacteriological profile and the resistance pattern of burn wound bacteria to the most common antibiotics. A total of 100 burn wound swabs were collected from burn patients from 4 hospitals in Baghdad province. After the swabs had been cultured on different media, conventional biochemical tests to identify bacterial isolates and antimicrobial sensitivity to the most common antibiotics were performed.

The total positive swabs for bacterial isolation was 89, of which, *Pseudomonas aeruginosa* was the most commonest pathogen [31.46%] followed by *Klebsiella spp.* [22.47%], *Staphylococcus aureus* [20.22%], *Escherichia coli* [15.73%] and *Proteus spp.* [10.11%]. Most isolates showed high resistance to cephalothin and trimethoprim, and high susceptibility to nalidixic acid and amikacin. Despite the rapid improvement in medical care, wound burn infection still represent a serious problem for burn patients, with many bacteria developed different degrees of resistance to most known antibiotics.

**Key words**: burn wounds, *Pseudomonas, Staphylococcus, Klebsiella*, antibiotics, susceptibility

INTRODUCTION

Burns are one of the most common and devastating forms of trauma [1]. Despite advances in the use of topical and parental antimicrobial therapy, and the practice of early tangential excision, bacterial infection remains a major problem in the management of burn victims[2]. In patients with severe burns over more than 40% of the total body surface area (TBSA), 75% of all deaths are currently related to sepsis from burn wound infection or other infection complications and/or inhalation injury[3,4,5,6]. The rate of nosocomial infections are higher in burn patients due to various factors like nature of the burn injury itself, invasive diagnostic and therapeutic procedures, and prolonged intensive...
care unit stay [7]. In addition, cross-infection results between different burn patients due to overcrowding in burn wards [8]. Although burn wound surfaces are sterile immediately following thermal injury, these wounds eventually become colonized with microorganisms [9]. Microorganisms colonizing burn wounds originate from the patient’s endogenous skin and gastrointestinal and respiratory flora [10,11]. Some microorganisms may also be transferred to a patient’s skin surface via contact with contaminated external environmental surfaces, water, fomite, air, and the solid hands of health care workers [12]. *Streptococcus pyogenes* was the most frequently recognized cause of burn wound sepsis in the early part of the last century. Over the years, however, *S. aureus* and *P. aeruginosa* have become the most frequent isolated organism in most burn units [13,14]. Emerging antimicrobial resistance trends in burn wound bacterial pathogens represent a serious therapeutic challenge for clinicians caring for burn patients [10,15]. Keeping in mind that infective agents and their susceptibility to antibiotic vary from time to time, it is desirable to carry out periodic reviews of the bacterial flora of the burn wounds. The aim of this study was to assess the current bacterial profile of burn wounds and their antibiogram in some Baghdad hospitals.

**MATERIALS AND METHODS**

A total of 100 burn patients [52 males and 48 females, mean age 21.8±12.07 years, range 4 months to 70 years] admitted to burn unit in four hospitals: Al-Kindy teaching hospital, Al-Karkh general hospital, Al-Yarmouk teaching hospital, and Al-Karama hospital/Baghdad [25 patients from each hospital] were used for this study during the period from October 2003 to January 2004. Mean total surface burned area was 15% [range 12-83%]. Wound swabs were taken from each patient. At direct patient contact, a protective gown and disposable gloves were used. The specimens were transported in sterile, leak-proof container to the laboratory. All specimens were inoculated on 5% blood agar and MacConkey agar plate and incubated overnight at 37°C aerobically. Bacterial pathogens were identified by conventional biochemical methods according to the standard microbiological technique [16].

Antimicrobial susceptibility was performed on Mueller-Hinton agar by the standard disk diffusion method recommended by the Clinical and Laboratory Standard Institute [CLSI] [17]. The antibiotics tested were: amikacin, ampicillin, cefotaxim, cephalothin, Chloramphenicol, ciprofloxacin, erythromycin, gentamicin, naladixic acid, tobramycin, and trimethoprim. Antibiogram of bacterial isolates were done according to Kirby Bauer method [18].
RESULTS AND DISCUSSION

Bacterial isolates were found in 89 [89%] samples and only 11 wound swabs were negative for bacterial culture. *P. aeruginosa* were the commonest pathogen isolates [31.46%] followed by *Klebsiella spp.* [22.47%], *S. aureus* [20.22%], *E. coli* [15.73%], and *Proteus spp.* (10.11%) (Table 1). Thermal destruction of the skin barrier and concomitant depression of local and systemic host cellular and humoral immune responses are pivotal factors contributing to infectious complications in patients with severe burns [19].

The results showed that the most common bacteria in burn wounds is *P. aeruginosa*. This finding is in accordance with previous studies which considered this bacteria as the commonest cause of burn wound infection[14,20,21]. However, other reports revealed that *Staph. aureus* was the most prevalent single organism colonizing burn wounds [22,23,24]. *P. aeruginosa* is part of a large group of free-living bacteria and has the ability to resist the effect of many disinfectants. The source of infection with this bacteria is from the patient’s endogenous gastrointestinal flora and/or an environment. Despite burn wound infection with this bacteria can take place anywhere, nosocomial infection is more frequent than any other places for many reasons, among which contamination, presence of multidrug resistant bacteria and crowding. Since as early as 1967, it was found that disinfectants used in many hospitals had contamination with *P. aeruginosa* [25]. Furthermore, certain subjects in hospitals such as sink-traps, floor, cloths, and mops were frequently contaminated with this bacteria. On the other hand, when similar areas were examined in domestic homes, *P. aeruginosa* was rarely isolated [26].

Within the infected wound, *P. aeruginosa* produces a number of cell-associated (adhesins, alginate, pili, flagella, and lipopolysaccharide) and extracellular (elastase, exoenzyme S, exotoxin A, hemolysins, Iron-binding proteins, leukocidins, and proteases) virulence factors that mediate a number of processes, including adhesion, nutrient acquisition, immune system evasion, leukocyte killing, tissue destruction, and bloodstream invasion [27]. These factors enable this bacteria to survive in burn wounds and transmit from patient to other within the burn unit. *Klebsiella spp* was found as one of major pathogen that infect burn wound [14,28]. The virulence factors the bacteria possessed enable it to invade the wound easily and resist the immune response of the body. The most important virulence factors are cell wall receptors, capsular polysaccharide, and endotoxin. First, the presence of cell wall receptors enables *Klebsiella* to attach to the host cell, thereby altering the bacterial surface so that phagocytosis by polymorphonuclear leukocytes and
macrophages is impaired and invasion of the non-phagocytic host cell is facilitated. Second, invasion of the host cell is also facilitated by the large polysaccharide capsule surrounding the bacterial cell; in addition this capsule acts as a barrier and protects the bacteria from phagocytosis. Third, this bacteria produces an endotoxin that appears to be independent of factors that determine receptors and capsular characteristics[29]. An eighteen isolates of *Staphylococcus aureus* which represents 22.2% of all isolates were found to be coagulase positive. Generally, *Staph. aureus* have a diverse array of virulence factors that facilitate adherence to host tissue, immune system evasion, and destruction of host cell and tissue, the most important of which is coagulase and hemolysin[30]. Burn wound infection with gram negative bacteria represented 77.52%, while gram positive bacteria accounts for only 22.47%. These results are in accordance with many previous studies(14,28). Prior to the discovery of antibiotic, gram positive bacteria (especially streptococci and *Staph. aureus*) was the most frequent cause of life-threatening burns. The use of broad-spectrum antibiotics led to the emergence of gram negative bacteria as a predominant organism causing invasive burn wound infections [31]. It is because this fluctuation in bacterial profile, a periodic assessment of burn wound infection must be assessed. Table- 2 shows the antimicrobial susceptibility pattern of the isolates. The study revealed that all isolates had resistance to trimethoprim and cephalothion [100%]. *P. aeruginosa* was unsusceptible to almost all antimicrobials. Among other gram negative bacilli, susceptibility percentage varied from 0% to cefotaxime, ampicillin to 100% to amikacin, chloramphenicol and nalidixic acid. However, *E. coli* showed high resistance to chloramphenicol, and moderate susceptibility to nalidixic acid (55.56%). *S. aureus* isolates were highly susceptible to amikacin and gentamicin (77.78% for both) and but not to cefotaxime [22.2%]. Risk factors for acquisition of an antibiotic-resistant organism include receipt of antibiotics prior to the development of infection, extended duration of hospitalization, previous hospitalization, invasive procedures, comatose state, and advancing age[32]. Microorganisms transmitted from the hospital environment tend to be more resistant to antimicrobial agents than those originating from patient’s normal flora[33]. Multidrug-resistant bacteria have frequently been reported as a cause of nosocomial outbreaks of infection in burn units or as colonizers of the wounds of burn patients [14] Based on National Nosocomial Infection Surveillance System (NNIS) criteria, all the burn patients are required to follow the distribution of bacterial species among burn isolates. The high percentage of multidrug resistant isolates is probably due to excessive and indiscriminate use of broad-spectrum antibiotics
These multi drug resistant strain establish themselves in the hospital environment in areas like sinks, taps, railing, mattress, toilets and thereby spread from patient to another. This is especially obvious in *P. aeruginosa* which showed high resistance to the all used antibiotics and this is attributable to a concerted action of multidrug efflux pumps with chromosomally-encoded antibiotic resistance genes (e.g. *mexAB-oprM, mexXY*) and the low permeability of the bacterial cellular envelopes. Besides intrinsic resistance, *P. aeruginosa* easily develop acquired resistance either by mutation in chromosomally-encoded genes, or by the horizontal gene transfer of antibiotic resistance determinants [34]. Therefore, routine microbiological surveillance and careful in vitro testing prior to antibiotic use may help in the prevention and treatment of multidrug resistant pathogen in burn infection.

<table>
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<tr>
<th>Table-1: Types of bacteria isolated from burn wounds</th>
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<tbody>
<tr>
<td><strong>Type of bacteria</strong></td>
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<tr>
<td><em>Pseudomonas aeruginosa</em></td>
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<tr>
<td><em>Klebsiella spp.</em></td>
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<tr>
<td><em>Staphylococcus aureus</em></td>
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<td><em>E. coli</em></td>
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<tr>
<td><em>Proteus spp.</em></td>
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<td><strong>Total</strong></td>
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<th>Table-2: Antimicrobial susceptibility pattern(%) of the isolates</th>
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<td><strong>Antibiotic</strong></td>
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<tr>
<td>P. aeruginosa</td>
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<tr>
<td><em>Ampicillin (AMP)</em></td>
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<tr>
<td><em>Cefotaxime (CTX)</em></td>
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<td><em>Cephalothin (KF)</em></td>
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<td><em>Chloramphenicol(C)</em></td>
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<td><em>Ciprofloxacin (CIP)</em></td>
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<td><em>Erythromycin (E)</em></td>
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<td><em>Gentamycin (G)</em></td>
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<td><em>Nalidixic acid (NA)</em></td>
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<td><em>Tobramycin (TM)</em></td>
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<td><em>Trimethoprim (W)</em></td>
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ND : not done

**REFERENCES**


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