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Prevalence and Antimicrobial-Resistant Patterns of *Pseudomonas aruginosa* among Burn Patients

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Abstract

Nosocomial infections are a significant global health issue, posing serious public health challenges in hospitals worldwide. The rise of drug-resistant bacteria causing these infections is increasingly problematic, exacerbated by inadequate hygiene practices and routine antimicrobial use, which contribute to heightened antimicrobial resistance. Pseudomonas aeruginosa is common causes of nosocomial infections. Furthermore, antimicrobial resistance is frequently observed in these pathogens, posing significant challenges to treatment. Pseudomonas aeruginosa is one of the commonest organisms causing different infections like wound infections, Lower Respiratory Tract Infection, Urinary tract infection, infections in burn patient in hospital setting. The increasing trend of antibiotic resistance in Pseudomonas aeruginosa poses a challenge to their empiric treatment with conventional agents. So, the objective of this study was to determine the prevalence and antimicrobial resistance pattern of Pseudomonas aeruginosa isolated from different clinical samples.

Keywords: Antimicrobial-Resistant, *Pseudomonas aruginosa*, Burn Patients

Introduction

Burns are one of the most common and devastating forms of trauma, often caused by heat, radiation, electricity or contact with chemicals. Burns remove the protective layer of the skin, disrupting the natural skin barrier and suppressing immune responses, and as a result the body is generally exposed to a variety of potential pathogens. Microbial infections after burns, where a large portion of the skin is damaged, are a very serious complication and often the main cause of death in patients [1, 2]. Infection in burn wounds is the leading cause of disability and death affecting all ages in both developed and developing countries [3]. The surfaces of burn wounds contain a large amount of dead tissue and a protein-rich environment that provides a suitable place for microbial colonization and multiplication [4]. Although the burn wound area is sterile immediately after thermal/burn injury, a complex and variable microbial environment

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develops rapidly thereafter within an average of 5 to 7 days [5]. Pseudomonas aeruginosa is a well-known opportunistic pathogen that causes serious infections and complications in burn patients worldwide, accounting for approximately 45% of deaths among these patients [6, 7]. The presence of necrotic and deformed tissues and a moist environment make the burn wound susceptible to infection by Pseudomonas aeruginosa [8]. In addition, the breach in the protective skin barrier, decreased immunity, and prolonged hospital stay are important factors responsible for the infection of burn wounds with such opportunistic pathogens, especially with multidrug-resistant Pseudomonas aeruginosa [9]. Treatment of infections caused by Pseudomonas aeruginosa is difficult and challenging because this organism has a natural sensitivity to a very limited number of antimicrobial agents and often requires combination therapy because high rates of antibiotic resistance are associated with Pseudomonas aeruginosa strains. In addition, the genetic changes and adaptive behavior of these bacteria within biofilms make them resistant to all known antimicrobial agents, making Pseudomonas aeruginosa infections more complex and life-threatening [10, 11].

There are several resistance mechanisms implicated in Pseudomonas aeruginosa strains; they exhibit high intrinsic resistance to a wide range of antibiotics (including aminoglycosides, fluoroquinolones, and beta-lactams), acquired resistance, and adaptive resistance [12]. The only major mechanism is resistance to carbapenems, which are widely used as the most important drugs for the treatment of Pseudomonas aeruginosa-associated infections. However, resistance to these compounds has also become a growing therapeutic problem. They are also resistant to all or nearly all antibiotics from the alpha-lactam group, aminoglycosides, and quinolones: cefepime, ceftazidime, imipenem, meropenem, piperacillin-tazobactam, ciprofloxacin, and levofloxacin. There is usually a combination of resistance mechanisms. The World Health Organization has classified carbapenem-resistant Pseudomonas aeruginosa as a critical target (priority 1) for which new treatments are being developed.[13-14]

Antimicrobial agents are required. Unfortunately, with the increasing use of broad-spectrum antibiotics, the incidence of multidrug-resistant Pseudomonas aeruginosa is increasing, and clinical treatment of these infections is becoming more challenging [13–16]. Pseudomonas aeruginosa is particularly susceptible to genetic changes that lead to antibiotic resistance and the resulting difficulties in immunocompromised individuals. The ability of Pseudomonas aeruginosa to use high levels of intrinsic and acquired resistance mechanisms to resist most antibiotics has made it increasingly difficult to eradicate and therefore there is a growing demand for the development of new alternatives to combat multidrug-resistant pathogens.[12,15]. These bacteria can simply develop resistance to all conventional antimicrobials against Pseudomonas aeruginosa through unique intrinsic and acquired resistance mechanisms. These bacteria often develop multiple resistant strains, which pose a serious threat to public health due to their limited treatment and lead to morbidity and mortality [17]. Furthermore, P. aeruginosa spreads from patient to patient and persists in patients throughout multiple antibiotic courses, which have been given to treat P. aeruginosa and non-P. aeruginosa infections [10, 18]. Therefore, antibiotic resistance of P. aeruginosa is currently a growing problem worldwide and raises serious concerns [18, 19]. Objectives of the study: This study was conducted to determine the pattern of antimicrobial resistance and prevalence of multidrug-resistant Pseudomonas aeruginosa infection among burn patients at Al-Hussein Teaching Hospital.

Burns are a serious public health problem worldwide. Pathogen colonization of burn wounds and invasion of the gastrointestinal tract can lead to severe complications and death, accounting for an estimated 180,000 deaths annually[20]. Infections following nonfatal burn injuries are a major cause of morbidity and mortality. Burn victims admitted to the hospital are also susceptible to infection. Hospital-acquired

infections have been a major contributor to the increased fatal outcomes[21]. Because thermal injury results in loss of the skin's protective barrier against microbial entry and a concomitant state of immune dysregulation, the burn wound surface provides a protein-rich environment conducive to the colonization and growth of endogenous and exogenous microorganisms [22]. Burn wound infections have long been a major challenge in burn care [23].

The most common pathogens responsible for burn wound infections are Staphylococcus aureus and Pseudomonas aeruginosa[24]. Burn departments have been reported to harbor multidrug-resistant strains of Pseudomonas aeruginosa that can colonize burn wounds and lead to infections [25]. This pathogen has been reported as the most common source of burn wound infections in the United States [26]. Pseudomonas aeruginosa is also the most common isolate among burn patients in Iraq [27]. This high prevalence of infection and the fact that the pathogen is resistant to many commonly used antibiotics make it imperative to monitor this infection in burn departments.

Pseudomonas aeruginosa (P.) is one of the most prevalent Gram-negative pathogens isolated from infected burn wounds, with a large reservoir of virulence factors and antimicrobial resistance traits [28]. Pseudomonas aeruginosa has evolved in parallel with the development of treatment options, and increasing antimicrobial resistance poses a threat to the lives of burn patients and is associated with a greater global burden on healthcare [29]. Burn victims are also at risk of infection with Pseudomonas aeruginosa bacteria.

Pseudomonas aeruginosa

Pseudomonas aeruginosa is a gram-negative rod measuring 0.5 to 0.8 µm by 1.5 to 3.0 µm. Almost all strains are motile by means of a single polar fla-gellum. It is a free-living bacterium, commonly found in soil and water. This bacterium, a member of the gamma proteobacteria, is a gram-negative, aerobic rod belonging to the bacterial family pseu-domonadaceae. Based on conserved macromole-1 cules (e.g. 16S ribosomal RNA), the family includes only members of the genus Pseudomonas which are cleaved into eight groups. P. aeruginosa is a typical species of its group which contains 12 other mem-bers. Almost all the clinical cases of P. aeruginosa infection can be associated with the compromise of host defense such as burn patients. While many cases of P. aeruginosa infection can be attributed to general immunosuppression (e.g. AIDS patients), 3.4 in neutropenic patients undergoing chemotherapy, such scenarios predispose the host to a variety of bacterial and fungal infections, and therefore do not yield information which is specific to the pathogene-sis of P. aeruginosa. In this respect, three of the more informative human diseases caused by P. aeruginosa are: 1) bacteremia in severe burn vic-tims; 2) chronic lung infection in cystic fibrosis pa-tients; and 3) acute ulcerative keratitis in users of extended-wear soft contact lenses. Observations and experimental evaluation of various bacterial viru-lence factors have shed a great deal of light on how P. aeruginosa is able to cause disease in a wide va-riety of organs, secondary to disruption of the nor-mal physiologic function.[30-33]

***P. aeruginosa* Bacteremia in Severe Burn Victims**

Bacterial infection following severe thermal injury can be most simplistically attributed to extensive breaches in the skin barrier. The fact that P. aerugi-nosa occurs so commonly in the environment makes it extremely likely that an individual suffering severe burns will be challenged with this microorganism be-fore the burns can heal. Burn hospitals often harbor multidrug-resistant P. aeruginosa that can serve as the source of infection.[34] P. aeruginosa has been found to contaminate the floors, bed rails, and sinks of hos-pitals, and has also been cultured from the hands of nurses. Besides transmission through fomites and vectors, bacterial flora can be carried into a hospital by the patient and can be an important source of in-fection for the same individual after injury." Regard-ing multidrug resistance, Hsueh et al. reported single multidrug-resistant strain of P. aeruginosa over a pe-riod of several years, and concluded that the strain was

carried by some patients asymptomatically through several rounds of antibiotic treatment which were administered to treat Pseudomonas and non-Pseudomonas infections.[35] This scenario can be worse during the spread of P. aeruginosa from one patient to another; the persistence of this strain takes place in patients throughout several courses of antibiotic treatment. It has been proved that during admission of patients in burn centers, a limited number of common strains cross-contaminate burn victims mostly when their lesions scrubbed in the bathroom.[36].

P. aeruginosa Virulence Factors in Burn Infection

Numerous P. aeruginosa virulence factors contribute to the pathogenesis of burn wound infection. Rahme et al. highlighted the occurrence of virulence factors of P. aeruginosa contributing to pathogenesis in burn wound infection of rodents. [37]A significant role has also been established for P. aeruginosa pili and fla-gella. Experiments comparing infection of burn wounds by pilus and flagellum deficient P. aerugi-nosa strains clearly demonstrate that the bacteria de-ficient in either of these structures have reduced viru-lence, both in their ability to persist at the wound site, and in their ability to disseminate throughout the host organism." Dissemination of P. aeruginosa through-out the host is also partially dependent upon produc-tion of bacterial elastase and other proteases. [38] Elas-tase has been shown to degrade collagen and non-collagen host proteins, and to disrupt the integrity of the host basement membrane. [39]Proteases can have adverse effects on several aspects of the innate and acquired host immune response. For example, elas-tase inhibits monocyte chemotaxis, which could ad-versely affect early clearance of P. aeruginosa from wound sites by phagocytosis, as well as subsequent presentation of bacterial antigens to the host immune system. The lasR gene encodes a protein critical for initiation of the quorum sensing response involved in virulence factor production and biofilm formation, indicating that other factors controlled by lasR are critical determinants of P. aeruginosa pathogenesis in burn wound infection.[40] Other P. aeruginosa viru-lence factors reported to be involved in pathogenesis of burn wound infection include phospholipase C,[41] the ferripyochelin-binding protein, 18 lipopolysaccha-ride (LPS), and exoproducts secreted by type III se-cretion apparatus. While the loss of the skin's barrier function is certainly an important factor in burn wound infection, its compromise fails to explain the relatively narrow range of bacterial pathogens which are typically cultured from infected burn wounds. It is, therefore, likely that additional host defense mechanisms specific to some pathogens are more compromised in severe burns. A reduction in infec-tion following local application of polyclonal human antibody to burn sites has been reported, [42] suggesting that in the untreated burn wound, immunoglobulin exists at subprotective levels. The possibility of a lo-cal deficiency of antibody-mediated immunity in burn wounds is further supported by an earlier report stat-ing that Fc receptor expression by polymorphonuclear leukocytes (PMNs) decreases by the fifth day post-injury in burn victims. Complement has also been shown to be depleted in burn wounds, [43] probably due to local consumption of complement components. Local deficiencies in protective antibody complement proteins, and PMN Fc receptors may explain the de-fects in random migration and chemotaxis of PMNs observed at burn wound sites. Taken together, these data suggest that the ability to colonize a burn wound depends upon the concerted impairment of several host immune mechanisms, and that the importance of P. aeruginosa in such infections is due to its ability to take advantage of the host immune compromise and secrete a variety of important virulence factors. P. aeruginosa produces two extracellular protein tox-ins, exoenzyme S and exotoxin A. Exoenzyme S hasthe characteristic subunit structure of the A-component of a bacterial toxin, and it has ADP-ribosylating activities.[44] Exoenzyme S is produced by the bacteria growing in the burned tissue and may be detected in the blood before the bacteria are present.[45] It has led to the suggestion that exoenzyme S may act to

impair the function of phagocytic cells in the blood-stream and internal organs as a preparation for invasion by P. aeruginosa. [46] Exotoxin A has exactly the same mechanism of action as the diphtheria toxin; it causes the ADP ribosylation of eucaryotic elongation factor 2, resulting in inhibition of protein synthesis in the af-fected cell.[47] Although it is partially-identical to diphthe-ria toxin, it is antigenically distinct. It utilizes a different receptor on host cells than diphtheria toxin does; oth-erwise, it enters the cells in the same manner and has the exact enzymatic mechanism. The production of exotoxin A is regulated by exogenous iron, but the de-tails of the regulatory process are distinctly different in C. diphtheriae and P. aeruginosa. Exotoxin A appears to mediate both local and systemic disease processes caused by P. aeruginosa. It has necrotizing activity at the site of bacterial colonization and is therefore thought to contribute to the colonization process.[48]

Candidate Vaccines for High-Risk People

Although antibiotic therapy has considerably improved the management of infectious diseases in general, many P. aeruginosa infections are not fully treated or eradicated by the application of anti-pseudomonal drugs and can, thus, become chronic infections. For instance, burn patients that survive the initial burn trauma can become colonized with antibiotic-resistant, hospital-derived P. aeruginosa strains that are not eas-ily eradicated with antibiotic therapy. [49] In cystic fi-brosis patients, when the strains are eventually selected out by antibiotic therapy to become multiply-resistant, an increase in the rate of decline in lung function is seen when compared to patients infected with antibi-otic susceptible strains. [50] Several P. aeruginosa anti-gens are used for vaccine development including lipopolysaccharide alone, polysaccharides alginate, extracellular proteins, exotoxin A, and killed whole cell. [51] However, none of them are clinically available to use for people who are at risk such as firefighters or infected patients (Immunocompromised and cystic fi-brosis patients).

Treatment of Infections

Topical antimicrobial therapy

It has been proved that an effective topical antim-icrobial agent substantially reduces the microbial load on the open burn wound surface and reduces the risk of infection. [52] Selection of topical antimicrobial therapy should be based on the agent's ability to in-hibit the microorganisms recovered from burn wound surveillance cultures and monitoring of the nosoco-mial infections acquired in the burn unit. Prescription is also based on the individual preparation of the topi-cal agent (e.g., ointment or cream versus solution or dressing) and its pharmacokinetic properties. Burn units may rotate the use of various topical antimicro-bial preparations on a regular basis to decrease the potential for development of antibiotic resistance. [53] Topical antibiotic agents should first be applied di-rectly to the patient's dressings before application to the burn wound to prevent contamination of the agent's container by burn wound flora. The inhibitory action of silver is due to its strong interaction with thiol groups present in the respiratory enzymes in the bacterial cell. [54] Silver has also been shown to inter-act with structural proteins and preferentially bind with DNA nucleic acid bases to inhibit replica-tion. 48.46 For this reason, silver has recently been shown to be highly toxic to keratinocytes and fibro-blasts and may delay burn wound healing if applied indiscriminately to debrided healing tissue areas. Moist exposure therapy, using a moisture-retentive, has been shown to act as an effective antibacterial agent while promoting rapid autolysis debridement and optimal moist wound healing in partial-thickness injury. [55] Moisture-retentive ointment also resulted in earlier recovery of keratinocytes with improved wound healing and decreased scar formation. Silver nitrate is most effective before the burn wound be-comes colonized. The burn wound needs to be cleansed of emollients and other debris before a mul-tilayered dressing is applied to the burn wound and subsequently saturated with silver nitrate solution. Effective use of this preparation, therefore, requires continuous

application with secondary occlusive dressings, making examination of the wound difficult.[56]

Mafenide Acetate

Topical mafenide acetate cream allows open burn wound therapy and regular examination of the burn wound surface because it is used without dressings. Mafenide acetate is applied a minimum of twice daily and has been shown to penetrate the burn eschar.4 The 5% solution must be applied to saturate gauze dressings, and these need to be changed every 8 hours for maximal effect. Mafenide acetate solution appears to be as effective as the cream preparation when used in this way. [57] Mafenide acetate (Sulfamylon) cream has a broad spectrum of activity against gram-negative bacteria, particularly P. aeruginosa, but it has little activity against gram-positive aerobic bacte-ria such as Staphylococcus aureus.[58] This agent also inhibits anaerobes such as Clostridium spp. Because protracted use of mafenide acetate favors the over-growth of C. albicans and other fungi, this agent should be used in combination with nystatin to pre-vent this complication due to its limited antifungal activity. [59] This compound is converted to p-sulfamylvanzoic acid by monoamide oxidase, a car-bonic anhydrase inhibitor, causing metabolic acidosis in the burn patient.[60]

Acticoat AB Dressing

This product is a specialized dressing, consisting of two sheets of high-density polyethylene mesh coated with nanocrystalline silver (e.g., ionic silver with a rayon-polyester core). [61] The more controlled and prolonged release of nanocrystalline silver to the burn wound area allows less-frequent dressing changes, reducing the risk of tissue damage, nosocomial infec-tion, patient discomfort, and the overall cost of topi-cal therapy. [62] Acticoat AB provides the most com-prehensive broad-spectrum bactericidal coverage against important burn wound pathogens of any topi-cal antimicrobial preparation currently marketed. [63] These dressings have a potent antibacterial activity against most aerobic gram negatives, including P. aeruginosa .

Resistance to Antimicrobial Agents

Resistance to topical antimicrobial agents

Although resistance to silver sulfadiazine in P. aeruginosa was reported, its resistance mechanism has not been determined. [64] It is suggested that resis-tance of Pseudomonas to silver based topical antim-icrobials in part is based on the mutation of outer membrane proteins that transport ions including silver across bacterial membrane [65] Gentamicin-resistant strains of P. aeruginosa which were isolated from burned patients have been reported.[66] These stains showed cross-resistance to silver sulfadiazine but their resistance was unstable and did not persist on subculture media. According to a report in USA, an epidemic sepsis of Enterobacter cloacae in burned patients occurred and resulted into 13 deaths.The MIC values of silver sulfadiazine for these strains were 3200 µg/ml whilst the strains isolated from non-burned patients were all sensitive to silver sulfadiaz-ine. Similarly, Rosenkranz et al. isolated two silversulfadiazine resistant strains of Entrobacter cloace in a burn unit where silver sulfadiazine was in use.[67]

Figure 1: Kirby-Bauer disc diffusion method for antimicrobial susceptibility testing of Pseudomonas aeruginosa isolates

Conclusion

P. aeruginosa infections identify those of a pathogen with many potentially virulent factors that allow it to colonize and infect essentially any mammalian tissue. The organism possesses a multitude of factors that promote adherence to host cells and mucins, damage host tissue, elicit inflammation and disrupt defense mechanisms. Due to impairment of the skin barrier in burn patients and frequent scrubbing, debridement and manipulation of the burn site, cross-contamination of MRD strains of Pseudomonas and colonizing of MDR strains is more likely. In spite of the ubiquitous nature of this microorganism and the frequency with which it is encountered, most human hosts counteract the infectious process effectively via the innate immune system. A more detailed molecular and cellular understanding of the bacterial and host factors is crucial to an overall comprehension of the pathogenic process of Pseudomonas, and will be of increasing importance to the development of preven-tative strategies to be sought for this major human pathogen. Selection of multi-drug resistant Pseudo-monas in burn centers can be facilitated through transmission from person to person as well as exten-sive applications of antipseudomonal antibiotics. To overcome inappropriate treatment of burn patients infected with P. aeruginosa, periodical antibacterial susceptibility surveys for the bacteria isolated from burn patient are warranted. The prevalence of P. aeruginosa infections in burn patients was found to be 54.9%, and the prevalence of multi-drug resistance among these isolates was 76.8. Such a high prevalence of MDR P. aeruginosa infection in burn patients is a cause for concern because it poses a serious therapeutic challenge due to very limited treatment options. This situation warrants the implementation of an efficient infection control program and regular surveillance of antimicrobial resistance in P. aeruginosa isolates in order to establish a rational antibiotic policy for the better management of such infections. Colistin still retains a high sensitivity and could, therefore, be used as a therapeutic alternative in case of multi-drug resistance. Our armamentarium against the MDR P. aeruginosa infections is severely restricted and has toxic side effects that can be lethal by themselves in severely compromised patients. Indiscriminate use of antibiotics, particularly from the β-lactams and carbapenem group needs to effectively curbed.

Recommendations

1. Conduct numerous epidemiological studies on the causes of the spread of resistance to both disinfectants and antibiotics by identifying the genes responsible for this resistance.

2. Raise health awareness and discourage the indiscriminate use of antibiotics without medical advice, which can lead to the emergence of resistant bacterial strains. Furthermore, adopt correct methods for preparing chemical solutions used for disinfection.

3. Use the optimal concentration of disinfectants, such as acetic acid and sodium hypochlorite, to treat burn injuries to avoid damaging living tissue at the site of injury.

4. Conduct biennial studies to determine the development of Pseudomonas aeruginosa resistance to modern antibiotics, in collaboration with the Hospital Infection Control Program.

5. Conduct a molecular study of the pathogenicity islands responsible for the production of microbial toxins in Pseudomonas aeruginosa isolates.

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