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Antibiotic and Antiseptic Susceptibility Profiles of Clinical *Pseudomonas aeruginosa* Isolates from Burn Wound Infections

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Abstract

Pseudomonas aeruginosa is a major cause of burn wound infections, often exhibiting multidrug resistance. This study evaluated the antibiotic and antiseptic susceptibility patterns of *P. aeruginosa* isolated from burn patients. Burn wound swabs (n = 30) were collected prospectively and cultured on cetrimide agar. Isolates were identified by cultural, morphological, and biochemical tests. Antibiotic susceptibility was determined using the Kirby-Bauer disc diffusion method against 16 antibiotics, while antiseptic susceptibility was assessed by agar cup method against Hexisol, 70 % ethanol, Dettol, and Savlon. Out of 30 samples, 12 were culture positive and 6 yielded *P. aeruginosa*. All isolates were susceptible to colistin; however, the majority of tested antibiotics - including streptomycin, ciprofloxacin, nalidixic acid, cephalosporins, cotrimoxazole, amoxiclav, and nitrofurantoin - showed limited or no effectiveness. The multiple antibiotic resistance (MAR) index ranged between 0.75 and 1.00, reflecting high selective pressure from antibiotic exposure. Among the antiseptics tested, Hexisol demonstrated the strongest bactericidal activity, whereas 70% ethanol, Dettol, and Savlon exhibited comparatively lower efficacy. These results indicate that *P. aeruginosa* isolates from burn wounds display extensive drug resistance, along with reduced susceptibility to commonly used antiseptics. Enhanced infection control measures and judicious antibiotic use are crucial to limit the dissemination of these resistant pathogens in healthcare facilities.

Keywords: *Pseudomonas aeruginosa*; Antibiotic resistance; MAR index; Antiseptic susceptibility; Burn wound infection.

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1. Introduction

Burn wound infections remain a major cause of morbidity and mortality among patients with thermal injuries worldwide, accounting for approximately 40-60 % of burn-related deaths, particularly in low- and middle-income countries [1-4]. The disruption of the skin barrier, prolonged hospitalization, invasive procedures, and burn-induced

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immunosuppression significantly increase susceptibility to microbial colonization and systemic infection. Consequently, effective infection control practices and appropriate antimicrobial management are essential components of burn care.

Although a wide range of microorganisms are implicated in burn wound infections, bacterial pathogens predominate, with Pseudomonas aeruginosa consistently reported as one of the most prevalent and clinically significant organisms in burn units globally [5-7]. The organism typically colonizes burn wounds after the first week of hospitalization and progressively becomes the dominant Gram-negative pathogen during prolonged hospital stays. Its pathogenic success is attributed to remarkable survival ability under harsh environmental conditions, colonize medical devices and hospital surfaces, and utilize minimal nutritional resources [8]. More importantly, the bacterium exhibits both intrinsic and acquired resistance to a wide range of antimicrobial agents through reduced membrane permeability, active efflux systems, enzymatic degradation, and genetic mutations [9]. In addition, the ability of P. aeruginosa to form biofilms within burn wounds further enhances its resistance to antibiotics and host immune responses, making infections difficult to eradicate and often leading to chronic wound complications [10].

In developing countries such as Bangladesh, the rising prevalence of antibiotic resistance is largely driven by inappropriate antibiotic use, including empirical treatment initiated prior to laboratory confirmation, improper dosing or premature discontinuation of therapy, and the routine administration of sub-therapeutic antibiotic levels in livestock for growth promotion [11]. Recent studies have reported a rapid increase in drug-resistant P. aeruginosa, including multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains in Bangladesh [12-15], highlighting the urgent need for systematic investigation of antimicrobial resistance in burn wound infections.

While antibiotics remain the cornerstone of treatment, antiseptics play a vital role in preventing wound colonization and reducing microbial burden in burn care. Commonly used topical antiseptics such as ethanol-based solutions, chlorhexidine formulations, and phenolic compounds are routinely applied in healthcare settings [16,17]. Nevertheless, recent studies have reported variable effectiveness of commonly used antiseptics against P. aeruginosa, and antiseptic susceptibility testing is often overlooked in routine surveillance.

Given the evolving resistance landscape and the limited regional studies, there is a critical need to simultaneously evaluate both antibiotic and antiseptic susceptibility patterns of *P. aeruginosa* isolates from burn wounds. Therefore, the present study aimed to investigate the susceptibility patterns of commonly used antibiotics and antiseptics against *P. aeruginosa* isolated from a small cohort (n = 30) of burn patients in Bangladesh. This work provides multidrug resistance profiles and antiseptic effectiveness, contributing to improved antimicrobial stewardship and burn wound management in healthcare settings.

2. Method and Materials

2.1. *Study area*

The study was conducted in the Department of Microbiology, Faculty of Life and Earth Sciences, Jagannath University, Dhaka, Bangladesh, from July 2018 to June 2019. Burn wound infection samples were collected from 30 patients admitted to Chittagong Medical College Hospital (CMCH), Chittagong, Bangladesh, after obtaining verbal informed consent from the patients or their attendants.

2.2. *Sample collection and isolation of Pseudomonas aeruginosa*

Prior to sample collection, burn wounds were gently cleaned with sterile normal saline. Wound swab samples were then collected aseptically using sterile cotton swabs to avoid contamination with skin commensals. Each swab was immediately immersed in Brain Heart Infusion (BHI) transport medium and transported to the laboratory for further processing. The collected samples were streaked onto cetrimide agar plates (HiMedia, India) for selective isolation of *P. aeruginosa* and incubated at 37 °C for 24 h. Presumptive identification was based on colony morphology and pigmentation. Final identification was performed using microscopic examination and standard biochemical tests according to Bergey's Manual of Determinative Bacteriology [18]. Conventional biochemical assays, including Gram staining, hydrogen sulfide (H_2S) production, indole test, motility test, methyl red (MR) test, Voges-Proskauer (VP) test, citrate utilization test, catalase test, and oxidase test, were conducted following standard protocols [19].

2.3. *Antibiotics susceptibility testing*

Antibiotic susceptibility of the *P. aeruginosa* isolates was determined using the Kirby–Bauer disc diffusion method on Mueller–Hinton agar (MHA) plates in accordance with standard guidelines [20]. A total of 16 antibiotics were tested: gentamicin, amikacin, streptomycin, azithromycin, ciprofloxacin, nalidixic acid, cefotaxime, ceftriaxone, ceftazidime, cefoxitin, cephradine, meropenem, cotrimoxazole, colistin, amoxiclav, and nitrofurantoin (Oxoid, UK). Bacterial suspensions were adjusted to 0.5 McFarland turbidity and uniformly swabbed onto MHA plates. Antibiotic discs were then placed aseptically on the inoculated plates. After incubation at 37 °C for 24 h, zones of inhibition were measured in millimeters. All experiments were performed in triplicate.

2.4. *Multiple antibiotic resistance (MAR) Index*

The multiple antibiotic resistance (MAR) index for each isolate was calculated by using the following formula [21]:

$$MAR\ INDEX = \frac{No.\ of\ antibiotics\ against\ to\ which\ the\ isolate\ is\ resistant}{Total\ number\ of\ antibiotics\ used}$$

2.5. *Antiseptic susceptibility test*

The susceptibility of *P. aeruginosa* isolates to antiseptic agents was evaluated using the agar well diffusion (agar cup) method on Mueller-Hinton agar [22]. After solidification, MHA plates were uniformly swabbed with standardized bacterial suspensions. Four wells were aseptically punched into each plate, and 100 µL of each commercial antiseptic solution - Savlon, Dettol, Hexisol, and 70 % ethanol - were added into the respective wells. The plates were allowed to stand at room temperature for 2 h to facilitate diffusion and subsequently incubated at 37 °C for 18-24 h in an upright position. Antimicrobial activity was assessed by measuring the diameter of the zones of inhibition around each well.

3. Results and Discussion

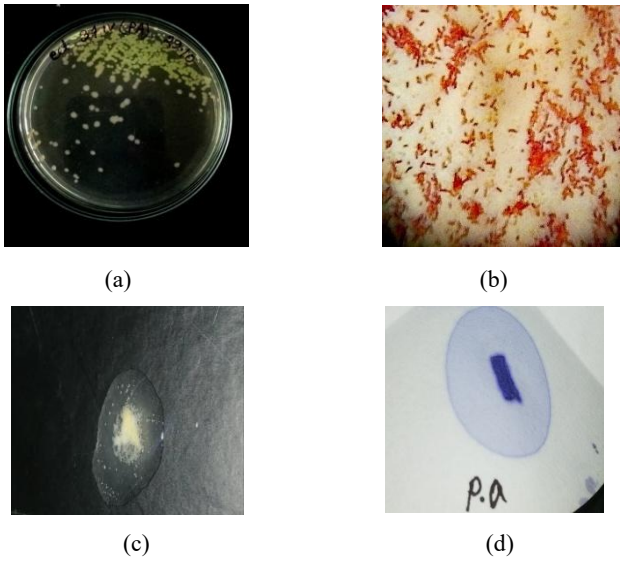
In the present study, bacterial growth was observed in 12 out of 30 burn wound samples, of which 50 % (n = 6) yielded *P. aeruginosa*, indicating its predominance among burn wound pathogens. This finding is consistent with several previous reports highlighting P. aeruginosa as one of the most frequently isolated organisms in burn wound infections [26–28]. The isolates were characterized based on cultural characteristics (colony color, form, margin, and elevation), morphological features, and biochemical properties (Table 1). All isolates were confirmed as *P. aeruginosa* by comparison with standard descriptions provided in Bergey's Manual of Determinative Bacteriology [16].

Table 1. Cultural, morphological, and biochemical characteristics of Pseudomonas aeruginosa isolates recovered from burn wound samples.

Criteria	Observation
Morphology	
Colony on cetrimide agar	Irregular, entire, convex, off-white and greenish colony
Gram staining	Gram negative
Cell arrangement	Short and Rod shaped
Biochemical test	
Indole test	Negative
Motility test	Motile
H_2S production	Negative
Citrate utilization	Positive
MR test	Negative
VP test	Negative
Oxidase test	Positive
Catalase test	Positive
Starch hydrolysis	Negative
Lipid hydrolysis	Positive

The antibiotic susceptibility pattern of *P. aeruginosa* isolates is shown in Figs. 1-2. The highest susceptibility of *P. aeruginosa* was observed against colistin (83.33 %) followed by azithromycin (50 %), gentamicin (33.33 %) and amikacin (16.67 %). In contrast, twelve antibiotics - namely cefotaxime, cotrimoxazole, ceftriaxone, ceftazidime, cefoxitin,

ciprofloxacin, amoxyclav, meropenem, cephradine, streptomycin, nalidixic acid, and nitrofurantoin - were ineffective against all isolates. The intrinsic resistance of *P. aeruginosa* to many of these agents is largely attributed to its low outer membrane permeability, active efflux pump systems (such as MexAB-OprM), and the production of chromosomally encoded β-lactamases, which collectively limit intracellular antibiotic accumulation and inactivate β-lactam antibiotics [23,24].



(a) (b)

(c) (d)

Fig. 1. Identification and biochemical characterization of *Pseudomonas aeruginosa* isolates.
(a) Growth of *P. aeruginosa* colonies on Cetrimide agar showing characteristic greenish pigmentation;
(b) Gram staining of *P. aeruginosa* revealing Gram-negative rod-shaped cells;
(c) Positive catalase test indicated by bubble formation;
(d) Positive oxidase test showing development of a purple color.

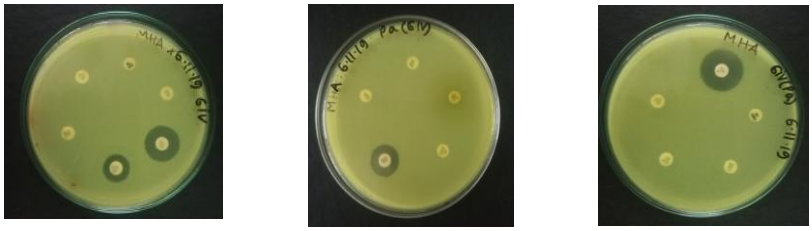


Fig. 2. Antibiotic susceptibility patterns of *Pseudomonas aeruginosa* isolates determined by the Kirby-Bauer disc diffusion method on Mueller-Hinton agar. Clear zones of inhibition surrounding certain antibiotic discs indicate *susceptibility*, whereas the absence or minimal zones around other discs indicate *resistance*.

The retained susceptibility to colistin is notable, as colistin exerts its bactericidal effect by interacting with the lipopolysaccharide (LPS) layer of the outer membrane in Gram-negative bacteria, displacing divalent cations (Ca^{2+} and Mg^{2+}), destabilizing membrane integrity, and leading to leakage of cellular contents and rapid bacterial death. This mechanism bypasses many classical resistance pathways associated with β-lactams and fluoroquinolones, which rely on intracellular targets. However, emerging colistin resistance has been increasingly reported and is frequently linked to modifications of LPS/lipid A, mediated by chromosomal regulators and transferable genes (e.g. pmr and mcr variants), which reduce colistin binding affinity and compromise its activity [25,26].

Unlike the present findings, several studies from Bangladesh and other countries have reported carbapenems, particularly meropenem, as highly effective against *P. aeruginosa* isolates from burn patients [12,27,28]. The complete resistance observed in this study suggests the emergence of carbapenem-resistant strains, potentially mediated by carbapenemase production, loss of outer membrane porin proteins (such as OprD), and overexpression of efflux pumps. These mechanisms significantly reduce intracellular drug concentrations and compromise carbapenem efficacy [29,30]. Similarly, the high resistance observed against cephalosporins including cefotaxime, ceftriaxone, ceftazidime, cefoxitin, and cephradine is consistent with β-lactamase-mediated hydrolysis of the β-lactam ring, particularly through extended-spectrum β-lactamases (ESBLs) and AmpC β-lactamase production. These enzymes degrade cephalosporins before they reach their target penicillin-binding proteins, rendering the antibiotics ineffective [23].

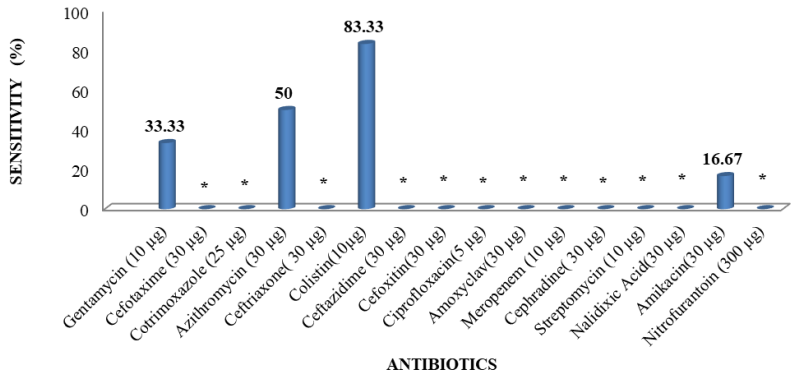


Fig. 3. Percentage susceptibility of *Pseudomonas aeruginosa* isolates to selected antibiotics as determined by the Kirby-Bauer disc diffusion assay. High sensitivity was observed to colistin, azithromycin, gentamicin, and amikacin, whereas resistance was noted against most cephalosporins, fluoroquinolones, carbapenems, and other agents.

Among the aminoglycosides, 33.33 % and 16.67 % (Fig. 3) of isolates were susceptible to gentamicin and amikacin, respectively, which is comparable to findings reported in earlier studies [31]. This partial effectiveness may be attributed to the bactericidal mechanism of aminoglycosides, which involves binding to the 30S ribosomal subunit, leading to the synthesis of defective proteins and disruption of bacterial cell membrane integrity [32,33].

However, resistance to aminoglycosides commonly arises through enzymatic drug modification, altered ribosomal binding sites, and reduced drug uptake due to membrane impermeability or efflux activity, which may explain the limited susceptibility observed in the majority of isolates [34,35].

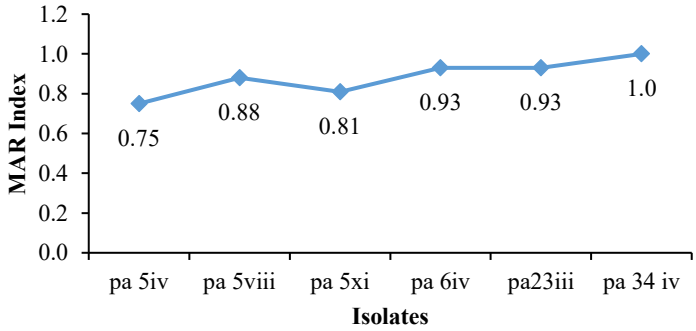


Fig. 4. Multiple antibiotic resistance (MAR) index values of individual *Pseudomonas aeruginosa* isolates. MAR indices ranged from 0.75 to 1.0, indicating high levels of multidrug resistance and strong antibiotic selection pressure among the isolates.

The MAR index analysis revealed values ranging from 0.75 to 1.0 for all isolates (Fig. 4), indicating exposure to multiple antibiotics and strong selective pressure within the clinical environment. High MAR indices are often associated with environments where antibiotics are frequently misused or overprescribed, promoting horizontal gene transfer of resistance determinants via plasmids, transposons, and integrons [21,36]. Such genetic exchange accelerates the emergence and persistence of multidrug-resistant strains in hospital settings.

In addition to antibiotics, the susceptibility of commonly used hospital antiseptics was evaluated. The highest antiseptic activity was observed with Hexisol, which inhibited all isolates (100 %). Dettol showed limited effectiveness (16.67 %), while no inhibitory effect was detected with Savlon or 70 % ethanol (Figs. 5-6). These findings indicate reduced efficacy of several routinely used antiseptics against *P. aeruginosa* and may reflect adaptive tolerance mechanisms in *P. aeruginosa*, including efflux pump overexpression, membrane modification, and stress response pathways. These findings indicate reduced efficacy of several routinely used antiseptics against *P. aeruginosa* and may reflect adaptive tolerance mechanisms in *P. aeruginosa*, including efflux pump overexpression, membrane modification, and stress response pathways. Recent studies have documented increasing tolerance of *P. aeruginosa* to multiple antiseptics and adaptation under sub-inhibitory exposures to commonly used agents [16]. Moreover, evaluations of antiseptic effectiveness against MDR *P. aeruginosa* biofilms highlight reduced activity even at clinically relevant concentrations [17]. Reviews suggest that biocides can trigger adaptive phenotypes and cross-resistance mechanisms that diminish antiseptic susceptibility [37], and molecular analyses show that antiseptic resistance genes co-occur with mobile elements, potentially enhancing tolerance and disease outbreaks in hospital settings [38].

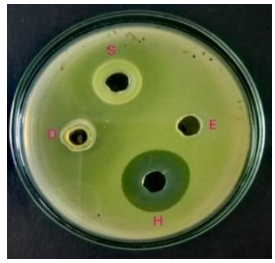


Fig. 5. Agar cup diffusion assay showing the susceptibility pattern of *Pseudomonas aeruginosa* isolates to commonly used hospital antiseptics. Prominent zones of inhibition were observed with Hexisol, whereas minimal or no inhibition was noted with Dettol, Savlon, and 70 % ethanol.

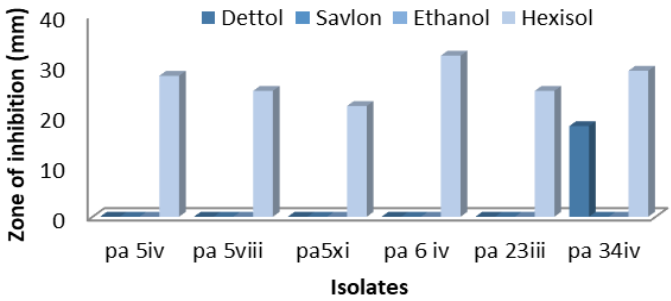


Fig. 6. Antiseptic susceptibility pattern of *Pseudomonas aeruginosa* isolates expressed as zones of inhibition (mm) obtained by the agar cup diffusion method. Hexisol exhibited the largest inhibition zones across all isolates, whereas minimal or no inhibition was observed with Dettol, Savlon, and 70 % ethanol.

Overall, the findings underscore the urgent need for regular microbiological surveillance, routine susceptibility testing prior to antibiotic administration, and the implementation of rational antibiotic policies in burn units. Additionally, the appropriate selection and use of antiseptics at recommended concentrations, along with strict adherence to infection control guidelines, are essential to minimize the spread of multidrug-resistant *P. aeruginosa* in healthcare settings.

4. Conclusion

In the present study, *P. aeruginosa* was isolated from 50 % of the culture-positive burn wound samples, confirming its predominance as a major pathogen in burn wound infections. Among the tested antibiotics, colistin exhibited the highest effectiveness (83.33 %), followed by azithromycin (50 %), while a high level of resistance to meropenem was observed, which is of significant clinical concern. The MAR index values ranging from 0.75 to 1.0 indicate extensive multidrug resistance among the *P. aeruginosa* isolates and reflect strong selective pressure from antibiotic overuse. Regarding antiseptic susceptibility, complete resistance was observed against commonly used agents such as Savlon and 70 % ethanol, whereas hexisol demonstrated significant antibacterial activity against all isolates.

These findings highlight the reduced efficacy of several routinely applied antibiotics and antiseptics against *P. aeruginosa* in burn wound settings. Overall, the results underscore the urgent need for continuous microbiological surveillance, routine susceptibility testing, and implementation of rational antibiotic and antiseptic use policies in healthcare facilities. Further investigations integrating molecular and computational approaches are recommended to elucidate the underlying resistance mechanisms and identify novel therapeutic targets for effective management of *P. aeruginosa* infections.

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