**Investigation of Antibiotic Resistance Patterns and Prevalence of bla*TEM* β-Lactamase Genes in *Pseudomonas aeruginosa* Strains Isolated from Burn Patients in Tabriz**

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**Abstract**

**Background and Objective:** A major contributor to hospital-acquired infections, particularly in burn units, is *Pseudomonas aeruginosa* (*P. aeruginosa*). Because this bacteria produces extended-spectrum β-lactamases (ESBLs), antibiotic resistance is a significant treatment concern. In this work, *P. aeruginosa* isolates isolated from burn victims in Tabriz were examined for antibiotic resistance patterns and the presence of the bla*TEM* gene.

**Methods and Materials:** For this descriptive-cross-sectional study, 100 clinical isolates of *P. aeruginosa* were collected from hospitalized patients in the burn wards of Tabriz hospitals over a six-month period. Standard biochemical methods were used for microorganism identification. Antibiotic resistance patterns were assessed by the disk diffusion technique according to clinical and laboratory standards institute protocols. Additionally, the presence of the bla*TEM* gene was investigated by Polymerase Chain Reaction, and ESBL production was confirmed by the combined disk test.

**Results:** The highest resistance rates were observed for levofloxacin (97%) and meropenem (92%), while the lowest was for ceftazidime (69%). Furthermore, 58% (58/100) of the isolates were ESBL-positive, and half of these (50%, 29/58) carried the bla*TEM* gene.

**Conclusion:** The results of this study indicate that *P. aeruginosa* strains in burn units of Tabriz exhibit high antibiotic resistance, and half of the ESBL-positive isolates carry the bla*TEM* gene. These findings highlight the need for continuous monitoring of antibiotic resistance patterns and prudent use of antibiotics.

**Keywords:** *Pseudomonas aeruginosa*، Drug Resistance, Bacterial، beta-Lactamases، Burns

**1. Introduction**

A rod-shaped, aerobic, Gram-negative bacterium with pili and a polar flagellum, *Pseudomonas aeruginosa* (*P. aeruginosa*) is essential for both bacterial movement and host cell attachment. Especially in burn wounds, it is one of the most prevalent organisms that cause nosocomial infections(1). *P. aeruginosa* is often responsible for nosocomial infections, including pneumonia, infections in immunocompromised patients, and infections in individuals with structural lung diseases such as cystic fibrosis(2). Burn patients face an elevated risk of serious infections and related mortality due to impaired skin barrier function and immune suppression(3). Treating *P. aeruginosa* infections in burn wounds is highly challenging because of the bacterium’s intrinsic and acquired resistance to antibiotics(4). Antibiotic resistance in *P. aeruginosa* has become a global problem, posing a serious public health threat by reducing treatment options and increasing mortality rates(5). Antimicrobial resistance (AMR) is defined as the ability of pathogenic bacteria to resist prescribed drugs, such as β-lactams. In Gram-negative bacteria, this resistance often arises from the production of extended-spectrum β-lactamases (ESBLs), which can inactivate β-lactam antibiotics(6). Among the most extensively studied AMR enzymes are β-lactamases(7). β-lactamases are classified based on their inhibitor response and substrate spectrum. ESBLs encode class A β-lactamases, leading to resistance against certain β-lactam antibiotics(8). These enzymes, encoded by bla genes in Gram-negative bacteria, constitute one of the most critical mechanisms of antibiotic resistance. They impair the efficacy of β-lactam antibiotics through hydrolysis of the β-lactam ring. Compounding this threat, bla genes exhibit a high potential for horizontal gene transfer and integration into multidrug-resistant plasmids, promoting the rapid spread of resistance within clinical settings (9). The clinical challenge of beta-lactam resistance in Gram-negative bacteria, largely driven by beta-lactamase production, has spurred significant pharmaceutical innovation. The development of novel compounds designed to inhibit or disrupt these enzymes has led to groundbreaking advancements with expanded therapeutic applications(10). This study aimed to accurately identify *P. aeruginosa* strains, determine antibiotic resistance patterns, and investigate the prevalence of the bla*TEM* gene in burn patients in Tabriz.

**2. Materials and Methods**

**2.1. Sampling and Bacterial Isolation**

A descriptive cross-sectional research was carried out on 100 burn patients who were hospitalized to Tabriz hospitals' burn units over the course of six months, from April to September 2024. Using sterile swabs, samples were obtained from burn wounds and immediately placed into Cary-Blair transport medium. To maintain optimal conditions for bacterial survival, samples were delivered to the laboratory on ice and processed within a strict 2-hour window from the time of collection. The inclusion criteria for this study required participants to be adult patients hospitalized with burn wounds in medical centers across Tabriz, northwest Iran, who exhibited clinical signs of active infection (such as purulent discharge, erythema, localized warmth, or systemic fever). Furthermore, enrollment was contingent upon obtaining written informed consent from the patient or their legal guardian. A definitive microbiological confirmation of *P. aeruginosa* infection, obtained from a sterilely collected burn wound sample and identified through standard cultural characteristics (e.g., grape-like odor, pyocyanin production), biochemical profiling (oxidase-positive), and automated systems, was mandatory for inclusion in the final analysis. Samples were initially cultured on sheep blood agar (containing 5% sheep blood) for the observation of hemolytic patterns and on MacConkey agar to selectively isolate Gram-negative bacteria, followed by incubation at 37°C for 24-48 hours. Suspected *P. aeruginosa* colonies were selected based on characteristic morphology: large size, smooth surface, irregular margins, and the distinctive green-blue pigmentation (pyocyanin). Final identification was confirmed through a series of standard biochemical tests. These included a positive oxidase reaction, which is a key diagnostic trait for *P. aeruginosa*; the ability to oxidize but not ferment glucose in Oxidation-Fermentation (OF) medium; alkaline/alkaline (K/K) reaction in Kligler Iron Agar (KIA) indicative of non-fermentative metabolism; positive citrate utilization; confirmed pyocyanin pigment production; and the ability to grow at 42°C, a test that helps differentiate it from other pseudomonads. Additional tests such as gelatin hydrolysis and urease activity were performed for differentiation from similar species. Using *P. aeruginosa* (American Type Culture Collection-ATCC: 27853) as a positive control, all processes were verified. At -80°C, confirmed isolates were kept in Tryptic Soy Broth with 15% glycerol. Exclusion criteria included patient unwillingness to continue cooperation, unconfirmed *P. aeruginosa* diagnosis in laboratory tests, and the presence of severe underlying diseases (e.g., uncontrolled diabetes, metastatic cancer, or advanced autoimmune disorders).The study was conducted in compliance with biosafety principles, ethical protocols, and approval code IR.IAU.TABRIZ.REC.1403.209.

**2.2. Antibiotic Susceptibility Testing by Disk Diffusion Method**

The standard agar disk diffusion technique (Kirby-Bauer) was used according to clinical and laboratory standards institute (CLSI) protocols. Mueller-Hinton agar and antibiotic disks manufactured by Padten Teb Iran were employed in this study. The tested antibiotics included β-lactams such as piperacillin (100 μg) and ceftazidime (30 μg); aminoglycosides including gentamicin (10 μg), tobramycin (10 μg), and amikacin (30 μg); fluoroquinolones such as ciprofloxacin (5 μg) and levofloxacin (5 μg); and other groups including cefepime (30 μg) and meropenem (10 μg). For quality control, standard strains of *Escherichia coli* (*E. coli*) (ATCC: 25922) and *P. aeruginosa* (ATCC: 27853) were used in each test batch. Results were interpreted based on CLSI guidelines.

**2.3. Phenotypic Identification of β-Lactamase-Producing Strains**

In this study, the identification of ESBL-producing isolates was performed using the standard Combination Disk Test method in accordance with CLSI protocols. Antibiotic disks of cefotaxime (30μg), cefotaxime with clavulanic acid (10μg), ceftazidime (30μg), and ceftazidime with clavulanic acid (10μg) (Mast Group, UK) were employed. Freshly prepared Mueller-Hinton agar was used as the culture medium. A bacterial suspension with a standard concentration of 0.5 McFarland (~1.5×10^8 CFU/ml) was prepared and uniformly inoculated onto the culture medium. Interpretation criteria were based on a ≥5 mm increase in the inhibition zone diameter for antibiotic disks combined with clavulanic acid compared to the antibiotic alone, indicating ESBL production. Final confirmation required positive results with both cefotaxime and ceftazidime disk pairs. For quality control, *Klebsiella pneumoniae* (ATCC 700603) served as the positive control and *E. coli* (ATCC 25922) as the negative control (All culture media were obtained from Merck, Germany).

**2.4. Genotypic Identification of bla*TEM* Gene-Producing Strains**

The frequency of bla*TEM* genes was investigated using polymerase chain reaction (PCR). Bacterial DNA was extracted using a kit from Invitek Stratec Business (Canada). DNA concentration was measured by Nanodrop, and purity was assessed by the OD260/280 ratio. Ratios between 1.8 and 1.9 indicated acceptable DNA purity. PCR reactions were carried out using 2x master mix from Sinaclon (Iran), containing 12.5 µl master mix, 1 µl forward primer (10 pmol), 1 µl reverse primer (10 pmol), 3 µl template DNA, and 7.5 µl nuclease-free distilled water. Specific primers for bla*TEM* gene amplification were designed, with sequences provided in Table 1. Primer design was performed using nucleotide databases (National Center for Biotechnology Information) and Allele ID7 software, and sequences were verified. PCR started with a 10-minute initial denaturation at 95°C. This was followed by 30 amplification cycles, which included 30 seconds of denaturation at 95°C, 60 seconds of annealing at 50°C, and 60 seconds of extension at 72°C. To make sure the PCR products were amplified completely, a final extension step was carried out for five minutes at 72°C. The negative control was distilled water. A 1.5% agarose gel was used for electrophoresis of the PCR products. The size marker was a 100 base pair (bp) ladder. V2 Safe Stain was applied to the gel, and a gel documentation system was used to see and record the DNA bands. Lastly, associations between genotypic and phenotypic data were evaluated using the chi-square test implemented in the Statistical Package for the Social Sciences (SPSS) version 23, with a statistical significance threshold set at p < 0.05.

**Table 1. Primers Used in This Study**

|  |  |  |
| --- | --- | --- |
|  (bp) size | Gene sequence  | Primer  |
| 403 | TTTCGTGTCGCCCTTATTCC ATCGTTGTCAGAAGTAAGTTGG | bla *TEM* –F bla *TEM* –R  |

**3. Results**

In this study, 100 isolates of *P. aeruginosa* were examined. The mean age of the patients was 45.9 ± 16.12 years, ranging from 20 to 78 years, including 38 females (38%) with a mean age of 49.61 ± 16.33 years and 62 males (62%) with a mean age of 39.84 ± 16.52 years. Statistical analysis showed no significant difference in the distribution of isolates between different age and gender groups (p > 0.05). Antibiogram results revealed a high resistance pattern to levofloxacin (97%), meropenem (92%), ciprofloxacin (88%), and tobramycin (87%), while the lowest resistance was observed to ceftazidime (69%) (Figure 1). The combined disk test identified 58 isolates (58%) as ESBL positive, including 22 isolates from females (37.93%) with a mean age of 45.45 ± 15.15 years and 36 isolates from males (62.07%) with a mean age of 39.30 ± 14.11 years. Among these 58 ESBL-positive isolates, 29 isolates (50%) carried the bla*TEM* gene (Figure 2). Statistical analyses found no significant association between ESBL status or the presence of the bla*TEM* gene with patients age or gender (p > 0.05). These results suggest that neither isolate distribution nor patterns of antibiotic resistance are substantially impacted by demographic variables. Nonetheless, the high frequency of important antibiotic resistance and the bla*TEM* gene's presence in 50% of ESBL-positive isolates are alarming and highlight the necessity of ongoing surveillance for antibiotic resistance and the execution of infection control initiatives.



**Figure 1. Antibiotic resistance of *Pseudomonas aeruginosa* isolates from burn patients in hospitals of Tabriz.**



**Figure 2. PCR product electrophoresis for the bla*TEM* gene. M: 100 bp marker, PC: positive control, 1 and 2: bla*TEM* positive samples, 3: bla*TEM* negative sample.**

 **4. Discussion**

One of the main causes of hospital-acquired infections in burn patients is the opportunistic Gram-negative bacterium *P. aeruginosa*. Since *P. aeruginosa* can acquire extensive antibiotic resistance, treating infections caused by this bacterium has become very challenging(11). Investigating the antibiotic resistance pattern of *P. aeruginosa* in the hospital setting and determining its susceptibility to commonly used hospital drugs helps establish initial treatment protocols and effectively manage drug resistance in this bacterium(12). *P. aeruginosa* is naturally resistant to several antimicrobial agents, making its control difficult. Various mechanisms contribute to this resistance(13). By producing beta-lactamase enzymes, bacteria become resistant to beta-lactam antibiotics(14). The significant increase of ESBL-positive *P. aeruginosa* strains among burn patients has posed challenges in treating this group(15, 16). The number of organisms producing bla*TEM* enzymes continues to rise, and treating infections caused by these bacteria remains highly problematic due to the antibiotic resistance conferred by these enzymes(17). In previous studies, samples were obtained from various sources such as urine, blood, infected wounds, and urinary tract infections(18). However, in this study, all clinical samples were collected from patients admitted to the burn unit. Among 100 isolates obtained from patients, 87 were resistant to at least three types of antibiotics. According to the findings of Abdelrahim et al.'s 2023 study, bla*TEM* was reported as the second most prevalent gene (46.2%) in ESBL-producing *P. aeruginosa* strains(19). The present study also identified this gene as one of the most prevalent resistance genes, with a frequency of 50%. In a study by Peymani et al. conducted in Tehran, 28.6% of ESBL-producing *P. aeruginosa* strains were reported (20), which is lower than the findings of the present study. In contrast to the study by Zhilong Chen et al., where ESBL-positive *P. aeruginosa* strains showed the highest resistance to ceftazidime (30%)(21), our study found this antibiotic to have the lowest resistance rate (69%). Bokaeian et al. in Zahedan reported that 25.86% of 116 isolates were resistant to at least one cephalosporin or aztreonam(22). Although this finding differs from the results of the current study in Tabriz, which demonstrated the highest resistance rates to levofloxacin (97%), meropenem (92%), ciprofloxacin (88%), and tobramycin (87%), both studies confirm a similar pattern of antibiotic resistance. In the present study, among 100 *P. aeruginosa* strains isolated from burn patients, 97 were resistant to levofloxacin, while the lowest resistance rate was observed against ceftazidime (69%). However, in a 2010 study by Fazeli et al. in Isfahan, 94% of *P. aeruginosa* isolates exhibited 100% resistance to ceftazidime(23), a discrepancy with our results. Bahrami et al. (2017) in Bandar Abbas found 47% resistance to meropenem among *P. aeruginosa* isolates(24), whereas this rate increased to 92% in the present study. Additionally, Bahrami et al. identified bla*TEM* as the most common beta-lactamase resistance gene with a frequency of 56.3% by PCR, which is relatively consistent with the current study’s 50%. Mustafa et al. (2002) reported a 41% resistance rate to meropenem(25), whereas our data reveal a dramatic rise to 92%, indicating a concerning expansion of carbapenem resistance over the past two decades. Adjei et al. (2018) reported an 82% resistance rate to meropenem(26), while Haghighifar et al. (2021) observed an increase to 91.3% (27). The results of the current study show that meropenem resistance is quite prevalent in the populations under investigation, which is in close agreement with both. The discrepancies between our findings and those of previous studies are likely influenced by several factors, including variations in sample size, the genetic diversity of local microbial populations, and distinct regional epidemiological patterns. Importantly, these same factors particularly our limited sample size and the specific geographical focus of our work in Tabriz represent key limitations of the present study that should be considered when interpreting the results. Consequently, future research with larger, multi-center cohorts is warranted to validate and generalize these findings.

**5. Conclusion**

This study reveals a concerning level of antibiotic resistance among *P. aeruginosa* isolates from burn patients in Tabriz, with over 90% resistant to meropenem and fluoroquinolones, and 58% of isolates identified as ESBL producers. Notably, half of the ESBL-positive strains harbored the bla*TEM* gene, highlighting its role in resistance mechanisms. Despite the lack of demographic correlations, the high prevalence of multidrug-resistant strains represents a significant public health challenge. These findings underscore the urgent need for systematic antibiotic resistance surveillance, strict infection control protocols in burn units, and evidence-based antimicrobial stewardship practices. Future research should focus on longitudinal monitoring of resistance trends and investigation of additional genetic factors, such as other ESBL and carbapenemase genes, to guide targeted interventions and reduce the spread of resistant.

**List of Abbreviation**

*P. aeruginosa*: *Pseudomonas aeruginosa*, ESBLs: Extended-spectrum β-lactamases, AMR: Antimicrobial resistance, ATCC: American type culture collection, CLSI: Clinical and laboratory standards institute, *E. coli*: *Escherichia coli*, PCR: Polymerase chain reaction.

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