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Antibiotic resistance and bla$_{TEM}$ β-lactamase gene carriage in *Pseudomonas aeruginosa* isolates from burn patients: A multicenter study in Tabriz, 2024

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Abstract

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

Methods: In this descriptive-cross-sectional study, 100 clinical isolates of *P. aeruginosa* were collected from patients hospitalized in the burn wards of Tabriz hospitals over a six-month period. Standard biochemical methods were used to identify microorganisms. 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, half of which (50%, 29/58) carried the bla$_{TEM}$ gene.

Conclusion: The results of this study indicated that *P. aeruginosa* strains in burn units of Tabriz hospitals exhibited high antibiotic resistance. Half of ESBL-positive isolates carried the bla$_{TEM}$ gene, highlighting the need for continuous monitoring of antibiotic resistance patterns and prudent use of antibiotics.

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Drug resistance
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Burns

Highlights

What is current knowledge?

- *P. aeruginosa* is a major cause of nosocomial infections in burn units and is recognized for its capacity to develop antibiotic resistance, including through the production of extended-spectrum beta-lactamases (ESBLs).

What is new here?

- This multicenter study offers recent and specific data from Tabriz, highlighting a concerningly high resistance rate to carbapenems (Meropenem, 92%) and fluoroquinolones (Levofloxacin, 97%).
- It is the first report in this region and among this patient group indicating that 50% of the ESBL-producing isolates possess the blaTEM gene, identifying a significant genetic mechanism for resistance.

Introduction

A rod-shaped, aerobic, Gram-negative bacterium with pili and a polar flagellum, *Pseudomonas aeruginosa* (*P. aeruginosa*) is a serious health concern in hospitals. Both pili and the flagellum are essential for the movement and host cell attachment of bacteria. Especially in burn wounds, *P. aeruginosa* is one of the most prevalent organisms causing nosocomial infections (1), including pneumonia and infections in immunocompromised patients or 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). Eradication of *P. aeruginosa* infecting burn wounds is a daunting task because of the bacterium's intrinsic and acquired resistance to antibiotics (4). Antibiotic-resistant *P. aeruginosa* has become a global problem, posing a serious public health threat associated with limited 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), which are classified based on their inhibitory responses and substrate spectra. 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, are responsible for one of the most critical mechanisms of antibiotic resistance (i.e., decapitating β-lactam antibiotics through degrading the β-lactam ring). Aggravating this threat, bla genes exhibit a high potential for horizontal gene transfer and integration into multidrug-resistant plasmids, promoting the rapid spread of resistance in clinical settings (9). The clinical challenge of beta-lactam resistance among Gram-negative bacteria, largely driven by beta-lactamase production, has spurred significant pharmaceutical innovations. The development of novel compounds designed to inhibit or disrupt these enzymes has led to groundbreaking accomplishments in expanding therapeutic interventions (10). This study aimed to accurately identify *P. aeruginosa* strains, determine their antibiotic resistance patterns, and investigate the prevalence of the bla$_{TEM}$ gene in burn patients in Tabriz.

Methods

Sampling and bacterial isolation

A descriptive cross-sectional study was conducted utilizing a convenience sampling method to recruit 100 burn patients admitted to the burn units of Tabriz hospitals 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 media. To maintain optimal conditions for bacteria survival, samples were transferred to the laboratory on ice and processed within a strict 2-hour window from the time of collection.

Inclusion criteria in this study included being adult patients hospitalized due to burn wounds in medical centers across Tabriz, northwest Iran, and showing clinical signs of active infections (Such as purulent discharge, erythema, localized warmth, or systemic fever). Furthermore, enrollment was contingent upon obtaining written informed consent from patients or their legal guardians. The definitive microbiological confirmation of *P. aeruginosa* infection, isolated from burn wound samples collected in a sterile manner, 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) to recognize 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 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, pyocyanin pigment production, and the ability to grow at 42°C (A trait that helps differentiate *P. aeruginosa* 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. Confirmed isolates were kept at -80°C in Tryptic Soy Broth with 15% glycerol.

Exclusion criteria included unwillingness to continue cooperation, unconfirmed laboratory detection of *P. aeruginosa*, 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 and ethical protocols and acquired the approval code of IR.IAU.TABRIZ.REC.1403.209.

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. Target 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.

Phenotypic identification of β-lactamase-producing strains

In this study, ESBL-producing isolates were identified using the standard Combination Disk test in accordance with CLSI protocols. The antibiotic disks of cefotaxime (30 µg), cefotaxime plus clavulanic acid (10 µg), ceftazidime (30 µg), and ceftazidime plus clavulanic acid (10 µg) (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. ESBL production was recognized based on a ≥5 mm increase in the inhibition zone diameter for antibiotic disks used along with clavulanic acid compared to the antibiotic alone. Final confirmation required positive results for both cefotaxime and ceftazidime disk pairs. *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).

Genotypic identification of bla$_{TEM}$-positive strains

The expression of bla$_{TEM}$ genes was investigated using polymerase chain reaction (PCR). Bacterial DNA was extracted using a kit (Invitek Stratec Business, Canada) and quantified by a Nanodrop device, and its purity was assessed by calculating the OD260/280 ratio. Ratios between 1.8 and 1.9 indicated acceptable DNA purity. PCR reactions were carried out using 2x master mix reactions 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 (Table 1). The primers were designed using nucleotide databases (National Center for Biotechnology Information) and Allele ID7 software, and their sequences were verified. PCR started with a 10-minute initial denaturation phase at 95°C. This was followed by 30 amplification cycles, encompassing 30 seconds of denaturation at 95°C, 60 seconds of annealing at 50°C, and 60 seconds of extension at 72°C. To ensure complete amplification, a final extension step was carried out for five minutes at 72°C. The negative control tube contained distilled water. A 1.5% agarose gel was used for electrophoresis of PCR products. The size marker included a 100-base pair (bp) ladder. V2 Safe Stain was applied to the gel, and a gel documentation system was used to visualize DNA bands. Associations between genotypic and phenotypic data were evaluated using the chi-square test implemented via Statistical Package for the Social Sciences (SPSS) software version 23 at a statistical significance threshold of $P < 0.05$.

Table 1. Primers used in this study

Primer	Sequence	Product size (bp)
bla $_{TEM}$ –F	TTTCGTGTCGCCCTTATTCC	403
bla $_{TEM}$ –R	ATCGTTGTCAGAAGTAAGTTGG	

Results

In this study, 100 isolates of *P. aeruginosa* were examined. The mean age of patients was 45.9 ± 16.12 years, ranging from 20 to 78 years. There were 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-Value > 0.05). Antibiogram results revealed a high prevalence of resistance to levofloxacin (97%), meropenem (92%), ciprofloxacin (88%), and tobramycin (87%), while the lowest rate of resistance was related to ceftazidime (69%) (Figure 1). The combined disk test identified 58 ESBL-positive isolates (58%), 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 58 ESBL-positive isolates, 29 (50%) carried the bla$_{TEM}$ gene (Figure 2). No significant association was found between ESBL or bla$_{TEM}$ status and patients' age or gender (P-Value > 0.05). Neither isolate distribution frequencies nor antibiotic resistance patterns were significantly associated with demographic variables. Nonetheless, the high frequency of antibiotic resistance and bla$_{TEM}$ positivity (50% of ESBL-positive isolates) highlights the necessity of ongoing surveillance for antibiotic resistance and infection control initiatives.

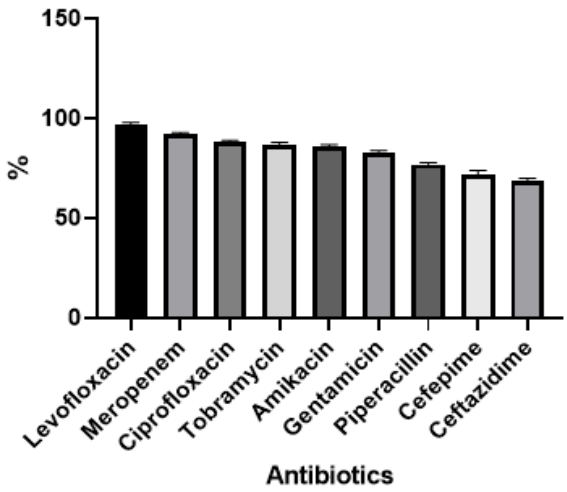

Figure 1. Antibiotic resistance of *Pseudomonas aeruginosa* isolates from burn patients in hospitals of Tabriz

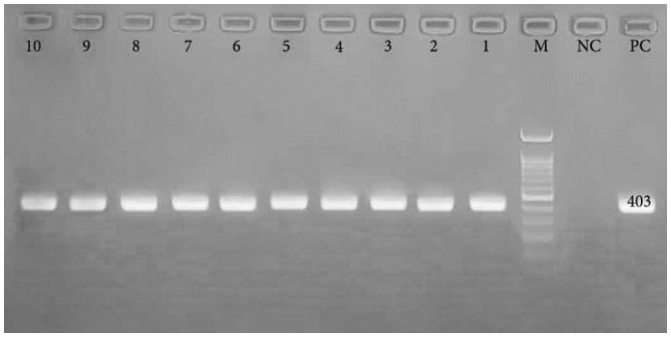

Figure 2. PCR product electrophoresis for the bla$_{TEM}$ gene. M: 100-bp marker, PC: positive control, NC: Negative control, 1 and 10: bla$_{TEM}$ positive samples

Discussion

One of the main sources 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 antimicrobials can help establish initial treatment protocols and effectively manage drug-resistant infections caused by this bacterium (12). *P. aeruginosa* is naturally resistant to several antimicrobial agents, making its control difficult. Various mechanisms contribute to this resistance (13), including producing beta-lactamase enzymes, resulting in resistance to beta-lactam antibiotics (14). The significant increase in ESBL-positive *P. aeruginosa* strains among burn patients has posed therapeutic challenges (15,16). The rate 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 had been 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. (2023), 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, the frequency of ESBL-producing *P. aeruginosa* strains was reported as 28.6% (20), which is lower than the rate recorded in the present study.

In contrast to a study by Chen et al., where ESBL-positive *P. aeruginosa* strains showed the highest resistance to ceftazidime (30%) (21), we found the lowest resistance rate (69%) to this antibiotic. Bokaeian et al. in Zahedan reported that 25.86% of 116 isolates were resistant to at least one cephalosporin or aztreonam antibiotic (22). 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%); however, both studies confirmed 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 complete resistance to ceftazidime (23), a discrepancy with our results. Bahrami et al. (2018) 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 was relatively consistent with the current study's 50% (24). Saleh et al. (2022) reported a 41% resistance rate to meropenem (25), whereas our data revealed 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 showed that meropenem resistance was quite prevalent in the populations under investigation, which was in close agreement with both above studies. The discrepancies between our findings and those of previous studies likely root in 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 on Tabriz population, represent the key limitations of the present study and should be considered when interpreting the results. Consequently, larger multi-center cohorts are warranted in the future to validate and generalize these findings.

Conclusion

This study revealed a concerning level of antibiotic resistance among *P. aeruginosa* isolates from burn patients in Tabriz, with over 90% resistance to meropenem and fluoroquinolones. Also, 58% of the isolates were identified as ESBL producers. Notably, half of ESBL-positive strains harbored the bla$_{TEM}$ gene, highlighting the high penetrance of this resistance mechanism. 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 regular antibiotic resistance surveillance, strict infection control protocols, and evidence-based antimicrobial stewardship practices in burn units. Future research should focus on longitudinal monitoring of resistance trends and investigating other genetic elements of resistance, such as other ESBL and carbapenemase genes, to guide targeted interventions and reduce the spread of resistant bacterial strains.

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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Ethical statement

Ethical approval was obtained from the Clinical Research Ethics Committee of Islamic Azad University, Tabriz Branch (Code: IR.IAU.TABRIZ.REC.1403.209). All participants were informed about the study's objectives and signed written informed consent before enrollment.

Conflicts of interest

The authors declared no potential conflict of interest with respect to the research, authorship, and/or publication of this article.

Author contributions

Concept/Design: A.J.S, M.P; Data acquisition: A.J.S; Data analysis and Interpretation: Z.G, A.G; Drafting the manuscript: Z.G, A.G, M.G.K.J, K.H.K, K.S, M.M.S.F; Critical revision of the manuscript: AJS; Final approval and Accountability: MP; Technical or Material support: A.J.S, M.P; Supervision: MP; Securing funding (If available): N/A.

Data availability statement

The data supporting the findings of this study can be obtained from the corresponding author upon a reasonable request.

References

1. Jafari-Sales A, Khaneshpour H. Molecular Study of BlaIMP and BlaVIM Genes in Pseudomonas Aeruginosa Strains, Producer of Metallo Beta Lactamases Isolated from Clinical Samples in Hospitals and Medical Centers of Tabriz. Paramedical Sciences and Military Health. 2020;14(4):18-25. [View at Publisher] [Google Scholar]

2. Reynolds D, Kollef M. The epidemiology and pathogenesis and treatment of Pseudomonas aeruginosa infections: an update. Drugs. 2021;81(18):2117-31. [View at Publisher] [DOI] [PMID] [Google Scholar]
3. Gong Y, Peng Y, Luo X, Zhang C, Shi Y, Zhang Y, et al. Different infection profiles and antimicrobial resistance patterns between burn ICU and common wards. Front Cell Infect Microbiol. 2021;11:681731. [View at Publisher] [DOI] [PMID] [Google Scholar]
4. Church D, Elsayed S, Reid O, Winston B, Lindsay R. Burn wound infections. Clin Microbiol Rev. 2006;19(2):403-34. [View at Publisher] [DOI] [PMID] [Google Scholar]
5. Abdi FA, Motumma AN, Kalayu AA, Abegaz WE. Prevalence and antimicrobial-resistant patterns of Pseudomonas aeruginosa among burn patients attending Yekatit 12 Hospital Medical College in Addis Ababa, Ethiopia. PloS one. 2024;19(3):e0289586. [View at Publisher] [DOI] [PMID] [Google Scholar]
6. Sah RSP, Dhungel B, Yadav BK, Adhikari N, Thapa Shrestha U, Lekhak B, et al. Detection of TEM and CTX-M Genes in Escherichia coli isolated from clinical specimens at tertiary care heart hospital, Kathmandu, Nepal. Diseases. 2021;9(1):15. [View at Publisher] [DOI] [PMID] [Google Scholar]
7. Mroczkowska JE, Barlow M. Fitness trade-offs in bla TEM evolution. Antimicrob Agents Chemother. 2008;52(7):2340-5. [View at Publisher] [DOI] [PMID] [Google Scholar]
8. Shalmashi H, Farajnia S, Sadeghi M, Tanoumand A, Veissi K, Hamishekar H, et al. Detection of ESBLs types blaCTX-M, blaSHV and blaTEM resistance genes among clinical isolates of Pseudomonas aeruginosa. Gene Rep. 2022;28:101637. [View at Publisher] [DOI] [Google Scholar]
9. Bush K, Jacoby GA. Updated functional classification of β-lactamases. Antimicrob Agents Chemother. 2010;54(3):969-76. [View at Publisher] [DOI] [PMID] [Google Scholar]
10. Bush K, Bradford PA. Epidemiology of β-lactamase-producing pathogens. Clin Microbiol Rev. 2020;33(2):10.1128/cmr. 00047-19. [View at Publisher] [DOI] [PMID] [Google Scholar]
11. Tchakal-Mesbahi A, Metref M, Singh VK, Almpani M, Rahme LG. Characterization of antibiotic resistance profiles in Pseudomonas aeruginosa isolates from burn patients. Burns. 2021;47(8):1833-43. [View at Publisher] [DOI] [PMID] [Google Scholar]
12. Espinal MA, Laszlo A, Simonsen L, Boulahbal F, Kim SJ, Reniero A, et al. Global trends in resistance to antituberculosis drugs. World Health Organization-International :union: against Tuberculosis and Lung Disease Working Group on Anti-Tuberculosis Drug Resistance Surveillance. N Engl J Med. 2001;344(17):1294-303. [View at Publisher] [DOI] [PMID] [Google Scholar]
13. Rahimzadeh Torabi L, Doudi M, Golshani Z. The frequency of blaIMP and blaVIM carbapenemase genes in clinical isolates of Pseudomonas aeruginosa in Isfahan medical centers. medical journal of mashhad university of medical sciences. 2016;59(3):139-47. [View at Publisher] [DOI] [Google Scholar]
14. Drawz SM, Bonomo RA. Three decades of β-lactamase inhibitors. Clin Microbiol Rev. 2010;23(1):160-201. [View at Publisher] [DOI] [PMID] [Google Scholar]
15. Empel J, Filczak K, Mrówka A, Hryniewicz W, Livermore DM, Gniadkowski M. Outbreak of Pseudomonas aeruginosa infections with PER-1 extended-spectrum β-lactamase in Warsaw, Poland: further evidence for an international clonal complex. J Clin Microbiol. 2007;45(9):2829-34. [View at Publisher] [DOI] [PMID] [Google Scholar]
16. Lin S-P, Liu M-F, Lin C-F, Shi Z-Y. Phenotypic detection and polymerase chain reaction screening of extended-spectrum β-lactamases produced by Pseudomonas aeruginosa isolates. J Microbiol Immunol Infect. 2012;45(3):200-7. [View at Publisher] [DOI] [PMID] [Google Scholar]
17. Shams E, Nateghi B, Eshaghiyan A, Behshood P. TEM gene Detection in Clinical Pseudomonas aeruginosa and Escherichia coli Samples. Research in Molecular Medicine. 2019;7(1):35-41. [View at Publisher] [DOI] [Google Scholar]
18. Jafarı-sales A, Mehdizadeh F, Fallah G, Pashazadeh M, Baghi HB. Examining the frequency of carbapenemase genes blaKPC, blaIMP, blaOXA-48, blaSPM, blaNDM, blaVIM, blaGES, blaBIC, blaAIM, blaGIM, blaSIM, and blaDIM in Pseudomonas aeruginosa strains isolated from patients hospitalized in northwest Iran hospitals. Journal of Experimental and Clinical Medicine. 2024;41(3):466-73. [View at Publisher] [Google Scholar]
19. Abdelrahim SS, Hassuna NA, Waly NG, Kotb DN, Abdelhamid H, Zaki S. Coexistence of plasmid-mediated quinolone resistance (PMQR) and extended-spectrum beta-lactamase (ESBL) genes among clinical Pseudomonas aeruginosa isolates in Egypt. BMC Microbiol. 2024;24(1):175. [View at Publisher] [DOI] [PMID] [Google Scholar]
20. Peymani A, Naserpour-Farivar T, Zare E, Azarhoosh KH. Distribution of blaTEM, blaSHV, and blaCTX-M genes among ESBL-producing P. aeruginosa isolated from Qazvin and Tehran hospitals, Iran. J Prev Med Hyg. 2017;58(2):E155. [View at Publisher] [PMID] [Google Scholar]
21. Chen Z, Niu H, Chen G, Li M, Li M, Zhou Y. Prevalence of ESBLs-producing Pseudomonas aeruginosa isolates from different wards in a Chinese teaching hospital. Int J Clin Exp Med. 2015;8(10):19400-05. [View at Publisher] [PMID] [Google Scholar]
22. Bokaeian M, Zahedani SS, Bajgiran MS, Moghaddam AA. Frequency of PER, VEB, SHV, TEM and CTX-M genes in resistant strains of Pseudomonas aeruginosa producing extended spectrum β-lactamases. Jundishapur J Microbiol. 2014;8(1):e13783. [View at Publisher] [DOI] [PMID] [Google Scholar]
23. Fazeli H, Moslehi Z, Irajian G, Salehi M. Determination of Drug resistance patterns and detection of bla-VIM gene in Pseudomonas aeruginosastrains Isolated from burned patients in the Emam Mosa Kazem hospital, Esfahan, Iran (2008-9). Iran J Med Microbiol. 2010;3(4):1-8. [View at Publisher] [Google Scholar]
24. Bahrami M, Mmohammadi-Sichani M, Karbasizadeh V. Prevalence of SHV, TEM, CTX-M and OXA-48 β-Lactamase genes in clinical isolates of Pseudomonas aeruginosa in Bandar-Abbas, Iran. Avicenna J Clin Microbiol Infect. 2018;5(4):86-90. [View at Publisher] [DOI] [Google Scholar]
25. Saleh MAA-jM, Naji HF. Detection of blatem, blactx-m, and blashv genes in clinical isolates of multidrug-resistant pseudomonas aeruginosa. Int J Health Sci . 2022;6(S7):3239-53. [View at Publisher] [DOI] [Google Scholar]
26. Adjei CB, Govinden U, Essack SY, Moodley K. Molecular characterisation of multidrug-resistant Pseudomonas aeruginosa from a private hospital in Durban, South Africa. Southern African Journal of Infectious Diseases. 2018;33(2):38-41. [View at Publisher] [DOI] [Google Scholar]
27. Haghighifar E, Dolatabadi RK, Norouzi F. Prevalence of blaVEB and blaTEM genes, antimicrobial resistance pattern and biofilm formation in clinical isolates of Pseudomonas aeruginosa from burn patients in Isfahan, Iran. Gene Reports. 2021;23:101157. [View at Publisher] [DOI] [Google Scholar]

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