AMERICAN JOURNAL OF BIOLOGY AND NATURAL SCIENCES
https://biojournals.us/index.php/AJBNS
ISSN: 2997-7185

Article

Identification of Integron and Antibiotic Resistance Genes in *Pseudomonas aeruginosa* by Genetic Methods in Burn Patients at Al-Hussien Teaching Hospital in Al Samawah, Iraq

Elaf Lateef Neamah[1], Huda J. Mohemmad[2], Bushra Ali Ghyadh[3], Zaid Mohammed Jasim[4], Amar Mousa Mandal[1,*]

[1]Department of Science, Faculty of Basic education, Al-Muthanna University, AL-Samawah,66001, Iraq
[2]Department of Pharmaceutical Sciences, College of Pharmacy, Thi-Qar University, Thi-Qar, Iraq.
[3]Department of Biology, Faculty of science, Thi-qar University, Thi-Qar, 64001, Iraq
[4]Presidency of Al-Muthanna University, AL-Samawah,66001, Iraq

Corresponding Author
Amar Mousa Mandal
mmarbiotech@mu.edu.iq

Citation: Neamah, E. L., Mohemmad, H. J., Ghyadh, B. A., Jasim, Z. M & Mandal, A. M. Identification of Integron and Antibiotic Resistance Genes in *Pseudomonas aeruginosa* by Genetic Methods in Burn Patients at Al-Hussien Teaching Hospital in Al Samawah, Iraq. American Journal of Biology and Natural Sciences 2026, 3(6), 163-172

Received: 08th Mar 2026
Revised: 21th Apr 2026
Accepted: 02th May 2026
Published: 20th Jun 2026

Abstract: The skin's defense mechanism against opportunistic infections is compromised by burn damage. One of the primary pathogens that colonize burn wounds and cause serious infections is *Pseudomonas aeruginosa* . Production of Extended Spectrum Beta-lactamase Enzymes (ESBLs) and Integron play a significant part in time wastage and selecting the best course of treatment. Using 16sRNA, sixteen *P. aeruginosa* isolates were detected by the conventional PCR technique. Amikacin (85%), Imipenem (70%), Tobramycin (65%), Azteronam, Cepfepime (63%), Ceftazidime (61%), were shown to have the greatest resistance levels, and the mordent resistance was demonstrated by Levofloxacin, Meropenem (55%), and Ciprofloxacin, Colistin (46%). The findings of the Double Disc Synergy Test (DDST) indicated that 10 (62.25%) among the isolates generated (ESBLs). All isolates were classified as Multidrug Resistance (MDR) 16(100%). The most common genotypes were Int-1 16(100%), Bla$_{TEM}$ 16(100%), and Bla $_{CTX-M}$ 13 (81.2%). Because Enterobacteriaceae produced a large number of ESBL and Integron Class-1 genes, Slowing the spread of antibiotic-resistant illnesses requires the application of high standards in both public health and laboratory practices.

Keywords: *Pseudomonas aeruginosa* , Burn wound infections, Extended Spectrum Beta-Lactamases (ESBLs), Multidrug resistance (MDR), PCR detection

Introduction

One of the most prevalent and severe types of trauma are burns, which are typically brought on by electricity, heat, chemical contact, or radiation [1]. Burn patients are more likely to contract hospital-associated infections because burn injuries impair the skin's ability to fend against infection and interfere with the immune system's normal functioning [2]. Infected sites from burn wounds provide an optimal habitat for opportunistic organisms, both native and alien. Burn patients may

become infected for a number of reasons, such as prolonged hospital stays, immunocompromised conditions, invasive hospital procedures, and exposed body surfaces [3]. The most common reason why burns patients experience difficulties from their injuries is polymicrobial infections. These hospitalized patients are at a heightened risk of contracting hospital-acquired infections, and extended hospital stays raise the possibility of contracting such resistant to treatment illnesses. *Pseudomonas aeruginosa* is an opportunistic pathogen found in various environments, including water and soil. It colonizes burn wounds, exhibits resistance to multiple antibiotics, and survives in various environmental stress , contributing to its pathogenicity and difficulty in treatment [4]. It is a Gram-negative bacterium found in soil, water, plants, and people non-fermentative, and rod-shaped aerobic. This resilient microorganism can endure temperatures between 4 and 42°C and thrive in conditions deficient in nutrients. It can survive for up to six months on dry, abiotic surfaces in hospitals thanks to its tenacious adaption and survival [5].

P. aeruginosa may develop acquired or innate resistance mechanisms to certain antibiotics. The development of efflux pump systems, a reduction in the outer membrane permeability, and the generation of enzymes which inhibit antibiotics are the reasons of intrinsic resistance. Furthermore, *P. aeruginosa* picks up resistance to antibiotics by mutational alterations and horizontal gene transfer of resistance genes. This bacterium has acquired antibiotic resistance primarily do so through conjugation, transduction, and transformation of genes producing Metallo-β-lactamases (MBLs) and Extended-spectrum β-lactamases (ESBLs) [6,7]. β-lactamases are categorized into four classes, A through D, according to the molecular makeup of each class. The serine residue at the active site in Classes A, C, and D have a, they are also known as serine β-lactamases (SBLs), which aids in bond hydrolysis. Conversely, class B β-lactamases are referred to as metallo-βlactamases (MBLs) due to the fact that required in their active sites one or two zinc ions which enhance the hydrolytic activity [8]. Among the class A enzymes are the following: Temoniera, the patient, is the name of the first β-lactamase encoding a plasmid that has been discovered in Gram-negative bacteria, known as TEM. Sulfhydryl variant (SHV), Cefotaximase (CTXM), and *K. pneumoniae* carbapenemase (KPC), which produces carbapenem, are other enzymes with functions comparable to those of TEM [9]. Recently, it has been discovered that a novel class of transportable genetic components is capable of transmitting antibiotic resistance genes. This genetic component, known as an integron, has the ability to encircle and transmit genes while they are within gene cassettes [10].

The first part which is required for transcription, it is integrin associated promoter (Pc), and expression of gene cassettes within the integron, the nearby recombination site attI, which is recognized by integrase, and the intI gene, which encodes an integrase (IntI), which is required for site-specific recombination, are the three essential parts of integrons. The genetic elements that encode genes for antibiotic resistance, are also referred to as 59-base elements known as Gene cassettes. They consist of a particular type of recombination that integrase identifies as attC [11]. Based on their integrase genes, integrons are categorized into four classes, with class 1 integrons being the most common [12]. Owing to the significance of Bla$_{TEM}$, Bla $_{SHV}$, and Integron class-1 (*Int-1*) in the dissemination of genes and antibiotic resistance, the study sought to determine whether these genes were present in P.aeruginosa isolated from burn wound patients at Al Hussein Teaching Hospital in Samawah Province, Iraq.

Materials and Methods

A total of 130 pus samples were collected from patients treated to the burn's unit of Al Hussein Teaching Hospital in Samawah Province, Iraq, between January and August of 2024. Sterile cotton swabs were used to gather samples from the purulent burned skin (burn wounds should be cleaned before samples are taken). Once collected, each specimen was placed in 5 ml of sterile broth and moved as quickly as possible to the next step of the procedure. Blood agar plates and MacConkey agar plates were used to inoculate all specimens immediately after they were collected. A combination of standard biochemical assays including the molecular method, oxidase, catalase, triple sugar iron agar (TSI), and gram stains were performed to identify isolates of *Pseudomonas aeruginosa* .

Susceptibility to Antibiotics

In this work, we employed an agar diffusion approach to characterize the antimicrobial susceptibility of bacteria and interpreted the results according to those outlined in the Clinical and Laboratory Standards Institute (CLSI) (CLI 2020) [13]. As part of this method, we placed 10 commercial antibiotic discs containing Aztreonam (ATM), Amikacin (AK), Ciprofloxacin (CIP), Cefepime (FEP) and Ceftazidime (CAZ) on each agar plate. Colistin (CT), Imipenem (IM), Levofloxacin (LEV), Meropenem (MEM) and Tobramycin (TOB) were also included in this process.

Extended-Spectrum β-Lactamases: phenotypic characterization

We phenotypically identified ESBLs by using the Double Disc Synergy Test as detailed in CLSI (2020) where we placed Amoxicillin/Clavulanic acid discs (20µg of Amoxicillin and 10µg of Clavulanic acid) in the center of our inoculated plates [14] and then placed Cefotaxime (30 µg) and Ceftazidime (30 µg) 20 mm apart on either side of the Amoxicillin/Clavulanic Acid disc. After then, the plates were maintained at 37°C for an additional day. An expansion of inhibition zone surrounding any cephalosporin by approximately 5 millimeters toward the Amoxicillin-Clavulanic acid disc indicated the development of ESBLs in the organisms [9].

Bacterial DNA extraction

Observing the guidelines provided by the manufacturer bacterial isolates had their bacterial DNA extracted using a DNA extraction kit (EasyPure® Bacteria Genomic DNA Kit/China). Ethidium bromide-stained 1% agarose gel electrophoresis was performed on the isolated DNA [15]. Using universal bacterial 16SrRNA primers, the *P. aeruginosa* 16 sRNA, ESBL genes [bla$_{CTXM}$, bla$_{TEM}$] and Integron gene [*Int-1*]) gene were amplified by PCR [16]. Targeted DNA was amplified using PCR reaction conditions in a 50 µl total volume reaction (tables 1, 2, 3, and 4) [17].

Table 1: *P.aeruginosa* 16sRNA Primer

Primer Sequences 5' ------'3	PCR product	Reference
F: TGCCTGGTAGTGGGGGATAA R: GGATGCAGTTCCCAGGTTGA `	**505 bp**	**[15]**

Table 2: Reaction Mix (50 microliters)

Component	Volume 50 µl
2xEasyTaq® PCR Super Mix	**25µl**
Forward primer	**1 µl**
Revers primer	**1 µl**
DNA	**2 µl**
Nuclease free water	**21 µl**
Total	**50 µl**

Table 3: Conditions for the PCR

Step	Temperature(˚C)	Time	No. of Cycles
Initial denaturation	**94**	**3 min**	1
Denaturation	**94**	**30sec**	35
Annealing	**Variables according to (primer's TM)**	**30 sec**	
Extension	**72**	**1 min**	
Final extension	**72**	**5 min**	1
Hold temperature	**4**	**∞**	-

Table 4: ESBLs and Integron genes were detected using primers.

Gene Name	Sequences Of Primer 5' ------'3	Size of Amplicon	Source
Bla CTX-M	**F GACGATGTCACTGGCTGAGC** **R AGCCGCCGACGCTAATAC**	**499 bp.**	**[18]**
Int 1	**F: GGTGTGGCGGGCTTCGTG** **R: GCATCCTCGGTTTTCTGG**	**480 bp.**	**[19]**
Bla TEM	**F: ATGAGTATTCAACATTTCCGTG** **R: TTACCAATGCTTAATCAGTGAG**	**861 bp.**	**[20]**

Results

P. aeruginosa infections hinder healing, result in invasive infections in burn patients, and even kill them. 16 (12.30%) of the 130 burn wound swabs had a positive results for *P.aeruginosa* by PCR method (figure 1); All test isolates were found to be resistant to three or more antibiotic classes, as demonstrated by disc diffusion data; as a result, they were classified as multidrug resistant (MDR), The highest resistance percentage in our findings were recorded for Amikacin and followed with that for Imipenem, Tobramycin, Azteronam, Cepfepime ,Ceftazidime, Levofloxacin, Meropenem , while the lowest one were recorded for Ciprofloxacin, Colistin Antibiotics (table 5).

The ESBLs producers by DDST in total 10(62.5%) in the current investigation (figure 2). As a result, it is advised to identify ESBL-positive isolates using the genotypic approach. Of all the isolates found in the current investigation, 13 (81.2%) and 16 (100%), respectively, had Bla CTXM and Bla TEM (figures 3 and 4). The current investigation's use of PCR amplification to screen for the presence of a certain class of integrase genes revealed that 16 (100%) of the isolates of *P.aeruginosa* had the int1 gene (figure 5).

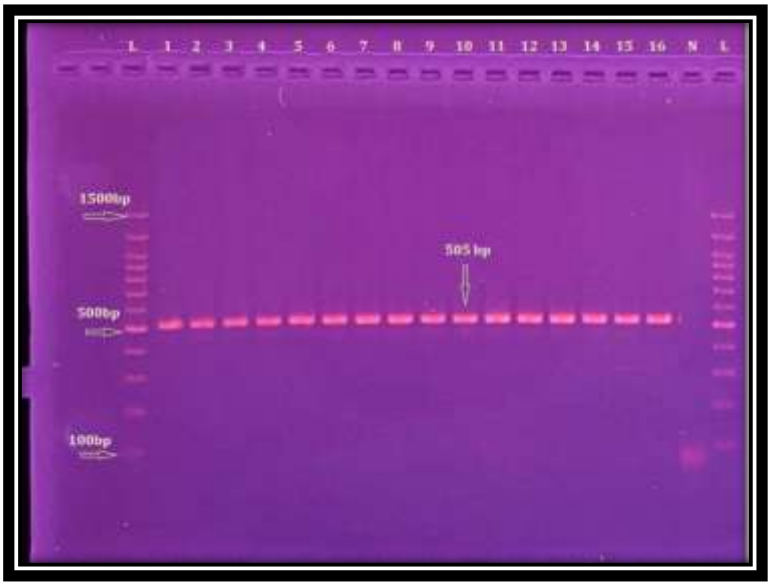

Figure-1: Gel Electrophoresis Image for *Pseudomonas aeruginosa* Primer

Table-5: Antibiotics Resistance Patterns for *Pseudomonas aeruginosa*

No.	Antibiotic Name	Resistance %
1.	Amikacin	**85%**
2.	Imipenem	**70%**
3.	Tobramycin	**65%**
4.	Azteronam	**63%**
5.	Cepfepime	**63%**
6.	Ceftazidime	**61%**
7.	Levofloxacin	55%
8.	Meropenem	55%
9.	Ciprofloxacin	41%
10.	Colistin	41%

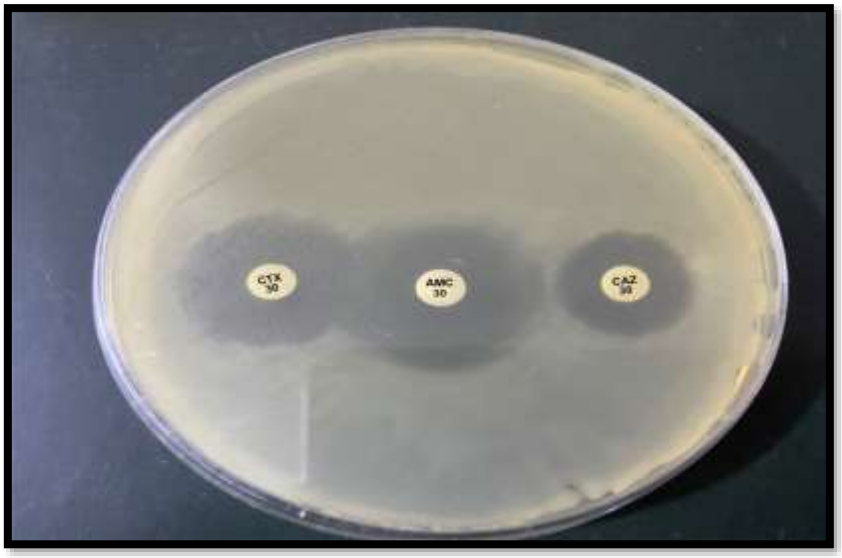

Figure 2: Phenotype ESBLs produced by *Pseudomonas aeruginosa* using DDST

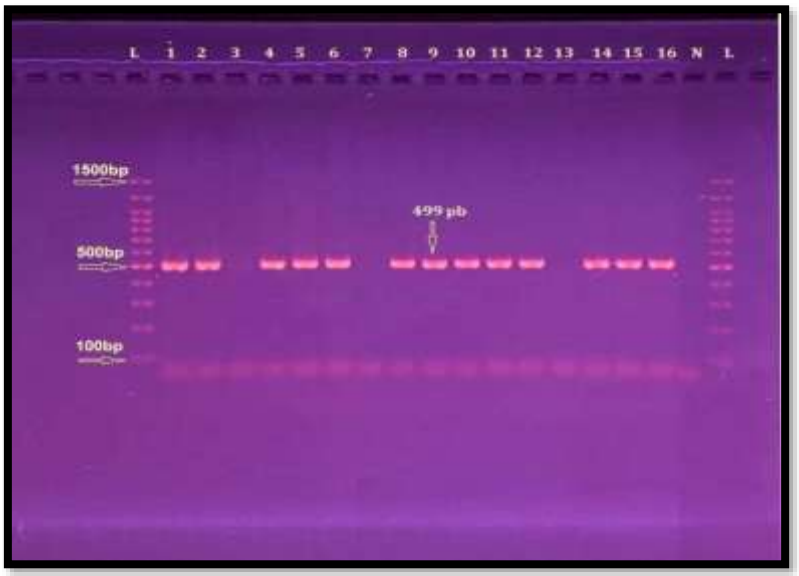

Figure 3: *Pseudomonas aeruginosa* Bla CTXM Primer Gel Electrophoresis Image

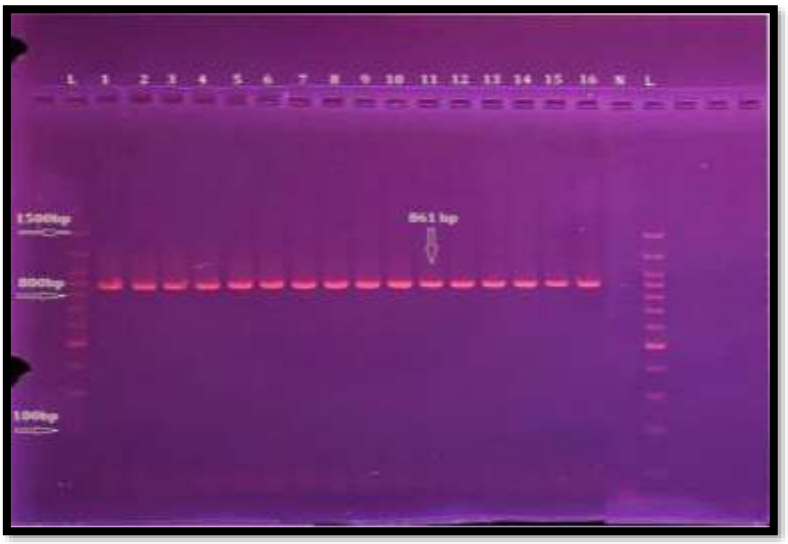

Figure 4: *Pseudomonas aeruginosa* Bla TEM Primer Gel Electrophoresis Image

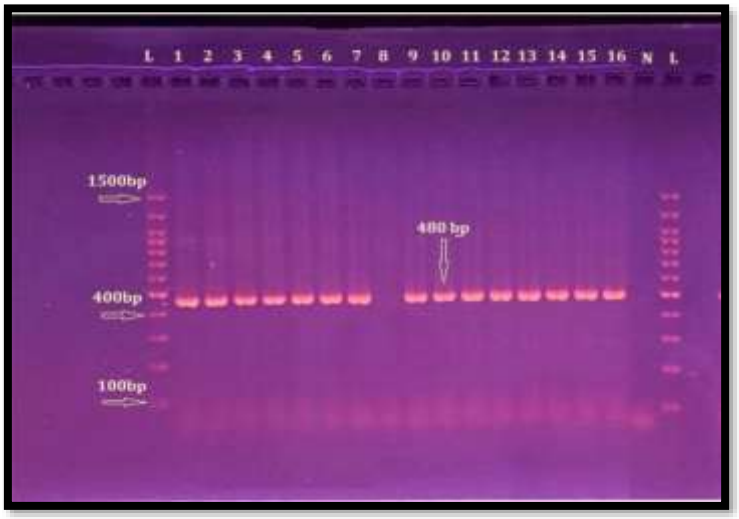

Figure 5: *Pseudomonas aeruginosa Int-1* Primer Gel Electrophoresis Image

A thorough examination of phenotypic and molecular data revealed a consistent and systematic resistance pattern among *Pseudomonas aeruginosa* isolates derived from burn wound infections. All isolates classified as multidrug-resistant exhibited resistance to multiple classes of antibiotics simultaneously, reflecting a widespread and stable resistance pattern rather than isolated or transient resistance events. High rates of resistance to aminoglycosides, carbapenems, and cephalosporins were particularly evident, indicating limited treatment options and underscoring the severity of the clinical condition associated with these infections.

A large number of isolates have been found to produce broad-spectrum beta-lactamases (ESBLs) according to phenotypic testing and the molecular testing strongly supported this phenotype. The high frequency of blaTEM and blaCTX-M genes can now provide definitive proof for the mechanism of resistance to beta-lactam antibiotics (broad-spectrum cephalosporin antibiotics). In addition, all isolates have an integron class I (intI1) gene, indicating a major role of the integron genetic element in establishing the resistance profile of *Pseudomonas aeruginosa* in burn units. The frequent co-

location of ESBL genes with integrons in the same isolate is indicative of an increased gene acquisition potential and an increased capability to maintain and express multiple resistance determinants. Such genetic structures likely result in enhanced horizontal gene transfer and provide the potential for persistence of multi-resistant strains in the face of constantly applied antibiotic selection pressures. Overall, these results demonstrate that antibiotic resistance in the studied isolates is genetically driven, highly structured, and widespread, highlighting a complex molecular basis.

Discussion

One of the primary issues with burn victims is *Pseudomonas aeruginosa* , an opportunistic bacterium that can cause serious nosocomial infections. This bacterium is frequently resistant to several medications and has extraordinary antimicrobial resistance. The current isolation percentage is consistent with findings who obtained 12% of *P.aeruginosa* from burn patients ,while [21] obtained 13.7% of *P.aeruginosa* from pus [22] while a study conducted by Othman *et al* . recorded higher isolation percentage than in our study, about 37% [23].

When treating nosocomial infections, there is growing worried over the proliferation of genes that confer antibiotic resistance among bacteria, including strains of *P. aeruginosa* [24]. Transposable resistance genes, which are mostly mediated by plasmids, are primarily responsible for resistance. These genes are said to have been encapsulated from the chromosomes of several bacterial species and shifted "horizontally" between bacteria by means of distinct mobile genetic components. A bacterial cell can acquire multiple resistance in a single movement when several resistance genes are carried on a single plasmid [25].

In addition, Antibiotic overuse and abuse in hospitals can lead to particular mutations that cause bacteria to become multidrug resistant to different antibiotics, especially when patients are receiving broad range or multiple medications. Abdi *et al*.recorded that about 33.33% of *P.aeruginosa* were MDR (1). and [26] reported that 76.8% of *P.aeruginosa* were MDR. The antibiotics resistance findings in the current study were consistent with other research, including that of [27-30].

The current (ESBLs) results concurred with [31] that reported that 70.73% of *P.aeruginosa* were ESBLs producers, While [32] reported that 28.6% of *P.aeruginosa* were ESBLs producers [32] and [33] reported that about 25% of *P.aeruginosa* were ESBLs producers , Despite being regarded as a dependable technique for ESBL detection, DDST has been demonstrated to have issues because the disc positioning distance is not standardized.

Another theory is that AmpC enzymes may target cephalosporins due to clavulanate, which stimulates them but does not effectively inhibit them, masking the synergy created by the inhibition of the ESBL. Using the phenotypic approach could lead to false negative results and interfere with the course of treatment because illnesses brought on by ESBL-positive bacteria can be treated with β-lactam antibiotics. These findings are consistent with (30) obtained 14.3% and 9.6% for Bla $_{CTXM}$ and Bla $_{TEM}$ respectively (36 ,While (34) obtained 41.6% and 86% for Bla $_{CTXM}$ and Bla $_{TEM}$ respectively .

Several resistance genes are present as gene cassettes on transposons and transmissible plasmids called integrons. The outcome of int I gene is consistent with (35) who reported that 92% of all isolates harbor the int1 gene recorded that 90% of *P.aeruginosa* had the int1 gene , and (36) reported that 95.7% of *P.aeruginosa* had the int1 gene . In light of our findings, the percentage of MDR isolates among integron-positive isolates was significantly greater (almost 100%), confirming the significance of these elements in the transmission of resistance genes across pathogens (13) revealed a significant proportion of MDR isolates among integron-positive, which is consistent with our data.

The results of this study indicate that antibiotic resistance in *Pseudomonas aeruginosa* burn-associated bacteria is caused by stable and coordinated genetic mechanisms, rather than sporadic resistance events. The concurrent presence of genes encoding broad-spectrum beta-lactamases (ESBLs) and class I introns suggests an enhanced ability of these isolates to acquire, maintain, and express multiple resistance factors under sustained antibiotic pressure.

Due to the unique environmental characteristics of burn units, it is a specialized setting where genetically modified organisms can thrive for extended periods of time and can then be easily spread. The presence of these genetically modified organisms (GMOs) has created challenges in the treatment of patients and ultimately increases the likelihood of the patient experiencing failure during the course of their treatment. This highlights the need to integrate molecular surveillance with antimicrobial stewardship programs when treating patients with multi-drug resistant *Pseudomonas aeruginosa* at any burn facility.

Conclusion

The high incidence of ESBLs, an enzyme produced by *P. aeruginosa* that is resistant to drugs, in current clinical settings is highlighted by the current study. Since the isolates generating these enzymes frequently have a sensitive phenotype in standard susceptibility testing, early discovery of these extended spectrum beta-lactamase producing isolates in a routine laboratory could help to avert treatment failure. The *int1* gene was widely distributed and appears to be crucial in the isolates' multidrug resistance. Antibiotic monitoring program implementation is therefore essential for selecting the right treatment and overseeing infection control procedures.

REFERENCES

[1] F. A. Abdi, A. N. Motumma, A. A. Kalayu, and W. E. Abegaz, "Prevalence and antimicrobial-resistant patterns of Pseudomonas aeruginosa among burn patients attending Yekatit 12 Hospital Medical College in Addis Ababa, Ethiopia," PLoS One, vol. 19, no. 3, p. e0289586, 2024, doi: 10.1371/journal.pone.0289586.

[2] D. Church, S. Elsayed, O. Reid, B. Winston, and R. Lindsay, "Burn wound infections," Clin. Microbiol. Rev., vol. 19, no. 2, pp. 403–434, Apr. 2006, doi: 10.1128/CMR.19.2.403-434.2006.

[3] N. A. Chaudhary, M. D. Munawar, M. T. Khan, K. Rehan, A. Sadiq, A. Tameez-Ud-Din, et al., "Epidemiology, bacteriological profile, and antibiotic sensitivity pattern of burn wounds in the burn unit of a tertiary care hospital," Cureus, vol. 11, no. 6, p. e4794, Jun. 2019, doi: 10.7759/cureus.4794.

[4] F. Jaafar, N. Bashar, L. Alhusseini, and H. Musafer, "Infections with Pseudomonas aeruginosa in burn patients: The host immune response," South Asian Res. J. Biol. Appl. Biosci., vol. 5, pp. 15–21, 2023.

[5] S. P. Diggle and M. Whiteley, "Microbe Profile: Pseudomonas aeruginosa: opportunistic pathogen and lab rat," Microbiology, vol. 166, no. 1, pp. 30–33, Jan. 2020, doi: 10.1099/mic.0.000860.

[6] E. B. Breidenstein, C. de la Fuente-Núñez, and R. E. W. Hancock, "Pseudomonas aeruginosa: All roads lead to resistance," Trends Microbiol., vol. 19, no. 8, pp. 419–426, 2011, doi: 10.1016/j.tim.2011.04.005.

[7] R. E. W. Hancock and D. P. Speert, "Antibiotic resistance in Pseudomonas aeruginosa: mechanisms and impact on treatment," Drug Resist. Updat., vol. 3, no. 4, pp. 247–255, Aug. 2000, doi: 10.1054/drup.2000.0152.

[8] H. Jassim, "The prevalence of blaCTX-M, blaTEM genes in bacteria isolated from bladder cancer patients with urinary tract infections," Univ. Thi-Qar J. Sci., vol. 10, 2023.

[9] H. Jassim, Y. Alkhafaji, and H. Alkafaji, "Assessment of beta-lactamases and integrons genes among bacteria isolated from bladder cancer patients with urinary tract infections," J. Popul. Ther. Clin. Pharmacol., pp. 9–23, 2023.

[10] A. C. Fluit and F. J. Schmitz, "Resistance integrons and super-integrons," Clin. Microbiol. Infect., vol. 10, no. 4, pp. 272–288, Apr. 2004, doi: 10.1111/j.1198-743X.2004.00858.x.

[11] S. Domingues, G. J. da Silva, and K. M. Nielsen, "Integrons: Vehicles and pathways for horizontal dissemination in bacteria," Mob. Genet. Elements, vol. 2, no. 5, pp. 211–223, Sep. 2012, doi: 10.4161/mge.22967.

[12] S. R. Partridge, G. D. Recchia, H. W. Stokes, and R. M. Hall, "Family of class 1 integrons related to In4 from Tn1696," Antimicrob. Agents Chemother., vol. 45, no. 11, pp. 3014–3020, Nov. 2001, doi: 10.1128/AAC.45.11.3014-3020.2001.

[13] M. Ebrahimpour, I. Nikokar, Y. Ghasemi, H. Sedigh Ebrahim-Saraie, A. Araghian, M. Farahbakhsh, et al., "Antibiotic resistance and frequency of class 1 integrons among Pseudomonas aeruginosa isolates obtained from wastewaters of a burn center in Northern Iran," Ann. Ig., vol. 30, no. 2, pp. 112–119, 2018, doi: 10.7416/ai.2018.2202.

[14] A. Schuetz, A. Ferrell, J. Hindler, R. Humphries, and A. Bobenchik, "Overview of changes in the Clinical and Laboratory Standards Institute Performance Standards for Antimicrobial Susceptibility Testing: M100 32nd and 33rd editions," J. Clin. Microbiol., vol. 63, 2025.

[15] T. Hussein Ali, A. Mousa Mandal, A. Alhasan, and W. Dehaen, "Surface fabrication of magnetic core-shell silica nanoparticles with perylene diimide as a fluorescent dye for nucleic acid visualization," J. Mol. Liq., vol. 359, p. 119345, 2022, doi: 10.1016/j.molliq.2022.119345.

[16] L. Shaabth, "Molecular identification and sequencing of Pseudomonas aeruginosa virulence genes among different isolates in Al-Diwaneyah hospital," Iraqi J. Vet. Sci., vol. 32, pp. 183–188, 2019.

[17] H. Kazemian, H. Heidari, R. Ghanavati, S. Ghafourian, F. Yazdani, N. Sadeghifard, et al., "Phenotypic and genotypic characterization of ESBL-, AmpC-, and carbapenemase-producing Klebsiella pneumoniae and Escherichia coli isolates," Med. Princ. Pract., vol. 28, no. 6, pp. 547–551, 2019, doi: 10.1159/000500311.

[18] S. M. Diene and J. M. Rolain, "Carbapenemase genes and genetic platforms in Gram-negative bacilli: Enterobacteriaceae, Pseudomonas and Acinetobacter species," Clin. Microbiol. Infect., vol. 20, no. 9, pp. 831–838, Sep. 2014, doi: 10.1111/1469-0691.12655.

[19] M. Bahrami, M. Mohammadi-Sichani, and V. Karbasizade, "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., vol. 5, pp. 86–90, 2018.

[20] M. Zarei-Yazdeli, G. Eslami, H. Zandi, M. Kiani, K. Barzegar, H. Alipanah, et al., "Prevalence of class 1, 2 and 3 integrons among multidrug-resistant Pseudomonas aeruginosa in Yazd, Iran," Iran. J. Microbiol., vol. 10, no. 5, pp. 300–306, Oct. 2018.

[21] M. Musyoki, M. Masika, W. Mutai, G. Wilfred, A. Kuria, and F. Muthini, "Antimicrobial susceptibility pattern of Acinetobacter isolates from patients in Kenyatta National Hospital, Nairobi, Kenya," Pan Afr. Med. J., vol. 33, 2019, doi: 10.11604/pamj.2019.33.146.16527.

[22] Y. Bayram, M. Parlak, C. Aypak, and I. Bayram, "Three-year review of bacteriological profile and antibiogram of burn wound isolates in Van, Turkey," Int. J. Med. Sci., vol. 10, no. 1, pp. 19–23, 2013, doi: 10.7150/ijms.4723.

[23] N. Othman, M. Babakir-Mina, C. Noori, and P. Rashid, "Pseudomonas aeruginosa infection in burn patients in Sulaimaniyah, Iraq: Risk factors and antibiotic resistance rates," J. Infect. Dev. Ctries., vol. 8, no. 11, pp. 1498–1502, 2014, doi: 10.3855/jidc.4886.

[24] B. Gu, M. Tong, W. Zhao, G. Liu, M. Ning, S. Pan, et al., "Prevalence and characterization of class I integrons among Pseudomonas aeruginosa and Acinetobacter baumannii isolates from patients in Nanjing, China," J. Clin. Microbiol., vol. 45, no. 1, pp. 241–243, Jan. 2007, doi: 10.1128/JCM.01318-06.

[25] M. H. Dirar, N. E. Bilal, M. E. Ibrahim, and M. E. Hamid, "Prevalence of extended-spectrum β-lactamase (ESBL) and molecular detection of blaTEM, blaSHV and blaCTX-M genotypes among Enterobacteriaceae isolates from patients in Khartoum, Sudan," Pan Afr. Med. J., vol. 37, p. 213, 2020, doi: 10.11604/pamj.2020.37.213.24584.

[26] P. Bhatt, K. Rathi, S. Hazra, A. Sharma, and V. Shete, "Prevalence of multidrug resistant Pseudomonas aeruginosa infection in burn patients at a tertiary care centre," Indian J. Burns, vol. 23, p. 56, 2015, doi: 10.4103/0971-653X.171977.

[27] M. Hakemi Vala, M. Hallajzadeh, A. Hashemi, H. Goudarzi, M. Tarhani, M. Sattarzadeh Tabrizi, et al., "Detection of Ambler class A, B and D β-lactamases among Pseudomonas aeruginosa and Acinetobacter baumannii clinical isolates from burn patients," Ann. Burns Fire Disasters, vol. 27, no. 1, pp. 8–13, Mar. 2014.

[28] M. Ahmad, M. Hassan, A. Khalid, I. Tariq, M. H. H. Bin Asad, A. Samad, et al., "Prevalence of extended spectrum β-lactamase and antimicrobial susceptibility pattern of clinical isolates of Pseudomonas from patients of Khyber Pakhtunkhwa, Pakistan," Biomed Res. Int., vol. 2016, p. 6068429, 2016, doi: 10.1155/2016/6068429.

[29] A. Kotwal, D. Biswas, B. Kakati, and M. Singh, "ESBL and MBL in cefepime resistant Pseudomonas aeruginosa: An update from a rural area in Northern India," J. Clin. Diagn. Res., vol. 10, no. 4, pp. DC09–DC11, Apr. 2016, doi: 10.7860/JCDR/2016/17742.7578.

[30] Z. Chen, H. Niu, G. Chen, M. Li, M. Li, and Y. Zhou, "Prevalence of ESBLs-producing Pseudomonas aeruginosa isolates from different wards in a Chinese teaching hospital," Int. J. Clin. Exp. Med., vol. 8, no. 10, pp. 19400–19405, 2015.

[31] X. Jiang, Z. Zhang, M. Li, D. Zhou, F. Ruan, and Y. Lu, "Detection of extended-spectrum beta-lactamases in clinical isolates of Pseudomonas aeruginosa," Antimicrob. Agents Chemother., vol. 50, no. 9, pp. 2990–2995, Sep. 2006, doi: 10.1128/AAC.01511-05.

[32] A. Peymani, T. Naserpour-Farivar, E. Zare, and K. H. Azarhoosh, "Distribution of bla(TEM), bla(SHV), and bla(CTX-M) genes among ESBL-producing P. aeruginosa isolated from Qazvin and Tehran hospitals, Iran," J. Prev. Med. Hyg., vol. 58, no. 2, pp. E155–E160, Jun. 2017.

[33] S. M. Abdelaziz, K. M. Aboshanab, I. S. Yahia, M. A. Yassien, and N. A. Hassouna, "Correlation between the antibiotic resistance genes and susceptibility to antibiotics among the carbapenem-resistant Gram-negative pathogens," Antibiotics, vol. 10, no. 3, 2021, doi: 10.3390/antibiotics10030255.

[34] K. Ghaima, A. Abdulhassan, and Z. Mahdi, "Molecular study of extended spectrum beta-lactamase (ESBL) genes in Pseudomonas aeruginosa isolated from burns," Biochem. Cell. Arch., vol. 18, 2018.

[35] P. Hosseini Pour, H. Momtaz, A. A. Serajyan, and E. Tajbakhsh, "Investigating class I, II and III integrons in multidrug resistance in Pseudomonas aeruginosa isolated from hospital infections in Ahvaz," Int. J. Med. Lab., vol. 2, no. 3, 2015.

[36] A. D. Khosravi, M. Motahar, and E. Abbasi Montazeri, "The frequency of class 1 and 2 integrons in Pseudomonas aeruginosa strains isolated from burn patients in a burn center of Ahvaz, Iran," PLoS One, vol. 12, no. 8, p. e0183061, 2017, doi: 10.1371/journal.pone.0183061.