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Correlation between Quorum Sensing Genes (lasI and lasR) and Antibiotics Resistance in *Pseudomonas aeruginosa* Isolated from Wound Infections

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Abstract

Background *Pseudomonas aeruginosa* (*P. aeruginosa*) is a common etiological agent of wound infection; it has been shown to increase the rate of mortality and morbidity in patients. It can potentially become multidrug-resistant (MDR) due to its ability to acquire different antimicrobial resistance mechanisms.

Objective To investigate the impact of Las Systems (lasI and lasR) on antibiotic resistance patterns in *P. aeruginosa*.

Methods Out of 117 clinical wound samples, 30 *P. aeruginosa* isolates were identified, and the antibiotic susceptibility and minimum inhibitory concentration (MIC) determination were evaluated. The conventional polymerase chain reaction (PCR) method was used for the detection of quorum-sensing genes using specific primer sequences after DNA extraction.

Results Out of the 30 *P. aeruginosa* isolates, 43% were identified as extensively drug-resistant (XDR), while 17% were classified as MDR. The highest level of antibiotic resistance was observed against Levofloxacin (60%), and the lowest level was against Colistin (23%). The occurrence of quorum-sensing genes among the isolates was as follows: lasI (96.67%), lasR (76.67%).

Conclusion No statistically significant correlation was observed between the presence of these genes and MDR or XDR phenotypes.

Keywords Pseudomonas aeruginosa, quorum-sensing, las genes, lasI, lasR

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List of abbreviations: MDR = MultiDrug-resistance, MDRPA = MDR *Pseudomonas aeruginosa*, MIC = Minimum Inhibitory Concentration, Qs = Quorum sensing, *P. aeruginosa* = *Pseudomonas aeruginosa*, PCR = Polymerase chain Reaction, XDR = Extensively drug-resistant, XDRPA = Extensively drug-resistant *Pseudomonas aeruginosa*

Introduction

Wound infections are a substantial burden in healthcare settings, especially when caused by multidrug-resistant (MDR) organisms. Among these, *Pseudomonas aeruginosa* (*P. aeruginosa*) remains a major opportunistic pathogen, often linked with chronic burn, and

surgical wound infections [1]. Its intrinsic resistance to a wide range of antibiotics, coupled with its ability to rapidly acquire new resistance mechanisms, makes treatment of *P. aeruginosa* infections particularly challenging [2].

P. aeruginosa has become a clinically significant bacterial pathogen in the past ten years, exhibiting widespread resistance to conventional antibiotics that has resulted in major global health problems [3].

One of the key factors contributing to the pathogenicity and antibiotic tolerance of *P. aeruginosa* is its quorum sensing (QS) system; a sophisticated bacterial communication mechanism that regulates the expression of various virulence factors [4]. The QS system in *P. aeruginosa* involves a network of signaling molecules, primarily N-acyl homoserine lactones (AHLs), and key regulatory genes such as lasI, lasR, rhlI, and rhlR [3]. The las system, consisting primarily of the lasI and lasR genes, is the top tier of this hierarchical QS network. It governs the production of elastases, proteases, and biofilms, all of which enhance the organism's ability to colonize, evade host defenses, and resist antibiotic treatment [5].

However, more than a third of *P. aeruginosa* infections are caused by extensively drug-resistant (XDR) and MDR strains, which have become more common in recent years. According to hospital data, 13% of serious infections linked to healthcare are caused by MDR *P. aeruginosa* (MDRPA); understanding the mechanisms underlying *P. aeruginosa* resistance is the top priority in the fight against it [6].

Many studies have referred to QS systems that may impact antibiotic resistance, directly by acting on efflux pumps regulation and indirectly through enhanced biofilm formation [7,8]. However, the precise relationship between las gene activity and antibiotic resistance remains unclear, especially in clinical isolates from wound infections [9].

This study aimed to explore the presence of lasI and lasR genes in *P. aeruginosa* isolates from wound infections and to assess their association with antibiotic resistance patterns.

Methods

The cross-sectional study was carried out between November 2024 and February 2025. A total of 30 *P. aeruginosa* clinical isolates were collected from 117 wound infection patients admitted to four hospitals in Baghdad, which has frequently served as a major referral center for patients from Baghdad and its suburbs.

Ethical approval was sought and given by the Institutional Review Board (IRB) in College of Medicine, Al-Nahrain University (No. IRB/38/0380).

The isolated bacteria were first determined according to morphological characteristics of the colonies, shape, size, odor, color, and pigment production. The final confirmation of isolates was achieved using the VITEK-2 system, which is also used for antibiotic susceptibility evaluation based on the company directions (BioMerieux, France).

Molecular detection of genes (lasI and lasR)

The diagnosed *P. aeruginosa* isolates were used for Genomic DNA extraction from a fresh overnight culture on Luria Bertani using the Geneaid Presto™ Mini gDNA Bacteria Kit (Taiwan). After that, DNA templates were used to target the (lasI, lasR) genes using specific primer sequences (Macrogen Korea), which are listed in table (1). The polymerase chain reaction (PCR) products were documented using electrophoresis on a 1.5% agarose gel (70 V, 60 min) (BDH England) plus ethidium bromide (0.3 µg/ml), and size forecasted by a DNA ladder (100-1000 bp) (Promega USA). The composition of the conventional PCR reaction mixture and the PCR thermocycling conditions employed in this study are summarized in tables (2 and 3), respectively.

Table 1. Primer sequence, thermal profile for detection of las gen

Gene	Primer (5′ 3′)	Amplicon size (pb)	Reference
lasI	F- CGT GCT CAA GTG TTC AAG G R- TAC AGT CGG AAA AGC CCA G	295	(10)
lasR	F- AAG TGG AAA ATT GGA GTG GAG R- GTA GTT GCC GAC GAC GAT GAAG	130	(10)

Table 2. The mixture of conventional PCR working

No	Components	Volume/µl	Concentration
1	Master Mix	0	
2	Forward Primer	1	10 µM
3	Reverse Primer	1	10 µM
4	Nuclease Free Water	16	
5	DNA	2	≤ 100 ng
	Total	20	

Table 3. The PCR thermos cycler program

Target gene	Initial denaturation (Temperature /time)	Denaturation (Temperature /time)	Annealing (Temperature /time)	Extension (Temperature /time)	Final extension (Temperature /time)	Cycle
lasI	94 °C/5 min	94 °C/1 min	56 °C/1 min	72 °C/1 min	72 °C/8 min	32
lasR	94 °C/5 min	95 °C/1 min	56 °C/50 s	72 °C/1 min	72 °C/8 min	32

Statistical analysis

The statistical package for social science (SPSS) version 24 (Chicago, USA) was used to analyze and present data. Categorical data is described by frequency and percentage. Chi-square was applied to find the correlation between two categorical variables. A p value of <0.05 was considered statistically significant.

Results

Antibiotic resistance of *P. aeruginosa*

Among 30 *P. aeruginosa isolates*, the resistance was to Levofloxacin (60%), Ciprofloxacin (57%), and Imipenem (53%). Moderate resistance was seen as Piperacillin and Ceftazidime (50%), Gentamicin (47%), and Amikacin (43%). Lower rates were noted for Cefepime (40%), Meropenem and Piperacillin-Tazobactam were (37%), with Colistin (23%) as shown in figure (1).

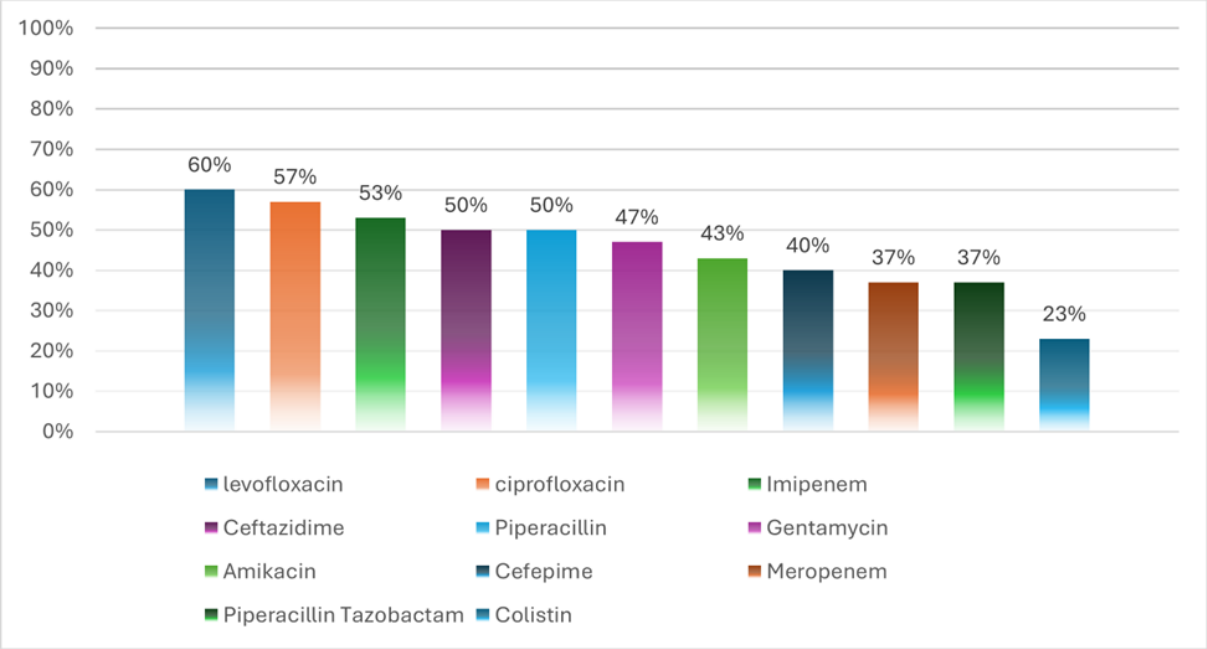

Figure 1. Antibiotic resistance rates of *P. aeruginosa* isolates from wound infection

Antibiotic resistance patterns of *P. aeruginosa*

The antibiotic resistance profile of the bacterial isolates revealed significant levels of XDR strains accounted for 13 isolates (43%), representing the highest proportion. Non-MDR/Non-XDR strains are bacteria that are not resistant to multiple classes of antibiotics, and they remain susceptible to most used antibiotics. Made up to 12 (40%) of the isolates. Additionally, MDR strains were identified in 5 (17%) of the cases (Figure 2).

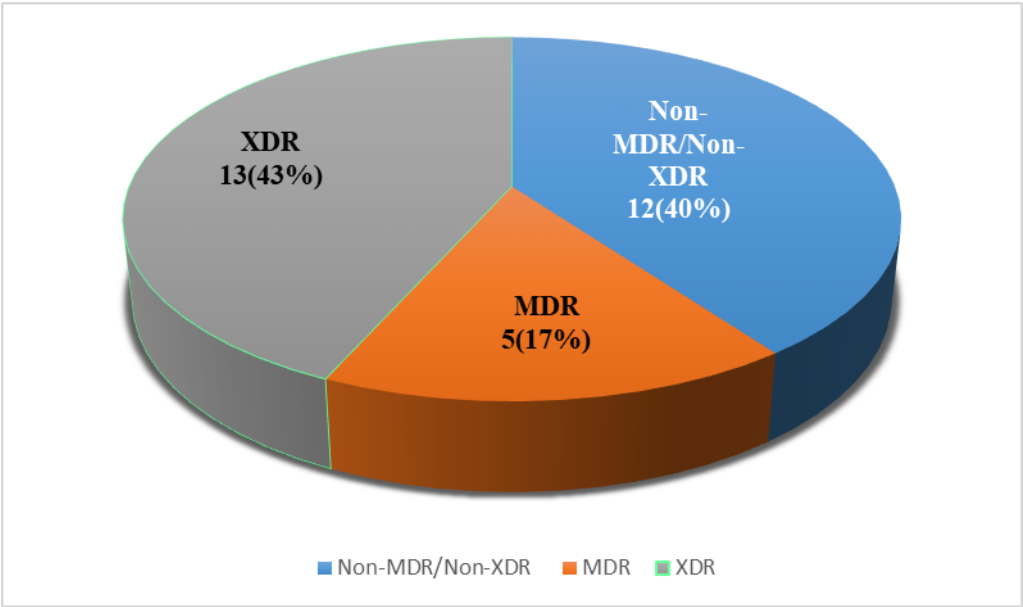

Figure 2. Distribution of antibiotic resistance patterns among *P. aeruginosa*
molecular detection of Las system Genes in *P. aeruginosa*

Molecular detection of Las system genes in *P. aeruginosa*

Molecular detection of QS genes under study in 30 *P. aeruginosa* isolates was 29 (96.67%) for lasI (295 bp), and 23 (76.67%) for lasR (130 bp) (Figures 3 and 4).

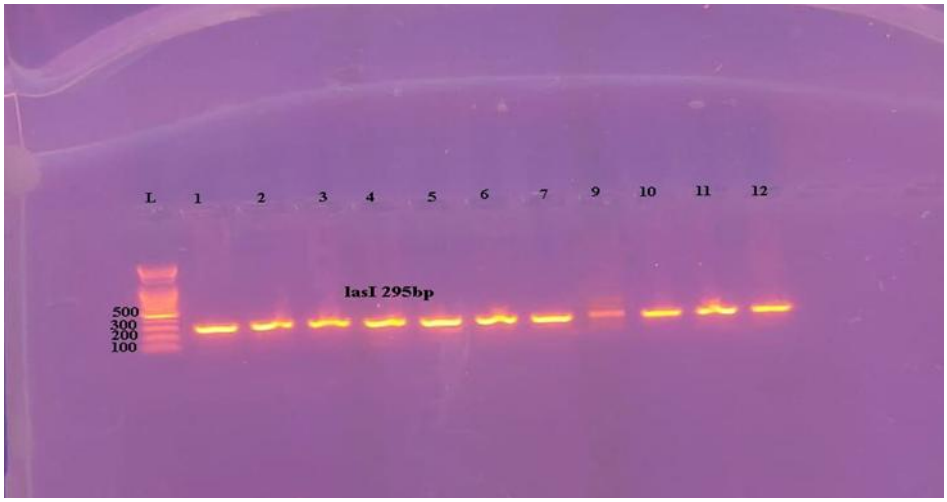

Figure 3. Gel electrophoresis (1.5 % agarose,70 V/cm for 60 min) amplification of the lasI gene, (L) 100 bp ladder lanes (1-12) PCR product at 295 bp

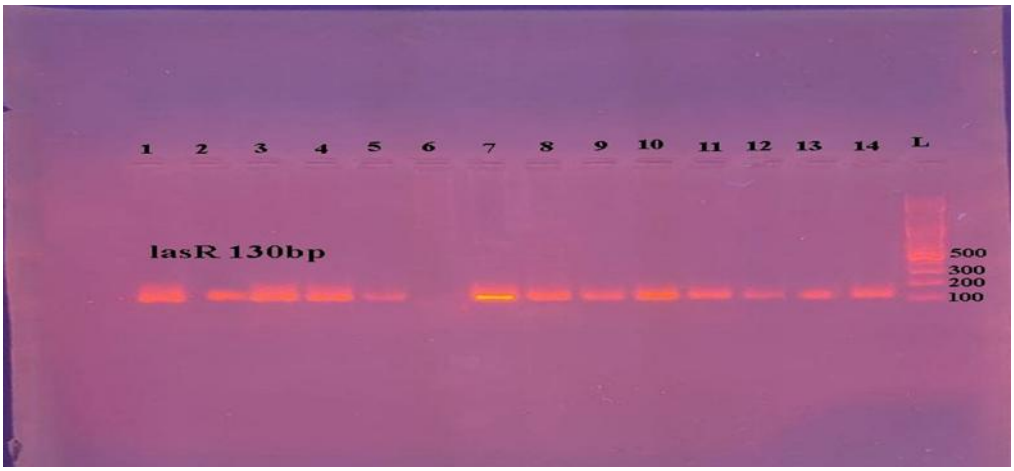

Figure 4. Gel electrophoresis (1.5 % agarose,70 V/cm for 60 min), amplification of the lasR gene, (L) 100 bp ladder lanes (1-14) PCR product at 130 bp

Correlation between las system genes (lasI, lasR) and antibiotic resistance patterns

The result indicates that the majority of XDR and MDR strains have las QS genes. However, lasI was positive in 5 (100%) MDR and 12 (92.3%) XDR, while lasR was positive in 4 (80%) and 10 (76.9%) respectively. Statistical analysis shows no significant correlation between the presence of these las QS and XDR, MDR ($p > 0.05$ for both genes) as shown in figure (5).

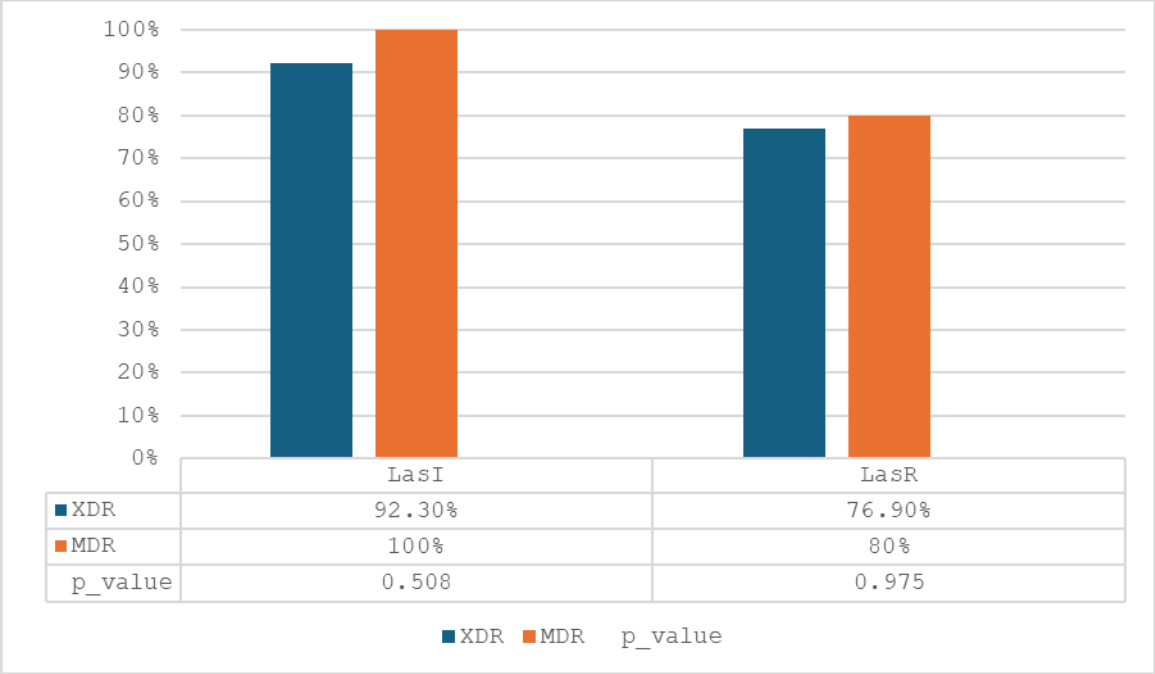

Figure 5. correlation between lasI, lasR and XDR, MDR for p. aeruginosa

Discussion

Antibiotic Susceptibility of *P. aeruginosa*

In this study, the highest resistance was observed for Levofloxacin (60%) and Ciprofloxacin (57%), which agree with the study in Baghdad, Iraq by Wadi and Ali, who recorded highest resistance 90% Levofloxacin and 77.5 % to Ciprofloxacin, and lowest resistance to Colistin was 22.5 % [11]. The notably low resistance rate to Colistin (23.3%) in isolates is concerning, as Colistin is often considered a last resort antibiotic for MDR and XDR infections. The emergence of Colistin resistance underscores the urgency for stringent antibiotic stewardship and the development of novel therapeutic strategies.

Resistance to Imipenem was 53%, consistent with findings in Kufa and Baghdad [12,13] but higher than the 5% reported in Erbil city, Iraq [14]. Meropenem (37%) and Cefepime (40%) resistance rates matched those reported by Ali et al. [15]. Piperacillin resistance was 50%, in line with Alaboudi and Aljwaid (56.5%) [16] but lower than Khudair and Mahmood. [17]. Ceftazidime resistance (50%) matched reports from Baghdad (54.7%) [18] and Iran (53.3%) [19]. Gentamicin (47%) and Amikacin (43%) resistance rates were consistent with previous Iraqi studies [17,20]. Piperacillin/tazobactam resistance was 37%, aligning with the Poland data (39.6%) [21]

The observed increase in antibiotic resistance in *P. aeruginosa*, particularly in Iraq, can be attributed to several key factors, such as unregulated antibiotic use, self-medication, lack of susceptibility testing, and weak infection control in some Iraqi healthcare settings.

Antibiotic resistance patterns of *P. aeruginosa*

Current result showed as follows; 13 (43%) was XDR, 5 (17%) MDR. In this respect, local modern studies, which have reported an occurrence rate of MDR, XDR patterns to be 30.8% and 33.3% in Diyala [22], 72.6% and 91.3% in Basrah [23], 37% and 22% in Sulaymaniyah [24], respectively. Also, a study in Baghdad by Hatif [25] recorded 83.92% as MDR. On the other hand, a study in Egypt by Samie [26] revealed that XDR was 54%, and MDR was 8.6%.

These variations in results related to MDR and XDR patterns may be due to many factors such as laboratory methods, sample size of bacterial

isolates, differences in infection control measures, and differences in hospital environments.

Molecular detection of Las system genes in P. *aeruginosa*

In this study, the occurrence of QS genes among *P. aeruginosa* isolates was: lasI 29 (96.67%), lasR 23 (76.67%).

For comparison, Ghazi and Jasim (Baghdad) reported frequencies of 96-100% for lasI, lasR genes [27]. Lima et al. [28] found 97.5% for lasI and 100% for lasR. Al-Kilabi et al. [29] reported lower rates in otitis media isolates (lasI: 87%, lasR: 80.6%). Ghanem et al. (Egypt) noted 81.6%, 80%, respectively [30]. Similarly, Elnegery et al. recorded lasR at 94% [31]. Another study of 120 isolates found lasI: 89.1%, lasR: 78.3%. [32]. The possible explanation for discrimination in such a result may be due to the fact that Qs occurrence rate can be influenced by genetic strain diversity, in which some bacteria naturally lose or mutant their genes, especially in long-standing wound infections [33].

Correlation between las system genes (LasI, LasR) and antibiotic resistance patterns

In the present study, the highest frequency of MDRPA was related to lasI with 100%, and lasR gene, with 80%. This agrees with a study in Iran by Hemmati et al., [32] that showed that 94.3% of them had lasI while 83% of them had lasR. Another study by Shravani et al., [34] showed 100% of MDRPA strains had las QS genes. also study in Egypt [30] for lasR, showed all MDRPA had lasR, and it was more virulent. On the other hand, the present study recorded 92.3% of XDR P. aeruginosa (XDRPA) had lasI and 76.9% had LasR. The study by Hemmati et al., [32] showed that all XDRPA had lasI and 90.5% of them had lasR.

Current results recorded no correlation between the las quorum sensing system and antibiotic resistance patterns, this indicates that even though the lasI and lasR genes were quite common in isolates of *P. aeruginosa*, there was no statistically significant correlation between their presence and the strain's MDR, XDR, or non-resistant status.

In other words, the frequency of las gene carriage was comparable in drug-resistant and drug-sensitive strains similar to an investigation by Hemmati et al. [32] that showed no correlation between the las system and XDR patterns, also another study by Lima et al. [28] and Shravani et al. [34]. They showed no significant differences were observed in the las gene detection between MDR and non-MDR strains of *P. aeruginosa*. Such results may refer primarily to the role of QS in regulating many virulence factors, and not have direct influence on antibiotic resistance genes like β-lactamases or efflux pumps, and the resistance mechanism of antibiotics can be independent of the QS system.

In conclusion, a high prevalence of las QS genes in P. aeruginosa isolated from wound infection. However, no statistically significant correlation was observed between the presence of these genes and MDR or XDR phenotypes.

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Author contribution

Kadhim: Conducted the sampling, isolation, and diagnosis, the molecular work, and wrote the manuscript. Dr. Hassan and Dr. Abdul Wahhab supervised the work, edited, and finalized the writing of the study. Hussein: Did the statistical analysis of work.

Conflict of interest

The authors declare that they have no competing interests.

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