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ORIGINAL ARTICLE

The Presence of Plasmid-Mediated Quinolone Resistance Genes in Pseudomonas Aeruginosa Strains Collected from Clinical Samples in Al-Al Diwaniyah City

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Key words:
P. aeruginosa; PMQR genes; Antibiotic resistance; qnr-A; qnr-B; MDR; XDR

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ABSTRACT

Background*: The growing challenge of antibiotics resistances caused by Pseudomonas aeruginosa (P. aeruginosa, an important opportunistic pathogen) is one of the global healthcare issues. For epidemiological purpose, the identification of resistance genes is crucial.* ***Objectives****: The present study aims to determine the prevalence of PMQR encoding genes (qnr-A, qnr-B) phenotypically and genotypically in P. aeruginosa to determine epidemiological prevalence of these genes in Al-Diwaniyah city.* ***Methodology****: The study identified P. aeruginosa isolates from wounds and burns exudates, as well as urine samples from urinary tract infections (UTI) by biochemical characterization and VITEK 2 system. Conventional PCR was used to detect the presence of these genes.* ***Results****: Burns had a greater prevalence of P. aeruginosa (67.44%) than wounds (20.93%) and UTIs (11.63%). Antibiotic resistance analysis showed that thoroughly drug-resistant (XDR) bacteria predominated in burn infections (72.4%), wound infections (61%), and UTIs (66.7%). Molecular investigation showed that qnr-B and qnr-A are the most common resistance determinants in clinical isolates (97.91%). Antibiotic resistance patterns in wound, burn, and UTI infections were variable, but qnr-A and qnr-B genes showed that wound and burn cases with positive expressions had significantly increased MDR and XDR resistances. Positive expression of the qnr-A and qnr-b genes was associated with increased antibiotic resistance in all infection types, with substantial differences. Antibiotic susceptibility and gene expression studies for P. aeruginosa showed that resistant isolates have consistently high frequencies of positive gene expressions for these genes, especially for Imipenem and Meropenem. All isolates were Nalidixic acid-resistant and expressed these genes more. Only a few P. aeruginosa isolates are MEROPENEM-resistant intermediates. P. aeruginosa was responsive to all antibiotics except Nalidixic Acid. Our results demonstrate a varied correlation between antibiotic susceptibility and infection type, with statistically significant differences.* ***Conclusion:*** *This study demonstrate a predominance of plasmid-mediated fluoroquinolone resistance genes in the examined isolates. It is advisable to optimize fluoroquinolone utilization to maintain their efficacy against multidrug-resistant bacteria.*

INTRODUCTION

Pseudomonal agents, particularly those derived from the bacterium *Pseudomonas aeruginosa*, present a significant problem in microbiology and the management of infectious disorders. This opportunistic disease is renowned for its flexibility and inherent resistance to a wide range of medicines, complicating treatment strategies. *Pseudomonas aeruginosa* is commonly found in several locations, including soil, water, and healthcare facilities, where it can cause opportunistic infections, especially in immunocompromised persons. The clinical ramifications of infections produced by pseudomonal agents are substantial, highlighting the imperative for a comprehensive understanding of their pathogenic mechanisms, resistance profiles, and the therapeutic approaches available to combat these formidable organisms. The rising prevalence of antibiotic-resistant *Pseudomonas* strains highlights the urgent necessity for better surveillance, research, and the advancement of novel antimicrobial medicines in the evolving global health landscape.[1,2]

The clinical ramifications of *Pseudomonas aeruginosa* resistance are substantial. Infections due to multidrug-resistant Pseudomonas aeruginosa are associated with elevated morbidity and mortality, prolonged hospital stays, and increased healthcare costs.

The conventional treatment strategy often employs combination medicines; however, these approaches are progressively losing efficacy as resistance mechanisms advance. Consequently, there is an imperative necessity for continuous investigation into novel therapeutic alternatives, encompassing the development of new antibiotics and alternative treatment methodologies.[4]

The resistance of *Pseudomonas aeruginosa* is multifaceted, involving a combination of intrinsic factors such as a unique outer membrane structure and efflux pump systems, alongside acquired resistance through horizontal gene transfer. s, such as the QNR-A AND QNR-B, which confer resistance to Quinolone — antibiotics often considered last-resort treatments for serious infections.[5,6]

This study aimed to determine the antimicrobial profile and the prevalence of PMQR gene carriage of *P. aeruginosa* isolated from different clinical samples

METHODOLOGY

Study design

The present cross-sectional investigation has been conducted at two major hospitals (Afak Hospital & the specialized center for burns in Diwaniyah) in Al-Diwaniyah province, Iraq. From November 2024 to May 2025. Various clinical samples were collected and transported for further analysis employing the standard microbiological and biochemical techniques.

Ethical approval

Ethics approvals were achieved by University of Karbala/College of Medicine, Ministry of Health, in addition to, the research ethics committees for each hospital (24,25/11/2024), in addition to the research ethics committees for each hospital. An informed consent has been taken from all patients before being enrolled in the current work.

Samples collection, Isolation and Identification of *P. aeruginosa*

The sampling protocol applied in the current investigation can be described as convenience sampling. Different clinical samples were collected from patients with infections, including burn exudate, wound exudate, and urine. Infections were considered as either, hospital acquired or community related consistent with standard epidemiological definitions established by Kumarasamy [7]. The samples were labeled and transported to Afak Hospital Microbiology Laboratory for processing as describe by Collee *et al* [8], midstream urine collected into sterile container while Burn and wound samples were taken by sterile swabs. Clinical samples were transported to the laboratory, cultured on MacConkey and blood agar media and were incubated aerobically at 37°C for 24 hours. Using standard loop method, streaking samples on above media, the urine culture was considered positive results, if the bacterial count was ≥105 (CFU)/ml. After 24 hours of incubation, the culture media were examined for the presence of *P. aeruginosa* growth. All isolates were initially identified by conventional methods [9, 10]; and species were identified, and confirmed with VITEK-2 automated system using the GN ID and AST 222 card (Biomerieux, France).

Antimicrobial sensitivity analysis

All *P. aeruginosa* isolates were tested for antimicrobial susceptibility using 7 antibiotics from two classes: Penems (Imipenem, Meropenem) and Quinolones (Fluoroquinolones, Ciprofloxacin, Norfloxacin, Levofloxacin, Nalidixic acid, and Ofloxacin). Three to five colonies were placed in a test tube with two ml of nutritional broth, cultured for two hours at 37°C, and then McFarland 0.5 was added to correct broth density with normal saline. After streaking Muller-Hinton agar in different directions on the plate with a sterile loop or disposable loop and left for five minutes to dry, the antibiotic disks were spread on the plate and incubated for 18-24 h at 37°C. CLSI evaluated colony inhibition zone with ruler or special instrument. MDR bacterium isolates were resistant to at least one antibiotic in three or more classes, while XDR isolates were resistant in all but one or two classes. The isolates had pan drug resistance (PDR) 19 to all antimicrobial classes. The minimum inhibitory concentration (MIC) of all isolates resistant to imipenem, meropenem, Levofloxacin, ciprofloxacin, and norfloxacin was tested by VITEK-2 automated system using GN AST 222 card (Biomerieux, France) and interpreted using CLSI break points.

Bacterial DNA extraction and genetic analysis

Genomic DNA was isolated from P. aeruginosa isolates using Geneaid Genomic DNA Purification Kit (Turkey) as directed. DNA samples were tested for purity using a NanoDrop™ One UV-Vis Spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA) and maintained at -20°C until PCR is performed. Each isolate's qnr-A and qnr-B genes were amplified using standard PCR. Table (1) lists the primers used to amplify the genes. DNA amplification for each gene was conducted in PCR tubes with 25µL of PCR master mix (Promega, USA), 3µL of DNA sample, 16µL of distilled deionized water, and 3µL of forward and reverse primers. PCR was performed in 50µL volume, and amplicons were seen on a 1.5% agarose gel to confirm their expected sizes.

Table 1. The primers used for the current study

Primer name	Oligo sequence (5'-3')	Gene name	Product size
QNR-A	F: ATTTCTCACGCCAGGATTTG	*Qnr-A*	516
	R: GATCGGCAAAGGTTAGGTCA		
QNR-B	R: GATCGGCAAAGGTTAGGTCA	Qnr-B	469
	R: ATGAGCAACGATGCCTGGTA		

Statistical analysis

The data analysis for present study was generated using The Statistical Package for the Social Sciences software, version 26 (IBM, SPSS, Chicago, Illinois, USA). Descriptive statistics was performed on the participants' data of each group. Data was presented as percentages; Chi-Square analysis was employed to compare between percentages. The results of all hypothesis tests with p-values <0.05 (two-side) were deem to be statistically significant. In addition, SPSS program and Microsoft Excel 2010 program was employed to draw chart figures

RESULTS

Distribution of *Pseudomonas aeruginosa* According to Infection Sites

Figure (1) displays the distribution of *P. aeruginosa* isolates according to infection sites. This figure shows that the highest (67.44%) prevalence of *P. aeruginosa* was in burns specimens, while the lowest (11.63%) prevalence was in UTIs specimens.

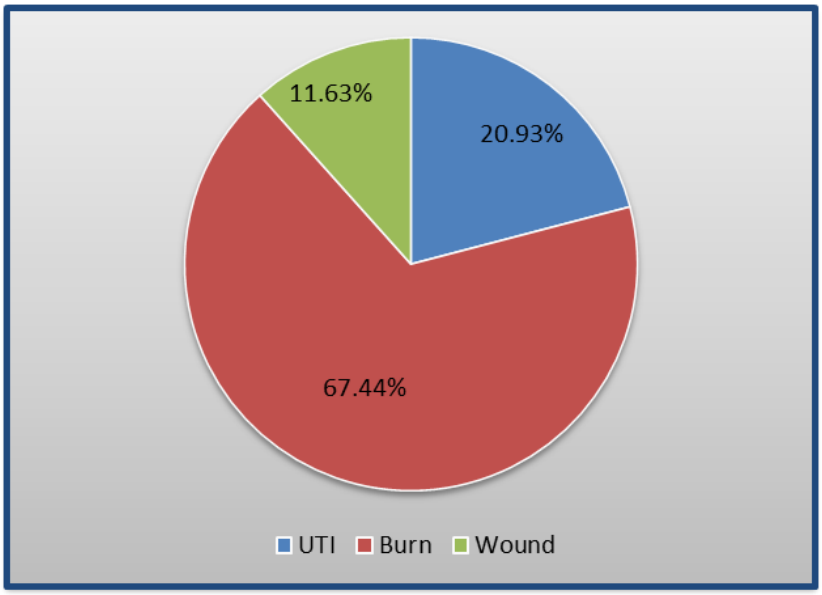

Fig. 1: Distribution of *Pseudomonas aeruginosa* According to Infection sites

Antibiotic resistance state for *Pseudomonas aeruginosa* in patients

Figure (2) shows the antibiotic resistance state for *P. aeruginosa* in patients with different infections. In burn cases, 72.4% of *P. aeruginosa* isolates have XDR resistance and 27.6% were MDR. In wound cases, 60% of isolates have XDR and 40% have MDR resistance. In UTIs cases, 66.7% of *P. aeruginosa* have MDR resistance, while only 33.3% have XDR, with notably highly significant (p=0.000) differences in each of them.

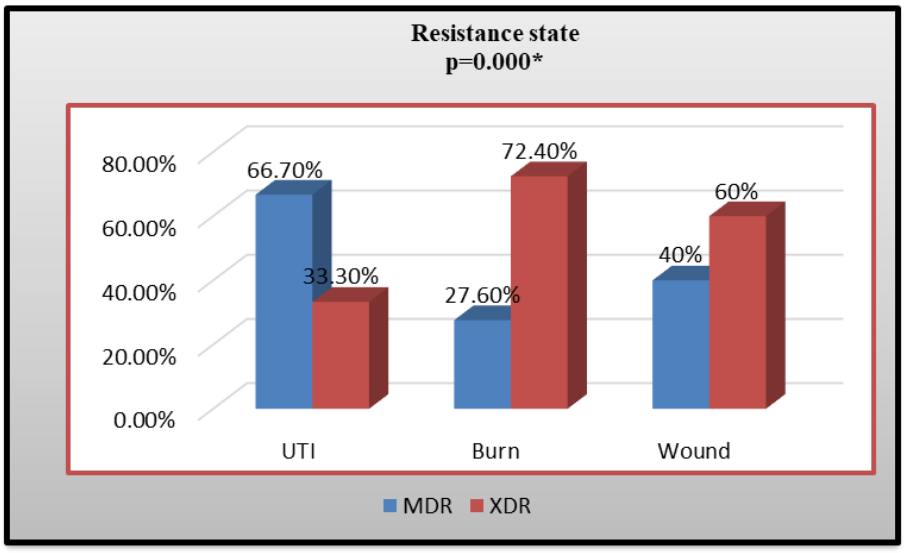

Fig. 2: Antibiotic resistance state for *Pseudomonas aeruginosa* in patients

Molecular detection of *PMQR* genes (QNR-A , QNR-B)

Figure (3) displays the PCR outcomes for 43 *P. aeruginosa* isolates, which tested for *qnr-A* , gene at the product size of 516bp, which was identified in 42 (97.91 %) of the clinical isolates of *P. aeruginosa*. The *qnr-B* gene was detected in 42 (97.91%) in the *P. aeruginosa* isolates, with a product size of 469bp figure (4). the distribution rates of these virulence genes for *P. aeruginosa* is displayed in Figure (5).

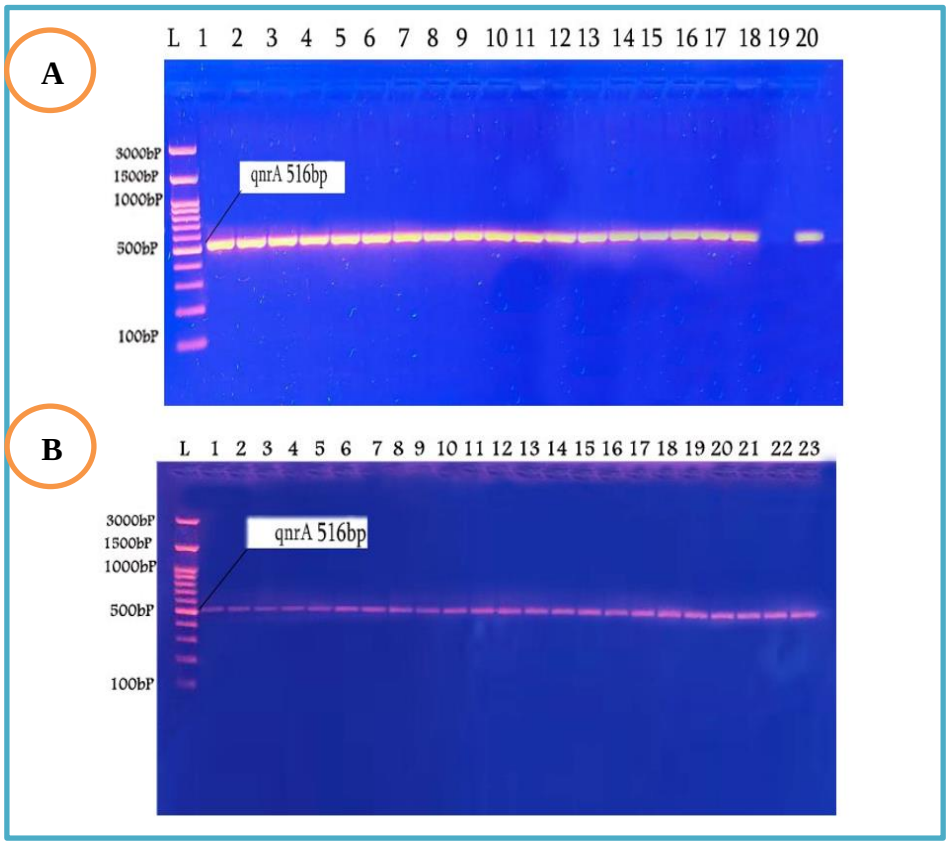

Fig. 3: Electrophoresis of amplified *QNR-A* (516bp). Agarose gel 1.5%, 75 volt for 1 h. A) The positive isolates were 1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17 18 and 20 the isolates 19 showed negative result. B) The positive isolates were 1, 2, 4, 5,6,7, 8, 9, 10, 11, 12, 13,14,15,16 17, 18, 19, 20, 21,22 and 23 .

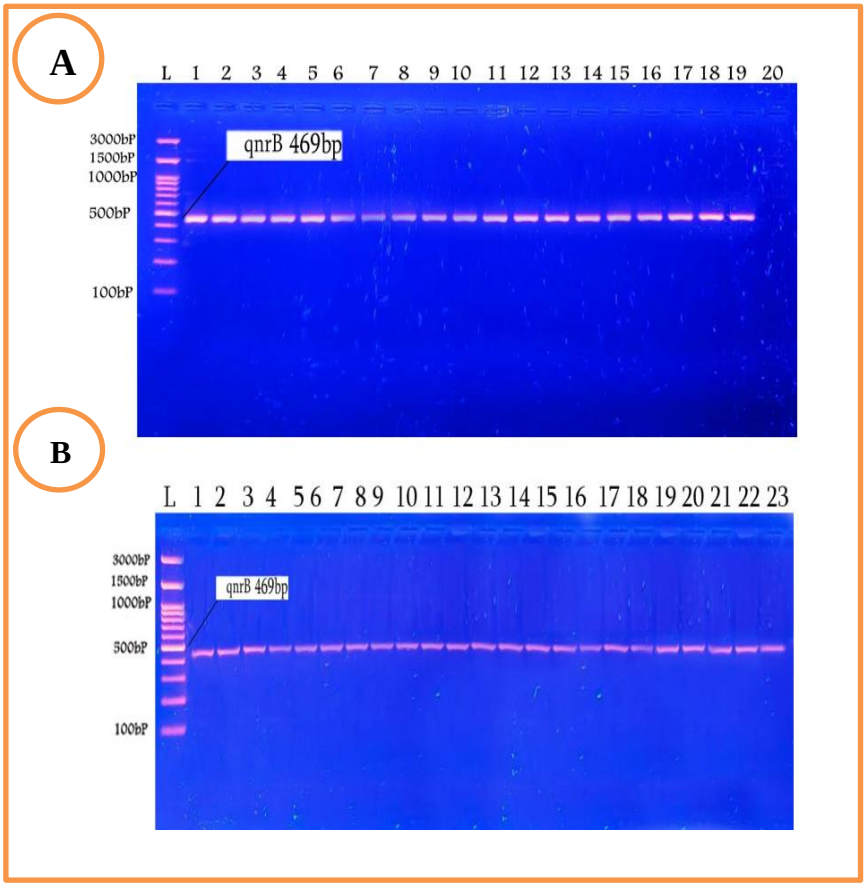

Fig. 4: Electrophoresis of amplified *QNR-B* (516bp). Agarose gel 1.5%, 75 volt for 1 h. A) The positive isolates were 1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18 and 19 the isolates 20 showed negative result. B) The positive isolates were 1, 2, 4, 5,6,7, 8, 9, 10, 11, 12, 13,14,15,16 17, 18, 19, 20, 21,22 and 23

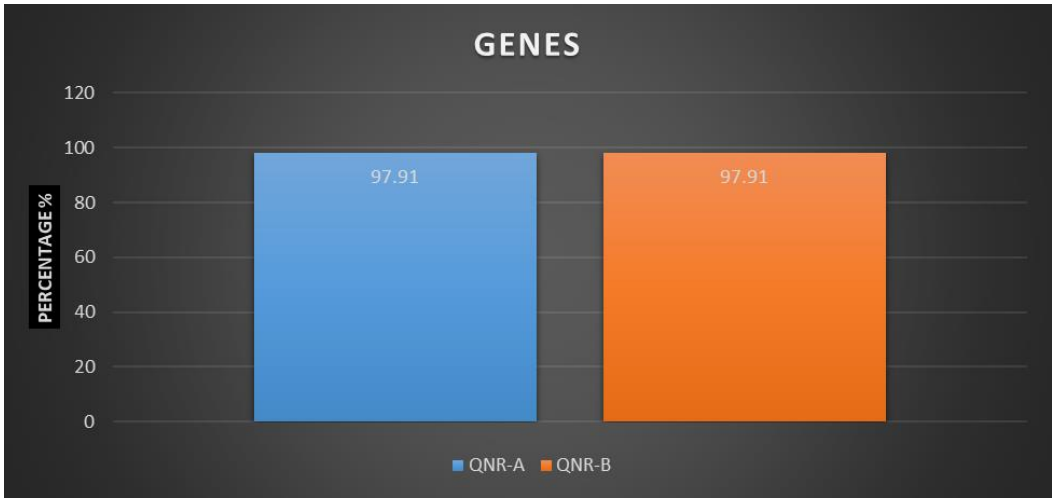

Fig. 5: Distribution of virulence genes among *P. aeruginosa* isolates from different clinical samples

Association between Studied Genes and Patterns of Antibiotic Resistance for *Pseudomonas aeruginosa* isolated from Wounds, Burns, and UTIs Infections

The present study analyzes the association between presence of *qnr-A* and *qnr-B* genes and antibiotics resistance in infections across wound, burn, and UTIs cases as explained in (table 2). The *qnr-A and qnr-B* genes indicated that in wound and burn cases their positive expressions were associated with significantly increase in both MDR and XDR resistances, while it's positive expression was more commonly linked to significantly increase in only MDR antibiotic resistance in UTIs cases. The qnr-A and qnr-B geneS showed a significant association between its

positive expression and escalated antibiotics resistance (both MDR and XDR) over all infection types, with P-values of 0.0001 in all these comparisons.

Antibiotic susceptibility for *Pseudomonas aeruginosa* in patients according to type of infection

Table (3) shows the antibiotic susceptibility for *P. aeruginosa* according to the type of infection, whether it is wounds, burns or UTIs. For IMPENEM, the highest percentage of isolates resistant to this antibiotic was those isolated from burns, at 93.1%, while the highest percentage of isolates sensitive to this antibiotic was those isolated from UTIs, at 44.9%. Regarding to MEROPENEM, as we mentioned above, there were three types of resistance isolates: resistant, intermediate, and sensitive. The highest percentage of resistant isolates was isolated from burns, at 86.2%. The isolates that showed intermediate resistance, which appeared only to this type of antibiotic, were the highest percentage of those isolated from UTIs, as well the highest percentage of isolates sensitive to this antibiotic was those isolated from UTI infections. As for LEVOFLOXACIN, CIPROFLOXACIN, OFLOXACIN, and NORFLOXACIN, the highest percentage of isolates resistant to this antibiotic was those isolated from burns, at 62.1%, while the highest percentage of isolates sensitive to this antibiotic was those isolated from UTIs, at 77.8%. According to NALIDIXIC ACID , all *P. aeruginosa* isolates were resistant to NALIDIXIC ACID, and the highest percentage was that isolated from burns, at 67.4%. All these associations showed statistically highly significant differences, ($p<0.05$).

Table 2. Association between Studied Genes and Patterns of Antibiotic Resistance of *Pseudomonas aeruginosa* in Wounds, Burns, and UTIs Infections

Gene type	Gene expression	Type of infection and Antibiotic resistance					
		UTI(n=9)		Burn (n=29)		Wound (n=5)	
		MDR	XDR	MDR	XDR	MDR	XDR
qnr_a	- ve	0 (0%)	0 (0%)	1(14.3%)	0 (0%)	0 (0%)	0 (0%)
	+ve	6(100%)	3(100%)	7(87.5%)	21(100%)	2(100%)	3(100%)
Total		6	3	8	21	2	3
P value		0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*
qnr_b	- ve	0 (0%)	0 (0%)	1(14.3%)	0(0%)	0 (0%)	0 (0%)
	+ve	6(100%)	3(100%)	7(87.5%)	21(100%)	2(100%)	3(100%)
Total		6	3	8	21	2	3
P value		0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*

*Significant difference at the 0.05 level by chi-square test NS: Non-significant difference

Table 3. Antibiotic susceptibility for *Pseudomonas aeruginosa* in Patients according to type of infection

Antibiotic	R/I/S	Types of infection No. (%)			Total	P value
		Wound	Burn	UTI		
IMPENEM	R	4 (80%)	27 (93.1%)*	5 (55.5%)	36 (83.7%)	0.0074*
	S	1(20%)	2 (6.9%)	4 (44.9%)*	7 (16.3%)	0.0001*
Total		5	29	9	43	
MEROPENEM	R	4 (80%)	25 (86.2%)*	3 (33.3%)	32 (74.4%)	0.0001*
	I	0 (0%)	2 (6.8%)	2 (22.2%)*	4 (9.3%)	0.0001*
	S	1 (20%)	2 (6.8%)	4 (44.4%)*	7 (16.3%)	0.0001*
Total		5	29	9	43	
LEVOFLOXACIN	R	3 (60%)	18 (62.1%)*	2 (22.2%)	23 (53.4%)	0.0001*
	S	2 (40%)	11 (37.9%)	7 (77.8%)*	20 (46.5%)	0.0001*
Total		5	29	9	43	
CIPROFLOXACIN	R	3 (60%)	18 (62.1%)*	2 (22.2%)	23 (53.4%)	0.0001*
	S	2 (40%)	11 (37.9%)	7 (77.8%)	20 (46.5%)	0.0001*
Total		5	29	9	43	
OFLOXACIN	R	3 (60%)	18 (62.1%)*	2 (22.2%)	23 (53.4%)	0.0001*
	S	2 (40%)	11 (37.9%)	7 (77.8%)*	20 (46.5%)	0.0001*
Total		5	29	9	43	
NALIDIXIC ACID	R	5 (11.6%)	29 (67.4%)*	9 (20.9%)	43 (100%)	0.0001*
NORFLOXACIN	R	3 (60%)	18 (62.1%)	2 (22.2%)	23 (53.4%)	0.0001*
	S	2 (40%)	11 (37.9%)	7 (77.8%)	20 (46.5%)	0.0001*
Total		5	29	9	43	

*Significant difference at the 0.05 level by chi-square test

DISCUSSION

According to the result presented in figure (1), the higher prevalence (67.44%), of *P. aeruginosa* was found in burn infections, followed by wound infections (20.93%) and UTIs (11.63%). This pattern emphasized the opportunistic nature of this pathogen and its ability to grow in environments with compromised tissues integrity and immune response.

The present findings are consistent with previous investigations that also showed high rates of *P. aeruginosa* in burn infections like those done in Yemen 46.5% [11], India 55% [12], and 62.70% from Nigeria [13]. On the other hand, the 67.44% isolation rate from the current investigation is much higher than those recorded in other previously done investigations where 6.25% in Nepal [14], 12.86% in Ethiopia [15], and 27% in Iraq [16]. Such differences in *P. aeruginosa* prevalence rates from burn wounds among investigations could be due to a combination of several reasons such as inclusion criteria, the use of selective/differential media to culture the *P. aeruginosa*, sampling protocol, and variations in provision the health care providing institutions including existence or absence of dedicated center to for burn injuries. The moderate prevalence rate of *P. aeruginosa* in wound infections is in line with finding of Phan *et al*. [17], which identified *P. aeruginosa* as a common bacterium in chronic wound infections globally. The presence of this bacterium in wounds can be attributed to its production of numerous virulence factors like elastase and pyocyanin, which detain healing and boost chronicity.

However, Mundhada *et al*. recorded a higher prevalence rate in postoperative wound infections, and highlighted the role of hospital-acquired strains to exacerbate wound infections [18]. The relatively low prevalence in UTIs is in consistence with Chowdhury *et al*, who reported that *P. aeruginosa* accounts for a smaller percent of UTI infections versus other uropathogens like *E. coli* [19]. This low prevalence rate may be due to the fact that this bacterium prefers the environments with higher moisture and nutrient availability, such as burn sites. However, Streeter and Katouli reviewed that *P. aeruginosa* could still cause considerable morbidity in nosocomial UTIs, especially, in immunocompromised individuals [21].

Regulatory authority's neglect, poor execution efforts and prescribing of antibiotic by poorly trained medical personnel individuals are considered as triggering factors that are implicated in the emergence of antibiotics resistance. The trend of usage of antibiotic is rising in different countries, hence, unsuitable using leads to emergence of resistant bacterial isolates that cause infections difficult to treat, accordingly cause financial burdens on health sectors and on societies [21]. The data displayed in (Figure 2) highlighted the antibiotics resistance patterns of *P. aeruginosa* in different infection types, revealing a highly significant differences (p=0.000) in resistance levels. With regard to wound infections, the observed resistance patterns are consistent with those presented by Monk *et al*, they reported that *P. aeruginosa* in chronic wounds often reveals giant levels of resistance as a result to biofilm production along with the expression of efflux pump [22], where these mechanisms lessen antibiotic penetration and efficacy, and contribute to the perseverance of MDR and XDR isolates. However, Al-Dahmoshi *et al*. suggested that resistance patterns in wound infections could vary according to healthcare setting and existence of co-infections [23]. The prevalence rate of XDR in burn infections is in line with Japoni *et al*, who emphasized the role of compromised tissues integrity, biofilm formation, and extended hospital stays in boosting resistance [25]. Burns represent an ideal environment to colonize *P. aeruginosa* due to the absence of the skin barriers along with presence of necrotic tissues, which enhance bacterial growth and resistance expansion. Similarly, Holder demonstrated that burn infections are mostly prone to resistance as a result of frequent use of broad-spectrum antibiotics, which have a selective pressure on bacterial population [26].

For urinary infections, the relatively lower rate of XDR as compared with burn and wound infections may reflect variations in the infection environment. In UTIs, *P. aeruginosa* is less likely to make biofilms as strong as those in burns or wounds strains, which may explain the higher rate of MDR isolates. This statement is supported by Metri *et al*. (2014), who reported that *P. aeruginosa* isolates in UTIs often respond better to antibiotic treatment versus isolates from other infection sites [26]. The present findings emphasize the adaptability of *P. aeruginosa* and its ability to promote resistance mechanisms tailored to certain infection environments. Accordingly, it is important to develop infection site-specific treatment strategies and there is an essential for strong infection control measures to reduce the spreading of MDR and XDR *P. aeruginosa* strains.

A 97.91% prevalence of qnr-a and qnr-b genes in *P. aeruginosa* strains is alarmingly high rate and enhances the important roles these genes in antibiotic resistance. The qnr-a and qnr-b genes are well-known to control the plasmid-based quinolone resistance, which can be transported between bacterial strains thus further helping to exacerbate the spreading of resistance. This high prevalence revealed that the majority of *P. aeruginosa* isolates in the investigated population possess the required mechanisms to evade quinolone-based treatments, constituting a significant challenge for clinical management. Such findings agree with [27] who showed the widespread presence of MDR and XDR phenotypes in clinical isolates, often associated to virulence genes like qnr. As well, the current investigation supported with the findings of [28] who

demonstrated the rising prevalence of plasmid-mediated resistance genes in *P. aeruginosa* isolates, especially in hospital-acquired infections. These investigations emphasized the impacts of qnr genes in conferring resistance not only to quinolones, but also to other antibiotic family due to their alliance with integrons that carried multiple resistance determinants. In fact, the implication of this high prevalence is extremely profound; clinically, it restricts the efficacy of quinolones, which are usually employed as first-line treatments for *P. aeruginosa* infections.

Various resistance mechanisms are significantly involved with diminishing antibiotics sensitivity in P. aeruginosa, including the possession of some genes like Plasmid Mediated Quinolone Resistance genes like *qnr-a*, *qnr-b*, *qnr-c* and *qnr-s*[29]. Surveillance of antimicrobial resistance in nosocomial pathogens like *P. aeruginosa* is crucial to explore medicinal interventions. Frequent prevalence of mobile genetic elements like these genes in nosocomial infections from high complexity hospitals could be a considerable concern[30]. The data provided in Table (2) reveals the association between the presence of *qnr-a* and *qnr-b* genes in *P. aeruginosa* and their roles in MDR and XDR resistance patterns infections over different infection types, including UTIs, burn, and wound infections. The results showed a statistically significant correlation between presence of *qnr-a* and *qnr-b* genes and antibiotic resistance profiles in *P. aeruginosa* isolates; where the lack of *qnr-a* and *qnr-b* genes corresponds to a lack of resistance in all infection types, as seen with 0% MDR and XDR. Contrariwise, the isolates having these genes are strongly expressed high resistance rates. The current findings are corresponding with [31]. who reviewed the roles of qnr genes in mediating quinolone resistance, often via plasmid-mediated mechanisms. Similarly,[30]. emphasized the lack of qnr genes in susceptible isolates and consolidating their association with resistance. Moreover [32] .assured the prevalence of qnr genes in MDR *P. aeruginosa* isolates, especially in hospital settings, emphasizing their clinical importance.

The (table 3) explores the association between infections (wound, burn, and UTI) and antibiotics resistance patterns in *P. aeruginosa*. It stands out the significant variations ($P \leq 0.05$) in resistance patterns (R, I, and S) to carbapenem family (imipenem and meropenem), fluoroquinolone (levofloxacin, ciprofloxacin, ofloxacin, norfloxacin), and nalidixic acid; where this outcomes underscored the impacts of infections sites on resistance phenotypes, and reflected the complex interplay of environmental and genetic determinants influencing *P. aeruginosa*. As for carbapenems, burn infections exhibited highest resistance rates 93.1% and 86.2% for imipenem and meropenem, respectively. Wound infections followed closely, with resistance rate of 80% for both imipenem and meropenem, while in *P. aeruginosa* from UTI samples both imipenem and meropenem showed significantly lower resistance rates (55.5% and 33.3%, respectively).

The present patterns align with previous findings reported by Alatoom *et al*, who found that carbapenem resistance in *P. aeruginosa* strains is driven by carbapenemase genes like *bla-OXA* and *bla-NDM*, especially in burn-associated infections [33]. The resistance toward fluoroquinolones is moderate in wound and burn infections but significantly lower in UTIs, where the susceptibility dominates with percent of 77.8%; this trend is align with Abdulameer and Abdulhassan, who attributed the ability of *P. aeruginosa* to resist the fluoroquinolone to efflux pump overexpression and mutation in target genes like *gyrA* and *parC*. Nalidixic acid resistance is highest in burn versus wound and UTI infections (67.4%, 11.6%, and 20.9% respectively) [34], further supports the intensify resistance in burns.

Burn infections consistently reveal the most concerning resistance pattrens in all examined antibiotics, followed by wound infections, whereas the isolates of UTI samples showed significantly increased susceptibility rates. The present findings indicated that infection type strongly affects antibiotic resistance in *P. aeruginosa*. This higher resistance rates in burn and wound infections can be assigned to the unique environmental pressures along with the expressions of resistance genes like *bla-CTX-M, bla-NDM*, and *bla-OXA*, which are often prevalent in *P. aeruginosa* isolates from these infection sites. Contrariwise, the higher susceptibility toward UTI infections may reflect the lower biofilm complexity, along with the relatively better permeation of antibiotics in the urinary tract. Previous investigations had assured the roles of biofilm formation and genotypes distribution to mediate the resistance phenotypes in *P. aeruginosa* [35, 36].

CONCLUSION

The high prevalence of *qnr-a* and *qnr-b* genes in *P. aeruginosa* isolates reveals their critical roles in antibiotic resistance, particularly MDR and XDR phenotypes across different infection types, including UTIs, burn, and wound. This emphasizes the urgent requirement for molecular surveillance, powerful infection control measures, and effectual antimicrobial stewardship programs to stop the spreading of these resistance genes and improve therapeutics outcomes in clinical settings.

Declarations

Consent for publication

All the authors gave consent for the publication of the work under the Creative Commons Attribution- Non-Commercial 4.0 license.

Availability of data and materials

The data and materials associated with this research will be made available by the corresponding author upon reasonable request.

Competing interests

We declare that there are no conflicts of interest associated with this manuscript.

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Author contributions

AHJ: Conceptualization, methodology, formal analysis, and manuscript writin, Data collection, investigation, and software analysis.

MM: Supervision, project administration, and final manuscript review

AJA: Literature review, validation, and manuscript editing.

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