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Antimicrobial Susceptibility and Biofilm Formation in Clinical Isolates of *P. aeruginosa* from Patients at Mosul Hospital

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ABSTRACT

Objective: *The study was designed to investigate the occurrence, distribution of samples, and antibiotic resistance and biofilm forming potential of P. aeruginosa.* ***Method:*** *This descriptive cross-sectional study, including 225 clinical samples, spanned from December 2018 to June 2019 and isolated P. aeruginosa were identified by traditional biochemical tests and confirmed by the VITEK 2 compact system. Antimicrobial susceptibility testing was done by Kirby-Bauer disk diffusion method according to CLSI 2025. Biofilm production was measured by Tissue Culture Plate method and Isolates were graded as weak, moderate and strong biofilm producers on the Basis of optical density.* ***Results:*** *A total of 12.8% (29/225) prevailed the recovery of P. aeruginosa and wound specimen displayed the maximum rate (22%, $\chi^2 = 18.55$, $p = 0.005$). Antimicrobial susceptibility results showed that imipenem and levofloxacin were the most active (62.1% each), while gentamicin was found to be the least resisted (96.6%). Multidrug-resistant (MDR) and pandrug-resistant (PDR) patterns were found in 58.6% and 20.7% of the isolates. All isolates were shown to produce biofilm, with 17% weak, 48% moderate and 34% strong biofilm producers. Nevertheless, there was no significant correlation between the degree of biofilm formation and the profile of resistance to antimicrobials (p 0.05).* ***Novelty:*** *Although biofilm production was not directly associated with resistance profiles, its implication in chronic infections highlights the importance of developing specific strategies to interfere with biofilm production in clinical applications.*

INTRODUCTION

Antimicrobial resistance (AMR) has rapidly emerged as a significant threat to global health, and MDR P. aeruginosa have been so far listed by the World Health Organization as a critical priority threat, together with other members of the ES KAPE group. Pseudomonas aeruginosa is considered to be MDR when it displays intermediate or resistant phenotype to at least one or more agents in ≥ 3 of the following antimicrobial classes: extended-spectrum cephalosporins (ceftazidime, cefepime); fluoroquinolones (ciprofloxacin, levofloxacin); aminoglycosides (amikacin, tobramycin); carbapenems (imipenem, meropenem, doripenem); and the piperacillin group (piperacillin-tazobactam, piperacillin) [1]. XDR P. aeruginosa extensively drug-resistant P. aeruginosa (XDR P. aeruginosa) is defined as P. aeruginosa exhibiting intermediate or resistant phenotypes to all but one or two, across all classes of agents. Pan drug resistant or PDR P. aeruginosa = P. aeruginosa with non-susceptibility to all agents in all classes. MBL-PA is a P. aeruginosa isolate that produces metallo-beta-lactamases. Carbapenem resistant P. aeruginosa (CR-PA) is defined as P. aeruginosa with an "I" or "R" result to at least one of meropenem, imipenem and doripenem when tested (P. aeruginosa is not recommended to be routinely tested for ertapenem). The definition of P. aeruginosa

phenotypes as P. aeruginosa with difficult to treat resistance (DTR). P. aeruginosa as phenotypes with resistance to all the anti-pseudomonas first line agents with the highest activity and least toxicity (for example carbapenems, β-lactams/β-lactamase inhibitors combinations, and fluoroquinolones) [2]. This conceptualization helps to differentiate better from MDR, XDR, PDR and single class resistant entities and should be modified over time [2].

Pseudomonas aeruginosa is a Gram-negative gamma proteobacterium of the genus Pseudomonas, which has evolved as a metabolically versatile and ecologically successful opportunistic pathogen causing a wide variety of community recognized [3] and nosocomial infections and, very rarely, infections in normal, immunocompetent individuals [1]. High morbidity and mortality rates in humans (depending on the host species) can be associated with Pseudomonas aeruginosa as a primary or secondary infectious agent. P. aeruginosa emerged as a predominant source of healthcare-associated infections (HAIs) and exploited modern therapeutic measures that prolong survival by mitigating the original life-threatening insult—chemotherapy, prosthetic device, ventilator, cannulas, among others—to induce infection in the weakened host eventually [4]. The development of MDR strains is also favoured by hospitalisation and use of numerous antimicrobial agents [4]. P. aeruginosa is a well-known source of healthcare-associated urinary tract infection especially due to catheterization. It has also been reported that P. aeruginosa accounts for 16% of the catheter associated urinary tract infections in ICU, which is responsible for about 10% of the urinary tract infections caused by catheterization [5]. P. aeruginosa-associated CAUTI is linked to significant morbidity and mortality, as the strain is commonly associated with MDR phenotypes and its infection frequency can be complicated by bacteremia. Due to coordinated mechanisms (efflux, permeability, and target modification) that are conserved across diverse taxa and incorporated even in wild-type or non-drug-resistant strains, P. aeruginosa is well known to be inherently resistant to many classes of antimicrobials, host-derived antimicrobial polypeptides, and disinfectants [3].

These strategies involve reduction in outer membrane permeability, enzymatic destruction, enhanced efflux and biofilm formation. In general, the resistome of p. aeruginosa is susceptible to mutations as well as the acquisition and incorporation of DNA through horizontal gene transfer [6].

Biofilm formation is an example of adaptive resistance mechanisms in Pseudomonas aeruginosa Biofilms are communities of bacteria (and occasionally yeasts) cells that have organized themselves into intra and inter species structures and that are embedded together within an extracellular polymeric substance (EPS) matrix and that are adherent to each other and to a surface, be it biotic or abiotic [7]. 80% of the bacterial infections involve biofilms. Polysaccharides from several P. aeruginosa biofilm isolates inhibit neutrophilic phagocytosis [7]. Bacteria living in biofilms are more resistant to the action of antibiotics than their planktonic counterparts, as most of the antibiotics target physiologic activities of actively growing bacteria, which would be primarily found in the periphery of a biofilm since they would have access to more nutrients compared to

those living in the centre of the three-dimensional biofilm. P. aeruginosa planktonic cells can be killed readily by antibiotics, but P. aeruginosa in biofilms exhibits a 1000(time or exponantial) higher resistance to antibiotics. The notion of biofilm associated antibiotic tolerance was initially proved by professor of biofilm pioneer Bill Costerton and his associates to bring hope to thousands if not millions of pacemaker patients, who were given no recourse for decades for life-threatening infections around pacemakers with antibiotics because the heart stopping bacteria were protected by biofilm: planktonic bacteria in blood were easily treatable with antibiotics, bacteria in biofilm on pacemakers were resistant to the very same treatment. P. aeruginosa biofilm are known to colonise biotic surfaces (conjunctiva, cochlear, mucous membrane epithelial cells, and chronic wounds) and abiotic surfaces (urinary catheter, pacemaker, CSF shunts, IV lines, breast implants, joints implants, endotracheal tubes, stents). Quorum sensing mediates P. aeruginosa biofilm formation. Bacterial communication is profoundly mediated by chemical signals called auto-inducers [7].

Upon reaching a quorum level (i.e., a threshold that can be detected), bacterial cells sense their population density and activate group behaviors such as biofilm development. Quorum sensing as a concept becomes the corner stone of socio-microbiology. Socio-microbiology is fundamentally an area of microbiology that deals with the community or social behaviors of bacteria. These auto inducers are small membrane diffusible molecules, secreted by individual bacterial cells and the concentration of signaling molecules is directly proportional to the density of bacterial cells and if the concentration of auto-inducers becomes high enough to activate specific downstream genes, it leads to a unified response throughout the entire bacterial population [7]. Three types of auto inducers namely; Pseudomonas quinolone signal, 3-oxo-dodecanoyl homoserine lactone and butyric homoserine lactone (C4 HSL) are secreted by P. aeruginosa [6], [8].

P. aeruginosa exhibits metabolically and morphologically different phenotypes at various stages of its biofilm development [9]. A recent review has summarised the different phases of biofilm formation in 5- As: adhesion, amplification, alienation, aging and allocation [10]. During stage one (adhesion stage), the planktonic P. aeruginosa cells are attracted to and attach to a substrate, whether biotic or abiotic, by twitching or swarming motility with its flagellum; the majority of the first contacts is transient, but the attachment is progressively stabilized by extracellular polymeric substance. Two (development) Amplification Two (development) Amplification Second stage, or expansion stage: stage two consists of the proliferation of P. aeruginosa micro colonies of similar cells on the substrate surface and secretion of EPS; sessile cells that have not lost their flagella yet migrate to the uppermost layers of the biofilm and the cells bind to a more stable surface, attachment to a surface induces changes in gene expression, including repression of flagella genes and promotion of multiple other P. aeruginosa genes including alg C; algC encodes phosphomanno-mutase, an enzyme that modulates P. aeruginosa LPS under environmental stresses such as exposure to antibiotics and host immune response by adding a phosphate moiety. Three (detachment) Alienage Four

(detachment) alienage The third epoch is characterized by detachment or fragmentation of parts of the incipient biofilm, emanating from at least one species of the anchor organism, from the surface into the surrounding milieu of the biofilm community, this is induced by nucleases produced by certain sessile cells within the biofilm population, digesting eDNA and inducing a mild retraction of the biofilm mass. In stage four or aging, the biofilm mass has undergone reconstruction and has acquired integrity, and is apparently not easily removed from its substratum by mechanical stress. With the final phase of the biofilm being the "stage five," or the "dispersion" phase; however, this stage does with the biofilm itself its end, but rather its beginning in a new cycle. Biofilm cells disperse as planktonic cells to seed new cycles in other locations [10].

RESEARCH METHOD

The Research Ethics Committee of equal to Mosul hospital granted ethical clearance for this cross-sectional descriptive study. Total of 225 clinical specimen (s) were obtained from August 2025 to October 2026.

From July to September of 2025 we collected 225 clinical samples that consisted of blood, urine, wound swabs, pleural fluid and others. Samples were plated onto required bacteriologic media Vis - à- Vis the specimen CLED, chocolate plate, blood plate and MacConkey plate.

Pseudomonas aeruginosa strain identification was accomplished on pooled presumptive strains. The colonial morphology, the pigment are classical features in phenotyping. This was followed by Gram staining and an array of conventional biochemical tests oxidase, catalase, urease, citrate utilization, triple sugar iron (TSI) agar tests and the ability to grow at 42°C [11]. After that, P. aeruginosa isolates were all frozen at -20^{o}C in 15% glycerol for storage in Tryptic Soy Broth (Oxoid) and were subsequently used in this study. Identification of the isolate was confirmed by BIOMÉRIEUX VITEK 2 compact system. A bacterial suspension was created from an individual isolate pure colony and was modified to a McFarland standard of 0.50–0.63 with a DensiCHEK Plus densitometer. The standardized suspension was filled in a Gram-negative identification (GN) card. The inoculated card is then sealed and inserted into the VITEK 2 system for analysis and performs (optical) assays of the 64 biochemical wells at interval of 15 min during the incubation using kinetic colorimetry. The identification results generated by the systems integrated database and software algorithm were interpreted and recorded upon completion of the cycle [12], [13].

Antimicrobial susceptibility testing was done manually by Kirby Baur Disc diffusion method as per CLSI guidelines. The following antibiotics were used for the purpose: Piperacillin-tazobactam 100/10µg (Oxoid, UK), ceftazidime 30µg (Oxoid, UK), cefepime 30µg (Oxoid, UK), imipenem 10µg (Oxoid, UK), meropenem 10µg (Oxoid, UK), ciprofloxacin 5µg (Oxoid, UK), levofloxacin 5µg (Oxoid, UK), tobramycin 10µg (Oxoid, UK), and gentamicin 10µg (Oxoid, UK). The AST result was read as per 2023 CLSI. The methods were confirmed with Pseudomonas aeruginosa ATCC 27853® (American Type Culture Collection) quality control strain.

The tissue culture plate method based on polystyrene microtiter plates was used for screening biofilm producers as described by Mathur et al. [14] with some modifications; Pseudomonas aeruginosa ATCC 27853® was used as positive control and sterile Luria Bertani broth as negative control and the procedure instructions were as follows:

1. Seeding the microtiter plate: Pure colonies of P. aeruginosa were isolated on Mueller-Hinton agar, three separate colonies were inoculated into Luria Bertani broth and the cultures were incubated at 37OC for 24hrs. The culture was diluted in a McFarland tube by 1: 40. 200µL of this diluted suspension was added to the wells of the microtiter plate and was incubated at 37^{o}C for 24hrs, along with positive and negative controls. The contents of the wells were aspirated with individual Pasteur pipettes. The well was washed with normal saline, 200µL 3 times per well, the plate was inverted and allowed to dry and blot at room temp.
2. Fixing the biofilm: The methanol-fixed air-dried attached biofilm mass was fixed with air.
3. Biofilm staining and dissolving the biofilm: The wells were stained with 3-(4, 5-dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide for 20 min at room temperature. The plate was rinsed with tap water and dried at room temperature for one hour. The stained and adhered biofilm mass was dissolved in 160µL ethanol by gently mixing (aspirating in and out of the pipette tip). The set up was allowed to stand for 15 mins at room temperature.
4. Measurement of biofilm mass: OD was measured at 550nm with Bio-Rad PR 4100 Absorbance Microplate Reader (ELISA plate reader).
5. Categorization of the biofilm: The isolates with OD beyond cut off OD up to 2X cut off OD were considered as weak biofilm former, those with OD beyond 2X cut off OD up to 4X the cut off OD were considered as moderate biofilm former and the OD values beyond 4X the cut off OD were considered as strong biofilm former. Cut off optical density was considered as three standard deviations above the mean OD of the negative control [13].

Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 30.0.0. Based on the level of measurement of the variables, suitable non-parametric tests were applied. Two-sided tests were used for all analyses with a 95% confidence level. A significance level (α) was estimated automatically by the software (=0.01 for some analyses, 0.05 for other analyses), dependent on the test requirements, 2-page view.

RESULT AND DISCUSSION

Results

Prevalence Demographics of P. aeruginosa Isolates: The total prevalence of P. aeruginosa among the clinical samples was 12.8% (29/225), which is represented in figure 1. The isolation rate was significantly different among body sites ($\chi 2 = 18.55$, $p = 0.005$).

The highest rate of isolation was from wound samples (22.0%) followed by pus (20.0%), and the lowest rates were from urine (6.7%) and HVS (10.0%), as shown.

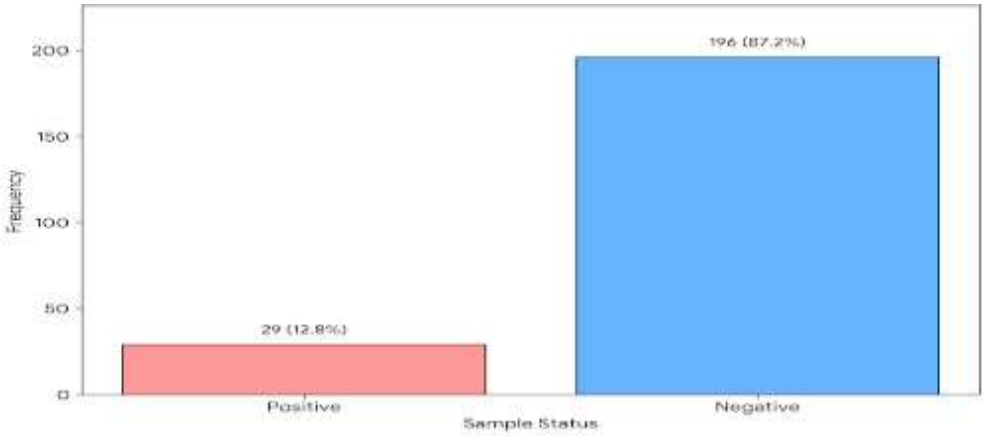


Figure 1. Frequency of *P. aeruginosa* clinical samples isolate status

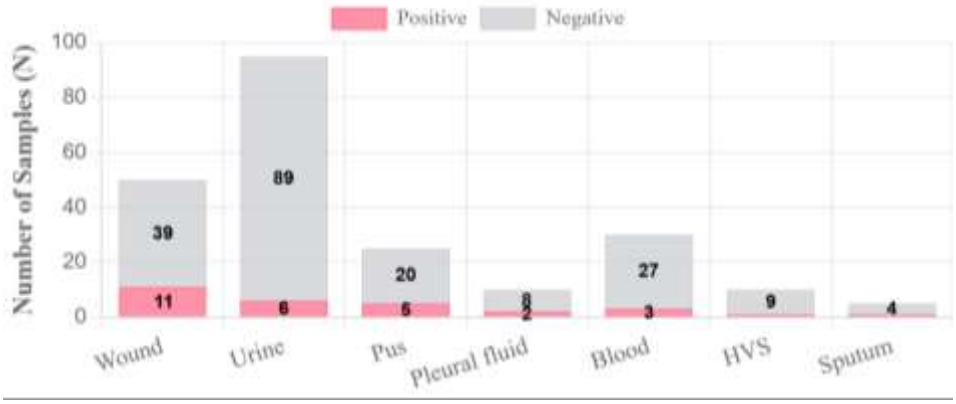


Figure 2. Frequency distributions of *P. aeruginosa* clinical specimen isolates

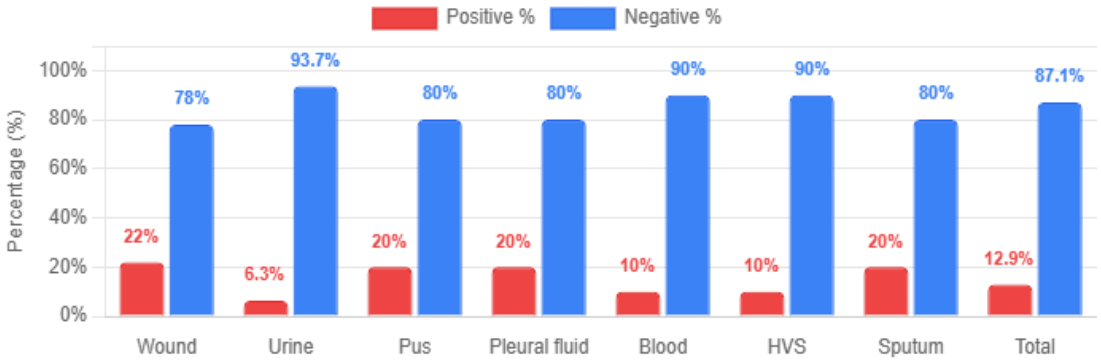


Figure 3. Percentage prevalence of *P. aeruginosa* across various samples analyses

Antimicrobial susceptibility of P aeruginosa isolates: Imipenem (a carbapenem β-lactam) and levofloxacin (a fluoroquinolone) showed the best susceptibility rate for

clinical isolates with the both comprised 62.1%. Instead, gentamicin (an aminoglycoside) had the greatest level of resistance (96%). Notably, ceftazidime, a third-generation cephalosporin, displayed a bipolar susceptibility distribution with no intermediates - 53 % of isolates were resistant and the rest were susceptible. These responses to resistance patterns.

Table 1. Antimicrobial Susceptibility Profile of the Clinical Isolates of *P. aeruginosa*

Antibiotic(µg)	**S (F)**	**S (%)**	**I (F)**	**I (%)**	**R(F)**	**R (%)**
Meropenem 10	14	48.3	1	3.4	14	48.3
Imipenem 10 a	18	62.1	2	6.9	9	31.0
Cefepime 30	16	55.2	2	6.9	11	37.9
Ceftazidime 30 c	12	41.4	0	0.0	17	58.6
Piperacillin-tazobactam100/10	13	44.8	3	10.3	13	44.8
Ciprofloxacin 5	10	34.5	5	17.2	14	48.3
Levofloxacin 5 a	18	62.1	5	17.2	6	20.7
Tobramycin 10	10	34.5	6	20.7	13	44.8
Gentamicin 10 b	0	0.0	1	3.4	28	96.6

Note. An indicates highest susceptibility, b indicates highest resistance, c indicates no intermediate "I" with high resistance. F stands for frequency.

Prevalence of AMR-Phenotypes in Clinical P. aeruginosa isolates: In case of P. aeruginosa clinical isolates, 58.6% were MDR, 20.7% were pan-drug resistant (PDR) and 3.4% were extensively drug-resistant (XDR) 17.3% were non-MDR and were susceptible to the majority of antibiotics tested. These results highlight the high level of resistance to antimicrobials in clinical P. aeruginosa, as condensed in Table 4.3.

Table 2. Distribution of AMR-Phenotypes of Clinical Isolates of *P. aeruginosa*

AMR-Phenotype	**Frequency**	**Percentage (%)**
Non-MDR	5	17.3
MDR	17	58.6
XDR	1	3.4
PDR	6	20.7
Total	**29**	**100%**

Biofilm Production among Clinical Isolates of Pseudomonas aeruginosa: Results: The degree of biofilm formation by these isolates is summarized in Table 3. Of these isolates, 17 weakly, 48 moderately, and 34 strongly produced biofilms.

Among biofilm clusters, there was a significant difference in isolation site (χ^2=20.323, p = 0.03), with a large effect size (Cramer's V = 0.592). Wound isolates were strong biofilm formers (> 50%) and sputum and HVS isolates were all or almost all weak formers. Similar to previous observations, we found no evidence of weak biofilm formers among the isolates derived from urine, blood and pleural fluids (Table 4), further

reinforcing the notion that P. aeruginosa from wounds has a greater tendency for potent biofilm formation.

Table 3. Strength of Biofilm Formation among Clinical Isolates of *P. aeruginosa*

Degree of Biofilm Formation	Frequency	Percentage (%)
Weak Biofilm Formers	5	17
Moderate Biofilm Formers	14	48
Strong Biofilm Formers	10	34
Total	**29**	**100**

Table 4. Distribution of Weak, Moderate and Strong Biofilm Formers across Different Sites of Isolation

Site of Isolation	WBF *n* (%)	MBF n (%)	SBF *n* (%)	Total *n* (%)	χ^2	*p*-value
Wound	1 (9)	4 (36)	6 (55)	11 (100)	20.323	0.03*
Urine	0 (0)	4 (67)	2 (33)	6 (100)		
Pus	2 (40)	2 (40)	1 (20)	5 (100)		
Blood	0 (0)	2 (67)	1 (33)	3 (100)		
Pleural fluid	0 (0)	2 (100)	0 (100)	2 (100)		
Sputum	1(100)	0 (0)	0 (0)	1 (100)		
HVS	1(100)	0 (0)	0 (0)	1 (100)		

Note. WBF: weak biofilm former, MBF: moderate biofilm former, SBF: strong biofilm former, *: Significant at 0.05 level. Cramer's V = 0.592,

Formation of biofilm and its relation with antimicrobial susceptibility: Biostatistics analyses based on Pearson's chi-square test with Fisher Freeman Haltom's corrections identified no significant correlation of biofilm formation and antimicrobial resistance pattern in clinical P. aeruginosa isolates (p 0.05) (Table 5). Some dissidence was observed in the susceptibility of biofilm-producing groups, among them more resistance to ceftazidime (90%) and cefepime (70%) in strong biofilm formers, however none of the evaluated antibiotics presented a significant association (p value between 0.166 and 0.818).

Table 5. Association between Antibiotic Susceptibility and Strength of Biofilm Formation among Clinical Isolates of *P. aeruginosa*.

Antibiotic	Strength of Biofilm Formation			χ^2	F.F.H	*p* value
	Weak	Moderate	Strong			
Meropenem	S 2 (40) I 0 (0) R 3 (60)	S 9 (64) I 0(0) R 5 (36)	S 4 (40) I 1 (10) R 5 (50)	3.302	3.463	.584
Imipenem	S 2 (40) I 0 (0) R 3 (60)	S 0 (0) I 1 (100) R 0 (0)	S 6 (60) I 0 (0) R 4 (40)	2.424	2.800	.660
Cefepime	S 2 (40) I 0 (0)	S 7 (50) I 2 (14)	S 3 (30) I 0 (0)	4.115	3.516	.410

Antibiotic	Strength of Biofilm Formation			χ^2	F.F.H	*p* value
	Weak	Moderate	Strong			
Ceftazidime	R 3 (60) S 2 (40) I 0 (0)	R 5 (36) S 7 (50) I 0 (0)	R 7 (70) S 1 (10) I 0 (0)	4.213	4.249	.166
P. tazobactam	R 3 (60) S 2 (40) I 0 (0)	R 7 (50) S 6 (43) I 1 (7)	R 9 (90) S 3 (30) I 2 (20)	1.898	1.760	.818
Ciprofloxacin	R 3 (60) S 2 (40) I 1 (20)	R 7 (50) S 7 (50) I 1 (10)	R 5 (50) S 2 (20) I 2 (20)	2.700	3.155	.651
Levofloxacin	R 2 (40) S 4 (80) I 1 (20)	R 6 (40) S 8 (57) I 2 (14)	R 6 (60) S 6 (60) I 1 (10)	2.030	2.240	.792
Gentamicin	R 0 (0) S 0 (0) I 1 (20)	R 4 (29) S 0 (0) I 1 (7)	R 3 (30) S 0 (0) I 0 (0)	2.079	2.147	.655
Tobramycin	R 4 (80) S 2 (40) I 2 (40) R 1 (10)	R 13 (93) S 9 (64) I 1 (7) R 4 (29)	R10 (100) S 3 (30) I 2 (20) R 5 (50)	5.025	4.841	.304

Note. F.F.H means Fisher Freeman Halton, S mean sensitive, I mean intermediate, R mean Resistance.

Discussion

The organism was isolated from a number of clinical samples in the present study, with statistical analysis revealing a significant association between the rate of isolation and the site from which the clinical specimen was taken (Chi square). There was considerable heterogeneity in the susceptibility of the isolates to the antibiotics. Imipenem (a carbapenem beta-lactam) and the fluoroquinolone, levofloxacin showed the highest rates of susceptibility. In contrast gentamicin (an aminoglycoside) was able to induce the strongest resistance. Ceftazidime (a 3rd generation cephalosporin) had a peculiar susceptibility profile with no intermediate susceptibility at all. The resistance pattern detected in the present study are significantly different from the previously published. In particular, our results are in contrast to those of Arabestani et al. [15] and Ugwuanyi et al. [16]. In Mosul who recorded universal resistance (100%) to ceftazidime among clinical isolates; and Arabestani et al. [15] in Iran, who found the highest resistance rates of 61.2% for levofloxacin. These differences might be due to the factors including: differences in regional antibiotic prescribing habits, geographical variability in the distribution of resistances genes, dissimilarities of the methodology of susceptibility testing and evolving bacterial adaptation exposed to different pressures of selection.

The review indicated disturbing levels of antimicrobial resistance phenotypes: half of the isolates qualified as multidrug resistant (MDR, resistant to ≥1 agent in ≥3

antimicrobial classes), and one-fifth qualified as pan-drug resistant (PDR). Only 1 isolate was thought to be extensively drug-resistant (XDR), with 1/5th being non-MDR. In summary, MDR and PDR phenotypes accounted for more than 2/3 of the clinical isolates, far emphasizing the large burden of the resistant isolates in this population. The prevalence of resistances observed in this study indicated a significant increase in comparison with Olaniran et al. [17], who found much lower rates in South West Nigeria (MDR: 25.4 %; XDR: 10.9 %; PDR: 5.98 %). This epidemiological divergence could be due to: variations in regional antimicrobial stewardship/diagnostic stewardship and possibly a steady rise of AMSB threats.

All the clinical P. aeruginosa isolates in our study were biofilm producers with different levels of production; this finding of 100% biofilm production is similar to that of Ugwuanyi et al. [16] in Nigeria, who also observed 100% biofilm forming ability in clinical isolates of P. aeruginosa but by genotyping method. Seventeen percent of isolates were weak biofilm formers, 48% were moderate and 34% were strong biofilm producers as per tissue culture plate method. The biofilm formation capacity varied significantly between the isolation sites. The intensity of biofilm formation was significantly related with the site of isolation according to the statistical analysis ($\chi^2 = 20.323$, $p = 0.03$) Wound isolates were the majority (60%) of strong biofilm producers, while other sites (pus, pleural fluid, blood, HVS and sputum) were mostly either weak or moderate biofilm producers. The large effect size measures Mathur et al. [14] (Cramer's V = 0.592) support a clinically meaningful association between site of isolation and the propensity for biofilm production.

CONCLUSION

Fundamental Finding: The study highlights a universal biofilm formation among clinical *P. aeruginosa* isolates, particularly from wound sources, alongside concerning rates of antimicrobial resistance. **Implication:** Its role in persistent infections underscores the need for targeted strategies. **Limitation:** Biofilm production did not correlate directly with resistance patterns. **Future Research:** Targeted strategies to disrupt biofilm formation in clinical settings.

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