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Original Research Article

Correlation Between Biofilm Formation and Antibiotic Resistance Among Clinical Isolates of Pseudomonas aeruginosa: A Cross-Sectional Study in Patna Medical College and Hospital, Patna

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Abstract:

Background: Pseudomonas aeruginosa is a major opportunistic pathogen associated with severe healthcare-associated infections. Its ability to form biofilms is a key virulence factor that contributes to persistence of infection and increased antibiotic resistance.

Aim: To determine the prevalence of biofilm formation among clinical isolates of P. aeruginosa and to evaluate its correlation with antibiotic resistance.

Methodology: A cross-sectional study was conducted on 94 non-repetitive clinical isolates of P. aeruginosa obtained from various specimens in a tertiary care hospital. Isolates were identified by standard microbiological methods. Antibiotic susceptibility testing was performed using the Kirby–Bauer disk diffusion method as per CLSI guidelines. Biofilm production was assessed by the Congo Red Agar method, and its association with antibiotic resistance was statistically analyzed.

Results: Biofilm formation was observed in 32 (34.0%) isolates, with 14 (14.9%) strong and 18 (19.1%) moderate producers. Biofilm producers were more frequent in ICU settings and in tracheal aspirate and urine samples. High resistance was noted against cefepime (80.9%) and ceftazidime (63.8%), while all isolates were sensitive to colistin. A strong association was observed between biofilm production and resistance to multiple antibiotics, including carbapenems, aminoglycosides, and fluoroquinolones.

Conclusion: Biofilm formation in P. aeruginosa is significantly associated with multidrug resistance, posing a serious therapeutic challenge. Routine detection of biofilms and rational antibiotic use are essential for effective infection management.

Keywords: Pseudomonas aeruginosa, biofilm, antibiotic resistance, multidrug resistance, clinical isolates, cross-sectional study.

Introduction

Pseudomonas aeruginosa is an aerobic, motile, nutritionally versatile bacterium with a Gram-negative phenotype that has a wide distribution in nature. It is an opportunistic human pathogen implicated in both community-acquired and healthcare-associated infections, carrying profound significance in morbidity, mortality, and healthcare expenditure [1]. Infections due to P. aeruginosa are very often life-threatening and notoriously difficult to treat because of its intrinsic resistance to multiple antimicrobials and the frequent emergence of MDR strains within healthcare settings [2, 3]. This multidrug resistance not only limits therapeutic options but also contributes to prolonged hospitalization, increased costs of treatment, and higher morbidity and mortality rates among infected patients.

Biofilm formation is considered one of the key virulence factors of P. aeruginosa. Biofilms are microbially derived sessile communities in which cells are irreversibly attached to a substratum or to each other, embedded within a self-produced extracellular polymeric substance (EPS) matrix, and exhibit an altered phenotype in terms of growth rate and gene transcription [4]. This biofilm mode of growth protects bacteria against host immune responses and antimicrobial agents, thereby enabling persistence of infection even under adverse conditions. Bacteria in biofilms communicate through chemical signaling molecules-what is

known as quorum sensing-which controls a wide array of physiological activities including virulence factor production and biofilm maturation [5].

The process of biofilm formation depends on a variety of environmental and bacterial factors, which include the availability of essential nutrients, chemotactic motility toward surfaces, the motility of bacteria, the presence of surface adhesins, and surfactants [6]. These factors together contribute to the initial attachment of bacterial cells onto surfaces, microcolony formation, and the establishment of a mature, structurally complex biofilm. Biofilm-associated infections are difficult to treat because the bacteria present in the biofilms are as much as 1,000 times more resistant to antibiotics compared to their planktonic counterparts [7].

Biofilms are central to the pathogenesis of many chronic and recurrent infections, such as those related to indwelling medical devices, dental plagues, upper respiratory tract infections, and urogenital infections [8]. The persistence of bacteria capable of forming biofilms in clinical settings has been associated with nosocomial infections and represents a serious challenge to infection control. Parallel to the aforementioned challenges, the global rise in multidrug-resistant organisms, including MDR strains of P. aeruginosa, has further complicated the management of infections due to this pathogen [9]. Resistant strains delimit the effectiveness of commonly used antibiotics, thus raising the need for new approaches toward therapy and further emphasizing the careful monitoring of resistance patterns.

The development of MDR P. aeruginosa is alarming because it narrows effective antimicrobial treatment options. Indeed, there is an increasing amount of evidence that a strong correlation between biofilm formation and antibiotic resistance exists. It was observed in many types of biofilm-producing strains that increased resistance to several classes of antibiotics could be identified. This correlation justifies the study of both biofilm formation and the determination of antibiotic susceptibility patterns in clinical isolates to better understand how such organisms cause persistent infections and to guide appropriate management and treatment approaches.

Given the clinical importance of infections caused by P. aeruginosa and the involvement of biofilms in the mediation of antibiotic resistance, this study was undertaken with the following objectives: isolation and identification of Pseudomonas aeruginosa from various clinical specimens, determination of the biofilm-forming capability of these strains, and analysis of the antibiotic susceptibility patterns among the isolates, correlating the same for biofilm production with antibiotic resistance. These objectives, when addressed, would add to the literature on the correlation between biofilm formation and antibiotic resistance among P. aeruginosa clinical strains and would have implications for better infection management and judicious use of antimicrobials.

Material and Methods

Study Design: This was a cross-sectional observational study conducted to assess the correlation between biofilm formation and antibiotic resistance among clinical isolates of Pseudomonas aeruginosa.

Study Area: The study was carried out in the Department of Microbiology, Patna Medical College and Hospital, Patna, Bihar, India.

Study Duration: The study was conducted over a period of 7 months from February 2025 to August 2025

Sample Size: A total of 94 non-repetitive clinical isolates of Pseudomonas aeruginosa obtained from various clinical specimens were included in the study.

Sample Population: Clinical specimens, including blood, urine, sputum, wound swabs, catheter tips, tracheal aspirates, pus, and other body fluids, were collected from patients attending Patna Medical College and Hospital during the study period.

Data Collection: All clinical samples submitted to the Department of Microbiology for routine diagnostic purposes were processed according to standard microbiological protocols. Isolation and identification of P. aeruginosa were performed using standard phenotypic techniques. Antibiotic susceptibility testing of all isolates was carried out by the Kirby-Bauer disk diffusion method on Mueller-Hinton Agar using a 0.5 McFarland turbidity standard. The antibiotics tested included Ceftazidime, Cefepime, Ceftazidime-clavulanic acid, Piperacillin-tazobactam, Imipenem, Meropenem, Gentamicin, Amikacin, Ciprofloxacin, Levofloxacin, Colistin, Fosfomycin, Norfloxacin, and Nitrofurantoin for urinary isolates. The zones of inhibition were measured and interpreted according to the Clinical and Laboratory Standards Institute (CLSI) guidelines. Biofilm formation was assessed using the Congo Red Agar (CRA) method, where isolates producing black colonies were considered biofilm producers.

Inclusion Criteria

- Non-repetitive clinical isolates of Pseudomonas aeruginosa from different clinical specimens.
- Isolates obtained from patients of all age groups and both sexes.

Exclusion Criteria

- Duplicate isolates from the same patient.
- Environmental or non-clinical isolates.

- Contaminated samples or those with mixed bacterial growth where P. aeruginosa could not be isolated.

Procedure: Clinical specimens were collected according to aseptic procedures and processed immediately in the microbiology laboratory. Pseudomonas aeruginosa isolates were identified using standard phenotypic methods, followed by antibiotic susceptibility testing using the Kirby-Bauer disk diffusion method. For biofilm detection, isolates were inoculated on Congo Red Agar plates prepared with Brain Heart Infusion broth, sucrose, agar, and Congo Red stain. Plates were incubated aerobically at 37°C for 24 hours, and the presence of black colonies was interpreted as indicative of biofilm formation. The resistance profiles of the isolates were then correlated with their biofilm-forming ability to evaluate the relationship between antibiotic resistance and biofilm production.

Statistical Analysis: Data obtained from the study were entered and analyzed using standard statistical methods. Descriptive statistics, such as percentages and proportions, were used to summarize the prevalence of biofilm formation and antibiotic resistance among the isolates. The association between biofilm formation and antibiotic resistance was analyzed using the Chi-square test or Fisher's exact test where appropriate. A p-value of less than 0.05 was considered statistically significant, indicating a meaningful correlation between the variables.

Result

Table 1 shows the distribution of Pseudomonas aeruginosa isolates obtained from different clinical samples. The majority of isolates were recovered from sputum samples, accounting for 36 cases (38.3%), followed by urine samples with 22 isolates (23.4%). Pus samples contributed 15 isolates (16.0%), while tracheal aspirates accounted for 9 isolates (9.6%). Smaller proportions were obtained from wound swabs (7.4%), catheter tips (3.2%), and blood samples (2.1%). Overall, respiratory and urinary specimens constituted the main sources of P. aeruginosa isolation in this study.

Table 1: Distribution of Pseudomonas aeruginosa isolates from different clinical samples (N = 94)

Clinical samples	Number of isolates (%)
Sputum	36 (38.3%)
Urine	22 (23.4%)
Pus	15 (16.0%)
Tracheal aspirate	9 (9.6%)
Wound swab	7 (7.4%)
Catheter tip	3 (3.2%)
Blood	2 (2.1%)
Total	**94 (100%)**

Table 2 summarizes the antibiotic resistance pattern of 94 Pseudomonas aeruginosa isolates. Complete susceptibility was observed to colistin, with all isolates (100%) being sensitive. High sensitivity was also noted for imipenem (83.0%), gentamicin (78.7%), and meropenem (76.6%). Moderate sensitivity was seen with piperacillin–tazobactam (70.2%), ciprofloxacin (69.1%), amikacin (67.0%), and levofloxacin (63.8%). In contrast, high resistance rates were observed for cephalosporins, particularly cefepime (80.9%) and ceftazidime (63.8%). Overall, the table highlights substantial multidrug resistance among P. aeruginosa, with colistin and carbapenems remaining the most effective therapeutic options.

Table 2: Antibiotic resistance pattern of Pseudomonas aeruginosa isolates (N = 94)

Antibiotics	Sensitive isolates (%)	Resistant isolates (%)
Imipenem	78 (83.0%)	16 (17.0%)
Meropenem	72 (76.6%)	22 (23.4%)
Piperacillin–tazobactam	66 (70.2%)	28 (29.8%)
Ceftazidime + clavulanic acid	61 (64.9%)	33 (35.1%)
Ceftazidime	34 (36.2%)	60 (63.8%)
Cefepime	18 (19.1%)	76 (80.9%)
Ciprofloxacin	65 (69.1%)	29 (30.9%)
Levofloxacin	60 (63.8%)	34 (36.2%)
Gentamicin	74 (78.7%)	20 (21.3%)
Amikacin	63 (67.0%)	31 (33.0%)
Colistin	94 (100%)	0 (0%)

Table 3 shows the distribution of biofilm production among Pseudomonas aeruginosa isolates assessed by the Congo Red Agar method. Out of 94 isolates, 14 (14.9%) were strong biofilm producers and 18 (19.1%) were moderate biofilm producers, while the majority, 62 isolates (66.0%), did not produce biofilm. Overall, one-third of the isolates (34.0%) demonstrated biofilm-forming ability, indicating a substantial presence of biofilm-producing P. aeruginosa in the study.

Table 3: Biofilm production among Pseudomonas aeruginosa isolates by Congo Red Agar method (N = 94)

Biofilm production	Number of isolates (%)
Strong	14 (14.9%)
Moderate	18 (19.1%)
Non-biofilm	62 (66.0%)
Total	**94 (100%)**

Table 4 shows the distribution of biofilm-producing and non-biofilm-producing isolates according to hospital location among 94 samples. Nearly half of the biofilm-producing isolates were recovered from the ICU (14/32, 43.8%), followed closely by the wards (13/32, 40.6%), while only 15.6% originated from the outpatient department (OPD). In contrast, non-biofilm-producing isolates were most commonly obtained from ward settings (32/62, 51.6%) and OPD (24/62, 38.7%), with relatively few from the ICU (6/62, 9.7%). Overall, Table 4 indicates that biofilm-producing isolates were disproportionately associated with ICU settings, suggesting a higher burden of biofilm-related infections in critically ill patients.

Table 4: Distribution of biofilm-producing and non-biofilm-producing isolates according to hospital location (N = 94)

Location	Biofilm producers n=32 (%)	Non-biofilm producers n=62 (%)
ICU	14 (43.8%)	6 (9.7%)
Ward	13 (40.6%)	32 (51.6%)
OPD	5 (15.6%)	24 (38.7%)
Total	**32 (100%)**	**62 (100%)**

Table 5 shows the distribution of biofilm formation across different clinical samples among 94 isolates. Biofilm production was most frequent in isolates from tracheal aspirates, with 6 out of 9 samples (66.7%) showing biofilm formation. Urine samples also demonstrated a relatively high proportion of biofilm producers (10/22, 45.5%), followed by pus samples (6/15, 40.0%). Lower rates were observed in wound swabs (28.6%) and sputum samples (19.4%). Catheter tip isolates showed biofilm formation in one-third of cases (33.3%), while none of the blood isolates exhibited biofilm production. Overall, Table 5 indicates that biofilm formation varies by clinical source and is more common in isolates from invasive or device-related samples.

Table 5: Biofilm formation in relation to different clinical samples (N = 94)

Clinical samples	Biofilm producers n (%)	Non-biofilm producers n (%)
Sputum (n=36)	7 (19.4%)	29 (80.6%)
Urine (n=22)	10 (45.5%)	12 (54.5%)
Pus (n=15)	6 (40.0%)	9 (60.0%)
Tracheal aspirate (n=9)	6 (66.7%)	3 (33.3%)
Wound swab (n=7)	2 (28.6%)	5 (71.4%)
Catheter tip (n=3)	1 (33.3%)	2 (66.7%)
Blood (n=2)	0 (0%)	2 (100%)

Table 6 shows the relationship between antibiotic resistance and biofilm production among resistant Pseudomonas aeruginosa isolates. A high proportion of resistant isolates were biofilm producers, particularly for meropenem (81.8%), piperacillin–tazobactam (78.6%), imipenem (75.0%), gentamicin (75.0%), and amikacin (74.2%). Ciprofloxacin-resistant isolates also showed substantial biofilm production (62.1%). In contrast, ceftazidime-resistant isolates demonstrated a moderate association with biofilm formation (56.7%), while cefepime-resistant isolates had a lower proportion of biofilm producers (39.5%). Overall, the table indicates a strong association between multidrug resistance and biofilm-forming ability in P. aeruginosa, suggesting that biofilm production may contribute to increased antimicrobial resistance.

Table 6: Correlation between antibiotic resistance and biofilm production among resistant isolates of Pseudomonas aeruginosa			
Antibiotics	**Resistant isolates (n)**	**Biofilm producers n (%)**	**Non-biofilm producers n (%)**
Imipenem	16	12 (75.0%)	4 (25.0%)
Meropenem	22	18 (81.8%)	4 (18.2%)
Piperacillin–tazobactam	28	22 (78.6%)	6 (21.4%)
Ceftazidime	60	34 (56.7%)	26 (43.3%)
Cefepime	76	30 (39.5%)	46 (60.5%)
Ciprofloxacin	29	18 (62.1%)	11 (37.9%)
Gentamicin	20	15 (75.0%)	5 (25.0%)
Amikacin	31	23 (74.2%)	8 (25.8%)

Discussion

Among the 94 clinical isolates of Pseudomonas aeruginosa included in the present study, sputum specimens were the highest contributors, totaling 38.3%, followed by urine, 23.4%, and pus, 16.0%. These observations are in agreement with previous literature reports highlighting the predominant respiratory and urinary tract infections that P. aeruginosa causes. Koirala et al. (2010) [10] also reported a higher recovery of P. aeruginosa from respiratory samples, mainly tracheal aspirates, illustrating the opportunistic nature of this organism in respiratory infections and its relation to intubation and mechanical ventilation. Meanwhile, Al-Ahmad et al. (2014) [11] obtained considerable isolation from both urinary tract and wound infections, which agrees with the fact that this bacterium is a major pathogen for both community- and hospital-acquired infections. However, in our study, only 2 isolates were recovered from the blood, a low proportion compared to other works where isolation from the bloodstream was quite higher, indicating that there could have been differences in patient populations or the way samples were obtained (Collie et al., 2009) [12]."

The antibiotic susceptibility patterns in our study showed variable resistance with carbapenems being the most effective. Imipenem and meropenem showed sensitivity rates of 83.0% and 76.6%, respectively, while cephalosporins, especially cefepime, showed high resistance (80.9%). Fluoroquinolones and aminoglycosides had moderate to high activity, while all isolates were sensitive to colistin, consistent with its role as a drug of last resort for multidrug-resistant P. aeruginosa. This is consistent with the work of Drenkard (2003) [13], who reported that carbapenems were relatively effective against P. aeruginosa, although resistance is increasingly reported due to both plasmid-mediated and chromosomal mechanisms. Contrarily, Al-Ahmad et al. (2014) [9] reported slightly higher resistance rates to carbapenems and aminoglycosides, which may be due to regional differences in antibiotic usage and hospital-specific selective pressure. The obtained resistance to cephalosporins, especially cefepime, agreed with Freeman et al. (1989) [14], where resistance to extended-spectrum cephalosporins is often related to the β-lactamase production ability and/or biofilm formation.

Our analysis of biofilm formation showed that 34% of the isolates were able to produce biofilm, with 14.9% showing strong production and 19.1% moderate. Noticeably, the majority, 66.0%, were non-biofilm producers. This distribution is in partial disagreement with that suggested by Costerton et al. (1999) [15], stating that the larger portion of clinical P. aeruginosa isolates from clinical sources is biofilm producers in nature, especially in conditions of chronic infections. The reason may be attributed to the source of specimens or detection methodologies since biofilm formation varies under different environmental conditions or isolate genotype. In the present study, the biofilm-producing isolates were mainly recovered in the ICU (43.8%), followed by wards (40.6%), therefore indicating the predisposing role of a critical care setting for biofilm-associated infection through invasive devices and protracted exposure to antibiotics. This result agrees with the suggestion made by Stapper et al. (2004) [16] that hospital environments with high selective pressure favor the emergence of biofilm-forming strains.

Biofilm formation thus varied significantly with the sample types. The tracheal aspirates formed the highest portion of biofilm producers, 66.7%, which illustrates the well-understood risk of biofilm development in ventilator-associated pneumonia as described by Drenkard, 2003 [13]. Urine samples also had a high proportion of biofilm production, at 45.5%, further highlighting the involvement of biofilms in urinary tract infections associated with catheters. Conversely, sputum isolates formed fewer biofilms at 19.4%, and thus planktonic development may predominate over biofilm formation during certain respiratory infections, or that biofilm formation is determined by host factors coupled with microbial interactions as stated by Costerton et al., 1999 [14]. These findings are in concert with Al-Ahmad et al. in 2014 [9], who observed that biofilm

formation is higher in device-associated infections compared to those that are non-device associated.

A significant positive correlation between biofilm formation and antibiotic resistance was observed. In carbapenem-resistant isolates, 75% of imipenem-resistant and 81.8% of meropenem-resistant strains were biofilm producers. Resistance to piperacillin–tazobactam, fluoroquinolones, and aminoglycosides revealed similar trends where 62–78% of the resistant isolates were biofilm producers. These findings support the fact that one of the mechanisms of multidrug resistance is facilitated through biofilm formation, which promotes poor antibiotic penetration, allows the development of microenvironments that can support the growth of persister cells, and enhances horizontal gene transfer Hoyle & Costerton, 1991; Drenkard, 2003) [17,9]. However, cefepime-resistant isolates showed a smaller percentage of biofilm production (39.5%), which indicates involvement of non-biofilm mechanisms of resistance, such as active efflux or target site modifications. The same pattern was also reported by Al-Ahmad et al. (2014) [9], indicating a complex relationship between biofilm formation and specific antibiotic resistance mechanisms.

Overall, our study results bring into focus the clinical challenge posed by biofilm-forming P. aeruginosa. The findings illustrate the need for incorporation of MBEC testing, a biofilm-specific antimicrobial testing, into routine diagnostics to guide appropriate therapy. Further, the data highlights the need for targeted infection control interventions in both ICU and device-associated infections to prevent the spread of multidrug-resistant, biofilm-forming strains. A new therapeutic strategy could be the combination of classical antibiotics with biofilm-disruptive agents, thus improving the outcome and reducing chronic infections.

Conclusion

This cross-sectional study underscores the high clinical relevance of Pseudomonas aeruginosa as a pathogen that is isolated from a wide variety of samples, especially respiratory, urinary, and those from invasive device-related specimens, reflecting its resilience in adapting to various hospital environments. The results showed a disturbing multidrug resistance pattern, with higher resistance rates against most commonly prescribed antipseudomonal antibiotics and preserved susceptibility to last-resort agents, indicating their continued importance in therapy. Biofilm formation was identified in a large proportion of the recovered isolates, with strong associations among the specimens recovered from critical care settings and those that were related to indwelling devices and lower respiratory tract infections, suggesting that hospital-related factors and invasive procedures play a role in biofilm development. Importantly, a clear association was found between biofilm production and antibiotic resistance, as biofilm-forming isolates had a greater tendency to resist multiple classes of antibiotics compared to non-biofilm producers. This association emphasizes the role of biofilms in enhancing bacterial survival and persistence and promoting treatment failure by limiting the penetration of antibiotics and enhancing the adaptive mechanisms of resistance. Overall, the study emphasizes that regular screening for biofilm production should be considered, along with judicious use of antibiotics and strict control of infection, especially in high-risk areas of the hospital, as measures that are necessary in managing infection by Pseudomonas aeruginosa and curbing the increasing challenge of antimicrobial resistance.

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