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Original Research Article

Unmasking Biofilm Associated Antimicrobial Resistance in Pseudomonas Aerugionosa: Insights from a Tertiary Care Hospital in Northern India

Mudhita Mahajan[1], Usra Jawaid[2], Sonal Jindal[3], Molly Madan[4]

[1]Third year Post graduate Resident, Department of Microbiology, Al-Falah Medical College, Faidabad Haryana, India

[2]Tutor, Department of Microbiology, Al-Falah Medical College, Faidabad, Haryana, India

[3]Professor & Head, Department of Microbiology, Radha Govind Institute of Medical Sciences, Meerut, Uttar Pradesh, India

[4]Professor & Head, Department of Microbiology, Al-Falah Medical College, Faidabad, Haryana, India

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Corresponding author: Dr. Sonal Jindal

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Abstract

Background: *Pseudomonas aeruginosa* is an opportunistic gram-negative bacillus with intrinsic and acquired resistance mechanisms, making it a major pathogen in both community and healthcare settings. Biofilm production further enhances its persistence, resistance to antimicrobials, and ability to cause chronic or recurrent infections. Surveillance of local antimicrobial susceptibility trends, especially in healthcare centres, is essential for guiding empirical therapy and infection control.

Aim and Objectives: To determine prevalence of *P. aeruginosa* isolates from all the clinical samples in a tertiary care hospital and to correlate between biofilm formation and antimicrobial resistance for these isolates.

Methods: This study was carried out at the Department of Microbiology of Al-Falah School of Medical Sciences, Faridabad associated with tertiary care hospital for a period of one year (January to December 2024). Out of total 4040 clinical samples processed, 82 isolates of *P. aeruginosa* identified using standard culture and biochemical tests. Biofilm detection done using Congo Red Agar (CRA) method and Microtiter Plate Assay (MPA). Conventional Antimicrobial susceptibility testing (AST) was performed using Mueller–Hinton agar Kirby–Bauer disc diffusion method as per CLSI 2025 guidelines. Statistical analysis was done using SPSS version 26.

Results: Out of total 82 Pseudomonas isolates, 46 (56.1%) were from male patients and 36 (43.9%) from female patients. Outpatient isolates (72.0%) outnumbered inpatient isolates (28.0%). Maximum isolates were from ear pus (38; 46.3%), followed by sputum (25; 30.5%), pus (10; 12.2%), urine (3; 3.7%), blood (3; 3.7%), and conjunctival swabs (3; 3.7%). Biofilm assays revealed 34 strong (41.5%), 22 moderate (26.8%), 16 weak (19.5%) producers, and 10 are non-biofilm producers (12.2%). Piperacillin–tazobactam demonstrated the lowest resistance (3.7%), while imipenem exhibited the highest (56.1%). Strong biofilm producers showed significantly higher resistance to all tested antibiotics was statistically significant ($p < 0.05$).

Conclusion: High rates of biofilm formation in *P. aeruginosa* correlated with multidrug resistance, particularly against carbapenems and ceftazidime. Piperacillin–tazobactam remains the most reliable agent. Regular surveillance, biofilm screening, and strict antimicrobial stewardship are critical to prevent treatment failures and the spread of multidrug-resistant strains.

Keywords: Pseudomonas aeruginosa, Biofilm Formation, Antimicrobial Resistance.

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Introduction

Pseudomonas aeruginosa is an aerobic, motile, gram-negative bacillus belonging to the family Pseudomonadaceae. It is non-fermentative, oxidase-positive, and ubiquitously present in soil, water, and hospital environments. Despite being an environmental organism, it has emerged as a leading opportunistic pathogen in humans, particularly in immunocompromised individuals, patients with prolonged hospitalisation, those with indwelling medical devices, and individuals with chronic underlying conditions such as diabetes, malignancy, and chronic obstructive pulmonary disease (COPD) [1]. The clinical significance of *P. aeruginosa* lies in its remarkable ability to resist diverse classes of antimicrobials. Intrinsic resistance mechanisms include reduced outer-

membrane permeability, efflux pumps, and production of antibiotic-inactivating enzymes. Furthermore, the bacterium readily acquires resistance determinants through plasmids, integrons, and transposons [2].

P. aeruginosa has been categorised as a Priority-1 "critical pathogen" by the World Health Organisation (WHO) due to the urgent need for novel therapeutic strategies [3,4].

One of the most concerning features of *P. aeruginosa* is its ability to produce biofilm. A biofilm is a structured community of bacterial cells embedded within a self-produced extracellular polymeric substance (EPS), primarily composed of polysaccharides, proteins, and extracellular DNA [5].

Biofilms confer protection against host immune responses, disinfectants, and antibiotics, often leading to chronic infections. In biofilm mode, *P. aeruginosa* can tolerate antibiotic concentrations up to 1000 times higher than those required to kill planktonic cells [6].

This explains its notorious involvement in chronic lung infections in cystic fibrosis patients, device-associated infections (catheters, prostheses), and persistent wound infections [7].

Biofilm production is not uniform across isolates. Some strains are strong biofilm formers, others are weak, and a subset may fail to form biofilms. Importantly, biofilm production is directly associated with multidrug resistance (MDR). Strong biofilm-forming strains often harbour resistance to carbapenems, aminoglycosides, and fluoroquinolones.

India, with its diverse healthcare settings, faces a disproportionate burden of antimicrobial resistance (AMR). Several tertiary centres have reported rising carbapenem resistance in P. aeruginosa, reaching 40–60% in recent years. However, rural and semi-urban healthcare systems remain underrepresented in surveillance studies. These centres often encounter challenges such as over-the-counter antibiotic use, lack of antimicrobial stewardship, poor infection-control infrastructure, and limited diagnostic capacity. As a result, the true prevalence of MDR and biofilm-producing *P. aeruginosa* in rural India remains poorly characterised.

The present study aimed to determine the prevalence of *P. aeruginosa* isolates from all the clinical samples in a tertiary care hospital in North India with the objectives of assessing the trend of biofilm production among these isolates and to evaluate the antimicrobial susceptibility pattern, with emphasis on the correlation between biofilm formation and resistance.

Rising threat of AMR and biofilm formation of Pseudomonas isolates in our region, prompted us to conduct this study, to understand bacterial resistance pattern in our geographic region. This data will aid clinicians in selecting adequate empiric therapy and strengthening antimicrobial stewardship in Indian healthcare settings.

Materials and Methods

Study Design and Setting: This study was carried out at the Department of Microbiology of Al-Falah School of Medical Sciences, Faridabad associated with tertiary care hospital for a period of one year (January to December 2024).

Ethical approval for this study was obtained from the Institutional Ethics Committee. (IEC code: IEC-AFSMSRC/RP/2024-18).

Informed consent was not applicable since the data were retrieved from laboratory records.

Sample Collection and Processing: Total 4040 of all clinical samples (pus, ear swabs, sputum, urine, blood, conjunctival swabs, wound swabs, and others) received from both outpatients (OPD) and inpatients (IPD), of all age groups ,genders was included in the study. Samples are collected under aseptic precautions and transported to the microbiology laboratory within two hours.

Specimens were inoculated on Blood agar, MacConkey agar, and Cetrimide agar and incubated aerobically at 37°C for 24-48 h. Colonies suggestive of *Pseudomonas aeruginosa* were identified using conventional standard methods: colony morphology, pigmentation, gram staining, oxidase test, motility, inability to ferment glucose, and biochemical tests including indole, methyl red, citrate, urease, triple sugar iron agar reaction, nitrate reduction, and arginine dihydrolase [8]. Production of characteristic diffusible bluish-green pigment (pyocyanin) in media was considered confirmatory for *P. aeruginosa*.

Biofilm Detection Methods

Two complementary assays were used:

1. Congo Red Agar (CRA) Method:

- CRA plates were prepared with brain heart infusion agar, sucrose, and Congo red dye.
- After inoculation, plates were incubated at 37°C for 24–48 h.
- Black, dry crystalline colonies were interpreted as strong biofilm producers; darkened but smooth colonies as moderate; pink to red colonies as weak/non-biofilm producers.

2. Microtiter Plate Assay (MPA):

- Isolates were inoculated in tryptic soy broth with glucose and incubated in 96-well flat-bottom plates for 24 h at 37°C.

- Wells were washed, fixed, and stained with 0.1% crystal violet.
- Optical density (OD) at 570 nm was measured using a microplate reader.
- Cut-offs were calculated as per Stepanović criteria [9].
- OD >0.24 was considered strong biofilm, 0.12–0.24 moderate, 0.06–0.12 weak, and <0.06 non-biofilm producer.

Antimicrobial susceptibility testing (AST): AST was performed by conventional method using Mueller–Hinton agar by the Kirby–Bauer disc diffusion method, following CLSI 2025 guidelines [8]. The following antibiotic discs (HiMedia, India) were tested:

- **β-lactams:** Piperacillin–tazobactam (110 µg), Ceftazidime (30 µg), Aztreonam (30 µg)
- **Carbapenems:** Imipenem (10 µg), Meropenem (10 µg)
- **Fluoroquinolones:** Ciprofloxacin (5 µg), Levofloxacin (5 µg)

P. aeruginosa ATCC 27853 was used as a control. Zone diameters were interpreted as susceptible, intermediate, or resistant.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using SPSS v26 (IBM, USA).

Chi-square test was used to assess associations between biofilm production and resistance. A p-value <0.05 was considered statistically significant.

Results

Distribution of isolates: Of 4040 processed samples, 82 (2.0%) yielded *P. aeruginosa*. Male patients accounted for 56.1%, and females were 43.9%. The majority of isolates were from OPD (72.0%) compared to IPD (28.0%).

Table 1: Gender and patient-wise distribution of *P. aeruginosa* isolates

Gender	OPD (n=59)	IPD (n=23)	Total (n=82)	% Distribution
Male	33	13	46	56.1%
Female	26	10	36	43.9%
Total	**59 (72.0%)**	**23 (28.0%)**	**82**	**100%**

Department-wise distribution: Maximum isolates were obtained from the ENT department, followed by chest medicine and medicine.

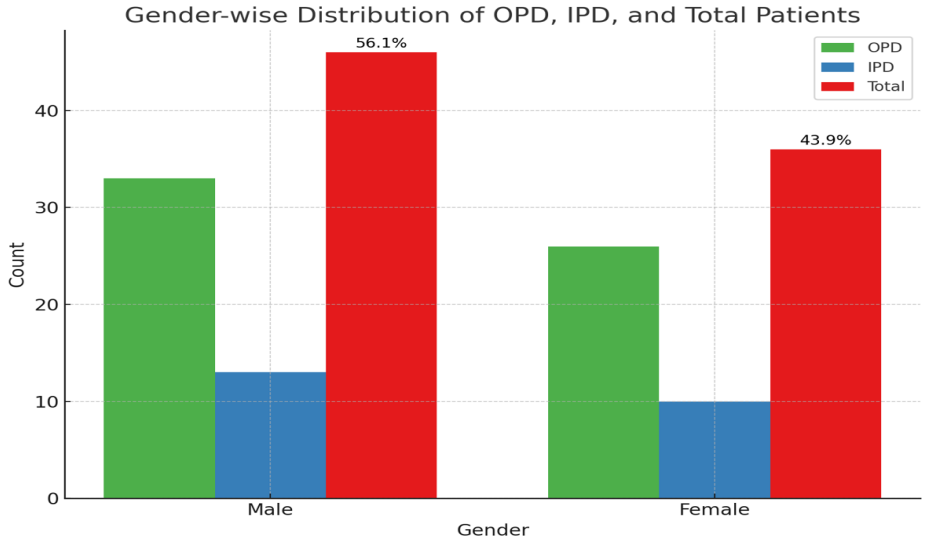

Figure 1: Gender wise distribution of OPD, IPD and Total patients

Table 2: Department-wise distribution of isolates

Department	OPD	IPD	Total	%
ENT	36	3	39	47.6%
Medicine	0	11	11	13.4%
Chest Med.	15	0	15	18.3%
Pediatrics	0	2	2	2.4%
Orthopedics	0	2	2	2.4%
Ophthalmology	2	0	2	2.4%
OBG	0	2	2	2.4%
Others	6	3	9	11.0%
Total	59	23	82	100%

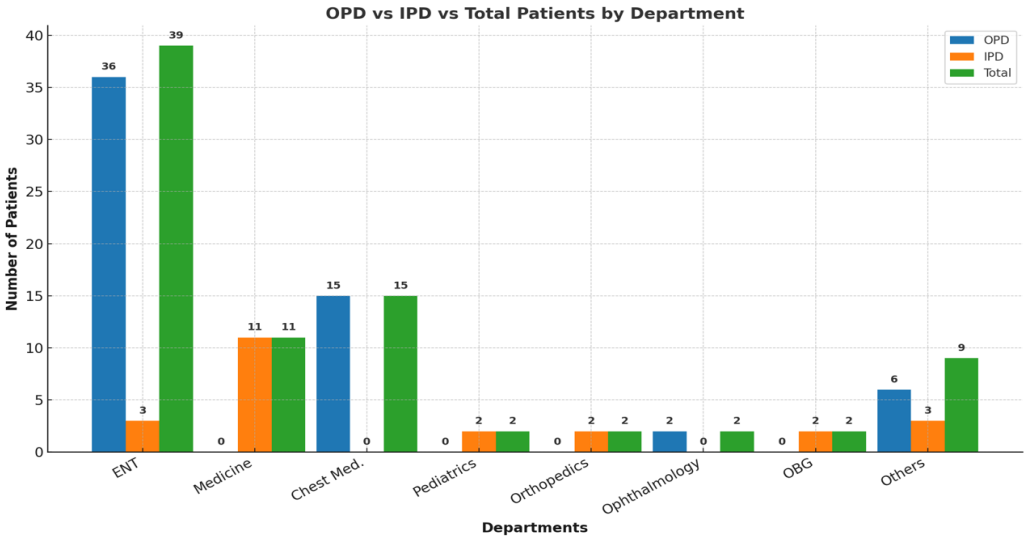

Figure 2: OPD vs IPD vs Total patients by department

Table 3: Distribution of isolates by clinical sample type

Sample type	No. of isolates	%
Ear pus	38	46.3%
Sputum	25	30.5%
Pus/wound swab	10	12.2%
Urine	3	3.7%
Blood	3	3.7%
Conjunctival swab	3	3.7%
Total	**82**	**100%**

Antimicrobial susceptibility: Resistance was highest to imipenem (56.1%), followed by ceftazidime (48.8%), ciprofloxacin (30.5%), levofloxacin (29.3%), aztreonam (23.2%), and meropenem (8.5%). Piperacillin–tazobactam showed the lowest resistance (3.7%).

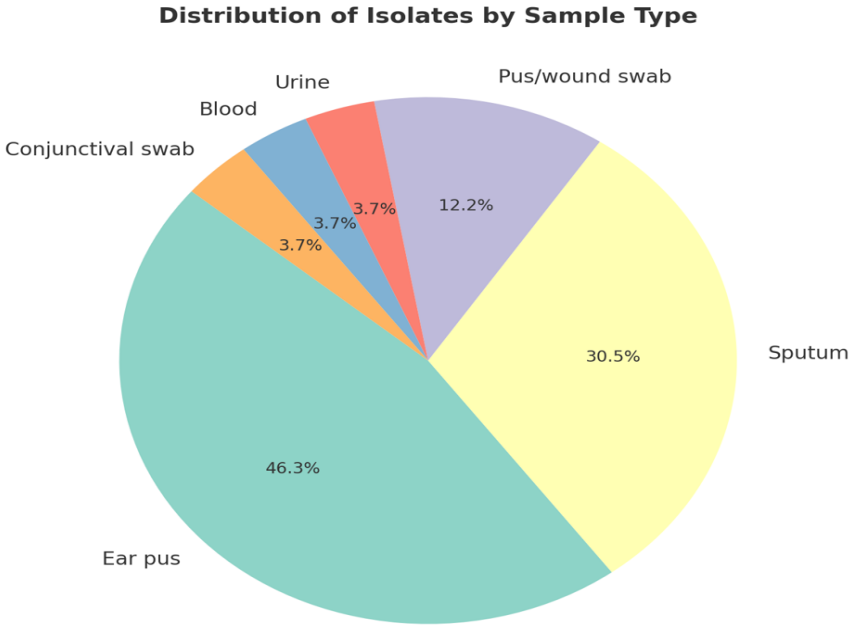

Figure 3: Distribution of isolates by sample type

Table 4: Antimicrobial susceptibility profile of *P. aeruginosa* isolates (n=82)

Antibiotic	Susceptible n (%)	Intermediate n (%)	Resistant n (%)
Piperacillin–Tazobactam	74 (90.2%)	5 (6.1%)	3 (3.7%)
Ceftazidime	36 (43.9%)	6 (7.3%)	40 (48.8%)
Ciprofloxacin	50 (61.0%)	7 (8.5%)	25 (30.5%)
Levofloxacin	51 (62.2%)	7 (8.5%)	24 (29.3%)
Aztreonam	53 (64.6%)	10 (12.2%)	19 (23.2%)
Meropenem	69 (84.1%)	6 (7.3%)	7 (8.5%)
Imipenem	30 (36.6%)	6 (7.3%)	46 (56.1%)

Biofilm production and resistance correlation: Biofilm assays revealed strong biofilm producers in 41.5% of isolates. Strong producers showed markedly higher resistance to carbapenems and fluoroquinolones.

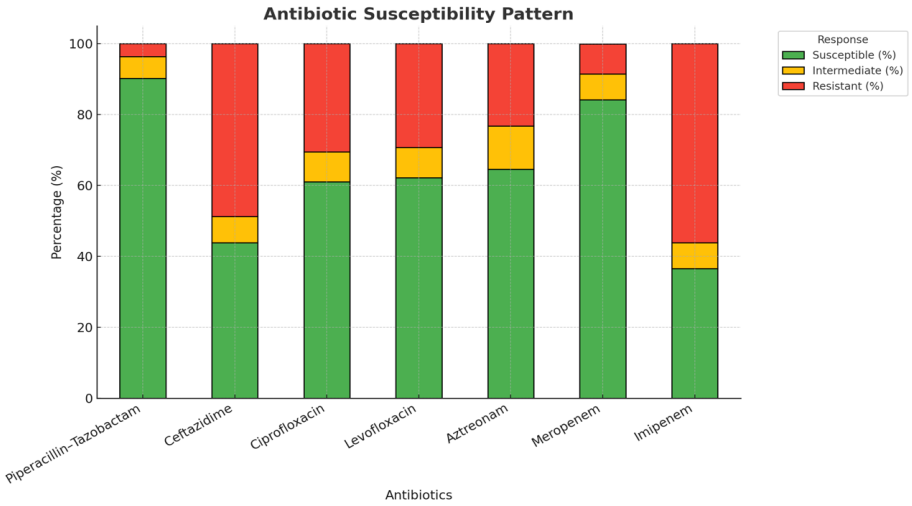

Figure 4: Antibiotic susceptibility pattern

Table 5: Correlation between biofilm production and resistance

Biofilm category	No. of isolates (%)	MDR rate (%)	High carbapenem resistance (%)
Strong	34 (41.5%)	85.3%	70.6% *
Moderate	22 (26.8%)	59.1%	40.9%
Weak	16 (19.5%)	31.2%	18.7%
Non-producer	10 (12.2%)	10.0%	0%

***p-value <0.05, statistically significant, showing the association of Strong Biofilm producer with more antimicrobial resistance.**

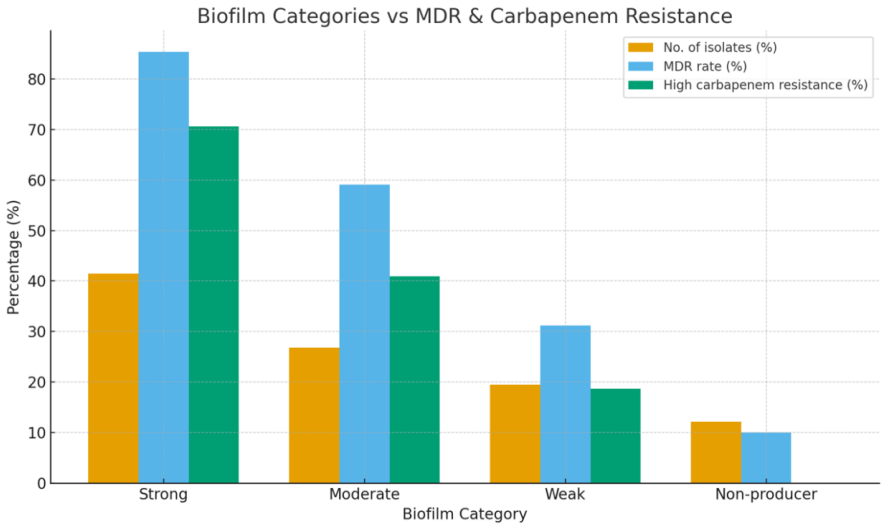

Figure 5: Biofilm categories vs MDR & Carbapenem Resistance

Discussion

The present analysis from a North Indian tertiary-care centre demonstrates two converging threats in *Pseudomonas aeruginosa*: (i) substantial resistance to key antipseudomonal agents—most notably carbapenems and third-generation cephalosporins—and (ii) a high prevalence of biofilm production with a clear association to multidrug resistance (MDR). Together, these findings help explain the clinical recalcitrance of many *P. aeruginosa* infections and underscore the urgency of strengthening diagnostics and stewardship in non-urban health systems.

Our overall resistance profile aligns with the broader literature describing *P. aeruginosa* as intrinsically difficult to treat due to a low-permeability outer membrane, inducible chromosomal AmpC, and robust efflux systems that collectively blunt the activity of several antibiotic classes [10]. The observed imipenem resistance (>50%) is consonant with hospital-based reports from South and Southeast Asia, including Thailand (~58%) and several Indian and Pakistani cohorts (42–50%), and exceeds the resistance reported in some Middle Eastern and African surveillance snapshots from earlier years. A plausible explanation for the comparatively higher imipenem resistance in our setting is the entrenched empirical use of carbapenems for severe sepsis syndromes in the absence of rapid diagnostics, a pattern that has been repeatedly associated with selection of carbapenem-resistant non-fermenters [11]. National programmatic data also point to a rising trend: the Indian Council of Medical Research (ICMR) network documented an increase in *P. aeruginosa* imipenem resistance from ~26% in 2017 to ~38.5% in 2023, suggesting that local peaks—such as those we report—may be harbingers of wider regional spread if unaddressed [12]. Ceftazidime resistance in our *Pseudomonas aeruginosa* isolates (~49%) is consistent with reports of high resistance rates (approximately 30–60%) from multicentre and regional surveillance studies, including data from Middle Eastern [13].While some centres report lower ceftazidime resistance (14–23%), these often reflect restricted formularies, lower antibiotic consumption, or earlier study periods, all of which temper selection pressure. By contrast, our rural context—with a high proportion of community-managed chronic ear and respiratory conditions—likely experiences repeated, unmonitored exposure to oral and parenteral anti-pseudomonal agents, fostering stepwise selection of β-lactam resistance.

In contrast to carbapenems and ceftazidime, piperacillin–tazobactam (PTZ) retained good activity, with resistance remaining in the single digits. This pattern is broadly consistent with reports from Singapore and several Indian centres that describe low-to-moderate PTZ resistance (≈5–12%) when stewardship programmes restrict indiscriminate use [14]. Nonetheless, other programmes have reported PTZ resistance above 30–40% in high-utilisation environments, signalling that the current susceptibility advantage is contingent and can be rapidly lost in the absence of local policy reinforcement [15]. Clinically, this supports a PTZ-first strategy for stable pseudomonal infections in our geography, reserving carbapenems for culture-proven indications or severe sepsis with high predicted resistance risk, as advocated by critical care and infectious disease guidance.

Fluoroquinolone non-susceptibility (~29% to ciprofloxacin and levofloxacin) occupies a middle ground relative to Indian and regional data: several tertiary centres report higher resistance (≈33–37%), whereas others—particularly where quinolones are less freely dispensed—document rates near 15–22% [16]. Given the pharmacokinetic advantages of quinolones in respiratory and ear infections, sustained stewardship attention is warranted; without it, resistance typically drifts upwards alongside cumulative community exposure.

A defining feature of our cohort is the prominence of biofilm-producing strains (≈61%), with strong producers showing markedly higher rates of MDR and carbapenem resistance than non-producers. This gradient mirrors mechanistic and clinical work demonstrating that biofilm architecture impedes antibiotic penetration, enriches slow-growing persister subpopulations, and facilitates horizontal exchange of resistance determinants—effects that can amplify phenotypic resistance by several orders of magnitude relative to planktonic cells [17]. Multiple Indian and neighbouring-country series report biofilm prevalence between 50% and 70% among *P. aeruginosa* clinical isolates and consistently identify a positive correlation between biofilm intensity and resistance, including to carbapenems and quinolones. Our data reinforce these observations and provide a rural-system context, with the highest biofilm yield from ear discharge and sputum—the very niches where chronic, surface-adherent infection biology dominates.

Specimen and service-line distribution further contextualise therapeutic challenges. ENT and chest medicine accounted for the largest share of isolates, and ear discharge was the single most common specimen in our series. Similar patterns have been recorded across Indian and Himalayan settings where chronic suppurative otitis media (CSOM) and chronic bronchitis syndromes remain prevalent and frequently under-treated or partially treated. In such conditions, biofilms frequently establish on epithelium and hardware (hearing aids, external ear devices), driving recurrent flares

despite apparently appropriate antibiotic courses. The male predominance we observed is in line with several Indian datasets and may reflect higher exposure to occupational and environmental reservoirs and care-seeking patterns in rural communities [18].

Two stewardship-relevant implications emerge. First, in biofilm-rich syndromes (CSOM, chronic tracheobronchitis, device-associated infections), systemic therapy should be paired with measures that disrupt biofilm—local debridement, topical antipseudomonal agents where appropriate, and strategies that improve penetration (e.g., optimised dosing, extended infusions for β-lactams). Second, empirical carbapenem use should be de-emphasised in favour of PTZ or aztreonam plus an antipseudomonal quinolone where local susceptibility supports it, with rapid de-escalation once culture data return [19].

Where laboratory capacity allows, reflex testing for carbapenemase production (e.g., mCIM/eCIM, Carba NP) and periodic molecular audits for class B (metallo-β-lactamases) and class D enzymes can guide formulary decisions and infection control priorities [20]. Our data also underscore the diagnostic value of incorporating biofilm phenotyping into routine workflows. Congo Red Agar (CRA) and semi-quantitative microtiter assays are low-cost, scalable platforms that correlate reasonably with clinical refractoriness and resistance risk, thereby informing early procedural adjuncts (e.g., drainage, debridement) and realistic counselling about treatment duration [21].

Although such assays are not yet standard in many laboratories, their incremental operational burden is modest relative to the clinical gains in chronic pseudomonal disease.

Compared with major academic centres, our hospital often operates with constrained infection-control staffing, fewer isolation facilities, and limited access to rapid diagnostics.

Over-the-counter antibiotic access and private-sector polypharmacy amplify selective pressure in the community. In this environment, even modest improvements—standardised specimen collection, timely culture prior to therapy, antibiogram-guided empiric pathways, and point-prevalence audits of carbapenem use—can deliver outsized benefit.

Our findings conclude *P. aeruginosa* in Northern India as a biofilm-proficient, increasingly drug-resistant pathogen for which PTZ retains useful activity, whereas imipenem and ceftazidime exhibit worrisome non-susceptibility. Concordance with multi-country data supports external validity, while the magnitude of carbapenem resistance and the biofilm-MDR linkage highlight a pressing need for locally tailored stewardship, diagnostic enhancement, and infection-control reinforcement [22].

Conclusion

P. aeruginosa remains a significant public health threat in Northern Indian healthcare settings. High prevalence of biofilm formation correlates strongly with antimicrobial resistance (p value <0.05), especially against carbapenems and ceftazidime. Piperacillin–tazobactam remains highly effective.

Limitations:

First only *Pseudomonas aeruginosa* was included in study using only Conventional method of pigment production. Other Pseudomonas species cannot be included in the study due to unavailability of automated detection methods. Secondly we did not perform phenotypic Carbapenemase confirmation or genotyping; thus, the specific enzymatic mechanisms (e.g., VIM, IMP, NDM, and OXA) remain undefined in our isolates. Thirdly we was not able to conclude outcomes (length of stay, relapse, and mortality).

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