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Prevalence and Antimicrobial Susceptibility Profile of Pseudomonas Aeruginosa Isolated from Diverse Clinical Specimens in A Tertiary Care Hospital

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Abstract:

Background: Pseudomonas aeruginosa is a ubiquitous, aerobic Gram-negative bacillus notorious for its capacity to colonise vulnerable patients and generate healthcare-associated infections that are notoriously difficult to manage. Its expanding multidrug-resistant (MDR) phenotype, underpinned by intrinsic low outer-membrane permeability, constitutive efflux pump expression, and a broad repertoire of acquired resistance mechanisms, has rendered empirical therapy increasingly unreliable and necessitates continuous local surveillance of susceptibility patterns.

Objectives: To isolate and confirm P. aeruginosa from diverse clinical specimens submitted to a tertiary care microbiology laboratory; to delineate the demographic and specimen-source profile of infected patients; and to generate a comprehensive, agent-specific antimicrobial susceptibility dataset against a representative panel of anti-pseudomonal antibiotics.

Methods: A prospective, descriptive, laboratory-based observational study was undertaken over six months (August 2025 to January 2026) at the Department of Microbiology, In a tertiary care hospital. A total of 174 non-duplicate P. aeruginosa clinical isolates were included. Species identification was performed by standard colonial morphology and a complete battery of biochemical tests (IMViC, oxidase, urease, TSI, OF test). Antimicrobial susceptibility testing was conducted by the Kirby-Bauer disk diffusion method on Mueller-Hinton agar in strict accordance with CLSI M100 guidelines (32nd edition, 2022).

Results: Male patients constituted 71.84% (n=125) of the study cohort. The >60-year age group yielded the highest number of isolates (32.76%). The majority of specimens originated from the in-patient department (83.33%). Sputum was the predominant specimen type (41.95%), followed by pus (17.24%) and ear swabs (15.52%). The General Medicine ward contributed the highest proportion of isolates (22.41%), followed by the ICU (18.97%). Among anti-pseudomonal agents tested, piperacillin-tazobactam demonstrated the highest sensitivity rate (87.93%), while the highest resistance rates were recorded for ceftazidime and aztreonam (42.53% each).

Conclusion: The study documents a concerning level of antimicrobial resistance in P. aeruginosa at this institution, particularly to third-generation cephalosporins and monobactams. Piperacillin-tazobactam and carbapenems retained relatively superior activity and may guide empirical treatment in clinically critical scenarios. Systematic susceptibility surveillance, stringent antimicrobial stewardship, and enhanced infection control measures are indispensable to curtail the dissemination of resistant clones.

Keywords: Pseudomonas aeruginosa; antimicrobial susceptibility; multidrug resistance; Kirby-Bauer disk diffusion; nosocomial infection; CLSI; piperacillin-tazobactam; antibiotic stewardship.

1. INTRODUCTION

The genus Pseudomonas, derived from the Greek words "pseudo" (false) and "monas" (single unit), encompasses a morphologically and metabolically diverse collection of aerobic, Gram-negative bacilli distributed across an extraordinary range of ecological niches. The species P. aeruginosa — whose epithet "aeruginosa" is derived from the Latin term for copper rust, describing its characteristic blue-green pigmentation — was first observed in 1850 by Sedillot, who noted the distinctive discolouration of contaminated surgical wound dressings. The causative organism was subsequently isolated in pure culture by Carle Gessard in 1882, who reported blue-green purulence in cutaneous wounds in two patients and formally associated the organism with the pathological process. [1,2]

Within the microbial kingdom, P. aeruginosa occupies a position of exceptional adaptability. It is an obligate aerobe capable of exploiting nitrate as a terminal electron acceptor under hypoxic conditions, facilitating survival in otherwise anaerobic microenvironments within tissues. The organism tolerates a broad temperature range of 4–42°C, with optimal growth between 37–41°C and can proliferate across a pH range of 5.6–8.0. Importantly, its capacity for growth at 42°C distinguishes it from closely related environmental pseudomonads such as P. putida and P. fluorescens, and forms the basis of a clinically useful phenotypic discriminator. [3,4].In immunocompetent individuals, P. aeruginosa functions essentially as an environmental commensal of limited virulence. However, in the setting of a compromised host — including patients with malignancy, burns, cystic fibrosis, or those receiving immunosuppressive therapies, broad-spectrum antibacterials, or undergoing invasive procedures — the organism exploits disrupted anatomical barriers and suppressed immune effectors to establish infection at virtually any anatomical site. The clinical spectrum ranges from localised skin and soft tissue infections, otitis externa, and corneal ulcers to severe and life-threatening syndromes including ventilator-associated pneumonia (VAP), catheter-associated urinary tract infection (CAUTI), bloodstream infection, and septic shock. [5,6].A defining characteristic of P. aeruginosa is its formidable capacity for antimicrobial resistance, expressed through three distinct biological tiers: intrinsic, acquired, and adaptive mechanisms. Intrinsic resistance arises from the inherently low permeability of its outer membrane, the constitutive expression of chromosomal AmpC beta-lactamase, and the inducible activity of four major resistance-nodulation-division (RND) family efflux pump systems — MexAB-OprM, MexCD-OprJ, MexEF-OprN, and MexXY-OprM — each conferring differential efflux of beta-lactams, quinolones, and aminoglycosides. Acquired resistance emerges through horizontal gene transfer of plasmid-encoded determinants, including extended-spectrum beta-lactamases (ESBLs) and metallo-beta-lactamases (MBLs), while adaptive resistance results from phenotypic reprogramming in response to environmental stimuli, most notably through biofilm formation and persister cell generation. [7,8]

The World Health Organization (WHO) has classified carbapenem-resistant P. aeruginosa among the "critical priority" pathogens for the development of new antimicrobial agents, reflecting the global threat posed by pan-drug-resistant strains that leave clinicians with no reliable therapeutic options. Surveillance data from the United States indicate approximately 51,000 healthcare-associated P. aeruginosa infections annually, with nearly 13% caused by MDR isolates. In European intensive care units, P. aeruginosa accounts for 15.9% of ventilator-associated pneumonia, 10.5% of urinary tract infections, and 27.8% of central-line-associated bloodstream infections, with overall infection-associated mortality approaching 20–50% depending on infection type and host vulnerability. [9,10,11]

In view of the escalating resistance burden and the consequent therapeutic challenges, ongoing institution-specific surveillance of P. aeruginosa prevalence, specimen source distribution, and antimicrobial susceptibility patterns is essential. Such localised data are critical for informing empirical treatment guidelines, identifying sentinel drug-resistant phenotypes, and directing targeted infection control interventions. Against this background, the present study was designed to characterise the clinical epidemiology and antimicrobial susceptibility landscape of P. aeruginosa at a tertiary care centre in northern India, with the specific objective of generating actionable evidence for local antibiotic stewardship and infection management programmes.

2. MATERIALS AND METHODS

2.1 Study Design, Setting and Duration: A prospective, descriptive, laboratory-based observational study was conducted over a six-month period from August 2025 to January 2026, in the Department of Microbiology, In a tertiary care hospital. The study protocol was reviewed and approved by the Institutional Ethics Committee. Written informed consent was secured from all enrolled patients prior to specimen collection.

2.2 Sample Size Estimation: The minimum required sample size was calculated using the standard formula for proportion estimation:

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$n = Z^2 \times p \times (1 - p) / d^2$

Applying a published prevalence of P. aeruginosa in tertiary care hospital isolates of 12.97% (p = 0.1297), a 95% confidence interval (Zα/2 = 1.96), and a margin of error of 5% (d = 0.05), the minimum sample size was calculated as n = 173.45, rounded to 174. [12]

2.3 Inclusion and Exclusion Criteria: Inclusion criteria encompassed: all clinical specimens yielding P. aeruginosa on primary culture, regardless of patient age, sex, ward, or admission category (in-patient or out-patient); only the first isolate per patient per infection episode was included to prevent duplication bias.

Exclusion criteria comprised: patients with documented prior antibiotic administration for the current infection episode (to avoid confounding susceptibility results); duplicate isolates of identical species from the same patient and specimen site; and specimens with insufficient volume for complete processing.

2.4 Specimen Collection and Processing: Clinical specimens — including sputum, pus, ear swabs, urine (midstream clean-catch in males; labial-separated mid-stream in females), blood (venipuncture into BACTEC bottles), bronchoalveolar lavage (BAL), endotracheal secretions, tracheal aspirates, pleural fluid, and tissue biopsies — were collected under stringent aseptic precautions and transported to the laboratory within two hours. When processing delay was unavoidable, urine and pus specimens were refrigerated at 4°C, while blood cultures were maintained at room temperature. [13]

2.5 Isolation and Identification of P. aeruginosa

2.5.1 Culture and Colonial Morphology: All specimens were inoculated onto 5% sheep Blood Agar (BA) and MacConkey Agar (MCA). Urine specimens were additionally plated onto HiCrome UTI Agar to facilitate direct colony identification. Plates were aerobically incubated at 37°C for 18–24 hours and examined for characteristic P. aeruginosa colonial morphology: large, flat, spreading colonies with a metallic sheen and distinct grape-like fruity odour on Blood Agar (often with beta-haemolysis), and lactose-non-fermenting colonies with blue-green pigmentation on MacConkey Agar, attributable to the production of the phenazine pigment pyocyanin. [14]

2.5.2 Microscopic Examination: Gram-stained smears were prepared from presumptive P. aeruginosa colonies. Organisms appearing as slender, non-sporing Gram-negative bacilli (0.5–1.0 µm × 1.5–5.0 µm), singly or in pairs, were selected for further biochemical characterisation. Motility was confirmed by the hanging drop method, with P. aeruginosa demonstrating brisk motility via polar flagella. [15]

2.5.3 Biochemical Identification: Definitive species identification was accomplished through a standardised biochemical test battery. P. aeruginosa was confirmed by: (i) positive oxidase reaction (blue-purple colour within 10 seconds with Kovac's reagent); (ii) negative Indole, Methyl Red, and Voges-Proskauer reactions; (iii) positive Citrate Utilisation (Simmons) test; (iv) positive urease; (v) alkaline/alkaline (K/K) pattern on Triple Sugar Iron (TSI) agar; (vi) oxidative (non-fermentative) glucose metabolism on Hugh-Leifson Oxidation-Fermentation (OF) medium; (vii) positive motility on semi-solid mannitol motility medium; and (viii) confirmed growth at 42°C, distinguishing P. aeruginosa from other pseudomonads. [16,17] Quality control was maintained throughout using ATCC reference strains (P. aeruginosa ATCC 27853, E. coli ATCC 25922, K. pneumoniae ATCC 700603).

2.6 Antimicrobial Susceptibility Testing: Antimicrobial susceptibility testing (AST) was conducted by the Kirby-Bauer disk diffusion method on Mueller-Hinton Agar (MHA), strictly adhering to the Clinical and Laboratory Standards Institute (CLSI) M100 performance standards, 32nd edition, 2022. [18] An inoculum suspension equivalent to the 0.5 McFarland turbidity standard (approximately 1–5 × 10^8 CFU/mL) was prepared from 24-hour pure cultures and uniformly inoculated onto MHA plates by three-directional swabbing. Antibiotic-impregnated disks were applied within 15 minutes of inoculation, gently pressed onto the agar surface, and incubated at 35°C ± 2°C for 16–18 hours. Zone diameters were measured to the nearest whole millimetre using calibrated calipers under reflected light and interpreted as Sensitive (S), Intermediate (I), or Resistant (R) according to CLSI species-specific breakpoints. [19] The following anti-pseudomonal agents were tested: ceftazidime (30 µg), cefepime (30 µg), aztreonam (30 µg), piperacillin-tazobactam (100/10 µg), amikacin (30 µg), gentamicin (10 µg), tobramycin (10 µg), ciprofloxacin (5 µg), levofloxacin (5 µg), imipenem (10 µg), and meropenem (10 µg). Quality control was performed using P. aeruginosa ATCC 27853.

Table 1: Antibiotic Disk Content and CLSI M100 (2022) Interpretive Breakpoints for P. aeruginosa

Antibiotic	Abbreviation	Disk (µg)	S (≥ mm)	I (mm)	R (≤mm)
Ceftazidime	CAZ	30	≥18	15–17	≤14
Cefepime	CPM	30	≥18	15–17	≤14
Aztreonam	AT	30	≥22	16–21	≤13
Piperacillin-Tazobactam	PIT	100/10	≥21	15–20	≤14
Amikacin	AK	30	≥17	15–16	≤14
Gentamicin	GEN	10	≥15	13–14	≤12
Tobramycin	TOB	10	≥15	13–14	≤12
Ciprofloxacin	CIP	5	≥25	19–24	≤18
Levofloxacin	LE	5	≥22	15–21	≤14
Imipenem	IMP	10	≥19	16–18	≤15
Meropenem	MRP	10	≥19	16–18	≤15

Table 1: Disk diffusion breakpoints per CLSI M100 (32nd ed., 2022). S=Sensitive; I=Intermediate; R=Resistant.

2.7 Statistical Analysis: All data were entered and managed in Microsoft Excel 2019 (Microsoft Corporation, Redmond, WA). Statistical analysis was performed using SPSS version 26.0 (IBM Corporation, Armonk, NY). Categorical variables were summarised as frequencies and percentages. Z-scores and corresponding p-values were calculated for sensitivity proportions of each antibiotic against a reference mean sensitivity proportion (76.51%) to identify agents with statistically significant deviation from the central tendency. A p-value ≤0.05 was used to denote statistical significance.

3. RESULTS

3.1 Demographic Profile: A total of 174 non-duplicate P. aeruginosa clinical isolates were collected and analysed over the six-month study period. Gender analysis revealed a pronounced male predominance: male patients contributed 125 isolates (71.84%) and female patients 49 (28.16%), yielding a male-to-female ratio of approximately 2.55:1 (Figure 1). This pattern was consistent across nearly all age strata.

Figure 1: Gender Distribution of P. Aeruginosa Clinical Isolates (n=174). Male patients constituted 71.84% of the cohort.

Age stratification revealed that the >60 year group carried the highest burden of infection, contributing 57 isolates (32.76%), followed by the 46–60 year cohort (47 isolates; 27.01%) and the 31–45 year group (33 isolates; 18.97%). The youngest age group (0–15 years) accounted for the smallest proportion (10 isolates; 5.75%). The complete age and gender distribution is presented in Table 2 and Figure 2.

Table 2: Age and Gender Distribution of P. aeruginosa Isolates (n=174)

S.No.	Age Group (yrs)	Male	Female	Total	Percentage (%)
1	0–15	7	3	10	5.75%
2	16–30	11	16	27	15.51%
3	31–45	23	10	33	18.97%
4	46–60	39	8	47	27.01%
5	>60	45	12	57	32.76%
—	**Total**	125	49	174	100.00%

Table 2: Complete age and gender distribution of 174 P. aeruginosa clinical isolates across five age strata.

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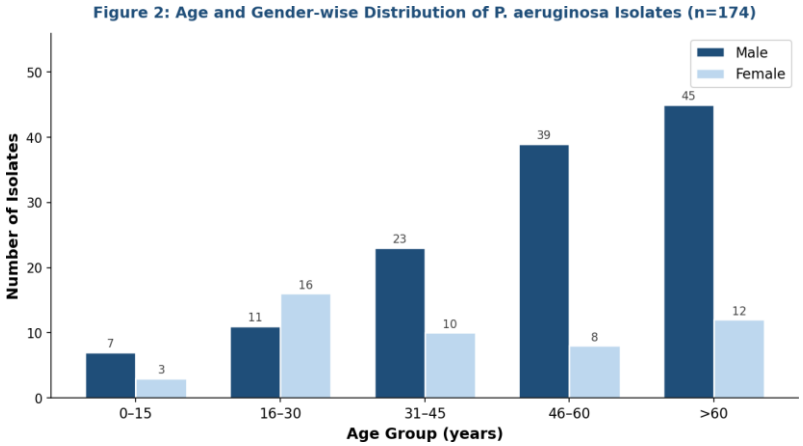

Figure 2: Age and Gender-wise Distribution of P. aeruginosa Isolates (n=174). The >60 year cohort demonstrated the greatest burden of infection.

3.2 In-Patient vs Out-Patient Distribution: The vast majority of P. aeruginosa isolates originated from hospitalised, in-patient department (IPD) patients: 145 of 174 isolates (83.33%) were from IPD sources, while only 29 (16.67%) were from the out-patient department (OPD), underscoring the predominantly nosocomial character of P. aeruginosa infection at this institution (Figure 6 and Table 3).

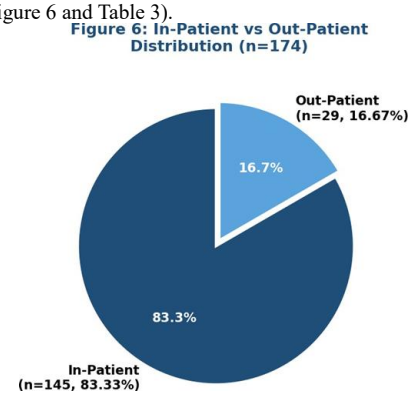

Figure 3: Distribution of P. aeruginosa Isolates by Patient Category (In-Patient vs Out-Patient) (n=174).

3.3 Department-wise Distribution: Among the 174 isolates, the highest number was recorded from the General Medicine ward (39 isolates; 22.41%), followed by the Intensive Care Unit (33; 18.97%), General Surgery (30; 17.24%), Respiratory ward (26; 14.94%), and Ear-Nose-Throat (ENT) department (23; 13.22%). The remaining contributions were from Emergency (10; 5.75%), Orthopaedics (8; 4.60%), Paediatrics (3; 1.72%), and Obstetrics and Gynaecology (2; 1.15%). Table 3 and Figure 3 illustrate the departmental distribution.

Table 3: Department-wise Distribution of P. aeruginosa Isolates (n=174)

Department	Total Isolates	OPD	IPD	Percentage (%)
General Medicine	39	5	34	22.41%
ICU	33	0	33	18.97%
General Surgery	30	4	26	17.24%
Respiratory	26	3	23	14.94%
ENT	23	13	10	13.22%
Emergency	10	0	10	5.75%
Orthopaedics	8	3	5	4.60%
Paediatrics	3	1	2	1.72%
Obs. and Gynaecology	2	0	2	1.15%
Total	174	29 (16.67%)	145 (83.33%)	100.00%

Table 3: Department-wise distribution of 174 P. aeruginosa isolates. OPD = Out-Patient Department; IPD = In-Patient Department.

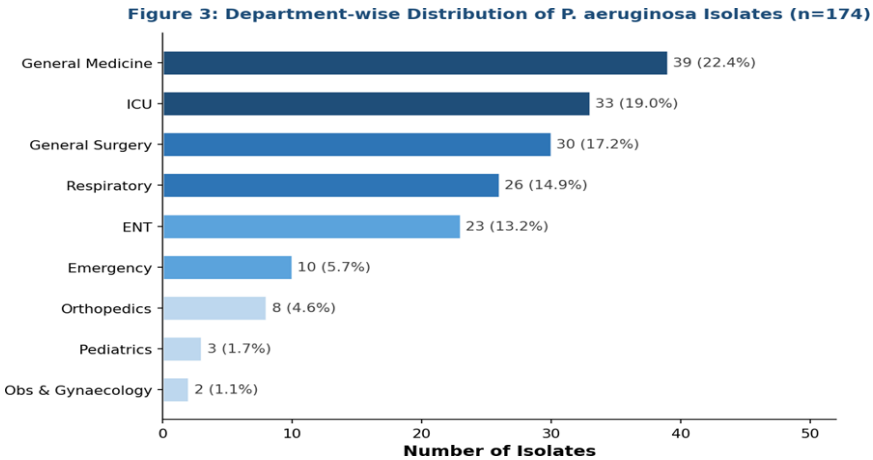

Figure 4: Department-wise Distribution of P. aeruginosa Clinical Isolates (n=174).

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3.4 Specimen-wise Distribution: Sputum specimens yielded the greatest number of P. aeruginosa isolates (73 isolates; 41.95%), consistent with the institution's substantial respiratory medicine and ICU caseload. Pus samples were the second most productive source (30; 17.24%), followed by ear swabs (27; 15.52%), urine (20; 11.49%), and BAL/ET secretions (13; 7.47%). Tracheal aspirates contributed 6 isolates (3.45%), and pleural fluid, blood, and tissue samples each contributed ≤2 isolates, collectively accounting for <2.9% of the total (Table 4 and Figure 4).

Table 4: Distribution of P. aeruginosa Isolates by Clinical Specimen Type (n=174)

Clinical Specimen	No. of Isolates	Percentage (%)
Sputum	73	41.95%
Pus	30	17.24%
Ear Swab	27	15.52%
Urine	20	11.49%
BAL / ET Secretions	13	7.47%
Tracheal Aspirate	6	3.45%
Pleural Fluid	2	1.15%
Blood	2	1.15%
Tissue	1	0.57%
Total	174	100.00%

Table 4: Specimen-wise distribution of 174 P. aeruginosa isolates. BAL=Bronchoalveolar Lavage; ET=Endotracheal.

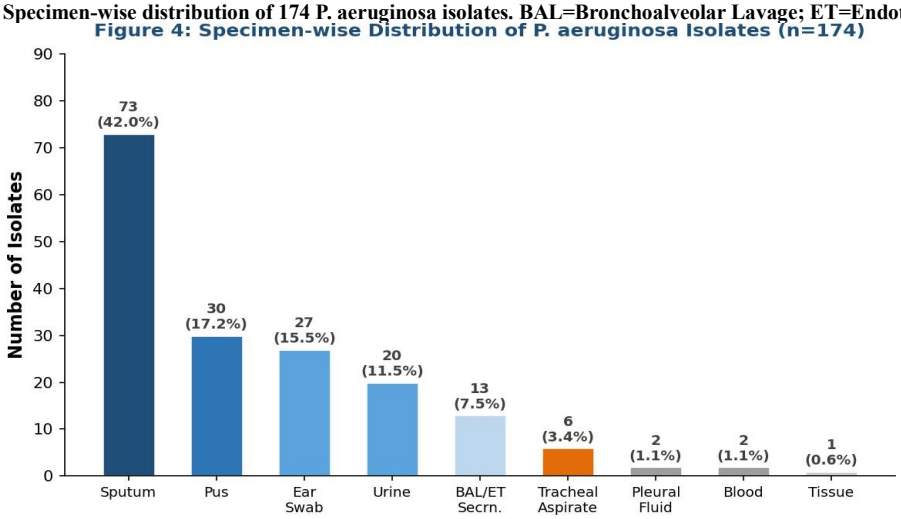

Figure 5: Specimen-wise Distribution of P. aeruginosa Isolates (n=174). Sputum was the dominant clinical source, accounting for 41.95% of total isolates.

3.5 Antimicrobial Susceptibility and Resistance Profile: Antibiogram analysis of all 174 P. aeruginosa isolates revealed a spectrum of resistance rates across the eleven anti-pseudomonal agents tested. The highest overall sensitivity was documented for piperacillin-tazobactam (153/174; 87.93%), followed by tobramycin and gentamicin (both 135/174; 77.59%), imipenem (133/174; 76.44%), cefepime (132/174; 75.86%), amikacin (131/174; 75.29%), meropenem (130/174; 74.71%), and ciprofloxacin and levofloxacin (both 126/174; 72.41%). The lowest sensitivity — and correspondingly highest resistance — was observed for ceftazidime and aztreonam (both 100/174 sensitive; 57.47% sensitivity; 42.53% resistance). Complete susceptibility data with statistical analysis are presented in Table 5 and Figure 5.

Table 5: Antibiogram Profile of 174 P. aeruginosa Isolates

Drug	Sensitive (n)	Resistant (n)	Sensitivity (%)	Resistance (%)	Z-Score	p-Value
Ceftazidime	100	74	57.47%	42.53%	-1.69	<0.05
Cefepime	132	42	75.86%	24.14%	-0.20	>0.05
Aztreonam	100	74	57.47%	42.53%	-1.69	<0.05
Pip-Tazobactam	153	21	87.93%	12.07%	1.14	<0.05
Amikacin	131	43	75.29%	24.71%	-0.20	>0.05
Gentamicin	135	39	77.59%	22.41%	0.16	>0.05
Tobramycin	135	39	77.59%	22.41%	0.16	>0.05
Ciprofloxacin	126	48	72.41%	27.59%	-0.40	>0.05
Levofloxacin	126	48	72.41%	27.59%	-0.40	>0.05
Imipenem	133	41	76.44%	23.56%	0.11	>0.05
Meropenem	130	44	74.71%	25.29%	-0.95	<0.05

Table 5: Complete antibiogram profile of 174 P. aeruginosa isolates. Z-scores and p-values reflect deviation from the mean sensitivity rate (76.51%). *p≤0.05 = statistically significant.

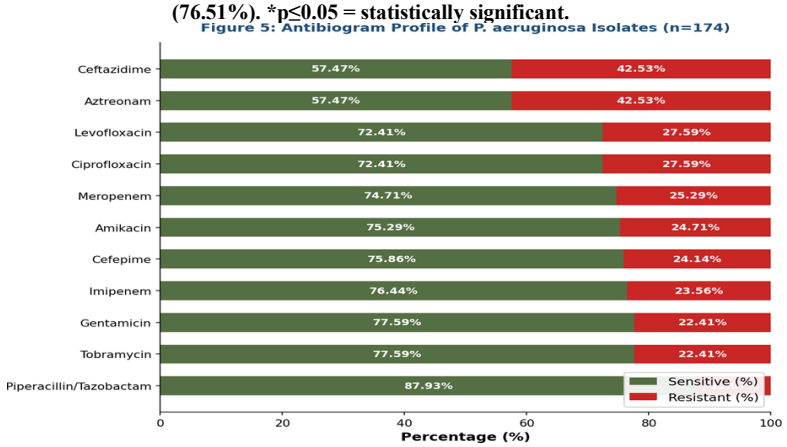

Figure 6: Antimicrobial Susceptibility and Resistance Profile of P. aeruginosa Isolates (n=174). Green = Sensitive; Red = Resistant.

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4. DISCUSSION

The present investigation, conducted over a six-month period, yielded 174 confirmed P. aeruginosa clinical isolates from a broad spectrum of specimen types, patient demographics, and clinical specialties at a northern Indian tertiary care facility. The findings present a clinically significant and nuanced picture of the local epidemiological and resistance landscape of this pathogen.

The pronounced male predominance observed in this cohort (71.84%) is consistently reported across Indian and international surveillance studies. Bindu and Saikumar (2022) [20] recorded 54.05% male prevalence in their southern Indian tertiary care series, while Tewari et al. (2020) [21] and Verma et al. (2021) [22] reported male predominance of 69.25% and 66.66%, respectively. The higher infection rates in males likely reflect greater occupational exposure to injury and wound contamination, higher rates of tobacco use predisposing to chronic lung disease and subsequent P. aeruginosa pulmonary colonisation, and potentially differential healthcare-seeking behaviour resulting in later, more advanced presentation. [23]

The age distribution in this study, with the peak burden in patients over 60 years (32.76%) and a secondary peak in the 46–60 year cohort (27.01%), is epidemiologically consistent with the well-established association between advancing age, immunosenescence, multiple comorbidities, and nosocomial P. aeruginosa infection. Salmani et al. (2015) [24] similarly identified patients above 60 years as the most frequently affected demographic. In the elderly, the convergence of declining mucosal defences, impaired cough reflexes, high rates of indwelling device utilisation, and longer hospital stays creates a particularly permissive environment for P. aeruginosa colonisation and invasive disease. [25]

The overwhelming in-patient dominance (83.33%) confirms the essentially nosocomial nature of P. aeruginosa infections at this institution, mirroring data from Tewari et al. (2020) [21] who reported 82% IPD origin, and Rahman et al. (2021) [26] who identified approximately 90% in-patient source. The ability of P. aeruginosa to persist in moist hospital environmental reservoirs — including sinks, ventilators, hydrotherapy equipment, and contaminated topical antiseptics — and to transmit via contaminated hands of healthcare workers sustains a continuous nosocomial risk cycle, particularly in ICUs and surgical wards. [27]

The department-wise distribution, with General Medicine (22.41%) and ICU (18.97%) as the two leading contributors, broadly aligns with previously published Indian data. Mullai et al. (2019) [28] identified surgical wards as the dominant source (60%), while Rahman et al. (2021) [26] documented surgical wards (19%), ICU (17%), and medical wards as leading contributors in their series. The prominence of the Respiratory ward (14.94%) and ENT department (13.22%) in the present study is noteworthy, reflecting the high burden of chronic respiratory disease and chronic suppurative otitis media (CSOM) in the community served by this institution, both of which are recognised clinical niches for P. aeruginosa colonisation and infection. [29]

The predominance of sputum as the leading specimen type (41.95%) in this study contrasts with many published series where pus and wound samples typically dominate. Senthamarai et al. (2014) [30] reported pus as the leading source (47.11%), and Kaur and Singh (2016) [31] identified pus as the primary source (36.4%). The sputum predominance in the present cohort is likely reflective of the institution's high respiratory medicine and ICU caseload, where ventilator-associated pneumonia and bronchiectasis represent the primary clinical contexts for P. aeruginosa respiratory infection. This epidemiological profile carries important therapeutic implications, as respiratory P. aeruginosa infections — particularly in ventilated patients — require systemic agents with adequate lung penetration rather than topical or oral formulations alone. [11,32]

The antibiogram findings reveal a nuanced resistance landscape across the eleven anti-pseudomonal agents evaluated. The lowest sensitivity rate, observed for ceftazidime and aztreonam (57.47% each, with a statistically significant negative deviation from the cohort mean, $p<0.05$), is particularly concerning from a clinical perspective, given that ceftazidime has historically been a cornerstone of empirical anti-pseudomonal therapy. The high resistance rate to ceftazidime is consistent with reports from Verma et al. (2021) [22] (63%), Tewari et al. (2020) [21] (70.19%), and Dash et al. (2014) [33] (77.7%). The parallel resistance to aztreonam suggests the production of broad-spectrum cephalosporinases or ESBLs in a substantial proportion of isolates, since both agents are primary targets of these enzymes. [7]

The relatively preserved sensitivity to carbapenems — imipenem (76.44%) and meropenem (74.71%) — in comparison to older anti-pseudomonal agents suggests that carbapenem resistance has not yet reached the alarming thresholds reported from some high-endemicity centres. Kaur and Singh (2016) [31] reported imipenem sensitivity of 82.2% and meropenem sensitivity of 66.9% in their series, while Ansari et al. (2015) [34] documented imipenem sensitivity of 88.99%. Although the carbapenem resistance rates in this study (23.56% and 25.29% for imipenem and meropenem, respectively) remain below levels defining pan-drug resistance, they represent a clinically significant elevation above the pre-2010 baseline and must be monitored longitudinally. [35]

Piperacillin-tazobactam emerged as the single agent with the highest sensitivity (87.93%) and a statistically significant positive deviation from the cohort mean (Z-score +1.14, $p<0.05$), suggesting its current utility as a reliable empirical option for P. aeruginosa infections at this institution. Yadav et al. (2016) [36] and Rahman et al. (2021) [26] similarly documented piperacillin-tazobactam sensitivity rates of 93.0% and 82.1%, respectively. The beta-lactamase inhibitor component (tazobactam) partially restores activity against ESBLproducing strains, though it does not reliably protect against MBL-producing or efflux pump-overexpressing isolates. The aminoglycosides — tobramycin and gentamicin (77.59% each) and amikacin (75.29%) — and fluoroquinolones — ciprofloxacin and levofloxacin (72.41% each) — occupy an intermediate tier, retaining meaningful but not optimal efficacy. [37]

5. CONCLUSION

This prospective observational study characterised the prevalence, demographic distribution, specimen-source epidemiology, and antimicrobial susceptibility profile of 174 P. aeruginosa clinical isolates recovered at a northern Indian tertiary care centre over a six-month period. Male sex (71.84%), elderly age (≥60 years; 32.76%), and in-patient status (83.33%) were identified as the dominant patient-level determinants of P. aeruginosa infection. Sputum represented the most productive specimen source, emphasising the central importance of respiratory tract infection in the clinical burden of P. aeruginosa at this institution.

The antibiogram data highlight piperacillin-tazobactam as the most reliably active anti-pseudomonal agent at this institution, while documenting concerning resistance levels against ceftazidime and aztreonam. The carbapenems retain intermediate efficacy, though the documented resistance rates underscore the imperative for antimicrobial stewardship to prevent further carbapenem resistance amplification. Immediate institutional priorities should include structured antibiotic policy reform, mandatory susceptibility-guided prescribing, comprehensive infection control bundles, and the integration of systematic P. aeruginosa resistance surveillance into the microbiology laboratory workflow to provide dynamic, real-time guidance for empirical therapy decisions.

Limitations

Several limitations inherent to the study design merit acknowledgement. The single-centre, six-month study window restricts temporal and geographic generalisability. Species-level biochemical identification was employed without molecular confirmation (e.g., 16S rRNA sequencing or MALDI-TOF MS), though the comprehensive biochemical panel used provides high diagnostic specificity for P. aeruginosa. MDR phenotype classification and genotypic characterisation of resistance mechanisms (ESBL, MBL, efflux pump overexpression) were not performed systematically, limiting mechanistic insight into resistance drivers. Clinical outcome data — including mortality, length of hospital stay, and treatment response — were not collected, precluding correlation of susceptibility profiles with patient prognosis. Future multi-centre longitudinal studies incorporating whole-genome sequencing and clinical outcomes are recommended to comprehensively address these limitations.

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