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ANTIMICROBIAL SUSCEPTIBILITY PATTERN OF PSEUDOMONAS AERUGINOSA ISOLATED FROM CLINICAL SPECIMENS: A RETROSPECTIVE OBSERVATIONAL STUDY AT TERTIARY CARE CENTER

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ABSTRACT **Background:** *Pseudomonas aeruginosa* is a leading opportunistic Gram-negative pathogen associated with healthcare-associated infections and is intrinsically resistant to several antimicrobial classes, with a well-documented capacity to acquire further resistance. Ongoing local surveillance of antimicrobial susceptibility is essential to guide empirical therapy and antimicrobial stewardship. **Methods:** We retrospectively analysed antimicrobial susceptibility data for 45 non-duplicate clinical isolates of P. aeruginosa recovered from patients attending our tertiary care healthcare center between January 2025 and July 2026. Isolates were identified and antimicrobial susceptibility testing was performed using the VITEK 2 automated system and Kirby-bauer disk diffusion method with results interpreted according to CLSI guidelines. Isolates were classified as susceptible (S), intermediate (I), or resistant (R) for each agent tested; multidrug resistance (MDR) was defined as non-susceptibility to at least one agent in three or more antimicrobial categories. **Results:** Isolates were most frequently recovered from pus (21, 46.7%), followed by urinary catheter (8, 17.8%) and urine (6, 13.3%). Susceptibility was highest to colistin (0% resistant; reported as intermediate under current breakpoints), amikacin (77.3% susceptible), and tobramycin (65.5% susceptible), and lowest to trimethoprim-sulfamethoxazole (95.5% resistant), tigecycline, nitrofurantoin, and nalidixic acid (all 100% resistant, though tested in few isolates). Among the carbapenems, resistance rates were 46.7% (meropenem), 48.9% (imipenem) and 53.5% (doripenem). Twenty-five of 45 isolates (55.6%) met criteria for multidrug resistance. Resistance to ceftazidime, meropenem, ciprofloxacin and piperacillin-tazobactam was numerically highest among urinary-catheter-associated isolates. **Conclusion:** Pseudomonas aeruginosa isolates in this cohort exhibited high rates of multidrug resistance and reduced susceptibility to carbapenems, antipseudomonal agents of last resort for severe infections. Amikacin and colistin retained the greatest in-vitro activity. These findings support continued local antimicrobial resistance surveillance and judicious carbapenem stewardship.

KEYWORDS : *Pseudomonas aeruginosa*; antimicrobial resistance; antibiogram; multidrug resistance; carbapenem resistance; clinical isolates

1.INTRODUCTION

Pseudomonas aeruginosa is a ubiquitous, non-fermenting Gram-negative bacillus and one of the most important opportunistic pathogens encountered in both community and healthcare settings. It is a major cause of healthcare associated infections including ventilator-associated pneumonia, catheter-associated urinary tract infection, surgical site and wound infections, bacteraemia, and infections in immunocompromised and critically ill patients. The organism was originally designated a critical-priority pathogen on the World Health Organization's 2017 priority pathogens list; in the WHO's 2024 update, carbapenem-resistant was reclassified to the high-priority tier, reflecting reported declines in global resistance, although it remains an important target for surveillance and antibiotic research and development given its intrinsic and acquired resistance to multiple antimicrobial classes and the limited pipeline of new agents active against it (WHO, 2024).

Pseudomonas aeruginosa possesses intrinsic resistance mechanisms including a restrictive outer membrane, constitutive efflux pump expression, and inducible AmpC β-lactamase, and it readily acquires additional resistance determinants such as extended-spectrum β-lactamases, carbapenemases, aminoglycoside-modifying enzymes, and mutations affecting porin expression and quinolone target enzymes. The consequence is frequent emergence of multidrug-resistant (MDR), extensively drug-resistant (XDR), and occasionally pandrug-resistant strains, which substantially narrow therapeutic options and are associated with increased morbidity, mortality, and healthcare costs.

Because resistance patterns vary considerably by geography, institution, specimen type, and over time, periodic local antibiogram analysis is essential to inform empirical antibiotic selection, antimicrobial stewardship programmes, and infection prevention & control policy. This study describes the antimicrobial susceptibility pattern of Pseudomonas aeruginosa isolated from clinical specimens, with the aim of characterising resistance to commonly used antipseudomonal agents and identifying isolates meeting criteria for multidrug resistance.

2. MATERIALS AND METHODS

2.1 Study design and setting

This was a retrospective study conducted in the Department of Microbiology at Bombay Hospital, Jaipur, between January 2025 and July 2026.

2.2 Sample collection and bacterial identification

Patients of all age groups attending Bombay Hospital, Jaipur, were included.

Blood agar, MacConkey agar, Mueller-Hinton agar, and HiChrome UTI culture media were used for isolation, and *Pseudomonas aeruginosa* was identified using the VITEK 2 automated system together with the oxidase test and pigment production on culture plates.

A total of 45 non-duplicate clinical isolates of *Pseudomonas aeruginosa* were included in the analysis. Specimen types comprised pus, urine, urinary catheter, blood, sputum, tracheal aspirate, bronchoalveolar lavage fluid, and pleural fluid (Table 1).

2.3 Antimicrobial susceptibility testing

Antimicrobial susceptibility testing (AST) was performed by automated broth microdilution using the VITEK 2 system, and results were interpreted according to the CLSI M100 breakpoint document, 35th edition (2025).

Isolates were tested against a panel of up to 27 antimicrobial agents spanning penicillins, β-lactam/β-lactamase-inhibitor combinations, cephalosporins, a monobactam, carbapenems, aminoglycosides, fluoroquinolones/quinolones, tetracyclines/glycylcyclines, a phenicol, a folate pathway inhibitor, a nitrofuran, and a polymyxin, according to laboratory testing protocols; not every isolate was tested against every agent. Results were recorded as susceptible (S), intermediate (I), or resistant ®.

2.4 Definitions

Multidrug resistance (MDR) was defined as non-susceptibility to at least one agent in three or more of the following antimicrobial categories, adapted from the standardised international definitions of Magiorakos et al. (2012) for P. aeruginosa: aminoglycosides, carbapenems, antipseudomonal cephalosporins, fluoroquinolones, antipseudomonal penicillins + β-lactamase inhibitor, monobactams, and polymyxins. Category-level classification was only assigned where at least one agent from that category had been tested.

2.5 Statistical analysis

Data were tabulated and analysed using descriptive statistics. For each antimicrobial agent, the percentage susceptible, intermediate, and resistant was calculated among isolates actually tested against that agent (denominators therefore vary by agent, as summarised in Table 2). Resistance rates for selected agents were stratified by specimen type for specimen categories with at least three isolates.

3. RESULTS

A total of 45 Pseudomonas aeruginosa isolates were analysed. The distribution by specimen type is shown in Table 1. Pus was the most common source (21 isolates, 46.7%), followed by sample collected from urinary catheter (8, 17.8%) and urine (6, 13.3%).

Table 1. Distribution of *Pseudomonas aeruginosa* isolates by specimen type (N = 45)

Specimen type	n	%
Pus	21	46.7
Urinary catheter	8	17.8
Urine	6	13.3
Sputum	3	6.7
Tracheal aspirate	3	6.7
Blood	2	4.4
Pleural fluid	1	2.2
Bronchoalveolar lavage	1	2.2

The overall antimicrobial susceptibility profile is summarised in Table 2. Susceptibility was best preserved to amikacin (77.3% susceptible) and tobramycin (65.5% susceptible) among the aminoglycosides. Carbapenem resistance was substantial: meropenem 46.7%, imipenem 48.9%, and doripenem 53.5%. Resistance to fluoroquinolones was high across all agents tested (ciprofloxacin 65.9%, ofloxacin 68.8%, levofloxacin 71.4%, norfloxacin 90.9%). Resistance to the antipseudomonal penicillin/β-lactamase-inhibitor combinations was 51.3% (piperacillin-tazobactam) and 59.0% (ticarcillin-clavulanate). No isolate was categorically resistant to colistin, although all 31 isolates tested returned an intermediate result rather than susceptible, which may reflect the revised (non-susceptible/susceptible-only) colistin breakpoint framework and warrants confirmation of the testing method used (see Limitations). Agents such as ampicillin, cefotaxime, ceftriaxone, doxycycline, tetracycline, nalidixic acid, nitrofurantoin, and tigecycline were tested in very few isolates (n = 1–20), limiting the precision of resistance estimates for these agents.

Table 2. Antimicrobial susceptibility of Pseudomonas aeruginosa isolates by agent (denominators vary by agent tested)

Antimicrobial agent	Class	n tested	S (%)	I (%)	R (%)	% R
Ceftazidime	3rd-gen (antipseudomonal) cephalosporin	45	19 (42.2)	1 (2.2)	25 (55.6)	55.6
Cefotaxime	3rd-gen cephalosporin	2	0 (0.0)	0 (0.0)	2 (100.0)	100.0
Ceftriaxone	3rd-gen cephalosporin	1	0 (0.0)	0 (0.0)	1 (100.0)	100.0
Cefepime	4th-gen cephalosporin	40	20 (50.0)	3 (7.5)	17 (42.5)	42.5
Amikacin	Aminoglycoside	22	17 (77.3)	0 (0.0)	5 (22.7)	22.7
Gentamicin	Aminoglycoside	21	12 (57.1)	1 (4.8)	8 (38.1)	38.1
Tobramycin	Aminoglycoside	29	19 (65.5)	0 (0.0)	10 (34.5)	34.5
Doripenem	Carbapenem	43	19 (44.2)	1 (2.3)	23 (53.5)	53.5
Imipenem	Carbapenem	45	20 (44.4)	3 (6.7)	22 (48.9)	48.9
Meropenem	Carbapenem	45	21 (46.7)	3 (6.7)	21 (46.7)	46.7
Cefoperazone-sulbactam	Cephalosporin + BLI	33	17 (51.5)	1 (3.0)	15 (45.5)	45.5
Ciprofloxacin	Fluoroquinolone	44	15 (34.1)	0 (0.0)	29 (65.9)	65.9
Levofloxacin	Fluoroquinolone	35	9 (25.7)	1 (2.9)	25 (71.4)	71.4
Norfloxacin	Fluoroquinolone	11	1 (9.1)	0 (0.0)	10 (90.9)	90.9
Ofloxacin	Fluoroquinolone	32	10 (31.2)	0 (0.0)	22 (68.8)	68.8
Trimethoprim-sulfamethoxazole	Folate pathway inhibitor	22	1 (4.5)	0 (0.0)	21 (95.5)	95.5
Tigecycline	Glycylcycline	20	0 (0.0)	0 (0.0)	20 (100.0)	100.0
Aztreonam	Monobactam	18	9 (50.0)	2 (11.1)	7 (38.9)	38.9
Nitrofurantoin	Nitrofuran	13	0 (0.0)	0 (0.0)	13 (100.0)	100.0
Ampicillin	Penicillin	2	0 (0.0)	0 (0.0)	2 (100.0)	100.0
Piperacillin-tazobactam	Penicillin + BLI	39	18 (46.2)	1 (2.6)	20 (51.3)	51.3
Ticarcillin-clavulanate	Penicillin + BLI	39	13 (33.3)	3 (7.7)	23 (59.0)	59.0
Chloramphenicol	Phenicol	8	1 (12.5)	1 (12.5)	6 (75.0)	75.0
Colistin	Polymyxin	31	0 (0.0)	31 (100.0)	0 (0.0)	0.0
Nalidixic acid	Quinolone	10	0 (0.0)	0 (0.0)	10 (100.0)	100.0
Doxycycline	Tetracycline	1	0 (0.0)	0 (0.0)	1 (100.0)	100.0
Tetracycline	Tetracycline	1	0 (0.0)	0 (0.0)	1 (100.0)	100.0

Twenty-five of the 45 isolates (55.6%) met the criteria for multidrug resistance (non-susceptibility to ≥1 agent in ≥3 antimicrobial categories). All 45 isolates had sufficient panel coverage (≥5 of 7 core categories tested) to permit this classification with reasonable confidence.

When resistance to selected key agents was stratified by specimen type (Table 3), isolates from urinary catheters showed the highest resistance across ceftazidime (100%), ciprofloxacin (100%), piperacillin-tazobactam (100%), and meropenem (75%), while isolates from pus specimens showed comparatively lower resistance rates (ceftazidime 28.6%, ciprofloxacin 52.4%, meropenem 23.8%). Urine isolates showed intermediate-to-high resistance across these agents.

Table 3. Resistance (%R) to selected antipseudomonal agents, by specimen type (n ≥ 3 isolates only)

Agent	Pus (n=21)	Urinary catheter (n=8)	Urine (n=6)	Sputum (n=3)	Tracheal aspirate (n=3)
Ceftazidime	6/21 (28.6%)	8/8 (100.0%)	4/6 (66.7%)	1/3 (33.3%)	2/3 (66.7%)
Meropenem	5/21 (23.8%)	6/8 (75.0%)	4/6 (66.7%)	1/3 (33.3%)	2/3 (66.7%)
Ciprofloxacin	11/21 (52.4%)	8/8 (100.0%)	5/6 (83.3%)	1/3 (33.3%)	1/2 (50.0%)
Piperacillin-tazobactam	3/16 (18.8%)	7/7 (100.0%)	3/6 (50.0%)	1/3 (33.3%)	2/3 (66.7%)

4. DISCUSSION

This analysis of 45 clinical Pseudomonas aeruginosa isolates demonstrates a substantial burden of resistance to multiple antipseudomonal agent classes, with just over half of isolates (55.6%) classified as multidrug-resistant. This is broadly consistent with global concern regarding Pseudomonas aeruginosa as a WHO high-priority pathogen and with reports from many settings describing MDR prevalence in the range of approximately one-third to two-thirds of clinical isolates, though rates vary considerably by country, patient population, and specimen source.

Carbapenem resistance (46.7–53.5% across meropenem, imipenem, and doripenem) is of particular clinical concern, as carbapenems are frequently reserved as agents of choice for serious Pseudomonas aeruginosa infections, including bacteraemia and ventilator-associated pneumonia. Mechanisms underlying carbapenem resistance in Pseudomonas aeruginosa include loss or downregulation of the OprD porin, upregulation of efflux pumps, AmpC overexpression, and acquisition of carbapenemases (e.g., VIM, IMP, NDM, or KPC types) (Del Barrio-Tofiño et al., 2024; Tsilipounidaki et al., 2024); this dataset did not include molecular characterisation, so the specific mechanism(s) driving resistance in these isolates cannot be determined and represents an important direction for further work. The high carbapenem and fluoroquinolone resistance rates observed here are consistent with recent Indian surveillance data, which similarly report escalating multidrug and extensive drug resistance among clinical P. aeruginosa isolates.

Fluoroquinolone resistance was high across all agents assessed, which may reflect widespread empirical or over-the-counter fluoroquinolone use in the study setting and is consistent with the well-documented propensity of Pseudomonas aeruginosa to acquire quinolone resistance through mutations in gyrA/parC and efflux upregulation.

This finding argues against empirical fluoroquinolone monotherapy for suspected *Pseudomonas aeruginosa* infection in this population pending susceptibility results.

Amikacin and tobramycin retained relatively better in-vitro activity than gentamicin, consistent with amikacin's reduced susceptibility to modification by many common aminoglycoside-modifying enzymes, and both may be reasonable options within combination regimens for susceptible isolates, particularly in critically-ill patients where source control and combination therapy are being considered. Colistin results warrant cautious interpretation: no isolate was reported as resistant, but all tested isolates fell into the intermediate category. Since 2020, colistin breakpoints for Enterobacterales and *Pseudomonas spp*. under EUCAST no longer include a susceptible category by disc diffusion, and reliable colistin susceptibility testing requires broth microdilution; the uniform 'intermediate' result across this dataset should prompt verification of the testing method used and, if disc diffusion was used, retesting by broth microdilution before this result informs clinical decision-making.

Resistance to ceftazidime, ciprofloxacin, piperacillin-tazobactam, and meropenem was numerically higher among urinary-catheter-associated isolates than among pus isolates in this dataset. This pattern is plausible given that catheter-associated isolates often arise in the context of prior antibiotic exposure, biofilm formation, and healthcare contact, all of which are recognised drivers of resistance selection (Taylor et al., 2014), but the small numbers in some specimen categories (particularly blood, pleural fluid, and bronchoalveolar lavage, each with 1–2 isolates) limit the statistical robustness of these comparisons and they should be regarded as descriptive/hypothesis-generating rather than confirmatory.

5. LIMITATIONS

This study has several limitations. First, the sample size (45 isolates) is modest, and several specimen categories contained very few isolates (n = 1–3), limiting the precision and generalisability of specimen-stratified comparisons. Second, not all isolates were tested against the full antimicrobial panel; and resistance percentages for sparsely tested agents should be interpreted with caution. Third, the dataset as provided did not include patient demographic or clinical information (age, sex, ward/unit, underlying diagnosis, prior antibiotic exposure, or outcome), precluding adjustment for potential confounders or clinical correlation. Fourth, the specific AST methodology and breakpoint standard require confirmation (see Methods), particularly for colistin. Fifth, this appears to be a single-setting, retrospective analysis without molecular characterisation of resistance mechanisms, so mechanistic conclusions cannot be drawn.

6. CONCLUSION

Pseudomonas aeruginosa isolates in this cohort showed high rates of resistance to fluoroquinolones, antipseudomonal penicillin/β-lactamase-inhibitor combinations, and — notably — carbapenems, with over half of isolates meeting criteria for multi-drug resistance. Amikacin, tobramycin, and colistin (pending confirmation of testing method) retained the greatest in-vitro activity. These findings underscore the need for carbapenem-sparing empirical strategies where locally appropriate, confirmatory susceptibility testing before finalising treatment, and continued antibiogram surveillance to guide institutional antimicrobial stewardship and infection prevention & control policy.

Author contributions

The author was involved in the conceptualization, study design, methodology, data curation, analysis and interpretation of data, and preparation, writing, reviewing of the article.

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