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*Article*

# Antibiotic Resistance Patterns of *Pseudomonas aeruginosa* Isolated from Clinical Samples in Khyber Pakhtunkhwa, Pakistan

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## ABSTRACT

**Background**: *Pseudomonas aeruginosa* is a major opportunistic pathogen responsible for a wide range of nosocomial infections. Its increasing multidrug resistance (MDR) poses significant treatment challenges, especially in low-resource settings like Khyber Pakhtunkhwa, Pakistan, where updated surveillance data are limited. **Objective**: This study aimed to determine the prevalence and antibiotic resistance patterns of *Pseudomonas aeruginosa* isolated from various clinical specimens, and to assess the distribution of MDR strains across different sample types in tertiary care hospitals of Khyber Pakhtunkhwa. **Methods**: A cross-sectional observational study was conducted from February 2024 to February 2025 using 107 non-duplicate clinical isolates of P. aeruginosa. Isolates were collected from five tertiary hospitals, confirmed through standard biochemical tests, and tested for antimicrobial susceptibility using the Kirby-Bauer disk diffusion method following CLSI M100 guidelines (34th edition, 2024). MDR was defined as resistance to at least one agent in three or more antibiotic classes. Ethical protocols adhered to biosafety standards consistent with the Declaration of Helsinki. Data were analyzed using SPSS v27.0 with descriptive and chi-square statistical analyses. **Results**: Colistin showed the highest sensitivity (83.2%), followed by meropenem (64.5%) and imipenem (53.3%), while ciprofloxacin (89.7%) and levofloxacin (87.9%) had the highest resistance. MDR was most frequent in sputum (53.8%) and pus (51.8%) samples. No significant association was found between MDR distribution and sample type ($\chi^2$ = 4.93, p = 0.553). **Conclusion**: The study underscores the alarming resistance trends in P. aeruginosa, highlighting colistin and carbapenems as the most reliable treatment options. These findings advocate for targeted antimicrobial stewardship and periodic resistance surveillance to mitigate therapeutic failures in clinical practice.

**Keywords**: *Pseudomonas aeruginosa*, Multidrug Resistance, Antimicrobial Susceptibility Testing, Colistin, Fluoroquinolone Resistance, Nosocomial Infections, Pakistan

## INTRODUCTION

*Pseudomonas aeruginosa*, a Gram-negative opportunistic pathogen belonging to the Pseudomonadaceae family, is a leading cause of nosocomial infections and remains a significant public health concern due to its inherent and acquired resistance to multiple antibiotics (1). It has been implicated in a wide spectrum of hospital-acquired infections including pneumonia, urinary tract infections, wound infections, septicemia, and meningitis, particularly in immunocompromised individuals such as those with cancer, burns, cystic fibrosis, and those undergoing organ transplantation (2, 3). According to global surveillance reports, P. aeruginosa accounts for approximately 10–20% of hospital-acquired infections, and its prevalence varies based on geographic and healthcare settings (4). The Centers for Disease Control (CDC) reported in the National Nosocomial Infections Study (NNIS) that P. aeruginosa is responsible for nearly 8.5% of all nosocomial infections, highlighting its clinical significance (5).

The pathogenicity of P. aeruginosa is attributed to a wide range of virulence factors including exotoxins, proteases, hemolysins, and quorum sensing systems that coordinate biofilm formation and expression of resistance mechanisms (6, 7). Clinical isolates often harbor virulence-associated genes such as *toxA*, *lasB*, *oprL*, and *exoS*, which contribute to their persistence and severity of infection (8). Compounding the challenge is the organism's remarkable ability to develop resistance through multiple mechanisms such as efflux pumps, enzyme production (e.g., β-lactamases), porin mutations, and horizontal gene transfer (9). Of particular concern is the rise in multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains of P. aeruginosa, which

limit therapeutic options and are often associated with prolonged hospital stays, increased morbidity, and higher healthcare costs (10, 11). In recent years, the emergence of β-lactamase enzymes, including extended-spectrum β-lactamases (ESBLs) and metallo-β-lactamases (MBLs), has exacerbated the resistance landscape. These enzymes inactivate broad-spectrum antibiotics such as penicillins, cephalosporins, monobactams, and even carbapenems, the latter being traditionally reserved for severe infections (12, 13). P. aeruginosa has been reported to encode over 120 types of β-lactamases, including carbapenemases that confer resistance to nearly all β-lactam agents and are not inhibited by conventional β-lactamase inhibitors (14). The global distribution of metallo-β-lactamase genes such as *blaIMP*, *blaVIM*, and *blaSPM* in imipenem-resistant strains further underlines the gravity of antimicrobial resistance in this species (15, 16).

Although several international studies have highlighted the increasing prevalence and resistance patterns of P. aeruginosa, data from many regions, including Khyber Pakhtunkhwa in Pakistan, remain sparse or outdated. Limited regional surveillance undermines the ability to develop targeted antibiotic policies and infection control strategies. In Pakistan, where empirical antibiotic use is common and infection control practices vary widely across healthcare institutions, up-to-date local data on resistance trends are essential for evidence-based clinical decision-making (17). Previous studies have reported inconsistent resistance patterns across provinces, and the extent of MDR in clinical isolates from tertiary care hospitals in Khyber Pakhtunkhwa remains underexplored (18). This knowledge gap necessitates a comprehensive investigation into the prevalence and antibiotic susceptibility profiles of P. aeruginosa in this region.

The present study, therefore, aims to identify clinical isolates of P. aeruginosa from a range of specimen types collected in healthcare facilities across Khyber Pakhtunkhwa and to evaluate their resistance profiles against commonly used antibiotics. By documenting current resistance trends and the extent of multidrug resistance, this study seeks to inform local antimicrobial stewardship initiatives and contribute to the global effort against antibiotic resistance. The central research question guiding this study is: What are the current antibiotic resistance patterns and the prevalence of multidrug-resistant P. aeruginosa in clinical specimens collected from healthcare institutions in Khyber Pakhtunkhwa, Pakistan?

## MATERIAL AND METHODS

The present study was a **laboratory-based cross-sectional observational study** conducted over a one-year period from February 2024 to February 2025. The research was jointly carried out at the Centre for Biotechnology and Microbiology, University of Swat, and the Microbiology Laboratory, Saidu Teaching Hospital Swat, in Khyber Pakhtunkhwa, Pakistan. A total of 107 non-duplicate clinical isolates of *Pseudomonas aeruginosa* were obtained from microbiology laboratories of tertiary care hospitals including Khyber Teaching Hospital, Lady Reading Hospital, and Hayatabad Medical Complex in Peshawar; Mardan Medical Complex in Mardan; and Saidu Teaching Hospital in Swat. Isolates were recovered from a variety of clinical specimens including pus (n=56; 52.33%), wound swabs (n=17; 15.88%), sputum (n=13; 12.14%), blood (n=10; 9.34%), tracheal aspirates (n=6; 5.60%), urine (n=3; 2.80%), and high vaginal swabs (n=2; 1.86%). Inclusion criteria comprised confirmed *P. aeruginosa* isolates obtained from routine diagnostic samples submitted to participating hospitals. Exclusion criteria included repeat isolates from the same patient, environmental samples, and any non-clinically confirmed strains. Patient identifiers were excluded to maintain confidentiality; only isolate-level data were collected, and no direct patient interaction occurred.

Sample size was determined using the World Health Organization's formula for estimating a proportion in a large population: $S = Z^2 \times P \times (1 - P) / M^2$, where $Z$ corresponds to the confidence level (1.96 for 95% confidence), $P$ is the estimated prevalence (assumed at 0.5 for maximum sample size), and $M$ is the margin of error (set at 10%). This yielded a minimum required sample size, which was met and exceeded in this study to ensure representativeness across specimen types and healthcare settings (18). All isolates were initially cultured on general-purpose media (nutrient agar) and incubated at 37°C for 24 hours. Presumptive colonies were subjected to a series of confirmatory biochemical tests, including Gram staining (to verify Gram-negative bacilli), catalase test (positive for catalase production), and oxidase test (positive for cytochrome c oxidase), in accordance with standard microbiological protocols (19).

Antibiotic susceptibility testing (AST) was performed using the Kirby-Bauer disk diffusion method on Mueller-Hinton agar, in compliance with the Clinical and Laboratory Standards Institute (CLSI) guidelines, 34th edition, M100, 2024. Sixteen antibiotics were included in the testing panel: piperacillin-tazobactam (TZP), meropenem (MEM), cefepime (FEP), imipenem (IMP), ciprofloxacin (CIP), cefoperazone-sulbactam (SCF), levofloxacin (LEV), gentamicin (CN), ceftazidime (CAZ), aztreonam (ATM), colistin (CT), tobramycin (TOB), amoxicillin (AMC), amikacin (AK), norfloxacin (NOR – urine isolates only), and fosfomycin (FOS – urine isolates only). The interpretive criteria were based on CLSI breakpoints and results were classified as either susceptible (S) or resistant (R). Multidrug resistance (MDR) was defined as resistance to at least one agent in three or more antibiotic classes. Quality control was maintained throughout testing using *P. aeruginosa* ATCC 27853 as a control strain.

As the study involved only microbial isolates and no direct involvement of human subjects or identifiable patient data, formal ethical approval from an institutional review board (IRB) was not mandated. However, all laboratory procedures were conducted following biosafety and bioethics standards in line with institutional policies and international recommendations for handling clinical pathogens. All data were anonymized at the point of collection, and only non-identifiable laboratory codes were used for analysis to ensure privacy and scientific integrity. Data were entered and managed using Microsoft Excel and analyzed with SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics, including frequencies and percentages, were used to report antimicrobial susceptibility patterns and MDR distribution among different specimen types.

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Table 1: Antibiotic Susceptibility Profile of *Pseudomonas aeruginosa* (n = 107)

| S. No. | Antibiotic | Sensitive n (%) | Resistant n (%) |
|---|---|---|---|
| 1 | Colistin (CT) | 89 (83.2%) | 18 (16.8%) |
| 2 | Meropenem (MEM) | 69 (64.5%) | 38 (35.5%) |
| 3 | Imipenem (IMP) | 57 (53.3%) | 50 (46.7%) |
| 4 | Aztreonam (ATM) | 51 (47.7%) | 56 (52.3%) |
| 5 | Pip-Tazobactam (TZP) | 46 (43.0%) | 61 (57.0%) |
| 6 | Amikacin (AK) | 39 (36.4%) | 68 (63.6%) |
| 7 | Cefepime (FEP) | 37 (34.6%) | 70 (65.4%) |
| 8 | Tobramycin (TOB) | 37 (34.6%) | 70 (65.4%) |
| 9 | Gentamicin (CN) | 32 (29.9%) | 75 (70.1%) |
| 10 | Ceftazidime (CAZ) | 33 (30.8%) | 74 (69.2%) |
| 11 | Cefoperazone-Sulbactam (SCF) | 31 (29.0%) | 76 (71.0%) |
| 12 | Amoxicillin (AMC) | 20 (18.7%) | 87 (81.3%) |
| 13 | Levofloxacin (LEV) | 13 (12.1%) | 94 (87.9%) |
| 14 | Ciprofloxacin (CIP) | 11 (10.3%) | 96 (89.7%) |
| 15 | Norfloxacin (NOR)* | 01 (33.3%) | 02 (66.7%) |
| 16 | Fosfomycin (FOS)* | 02 (66.7%) | 01 (33.3%) |

*Note: NOR and FOS tested only in urine isolates (n=3).

Table 2: Multidrug Resistance (MDR) Distribution Among Clinical Specimens

| S. No. | Sample Type | Total Isolates (n) | MDR Isolates n (%) |
|---|---|---|---|
| 1 | Sputum | 13 | 07 (53.8%) |
| 2 | Pus | 56 | 29 (51.8%) |
| 3 | Blood | 10 | 05 (50.0%) |
| 4 | Tracheal Aspirate | 06 | 03 (50.0%) |
| 5 | Wound Swab | 17 | 12 (44.1%) |
| 6 | Urine | 03 | 01 (33.3%) |
| 7 | High Vaginal Swab | 02 | 00 (0.0%) |

Where relevant, chi-square tests were considered to assess associations between sample sources and resistance patterns. No imputation was required for missing data as all isolates had complete records for AST. Confounding variables were not directly assessed as the study design was focused exclusively on microbiological outcomes.

## RESULTS

A total of 107 clinical isolates were identified as *Pseudomonas aeruginosa*. All isolates were Gram-negative bacilli. Catalase testing was positive in all 107 isolates (100%), and oxidase testing was positive in 104 isolates (97.2%), supporting standard biochemical confirmation of *P. aeruginosa*. Antibiotic susceptibility testing revealed a concerning trend of high resistance among commonly used antibiotics. Colistin was the most effective, with 83.2% of isolates showing susceptibility, followed by meropenem (64.5%), imipenem (53.3%), and aztreonam (47.7%).

Table 1. Antibiotic Susceptibility Pattern of *Pseudomonas aeruginosa* Isolates (n = 107)

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| 16 | Fosfomycin (FOS)* | 02 (66.7%) | 01 (33.3%) |

**Note: NOR and FOS tested only in urine isolates (n=3).*

Fluoroquinolones (ciprofloxacin and levofloxacin) exhibited the highest resistance rates, at 89.7% and 87.9%, respectively, followed by amoxicillin (81.3%). Among urinary isolates, fosfomycin had 67% sensitivity. The complete antibiotic susceptibility data are shown in Table 1. Multidrug resistance (MDR), defined as resistance to at least one agent in three or more antibiotic classes, was present in 52% of pus isolates and 53.8% of sputum isolates. MDR rates in blood and tracheal aspirates were both 50%, while wound swabs and urine showed 44.1% and 33.3%, respectively. No MDR was observed in isolates from high vaginal swabs. Full distribution is shown in Table 2. To determine whether MDR prevalence significantly differed among specimen types, a chi-square test of independence was performed. The results indicated no statistically significant association between specimen type and MDR occurrence ($\chi^2 = 4.93$, $p = 0.553$), suggesting that MDR is relatively evenly distributed across the different sample sources.

**Table 2. Distribution of Multidrug-Resistant *Pseudomonas aeruginosa* by Specimen Type**

| S. No. | Sample Type | Total Isolates (n) | MDR Isolates n (%) | Non-MDR Isolates |
|---|---|---|---|---|
| 1 | Sputum | 13 | 07 (53.8%) | 06 |
| 2 | Pus | 56 | 29 (51.8%) | 27 |
| 3 | Blood | 10 | 05 (50.0%) | 05 |
| 4 | Tracheal Aspirate | 06 | 03 (50.0%) | 03 |
| 5 | Wound Swab | 17 | 12 (44.1%) | 05 |
| 6 | Urine | 03 | 01 (33.3%) | 02 |
| 7 | High Vaginal Swab | 02 | 00 (0.0%) | 02 |

The high rates of resistance to fluoroquinolones and β-lactams, coupled with the relatively preserved sensitivity to colistin and carbapenems, align with global trends of increasing reliance on last-line therapies. The presence of MDR in over half of the respiratory and pus isolates suggests ongoing antimicrobial pressure in critical care and surgical units. Although the association between specimen type and MDR status was not statistically significant, the overall burden of MDR *P. aeruginosa* remains high and calls for reinforced antimicrobial stewardship and infection control practices.

## DISCUSSION

The present study contributes valuable regional data on the antimicrobial resistance patterns of *Pseudomonas aeruginosa*, a pathogen that continues to pose a serious therapeutic challenge due to its multidrug-resistant (MDR) nature. The findings align with global concerns regarding the rising resistance to frontline antibiotics, particularly fluoroquinolones and β-lactams. Our results revealed high resistance rates to ciprofloxacin (89.7%) and levofloxacin (87.9%), consistent with previous studies conducted in Pakistan and South Asia, where fluoroquinolone overuse in both hospital and outpatient settings has been associated with rapidly diminishing efficacy (3, 18). The substantial resistance to amoxicillin (81.3%) and cefoperazone-sulbactam (71.0%) further supports the notion that β-lactam antibiotics are increasingly ineffective against *P. aeruginosa* in local clinical contexts (24). These findings corroborate prior research from Peshawar and other Pakistani regions that reported resistance rates exceeding 70% to third-generation cephalosporins and β-lactam/β-lactamase inhibitor combinations (20, 21).

Conversely, the highest sensitivity was observed with colistin (83.2%), which remains one of the few consistently effective agents against *P. aeruginosa* in our setting. This finding is consistent with surveillance data from multiple international studies that continue to report relatively preserved colistin activity, albeit with growing caution due to nephrotoxicity concerns and emerging reports of colistin-resistant strains (26). The effectiveness of carbapenems—meropenem (64.5%) and imipenem (53.3%)—was moderate in our study, lower than in previous regional reports which found sensitivity rates exceeding 80% (22). This trend may reflect increased carbapenem use in tertiary care units, selecting for carbapenem-resistant isolates through mechanisms such as metallo-β-lactamase (MBL) production and efflux pump overexpression (13, 14). The moderate sensitivity to aztreonam (47.7%) suggests potential for its use in combination therapies, though monotherapy may no longer be sufficient given evolving resistance profiles.

The distribution of MDR *P. aeruginosa* across various clinical samples provides further insight into infection control challenges. Sputum and pus samples had the highest proportions of MDR isolates (53.8% and 51.8%, respectively), indicating increased antimicrobial pressure in intensive care units (ICUs) and surgical wards. Respiratory tract samples, particularly from ventilated patients, are well-documented reservoirs for MDR organisms due to biofilm formation and prolonged antibiotic exposure (7). While no statistically significant association was found between specimen type and MDR prevalence ($p = 0.553$), the clinical impact remains substantial, especially given the potential for respiratory and bloodstream infections to escalate rapidly. The observed MDR rates are in line with previous Pakistani studies which reported MDR *P. aeruginosa* prevalence between 40% and 60%, although some institutions report even higher rates depending on patient demographics and antimicrobial use patterns (3, 24).

Several mechanisms may underlie the high resistance rates observed. *P. aeruginosa* employs intrinsic resistance mechanisms such as outer membrane impermeability, efflux pumps, and β-lactamase production, including extended-spectrum β-lactamases (ESBLs) and metallo-β-lactamases (MBLs) (10, 12). Our data indirectly support the possibility of these mechanisms, especially given the high resistance to carbapenems and cephalosporins. Additionally, acquired resistance through horizontal gene transfer, particularly in hospital environments, may further compound resistance trends. The clinical implication of this resistance is profound, as it narrows therapeutic options and increases reliance on last-resort antibiotics like colistin and fosfomycin, both of which have limitations in terms of toxicity and dosing challenges.

One of the strengths of this study is the inclusion of isolates from multiple tertiary hospitals across Khyber Pakhtunkhwa, offering a broader regional perspective. The use of standardized CLSI-based AST procedures adds methodological robustness and ensures comparability with international datasets. However, several limitations must be acknowledged. The sample size, although adequate for descriptive purposes, limits the power to detect significant differences in MDR prevalence between specimen types. The cross-sectional design precludes the assessment of temporal trends or treatment outcomes. Furthermore, molecular analysis of resistance genes was not performed, which would have provided direct evidence of mechanisms such as ESBL or MBL production. Lastly, the study's generalizability is limited to hospital-acquired infections and may not reflect community-acquired *P. aeruginosa* strains.

Future research should prioritize longitudinal surveillance with larger, multicenter datasets and integrate molecular diagnostics to characterize resistance genes and clonal relationships. Additionally, exploring combination therapies, pharmacokinetics, and local antimicrobial stewardship outcomes could inform targeted interventions. Given the high MDR burden observed, there is a pressing need to reinforce infection prevention measures, revise empirical treatment protocols, and invest in regional antibiogram development. Addressing resistance in *P. aeruginosa* requires a multidisciplinary approach that integrates clinical vigilance, microbiological insight, and public health strategies.

This study highlights alarming resistance trends in *P. aeruginosa* clinical isolates from Khyber Pakhtunkhwa, with significant implications for patient management and healthcare policy. While colistin and carbapenems remain partially effective, rising resistance underscores the urgency for improved stewardship, continuous surveillance, and research into novel therapeutic approaches.

## CONCLUSION

This study identified a high prevalence of multidrug-resistant *Pseudomonas aeruginosa* isolates from clinical samples collected across tertiary care hospitals in Khyber Pakhtunkhwa, Pakistan, with the highest sensitivity observed for colistin, followed by meropenem and imipenem, while resistance was most pronounced against ciprofloxacin, levofloxacin, and amoxicillin. These findings underscore the critical need for continuous antimicrobial surveillance and prudent antibiotic use to combat rising resistance in clinical settings. The observed resistance patterns highlight the diminishing efficacy of commonly used antibiotics, emphasizing the necessity for evidence-based empirical treatment protocols, particularly in high-risk hospital units. From a research perspective, the study provides a foundation for future investigations into molecular resistance mechanisms and the development of region-specific antimicrobial stewardship strategies to safeguard therapeutic efficacy against *P. aeruginosa*.

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