

OCCURRENCE AND ANTIBIOGRAM STUDIES OF PSEUDOMONAS AERUGINOSA FROM URINARY TRACT INFECTION

Original Research (ID: 1711)

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ABSTRACT

Background: *Pseudomonas aeruginosa* is an important opportunistic pathogen associated with complicated and healthcare-associated urinary tract infections. Its intrinsic resistance, capacity to acquire additional resistance mechanisms, and persistence in moist clinical environments can limit treatment options and increase the risk of therapeutic failure. Local surveillance is therefore essential because antimicrobial susceptibility patterns vary across institutions and geographical areas. Reliable regional data can support empirical treatment, antimicrobial stewardship, and timely selection of effective antipseudomonal therapy for affected patients.

Objective: The study determined the occurrence of *P. aeruginosa* in urine specimens from patients with suspected urinary tract infection and assessed the antimicrobial susceptibility profile of confirmed isolates.

Methods: A multicentre, laboratory-based cross-sectional study processed 565 clean-catch midstream urine specimens collected from four public and private laboratories in Punjab, Pakistan, between January and March 2020. Specimens were cultured on cetrimide agar, and suspected isolates were identified through colony morphology, Gram staining, motility, and biochemical testing. Antimicrobial susceptibility to 14 agents was assessed by the modified Kirby–Bauer disc diffusion method using CLSI 2019 interpretive criteria. Frequencies and percentages were calculated.

Results: Fifty-two specimens were positive for confirmed *P. aeruginosa*, giving an occurrence of 9.2%. Of the confirmed isolates, 30 (57.7%) were obtained from females and 22 (42.3%) from males. Susceptibility was highest to colistin at 100%, followed by tobramycin at 88.5%, meropenem at 78.8%, piperacillin–tazobactam and cefepime at 73.1% each, amikacin at 71.2%, imipenem at 69.2%, and gentamicin at 67.3%. Resistance was highest to amoxicillin–clavulanate at 94.2% and ceftriaxone at 82.7%, followed by levofloxacin at 53.8%, ofloxacin at 50.0%, ciprofloxacin at 46.2%, and ceftazidime at 44.2%.

Conclusion: *P. aeruginosa* showed substantial variation in antimicrobial susceptibility, supporting culture-guided therapy and regular local antibiogram surveillance. Rational prescribing and strengthened antimicrobial stewardship remain necessary to limit further resistance and preserve effective treatment options.

Keywords: Anti-Bacterial Agents; Drug Resistance, Bacterial; Microbial Sensitivity Tests; *Pseudomonas aeruginosa*; Pseudomonas Infections; Urinary Tract Infections; Urine

INTRODUCTION

Urinary tract infections (UTIs) are among the most frequently encountered bacterial infections in clinical practice and affect individuals of all age groups. Although both males and females may develop a UTI, the incidence is generally higher among females because of anatomical and physiological factors that facilitate the entry of microorganisms into the urinary tract (1,2). Globally, UTIs are estimated to affect approximately 150 million people each year, creating a substantial burden on patients, healthcare services and national economies. The burden is particularly important in hospitals, where urinary tract infections are commonly associated with prolonged admission, invasive procedures, urinary catheterization and exposure to broad-spectrum antimicrobial therapy. Hospital-acquired UTIs account for approximately 12.9% of healthcare-associated infections in the United States, 19.6% in Europe and nearly 24% in developing countries, indicating a disproportionately greater problem in resource-limited healthcare systems (3). A wide range of bacterial species may cause urinary tract infections; however, *Pseudomonas aeruginosa* is of particular concern because of its association with complicated, recurrent and healthcare-associated infections. The organism is an aerobic, motile, encapsulated, non-fermenting Gram-negative bacillus belonging to the family *Pseudomonadaceae*. It is widely distributed in natural and healthcare environments and can survive under conditions that are unsuitable for many other microorganisms. Within hospitals, it may be recovered from contaminated irrigation fluids, respiratory-care equipment, urinary catheters, cleaning solutions, diluted antiseptics, infusions, cosmetics and even liquid soap (4). Its ability to persist in moist environments allows it to colonize sinks, drainage systems and medical equipment, creating multiple opportunities for transmission to susceptible patients.

The clinical importance of *P. aeruginosa* extends beyond urinary tract infections. It is an established cause of ventilator-associated pneumonia, bloodstream infections, wound infections, burn-related infections and infections of the respiratory tract. Nevertheless, its role in UTIs is especially important among hospitalized patients, older adults, immunocompromised individuals and those with structural or functional abnormalities of the urinary system. Patients with indwelling urinary catheters are at increased risk because catheters can provide both a route of entry and a surface for bacterial attachment and biofilm formation. As the organism is capable of surviving on urinary devices and other moist medical equipment, catheter-associated urinary tract infections represent an important route through which *P. aeruginosa* may spread in healthcare facilities (5). The clinical presentation of a UTI caused by *P. aeruginosa* may resemble that of infections caused by other urinary pathogens. Common manifestations include dysuria, increased urinary frequency, urgency and suprapubic tenderness. In more complicated cases, patients may develop fever, flank pain, systemic inflammatory responses or bloodstream infection, particularly when diagnosis and treatment are delayed (6). The pathogenic potential of *P. aeruginosa* is related to a combination of structural and secreted virulence factors. The organism produces proteases, toxins and tissue-damaging enzymes, while its surface characteristics and biofilm-forming capacity support adherence, persistence and protection from host immune responses. Its ability to avoid or withstand phagocytosis further contributes to prolonged infection and treatment failure (7).

Treatment of *P. aeruginosa* infections presents a major clinical challenge because the organism demonstrates both intrinsic and acquired resistance to multiple antimicrobial agents (8). Its natural resistance is supported by low outer-membrane permeability, antimicrobial efflux systems and the production of enzymes capable of inactivating certain drugs. Additional resistance may develop through mutations or the acquisition of resistance genes under antimicrobial selection pressure. Consequently, isolates that were previously susceptible may become resistant during treatment, particularly when antimicrobial therapy is prolonged, inadequately dosed or used without culture and susceptibility testing. Antimicrobial agents commonly considered for the treatment of susceptible *P. aeruginosa* infections include carbapenems such as meropenem and imipenem, aminoglycosides such as gentamicin and amikacin, antipseudomonal cephalosporins such as cefepime and ceftazidime, antipseudomonal penicillins such as piperacillin and ticarcillin, and fluoroquinolones such as ciprofloxacin and ofloxacin (9). The selection of an appropriate drug depends on the site and severity of infection, the susceptibility profile of the isolate, previous antimicrobial exposure and the clinical condition of the patient. Colistin has also regained importance for the management of multidrug-resistant Gram-negative infections. Although its use has historically been limited by nephrotoxicity and neurotoxicity, it may remain one of the few therapeutic options for infections caused by extensively resistant strains of *P. aeruginosa*. Its use therefore requires careful clinical assessment, dose adjustment and monitoring of renal function.

The increasing resistance of *P. aeruginosa* has serious implications for empirical treatment. When local resistance patterns are unknown, patients may initially receive an antimicrobial agent that is ineffective against the infecting strain. Inappropriate empirical therapy may delay clinical improvement, prolong hospitalization, increase treatment costs and contribute to serious complications. Continuous surveillance of antimicrobial susceptibility is therefore essential for detecting changes in resistance patterns and supporting the development of evidence-based institutional treatment guidelines. Regular antibiogram studies can assist clinicians in selecting appropriate empirical therapy while culture results are pending and can also strengthen antimicrobial stewardship programmes by discouraging unnecessary or ineffective antibiotic use (10). Antimicrobial resistance is particularly concerning in developing countries, where unregulated access to antibiotics, self-medication, incomplete treatment courses, irrational prescribing and the circulation of substandard or counterfeit medicines may accelerate the emergence of resistant organisms (2). In addition, limited microbiology services and inconsistent infection-prevention practices may allow resistant strains to spread within healthcare facilities. The consequences are clinically significant, as mortality has been reported to be higher among patients infected with multidrug-resistant *P. aeruginosa* than among those infected with drug-susceptible strains. Mortality rates of approximately 34% have been reported in patients with multidrug-

resistant infections, compared with approximately 22% among patients with susceptible infections, highlighting resistant *P. aeruginosa* as a potential marker of poor outcomes in hospitalized populations (11).

The global importance of this threat is reflected in the recognition of carbapenem-resistant *P. aeruginosa* as a critical-priority pathogen by the World Health Organization. This classification emphasizes the urgent need for new antimicrobial agents, improved diagnostic capacity, effective infection-control measures and continuous resistance surveillance (12). Despite this concern, the occurrence and antimicrobial susceptibility of urinary *P. aeruginosa* isolates may differ considerably between hospitals, geographical areas and patient populations. Local data are therefore necessary because susceptibility findings from one region may not accurately guide treatment in another. The limited availability of recent institution-specific evidence creates uncertainty regarding the burden of *P. aeruginosa* in urinary infections and the antibiotics that remain effective against circulating isolates. Against this background, the present study sought to determine the occurrence of *Pseudomonas aeruginosa* among urine samples obtained from patients with suspected urinary tract infection and to evaluate the antimicrobial susceptibility pattern of the identified isolates. The research question was whether *P. aeruginosa* represented a meaningful proportion of urinary isolates during the specified study period and which commonly used antipseudomonal antibiotics retained adequate activity against it. The study was therefore undertaken to generate a local antibiogram that could support timely treatment decisions, guide empirical antibiotic selection, contribute to antimicrobial stewardship and assist in reducing the clinical consequences of multidrug-resistant urinary tract infections.

METHODOLOGY

A multicentre, laboratory-based descriptive cross-sectional study was conducted to determine the occurrence and antimicrobial susceptibility profile of *Pseudomonas aeruginosa* isolated from urine specimens of patients with suspected urinary tract infection. A total of 565 urine samples were obtained from selected public and private diagnostic laboratories in Punjab, Pakistan. Of these, 100 samples were collected from Chughtai Laboratory, Sheikhupura; 165 from Chughtai Laboratory, Faisalabad; 100 from the Microbiology Laboratory of Quaid-e-Azam Medical College, Bahawalpur; and 200 from the Microbiology Laboratory of Jinnah Hospital, Lahore. Patients of either sex and all age groups who were clinically suspected of having a urinary tract infection and whose urine specimens were submitted for culture were considered eligible. Properly labelled, adequately collected and sufficient-volume urine specimens were included, whereas leaking, unlabelled, improperly stored, visibly contaminated or duplicate specimens from the same patient were excluded. Urine specimens were collected using the clean-catch midstream technique to minimize contamination by periurethral flora. Patients were instructed to clean the periurethral area, discard the initial portion of urine and collect the midstream portion in a sterile, wide-mouthed, leak-proof container (13,14). Specimens were transported to the respective microbiology laboratories and processed as soon as possible after collection. Each specimen was assigned a laboratory identification number, and patient identifiers were not used during microbiological analysis.

Isolation of *P. aeruginosa* was performed using cetrimide agar supplemented with glycerol, a selective medium that promotes the growth and characteristic pigment production of *Pseudomonas* species (15). The medium was prepared according to the manufacturer's instructions by dissolving 45.3 g of dehydrated cetrimide agar base in the recommended volume of distilled water, followed by the addition of 10 mL glycerol. The mixture was heated until completely dissolved and sterilized in an autoclave at 121°C for 15 minutes. After sterilization, the medium was allowed to cool and was aseptically poured into sterile Petri dishes inside a biosafety cabinet. The plates were left undisturbed until the medium had completely solidified (16). Using a sterile calibrated device, 0.1 mL of each urine specimen was inoculated onto cetrimide agar and evenly spread over the surface. The inoculated plates were incubated aerobically at 35–37°C for 18–24 hours. Colonies showing morphological characteristics suggestive of *P. aeruginosa*, including flat or spreading growth and characteristic pigment production, were selected for further examination. Suspected colonies were subcultured using the four-quadrant streaking technique to obtain pure isolates (17).

Presumptive identification was confirmed through colony morphology, Gram staining, motility assessment and conventional biochemical testing. Gram-stained smears were initially examined under low power and subsequently under the 100× oil-immersion objective to identify Gram-negative bacilli. The biochemical identification panel included oxidase, catalase, citrate utilization, indole, methyl red and Voges–Proskauer tests (18,19). Isolates exhibiting the expected biochemical profile of *P. aeruginosa*, including oxidase positivity, catalase positivity, citrate utilization and negative indole and methyl red reactions, were identified as *P. aeruginosa*. Motility was assessed using a fresh culture prepared in nutrient broth and incubated for less than 24 hours, because ageing cultures may show reduced motility. The organism was considered motile when active directional movement or growth extending away from the inoculation line was observed (20,21). Antimicrobial susceptibility testing was performed using the modified Kirby–Bauer disc diffusion method on Mueller–Hinton agar in accordance with the Clinical and Laboratory Standards Institute guidelines available in 2019. Mueller–Hinton agar was prepared according to the manufacturer's instructions, sterilized at 121°C for 15 minutes and adjusted to a pH of 7.2–7.4. A 0.5 McFarland turbidity standard was prepared by mixing 0.05 mL of 1.18% barium chloride dihydrate with 9.95 mL of 1% sulphuric acid. This standard represented an approximate bacterial density of 1.5×10^8 colony-forming units per millilitre.

For each confirmed isolate, several well-isolated colonies from a fresh culture were emulsified in sterile normal saline. The turbidity of the suspension was visually adjusted to match the 0.5 McFarland standard. A sterile swab was dipped into the standardized suspension, excess fluid was removed by pressing the swab against the wall of the tube, and the entire surface of a Mueller–Hinton agar plate was

inoculated in multiple directions to produce a uniform bacterial lawn. Antimicrobial discs were placed aseptically on the inoculated agar surface, ensuring adequate spacing between discs. The plates were incubated aerobically at 35–37°C for approximately 16–18 hours. The antimicrobial agents tested included amoxicillin–clavulanic acid (20/10 µg), ceftriaxone (30 µg), levofloxacin (5 µg), ofloxacin (5 µg), colistin (10 µg), piperacillin–tazobactam (100/10 µg), cefepime (30 µg), meropenem (10 µg), tobramycin (10 µg), amikacin (30 µg), gentamicin (10 µg), ciprofloxacin (5 µg), ceftazidime (30 µg) and imipenem (10 µg) (22). Following incubation, inhibition-zone diameters were measured in millimetres and interpreted according to the applicable CLSI 2019 breakpoints. Each isolate was categorized as susceptible, intermediate or resistant where validated interpretive criteria were available. The occurrence of *P. aeruginosa* was calculated as the proportion of confirmed isolates among the 565 urine specimens. Antimicrobial susceptibility findings were summarized using frequencies and percentages for each tested antibiotic.

Table 1: 0.5 McFarland turbidity standards preparation

Reagents	Quantity (ml)
H ₂ SO ₄ (1.0%)	9.95
BaCl ₂ . 2H ₂ O (1.18%)	0.05

Table 2: Constituents of McFarland Turbidity Standards

Standard no.	Volume (ml)		Bacterial count per ml (10 ⁸) Represented
	BaCl ₂ . 2H ₂ O (1.18%)	H ₂ SO ₄ (1.0%)	
0.5	0.05	9.95	1.5
1	0.1	9.9	3
2	0.2	9.8	6
3	0.3	9.7	9
4	0.4	9.6	12
5	0.5	9.5	15

RESULTS

A total of 565 urine specimens obtained from patients with suspected urinary tract infection were processed between January and March 2020. The specimens were received from four public and private microbiology laboratories in Punjab, Pakistan. Of the total samples, 200 (35.4%) were collected from Lahore, 165 (29.2%) from Faisalabad, 100 (17.7%) from Sheikhupura and 100 (17.7%) from Bahawalpur. Initial culture produced 54 presumptive *Pseudomonas aeruginosa* isolates, corresponding to 9.6% of all urine specimens. Following confirmatory morphological and biochemical testing, 52 isolates were identified as *P. aeruginosa* and were included in the final analysis, resulting in a confirmed occurrence of 9.2%. The remaining 513 specimens (90.8%) were negative for confirmed *P. aeruginosa*. Among the 52 confirmed isolates, 30 (57.7%) were recovered from female patients and 22 (42.3%) from male patients. Thus, the female-to-male ratio among patients with confirmed *P. aeruginosa* urinary isolates was approximately 1.36:1. Laboratory-specific numbers of positive isolates were not available in the reported dataset; therefore, the occurrence of *P. aeruginosa* could not be calculated separately for Lahore, Faisalabad, Sheikhupura and Bahawalpur.

Growth on cetrimide agar produced round, slightly mucoid colonies with characteristic blue-green to yellow-green pigmentation. The observed pigmentation was consistent with the production of pyocyanin and pyoverdine. A characteristic fruit-like odour was also noted during culture examination. On microscopic examination of Gram-stained preparations under the 100× oil-immersion objective, the isolates appeared as pink, Gram-negative bacilli. All confirmed isolates demonstrated motility. The isolates showed a consistent biochemical profile. Citrate utilization, oxidase and catalase reactions were positive, whereas indole, methyl red and Voges–Proskauer reactions were negative. No acid or gas production was detected on triple sugar iron agar, supporting the non-fermentative characteristics of the isolates. The combined colony morphology, microscopic appearance, motility and biochemical findings were used to confirm the identification of *P. aeruginosa*.

Antimicrobial susceptibility testing was completed for all 52 confirmed isolates against 14 antimicrobial agents. Colistin showed the highest recorded activity, with all 52 isolates classified as susceptible and none classified as resistant. Tobramycin was the second most active agent, with 46 isolates (88.5%) susceptible and 6 (11.5%) resistant. Meropenem was active against 41 isolates (78.8%), while 11 (21.2%) were resistant. Piperacillin–tazobactam and cefepime showed comparable activity. Piperacillin–tazobactam was effective against 38 isolates (73.1%), whereas 14 (26.9%) were resistant. Cefepime also showed susceptibility in 38 isolates (73.1%) and

resistance in 14 (26.9%). Amikacin was effective against 37 isolates (71.2%), while 15 (28.8%) were resistant. Imipenem susceptibility was recorded in 36 isolates (69.2%), with resistance in 16 (30.8%). Gentamicin was active against 35 isolates (67.3%), whereas 17 (32.7%) were resistant.

Lower susceptibility was observed for ceftazidime and the tested fluoroquinolones. Ceftazidime was effective against 29 isolates (55.8%), while 23 (44.2%) were resistant. Ciprofloxacin susceptibility was detected in 28 isolates (53.8%), compared with resistance in 24 (46.2%). Ofloxacin showed equal susceptibility and resistance, with 26 isolates (50.0%) in each category. Levofloxacin was active against 24 isolates (46.2%), whereas 28 (53.8%) were resistant. The lowest recorded activities were observed for ceftriaxone and amoxicillin–clavulanate. Only 9 isolates (17.3%) were categorized as susceptible to ceftriaxone, while 43 (82.7%) were resistant. Amoxicillin–clavulanate showed activity against 3 isolates (5.8%), whereas 49 (94.2%) were resistant. Overall, the greatest recorded susceptibility was observed with colistin, tobramycin and meropenem, while the highest resistance frequencies were recorded for amoxicillin–clavulanate, ceftriaxone and levofloxacin.

Table 3: Antimicrobial Susceptibility Profile of *Pseudomonas aeruginosa* Isolates from Urine Samples

Antibiotics	Potency	Zone of inhibition Diameter (mm)		Resistant Number (%)	Sensitive Number (%)
		Sensitive	Resistant		
Gentamicin	(10 µg)	≥15	≤12	17 (33%)	35 (67%)
Tobramycin	(10µg)	≥15	≤12	6 (12%)	46 (88%)
Amikacin	(30 µg)	≥17	≤14	15 (29%)	37 (71%)
Colistin	(10 µg)	≥11	≤10	0 (0%)	52 (100%)
Imipenem	(10 µg)	≥23	≤19	16 (31%)	36 (69%)
Meropenem	(10 µg)	≥19	≤15	11 (21%)	41 (79%)
Ceftazidime	(30 µg)	≥18	≤14	23 (44%)	29 (56%)
Cefepime	(30 µg)	≥18	≤14	14 (27%)	28 (73%)
Ceftriaxone	(30 µg)	≥18	≤13	43 (83%)	9 (17%)
Ciprofloxacin	(5 µg)	≥21	≤15	24 (46%)	28 (54%)
Levofloxacin	(5 µg)	≥17	≤13	28 (54%)	24 (46%)
Ofloxacin	(5 µg)	≥16	≤12	26 (50%)	26 (50%)
Amoxicillin clavulanic acid ⁺	(20,10 µg)	≥18	≤13	49 (94%)	3 (6%)
Piperacillin+ tazobactam	(100,10 µg)	≥21	≤14	14 (27%)	38 (73%)

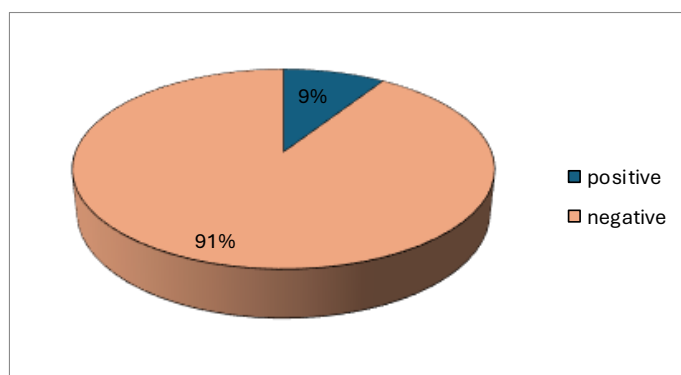


Figure 1: Occurrence of *Pseudomonas aeruginosa* in UTIs

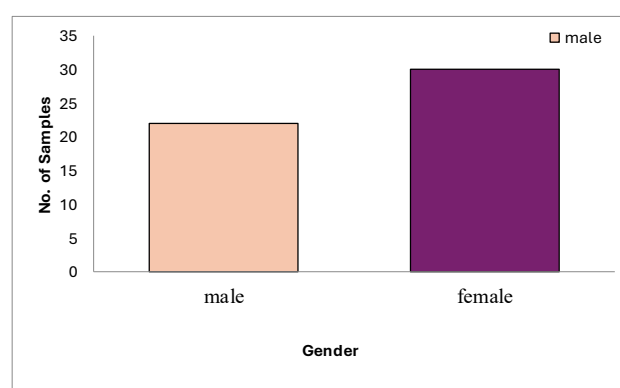


Figure 2: Gender based prevalence of UTIs

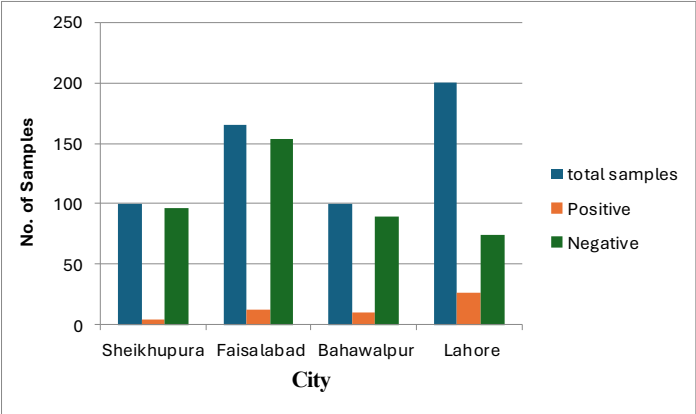


Figure 3: City wise distribution of *Pseudomonas aeruginosa*

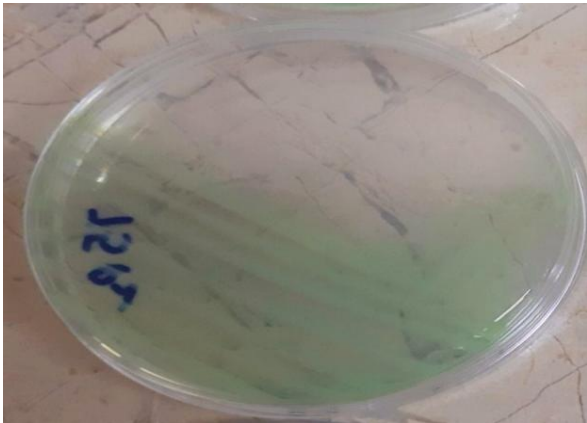


Figure 4: Colony morphology of *pseudomonas aeruginosa* showing blue green colonies

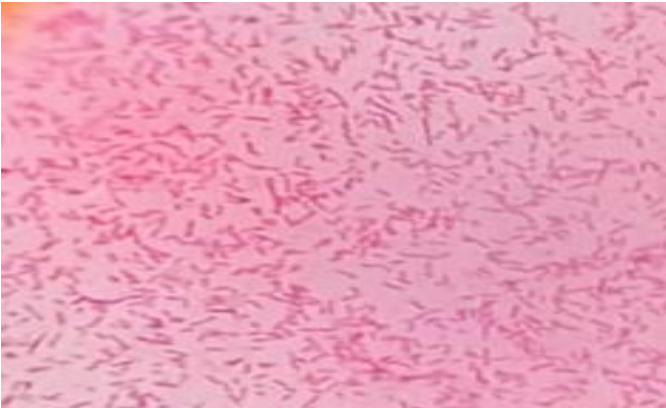


Figure 5: Microscopic view of gram negative pink colored rods (*Pseudomonas aeruginosa*).



Figure 6: Oxidase positive

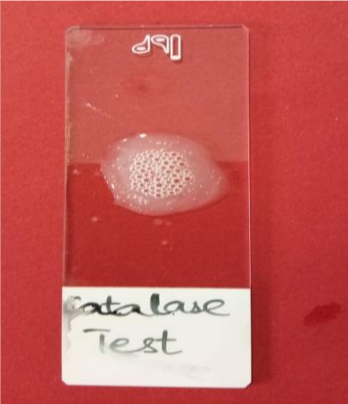
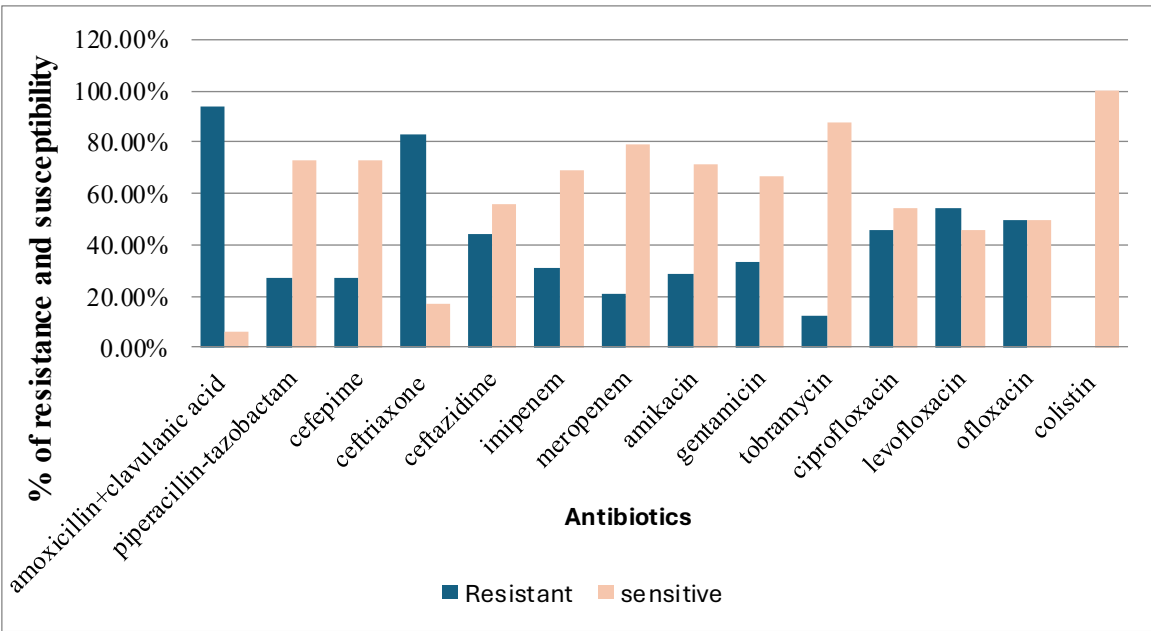


Figure 7: Catalase positive



Effectiveness of antibiotics tested against *Pseudomonas aeruginosa*

DISCUSSION

The present study evaluated the occurrence and antimicrobial susceptibility profile of *Pseudomonas aeruginosa* isolated from urine specimens collected from patients with suspected urinary tract infection in different cities of Punjab, Pakistan. The findings confirmed that *P. aeruginosa* represented an important urinary pathogen, accounting for approximately 9.2% of the processed specimens. Although the proportion was lower than that reported in several hospital-based studies, it remained clinically relevant because infections caused by this organism are frequently associated with urinary catheterization, prolonged hospitalization, prior antimicrobial exposure and underlying urinary tract abnormalities. As an opportunistic pathogen, *P. aeruginosa* has been implicated in a wide range of healthcare-associated infections, including urinary tract infection, pneumonia, wound infection and bloodstream infection. Its clinical importance is further increased by its ability to persist in moist hospital environments and develop resistance to multiple antimicrobial classes. The occurrence observed in the present study was within the broad range reported in previous literature. Earlier studies documented proportions of approximately 18% among isolated uropathogens, 29.6% among urine specimens and 20.5% among patients with suspected UTI (23,24). Other investigations reported lower occurrence rates of nearly 2%, 5.4% and 6%, which were closer to the present finding (25,26). In contrast, markedly higher proportions, including 21.6%, 35% and 49.3%, were reported in studies conducted among hospitalized or catheterized patients (27). These variations were likely related to differences in study populations, hospital settings, specimen-selection criteria, catheter use, antimicrobial exposure, local infection-control practices and methods of bacterial identification. Studies restricted to catheterized or critically ill patients would be expected to yield higher proportions than studies involving a broader group of patients with suspected uncomplicated and complicated urinary infections.

The higher number of isolates recovered from female patients was consistent with the generally greater burden of urinary tract infection among females. However, this finding required cautious interpretation because the total number of male and female urine specimens submitted for culture was not available. Therefore, the observed sex distribution represented the proportion of isolates among confirmed cases rather than sex-specific prevalence. A reliable comparison between male and female patients would have required separate denominators for both groups and adjustment for age, catheterization, hospitalization and underlying urinary disease. The antimicrobial susceptibility findings demonstrated considerable variation across the tested agents. The greatest recorded activity was observed with colistin, tobramycin and meropenem, followed by piperacillin–tazobactam, cefepime, amikacin and imipenem. Complete susceptibility to colistin was comparable with earlier studies in which colistin remained one of the most active agents against multidrug-resistant *P. aeruginosa* isolates (24). Another investigation similarly reported approximately 95% susceptibility to colistin and relatively favorable activity of piperacillin–tazobactam and imipenem (28,29). These findings supported the continued in-vitro activity of last-resort or broad-spectrum antipseudomonal agents. Nevertheless, colistin susceptibility in the present study was determined by disc diffusion, a method now considered unreliable because colistin diffuses poorly through agar. The apparent 100% susceptibility should therefore have been interpreted cautiously and ideally confirmed through broth microdilution.

Meropenem showed greater activity than imipenem, although resistance to both carbapenems was present. This finding was clinically important because carbapenem-resistant *P. aeruginosa* has been recognized as a critical-priority pathogen requiring intensified surveillance and development of new therapeutic options. Reduced carbapenem susceptibility may arise through loss or alteration of outer-membrane porins, overexpression of efflux pumps, increased AmpC production and acquisition of carbapenemases, particularly metallo-beta-lactamases. The coexistence of these mechanisms may lead to multidrug resistance and limit the effectiveness of commonly available therapies (23–25). The observed resistance to carbapenems therefore indicated a need for culture-directed treatment and stronger antimicrobial stewardship. The fluoroquinolones showed moderate to poor activity. Ciprofloxacin remained effective against slightly more than half of the isolates, whereas levofloxacin and ofloxacin showed lower susceptibility. These findings were consistent with increasing fluoroquinolone resistance reported in clinical isolates of *P. aeruginosa*. Such resistance may reflect frequent empirical use of oral fluoroquinolones for urinary infections, incomplete treatment courses and repeated exposure among patients with recurrent disease. The reduced activity of ceftazidime and gentamicin also suggested that these agents should not be selected empirically without considering local susceptibility data.

The highest recorded resistance occurred against amoxicillin–clavulanate and ceftriaxone. These results were not unexpected because *P. aeruginosa* is intrinsically resistant to several beta-lactam agents that lack antipseudomonal activity. Consequently, these drugs have limited therapeutic or interpretive relevance for this organism, and their inclusion may have exaggerated the apparent resistance burden. Future antibiogram studies should prioritize agents with recognized antipseudomonal activity and should apply current organism-specific interpretive standards. The study had several strengths. It included a relatively large number of urine specimens and collected samples from multiple public and private laboratories across different cities, thereby providing broader regional information than a single-centre investigation. The use of selective culture media, biochemical confirmation and standardized disc diffusion methods also supported consistency in isolate identification and susceptibility testing.

Several limitations remained. The study lacked detailed clinical information regarding age, hospitalization, catheter use, previous antibiotic exposure, recurrent UTI and comorbid conditions. It therefore could not identify factors associated with infection or resistance. Laboratory-specific positive counts were unavailable, preventing comparison between cities and institutions. Intermediate susceptibility results, multidrug-resistance definitions and resistance combinations were not reported. Molecular testing for resistance genes and

confirmatory methods for colistin were also not performed. In addition, the discrepancy between presumptive and confirmed isolate counts required clarification from the original laboratory records. Overall, the findings emphasized that *P. aeruginosa* remained a clinically important urinary pathogen with substantial resistance to several commonly used antibiotics. The results supported routine urine culture, local antibiogram development and avoidance of inappropriate empirical therapy. Future multicentre studies should include patient-level risk factors, standardized susceptibility methods, molecular characterization of resistance mechanisms and periodic surveillance to identify changes in antimicrobial effectiveness over time.

CONCLUSION

The study confirmed that *Pseudomonas aeruginosa* was an important cause of urinary tract infection and demonstrated considerable variation in its response to commonly used antimicrobial agents. Several routinely prescribed antibiotics showed limited activity, whereas selected antipseudomonal drugs remained comparatively effective. These findings highlighted the importance of culture-based diagnosis and local antimicrobial susceptibility testing before treatment initiation. Regular surveillance, rational antibiotic prescribing and strengthened antimicrobial stewardship are essential to preserve the effectiveness of available therapies and reduce the emergence and spread of multidrug-resistant *P. aeruginosa*.

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