ANTIMICROBIAL RESISTANCE PROFILE AND MOLECULAR SCREENING OF *bla*KPC GENE AMONG CLINICAL ISOLATES OF PSEUDOMONAS AERUGINOSA FROM A TERTIARY CARE HOSPITAL, NEPAL

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ABSTRACT

Pseudomonas aeruginosa is a major opportunistic pathogen with increasing antimicrobial resistance, creating significant treatment challenges in hospital settings. This study aimed to determine the antimicrobial resistance profile of *P. aeruginosa* and screen for the *blaKPC* gene among meropenem-resistant isolates. A descriptive cross-sectional study was conducted from December 2021 to May 2022, yielding 75 non-duplicate isolates identified through standard microbiological methods; antimicrobial susceptibility testing was performed using the Kirby-Bauer technique, and 17 meropenem-resistant isolates underwent PCR for *bla*KPC detection. Sputum (33.33%) and pus (29.33%) were the most common specimen sources, and multidrug resistance was observed in 54.7% of isolates, particularly among inpatients and ICU cases. High-rate resistance to ceftazidime and piperacillin was noted, while carbapenem resistance remained moderate (21-23%). None of the meropenem-resistant isolates carried the *bla*KPC gene, indicating alternative carbapenem resistance mechanisms. These findings highlight a substantial burden of MDR *P. aeruginosa* and underscore the need for ongoing surveillance and strengthened infection control practices.

KEYWORDS

Pseudomonas aeruginosa, MDR, ESBL, *bla*KPC gene, Nepal

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INTRODUCTION

Pseudomonas aeruginosa is an aerobic, rod-shaped, non-fermenting Gram-negative bacterium widely distributed in the environment, including soil, plants, and hospital water systems such as sinks, showers, and toilets.[1] It is a major opportunistic pathogen responsible for severe infections in immunocompromised and hospitalized patients.[1,2] The organism exhibits remarkable intrinsic resistance and a strong ability to acquire new resistance determinants, leading to limited therapeutic options and posing significant treatment challenges.[2]

Antimicrobial resistance (AMR) is a growing global health threat, driven by the emergence and spread of multidrug-resistant (MDR) bacteria.[3] Among these, *P. aeruginosa* is of particular concern due to its increasing rate of resistance to multiple antibiotics.[4,5] The bacterium employs several mechanisms to evade antimicrobial action, including efflux pump overexpression (MexAB-OprM), production of chromosomal AmpC β-lactamase, loss of the OprD porin, and production of carbapenemases belonging to Ambler classes A (*bla*KPC), B (*bla*IMP, *bla*VIM, *bla*NDM, *bla*SIM, *bla*GIM, *bla*SPM) and D (*bla*OXA-48).[6]

Klebsiella pneumoniae carbapenemases (KPCs), encoded by *bla*KPC gene, hydrolyze penicillins, cephalosporins and carbapenems.[7,8] Although KPCs are more common in Enterobacteriaceae, their emergence in *P. aeruginosa* has been increasingly reported worldwide, often associated with plasmid-mediated horizontal gene transfer, facilitating rapid dissemination.[7-10] Globally, the coexistence of *bla*KPC with other carbapenemase genes such as *bla*IMP, *bla*VIM, *bla*NDM has been documented in *P. aeruginosa* isolates.[9,10] In Nepal, several studies have reported *bla*IMP-1, *bla*VIM-2, and *bla*NDM-1 among *P. aeruginosa*[11-13], but reports of *bla*KPC-positive strains remain limited.[11-13]

Although KPCs are not the only mechanism conferring carbapenem resistance, they are clinically significant because they are often undetected by conventional testing and have a high potential for rapid spread. Their presence severely limits treatment options and complicates infection control efforts. Therefore, this study aims to determine the antimicrobial susceptibility profile and detect the *bla*KPC gene among clinical isolates of *P. aeruginosa* to provide evidence for empirical therapy and strengthen infection control strategies in healthcare settings.

MATERIALS AND METHODS

A descriptive cross-sectional study was conducted from December 2021 to May 2022 in the Clinical Microbiology Laboratory, Nepal Medical College Teaching Hospital (NMCTH), Kathmandu, Nepal. A total of 75 non-duplicate *P. aeruginosa* isolates were obtained from various clinical specimens, including pus, blood, urine, sputum, and body fluids collected from patients attending NMCTH.

Isolation and identification: All specimens were processed following standard microbiological procedures. Briefly, samples were inoculated onto appropriate culture media [urine on Cystine Lactose Electrolyte Deficient (CLED) agar, pus on blood and MacConkey agar, sputum and body fluids on blood, MacConkey and chocolate agar] and incubated aerobically at 37^{0}C for 24 hours. Blood cultures were incubated at 37^{0}C and sub-cultured every alternate day for up to seven days. Identification of *P. aeruginosa* was done based on colony characters, Gram staining morphology and conventional biochemical tests.[14]

Antimicrobial susceptibility testing: Antimicrobial susceptibility testing was performed by the Kirby-Bauer disc diffusion method on Mueller-Hinton agar (MHA) following the guidelines of Clinical and Laboratory Standards Institute (CLSI).[15] The following antibiotic discs (Hi-Media Laboratories, Mumbai, India) were used: piperacillin (100 µg), ceftazidime (30 µg), tobramycin (30 µg), ciprofloxacin (5 µg), ofloxacin (5 µg), cotrimoxazole (1.25/23.75 µg), imipenem (10 µg), meropenem (10 µg) and piperacillin-tazobactam (100/10 µg).

Screening for multidrug resistance: Isolates resistant to at least one antimicrobial agent in three or more different classes were categorized as multidrug-resistant (MDR).[16]

Molecular detection of *bla*KPC gene: Of the 75 *P. aeruginosa* isolates included in the study, the 17 isolates that demonstrated phenotypic resistance to meropenem were further subjected to molecular testing for the detection of the *bla*KPC gene at Molecular Laboratory of Annapurna Research Center, Kathmandu.

Detection of the *bla*KPC gene was carried out by conventional polymerase chain reaction (PCR) following standard protocols. Genomic DNA was extracted using the Qiagen DNA extraction kit (Qiagen, Germany). PCR amplification was performed using forward primer

Uni-KPC-F (5'-ATGTCACTGTATCGCCGTCT-3') and reverse primer Uni-KPC-R (5'-TTACTGCCCGTTGACGCCC-3'). The reaction mixture contained DNA template, nuclease-free water, primers, and master mix (Thermo-Fisher Scientific, USA). PCR products were subjected to gel electrophoresis, and a band size of 239 base pairs was considered positive for *bla*KPC.[17]

Data analysis: Data entered in excel and analyzed using STATA-14 software. Descriptive statistics were used to summarize data, with categorical variables expressed as frequencies and percentages, and continuous variables as medians and interquartile ranges (IQR). Association between variables, including patient category, specimen type, and hospital unit, were evaluated using the Chi-square (χ^2) test or Fisher's Exact test when appropriate. A *p*-value <0.05 was considered statistically significant.

Ethical considerations: Ethical approval for this study was taken from the Institutional Review Committee of NMCTH (Ref. No.: 042-078/079). All data and information were handled with strict confidentiality and used solely for research purposes. Patient identifiers were excluded, and numerical codes were assigned to ensure anonymity and protect privacy throughout the study.

RESULTS

A total of 6,105 clinical specimens (urine: 3268, blood: 1509, sputum: 705, pus: 532 and body fluids: 94) from (both inpatient and outpatient) all age groups received for aerobic bacterial culture and antimicrobial susceptibility testing at Clinical Microbiology Laboratory of NMCTH were included in the study. Of the total specimens processed, 1,173 (502 inpatient's and 671 outpatient's) clinical samples showed bacterial growth with a growth positivity rate of 19.21%.

The prevalence rate of *Pseudomonas* spp. was 6.39% (n=75/1,173) among the total bacterial isolates and 1.22 % among the total clinical specimen processed (n=75/6,105). Of the total 75 pseudomonal isolates, 64.00% (n=48/75) were from inpatients and 36.00% (n=27/75) from outpatients. Likewise, 56.00% (n=42/75) were from males and 44.00% (n=33/75) were from females. The isolates obtained were 25 (33.33%), 22 (29.33%), 20 (26.67%), 6 (8.00%), and 2 (2.66%) from sputum, pus, urine, blood, and body fluids, respectively. The highest positivity rate among the processed sample was found in the pus sample 4.13% (n=22/532) followed by sputum 3.54% (n= 25/705), body fluids 2.12% (n=2/94), urine 0.61% (n=20/3268) and blood 0.40% (n= 6/1509). The rate of isolation of *P. aeruginosa* was higher among the isolates from inpatient 9.56% (n= 48/502)

Table 1: Distribution of clinical isolates of *P. aeruginosa* according to the age of patients (n=75)

Age of patients (years)	*n* of isolates (%)
≤20	5 (6.67)
21-40	20 (26.67)
41-60	28 (37.33)
>60	22 (29.33)

Table 2: Distribution of MDR *P. aeruginosa* isolates according to patient category, specimen type, and hospital unit (n = 41)

Variables		Category	Number of MDR isolates (n=41)	%
Specimen type		Urine	10	65.85
		Blood	1	2.44
		Pus	18	43.90
		Sputum	11	26.82
		Body-fluids	1	2.44
Total			41	
Patient type	Inpatients (n=27)	Medical ICU	8	19.50
		Surgical ICU	7	17.10
		Medical Ward	2	4.87
		Gynecology ward	3	7.32
		Post operative ward	5	12.19
		Neuro ward	2	4.87
	OPD patients (n=14)		14	34.15

Table 3: Resistance pattern of *P. aeruginosa* to various antibiotics (n=75)

Antibiotics	*n* of resistance (%)
Piperacillin	38 (50.66)
Ceftazidime	40 (53.33)
Tobramycin	27 (36.00)
Ciprofloxacin	35 (46.66)
Ofloxacin	33 (44.00)
Piperacillin-tazobactum	18 (24.00)
Imipenem	16 (21.33)
Meropenem	17 (22.66)

than the isolates from OPD 4.02% (n= 27/671). The median age of the patients was 52 years, with an interquartile range (IQR) of 36 to 66. The distribution of the isolates according to the age group of patients is shown in Table 1.

The prevalence of MDR *P. aeruginosa* was 54.66% (n=41/75). Among the MDR *P. aeruginosa* isolates, the majority 65.85% (n=27/41) were recovered from inpatients, while 34.15% (n=14/41) were from outpatients. The highest rate of isolation was in pus samples 43.90% (n=18/41). Based on hospital distribution, MDR isolates were most frequently obtained from patients admitted to the Medical Intensive Care Unit (MICU) 19.50% (n=8/41), Surgical ICU (SICU) 17.10% (n=7/41) and post-operative ward 12.19% (n=5/41) (Table 2).

The antimicrobial susceptibility pattern of *P. aeruginosa* showed: more than half of the isolates resistant to ceftazidime (53.33%) and piperacillin (50.66%). Resistance to piperacillin-tazobactam, imipenem and meropenem remained comparatively low (21.00%-24.00%) (Table 3)

PCR analysis of the 17 meropenem-resistant *P. aeruginosa* isolates did not detect the *bla*KPC gene in any of the samples which is shown in Fig. 1.

DISCUSSION

In this study, we investigated the antimicrobial resistance profile of *P. aeruginosa* isolates from a tertiary care hospital in Nepal and screened meropenem-resistant isolates for the presence of *bla*KPC gene. Among 6105 clinical specimens processed, 19.21% (n=1,173) demonstrated bacterial growth, with *P. aeruginosa* accounting for 6.39% of the total bacterial isolates (n=75) and 1.22% of all specimens processed. The overall prevalence of *P. aeruginosa* in our study is consistent with previous reports from tertiary care settings in the region,[18-20] reflecting its role as an opportunistic pathogen in hospitalized patients and its capacity to colonize diverse clinical sites.[20]

The majority of *P. aeruginosa* isolates were recovered from inpatients (n=48), compared to outpatients (n=27), and the median age of affected patients was 52 years. Similar findings showing the common prevalence of *P. aeruginosa* among inpatients have been reported in various studies conducted in various durations.[19-21] The predominance of *P. aeruginosa* isolates among inpatients reflects its established role as a major healthcare associated pathogen, similar to the report by others.[20,21] Hospitalized patients, especially those exposed to invasive devices or broad-spectrum antibiotics are more susceptible to colonization and infection due to disrupted host defenses and increased environmental exposure within hospitals.[21,22] The median age of 52 years further indicates greater vulnerability among older individuals, who commonly have impaired immunity and multiple comorbidities

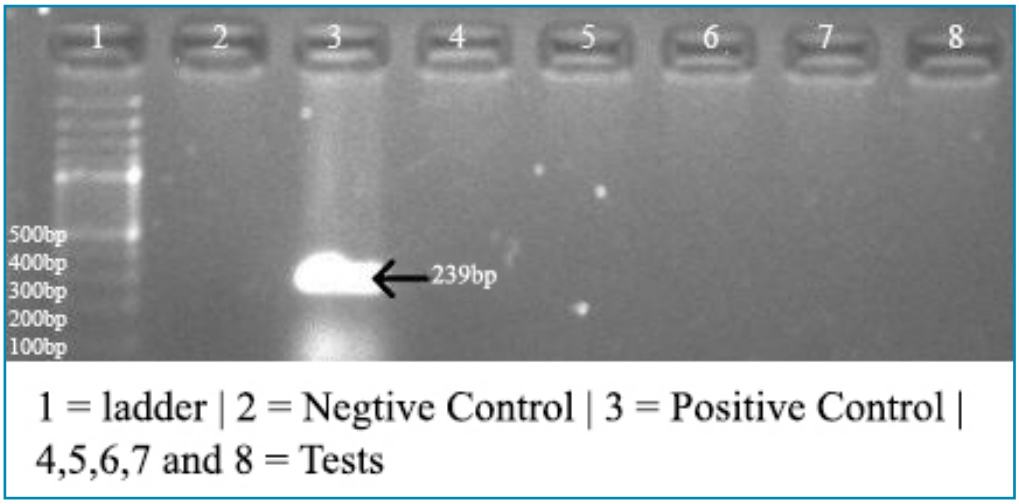

Fig. 1: Gel image for detection of *bla*KPC gene

that facilitate opportunistic infections.[23] These findings highlight the combined influence of hospitalization and advancing age on the risk of *P. aeruginosa* infection, highlighting the need for strengthened infection control and antimicrobial stewardship in high-risk groups.[23,24]

This study showed sputum (33.33%) and pus (29.33%) were the most frequent sources of *P. aeruginosa*, with the highest positivity rates in pus (4.13%) and sputum (3.56%). Similar report was found from the same set-up in the previous study[20] however, varying results have been reported from various regions.[2,4,7,18,19] The predominance of *P. aeruginosa* in sputum and pus is consistent with its established role in respiratory tract and post-surgical wound infections. Recent studies underscore its clinical impact in these niches for instance, its involvement in ventilator-associated pneumonia with significant mortality.[24] Likewise, postoperative wound infections remain a major concern; *P. aeruginosa* has been identified as an emerging threat in surgical sites.[25]

Our study revealed a substantial prevalence of MDR *P. aeruginosa* (54.66%). MDR isolates were more frequently obtained from inpatients, particularly from the MICU (19.51%), SICU (17.10%), and post-operative wards (12.19%), underlining the role of critical care settings as reservoirs of resistant strains. These findings are comparable to previously reported data from Nepal, where MDR prevalence in *P. aeruginosa* ranged around 50.0%-65.0% in both hospital and ICU settings.[18-20]

This highlights that ICU patients as a high-risk population for acquiring MDR *P. aeruginosa*, likely due to invasive procedures, prolonged hospitalization, and frequent antibiotic exposure.[21,22] Among MDR isolates, pus samples accounted for the highest proportion (43.90%), reflecting the burden of wound infections with resistant strains.[25]

The antimicrobial susceptibility profile demonstrated that more than half of the isolates were resistant to ceftazidime (53.33%) and piperacillin (50.66%), while resistance to fluoroquinolones (ciprofloxacin 46.66%, ofloxacin 44.0%) and aminoglycosides (tobramycin 36.0%) was moderate. Similar susceptibility trends have been documented in Nepalese hospitals, although the exact resistance rates can vary by location and time.[18-20,27,28] Resistance rates to β-lactam/β-lactamase inhibitor combinations (piperacillin-tazobactam 24.0%) and carbapenems (imipenem 21.33%, meropenem 22.66%) were comparatively lower, suggesting that these agents remain viable therapeutic options in this setting. However, the presence of carbapenem resistance (22.66%) is worrisome, as carbapenems are typically reserved for severe infections caused by resistant gram-negative pathogens.[6] The high level of resistance to ceftazidime and piperacillin is concerning, as these antibiotics are frequently used as first-line agents for severe *P. aeruginosa* infections.[26] These resistance patterns may be attributed to the intrinsic adaptive mechanisms of *P. aeruginosa*, including efflux pump overexpression, β-lactamase production, and porin modifications.[5,6]

In this study, only meropenem-resistant *P. aeruginosa* isolates (n=17) were selected for *bla*KPC detection because carbapenem resistance is a strong phenotypic indicator of possible carbapenemase production.[11-13] Screening all 75 isolates regardless of susceptibility would be resource-intensive and is not routinely recommended in diagnostic algorithms.[11,12] By focusing on the 17 meropenem-resistant isolates, the study targeted the subgroup most likely to harbour KPC-type β-lactamases, thereby improving the diagnostic yield and ensuring optimal use of molecular testing resources. However, on molecular screening there was absence of PCR amplification and lack of bands aligning with the positive control in gel electrophoresis. This indicates that KPC-type carbapenemases are not contributing to carbapenem resistance in the studied isolates.

The absence of *bla*KPC among the study isolates may be attributed to the relatively small sample size and the single-center study design, which may not fully represent the broader epidemiological trends of carbapenemase distribution in the region. Since *bla*KPC remains uncommon in Nepal and South Asia compared to other carbapenemase genes such as *bla*IMP, *bla*VIM, and *bla*NDM,[7,11-13,29] its detection often requires larger datasets to capture low-prevalence resistance determinants.[29,30]

Furthermore, variations in antimicrobial pressure, infection control practices, and local transmission dynamics across different hospitals may influence the emergence and spread of KPC-producing strains.[6,19,21-23] Therefore, a larger, adequately powered study involving a multicentric approach across diverse healthcare settings would provide a more comprehensive understanding of the prevalence and molecular epidemiology of *bla*KPC in *P. aeruginosa*. Expanded surveillance would also help determine whether the current

absence reflects a true epidemiological pattern or simply under-detection due to limited sampling.

The lack of *bla*KPC in our isolates also suggests that other mechanisms are driving cabpenem resistance. In *P. aeruginosa*, loss of inactivation of the OprD porin, overexpression of efflux pumps, and production of metallo-β lactamases like VIM, IMP, NDM are well-recognized contributors.[21,24] Evidence shows that disruptions in the *opr*D gene can markedly reduce carbapenem uptake, leading to high-level resistance.[30] This finding is consistent with regional reports, where MBLs are commonly identified while KPC-type carbapenemases remain uncommon.[5,7,11,12] This further underscores the need to expand molecular surveillance to include a broader spectrum of carbapenemase genes beyond KPC-type, in order to obtain a more accurate understanding of the molecular epidemiology of carbapenemase-producing *P. aeruginosa* in the region.

From a public health and clinical management perspective, the high burden of MDR *P. aeruginosa*, particularly in ICU and post-surgical settings, signifies the need for robust infection prevention and control interventions and antimicrobial stewardship.[6,23,24,26] Empirical therapy in our hospital should be guided by the local resistance data we have generated; for example, carbapenems and piperacillin-tazobactam may retain utility but should be used judiciously to prevent further selection pressure.

This study demonstrates a high prevalence of MDR *P. aeruginosa* in a tertiary care hospital in Nepal, with significant resistance to commonly used antipseudomonal agents. Although *bla*KPC was not detected among meropenem-resistant isolates, likely through alternative mechanisms or under-detection due to limited samples, carbapenem resistance persists. Continuous surveillance, targeted molecular screening, and stringent infection control practices are critical to mitigate the spread of resistant *P. aeruginosa* strains in hospital settings.

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