ANTIMICROBIAL RESISTANCE AMONG *PSEUDOMONAS AERUGINOSA* ISOLATED AT A TERTIARY CARE HOSPITAL, NEPAL: A DESCRIPTIVE CROSS-SECTIONAL STUDY

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ABSTRACT

Pseudomonas aeruginosa is one of the common isolates from various clinical samples. Infections by it, are usually difficult to treat due to its emerging acquisition of drug resistance, limiting the therapeutic options. This study was done to determine the prevalence of *P. aeruginosa* and its antibiotic susceptibility patterns in our setup. A descriptive cross-sectional study was conducted over a year from April 2019 to March 2020 in the Microbiology Laboratory of Nepal Medical College Teaching Hospital, Kathmandu, Nepal in 121 non-repetitive clinical isolates of *P. aeruginosa.* Identification of the isolates and performance of antimicrobial susceptibility testing were done by using standard microbiological procedures. The prevalence rate of *P. aeruginosa* was 5.98% (n=121) among the total bacterial isolates (n=2021) and 1.02% among the total clinical specimens processed (n=11,880). Of the total 121 isolates, 84 (69.42%) were from inpatients and 37 (30.58%) from outpatient clinical samples. The prevalence of MDR and ESBL-producing *P. aeruginosa* was 53.71% (n=65) and 20.60% (n=25), respectively. Except, for carbapenems and piperacillin-tazobactam, the susceptibility of the isolates towards other commonly prescribed and tested antibiotics like piperacillin, tobramycin, ceftazidime, ciprofloxacin and ofloxacin were around 50.00%. The presence of MDR and ESBL producing *P. aeruginosa* and their resistance against most of the commonly used antibiotics in our set-up necessitates urgent and coordinated efforts to implement effective antimicrobial stewardship, strengthen infection control practices and enhance surveillance system. Addressing these issues are essential to preserve the efficacy of existing antibiotics and safeguard public health.

KEYWORDS

Pseudomonas aeruginosa, MDR, ESBL, Nepal

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INTRODUCTION

Pseudomonas aeruginosa is an aerobic, rod-shaped, non-fermenting gram-negative bacteria found in diverse environmental settings like soil, plants, and hospital reservoirs of water including showers, sinks, and toilet water.[1] It is an opportunistic pathogen and is one of the leading causes of infections like pneumonia, wound infection, urinary tract infection, and bacteremia, especially among immune-suppressed and hospitalized patients.[2,3] Prolonged hospital stays, mechanical ventilation in ICU setup, use of medical devices, and lack of proper antisepsis during medical procedures are the common risk factors for its infections which are often severe and life-threatening.[4]

Intrinsic resistance to commonly used antimicrobials and the ability to easily acquire antibiotic resistance in these bacteria has made limited antibiotic options and has posed a great challenge to treat its infections.[5] Ubiquitous presence,[1] common association with varieties of infections,[2,3] and extensive use of antibiotics have generated selective pressure to encourage the development of resistance in these bacteria because they have the genetic capacity to express a wide repertoire of resistance mechanisms and mutation in chromosomal genes.[4,5] Similarly, they can easily acquire resistance genes from other organisms via plasmid, bacteriophages, and transposons resulting in overexpression of efflux-pumps,[5] production of antibiotics inactivating enzymes like extended-spectrum beta-lactamases (ESBL), Amp-C beta-lactamases and metallo-beta-lactamases (MBL) making the antibiotics not effective.[5,6]

The use of appropriate antibiotics is still the mainstay to treat infections by *P. aeruginosa* however, the emergence of multidrug resistance in these bacteria worldwide, which may vary in different geographical settings has jeopardized the convenient therapy against their infections.[7] So, it is necessary to perform region-specific studies to generate data locally that may aid the clinicians to choose the accurate treatment regimen.

In a developing country like Nepal, unnecessary, excessive, and injudicious use of antibiotics plays an important role in the development of resistance among pathogens like *Pseudomonas*.[8-10] Several studies on antimicrobial susceptibility patterns of *P. aeruginosa* have been conducted in Nepal so far,[8-12] however, the data available is not enough to explore the true scenario in local settings. Continued studies on antimicrobial resistance among *P. aeruginosa* are crucial to determine the susceptibility pattern against commonly prescribed antibiotics in Nepal and help the clinician to choose the appropriate treatment options. Therefore, this descriptive cross-sectional study was done to determine the distribution rate and antimicrobial susceptibility pattern of *P. aeruginosa* isolated from various clinical samples of the patients attending Nepal Medical College Teaching Hospital (NMCTH).

MATERIALS AND METHODS

A descriptive cross-sectional study was conducted over a year from April 2019 to March 2020 in the Clinical Microbiology laboratory of NMCTH, Kathmandu, Nepal. The study was done in 121 non-repeated bacterial isolates of *P. aeruginosa* from clinical specimens (pus, blood, urine, sputum, and body fluids) from patients attending NMCTH.

Isolation and identification: All the clinical samples received in the Clinical Microbiology Laboratory for culture and sensitivity were processed as a routine diagnostic process by standard microbiological techniques. In brief, the specimens were inoculated in culture plates (urine in CLED medium, pus in blood agar and MacConkey agar, sputum, and body fluids in blood agar, MacConkey agar and chocolate agar). All inoculated plates were incubated at 37^{0}C for 24 hours aerobically. Blood culture bottles received were incubated at 37^{0}C and after 24 hours, sub-cultured in blood agar and MacConkey agar every alternate day for seven days. Bacterial isolates of *P. aeruginosa* were then identified further by studying colony characters, gram stain, and biochemical tests.[13]

Antimicrobial susceptibility test: The antimicrobial susceptibility testing was done by Kirby-Bauer disc diffusion method in Mueller Hinton agar (MHA) as per the Clinical and Laboratory Standards Institute (CLSI) guidelines[14] by using the following commercially available antimicrobial discs from Hi-media, Laboratories, Mumbai, India. Piperacillin (100μg), Ceftazidime (30μg), Tobramycin (30μg), Ciprofloxacin (5μg), Ofloxacin (5μg), Cotrimoxazole (1.25/23.75μg), Imipenem (10μg), Meropenem (10μg), Piperacillin–Tazobactam (100/10μg).

Screening of MDR and potential ESBL producers: In this study, the isolates that are resistant to at least one agent of three different classes of commonly used antimicrobial agents, were regarded as multi-drug resistant (MDR).[15] The bacterial isolates with a zone of inhibition

(ZOI) ≤25mm for Ceftriaxone, ≤22mm for Ceftazidime, and/or ≤27mm for Cefotaxime were considered as a potential ESBL producer as recommended by CLSI .[14]

Phenotypic confirmation of ESBL: Isolates that were considered as potential ESBL producers by initial screening were emulsified in nutrient broth to adjust the inoculum density equal to that of 0.5 McFarland turbidity standards. Combination disk test (CDT), as recommended by the CLSI, was performed in all isolates presumed to be ESBL producers. In this test, Ceftazidime (30μg) and Cefotaxime (30μg) disks alone and in combination with Clavulanic acid (Ceftazidime + Clavulanic acid, 30/10μg and Cefotaxime + Clavulanic acid, 30/10μg) disks were applied onto a plate of MHA with the test strain and then incubated in ambient air for 18 hours of incubation at 37^{0}C. Isolate that showed an increase of ≥5 mm zone of inhibition either in one or both the combination disks in comparison to that of the disk used alone was considered as ESBL producer.[14]

RESULTS

A total of 11,880 clinical specimens (urine: 6650, blood: 2305, sputum: 1350, pus: 1190 and body fluids: 385) from both inpatients and outpatients of all age groups received for aerobic bacterial culture and antimicrobial susceptibility testing at Clinical Microbiology Laboratory of NMCTH from April 2019 to March 2020 were included in the study. Of the total specimens processed 2021 (701 inpatient's and 1,320 outpatient's) clinical samples showed bacterial growth with a growth positivity rate of 17.01%.

Table 1: Distribution of clinical isolates of *P. aeruginosa* according to the age of patients (n=121)

Age of patients (years)	*n* of isolates (%)
<20	18 (14.88)
21-40	26 (21.49)
41-60	37 (30.58)
>60	40 (33.05)

The prevalence rate of *Pseudomonas* spp. was 5.98% (n=121) among the total bacterial isolates and 1.02 % among the total clinical specimen processed. Of the total 121 (84 from inpatients and 37 from outpatients) pseudomonal isolates 71 were from males and 50 were from females. The isolates obtained were 50 (41.32%), 45 (37.19%), 16 (13.22%), 8 (6.61%), and 2 (1.65%) from sputum, urine, pus, blood, and body fluids, respectively. The highest positivity rate among the processed sample was found in the sputum sample (3.70%) followed by pus (1.34%), urine (0.68%), body fluids (0.52%), and blood (0.35%). The rate of isolation of *P. aeruginosa* was higher among the isolates from inpatient (n=701) than the isolates from OPD (n=1320) (11.98% vs 2.80%). The distribution of the isolates according to the age group of patients is shown in Table 1.

Table 2: Resistance pattern of *P. aeruginosa* to various antibiotics

Antibiotics	*n* of resistance (%)
Piperacillin	60 (49.58)
Ceftazidime	66 (54.54)
Tobramycin	40 (33.00)
Ciprofloxacin	54 (44.62)
Ofloxacin	50 (41.32)
Piperacillin-Tazobactum	24 (19.83)
Imipenem	16 (13.22)
Meropenem	17 (14.04)

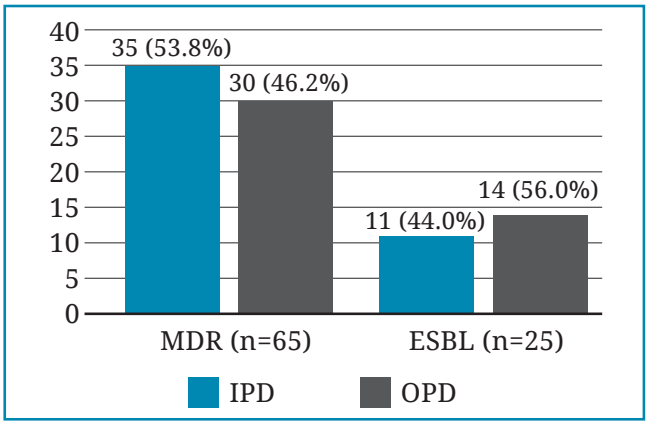

Fig. 1: Rate of MDR and ESBL producing *P. aeruginosa* in clinical samples from inpatients and outpatients.

The prevalence of MDR and ESBL producing *P. aeruginosa* was 53.71% (n=65) and 20.60% (n=25), respectively. All ESBL producing pseudomonal isolates were MDR and their rate is higher in the outpatient sample. The multidrug resistance rate was higher in isolates among the clinical samples collected from inpatients (Fig. 1). The antimicrobial susceptibility pattern of *P. aeruginosa* is shown in Table 2.

DISCUSSION

Ubiquitous presence,[1] easy colonization from external sources in patients and healthy individuals,[2] biofilm production,[2,4] survival even at low-level disinfectants and at a wide

range of temperature,[1] intrinsically resistant to common antibiotics,[5] etc. explains the common presence of *P. aeruginosa* especially in hospital set-up resulting in their frequent association with varieties of infections.

In this study, the prevalence of *P. aeruginosa* was 5.98% among the total bacterial isolates and 1.02% among the total clinical specimen processed which was relatively higher than the previous study done in the same set-up in 2015.[12] Similarly, a higher rate of prevalence has been reported in other studies conducted in Nepal[16,17] and India.[18,19] Previous colonization, rate of hospitalization and hospital stay, infection prevention practices in the hospitals, patient's underlying conditions etc. are the risk factors for pseudomonal infections which may vary with different geographical and hospital settings.[2,4] However, their common presence in various clinical infections sounds for extensive studies to be done analyzing the risk factors for their infections so that preventive measures can be applied to decrease the threat of pseudomonal infections.

Many studies suggested that Pseudomonal infections are more common in males than females.[12,18-20] A similar result was found in this study also. However, there was no significant association of *P. aeruginosa* infection with gender (P>0.05).

Community, as well as hospital-acquired *P. aeruginosa* infections, are increasing nowadays worldwide.[21,22] This study showed the higher number of isolates from inpatient samples as compared to outpatient samples which had a significant association between outpatient/ inpatient and *P. aeruginosa* infection (P<0.05). This is in accordance with several studies conducted in different geographical settings on different time frames.[12,16,19,20] *P. aeruginosa* as compared with other pathogens, can easily thrive in the hospital environment as they are relatively resistant to low-level antiseptics[1] and common antibiotics,[5] can resist a wide range of temperature variations,[1] and can easily form biofilm on catheters and prosthetic devices.[1,2,4] This explains their common presence in the hospital environment and association with infections in hospitalized patients who are relatively immunocompromised due to disease conditions and other underlying medical interventions.

In this study, the highest number of *P. aeruginosa* was isolated from sputum (41.32%) followed by urine (37.19%), pus (13.22%), blood (6.61%), and body fluids (1.65%) which is similar to the findings reported by Mahaseth *et al.*[17] Ojha *et al.*[23] and Sharma *et al.*[24] However, this result was not consistent with the findings by Subha *et al*,[12] Senthamaria *et al.*[25] who reported a greater number of isolates from pus samples. The distribution of specimens may vary with each hospital and type of patient as each hospital facility has a different environment associated with it. Moreover, *P. aeruginosa* is more common to cause nosocomial pneumonia[3] due to its common presence in hospital environments,[3,4] easy colonization in the oral cavity, and endotracheal tubes,[2,4] predisposing the infections. *P. aeruginosa* infection is more common in the old age group patients[12,24] We found the infection higher in the age group of patients who were more than 60 years of age (33.05%) which is in line with the study made by Ojha *et al*[23] (39.0%) and Shrestha *et al* (35.3%).[12] This could be due to decreased immunity, prolonged hospitalization, and other associated co-morbidities in these age groups.[2,4]

The current study reveals a concerning prevalence of multidrug-resistant (MDR) *P. aeruginosa* (53.71%) and ESBL producing strains (20.6%). These findings underscore the growing threat posed by antimicrobial resistance in *P. aeruginosa*, an opportunistic pathogen commonly implicated in healthcare-associated infections. The high MDR rate is consistent with previous studies from both developing and developed countries, where resistance in *P. aeruginosa* has been increasingly reported, particularly in nosocomial settings.[9,18,23,24,26]

Importantly, all ESBL producing isolates in our study were also multidrug-resistant, indicating a strong association between beta-lactamase production and resistance to other antibiotic classes.[5] This co-resistance pattern has been documented in prior research and is believed to result from the co-selection of resistance genes, often carried on mobile genetic elements such as plasmids and integrons.[6,23] The clinical implication is significant, as infections caused by ESBL producing MDR strains are associated with higher morbidity, mortality and treatment costs due to limited therapeutic options.[4,5,7]

Interestingly, the prevalence of ESBL producing *P. aeruginosa* was notably higher in outpatient samples, which may reflect community acquisition of resistant strains.[27] This observation contrasts with the traditional hospital-centric view of ESBL and MDR emergence but aligns with more recent evidence of resistance spreading into community settings, likely facilitated by overuse or misuse of antibiotics in outpatient care. This finding warrants further investigation and highlights

the need for robust antimicrobial stewardship programs beyond hospital environments.

In contrast, the MDR rate was higher in isolates obtained from inpatients, which is in agreement with previous literature demonstrating that prolonged hospital stays, invasive procedures, and intensive antibiotic exposure contribute to the selection and propagation of resistant organisms in clinical settings.[19,20,26] This reinforces the need for strict infection control measures and periodic surveillance to monitor resistance trends within hospitals.

The antimicrobial resistance patterns observed in *P. aeruginosa* isolates from our study highlight a concerning trend in the prevalence of resistant strains. These findings are consistent with global trends indicating increasing resistance of *P. aeruginosa* to commonly used antibiotics.[6,20,25] A study conducted in Nepal reported resistance rates of 59% to Piperacillin and 78.3% to Ceftazidime among *P. aeruginosa* isolates.[28] Similarly, resistance rates to fluoroquinolones and aminoglycosides were reported to be 43.4% and 36.3%, respectively, in the same study.

The relatively lower resistance rates observed in our study for carbapenems (Imipenem and Meropenem) and Piperacillin-Tazobactam suggest that these antibiotics might still be effective options for treating *P. aeruginosa* infections. However, the increasing resistance to beta-lactams[22,25,26] and aminoglycosides[24,26] is alarming and underscores the need for continuous surveillance and prudent antibiotic use.

The high resistance rates observed in our study could be attributed to several factors, including overuse and misuse of antibiotics,[4] inadequate infection control measures,[26] and the ability of *P. aeruginosa* to acquire resistance genes through horizontal gene transfer.[5] Additionally, the presence of resistance mechanisms such as beta-lactamases and efflux pumps contribute to the pathogen's ability to withstand antimicrobial agents.[5,6]

In conclusion, the findings of our study highlight the urgent need for implementing effective antimicrobial stewardship programs, enhancing infection control practices, and conducting regular surveillance to monitor resistance patterns. Further research is needed to explore the molecular mechanisms for underlying resistance and to develop strategies to combat the growing threat of antimicrobial resistance in *P. aeruginosa*.

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