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Molecular Characterization of Colistin Resistance in *Pseudomonas aeruginosa* Isolates: Insights from a Study in Bangladesh

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Abstract

Background: *Pseudomonas aeruginosa* is a major opportunistic pathogen responsible for severe hospital-acquired infections. Increasing antimicrobial resistance, particularly to colistin, which is the last-resort therapy for multidrug-resistant (MDR) Gram-negative bacteria, poses a critical challenge.

Objective: This study aimed to determine the prevalence, resistance profile, and colistin resistance genes among *P. aeruginosa* isolates from clinical samples.

Methodology: This cross-sectional study was conducted at the Department of Microbiology and Immunology at Dhaka Medical College Hospital (DMCH), Dhaka, Bangladesh. The research was conducted over one year, from January to December 2022. A total of 330 clinical specimens were subjected to culture and sensitivity testing. PCR screened colistin-resistant isolates for *pmrA*, *pmrB*, *pmrC*, *phoP*, and *phoQ* genes.

Results: Culture positivity was observed in 64.24% of samples, with wound swabs and pus showing the highest rate (72.41%), followed by endotracheal aspirates (70.00%). *P. aeruginosa* constituted 24.05% of isolates, predominantly from wound and urine samples. High resistance rates were noted for ciprofloxacin (88.23%), ceftazidime (84.31%), gentamicin (68.62%), and carbapenems (imipenem 56.86%, meropenem 60.78%). Colistin resistance was detected in 25.49% of isolates. Among these, 56.86% were MDR, 21.56% extensively drug-resistant (XDR), and 13.72% pandrug-resistant (PDR). The *pmrA* gene was most frequent (46.15%), followed by *pmrB* (38.46%), *pmrC* and *phoP* (30.76% each), and *phoQ* (23.07%).

Conclusions: The predominance of MDR and XDR *P. aeruginosa* underscores an urgent need for antimicrobial stewardship and surveillance. Detection of multiple regulatory genes in colistin-resistant isolates suggests complex molecular mechanisms requiring continued genomic monitoring.

Keywords: *Pseudomonas aeruginosa*; Molecular characterization; Bangladesh; Colistin resistance

Introduction

The increasing prevalence of multidrug-resistant (MDR) bacterial pathogens represents a major global health concern, limiting therapeutic options and leading to high morbidity and mortality rates [1]. Among these pathogens, *Pseudomonas aeruginosa* is particularly problematic due to its remarkable ability to acquire and express diverse resistance mechanisms, rendering many classes of antibiotics ineffective. This opportunistic pathogen is associated with severe infections in immunocompromised and critically ill patients, including ventilator-associated

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pneumonia, bloodstream infections, urinary tract infections, and wound infections [2].

With the diminishing efficacy of β-lactams, aminoglycosides, and fluoroquinolones, colistin (polymyxin E) has re-emerged as a last-line therapeutic option against MDR and XDR *P. aeruginosa* [3]. Colistin acts by disrupting the integrity of the bacterial outer membrane through binding to lipid A of lipopolysaccharides. However, increasing reports of colistin resistance have raised alarm, as resistance to this drug severely compromises treatment outcomes and leaves clinicians with few viable alternatives [4]. These resistance mechanisms have been reported in various Gram-negative microorganisms, including *Salmonella enterica*, *K. pneumoniae*, *A. baumannii*, *P. aeruginosa*, and *E. coli*. They are involved in the two-component system genes *phoP*/*phoQ* and *pmrA*/*pmrB* [5,6], PhoQ and PmrB proteins possess tyrosine kinase activity, which phosphorylates the regulator protein (PhoP or PmrA), activates the *pmrHFIJKLM* operon, and finally modifies the surface of bacteria by adding L-Ara4N or pEtN to lipid A [7]. PhoP/PhoQ is also regulated by the ColR/ColS and CprR/CprS systems. Mutations in these regulatory systems can lead to overexpression of PhoP/PhoQ in *P. aeruginosa* [8]. ParR/ParS is also involved in colistin resistance in *P. aeruginosa*, with upregulation of the LPS modification operon at sub-inhibitory concentrations of polymyxins [9]. The two-component systems found in *P. aeruginosa* are PhoP/PhoQ and PmrA/PmrB [10].

In this study, we investigated the amino acid substitution of PmrA-PmrB and PhoP-PhoQ in colistin-nonsusceptible *P. aeruginosa* (CNPA). Understanding the genetic basis of colistin resistance will aid in enhancing surveillance, promoting antimicrobial stewardship, and formulating strategies to curb the dissemination of resistant strains.

Materials and Methods

Study design and setting

This cross-sectional study was conducted in the Department of Microbiology and Immunology at Dhaka Medical College Hospital (DMCH), Dhaka, Bangladesh. The research was carried out over a year, from January to December 2022.

Isolation and Identification of *P. aeruginosa*

Clinical specimens, including wound swabs, urine, wound swabs, pus, tracheal aspirates, sputum, blood, and other body fluids, submitted to the Microbiology Laboratory at DMCH were processed for bacterial isolation. A total of 51 consecutive, non-duplicate isolates of *P. aeruginosa* were obtained from hospitalized patients during the study period. Phenotypic identification of *P. aeruginosa* was done by observing colony morphology on blood agar (white or cream-coloured, smooth to mucoid colonies, hemolytic), on MacConkey agar (generally form colourless colonies), Gram staining (gram negative bacilli), produces blue or green pigment and biochemical tests like-Oxidase test (positive), catalase tests (positive), TSI agar (butt-red, slant-red, no H_2S or gas production), urease production (negative), indole test (negative), motility (motile) und citrate utilization test (positive). Growth at 42°C on agar was also done for confirmation of *P. aeruginasa*. For quality control, the reference strain *P. aeruginosa ATCC 27853* was used during culture, biochemical testing, and phenotypic confirmation of clinical isolates. The study received ethical approval from the institutional review board of Dhaka Medical College, and written informed consent was secured from all participating patients.

Antimicrobial Susceptibility Testing

The antimicrobial susceptibility profiles of the *Klebsiella pneumoniae* isolates were determined using the standard Kirby-Bauer disk diffusion method on Mueller-Hinton agar (Oxoid Ltd., UK) [11]. A range of commercially prepared antibiotic discs was employed, including Amikacin (30 µg), Aztreonam (30 µg), Ceftazidime (30 µg), Cefepime (30 µg), Colistin (10 µg), Ciprofloxacin (5 µg), Gentamicin (10 µg), Imipenem (10 µg), Meropenem (10 µg), Fosfomycin (200µg), Piperacillin-tazobactam (100 µg/10 µg). Interpretation of inhibition zones was carried out under guidelines established by the Clinical and Laboratory Standards Institute (CLSI) and the European Committee on Antimicrobial Susceptibility Testing (EUCAST). To identify ESBL-producing strains, the double-disk synergy test was applied. Furthermore, isolates were categorized as multidrug-resistant (MDR), extensively drug-resistant (XDR), or pandrug-resistant (PDR) based on criteria outlined by the Centers for Disease Control and Prevention (CDC) [12,13].

MDR

Multidrug-resistant (MDR) isolates were defined as those that showed resistance to three or more classes of antipseudomonal agents (carbapenems, fluoroquinolones, penicillins/cephalosporins, and aminoglycosides) [13].

ESBL

ESBL production in all of the isolates was detected by the double disk synergy test as described by Jarlier [14]. Synergy was determined between a disk of amoxiclav (20µg amoxicillin and 10µg clavulanic acid) and a 30µg disk of each third-generation cephalosporin. The test antibiotic was placed 20 mm apart on a lawn culture of the isolate under test on Mueller-Hinton agar. The test organism was considered to produce ESBL if the zone size around the antibiotic disk increased towards the amoxiclav disk. This criterion also fulfills the CLSI guidelines [15]. This increase occurs because the clavulanic acid present in the amoxiclav disk inactivates the ESBL produced by the test organism [16].

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Detection of colistin-resistant genes by Polymerase Chain Reaction (PCR)

Conventional polymerase chain reaction (PCR) was used to detect Colistin-resistant genes (*pmr*A, *pmr*B, *Pmr*C, *pho*P, *pho*Q) in *P. aeruginosa* isolates. Genomic DNA was extracted using the boiling method. Amplified PCR products were visualized by agarose gel electrophoresis to confirm the presence of target genes.

Data analysis

All collected data were analyzed using IBM SPSS Statistics version 23. Categorical variables were summarized as frequencies and percentages. Graphs and visualizations were generated using the matplotlib library in Python.

Result

A total of 330 clinical samples were processed during the study period. Out of these, 212 (64.24%) yielded positive microbial growth. The culture positivity rate varied notably across sample types. The highest positivity was observed in wound swabs and pus samples (72.41%), followed by endotracheal aspirates (70.00%), and urine samples (58.33%). The lowest rates were found in blood (45.00%) and sputum (46.66%) samples (Table 1).

A Chi-square test for independence was performed to assess whether the culture positivity rate was associated with sample type. The analysis revealed a statistically significant association between sample type and culture positivity ($\chi^2 = 13.42$, df = 4, $p = 0.009$).

Among the 212 culture-positive samples, 51 (24.05%) were *P. aeruginosa* (Table 2), of which 45 (88.23%) were resistant to ciprofloxacin, 43 (84.31%) were resistant to ceftazidime, 20 (39.21%) were resistant to piperacillin-Tazobactum, and 13 (25.49%) were resistant to colistin (Figure 1).

Among the 51 *P. aeruginosa* isolates, 29 (56.9%) were multidrug-resistant (MDR), 11 (21.6%) were extensively drug-resistant (XDR), and 7 (13.7%) were pan-drug resistant (PDR). A chi-square goodness-of-fit test demonstrated that the distribution of resistance patterns was significantly different from equal proportions ($\chi^2 = 17.53$, df = 2, $p < 0.001$), with MDR being the predominant phenotype (Table 3).

Here, *pmr*A, *pmr*B, *pmr*C, *pho*P, and *pho*Q genes from different isolates of colistin-resistant *P. aeruginosa* were analyzed by PCR. Among 13 colistin-resistant isolates, 6 (46.15%) were positive for *pmr*A, 5 (38.46%) for *pmr*B, 4 (30.76%) for *pmr*C, 4 (30.76%) for *pho*P, and 3 (23.07%) for *pho*Q gene (Table 4).

Table 1: Culture positivity from different clinical samples (N = 330).

Sample Type	Total Samples (n)	Culture Positive (n)	Culture Positive (%)
Wound swab and pus	145	105	72.41
Urine	120	70	58.33
Endotracheal aspirate	30	21	70.00
Blood	20	9	45.00
Sputum	15	7	46.66
Total	**330**	**212**	**64.24**

Differences between the growth rate of various clinical samples are statistically significant (p<0.0001).

Table 2: Distribution of organisms isolated from different samples by biochemical tests (N = 212).

Organisms	WS & Pus (105) n (%)	Urine (70) n (%)	ETA (21) n (%)	Blood (09) n (%)	Sputum (07) n (%)	Total (212) N (%)
P. aeruginosa	35 (33.33)	12 (17.14)	03 (14.28)	01 (11.11)	0 (0.00)	51 (24.05)
E. coli	16 (15.23)	41 (58.57)	0 (0.00)	0 (0.00)	0 (0.00)	57 (26.88)
Klebsiella spp	16 (15.23)	03 (4.28)	06 (28.57)	0 (0.00)	05 (71.42)	30 (14.15)
Acinetobacter spp	10 (9.52)	04 (5.71)	07 (33.33)	02 (22.22)	02 (28.57)	25 (11.79)
Enterobacter spp	06 (5.71)	03 (4.28)	03 (14.28)	02 (22.22)	0 (0.00)	14 (6.60)
Proteus mirabilis	05 (4.76)	02 (2.85)	0 (0.00)	0 (0.00)	0 (0.00)	07 (3.30)
Proteus vulgaris	01 (0.95)	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)	01 (0.47)
Salmonella spp	0 (0.00)	0 (0.00)	0 (0.00)	03 (33.33)	0 (0.00)	03 (1.41)
Staph. aureus	14 (13.33)	02 (2.85)	02 (9.52)	01 (11.11)	0 (0.00)	19 (8.96)
Other Pseudomonas spp.	02 (1.90)	01 (1.42)	0 (0.00)	0 (0.00)	0 (0.00)	03 (1.41)
Coagulase-negative Staphylococcus	0 (0.00)	02 (2.85)	0 (0.00)	0 (0.00)	0 (0.00)	02 (0.94)

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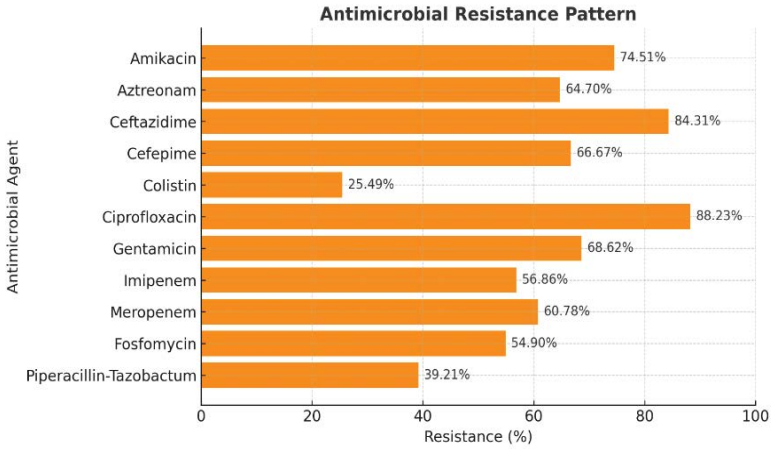

Figure 1: Antibiotic resistance pattern of isolated *P. aeruginosa* by disc diffusion method (N = 51).

Table 3: Types of antibiotic resistance patterns among the isolated *P. aeruginosa* (N = 51).

Types of resistance	n (%)
MDR	29 (56.86)
XDR	11 (21.56)
PDR	07 (13.72)

Table 4: Distribution of *pmr*A, *pmr*B, *pmr*C, *pho*P, *and pho*Q genes among the colistin-resistant *P. aeruginosa* by PCR (N = 13).

Samples	*pmr* A n (%)	*pmr* B n (%)	*pmr* C n (%)	*pho* P n (%)	*pho* Q n (%)
Wound swab & Pus (N = 8)	4 (50.00)	2 (25.00)	1 (12.50)	2 (25.00)	1 (12.5)
Urine (N = 2)	0 (0.00)	1 (50.00)	2 (100.00)	1 (50.00)	0 (0.00)
ETA (N = 2)	1 (50.00)	2 (100.00)	0 (0.00)	1 (50.00)	1 (50.00)
Blood (N = 1)	1 (100.00)	0 (0.00)	1 (100.00)	0 (0.00)	1 (100.00)
Sputum (N = 0)	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)	0 (0.00)
Total (N = 13)	6 (46.15)	5 (38.46)	4 (30.76)	4 (30.76)	3 (23.07)

Discussion

330 clinical samples were analyzed in the present study to determine the distribution and antimicrobial resistance profile of *Pseudomonas aeruginosa*, with particular emphasis on colistin resistance and associated regulatory genes. The findings revealed a concerning positivity rate of 64.24% for bacterial growth across the clinical samples, pointing to a considerable infection burden in the studied population, which is consistent with the findings of Munny et al. [17]. Notably, wound swabs and pus samples exhibited the highest culture positivity at 72.41%, followed closely by endotracheal aspirates, which showed a 70.00% positivity rate which is similar to the study of Siddiqua et al. [18]. This trend aligns with *P. aeruginosa*'s known preference for moist environments and its opportunistic role as a pathogen, particularly among vulnerable patients, including those with burns, chronic wounds, and conditions leading to ventilator-associated pneumonia.

Among the culture-positive isolates, *P. aeruginosa* accounted for 24.05%, representing a significant proportion of the Gram-negative isolates. This prevalence aligns with previous reports highlighting *P. aeruginosa* as a major nosocomial pathogen responsible for a wide spectrum of infections, including wound, urinary, respiratory, and bloodstream infections. The relatively higher isolation rate from wound swabs and pus (33.33%) compared to other samples suggests that *P. aeruginosa* remains a predominant cause of wound and soft-tissue infections due to its intrinsic resistance mechanisms and its ability to form biofilms. About 90% of the *P. aeruginosa* isolates of this study were obtained from only three important specimens, e.g., wound swab, pus, and urine. Similar results had been obtained in India, reported by Andhale et. al. [19] and Pathi et. al. [20] in different studies.

Antimicrobial susceptibility testing revealed alarmingly high resistance rates among *P. aeruginosa* isolates. Ciprofloxacin resistance was most frequent (88.23%), followed by ceftazidime (84.31%), gentamicin (68.62%), cefepime (66.67%), and aztreonam (64.70%). Resistance to carbapenems, namely imipenem (56.86%) and meropenem (60.78%), further underscores the limited therapeutic options available for treating these infections. Gill et al. [21] reported that 70% *P. aeruginosa* were resistant to ceftazidime and 80% were resistant to amikacin, which is in agreement with the present study. Bhatt et al. [22] in India reported 77% resistance to ceftazidime which is also almost similar to the present study.

Although colistin resistance was relatively lower (25.49%), this is still clinically concerning, given that colistin

is considered a last-resort antibiotic against multidrug-resistant (MDR) Gram-negative bacteria. Similar trends have been observed in Abd El-Baky et al. in Egypt (21.3%) [23].

More than half (56.86%) of the isolates exhibited a multidrug-resistant (MDR) phenotype, while 21.56% were extensively drug-resistant (XDR) and 13.72% were pandrug-resistant (PDR). Gill et al. [21] reported that 50% isolates were multidrug resistant, which is almost similar to the findings in the present study (21). Owlia et al. [24] in Iran reported that 54.5% of isolates were multidrug resistant and 33% were extensively resistant, which are also similar to the findings of this study. The predominance of MDR strains indicates a significant threat to effective antimicrobial therapy and highlights the urgent need for antimicrobial stewardship and infection control strategies. The emergence of XDR and PDR strains further emphasizes the adaptability of *P. aeruginosa* in acquiring multiple resistance determinants.

Molecular analysis of the 13 colistin-resistant *P. aeruginosa* isolates demonstrated the presence of various two-component regulatory system genes associated with colistin resistance, including *pmrA*, *pmrB*, *pmrC*, *phoP*, and *phoQ*. Among these, *pmrA* was the most frequently detected (46.15%), followed by *pmrB* (38.46%), *pmrC* and *phoP* (30.76% each), and *phoQ* (23.07%). Mostofa et al. found that 61.53% colistin-resistant *P. aeruginosa* were positive for *pmr*A, 38.5% for *pmr*B, 38.46% for *pho*P and 23.07% for *pho*Q [25]. Goli et al. [26] in Iran showed that the mutation of *pmr*B gene and expression change of *pmr*AB or *pho*PQ have occurred in colistin-resistant isolates [26]. In *P. aeruginosa*, mutations occurring in two-component regulatory systems are the primary mechanisms attributed to the development of resistance against colistin, according to Olaitan et al. [27]. These genes are known to mediate lipid A modification of lipopolysaccharides, leading to reduced colistin binding affinity and resistance. The detection of multiple resistance-associated genes within individual isolates suggests a complex regulatory interplay that contributes to colistin resistance. Similar findings have been reported in studies where mutations or overexpression of these genes were correlated with resistance phenotypes in *P. aeruginosa*.

Conclusion

The present study provides a comprehensive overview of the prevalence, antimicrobial resistance patterns, and molecular mechanisms underlying colistin resistance in *Pseudomonas aeruginosa* isolated from diverse clinical samples. The high culture positivity rate and the predominance of *P. aeruginosa* among Gram-negative isolates highlight its continued role as a major nosocomial pathogen, particularly in wound and soft-tissue infections. The alarming resistance rates to commonly used antibiotics, including fluoroquinolones, β-lactams, aminoglycosides, and carbapenems, reflect the growing challenge in managing *P. aeruginosa* infections within clinical settings.

Although the observed rate of colistin resistance was comparatively lower, its presence indicates significant clinical concern, as it is the last-line therapeutic option. The detection of multidrug-resistant, extensively drug-resistant, and pandrug-resistant phenotypes underscores the urgent need for enhanced antimicrobial stewardship and effective infection control measures. Molecular characterization further revealed the involvement of two-component regulatory system genes, particularly *pmrA*, *pmrB*, *pmrC*, *phoP*, and *phoQ*, in mediating colistin resistance, which is consistent with global reports implicating these genes in lipid A modification and reduced colistin susceptibility.

Overall, these findings emphasize the adaptive potential of *P. aeruginosa* and the necessity of continuous surveillance, rational antibiotic use, and molecular monitoring to mitigate the spread of resistant strains and preserve the efficacy of existing antimicrobial agents.

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