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Antimicrobial Resistance Patterns and Phenotypic Profiles of Pseudomonas aeruginosa Isolated from Clinical Samples

Sefat-E-Rabbi Eva[1], S M Shamsuzzaman[2], Shatabdi Mallick[3], Moshabha Akter Anika[4], Ziaul Hasan[5]

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*Corresponding author

ABSTRACT

Background: Pseudomonas aeruginosa is a significant concern among hospital-acquired pathogens, due to the growing resistance to antibiotics, particularly colistin and carbapenems. The goal of this study was to determine the resistances and phenotypes of P. aeruginosa strains obtained from clinical samples. **Methods & Materials:** This cross-sectional study, conducted at the Department of Microbiology, Dhaka Medical College in 2019, analyzed 350 clinical samples using standard microbiological and molecular techniques to identify Pseudomonas aeruginosa and assess antimicrobial resistance. Susceptibility testing was performed via disc diffusion and MIC methods, with PCR used to detect resistance genes. In vivo efficacy of various antibiotic regimens was evaluated in mice models. Data were analyzed using SPSS version 26. **Results:** Among 236 bacterial isolates, P. aeruginosa was found to constitute 26.69% (n=63), mostly from wound swabs (66.67%). High levels of resistance were found to ceftriaxone (82.54%), ceftazidime (74.60%), and amikacin (74.60%). The incidence of multi-drug resistance was found to be 61.90%, of which 19.05% had extensive drug resistance, while 6.35% had pan-resistance. The overall colistin resistance was found to be 19.05%, while the detection of both pmrC and phoP genes was found to occur in 50% of colistin-resistant strains. Carbapenemase was detected in 85.71% of imipenem-resistant strains. **Conclusion:** High levels of multidrug, extensive, and pan-resistance in P. aeruginosa isolates were alarming. The colistin-imipenem combination appeared to possess encouraging effectiveness against resistant isolates, justifying assessment for therapeutic use.

Keywords: *Pseudomonas aeruginosa, Antimicrobial resistance, Colistin resistance, Carbapenemase.*

1. *Assistant Professor, Department of Microbiology, Sir Salimullah Medical College, Dhaka, Bangladesh (ORCID: 0009-0009-1929-6511)*
2. *Professor, Department of Microbiology, Dhaka Medical College, Dhaka, Bangladesh*
3. *Assistant Professor, Department of Microbiology, Khulna Medical College, Khulna, Bangladesh*
4. *Assistant Surgeon, OSD(DGHS), Dhaka, Bangladesh*
5. *Consultant, National Institute of Traumatology and Orthopaedic Rehabilitation, Dhaka, Bangladesh*

INTRODUCTION

Pseudomonas aeruginosa is a Gram-negative opportunistic pathogen that results in morbidity and fatality in nosocomial related infections, particularly in immunocompromised patients, and at intensive care units [1]. The Pseudomonas species that is widely distributed possess a high level of adaptability and intrinsic resistance to a large range of drugs. Pseudomonas has a capability of forming biofilms, which helps it to survive in hospital conditions [2]. The adaptability of this versatile pathogen to survive in the low nutrient environment makes it stand out and be a significant cause of nosocomial infections in many countries across the world [3]. The world has been experiencing a significant problem in treatment, the development of multi-drug resistant (MDR), extensively drug-resistant (XDR), and pan-drug-resistant (PDR) strains of P. aeruginosa, which in turn have been increasing the cost of healthcare as managing the infection of such bacteria is becoming more difficult [4]. The resistance to carbapenems as the last-resort antibiotics in the infection caused by Pseudomonadales has been increasing due to the evolution of carbapenemase-producing bacteria through gene transfer and chromosomal mutations that altered the susceptibility of carbapenems in the targeted bacteria because of the changes that occurred in the cell wall permeability of the bacteria [5]. Also rediscovered in response to the increased cases of resistance, colistin, a polymyxin antibiotic, has been named as a prime drug against MDR/Xenodrug-resistant (XDR) Gram-negative pathogens [6,7]. Despite this, there has been increase in resistance to colistin in P. aeruginosa, which is due to the mutations in two-component regulatory systems as pmrAB and phoPQ, resulting in lipopolysaccharides changes, which inhibits the binding of antibiotics [8]. Even more importantly, the discovery of plasmid-mediated colistin resistance genes (mcr-1 through mcr-5) in Enterobacteriaceae has been a cause of concern, whereas in Pseudomonas, their functions are ill-defined [9]. Antimicrobial resistance is also a very high issue in resource limited settings such as the South Asian region because of numerous factors including the inappropriate use of antimicrobials, poor infection control practices, and the lack of competence in diagnosis [10]. It is known that Bangladesh is experiencing dramatic issues in terms of hospital-acquired infections as well as antimicrobial resistance, yet the information about the resistance profile and the mechanism of P. aeruginosa is horrendously scarce and untapped [11]. There is increasing resistance to the use of the available antimicrobials, and the inability to respond to treatment is accumulating. Combination antimicrobial therapy has taken a fresh significance in terms of its suitability in combating any resistant strain and its suitability to prevent the development of resistance. The objective of the research is to determine the patterns of antimicrobial resistance of P. aeruginosa of clinical origin, resistance phenotyping, including MDR, XDR, and PDR, genes involved in colistin resistance, and combined antibiotics against MDR.

METHODS & MATERIALS

The current cross-sectional research was conducted in the Department of Microbiology, Dhaka Medical College, between January and December 2019 on 236 samples of culture-positive bacterial samples to detect P. aeruginosa. The samples taken included wound swabs, urine, endotracheal aspirates (ETA) and blood samples of adult inpatients and ICU patients, which were taken aseptically. The data gathered such as the demographics was captured on a pre-designed sheet and the data was processed and analysed using Microsoft Excel. The culture and sensitivity tests were done under the standard protocols

of microbiological tests using media like blood agar, MacConkey agar and Mueller-Hinton agar. The identification of P. aeruginosa and other Gram-negative organisms was performed based on the study of the colony features, Gram staining, and biochemical analysis. The susceptibility tests were done by modified Kirby- Bauer disk diffusion for antimicrobial susceptibility tests, and through agar dilution minimum inhibitory concentration of colistin resistance was determined. Validation was done by using control strains (E. coli ATCC 25922). Producers of extended-spectrum beta-lactamase, carbapenemase and Metallo beta-lactamase were identified using the double-disk synergy, combined disk and modified Hodge tests. Combination studies of antibiotic combinations such as imipenem, colistin and amikacin were evaluated using agar dilution and FICI values were read. Detection of resistance genes mcr-1 through mcr-5, pmrAB, C, phoPQ was done using Polymerase chain reaction where DNA was extracted using E. coli pellets, amplified using thermal cyclers and investigated using agarose gel electrophoresis. To determine homology in the sequenced E. coli strains, the sequenced strains were validated and subsequently confirmed through the use of BLAST analysis. In vivo experiments were carried out to investigate the in vivo emergence resistances in which Swiss Albino mice were exposed to resistant strains. Survivability was also monitored, and microbicidal clearance after the therapy was done by the blood culture. DMC provided ethical clearance, and the patients were kept confidential. Data analysis and processing were performed using SPSS software v 26.

RESULTS

Table I shows that *P. aeruginosa* was the most prevalent, constituting 26.69% (n=63). *Escherichia coli* was the next, making up 19.07% isolates, while *Klebsiella pneumoniae* was the third most prevalent, constituting 14.41%. Gram-positive bacteria, *Staphylococcus aureus* being the most preponderant, constituting 10.59% isolates, while other gram-negative organisms included *Enterobacter cloacae* (7.63%) and *Acinetobacter baumannii* (6.36%), respectively (*Table I*).

Table I
Distribution of organisms isolated from different samples (*n*=236).

Isolated Bacteria	n (%)
P. aeruginosa	63 (26.69)
Non-aeruginosa pseudomonas	4 (1.69)
E. coli	45 (19.07)
K. pneumoniae	34 (14.41)
K. oxytoca	2 (0.85)
P. mirabilis	11 (4.66)
P. vulgaris	3 (1.27)
Acinetobacter baumannii	15 (6.36)
C. freundii	4 (1.69)
C. koseri	2 (0.85)
E. cloacae	18 (7.63)
E. aerogenes	6 (2.54)
S. typhimurium	2 (0.85)
S. aureus	25 (10.59)
Coagulase-negative staphylococci	2 (0.85)

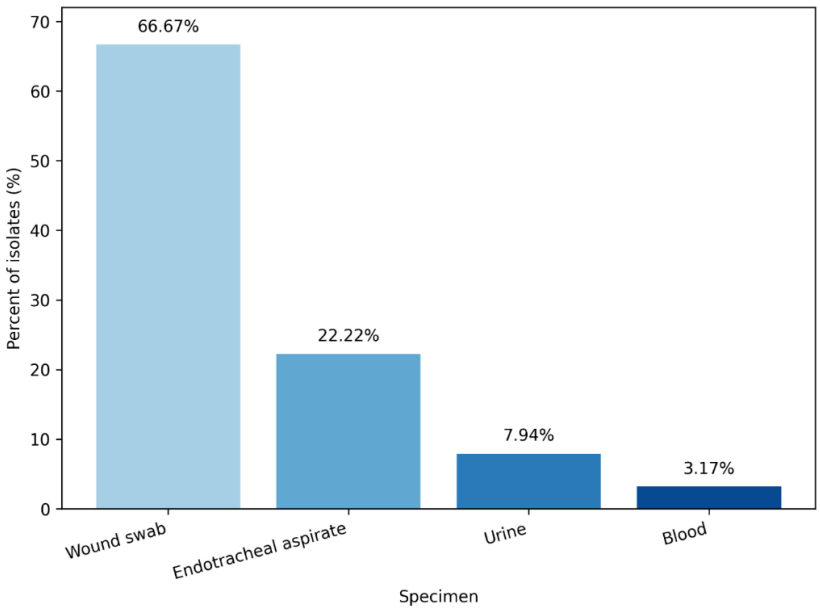

Figure 1 Distribution of *P. aeruginosa* isolates by specimen (*n*=63).

Figure 1 reveals that the isolates of *P. aeruginosa* were based on the type of samples, which gave dominance in wound swab samples, accounting for 66.67% of the isolates. The isolates from endotracheal aspirates comprised 22.22%, which is quite high, showing its prevalence in the respiratory tract, especially within ICUs. Urinary tract infection isolates comprised 7.94%, while blood isolates comprised the least, which is 3.17%.

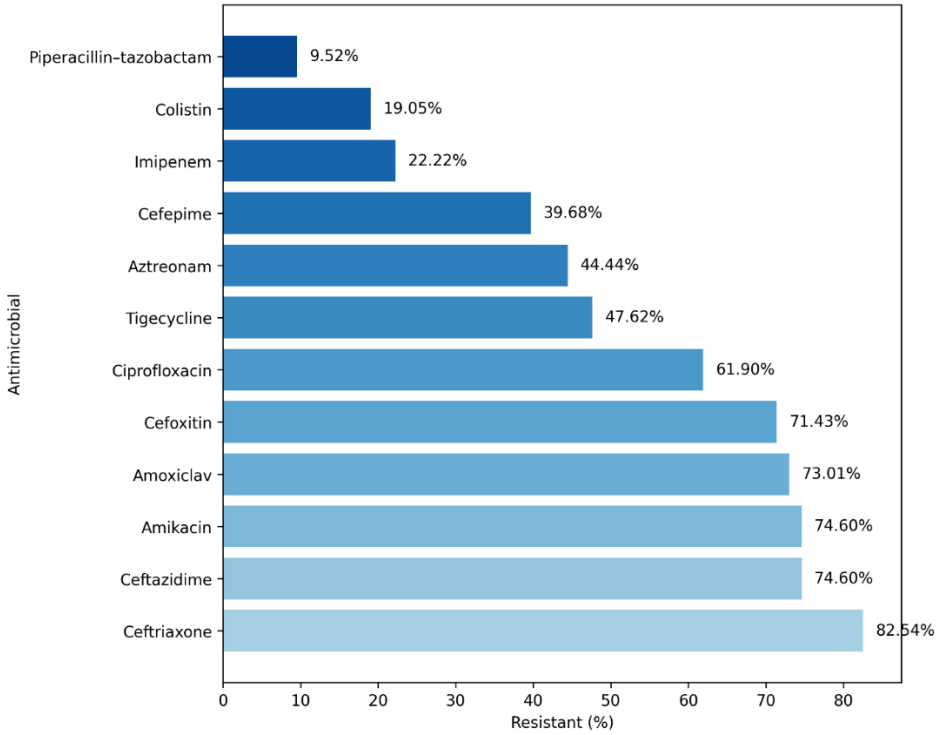

Figure 2 Antimicrobial resistance pattern in *P. aeruginosa* (*n*=63).

Figure 2 exhibits very high levels of antimicrobial resistance among the isolates of *P. aeruginosa*. Resistance to ceftriaxone showed a high frequency of 82.54% among isolates, followed by ceftazidime and amikacin, exhibiting resistances of both 74.60% respectively. Carbapenems, specifically imipenem, showed resistance among 22.22% isolates, and colistin among 19.05% isolates. It is notable that the lowest level of resistance was shown by piperacillin and tazobactam among all agents tested, with a value of 9.52%.

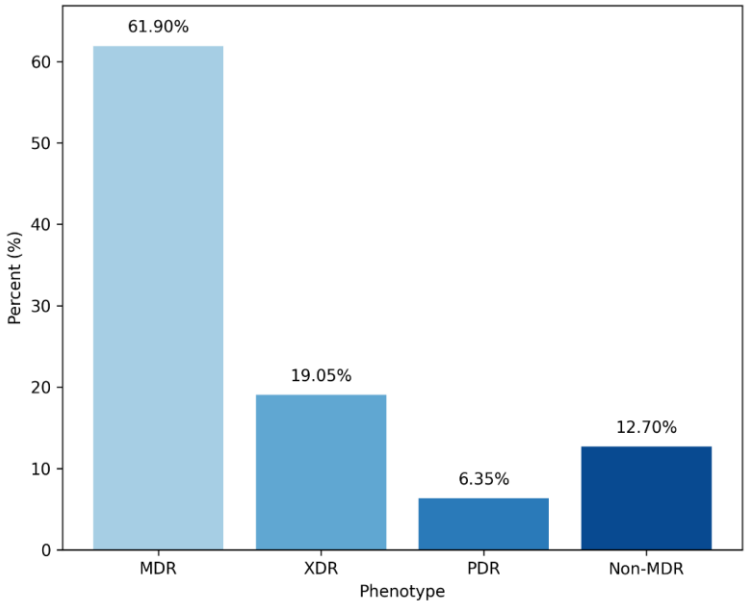

Figure 3 Resistance phenotype categories in *P. aeruginosa* (*n*=63).

Figure 3 illustrates that 61.90% of the isolates belonged to the MDR category, showing non-susceptibility to at least one drug in three or more classes of antimicrobials. The ExDR isolates contributed 19.05%. The pan-drug-resistant isolates showed resistance to all the tested drugs and contributed 6.35%. Only 12.70% belonged to the non-MDR category.

Table II indicates that among the 12 colistin-resistant *P. aeruginosa* isolates, wound swabs accounted for the highest proportion (58.33%), followed by endotracheal aspirates (25%) and urine (16.67%). All colistin-resistant isolates exhibited 100% co-resistance to ceftriaxone and ceftazidime, while resistance to amoxiclav was 91.67%. High levels of co-resistance were also observed with ciprofloxacin (83.33%), amikacin and cefoxitin (75%), and imipenem (58.33%). Moderate resistance was noted for cefepime and aztreonam (50% each), with lower rates for tigecycline (41.67%) and piperacillin–tazobactam (25%), reflecting a concerning multi-drug resistance pattern.

Table II
Colistin resistance in *P. aeruginosa*, distribution and co-resistance (*n*=12).

A. Distribution by specimen, plus specimen-level prevalence	
Specimen	**Colistin-resistant isolates, n (%)**
Wound swab	7 (58.33)
Endotracheal aspirate	3 (25.00)
Urine	2 (16.67)
Blood	0 (0.00)
B. Co-resistance pattern among colistin-resistant isolates (*n*=12)	
Antimicrobial	**Resistant, n (%)**
Ceftriaxone	12 (100.00)
Ceftazidime	12 (100.00)
Amoxiclav	11 (91.67)
Ciprofloxacin	10 (83.33)
Amikacin	9 (75.00)
Cefoxitin	9 (75.00)
Imipenem	7 (58.33)
Cefepime	6 (50.00)
Aztreonam	6 (50.00)
Tigecycline	5 (41.67)
Piperacillin–tazobactam	3 (25.00)

Table III depicts that the production of Extended-spectrum β-lactamases (ESBLs) was observed in 13 isolates of *P. aeruginosa* (20.63%). The highest proportion of ESBL-producing isolates was isolated from the samples of patient wound swabs (19%), and one from the endotracheal aspirates (1.5%).

Table III
ESBL phenotype in *P. aeruginosa* (*n*=63).

Specimen	ESBL-positive isolates, n (%)
Wound swab	12 (19)
Endotracheal aspirate	1 (1.5)
Urine	0 (0)
Blood	0 (0)

Table IV demonstrates that the total of 14 imipenem-resistant isolates of *P. aeruginosa*, the highest percentage of specimens was from the swabs of the wounds (78.57%), and the second highest was from the endotracheal aspirates (14.29%). Results from the carbapenemase test using the phenotypic technique showed that the highest positivity was seen in the combined disc test (85.71%), followed by the double-disc synergy test (64.29%), and then the modified Hodge test (28.57%). According to the imipenem MIC distribution, half of the isolates (50%) had a MIC of 32 µg/ml, while a significant portion showed high-level resistance with MIC values ≥128 µg/ml in 28.6% of cases, indicating significantly lower susceptibility among the isolates under study.

Table IV
Imipenem resistance and carbapenemase phenotype in *P. aeruginosa* (*n*=14).

A. Distribution of imipenem-resistant isolates	
Specimen	**Imipenem-resistant isolates, n (%)**
Wound swab	11 (78.57)
Endotracheal aspirate	2 (14.29)
Urine	1 (7.14)
Blood	0 (0.00)
B. Phenotypic carbapenemase detection among imipenem-resistant isolates (*n*=14)	
Method	**Positive, n (%)**
Combined disc assay	12 (85.71)
Double disc synergy test	9 (64.29)
Modified Hodge test	4 (28.57)
C. Imipenem MIC distribution (*n*=14)	
Imipenem MIC (µg/ml)	**n (%)**
≥256	2 (14.29)
128	2 (14.29)
64	1 (7.14)
32	7 (50.00)
16	1 (7.14)
8	1 (7.14)

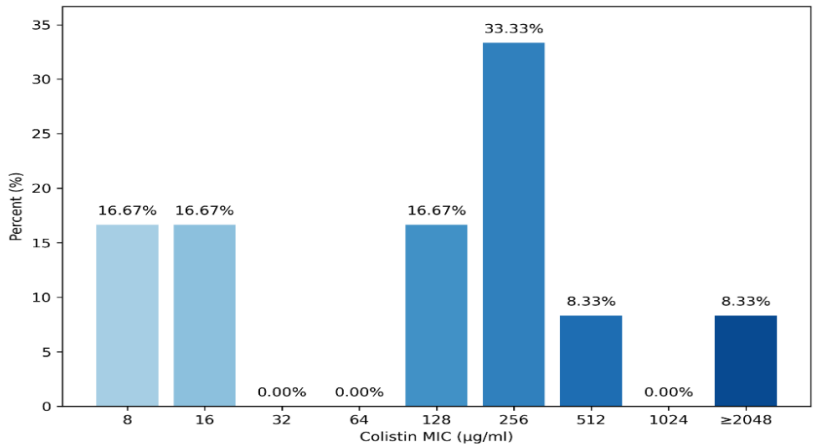

Figure 4 (A) Colistin MIC distribution (*n*=12).

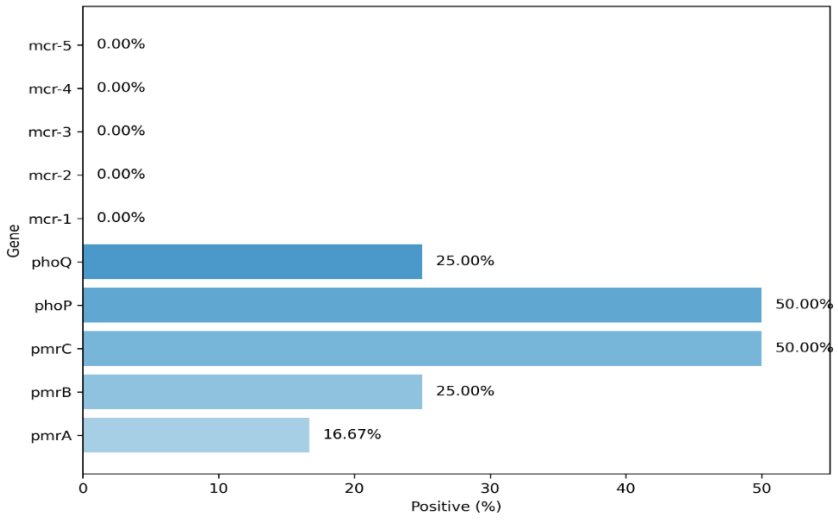

Figure 4 (B) PCR detection of resistance-associated genes among colistin-resistant isolates (*n*=12).

Figure 4 depicts the distribution of colistin MIC among the resistant isolates showed that there is heterogeneity, with 33.33% having an MIC of 256 µg/ml. There is one isolate that showed high resistance, with an MIC of >2048 µg/ml. Characterization of the resistant isolates showed that the genes pmrC and phoP were present in 50%, while the pmrB and phoQ genes were present in 25%, and the pmrA gene was present in 16.67% of the isolates that showed resistance to colistin.

Table V shows that colistin-imipenem displayed 100% synergy in vitro with a median FICI of 0.38, as well as complete in vivo microbial eradication. The imipenem-amikacin pair indicated 75% synergy (median FICI = 0.50) with complete in vivo efficacy. On the contrary, the results of colistin-amikacin indicated mostly indifferent interactions (75%) with a median FICI of 2.00 and a lack of in vivo efficacy. These results show that the imipenem/colistin pair has considerably higher therapeutic value against resistant strains of *P. aeruginosa*.

Table V
Combination efficacy against resistant *P. aeruginosa*, in vitro and in vivo summary.

Combination	Agar dilution MIC reduction, n (%) (n=4)	Checkerboard outcome, n (%) (n=4)	FICI median (range)	In vivo synergy (%)	In vivo blood culture negative, n=5, n (%)
Colistin + Imipenem	8-fold: 2 (50.00), 4-fold: 2 (50.00)	Synergy: 4 (100.00)	0.38 (0.25-0.50)	100	5 (100)
Imipenem + Amikacin	4-fold: 3 (75.00), 2-fold: 1 (25.00)	Synergy: 3 (75.00), Additive: 1 (25.00)	0.50 (0.50-1.00)	100	5 (100)
Colistin + Amikacin	2-fold: 1 (25.00), none: 3 (75.00)	Additive: 1 (25.00), Indifferent: 3 (75.00)	2.00 (1.00-2.00)	0	0 (0)

DISCUSSION

This paper presented thorough information about the pattern of antibiotic resistance and the phenotypic features of P. aeruginosa in Bangladesh. P. aeruginosa (26.69) was second in the list of clinical isolates because it is commonly known to be among the most common nosocomial infections found in healthcare facilities globally [12]. The high rate of isolates due to wound infections (66.67) goes hand in hand with its inherent virulence factor of colonization of injured epithelial surfaces, especially in surgical patients and persons with persistent wounds [13]. The resistance rates against the first-line antibiotics were surprisingly high with 82.54% resistant to ceftriaxone and 74.60% to ceftazidine. These levels of resistance are way higher in a report by Ranjar et al. [14]. But this should be the case, as the antimicrobial stewardship programs are not yet optimal in the majority of South Asian clinical facilities [15]. This trend is supported by the prevalence of healthcare settings that promote MDR, XDR and PDR phenotypes. It is also important to note that carbapenem resistance, which is also identified in the study as present in 22.22% of isolates is of particular concern in terms of therapeutic implications, since carbapenems were traditionally viewed as the last-line agents in the treatment of complicated Pseudomonas infections [16]. The phenotypic carbapenemase test yielded the expected results that only 85.71% of imipenem-resistant strains had enzyme production that is a strong indication that enzymatic mechanisms rather than impermeability/efflux mechanisms are the dominant mechanisms of resistance [6]. The variation in the ranges of distribution (16≥256 µg/ml) could either be due to fluctuating expression levels of the carbapenemase-producing gene or could also be due to the fact that the level of colistin resistance of 19.05% is a cause of concern due to the significance of the drug being a last resort drug in the presence of MDR and XDR gram negatives [17]. These are bigger than the results of Biswas et al., which is in line with the increasing figures of the countries where colistin is a leading consumer [18]. Genus level was characterized by chromosomal mutations in two-component systems of regulation, particularly in the genes that encode pmrC and PhoP (50% of the isolates positive) that mediate the alteration of lipopolysaccharides and thus reduces the affinity of colistin binding [19]. It is clear that the isolates tested negative in the mcr gene and this could mean that the colistin resistance in this area could be chromosome-based and that the possibility of horizontal transmission could be reduced compared to the mcr gene-carrying Enterobacteriaceae [20]. The co-resistance between the colistin-resistant isolates, e.g. 100% resistance to cephalosporins, and 58.33% resistance to carbapenems is very high, which is a dramatic decrease in the available chemotherapeutic options [21]. Instead, the fact that the piperacillin-tazobactam that remained 75 per cent susceptible in colistin-resistant isolates, points to efficacy, which has to be confirmed by the clinical outcomes studies and then definitive guidance can be provided [22]. Combination therapy with antibiotics was also a productive field, with colistin-imipenem being very synergistic (100% in vitro combination with 50% median FICI of 0.38) and full microbiologic regeneration of mice models. The explanation of this is probably due to the fact that colistin generates the degradation of outer membrane integrity since now imipenem can get in and inhibit cell wall synthesis as a mechanism of action [23]. It is remarkable that their profile of stronger combination is in comparison with colistin-amikacin (which is largely non-selective) creating rationale in methodologically-based combination therapy strategies. It is important to note that, on such data, such colistin-carbapenem combination therapy regimens must be investigated in clinical practice in infections caused by the susceptible or provisionally resistant strains, etc. [24].

LIMITATIONS

The study could be limited in terms of generalization because only one center was studied and the study is based on identification of carbapenemase using the phenotypic technique and does not involve the molecular examination of the beta-lactamase genes. The information in animal models would also need confirmation.

CONCLUSION

P. aeruginosa isolates were found to have exceptionally high proportions in relation to multidrug resistance, extensively drug-resistance, pan-drug resistance, high colistin resistance, and carbapenem resistance, effected mostly because of chromosome mutation and not plasmid-located genes. Colistin/imipenem combination exhibited synergistic interactions in vitro and was completely effective in vivo, and this would be useful in designing an effective treatment regimen in case of resistant isolates. These findings highlight the importance of proper antimicrobial use/utilization, strict infection and conscientiousness in selection of combination regimens in the management of severe cases of P. aeruginosa infection in resource- constrained settings.

RECOMMENDATIONS

Multi-center surveillance studies based on whole-genome sequencing should be employed in future studies that attempt to understand how resistance spreads. Conversely, the prospective studies will play an imperative role in the establishment of the most suitable treatment modalities in P. aeruginosa-resistant patients.

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CONFLICT OF INTEREST

None declared

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