Molecular epidemiology of *Pseudomonas aeruginosa* in intensive care units

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Summary

Background: *Pseudomonas aeruginosa* is a major nosocomial pathogen that is hard to treat because of its multiple antibiotic resistance. In this study, we aimed to investigate the molecular epidemiology and antibiotic susceptibility of clinical *P. aeruginos*a isolates from Intensive Care Unit (ICU) patients in order to contribute to developing rational antibiotic usage policies and monitoring infection control precautions.

Materials and Methods: seventy-five *P. aeruginosa* isolates from clinical specimens of patients in ICUs of Erciyes University Medical Faculty Hospital were studied. Bacterial identification was made using conventional methods and/or an automated Phoenix ID system (BD Diagnostics, Franklin Lakes, USA). The Minimal Inhibitory Concentration (MIC) for antibiotics was determined by broth microdilution. Repetitive sequence-based Polymerase Chain Reaction (rep-PCR) was used to assess the clonal relationship of the strains.

Results: the resistance rates of the strains for amikacin, gentamicin, ciprofloxacin, levofloxacin, ceftazidime, cefepime, imipenem, meropenem, colistin were 2.7%,18.7%, 24%, 22.7%, 16%, 5.3%, 64%, 60%, and 1.3% respectively. According to rep-PCR results, 21 clones (A-U) were identified. It was found that 33.3% of the strains were in clone B, which was the dominant type. It was found that antibiotic susceptibility patterns of some clonally related isolates were similar.

Conclusions: it is important to monitor resistance rates of infectious agents for guiding clinicians in empirical therapy and making rational antibiotic usage policies, it is also important to investigate clonal relationships for tracing the spread of infectious agents, especially multidrug-resistant pathogens and infection control precautions.

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Introduction

P. aeruginosa is a non-fermentative bacterium commonly found in natural environments, including soil, moist surfaces, plants, and water. It has the ability to reproduce on medical instruments, in the hospital environment and even in disinfectants [15,18]. It poses a risk, especially for patients whose immune system is suppressed and who receive chemotherapy, mechanical ventilation, and antibiotic therapy. It is mostly isolated from patients hospitalized in intensive care, burn, hematology, oncology, and surgery units [8]. It causes bacteremia, external otitis, pneumonia, urinary tract, burn wounds, and skin and soft tissue infections [1,3]. It has the ability to colonize permanently in a variety of environments due to the formation of a resistant biofilm. Infections caused by this virulent organism are difficult to control and treat due to intrinsic resistance [15,18].

In general, while carbapenems are reliable agents in the treatment of *P. aeruginosa* infections, there is a significant increase in the prevalence of carbapenem-resistant *P. aeruginosa* nowadays, associated with the increased use of all beta-lactam antibiotics. Fluoroquinolones, aminoglycosides, and some beta-lactam antibiotics are still active compounds against this microorganism. However, it can develop resistance to these agents with acquired resistance mechanisms [18].

Establishing rational antibiotic use policies and ensuring compliance is necessary to prevent the rise in resistance rates and mitigate the spread of antimicrobial-resistant bacteria. Epidemiological studies are carried out to monitor the resistance status of infectious agents and determine the resistance characteristics. This is very important in guiding the clinician in empirical treatment and establishing rational antibiotic use policies. Evaluation of clonal relationships enables the spread of infectious agents and monitoring of infection control measures [5].

This study aimed to investigate the antibiotic resistance pattern and molecular epidemiology of *P. aeruginosa* strains isolated from patients hospitalized in Intensive Care Units (ICU) by Repetitive sequence-based Polymerase Chain Reaction (rep-PCR) (DiversiLab, bioMérieux, France).

Materials and Methods

Bacterial isolates and patients

Between December 2013 and March 2015, 75 *P. aeruginosa* strains isolated from clinical samples of patients in ICUs of Erciyes University Medical Faculty Hospital were included in the study. Microorganisms were identified by colony morphology, Gram staining, oxidase character, and by using Phoenix ID (BD Diagnostics, Franklin Lakes, USA) fully automated identification system. Only one *P. aeruginosa* isolate was included from each patient. The isolates were stored in Microbanks at -70°C until the day of the study. The clinical data of the patients were obtained retrospectively from the hospital information system. Ethical approval for this study was obtained from the Erciyes University Clinical Research Ethics Commitee (09.05.2014, decision no: 2014/287).

Determination of antibiotic sensitivities

Antibiotic susceptibilities for amikacin, gentamicin, ciprofloxacin, levofloxacin, ceftazidime, cefepime, imipenem, meropenem, and colistin were determined in accordance with the criteria of the Clinical and Laboratory Standards Institute (CLSI) [17]. Potent powder formulations of antibiotics were obtained from authorized companies. In the broth microdilution method, two-fold serial dilutions of antibiotics were made using cation-adjusted Mueller Hinton Broth (Merck, Germany) in sterile U-bottom 96-well microplates. Twelve different concentrations of antibiotics, from 0.06 µg/mL to 128 µg/mL, were evaluated. *P. aeruginosa* ATCC 27853 standard strain was used for quality control.

Molecular epidemiological typing

For molecular epidemiological typing, the automated rep-PCR method DiversiLab (bioMérieux, Marcy l'Étoile, France) was used. Nucleic acid extraction was made by using UltraClean Microbial DNA Isolation Kit (Mo Bio, Carlsbad, USA). After the amplification by rep-PCR method using the DiversiLab Pseudomonas DNA Fingerprinting Kit (bioMérieux, France), microfluidic electrophoresis was performed using the DiversiLab DNA LabChip kit. The loaded chips were transferred to the Agilent Bioanalyzer, and the rep-PCR-based fingerprint patterns of *P. aeruginosa* isolates were evaluated with the internet-based software (2100 Expert Software version 3.6). The results were analyzed by creating a similarity matrix with the Pearson Correlation analysis, and the similarity of the isolates was interpreted. Isolates that were more than 90% (1-4 bands difference) similar to each other were considered as a main clone. Among the main clones, more than 95% (1-2 band difference) of similar clones were accepted as a subclone type. Strains with similarity rates below 90% (>4 bands difference) were considered as different clones.

Results

A total of 75 *P. aeruginosa* isolates from blood, pleural fluid, catheter tip, urine, Bronchoalveolar Lavage (BAL), Endotracheal Aspirate (ETA), and wound samples of patients in the ICUs were analyzed. Table 1 shows the distribution of *P. aeruginosa* isolates according to clinical samples and ICUs. Table 2 presents the characteristics and risk factors of the patients. Table 3 shows the antibiotic susceptibility, $MIC^{50,}$ and MIC^{90} values of the isolates.

According to rep-PCR analysis, 10 main clones (A-J) with a total of 64 isolates were identified while the other 11 isolates were found to be sporadic. Four subclones in clone B, three subclones in clone H, and two subclones each in clones C, D, and J were identified. Clone B was identified as the dominant clone, containing 33% (n=25) of the isolates. The isolates in this clone persisted for one year and were isolated from seven different ICUs. The six *P. aeruginosa* isolates in clone H were obtained from four different ICUs over a period of one year. It was observed that all nine isolates in clone J persisted for one year and were isolated from internal medicine and general surgery ICUs. In some of the other clones, it was determined that the isolates were obtained in a short time period, such as one month.

The susceptibility profile of the isolates belonging to clone J was identical (isolates with intermediate susceptibility phenotype

Table 1. Distribution of *P. aeruginosa* isolates according to clinical samples and ICUs.

	Clinical samples n (%)							
Intensive Care Units	Blood	Urine	BAL	ETA	Pleural fluid	Catheter tip	Wound	Total
IMICU	13 (65)	1 (50)	2 (22.2)	9 (32.1)		2 (66.7)	1 (11.1)	28 (37.3)
GSICU	2 (10)		1 (11.1)	3 (10.7)	1 (25)		3 (33.3)	10 (13.3)
AICU			3 (33.3)	5 (17.9)			2 (22.2)	10 (13.3)
CDICU	1 (5)		1 (11.1)	4 (14.3)				6 (8)
TSICU			1 (11.1)		3 (75)			4 (5.3)
PICU	2 (10)		1 (11.1)	1 (3.6)		1 (33.3)	1 (11.1)	6 (8)
BICU							2 (22.2)	2 (2.7)
NICU	2 (10)	1 (50)		6 (21.4)				9 (12)
Total	20	2	9	28	4	3	9	75 (100)

ICU, Intensive Care Unit; IMICU, Internal Medicine ICU; GSICU, General Surgery ICU; AICU, Anesthesia ICU; CDICU, Chest Diseases ICU; TSICU, Thoracic Surgery ICU; PICU, Pediatrics ICU; BICU, Burns ICU; NICU, Neurosurgery ICU.

were accepted as resistant). These strains were isolated from two different ICUs (internal medicine and general surgery ICUs). The antibiotic susceptibilities of the clonally related isolates are presented in Table 4.

In this study, 27 isolates were resistant only to carbapenems (imipenem or meropenem I/R). Of these isolates, 13 were in clone B, and five were in clone H. Most of the carbapenem-resistant isolates (n=8) in clone B were isolated from the internal medicine ICU within a month. Similarly, most of the carbapenem-resistant strains in clone H and all strains (n=3) in clone F, which were found to be carbapenem-resistant, were also isolated from the internal medicine ICU.

Twenty isolates with multidrug resistance (at least three antimicrobial groups I or R) were detected, eight of which were in clone B. Within two months, most of these strains were detected in internal medicine and general surgery ICUs.

Table 2. Characteristics and risk factors of patients with *P. aeruginosa* isolated.

Age n (%)	Gender n (%)	Days of hospitalization	Comorbidities n (%)	Previous treatments n (%)	Total parenteral nutrition n (%)	Immunosuppressive therapy n (%)	Invasive medical procedures n (%)
Adult 64 (85.3)	Male 49 (65.3)	33.37±28.82	Trauma 7 (9.3)	3rd generation cephalosporins 25 (33.3)	15 (20)	4 (5.3)	Major surgery 20 (26.7)
Children 11 (14.7)	Female 26 (34.7)		Acute/chronic renal failure 7 (9.3)	Cefepim 19 (25.3)			Mechanical ventilation 60 (80)
			Cancer 18 (24)	Aminoglycosides 21 (28)			Tracheostomy 28 (37.3)
			Respiratory failure 39 (52)	Fluoroquinolones 23 (30.6)			Central venous catheter 61 (81.3)
			Diabetes mellitus 13 (17.3)	Carbapenems 41 (54.6)			Hemodialysis catheter 18 (24)
				Glycopeptides 15 (20)			Arterial catheter 9 (12)
				Antifungal drugs 8 (10.6)			Urine catheter 69 (92)
				Colistin 12 (16)			

Table 3. Sensitivity of *P. aeruginosa* strains to the tested antibiotics.

Antibiotic name	MIC_{50}	MIC_{90}	MIC range	S		I		R		CLSI limit values[17]	
				n	%	n	%	n	%	S	R
Amikacin	1	8	0.25-64	69	92	4	5.3	2	2.7	≤16	≥64
Gentamicin	2	>128	0.50->128	53	70.7	8	10.7	14	18.7	≤4	≥16
Ciprofloxacin	0.125	32	<0.06-32	55	73.3	2	2.7	18	24	≤1	≥4
Levofloxacin	1	32	0.125-128	56	74.7	2	2.7	17	22.7	≤2	≥8
Ceftazidime	4	32	<0.06>128	56	74.7	7	9.3	12	16	≤8	≥32
Cefepim	4	8	0.25-128	68	90.7	3	4	4	5.3	≤8	≥32
İmipenem	8	16	0.125-128	19	25.3	8	10.7	48	64	≤2	≥8
Meropenem	8	32	<0.06-128	19	25.3	11	14.7	45	60	≤2	≥8
Colistin	0.25	1	<0.06-16	73	97.3	1	1.3	1	1.3	≤2	≥8

MIC, Minimum Inhibitory Concentration.

Table 4. Clonal relatedness and antibiotic susceptibility of the isolates.

		Antibiotic susceptibility								
Clone	Isolate ID	AN	GN	CIP	LEV	CAZ	FEP	IMP	MER	CO
A	PA53	S	S	R	S	S	S	R	R	S
	PA36	S	S	S	S	S	S	R	I	S
B1	PA33	S	S	S	S	S	S	R	I	S
	PA8	S	S	S	S	S	S	S	S	S
	PA20	S	S	S	S	S	S	R	I	S
	PA4	S	S	S	S	S	S	R	S	S
	PA7	S	S	S	S	S	S	R	S	S
	PA1	S	S	S	S	S	S	R	R	S
	PA6	S	S	S	S	S	S	R	R	S

To be continued on next page

Table 4. Continued from previous page.

Clone	Isolate ID	Antibiotic susceptibility								
		AN	GN	CIP	LEV	CAZ	FEP	IMP	MER	CO
B2	PA23	S	R	R	R	S	S	R	R	I
	PA19	S	S	S	S	S	S	R	I	S
	PA59	R	R	R	R	S	S	R	R	S
	PA13	S	S	S	S	I	S	S	S	S
	PA9	S	S	S	S	S	S	S	S	S
	PA35	S	R	S	S	R	S	I	R	S
	PA55	I	R	R	R	S	S	R	R	S
	PA37	I	R	R	R	I	S	R	R	S
	PA34	I	R	R	R	S	S	R	R	S
	PA57	R	R	R	R	S	S	R	R	S
B3	PA29	S	S	S	S	S	S	R	S	S
	PA24	S	S	S	S	S	S	R	S	S
	PA26	S	I	S	S	R	R	R	R	S
	PA21	S	I	S	S	I	S	S	S	S
	PA44	S	S	S	S	S	S	R	I	S
	PA66	S	S	S	S	S	S	R	R	S
B4	PA27	S	S	S	S	S	S	R	S	S
	PA67	S	S	S	S	S	S	R	R	S
C1	PA5	S	S	S	S	S	S	R	R	S
	PA22	S	S	R	R	R	S	S	I	S
	PA16	S	S	I	R	I	I	I	R	S
	PA17	S	S	S	S	S	S	R	R	R
	PA11	S	S	S	S	I	S	S	S	S
C2	PA12	S	S	S	S	S	S	S	S	S
D1	PA10	S	S	S	S	S	S	S	S	S
	PA70	S	S	R	R	R	S	S	I	S
D2	PA25	S	S	I	R	R	S	R	S	S
	PA63	S	S	R	R	R	S	R	R	S
E	PA32	S	S	S	S	R	R	S	S	S
	PA72	S	S	R	R	S	S	R	R	S
	PA14	S	S	S	S	S	S	R	R	S
	PA30	S	S	S	S	S	S	S	S	S
	PA56	S	S	R	S	S	S	R	R	S
F	PA61	S	S	S	S	S	S	R	R	S
	PA31	S	S	S	S	S	S	R	R	S
	PA18	S	S	S	S	S	S	I	S	S
G	PA52	S	S	R	S	S	S	R	I	S
	PA54	S	S	S	I	S	S	I	R	S
H1	PA69	S	S	S	S	S	S	R	R	S
	PA74	S	S	S	S	S	S	R	S	S
H2	PA51	S	S	S	S	S	S	I	I	S
H3	PA48	S	S	S	S	S	S	S	R	S
	PA73	S	S	S	R	R	S	R	R	S
	PA60	S	S	S	S	S	S	S	I	S
I	PA49	S	S	R	R	I	I	R	R	S
	PA45	S	S	S	S	R	S	S	S	S
J1	PA68	S	R	S	S	S	S	I	R	S
	PA47	S	I	S	S	S	S	R	R	S
	PA40	S	R	S	S	S	S	R	R	S
	PA39	S	R	S	S	S	S	R	R	S
	PA58	S	I	S	S	S	S	R	R	S
	PA50	S	R	S	S	S	S	R	R	S
	PA64	S	I	S	S	S	S	R	R	S
	PA43	S	I	S	S	S	S	R	R	S
J2	PA62	S	I	S	S	S	S	R	R	S
K	PA15	S	S	S	S	S	S	S	S	S
L	PA71	S	S	R	S	I	S	I	R	S
M	PA65	S	I	R	R	R	I	I	R	S
N	PA3	S	R	S	S	S	S	R	R	S
O	PA41	I	R	R	R	R	R	R	R	S
P	PA28	S	R	R	R	R	R	S	R	S
Q	PA46	S	S	S	S	S	S	S	S	S
R	PA38	S	S	S	S	S	S	S	R	S
S	PA2	S	S	S	S	S	S	R	R	S
T	PA75	S	S	S	I	I	S	S	R	S
U	PA42	S	S	S	S	S	S	S	R	S

PA, *P. aeruginosa*; AN, Amikacin; GN, Gentamicin; CIP, Ciprofloxacin; LEV, Levofloxacin; CAZ, Ceftazidime; FEP, Cefepime; IMP, Imipenem; MER, Meropenem; CO, Colistine; R, Resistant; I, Intermediate; S, Susceptible.

Discussion

P. aeruginosa causes infections with high mortality and morbidity in patients hospitalized in ICUs [4]. It has been reported that *P. aeruginosa* infections are mostly associated with invasive tools and procedures [19]. It was determined that all patients in our study had a history of at least one invasive intervention. Urinary and central venous catheterization, and mechanical ventilation were the most frequent among these interventions. In addition, antibiotic use and comorbid conditions such as chronic obstructive pulmonary disease are other important risk factors [20]. Carbapenem use was present in more than half of the patients in our study, and respiratory failure, malignancy, and diabetes were prominent among the comorbid conditions.

According to antibiotic susceptibility studies conducted in our country, the resistance rates for amikacin, gentamicin, ciprofloxacin, levofloxacin, ceftazidime, cefepime, imipenem, meropenem, and colistin were found to be between 3-38%, 6-34%, 17-30%, 18-52%, 19-35%, 17-35%, 19-32%, 15-47%, and 0-7%, respectively [1,2,3,8,10,14]. In studies involving intensive care units in Turkey, antibiotic resistance rates for amikacin, gentamicin, ciprofloxacin, levofloxacin, ceftazidime, cefepime, imipenem, meropenem, and colistin were found to be between 8-55%, 19-45%, 34-38%, 43-49%, 29-41%, 28-55%, 27-70%, 27-70%, and 1-5%, respectively [4,7,11,13,16]. According to these results, it can be observed that the antibiotic susceptibility rates of *P. aeruginosa* strains isolated in ICUs are lower than in other clinics, which can be elucidated by the higher use of antibiotics in ICUs. The antibiotic group with the highest resistance rate was carbapenems; nevertheless, amikacin and colistin were found to be the most effective antibiotics against *P. aeruginosa*. While the carbapenem resistance rates in our study were above the average of Turkey's ICU data, the rates of resistance to aminoglycosides, quinolones, and cephalosporins were found at the lower values of these ranges. This result can be related to the prevalence of antipseudomonal carbapenem use in the treatment of *P. aeruginosa* infection. These variable rates in antibiotic susceptibility reveal the necessity for each hospital to determine its own resistance pattern in order to determine treatment protocols.

In the light of the results obtained by molecular methods and classical epidemiological information, the distribution of the clones is determined, and the source of the isolates is defined. For this purpose, PFGE is the most commonly used and accepted as the gold standard method. However, the rep-PCR method was preferred in this study. It has been shown that the results obtained by both methods are compatible. Rep-PCR method has been suggested as a reliable, faster, and easier method in *P. aeruginosa* genotyping due to the disadvantages of the Pulsed-Field Gel Electrophoresis (PFGE) method, such as time consumption, difficulty of standardization between laboratories, and application of gel electrophoresis [6]. It has been reported that the rep-PCR method gave successful results when compared with PFGE in the genotyping of *P. aeruginosa* isolates [12].

In a study conducted in 30 Brazilian hospitals, 92.7% of the 161 carbapenem-resistant *P. aeruginosa* isolates showed >95% genetic similarity, which indicates a clonal spread according to the rep-PCR genotyping [9]. In Turkey, Yetkin *et al.* [19] investigated the epidemiological relationship between 105 *P. aeruginosa* strains using the PFGE method. They stated that 16 patients were epidemiologically related and that the patients might have acquired the microorganism by cross-contamination, which can occur horizontally with the hands of personnel or contaminated medical devices. In another study from Turkey, *P. aeruginosa* strains with similar antibiotic sensitivities were isolated from the plastic surgery clinic and ICU of a hospital, and to investigate the possible nosocomial outbreak, 26 carbapenem-resistant *P. aeruginosa* isolates were analyzed with PFGE. The strains were reported to be clustered in four main clones. The emphasis was placed on the increase in the isolation of bacteria exhibiting the same antibiotic resistance pattern within the same time interval, serving as a warning for a potential epidemic [5]. In our study, it was determined that there were isolates showing clonality at different times and in different ICUs, and it was thought that these strains might be the members of the hospital flora and might cause cross-contamination among patients.

Phenotypic similarities between isolates, such as antibiotic susceptibility, should draw attention to possible clonal spread and should be confirmed by molecular methods. 8 isolates from clone B were detected within a month. No significant reason could be found to explain this clonality. However, since it does not mean there will be no epidemic in the future, attention should be paid to the issues that need to be considered. Evaluation of clonality among pathogens isolated from patients in ICUs is important in terms of monitoring the infection control measures, and the spread of multi-resistant pathogens. Although phenotypic similarities between strains, such as antibiotic susceptibility solely, do not indicate the same genetic source, high similarity in antibiotic susceptibilities, or an unexpected resistance profile should alert clinicians to the need for an epidemiological investigation. There were 20 multidrug-resistant strains in different clones, and two units emerged within two months. An epidemic cannot occur in a long period of 16 months, but it is important to recognize clonal spread in advance. It doesn't mean there won't be an outbreak later.

Monitoring the antibiotic resistance of infectious agents guides the clinician in empirical treatment and ensures rational antibiotic use policies. Evaluation of molecular epidemiology is important in terms of monitoring the infection control measures and the clonal spread of infectious agents, especially multi-resistant pathogens. A higher number of isolates would have made the results more valuable in terms of resistance epidemiology. Another limitation of the study is that the rep-PCR method is old. It is necessary to continue clonal affinity and resistance epidemiology studies with higher numbers and new methods.

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