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Changes in the Dependence of *Pseudomonas aeruginosa* O Serogroup Strains and Their Resistance to Antibiotics in a University Hospital During a 5-year Period

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Key words: *Pseudomonas aeruginosa; O serogroup; antibiotic resistance.*

Summary. *The aim of our study was to determine the changes in antibiotic resistance and O serogroup dependence of P. aeruginosa strains isolated from lower respiratory tract specimens of patients in 2003 and 2008; the patients were treated in intensive care units of the biggest treatment facility in Lithuania (Hospital of Lithuanian University of Health Sciences, HLUHS, former Hospital of Kaunas University of Medicine)*

Material and Methods. The study included 90 P. aeruginosa strains serotyped in 2003 and 101 P. aeruginosa strains serotyped in 2008, which were randomly selected. The resistance of P. aeruginosa strains was determined by the disc diffusion method based on the standard guidelines. The sizes of inhibition zones were interpreted according to the National Committee for Clinical Laboratory Standards (M_2-A_6). Isolates were serotyped using sera with specific antibodies against the O antigens of P. aeruginosa (Bio-Rad, France).

Results. Comparison of changes in the distribution of P. aeruginosa serogroups in 2003 and 2008 showed that P. aeruginosa strains of serogroups O:1, O:2, and O:3 were more prevalent in 2003 as compared with 2008 (23.3%, n=21; 27.8%, n=25; 12.2%, n=11 vs. 9.9%, n=10; 10.9%, n=11; 4.0%, n=4, P<0.05). P. aeruginosa strains of serogroups O:6 and O:11 were isolated more frequently in 2008 than 2003 (26.7%, n=27; 34.7%, n=35 vs. 4.4%, n=4; 10.0%, n=9, P<0.001). The results showed that 18 of the 90 P. aeruginosa strains in 2003 and 25 of the 101 P. aeruginosa strains in 2008 were resistant to three or more antibiotics tested, i.e., they were multidrug-resistant. Analysis of the distribution of serogroups among these P. aeruginosa strains isolated in 2003 and 2008 revealed a significantly higher frequency of O:11 serogroup than other serogroups. Meanwhile, in the group of nonmultidrug-resistant P. aeruginosa strains, P. aeruginosa O:11 serogroup strains were identified less frequently and accounted only for 2.8% (n=2, P<0.001) of the isolates in 2003 and 27.6% (n=21, P<0.01) in 2008.

Conclusions. During the 5-year period, the isolation rate of P. aeruginosa strains belonging to serogroup O:11 increased. P. aeruginosa strains isolated in 2003 and 2008 belonging to serogroup O:11 were more frequently multidrug resistant. The increasing resistance of P. aeruginosa to reserve antibiotics of carbapenem group was observed.

Introduction

Pseudomonas aeruginosa (*P. aeruginosa*) strains have an innate characteristic of the resistance to antibiotics and capability to rapidly acquire the resistance to antibiotics with antipseudomonal activity. Due to these reasons, *P. aeruginosa* remains an important nosocomial pathogen and still causes considerable problems in most countries including Lithuania (1–4). It has been demonstrated that resistance patterns of *P. aeruginosa* strains to antibiotics differ depending on lipopolysaccharide structure of the bacterial outer membrane, which is part of the bacterial cell wall and O serogroup (according to O antigen), to which they belong (5, 6). However, an important role in the development of the resistance to antibiotics among *P. aeruginosa* strains is played by antibiotic policy and prescribing strategies in hospitals and within a country. There is a large body of evidence that frequent usage of antibiotics can cause alterations in *P. aeruginosa*

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strains and can drive transformation to strains having more pathogenicity factors and strains more resistant to antibiotics (7, 8). Due to the effect of antibiotics, resistant strains of *P. aeruginosa* emerge. Therefore, the antibiotic resistance of *P. aeruginosa* strains, belonging to the same serogroup, isolated from the specimens of patients treated in different clinical settings can differ as different antibiotics and their dosages are administered. In Poland, *P. aeruginosa* strains resistant to aminoglycosides and beta-lactam antibiotics were serotyped as O:12 more frequently (6); in Greece, *P. aeruginosa* strains resistant to antibiotics belonged to O:11 serogroup more frequently (9). The findings of our previous studies showed that *P. aeruginosa* strains resistant to most antipseudomonal antibiotics (multidrug resistant, MDR) belonged to O:E serogroup (O:11) more frequently (10). It is important to monitor the changes in the serogroup dependence and antibiotic resistance of *P. aeruginosa* strains in a larger treatment facility over a period of several years since knowledge of these changes can contribute to a successful choice of empirical treatment for critically ill patients. Our study aimed at determining the changes in antibiotic resistance and O serogroup dependence of *P. aeruginosa* strains isolated from lower respiratory tract specimens of patients in 2003 and 2008 treated in intensive care units of the biggest treatment facility in Lithuania (Hospital of Lithuanian University of Health Sciences, HLUHS).

Material and Methods

P. aeruginosa Strains. According to the data of the Laboratory of Microbiology at the HLUHS, a total of 145 *P. aeruginosa* strains in 2003 and 151 in 2008 were isolated from lower respiratory tract specimens (bronchial or bronchoalveolar lavage) of patients treated in the intensive care units (central, neurosurgery, and cardiac surgery). The strains were identified according to the standard methodology. The study included 90 *P. aeruginosa* strains serotyped in 2003 and 101 *P. aeruginosa* strains serotyped in 2008, which were randomly selected; one patient, one strain. The molecular fingerprinting was not performed, and copy strains were not excluded. Although only noncopy strains were included in this study, it cannot be excluded at this stage that clonal strains bearing certain serotypes spread locally. Appropriate studies to test the clonal diversity of *P. aeruginosa* strains are needed.

Susceptibility Testing. The resistance of *P. aeruginosa* strains was determined by the disc diffusion method based on the standard guidelines (11). The sizes of inhibition zones were interpreted according to the National Committee for Clinical Laboratory Standards (M_2–A_6). Standard *P. aeruginosa* ATCC 27853 strain was used as a control strain.

Serotyping. Isolates were serotyped using sera with specific antibodies against O antigens of *P. aeruginosa* (Bio-Rad, France). Serogroups were denominated by Arabic numerals from O:1 to O:16 according to the classification of the ICSB Subcommittee on Pseudomonas and Related Organisms. At the same time, serogroups were identified using the agglutination method according to the methodology approved by the manufacturer of specific antibodies (Bio-Rad, France). At the beginning, a bacterial suspension was prepared: no later than after 24 hours, the isolated P. *aeruginosa* colony grown on the nonselective growth medium was diluted with sterile physiological saline. One drop of a polyvalent serum (pooled conjugate for several specific *P. aeruginosa* serogroups) was mixed with 10 μL of the bacterial suspension solution on a glass slide; by rocking the slide gently in a rotary motion, no later than after 5 minutes, it was observed if agglutination occurred. If agglutination was observed during the testing with a polyvalent serum, further testing was carried out by employing particular specific sera belonging to that group of a polyvalent serum. Physiological saline without serum was used as a negative control. If agglutination occurred later than after 5 minutes or bacterial suspension agglutinated with more than one specific serum, the testing was repeated by heating the suspension for 30 minutes at 120°C before agglutination.

The serogroups obtained by Bio-Rad corresponded to the following Homma serogroups: O:1 (O:I), O:2 (O:B), O:3 (O:A), O:4(O:F), O:6 (O:C), O:7 (O:G), O:9 (O:D), O:10 (O:H), and O:11 (O:E), respectively.

Statistical Analysis. Statistical analysis was conducted using the SPSS (Statistical Package for Social Sciences, Microsoft Inc., USA) software, version 10.0 for Windows. While analyzing differences in frequency, nonparametric statistical criterion χ^2 and Fisher exact test were used. The differences between the groups were considered significant if P was <0.05.

Results

Distribution of O Serogroups. Analysis of serogroup dependence of *P. aeruginosa* strains isolated in 2003 showed that *P. aeruginosa* strains belonged to 10 of the 16 O serogroups: O:1 (23.3%, n=21); O:2 (27.8%, n=25), O:3 (12.2%, n=11), O:6 (4.4%, n=4), O:7 (2.2%, n=2), and O:11 (10%, n=9); there were four more strains each belonging to O:4, O:9, O:12, and O:13 serogroups (in each case 1.1%). In 15.7% (n=14) of cases, *P. aeruginosa* strains did not agglutinate with specific sera; therefore, the serogroup was not identified. *P. aeruginosa* strains isolated in 2008 belonged to 8 O serogroups: O:1 (9.9%, n=10); O:2 (10.9%, n=11), O:3 (4.0%, n=4), O:4 (2.0%, n=2), O:6 (26.7%, n=27), O:7 (1.0%, n=1), O:11 (34.7%, n=35); 10.8% (n=11) of *P. aeruginosa* strains could not be serotyped.

Comparison of changes in the distribution of *P.*

aeruginosa serogroups in 2003 and 2008 showed that *P. aeruginosa* strains of O:1, O:2, and O:3 serogroups were more prevalent in 2003 as compared with 2008 (23.3%, n=21; 27.8%, n=25; 12.2%, n=11 vs. 9.9%, n=10; 10.9%, n=11; 4.0%, n=4, $P<0.05$). *P. aeruginosa* strains of O:6 and O:11 serogroups were isolated more frequently in 2008 than in 2003 (26.7%, n=27; 34.7%, n=35 vs. 4.4%, n=4; 10.0%, n=9, $P<0.001$).

O Serogroup and Multidrug Resistance. The results showed that 18 of the 90 *P. aeruginosa* strains in 2003 and 25 of the 101 *P. aeruginosa* strains in 2008 were resistant to three or more antibiotics tested, i.e., they were MDR. Analysis of the distribution of *P. aeruginosa* strains isolated in 2003 and 2008 by serogroup revealed that these strains agglutinated with O:11 serogroup serum more frequently than with sera for other serogroups. Meanwhile, in the group of non-MDR *P. aeruginosa* strains, O:11 serogroup strains were identified less frequently and accounted only for 2.8% (n=2, $P<0.001$) of the isolates in 2003 and 27.6% (n=21, $P<0.01$) in 2008. Table 1 shows the distribution of MDR and non-MDR *P. aeruginosa* strains by serogroups.

O serogroup and Resistance to Antibiotics. In the evaluation of serogroup dependence of *P. aeruginosa* strains resistant to separate antibiotics, it was determined that most of ceftazidime-resistant *P. aeruginosa* strains belonged to O:11 serogroup (40%, n=2 in 2003 and 50%, n=7 in 2008). In 2008, a significant increase in the proportion of *P. aeruginosa* O:11 serogroup strains resistant to piperacillin and piperacillin/tazobactam as compared with the piperacillin- and piperacillin/tazobactam-resistant *P. aeruginosa* O:11 serogroup strains isolated in 2003 was observed (66.7%, n=12 and 55.6%, n=5 vs. 14.3%, n=3 and 8.3%, n=1, respectively; $P<0.05$) (Table 2).

While analyzing the serogroup dependence of *P. aeruginosa* strains resistant to aminoglycosides, a significant difference comparing the years 2003 and 2008 was observed only in the prevalence of gentamicin-resistant *P. aeruginosa* strains, which were serotyped as O:6. The analysis of *P. aeruginosa* strains isolated in 2003 showed that there were no gentamicin-resistant *P. aeruginosa* strains belonging to O:6 serogroup, and in 2008, even 25% (n=5) of gentamicin-resistant strains belonged to this serogroup ($P<0.05$).

Table 1. Distribution of Multidrug-Resistant and Nonmultidrug-Resistant *Pseudomonas aeruginosa* Strains by Serogroups in 2003 and 2008

O Serogroup of *P. aeruginosa* Strains	*Pseudomonas aeruginosa* Strains							
	2003				2008			
	Total N=90 n (%)	MDR N=18 n (%)	Non-MDR N=72 n (%)	*P*	Total N=101 n (%)	MDR N=25 n (%)	Non-MDR N=76 n (%)	*P*
1	21 (23.3)	2 (11.1)	19 (26.4)	0.223	10 (0.1)	5 (20.0)	5 (6.6)	0.115
2	25 (27.8)	1 (5.6)	24 (33.3)	0.019	11 (10.9)	0	11 (14.5)	0.061
3	11 (12.2)	2 (11.1)	9 (12.5)	1.0	4 (4.0)	1 (4.0)	3 (3.9)	1.0
6	4 (4.4)	0	4 (5.6)	0.58	27 (26.7)	3 (12.0)	24 (31.6)	0.055
11	9 (10)	7 (38.9)	2 (2.8)	<0.001	35 (48.6)	14 (56.0)	21 (27.6)	0.01
4, 7, 9, 12, 13	6 (6.7)	2 (11.1)	4 (5.6)	0.595	3 (3.0)	0	3 (3.9)	0.572
Nontypable	14 (15.6)	4 (22.2)	10 (13.9)	0.467	11 (10.9)	2 (8.0)	9 (11.8)	0.727

MDR, multidrug-resistant *Pseudomonas aeruginosa* strains.

Table 2. Distribution of *Pseudomonas aeruginosa* Strains Resistant to Beta-Lactam Antibiotics by Serogroups in 2003 and 2008

O Serogroup of *P. aeruginosa* Strains	*Pseudomonas aeruginosa* Strains Resistant to Antibiotics					
	Ceftazidime		Piperacillin		Piperacillin/Tazobactam	
	2003 N=5 n (%)	2008 N=14 n (%)	2003 N=21 n (%)	2008 N=18 n (%)	2003 N=12 n (%)	2008 N=9 n (%)
1	1 (20.0)	4 (28.6)	3 (14.3)	3 (16.7)	3 (25.0)	3 (33.3)
2	1 (20.0)	0	4 (19.0)	0	2 (16.7)	0
3	0	0	3 (14.3)	1 (5.6)	3 (25.0)	1 (11.1)
6	0	2 (14.3)	0	2 (11.1)	0	0
11	2 (40.0)	7 (50.0)	3 (14.3)	12 (66.7)*	1 (8.3)	5 (55.6)*
4, 7, 9, 12, 13	0	0	2 (9.5)	0	0	0
Nontypable	1 (20.0)	1 (7.1)	6 (28.6)	0	3 (25.0)	0

*$P<0.05$, the year 2008 versus the year 2003.

Table 3. Distribution of *Pseudomonas aeruginosa* Strains Resistant to Aminoglycosides and Ciprofloxacin by Serogroups in 2003 and 2008

O Serogroup of *Pseudomonas aeruginosa* Strains	*Pseudomonas aeruginosa* Strains Resistant to Antibiotics					
	Gentamicin		Amikacin		Ciprofloxacin	
	2003 N=18 n (%)	2008 N=20 n (%)	2003 N=4 n (%)	2008 N=6 n (%)	2003 N=14 n (%)	2008 N=40 n (%)
1	2 (11.1)	4 (20.0)	1 (25.0)	2 (33.3)	1 (7.1)	3 (7.5)
2	1 (5.6)	0	0	0	1 (7.1)	0
3	4 (22.2)	1 (5.0)	0	0	1 (7.1)	2 (5.0)
6	0	5 (25.0)*	0	1 (16.7)	0	2 (5.0)
11	6 (33.3)	6 (30.0)	1 (25.0)	2 (33.3)	6 (42.9)	28 (70.0)*
4, 7, 9, 12, 13	2 (11.1)	2 (10.0)	0	0	1 (7.1)	2 (5.0)
Nontypable	3 (16.7)	2 (10.0)	2 (50.0)	1 (16.7)	4 (28.6)	3 (7.5)

*$P<0.05$, the year 2008 versus the year 2003.

Table 4. Distribution of *Pseudomonas aeruginosa* Strains Resistant to Carbapenems by Serogroups in 2003 and 2008

O Serogroup of *Pseudomonas aeruginosa* strains	*Pseudomonas aeruginosa* Strains Resistant to Antibiotics			
	Imipenem		Meropenem	
	2003 N=7 n (%)	2008 N=40 n (%)	2003 N=15 n (%)	2008 N=29 n (%)
1	3 (42.9)	4 (10.0)	3 (20.0)	3 (10.3)
2	0	0	0	0
3	0	0	0	0
6	0	11 (27.5)	0	7 (24.1)
11	0	19 (47.5)*	5 (33.3)	15 (51.7)
4, 7, 9, 12, 13	2 (28.6)	0	3 (20.0)	0
Nontypable	2 (28.6)	6 (15.0)	4 (26.7)	4 (13.8)

*$P<0.05$, the year 2008 versus the year 2003.

Among ciprofloxacin-resistant *P. aeruginosa* isolates, the most frequently encountered serogroup was O:11 in both 2003 and 2008 (42.9%, n=6 and 70%, n=28; $P<0.05$) (Table 3).

During the study, among *P. aeruginosa* strains isolated in 2003, there were 7 strains resistant to imipenem, and only 5 of them were serotyped. In 2003, none of the imipenem-resistant *P. aeruginosa* strains, which belonged to O:11 serogroup, was isolated. Whereas, among *P. aeruginosa* strains isolated in 2008, nearly half (47.5%, n=19) of the imipenem-resistant *P. aeruginosa* strains belonged to O:11 serogroup ($P<0.05$). In 2003, there was no difference in the distribution of meropenem-resistant *P. aeruginosa* strains by serogroup; however, in 2008, more than half of these strains (51.7%, n=15) belonged to O:11 serogroup (Table 4).

Analysis of changes in the resistance of *P. aeruginosa* strains to beta-lactam antibiotics in 2003 and 2008 revealed that there was an increase in the rates of the resistance to ceftazidime (from 5.6% to 13.9%, $P<0.05$), imipenem (from 7.8% to 39.6%, $P<0.001$), and meropenem (from 16.7% to 28.7%, $P<0.05$) during the 5-year period. However, there were no changes in the resistance of *P. aeruginosa* strains to piperacillin and piperacillin/tazobactam (Fig. 1).

While evaluating the resistance of *P. aeruginosa* strains to antibiotics of aminoglycoside group, no changes were observed during the 5-year period. However, the resistance of *P. aeruginosa* strains to ciprofloxacin increased from 15.6% in 2003 to 39.6% in 2008 ($P<0.001$) (Fig. 2).

Discussion

The increasing resistance of *P. aeruginosa* strains to antibiotics has become a recognized problem not only in most European countries, but also across the world; it has also been linked to the frequent usage of antibiotics (12–16). Bacteria have an innate characteristic enabling them to exchange diverse mobile genetic elements, which are responsible for the resistance to antibiotics, and this leads to the emergence of multidrug-resistant strains spreading in a hospital environment (17, 18). The increasing resistance of *P. aeruginosa* strains even to reserve antibiotics – carbapenems – causes consider-

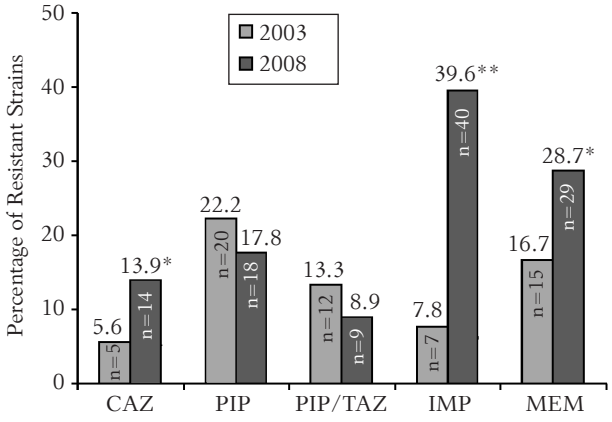

Fig. 1. Resistance of *Pseudomonas aeruginosa* strains, isolated in 2003 and 2008, to beta-lactam antibiotics

CAZ, ceftazidime; PIP, piperacillin; PIP/TAZ, piperacillin/tazobactam; IMP, imipenem; MEM, meropenem.
*$P<0.05$, the year 2008 versus the year 2003;
**$P<0.001$, the year 2008 versus the year 2003.

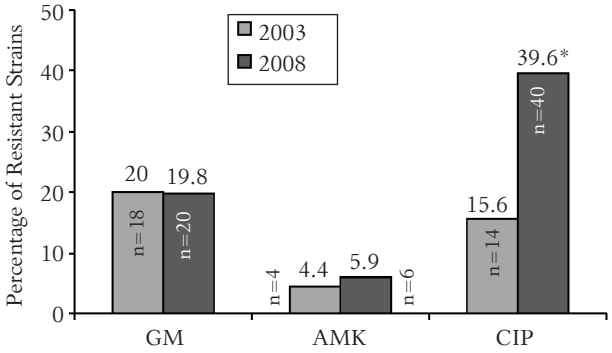

Fig. 2. Resistance of *Pseudomonas aeruginosa* strains, isolated in 2003 and 2008, to aminoglycosides and ciprofloxacin

GM, gentamicin; AMK, amikacin; CIP, ciprofloxacin.
*$P<0.01$, the year 2008 versus year the 2003.

able problems in choosing the empirical treatment of infections caused by *P. aeruginosa* (18, 19). It has been proved that pathogenicity factors are found more frequently in *P. aeruginosa* strains with the outer membrane formed from O antigen-containing lipopolysaccharides (20, 21). However, there are scarce data regarding capability of *P. aeruginosa* strains of different O serogroups to acquire the resistance to antibiotics. Findings of our previous studies have shown that MDR *P. aeruginosa* strains were serotyped as O:11 (Homma O:E), and these data were consistent with the findings from a study of *P. aeruginosa* strains prevalent in Greek hospitals (9, 10). However, the surveillance of changes in the resistance to antibiotics in every treatment facility is crucial, and these changes can often be dependent on the changes in the prevalence of *P. aeruginosa* strains of different serogroups. During this study, a comparison of distribution of *P. aeruginosa* strains by serogroups in 2003 and 2008 revealed a considerable increase in the number of *P. aeruginosa* strains belonging to O:11 serogroup in the studied hospital in 2008. As *P. aeruginosa* strains of this serogroup remain more resistant to most antipseudomonal antibiotics, it is interesting to note that no significant increase in the resistance of *P. aeruginosa* strains to antibiotics, except for imipenem, was observed during the 5-year period. It is important to note that despite an increase in the isolation rate of *P. aeruginosa* strains belonging to O:11 serogroup, the spread of multidrug-resistant *P. aeruginosa* strains was not observed. Our previous data demonstrated that although *P. aeruginosa* strains of O:11 (Homma O:E) serogroup were resistant to most of antipseudomonal antibiotics more frequently, convincing evidence could not be found to prove that they were more pathogenic – the occurrence rate of hospital-acquired pneumonia or colonization rate of the lower respiratory tract was the same (10). Analysis of our data revealed that during the 5-year period, the resistance of *P. aeruginosa* strains to meropenem increased from 16.7% to 28.7%, and currently it does not differ from the resistance documented in other countries (22). However, compared with the data of other studies, the resistance rate of *P. aeruginosa* strains to imipenem, which was markedly lower in 2003 (7.8%), has recently reached 39.6% and has exceeded resistance rates reported by other countries (22–24). Interestingly, there was no imipenem-resistant strains belonging to O:11 serogroup among *P. aeruginosa* strains isolated in 2003; meanwhile, 47.5% of imipenem-resistant *P. aeruginosa* strains serotyped in 2008 belonged to O:11 serogroup.

Although the present study shows that the resistance to ceftazidime increased to 13.9% during the 5-year period, it is still lower than in some other centers reaching even 48.9% (23).

Conclusions

During the 5-year period, the isolation rate of *P. aeruginosa* strains belonging to O:11 serogroup increased. *P. aeruginosa* strains both isolated in 2003 and 2008 belonging to O:11 serogroup were more frequently MDR. The increasing resistance of *P. aeruginosa* to reserve antibiotics of carbapenem group was observed.

Statement of Conflict of Interest

The authors state no conflict of interest.

Pseudomonas aeruginosa padermių O serogrupių priklausomybės ir atsparumo antibiotikams pokyčiai universitetinėje ligoninėje per penkerius metus

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Raktažodžiai: *Pseudomonas aeruginosa,* O serogroupės, atsparumas antibiotikams.

Santrauka. *Tyrimo tikslas.* Nustatyti *P. aeruginosa* padermių, išskirtų iš didžiausioje Lietuvos gydymo įstaigoje (buvusiose Kauno medicinos universiteto klinikose, KMUK) intensyviosios terapijos skyriuose (ITS) gydytų pacientų apatinių kvėpavimo takų (AKT) sekreto, O serogrupių priklausomybės ir atsparumo antibiotikams pokyčius 2003 ir 2008 m.

Metodai. Į tyrimą įtrauktos *P. aeruginosa* padermių serogrupės, kurių 2003 m. nustatyta 90, 2008 m. – 101, jos buvo atrinktos atsitiktinės atrankos būdu. *P. aeruginosa* padermių atsparumas antibiotikams nustatytas diskų difuzijos metodu remiantis standartine metodika. Slopinimo zonų dydžiai interpretuoti pagal Nacionalinio klinikinių laboratorijų standartų komiteto (NCCLS – angl. *National committee for clinical laboratory standards*) M_2–A_6 lenteles. Serogrupėms nustatyti naudoti serumai, turintys specifinių antikūnų prieš *P. aeruginosa* O grupės antigenus (Bio-Rad, Prancūzija).

Rezultatai. Tyrimo metu lyginant *P. aeruginosa* padermių serogrupių priklausomybės pokyčius 2003 ir 2008 m., nustatyta, kad *P. aeruginosa* O:1, O:2 ir O:3 serogrupės padermės dažniau buvo išskirtos 2003 m. lyginant su 2008 m. (23,3 proc., n=21; 27,8 proc., n=25; 12,2 proc., n=11 ir 9,9 proc., n=10; 10,9 proc., n=11; 4,0 proc., n=4, p<0,05). *P. aeruginosa* O:6 ir O:11 serogrupių padermės dažniau buvo išskirtos 2008 m. nei 2003 m. (26,7 proc., n=27; 34,7 proc., n=35 ir 4,4 proc., n=4; 10,0 proc., n=9, p<0,001). Ištyrus 2003 m. ir 2008 m. išskirtų dauginio atsparumo (MDR) *P. aeruginosa* padermių serogrupių priklausomybę, nustatyta, kad šios padermės dažniau agliutinavo su O:11 serogrupės serumu nei su kitais serogrupių serumais. Tuo tarpu *P. aeruginosa* padermių, nepriklausančių MDR, grupėje O:11 serogrupės *P. aeruginosa* padermių patikimai rečiau nustatyta ir sudarė tik 2,8 proc. (n=2, p<0,001) 2003 m., ir 27,6 proc. (n=21, p<0,01) 2008 m.

Išvados. Penkerių metų laikotarpiu padaugėjo O:11 serogrupei priklausančių *P. aeruginosa* padermių išskyrimo dažnis. Tiek 2003 m., tiek 2008 m. išskirtos O:11 serogrupei priklausančios *P. aeruginosa* padermės dažniau buvo MDR. Didėja *P. aeruginosa* atsparumas rezerviniams karbapenemų grupės antibiotikams.

References

1. Di Martino P, Garniere H, Berry H, Bret L. Antibiotic resistance and virulence properties of Pseudomonas aeruginosa strains from mechanically ventilated patients with pneumonia in intensive care units: comparison with imipenem-resistant extra-respiratory tract isolates from uninfected patients. Microbes Infect 2002;4:613-20.
2. Matar GM, Chaar MH, Araj GF, Srour Z, Jamaleddine G, Hadi U. Detection of a highly prevalent and potentially virulent strain of Pseudomonas aeruginosa from nosocomial infections in a medical center. BMC Microbiology 2005;5:29.
3. Kareivienė V. Dažniausių pūlinės infekcijos sukėlėjų, nustatytų Kauno miesto gydymo įstaigose, atsparumo antibiotikams analizė. (Analysis of most common purulent infection agents resistance to antibiotics aquired at Kaunas health institutions.) [Dissertation]. Kaunas: KMU; 2003.
4. Gailienė G. Pseudomonas aeruginosa atsparumo antibiotikams ir serogrupinės priklausomybės ypatumų sąsajos. (The links of pecularities of Pseudomonas aeruginosa resistance to antibiotics and of its different serogroups.) [Dissertation]. Kaunas: KMU; 2004.
5. Tassios PT, Gennimata V, Maniatis AN, Fock C, Legakis NJ. Emergence of multidrug resistance in uniquitous and dominant Pseudomonas aeruginosa serogroup O:11. The Greek Pseudomonas Aeruginosa Study Group. J Clin Microbiol 1998;36:897-901.
6. Patzer J, Dzierzanowska D. The resistance patterns and serotypes of Pseudomonas aeruginosa strains isolated from children. J Antimicrob Chemother 1991;28:869-75.
7. Alverdy J, Zaborina O, Wu L. The impact of stress and nutrition on bacterial-host interactions at the intestinal epithelial surface. Curr Opin Clin Nutr Metab Care 2005;8:205-9.
8. Zaborina O, Kohler JE, Wang Y, Bethel C, Shevchenko O, Wu L, et al. Identification of multi-drug resistant Pseudomonas aeruginosa clinical isolates that are highly disruptive to the intestinal epithelial barrier. Ann Clin Microbiol Antimicrob 2006;5:14.
9. Farmer JJ, Weinstein RA, Zierdt CH, Brokopp CD. Hospital outbreaks caused by Pseudomonas aeruginosa: importance of serogroup O:11. J Clin Microbiol 1982;16(2):266-70.
10. Vitkauskienė A. Pathogenicity factors of potential hospital-acquired pneumonia pathogens, Pseudomonas aeruginosa and Klebsiella pneumoniae, and their influence on the course of disease. (Potencialių hospitalinės pneumonijos sukėlėjų Pseudomonas aeruginosa ir Klebsiella pneumoniae patogeniškumo veiksniai ir jų įtaka ligos eigai.) [Dissertation]. Kaunas: KMU; 2008.
11. Klinikinių mikrobiologinių tyrimų metodinės rekomendacijos. Kauno medicinos universitetas Klinikinės mikrobiologijos centras, Lietuvos Respublikos sveikatos apsaugos ministerija, Higienos institutas. Vilnius; 1999.
12. Gaynes R, Edwards JR; National Nosocomial Infections

Surveillance System. Overview of nosocomial infections caused by gram-negative bacilli. Clin Infect Dis 2005;41: 848-54.
13. Karlowsky JA, Draghi DC, Jones ME, Thornsberry C, Friedland IR, Sahm DF. Surveillance for antimicrobial susceptibility among clinical isolates of Pseudomonas aeruginosa and Acinetobacter baumannii from hospitalized patients in the United Stated, 1998 to 2001. Antimicrob Agents Chemother 2003;47(5):1681-8.
14. Leroy O, Giradie P, Yazdanpanah Y, Georges H, Alfandari S, Sanders V, et al. Hospital-acquired pneumonia: microbiological data and potential adequacy of antibiotic regimes. Eur Respir J 2002;20:432-9.
15. Micek ST, Ward S, Fraser VJ, Kollef MH. A randomized controlled trial of an antibiotic discontinuation policy for clinically suspected ventilator-associated pneumonia. Chest 2004;125:1791-9.
16. Alonso A, Campanario E, Martinez JL. Emergence of multidrug-resistant mutants is increased under antibiotic selective pressure in Pseudomonas aeruginosa. Microbiology 1999;145:2857-62.
17. Hibbertrogers LCF, Heritage J, Gascoynebinzi DM, Hawkey PM, Todd N, Lewis IJ, et al. Molecular epidemiology of ceftazidime resistant Enterobacteriaceae from patients on a paediatric oncology ward. J Antimicrob Chemother 1995; 36:65-82.
18. Paterson DL, Ko WC, Von Gottberg A, Mohapatra S, Casellas JM, Goossens H, et al. Antibiotic therapy for Klebsiella pneumoniae bacteremia: implications of production of extended-spectrum β-lactamases. Clin Infect Dis 2004; 39(1):31-7.
19. Paterson DL, Ko WC, Von Gottberg A, Casellas JM, Mulazimoglu L, Klugman KP, et al. Outcome of cephalosporin treatment for serious infections due to apparently susceptible organisms producing extended-spectrum beta-lactamases: implications for the clinical microbiology laboratory. J Clin Microbiol 2001;39(6):2206-12.
20. Cryz SJ, Pitt TL, Fürer E, Germanier R. Role of lipopolysaccharide in virulence of Pseudomonas aeruginosa. Infect Immun 1984;44:508-13.
21. Dasgupta T, de Kievit TR, Masoud H, Altman E, Richards JC, Sadovskaya I, et al. Characterization of lipopolysaccharide-deficient mutants of Pseudomonas aeruginosa derived from serotypes O_3, O_5, and O_6. Infect Immun 1994;62:809-17.
22. Goossens H. MYSTIC (Meropenem Yearly Susceptibility Test Information Collection) results from Europe: comparison of antibiotic susceptibilities between countries and centre types. J Antimicrob Chemother 2000;46(Suppl B):39-52.
23. Savas L, Duran N, Savas N, Ölner Y, Ocak S. The prevalence and resistance patterns of Pseudomonas aeruginosa in Intensive care units in a University hospital. Turk J Med Sci 2005;35:317-22.
24. Leclercq R. Safeguarding future antimicrobial options: strategies to minimize resistance. Clin Microbiol Infect 2001;7: 18-23.

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