Antimicrobial resistance patterns of Pseudomonas aeruginosa isolated from canine clinical cases at a veterinary academic hospital in South Africa

Although Pseudomonas aeruginosa (P. aeruginosa) can infect both animals and humans, there is a paucity of veterinary studies on antimicrobial resistance of P. aeruginosa in South Africa. Secondary data of canine clinical cases presented at the hospital from January 2007 to December 2013 was used. The following information was recorded: type of sample, the date of sampling and the antimicrobial susceptibility results. Frequencies, proportions and their 95% confidence intervals were calculated for all the categorical variables. In total, 155 P. aeruginosa isolates were identified and included in this study. All the isolates were resistant to at least one antimicrobial (AMR), while 92% were multi-drug resistant (MDR). Most isolates were resistant to lincomycin (98%), penicillin-G (96%), orbifloxacin (90%), trimethoprim-sulfamethoxazole (90%) and doxycycline (87%). A low proportion of isolates was resistant to imipenem (6%), tobramycin (12%), amikacin (16%) and gentamicin (18%). A high proportion of MDR-P. aeruginosa isolates was resistant to amoxicillin-clavulanic acid (99%), tylosin (99%), chloramphenicol (97%) and doxycycline (96%). Few (6%) of MDR-P. aeruginosa isolates were resistant to imipenem. Pseudomonas aeruginosa was associated with infections of various organ systems in this study. All P. aeruginosa isolates of P. aeruginosa exhibited resistance to β-lactams, fluoroquinolones and lincosamides. Clinicians at the hospital in question should consider these findings when treating infections associated with P. aeruginosa.

Keywords: antimicrobial resistance; Pseudomonas aeruginosa; dogs; multi-drug resistance; veterinary.

Background

Pseudomonas aeruginosa (P. aeruginosa) is a gram-negative, saprophytic and opportunistic pathogen capable of infecting both humans and animals (Alhazmi 2015). The organism is ubiquitous in moist environments such as water and soil (Iregbu & Eze 2015).

In human medicine, P. aeruginosa has been associated with nosocomial infections of the urinary tract, surgical wounds and bloodstream (Peleg & Hooper 2010). In addition, the organism has been isolated in patients with severe burn wounds, meningitis, brain abscesses and other underlying clinical conditions (Hauser & Ozer 2011; Strateva & Yordanov 2009; Türkyilmaz 2008). In veterinary medicine, P. aeruginosa has been isolated from dogs with chronic otitis externa, pyoderma, conjunctivitis, septicemia, lower urinary tract infections, pneumonia and bacterial endocarditis (Dégi, Cristina & Stancu 2010; Petrov et al. 2013). Dogs with compromised immune systems and co-morbid conditions are at a higher risk of P. aeruginosa colonisation (Musser & Beamer 1961).

Although P. aeruginosa infections in human medicine are well documented in South Africa (Mudau et al. 2013; Odjadjare et al. 2012; Perovic et al. 2008), studies of P. aeruginosa infections in veterinary medicine could not be sourced. This is despite P. aeruginosa organisms having been reported as having high levels of resistance to commonly used antimicrobial agents such as penicillins, tetracyclines, fluoroquinolones and aminoglycosides (Prescott et al. 2003; Vingopoulou et al. 2018).

This study investigated the antimicrobial resistance patterns of P. aeruginosa from clinical samples obtained from dogs presented to a veterinary academic hospital in South Africa between January 2007 and December 2013. The results of this study will help guide empirical antimicrobial selection for the treatment of dogs infected with P. aeruginosa in veterinary medicine. In addition, the
information generated from this study will contribute to antimicrobial resistance surveillance programmes in animal health.

**Methods**

**Study area**

This study was conducted at a veterinary academic hospital located in Pretoria, South Africa. The veterinary hospital provides services for multiple veterinary disciplines, including internal medicine and surgical procedures. It offers training in companion, livestock and wildlife studies, and serves as a referral centre for complicated medical and surgical cases from other parts of the city, country and neighbouring countries.

**Data collection**

This study used secondary data of *P. aeruginosa* clinical isolates from dogs admitted to the veterinary academic hospital between January 2007 and December 2013. The hospital requires clients to sign consent forms granting the hospital permission to use information obtained from their animals for purpose of teaching and research. Information such as patient unique number, type of sample, date of sample collection, bacterial culture and antimicrobial susceptibility of the isolates was extracted from paper records submitted during the study period. The records of all the patients that yielded samples positive for *P. aeruginosa* (*n* = 155) were reviewed and included in this study.

**Bacterial isolates and antimicrobial susceptibility testing**

The bacteriology laboratory cultures all the submitted clinical samples to isolate *P. aeruginosa* using standard bacteriological methods as described by Quinn et al. (1994). Isolates were then subjected to a panel of 19 antimicrobial agents using the disk diffusion method to establish their susceptibility profiles. The bacteriology laboratory follows the Clinical Laboratory Standards Institute guidelines (Clinical Laboratory Standards Institute 2007, 2008, 2009, 2010, 2011, 2012) to isolate and conduct antimicrobial susceptibility testing.

Antimicrobials included in the test panel were the following: 30 µg-aminoglycosides, 20/10 µg ampicillin, 100 µg carbenicillin, 30 µg ceftazidime, 30 µg cephalothin, 30 µg chloramphenicol, 30 µg doxycycline, 5 µg enrofloxacin, 10 µg gentamicin, 30 µg imipenem, 30 µg kanamycin, 2 µg lincomycin, 100 µg Lincospectin, 5 µg orbifloxacin, 10 µg penicillin-G, 100 µg piperacillin, 25 µg trimethoprim-sulfamethoxazole, 20/10 µg amoxycillin-clavulanic acid, 10 µg tobramycin and 15 µg tylosin.

The laboratory classifies the results of the antibiogram as intermediate, sensitive or resistant, following the Clinical and Laboratory Standards Institute guidelines (Clinical and Laboratory Standards Institute 2007, 2008, 2009, 2010, 2011, 2012). However, for this study, isolates that had been classified as having intermediate susceptibility were reclassified as being resistant. Multi-drug resistance (MDR) was defined as resistance to at least one antimicrobial in three or more antimicrobial categories (Magiorakos et al. 2011).

Antimicrobial agents such as penicillins, cephalosporins, aminoglycosides and sulfamethoxazole-trimethoprim, which the organism showed inherent resistance to, were excluded from the MDR analysis (Pang et al. 2019). Lincospectin and lincomycin were also removed from the analysis because they are mainly efficacious against gram-positive bacteria (Farrington 2012). However, the newer generation β-lactams (imipenem) were included in the analysis because they have a broad-spectrum activity that allows them to be active against gram-negative organisms. Furthermore, amoxycillin-clavulanic acid was included in the calculation of MDR because clavulanic acid was shown to be effective against beta-lactamase enzymes.

**Data management and analysis**

The dataset was assessed for duplicates and missing information such as the lack of antibiogram results. None of the isolates had missing information and there were no duplications in the dataset.

All statistical analyses were performed using Statistical Package for Social Sciences (SPSS) (IBM SPSS statistics version 25). ‘Specimen types’ with a frequency of less than 4% were recategorised into a new category called ‘others’. Thus, the category ‘others’ included specimen types such as aspirates, semen, prostate fluid, vaginal swabs, lung or pleural fluid, bile samples, foreign-object swabs, bone, nasal swabs, tracheal aspirates and oral-cavity swabs. Descriptive statistics (i.e. frequencies and proportions) were computed and presented using tables.

**Ethical consideration**

This article followed all ethical standards for a research without direct contact with human or animal subjects.

**Results**

Thirty-four percent (34%, 52/155) of the *P. aeruginosa* isolates included in this study were recovered from ear canal samples followed by urine (22%, 34/155) and skin (10%, 16/155) samples. Abscesses contributed to the lowest proportion of isolates (4%, 6/155). Meanwhile, 30% (46/155) of the positive samples were categorised as ‘others’.

Almost all *P. aeruginosa* isolates included in this study were resistant to lincomycin (98%, 150/153), penicillin-G (96%, 148/154), amoxycillin-clavulanic acid (93%, 142/152), carbenicillin (92%, 93/101), cephalothin (90%, 140/154) and doxycycline (87%, 134/154). However, lower levels of resistance were observed to imipenem (6%, 6/100), tobramycin (12%, 12/96) and gentamicin (18%, 29/154) (Table 1).

**Multi-drug resistance**

Almost all (92%, 142/155) *P. aeruginosa* isolates were MDR, with a high proportion of these MDR isolates exhibiting
Resistance of Pseudomonas aeruginosa to β-lactams

We observed that a high proportion (86%) of P. aeruginosa isolates was resistant to piperacillin. This is contrary to the 14% piperacillin resistance amongst P. aeruginosa isolated from human cancer, burn wounds and cardiac-neuro-pediatric surgical patients observed in Kuwait (Mokaddas & Sanyal 1999). A high proportion (92%) of P. aeruginosa resistant to amoxicillin-clavulanic acid was also observed in this study, which is consistent with the observation by Gad, El-Domany and Ashour (2008) who reported 95% amoxicillin-clavulanic acid resistance amongst P. aeruginosa isolated from human patients with respiratory tract, urinary tract and skin infections in Egypt. Human studies have also reported high proportions of P. aeruginosa isolates resistant to cepazidime (Khan, Khan & Kazmi 2005; Pintari et al. 2011; Pintarić et al. 2017). This is consistent with the 77% resistance observed in this study.

The results of this study and other studies suggest that resistance against β-lactams is common amongst P. aeruginosa (Ansari et al. 2016; Mishra et al. 2012; Rafiee et al. 2014). This resistance is attributed to intrinsic resistance mediated by low membrane permeability and production of AmpC beta-lactamase amongst P. aeruginosa isolates (Pechère & Köhler 1999).

In contrast, we observed low levels (6%) of imipenem resistance amongst P. aeruginosa isolates. Our findings are consistent with the 10% imipenem resistance amongst P. aeruginosa from dogs with otitis externa in Brazil (Oliveira et al. 2005). In the light of these findings, imipenem remains the most effective drug for the treatment of β-lactam resistant P. aeruginosa and would most likely lead to a successful treatment outcome if used to treat P. aeruginosa exhibiting MDR at the veterinary hospital under study (Papp-Wallace et al. 2011).

Resistance to fluoroquinolones and tetracyclines

In this study, a higher proportion of resistance to enrofloxacin (73%) and orbifloxacin (90%) was observed amongst P. aeruginosa isolates from dogs as compared to 53% enrofloxacin resistant P. aeruginosa isolates from dogs reported by Pintarić et al. (2017). Similarly, Rubin et al. (2008)

Table 1: Antimicrobial resistance profile of Pseudomonas aeruginosa isolates from dog clinical cases tested at a veterinary academic hospital, South Africa.

| Antimicrobial category | % | n/N | 95% CI Lower | 95% CI Upper |
|-------------------------|---|-----|--------------|--------------|
| Aminoglycosides         |   |     |              |              |
| Amikacin               | 16| 26/156| 11.64 | 23.39 |
| Gentamicin             | 18| 29/154| 13.44 | 25.74 |
| Kanamycin              | 89| 134/150| 83.38 | 93.33 |
| Tobramycin             | 12| 12/96 | 7.29  | 20.59 |
| Penicillins            |   |     |              |              |
| Carbenicillin          | 92| 93/101| 85.14 | 95.93 |
| Penicillin-G           | 96| 148/154| 91.76 | 98.20 |
| Piperacillin           | 86| 80/93 | 77.54 | 91.65 |
| Amoxicillin/Ampicillin | 92| 133/144| 86.84 | 95.68 |
| Carbapenem             | 6 | 6/100 | 2.78  | 12.48 |
| Cephalosporins         |   |     |              |              |
| Cephalothin            | 90| 140/154| 85.32 | 94.51 |
| Ceftazidime            | 77| 78/101| 68.93 | 85.00 |
| Combination            |   |     |              |              |
| Amoxicillin-clavulanic acid | 93| 142/152| 88.31 | 96.39 |
| Tetracycline           | 87| 134/154| 80.79 | 91.43 |
| Amphenicols            |   |     |              |              |
| Chloramphenicol        | 89| 132/148| 83.16 | 93.24 |
| Fluoroquinolones       |   |     |              |              |
| Orbifloxacin           | 90| 137/152| 84.36 | 93.93 |
| Enrofloxacin           | 73| 113/154| 65.89 | 79.73 |
| Macrolide              |   |     |              |              |
| Tylosin-tartrate       | 92| 143/154| 87.66 | 95.96 |
| Lincomamides           |   |     |              |              |
| Lincomycin             | 98| 150/153| 94.39 | 99.33 |
| Lincomamide-aminoglycoside | 90| 138/153| 84.46 | 93.97 |

Cl, confidence interval.

Table 2: Proportions of various antimicrobials that were involved in the multi-drug resistance combinations.

| Antimicrobial agent | % | n/N | 95% CI Lower | 95% CI Upper |
|---------------------|---|-----|--------------|--------------|
| Chloramphenicol     | 97.7| 130/133| 95.2 | 100.0 |
| Doxycline           | 96.4| 134/139| 93.3 | 99.5 |
| Enrofloxacin        | 78.8| 111/139| 73.1 | 86.5 |
| Imipenem            | 6.0 | 6/99 | 1.3 | 10.7 |
| Orbifloxacin        | 96.3| 132/137| 93.2 | 99.4 |
| Amoxicillin-clavulanic acid | 99.2| 132/133| 97.7 | 100.0 |
| Tylosin             | 98.5| 137/139| 96.5 | 100.0 |

Cl, confidence interval.

Discussion

We investigated the antimicrobial resistance patterns of P. aeruginosa in samples from canine clinical cases presented at a veterinary academic hospital in South Africa. Most P. aeruginosa isolates included in this study were isolated from ear-canal swabs and urine samples. Several other studies have also reported P. aeruginosa involvement in otitis externa (Dégi et al. 2010; Mekić, Matanović & Šeol 2011; Pye 2018; Steen & Paterson 2012) and urinary tract infections in dogs (Thompson et al. 2011; Wong, Epstein & Westropp 2015). However, given that P. aeruginosa is a secondary pathogen, more clinical and laboratory information is needed to determine the significance of these results (Weese et al. 2019). Although the presence of P. aeruginosa at these body sites could be attributed mainly to the increased sensitivity to infection because of the easy access (Cabassi et al. 2017), early diagnosis and implementation of the correct treatment are still important for improved prognosis (Marza et al. 2006).
reported lower proportions of \( P. \) \( \text{aeruginosa} \) from canine clinical isolates that were resistant to enrofloxacin (31%) and orbifloxacin (52%). Although we are not able to explain the difference between our results and those of other researchers, the resistance to fluoroquinolone observed in \( P. \) \( \text{aeruginosa} \) is generally attributed to the low permeability of the bacteria’s outer membrane that limits the rate of penetration of antibiotic molecules into the cells (Nicas & Hancock 1983). We also observed a high proportion (87%) of doxycycline resistant \( P. \) \( \text{aeruginosa} \). This is comparable with the 91.07% and 99.6% resistance reported by Javiya et al. (2008) and Shah, Wasim and Abdullah (2015), respectively. Similarly, a high proportion (100%) of doxycycline resistant \( P. \) \( \text{aeruginosa} \) isolates from dogs with otitis externa was reported by Petrov et al. (2013). The high level of resistance to doxycycline observed in this study suggests that clinicians at the veterinary academic hospital under study might have to reconsider prescribing doxycycline for the treatment of \( P. \) \( \text{aeruginosa} \) infections in dogs presented at this the hospital.

**Resistance to aminoglycosides**

In comparison with resistance levels to other drugs observed and discussed above, low resistance levels to amikacin (16%), gentamicin (18%) and tobramycin (12%) were observed amongst \( P. \) \( \text{aeruginosa} \) isolates. This is consistent with the findings by Yukawa et al. (2017) who also reported low levels of \( P. \) \( \text{aeruginosa} \) resistance to amikacin (2.5%) and gentamicin (4.5%) in clinical cases of dogs and cats in Japan. Khan and Faiz (2016) also reported low proportions of \( P. \) \( \text{aeruginosa} \) isolates resistant to amikacin (7.4%) and gentamicin (11.6%) in various human clinical cases in Saudi Arabia. This is contrary to the view of some authors that \( P. \) \( \text{aeruginosa} \) tends to exhibit intrinsic resistance to aminoglycosides (Pang et al. 2019). The latter view is supported by studies that have reported very high proportions of \( P. \) \( \text{aeruginosa} \) isolates that are resistant to aminoglycosides. For example, Penna et al. (2011) in Brazil reported a high proportion of \( P. \) \( \text{aeruginosa} \) isolates from dog clinical cases that were resistant to amikacin (70%), gentamicin(71%) and tobramycin (65%). Javiya et al. (2008) in India also reported high proportions of \( P. \) \( \text{aeruginosa} \) isolates from human clinical cases that were resistant to amikacin (50%), gentamicin (67%) and tobramycin (66%). Similarly, 89% of \( P. \) \( \text{aeruginosa} \) isolates in this study were resistant to kanamycin. This is comparable with the 90% resistance to kanamycin amongst canine clinical isolates reported by Rubin et al. (2008) in the United States. The higher proportion of resistance to kanamycin compared with other aminoglycosides that was observed in this study could be attributed to chromosomal \( \text{aphA} \)-encoded aminoglycoside phosphoryl transferase (\( \text{APH(3')} \) llb), which are enzymes that inactivate the action of antimicrobials, leading to resistance (Morita, Tomida & Kawamura 2013). Therefore, the results of this study support the theory of variations in the susceptibility of \( P. \) \( \text{aeruginosa} \) to different aminoglycosides based on their mechanism of action. In view of this, the observations cast doubt on the efficacy of kanamycin in the treatment of \( P. \) \( \text{aeruginosa} \) infections amongst clinical cases presented at the veterinary academic hospital (Poole 2005). Despite the widely accepted view that \( P. \) \( \text{aeruginosa} \) exhibits intrinsic resistance to aminoglycosides, available evidence suggests aminoglycosides such as amikacin or gentamicin are useful in the treatment of respiratory infections (Poole 2005). Furthermore, commercial topical preparations for treatment of ear infections also contain aminoglycosides that are known to be effective (Boyd, Santoro & Gram 2019).

**Multi-drug resistance**

Overall, 92% of \( P. \) \( \text{aeruginosa} \) isolates included in this study were MDR. Several other authors have also reported MDR levels of up to 97.9% amongst \( P. \) \( \text{aeruginosa} \) clinical isolates from humans (Shokri et al. 2016). In contrast, other authors have reported low proportions of MDR-\( P. \) \( \text{aeruginosa} \), ranging from 14% to 29% in human studies conducted in Pakistan and Saudi Arabia (Gill et al. 2011; M.A. Khan & Faiz 2016; Tam et al. 2010; Ullah, Malik & Ahmed 2009).

*Pseudomonas aeruginosa* is known to exhibit intrinsic resistance against \( \beta \)-lactams, fluoroquinolones, tetracyclines, aminoglycosides and lincosamides (Iregbu & Eze 2015; Mekić et al. 2011; Steen & Paterson 2012; Türkyılmaz 2008). Intrinsic resistance is caused by factors such as low outer membrane permeability, the production of AmPc \( \beta \)-lactamase and the presence of efflux systems MexA-MexB-OprM, MexC-MexD-OprJ, MexE-MexF-OprN and MexX-MexY-Op (Morita et al. 2001, 2013). Because drugs against which \( P. \) \( \text{aeruginosa} \) exhibiting intrinsic resistance were not included in the determination of MDR, the high proportion of MDR-\( P. \) \( \text{aeruginosa} \) (92%, 142/155) observed in the present study is most likely explained by acquired resistance. This view is supported by the observation that antimicrobials that were frequently involved in MDR combinations such as enrofloxacin, imipenem, orbifloxacin and amoxycillin-clavulanic acid are drugs that \( P. \) \( \text{aeruginosa} \) is known not to exhibit intrinsic resistance to. Furthermore, because the veterinary hospital where the study was conducted is a teaching and referral hospital, it is also possible that by the time most of the dogs from which the samples were collected were presented at the hospital, they would already have been exposed to antimicrobial treatment. It is known that exposure to antimicrobials is a risk factor for the development of resistance. However, it is not possible to confirm this assertion because of lack of information on previous antimicrobial exposure amongst the dogs that were sampled.

**Limitations of the study**

The study was limited to only one veterinary academic hospital and did not include isolates from other veterinary hospitals in the vicinity of the study area. In view of this, findings reported in this study cannot be generalised to the whole of the Gauteng province. A history of previous antimicrobial usage amongst the dogs tested was also not available to the researchers; therefore, it was not possible to
Data availability statement

The dataset that supports the findings of this study is available from Prof. Daniel Nenene Qekwana at the University of Pretoria and all the documentations have been approved and are in line with the regulations of the University of Pretoria.

Disclaimer

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