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The Prevalence Of Multi-Drug Resistant *Pseudomonas Aeruginosa* In The Burn's Unit Of Pelonomi Hospital.

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Abstract

Introduction

The occurrence of multi-drug resistant Pseudomonas aeruginosa is increasing more daily that possess potential health threat worldwide. Research shows that the resistance occurs through the overuse and misuse of broad-spectrum antibiotics. This study will determine the prevalence of multi-drug resistant bacteria in the burn's unit of Pelonomi hospital.

Methodology

This is a cohort observational study and therefore data was only collected and observed. The data was collected for all patients 18 years and older that were admitted to the burn's unit of Pelonomi hospital that tested positive for Pseudomonas aeruginosa and that was tested for sensitivity. A total of 85 samples were collected for this study over 4 months because this was done as part of 4th year module for the qualification.

Results and discussion

As part of inclusion criteria all 85 patients that were admitted to the burn's unit of Pelonomi hospital tested positive for Pseudomonas aeruginosa. The sensitivity profile of each patient was recorded for each antibiotic and subjected to statistical analysis. The outcomes would then be classified as resistance, intermediate, and sensitive when compared to the interpretation standards. The observation and calculations on each antibiotic tested, sulfamethoxazole was found to be the least active antibiotic on all the samples (100%), tazobactam resistance (48.24%), imipenem (17.65%), ceftazidime (11.76%), cefepime (8.24%), meropenem (5.88%) and ciprofloxacin and amikacin had (2.35%) resistance each. There was an overall resistance of Pseudomonas aeruginosa towards all their antibiotics of 24.56% and a sensitivity of 70.15% and 5.29% was intermediate. The results clearly show that there is a high percentage of resistance towards antibiotics that are supposed to work against the bacterium.

Conclusion

The prevalence of antimicrobial resistant P. aeruginosa in the burn's unit of Pelonomi hospital between the months of 1 March 2023 and 31 August 2023 was 24.56%. This indicates that there is an overall resistance to the antibiotics that are supposed to work against the bacterium. The results correlate with what was reported by Moradali et al showing that P. aeruginosa is associated with mortality in immunocompromised patients and its presence needs monitoring (Moradali et al., 2017). This poses as a threat for the further development of resistance against these antibiotics and is live threatening for patients that already have a compromised immune system in South Africa.

Keywords: *Pseudomonas aeruginosa, antimicrobial resistance, multi-drug resistance.*

INTRODUCTION

Globally there is an increase in antimicrobial resistant organisms which is attributed by the development of resistance to broad-spectrum antibiotics due to the misuse and non-adherence to treatment by the patients (Davids, 2021). Resistance to antibiotics occurs when bacteria does not respond to the antibiotics that are developed to kill it and they survive in the presence of the antibiotics and develops genes that codes for resistance (CDC, 2021). These types of organisms, cause infections that are difficult, to treat and causes longer hospital stays that are unnecessary including additional follow-up doctor visits, and it could even be fatal (CDC, 2021). South Africa is the country that imports the highest amount of antibiotics in the world (21 149 standard units per 1000/population more than other countries) and around 80% of the antibiotics that are prescribed are broad-spectrum (Health, 2018). One organism that can be seen as a leading cause of mortality and morbidity in South

Africa is *Pseudomonas aeruginosa* (Moradali , et al., 2017). This is an opportunistic pathogen that infects especially immunocompromised individuals and is also a commonly seen hospital acquired infection (Moradali , et al., 2017). This type of bacteria can be seen as a health threat worldwide due to the fact that it is becoming resistant to more types of antibiotics, its mechanisms for adaptation and survival is causing this (Moradali , et al., 2017). A study that was done in 2021 revealed that there was 7.5 million people of all ages that are HIV positive in South Africa at that exact moment (UNAIDS, 2023). Another study that was performed also revealed that another 304 000 South African citizens have TB (Voight, 2022). Without including any other disease that makes people immunocompromised (for example cancer) it is estimated that in 2021 around 13% of the South African population was immunocompromised either due to HIV or TB (Voight, 2022).

Since *Pseudomonas aeruginosa* is an organism that causes opportunistic infections in immunocompromised individuals it is a threat for people who are immunocompromised. *Pseudomonas aeruginosa* are becoming more resistant to a whole list of antibiotics that are supposed to work and this is deadly to all immunocompromised individuals (Voight, 2022). The prescriptions for antibiotics by health professionals should be decided based on a test that was done for antimicrobial susceptibility to ensure that the correct treatment is provided for the patient. If the wrong antibiotics are being prescribed to the patient it will only cause the bacteria to become even more resistant and it could lead to death of the patient. This research will be done to proof to healthcare professionals that *Pseudomonas aeruginosa* is already resistant to a wide range of antibiotics and that the prescriptions should be given with caution to patients to prevent any further resistance and prevent *Pseudomonas aeruginosa* from becoming resistant to all types of antibiotics.

The results of this research will prove that *Pseudomonas aeruginosa* is becoming multi-drug resistant. This will make healthcare professionals and also members of the public aware of the dangers of this occurring since it could cause havoc on our health system if there is an outbreak of this type of *Pseudomonas aeruginosa* that is resistant to all types of antibiotics. So therefore, care should be taken by health professionals to ensure that the necessary tests have been done to ensure that the correct antibiotics are being prescribed to the patient for this type of infection. If this research is taken seriously and healthcare professionals decide to confirm the type of *Pseudomonas aeruginosa* is sensitive to the type of antibiotic, they want to prescribe it will slow down the development of multi-drug resistance and reduce the number of mortality and morbidity caused by this type of bacteria. Another benefit is that this research will reduce the longer hospitals stay in public hospitals that will free up more funding from the government that could be allocated else whare in the hospitals where there is a lack of funding for example at clinics. The effect of this will only be seen in the following 10 to 15 years if all general practitioners in South Africa decide that they want to help fight this unseeable enemy of antimicrobial resistant *Pseudomonas aeruginosa*.

Problem statement:

Pseudomonas aeruginosa is globally known to cause problems in the hospital setting with most health care workers being asymptomatic carriers of it. The last study done by Moradali et al in 2017 showed that this organism needs monitoring as it causes a lot of mortality in immunocompromised patients. In this study we want to establish the presence of *Pseudomonas aeruginosa in* Pelonomi hospital burn unit and determine its resistance profile.

Aim

The research aim was to do observatory study and determine the bacterial profile and the prevalence of antimicrobial resistant *Pseudomonas aeruginosa* in Pelonomi hospital from the burn's unit during the months of March until August of 2023.

Objective

1. To determine the organism(s) associated with infection in the burn's unit at Pelonomi hospital.
2. To record and perform statistical calculation to determine the prevalence of antimicrobial resistant pattern among *Pseudomonas aeruginosa* between the months of March until August of 2023.

LITERATURE REVIEW

Pseudomonas is a bacterium that can be found in the environment such as in the soil or in water. But Pseudomonas aeruginosa is the most common organism that causes infections in the lungs, blood or other parts of the human body post-surgery. This bacterium lives in environments such as soil, water and on surfaces in hospital environments. So, this can also be spread in the environment from person to person through contaminated surfaces, equipment or hands. The patients in hospitals that are on breathing machines, patients that have devices in them such as catheters and burns patients or patients that had surgery are more at risk for infection (Centre for Disease Control , 2019).

Most infections occur in hospitals and therefore healthcare providers are responsible for preventing patients from getting this infection. When treating and touching patients they should clean their hands before touching a patient by washing it with water and soap or by using alcohol-based hand sanitizer. Constant cleaning of medical devices should be performed before every procedure and in between patients. All surfaces in the hospital, including the patients' rooms should be cleaned on a regular basis to decrease the growth of this bacterium. Another way to reduce the risk of infection is to ensure that water management plans are in place to identify this organism and reduce the risk of spreading the bacterium (Centre for Disease Control , 2019).

Pseudomonas aeruginosa infection can be seen as a nosocomial infection, in other words it is a healthcare-associated infection. This type of infection is obtained when a patient is receiving health care, but the infection was not present before this period. This could occur during hospital stays, in an ambulance, long term care facilities and it normally appears after discharge. Daily staff members in hospital environments are also susceptible to these types of infections. In order for a patient to be susceptible to these infections they should have a weakened immune system. Surgery is also a common mode of transmission, along with indwelling medical devices and prosthetic devices. A nosocomial infection is not limited to bacterial infection, but it could also be fungal or viral. These types of infections affect patients' safety and cause increased morbidity and mortality, not even to mention the financial and emotional burden that is placed on these patients and their family members. Due to the increase in nosocomial infections, there is also a tremendous increase in multi-drug resistant organisms. These types of infections affect 3.2% of all the patients that are hospitalized in the United States and 6.5% of patients in the European region, but the worldwide prevalence is much higher than this. Due to the lack of surveillance systems in the world for nosocomial infections, it is not clear how this affects patients across the world (Sikora & Zahra, 2023).

Patients that are infected through hot tubs or pools get a type of infection called *Pseudomonas* folliculitis. The main characteristics are breast tenderness, fever, maculopapular pruritic rash and axillary lymphadenopathy. Another scenario is when the infection is contracted through a puncture wound in the foot by a piece of glass or a nail. There might be a delay in signs and symptoms, and this may cause a delay in diagnosis that causes serious complications that includes osteomyelitis or septic arthritis. But symptoms could also include purulent discharge and local tenderness around the wound area. In 60% of patients with cystic fibrosis chronic infection occurs with *Pseudomonas aeruginosa*. This causes a higher mortality rate.

Pseudomonas aeruginosa is also commonly seen in burns patients and the common signs are a purulent discharge that is green blue in color with local and systemic inflammation that could progress to sepsis and eventually septic shock.

Patients that have diabetes are prone to develop malignant otitis externa that reveals granulated tissue inside the ear that is caused by *Pseudomonas aeruginosa.*

All patients that had organ transplants are at high risk for developing *Pseudomonas aeruginosa* infection after surgery especially urinary tract infections due to catheters. Patients with *Pneumoniae* are also at a higher risk for infection by *Pseudomonas aeruginosa* due to their compromised immune system. (Wilson & Pandey , 2022)

What is a multi-drug resistant bacterium and how is *Pseudomonas aeruginosa* treated?

In the laboratory specialized methods are used to grow microorganisms by using growth mediums known as cultures. This is then used to determine the resistance and sensitivity of these pathogens to a wide range of antimicrobial agents. Thereafter a list of appropriate antimicrobial agents is sent to the doctor as a guide to what antibiotics could be used to treat the patient. The appropriate name for this procedure is antimicrobial susceptibility testing. This is performed by medical technologists (or also called medical laboratory scientists). When looking at the larger picture, this procedure is performed to ensure that patients receive the appropriate treatment and to prevent the rapid spread of infections and the development of multi-drug resistant bacteria. There are continuous surveillance systems in place for the resistance patterns due to mutations in bacterial DNA. The two main methods that are used in laboratories include the minimum inhibitory concentration method (this is done to determine the minimum concentration of antibiotics that could be used to kill the bacterium) and the disk diffusion method to determine what type of antibiotics would kill the bacterium (Bayot & Bragg, 2022).

Although it is always advised that antimicrobial susceptibility testing should be performed before treatment is started, there are still some agents that could provide coverage for the bacterium. These agents include cephalosporins, carbapenems, aminoglycosides and fluroquinolones. These bacteria are used as the first line of therapy until the sensitivity and culture results are available. When other drugs are given to patients without determining what bacterium it is, or what antibiotics would word it causes resistance in these bacteria. The

bacteria adapt and grow in the presence of the antibiotics and through this method it develops resistance. Multi-drug resistance is when a bacterium is resistant to most of the drug that are supposed to work for it, and this makes treatment increasingly difficult for these patients (Wilson & Pandey , 2022).

What is the mechanism of action for Pseudomonas aeruginosa to develop resistance?

This bacterium does not have one mode of resistance, but an entire list of mechanisms to make it extremely resistant to a whole range of antibiotics. The first type of resistance is due to adaptive resistance, and this includes continuous exposure to antibiotics and overexposure to environmental stress. The second type acquired resistance due to a mutation and horizontal gene transfer. And the last type is intrinsic resistance by antibiotics inactivating enzyme, over expression of efflux pump and low outer membrane permeability (Pachori , et al., 2019).

Efflux system

This mechanism is part of Pseudomonas aeruginosa, and it expresses this system that expels certain drugs and other substances out of the cell through the bacterial cell wall (Pachori , et al., 2019).

ß-Lactam resistance

The group of ß-lactam antibiotics include penicillin's, 3rd generation cephalosporins (ceftazidime), monobactam (aztreonam), cefepime (4th generation cephalosporins), imipenem, meropenem and doripenem. There are the antibiotics that are most commonly used, and are the most effective, against *Pseudomonas aeruginosa*. *Pseudomonas aeruginosa* contains an enzyme known as ß-lactamases that are able to break the ß-lactam ring in the antibiotic and through this inhibiting it. Until thus far there has been four classes of ß-lactamases identified (A, B, C, and D) each with its own individual mode of action (Pachori , et al., 2019).

AmpC Beta lactamase

Benzyl penicillin, narrow spectrum cephalosporin and imipenem induces the production of endogenous beta lactamase such as AmpC beta lactamase in *Pseudomonas aeruginosa*. Although *P. aeruginosa* is susceptible to aztreonam, carboxypenicillins and ceftazidime it is also able to acquire resistance by gene mutations that causes AmpC beta-lactamase hyper production. The bacterium produces an inducible chromosome-encoded AmpC beta-lactamase and this is normally produced in low quantities and determines resistance to majority of the early cephalosporins and aminopenicillins. In the presence of inducing beta-lactams the production of chromosomal cephalosporins could increase from 100 to 1000 times. Beta-lactamase inhibitors does not inhibit the AmpC cephalosporinase activity (Pachori , et al., 2019).

Resistance to aminoglycosides

Microbial protein synthesis is inhibited by the aminoglycosides that causes binding to the bacterial 30s ribosomal subunit and the protein synthesis cannot be initiated then. Transferable aminoglycoside modifying enzymes (AME's), efflux systems, low membrane permeability and rarely target modification mediate the resistance to aminoglycoside in *Pseudomonas* (Pachori , et al., 2019).

Aminoglycoside modifying enzymes

The AMEs attaches a phosphate, adenyl or acetyl radical of the antibiotic molecule to inactivate the aminoglycoside. And therefore, the modified antibiotics have a decreased binding affinity to its corresponding target inside the bacterial cell wall (the 30S ribosomal subunit) (Pachori , et al., 2019).

Low membrane permeability

In cystic fibrosis patients, clinical isolates have been reported to have resistance to aminoglycosides independent from AMEs. This is due to impermeability resistance and this type of resistance is when there is reduced uptake through a decrease in the permeability of the membrane (Pachori , et al., 2019).

Target modification

A low affinity of certain drugs for the bacterial ribosome causes bacteria to be resistant to aminoglycosides. Methylation of the 16S rRNA is done to accomplish this target modification. For Pseudomonas aeruginosa there have been different 16S rRNA methylases described. Firstly, RmtA is one that has been reported in aminoglycoside resistant clinical isolates and it confers resistance to all parenterally administered aminoglycosides (this includes tobramycin, amikacin, kanamycin, arbekacin, gentamycin and isepamicin). There are also other 16S rRNA methylases such as RmtB, RmtD and ArmA (29) (Pachori , et al., 2019).

METHODOLOGY

Study location

The study location was Pelonomi hospital burn's unit This site was chosen since it will give a more accurate representation of urban and rural populations in the Free State since majority of people go to Pelonomi hospital.

This will also give an accurate representation of all people from different backgrounds and rases. This is not only a specialist hospital but also a hospital that gives training to health professionals.

Study design

This is a cohort observational study. This means that all the tests have already been done for the first 9 months of the year and this is the data that will be used and no changes to this data will be made. This study is also quantitative since it will be focusing on the amount of *Pseudomonas aeruginosa* that are antimicrobial susceptible, resistant, or intermediate in nature. All of the participants that were chosen for this study must be 18 years or older and a patient in the burn's unit of Pelonomi hospital between 1 March 2023 and 31 August 2023.

Study population

The number of participants

The number of patients in the burn's unit of Pelonomi hospital increases drastically over the winter period. This is also the reason why this study was chosen over the months of March and August to have the most patients possible included in the study. There were no certain number of participants chosen for this study. A guide was however chosen to say that all patients admitted into the burn's unit over the age of 18 years old between 1 March 2023 and 31 August 2023 will be included in this study if they tested positive for *Pseudomonas aeruginosa*.

Participant identification

The researcher went through all of the patients results on the NHLS laboratory's computer filing system (LIS) and only chose the patients that were admitted in the burns unit over the age of 18 years old between 1 March 2023 and 31 August 2023 will be included in this study if they tested positive for *Pseudomonas aeruginosa*.

In- and exclusion criteria

Inclusion criteria

1. Patients admitted into Pelonomi hospital burns unit between 1 March 2023 and 31 August 2023.
2. The patient must be 18 years or older.
3. The patient must have tested positive for *Pseudomonas aeruginosa*.

Exclusion criteria

1. Patients that were not admitted to the burn's unit, or that was not admitted in the correct time interval.
2. The patient was below 18 years of age.
3. The patient did not test positive for *Pseudomonas aeruginosa*.

METHODS AND MATERIAL

Preparation

A data sheet was compiled by the researcher to collect the data for this study. On this sheet there was no private information regarding the patients and a new number was given to each patient rather than using their laboratory number. The simulated data was also provided by the study leaders, so no patient information was used for this research.

Data collection

The data was collected by the researcher on a weekly basis. The samples are processed every Thursday in the laboratory, so every Monday the data was collected. This was done by going through all of the forms for that day and only identifying the patients that meet the inclusion criteria. The information that was captured on the data collection sheet was the patient's laboratory number and also the antimicrobial sensitivity testing results.

Data management and statistical analysis

There will be primary data collected from the laboratory for the swabs that was sent to be tested for *Pseudomonas aeruginosa* antimicrobial susceptibility of 1 March 2023 until 30 August of 2023. A copy will be made of the results for each patient that was tested for *Pseudomonas aeruginosa* antimicrobial susceptibility. The copy that is made is only of the back form, this only gives the patient laboratory number with the results of the tests performed. This information is then captured on the data collection sheet that was compiled on a word document. The computer is password protected.

When *Pseudomonas aeruginosa* is tested it is tested for susceptibility of 8 types of antibiotics. So, for each antibiotic a percentage will be calculated and displayed in a pie chart to see the percentage of resistant and the percentage of sensitive bacteria for that specific type of antibiotic. Another last calculation will be done to show the total percentage of bacterial resistance for *Pseudomonas aeruginosa.*

ETHICAL ASPECTS AND GOOD CLINICAL PRACTICE

Ethical clearance

Ethical approval was requested from the Health Sciences Research Ethics Committee (HSREC) and permission from the manager of Pelonomi National Health Laboratory Services (NHLS) was granted. The research only commenced once the necessary permission was granted, but for this research simulated data was used.

Safety variables

Project safety

There were no interviews carried out in this study and all of the data that was collected for the patients was kept confidential.

Patient's safety

This research poses no risk to patients since only patient information was gathered.

Premature discontinuation of the study

This study was not prematurely discontinued since it did not pose risk or harm any patients and the patient's confidentiality was maintained at all times.

Good clinical practice (GCP) / Quality assurance

This study was subject to the GCP guidelines and the declaration of Helsinki's basic principle number three states that scientifically qualified people should conduct research, and this should be done under the supervision of adequately qualified people.

Financial implications to the participant

There were no financial implications to the patients.

Withdrawal criteria

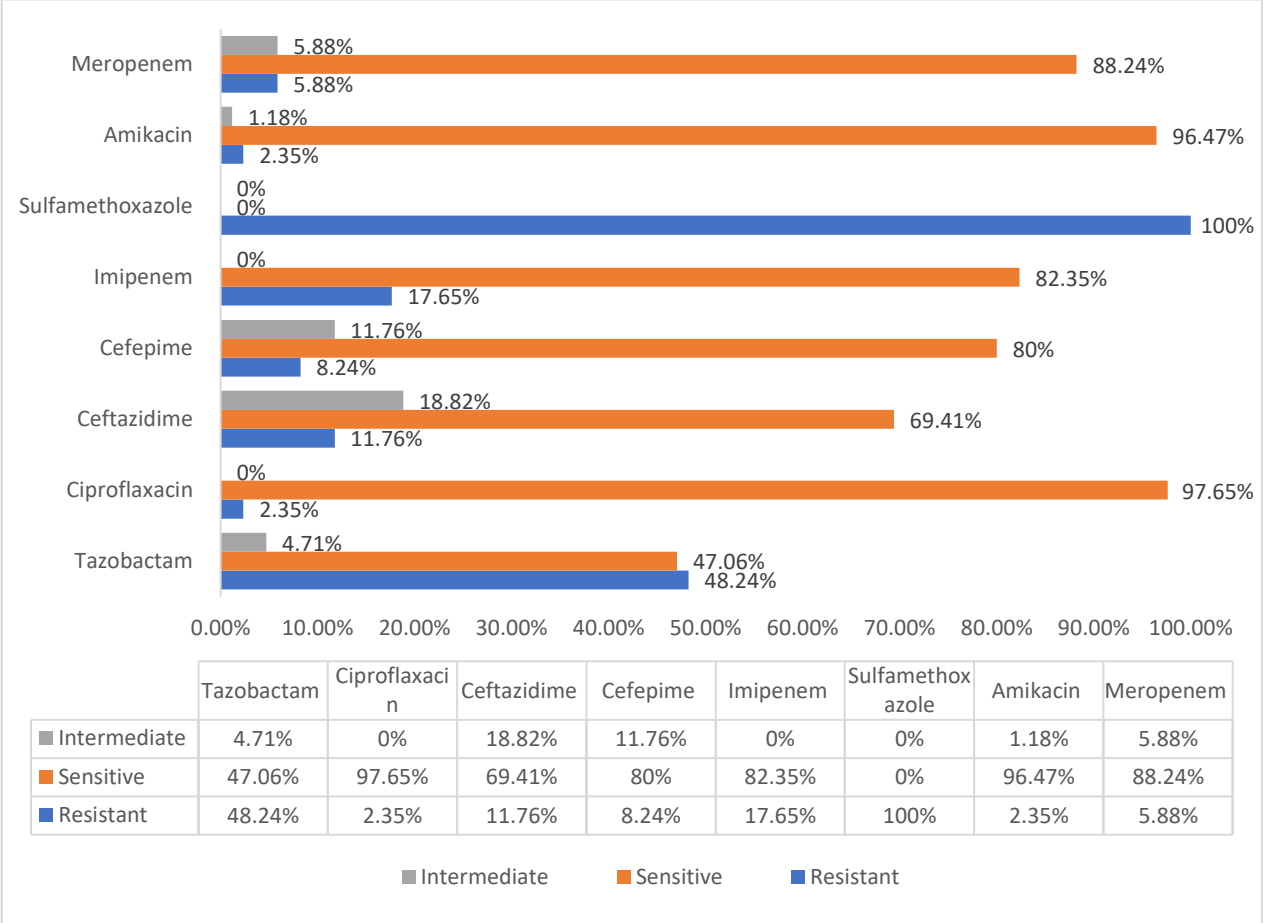
Not applicable to this study.

Subject information and informed consent

Not applicable to this study.

Confidentiality

Each patient's hospital number was replaced with a number according to the order in which the data was collected. The data was kept in a secure folder on a password-protected computer.



	Tazobactam	Ciproflaxacin	Ceftazidime	Cefepime	Imipenem	Sulfamethoxazole	Amikacin	Meropenem
Intermediate	4.71%	0%	18.82%	11.76%	0%	0%	1.18%	5.88%
Sensitive	47.06%	97.65%	69.41%	80%	82.35%	0%	96.47%	88.24%
Resistant	48.24%	2.35%	11.76%	8.24%	17.65%	100%	2.35%	5.88%

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RESULTS

For the months between 1 March 2023 and 31 August 2023 there was a total of 85 patients that were admitted to the burn's unit of Pelonomi hospital and tested positive for *Pseudomonas aeruginosa*. For each patient the sensitivity and resistance were tested for *P. aeruginosa* and the results were obtained. The percentage of resistance, sensitivity and intermediate was calculated for each antibiotic. This table can be seen as the bacterial profile for *P. aeruginosa* that was tested for sensitivity over these months.

The *P. aeruginosa* was 88.24% sensitive and a 5.88% resistant to meropenem, 96.47% sensitive and 2.35% resistant to amikacin, 0% sensitive and 100% resistant to sulfamethoxazole, 82.35% sensitive and 17.65% resistant to imipenem, 80% sensitive and 8.24% resistant to cefepime, 69.41% sensitive and 11.76% resistant to ceftazidime, 97.65% sensitive and 2.35% resistant to ciprofloxacin and 47.06% sensitive and 48.24% resistant to tazobactam.

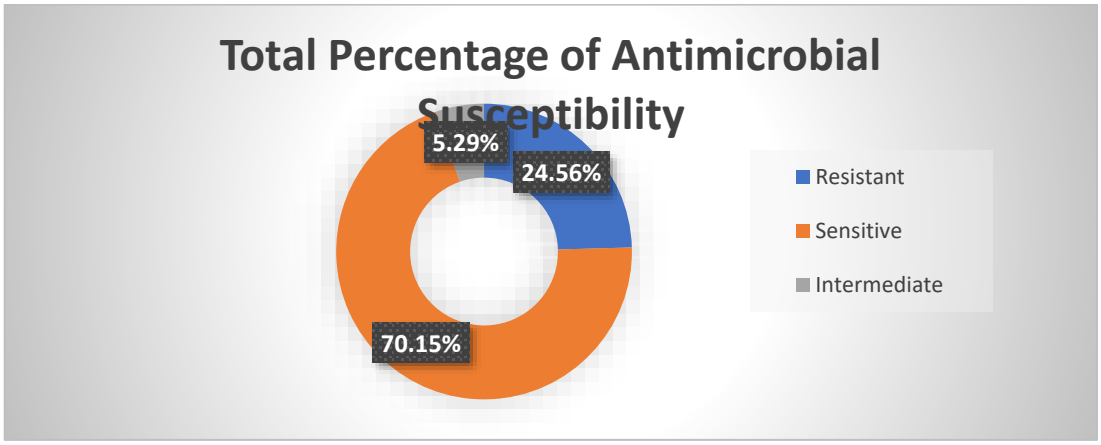

Figure 1: Total percentage of antimicrobial susceptibility

This pie chart can be used to determine the prevalence of resistant *P. aeruginosa*. When looking at the total profile of the resistance pattern for all of the antibiotics together it was noted that *Pseudomonas aeruginosa* was 24.56% resistant to the antibiotics and 70.15% sensitive to the antibiotics it was tested for and 5.29% intermediate. The antibiotics for which it was tested for are all supposed to kill the bacterium, so a 24.56% resistance is concerning since it is not supposed to be this high due to the fact that it is supposed to be sensitive to all of the antibiotics.

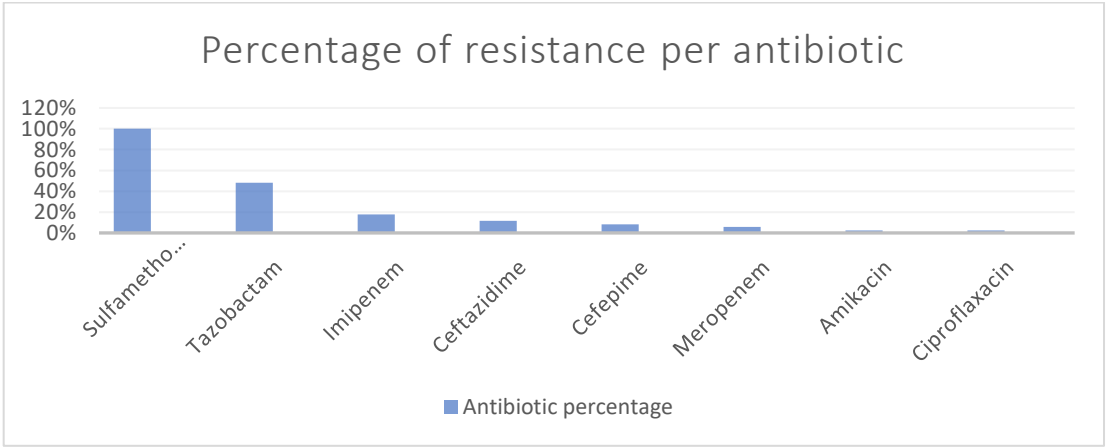

Figure 2: Resistance percentage per antibiotic

The resistance pattern of *P. aeruginosa* is shown in this table by sorting the antibiotics from the highest percentage of resistance to the lowest percentage. In descending order, it can be seen that *P. aeruginosa* had the highest level of resistance towards sulfamethoxazole/ trimethoprim at 100%, then tazobactam at 48.24%, imipenem at 17.65%, ceftazidime at 11.76%, cefepime at 8.24%, meropenem at 5.88% and lastly amikacin at 2.35% and also ciprofloxacin at 2.35%. It can clearly be seen in the data that due to the fact that *P. aeruginosa* had a 100% resistance towards sulfamethoxazole/ trimethoprim that it could no longer be considered as an antibiotic to treat the bacterium since there was not one case over these months where it was sensitive towards the antibiotic.

DISCUSSION

P. aeruginosa is an opportunistic pathogen that infects immunocompromised individuals and is also a commonly seen hospital acquired infection. This type of bacteria can be seen as a health threat worldwide due to the fact that it is becoming resistant to more types of antibiotics, its mechanisms for adaptation and survival is causing this (Moradali , et al., 2017). A study that was done in 2021 revealed that there was 7.5 million people of all ages that are HIV positive in South Africa at that exact moment (UNAIDS, 2023). Another study that was performed also revealed that another 304 000 South African citizens have TB (Voight, 2022). Without including any other disease that makes people immunocompromised (for example cancer) it is estimated that in 2021 around 13% of the South African population was immunocompromised either due to HIV or TB. Each patient that tested positive for P.aeruginosa should firstly be tested for sensitivity or resistance before any type of antibiotic could be given to the patient to ensure that the bacterium does not develop any further resistance. If any of these patients that are immunocompromised are admitted into the hospital and obtain this nosocomial infection it could be life threatening to them. This could cause a strain on the government due to extra funding being spent to treat these patients and it could also cause a lot of unnecessary deaths.

There is a long list of antibiotics that are prescribed against *Pseudomonas aeruginosa*. But the bacterium should firstly be tested for sensitivity to determine what antibiotics and at what concentrations would kill the bacteria. These antibiotics include carbapenems (imipenem, meropenem), cephalosporins (Amikacin, ceftazidime, cefepime,), quinolone (ciprofloxacin), beta-lactam inhibitors (tazobactam) and sulfonamide (sulfamethoxazole/ trimethoprim) (Wilson & Murray, 1998).

Piperacillin/Tazobactam is part of the beta-lactamase inhibitor class. Tazobactam is generally not prescribed alone and is usually combined either with piperacillin or ceftolozane due to the fact that tazobactam has little antimicrobial activity by itself. The combination of the two antibiotics makes them effective at bacteria that expresses beta-lactamase. The antibiotic combination would degrade the bacteria by inhibiting the beta-lactamase enzymes irreversibly (Personal Helath Analytics , 2022).

Amikacin is an aminoglycoside and causes the inhibition of bacterial protein synthesis. It achieves this by binding the 30s ribosome subunit causing a misreading of the bacterial mRNA. This then results in the production of abnormal peptides that gather in the bacterial cell eventually causing cell death (Moore , 2023).

Imipenem and meropenem are carbapenems and they function by inhibiting cell wall synthesis. Penicillin binding proteins (PBP) are enzymes that catalyze the formation of peptidoglycan in the bacterial cell wall. The peptidoglycan in the cell wall helps maintain cell integrity and protects the bacteria from external stressors. When the carbapenems binds the PBP's it weakens the peptidoglycan and eventually the bacterial cell will burst due to the osmotic pressure (Bonomo , et al., 2011).

Cefepime is a fourth-generation cephalosporin and ceftazidime is a third generation cephalosporin. Fourth generation cephalosporins have the same coverage as the third generation but with additional coverage against gram negative bacteria with resistance. Cephalosporins are bactericidal and are a type of beta-lactam antibiotic. The antibiotic disrupts the peptidoglycan layer synthesis of the bacterial cell wall. The peptidoglycan is an important structure of the bacterial cell wall and when it is compromised the entire cell wall will be compromised resulting in lysis and then death of the cell. Human cell do not have peptidoglycan and are therefore not targeted by the antibiotics (Port, 2015).

Ciprofloxacin is a second-generation fluroquinolone. These are the only class of antimicrobial agents that directly inhibits bacterial DNA synthesis. DNA gyrase and topoisomerase IV are the target of the antimicrobial agent and bind to specific domains of the DNA. The results are blockage of DNA replication apparatus and produces double breaks in the DNA that are important in bacterial activity (Hooper & Jacoby , 2016).

The antibiotic that had the most resistance was sulfamethoxazole/ trimethoprim when looking at figure 1. During previous research that was done on antibiotics it was noted that bacteria are obligate synthesizers for folic acid. These two antibiotics are used together to achieve the best possible results. The physiologically active form of folic acid is tetrahydrofolic acid. This is used in the production of purines, thymidine, and the DNA of bacteria. The combination of sulfamethoxazole/ trimethoprim inhibits bacterial synthesis by inhibiting the synthesis of tetrahydrfolic acid. Sulfamethoxazole inhibits the synthesis of dihydrofolic acid from its precursor. Trimethoprim competitively inhibits dihydrofolate reductase since it is a structural analogue of the pteridine portion of dihydrofolic acid. As a consequence, tetrahydrofolic acid is inhibited from being produced from dihydrofolic acid. The combination of these antibiotics blocks 2 enzymes in one pathway and therefore the organism dies (Masters , et al., 2003).

When looking at how the bacteria developed resistance there is 5 main mechanisms seen: acquired resistance by drug-resistant target enzymes, changes in the target enzyme, mutational changes or recombination in the target enzymes, naturally insensitive target enzymes and lastly the permeability barrier and/or efflux pumps. When looking at *P.aeruginosa* its mechanisms of resistance are due to an efflux pump and a permeability barrier to sulfamethoxazole/ trimethoprim. The influence of these two mechanisms are difficult to separate and therefore it is said that *P.aeruginosa* uses both these mechanisms as a form of resistance (Eliopoulos & Huovinen , 2001).

CONCLUSION

The prevalence of antimicrobial resistant *P.aeruginosa* in the burn's unit of Pelonomi hospital between the months of 1 March 2023 and 31 August 2023 was 24.56%. This indicates that there is an overall resistance to the antibiotics that are supposed to work against the bacterium. This poses as a threat for the further development of resistance against these antibiotics and is live threatening for patients that already have a compromised immune system in South Africa.

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Conflict of interest
No conflict of interest

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