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By Universitas Muhammadiyah Sidoarjo

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Isolation and Antibiotic Susceptibility Patterns of Pseudomonas aeruginosa From the Patients with Respiratory Tract Infection

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Abstract

General Background: Respiratory tract infections caused by bacterial pathogens contribute significantly to global morbidity and mortality, with *Pseudomonas aeruginosa* being a major nosocomial pathogen frequently isolated from respiratory infections. This Gram-negative bacterium exhibits intrinsic antibiotic resistance and can develop multiple resistance mechanisms, making treatment increasingly challenging. **Knowledge Gap:** Although *P. aeruginosa* is recognized for multidrug resistance, periodic monitoring of antibiotic susceptibility patterns in specific geographic regions remains essential as resistance profiles vary temporally and geographically. Aims: This study aimed to isolate and identify *P. aeruginosa* from sputum samples of patients with respiratory tract infections and determine antibiotic susceptibility patterns against thirteen commonly used antibiotics. **Results:** Among 110 sputum samples collected from Al-Diwaniyah Teaching Hospital (March-July 2025), *P. aeruginosa* was isolated from 33.33% of culture-positive samples, demonstrating highest resistance to ceftazidime (76.92%), cefepime (73.07%), and levofloxacin (76.92%), while showing greater sensitivity to amikacin (61.53%) and colistin (53.84%). **Novelty:** This investigation provides current antibiotic resistance data specific to the Al-Diwaniyah region, revealing alarmingly high resistance rates to modern-generation cephalosporins. **Implications:** These findings necessitate updating regional antibiotic treatment protocols for respiratory infections and recommend amikacin-colistin combination therapy to mitigate further resistance development.

Keywords : Pseudomonas Aeruginosa, Antibiotic Resistance, Respiratory Tract Infections, Antimicrobial Susceptibility, Multidrug Resistance

Highlight :

- P. aeruginosa accounted for 33.33% of bacterial respiratory tract infections studied.
- Highest resistance observed to Ceftazidime (76.92%), Levofloxacin (76.92%), and Cefepime (73.07%).
- Amikacin and Colistin demonstrated greatest effectiveness with 61.53% and 53.84% sensitivity rates.

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Introduction

Bacterial pathogens that induced respiratory tract infections are common worldwide and raise rates of morbidity and mortality, therefore identification of these bacterial pathogens is important for treatment management and prevention [1]. Both Gram-positive and Gram-negative bacteria, which include a wide variety of bacterial genera, are significant bacterial pathogens of respiratory tract infections such as *Streptococcus pneumonia, Streptococcus agalactiae*, *Staphylococcus aureus*, *Bordetella parapertussis*, *Bordetella pertussis*, *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* [2].

P. aeruginosa is a Gram-negative pathogen that plays a major contributor in causing hospital-acquired infections [3]. Due to its presence and isolation from secretions of patients with bronchitis, asthma and recurrent infections, this bacterium has been identified as a pathogen for respiratory tract infections [4]. One of the most important characteristics of this bacterium resistance to antibiotics and capacity to endure in harsh environmental conditions [5]. Despite the development and discovery of numerous antibiotics, these bacteria are hard to treat, cause health problems, and a high percentage of fatalities because of their widespread resistance to antibiotics [6]. One of the main obstacles its capacity to resist antibiotics, which results from the presence of antibiotic resistance genes [7]. It works by regulating resistance mechanisms, which enable it to harm body tissues and alter its target to eliminate the effects of antibiotics, which is one of the main therapeutic challenges [8].

One of the most widely used treatments for bacterial respiratory tract infections is antibiotics, because antibiotics have multiple mechanisms against bacterial pathogens [1]. Beta-lactam antibiotics, which are frequently used to treat infections brought on by microbes all over the world, are among the most significant of these treatments [9]. Its mode of action involves using penicillin-binding proteins (PBPs) to stop the bacterial cell wall from forming, these include four main types penicillins, cephalosporins, carbapenems and monobactams, which differ in their effectiveness and uses [10]. A beta-lactam antibiotic called Piperacillin is used to treat severe *P. aeruginosa* infections and inflammations, in addition to Tazobactam, which is an inhibitory beta-lactam antibiotic [11]. Also many infections brought on by Gram-negative bacteria are treated with Avibactam, a broad-spectrum, inhibitory beta-lactam antibiotic [12].

Another class of antibiotics that works well against Gram-negative bacteria such as *P. aeruginosa* are cephalosporins, it has been discovered that the third and fourth generation Cephalosporins, Ceftazidime and Cefepime are broad-spectrum antibiotics against bacteria, it has also been discovered that contemporary cephalosporins like Ceftolozane are effective against bacteria [13].

Additionally, it was discovered that among the first-line therapies for infections brought on by the *P. aeruginosa* bacteria are carbapenem antibiotics, because of their high effectiveness against the bacteria and wide range of activity [14]. It targets the cell wall by preventing the transport of peptides that are vital to the bacterial cell wall, It is a bactericidal antimicrobial and is used for Gram-negative bacteria such as *P. aeruginosa* [15]. Among the most widely used carbapenems are Imipenem and Meropenem [16]. Aminoglycosides are effective antibiotics in treating respiratory infections like pneumonia brought on by the *P. aeruginosa* [17]. Like the antibiotics Gentamicin and Amikacin, which prevent the synthesis of proteins, they also interfere with translation by attaching to the bacterial 30S ribosomal subunit, thus having a bactericidal effect [18]. Ciprofloxacin and levofloxacin are among the class of antibiotics known as fluoroquinolones, which have a bactericidal effect, by inhibiting DNA gyrase enzyme, they stop DNA replication in Gram-negative bacteria [19]. Colistin a narrow-spectrum antibiotic that works well against Gram-negative bacteria, is one of the useful medications used to treat *P. aeruginosa* infections [20], it is an polymyxin antibiotic that works by inhibiting the cell membrane and endotoxins of bacteria [21].

Because *P. aeruginosa* a pathogenic agent of respiratory tract infections and exhibits distinct patterns of interaction with antibiotics, this study sought to isolate and diagnose *P. aeruginosa* from patients with respiratory tract infections and examine the bacterium's reaction to antibiotics. Periodic examination and ongoing monitoring of the patterns of response to antibiotics of this bacterium are crucial for effectively managing treatment for the infection because the nature of the effect of antibiotics varies from time to time.

Work Methods

A. Ethical approval

In order to protect patient's physical and psychology well-being general ethics were adhered to when interacting with them and when collecting samples in this study, which is consistent with instructions of the ethics committees at Al-Furat Al-Awsat Technical University.

B. Sample Collection

Samples were taken from patients who visited Al-Diwaniyah Teaching Hospital in Al-Diwaniyah Governorate with respiratory infections from 1/3/2025 to 1/7/2025. According to the specialist physician's diagnosis 110 sputum samples were collected, patients were instructed to cough deeply to collect sputum sample by using sterile, sealed plastic containers and the samples were brought to the laboratory, to perform laboratory culture.

C. Sample Culture

Sputum samples were cultivated on solid culture media, specifically blood agar and MacConkey agar, prepare in accordance with the manufacturer's guidelines (Himedia/India). Using sterile cotton swabs, the samples were striated into plates for culture. The plates were then incubated for a full day 24 hour at 37°C [22].

D. Isolation and Morphological Diagnosis

Colony size and shape on culture media, hemolytic activity on blood agar, and lactose fermentation on MacConkey agar were used to identify bacterial isolates. purified bacterial colonies were subsequently smeared onto sterile glass slides for microscopic analysis to determine the morphology of bacterial cells and colonies as well as their response to Gram stain, [22]. Catalase, oxidase, and urea tests were among biochemical tests used to identify isolates. In addition to the IMVIC test suite, which comprised the indole, methyl reed, Voges-Proskaur, and Simmons' citrate growth test (identifying a shift in the medium's color from green to blue) [23]. The VITEK test, which is a crucial, precise, and confirmatory test, was then used to confirm diagnosis of *P. aeruginosa* bacterial isolates, the test was conducted in accordance with the guidelines on the prepared kit.

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E. Sensitivity Test of Antibiotics

The antibiotic tablets and dose in microgram used in the study are listed in table (1). The test was conducted by filling tubes with a bacterial suspension prepar of a saline solution containing purified bacterial colonies until the turbidity of the solution in the tubes matched the standard turbidity constant solution (McFarland standard). The culture plates containing Muller-Hilton medium were then inoculated with bacterial suspension using sterile cotton swabs. The antibiotic tablets were then applied to the plates using sterile forceps, and plates were incubated for 24 hours at 37°C. After that, circumference of the antibiotic-resistant and inhibited regions was measured and compared to standard values using [24].

Table (1) Antibiotic tablets made by Himedia

Antibiotic Name	Symbol	Dose
Piperacillin	PRL	100
Avibactam	AVI	20
Tazobactam	TZ	10
Cefepime	FEP	10
Ceftazidime	CAZ	30
Ceftolozane	CTZ	30
Imipenem	IPM	10
Meropenem	MEM	10
Amikacin	AK	30
Gentamicin	CN	10
Ciprofloxacin	CIP	10
Levofloxacin	LEV	5
Colistin	CT	10

F. Analysis of Statistical

SPSS version 32 was used for the statistical analysis, and the chi-square test was used to analyze the findings. Because it is statistically significant, a p-value of less than 0.05 was employed [25].

Results

A. Isolation and Diagnosis

110 sputum samples from patients suffering from respiratory tract infections were gathered for this study investigation. According to table (2), which displays the results of bacterial culture on culture media, 78 samples (70.9%) showed a positive result for growth, while 32 samples (29.1%) showed a negative result and did not show bacterial growth on culture media.

Table (2) Distribution of samples based on bacterial growth on culture media

Culture results	Number	Percentage
Cases of positive culture	78	70.9%
Cases of culture negative	32	29.1%
Total	110	100%
X2	38.47	
P value	<0.0001	

The morphological traits of bacterial isolates, such as color and shape of the colonies on culture media, were used to identify the bacterial isolates that were causing respiratory tract infections in patients. Since the goal was to isolate *P. aeruginosa* the focus was on this bacterium. They were

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discovered to be hemolytic colonies on blood agar medium, encircled by a transparent halo and non-lactose fermenting colonies on MacConkey agar medium, appearing as small, pale-colored colonies. Under a microscope examination, bacterial cells shape revealed that they were rod-shaped and Gram-negative cells. As for the biochemical tests, it was discovered that adding drops of hydrogen peroxide to the colonies produced air bubbles, which indicated a positive result for the catalase test. Additionally, oxidase test yielded a positive result because the colonies turned purple 10 to 30 seconds after drops of hydrogen peroxide reagent were added. These bacteria showed a negative result for the indole test, as no red ring appeared at the top of the medium following the addition of kovacs reagent, similarly methyl red test yielded a negative result, as the medium did not turn color from yellow to red after adding the methyl red reagent. The results demonstrated that this bacterium is agile for growth testing on simmons citrate agar, because medium's color changed from green to blue, indicating the use of citrate as a source of carbon. The diagnostic results also demonstrated that *P. aeruginosa* were negative for the urease test after being cultivated on urea agar, because medium did not change color from yellow to pink, which is a result of urease production [22; 23]. As shown in Table (3), which shows the results of the tests for this bacterium. Additionally, since the VITEK device is one of the tests that provides accurate results, the bacterial isolates were diagnosed using it to confirm diagnosis. The findings demonstrated that 99% of the isolates were related to *P. aeruginosa* bacteria.

Table (3) Biochemical tests used in diagnosis *P. aeruginosa*

Bacteria	Tests	Result
P. aeruginosa	Gram stain	-
	Oxidase	+
	Catalase	+
	Hemolysis	+
	Urease production	-
	Indol	-
	Methyl red	-
	Voges – Proskauer	-
	Simmon's Citrate	+
	H_2S Production	-

- : Negative + : Positive

Table (4) and Figure (1) show the bacterial species isolated from patients with respiratory tract infections that were diagnosed during the isolation and diagnosis of *P. aeruginosa*. It was found that *Klebsiella. pneumoniae* had the highest isolation rate (37.17%), followed by *P. aeruginosa* (33.33%), *Staphylococcus aureus (*19.23%), and *Moraxella spp.* (6.41%). *Streptococcus agalactiae* had lowest rate at 3.84%. At a significance level of P<0.0001, statistical analysis showed highly significant differences between the isolated bacterial species.

Table (4) Bacterial species isolated from respiratory tract infections

Bacterial Species	Number	Percent%
Klebsiella pneumonia	29	37.17
Pseudomonas aeruginosa	26	33.33
Staphylococcus aureus	15	19.23
Moraxella spp.	5	6.41
Streptococcus agalactiae	3	3.84
Total	78	100
X^2	44.8	
P value	<0.0001(HS)	

HS: Highly significant difference at P<0.05

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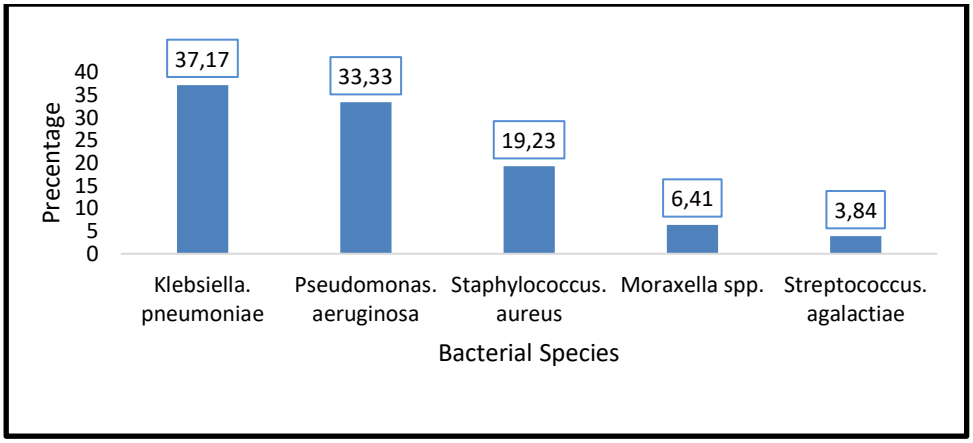

Figure (1) shows the bacterial species isolated from respiratory tract infections

B. Antibiotic susceptibility patterns in *P. aeruginosa*

The Kirby-Bauer disc diffusion method was used to test susceptibility of 26 *P. aeruginosa* isolates against 13 different antibiotics on Mueller-Hinton agar. table (5) and figure (2) show the different degrees of antibiotic susceptibility and resistance exhibited by *P. aeruginosa* isolates. The results demonstrated that isolates of *P. aeruginosa* had a high level of beta-lactam antibiotic resistance. They were found to have a resistance rate of 65.38% for both Avibactam and Tazobactam and 61.53% for the antibiotic Piperacillin. While the isolates sensitivity rates to the Piperacillin and Avibactam and Tazobactam antibiotics were determined to be 38.46% and 34.61%, respectively. The findings also revealed a high rate of resistance to cephalosporin antibiotics, with Ceftazidime having highest resistance rate at 76.92%, Cefepime at 73.07% and Ceftolozone at 69.23%, while the sensitivity rate was low, it was 23.07% for Ceftazidime, 26.92% and 30.76% for Cefepime and Ceftolozone. Additionally, these results showed that *P. aeruginosa* isolates exhibited resistance to Carbapenems, specifically Imipenem and Meropenem, at rates of 53.84% and 57.69% respectively, while their sensitivity to these antibiotics was 46.15% and 42.3% respectively.

Regarding the aminoglycoside antibiotics, specifically Amikacin and Gentamicin, the results indicated that *P. aeruginosa* isolates had the highest sensitivity rate to Amikacin, with a sensitivity rate of 61.53% and a resistance rate reached 38.46%, and the isolates exhibited a high level of Gentamicin resistance (61.53%). As for fluoroquinolone antibiotics, the results revealed that *P. aeruginosa* isolates had a highest resistance rate of 76.92% to Levofloxacin and a sensitivity rate of 23.07% to this antibiotic, whereas isolates had a moderate resistance to Ciprofloxacin, with the isolates and sensitivity being equal at 50%. Additionally, *P. aeruginosa* isolates demonstrated a resistance rate of 46.15% and a sensitivity of 53.84% to colistin, which is polymyxin antibiotic.

Table (5) Antibiotic Susceptibility Patterns of *P. aeruginosa* Isolates

Name of Antibiotics	**Patterns of *P. aeruginosa* to Antibiotics**	
	Resistance (%)	**Sensitive (%)**
Piperacillin	16(61.53)	10(38.46)
Avibactam	17(65.38)	9(34.61)
Tazobactam	17(65.38)	9(34.61)
Cefepime	19(73.07)	7(26.92)
Ceftazidime	20(76.92)	6(23.07)
Ceftolozone	18(69.23)	8(30.76)
Imipenem	14(53.84)	12(46.15)
Meropenem	15(57.69)	11(42.3)
Amikacin	10(38.46)	16(61.53)
Gentamicin	16(61.53)	10(38.46)
Ciprofloxacin	13(50)	13(50)
Levofloxacin	20(76.92)	6(23.07)
Colistin	12 (46.15)	14(53.84)

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X^2	18.29
P value	0.107(NS)

NS: No significant difference at P<0.05

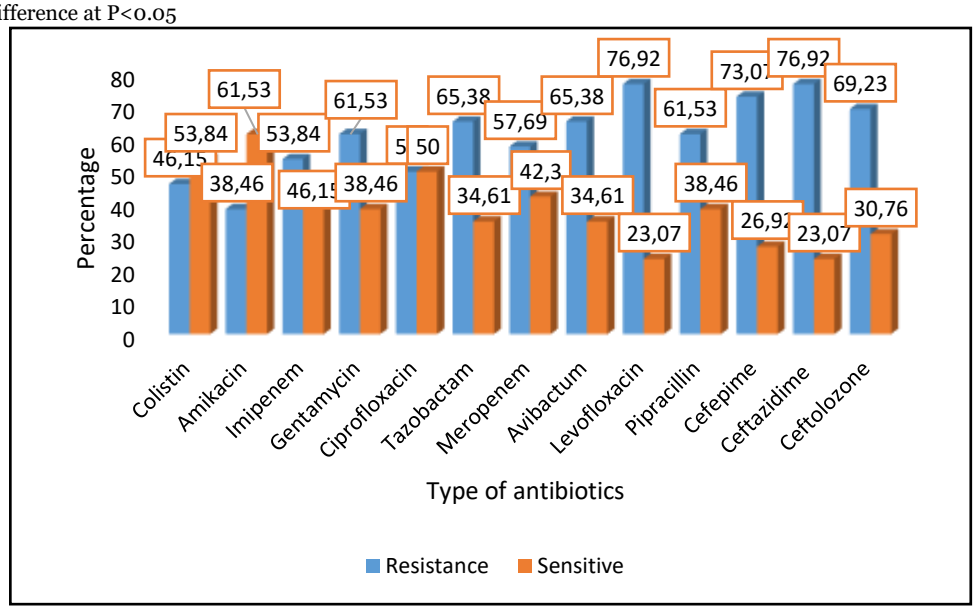

Figure (2): Resistance and sensitivity patterns rates to antibiotics in *P. aeruginosa*

Discussion

Since respiratory tract infections are frequently linked to bacterial pathogens, particularly *P. aeruginosa*, which is known for its multidrug resistance and is therefore challenging to treat, this study aim to isolate and diagnose *P. aeruginosa* from patients with respiratory tract infections [26]. It was discovered that *P. aeruginosa* causes serious infections in patients and antibiotic resistance, which may have life-threatening effects for patients and public health [27]. According to the study's findings, 78 sputum samples (70.9%) had positive results, while32 samples (29.1%) had negative growth on culture media. Additionally, the results indicated that 33.33% of sputum samples contained *P. aeruginosa*. These findings were consistent with those of [28], who discovered that this bacterium was isolated from respiratory tract infections at a rate of 26%, while our findings were higher than those of [29], who discovered that *P. aeruginosa* was responsible for 10% of respiratory tract infections. This variation in isolation rates could be caused by variations in number of pathological samples being examined, as well as variations in time and location, sample collection techniques and laboratory bacterial culture conditions, all of which could have an impact on the growth and appearance of bacteria.

Patterns of antibiotic susceptibility in isolates of *P. aeruginosa*

The study's findings showed that different *P. aeruginosa* isolates had varying levels of antibiotic efficacy, this variation in antibiotic resistance may be attributed to the misuse of antibiotics by individuals and the prescription of multiple antibiotics, which leads to the pathogen becoming resistant to a variety of antibiotics. Specifically, *P. aeruginosa* is a bacterial pathogen that exhibits self-resistance in addition to developing numerous antibiotic resistance mechanisms, which results in the persistence of infection and treatment failure [30]. The findings demonstrated that isolates of these bacteria had a high rate of resistance to beta-lactam antibiotics, including Tazobactam, Avibactam and Piperacillin. These findings are in line with those of [31], who discovered that these bacteria had a high rate of beta-lactam antibiotic resistance. Additionally, it was discovered *P. aeruginosa* exhibited high resistance to modern-generation cephalosporins, Ceftazidime had the highest resistance rate of 76.92% when compared to other antibiotics in the study, followed by Cefepime at 73.07% and Ceftolozone at 69.23%. Because *P. aeruginosa* is the causative agent of infections isolated from respiratory tract infections, thus, these antibiotics are not thought to be among the first treatment options. Genetic mutations that result in elevated MexAB-OprM outflow pump gene expression could be the cause of this high resistance to Ceftazidime [32]. Our findings were different from those of [33] study, which discovered that these bacteria had extremely low resistance rates of 22% and 13% for Cefepime and Ceftazidime, respectively. This difference, which appears in comparison with the results in other studies, is due to difference in the nature of isolates in terms of place and time. It may be the result of *P. aeruginosa* isolates from sputum samples being exposed to beta-lactam antibiotics frequently, especially cephalosporins and thus leading to the emergence of multi-resistance.

Additionally, it was discovered *P. aeruginosa* isolates had a high level of resistance to carbapenem antibiotics, with Imipenem at 53.84% and Meropenem at 57.69%. *P. aeruginosa* resistant to carbapenem antibiotics was found to be one of the pathogens linked to infections related to healthcare [34]. These findings contrasted with those of [35], who discovered that 76% of isolates were sensitive to the antibiotic Imipenem and 24% of isolates were resistant to it. The findings of our investigation also differed from those of [36], who reported that 91.55% of *P. aeruginosa* respiratory infections were sensitive to the antibiotic Meropenem. This variation in sensitivity rates and existence of resistant isolates could be caused by *P. aeruginosa* resistance to these antibiotics due to the production of metallo-β-lactamases (MBL), which reduces the effectiveness of these medications, as well as the efflux pump expressed as a result of decreased membrane permeability [37].

According to the study findings, *P. aeruginosa* isolates had a resistance rate of 38.46% to the antibiotic Amikacin and a high resistance rate of 61.53% to the Gentamicin, It indicates that the aminoglycoside antibiotic Amikacin is more effective than Gentamycin against isolates of *P.*

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aeruginosa. This is consistent with the findings of [38], who discovered that Amikacin exhibits activity and an efficient effect against Gram-negative bacteria, such as *P. aeruginosa*. This could be because the Amikacin structure contains an aminohydroxybutyryl group, which prevents the antibody from being enzymatically modified at various locations without affecting its binding to the A site in rRNA [39].

Additionally, results demonstrated that the *P. aeruginosa* isolates showed very high resistance to the antibiotic Levofloxacin, reaching 76.92%, while the sensitive isolates reached 23.07%. In contrast, the rates of resistance and sensitivity to the antibiotic Ciprofloxacin were equal reaching 50%, from this we find that the antibiotic Ciprofloxacin may be more effective than Levofloxacin in treating isolates of *P. aeruginosa*. Mutations in the *gyrA* gene, which codes for DNA gyrase's a subunit, could be the cause of resistance's emergence [40]. Furthermore, the widespread use of fluoroquinolones causes isolates to become resistant. This is consistent with the findings of [41], who discovered that the overuse of these antibiotics in hospitals causes resistant *P. aeruginosa* isolates to emerge. The findings of our study were different from those of [42], who discovered that the sensitivity rate to Levofloxacin was 70% and resistance rate was 30%. Because 65% of the isolates were sensitive to ciprofloxacin, our findings were comparable to theirs. Additionally, it was discovered that *P. aeruginosa* isolates had a low resistance rate and a high sensitivity rate to Colistin. This is in line with findings of [43] and [44], who discovered that Colistin is a successful treatment for these bacteria, which are known for their multidrug resistance. Nevertheless, some of *P. aeruginosa* isolates demonstrated resistance to this antibiotic, according to the study's findings. This may be because enzymatic modification of lipids alters the outer membrane's permeability and negative charge, which lowers the anticolistin's affinity for the membrane and causes resistance to develop. Additionally, genetic mutations or the presence of antibody-resistant genes could be the cause of isolates' resistance to this antibiotic [45].

Conclusions

The study's findings lead us to conclusion *P. aeruginosa* is a common pathogen that causes bacterial respiratory tract infections. The study's findings also demonstrated *P. aeruginosa* isolates from patients with respiratory infections in the Al-Diwaniyah Governorate showed high resistance to a variety of antibiotics. It was discovered that it had a high rate of resistance to beta-lactam antibiotics, particularly modern-generation cephalosporins, with Ceftazidime showing the highest rate of resistance and Levofloxacin showing the same high rate of resistance and Cefepime thus, these antibiotics are not thought of as initial treatments for *P. aeruginosa* infections. While both Amikacin and Colisin were found to be effective against these bacteria and their use is recommended, as *P. aeruginosa* isolates showed high sensitivity to them, it is preferable to administer them in combination with other less effective antibiotics as a therapy to reduce the emergence of resistance.

Recommendations

Because *P. aeruginosa* isolates have developed resistance to most antibiotics, this study recommends using antibiotics sparingly and appropriately to prevent emergence and spread of resistance in *P. aeruginosa* strains. In order to help control infections caused by *P. aeruginosa* and develop new treatment strategies, study highlights the significance of hospitals routinely updating their antibiotic protocols for treating respiratory tract infections, identifying effective antibiotics and those facing resistance. Additionally, we recommend research to understand and investigating the molecular and genetic underpinnings of antibiotic resistance in *P. aeruginosa* that causes respiratory tract infections.

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Authors' Contributions

The researcher designed the study, wrote the research, collected data, analyzed the results, and then reviewed the research and gave approval for publication.

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Conflict of interests

There is no conflict of interest

References

1. A. Eltayeb and S. Leaver, "Bacterial Infections of the Respiratory Tract and Their Pharmacological Treatment," Anaesthesia & Intensive Care Medicine, vol. 25, no. 9, pp. 623-629, 2024. https://doi.org/10.1016/j.mpaic.2024.06.006
2. Y. Long, Y. Zheng, C. Li, Z. Guo, P. Li, F. Zhang, W. Liu, and Y. Wang, "Respiratory Pathogenic Microbial Infections: A Narrative Review," International Journal of Medical Sciences, vol. 21, no. 5, pp. 826-836, 2024.
3. D. Reynolds and M. Kollef, "The Epidemiology and Pathogenesis and Treatment of Pseudomonas Aeruginosa Infections: An Update," Drugs, vol. 81, pp. 2117-2131, 2021. https://doi.org/10.1007/s40265-021-01635-6
4. M. G. Clemente, D. D. L. Rosa, L. Maiz, R. Giron, M. Blanco, C. Olveira, R. Canton, and M. A. M. Garcia, "Impact of Pseudomonas Aeruginosa Infection on Patients with Chronic Inflammatory Airway Diseases," Journal of Clinical Medicine, vol. 9, no. 3800, pp. 1-32, 2020.
5. P. Laborda, F. Sanz-Garcia, S. Hernando-Amado, and J. L. Martinez, "Pseudomonas Aeruginosa: An Antibiotic Resilient Pathogen with Environmental Origin," Current Opinion in Microbiology, vol. 64, pp. 125-132, 2021. https://doi.org/10.1016/j.mib.2021.09.010
6. H. W. Boucher, G. H. Talbot, J. S. Bradley, et al., "Bad Bugs, No Drugs: No ESKAPE! An Update from the Infectious Diseases Society of America," Clinical Infectious Diseases, vol. 48, no. 1, pp. 1-12, 2009. https://doi.org/10.1086/595011
7. P. Pachori, R. Gothalwal, and P. Gandhi, "Emergence of Antibiotic Resistance Pseudomonas Aeruginosa in Intensive Care Unit; A Critical Review," Genes & Diseases, vol. 6, pp. 109-119, 2019. https://doi.org/10.1016/j.gendis.2019.04.001
8. N. Sathe, P. Beech, L. Croft, C. Suphioglu, A. Kapat, and E. Athan, "Pseudomonas Aeruginosa: Infections and Novel Approaches to Treatment - Knowing the Enemy - The Threat of Pseudomonas Aeruginosa and Exploring Novel Approaches to Treatment," 2023.
9. A. K. Chandel, L. V. Rao, M. L. Narasu, and O. V. Singh, "The Realm of Penicillin G Acylase in Beta-Lactam Antibiotics," Enzyme and Microbial Technology, vol. 42, pp. 199-207, 2008.

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10. L. M. Lima, B. N. M. D. Silva, G. Barbosa, and E. J. Barreiro, "Beta-Lactam Antibiotics: An Overview from a Medicinal Chemistry Perspective," European Journal of Medicinal Chemistry, vol. 208, pp. 112829, 2020. https://doi.org/10.1016/j.ejmech.2020.112829
11. G. M. Pacifici, "Clinical Pharmacology of Piperacillin/Tazobactam," Journal of Clinical and Biomedical Investigation, vol. 3, no. 1, pp. 11-19, 2023. https://doi.org/10.5216/jcbi234024
12. M. Sargianou, P. Stathopoulos, C. Vrysis, I. D. Tzvetanova, and M. E. Falagas, "New Beta-Lactam/Beta-Lactamase Inhibitor Combination Antibiotics," Pathogens, vol. 14, no. 4, pp. 307, 2025. https://doi.org/10.3390/pathogens14040307
13. A. Rusu and L. Ioana-Andreea, "The New Fifth-Generation Cephalosporins - A Balance Between Safety and Efficacy," Romanian Journal of Pharmaceutical Practice, vol. 13, no. 3, pp. 121-126, 2020. https://doi.org/10.37897/RJPhP.2020.3.2
14. T. Takahashi, T. Tada, S. Shrestha, T. Hishinuma, J. B. Sherchan, M. Tohya, et al., "Molecular Characterisation of Carbapenem-Resistant Pseudomonas Aeruginosa Clinical Isolates in Nepal," Journal of Global Antimicrobial Resistance, vol. 26, pp. 279-284, 2021. https://doi.org/10.1016/j.jgar.2021.07.003
15. V. Trebosc, B. Schellhorn, J. Schill, V. Lucchini, J. Buhler, et al., "In Vitro Activity of Rifabutin Against 293 Contemporary Carbapenem-Resistant Acinetobacter Baumannii Clinical Isolates and Characterization of Rifabutin Mode of Action and Resistance Mechanisms," Journal of Antimicrobial Chemotherapy, vol. 75, pp. 3552-3562, 2020.
16. M. A. Lamb, S. Wiedbrauk, E. Kathryn, and R. S. C. Fairfull-Smith, "Approaches to Enhance the Antimicrobial Activity of Carbapenems Within Bacterial Biofilms," Pharmaceuticals, vol. 1, no. 4, pp. 622-644, 2024. https://doi.org/10.1039/d4pm00141a
17. A. W. Serio, T. Keepers, L. Andrews, K. M. Krause, and K. Bush, "Aminoglycoside Revival: Review of a Historically Important Class of Antimicrobials Undergoing Rejuvenation," EcoSal Plus, vol. 8, no. 1, 2018. https://doi.org/10.1128/ecosalplus.esp-0002-2018
18. K. M. Krause, A. W. Serio, T. R. Kane, and L. E. Connolly, "Aminoglycosides: An Overview," Cold Spring Harbor Perspectives in Medicine, vol. 6, no. 6, pp. 027029, 2016. https://doi.org/10.1101/cshperspect.a027029
19. F. Vuotto, J. P. Bru, E. Canoui, M. Caseris, M. C. C. Chopin, R. Cohen, S. Diamantis, A. Dinh, P. Fillatre, R. Gauzit, Y. Gillet, A. P. Jonville-Bera, M. Lafaurie, P. Lesprit, M. Lorrot, J. Lourte, L. Maulin, D. Poitrenaud, A. Pariente, J. Raymond, and T. E. Bonnet, "The Latest Updates on the Proper Use of Fluoroquinolones - Actualisation 2025 Update by the SPILF and the GPIP," Infectious Diseases, vol. 55, no. 4, pp. 105062, 2025. https://doi.org/10.1016/j.idnow.2025.105062
20. M. Talaat, S. Tolba, E. Abdou, M. Sarhan, M. Gomaa, and Y. J. F. Hutin, "Over-Prescription and Overuse of Antimicrobials in the Eastern Mediterranean Region: The Urgent Need for Antimicrobial Stewardship Programs with Access, Watch, and Reserve Adoption," Antibiotics, vol. 11, no. 12, pp. 1773, 2022. https://doi.org/10.3390/antibiotics11121773
21. World Health Organization, "The WHO AWaRe (Access, Watch, Reserve) Antibiotic Book," Geneva, Switzerland: World Health Organization, 2022.
22. J. F. MacFaddin, "Biochemical Tests for Identification of Medical Bacteria," 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2000.
23. B. A. Forbes, F. S. Daniel, and S. W. Alice, "Bailey and Scott's Diagnostic Microbiology," 12th ed. USA: Mosby Elsevier Company, 2007.
24. Clinical and Laboratory Standards Institute, "Performance Standards for Antimicrobial Susceptibility Testing," 34th ed., 2024.
25. J. Rahman, "Brief Guidelines for Methods and Statistics in Medical Research," 1st ed. Singapore: Springer Singapore Heidelberg, 2015.
26. H. K. Yum, I. N. Park, B. M. Shin, and S. J. Choi, "Recurrent Pseudomonas Aeruginosa Infection in Chronic Lung Diseases: Relapse or Reinfection?" Tuberculosis and Respiratory Diseases, vol. 77, no. 4, pp. 172-177, 2014. https://doi.org/10.4046/trd.2014.77.4.172
27. N. Maharjan, "Pseudomonas Aeruginosa Isolates Among Clinical Samples Showing Growth in a Tertiary Care Centre: A Descriptive Cross-Sectional Study," Journal of Nepal Medical Association, vol. 60, no. 252, pp. 676-680, 2022.
28. S. Prajapati and S. Mehta, "Antimicrobial Susceptibility of P. Aeruginosa Isolated from Patients with Respiratory Tract Infections," IP International Journal of Medical Microbiology and Tropical Diseases, vol. 5, no. 3, pp. 169-173, 2019.
29. M. S. Kadhim and L. H. H. Alshaibah, "Isolation and Identification of Pseudomonas Aeruginosa from Nosocomial Infections Patients in Al Diwaniyah Province," Al-Qadisiyah Journal of Pure Science, vol. 29, no. 2024, pp. 1-7, 2024.
30. E. Rossi, R. La Rosa, J. A. Bartell, R. L. Marvig, J. A. J. Haagensen, L. M. Sommer, S. Molin, and H. K. Johansen, "Pseudomonas Aeruginosa Adaptation and Evolution in Patients with Cystic Fibrosis," Nature Reviews Microbiology, vol. 19, pp. 331-342, 2021.
31. M. F. Al-Saffar and E. M. Jarallah, "Isolation and Characterization of Pseudomonas Aeruginosa from Babylon Province," Biochemistry and Cell Archives, vol. 19, no. 1, pp. 203-209, 2019. www.connectjournals.com/bca
32. K. A. Glen and I. L. Lamont, "Beta-Lactam Resistance in Pseudomonas Aeruginosa: Current Status, Future Prospects," Pathogens, vol. 10, no. 12, pp. 1638, 2021. https://doi.org/10.3390/pathogens10121638
33. C. Liao, N. Lee, H. Tang, S. Lee, C. Lin, P. Lu, J. Wu, W. Ko, W. S. Lee, and P. Hsueh, "Antimicrobial Activities of Ceftazidime-Avibactam, Ceftolozane-Tazobactam, and Other Agents Against Escherichia Coli, Klebsiella Pneumoniae, and Pseudomonas Aeruginosa Isolated from Intensive Care Units in Taiwan: Results from the Surveillance of Multicenter Antimicrobial Resistance in Taiwan in 2017," Infection and Drug Resistance, vol. 12, pp. 1-8, 2019. https://doi.org/10.2147/IDR.S193638
34. M. Al-Orphaly, H. A. Hadi, F. K. Eltayeb, et al., "Epidemiology of Multidrug-Resistant Pseudomonas Aeruginosa in the Middle East and North Africa Region," mSphere, vol. 6, no. 3, pp. e00202-21, 2021. https://doi.org/10.1128/mSphere.00202-21
35. A. Fatima, S. B. Naqvi, S. A. Khaliq, S. Perveen, and S. Jabeen, "Antimicrobial Susceptibility Pattern of Clinical Isolates of Pseudomonas Aeruginosa Isolated from Patients of Lower Respiratory Tract Infections," SpringerPlus, vol. 1, no. 70, pp. 2-4, 2012. http://www.springerplus.com/content/1/1/70
36. A. Samad, T. Ahmed, A. Rahim, A. Khalil, and I. Ali, "Antimicrobial Susceptibility Patterns of Clinical Isolates of Pseudomonas Aeruginosa Isolated from Patients of Respiratory Tract Infections in a Tertiary Care Hospital, Peshawar," Pakistan Journal of Medical Sciences, vol. 33, no. 3, pp. 670-674, 2017. https://doi.org/10.12669/pjms.333.12416
37. P. D. Lister, D. J. Wolter, and N. D. Hanson, "Antibacterial-Resistant Pseudomonas Aeruginosa: Clinical Impact and Complex Regulation of Chromosomally Encoded Resistance Mechanisms," Clinical Microbiology Reviews, vol. 22, no. 4, pp. 582-610, 2009. https://doi.org/10.1128/CMR.00040-09
38. A. Endo, A. Nemoto, K. Hanawa, Y. Maebayashi, Y. Hasebe, M. Kobayashi, A. Naito, Y. Kobayashi, S. Yamamoto, and K. Isobe, "Relationship Between Amikacin Blood Concentration and Ototoxicity in Low Birth Weight Infants," Journal of Infection and Chemotherapy, vol. 25, no. 1, pp. 17-21, 2019.
39. L. P. Kotra, J. Haddad, and S. Mobashery, "Aminoglycosides: Perspectives on Mechanisms of Action and Resistance and Strategies to Counter Resistance," Antimicrobial Agents and Chemotherapy, vol. 44, no. 12, pp. 3249-3256, 2000. https://doi.org/10.1128/aac.44.12.3249-3256.2000
40. R. M. Farahi, A. A. Ali, and S. Gharavi, "Characterization of gyrA and parC Mutations in Ciprofloxacin-Resistant Pseudomonas Aeruginosa Isolates from Tehran Hospitals in Iran," Iranian Journal of Microbiology, vol. 10, no. 4, pp. 242, 2018. PMID: 30483376 PMCID:

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41. R. Salma, F. Dabboussi, I. Kassaa, R. Khudary, and M. Hamze, "gyrA and parC Mutations in Quinolone-Resistant Clinical Isolates of Pseudomonas Aeruginosa from Nini Hospital in North Lebanon," Journal of Infection and Chemotherapy, vol. 19, no. 1, pp. 77-81, 2013. https://doi.org/10.1007/s10156-012-0455-y
42. M. A. Al-Mayali and E. D. Salman, "Bacteriological and Molecular Study of Fluoroquinolones Resistance in Pseudomonas Aeruginosa Isolated from Different Clinical Sources," Iraqi Journal of Science, vol. 61, no. 9, pp. 2204-2214, 2020. https://doi.org/10.24996/ijs.2020.61.9.7
43. Z. Pang, R. Raudonis, B. R. Glick, T. J. Lin, and Z. Cheng, "Antibiotic Resistance in Pseudomonas Aeruginosa: Mechanisms and Alternative Therapeutic Strategies," Biotechnology Advances, vol. 37, no. 1, pp. 177-192, 2019. https://doi.org/10.1016/j.biotechadv.2018.11.013
44. S. Ibrahim, N. Al-Saryi, I. Al-Kadmy, and S. N. Aziz, "Multidrug-Resistant Acinetobacter Baumannii as an Emerging Concern in Hospitals," Molecular Biology Reports, vol. 48, pp. 6987-6998, 2021. https://doi.org/10.1007/s11033-021-06690-6
45. M. S. Arcilla, J. M. V. Hattem, S. Matamoros, D. C. Melles, J. Penders, M. D. de Jong, et al., "Dissemination of the mcr-1 Colistin Resistance Gene," The Lancet Infectious Diseases, vol. 16, no. 2, pp. 147-149, 2016. https://doi.org/10.1016/S1473-3099(15)00541-1

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