Study of the prevalence of antibiotic resistance in *Pseudomonas aeruginosa* in different regions in Diyala Governorate

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Summary

Doctors and health workers in Diyala Governorate hospitals face a great challenge due to the difficulty of choosing the appropriate treatment for various infectious diseases that affect children and adults, and this challenge becomes more difficult year after year. Especially among fast-spreading bacteria, opportunistic pathogens and nosocomial infections such as Pseudomonas aeruginosa, with their great potential to develop resistance to diverse classes of antibiotics. In fact, one of the most common problems facing health workers is the ability of bacteria to quickly develop resistance during the treatment of infections. This study focused on the prevalence of antibiotic resistance in Pseudomonas aeruginosa in Diyala Governorate. The results of this study showed that amoxicillin and amoxicillin/clavulanic acid recorded (58.4%) each within the penicillin group. While cefuroxime and cefotaxime recorded a percentage of (52.3% and 50.7%),

respectively, based on the cephalosporin class. The results showed that the least resistance was recorded in colistin (9.2%) among the Polycationic piptide class.

List of Contents

List of Tables

List of Figures

1- Introduction

Pseudomonas aeruginosa (*P.aeruginosa*) is a gram-negative, motile, aerobic bacillus, some of which produce water-soluble pigments (Salman. *et al.*, 2017). It is considered an opportunistic bacterium associated with nosocomial infections, such as ventilator-associated pneumonia (VAP), infections in intensive care units, patients with central lines, and infections at surgical sites. (Yousef. *et al.*, 2023). *P. aeruginosa*, a highly pathogenic bacterium, has cell surface structures, secreted compounds, and biofilm formation that contribute to its pathogenicity (Abd-Al-Absawe and Tuwaij , 2022).

P. aeruginosa has a distinctive characteristic that has become apparent nowadays, which is resistance to antibiotics (Salman. *et al*; 2017). Its resistance is due to a group of factors, including low permeability of its cell wall, the bacteria possessing the genetic ability to express a wide repertoire of resistance mechanisms, and the occurrence of mutation also contributes to the development of resistance as well as the acquisition of additional resistance genes from other organisms through plasmids, transposons, and bacterial phages (Lambert, 2002; Verdial, 2023). In some previous research, the results showed a high prevalence of resistance 79% to 100%, which is higher than those reported by other studies in Iran 16.5 - 41%, Iraq 12.4%, Brazil 71.4%, and Egypt 70% (Alkhulaifa and Mohammed, 2023). According to that mentioned above, this study was aimed to investigate the phenotypic pattern of antibiotic resistance among *Pseudomonas aeruginosa* in in different regions in Diyala Governorate.

This was achieved according to the following steps:

1) Collection of clinical Specimens for isolation of *Pseudomonas aeruginosa* from three districts (Baqubah, Al-Khalis, and Muqdadiyah).

2) Identification of Pseudomonas aeruginosa according to their morphological characteristic, biochemical tests, identification by using Vitek-2 system.

3) Investigating the antibiotic susceptibility pattern of bacterial isolates against different antibiotics belongs to different groups.

2. Literature Review

2-1 *Pseudomonas aeruginosa*

P. aeruginosa is a rod-shaped, aerobic, gram-negative bacterium that belongs to the Pseudomonadaceae family. It is not a fermentable carbohydrate but does create acid from sugars such xylose, glucose, and fruct, but not lactose or sucrose. (Verdial. *et al.*, 2023). If nitrate is present, *p.aeruginosa* can also grow anaerobically (Lodise and Bidell, 2019). Although 37°C is ideal for *p.aeruginosa*, it has also been observed to grow 42°C and 4°C, itis different from other Pseudomonas fluorescens species in that it can grow at 42°C (Sekhi, 2022).

P. aeruginosa is linked to significant morbidity and fatality rates, particularly in people with impaired immune systems (Serretiello. *et al*., 2023). Although the precise route of transmission and site of infection are frequently unknown in majority of patients, it is also regarded as an apportunistic pathogen. (Yousef. *et al*, 2023). In general, transmission happen when patient is contaminated by food or water, by direct contact with contaminated tanks, or by hospital personnel handling another pat ient. In most cases, *p.aeruginosa* typically enter humans through mouth or respiratory system. (Lodise and Bidell, 2019). *P. aeruginosa* secretes a range of virulence factors in order to adapt to unfvoarable host environments, which aid in pathogenesis and effective infection of the host (Qin. et al., 2022).

2-2 **Virulence factors**

One of the main causes of P. aeruginosa's enhanced pathogenicity is thought to be virulence factors , as these factors are catecorized into cell-related factors, including adhesives and lipopolysaccharides (Edward, *et al*., 2023). As well as secreted substances, such as exotoxin A, proteases, exoenzymes, and phospholipases. C, pyocyanin, alginate, and DNase (Strateva and Mitov, 2011). Exotoxin S can cause cell death and exotoxin A is crucial in blocking the creation of proteins (Hauser,

2009 ; Shaver and Hauser, 2004). It's also thought that polysaccharide (alginate) is primary cause of mucus in cystic fibrosis patients (Edward, *et al.*, 2023). The phospholipase C enzyme may also cause surface protein degradation. (López, *et al.*,2010). Pyocyanin can produce reactive oxygen groups which cause tissue damage by triggering apoptosis (Muller, 2002; Edward, *et al*., 2023). The enzyme DNase can also break down DNA , and the enzyme hemolysin aids in breakdown of cells , which make them more harmful (Mulcahy, *et al*., 2010). The patient's tissue proteins are then broken down by the enzyme protease , which suppress the immune system.(Macin, *et al*., 2017). Howover as a result of biofilm formation, resistance rates may rise in patients , particularly in cases of acute and chronic infections This raises the risk of infection and death (Chen and Wen, 2011).

2-3 Acquisition of Antibiotic Resistance

P. aeruginosa developed antibiotic resistance through multiple variety of innate and acquired preocesses, such as the creation the biofilm-mediated resistant and multi-drug-resistant persistent cells (Qin. *et al.*,2022). Intrinsic, acquired, or adaptive resistance mechanisms are existing in *P. aeruginosa* as following:

1. **Intrinsic resistance :** Innate antibiotic resistance defined as the inherent qualities of bacteria that enable them to withstand the effect of antibiotics and other antimicrobial agents (Verdial. *et al*., 2023) . This type originte from genes that encode the fundamental characteristics of cell composition and structures that offer defense against harmful substances and antibiotics (Lodise and Bidell, 2019) . This kind of resistance does not arise from prior exposure of inhibitory substances ; rather, itis depending on architecture of bacterial cells .(Verdial. *et al* ; 2023). P. aeruginosa expresses efflux pump system , produces enzymes that inactivate antibiotics and has intrinsic

resistance to number of antibiotics due to its lower permeability, (Pang . *et al* ; 2019).

2. **Acquired Resistance**: mutation of intrinsic genes or horizontal acquisition from other bacteria via the transfer of plasmids containing genetic materials encoding for antibiotic resistance are the two ways that acquired resistance mechanisms arise . (Lodise and Bidell, 2019). The exposure to antibiotics is one of an external stressor that significant impact on the development of acquired resistance . (Verdial.*et al*.,2023). These mechanisms are durable and can be spreed horizontally such as resistance genes carried by plasmids or vertically such as during bacterial replication (Fernandez- Billon.,2023)

3. **Adaptative Resistance:** Bacteria use mechanism called adaptive resistance to momentarily strengthen their resistance to effect of drugs and other stresses This kind of resistance mostly depends on artificially produced changes in gene expression, which raise the production of proteins or changes in targets of antibiotics (Pang . *et al* ; 2019).

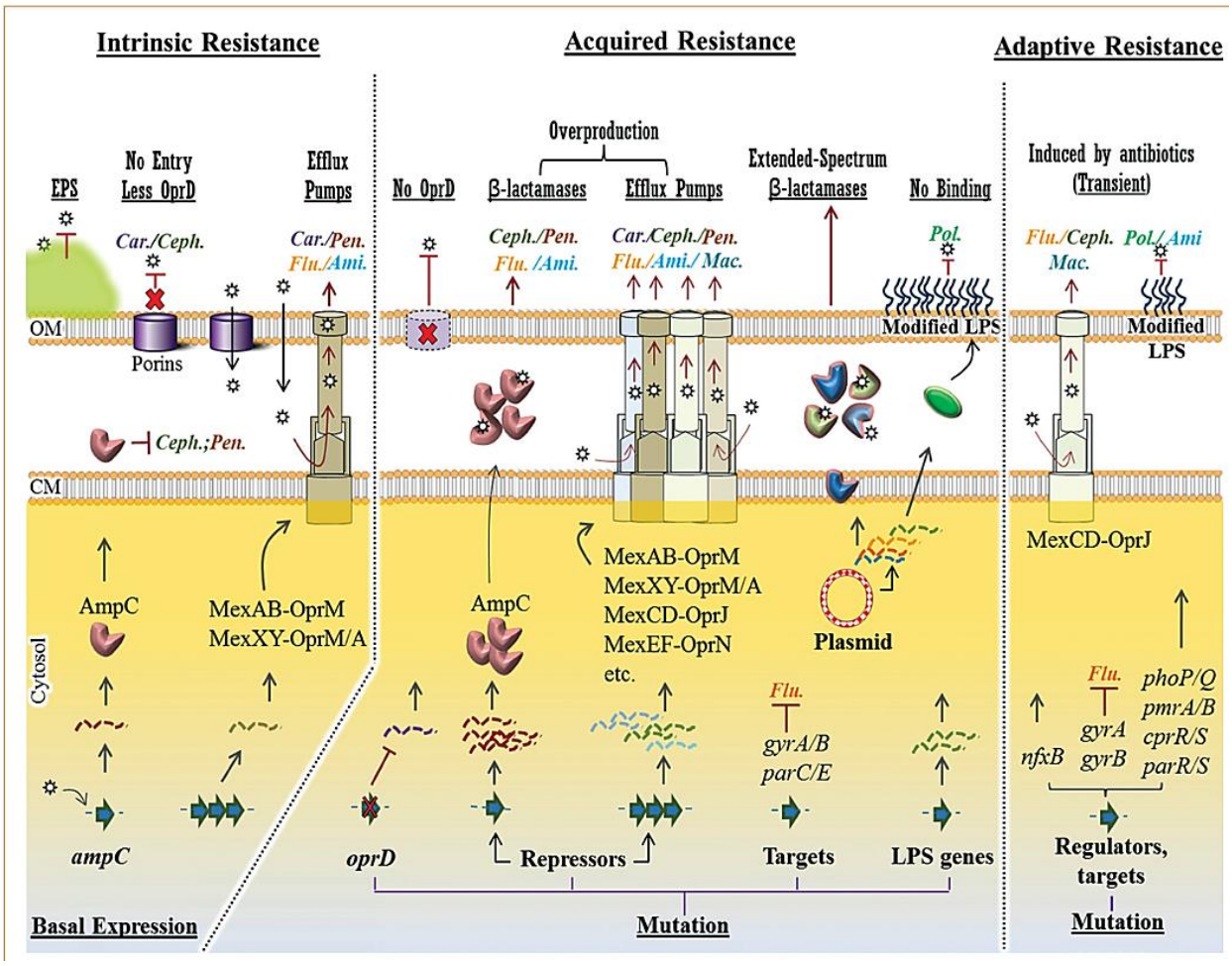

Figure (2-1): Intrinsic, acquired, and adaptive mechanisms confer antibiotic resistance in *P. aeruginosa.* (Lodise and Bidell, 2019).

2-4 Mechanisms of antimicrobial resistance in P. aeruginosa

The P.aeruginosa's antimicrobial resistance mechanisms are best understood in terms of their effects on efflux pumps , taeget binding site mutations , antibiotic inactivating enzymes and outer membrane porin and permeability changes . (Hernando-Amado and Martinez,2023).as following :

2. Literature Review

1. Permeability Alterations and outer membrane porins

Because its outer membrane is entirely in charge of aborping particular antibiotics that rely on different porin activities *P.aeruginosa* outer membrane may act as particular barrier inhibiting the entrance of antibiotics.(Dweh and Rayanoothala.,2023). *P. aeruginosa* innate resistance is because of the relative imperviousness of P.aeruginosa's outer membrane to numerous antibiotics (Sekhi, 2022). *P. aeruginosa* manipulates different purines to promote antibiotic resistance and decrease their permeability , OprF improve *p.aeruginosa* biofilm formation and attachment function formation.(Qin, *et al*., 2022). Aminoglycoside resistance is thought to be influenced by mutations that alter polarity of the outer cell membrane , alter the size of the porin channel , reduce quality of porins or completely eliminate porins. (Lambert,2002).

2. Efflux Pump Systems

One important conserved method for eliminating antibiotics is efflux pump which also has the ability to control virulence (QS) genes to enhance antibiotic resistance and preserve bacterial homeostasis.(Edward.,2023). There are multiple multidrug efflux systems in P.aeruginosa .usually These efflux systems consist of three components: a cytoplasmic membrane pump, a cytoplasmic membrane “exit” porin, and a binding protein (Fernandez-Billon, *et al*.,2023). The efflux pump family has five components : ATP binding cassette (ABC) family, master facilitator family (MFS), multidrug and toxic compound extrusion (MATE), resistance node division (RND), and small multidrug resistance-SMR (Lambert,2002). Due to the strict regulation of genetic changes encoding greater pump expression, p umping regulation may occasionally be linked to increased pump efficiency throug h enhanced affinity for particular antibiotic substrates.(Lodise and Bidell, 2019).

3. Enzyme Mediated

Antibiotics frequently contain chemical bonds (such as amides and esters), and bacteria can produce extended-spectrum beta lactamases, which hydrolyze or modify the antibiotic and cause resistance to penici llin, cephalosporin, and aztreonam, among other antibiotics.(Ghoul.*et al*,2023)

4. Chromosomally Mediated

P. aeruginosa has intrinsic resistance to amino-penicillins and first- and second-generation cephalosporins because to the presence of chromosomally encoded catalytic molecular class C AmpC β-lactamases (Dweh and Rayanoothala.,2023). *P. aeruginosa* ESBL is thought to be the most significant mechanism for controlli ng antibiotics, and it will be a key focus for the creation of more potent antimicrobi al medications. (Qin, et al., 2022). In *P. aeruginosa* it is a class D molecular enzyme, OXA-50 , this is a relatively narrow-spectrum oxacillinase that is inconsistent with nonsensitivity to ampicillin and first and second generation cephalosporins (Lambert,2002).

5. Acquired ß-Lactamases

Penicillinase PSE(pseudomonas-specific enzyme) is the most often acquired beta-lactamase and is a member of the molecular class.The activity of narrow-spectrum β-lactams seems to be impacted by PSE pencillinases, but not that of broad spectrum cephalosporins , menobactams ,or carbapenem (Dweh and Rayanoothala.,2023).

6. Aminoglycoside modification enzymes

Antibiotic resistance can be produced by *P. aeruginosa* by altering the amino and g lycosidic groups of aminoglycoside drugs by three mechanisms :: aminoglycoside phosphotransferase (APH), aminoglycoside acetyltransferase (AAC), and aminoglycoside nucleotide transferase (ANT) (Sekhi, 2022), While plasmids are typically the source of aminoglycoside resistance, other transp osable genetic elements such as integrons and transposons can also impart this resi stance. (Lodise and Bidell, 2019). APH can render streptomycin inactive through transferring a phosphate group to the 3′-hydroxyl group of aminoglycosides (MacKinnon,2011). By adding an acetyl group to the amino group at aminoglycosides' 3′ and 6′ sites, AAC may result in gentamicin resistance. Through the transfer of adenosine groups to amino or hydroxyl groups of these aminoglycosides ANT gives P. aeruginosa resistance to amikacin (Qin, *el at*., 2022).

7. Target site mutations

This resistance mechanism is consequence of mutational in target enzymes that preserve their essential function in cell metabolism but make them immune to the selective inhibition caused by antibiotics. (Lambert; 2002). The gene structure of bacteria can undergo persistent modifications due to induced or spontaneous mutations. (Fernandez- Billon, et al., 2023).

P. aeruginosa mutations can change porin channels or antibiotic targets, which can result in altered antibiotic absorption or increased production of resistance genes (Verdial. *et al* ; 2023). High-level resistance linked to one or more point mutations in the same gene (gyrA), or mutations affecting many genes (gyrA and parC). (Abd Al- Absawe and Tuwaij., 2022). *Pseudomonas aeruginosa* is more prone to mutational resistance than Enterobacteriaceae because of their innately lower

vulnerability to permeability and efflux mechanisms. (Yousef. *et al.*, 2023). Mutations in the genes producing topoisomerase IV (parC and parE) and DNA gyrase (gyrA and gyrB) are linked to P. aeruginosa having less resistance to quinolones (Verdial.*et al.*,2023). These mutations may result in changes to these antibiotics target locations which would lower their binding affinity (Dweh and Rayanoothala., 2023). The above mechanisms of antimicrobial resistance are shown in Figure 2.

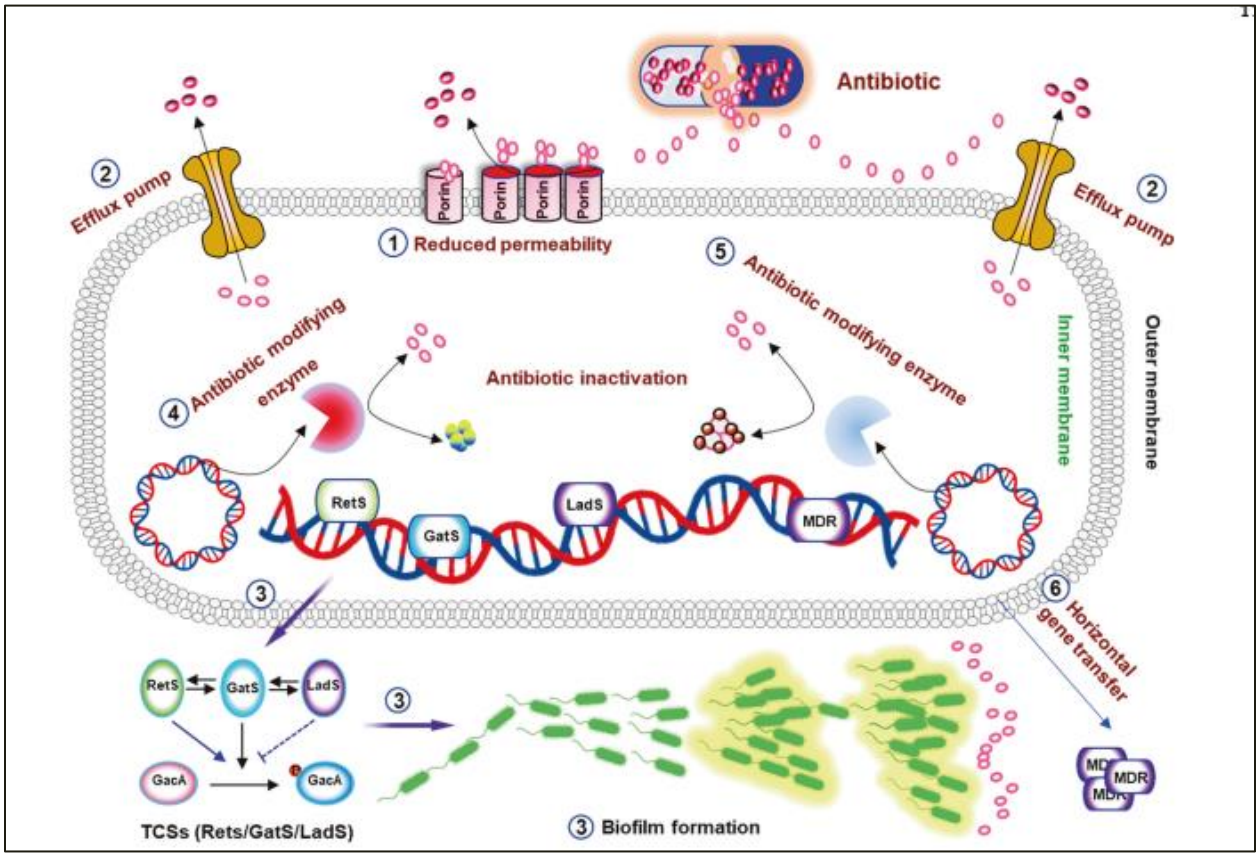

Figure (2-2): Mechanisms of antimicrobial resistance in *P. aeruginosa* (Qin, el at., 2022)

Drug penetration into cells is inhibited by altering the outer membrane protein porins, which lowers membrane permeability. Drugs are directly pumped out via the

efflux mechanism Enzymes that hydrolyze and modify drugs make them inactive. Similarly,Certain enzymes change the target so that the medication is unable to attach to it, which renders the medication inactive (Abd Al- Absawe and Tuwaij., 2022).

Horizontal gene transfer can result in the acquisition of antibiotic resistance genes on plasmids from either the same or distinct bacterial species.Antibiotics cannot en ter cells because biofilms function as physical barriers that are triggered qourum sensing signalin molecules (Hernando-Amado and Martinez, 2023).

2-5 Causes of antimicrobial resistance

Antibiotic resistance has developed since the middle of 20th century when antibiotics wewre first introduced and have benn ineffective in treating a number of illnesses in human. (Said *et al*.; 2023). Inappropriate usage of antibiotics is primary cause of antimicrobial resistanceHuman usage of antibiotics grew by 36% globally between 2000 an 2010 (Hoffman *et al;* 2015). In addition, extensive use of antibiotics in aquaculture and cattle , food chain transmission and direct contact are factors imact AMR . (Endale, *et al* ;2023). Half of this high usage is considered as unnecessary, e.g. when antibiotics are used to treat illnesses like infleunza that are caused by viruses, where antibiotics not effect on it (Hoffman *et al ;* 2015). Wildlife-related factors (transmission through interaction with wildlife and habitat encroachment) and environmental factors (release of antibiotics and resistant bacteria into water bodies , soil , and waste systems) are addressed .(Endale. *et al* ;2023). antibiotics are not controlled and available over the counter without a prescription in many countries . This lack of control results in antibiotics that are readily attaiable and inexpensive , which trigger overuse (Ventola:2023).

3- Materials and devices

Table (3-1) shown Materials and devices used in this study:

No.	Device name	The company
1.	Petri dish	China
2.	Incubator	German
3.	Vitek-2-compact	Franch
4.	Hood	Switzerland
5.	Autoclave	Japanese
6.	Loop	China
7.	Burner	China
8.	Mackoncky agar	Indian
9.	Blood agar	Indian
10.	Mueller Hinton Agar	Indian

3-1 Culture media preparation (Al-Khshali, 2020)

3-1-1 Blood agar medium

The blood agar medium was prepared by dissolving 33g of blood agar base in 950ml of D.W, pH was balanced to 7.0 and disinfected by autoclaving, then cooled to 45°C, after that 50ml of fresh blood was added, mixed well and poured into sterile petri dishes.

3-1-2 MaCconcky agar

Suspend 52 g of dehydrated macconky agar medium in 1000 ml of distilled water. Then heating to boiling to dissolve the medium completely. Sterilized by autoclaving pressure (121°C) for 15 minutes. The cooled to 45°C -50°C.Mixed well before pouring into sterile Petri plates

3-1-3 Mueller Hinton Agar

Suspend 38 g in 1 litre of distilled water, bring to the boil to dissolve the medium completely and sterilize by autoclaving at 121°C for 15 minutes. Mix and dissolve them completely. Pour the liquid into the petri dish and wait for the medium to solidify

3-2 Sterilization method

1- Wet heat sterilization [autoclaving]: It used for media ,solution at 121C for 15 min. pressure 15 psi
2- Dry heat sterilization for glassware and some tool in oven at 180C for 2 hours

3-3 Collection of clinical specimens

Samles were collected from november 2023 to february 2024 from various clinical Specimens (adult and children) in bequbah teaching hospital, Al-khalis general hospital and Al-Muqdadiya general hospital. These sample were transferred to the microbiology laboratory in these hospitals and cultured directly on blood agar ;MaCconkey agar and incubated for 24 hours at 37C◦.

3- 4 Identification of *P. aeruginosa* isolates

Isolates were examined depending on their color , size , shape , pigments and thier hemolytic ability for identification the type of bacteria:

3-4-1 Colonial morphology

The grown bacteria on the nutrient agar and Muller- Hinton agar are characterized by the following features: the bacterial morphology (smooth mucoid), grape odor, diffusible pigments on Muller-Hinton agar (bluish green or yellowish green)

3-4-2 Microscopic examination

The microscopic examination includes gram–stain reaction (negative), shape (rods), cells arranged with each other , presence of capsule and motility (mobile using two distinct forms of motility, swimming and twitching pattern).

3-4-3 Biochemical tests

3-5 Antibiotic susceptibility testing

P. aeruginosa isolates were tested for their sensitivity to commonly used antibiotics by minimum inhibitory concentration (MIC) method using vitek-2 compact device.

3-6 Identification of *P. aeruginosa* Isolates via VITEK2

3 ml of normal saline was placed in test tube and inoculated with a lope full of isolated colony. Then the test tube was inserted into a Dens Check machine for calibration of colony to McFarland standard solution (1.5×108 cell/ml). The calibrated solusion was placed in the cassette and a sample identification number scored into the computer software via barcode. The VITEK 2 compact type was then read from barcode placed on the card during production and the card was thus connected to the sample ID. The cassette was placed in the filler module, when the cards were filled, transferred the cassette to the reader/ incubator module.subsequent steps were handled by the instrument; the instrument controls the incubation temperature and the concentration of the used antibiotics .

Statistical analysis

Excel 2016 software was used to analysis the data we collected. We expressed the quantitative data by frequencies and percentages.

4. Results and Discussion

4.1 Collection and Identification of Bacterial Isolates

(345) different samples (urine, ear swab, throat swab and wounds) were collected from patients of different age groups and gender admitted to Baqubah Teaching Hospital, Al-Muqdadiya, and Al-Khalis Hospital in Diyala Governorate.
(183) isolates of *Pseudomonas aeruginosa* were obtained from the total number of samples. These samples were subjected to various laboratory tests to confirm isolation. Then we conducted a sensitivity test to different antibiotics to identify resistance rates

4.2 Identification of *P.aeruginosa*

4.2.1 Morphological and Microscopic Examinations

Results of morphological identification showed that growing colonies on plates were smooth mucoid , grape odor, bluish green or yellowish green on Muller-Hinton agar , under microscope the were apear in rode shape cells ,Gram negative , mobile using two distinct forms of motility (swimming and twitching pattern).

4.2.2 Biochemical tests

Pseudomonas aeruginosa bacteria have been identified based on their biochemical characteristics. The results are shown in Table (4-1).
All of which confirmed that the isolation was due to *Pseudomonas aeruginosa*, according to the instructions of the manufacturer of the kit prepared for the test.

Table (4-1) show Biochemical properties of *P. aeruginosa* isolates in this study

Biochemical details																
١	APPA	+	٩	ADO	-	١٧	PyrA	-	٢٥	IARL	-	٣٢	dCEL	-	٤٠	BGAL	-
٢	**H2S**	-	١٠	BNAG	-	١٨	AGLTp	-	٢٦	dGLU	+	٣٣	GGT	+	٤١	OFF	-
٣	BGLU	-	١١	dMAL	-	١٩	dMAN	+	٢٧	dMNE	+	٣٤	BXYL	-	٤٢	BALp	+
٤	**proA**	+	١٢	LIP	+	٢٠	PLE	-	٢٨	TyrA	+	٣٥	URE	-	٤٣	dSOR	-
٥	SAC	-	١٣	dTAG	-	٢١	dTRE	-	٢٩	CIT	+	٣٦	MNT	+	٤٤	5KG	-
٦	ILATK	+	١٤	AGLU	-	٢٢	SUCT	+	٣٠	NAGA	-	٣٧	AGAL	-	45	PHOS	-
٧	**GlyA**	-	١٥	ODC	-	٢٣	LDC	-	٣١	IHISa	-	٣٨	CMT	+	٤٦	BGUR	-
٨	O129R		١٦	GGAA	+	٢٤	IMLTa	+	٣٢	ELLM	-	٣٩	ILATa	+			

4.3 Susceptibility of *P .aeruginosa* to different Antibiotics

Our study showed increased levels of antibiotic resistance in *P.aeruginosa* among some of the groups under study, which included (penicillin, cephalosporins, carbapenems, aminoglycosides, fluoroquinolones, and polycationic peptides) as shown in Table (4.2):

Table (4-2): show Percentages of resistance in antimicrobial drugs.

NO	Antibiotic		NO. R (%)	NO. S (%)
	Class	Type		
1.	Penicillin	Amoxicillin	38 (58.4%)	27 (41.5%)
2.		Amoxicillin/ Clavulanic acid	38 (58.4%)	27 (41.5%)
3.		Ticarcillin	17 (26.1%)	48 (73.8%)
4.		Ticarcillin/ Clavulanic acid	9 (13.8%)	56 (86.1%)
5.		Piperacillin	18 (27.6%)	47 (72.3%)
6.		Piperacillin/ Tazobactam	14 (21.5%)	51 (78.4%)
7.	Cephalosporin	Cefuroxime	34 (52.3%)	31 (47.6%)
8.		Cefixime	31 (47.6%)	34 (52.3%)
9.		Cefotaxime	33 (50.7%)	32 (49.2%)
10.		Ceftazidime	9 (13.8%)	56 (86.1%)
11.		Ceftriaxone	31 (47.6%)	34 (52.3%)
12.		Cefepime	12 (18.4%)	53 (81.5%)
13.	Carbapenem	Imipenem	13 (20.0%)	52 (80.0%)
14.		Meropenem	13 (20.0%)	52 (80.0%)
15.	Aminoglycosides	Amikacin	12 (18.4%)	53 (81.5%)
16.		Gentamicin	9 (13.8%)	56 (86.1%)
17.		Tobramycin	12 (18.4%)	53 (81.5%)
18.	Fluoroquinolones	Ciprofloxacin	15 (23.0%)	50 (76.9%)
19.		Gemifloxacin	19 (29.2%)	46 (70.7%)
20.		Levofloxacin	15 (23.0%)	50 (76.9%)
21.		Moxifloxacin	14 (21.5%)	51 (78.4%)
22.	Polycationic piptide	Colistin	6 (9.2%)	59 (90.7%)

4-4 Penicillin group

Results of this study showed the amoxicillin and amoxicillin/ clavulanic acid scored highest resistance (58.4%) for each them. among penicillin class compared to ticarcillin/ clavulanic acid that scored lowest percentage (13.8%) as shown in following figure (4-1) :

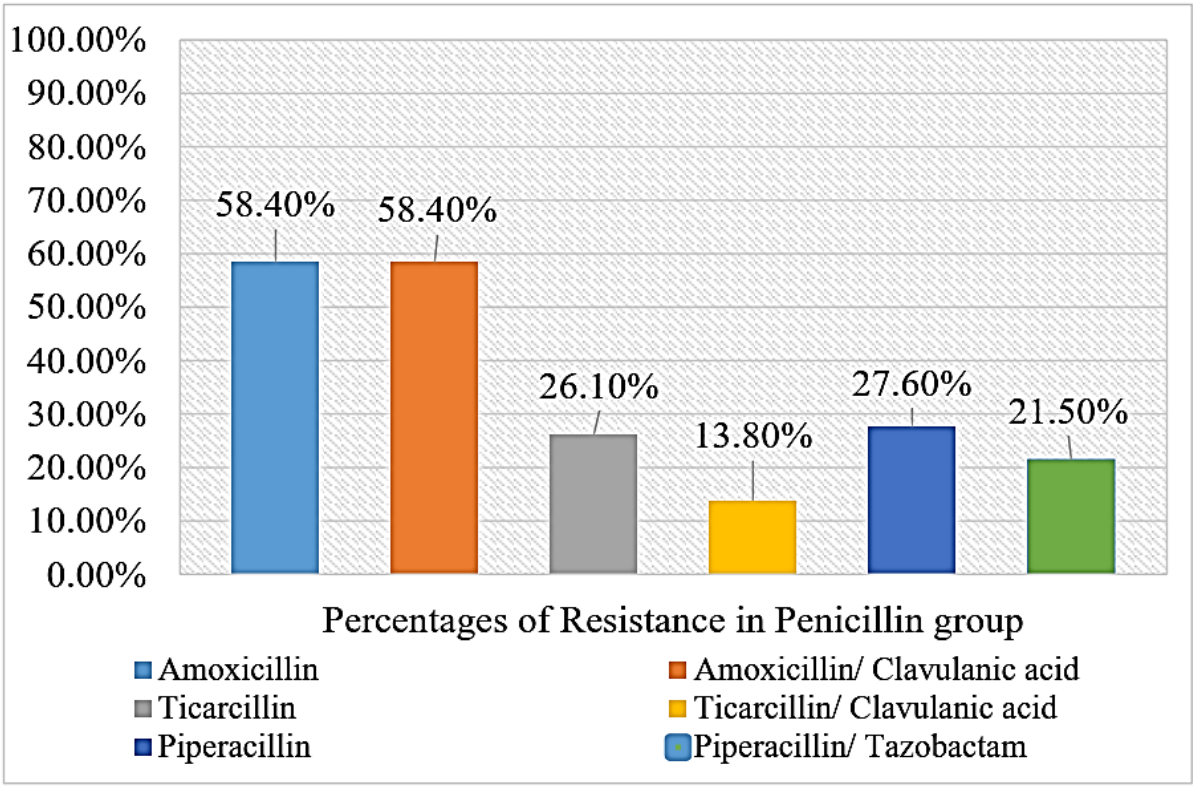

Figure (4-1): Percentages of resistance in Penicllin group

4-5 Cephalosporin group

Based on cephalosporin class, present findings mentioned the cefuroxime and cefotaxime scored highest resistance (52.3% and 50.7%) respectively, compared to

ceftazidime that scored lowest percentage (13.8%) as shown in following figure (4-2):

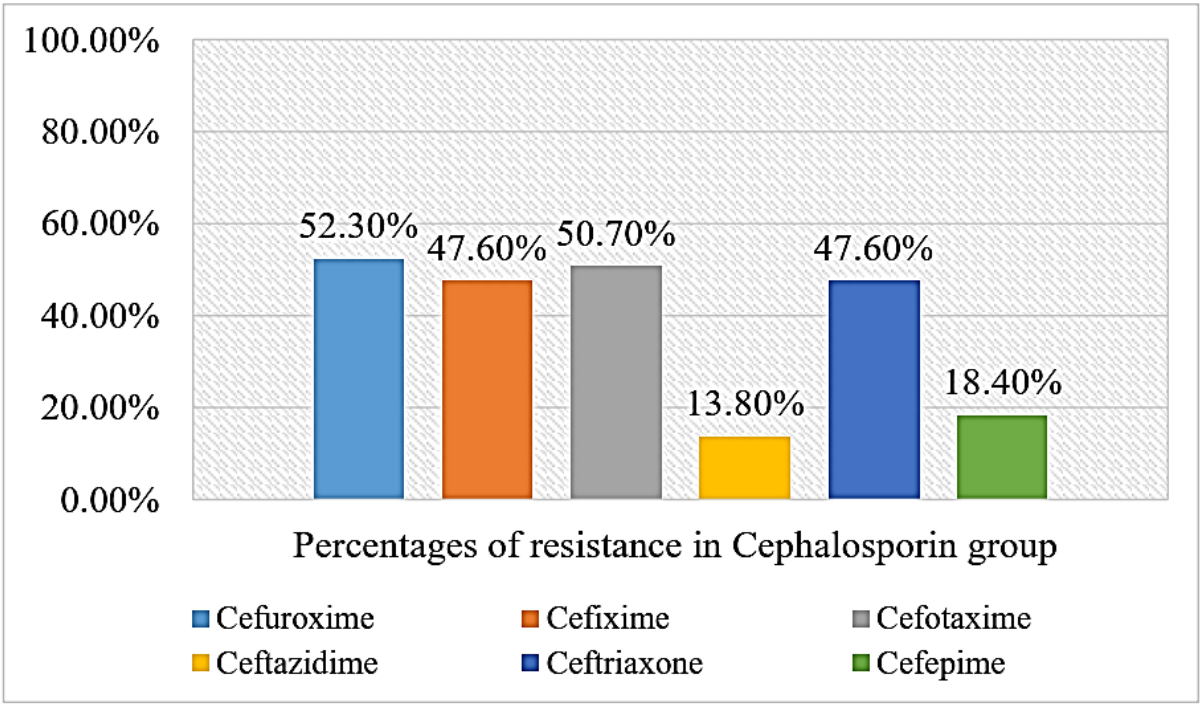

Figure (4-2): Percentages of resistance in cephalosporin group

4-6 Carbapenem and Aminoglycosides group

Present outcomes showed the imipenem and meropenem scored highest resistance (20.0% and 20.0%) among carbapenem group in participants.

While according to the amikacin and tobramycin scored highest resistance (18.4% and 18.4%) among aminoglycosides group in participants than gentamicin that scored lowest percentage (13.8%) as shown in following figure (4-3):

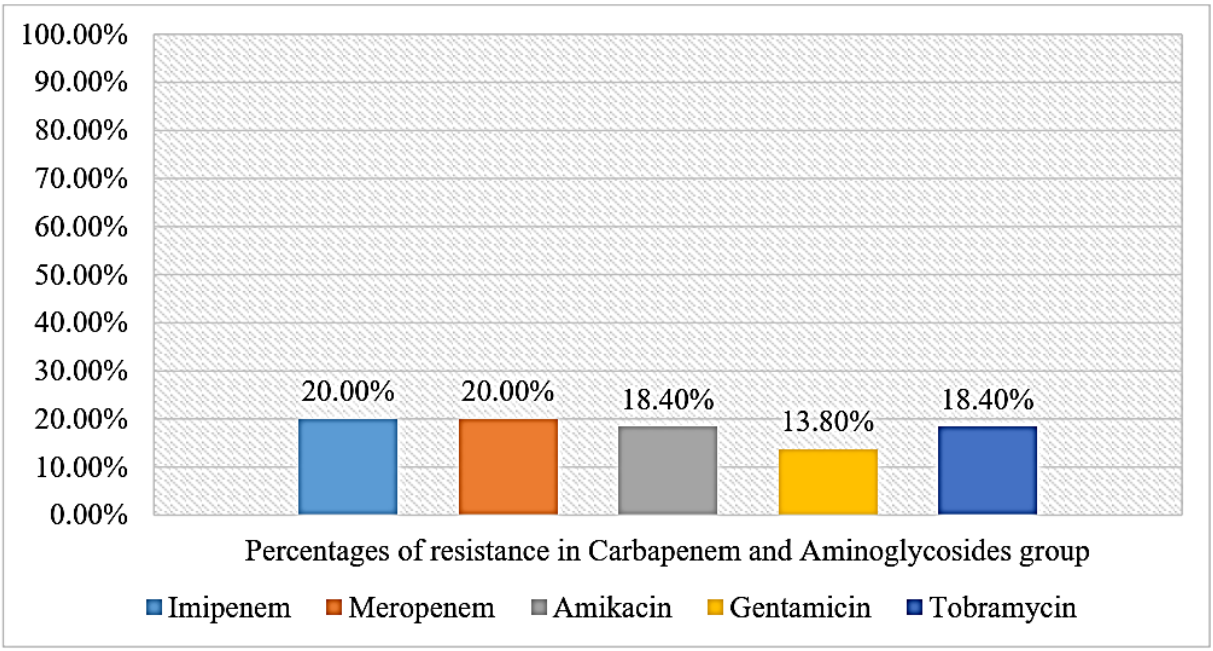

Figure (4-3): Percentages of resistance in Carbapenem and Aminoglycosides group

4-7 Fluoroquinolones and Polycationic piptide group

The research mentioned the gemifloxacin scored highest resistance (29.2%) among fluoroquinolones class in participants than moxifloxacin that scored lowest percentage (21.5%). Finally, colistin scored lowest resistance (9.2%) among polycationic piptide class as shown in following figure (4-4):

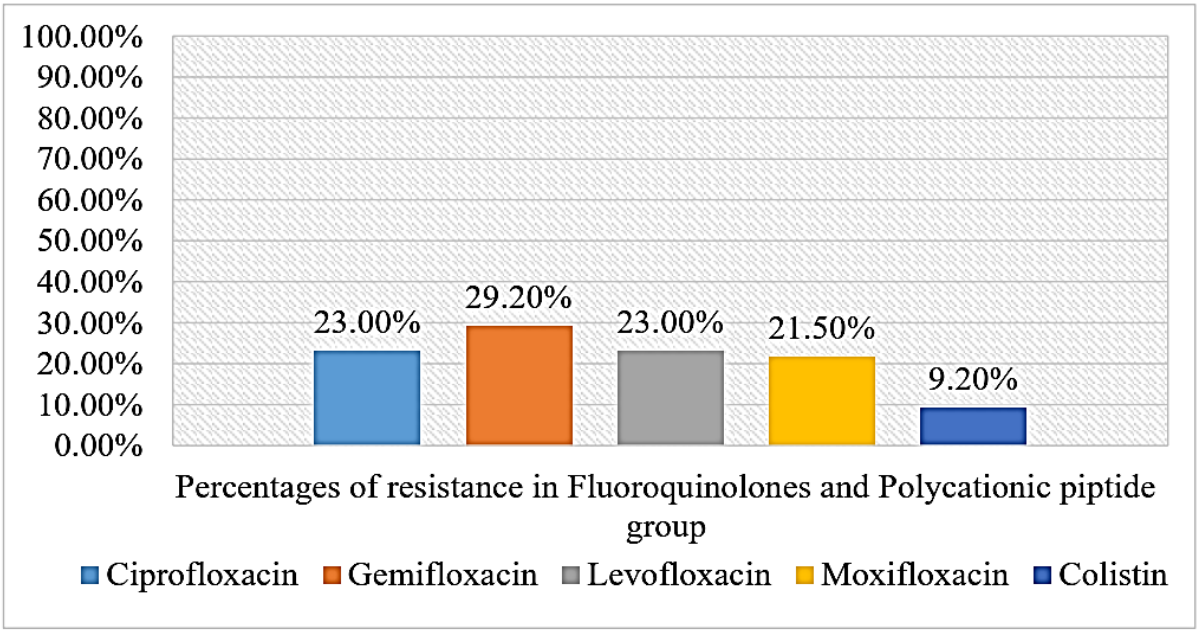

Figure (4-4): Percentages of resistance in fluoroquinolones and Polycationic piptide group

The reason for the high rates of resistance can be explained by the fact that bacteria are quick to adapt to different types of environments and are quick to spread due to their possession of different resistance mechanisms through which they were able to overcome various conditions and cause pathogenicity. The correct interpretation of antibiograms allows for the selection of the most suitable antibiotic therapy, enhancing patient outcomes and minimizing the risk of treatment failure. Conversely, misinterpretation can lead to inappropriate antibiotic use, inadvertently promoting the selection of resistant strains and exacerbating the issue of antibiotic resistance. Such mistakes endanger the patient's life, in addition to widespread negative effects on public health as the battle against *P. aeruginosa* continues.