Correlation between efflux pump and resistance to antibiotics in *Pseudomonas aeruginosa* isolated from different clinical cases

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ABSTRACT

Background: *Pseudomonas aeruginosa* is an opportunistic bacterium that causes nosocomial infections in hospitals. It can develop high-level multidrug resistance (MDR). The *MexA* gene is a constitutive chromosomal gene in the Resistance Nodulation Division (RND) family of *P. aeruginosa*. **Objectives:** This study aimed to detect the *MexA* efflux pump resistance gene in *P.aeruginosa* isolates. **Methodology:** A total of 165 specimens were collected from patients aged 5-65 years who suffered from burns, wounds, otitis media, and UTIs in two hospitals in Iraq during the period from November 2023 to January 2024. An antibiotic susceptibility test was performed using the Kirby-Bauer disk diffusion method in line with CLSI-2023. Finally, PCR was used to investigate the presence of the *MexA* gene in (25) *P. aeruginosa* isolates, which were highly resistant to Beta-lactam and quinolone antibiotics (MDR). **Results:** Out of 165 specimens, only 116 (70.30%) showed similar morphological traits and biochemical testing for *Pseudomonas aeruginosa*. Burn infections showed the highest isolation ratio, followed by wound infections, otitis media, and urinary tract infections (34.54, 17.59, 11.51, and 6.66) % respectively. Antibiotic resistance profile of (25) *P. aeruginosa* were 100% resistance to Ceftriaxone, Cefotaxime, and Amoxicillin-Clavulanic acid, while only 20% were resistant to Meropenem and Imipenem. *MexA* gene was found in 96% of *P.aeruginosa* isolates. **Conclusion:** The extensive identification of the *MexA* gene in *P.aerogenosa* indicates resistance to antibiotics associated with the function of the efflux pump gene and regulatory proteins.

Keywords: *P.aeruginosa*, MDR, RND, *MexA* efflux pump gene.

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INTRODUCTION

Pseudomonas aeruginosa is a rod-shaped, Gram-negative bacterium that is frequently encountered in medical environments. Due to its opportunistic nature, it presents significant hazards since it can cause nosocomial illnesses, which are associated with elevated death rates (1). It typically causes blood, urinary tract, and airways infections, and is the most common cause of burn infections, hot tub dermatitis, and outer ear infections (2). Antibiotic resistance is a major problem in treating and/or preventing illnesses caused by this bacterium. Multiple antibiotic resistance mechanisms, including efflux pump systems, have been demonstrated in *P. aeruginosa*, which can develop multidrug resistance (3). It has been suggested that *P. aeruginosa* multidrug resistance is influenced by the overexpression of efflux pumps (4). Efflux pumps are bacterial transport proteins involved in the extrusion of substrates from the cellular interior to the external environment (5). The majority of Gram-negative bacteria possess genes encoding efflux pumps of the resistance- Nodulation-Division (RND) family (6). The system comprises three components: a membrane fusion protein (MFP), an outer membrane factor (OMF), and a cytoplasmic membrane carrier. RND regulates molecules' movement between the cytoplasm and periplasm. RND complex genes are

organized in operons on the *P. aeruginosa* chromosome (7). Twelve systems from the RND family have been reported for *P. aeruginosa.* MexAB-OprM, MexCD-OprJ, MexEF-OprN, or MexXY (-OprA). Overexpression of specific RND efflux pumps leads to reduced susceptibility to their corresponding substrate antibiotics (8). i.e., Resistance to B-Lactam and Quinolone antibiotics is mediated by MexAB-OprM, while resistance to Fluoroquinolones, Tetracyclines, Macrolides, and some β-lactams, such as Cefepime, is mediated by MexCD-OprJ; on the other hand, Fluoroquinolone, Chloramphenicol, and Trimethoprim antibiotic resistance is influenced by MexEF-OprN, while MexXY-OprM is responsible for resistance toward Aminoglycosides, Cefepime, Ciprofloxacin, and Levofloxacin (9). Adaptive, acquired, and intrinsic resistance are examples of resistance mechanisms in *P. aeruginosa* (10). *P. aeruginosa* develops adaptive resistance through the production of biofilm, which offers protection against numerous antibiotics. *P. aeruginosa*'s adaptive resistance is enhanced by the production of biofilm, which offers protection from numerous antibiotics. *P. aeruginosa* might acquire antibiotic resistance genes from its environment through horizontal gene transfer, and mutations are contributing to this occurrence (11). Two primary factors contribute to *P. aeruginosa* intrinsic resistance: the production of enzymes that render drugs inactive and the increased expression of efflux pumps, which decreases outer membrane permeability. (12). This study aimed to detect the *MexA* efflux pump gene in *P. aeruginosa.*

METHODOLOGY

- **Bacterial isolation and identification**

During this study, a total of 165 specimens were collected from patients aged 5-65 years with burns, wounds, otitis media, and UTIs in two hospitals in Iraq: Al-Yarmouk Teaching Hospital and Burns Center at Al-Diwanya Hospital, during the period from 11/9/2023 to 30/12/2023. The specimens were obtained from burns, wounds, and otitis media using swabs from the infected area, while a urine sample was taken from UTI patients. Swabs were cultured on nutrient agar and MacConkey agar plates and incubated overnight at 37°C.

- **Ethical approval**

The research was approved by the Research Ethics Committee of the Diyala Health Directorate (No.273, dated 16/10/2023). The committee confirmed compliance with approved scientific and ethical standards.

- **Bacterial diagnosis methods**

Lactose-non-fermented colonies from MacConkey agar were cultured on cetrimide agar plates for isolation of *P. aeruginosa at* 37 °C overnight. Later, pure isolated colonies were subcultured on blood agar plates for an additional 18 hours at 37 °C. The isolated bacteria were identified based on their cultural features, biochemical tests, and by the VITEK 2 system. (13)

- **Antimicrobial sensitivity examination**

P. aeruginosa isolates were subjected to the antibiotic susceptibility tests by using the Kirby-Bauer disk diffusion method. (14)

Table 1: List of antibiotic discs

Antibiotics	Concentration	Family	Sign
Ceftriaxone	**30 (µg)**	ß -lactam(cephalosporin)	**CRO**
Cefotaxime	**30 (µg)**		**CTX**
Ceftazidime	**30 (µg)**		**CAZ**
Cefepime	**30 (µg)**		**FEP**
Imipenem	**10 (µg)**	ß-lactam (carbapenem)	**IPM**
Meropenem	**10 (µg)**		**MEM**
Piperacillin-tazobactam	**100/10 (µg)**	ß-lactam (penicillin)	**TZP**
Amoxicillin – clavulanic acid	**20/10 (µg)**		**AMC**
Aztreonam	**30 (µg)**	ß-lactam (monobactams)	**ATM**
Ciprofloxacin	**5 (µg)**	Fluoroquinolone	**CIP**
Levofloxacin	**5 (µg)**		**LVX**
Amikacin	**30 (µg)**	Aminoglycoside	**AK**
Gentamicin	**10 (µg)**		**CN**
Tobramycin	**10 (µg)**		**TOB**

- **MOLECULAR STUDY**
  - DNA Extraction

Five milliliters of overnight cultures from single colonies of each isolate were obtained in brain heart infusion broth. Cells were collected by centrifugation for 2 minutes at 12000 rpm. The pelleted cells were subjected to DNA extraction using the Wizard® Genomic DNA Purification Kit according to the company's instructions (Promega ,USA). The purified DNA was quantitatively assessed for purity and concentration (ng/µl) using a Nano Drop spectrophotometer.

  - Detection of efflux pump resistance *MexA* gene.

MexA gene detection was performed for each *P. aeruginosa* isolate, and a specific primer was designed based on the reference genome of *P. aeruginosa* PAO1 (GenBank accession no.NC_002516.2:472024-473175), using Primer3Plus version 3.3.0 online, as illustrated in Table 2. The polymerase chain reaction (PCR) was performed with a final volume of 20 µL under the optimized conditions as shown in Table 2. PCR products were separated on 1% agarose gel, and their predicted sizes were evaluated.

Table 2: The sequence of designed primers

Name of gene	Primer sequence (5´- 3´)	Product size (bp)	References
MexA	F: AGACGGTGACCCTGAATACC	108bp	This study
	R: CTTCCTTGAACAGGCGCTTG		

Table 3: Condtions of PCR reaction

Step	Temperature (°C)	Time (minutes)	No. of cycles
Initial Denaturation	95	5	1
Denaturation	95	0.5	35
Annealing	62	0.5	
Extension	72	0.5	
Final Extension	72	5	1

Table 4: The reaction mixture for PCR

Components	Volume (µl)	Final concentration
Go Taq®Green Master Mix (2X)	10	1X
Forward primer (10 µM)	1	0.5 µM
Reverse primer (10 µM)	1	0.5 µM
DNA template	2	< 250 ng
Nuclease free water	6	------
Final volume	20	------

RESULTS

- Isolation and identification of *P. aeruginosa*

116 *P.aeruginosa* were isolated from various clinical specimens. The source and percentage of these isolates are displayed in Table 5.

Table 5: Isolation number and ratio of *P. aeruginosa* from several clinical situations

Source of specimen	No.of specimens	No. of *P. aeruginosa* isolates	Percentage of total specimens %
Burns	65	57	34.54%
Wounds	40	29	17.59%
Otitis media	33	19	11.51%
UTIs	27	11	6.66%
Total	165	116	70.30%

Of the 165 clinical specimens with burns, otitis media, UTIs, and wounds, 116 isolates tested positive for *P. aeruginosa*. The highest isolation ratio was observed in burn infections, which were followed in order of severity by wound infections, otitis media, and urinary tract infections (34.54%, 17.59%, 11.51%, and 6.66%, respectively).

- Morphological characteristics

P. aeruginosa colonies on Cetrimide agar exhibited mucoid development, a smooth form with flat margins and an elevated center, a fruity odor, and yellow-green colonies. Bacterial culture on MacConkey agar medium, which distinguishes lactose-fermenting from non-lactose-fermenting bacteria, showed growth of small, pale colonies of *P. aeruginosa*, indicating that the bacteria do not ferment lactose. *P. aeruginosa* isolates under the microscope revealed a single, non-spore-forming, Gram-negative, rod-shaped bacterium.

- Biochemical tests

P. aeruginosa isolates were positive for Oxidase and Catalase, with a variable blood hemolysis pattern, while the Vitek 2 test detected *P. aeruginosa* in 116 out of 165 individuals with various illnesses, with a 95-99% identification rate.

- Antibiotic susceptibility test

The profile of antibiotic resistance in 25 isolates of *P. aeruginosa* revealed distinct patterns of resistance to various antibiotics Figure 1. A high level of bacterial resistance was observed against Ceftriaxone, Cefotaxime, and Amoxicillin-Clavulanic acid in 25(100%), Ciprofloxacin and Aztreonam 23(92%), Levofloxacin 22(88%), Tobramycin and Gentamicin 21(84%), Ceftazidime 18(72%), and Cefepime 16(64%). Meropenem and Imipenem 5(20%), Amikacin 7(28%), and Piperacillin-Tazobactam 9(36%) were found to have low levels of bacterial resistance.

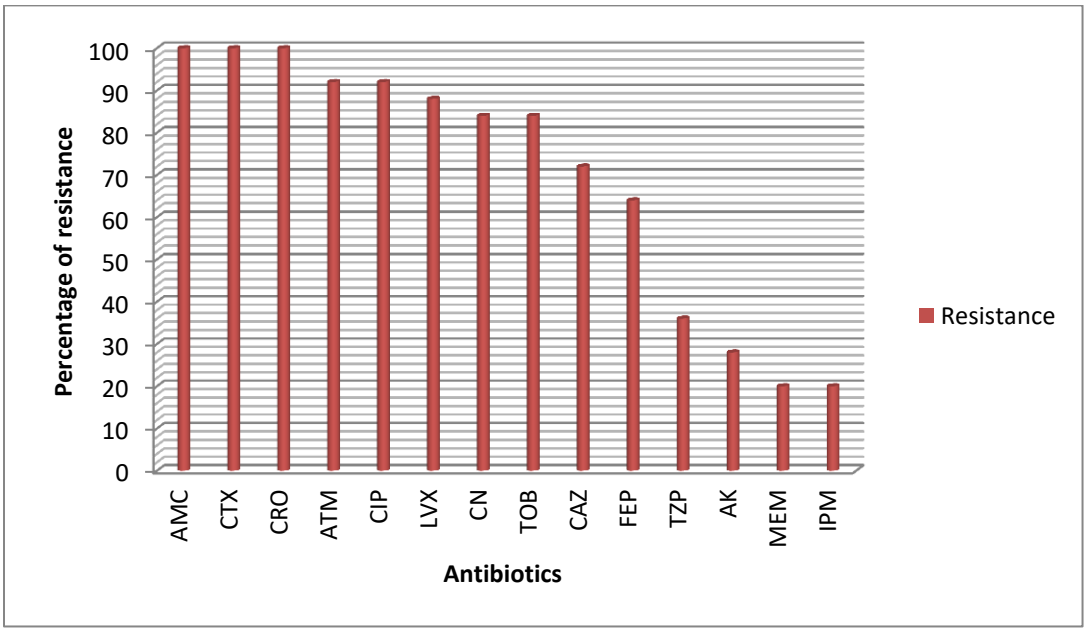

Figure (1): Percentage of *P.aeruginosa* isolates resistant to tested antibiotics.

Detection of *P. aeruginosa* efflux pump resistance *MexA* gene by PCR

The *MexA* efflux pump gene has been detected by PCR in 24 out of 25 *P. aeruginosa* isolates. As indicated in Figure. 2, as evidenced by the MexA band at 108 bp.

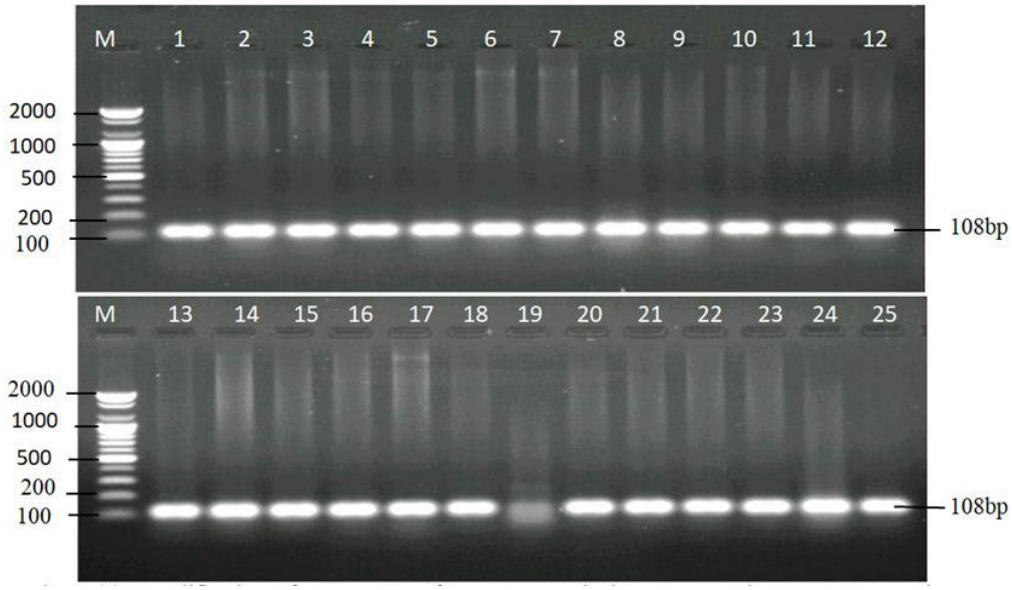

Figure (2): Amplification of *MexA* gene of *P. aeruginos* isolates separated on 1% agarose gel electrophoresis stained with Eth.Br. Lane M: 100 bp ladder marker. Lanes 1-25 resemble 108bp PCR products.

DISSCUSION

Ten to fifteen percent of nosocomial infections globally are caused by *P.aeruginosa*. The innate resistance of the species and its extraordinary capacity to develop new resistance mechanisms to a variety of antimicrobial drugs make these infections difficult to cure in many cases (15). In the current investigation, isolation results showed that 116 (70.30%) of the *P. aeruginosa* isolates were obtained from individuals with various clinical manifestations, indicating that *P. aeruginosa* was associated with infections across patient populations, and the highest isolation ratio was observed in burn infections, followed, in order of severity, by wound infections, otitis media, and urinary tract infections. The majority of *P. aeruginosa* isolates were obtained from burn patients, leading to the suggestion that this bacterium was the primary cause of nosocomial infections in burn units. (16,17)

These results were close to those of Qader (18), who isolated *P. aeruginosa* from burn wound infections at a rate of 39.6% from hospitals in Duhok and Erbil, Iraq. According to AL-Shamaa (19), the majority of *P. aeruginosa* isolates were isolated from burns (25/31) and wounds (6/31) in two hospitals in Baghdad. The results are consistent with another Iraqi study (20), which found that, out of 50 distinct patient samples recovered from Al-Diwanyia hospital, burns accounted for the greatest number and percentage of *P. aeruginosa* isolates (14/20), followed by otitis (11/16, 68%) and wounds(6/14, 42%). This variation in the presence of *P.aeruginosa* among the infected isolates can be attributed to a number of significant factors, such as variations in the type of samples, sampling technique and period, quantity of samples collected, patient age, sex, geographic location from which the samples were obtained, and other variables that vary among studies of this kind(20).

The results of the antibiotic sensitivity tests in this investigation indicate that all the isolates of *P. aeruginosa* were completely resistant (100%) to two antibiotics (Ceftriaxone and Amoxicillin-Clavulanic Acid). These findings corroborated the studies by Abbas and Hasan (16,17). Also, the results show that Meropenem and Imipenem have the highest effectiveness rate against *P. aeruginosa* isolates, which agrees with Hameed's results (21). These two antibiotics, which belong to the Carbapenems (β-lactam class of antibiotics), are commonly used to treat *P.*

aeruginosa infections. The mechanism of action involves inhibiting the transpeptidases that assemble peptidoglycans, also known as penicillin-binding proteins (PBPs), which are located on the outer layer of the plasma membrane (22) *MexA* is a constitutive chromosomal gene, efflux pump gene, that belongs to the membrane fusion protein (MFP) family located on the chromosome, which is thought to be in charge of bridging the inner and outer membranes. It's among the genes linked to *P. aeruginosa* drug resistance (23,24). Gene prevalence in this study was (24/25, 96%).These *MexA* gene finding were consistent with a local study by Abdulhady and Kadhim in Al-Diwaniyah city, Iraq, which found that 93.75% of *P. aeruginosa* isolates from various clinical cases had the *MexA* gene (25). However, only 96.2 percent of *P. aeruginosa* isolates from burn, wound, urine, ear, and sputum possessed the *MexA* gene, according to a local analysis carried out in Babylon, Iraq by Jameel (26). Another study carried out in Iran by Dorri, discovered that all *P. aeruginosa* isolates from burns, throats, and noses had *MexA* gene (27).

Conclusion: The extensive identification of the *MexA* gene in *P. aerogenosa* indicates antibiotic resistance associated with efflux pump function and regulatory proteins.

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العلاقة بين مضخات التدفق والمقاومة للمضادات الحيوية في الزوائف الزنجارية المعزولة من مصادر سريرية مختلفة

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الخلاصة

خلفية عن الموضوع : الزائفة الزنجارية هي بكتيريا انتهازية تسبب عدوى المستشفيات في المستشفيات. كما أنها واحدة من الكائنات الحية التي يمكنها تطوير مستوى عالٍ من مقاومة الأدوية المتعدد .MexA هو جين كروموسومي. يعد جين MexA أحد الجينات المرتبطة بمقاومة الزائفة الزنجارية للأدوية وهو جزء من عائلة (RND). **الهدف من الدراسة:** تهدف هذه الدراسة إلى الكشف عن وجود جين مقاومة مضخة التدفق MexAفي عزلات الزائفة الزنجارية .**المواد وطرق العمل:** تم جمع (165) عينة من مرضى بأعمار (5-65) سنة يعانون من الحروق والجروح والتهاب الأذن الوسطى والتهاب المسالك البولية في مستشفيين في العراق للمدة من 11/9/2023 إلى 30/12/2023. اختبار الحساسية للمضادات الحيوية باستخدام طريقة الانتشار القرصي Kirby-Bauer المتوافقة مع .CLSI-2023 وأخيراً تم استخدام PCR لفحص وجود جين MexAفي (25) عزلة من الزائفة الزنجارية وتم اختيار هذه العزلات اعتماداً على نمط مقاومتها (المقاومة العالية) للمضادات الحيوية بيتا لاكتام والكينولون (MDR) في اختبار الحساسية للمضادات الحيوية. **النتائج:** من بين 165 عينة، أظهرت 116 فقط (70.30%) سمات شكلية واختبارات كيميائية حيوية مماثلة لبكتيريا *Pseudomonas aeruginosa.* أظهرت إصابات الحروق أعلى نسبة عزل، تليها إصابات الجروح، والتهاب الأذن الوسطى، ثم التهابات المسالك البولية (34.54%، 17.59%، 11.51% و6.66% على التوالي) . أظهرت مقاومة المضادات الحيوية لـ 25 عزلة مقاومة بنسبة 25 (100٪) للسيفترياكسون والسيفوتاكسيم وحمض الأموكسيسيلين-الكلافولانيك، بينما كانت المقاومة 5 (20٪) فقط للميروبينيم والإيميبينيم. تم العثور على جين MexA بنسبة (96%) في عزلات الزائفة الزنجارية0 **الاستنتاج:** إن التحديد الشامل لجين MexA في بكتريا الزائفة الزنجارية يشير إلى مقاومة المضادات الحيوية المرتبطة بوظيفة جين مضخة التدفق والبروتينات التنظيمية.

الكلمات المفتاحية: الزائفة الزنجارية ، مقاومة الأدوية المتعددة ، قسم مقاومة العقد ، MexAاحد جينات مضخة التدفق.