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Antibiogram of *Pseudomonas aeruginosa* Isolated from Burn & Wound Infections Among Inpatients and Outpatients Attending to Ramadi Teaching Hospital in Ramadi, Iraq.

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ABSTRACT

ground: *Pseudomonas aeruginosa* is Gram- Negative bacterium and one of ng of Hospital and community-acquired infections. Over and improper use of iotics leads to significant changes in microbial genetic ecology of the rium that leads to the spread of multidrug resistance which become now a l problem. The study aimed to determine the prevalence of *Pseudomonas ginosa* burn &wound infections and their antibiograms toward common nercial antibiotics.

nts and Methods: Swabs were taken from patients with burn and wound ions. Specimens were examined microscopically as soon as possible (within our) by direct Gram-stained smears and indirectly by cultivation aerobically suitable culture media. Bacterial isolates were diagnosed and confirmed suitable diagnostic techniques. The antibiotics susceptibility was determined ne Kirby Bauer Disc diffusion method and the results were interpreted ding to the Clinical and Laboratory Standards Institute (CLSI) lines2018.

lts: A total of 34 isolates of *Pseudomonas aeruginosa* were isolated from a and wound infections during a period of six months, from September 20l8 to h 20l9. Antibiogram of *Pseudomonas* indicated that most of isolates were ant to Ceftriaxone (94.1%), Ceftazidime (94.1%), Cefotaxime (91.2%), acin(61.8%), piperacillin(52.9%) and to a lesser extent to Gentamicin(35.3% treonam(29.4%), and Tobramycin(26.5%). On the other hand most of the es were sensitive to Norfloxacin(76.5%), Ciprofloxacin(85.3%), Meropenem mipenem(91.2%)..

lusion: Carbapenems and fluoroquinolone antibiotics appeared to be the effective agent against *P. aeruginosa* isolates. On the other hand, losporins were quite not effective against *P.aeruginosa*.

INTRODUCTION

Pseudomonas aeruginosa is an opportunistic pathogen that can cause several infections in humans to include acute and chronic, and it has become a very important cause of Hospital-acquired infections and antibiotic resistance(Al-Wrafy et al., 2017). *P.aeruginosa*

has been considered as a ubiquitous organism because abilities to adaptation in a wide range of environments include soil, water, sewage, and hospitals (Mahmoud et al., 2013). This bacterium was able to multiply in many water sources such as seawater, rivers water, and even bottled water(Tirodimos et al., 2010). In addition, it's high resistance to most disinfectants (Ali et al., 2015). Among all gram-negative bacteria, *P.aeruginosa* has been recognized as a predominant opportunistic pathogen, which usually infects persons having some underlying diseases and compromised immune status (Porras-Gómez et al., 2012). In the hospital, *P. aeruginosa* usually attacks the patients with burn and wound infections, where further complicate of the primary condition, may occur and sometimes can cause bacteremia(Inacio et al., 2014).

Individuals most at high risk include those with an immunosuppressed patient, burn wound and cystic fibrosis patients(Church et al., 2006 .,LiPuma ,2010). Pathogenesis of the bacterium is mediated by multiple virulence factors that facilitate adhesion and disrupt host cell signaling pathways while targeting the extracellular matrix (Alhazmi, 2015). These virulence factors include Type IV Pili, lipopolysaccharide, Exopolysaccharide (Alginate), Type III secretion system, protease, Pigments, and Exotoxin A. These virulence factors play an important role in pathogenesis by facilitating colonization of bacterium to surface, survival and invasion bacterium of host tissues (Hogardt and Heesemann ,2011). Infections caused by *P. aeruginosa* are particularly problematic because the bacterium is inherently resistant to many classes of drug and has the ability to acquired resistance to many antimicrobial drugs(Baron ,1996).

The mechanism of antibiotic resistance in *P. aeruginosa* is multi-factorial and due to either intrinsic resistance or acquired resistance. The intrinsic pathway of resistance includes several mechanisms such as decreases in membrane permeability, efflux mechanism that pumping the antimicrobial agents outside of the cell wall and production of enzymes that cause inactivation of antibiotics (Breidenstein et al., 2011). Acquired pathway of resistance is the acquisition of resistance mechanisms via horizontal gene transfer and can occur during chemotherapy or is a consequence of mutational changes (Poole ,2011). The critical way to acquire drug resistance in multi-drug resistant *P.aeruginosa* is through the acquisition of plasmid. Plasmid-mediated resistance has been documented by several authors for the genetic transfer of several drug resistance genes(Shahid and Malik ,2003).

MATERIALS AND METHODS

Samples Collection, Isolation, and Identification:

Ninety-two skin swabs collected from inpatients and outpatients with a wound and burn infections from both sexes. Patients were attending to Ramadi Teaching Hospital and Private Clinics of Dermatology in Ramadi City , west of Iraq during the period extended from September to March 2019. Initial identification of *P. aeruginosa* bacterium was done on MacConkey agar, blood agar and cetrimide agar (Oxoid, Himedia). Biochemical identification of isolates was carried out by different biochemical test include catalase and oxidase test, IMVC test, KIA test .The diagnosis confirmed by using the VITEK 2 system.

Antimicrobial Susceptibility Test:

12 commercial common antibiotics including β-Lactam group, aminoglycoside group, monobactam group, and quinolones group had been tested to determine the sensitivity of *P.aeruginosa* by Kirby Bauer disc diffusion method. Suspensions of the isolates of 0.5 McFarland turbidity standard were made and Mueller Hinton Agar (MHA) plates were inoculated. Antibiotic discs of Meropenem (10 µg), Imipenem (10 µg), Amikacin(10µg),Gentamicin(10µg),Tobramycin(10µg),Piperacillin (100µg), Aztreonam (30µg),Ceftazidime(30µg),Cefotaxime (30µg), Ceftriaxone (10µg) Ciprofloxacin

(10 µg), Norfloxacin (10 µg) were applied on the plates.

The incubation of bacterium was done at the temperature of 37°C in aerobic conditions for18-24 hours(Rao and Rao , 2009) . The results were interpreted according to the Clinical and Laboratory Standards Institute (CLSI) guidelines2018. The inhibition zones were controlled with the reference *Escherichia coli* ATCC10536 and *Pseudomonas aeruginosa* ATCC154427.

RESULTS AND DISCUSSION

Sex and Hospitalization:

Ninety-two skin swabs obtained from inpatient admitted to Ramadi Teaching Hospital and outpatient attending both Consulting Clinic in the same Hospital and Dermatology private clinics in Ramadi city were randomly collected and examined for detection *P.aeruginosa* bacterium. A total of (N=60, 65.2%) represented skin swabs from hospitalized patients, while (N=32 , 34.8%) were represented skin swabs from non-hospitalized patients. On the other hand, there were (n=48, 52.2 %) skin swabs from males and (n=44 , 47.8%) skin swabs from females (Table1).

Table 1: The source of specimens regarding hospitalization & gender of the patient

Gender	Hospitalization					
	Inpatient		Outpatient		Total	
	No	%	No	%	No	%
Male	31	51.7	17	53.1	48	52.2
Female	29	48.3	15	46.9	44	47.8
Total	60	100	32	100	92	100
P=0.814 (Not significant using Pearson Chi-square test at 0.05 level.						

Isolation and Identification of *Pseudomonas aeruginosa*

Thirty-four isolates of *P. aeruginosa* were obtained of the total (92) clinical specimens (wounds & burns). The preliminary cultural diagnosis was done on blood agar and MacConkey agar. Most of the isolates appeared β-hemolysis on blood agar while others isolates were non-hemolysis. All isolates grew on MacConkey agar but appeared pale color colony because of an inability to ferment lactose sugar. Suspected *P.aeruginosa* samples re-cultured in Cetrimide agar medium to confirmed diagnosis of the bacterium, where *P.aeruginosa* bacterium differ from other *Pseudomonas* species by growing on cetrimide agar which considered selective medium for this bacterium because *P.aeruginosa* has the ability to resist cetrimide material which considered a toxic material for other bacteria(Forbes et al.,2007) . Also, it produced Greenish-yellow color through growth in this medium. All the isolate grew on the Muller- Hinton agar with produced the diagnostic pigments .The pigments varied from yellowish-green to bluish green (figure 1). Also, the isolates produced a sweat grape-like odor. In this study, the biochemical tests were carried out and the result compared with standard result documented by(Church, 2016). The diagnosis was confirmed by using the VITEK 2 system. Classified all isolates of *P. aeruginosa* by a source of infection (Table 2).

Table (2) *P. aeruginosa* according to a source of infection

Sample	Total	NO. of positive *p.aeruginosa isolates*	Percentage %
Burn	32	15	46.8
Wound	60	19	31.6
Total	92	34	

The results of this study indicated that the largest proportion of the isolates were within the burn samples 15 (46.8%) isolates of total 32 burn swabs, while the proportion of isolates in the wounds samples were 19(31.6%) isolates of total 60 wound swabs. These results agreed with(Alkaabi ,2013) who found that *P.aeruginosa* is one of the more common bacterial species that causes burns and wounds infection in hospitals. On the other hand disagreed with (Negi et al., 2015) that recovered low percentage of 7.9% of *P.aeruginosa* isolate obtained of SSI in the previous study carried out in India. *P. aeruginosa* is the leading cause of invasive infections in burn patients; 75% of all deaths in patients with severe burn are related to sepsis from invasive burn wound infection (Barrow et al., 2004)

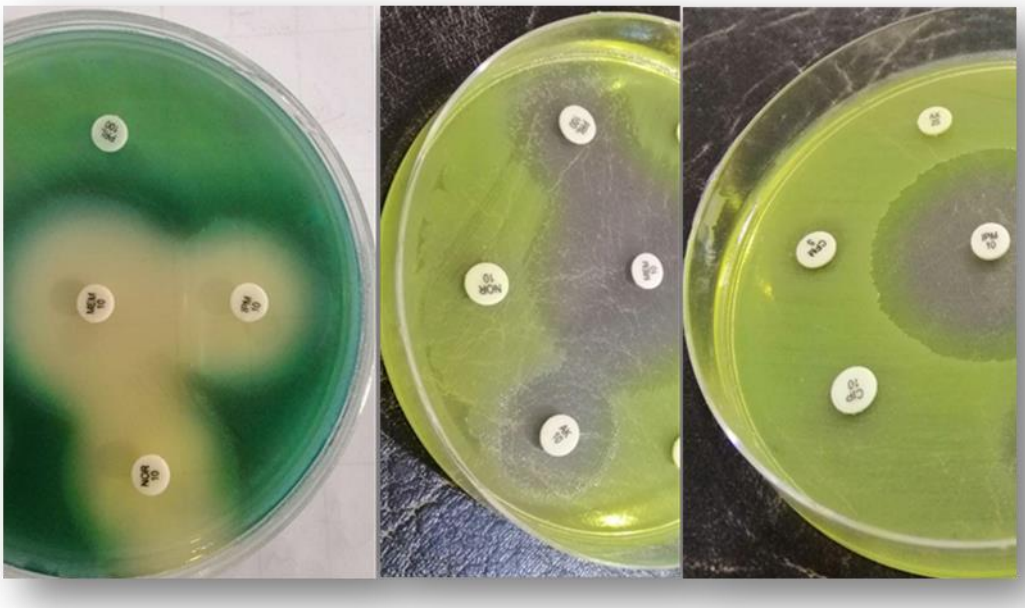

Fig. 1: yellowish-green to bluish green pigments produced by *P.aeruginosa* isolates on Muller- Hinton agar medium.

Antimicrobial Susceptibility Test for *P.aeruginosa*:

Standard disk diffusion method was used to determine the sensitivity of *P.aeruginosa* against 12 commercial common antipseudomonal antibiotics. Antimicrobial susceptibility testing was carried out for 34 isolates and the results are depicted in [Table3/Fig-2].

P.aeruginosa has the ability to resist many of antibiotics, and this ability either be normal or may be acquired through mutations in

their genetic material or through the horizontal genes transferees (Saderi and Owlia, 2015). The antimicrobial susceptibility profile of the *P. aeruginosa* isolates revealed that most of the isolates were resistant to Cephalosporins such as Ceftriaxone, Ceftazidime, and Cefotaxime with percentage 94.1 %, 94.1%, 91.2% respectively. These results agreed with (Shaikh et al., 2015) who reported resistance to Ceftazidime was (91.49 %) and (Qasim, 2006) as the ratio of resistance to Cefotaxime 92%, also (AL-Taai, 2016) who reported high percentage of resistance to Cefotaxime (85.71%) and Ceftriaxone (85.71%), while these results were different from that of (Al-Gherawi, 2009) who reported the percentage of Cefotaxime resistance were 66.7% .

Results showed that the percentage of *P. aeruginosa* isolates were resistant to Piperacillin (52.9%), and this result was Close with(Negi et al., 2015) who reported (45.5 %) of Piperacillin resistance and disagreed with (Abdullah et al., 2010).

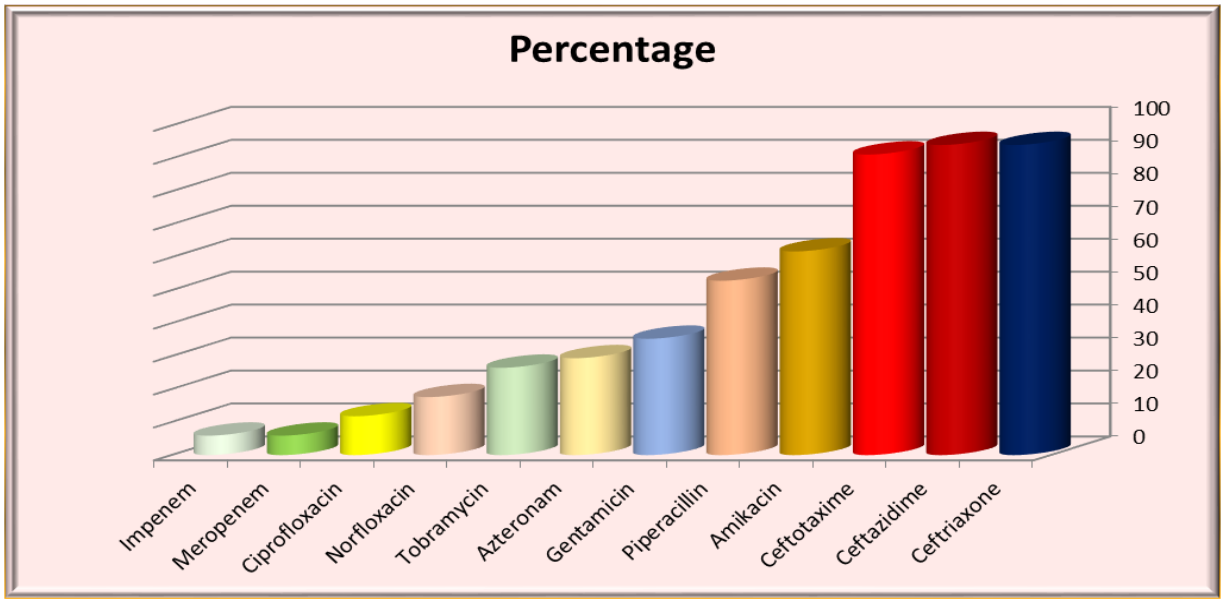

Fig. 2: The distribution of resistant *ps. aeruginosa* using Disc Diffusion Method (n=34)

The high resistance of *P.aeruginosa* isolates to β-lactam and cephalosporin's antibiotics may be due to the breakdown of the β-lactam ring which interferes with antibiotics structure and inactivate it by their production of β-lactamase enzymes. This is plasmid-mediated or by decreasing membrane permeability towards the antimicrobial agents(Al-Falahy ,2000). Excessive and improper use of antibiotics may play an important role in the development of resistance to β-lactam antibiotics. A previous study found a relationship between excessive use of antibiotics and the frequency of resistance toward antibiotics(Smith et al., 2000).

In the present study, it was found that *P.aeruginosa* isolates exhibited different rates of resistance towards the aminoglycoside antibiotics which include Amikacin, Gentamicin, and Tobramycin with a percentage (61.8 %, 35.3%, and 26.5%) respectively. This result agreed with (Amutha et al., 2009) who reported the highest resistance of *P.aeruginosa* strains against Amikacin (62.2%) and (Elhariri et al., 2017) who found (28.5%) resistance rate of Gentamicin, while the results disagreed with (AL-Taai ,2016) who reported resistance rate of Amikacin (30.35%) and Gentamicin (60.71%) and (AL-Salihi et al., 2014) who found *P.aeruginosa resistance* to

Amikacin, gentamicin, and Tobramycin were 75 %, 97.3%, and 87.5% respectively .

The current study revealed that antipseudomonal effect of gentamycin was higher than amikacin. This finding uncorrelated with other studies conducted by (Smitha et al., 2005) and (Poole, 2005) who found that resistance to amikacin of *P. aeruginosa* was still lower than to gentamicin. The mechanisms of resistance to aminoglycoside antibiotics in clinical isolates are usually controlled by enzymatic inactivation of the antibiotic or due to abundant secretions of alginate by the bacterium which linked with the positive charge antibiotics and prevents its spread into the cell(Lambert, 2002).

Fluoroquinolone antibiotics (Ciprofloxacin, Norfloxacin) gave good effectiveness towards most of *P.aeruginosa* isolates, where the percentage of sensitivity was (85.3 %, 76.5%) and the percentage of resistance was (11.8%, 17.6%) respectively. This result agreed with (Al-Qasi ,2012) who found that only 8.6 % of isolates were resistant to Ciprofloxacin and disagreed with (Haleem et al., 2011) who observed resistance percentage of ciprofloxacin (31.25%) and Norfloxacin(37.5%). The main mechanisms of resistance are mutations in the target genes (Williams et al., 2006).

Table 3 : The antibiogram of *p. aeruginosa* isolates.

		No (%)		
Antibiotics		**Resistant**	**Intermediate**	**Susceptible**
Symbol	**Name**			
PRL	Piperacillin	18(52.9)	6 (17.6)	10 (29.4)
AK	Amikacin	21 (61.8)	6 (17.6)	7 (20.6)
CN	Gentamicin	12 (35.3)	7 (20.6)	15(44.1)
TOB	Tobramycin	9 (26.5)	1 (2.9)	24 (70.6)
CAZ	Ceftazidime	32 (94.1)	0 (0)	2 (5.9)
CRO	Ceftriaxone	32 (94.1)	0 (0)	2 (5.9)
CTX	Ceftotaxime	31 (91.2)	1 (2.9)	2 (5.9)
CIP	Ciprofloxacin	4 (11.8)	1 (2.9)	29 (85.3)
NX	Norfloxacin	6 (17.6)	2 (5.9)	26 (76.5)
ATM	Azteronam	10 (29.4)	1 (2.9)	23 (67.6)
IMP	Impenem	2 (5.9)	1 (2.9)	31 (91.2)
MEM	Meropenem	2 (5.9)	1 (2.9)	31 (91.2)

Most of the isolates were found to be highly sensitive to Carbenem antibiotics including Imipenem and Meropenem, where the sensitivity rate was (91.2%), in the same times the resistance rate was (5.9%). This result agreed with (Begum et al., 2013) who reported sensitivity rate toward Imipenem (93.3%) and was slightly different from an earlier report by (Amutha et al., 2009) who reported resistance rate of *P.aeruginosa* strains against Imipenem (5%), Meropenem (17%), while this finding was too far than (Strateva et al., 2007) who found the resistance rate of *P.aeruginosa* against imipenem (42.3%), meropenem (45.5%) in a previous study carried out in Sofia, Bulgaria. The result of this study showed that Carbapenems and Fluoroquinolone antibiotics had remarkable activity against *P.aeruginosa* and this could be due to its proper and infrequent use in the treatment so can be considered as the drug of choice for treatment *P.aeruginosa* infections

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ARABIC SUMMARY

اختبار حساسية المضادات الحيوية لبكتريا الزائفة الزنجارية المعزولة من أخماج الجروح والحروق للمرضى الراقدين وغير الراقدين الحاضرين الى مستشفى الرمادي التعليمي في مدينة الرمادي-غرب العراق.

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جمعت اثنان و تسعون عينة شملت اخماج سريرية (الجروح والحروق) من المرضى الراقدين وغير الراقدين في مستشفى الرمادي التعليمي خلال الفترة من أيلول 2018 الى أذار 2019 . زرعت جميع هذه العينات على اوساط أكار الدم المغذي , وسط الماكونكي أكار ووسط السترمايد أكار وحضنت جميع الاطباق هوائيا وبدرجة حراره 37 درجة مئوية لمدة تتراوح من 18 الى 24 ساعة . اظهرت نتائج العزل والتشخيص وجود (34) عزلة من بكتريا الزائفة الزنجارية *Pseudomonas aeruginosa* وتم تأكيد التشخيص باستخدام نظام VITEK 2. أظهرت نتائج الحساسية للمضادات الحيوية بأن غالبية عزلات الزائفة الزنجارية كانت مقاومه للسيفالوسبورينات وبنسبة (94.1%) لكل من Ceftriaxone وCeftazidime , ونسبة(91.1%) لمضاد Cefotaxime . كما أن هذه العزلات كانت مقاومه لعدد من المضادات وبالنسب التالية Amikacin(61.8%), Piperacillin(52.9%) , Gentamycin(35.3%), Aztreonam(29.4%), Tobramycin(26.5%) . في حين غالبية عزلات الزائفة الزنجارية كانت حساسة تجاه مضادات Norfloxacin (76.5%) , Ciprofloxacin(85.3%). كما كانت عالية الحساسية لمضادات Impenem و Meropenem وبنسبة (% 91.2) لكليهما .