International Journal of Research Studies in Science, Engineering and Technology
Volume 4, Issue 12, 2017, PP 30-36
ISSN : 2349-476X

Isolation and Identification of Multi Drug Resistant Pseudomonas *Aeruginosa* Causing Wound Infection in Erbil City

Fattma A. Ali, Zainab Abdulwahab, Sara Asaad, Shkofa Muhammad

Hawler Medical university/College of Health Sciences

***Corresponding Author:** Fattma A. Ali, Hawler Medical university/College of Health Sciences, Egypt.*

ABSTRACT

Gram-negative bacilli Pseudomonas aeruginosa is an important pathogen in hospitalized patients, contributing to their morbidity and mortality due to its multiple resistance mechanisms. Therefore, as therapeutic options become restricted, the search for new agents is a priority. Latterly an accelerated increase in frequency of multidrug-resistant clinical strains has severely limited the availability of therapeutic options. In this study one hundred and forty four samples of wound were collected from patients admitted to Hawler teaching hospital, Rizgary hospital, Par hospital in Erbil city, during this period of September to march 2016. Twenty isolated of pseudomonas aeruginosa were identified by using cultural, morphological, characteristics, and biochemical test and Vitek 2 system. Results of pigments production revealed the ability of Pseudomonas aeruginosa to produce various pigments including blue/green, yellow/green, and brown/blue. The highest percentage of P.aeruginosa in female 11(19.64) were higher than those in male patients 9(10.22).Susceptibility of Pseudomonas aeruginosa isolates to different antibiotic was examined. Imipenem and pip/Tazo was the most effective antimicrobial agent against Pseudomonas aeruginosa isolates and most of isolates showed that high resistance to Trimeth/sulfa 80%.On the other hand our results indicated that most isolates of P.aeruginosa were multi resistance which show resistance to≥4 antibiotics. The problem of antimicrobial resistance in bacterial pathogens has been fairly described as a growing global crisis. Rate of reported resistance in common pathogens are reaching levels in many corners of the world preclude the empirical use of many our most potent and reliable antimicrobial agent.

Keywords: *Pseudomonas aeruginosa, wound infection, antimicrobial resistance*

INTRODUCTION

Pseudomonas aeruginosa is a G-negative bacteria belonging to the proteobacteria phylum [1]. *P. aeruginosa*is one of the most virulent opportunistic infectious agent of man [2].Which involved in hospital infections and causing illness in immunocomprised patients One of the major causes of serious infections in burn patients, resulting in mortality as high as 50% [3]. *P.aeruginosa* is primarily a nosocomial pathogen. According to the centers for Disease Control and Prevention (CDC), the overall incidence of *P.aeruginosa* infection in United States hospital averages about 0.4 percent (4 per 1000 discharges), and the bacterium is the fourth most commonly – isolated nosocomial pathogen accounting for 10.1 percent of all hospital- acquired infection .It has been estimated that about ten percent of all hospital-acquired infections are caused by *P. aeruginosa,* and for immune-compromised patients the mortality rate range from 20 to 70 % [4].When pathogenic bacteria are entering host tissue, different substances called virulence factors are released from the bacteria. Virulence factors cause damage to tissues through toxicity and invasiveness and allow for establishment of bacteria. *P. aeruginosa* produce a variety of virulence factors, both extracellular and cell-associated products. [5]. Pyocyanin is the major phenazine pigment produced by *P. aeruginosa*and has been shown to contribute to its pathogenicity. The presence of pyocyanin is easy to detect due to its blue color that turns stationary phase cultures of *P. aeruginosa* green, and is commonly found to stain infected tissues, pus, or dressings [6]._Examined wound dressings from burn patients infected with *P.aeruginos a*nd found that four of seven dressings contained pyocyanin. A wound is said to be infected when the invading

microorganisms cause notably impaired wound healing. When infectious bacteria are invading a host, toxic substances are produced by the microorganisms that cause damage to the host tissues. These substances are called virulence factors and allow the bacteria to establish in the host. The host responds to the bacterial invasion with attack of inflammatory cells such as neutrophils which release cytotoxic enzymes, oxygen radicals and inflammatory mediators which cause further damage to host tissue. This host response mechanism is also contributing to the non-healing stage of the infected wound [7]..Once established in a wound, the bacterium is almost impossible to eliminate with antibiotics due to biofilm formation [8].All wounds are contaminated by bacteria, meaning that microorganisms are present but not replicating. Wounds become contaminated from endogenous sources such as the gastrointestinal tract, the surrounding skin, the environment or from the healthcare provider (Sibbald *et al*., 2003). The classic signs of infection in wounds include swelling, redness, pain, heat, purulence, and impaired function. Chronic wounds can also show symptoms such as low transcutaneous oxygen tension, development of necrotic tissue, foul odor and wound breakdown as well as deterioration and discoloration of granulation tissue and increased friability [9].One of the most common pathogens in chronic wounds is, *P.aeroginosa*. A very problematic microbe due to its ability to form resistant biofilms [10].However wounds or burns disrupt the barrier and weaken the immune system, allowing opportunistic pahogenes such as *P.aeroginosa* to take advantage. The hospital environment tends to cultivate the multi drug resistance *P.aeroginosa* strains, increase the rate of complications caused by MDR pathogens. *P. aeruginosa*is an opportunistic pathogen, often acquired in hospital environments and is often associated with respiratory and urinary infections, in burn damage wounds, and in chronic wounds [1].Mechanisms of resistance to antibiotics in *P.aeruginosa* are either based on non-mutational intrinsic resistance or mutational acquired resistance .Fluoroquinolones and Aminoglycosides are two important classes of antibiotics used in the treatment of *Pseudomonas* infection. *Pseudomonas* readily develops resistance to these agent, reducing the antibiotic effectiveness [11].Haleem *et al.*, [12].obtained 48 *P. aeruginosa* isolates from wound and burn. They were resistant (100%) to Ampicillin, Cefotaxime, Chloramphenicol, Penicillin, Doxycycline, and Erythromycin, while they exhibited moderate resistance to Amikacin 19(35.5%), Ciprofloxacin 15(31.26%) and Polymyxine 29(40%), but they were sensitive to Pipracillin, Ticarcilline. Mukerjee *et al*., [13].found that all isolates *of P.aeruginosa* obtained from calculate medical college and hospital were multiple resistant to many antibiotic including several B- Lactams , Cephalosporins , Aminglycosidws, Fluoroquinolones ; however , Piperacillin, Carbenicillin, Amikacin and Ciprofloxacin were the only drugs whose resistance levels much lower . demonstrated that plasmid was identified in *P.aeruginosa* as an agent confer resistance to the Streptomycin, Sulfanilamide , Gentamycin and Carbencillin . It has been classed as abroad range plasmid due to its ability to replicate in both *E.coli* and *P. aeruginosa* .

MATERIALS AND METHODS

Sample Collection

From September throughout march2016, a total of 144 sample, taken from one source (wound) from patient admitted to Hawler teaching hospital, Par hospital, and Rizgary hospital of Kurdistan Region in province of Erbil.

Phenotyping identifications of *Pseudomnas Aeruginosa*

Swabs from specimens were plated on blood agar and MacConkey agar, a single transparent non-lactose fermenting colony was selected then subculture on appropriate medium. First, a Gram-stain was performed, Gram-negative rods with no particular arrangements. The organism was again grown on MacConkey agar plate to produce colorless colonies non lactose fermenter, *P. aeruginosa* expresses the expigment pyocyanin, which was blue-green in color, and the colonies will appeared flat, large and oval. It has also a characteristic fruity smell, when identified provisionally as *P.aeruginosa* it was sub cultured on nutrient agar slant after incubated at 37 C^0 for 24 hours, then stored at $4C^0$ in refrigerator [14].

Vitek2 Compact System

The newly redesigned colorimetric Vitek2 compact system with updated advanced expert system (AES) (bioMerieux, Marcy l'Etoile, France) was evaluated for its accuracy and rapidity to identify clinical isolates and to detect several antimicrobial resistance [15].

Principles of the Vitek2 is an automated microbiology system utilizing growth-based technology. This system accommodate the colorimetric reagent cards that are incubated and interpreted automatically.

Overall, the Vitek2 gave 95.8% of compatibility with the reference API strips (bioMerieux) in the identifications (ID) s of the Gram- positive cocci (GPC), Gram-negative rods (GNR), and yeasts. The accuracy was finally estimated to 98.3% through additional confirmatory tests. Also, >90% of identifications of GPC and GNR were obtained within 7 hours of incubation. The most resistant isolates were identified within 12 hours of incubation. In conclusion, the new colorimetric Vitek2

Identified within 12 hours of incubation. In conclusion, the new colorimetric Vitek 2 compact system with AES greatly improved is accuracy in species identification and detection of antimicrobial resistances, and it will be highly acceptable to clinical microbiology laboratory function [16].

Antimicrobial Susceptibility Testing

Disc diffusion method, also known as the Kirby-Bauer method was carried out according to the Clinical and Laboratory Standard Institute guidelines (CLSI), formerly the National Committee for Clinical Laboratory Standards (NCCLS) [17].

- Muller Hinton agar was prepared and sterilized then the medium was cooled to 45˚C and poured to sterilized Petri dish.
- Muller Hinton agar containing 5% blood is used for testing Streptococci.
- Inoculums suspension were prepared using colony suspension standardized to
- Match the turbidity of a McFarland 0.5 standard.
- The plates were inoculated by a sterile cotton swab dipped in bacterial suspension, then swabbed evenly across the surface of a Muller Hinton agar plates.
- Sterile forceps was used to place the antibiotic disc on the surface of the inoculums.
- The inoculated plates were incubated at 37˚C for 18-24 hours.
- After incubation the diameters of inhibition zone produced by antimicrobial inhibition of bacterial growth were measure using a ruler.
- Each inhibition zone interpreted chart table which is recommended by NCCLS

Results and Discussion

Frequency of *Pseudomonas Aeruginosa* Isolated from Wound Sample

A total of 144 samples were collected from patient attending Par hospital, Hawler teaching hospital and Rizgary hospital in Erbil city/Iraq from November to march 2016 in Erbil city. Our results showed that among 144 samples 20(13.88%) were culture positive with *pseudomonas aeruginosa*, while 124(86.12%) were cultured negative as in table and figure (1).All bacterial isolates were subjected to a series of confirming tests to ensure that these isolates recovered belong to *P.aeruginosa species* .These bacterial cells from smear preparation are gram negative rods, motile, non-spore forming, arranged in single or short chain. All *p.aeruginosa.* isolated and identified by using microscopical, morphological, biochemical tests and Vitek 2 .The isolates were grown on a variety of media to investigate pigment production. The colors produced by the isolates when they were grown on selective media at 37C° were noted. The isolates produced yellow/green, indicate of the pyocyanin production, and brown/blue, indicate of pyomelanin production .Biochemical test employed were urease production, citrate utilization and. Indole test oxidase, catalase and nitrate were also carried out. Besides these tests, motility and growth of organism in potassium cyanide were also checked. One hundred forty four *P.aeruginosa* isolates isolated from one source of infection from wound. The positive cultures of *P.aeruginosa* were (20) isolates. Kheder, [18].obtained *P.aeruginosa* 20% from wound which higher than to our result. Also our results agreed with Salih, [19].who collected fifty isolates from 200 burn samples in Sulaimaniya hospitalized patient. Othman, [20].reported that the percent of *P.aeruginosa* was 32% from 156 burn patient which higher than our results.

Table1. *Frequency of p.aeruginosa isolated from wound samples.*

Wound infection	Number	%
Infected	20	13.88%
Unaffected	124	86.12%
Total	144	100%

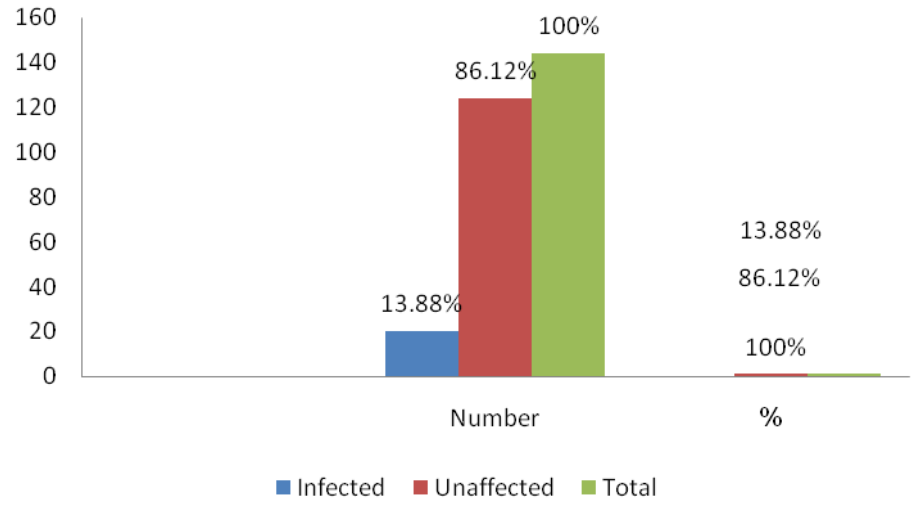

Figure1. *Frequency of P.aeruginosa isolated from wound samples.*

Relation between *P.aeruginosa* and Gender (Female, Male)

The highest percentage of infection by *P.aeruginosa* in female 11(19.64%) were higher than those in male patients 9(10.22%) as in table 2.Our results agreement with results reported by Langeotz *et al.*,[21].in Berlin, Germany who found that the infectious rate *of Psudomonas aeruginos*a in wound of surgical site in female 258(6.9%) more than male 182(5.3%) but relatively lower than our results.

Table2. *Relation between P.aeruginosa and gender (female, male)*

	Patient	wound positive		wound negative		Total
	number	%	Number	%	Number	%
Male	9	10.22%	79	89.77%	88	61.11%
Female	11	19.64%	45	80.35%	56	38.88%
Total	20	13.88	124	86.11%	144	100%

All *p.aeruogenosa* isolates were tested for antimicrobial sensitivity testing and the results were interpreted according to standard value by clinical and laboratory standard institute of antimicrobial sensitivity testing (C.L.S.I) as shown in table (3) the most sensitive antibiotics were Pip/Tazo, Imipenem (80%) followed by Meropenem (75%), Aimikacin (75%) while the most resistance antibiotic were Trimeth/sulpha (20%) as in figure (3).

Table3. *Antimicrobial susceptibility testing for P.aeruginosa*

Antibiotic	Sensitive	Resistance
pip/Tazo	(16)80%	(4)20%
Ciftazidime	(13)65%	(7)35%
Cefepime	(13)65%	(7)35%
Imipenem	(16)80%	(4)20%
Meropenem	(15)75%	(5)25%
Amikacin	(15)75%	(5)25%
Gentamicin	(13)65%	(7)35%
Tobramycin	(13)65%	(7)35%
Ciprofloxacin	(13)65%	(7)35%
Trimeth/sulfa	(4)20%	(16)80%

Regarding antibiotic resistance pattern in *P.aeruginosa* isolates twenty isolates of *P.aeruginosa* were screened for their resistance to ten used antibiotics (Pip/Tazo, ciftazidine,

Cefepime, Imipenem, Meropenem, Amikacin, Gentamicin, Tabromycin, Ciprofloxacin, Trimeth/sulfa).Results indicate that most effective antibiotic were Pip/Tazo 80%, Imipenem 80%, followed by Meropenem 75%, Amikacin 75%. As in table (3) revealed the highest resistant percentage (80%) was to Trimeth/sulfa. It is note worthy to mention that spreading of resistant *P.aeruginosa* in the last few decades is incredibly increased which limits the choice of the therapeutic option for the treatment of infection caused by this microorganism. Henwood *et al.*, [22].reported that the *P.aeruginosa* is inherently resistant to many antimicrobial agent owing to impermeability multidrug efflux pump and a chromosomal AmpC Beta-lactamase and useful activity is seen among Aminopenicillin, firth-fourth generation of Cephalpsporin, monobactam , Carbapenem, Amino Glycosides and Flornquinolones. Olayinka *et al.*, [23].reported that 20% of *P.aeruginosa* isolated from clinical sample obtained from the surgical units of Ahmadu Bello university teaching hospital in Nigeria were sensitive to Imipenem which is in good agreement with our results that among 20 isolates 20% were resistant to Imipenem. Othman [20]. pointed that more than 50 isolates of *P.aeruginosa* among different clinical specimen resist 98% to Amikacin, 96% to Cephotaxime, 80% to rifampicin, 70% to ampicillin, 70% to augment and 60% to doxycycline respectively. Haleem *et al.*, [12] reported that *Pseudomonas aeruginosa* isolated from different clinical cases were fully resistant (100%) for each of the following antibiotics ampicillin , Cefotaxine, Chloramphenicol, Penicillin, Doxycycline and Erythromycin while exhibited moderate resistant's to Amikacin 19 (39.5%) ,Ciprofloxacin 15 (31.26%) and Polymyxin 29(40%) and were sensitive to Pipracillin, Ticarciline in a percentage rate (20.08%)for each antibiotic. The mechanisms of bacterial resistant to Aminoglycoside antibiotics clinical isolates is usually controlled by enzymatic in activation of the antibiotic, since nine different enzymes that catalyze the phosphorylation, Acetylation, Coradenylylation of Aminoglycosides have now been identified in bacteria Clinicians also frequently commence patients on antibiotic therapy before sending samples to the microbiology laboratory such that many of the samples are negative when cultured. Abuse and misuse of antibiotics have been known to contribute to the development of antibiotic resistance. The mechanisms of bacterial resistant to Aminoglycoside antibiotics clinical isolates is usually controlled by enzymatic in activation of the antibiotic, since nine different enzymes that catalyze the phosphorylation, Acetylation, Coradenylylation of Aminoglycosides have now been identified in bacteria [24].Clinicians also frequently commence patients on antibiotic therapy before sending samples to the microbiology laboratory such that many of the samples are negative when cultured. Abuse and misuse of antibiotics have been known to contribute to the development of antibiotic resistance.

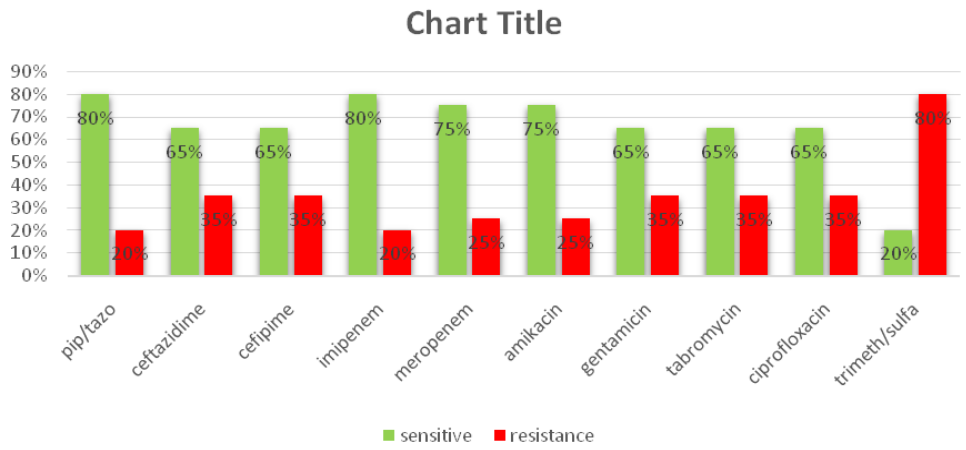

Figure2. *Antimicrobial susceptibility testing for P.aeruginosa*

The Number of Antibiotics (Sensitive and Resistance) for *P.Aeruginosa*

As shown in table (5), 7 isolates of *P.aeruginosa* isolated from wound samples were multi resistance which showed resistance to four or more antibiotic 4, while 2 isolates were resistance to 10 antibiotics .In current study 7 isolates of *P.aeruginosa* isolated from wound samples were multi resistance to four or more antibiotics. The evolution of multi

resistant *P.aeruginosa* and its mechanisms of antibiotic resistance mechanisms include reduced cell permeability, efflux pumps, change in the target enzymes and inactivation of the antibiotics Matsuo *et al*., [25]. The indiscriminate use of antimicrobials over prolonged periods has led to the emergence of multi drug resistant (MDR) strains. Whenever a new and effective antibiotic is introduced, bacteria after exposure to this antimicrobial, acquire resistance through different mechanisms

Table4. *The number of antibiotics (sensitive and resistance) for P.aeruginosa.*

P	PT	COZ	CFPM	IMI	MEM	AMI	G	TOB	CIP	SXT	Number Of Resistant	Number Of Sensitive
P1	S	S	S	S	S	S	S	S	S	R	1	9
P2	S	S	S	S	S	S	S	S	S	R	1	9
P3	S	S	S	S	S	S	S	S	S	R	1	9
P4p	S	S	S	S	S	S	S	S	S	S	-	10
P5	S	S	S	S	S	S	S	S	S	R	1	9
P6	S	S	S	S	S	S	S	S	S	R	1	9
P7	S	S	S	S	S	S	S	S	S	R	1	9
P8	S	R	R	S	S	S	S	S	R	S	3	7
P9	R	R	R	S	R	S	R	R	R	R	8	2
P10	S	S	S	S	S	R	S	S	S	R	2	8
P11	R	R	R	S	S	S	S	S	S	R	4	6
P12	R	R	R	R	R	R	R	R	R	R	10	-
P13	S	S	S	S	S	S	R	R	R	R	4	6
P14	S	S	S	S	S	S	S	S	S	S	-	10
P15	S	S	S	S	S	S	R	R	S	R	3	7
P16	S	R	R	R	R	R	R	R	R	R	9	1
P17	R	R	R	R	R	R	R	R	R	R	10	-
P18	S	S	R	S	S	R	S	S	S	R	3	7
P19	S	S	S	R	R	S	R	R	S	R	5	5
P20	S	R	S	S	S	S	S	S	R	S	2	8

P=Isolated pathogens S=sensitive R=resistance Pip/Tazo: Piperacillin+Tazobactam, Coz: Ciftazidime, Cfpm: Cefepiem, IMI: Imepenem, MEM: Meropenem, AMI:Amikacin, G:Gentamicin, Tob:Tobramycin, Cip: Ciprofloxacin, SXT: Trimeth/Sulpha

REFERENCES

[1] Madigan M.T, Martinko J.M and Brock. (2006). Biology of microorganisms. 11th edition. Pearson Prentice Hall. New Jersey, USA.

[2] Engel, J. (2007). *Pseudomonas aeroginosa* Internalization by Non-Phagocytic Cells. American Journal of Medicine and Microbiology and Immunology; 5: 343-368.

[3] Mahboobi. M. Shahcheraghi, F. and Feizabadi.M.M. (2006). Bactericidal Effects of Essential Oils from Clove, Lavender and Geranium on multi drug resistance isolates from *Pseudomonas aeruginosa. Iranian J.Biotechno.* 4(2):137-140.

[4] Parsons J.F, Greenhagen B.T, Shi K, Calabrese K, Robinson H, and Ladner J.E. (2007) .Structural and functional analysis of the pyocyanin biosynthetic protein PhzM from *Pseudomonas aeruginosa*. Biochemistry. ; 469(7): 1821-1828.

[5] Kipnis E and Sawa T. (2006).Targeting mechanisms of *Pseudomonas aeruginosa* pathogenesis. Médecineet maladies infectieuses. Wiener-Kronish J.; 36: 78-91.

[6] Wilcox MH, Winstanley TG, Spencer RC (1994). Epidemology of drug resistance: Implications for a post-antimicrobial era. Journal science; 257:1050-55.

[7] Bjarnsholt T, Kirketerp-M K, Jensen PØ, Madsen K.G, Phipps R, (2008). Why chronic wounds will not heal: a novel hypothesis. Wound repair and regeneration. 16(1): 2-10.

[8] Kirketerp-M K, Jensen PØ, Fazli.M, Madsen K.G, and Pedersen J. T. (2008).Distribution, organization, and ecology of bacteria in chronic wounds. Journal of clinical microbiology; 46 (8):2717-22.

[9] Sibbald R.G, Orsted H, Schultz G.S, Coutts P, andKeast D. (2003); preparing the wound bed (2003): Focus on infection and inflammation. Ostomy Wound Manage 49; 11.

[10] Thomsen T.R, Aasholm M.S, Rudkjøbing V.B, Saunders A.M, Bjarnsholt T., and Givskov. (2010). the bacteriology of chronic venous leg ulcer examined by culture-independent

molecular methods. Wound repair and regeneration. ; 18(1):38-49.

[11] Schweizer HP (2003). Efflux as a mechanism of resistance to antimicrobials in *Pseudomonas aeruginosa* and related bacteria. Unanswered questions. Genetic molecular research; 2:48:-62.

[12] Haleem H, Tarrad Jk, Banyan IA (2011). Isolation of *Pseudomonas aeruginosa* from clinical cases and environmental samples, and analysis of its antibiotic resistant spectrum at hilla teaching hospital. Medical Journal of Babylon; 8(4):45-52.

[13] Mukerjee S, Chaki S, Barman S, Das S, Koley H, Dasidar G (2012). Effective Elimination of Drug Resistance Genes in *Pseudomonas aeruginosa* by antipsyotic Agents Thioridazine. Current Research in Bacteriology; 5(1):36-41.

[14] Kleyn B (2003). Microbiology Experiments: A health science Perspective. 4th Ed. The McGraw-Hill Companies. New York.

[15] Nakasone I, Kinjo T, Yamane N, Kisanuki K and Shiohira C M. (2007). Laboratory- based evaluation of the colorimetric VITEK 2 Compact System for species identification and of the Advanced Expert System for detection of antimicrobial resistances: VITEK 2 Compact System identification and antimicrobial susceptibility testing. Diagnosis Microbiology Infection Disease Journal; 58: 191-198.

[16] Kasse M, Baars B, Friedrich S, Szabados F and Gatermann S G. (2009). Performance of MicroScan Walkaway and Vitek 2 for Detection of Oxacillin Resistance in a Set of Methicillin- Resistant *Staphylococcus aureus* Isolates with Diverse Genetic Backgrounds. Journal of Clinical Microbiology; 47 (8): 2623-2625.

[17] Wayne P A. (2005). Clinical and laboratory standards institute. Performance standard for antimicrobial susceptibility testing. 15th ed. Informational Supplement. CLSI / NCCLS M 100- S 15. The institute.

[18] Kheder AK (2002). Studies on Antibiotic Resistance by Plasmids of *Pseudomonas aeruginosa*. Ph. D. Thesis, Science Education College Salahaddin University-Erbil, Kurdistan, Iraq.

[19] Salih SSH (2008). Some bacteriological and molecular genetic *studies of Pseudomonas aeruginosa* isolated from different environments. M.Sc. thesis, the higher academy of human and scientific studies, Iraq.

[20] Othman A (2011). Anti plasmid (curing) effect of alcoholic extract of *rosmarinus officinalis* on resistant isolate of *Pseudomonas aeruginosa* M.Sc thesis in sciences in medical Microbiology College of medicine at Hawler Medical University

[21] Langelotza C, Mueller-Raua, Terziyskia S, RauaB, Petra A, Gastmeierc,d Geffersc,d (2014).Gender-Specific Differences in Surgical Site Infections: An Analysis of 438,050 Surgical Procedures from the German National Nosocomial Infections Surveillance System.Viszeralmedizin ;30:114–117.

[22] Hen Wood C, Livermore D, James D, Waner M (2001).Antimicrobial susceptibility of *pseudomonas aeruginosa:* results of a UK survey and evaluation of the British. Journal of Antimicrobial Agents and Chemotherapy; 47:789-799.

[23] Olayinka AT, Olayinka BO, Onile BA (2009). Antibiotic susceptibility and plasmid pattern of *Pseudomonas aeruginosa* from the surgical unit of a university teaching hospital in north central Nigeria. Journal of medicine and medical sciences; 1(3):079-083.

[24] Pollack M, Mandell GL, Bennett JE, Dolin R (2000) *Pseudomonas aeruginosa.* In G.L. Mandell JE, Eennet V, Dolin R. Principles and Practice of Infectious Diseases.

[25] Matsuo Y, Eda S, Gotoh N, Yoshihara E, Nakae T (2004). Mex Zmedited regulation of mexXY multidrug efflux pumps expression in membrane protein profiles of *Xanthomonas maltophilia* isolates displaying temperature. Dependent susceptibility to gentamicine. Journal antimicrobial agents Chemotherapy; 33: 663-666.

Citation: *A. Fattma, A. Zainab, A. Sara and M. Shkofa, "Isolation and Identification of Multi Drug Resistant Pseudomonas Aeruginosa Causing Wound Infection in Erbil City", International Journal of Research Studies in Science, Engineering and Technology, vol. 4, no. 12, pp. 30-36, 2017.*