Global Science Research Journals

ISSN: 2505-001X Vol. 4(2), pp. 147-152, February, 2016.

http://www.globalscienceresearchjournals.org/

Global Journal of Biological and Biomedical Research

Full Length Research Paper

Antibiotic susceptibility of *P. aeruginosa* isolated from burns and wounds of patients in Iraq

Ghaith Al-Daradji

Southern Technical University, Shatrah Technical Institute, Iraq.

Accepted 05 January, 2016

A total of one hundred and forty two swab samples (92 clinical and 50 from hospital environment) were collected for the detection of *Pseudomonas aeruginosa*. Out of the total samples, 29 isolates of *P. aeruginosa* were isolated and recorded an overall prevalence rate of 20.42% (29/242) of which 18 (19.56%) were from wounds and burns swabs of patients, and 11 (22%) were from hospital environment. The highest rate of *P. aeruginosa* (60%) identified from hospital environmental specimens were from door handles followed by ward sinks (57.15%) and the least (10.53%) from patients' beds and table tops. According to gender and age group, the study showed the highest rate of *P. aeruginosa* in the male (55.6%), and in young patients (38.9%) between the ages of 5 and 25 years compared to the elderly; while the lowest rate 27.8% were from those age 45 years and above. Results showed that all isolates from patients and hospital environment were resistant to ticarcillin and ceftazidime (100%). Also, *P. aeruginosa* from patients demonstrated high resistance to cefepime, ofloxacin, gentamycin, tobramycin, ciprofloxacin, lomefloxacin, norfloxacin, levofloxacin and amikacin in the following order respectively :88.8, 77.7, 61.1, 50.0, 44.4, 44.4, 38.8, 38.8 and 33.3%; whereas showed low resistance (16.6 and 11.1%) to each of ticarcillin/clavulanate and meropenem, and only 5.5% to imepenem. Generally, this study pointed that *P. aeruginosa* isolates from hospital environment were more resistant to particular antibiotics than that of clinical isolates. It was also revealed that *P. aeruginosa* have high sensitivity to imepenem, meropenem and ticarcillin /clavulanate and these should be considered in the treatment of this bacterium.

Key words: *Pseudomonas aeruginosa*, antibiotic resistance, clinical samples, surgical wards, hospital environment.

INTRODUCTION

Pseudomonas aeruginosa is an opportunistic nosocomial pathogen, it has higher prevalence and mortality rate in hospital environment, especially among patients, particularly those with burns, wounds and cancer and in

*Corresponding author: E-mail: dara.ghaith@hotmail.com

Table 1. Frequency of *P. aeruginosa* isolates amo–ng patients and hospital environment specimens, 2012.

Specimen type	Specimens no.	*P. aeruginosa* isolates	
		NO.	%
Clinical samples (Burns and wounds)	92	18	19.56
Environment	50	11	22
Total	142	29	20.42

the critically ill admitted in intensive care unit. *P. aeruginosa* is resistant to several antimicrobial agents and therefore become dominant and important when more susceptible bacteria of the normal flora are suppressed (Brooks et al., 2011; Meenakumari et al., 2011). Contaminated medical devices and the hospital environment have often been suggested as potential sources infection with *P. aeruginosa* and the high mortality associated with these infections is due to a combination of the bacterial resistance to antibiotics and a weak host defense system (Meenakumari et al., 2011; Orsi et al., 1995). Wound and burn infections is a major contributor to nosocomial infections. Furthermore, these infections prolong the burden of the disease by increasing cost of treatment, hospital stay and sometimes may lead to death particularly when complicated with septicemia and tetanus (Sule et al., 2002). The current spread of multi-drug resistant bacteria pathogens such as *P. aeruginosa* has added a new side to the problem of wound and burn infections (Sule and Olusanya, 2000). These strains of *P. aeruginosa* establish themselves in the hospital environment in areas like, door handles, patients' beds, sinks, table tops, toilets and others, thereby spreading from one patient to another: and it has a high rate for developing resistance to most of the antimicrobial agents (Haghi et al., 2010; Falagas et al., 2005). Therefore, this study aimed to detect the antibiotic susceptibility of *P. aeruginosa* isolated from burns and wounds of patients and hospital environmental samples from Al Hussein Teaching Hospital in Nasiriyah, Iraq.

MATERIALS AND METHODS

Isolation and identification of bacteria

Clinical swabs from wounds and burns were collected from hospitalized patients in burns and wounds wards and from items in the hospital environment, and subsequently examined from May to August, 2012. One hundred and forty two samples (92 from patients and 50 from items in hospital environment: patients' beds, door handles, sinks and table tops) were collected for detection of *P. aeruginosa.* A performa which include age, gender, health status and relevant data were obtained from each patient, and the consent was taken from all patients before the collection of samples. Sterile cotton swabs (dipped in normal saline 0.9) were used to swab patients' burns and wounds and the surfaces of frequently handled items. Swabs were inoculated onto MacConky and blood agar plates (Oxoid and Himedia) and incubated at 37°C between 18 to 24 h. Colonies grown on culture plates were identified by the morphology of colonies, standard biochemical tests and gram staining (MacFaddin, 2000). Colonies that produce pyocyanin and pyoverdin pigments were confirmed by being transferred to nutrient agar (Oxoid, UK) and subcultured more than once to obtain pure cultures. *P. aeruginosa* isolates were identified using conventional biochemical tests such as oxidase test, catalase test, motility test, haemolysin production and other biochemical tests (Garcia and Isenberg, 2007; Elmer et al., 2006; Atlas and Synder, 2006).

Antibiotic susceptibility testing

The susceptibilities of the isolates to 14 antibiotics (Himedia, India): ticarcillin 75 µg, ceftazidime 30 µg, cefepime 30 µg**,** ofloxacin 5 µg, gentamycin 10 µg, tobramycin 10 µg, ticarcillin/clavulanate 75/10 µg, meropenem 10 µg, imepenem 10 µg, ciprofloxacin 5 µg, lomefloxacin 10 µg, norfloxacin 10 µg, levofloxacin 5 µg and amikacin 30 µg, were determined on Mueller-Hinton agar by the Kirby Bauer disk diffusion method. The zone of inhibition diameter was measured and the results were interpreted based on the guidelines by the Clinical and Laboratory Standards' institute CLSI 2011 and 2012.

Statistical analysis

The Microsoft Excel data analysis tool was used and the relevant data were collectively documented on a questionnaire and values expressed in means and percentage.

RESULTS

A total of 142 swab samples (92 from patients and 50 from hospital environment item) were collected for detection of *P. aeruginosa*. Among the total samples, 29 (20.42%) isolates of *the bacterium* were isolated; 18 (19.56%) from wounds and burns swabs of patients and 11 (22%) from hospital environment (Table 1). The distribution of *P. aeruginosa* isolates among different hospital environment items samples are shown in Table 2. The rate of *P. aeruginosa* was: 3(60%) in door handles, 4(57.15%) in ward sinks and 2 (10.53%) in both the patients' beds and table tops.

Table 3 demonstrated the distribution of *P. aeruginosa* according to age group and gender of patients. The results showed the high prevalence of *the bacterium* isolates among the male than female (55.6 and 44.4%, respectively), and the high rate of isolates were found among the age group 5 to 25 years (38.9%) and the low rate among age group <45 years (27.8%). Antibiotics

Table 2. Distribution of *P. aeruginosa* isolates among hospital environment specimens, 2012.

Specimen type	Specimens no.	*P. aeruginosa* isolates	
		No.	(%)
Patients beds	19	2	10.53
Door handles	5	3	60
Ward Sinks	7	4	57.15
Tables tops	19	2	10.53
Total	50	11	22

Table 3. Age and gender distribution of *P. aeruginosa* isolates for patients burns and wounds, 2012.

Variable	Total n=92	*P. aeruginosa* (n=18)	
		No.	%
Age/year			
5- 25	50	7	38.9
26-45	23	6	33.3
<45	19	5	27.8
Sex			
Female	38	8	44.4
Male	54	10	55.6

Table 4. Antibiotics resistance patterns of *P. aeruginosa* isolates.

Antibiotic	Clinical isolates	(18)	Environmental isolates (11)	
	Resistant No.	Resistant %	Resistant No.	Resistant %
Ticarcillin 75 µg	18	100	11	100
Ceftazidime 30 µg	18	100	11	100
Cefepime 30 µg	16	88.8	10	90.9
Ofloxacin 5 µg	14	77.7	9	81.8
Norfloxacin 10 µg	7	38.8	5	45.4
Ciprofloxacin 5 µg	8	44.4	7	63.6
Levofloxacin 5 µg	7	38.8	5	45.4
Lomefloxacin 10 µg	8	44.4	6	54.5
Amikacin 30 µg	6	33.3	5	45.4
Tobramycin 10 µg	9	50,0	7	63.6
Gentamycin 10 µg	11	61.1	7	63.6
Ticarcillin/ clavulanate75/10 µg	3	16.6	3	27.2
Meropenem 10 µg	2	11.1	2	18.1
Imipenem 10 µg	1	5.5	1	9

susceptibility tests of *P. aeruginosa* against 14 different types of antibiotics is demonstrated in Table 4. All the 29 isolates screened from wounds and burns of patients and from hospital environment were resistant to ticarcillin and ceftazidime (100%). In *P. aeruginosa* from patients, resistance to cefepime, ofloxacin, gentamycin, tobramycin, ciprofloxacin, lomefloxacin, norfloxacin, levofloxacin and amikacin was 88.8, 77.7, 61.1, 50.0, 44.4, 44.4, 38.8, 38.8 and 33.3%, respectively. Whereas, the results showed that there was low resistance (16.6

and 11.1%) to each of ticarcillin/clavulanate and meropenem, and 5.5% of isolates were resistant to imepenem. Results showed that isolates of the bacterium from hospital environment were more resistant to antibiotics than those from the clinical environment. The level of resistance to cefepime, ofloxacin, gentamycin, tobramycin, ciprofloxacin, lomefloxacin, norfloxacin, levofloxacin, amikacin, ticarcillin/clavulanate, meropenem and imipenem were 90.9, 81.8, 63.6, 63.6, 63.6, 54.5, 45.4, 45.4, 45.4, 27.2, 18.1 and 9%, respectively.

DISCUSSION

P. aeruginosa is ranked second among gram-negative bacteria isolated from hospital environment, and leading cause of nosocomial infections responsible for high rate of morbidity and mortality (Meenakumari et al., 2011; Okon et al., 2009). The *bacterium* can cause serious infections in immunocompromised patients such as those with surgery wounds or severe burns (Sule et al., 2002; Dale et al., 2004). Result of this study showed that the prevalence of *P. aeruginosa* was 20.42% (29/142) among all the samples for patients and hospital environment, which is less than that reported in other studies; (39.1%) to that obtained by Okon et al. (2009) from wound swabs in Nigeria and 25.5% was reported in Cameron (Ndip et al., 2005). But these results were higher than 17.85% recorded in the teaching hospital of Al-Sulaimania city, Iraq (Ekrem et al., 2014) and 18.6% reported in Egypt (Gad et al., 2007). This disparity in prevalence rate among several studies can be attributed to differences in hygienic practices and geographical location.

The detection rate of *P. aeruginosa* in clinical samples 19.56% was not largely different from that of 22% from hospital environmental specimens and this result was similar to other studies in different area of the world (Ndip et al., 2005; Ekrem et al., 2014; Gad et al., 2007; Savaş et al., 2005). The results of this study agree with that of several other studies. (Meenakumari et al., 2011; Orsi et al., 1995; Sule et al., 2002) explain that *P. aeruginosa* is a common cause of infections in wound and burns contacted from the hospital environment or from patients own normal flora, and these infections leading to the longer hospital stays and increasing the treatment costs and mortality rate. The highest rate of *P. aeruginosa* which was identified from hospital environmental specimens in this study were from door handles (60%) followed with ward sinks (57.15%), This result could be explained by the fact that bacteria grow very well at sites with adequate amount of moisture and where people commonly come in contact with, while the result showed the least rate of *P. aeruginosa* (10.53%) in the patients' beds and tables tops, which are mostly kept dry. According to gender and age group, the result of this study shows the highest rate of *P. aeruginosa* in the male (55.6%), and 38.9% in the young patients (ages 5 to 25 years) compared to the elderly, while the lowest rate (27.8) was found among age group of forty five years and above, which indicates that males in this age group are more active and involve in different clinical hygiene practices, even in hospital environment. This result is comparable with the study of Okon et al in Nigeria, which recorded that male patients showed a record of 52.8% and the highest frequency of this bacterium (20.7%) was found in age group of 29 years and below (Okon et al., 2009). On the other hand, these results disagree with studies of Shewatatek et al. (2014) in Ethiopia and Ekrem and Rokan in Al-Sulaimania city, Iraq, where results of the studies showed higher occurrence of the bacterium in female and elderly patients (Shewatatek et al., 2014; Ekrem et al., 2014).

Results in Table 4 show the antibiotic susceptibility testing profile of *P. aeruginosa* that, 100% of isolates of clinical samples and hospital environment were resistance to ticarcillin and ceftazidime. The resistance of *P. aeruginosa* against the Beta-lactam antibiotics was higher than that of non Beta-lactam, this result can be attributed to the hyper production of Beta lactamase through the resistance genes and mutational processes (CDC, 2010; Lister et al., 2009; Okon et al., 2009). Also, *P. aeruginosa* isolates from patients demonstrated high resistance to cefepime, ofloxacin, gentamycin, tobramycin, ciprofloxacin, lomefloxacin, norfloxacin, levofloxacin and amikacin at 88.8, 77.7, 61.1, 50.0, 44.4, 44.4, 38.8, 38.8 and 33.3%, respectively, whereas the results showed low resistance (16.6 and 11.1%) to each of ticarcillin/clavulanate and meropenem, and only (5.5%) were resistant to imepenem. Generally, *P. aeruginosa* isolates from hospital environment were more resistant to particular antibiotics than that of clinical isolates. This result was similar with other studies done in Iraq (Ekrem et al., 2014), Egypt (Gad et al., 2007), Cameron (Ndip et al., 2005) and Nigeria (Okon et al., 2009), but differs with others such as study of Shewatatek et al. (2014) who recorded low resistance to these antibiotics. Difference in the resistance rate among several studies may be attributed to factors like hygienic culture of population, type of clinical specimen examined and exposure to antibiotics.

The resistance rate of Cefepime and ofloxacin in clinical and hospital environment isolates used in this study was recorded between 77 to 90.9%. This rate agree with the result of the study done in India (Prakash et al., 2012), but higher than that of Egypt (Gad et al., 2007) and Belgium (Van, 2003). Results in Table 4 also show that resistance rate to gentamycin was 61.1 and 63.6%, this finding is lower than those of studies reported in Egypt (67.5%) (Gad et al., 2007), Cameroon (66.7%) (Ndip et al., 2005) and Jordan (72%) (Masaadeh and Jaran, 2009). Whereas, the results are higher than the results of the study in Iraq (40%) (Ekrem and Rokan, 2014). The high resistance of *P. aeruginosa* isolates to gentamycin can be attributed to the wide use of this antibiotic in hospital

and the modification in bacteria enzymes. *P. aeruginosas* resistance to meropenem and imepenem was lower than other Beta-lactams used in the present study, in both patients and environment isolates. This result agrees with study of Ekrem and Rokan in 2014 that recorded 0% resistance rate to meropenem and imipenem in Iraq. The high sensitivity of these antibiotics can be attributed to low exposure because of the limited usage in Iraqi hospitals (Alzaidi and Alsulami, 2014). Meropenem and imipenem may be active therapy for *P. aeruginosa* infection.

Increasing resistance to different antibiotics especially among nosocomial pathogens has been reported world-wide and become important therapeutic challenge in the treatment of disease (Jones et al., 2002; Orrett, 2004). This fact agrees with the study of WHO which pointed that the widespread use of antibiotics both outside and inside of medicine is playing a significant role in the emergence of resistant bacteria by developing several resistance mechanisms such as production of Beta-lactamase enzymes that destroy these antibiotics (WHO, 2002). The major problem of the resistant bacteria emergence is due to overuse and misuse of antibiotics by patients as well as doctors (Goossens et al., 2005; Iduh et al., 2015), and may be related to random use of antibiotics without antibiotic sensitivity test and laboratory diagnosis.

Conclusion

The high rate and multidrug resistance of *P. aeruginosa* which were isolated from clinical and hospital environment probably occur as a result of wide use and abuse of antibiotics. Therefore the result of this study may be as a recommendation to the correct use of antibiotics in treatment of patients and also has to be considered as a part of infection control measures in hospital environment in order to reduce the risk of resistance development of *P. aeruginosa* infection.

Conflict of Interests

The authors have not declared any conflict of interests.

REFERENCES

Alzaidi JR, Alsulami AA (2014). Detection of methicillin resistant Staphylococcus aureus (MRSA) in the surgical wards by Chromogenic agar medium and PCR assay. Int. J. Biomed. Life Sci. 5(3):381-397.

Atlas RM, Snyder JW (2006). Handbook of media for clinical microbiology. 2nd ed. CRC Publisher. New York. Pp.278-339.

Brooks GF, Karen CC, Janet SB, Stephen AM (2011). Medical Microbiology. Jawetz, Melnick, & Adelberg. 25th edition, McGraw-Hill LANG. Chapter 16.

CDC-Centers for Disease Control and Prevention (2010). Antibiotic resistance questions and answers. Retrieved from http://www.cdcgov/getsmart/antibiotic-use/anitbiotic-resistance-faqs.html.

CLSI-Clinical and Laboratory Standards Institute (2011). Performance standards for antimicrobial susceptibility testing. Twenty-first Information supplement, CLSI document M100-S21. 31(1); M02-A10, M07-A8, Wayne PA.USA.

CLSI-Clinical and Laboratory Standards Institute (2012). Performance standards for antimicrobial susceptibility testing. Twenty-second Information supplement, CLSI document M100-S22. 32 (3); M02-A11, M07-A9, with M23-A3, M39-A3,M45-A2, Wayne PA.USA.

Dale RMK, Schnell G, Wong JP (2004). Therapeutic efficacy of antibiotics against burn wound infection by *Pseudomonas aeruginosa* Antimicrob. Agents Chemother. 48(8):2918-2923.

Ekrem K, Rokan DK (2014). Antibiotic susceptibility patterns of *Pseudomonas aeruginosa* strains isolated from various clinical specimens. Sky J. Microbiol. Res. 2(2):13-17.

Elmer K, Washington W, Stephen A Jr., William J, Gary P, Paul S, Gail W (2006). Konemans color atlas and textbook of diagnostic microbiology. Sixth ed. Lippincott Williams and Wilkins, London.

Falagas ME, Bliziotis IA, Kasiakou SK, Samonis G Athanassopoulou P, Michalopoulos A (2005). Outcome of infections due to pandrug-resistant (PDR) Gram-negative bacteria. BMC Infect. Dis. 5:24.

Gad GF, Domany RA, Zaki S, Ashour HM (2007). Characterization of *Pseudomonas aeruginosa* isolated from clinical and environmental samples in Minia, Egypt: prevalence, antibiogram and resistance mechanisms. J. Antimicrob. Chemother. 60:1010-1017.

Garcia LS, Isenberg HD (2007). Aerobic bacteriology. Clinical microbiology procedures handbook. 2nd ed. update, ASM Press. Washington C, D. 3:1.

Goossens H, Ferech M, Vander Stichele R, Elseviers M (2005). Outpatient antibiotic use in Europe and association with resistance: a cross-national database study. Lancet 365(9459):79-87.

Haghi M, Maadi H, Delshad R, Nezhady M, Golizade S (2010). Antibiotic resistance pattern of *Escherichia coli Staphylococcus aureus* and *Pseudomonas aeruginosa* isolated from burnt patients Urmia, Iran. Int. J. Acad. Res. 2:377-380.

Iduh UM, Chollom CS, Nuhu A, Spencer TH, Nura MB, Ashcroft OF (2015). Nosocomial infections in post-operative wounds due to *Staphylococcus aureus* and Pseudomonas aeruginosa in Benue State Nigeria. Afr. J. Microbiol. Res. 9(36):1989-1996.

Jones RN, Kirby JT, Beach ML, Biedenbach DJ, Pfaller MA (2002). Geographic variations in activity of broad-spectrum lactams against *Pseudomonas aeruginosa*: Summary of the worldwide SENTRY antimicrobial surveillance program 1997–2000. Diagn. Microbiol. Infect. Dis. 43:239-243.

Lister PD, Wolter DJ, Hanson ND (2009). Antibacterial-Resistant *Pseudomonas aeruginosa:* Clinical impact and complex regulation of chromosomally encoded resistance mechanisms. Clin. Microbiol. 22(4):582-610.

MacFaddin JF (2000). Biochemical tests for identification of medical bacteria. Baltimore, MD: Lippincott Williams and Wilkins.

Masaadeh HA, Jaran AS (2009). Incident of *Pseudomonas aeruginosa* in post-operative wound infection. Am. J. Infect. Dis. 5:1-6.

Meenakumari S, Verma Sh, Absar A, Chaudhary A (2011). Antimicrobial susceptibility pattern of clinical isolates of *Pseudomonas aeruginosa* in an Indian cardiac hospital. Int. J. Eng. Sci. Technol. 3:7117-7124.

Ndip RN, Dilonga HM, Ndip LM, Akoachere JF, Akenji TN (2005). *Pseudomonas aeruginosa* isolates recovered from clinical and environmental samples in Buea, Cameroon: current status on biotyping and antibiogram. Trop. Med. Int. Health 10:74-81.

Okon K, Agukwe P, Oladosu W, Balogun S, Uba A (2009). Antibiotic resistance pattern of *Pseudomonas aeruginosa* isolated from clinical specimens in a tertiary hospital in northeastern Nigeria. J. Microbiol. 8(2):5-7.

Orrett FA (2004). Antimicrobial susceptibility survey of *Pseudomonas aeruginosa* strains isolated from clinical sources. J. Natl. Med. Assoc. 96(8):65-69.

Orsi GB, Tomao P, Visca P (1995). In vitro activity of commercially manufactured disinfectants against *Pseudomonas aeruginosa*. Eur. J. Epidemiol. 11:453-457.

Prakash HR, Rashmi B, Neena K, Suresh S, Anitha MR5, Vijayanath V

(2012). Antimicrobial susceptibility pattern of *Pseudomonas aeruginosa* strains isolated from clinical sources. J. Pharm. Biomed. Sci. 14:5.

Savaş L, Duran N, Savaş N, Onlen Y, Ocak S (2005). The prevalence and resistance patterns of *Pseudomonas aeruginosa* in intensive care units in a University hospital. Turk. J. Med. Sci. 35:317-322.

Shewatatek G, Gizachew T, Molalegne B, Terefe G (2014). Drug sensitivity of *Pseudomonas aeruginosa* from wound infections in Jimma university specialized hospital, Ethiopia. J. Med. Med. Sci. Res. 3(2):13-18.

Sule A, Olusanya O (2000). In-vitro antimicrobial activities of fluoroquinolones compared with common antimicrobial agents against clinical bacterial isolates from parts of South Western Nigeria. Niger. Q. J. Hosp. Med. 10(1):18-21.

Sule A, Thanni L, Sule-Odu O, Olusanya O (2002). Bacterial pathogens associated with infected wounds in Ogun state university teaching hospital, Sagamu, Nigeria. Afr. J. Clin. Exp. Microbiol. 3(1):13-16.

Van EJ (2003). Multicentre surveillance of Pseudomonas aeruginosa susceptibility pattern in nosocomial infection. J. Antimicrob. Chemother. 51:347-352.

WHO (2002). Humans. World Health Organization.