Antibiotics Susceptibility pattern of *Pseudomonas aeruginosa* that isolated from ear, wound and urine samples

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Abstract:

A total of 181 isolates of *Pseudomonas aeruginosa* were obtained from 781 clinical samples from Al-Sader Medical city. The isolates were collected from ear swab(85 isolates), wound swab (70 isolates) and urine sample (26 isolates). *P. aeruginosa* was isolated from ear swab in high percentage 85 (46.96%) as compared with wound swab 70 (38.67%) and urine sample 26 (14.99%). The isolates of *P. aeruginosa* were subjected to antibiotic sensitivity test. Among various antibiotics tested, the strains showed highest resistance to ampicillin (100%), followed by Cephalothin (92.82%), Cefotaxem (84.53), Gentamycin(69.61) and Trimethoprim (67.99), Carbencillin (67.95), Ceftriaxon (24.86), Ciprofloxacin (20.99), Cefamandol (19.33), Tobramycin(14.91), Amikacin(14.3), and Piperacillin (12.7). Among most effective antibiotics against *P.aerugionsa*, with low resistant rate are Tobramycin (14.91), Amikacin (14.3), and Piperacillin (12.7) respectively. Out of 50 *P.aerugionsa* isolates (retested for ESBL production)13 (26%) were found to be ESBL producers and non-ESBL were 37 (74%). Extended spectrum beta-lactamases enzymes (ESBL) was performed by double disc diffusion method .

Keywords: Resistant; *Pseudomonas aeruginosa*; ESBL

Introduction:

P. aeruginosa has emerged as a major cause of infection in the last few decades. It is an increasingly prevalent opportunistic pathogen and is the fourth most frequently isolated nosocomial pathogen accounting for 9.9% of all hospitals acquired infections (Khan *at al*, 1998; Gordon *et al*, 998). In burn units, *P.aeruginosa* outbreak leads to high mortality of up to 60 % (Richard *et al*, 1994).. The pathogenicity of these organisms is based on its ability to produce a variety of toxins and proteases. It also depends upon its ability to resist phagocytosis (Baltimore, 2000).Normally human faecal carriage of *P. aeruginosa* is low, around 3% (Botzenhart and Doring, 1993). However, carriage increases with the length of stay in hospital, reaching 30 - 50% after 3

weeks and thus can present a distinct risk of endogenous infection (Neu, 1983). *P. aeruginosa* can infect almost any external site or organ, and therefore, can be isolated from various body fluids such as sputum, urine, wounds, eye or ear swabs and from blood (Hugbo and Olurinola,1992). *P. aeruginosa* is inherently resistant to many antibiotics and can mutate to even more resistant strains during therapy. Although numerous resistance mechanisms have been identified, the mutation of porin proteins constitutes the major mechanism of resistance. Penetration of antibiotics in to the Pseudomonad cell is primarily through pores in the outer membrane. If the proteins forming the walls of these pores are altered to restrict flow throw the channels, resistance to many classes of antibiotics can develop. *P. aeruginosa* also produces a number of different beta lactamases that can inactivate many beta lactam antibiotics (eg. penicillins,cepalosporins, and carbapenems). The accelerated emergence of antibiotic resistance among the prevalent pathogens is the most serious threat to the management of infectious diseases. β-lactamases antibiotics are the most common treatment for bacterial infections (Blanc, *et al.1998*). *P. aeruginosa* represents a phenomenon of antibiotic resistance, demonstrating practically all known enzymic and mutational mechanisms of bacterial resistance. These mechanisms are often present simultaneously, conferring combined resistance to many strains (McGowan, 2006).Unfortunately, extended-spectrum β-lactamases (ESBLs) have been widely spread throughout serious infections of Gram-negative bacteria in the 1980's (Bradford, 2001). The first report of plasmid-encoded β- lactamases capable of hydrolyzing the extended spectrum cephalosporins was published in 1983 (Khan, *et al.,1998*). Hence, ESBLs are able to hydrolyze a broader spectrum of β-lactamases antibiotics than the simple parent β-lactamases from which they are derived. ESBLs have an ability to inactivate antibiotics containing an oxyimino-group such as oxyimino-cephalosporins (*e.g.*, ceftazidime, ceftriaxone, cefotaxime) as well as oxyimino-monobactam (aztreonam) (Bradford, 2001); however, they are not active against cephamycins and carbapenems. Generally, they are inhibited by β-lactamase-inhibitors such as clavulanate and tazobactam (Yan, *et al*, 2006). Various types of ESBLs have been identified in *P. aeruginosa* including class A ESBLs, such as TEM,SHV, PER and VEB ESBLs, and class D, such as OXA-type ESBLs.. Among them, OXA–type ESBLs have been encountered most commonly in *P. aeruginosa* while class A ESBLs were found uncommon (Livermore,2002,De Champs,2002). Furthermore, most OXA-type ESBLs are OXA-2 and OXA-10 derivatives (Livermore,2002).The OXA-type ESBLs were originally discovered in *P. aeruginosa* isolates from a single hospital in Ankara, Turkey (Hall *et al.*, 1993). Several OXA-type ESBLs have been derived from the original OXA-10 β-lactamase (*e.g.*, OXA -11, 14, 6 and 17). OXA-type β-

lactamases are characterized by their high hydrolytic activity against oxacillin and cloxacillin and are poorly inhibited by clavulanic acid. Extension of the hydrolytic spectrum of oxacillinase to oxyimino cephalosporins has been reported in OXA-2 and OXA-10 extended-spectrum derivatives. In contrast to the majority of OXA-type ESBLs, which confer resistance to ceftazidime, the OXA-17 β-lactamase confers resistance against cefotaxime and ceftriaxone but provides only marginal protection against ceftazidime (Danel, *et al.*, 1999).

This research was designated to detect the percentage of infection of *P. aeruginosa* in ear, wound and urine samples and study the sensitivity of *P. aeruginosa* to various types of antibiotics as well as the determination the ability of *P. aeruginosa* to produce ESBL enzymes.

Materials &Methods:

Sample Collection Techniques

Seven hundred and eighty one samples of ear, wound swabs and urine samples submitted to the Microbiological Laboratory of Al-Sader medical city were used for this study .Urine samples were collected in sterile universal bottles; patients suffering from urinary tract infection were instructed on how to collect a clean proof universal container. Specimens of ear discharge were collected using sterile cotton swab sticks by the assistance of medical officer. Wounds swabs were collected using sterile swabs sticks.

Isolation Procedures:

Urine: Samples were mixed thoroughly by inverting the container several times, using a sterile wire loop, the sample was inoculated on blood agar and MacConkey agar plates. The plates were incubated over night at 37ºC.

Ear discharges, and wound swabs:

The swabs were streaked on sterile blood agar, and MacConkey agar plates by first made primary inoculums and then streaked out using sterile wire loop to give a discrete colonies. The plates were incubated at 37ºC for 24 hours. Suspected colonies were stored on slant for further tests.

Identification of the Isolates

The isolates were identified on the basis of macroscopic, microscopic, physiological, and biochemical tests.(Macffadin,2000).

Antibiotic Sensitivity Test

Susceptibility of the isolates to antimicrobial agents was determined by the agar diffusion techniques using Kirby-Bauer disc method (Prescolt et al., 2002) was used for antibiotic susceptibility testing for each bacterial isolate on Muller Hinton agar. Medium was prepared and sterilized by autoclaving at 121 ºC for 15 min. 25 ml of media was poured in 90 mm sterile Petri dishes and incubated at 37ºC overnight to check sterility. Using ampicillin (30 µg), amikacin (30 µg),

Carbencillin (100 µg), cefamandol (30 µg), ceftriaxon (30 µg), Trimethoprim (25 µg), cefotaxime (30 µg), ciprofloxacin (10 µg), gentamycin (10 µg), piperacillin (10 µg),Tobramycin(10 µg) and Cephalothin (30 µg). The plates were incubated at 37ºC for 18 h and after incubation, plates were examined and zones of inhibition and reported the organism sensitive, intermediate, resistant according to national committee for control laboratory standards (NCCLS, 2007). After 18 h of incubation, plates were examined and zones of inhibition were measured. Results were interpreted on the basis of zone sizes, as sensitive, intermediate, resistant according to National Committee for Control Laboratory standards (NCCLS, 2007).

Detection of Extended Spectrum Betalactamases (ESBLs)

Double disc diffusion method was used to detect the extended spectrum beta lactamases (ESBL). A single, separated colony of the test organism was picked and emulsified in 0.9% normal saline in a test tube, the turbidity of the test organism was matched with 0.5% McFarland's Standard. The suspension of test organism was spread on the Mueller - Hinton agar surface in a petri plate with the help of cotton swab soaked in suspension tube. A disk of co - amoxicillin (20 µg amoxicillin/10 µg clavulanic acid) was placed in the center of the agar surface. The discs of cephotaxime, ceftriaxone, ceftazidime and aztreonam (30 µg) were arranged in such a way that the distance between the central disc and surrounding discs was approximately 30 mm. The plates were incubated at 37ºC for 24 h. After an overnight incubation, the zones observed. If the inhibition zone around one or more cephalosporins discs was extended on the side nearest to the co-amoxiclave disc,the organism showing this synergism is an ESBL - producer. When there was no extension of zones, the test was repeated by reducing the distance between the cephalosporins and aztreonam, amoxiclave discs to 20 mm or even less. Zones of inhibition were again observed next day. If no extension of 3rd generation of cephalosporins and aztreonam towards co-amoxiclav discs was observed, the organisms were considered as non-producer of ESBLs. (Kader et. al.,2006).

Results:

A total of 181 isolates of Pseudomonas aeruginosa were obtained from 781clinical samples collected from ear, wound and urine samples., 85 isolates of Pseudomonas aeruginosa were from ear samples, 70 isolates from wound samples and 26 isolates from urine samples. (Table 1). The distribution of Pseudomonas aeruginosa according to the site of infection showed that the percentage of infection of Pseudomonas aeruginosa in ear swab 85(46.96%) was higher than in wound samples 70(38.67%) and urine samples 26(14.99%) respectively. (Table 2).

Table (1):Types of samples and number of isolated bacteria.

Types of samples	Total No. of isolated Bacteria No=781	No. of isolated Pseudomonas aerugenosa
Ear swab	160	85
Wound swab	226	70
Urine samples	395	26
Total	781	181

Table (2):Distribution of Pseudomonas aeruginosa according to sites of infection.

Types of samples	No. of isolated Pseudomonas aeruginosa	Percentage of infection %
Ear swab	85	46.96
Wound swab	70	38.67
Urine samples	26	14.36
Total	181	99.99

In the present study, the susceptibility of 181clinical isolates of P. aeruginosa showed highest resistance (100%) was found against ampicillin . The next most resistant antibiotics were Cephalothin (92.81%), Cefotaxem (84.53), Gentamycin (69.61) and Trimethoprim (67.99), Carbencillin (67.95), Ceftriaxon (24.86), Ciprofloxacin (20.99), Cefamandol (19.33), Tobramycin (14.91), Amikacin (14.3), and Piperacillin (12.7). Among most effective antibiotics against P.aerugionsa, with low resistant rate are Tobramycin(14.91), Amikacin (14.3) and Piperacillin (12.7) respectively. (Table 3).

The present study was conducted to determine the ESBLs among P. aeruginosa isolates. In our study 50 isolates of P.aerugionsa retested to The detection the ability of this bacteria to production the ESBL(Figure-1), where13 (26%) were found to be ESBL producers and 37 (74%) were non-ESBL producers.(Figure-2). ESBL was performed by double disc diffusion.

Table (3):Antibiotic susceptibility pattern of Pseudomonas aeruginosa that isolated from urine, ear and wound swab samples

Types of antibiotics	Resistant	sensitive	intermittent
Piperacillin(PRL10µg)	23 (12.7)	150 (82.87)	8 (4.19)
Amikacin(AK30 µg)	26(14.3)	137 (75.69)	18 (9.94)
Tobramycin(TOB10µg)	27 (14.91)	131 (72.37)	23 (12.7)
Cefamandol (CEF30µg)	35 (19.33)	124 (68.5)	22 (12.15)
Ciprofloxacin (CF10µg)	38 (20.99)	138(76.2)	5 (2.76)
Ceftriaxon (CRO 30 µg)	45(24.86)	136(75.13)	-
Carbencillin(PY100µg)	123 (67.95)	52(29.28)	6 (3.31)
Trimethoprim(SXT25µg)	123 (67.99)	49 (27.07)	9(4.97)
Gentamycin (CN10µg)	126 (69.61)	46 (25.41)	9 (4.97)
Cefotaxem (CTX30 µg)	153 (84.53)	28 (15.45)	-
Cephalothin (KF 30 µg)	168 (92.81)	13 (7.18)	-
Ampcillin (Am 30 µg)	181(100)	-	-

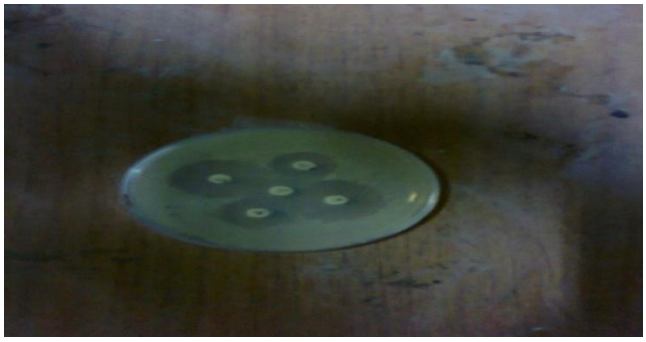

Figure-1: ESBL producing P. aeruginosa. Enhancement of zone of inhibition produced by susceptible strain of P. aeruginosa to 3rd generation cephalosporins and aztreonam to wards amoxyclav disc placed at the centre.

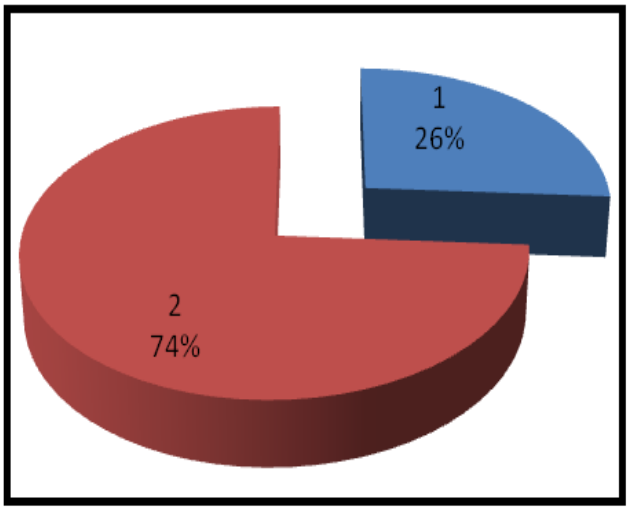

Figure -2: ESBL producers among P. aeruginosa 1. ESBL producer. 2. Non ESBL producer.

Discussion:

The knowledge of the bacteriology of an infection and the laboratory susceptibility testing of micro-organism implicated could make drug selection in antimicrobial chemotherapy more rational. Supportive infection of the skin and ear are common occurrences in hospitalized and out patients. (Dionigi et al., 2001).

In this study a significant increase was found in the number of P. aeruginosa strains isolated from ear followed by wound and urine, similar kind of results was reported by(Adedeji et.al.,2007)who reported that percentage of infection with P. aeruginosa in ear site was high(50%) compared with the percentage of

infection of P. aeruginosa in wound(16 %) and urine samples (8.7 %) but in a study conducted by (Anjum and Mir. ,2010), P. aeruginosa was mostly isolated from pus sample (41%)and urine sample (32 %). Olayinka, (2004) also observed that P.aeruginosa was mostly isolated from urine samples. This could be attributed due to differences in geographical location and hygienic measures or due to the fact that most patients going for major surgery tend to get catheterized.

The results obtained also agreed with the findings of Selina (2002) they rated P. aeruginosa as the most common bacteria isolated from mild to severe form of external otitis and chronic supportive otitis media. The increase of infection with P. aeruginosa in ear site as compared with skin infection or urinary tract infection may be explain on the bases the anatomical structure of ear, where the construction of the ear canal from bone growth can trap debris leading to infection (Wang et. al.,2005).Saturation divers have reported otitis external during occupational exposure (Ahlen et. al.,1998).Even without exposure to water, the use of objects such as cotton swab or other small objects to clear the ear canal is enough to cause breaks in the skin, and allow the condition to develop.(Zichichi et. al.,2000) Once the skin of the ear canal is inflamed, external otitis can be drastically enhanced by either scratching the ear canal with an object, or by allowing water to remain in the ear canal for any prolonged length of time.

An important striking feature found in this study was increased resistance to most of the antibiotics tested by disk diffusion method.Similer finding were reported by (Poirel et al., 2002; Amutha et al., 2009) in which P. aeruginosa isolates were resistant to various types of antibiotics .On the other hand, the most isolates of P.aeruginosa in this study were sensitive to Piperacillin(82.87),amikacin(75.69 %),and tobramycin (72.37%)but Anjum and Mir in(2010) showed that most isolates of P.aeruginosa were sensitive to amikacin(79%),tobramycin(70%) and Piperacillin(65%).There are multiple factors, which contribute to the global spread of resistance of P.aeruginosa to the most types of antibiotics, decreasing unnecessary antibiotic use, treating with narrow spectrum agents, improving compliance with therapy, decreasing use of antibiotic in animal and agriculture, and improving infection control all have a role in confronting this problem. In addition, immunization may diminish the impact of resistance by preventing infection and also the carriage of transmission. However, P. aeruginosa exhibits intrinsic resistance to several antimicrobial agents. It poses some multi-drug efflux systems, including MexAB-OprM and MexXY-OprM.(Li et. al.,1995) Furthermore, acquired antimicrobial resistance constitutes a major challenge for anti-pseudomonas therapy, especially when it is associated with resistance to other classes of

drugs.(Pagani et.al.,2004) Antimicrobial resistance among clinical isolates of P. aeruginosa may complicate the treatment of infections. New antimicrobial agents with activity against P. aeruginosa will not be available in the near future, making ongoing surveillance of the activities of currently available agents of critical importance.(Flamm et.al.,2004) Although numerous resistance mechanisms have been identified, the mutation of porin proteins constitutes the major mechanism of resistance. Penetration of antibiotics in to the Pseudomonad cell is proteins forming the walls of these pores are altered to restrict flow throw the channels, resistance to many classes of antibiotics can develop. P. aeruginosa also produces a number of different beta lactamases that can inactivate many beta lactam antibiotics (eg. penicillins, cepalosporins, and carbapenems).The extended spectrum of beta lactamases (ESBL) producing organisms are a breed of drug resistant pathogens that are rapidly becoming important globally in the area of hospital acquired infections. ESBLs have been described in P. aeruginosa only recently (Nordmann & Naas,1994;Jarlier et al.,1998). ESBL which are very important as these strains may often cause morbidity and mortality in patients underlying diseases or limit therapeutic options due to the high degree of multidrug resistance. In the present study 13(26 %) isolates of P. aeruginosa showed ESBL production .Similar findings were reported by Anjum and Mir. In (2010) but Amutha et al., in (2009) reported 40.9 % of P. aeruginosa isolates to be ESBL producers. In conclusion ,it was observed that high number of P. aeruginosa isolated from ear samples and low number isolated from urine samples, the isolates of P. aeruginosa were highly resistance to different types of antibiotics, but effective antibiotics in this study are Piperacillin, Amikacin and peperacilline.26% of isolated strains of P. aeruginosa were ESBLs positive.

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الخلاصة:

تم جمع 181عزله لبكتريا *P.aeruginosa* من مجموع العينات ألسريريه البالغ عددها 781 من مدينه الصدر الطبية، وقد جمعت هذه العينات مِن مسحات الإذن (85عزله)، مسحات الجروح (70عزله) ومن عينات الإدرار(26عزله). لقد سجلت أعلى نسبه عزل لبكتريا *P.aeruginosa* في العينات التي تم أخذها من مسحات الاذن85(46.96%) مقارنه بنسبه عزلها من مسحات الجروح 70 (38.67%) و عينات الادرار26(14.99%) .كما تم اختبار حساسية بكتريا *P.aeruginosa* لأنواع مختلف من مضادات الحياة وقد سجل المضاد Ampicillin أعلى نسبه مقاومه(100%) يليه المضاداتCephalothin (92.82%) ، Cefotaxem (84.53%) ، Gentamycin، (69.61%) Trimethoprim، (67.99%) ، Carbencillin (67.95%) Ceftriaxon، (24.86%) Ciprofloxacin، (20.99 %) ، Cefamandol (19.33%) Tobramycin، (14.91 %) Amikacin، (14.3%) ، Piperacillin (12.7%) وعدت المضادات Tobramycin، Amikacin، Piperacillin من بين أفضل المضادات المؤثرة ضد *P.aeruginosa* إذ سجلت اقل نسبه مقاومه (14.91%)، (14.3%) ، (12.7%) على التوالي. تم أعاده فحص 50 عزله لبكتريا *P.aeruginosa* لتحري عن قابليتها لإنتاج إنزيمات Extended spectrum beta-lactamases enzymes(ESBL) إذ كانت 13(26%) من العزلات منتجه لإنزيم (ESBL) و37(74%) غير منتجه لإنزيم (ESBL) وقد اجري هذا الفحص بطريقه Double disk diffusion method.