Available online www.jocpr.com

Journal of Chemical and Pharmaceutical Research, 2015, 7(8):186-190

Research Article

ISSN : 0975-7384
CODEN(USA) : JCPRC5

Antimicrobial susceptibility pattern of *Pseudomonas aeruginosa* from clinical isolates at a tertiary care centre in Vijaypur, Karnataka

Preeti B. Mindolli[1] and Manjunath P. Salmani[2]

[1]*Department of Microbiology, Al Ameen Medical College, H&RC, Athani Road, Vijaypur, Karnataka, India*
[2]*Department of Microbiology, Shri B.M. Patil Medical College, H&RC, Solapur Road, Vijaypur, Karnataka, India*

ABSTRACT

Pseudomonas aeruginosa is emerging as a nososcomial pathogen. It has intrinsic as well as acquired resistance to many antimicrobial drugs. This study investigated the antimicrobial resistance pattern of P. aeruginosa from clinical isolates. It is a retrospective study carried out from June 2014 – December 2014 in Department of Microbiology at Al-Ameen Medical College, H&RC, Vijaypur. Out of 396 culture positive samples, 78 were identified as P. aeruginosa (by standard bacteriological identification procedures). These isolates were recovered from various specimens like pus, urine, sputum, broncho-alveolar lavage (BAL) and tracheal aspirate. Antimicrobial sensitivity testing was done by Kirby-Bauer disc diffusion method. Out of 396 cultures positive, 78 were positive for P. aeruginosa. The isolation rate was 19.69%. P. aeruginosa were more sensitive to combination drugs like piperacillin+tazobactum (93.5%) and cefoperazone+sulbactum (92.3%) followed by imipenem (88.2%), meropenem (87.1%). Sensitivity to amikacin, tobramycin, gentamicin and ceftazidime ranges from 35%-55%. Highest resistance rate was seen for amoxicillin followed by doxycycline. From our study, we concluded that P. aeruginosa is one of the most common nosocomial pathogen. It is sensitive to combination drugs like piperacillin+tazobactum and cefoperazone+sulbactum. It is also sensitive to carbapenems like imipenem, meropenem and aminoglycosides like amikacin, tobramycin, gentamicin, and cephalosporins like ceftazidime. Rational use of these drugs is necessary to prevent further spread of antimicrobial resistance among P. aeruginosa strains and also emergence of multi drug resistance.

Key words: Antimicrobial sensitivity testing; Drug resistance; Pseudomonas aeruginosa

---

INTRODUCTION

Pseudomonas aeruginosa (P. aeruginosa) is a classic opportunistic pathogen with innate resistance to many antibiotics and disinfectants. [1] It is found in moist environment and disinfectant solution due to its ability to utilize different organic compounds and survive in nutrient deficient conditions. [2] P. aeruginosa is a notable cause of nosocomial infection of the respiratory tract, urinary tract, wound, blood stream and central nervous system. For immunocompromised patients, such infections are often severe and life threatening. [3]

Mechanisms that cause antimicrobial drug resistance in P. aeruginosa is due to acquisition of resistance genes (e.g.: those encoding betalactamase), [4] and aminoglycoside modifying enzymes [5] via horizontal gene transfer and mutation of chromosomal genes (target site efflux mutations). Last one is the mechanism of resistance in fluoroquinolones particularly ciprofloxacin. [6] Biofilm production especially in case of pulmonary infections in patients with cystic fibrosis contributes to its resistance to antimicrobial agents. [7]

Multiple antibiotic resistances in bacterial population is a growing clinical problem which is a threat to public health. Hence there is a need to conduct studies to profile different pathogens responsible for specific infections and their resistance patterns so as to generate data that would help clinicians to choose correct antibiotics for treatment. [8]

EXPERIMENTAL SECTION

It is a retrospective study carried out from June-December 2014 in Department of Microbiology at Al Ameen Medical College, H&RC, a tertiary health care centre, vijay pur, Karnataka.

Specimen: A total of 1087 non-duplicate samples from hospitalized patients admitted in different wards of hospital were investigated for bacterial culture and identification. Specimens were taken from various sources like sputum, urine, pus, catheter, broncho alveolar lavage and tracheal aspirate.

Laboratory identification of isolates: Identification of bacterial isolates was done by standard microbiological procedure. Specimens were inoculated on Nutrient agar, Blood agar, MacConkey agar and were studied for colony morphology. A battery of tests were performed that included Gram stain, oxidase test, motility test, production of pyocyanin, growth at 42^0C, oxidative metabolism of glucose and arginine hydrolase.

Antibiotic susceptibility tests: Antibiotic susceptibility test was done by Kirby-Bauer disk diffusion method as per CLSI guidelines. Paper discs were impregnated with antibiotics of standard strength as below:

Penicillins: Amoxycillin (20µg), Piperacillin (100µg), Ticarcillin(>75µg)
Fluoroquinolones: Ciprofloxacin (5µg), Levofloxacin (5µg)
Tetracyclines: Doxycycline (30µg)
Macrolides: Azithromycin (15µg)
Cephalosporins: Cefoperazone (75µg), Ceftazidime (30µg), Cefepime (30µg), Ceftriaxone (30µg)
Carbapenems: Imipenem (10µg), Meropenem (10µg)
Aminoglycosides: Gentamicin (10µg), Tobramycin (10µg), Amikacin (30µg)
Combination drugs: Piperacillin+Tazobactum (100/10µg), Cefoperazine+Sulbactum (75/30 µg)

They were incubated overnight at 37^0C. The diameter of the zone of inhibition was measured and results were interpreted as sensitive, intermediate and resistant strain.

RESULTS

A total of 1087 nonduplicate samples from hospitalized patients were processed, of which 396 were culture positive. Out of 396 culture positive samples, 78 were identified as P. aeruginosa bystandard microbiological procedures. The rate of isolation of P. aeruginosa is 19.69%. Out of 78 culture identified P. aeruginosa, 36 (46.1%) were from male and 42 (58.4%) from females (Table 1).

Table 1: Age and gender wise distribution of P. aeruginosa isolates

Age (In Years)	Number of Males	Number of Females	Total (In %)
<10 Years	1	2	3 (3.8%)
11-20	6	4	10 (12.8%)
21-30	5	2	7 (8.9%)
31-40	8	3	11 (14.1%)
41-50	7	13	20 (25.6%)
51-60	4	7	11 (14.1%)
>60	5	11	16 (20.5%)

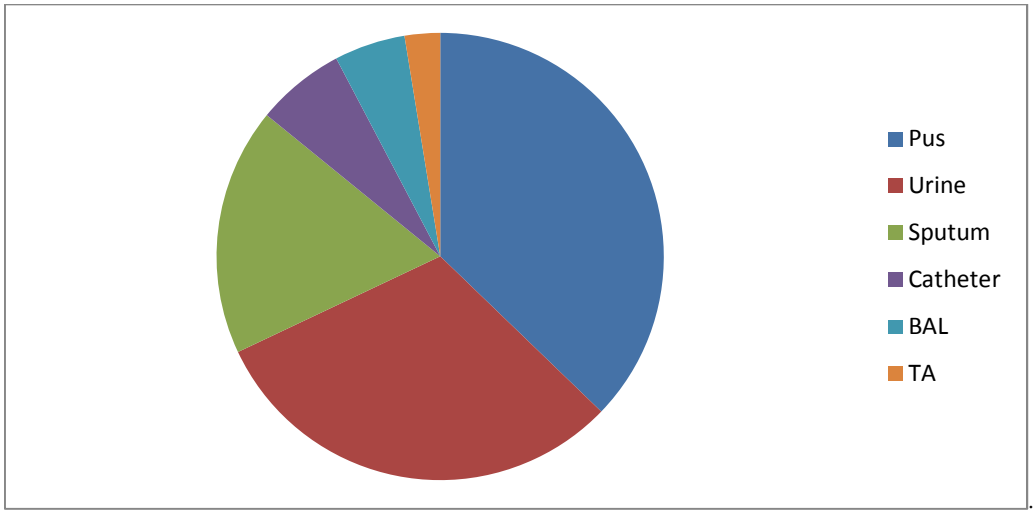
The rate of isolation was more in the age group of 41-50 years. Major source of these isolates were from wound/pus [29 (37.17%)] followed by urine, sputum, catheter, broncho-alveolar lavage and tracheal aspirate (Table 2).

Table 2: Frequency of distribution of P. aeruginosa from specific sites

Source of specimen	Number of Samples	Percentage (%)
Pus/Wound	29	37.17%
Urine	24	30.76%
Sputum	14	17.9%
Catheter	5	6.41%
BAL	4	5.12%
Tracheal aspirate	2	2.56%

***BAL**=Broncho Alveolar Lavage*

Figure 1: Distribution of P. aeruginosa from specific site



***BAL**-Broncho Alveolar Lavage; **TA**-Tracheal Aspirate*

Table 3: Antibiotic sensitivity pattern of P. aeruginosa against different class of antibiotics

Antibiotics	Sensitive	Intermediate	Resistant
Penicillin			
Amoxycillin (20µg)	1 (1.28%)	1 (1.28%)	76 (97.4%)
Piperacillin(100µg)	22 (28.2%)	3 (3.8%)	53 (67.9%)
Ticarcillin(75µg)	31 (39.74%)	2 (2.56%)	45 (57.6%)
Fluoroquinolones			
Ciprofloxacin(5µg)	30 (38.4%)	1 (12.0%)	47 (60.25%)
Levofloxacin(5µg)	35 (44.87%)	2 (2.56%)	41 (52.56%)
Tetracyclines			
Doxycycline(30µg)	7 (8.97%)	2 (2.56%)	69 (88.46%)
Macrolides			
Azithromycin(15µg)	33 (42.3%)	3 (3.8%)	42 (53.8%)
Cephalosporin			
Cefoperazone(75µg)	43 (55.1%)	3 (3.8%)	32 (41.02%)
Ceftazidime(30µg)	42 (53.84%)	2 (2.56%)	34 (43.58%)
Cefepime(30µg)	39 (50.0%)	2 (2.56%)	37 (47.4%)
Ceftriaxone(30µg)	38 (48.7%)	4 (5.2%)	36 (46.15%)
Carbapenems			
Imipenem(10µg)	69 (88.2%)	3 (3.8%)	6 (7.9%)
Meropenem(10µg)	68 (87.1%)	2 (2.56%)	8 (10.25%)
Aminoglycosides			
Gentamicin(10µg)	38 (48.1%)	2 (2.56%)	38 (48.1%)
Tobramycin(10µg)	41 (52.56%)	3 (3.84%)	34 (43.58%)
Amikacin(30µg)	43 (55.12%)	2 (2.56%)	33 (42.3%)
Combination drugs			
Piperacillin+Tazobactum(100/10µg)	73 (93.5%)	2 (2.56%)	3 (3.8%)
Cefoperazone+Sulbactum(20µg)	72 (92.3%)	2 (2.56%)	4 (5.12%)

DISCUSSION

In our study, 78 isolates were isolated and identified as P. aeruginosa by standard biological procedures. The rate of isolation is 19.69%. The rate of isolation was more in the age group of 41-50 years (25.6%) followed by elderly age group. This may be due to decreased immunity, prolonged hospitalization and associated co-morbidities. [6] A study done by Rajat et al in 2012 shows 29% [9] isolation rate in the age group of 31-45 years which is similar to our study and study done by Chander Anil et al [6] shows 20% in age group of 21-40 years. Out of 78, 36 (46.1%) were from male and 42 (53.84%) from female which is same as Chander Anil et al. [6]

In our study, 37.17% of isolates were obtained from pus/wound followed by 30.76% from urine specimen. In a study done by Anuradha et al in 2014 [2] shows 39.39% from pus samples and 37.87% from urine samples. Another study by Javiya et al in 2008 [8] reported highest number of P. aeruginosa from urine followed by pus and sputum. In a study Anuprabha et al in 2006 [10] showed 32% isolation rate from pus. Similar results are also reported by Mohana Soundaram and Arora et al. [11, 12] This indicates that wound infections and urinary tract infection are most common hospital acquired infections. These are most common cause for morbidity in hospitalized patients. P. aeruginosa is a common cause of wound infection especially in burns patients because burns have large exposed area of dead tissue free of any defence, so ideal for P. aeruginosa infection. [13]

The resistance profile of P. aeruginosa to the antimicrobial agents tested varied among the isolates investigated. [6] Highest sensitivity was seen to combination drugs like piperacillin+tazobactum (93.5%) and cefoperazone+sulbactum (92.3%). Sensitivity to carbapenems like imipenem (88.2%) and meropenem (87.1%) was comparatively high. Similar studies like Al-Jasser et al in 2004 [14] showed sensitivity to meropenem (91.6%), imipenem (90.2%) and piperacillin+tazobactum (81.3%). Raja NS et al [15] showed sensitivity to imipenem (90.1%) and piperacillin+tazobactum (90.6%). Ansary SP et al [16] showed sensitivity to cefoperazone+sulbactum (82%) which is similar to our study.

Sensitivity to aminoglycosides (gentamicin, tobramicin, amikacin) and cephalosporins (cefoperazone, ceftazidime, cefipime, ceftriaxone) ranges from 45-55% which is same as study conducted by Garba et al in 2006. [13]

Highest resistance was seen to amoxicillin (97.4%) followed by doxycycline (88.46%) which is similar to study conducted by Garba I et al in 2006. [13] Resistance to quinolones (ciprofloxacin & levofloxacin) ranges from 50-60% and azithromycin was 53.8%.

The selective pressure from use of antimicrobial agents is a major determinant for emergence of resistant strains. The subinhibitory antibiotic concentration in wounds, due to administration of inappropriate dosage of beta-lactum antibiotic or regular administration of aminoglycosides in combination with beta lactum, provides optimal conditions for selection and persistence of multidrug resistant strains. [10]

CONCLUSION

P. aeruginosa is a leading cause of nosocomial infection. Indiscriminate use of antibiotics has led to emergenece of multidrug resistant srains. In our study, strains are more sensitive to combination drugs like piperacillin+tazobactum and cefoperazone+sulbactum and carbapenems like imipenem and meropenem. A more restricted and rational use of these drugs is necessary. A regular monitoring of antimicrobial susceptibility pattern is essential to guide the physicians in prescribing right combination of drugs and emergence of multidrug resistance strains of P. aeruginosa.

REFERENCES

[1] JG Collee, AG Fraser, BP Marmion, A. Simmons. Pseudomonas, Stenotrophomonas, Burkholderia. Mackie and Mccartney Practical Medical Microbiology, 14th edition, Churchill Livingstone publication, New York, **2007**: 413-425.
[2] Anuradha B, Afreen U, Praveen M. *Global J of Med Res*, **2014**; 14 (7): 23-28.
[3] BA Forbes, DF Sahm, SA Weissfeld. Bailey and Scott's diagnostic Microbiology. 12th edition, Mosby Inc, St. Louis, **2007**: 334-338.
[4] WH Zhao, ZQ Hu. *Crit Rev Microbiol*, **2010**; 36 (3): 245-258.
[5] Poole K. *Antimicrob Agents Chemother*, **2005**; 49: 479-87.

[6] A Chander, MS Raza. *Asian J Pharm Clin Res*, **2013**; 6 (3): 235-38.
[7] JC Davies, D Bilton. *Respir care* **2009**; 54 (5): 628-640.
[8] VA Javiya, SB Ghatak, KR Patel, JA Patel. *Indian J Pharmacol*, **2008**; 40 (5): 230-234.
[9] RM Rajat, GL Ninama, K Mistry, R Parmar, K Patel, MM Vegad. *National J Med Res*, **2012**; 2 (2): 156-159.
[10] Anupurba S, A Bhattacharjee, A Garg, MR Sen. *Indian J Dermatol*, **2006**; 51 (4): 286-288.
[11] KM Mohana Soundaram. *J Clin Diagn Res*, **2011**; 5 (3): 491-494.
[12] D Arora, N Jindal, R Kumar, T Romi. *Int J Pharm Pharm Sci*, **2011**; 3(2): 82-84.
[13] I Garba, YH Lusa, E Bawa, MB Tijjani, MS Aliyu, UU Zango et al. *Nigerian J Basic and App Sci*, **2012**; 20 (1): 32-34.
[14] AM Al-Jasser, NA Elkhizzi. *Saudi Med J* **2004**; 25 (6): 780-784.
[15] NS Raja, NN Singh. *J Microbiol Immunol Infect*, **2007**; 40 (1): 45-49.
[16] SP Ansary, R Haque, AA Faisal. *Tropical Doctor*, **1994**; 24: 188.