VOLUME - 10, ISSUE - 01, JANUARY - 2021 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Microbiology

ISOLATION OF CEFTAZIDIME RESISTANT *PSEUDOMONAS AERUGINOSA* ISOLATES FROM PUS SAMPLES

Priyanka Sharma Demonstrator, Department of Microbiology, Government Medical College & Hospital, Jammu.

Tomar R Demonstrator, LLRM Medical College, Meerut (U.P.).

Perika Sharma* Demonstrator , Department of Microbiology, Government Medical College & Hospital, Jammu. *Corresponding Author

ABSTRACT This study aimed to detect Ceftazidime resistant Pseudomonas aeruginosa isolates from pus samples

KEYWORDS :

INTRODUCTION

Pseudomonas aeruginosa is a non fermenter, oxidase positive, pigment producing gram negative bacilli which is a major nosocomial pathogen. It produces manifestations such as Ventilator associated pneumonia (VAP), Chronic respiratory tract infections (especially in cystic fibrosis patients), Bacteremia, Infective endocarditis, Ear infections such as swimmer's ear (among children) and malignant otitis externa (in elderly diabetic patients), Corneal ulcers (in contact lens wearers), Shanghai fever, Skin and soft tissue infections (in burn patients), Ecthyma gangrenosum, Dermatitis, Toe-web infection, Green nail syndrome, Cellulitis. It is also implicated in the causation of Osteomyelitis, Septic arthritis, Meningitis and Urinary tract infection. Pathogenesis of Pseudomonas is greatly attributed to its ability to develop widespread resistance to multiple antibiotics and disinfectants. This species is inherently resistant to most of the antibiotics and only limited antimicrobial agents have antipseudomonal action such as Penicillins (Piperacillin, Mezlocillin, Ticarcillin), Cephalosporins (Ceftazidime, Cefoperazone, Ceftolozane and Cefepime), Beta-lactam/Beta-lactamase inhibitor combination (Piperacillin-Tazobactam, Cefoperazone-sulbactam), Carbapenems (Imipenem, Doripenem, Meropenem), Monobactams (Aztreonam), Aminoglycosides (Tobramycin, Gentamicin, Amikacin), Quinolones (Ciprofloxacin, Levofloxacin), Polymyxins (Polymyxin B, Colisitn)[1].

As a third generation cephalosporin, Ceftazidime (CAZ) has broad-spectrum activity and inhibits cell wall synthesis by binding to penicillin-binding proteins (PBPs) of Gram-negative bacilli[2].A surge in ceftazidime resistance in human clinical isolates of P. aeruginosa results from the production of acquired β-lactamase, the constitutive overproduction of AmpC, or an activationof the MexAB-OprM or MexXY-OprM efflux systems[3,4,5,6,7,8,9,10].Studies have shown that P. aeruginosa is exceptionally problematic in terms of antimicrobial resistance because of its rapid ability to develop resistance and the multiple mechanisms by which it can become resistant to a variety of antimicrobials[11].

Antimicrobial susceptibility pattern of microorganisms varies with time, place and depends on the emergence of new resistant strain. Ceftazidime is an important and effective antimicrobial agent for the therapy of serious infections due to multidrug resistance in P. aeruginosa. It is important to consider resistance to this antimicrobial when selecting the regimen. Thus, consistent data on the same is mandatory for clinicians to decide appropriate treatment strategy. This will eventually help in time management, accurate administration of drug; reduce possibility of drug resistance and therapy failure. The widespread use of broad-spectrum antibiotics in the hospital is probably responsible for the emergence of resistant strain.

Thus, this study was designed to isolate Ceftazidime resistant Pseudomonas aeruginosa strains from pus samples.

MATERIALS AND METHODS :

The present study was done in the Bacteriology section of the Dept. of Microbiology, GMC, Jammu spanning a period of 6 months .

Various pus samples received in the Bacteriology Lab from various wards were processed as per standard protocols. Swabs were processed for direct examination by Gram's Stain, then inoculation was done by Streak method on Blood Agar and Mac Conkey Agar plates.

Samples like Endotracheal tip culture were inoculated by roll plate technique. Inoculated plates were subjected to aerobic incubation at 37 ° C for 24 hours.

Next day identification was done by colony morphology, Gram's staining and conventional biochemical tests as per standardized protocols of our laboratory .

Antibiotic sensitivity was performed by using Kirby- Bauer disc diffusion method as per Clinical Laboratory Standards Institute (CLSI) guidelines. Antibiotic discs were applied on Mueller- Hinton Agar. Antibiotic discs tested were Ampicillin, Ceftazidime, Imipenem, Piperacillin- Tazobactam, Gentamycin, Amikacin, Levofloxacin.

RESULTS :

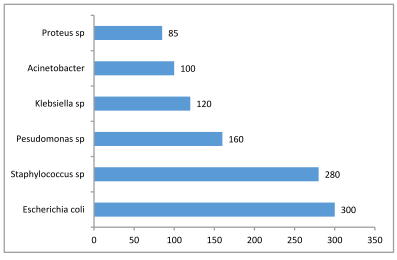

Figure 1 : Graph demonstrating various organisms isolated from different samples

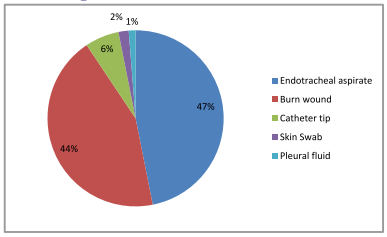

Figure 2 : Depicting distribution of Pseudomonas according to the type of specimen

Table 1: Demonstrating the distribution of Pseudomonas sp

Total Pseudomonas isolates	160
Pseudomonas aeruginosa	140
Other Pseudomonas sp	20

Table 2: Antibiotic Susceptibility profile of Psedomonas aeruginosa

ANTIBIOTIC	SENSITIVE (%, n=140)	RESISTANT (%, n= 140)	INTERMEDIATE
Ceftazidime	2 (1.42)	130 (92.8)	8
Aztreonam	80 (57.1)	60 (42.8)	-
Imipenem/ Meropenem	110 (78.5)	30 (21.4)	-
Piperacillin-Tazobactam	40 (28.5)	90 (64.2)	1
Gentamycin	67 (47.8)	73 (52.1)	-
Netilmicin	90 (64.2)	50 (35.7)	-
Amikacin	80 (57.14)	60 (42.8)	-
Levofloxacin	78(55.7)	62 (44.2)	-
Ampicillin	20 (14.2)	120 (85.7)	-

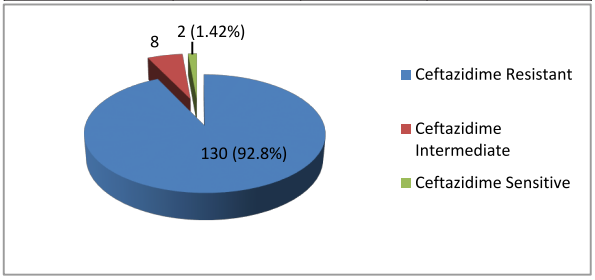

Figure 3 : Depicting the percentage of Ceftazidime resistant isolates

DISCUSSION :

Pseudomonas aeruginosa is an important cause of nosocomial infections associated with high mortality rates [12]. This high pathogenicity is attributed to its intrinsic resistance to a wide array of antibiotics and the ability to develop multidrug resistance in the hospital environment [13]. Ceftazidime belongs to the third generation Cephalosporin group and is considered as one of the major antimicrobials in the treatment of Pseudomonas aeruginosa infections [14,15]. But resistance to this antibiotic is emerging very fast and is responsible for occurrence of resistant infections in the hospital environment. Ceftazidime resistant isolates are known to arise through the horizontal acquisition of _β-lactamases or altered expression of the chromosomal drug-inducible wide-spectrum class C _β-lactamase AmpC [16]. Hence , our study was planned to recognise the percentage of such resistant isolates from the hospital environment.

Our study demonstrated a high percentage of Ceftazidime resistance (92.8 %) in Peudomonas aeruginosa which is consistent with study by Mahmoud et al (91%) [17,18] while a study by Gupta et al 2016 [19] showed 68.5 % of Ceftazidime resistance. This high resistance is attributed to unchecked use of antibiotics in the hospital. High level resistance was also seen with Ampicillin (85 %), Piperacillin- Tazobactam (64.2 %). Low resistance was seen with Imipenem (21.4 %). Netilmicin (35.7 %), Amikacin (42.8 %). This was comparable with study by Hasuuna et al 2015 [20].

Our study showed that most important risk factors significantly associated with Pseudomonas infections were endotracheal incubation (47 %) followed by burn wounds (44 %). This was comparable with study by Gupta et al 2016[19].

Therefore, management of infections due to Pseudomonas sp represents a major therapeutic challenge due to increasing resistance to a wide range of antibiotics and presence of significant risk factors.

CONCLUSION:

Our study was planned to highlight rapidly emerging problem of Antimicrobial resistance in Pseudomonas aeruginosa which is an important nosocomial pathogen.

High degree of ceftazidime resistance seen from our study calls for the use of newer drugs for the treatment of such multi-drug resistant Pseudomonas infections .

This study could guide Hospital Infection Control Committee in framing proper antibiotic policies for the hospital and recognising the resistant hospital strains to curtail the spread of infection and take appropriate management strategies.

REFERENCES

1. Apurba S Sastry, Sandhya Bhat. Essentials of Medical Microbiology, Second Edition, p352-355.
2. Clarke AM, Zemcov SJ. Ro 13-9904 and GR 20263, two new cephalosporins with broad-spectrum activity: an in vitro comparison with other beta-lactam antibiotics. *J Antimicrob Chemother* 1981; 7:515–6.
3. Lindberg F, Lindquist S, Normark S (1987): Inactivation of the ampD gene causes semiconstitutive overproduction of the inducible Citrobacterfreundii -lactamase. *Journal of Bacteriology*, 169, 1923–1928.
4. Li XZ, Ma D, Livermore DM, Nikaido H (1994): Role of efflux pump(s) in intrinsic resistance of Pseudomonas aeruginosa: active efflux as a contributing factor to betalactam resistance. *Antimicrobial Agents and Chemotherapy*, 38, 1742–1752.
5. Stapleton P, Shannon K, Phillips I (1995): DNA sequence differences of ampD mutants of Citrobacterfreundii. *Antimicrobial Agents and Chemotherapy*, 39, 2494–2498.
6. Nordmann P, Guibert M (1998): Extended-spectrum -lactamase in Pseudomonas aeruginosa. *The Journalof Antimicrobial Chemotherapy*, 42, 128–131.
7. Aires JR, Kohler T, Nikaido H, Plesiat P (1999): Involvement of an active efflux system in the natural resistance of Pseudomonas aeruginosa to aminoglycosides. *Antimicrob Agents Chemother*, 43, 2624–2628.
8. Kuga A, Okamoto R, Inoue M (2000): ampR gene mutations that greatly increase class C -lactamase activityin Enterobacter cloacae. *Antimicrobial Agents and Chemotherapy*, 44, 561–567.
9. Masuda N, Sakagawa E, Ohya S, Gotoh N, Tsujimoto H, Nishino T (2000): Substrate specificities of MexABOprM, MexCD-OprJ, and MexXY-OprM efflux pumps in Pseudomonas aeruginosa. *Antimicrob Agents Chemother*, 44, 3322–3327.
10. Livermore DM (2002): Multiple mechanisms of antimicrobial resistance in Pseudomonas aeruginosa: our worst nightmare? *Clinical Infectious Diseases*, 34, 634–640.
11. Lister PD, Wolter DJ, Hanson ND. Antibacterial-resistant Pseudomonas aeruginosa: clinical impact and complex regulation of chromosomally encoded resistance mechanisms. *ClinMicrobiol Rev* 2009;22:582–610.
12. Lambert ML, Suetens C, Savey A, Palomar M, Hiesmayr M, et al. (2011) Clinical outcomes of health-care-associated infections and antimicrobial resistance in patients admitted to European intensive care units: a cohort study. *Lancet Infect Dis* 11: 30-38.
13. Kallen AJ, Hidron AI, Patel J, Srinivasan A (2010) Multidrug resistance among gram-negative pathogens that caused healthcare-associated infections reported to the National Healthcare Safety Network, 2006-2008. *Infect Control Hosp Epidemiol* 31: 528-531.
14. Liao X, Hancock RE. 1997. Susceptibility to beta-lactam antibiotics of *Pseudomonas aeruginosa* overproducing penicillin-binding protein 3. Antimicrob Agents Chemother **41:**1158–1161.
15. Hayes MV, Orr DC. 1983. Mode of action of ceftazidime: affinity for the penicillin-binding proteins of *Escherichia coli* K12, *Pseudomonas aeruginosa* and *Staphylococcus aureus*. *J Antimicrob Chemother* **12:**119–126. http://dx.doi.org/10.1093/jac/12.2.119.
16. Fisher JF, Mobashery S. 2014. The sentinel role of peptidoglycan recycling in the beta-lactam resistance of the Gram-negative *Enterobacteriaceae* and *Pseudomonas aeruginosa*. *Bioorg Chem* **56:**41–48. http://dx.doi .org/10.1016/j.bioorg.2014.05.011.
17. Mahmoud BA, Zahran WA, Hindawi GR, Labib AZ and Galal R (2013) Prevalence of Multidrug-Resistant Pseudomonas aeruginosa in Patients with Nosocomial Infections at a University Hospital in Egypt, with Special Reference to Typing Methods. *Journal of Virology & Microbiology* 2013: 1-13.
18. Zafer MM, Al-Agamy MH, El-Mahallawy HA, Amin MA, Ashour MS (2014) Antimicrobial resistance pattern and their beta-lactamase encoding genes among Pseudomonas aeruginosa strains isolated from cancer patients. *Biomed Res Int*
19. Gupta R, Malik A, Rizvi M, Ahmed SM. Incidence of multidrug- resistant pseudomonas spp. In ICU patients with special reference to ESBL, AMPC, MBL and biofilm production. *J Global Infect Dis* 2016; 8:25-31
20. Hassuna N, Ibrahim A , Eleuoon SM, Rizk HA. High Prevalence of Multidrug Resistant Pseudomonas aeruginosa Recovered from Infected Burn Wounds in Children. *Arch Clin Microbiol* 2015; 6(4).