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PREVALENCE AND ANTIBIOGRAM OF HOSPITAL ACQUIRED AND COMMUNITY ACQUIRED PSEUDOMONAS AERUGINOSA: A HOSPITAL BASED CROSS SECTIONAL STUDY

Microbiology

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ABSTRACT

BACKGROUND: Knowing epidemiology of hospital acquired and community acquired *P.aeruginosa* is important for deciding empirical treatment.

AIM: To know prevalence and antibiogram of hospital acquired and community acquired *P.aeruginosa*.

METHODS: A study done in Microbiology department of Shri Shankaracharya Medical College, Bhilai; from 01/1/2017 to 31/12/2017. Processing of sample was done by standard protocol and antibiotic sensitivity test was done according to CSLI guidelines.

RESULTS: Isolation rate of *P.aeruginosa* in our study was 3.35%. Hospital acquired strains were more resistant than community acquired. Carbapenems were the most sensitive drugs after polymixin B and Colistin.

CONCLUSION: Regular periodic surveillance to formulate rational treatment of *P.aeruginosa* is necessary because of their changing pattern of antimicrobial resistance.

KEYWORDS

P.aeruginosa, Antibiogram, Hospital Acquired P.aeruginosa,community Acquired P.aeruginosa, Carbapenem

INTRODUCTION

Pseudomonas aeruginosa, is a well known nosocomial ESKAPE pathogen.[1,2] It is now increasingly implicated in community acquired infections.[3] Approximately 4% of normal adults can be carrier of *P.aeruginosa* in their pharynx, colon or skin.[4] 28.3% carriage rate on hands of health care worker had been reported in India.[5] *P.aeruginosa* causes broad spectrum of infections including respiratory tract, gastrointestinal, wound, sepsis and urinary tract.[6] Multidrug resistance is common in *P.aeruginosa* infections.[7] There is rising trends of carbapenem resistance in *P.aeruginosa* which call for rapid detection and judicious use of antibiotics.[8] *P.aeruginosa* infections outcome can be improved by timely administration of adequate antibiotic based on susceptibility pattern.[9-10] This study was conducted to find out antibiogram of community and hospital acquired *P.aeruginosa* which inturn will help physician to decide empirical treatment in these situations and may serve as a base for further research and findings to ensure rational treatment of highly resistant pathogens.

MATERIALS AND METHODS:

The study was carried out in the Microbiology department, Shri Shankaracharya Institute of Medical Sciences, Bhilai. It was a cross sectional study done from 01 Jan 2017 to 31 Dec 2017.

SAMPLE PROCESSING: Samples were cultured on blood and MacConkey agar. Colonies were identified by Gram staining, colony characteristics, motility and biochemical reactions.[11] 146 *P.aeruginosa* were isolated from 4360 clinical samples in microbiology laboratory during study period were included in study. Duplicate isolates were excluded from the study. Antibiotic sensitivity test was performed by Kirby-Bauer disk diffusion method(CLSI,2012).[12]

All 146 patients isolating *P. aeruginosa* were reevaluated at time of report dispensing to categorize them in hospital and community acquired *P.aeruginosa.*

P.aeruginosa isolated after 48 h of hospitalization or from a patient with a history of hospitalization or of a residence in a long-term care facility within 1 year of the culture date were considered hospital acquired and rest as community acquired *P.aeruginosa* .[13]

Multidrug resistant *P.aeruginosa* : Isolates were considered MDR pseudomonas if they are non susceptible to atleast one agent in three different antimicrobial classes.[14]

RESULTS:

During the study period, 146 strains of *P.aeruginosa* were isolated from 4360 various samples received in microbiology laboratory in our hospital. Thus, the isolation rate of *P.aeruginosa* was found to be 3.35%. Among them 129 were hospital acquired and 17 were community acquired. Gender and age analysis revealed that Male and female ratio was 101:45and mean age was 50±2.5.

Maximum isolation was from pus(60.27%) followed by urine(19.86%), ear discharge/swab(12.33%), blood(4.11) and sputum/BAL/pleural fluid(3.42).

Table showing resistance pattern of *P.aeruginosa in various samples:*

ANTIBIOTICS	PUS		URINE		EAR DISCHARGE		BLOOD		SPUTUM		TOTAL	
	HA	CA	HA	CA	HA	CA	HA	CA	HA	CA	HA	CA
	N=86 (%)	N=02 (%)	N=26 (%)	N=03 (%)	N=08 (%)	N=10 (%)	N=06 (%)	N=00 (%)	N=03 (%)	N=02 (%)	N=129 (%)	N=17 (%)
Piperacillin	43(50)	01 (50)	13(50)	03(100)	06(75)	06(60)	04(66.67)	-	03(100)	01(50)	69(53.49)	10(58.82)
Piperacillin+Tazobactam	35(40.70)	00	03(11.53)	01(33.33)	01(12.5)	01(10)	02(33.33)	-	01(33.33)	00	42(32.56)	02(11.77)
Ceftazidime	61 (70.93)	01 (50)	15(57.70)	02(66.67)	06(75)	05(50)	05(83.33)	-	02(66.67)	01(50)	89(68.99)	9(52.94)
Cefepime	69(80.23)	2(100)	18(69.23)	02(66.67)	05(62.5)	05(50)	05(83.33)	-	03(100)	01(50)	100(77.52)	10(58.82)

Ciprofloxacin	58(67.44)	2(100)	11(42.30)	03(100)	06(75)	05(50)	04(66.67)	-	03(100)	01(50)	82(63.56)	11(64.70
Levofloxacin	65(75.58)	1(50)	05(19.23)	01(33.33)	04(50)	04(40)	03(50)	-	01(33.33)	00	78(60.46)	6(35.29)
Amikacin	37(43.02)	1(50)	07(26.92)	01(33.33)	03(37.5)	01(10)	03(50)	-	01(33.33)	00	51(39.53)	03(17.65
Gentamycin	52(60.46)	02(100)	08 (30.77)	02 (66.67)	04(50)	04(40)	04(66.67)	-	02(66.67)	01(50)	70(54.26)	09(52.94)
Imipenem	03(3.49)	00	00	00	00	00	00	-	00	00	3(2.32)	00
Meropenem	01(1.16)	00	00	00	00	00	00	-	00	00	01(.77)	00
Nystatin	00	00	00	00	00	00	00	-	00	00	00	00
Polymyxin B	00	00	00	00	00	00	00	-	00	00	00	00

*HA- hospital acquired; CA- community acquired

Multidrug resistance was seen in 36(24.65%) *P.aeruginosa* strains.

Discussion:

In our study, isolation rate of *P.aeruginosa* was 3.35%, which is consistent with study done in Indore.[15] Among them, hospital acquired *P.aeruginosa* was more than community acquired *P.aeruginosa* which is similar to studies done in India.[16,17] Males were more affected than females and mean age of patients was 50±2.5 which is in accordance with study done by Rajput et al.[18]

In our study, isolation rate was highest from pus(60.27) followed by urine, ear discharge, blood and sputum which is in accordance to study done by Banjare et al.[19] In community acquired ,isolation rate was common in ear discharge. This can be due to community acquired *P.aeruginosa* is most frequent etiology of otitis externa.[20]

According to our findings, hospital acquired strains were more resistant to almost all the antibiotics evaluated then community acquired strains which shed light on the fact that hospital acquired strains are more resistant then community acquired strains.[21] Reason to this is selective pressure caused by regular use of antibiotics in hospitals. Among different groups of antibiotics, all the isolates were sensitive to polymyxin B and colistin. After that, carbapenems was the most sensitive which is similar to studies done all over.[22,23] There was 3.49% resistance to imipenem among *P.aeruginosa* which is similar to findings of Lister PD.[24] Drug resistance to piperacillin was high but resistance to piperacillin+ tazobactam was 32.56, which can be compared to study done by Chander et al.[25] Ceftazidime, group A drug for *P.aeruginosa,* come out to be considerably resistant in this study.[12] Another cephalosporin, cefepime also showed high resistance which is similar to study done by Chander et al.[25] Among fluroquinolones, ciprofloxacin was higher resistant than levofloxacin in both community and hospital acquired strains. MDR was seen in 24.65% isolates, which can be compared with study done in India.[26]

In our findings, higher antibiotic resistance was seen in pus (60.27%), but for confirmation further study is required involving larger sample size.

According to our findings, although as whole *P.aeruginosa* showed higher resistance but community acquired *P.aeruginosa* can be empirically treated with amikacin,levofloxacin, aztreonam and piperacillin+ tazobactam and hospital acquired *P.aeruginosa* by amikacin, piperacillin+ tazobactam and carbapenems. Polymixyn B and colistin can be preserved for carbapenem resistant strains.

CONCLUSION:

Our study deduce that prescription of antibiotics should be done according to local antibiotic susceptibility data followed by routine culture and sensitivity test to contain the spread of multidrug resistant *P.aeruginosa.*

REFERENCE:

1. Rice LB. Federal funding for the study of antimicrobial resistance in nosocomial pathogens: no ESKAPE. J Infect Dis 2008;197:1079–1081.
2. Pogue JM, Kaye KS, Cohen DA, Marchaim D. Appropriate antimicrobial therapy in the era of multidrug-resistant human pathogens. Clin Microbiol Infect 2015;21:302–312.
3. Javiya, V.A., Ghatak, S.B., Patel, K.R., Patel., J.A. 2008. Antibiotic susceptibility patterns of Pseudomonas aeruginosa at a tertiary care hospital in Gujarat, India. Indian J. Pharmacol., 40: 230 234.
4. Rose, H. D., Franson, T. R., Sheth, N. K., Chusid, M. J., Macher, A. M. & Zeirdt, C. H. (1983). Pseudomonas pneumonia associated with use of a home whirlpool spa. JAMA 250, 2027–2029.
5. Dwivedi M, Mishra A, Singh RK, Azim A, Baronia AK, Prasad KN.Nosocomial cross transmission of Pseudomonas aeruginosa between patients in a tertiary intensive care unit. IJPM 2009; - 5 2 (4):509-13
6. Pollack M. Pseudomonas aeruginosa. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and Practice of Infectious Diseases. New York, NY: Churchill Livingstone; 1995:1980-2003.
7. Doyle JS, Buising KL, Thursky KA, Worth LJ, Richards MJ. Epidemiology ofinfections acquired in intensive care units. Semin Respir Crit Care Med2011;32(2):115–38.
8. Hattemer A, Hauser A, Diaz M, Scheetz M, Shah N, Allen JP, Porhomayon J, El-Solh AA. Bacterial and Clinical Characteristics of Health Care- and Community-Acquired Bloodstream Infections Due to Pseudomonas aeruginosa. Antimicrobial Agents and Chemotherapy 2013;57(8): 3969–3975
9. Bisbe J, Gatell JM, Puig J, Mallolas J, Martinez JA, Jimenez de Anta MT, Soriano E. 1988. Pseudomonas aeruginosa bacteremia: univariate and multivariate analyses of factors influencing the prognosis in 133 episodes. Rev. Infect. Dis. 10:629–635.
10. Chatzinikolaou I, Abi-Said D, Bodey GP, Rolston KV, Tarrand JJ, Samonis G. 2000. Recent experience with Pseudomonas aeruginosa bacteremia in patients with cancer: retrospective analysis of 245 episodes. Arch. Intern. Med. 160:501–509.
11. Forbes BA, Sahm DF and Weissfeld AS. Bailey and Scott's Diagnostic Microbiology, 11th edition. St. Louis: Mosby. 2002.148-168 pp.
12. CLSI. Performance standards for antimicrobial disc susceptibility tests; approved standard-11th ed., CLSI 2012; Document M02-A11
13. ASM microlibrary (http://www.microbelibrary.org/library/laboratory-test/3656-polymerase-chain-reaction)
14. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect., 2012; 18(3): 268–81.
15. Jain K, Rajput MS, Chavan N et. al. ESBL- a great concern in urinary tract infections in tertiary care hospital, Indore. Int J Health Sci Res. 2015; 5(8):217-223.
16. Anupurba, S., Battacharjee, A., Garg, A., Ranjansen, M. 2006. The antimicrobial susceptibility of Psuedomonas aeruginosa isolated from wound infections. Indian J. Dermatol., 51(4): 286 288.
17. Prashant Durwas Peshattiwar, Basavaraj Virupaksappa Peerapur, 2011. ESBL and MBL Mediated Resistance in Pseudomonas aeruginosa: An emerging threat to clinical therapeutics. J. Clin. Diagn. Res., 5(8): 1552 1554.
18. Rajput M. S., Kumar P., Thanna R. C., Resistance Pattern of Pseudomonas Aeruginosa Isolates from Surgical Wounds. Indian J Microbiol Res 2015;2(1):46-49.
19. Banjare B , Barapatre R. Incidence of carbapenem-resistant pseudomonas aeruginosa in clinical samples International Journal of Biomedical Research 2015; 6(08): 567-569.
20. Brook I, Fraizer E,Thompson D. Aerobic and anaerobic microbiology of external otitis. Clin Infect Dis. 1992;15:955-958.
21. Arshi Syed, Manzoor Thakur, Syed Shafiq, Assad Ullah Sheikh, 2007. In-vitro sensitivity patterns of Pseudomonas aeruginosa strains isolated from patients at skims - role of antimocribials in the emergence of multiple resistant strains. JK-Pract., 14(1): 31 34.
22. Satyajeet, K., Pawar, P., Mane, M., Ravindra, V., Shinde, H., Patil,V., Patil, S.R., Karande, G.S., Mohite, S.T. 2014. Pseudomonas aeruginosa and its antibiogram from clinical isolates in a tertiary teaching hospital from Western Maharashtra, India. J. Evidence Based Med. Healthcare, 1(7): 574 581.
23. Sibhghatulla Shaikh, Jamale Fatima, Shazi Shakil, Syed Mohd. Danish Rizvi, Mohammad Amjad Kamal, 2014. Prevalence of multidrug resistant and extended spectrum beta-lactamase producing Pseudomonas aeruginosa in a tertiary care hospital. Saudi J. Biol. Sci., http://dx.doi.org/10.1016/j.sjbs.2014.06.0 01
24. Lister PD. Beta-lactamase inhibitor combinations with extended-spectrum penicillins: factors influencing antibacterial activity against enterobacteriaceae and Pseudomonas aeruginosa. Pharmacotherapy;2000:20:213-218.
25. Chander Anil, Raza Mohammad Shahid. Antimicrobial susceptibility pattern of Pseudomonas aeruginosa clinical isolates at a tertiary care hospital in Kathmandu, Nepal., 2013; 6: 235-38.
26. Prakash V, Mishra PP, Premi HK, Walia A, Dhawan S, Kumar A. Increasing incidence of multidrug resistant Pseudomonas aeruginosa in inpatients of a tertiary care hospital. Int J Res Med Sci., 2014; 2(4): 1302-1306.