

How to Cite:

Patil, S. S., Pawar, S. K., Kadam, S. H., & Kakade, S. V. (2022). *Pseudomonas aeruginosa* from intensive care units: Prevalence, clinical and antimicrobial profile. *International Journal of Health Sciences*, 6(S1), 9846–9853. <https://doi.org/10.53730/ijhs.v6nS1.7296>

***Pseudomonas aeruginosa* from intensive care units: Prevalence, clinical and antimicrobial profile**

Shweta S. Patil

Dr. Satyajeet K. Pawar (Corresponding Author)
Associate Professor, Department of Microbiology, KIMS, Karad
Email: drskpawar@gmail.com

Shamala H. Kadam

Dr. Satish V. Kakade

Abstract---Infections caused by resistant organisms are increasing in hospitalised patients. *Pseudomonas aeruginosa* is one the important pathogen which is resistant to many antimicrobials and associated with infections in intensive care units with higher rates of morbidity and mortality. It can cause wide range of infection in ICU patients like, wound infections, septicaemia, urinary tract infection, cystitis, and rarely pneumonia. The study was carried out with an aim to study prevalence, clinical and antimicrobial profile of ICU infections caused by *P. aeruginosa*. Bacteriological study of total 589 clinical specimens from different ICU s was done. Prevalence of *P. aeruginosa* infection was 13.66 %. Age group 21- 40 and 41 to 60 was most affected (36.25 %). Maximum isolates were from medicine ICU (51.25 %) and from urine specimen (37.5 %). Piperacillin (48.75 %) and Amikacin (47.5 %) showed most susceptibility pattern. To conclude it is very important to have routine surveillance of ICU infections to prevent pan drug resistant *Pseudomonas aeruginosa* infection.

Keywords---*Pseudomonas aeruginosa*, antimicrobial, infections.

Introduction

Infections caused by resistant organisms are increasing in hospitalised patients. More so the infection with these organisms is increasing exponentially among intensive care unit (ICU) patients.¹ *Pseudomonas aeruginosa* is one the important pathogen which is resistant to many antimicrobials and associated with infections in intensive care units with higher rates of morbidity and mortality.² Overuse of

antimicrobials for treatment of intensive care unit patients results in selection pressure causing resistant bugs development and their spread.³ Among ICU pathogens from ESKAPE group (Enterococcus, Staphylococcus, Klebsiella, Acinetobacter, Enterobacter) *Pseudomonas aeruginosa* has become one of the leading causative agent.⁴ *Pseudomonas aeruginosa* can cause wide range of infection in ICU patients like, wound infections, septicaemia, urinary tract infection, cystitis, and rarely pneumonia.

From the genus *Pseudomonas*, commonly known as Pseudomonads, the *Pseudomonas aeruginosa* is commonly isolated organism from clinical specimens. This is because of its stubborn nature, ubiquitous availability, innate mechanism of resistance to many antimicrobials and antiseptics, ability to remain alive in moist environment and last but not the least of acquiring drug resistance against the newer antimicrobials.⁵

Innate mechanism of resistance in *P. aeruginosa* is based on its low permeability of the outer member and overexpression of efflux pumps. The acquired resistance includes mutational changes or acquisition of genes encoded for penicillin binding proteins, efflux pumps, porins and beta-lactamase production.⁶ It is therefore important to have proper empirical treatment policy for any ICU infection caused by *P. aeruginosa*. Present study was conducted in a tertiary care hospital with an aim to study prevalence, clinical and antimicrobial profile of ICU infections caused by *P. aeruginosa*. Males (66 %) and were more affected

Material and Methods

This hospital based prospective study was carried out between June 2020 and May 2021. Total 589 clinical specimens from different ICU s from Krishna Hospital and Medical Research Centre (KH & MRC) were included in the study and were processed in the Department of Microbiology, Krishna Institute Medical Sciences, Karad after ethical clearance from the institute. All samples were collected under aseptic precautions by standard procedures and processed according to standard guidelines. Brain heart infusion broth was used for the blood culture. The broth which showed turbidity was sub-cultured onto the MacConkey agar and Blood agar media using sterile technique. All other specimens were processed on Blood agar, MacConkey and Chocolate agar. Colony morphology was read out after 24 hours of incubation at 37 ° C and were identified by standard methodology.⁷ From the total 589 clinical specimens, *P. aeruginosa* isolated were 80 in number. These isolates were further studied for demographic data and clinical and antimicrobial profile. The antimicrobial sensitivity testing was done by Kirby Bauer disc diffusion method according to the CLSI guidelines.⁸

Observations and Results

Over a period of 1-year, bacterial isolates obtained from patients admitted in various intensive care unit (ICU) were studied in the Department of Microbiology, Krishna Institute of Medical Sciences, Deemed University, Karad. Total 589 clinical specimens from different ICU s were included in the study. *Pseudomonas*

aeruginosa was isolated from 80 specimens. Isolation rate for *Pseudomonas aeruginosa* was 13.66 %.

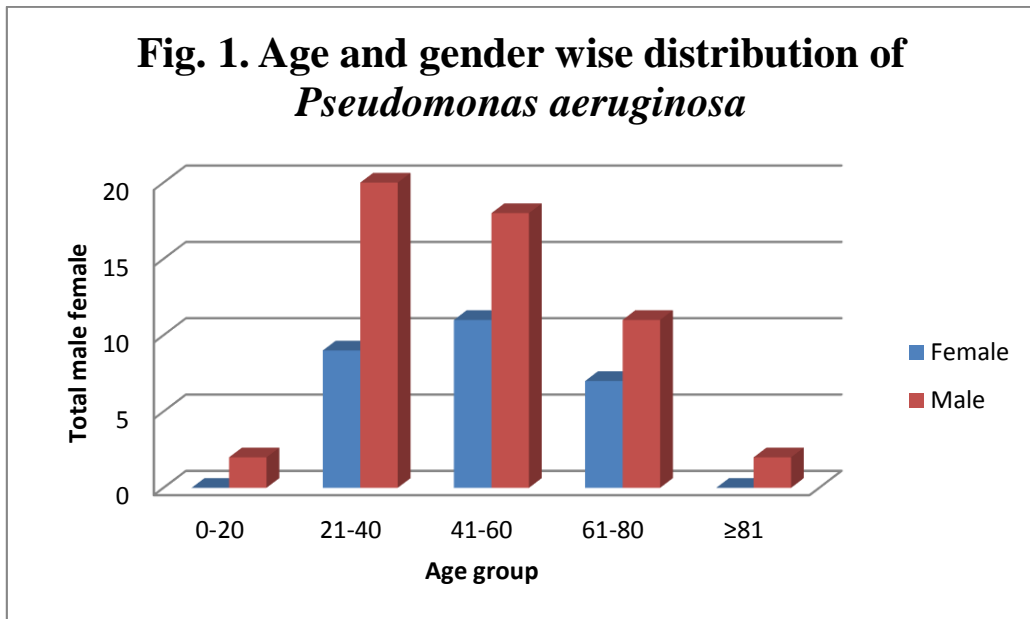
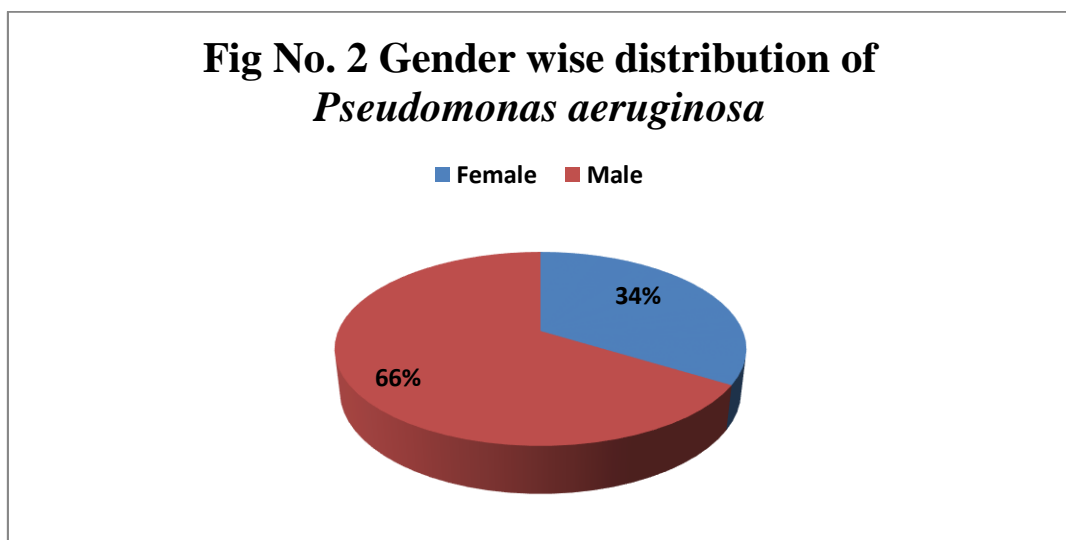


Fig.1 shows age and gender wise distribution of *Pseudomonas aeruginosa*.

Maximum isolates were from 21-40 age group 29 (36.25%), 41-60 age group 29 (36.25%), followed by 61-80 age group 18 (22.5%), 0-20 age group 2(2.5%), ≥81 age group 2(2.5%). Maximum females were from 41-60 age group 11(40.74%), followed by 21-40 age group 9 (33.33%), 61-80 age group 7 (25.92%). Maximum males were from 21-40 age group 20 (37.73%), followed by 41-60 age group 18 (33.96%), 0-20 age group 2(3.77%), ≥81 age group 2(2.5%).



Fig,2: Gender wise distribution of *pseudomonas aeruginosa*

Fig No. 2 shows gender wise distribution of *Pseudomonas aeruginosa*. Maximum isolates were from males 53 (66.25%) followed by females 27 (33.75%)

Table no. 1
Sample wise distribution of *pseudomonas aeruginosa*

Specimens	Total Number	Percentage (%)
Blood	6	7.5
Asitic Fluid	1	1.25
Discharge Swab	2	2.5
ETT	9	11.25
Peritoneal Fluid	1	1.25
Pus	19	23.75
Sputum	8	10
Catheter tip	1	1.25
Urine	30	37.5
Wound Swab	3	3.75
Total	80	100%

Majority of the isolates were from Urine 30 (37.5%) followed by Pus 24 (30%), ETT 9 (11.25%), Sputum 8 (10%), Blood 6 (7.5%) and Catheter tip 1 (1.25). (Table No.1)
Maximum isolates from medicine ICU followed by surgery ICU (Fig.3)

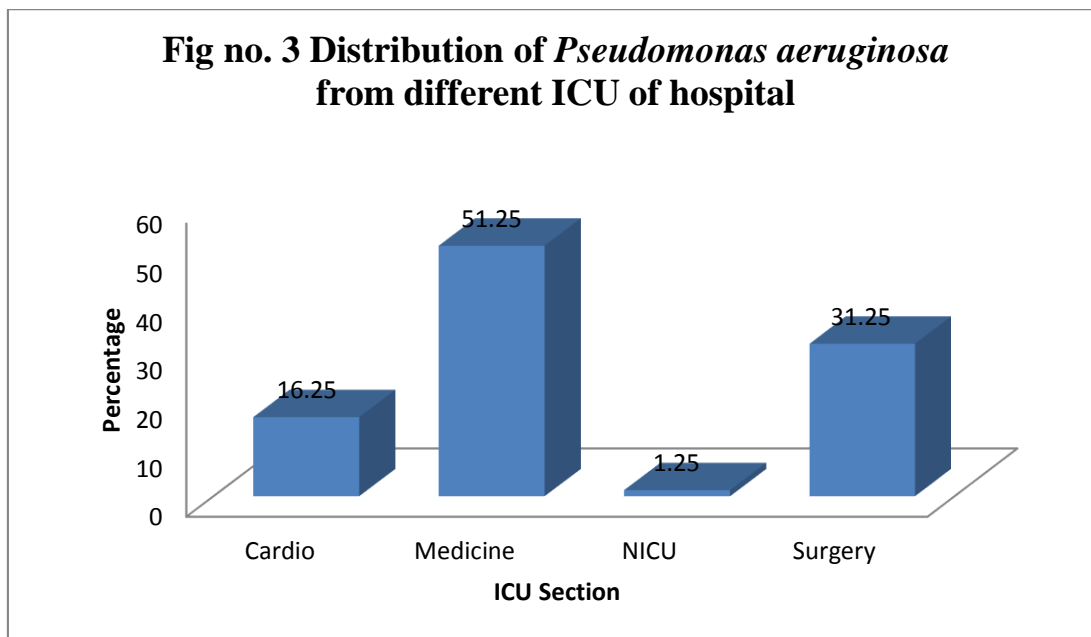


Fig. 3: Distribution of *pseudomonas aeruginosa* isolated from different ICU of hospital

Table no. 2
Antibiotic sensitivity profile of *Pseudomonas aeruginosa*

Antibiotics	Sensitive (%)	Resistance (%)
Amikacin	38 (47.5)	42 (52.5)
Gentamicin	36 (45)	44 (55)
Piperacillin/Tazobactam	27 (33.75)	53 (66.25)
Imipenem	28 (35)	52 (65)
Ceftazidime	28 (35)	52 (65)
Cefoperazone	33 (41.25)	47 (58.75)
Ceftriaxone	27 (33.75)	53 (66.25)
Cefotaxime	13 (16.25)	67 (83.75)
Cefepime	33 (41.25)	47 (58.75)
Amoxicillin/ Clavulanic	26 (32.5)	54 (67.5)
Ciprofloxacin	24 (30)	56 (70)
Levofloxacin	18 (22.5)	62 (77.5)
Piperacillin	39 (48.75)	41 (51.25)
Co-Trimoxazole	11 (13.75)	69 (86.25)

The bacterial isolates were tested against antimicrobial agents and their resistance patterns was observed. (Table No. 2) *Pseudomonas aeruginosa* showed maximum sensitivity to Piperacillin 39 (48.75%) followed by Amikacin 38 (47.5%), whereas, maximum resistance was to Co-Trimoxazole 69 (86.25%), followed by Cefotaxime 67 (83.75%).

Discussion

Pseudomonas aeruginosa infection in ICU are of constant concern. Colonization with this organism often precedes infection and the prevention is therefore extremely important. The choice of empiric antibiotics in the ICU setting is difficult. There needs to be balance between excessively broad coverage and too narrow coverage.

To prevent development of drug resistant *Pseudomonas aeruginosa* infection in ICU infections, patients require broad spectrum empiric coverage. In our study, total of 80 *Pseudomonas aeruginosa* isolates were processed. Prevalence rate of *Pseudomonas aeruginosa* infection was 13.6 % from ICU. Almost similar findings (14.7 %) were seen by Gill JS *et al.*⁹

Out of 80 patients, 53 (66.25%) were males and 27 (33.75%) females. Similarly, Loureiro M. M. *et al.*¹⁰ has found males 59.4% and females 40.6%. Among Indian studies Javiya VA *et al.* had found a 62.5 % of males and 37.5 % of females infected with *P.aeruginosa* which were matching findings in the present study.¹¹

In our study, maximum number of isolates were from urine 30 (37.5%) followed by pus 19 (23.75%), ETT 9 (11.25%), sputum 8 (10%), blood 6 (7.5%), wound swab 3 (3.75%), discharge swab 2 (2.5%), catheter tip 1 (1.25). Gill JS *et al.* had isolated from 53.4 % of urine and 43.18 % of wound specimens.⁹ *P.aeruginosa* is one of the leading uropathogen which is responsible for urinary tract infections worldwide.¹² Urinary catheterizations, long term admission in ICU are few of the

predisposing factors responsible for *P. aeruginosa* infections.¹³ More no of cases 29 (36.25 %) cases, were seen between 21-40 years and 41 to 60 years age group. This is in accordance with various other studies as reported by Senthamarai S. et al. (39.42 %) ¹⁴, Anupurba S et al., ¹⁵ (45.88%) and Okon K.O et al., (24.6%). ¹⁶ The maximum patients in these studies were also from age group between 21–40 years. Maximum isolates (n=41) of *Pseudomonas aeruginosa* were from Medicine ICU (51.25%) followed by Surgery ICU (n=25, 31.25 %)

In our study, *Pseudomonas aeruginosa* showed maximum resistance to Co-trimoxazole 86.25% followed by Cefotaxime 67 (83.75%), Levofloxacin 62 (77.5%), Ciprofloxacin 56 (70%), Amoxicillin/Clavulanic 54 (67.5%), Ceftriaxone 53 (66.25%), Piperacillin/tazobactam 53(66.25%), Imipenem 52 (65%), Ceftazidime 52 (65%), Cefepime 47 (58.75%), Cefoperazone 47 (58.75%), Gentamicin 44 (55%), Amikacin 42 (52.5%), Piperacillin 41 (51.25%).

The study of Shah DA et al. has reported resistance to Co-trimoxazole 99.2%.¹⁷ Sivanmaliappan TS et al. has reported resistance of *Pseudomonas aeruginosa* to Co-trimoxazole 66.6%.¹⁸

In the study by Senthamarai S. et al. ¹⁴ had found resistance of Ceftazidime (65.38%), Cefotaxime (51.92%), Ceftriaxone (55.76%) and Piperacillin (59.61). Ibukun et al., (79.4%) ¹⁹, Yapar et al., (84%) ²⁰ and K.M Mohanasundaram et al., (84.6%) ²¹, had more resistance report against ceftazidime in their study. Our study finding is in line with the findings of Senthamarai S. et al. ¹⁴ and Dwivedi et al. (63%).²²

As a broad-spectrum empirical therapy, there is indiscriminate use of 3rd generation cephalosporin resulting in secretion of ESBL enzymes. This mediate the resistance by breaking or hydrolysis of β -lactam ring of the β -lactam antibiotics.

Conclusion

Pseudomonas. aeruginosa is one of the leading pathogen in infections in ICU setup. Major health concern factor is these isolates are associated with multidrug resistance. Antipseudomonal Penicillins like Piperacillin, aminoglycosides and combination drugs like Amoxicillin and Clavulanic acid gives options for effective empirical antimicrobial therapy but will vary depending on local antibiogram in a hospital. It is therefore very important to have routine surveillance of antibiogram in any ICU set up to prevent development of pan drug resistant *P. aeruginosa*.

References

1. Boucher HW, George H, Talbot J, et al. Bad bugs, No drugs: No ESKAPE! An update from the infectious diseases society of America. Clin Infect Dis. 2009;48(1):1e12.
2. Dereli N, Ozayar E, Degerli S, Sahin S, Koç F. Three-Year Evaluation of Nosocomial Infection Rates of the ICU. Braz J Anesthesiol. 2013;63(1):73-8.

3. Esposito S, Leone S. Antimicrobial treatment for intensive care unit (ICU) infections including the role of the infectious disease specialist. *Int J Antimicrob Agents*. 2007;29:494-500.
4. Moore LS, Freeman R, Gilchrist M, et al. Homogeneity of antimicrobial policy, yet heterogeneity of antimicrobial resistance: antimicrobial non-susceptibility among clinical isolates from primary, secondary and tertiary care patients in London. *J Antimicrob Chemother*. 2014;69(12):v3409e3422
5. Pachori P, Gothwal R, Gandhi P. Emergence of antibiotic resistance *Pseudomonas aeruginosa* in intensive care unit; a critical review. *Genes & diseases*. 2019 Jun 1;6(2):109-19.
6. Oie S, Fukui Y, Yamamoto M, Masuda Y, Kamiya A. In vitro antimicrobial effects of aztreonam, colistin, and the 3-drug combination of aztreonam, ceftazidime and amikacin on metallo β -lactamase-producing *Pseudomonas aeruginosa*. *BMC Infect Dis*. 2009;9:123.
7. Collee JG, Miles RS, Watt B. Tests for identification of bacteria. In: Collee JG, Fraser AG, Marmion BP, Simmons A, Mackie, McCartney's, editors. *Practical Medical Microbiology*. 14th ed. Edinburgh: Churchill Livingstone; 1996. 131-150.
8. Clinical and Laboratory Standards Institute. Performance Standards for Antimicrobial Susceptibility Testing; CLSI document M100-S30.2020
9. Gill JS, Arora S, Khanna SP, Kumar KH. Prevalence of multidrug-resistant, extensively drug-resistant, and pandrug-resistant *Pseudomonas aeruginosa* from a tertiary level intensive care unit. *Journal of global infectious diseases*. 2016 Oct;8(4): 155-159.
10. Loureiro MM, De Moraes BA, Mendonça VL, Quadra MR, Pinheiro GS, Asensi MD. *Pseudomonas aeruginosa*: study of antibiotic resistance and molecular typing in hospital infection cases in a neonatal intensive care unit from Rio de Janeiro City, Brazil. *Memórias do Instituto Oswaldo Cruz*. 2002; 97:387-94.
11. Javiya VA, Ghatak SB, Patel KR, Patel JA. Antibiotic susceptibility patterns of *Pseudomonas aeruginosa* at a tertiary care hospital in Gujarat, India. *Indian journal of pharmacology*. 2008 Oct;40(5):230-234
12. Peix, A., Ramírez-Bahena, M. H., & Velázquez, E. Historical evolution and current status of the taxonomy of genus *Pseudomonas*. *Infection, Genetics and Evolution*, 2009; 9(6), 1132-1147.
13. Moss, W. J., Beers, M. C., Johnson, E., Nichols, D. G., Perl, T. M., Dick, J. D., ... & Willoughby, R. E. Pilot study of antibiotic cycling in a pediatric intensive care unit. *Critical care medicine*, 2002; 30(8), 1877-1882.
14. Senthamarai S. Resistance pattern of *Pseudomonas aeruginosa* in a tertiary care hospital of Kanchipuram, Tamilnadu, India. *Journal of clinical and diagnostic research: JCDR*. 2014 May;8(5): DC30-DC32
15. Anupurba S, Battacharjee A, Garg A, Ranjansen M. The antimicrobial susceptibility of *Pseudomonas aeruginosa* isolated from wound infections. *Indian J Dermatol*. 2006; 51(4): 286-88.
16. Okonk O, Aguwe PC, Oladosu W, Balogun, Uba A, Antibiotic resistance patterns of *Pseudomonas aeruginosa* isolated from clinical specimens in a tertiary care hospital in Northeastern Nigeria. *Journal of microbiology and antimicrobials*. 2009, Vol 1(2); 019:026
17. Shah DA, Wasim S, Abdullah FE. Antibiotic resistance pattern of *Pseudomonas aeruginosa* isolated from urine samples of Urinary Tract

- Infections patients in Karachi, Pakistan. Pakistan journal of medical sciences. 2015 Mar;31(2):341-346
18. Sivanmaliappan TS, Sevanan M. Antimicrobial Susceptibility Patterns of *Pseudomonas aeruginosa* from Diabetes Patients with Foot Ulcers. International Journal of Microbiology. 2011 Nov 17; 2011:605195-
 19. Ibukun A, Tochukwu N, Tolu O. Occurrence of ESBL and MBL in clinical isolates of *Pseudomonas aeruginosa* From Lagos, Nigeria. Journal of American Science. 2007; 3(4): 81-85.
 20. Ayse Yüce, Nur Yapar, Oya Eren Kutsoylu. Evaluation of antibiotic resistance patterns of *pseudomonas aeruginosa* and *Acinetobacter* spp. strains isolated from intensive care patients between 2000-2002 and 2003-2006 periods in Dokuz Eylul University Hospital, Izmir Mikrobiyol Bul. 2009; 43(2):195-202
 21. Mohanasundaram KM. The antimicrobial resistance pattern in the clinical isolates of *Pseudomonas aeruginosa* in a tertiary care hospital: 2008-2010(a 3-year study). Journal of Clinical and Diagnostic Research. 2011, Vol-5(3):491-94
 22. Dwivedi M, Mishra A, Singh RK, Azim A, Baronia AK, Prasad KN. The nosocomial cross - transmission of *Pseudomonas aeruginosa* between patients in a tertiary intensive care unit. Indian J Pathol Microbiol. 2009; 52(4): 509-13.