

Study of Ventilator Associated Pneumonia Its Etiology and Antibioqram ProfileSri Ram Chaudhari¹, Ritesh Kumar^{2*}, Karemera K. John³, Sandesh Shrestha⁴¹⁻³Postgraduates, Department of Microbiology, Sharda University, Greater Noida, U.P, India⁴Demonstrators, Department of Biochemistry, Rama Medical College, Hapur, U.P, India**Original Research Article*****Corresponding author**

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Abstract: Pneumonia seems to have been recognized either as nosocomial or community-acquired infection. Nosocomial pneumonia (NP) is further differentiated into Ventilator-Associated Pneumonia (VAP) if the process arose after the patient had been receiving at least 48 hours of mechanical ventilation. To isolate, identify and study the antibiogram of the organisms causing Ventilator-Associated Pneumonia, A Cross-sectional study. The study was approved by the institutional ethical committee. A total of 69 ETA samples obtained who were on mechanical ventilation for ≥ 48 hours. Out of a total of 69 patients on mechanical ventilation for ≥ 48 hours, only 21 (30.4%) were found to have suffered from Ventilator Associated Pneumonia (VAP) by using ICU scoring system Clinical Pulmonary Infection Score (CPIS). Of the total 29 isolates, 21 (72.4%) were Gram-negative bacilli, 5 (17.2%) were Gram-positive cocci and 3 (10.3%) were Candida. The study confirmed the magnitude of the problem of ventilator-associated pneumonia, in our setup, *Acinetobacter baumannii*, *Pseudomonas aeruginosa* and *Klebsiella* species were found to be potential pathogens causing ventilator-associated pneumonia (VAP) in our ICU.

Keywords: Nosocomial, ventilator, antibiogram, clinical pulmonary infection score.

INTRODUCTION

Ventilator-Associated Pneumonia (VAP) refers to the development of parenchymal lung infection after a patient has undergone intubation or tracheostomy and received mechanical ventilation (MV) after 48 hours [1, 2].

It is the second most common cause of nosocomial infection after urinary tract infection among pediatric and neonatal intensive care unit (NICU) patients and most common among ventilated patients [3]. It carries high mortality rate ranging (6%-68%) and may be as high as (74%) in high-risk population, indicating a serious health hazard among ventilated patients [4,5].

The diagnosis of VAP varies among hospitals and providers but usually requires a new infiltrate on chest X-ray, plus two or more other factors. The diagnosis of VAP was made on clinical and biological criteria. A clinical diagnosis of VAP was made by using Modified Clinical Pulmonary Infection Score (CPIS) ≥ 6 [6,7]. Detection of the causative organism is crucial for the diagnosis. Collecting the lower respiratory tract sample either by invasive (protected specimen brush (PSB) or broncho-alveolar lavage {BAL}) or non-invasive (endotracheal aspirate {ETA}) technique and cultured suitable medium. The American thoracic society (ATS) guidelines recommended that quantitative cultures can be performed on endotracheal aspirate (ETA) or a sample collected by non

bronchoscopically presence of Hospital-acquired pneumonia (HAP) increases hospital stay by an average of 7-9 days per patient[8].

Despite the advancements in the antimicrobial therapy, VAP remains substantial reason of morbidity and mortality. Rapid diagnosis and initiation of appropriate antibiotic treatment, as there is an adverse effect of inadequate treatment on patient's prognosis and the emergence of multidrug-resistant (MDR) pathogen[9]. Therefore, this study was carried out to isolate, identify and determine the antibiogram profile of the organisms causing Ventilator-Associated Pneumonia.

MATERIALS AND METHODS

The study was conducted in the Department of Microbiology, School of Medical Sciences & Research, Sharda Hospital, Greater Noida. Study type: Cross-sectional study. Study duration: The study was conducted for a period of 1 year (2016-2017). Study population: All patients who fall under predefined inclusion criteria of CPIS and were on mechanical ventilation for ≥ 48 hours in ICU, Sharda hospital.

Statistic: Microsoft Excel 2008 was used to analyse the data obtained from the total 69 ETA samples. Ethical consideration: Ethical approval for the study was obtained from the Ethics and Research Committee of the Sharda University.

RESULTS

Distribution of Ventilated patients on the basis of CPIS score result

Total of 69 patients on mechanical ventilation for ≥ 48 hours, only 21 (30.4%) were found to be suffering from Ventilator Associated Pneumonia (VAP) by using ICU scoring system Clinical Pulmonary Infection Score (CPIS).

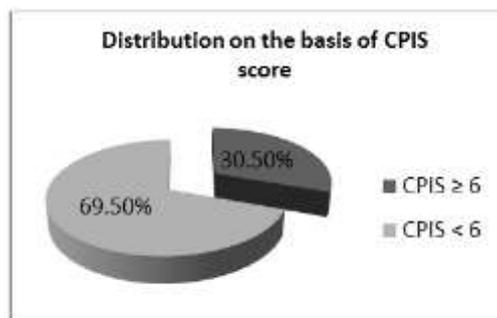


Fig-1: Showing distribution on the basis of CPIS score

Table-1: Distribution of isolated microorganism

Isolated Organism	Number	Percentage (%)
Gram Positive Cocci (GPC)	5/29	17.24%
<i>S.aureus</i>	3	10.34%
<i>CoNS</i>	2	6.89%
Gram Negative Baccili (GNB)	21/29	72.41%
<i>Acinetobacter baumannii</i>	7	24.13%
<i>Klebsiella species</i>	6	20.68%
<i>Pseudomonas aeruginosa</i>	4	13.79%
<i>E.coli</i>	3	110.34%
<i>Proteus species</i>	1	3.44%
Candida	3/29	10.34%

Table-2: Antibiotic sensitivity of Gram Positive Cocci (GPC)

Antibiotics	Sensitivity		Total sensitivity (%)
	CoNS (n=2)	S.aureus(n=3)	
Penicillin	1(50%)	-	(20%)
Vancomycin	2 (100%)	3 (100%)	(100%)
Amikacin	2 (100%)	2 (66.6%)	(80%)
Erythromycin	1 (50%)	1 (33.3%)	(40%)
Ciprofloxacin	1 (50%)	2 (66.6%)	(60%)
Clindamycin	1 (50%)	1(33.3%)	(40%)
Cefoxitin	1 (50%)	1(33.3%)	(40%)
Linezolid	2 (100%)	3 (100%)	(100%)

All Gram-positive cocci demonstrated highest sensitivity against Vancomycin (100%), Linezolid (100%) followed by Amikacin (80%) and Ciprofloxacin

(60%). Most of them were found to be resistant against Penicillin (80%).

Table-3: Antibiotic sensitivity of Gram negative bacilli (GNB)

Name of Antibiotics	<i>Acinetobacter</i> (n = 7)		<i>Klebsiella</i> (n = 6)		<i>E.coli</i> (n = 3)		<i>Proteus</i> (n = 1)		Sensitivity(%)	
	S	R	S	R	S	R	S	R	Total S	TotalR
Amoxicillin	1(14.2%)	6(85.8%)	3(50%)	3(50%)	1(33.3%)	2(66.6%)	1(100%)	-	(35.2%)	(64.8%)
Cefixime	4(57.1%)	3(42.9%)	2(33.3%)	4(66.6%)	1(33.3%)	2(66.6%)	1(100%)	-	(47.05%)	(52.9%)
Cefotaxime	2(28.5%)	5(71.5%)	3(50%)	3(50%)	2(66.6%)	1(66.6%)	1(100%)	-	(47.05%)	(52.9%)
Cefuroxime	1(14.2%)	6(85.8%)	2(33.3%)	4(66.6%)	1(33.3%)	2(66.6%)	-	1(100%)	(23.5%)	(76.5%)
Imipenem	7(100%)	-	6(100%)	-	3(100%)	-	1(100%)	-	(100%)	-
Meropenem	7(100%)	-	6(100%)	-	3(100%)	-	1(100%)	-	(100%)	-
Gentamicin	3(42.8%)	4(57.2%)	2(33.3%)	4(66.6%)	1(33.3%)	2(66.6%)	-	1(100%)	(35.2%)	(64.8%)
Ciprofloxacin	4(57.1%)	3(42.9%)	4(66.6%)	2(33.3%)	2(66.6%)	1(33.3%)	100%	-	(64.7%)	(35.3%)
Levofloxacin	4(57.1%)	3(42.9%)	3(50%)	3(50%)	2(66.6%)	1(33.3%)	1(100%)	-	(58.9%)	(41.1%)

Gram-negative bacilli which include *Acinetobacter baumannii* (7), *Klebsiella* species (6), *E. coli* (3), and *Proteus* (1) demonstrated highest sensitivity against Imipenem (100%) and Meropenem (100%) followed by Ciprofloxacin (64.7%) and Levofloxacin (58.9%). High resistance was noted against Cefuroxime (76.5%), Gentamicin (64.8%) followed by Amoxicillin (35%)

DISCUSSIONS

When the bacteriological profile of ventilator-associated pneumonia was studied, it was found that *Acinetobacter baumannii* (24.1%) was the predominant pathogen that responsible for ventilator-associated pneumonia followed by *Klebsiella species* (20.68%), *Pseudomonas aeruginosa* (13.34%) and *E.coli* (10.34%) were the next major pathogens followed by *Staphylococcus aureus* (10.24%), *CoNS* (6.89%) and *Proteus* (3.44%). These results were in agreement with a prospective study of 90 patients conducted by Set *et al.*, in which it was found that (19.7%) and (9.37%) for *Acinetobacter baumannii* and *Staphylococcus aureus* caused VAP respectively [10].

Another study reported by Golia *et al.* which showed *Acinetobacter baumannii* and *Pseudomonas aeruginosa* (non-fermenter) (8%) and (20%) respectively, as the most predominant isolated causing both early onset and late onset ventilator-associated pneumonia [11]. A similar finding was reported by Rajasekhar *et al.* in which it was found (26.5%) and (14.5%) for *Acinetobacter baumannii* and *E.coli* respectively as the most predominant isolates causing ventilator-associated pneumonia [12].

However, our bacteriological profile was dissimilar to the profile observed by Mohanty *et al.* in which the most infecting microorganism was *Pseudomonas aeruginosa* (30%) followed by

methicillin resistant *Staphylococcus aureus* (23%), *Klebsiella* species (20%) and *Acinetobacter baumannii* (17%) [1]. This difference in the bacteriological profile may be due to the difference in the geographical regions and also in the procedures conducted and the antibiotics policies being followed in different hospitals.

CONCLUSION

The present study confirms the magnitude of the problem of ventilator-associated pneumonia, in our setup, *Acinetobacter baumannii*, *Pseudomonas aeruginosa* and *Klebsiella* species were found to be potential pathogens causing VAP in our ICU. The best approach to manage this problem seems to be an adaptation of preventive strategies.

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REFERENCES

1. Society AT, America IDSo. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171:388-416.
2. Mohanty D, Routray SS, Mishra D, Das A. Ventilator associated pneumonia in a ICU of a tertiary care Hospital in India. Journal of Contemporary Medical Research. 2016;3(4):1046-9.
3. Modi PP, Javadekar TB, Nanda S, Pandya NN. A Study on Ventilator Associated Pneumonia in Pediatric Age Group in a Tertiary Care Hospital, Vadodara. National Journal of Medical Research. 2012;2(3):318-21.
4. MJ, Munro CL, Hummel RS, Elswick R, McKinney JL, Sessler CN. Effect of backrest

- elevation on the development of ventilator-associated pneumonia. *American Journal of Critical Care*. 2005;14(4):325-32.
5. Kumar A, Ghauri MI, Razzaque S, Jamali A, Mohammad JS. Ventilator associated pneumonia; prevalence and microbial patterns. *Pakistan Journal of Chest Medicine*. 2015;20(2).
 6. Koenig SM, Truitt JD. Ventilator-associated pneumonia: diagnosis, treatment, and prevention. *Clinical microbiology reviews*. 2006;19(4):637-57.
 7. Fartoukh M, Maître B, Honoré S, Cerf C, Zahar J-R, Brun-Buisson C. Diagnosing pneumonia during mechanical ventilation: the clinical pulmonary infection score revisited. *American journal of respiratory and critical care medicine*. 2003;168(2):173-9.
 8. Rello J, Ollendorf DA, Oster G, Vera-Llonch M, Bellm L, Redman R, et al. Epidemiology and outcomes of ventilator-associated pneumonia in a large US database. *CHEST Journal*. 2002;122(6):2115-21.
 9. Jakribettu RP, Bloor R. Characterisation of aerobic bacteria isolated from endotracheal aspirate in adult patients suspected ventilator associated pneumonia in a tertiary care center in Mangalore. *Saudi journal of anaesthesia*. 2012;6(2):115.
 10. Set R, Bobade O, Shastri J. Bacteriology profile among patients with ventilator-associated pneumonia from a medical intensive care unit at a tertiary care center in Mumbai. *Indian Journal of Pathology and Microbiology*. 2011;54(2):432.
 11. Golia S, Sangeetha K, Vasudha C. Microbial profile of early and late onset ventilator associated pneumonia in the intensive care unit of a tertiary care hospital in bangalore, India. *J Clin Diagn Res*. 2013;7(11):2462-6.
 12. Rajasekhar T, Anuradha K, Suhasini T, Lakshmi V. The role of quantitative cultures of non-bronchoscopic samples in ventilator associated pneumonia. *Indian journal of medical microbiology*. 2006;24(2):107.