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Clinical study of ventilator-associated pneumonia in tertiary care hospital, Kolhapur, Maharashtra, India

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ABSTRACT

Background: Ventilator-associated pneumonia (VAP) is the most common nosocomial infection acquired by patients admitted in the intensive care unit (ICU). However, there is very less information or clinical data available on the occurrence of VAP in Kolhapur, Maharashtra.

Methods: study aims to determine the Incidence of VAP in ICU, to study the association between causative microorganism and sensitivity, and to study the association between prognosis and incidence of VAP. Settings and Design: Tertiary level, medical-surgical ICU; prospective, observational study.

Results: Patients coming to Medicine Department of the hospital subjected to mechanical ventilation for more than 48 hours in critical Care Facility during the period of two years May 2014 to April 2016.

Conclusions: In the incidence of VAP was found to be 78% among ICU patients. Majority (36%) patients had diabetes mellitus, 30% had hypertension before the admission. It is observed that chances of developing VAP were more in patients with co-morbid conditions. The microbiological results of Endotracheal Aspirate showed that, majority 36% had pseudomonas, 26% had Acinetobacter, 22% had no growth, 14% staphylococci (Staphylococcus is a gram-positive, round-shaped bacterium that is a member of the Firmicutes, and is frequently found in the nose, respiratory tract, and on the skin), 2% proteus mirabilis as compared to similar studies. Out of all, Pseudomonas is the most commonly isolated organism. This could be attributed to decreased immunity and a compromised general condition due to associated illness. Also, prolonged hospital stay is also of significance.

Keywords: Antibiotics, Incidence, Infection, ICU, microbiological profile, Outcome, VAP

INTRODUCTION

Pneumonia is the second most common nosocomial infection among critically ill patients, affecting 27% of all critically ill patients¹. It is one among the leading cause of morbidity and mortality among the hospital acquired infections.² Ventilator associated pneumonia (VAP) refers to hospital acquired pneumonia that occurs within 48 hours or longer after mechanical ventilation (MV). It

is characterized by the presence of new or progressive infiltrate, sign of systemic infection (fever, altered white cell count), changes in sputum characteristics.³ Ventilator-associated pneumonia (VAP) is the most commonly seen nosocomial infection among mechanically ventilated patients and is the biggest concern for critical care specialists. Eighty-six percent of nosocomial pneumonias are associated with mechanical ventilation. Though the incidence of VAP has declined in the developed countries, it continues to be unacceptably high in the developing world.^{4,5} VAP that occurs within 48 to 72 hours of MV is termed as early onset VAP. VAP that occurs after this period is considered late onset VAP. VAP is the second most common nosocomial infection in the intensive care unit (ICU) and the most common in mechanically ventilated patients.⁶ The incidence of VAP increases with the duration of MV.⁷

Approximately 10-28% of critical care patients develop VAP during their stay in the critical care unit.¹ The incidence of VAP increases with the duration of MV.⁷ VAP may account for up to 60% of all Healthcare-Associated Infections out of the total. The VAP increases the length of ICU stay of a patient by around 28% and doubles the risk of mortality as compared with patients without VAP.⁸ The crude mortality rate for VAP is 27 to 76 %.⁹ The Studies in the past have consistently shown that a delay in initiation of appropriate antibiotic therapy is found to increase the mortality among the patients developing VAP.¹⁰

Pseudomonas (Pseudomonas is a genus of Gramaerobic negative, Gammaproteobacteria) and Acinetobacter (Acinetobacter is a genus of Gramnegative bacteria belonging to the wider class of Gammaproteobacteria) pneumonia is associated with increased mortality rates when compared to other organisms.¹¹ The epidemiology and outcomes of VAP were addressed in few studies in India; however, no research has been found regarding VAP in Kolhapur region, Maharashtra, India. Hence, this study aims to determine the Incidence of VAP in ICU, to study the association between causative microorganism and sensitivity, and to study the association between prognosis and incidence of VAP.

METHODS

A hospital-based prospective observational study was carried out in D.Y.Patil Medical College and Hospital, Kolhapur, during the period from May 2014 to April 2016 to detect the incidence, causative organisms, and outcomes of VAP. All patients admitted to the Intensive care unit at D.Y.Patil Hospital, Kolhapur during a period of two years requiring Mechanical Ventilation for longer than 48 hrs. were enrolled for the study.

Inclusion criteria

• All patients subjected to mechanical ventilation for more than 48 hours in critical Care Facility.

Exclusion criteria

• Patients having Pneumonia prior to Mechanical ventilation, patients having pulmonary oedema prior to Mechanical ventilation and patients having acute respiratory distress syndrome (ARDS) prior to MV were excluded from the study.

A data collection sheet was used to collect and record the following: detailed history including name, age, sex, underlying clinical conditions, data of admission, and baseline investigations like Blood counts, renal function tests, blood glucose levels, chest x-ray, ECG, endotracheal aspirate for gram staining and culture, and ABG were done. Patients were evaluated for the related factors concomitant diseases, immunosuppression (like chronic renal failure, diabetes mellitus and steroid therapy), indication for MV, the ratio of PaO2 to FiO2 prior to onset of VAP and clinical pulmonary infection score (CPIS).

Chest radiograph were taken at the time of connecting the patient to the ventilator and after 48 hours of mechanical ventilation. Later serial chest radiographs were taken every 24 hours to look for evidence of pneumonia. The diagnosis of VAP was based on clinical and microbiological criteria. Written and informed consent was signed from participant or care taker.

Statistical Analysis

The collected data was compiled in Microsoft Excel 2010 and statistical analysis of the pre-coded data was done using SPSS (Statistical Programme for Social Sciences) software 15 version and Open Epi Software Version 2.3. The statistical analysis was performed using standard tests. Data were summarized using the mean and standard deviations for quantitative variables and frequency and percentage for qualitative variables. Fisher's exact test or x2 statistic was applied when two or more set of variables were compared. P<0.05 was considered to be statistically significant.

RESULTS

A total of 50 patients who met all the inclusion criteria were enrolled in the current study. The mean age of the patients was 54.26+11.6 years. Majority (78%) of the respondents were in the age group more than 40years. Majority (68%) of the respondents were male. A total of 24 (48%) were alcohol users and 36% were tobacco consumers (Table 1). A total of 39 (78%) respondents developed VAP during the study period. VAP incidence rate per 100 patients was calculated as 39/50X100=78%.

A total of 18 (36%) respondents had diabetes and 30% had hypertension before admission. Among those who developed VAP, diabetes population was 44% and hypertension cases were 31%. Pre-morbid conditions like Hepatic encephalopathy, OP poisoning was present in 14% and 12% study population respectively. VAP was developed in few patients (13%) with GB syndrome, and 10% VAP developed patients had IC bleeding, Left side CVA before admission. In general examination, it was found that 30% study respondents hand oedema. Among VAP developed patients 26% had oedema during the general examination (Table 2).

Table 1: Demographic characteristics and proportionof VAP cases among study population.

Variables		Total sample n=50 (%)
Age	More than 40 years	39 (78)
	Less than 40 years	11 (22)
Sex	Male	34 (68)
	Female	16 (32)
Alcohol	Yes	24 (48)
usage	No	26 (52)
Tobacco	Yes	18 (36)
usage	No	32 (64)
VAP	Developed	39 (78%)
	Not Developed	11 (22%)

Table 2: General examination of the study population.

Variables		VAP
	sample	n=39
	n=50 (%)	(%)
Diabetes mellitus	18 (36)	17 (44)
Hypertension	15 (30)	12 (31)
Cardio-vascular attack	4 (8)	3 (8)
Renal failure	2 (4)	2 (5)
Pulmonary tuberculosis	1 (2)	1 (3)
Hepatic encephalopathy	7 (14)	4 (10)
OP Poisoning	6 (12)	2 (5)
GB Syndrome	5 (10)	5 (13)
IC bleeding	5 (10)	4 (10)
Acute pancreatitis	4 (8)	3 (8)
Left side CVA	4 (8)	4 (10)
CKD	3 (6)	3 (8)
DKA	3 (6)	3 (8)
Hepato-renal syndrome	2 (4)	2 (5)
TBM	2 (4)	2 (5)
Right side CVA	2 (4)	2 (5)
NASH	2 (4)	0 (0)
Acute MI with cardiogenic shock	1 (2)	1 (3)
Acute appendicitis	1 (2)	0 (0)
TTP	1 (2)	1 (3)
Unknown poisoning	1 (2)	0 (0)
Viral	1 (2)	1 (3)
encephatopathy		
Oedema	15(30)	10 (26)
Oedema Pallor	15(30) 13 (26)	10 (26) 10 (26)
	Diabetes mellitus Hypertension Cardio-vascular attack Renal failure Pulmonary tuberculosis Hepatic encephalopathy OP Poisoning GB Syndrome IC bleeding Acute pancreatitis Left side CVA OKA Hepato-renal syndrome TBM Right side CVA NASH Acute MI with cardiogenic shock Acute appendicitis TTP Unknown poisoning Viral encephalopathy	Total sample $n=50 (%)$ Diabetes mellitus18 (36)Hypertension15 (30)Cardio-vascular attack4 (8)attack4 (8)attack1 (2)Pulmonary tuberculosis1 (2)Hepatic encephalopathy7 (14)OP Poisoning6 (12)GB Syndrome5 (10)IC bleeding5 (10)Acute pancreatitis4 (8)Left side CVA4 (8)CKD3 (6)DKA3 (6)Hepato-renal syndrome2 (4)Right side CVA2 (4)NASH2 (4)Acute MI with cardiogenic shock1 (2)TTP1 (2)Unknown poisoning1 (2)Viral1 (2)Viral1 (2)

Figure 1 shows endotracheal aspirate, where it was found that majority (36%) had pseudomonas, 26% had acinetobacter, 22% had no growth, 14% staphylococciand 2% proteus mirabilis.



Figure 1: Endotracheal aspirate among the study population.

Table 3 shows Association between outcome and incidence of VAP. Among 39 VAP developed patients died and of the 11 non-VAP developed patients 1 died. This was found to be not statistically significant in chi square test.

Table 3: Association between prognosis(outcome) and
among VAP and non-VAP patients.

Prognosis (Outcome)	VAP Present	VAP Absent	Total Samples
/ VAP	n=39 (%)	n=11 (%)	n=50 (%)
Recovered	34 (87%)	10 (91%)	44 (88%)
Died	5 (13%)	1 (9%)	6 (12%)

Figure 2 shows the comparison between microorganism and sensitivity among VAP developed cases. A total of 33 out of 39 patients were not resistant to all, while 11 out of 39 patients had resistance. Among non-resistance cases, it was found that 17 were due to pseudomonas, 11 due to acinetobacter and 5 were due to staphylococci.



Figure 2: Association between microorganism and sensitivity among VAP developed patients.

A total of 6 out of 39 patients were resistant to all, of which 2 had acinetobacter, 1 was pseudomonas, 2were staphylococci and 1 was proteus mirabilis resistance. This difference between resistance and non-resistance cases of VAP was analysed using chi square test, and it was found to be statistically significant (p value<0.05).

DISCUSSION

The observed incidence of VAP was 78% in the current study. In studies by Nagar et al Dey and Joseph distribution was same in every age group and there was no sex predilection to VAP in other studies conducted by Wagh et al and Dey et al.^{7,11-13} Incidence of VAP among ICU patients is at the higher end of the range of 15-58% reported in other studies.^{2,14-16} This high incidence of VAP can be attributed to several factors such as difference in study population.

The high incidence of VAP in our study may be due to pre-morbid conditions and associated co-morbidities which have influence the incidence. In our present the duration of mechanical ventilation is found to be important risk factors for VAP. In a study by Ranjan, et al.² 85.17% patients developed VAP when they were on mechanical ventilation for >15 days.

In present study, majority 18 respondents had DM, 15 had HTN, 4 had CVA, 2 had renal failure, 1 had PTB. It is observed that chances of developing VAP were more in patients with co morbid conditions. Similar observations were mentioned in studies conducted by Rakshit and Heyland et al.^{8,11} This could be attributed to decreased immunity and a compromised general condition due to associated illness. Also, prolonged hospital stay is also of significance.

In present study, the microbiological results of Endotracheal Aspirate showed that, majority 36% had pseudomonas, 26% had acinetobacter, 22% had no growth, 14% staphylococci, 2% proteus mirabilis as compared to similar studies by Fagon et al and Torres et al .^{17,18} Pseudomonas being the most common organism isolated. Similar observations were also seen in studies by Jordi et al and Brennan et al.^{19, 20}

Airway intubation is found to be significant risk factors for developing VAP due to increased frequency of colonization by non-fermenters like Pseudomanasspp and Acinetobacter, spp. Nevertheless, in the era of advance diagnoses and early management of possible complications the incidence of 78% is very high and it needs serious introspection, and this high incidence of VAP could also be possibly due to poor sepsis practice in the hospital/ICU. The overall mortality in patients with VAP in our study was 13% while in the non VAP patients the mortality was 9% (p<0.05). This mortality rate was lesser than in previous studies by Ranjan et al and Mukhopadhyayet al. ^{2,16}

CONCLUSION

VAP is a serious problem in ICU leading to prolonged hospitalization and its associated financial implications, and mortality. Effective sepsis practice like hand washing is widely considered as an important but underutilized measured to prevent nosocomial infections like VAP. Main causative organism is pseudomonas in endotracheal aspirate, which needs to be diagnosed and treated as early as possible. Hence, better knowledge of local patterns of pathogens causing VAP can help the clinicians facilitate treatment options. Further studies are needed to under the condition of ICU in detail.

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REFERENCES

- 1. Koenig SM, Truwit JD. Ventilator-associated pneumonia: diagnosis, treatment, and prevention. Clin Microbiol Rev. 2006;19(4):637-57.
- Ranjan N, Chaudhary U, Chaudhry D, Ranjan KP. Ventilator-associated pneumonia in a tertiary care intensive care unit:Analysis of incidence, risk factors and mortality. Indian J Crit Care Med. 2014;18(4):200-4.
- 3. Saravu K, Preethi V, Kumar R, Guddattu V, Shastry AB. Determinants of ventilator associated pneumonia and its impact on prognosis:A tertiary care experience. Indian J Crit Care Med. 2016;17(6):337-42.
- 4. Gupta A, Agrawal A, Mehrotra S, Singh A, Malik S, Khanna A. Incidence, risk stratification, antibiogram of pathogens isolated and clinical outcome of ventilator associated pneumonia. Indian J Crit Care Med. 2011;15(2):96-101.
- 5. Khilnani GC, Jain N. Ventilator Associated Pneumonia :Changing microbiology and implications. Indian J Crit Care Med. 2013;17(6):331-2.
- 6. Kalanuria AA, Zai W, Mirski M. Ventilatorassociated pneumonia in the ICU. Crit Care, Bio Med Cent. 2014;18:208.
- Wagh H, Acharya D. Ventilator Associated Pneumonia - an Overview. Br J Med Pract. 2009;2(2):16-9.
- 8. The Canadian Critical Care Trials Group. A Randomized Trial of Diagnostic Techniques for

Ventilator-Associated Pneumonia. N Engl J Med. 2006;355(25):2619-30.

- 9. Peter J, Chacko B, Moran J. Comparison of closed endotracheal suction versus open endotracheal suction in the development of ventilator-associated pneumonia in intensive care patients: an evaluation using meta-analytic techniques. Indian journal of medical sciences. 2007;61(4):201-11.
- 10. Wip C, Napolitano L. Bundles to prevent ventilatorassociated pneumonia: how valuable are they? Curr Opin Infect Dis. 2009;22(2):159-166.
- Rakshit P, Nagar VS, Deshpande AK. Incidence, clinical outcome, and risk stratification of ventilatorassociated pneumonia - a prospective cohort study. Indian J Crit Care Med. 2005;9(4):211-6.
- Rodrigues DO, Cezário RC, Filho PPG. Ventilator-Associated Pneumonia (VAP) caused by Multidrug-Resistant (MDR) Pseudomonas aeruginosa vs. other microorganisms at an adult clinical-surgical intensive care unit in a Brazilian University Hospital: Risk factors and outcomes. Int J Med Med Sci. 2009;1(10):432-7.
- Joseph NM, Sistla S, Dutta T, Badhe A. Ventilatorassociated pneumonia in a tertiary care hospital in India: Incidence and risk factors. J Infect Dev C tries. 2009;3(10):771-7.
- 14. Morehead RS, Pinto SJ. Ventilator-Associated Pneumonia. Arch Intern Med. 2000;160:1926-36.
- 15. Gadani H, Vyas A, Kar AK. A study of ventilator associated pneumonia: Incidence, outcome, risk

factors and measures to be taken for prevention. Indian J Anaesth. 2010;54(6):535-40.

- Mukhopadhyay C, Bhargava A, Ayyagari A. Role of mechanical ventilation and development of multidrug resistant organisms in hospital acquired pneumonia. Indian J Med Res. 2003;118:229-35.
- 17. Chastre J, Fagon J. Ventilator-associated Pneumonia. Am J Respir Crit Care Med. 2002;165:867-903.
- Torres A, Carlet J. Ventilator-associated pneumonia. European Task Force on ventilator-associated pneumonia. Eur Respir J. 2001;17(5):1034-45.
- Rello J, Ollendorf DA, Oster G, Vera Llonch M, Bellm L, Redman R, Kollef MH. Epidemiology and outcomes of ventilator-associated pneumonia in a large US database. Chest Journal. 2002;122(6):2115-21.
- Brennan MT, Bahrani-Mougeot F, Fox PC, Kennedy TP, Hopkins S, Boucher RC, Lockhart PB. The role of oral microbial colonization in ventilatorassociated pneumonia. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2004;98(6):665-72.

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