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Original Article

A Study to Find out Incidence, Etiology, Diagnosis and Outcome of Ventilator Associated Pneumonia

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ABSTRACT

Background: *Ventilator-associated pneumonia (VAP) is defined as pneumonia that occurs 48-72 hours or thereafter following endotracheal intubation. VAP contributes to approximately half of all cases of hospital-acquired pneumonia. VAP is estimated to occur in 9-27 % of all mechanically ventilated patients. Hence the present study was undertaken to study incidence, etiology, diagnosis and outcome VAP.*

Methods: *A total of 100 patients who will be kept on mechanical ventilator will be selected in an intensive care unit (ICU). Cases included will be patients of both sexes who were kept on mechanical ventilator for more than 48 h, having the age of >14 years. Informed Written consent will be obtained from all the study subjects. A detailed clinical evaluation including thorough history, physical and general examination will be done on each subject. Growth >105 CFU/ml was taken as the cut-off threshold for ETAs while growth >104 CFU/ml was taken as the cut-off for BAL. All patients who will be included in the study will be monitored at frequent intervals (every three days) for the development of VAP using clinical and microbiological criteria until either discharge or death. The clinical parameters will be recorded from their medical records and bedside charts. Details of antibiotic therapy, surgery, use of steroids, duration of hospitalization, presence of neurological disorders, and impairment of consciousness will also be noted.*

Results: *Out of 100 patients VAP was found to be in 43 patients . in early onset VAP pseudomonas aeruginosa and acinetobactor baumanii are the chief causative organisms (36% each) .in late onset VAP pseudomonas, klebsiella were most common causative organisms(52% each) followed by acinetobactor baumanii.*

Conclusions*: This proves that non-invasive ventilatory support is an effective tool in the management of acute exacerbation of COPD.*

Keywords: *Ventilator Associated Pneumonia, hospital-acquired pneumonia*

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INTRODUCTION

Ventilator-associated pneumonia (VAP) refers to bacterial pneumonia developed in patients who have been mechanically ventilated for a duration of more than 48 h.[1] It ranges from 6 to 52% and can reach 76% in some specific settings.[2] Hospital-acquired pneumonia (HAP) is the pneumonia after 48 h or more after admission, which did not appear to be incubating at the time of admission. For a long time it was common to distinguish between an early onset (the first 4 days) and a late onset (after the 4th day) of ventilator-associated pneumonia (VAP). Early-onset nosocomial pneumonia was believed to be due primarily to gram-negative bacteria, such as *Haemophilus influenzae*, and methicillin-sensitive *Staphylococcus aureus* (MSSA) and *Streptococcus pneumoniae*. For late-onset nosocomial pneumonia, the most commonly encountered causative pathogens reported were higher-level antibiotic-resistant gram-negative bacteria, such as *Pseudomonas aeruginosa*,

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Acinetobacter spp., or *Methicillin-resistant S. aureus* (MRSA).

The Clinical Pulmonary Infection Score (CPIS) was developed by Pugin and colleagues to facilitate the diagnosis of VAP using clinical variables. It gives a score of 0–3 for temperature, leucocytosis, ratio, chest radiography, tracheal secretions, and culture of tracheal aspirate. The maximum score that can be obtained is 12 and a score >6 is diagnostic of VAP.

METHODS

A total of 100 patients who will be kept on mechanical ventilator will be selected in intensive care unit (ICU) . Cases included will be patients of both sexes who were kept on mechanical ventilator for more than 48 h, having the age of >14 years.

INCLUSION CRITERIA

1. Patients who are mechanically ventilated for more than 48 hours, with occurrence of new and persistent infiltration in the chest roentgen, together with any two of the following:
2. Patients above age 14 years.
3. Fever, defined as temperature >38°C
4. Leucocytosis, defined as total leukocyte count >11 X103 or leucopenia defined as total leukocyte count <4x103
5 . Purulent tracheal aspirates.

EXCLUSION CRITERIA. Patients who die within 48 hours.2. Develops pneumonia within 48 hr 3. Those who are admitted with pneumonia at the time of admission 4. Those who were admitted to ICU with diagnosis of pneumonia or had any infiltrations in the chest X-ray at the time of ICU admission were also excluded. 5. Patients below 14 yrs of age or temp. Below 38 deg cent or leukocyte count between 4x103 to 11X103 were also excluded.

Utility of CPIS for diagnosis of VAP

The diagnosis of VAP will be based on clinical and microbiological criteria . A clinical suspicion of VAP will be made in patients with a Modified Clinical Pulmonary Infection Score (CPIS) > 6, the diagnosis will be confirmed by performing a quantitative culture of the endotracheal aspirate and observing ≥ 105 cfu/ ml.

RESULTS

out of 48 supine patients 33 developed VAP with 68% conversion and out of total 52 semi recumbent patients only 10 developed VAP. Only 10% semi recumbent subjects developed VAP while 69% supine positioning patients developed VAP.

Table 1: Distribution of Patients.

Position	Total cases	Total vap	Percentages	P value
Supine	48	33	69%	0.001*
Semi recumbent	52	10	19%	
Total	100	43		

Table 2: Sex Distribution

ETIOLOGIC AGENT	EARLY ONSET VAP	LATE ONSET VAP	TOTAL VAP
Pseudomonas aeruginosa	5	15	20
Klebsiella pneumoniae	3	15	18
Acinetobactor baumanii	5	8	13
Staph aureus(MRSA)	1	2	3
Staphylococcus epidermidis	1		1

Out of study population of 100 ,43 developed VAP. Out of 43, 29 (67%) developed late Onset VAP while 14 (33%) developed early onset VAP. Out of 14 early onset 4 were expired,8 recovered and 2 went LAMA. out of 29 late onset, 20 were expired, 5 recovered and 4 went LAMA.

Table 3: Type of Treatment

Outcome	Early Onset Vap	Late Onset Vap	P Value	Total Vap	Non Vap	P Value
Expired	4	20	0.0157*	24	17	0.029*
Recovered	8	5		13	30	
Lama	2	4		6	10	
Total	14	29		43	57	

DISCUSSION

A total of 100 patients who will be kept on mechanical ventilator will be selected in intensive care unit (ICU). Cases included will be patients of both sexes who were kept on mechanical ventilator for more than 48 h, having the age of >14 years .

intensive care unit education on the prevention of VAP is essential, because the occurrence of nosocomial infections is directly related to the adequacy of staff.[3] Nurses need to understand the pathophysiology of VAP, risk factors for this type of pneumonia, and strategies that may prevent the disease. The primary advantage of a clinical strategy for diagnosing VAP is that it does not require specific expertise or specialized equipment or techniques and is non-invasive. Therefore, such an approach can be utilized anywhere. However, because of the poor specificity of clinical signs and symptoms of VAP and of non-quantitative or semi quantitative cultures of tracheal secretions, relying on the clinical approach would be expected to result in treating non-infectious processes with broad-spectrum antibiotics as well as potentially failing to recognize and pursue non-infectious mimics of VAP and non-pulmonary infections.

Principles to apply when choosing appropriate therapy for VAP include knowledge of organisms likely to be present, local resistance patterns within the ICU, a rational antibiotic regimen, and a rationale for antibiotic de-escalation or stoppage. Although the clinician could know the organisms and sensitivities prior to the development of VAP, this is often not the case. In the latter situation, empirical choices that provide adequate coverage are critical. Early effective therapy for VAP is associated with reduced mortality.

Luna et al. demonstrated that inadequate therapy during the initial 48 h, despite provision of adequate therapy after BAL results, was associated with a mortality rate of 91%.[4] In our study we have taken 100 patients who were on mechanical ventilation for more than 48 hours and were following our inclusion criteria, out of 100 study population 43 patients developed VAP(CPIS>6).

Out of these 43 VAP patients 29 developed late onset VAP while 14 developed early onset VAP.

Crude ICU mortality rates of 24 to 76% have been reported for VAP at a variety of institutions.[5-9] ICU ventilated patients with VAP appear to have a 2- to 10-fold higher risk of death compared with patients without pneumonia. In 1974, fatality rates of 50% for ICU patients with pneumonia versus 4% for patients without pneumonia were reported.[10] The results of several studies conducted between 1986 and 2001 have confirmed that observation: Despite variations among studies that partly reflect the populations considered, overall mortality rates for patients with or without VAP were, respectively: 55 versus 25%.[10] 33 versus 19%[11] 37 versus 9%[6], and 44 versus 19%[9]. Pseudomonas aeruginosa and klebsiella pneumonia both were the causative isolates of most of late onset VAP (15/29, 52% each) followed by *acinetobactor baumanii* (table 2).while in early onset VAP pure isolates were more common and *acinetobactor baumanii*, *Pseudomonas aeruginosa* were chief causative isolates (36% each) followed by *Klebsiella pneumonia* (22%) and *S. epidermidis*.

Coming to the MDR pathogens, it was concluded in our study that 63% of VAP causes by MDR pathogens and ratio was higher in late onset VAP(20 from late onset VAP, 7 from early onset).concerned about individual pathogens, out of 20 cases of pseudomonas[10] were caused by MDR pseudomonas, out of 10, 5 were positive for ESBL and 5 for AmpC among early onset VAP pseudomonas was predominant MDR pathogen. As the incidence of MDR-pathogens was quite high (63%) in our study, this indicates that need for appropriate empirical treatment of VAP with proper antibiotics, effective against these MDR pathogens are required.[12] Production of various forms of beta-lactamases like ESBL, MBL, and AmpC beta-lactamase were responsible for this MDR. Similar to other studies.[13,14] ESBL and and MBL were produced by most of the non-fermenter.

PAO_2 /FIO_2 ratio

In our study out of 43 VAP patients 33(77%) has PAO_2/FIO_2 <240 while 10 (23%) patients having PAO_2/FIO_2 > 240. The PaO_2/FiO_2 ratio was assessed during the course of ventilator support and it was observed that the ratio dropped at least 12–24 h before the onset of the clinic radiologic picture suggestive of VAP. Thus, a decline in the PaO_2/FiO_2 ratio was found to be an early indicator of onset of VAP. Reintubation resulted in a very high incidence of VAP and proved to be an independent risk factor in various studies.

CONCLUSION

Incidence is directly proportional to duration of mechanical ventilation and re-intubation is a strong risk factor for development of VAP. Therefore, duration of ventilation has to be reduced to get rid of morbidity and mortality associated with mechanical ventilation, which can be achieved by administering a proper weaning protocol and titrating sedation regimens as per the need of the patients. Promoting nasogastric feeding. Although necessary for critically ill patients, it should be given keeping the patients in a semi recumbent position with the head end elevated to 45° because the supine position promotes aspiration. A decrease in the PaO_2/FiO_2 ratio is an early predictor of VAP. Pseudomonas is the most common organism in our institution.

Late-onset VAP is associated with poor prognosis as compared to the early-onset variety.

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