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A STUDY ON INCIDENCE AND ETIOLOGY OF VENTILATOR ASSOCIATED PNEUMONIA

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ABSTRACT

BACKGROUND: *Ventilator Associated Pneumonia (VAP) is a subset of pneumonia and the term refers to nosocomial pneumonia in a patient on mechanical ventilator support by endotracheal tube or tracheostomy for greater than or equal to 48 hours. VAP continues to be a major threat to patients admitted in intensive care units and receiving mechanical ventilation.*

AIM: *To study the incidence and etiology of VAP. To analyze the underlying risk factors for VAP. To study the percentage of early and late onset pneumonia in these patients. To study the morbidity and mortality attributed by VAP.*

MATERIALS AND METHODS: *The study comprised of 50 patients who were admitted in IMCU and had underwent mechanical ventilation for 48 hours and clinical criteria was applied to diagnose VAP.*

RESULTS AND CONCLUSION: *The incidence of VAP increases with the duration of mechanical ventilation. The major precipitating factor for developing VAP was aspiration. Diabetes is one of the major risk factor to develop VAP. There was a High incidence of MDR organisms in patients with VAP unlike in community acquired pneumonia.*

KEYWORDS : Ventilator Associated Pneumonia (VAP), Multi Drug Resistant Organisms (MDR), Aspiration

INTRODUCTION:

Pneumonia is the second most common nosocomial infection affecting 27% of all critically ill patients. Pneumonia is defined as Nosocomial when it occurs more than 48 hours after the patient's admission to the hospital and when it was not in incubation at the time of hospitalisation[1]. VAP is a subset of pneumonia and the term refers to nosocomial pneumonia in a patient on mechanical ventilator support by endotracheal tube or tracheostomy for greater than or equal to 48 hours.

VAP continues to be a major threat to patients admitted in intensive care units (ICU) and receiving mechanical ventilation. Eighty-six percent of nosocomial pneumonias are associated with mechanical ventilation and are termed VAP[2].

A VAP arising 48 to 96 hours after tracheal intubation usually is called "early-onset VAP", and the one that occurs after this period as the "late-onset VAP" .Generally, early-onset VAP has a better prognosis and is more likely to be caused by aspiration of antibiotic-sensitive bacteria colonizing the oropharynx. Late-onset VAP may be caused by more unusual or multidrug-resistant (MDR) pathogens and is associated with greater morbidity and mortality.

Endotracheal intubation has been identified as a risk factor for developing VAP. Critically ill patients who are intubated for more than 24 hours were found to be at 6 to 21 times higher risk of developing ventilator-associated pneumonia and those patients intubated for less than 24 hours are at 3 times the risk of VAP, compared to non-intubated patients. Other risk factors for VAP include decreased level of consciousness, gastric distention, and presence of gastric or small intestine tubes, trauma, or COPD. VAP is reported to occur at rates of 10 to 35 cases / 1000 ventilator days, depending on the clinical situation.

Aspiration of oral and /or gastric fluids is recognized to be an essential step in the development of VAP. Pulmonary aspiration is increased by supine positioning and pooling of secretions above the ET tube cuff. Estimates of attributable mortality are variable, but increased duration of ventilation is a consistent finding, along with the corresponding increase in hospital days and cost.

A major component of the problem is the ineffectiveness of therapy once VAP is diagnosed. Brun-Buisson et al have demonstrated failure rates of 49 to 62% despite the use of standard antibiotic combinations. Given the burden of VAP, both physical and financial, and the difficulties in treatment, prevention strategies would appear to be of paramount importance.

PATHOPHYSIOLOGY:

VAP primarily occurs because the endotracheal or tracheostomy tube allows free passage of bacteria into the lower segments of the lung in a person who often has underlying lung or immune problems. Bacteria travel in small droplets both through the endotracheal tube and around the cuff[3,4]. Bacteria colonize the endotracheal or tracheostomy tube and are embolized into the lungs with each breath. Bacteria may also be brought down into the lungs with procedures such as deep suctioning or bronchoscopy.

Whether bacteria also travel from the sinuses or the stomach into the lungs is, controversial[5]. However spread to the lungs from the blood stream or the gut is uncommon. Once inside the lungs, bacteria then take advantage of any deficiencies in the immune system (such as due to malnutrition or chemotherapy) and multiply. A combination of bacterial damage and consequences of the immune response lead to disruption of gas exchange with resulting symptoms.

The main route of VAP occurrence is aspiration of pathogenic gram-

positive and gram-negative bacteria, and colonization on the oropharynx and gastrointestinal tract.

Under normal conditions, the host defense, including filtration and humidification of air in the upper airways, epiglottic and cough reflexes, ciliary transport by respiratory epithelium, phagocytes in distal lung, and systemic cell mediated and humoral immunity, prevent bacterial invasion.

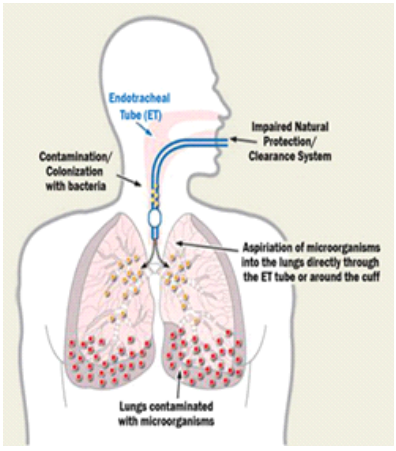

IMAGE 1: Pathophysiology of VAP

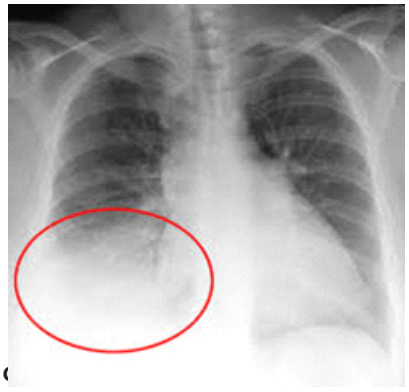

IMAGE 2: (

In intensive care units, the host defenses of patients are usually distorted because of their underlying diseases and invasive devices that are used. Patients are not able to cough efficiently due to sedation or underlying disease.

Also when they are intubated, the endotracheal tube holds the vocal cords open and facilitates aspiration. As a consequence, the endotracheal or tracheostomy tube allows free passage of bacteria into the lower segments of the lung in a person who often has underlying lung or immune problems. Bacteria travel in small droplets both through the endotracheal tube and around the cuff.

Once bacteria reach the distal lung, they multiply and cause invasive disease. Moreover, bacteria then take advantage of any deficiencies in the immune system of the host to continue to multiply and worsen the condition. A combination of bacterial damage and consequences of the immune response lead to disturbances of gas exchange with resulting symptoms.

Morbidity and mortality associated with the development of VAP is high, with mortality rates ranging from 20 to 41%. It has been shown that the development of VAP increases the length on the mechanical ventilator by 4 days, critical care and hospital lengths of stay (LOS) by 4 and 9 days[6].

DIAGNOSIS[7,8]:

VAP should be suspected in any person developing fever, increasing numbers of white blood cells and new shadows (infiltrates) on a chest x-ray. Blood cultures may reveal the microorganism causing VAP.

DIAGNOSTIC CRITERIA:

A new and persistent (>48 hours) infiltrate on chest radiograph 48 hours after admission to hospital not explained by other pathology such as pulmonary edema and not deemed to be incubating at the time of admission into hospital, and Plus two or more of the three criteria:

- Fever of >38.3°C,
- Leukocytosis of $>12 * 10^9$/L, and/or
- Purulent tracheobronchial secretions

This criteria has sensitivity of 69% and a specificity of 75% for establishing the diagnosis of VAP.

Because of the poor specificity of the clinical diagnosis of VAP and of qualitative evaluation of ET Aspirates, Pugin et al. developed a composite clinical score, called the clinical pulmonary infection score (CPIS), based on six variables:

- Temperature,
- Blood leukocyte count
- Volume and purulence of tracheal secretion
- Oxygenation,
- Pulmonary radiography, and
- Semi quantitative culture of tracheal aspirate.[7,8]

The score varied from 0 to 12. A CPIS of >6 had a sensitivity of 93% and a specificity of 100%.

CPIS Score

Day	Parameter	Value for score of	
		1 point	2 points
1	Temp (°C)	38.5 to 38.9	≥39 or ≤36
	White blood cells/mm3	<4,000 or >11,000	<4,000 or > 11,000 & ≥ 50% bands
	Secretions		
	PaO2/FiO2	Non-purulent	Purulent
	Chest X-ray infiltrates	Diffuse or patchy	≤240 & no ARDS Localized
3	Temp (°C)	38.5 to 38.9	≥39 or ≤36
	White blood cells/mm3	<4,000 or >11,000	<4,000 or > 11,000 & ≥ 50% bands
	Secretions		
	PaO2/FiO2	Non-purulent	Purulent
	Chest X-ray infiltrates		≤240 & no ARDS
	Progression of chest X-ray infiltrates	Diffuse or patchy	Localized
	Sputum		Yes
		Culture >1+	Culture >1+ and same organism on Gram stain

As multiple etiologies may explain why patients develop a fever and pulmonary infiltrates while receiving mechanical ventilation, we have to search for other infectious and non- infectious etiologies concurrently with evaluation for VAP. The extent of this investigation is dictated by the clinical circumstances, including physical examination, laboratory findings, and the severity of illness.

MATERIALS AND METHODS:

Patients who were admitted in IMCU had underwent mechanical ventilation for 48 hours were visited and the clinical criteria was applied to diagnose VAP.

The organism that caused VAP was defined as the organism which was Isolated from the sputum or endotracheal aspirate which was sent for culture and sensitivity. The day of onset of VAP was noted to classify into EOP (5 - 7 days) or LOP (>7 days) by revisiting the patients on day 7.

Inclusion Criteria:
Patients who were admitted and underwent mechanical ventilation for 48 hours in medical intensive care unit age > 16 years.

Exclusion Criteria:
- Age < 16 years
- Patients who have got lower respiratory tract infection on admission
- Pulmonary tuberculosis
- COPD
- ARDS
- Bronchial asthmatics

RESULTS:

Table 1: Incidence of VAP

	No. of patients	Percentage
VAP	20	40%
N-VAP	30	60%
Total	50	100%

Table 2: X-Ray Findings

Lobar distribution	Frequency	Percentage
Right side	14	70%
Left side	2	10%
Bilateral	4	20%

Table 3: Early Vs Late VAP

	Frequency	Percentage
Early	7	35%
Late	13	65%

Table 4: Risk factors

Risk Factor	Frequency	Percentage
Diabetes	9	64.3%
H/o Transfer out	2	14.28%
Elderly	1	7.14%
Shock in the first two days of admission	2	14.28%

Table 5: Profile of Organism

	Frequency	Percentage
Gram Negative	12	60%
Gram Positive	5	25%
Polymicrobial	3	15%

Table 6: Age Distribution and Outcome

Age	Death		Recovery	
	Number	%	Number	%
16 - 30 Years	3	20%	2	40%
31 - 60 years	11	73.3%	3	60%
>60 years	1	6.7%	0	0%

DISCUSSION:
Incidence of Ventilator Associated Pneumonia is a well-known entity in the ICU setup. The aim and objective of the study was to find the incidence and etiology of VAP and the underlying risk factors. The data was collected from the sample of 50 subjects admitted in the ICU.

The results of this study revealed the total of 40% of incidence of VAP. The majority of the patients are found to be in the 31 to 60 age group and most common cause was found to be aspiration as shown by the high frequency of patients having infiltrates in the right lung in chest X-Ray (70%). There was increased incidence of VAP among patients who had a long duration of admission in ICU on ventilator. MDR gram negative organisms were found to be more prevalent on culture in this study. Mortality was also increased among the age group 31 to 60 years, the main risk factor found to be diabetes followed by shock.

From this study Ventilator Associated Pneumonia is the major cause of death among patients admitted in the ICU on ventilator. Thus preventive measures, early diagnosis and treatment must be advocated.

CONCLUSION:
- The incidence of VAP increases with the duration of mechanical ventilation.
- Incidence of VAP did not change with either emergency or elective intubation.
- The major precipitating factor for developing VAP was aspiration.
- Diabetes is one of the major risk factor to develop VAP.
- There was a High incidence of MDR organisms in patients with VAP unlike in community acquired pneumonia.
- The Patients admitted in ICU with VAP has a longer duration of stay than N-VAP.

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