Original Research Article

A study on outcomes of ventilator associated pneumonia in children in a tertiary care hospital

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Abstract

Basing the diagnosis of VAP on new or changing Pulmonary infiltrate on CXR and one clinical feature of hospital acquired pneumonia has a good sensitivity but poor specificity. Increasing the number or criteria required increases the ability to distinguish hospital acquired pneumonia from other entities that mimic pneumonia clinically. All patients on mechanical ventilator admitted to the pediatric intensive care unit during the prescribed study period were considered for case identification and study was prospective study. Mortality in present study was 41(27.3%), with almost similar number male and female patients. In our study mean duration hospital stay was 13 days in patients with VAP as compared to 10 days in non VAP group patients. This shows VAP patients had significantly prolonged length of hospital stay as showed in other pediatric studies

Key Word: VAP, Hospital Acquired Pneumonia, Outcome

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Received Date: 14/07/2018 Revised Date: 08/08/2018 Accepted Date: 01/09/2018
DOI: https://doi.org/10.26611/1014731

Access this article online

Quick Response Code:

Website: www.medpulse.in

Accessed Date: 03 September 2018

INTRODUCTION

Major clinical features suggestive of hospital acquired pneumonia are fever, purulent sputum and a new or changing pulmonary infiltrates on chest radiograph (CXR). Although the sensitivity of a clinical diagnosis of VAP is high, the specificity is low. Specificity is a problem in patients with pre-exisitng pulmonary infiltrates such as patients with ARDS. Although an abnormal CXR is essential, the diagnosis of VAP cannot be based on CXR alone. No single radiographic sign or combination of signs increases the likelihood of a diagnosis of hospital acquired pneumonia. There is poor agreement between readers and the interpretation of infiltration is affected by alteration of ventilator settings. The combination of any one clinical feature with an abnormal chest x-ray is associated with a high likelihood of hospital acquired pneumonia.[1] Basing the diagnosis of VAP on new or changing Pulmonary infiltrate on CXR and one clinical feature of hospital acquired pneumonia has a good sensitivity but poor specificity. Increasing the number or criteria required increases the ability to distinguish hospital acquired pneumonia from other entities that mimic pneumonia clinically.[2] The radiographic interpretation of pulmonary disease in ICU setting is challenging as abnormal immune response often alters the appearance of the chest radiographic findings of the pneumonias. Although portable chest X-ray are the most frequently performed radiographic examinations they are often of suboptimal quality despite recent advances in diagnostic imaging. Inadequate exposure, breathing pattern and variations in techniques such as poor patient positioning, different degrees of inspiration and short focus film distance, all may compromise film quality. Many ICU patients who develop pneumonia show diffuse parenchymal opacifications with either interstitial, alveolar or mixed patterns. Resolution after therapy in these patients often is delayed. The radiographic changes in aspiration pneumonia generally develop from 12-24 hrs after aspiration. In supine patients, the abnormal opacities usually develop in the posterior aspect of the upper lobes and in the superior and posterior basal segments of the lower lobes.[3] Lobar type of pattern (non-segmental peripheral airspace consolidation) is frequently caused by bacteria

How to cite this article: Shashikant, Manoj G M. A study on outcomes of ventilator associated pneumonia in children in a tertiary care hospital. *MedPulse International Journal of Pediatrics.* September 2018; 7(3): 47-50. http://medpulse.in/Pediatrics/index.php

(commonly Streptococcus pneumonia and Klebsiella pneumonia). Most hospital acquired pneumonia, begin as bronchopneumonia and are caused by gram positive and Gram negative bacteria. Other infections (eg Fungal, viral, rickettsial and tubercular) and noninfectious disorders (pulmonary thromboembolism, vasculitis, drug reactions) may also be responsible for similar shadows.[4] Interstitial pneumonia is often viral. Radiologically these findings appear as peribronchial cuffing, increased reticular makings or ill defined nodules. Atelectasis is common in post thoracic surgery and upper abdominal surgery but is also seen in other patients in ICU. Most commonly it is due to inspissated mucus plug which is due to excessive secretion, poor respiratory effort and poor cough reflex due to anesthesia and analgesics. The lobar atelectasis more commonly involves lower lobes with right lower lobe 5 times more commonly involved than the left. After lobar atelectasis evidence of volume loss appears within 18 to 24 hours and complete lobar atelectasis may not appear radiologically for days to weeks.[5] CT is definitely more sensitive than plain X-rays but is not routinely available. It is helpful in diagnosing lung abscess, pleural effusion and pneumothorax more efficiently than X-rays. Gallium scanning and Indium radionucleotide labeled leucocyte imaging is often used as screening procedure for detecting area of infection. Although these scans are quite sensitive but they lack specificity and are best used for detecting extra thoracic foci of infection in these patients Nosocomial pneumonia is usually defined pathologically as foci of consolidation with intense leukocyte accumulation in the bronchioles and adjacent alveoli. The collection of pathogenetic material in the air filled alveoli is called consolidation. The pathogenetic material may be inflammatory cells, blood and exudates.[6] In a study, Johanson*et al*[7] classified VAP as mild, moderate, or severe bronchopneumonia. Mild bronchopneumonia was defined as the presence of scattered neutrophilic infiltrates localized to terminal bronchioles and surrounding alveoli. Moderate bronchopneumonia was defined as extension of this process with gross confluence of infiltrates between adjacent lobules, purulent sputum was often present in bronchioles. Severe bronchopneumonia was diagnosed when this process was extensively confluent both grossly and microscopically and was occasionally associated with tissue necrosis. Recent postmortem human studies have used modified histologic criteria to define VAP. In a postmortem study of mechanically ventilated patients, the author group defined VAP as a consolidation at the level of secondary lobules with intense accumulation of polymorphonuclear leukocytes, fibrinous exudate, and cellular debris within alveolar spaces.[8]

METHODOLOG

All patients on mechanical ventilator admitted to the pediatric intensive care unit during the prescribed study period were considered for case identification and study was prospective study.

Methods of collection of data: Sample Size: 150 children aged between 1 month and 16 years Sampling procedure: Consecutive patients in pediatric intensive care unit on mechanical ventilator who developed pneumonia fulfilling inclusion criteria were studied.

Selection Criteria

Inclusion criteria:

- The children aged between 1 month and 16 years who are included in this study are those who are on mechanical ventilator for more than 48 hours

Exclusion Criteria:

- Patients who developed respiratory infections in less than 48 hours of mechanical ventilation, those who are discharged from PICU in less than 48 hours or died within 48 hours are excluded.
- Children of Parents who have not given consent

RESULTS

Table 1: Duration of mechanical ventilation (days) of patients studied

Duration of ventilation (days)	Gender		Total
	Female	Male	
1-2	1(1.4%)	5(6.4%)	**6(4%)**
3-4	48(66.7%)	45(57.7%)	**93(62.0%)**
5-6	23(31.9%)	28(35.9%)	**51(34.0%)**
Total	**72(100%)**	**78(100%)**	**150(100%)**

P=0.249

Ninety three (62.0%), patients were ventilated for 3-4 days duration.

Table 2: Duration of stay in Hospital (days) of patients studied

Duration of stay in Hospital (days)	Gender		Total
	Female	Male	
1-2	0(0%)	0(0%)	**0(0%)**
3-7	19(26.4%)	14(17.9%)	**33(22%)**
8-14	46(63.9%)	53(67.9%)	**99(66%)**
>14	7(9.7%)	11(14.1%)	**18(12%)**
Total	**72(100%)**	**78(100%)**	**150(100%)**

P=0.386

Ninety nine (66%) patients stayed in hospital for 8-14 days duration.

Table 3: Outcome of patients studied

Outcome	Gender		Total
	Female	Male	
Improved	53(73.6%)	56(71.8%)	**109(72.7%)**
Death	19(26.4%)	22(28.2%)	**41(27.3%)**
Total	**72(100%)**	**78(100%)**	**150(100%)**

P=0.803

Mortality in present study was 41(27.3%), with almost similar number male and female patients.

DISCUSSION

Table 4: study in comparison of duration of hospital stay with other studies

Study	VAP	NON VAP
Present study(N =150)	13	10
Edward I. Broughton *et al*[8](N=96)	27.9	12
MahaAlmuneefa *et al*[9](N=360**)**	34	15
RamyaSrinivasan *et al*[10](N=58)	25	23

In our study mean duration hospital stay was 13 days in patients with VAP as compared to 10 days in non VAP group patients. This shows VAP patients had significantly prolonged length of hospital stay as showed in other pediatric studies Almuneef *et al* (34 vs 15)and Elward *et al* (27.9 vs 12). This shows impact of VAP on hospital expenses and importance of it preventing to reduce the health care costs though in present study the average duration hospital stay was less as compared to other studies, as the mortality of the overall study was too high and mortality among VAP group was too high as more severely critically ill children were under study.

Table 5: study in comparison with duration of mechanical ventilation with other studies.

Study	VAP	NON VAP
Present study (N =150)	6DAYS	3.5DAYS
MahaAlmuneefa *et al*[11](N=360)	16 DAYS	10 DAYS
RamyaSrinivasan *et al*[10](N=58)	12 DAYS	09DAYS

In our study mean duration mechanical ventilation was 6 days in patients with VAP as compared to 3.5 days in non VAP group this shows VAP patients had significantly prolonged length of mechanical ventilation, confirming data from other pediatric studies by Almuneef *et al* (21 vs 10) and Ramyasrinivasan *et al*(18 vs 9).This shows impact of VAP on morbidity and hospital expenses and importance of it preventing to reduce the health care costs but average duration Mechanical ventilation in our study was less as compared to other studies as our study population was critically ill children and mortality was too high in VAP group. Overall mortality was also high in compared to other studies.

Table 6: comparison of mortality in VAP and non VAP group in present study with other studies

Study	VAP	NON VAP GROUP
Present study(N=150)	64%	9.3%
Craven DE *et al*[12](N=105)	55%	25%
Torres A *et al*[13](N=190)	71%	28%
Fagon JY *et al*[14](N=240)	33%	19%

In present study showed there was definite higher mortality in VAP than non VAP group as showed in previous studies Craven *et al* (55% vs 25%,), Torres *et al* (71% vs 28%) and Fagon *et al* (33% vs 19%).These rates correspond to increased risk ratios of mortality of VAP patients although these statistics indicate that VAP is a severe disease, previous studies have not clearly demonstrated that pneumonia is indeed responsible for the higher mortality rate of these patients. Two independent factors make it difficult to assign responsibility unambiguously. The first is, once again, the difficulty in establishing a firm diagnosis, that is, to clearly identify patients with VAP; thus, the widely diverging VAP mortality rates reported might reflect not only differences in the populations studied but also differences in the diagnostic criteria used. Second, numerous studies have demonstrated that severe underlying illness predisposes patients in the ICU to the development of pneumonia, and their mortality rates are, consequently, high .Therefore, it is difficult to determine whether such patients would have survived if VAP had not occurred.

CONCLUSION

Children developing VAP required more duration of mechanical ventilation as compared to non VAP group. Mortality was 41% among the mechanically ventilated patients. Mortality among VAP patients was higher as compared to non VAP patients.

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Source of Support: None Declared
Conflict of Interest: None Declared