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Original Research Article

Ventilator associated pneumonia: An enduring hitch in intensive care units!! A study from a tertiary care center

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ABSTRACT

Introduction: Ventilator-associated pneumonia (VAP) is a serious health care-associated infection. It prolongs hospital stay and drives up hospital costs reporting high morbidity and mortality. VAP is defined as pneumonia that occurs 48h or more after endotracheal intubation or tracheostomy, caused by infectious agents not present or incubating at the time mechanical ventilation.VAP requires rapid diagnosis and initiation of the appropriate antibiotics.

Materials and Methods: The present study was done in the department of Microbiology, Rajarajeswari Medical college, Bangalore. All the clinically suspected cases of VAP from intensive care units over a period of one year were included in the study. Endotracheal aspirate (ETA) and bronchoalveolar lavage (BAL) samples were collected from all patients and processed . Identification was carried out according to standard biochemical tests. Sensitivity pattern was determined using Kirby-bauer disc diffusion according to CLSI guidelines.

Results: Out of 160 patients, who were on mechanical ventilation, 7 patients fulfilled the clinical and microbiological criteria. Incidence of VAP in our study is 4.4 and incidence density is 10.5 for 1000 ventilator days.57% of bacterial isolates were found to be *Acinetobacter* spp. followed by *Pseudomonas aeruginosa* 29% and *Klebsiella pneumoniae* 14%. Among 7 cases, 3(43%) were Early onset, 4(57%) were late onset VAP.

Discussion and Conclusion: Even in the era of advanced medical care VAP remains a major challenge. The risk of developing VAP can be reduced by VAP prevention care bundles. Timely diagnosis is a major step to initiate appropriate antibiotics for better outcomes. Both patients and units are at risk of developing multidrug–resistant organisms and therefore appropriate antibiotic stewardship is essential. Better knowledge of local patterns of pathogens causing VAP can help facilitate treatment choice, in turn reducing the ventilator days and hospital stay.

1. Introduction

Nosocomial infection in the intensive care units remain a major threat. The patients in the intensive care units would fall as a prey not only for their critical illness but also for the nosocomial infections. According to the reports, 27% of the critically ill patients suffer from pneumonia and it stands as the second most common nosocomial infection in critically ill patients.[1] Eighty-six percent of nosocomial pneumonias are associated with mechanical ventilation and are termed ventilator-associated pneumonia (VAP).[2] VAP is defined as pneumonia that occurs 48h or more after endotracheal intubation or tracheostomy, caused by infectious agents not present or incubating at the time mechanical ventilation.[3]

Ventilator-associated pneumonia (VAP) is a serious health care-associated infection. It causes prolong hospital stay (ranging from 4 days to 14 days) and ride up hospital costs reporting high morbidity and mortality. It also leads to increased antibiotic pressure. It accounts for the physical, psychological, financial burden to the patient as well to the family.[2] International Nosocomial Infection Control Consortium suggest that the overall rate of VAP is 13.6 per 1000 ventilator days.[4] However, the individual rate varies according to patient group, risk factors, and hospital setting.

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On the average VAP develops after 5-7 days of mechanical ventilation, with a mortality rate between 24% and 76%.[5] Early-onset VAP, is defined as that occur within the first four days of mechanical ventilation.[6] This is usually attributed to the antibiotic-sensitive community-acquired bacteria such as Haemophilus and *Streptococcus*. Whereas VAP that develops after 5 days of mechanical ventilation is termed as late onset sepsis, caused by multidrug – resistant bacteria such as *Pseudomonas aeruginos*a.

VAP appeals early diagnosis and initiation of appropriate antibiotics. Over the past several decades, our knowledge of VAP has grown significantly regarding risk factors, pathogenesis, microbiological profile and its prevention. This study was done to detect the microbiological profile, incidence and incidence density of VAP in our centre.

2. Objective

1. Determine the incidence of VAP
2. Determine the incidence density
3. Identify various bacterial pathogens causing VAP.

3. Materials a nd Methods

3.1. Inclusion criteria

All the clinically suspected cases of VAP (Fever, leucopenia, change in respiratory secretions, respiratory distress and bradycardia or tachycardia) from intensive care units.

3.2. Exclusion criteria

Cases other than VAP

3.3. Procedure

The present study was done in the department of Microbiology, Rajarajeswari Medical college, Bangalore, Karnataka, India. Endotracheal aspirate (ETA) was collected with a mucus extractor by deep suctioning in a patient who was intubated. Bronchoalveolar lavage (BAL) was collected by wedging a bronchoscope or catheter into a bronchus and isolating the distal airway. A volume of saline is instilled and the fluid is aspirated back from the airway using gentle suction. The smears are made from secretions in the sputum cup and smears are stained with both Gram's stain and acid fast staining. Endotracheal tips(ET) were transferred to sterile centrifuge tubes. ET tips were rinsed with 1 ml normal saline, so that washed fluid collects within the centrifuge tube. centrifuge tube was vortexed for dislodging collection within the ET tip and to disperse organisms into the saline. Calibrated 2 mm loop that holds 5 μL was taken and inoculated on to blood agar (BA), and MacConkey agar(MA) plates. The sample was inoculated in the thioglycollate tube. The media was incubated at 37^0 c for 24 hours Plate s were examined after 24hrs. If there is no growth on the plates they are reincubated for another 24 hours. Thioglycollate tubes were incubated for 7 days if plates do not show growth. Examined daily for turbidity. If thioglycollate media shows turbid ity, smear and Gram stain was done and subcultured. Identification was carried out according to standard biochemical tests. Antibiotic sensitivity tests were done by Kirby-Bauer disc diffusion method. The following antibiotics were used for susceptibility testing: ampicillin, amoxyclav, cefuroxime, ceftazidime, cefepime, ciprofloxacin, gentamicin, amikacin, piperacillin, piperacillin+tazobactam, meropenem, imipenem, aztreonam, netilmycin, tigecycline, colistin, and co-trimoxazole. All the discs were procured from [Hi-media laboratories limited]. The diameter of the zone of inhibition was measured and interpreted according to the CLSI guidelines. Incidence and incidence density are calculated.[1,7–9]

3.4. Statistical analysis

The statistical analysis was performed using standard tests. Fisher's exact test was applied. $P < 0.05$ was considered to be statistically significant.

4. Results

Out of 160 patients, who were on mechanical ventilation, 7 patients fulfilled the clinical and microbiological criteria. Out of 7 VAP cases five were males and two were females. The mean age of the patient was 40years and showed male preponderance. Gender description is as shown inTable 1. Out of 7, 2 patients were admitted in respiratory intensive care unit (RICU), 4 in medical intensive care unit (MICU) and one patient was admitted in surgical intensive care unit (SICU). The distribution of patients is as shown in Table 2. Among seven culture positive cases, *Acinitobacter* species was grown in four (57%) samples, *Pseudomonas aeruginos*a in 2(29%),and *klebsiella pneumonia*e in 1(14%) sample. The bacteriological distribution is as shown in Table 3. The growth of *Pseudomonas, acinitobacter* and *Klebsiella* is as depicted in Figures 1, 2 and 3 respectively. Antibiotic susceptibility testing done on Kirby-baur disc diffusion method is as shown in Figure 4. All the isolates were sensitive to colistin and tigecycline, 97% were sensitive to imipenem, 90% to meropenem, 85% to tobramycin, 85% to netilmycin, 66% ceftazidime, 66% to ofloxacin, 85% aztreonem. Among 7 cases, 3(43%) were early onset 4(57%) were late onset VAP. Incidence of VAP in our study is 4.4 and incidence density is 10.5 for 1000 ventilator days.

Table 1: Gender distribution of the VAP cases

Gender	Number of cases
Male	5
Female	2
Total	7

Table 2: Distribution of VAP cases in various critical care units

Name of critical care unit	Number of cases
MICU	4
RICU	2
SICU	1
Total	7

Table 3: The bacteria distribution in VAP samples

Name of the bacteria	Number of samples	Percentage
Acinetobacter species	4	57%
*Pseudomonas aeruginos*a	2	29%
Klebsiella pneumoniae	1	14%
Total	7	100%

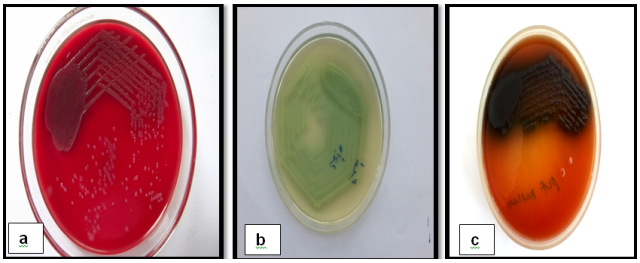

Fig. 1: Growth of *Pseudomonas aeruginos*a on culture media; **a):** Blood agar: large, irregular, beta-hemolytic colonies with iridescence; **b):** Nutrient agar : large, irregular colonies with greenish diffusible pigmen; **c):** Mac Conkey agar: large, irregular, non lactose fermenting colonies

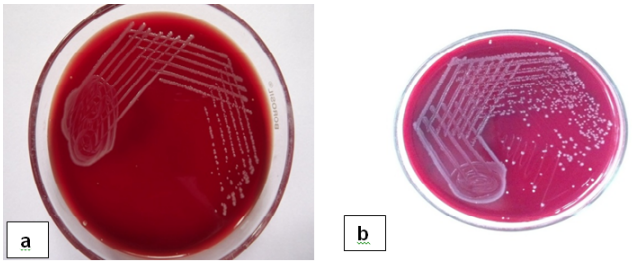

Fig. 2: Growth of *Acinetobacter species* on culture media; **a):** Blood Agar: translucent to opaque, smooth raised colonies; **b):** Mac Conkey Agar: Lactose non-fermenting colonies with faint pink tint

5. Discussion

Even with the implementation of strict hospital infection control practices VAP remain as an enduring hitch in the critical care units. 28% of patients who receive mechanical ventilation in the critical care units are at risk of developing

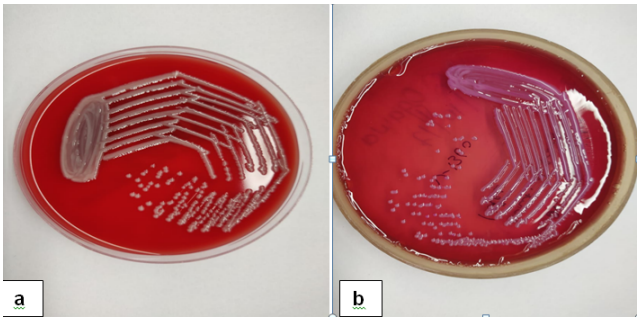

Fig. 3: Growth of *Klebsiella pneumoniae* species on culture media ; **a):** Blood Agar: Opaque, smooth raised, mucoid colonies ; **b):** Mac Conkey Agar: Lactose fermenting,mucoid colonies

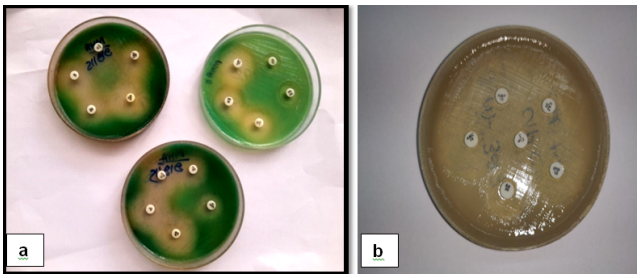

Fig. 4: Antibiotic sensitivity plate ; **a):** Pseudomonas aeruginosa; **b):** Klebsiella pneumoniae

VAP. The incidence of VAP is directly proportional to the duration of mechanical ventilation. Estimated rates are 3% per day for the first 5 days, 2% per day for days 6-10, and 1% per day after

day 10.[10] Knowledge on local pathogens and their sensitivity patterns causing VAP is a preliminary requisite to facilitate treatment choice, thus reducing the ventilator days and hospital stay.

In our study, the mean age of the patient was 40years and showed male preponderance. This is in concordance with the study done by Hina Gadani et al.[11] The incidence and incidence density in our study was 4.4 and 10.5 for 1000 ventilator days. This is lower when compared to the same done by Hina gadina et al (incidence 37%)[11] and Neelima R et al(incidence 57.14, 31.7/1000 incidence density).[12] This could be because of strict vigilance on infection control practices, adequate nursing staff and stern convenance of VAP prevention care bundles. In our study growth from VAP samples yielded *Acinetobacter* species, *Pseudomonas aeruginos*a and *klebsiella* species, this is in correlation with the bacteriological profile of study done by Neelima R et al.[12] *Pseudomon as or Acinetobacte*r pneumonia when compared to other organisms are associated with higher mortality. Studies have shown that mortality risk significantly escalates with delay in starting appropriate and adequate dose of antibiotics. Reports state that, antibiotic use prior to the onset of ventilator-associated pneumonia

(VAP) would substantially increase the probability of infection with multidrug-resistant (MDR) pathogens.[10] Hence prompt and early diagnosis is required to incite appropriate an tibiotics for improved outcomes.[13]

Endotracheal tube or tracheotomy interferes with the normal anatomy and physiology of the respiratory tract, which acts as main culprit for the development of VAP. The level of consciousness is impaired in intubated patients that hinders the voluntary clearance of secretions. This leads to the macro aspiration and micro aspiration of contaminated oropharyngeal secretions that are rich in harmful pathogens. They eventually reach the lower airways leading to the development of a pneumonia.[13] Several studies reported Decontamination of the digestive tract reduces the incidence of VAP by decreasing colonization of the upper respiratory tract. Methods used include antiseptics, such as chlorhexidine in the oropharynx. The aim of this method is to eradicate potentially harmful pathogens like aerobic gram-negative microorganisms and *methi cillin-sensitive Staphylococcus aureus* from oropharyngeal or gastrointestinal tract. Practice of regular oral care and habitual maintainance of basic hygiene minimize dental plaque and colonization with aerobic pathogens thus bring down the mortality and lessen the antibiotic resistance in ventilated patients from cririical care units

An effective strategy should be adopted which aims at infection control at various perspectives like education of the medical staff, universal hand hygiene, use of personal protective equipment and a protocol for microbiological surveillance, prompt reporting, implementing the preventive care bundles in the critical care unit. Basic preventative measures include minimizing time on a ventilator via the implementation of an early weaning protocol, providing regular sedation breaks[13] Utilization of appropriate laboratories facilities and procedures and an immediate implementation of necessary infection control measures also help in restrain the spread of infection right at its source. Knowledge about the local factors leading to VAP and the microbiologic milieu of a given unit is statuary nip the bud.

6. Conclusion

VAP remains a significant risk to the critically ill ventilated patient. Simple and effective preventive measures should be followed. Strict vigilance on early diagnosis, treatment and prevention should be adopted to bring down the VAP rates.

7. Source of Funding

None.

8. Conflict of Interest

None.

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