Ventilator Associated Pneumonia – an Overview

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Summary

Ventilator Associated Pneumonia (VAP) is pneumonia occurring in a patient within 48 hours or more after intubation with an endotracheal tube or tracheostomy tube and which was not present before. It is also the most common and fatal infection of ICU. VAP increases length of ICU stay by 28% and each incidence of VAP is estimated to generate an increased cost of £6000-£22000.

The NICE in collaboration with NPSA is examining four technical patient safety solutions for the prevention of VAP. The Department of Health published a 'High impact intervention' for ventilated patients in June 2007. Eliminating or reducing the unnecessary use of antibiotics should be the primary goal in reducing antibiotic-resistant nosocomial infections.

Ventilator Associated Pneumonia (VAP) is defined as pneumonia occurring in a patient within 48 hours or more after intubation with an endotracheal tube or tracheostomy tube and which was not present before.

Early onset VAP occurs within 48 hours and late onset VAP beyond 48 hours of tracheal intubation.

Incidence

Between 5-15% of hospital in-patients develop infection during admission to ICU. Patients are 5-10 times more likely to acquire nosocomial infections than patients in the wards and approximately 86% of hospital associated pneumonia is linked with mechanical ventilation.

Approximately 10-28% of critical care patients develop VAP. VAP is also the most common and fatal infection of ICU and in the United States it affects 9-27% of intubated patients and doubles the risk of mortality as compared with similar patients without VAP.

VAP may account for up to 60% of all Healthcare-Associated Infections. VAP increases length of ICU stay by 28% and each incidence of VAP is estimated to generate an increased cost of £6000-£22000.

Diagnosis

Despite the high incidence, diagnosis remains challenging because many conditions common to ICU patients like ARDS, sepsis, cardiac failure and lung atelectasis have similar clinical signs. More than 50% of patients diagnosed with VAP do not have the disease whereas upto one-third are not diagnosed. Unfortunately there is no clearly accepted gold standard for diagnosis of VAP.

Centres for disease control and prevention (CDC) national healthcare safety network definition for VAP

Radiology signs (2 or more serial chest x-rays with at least one of the following)
1) New or progressive and persistent infiltrate
2) Consolidation
3) Cavitation

Clinical signs
At least one of the following
1) Fever (temperature > 38 deg C with no other recognised cause)
2) Leucocytosis > 12000 WCC/µL or leucopenia (<4000 WCC/µL)
3) For adults 70 years or older, altered mental status with no other recognisable cause and at least 2 of the following
   1) New onset of purulent sputum, or change in character of sputum, or increased respiratory secretions, or increased suctioning requirements
   2) New-onset or worsening cough, or dyspnoea or tachypnoea
   3) Rales or bronchial breath sounds
   4) Worsening gas exchange (eg. O2 desaturations [PaO2/FiO2 ≤ 240], increased O2 requirements, or increased ventilation demand)

Microbiological criteria (optional)
At least one of the following:
1) Positive growth in blood culture not related to another source of infection
2) Positive growth in culture of pleural fluid
3) Positive quantitative culture from bronchoalveolar lavage (≥104 colony forming units/ml) or protected specimen brushing (≥103 colony forming units/ml)
4) 5% or more of cells with intracellular bacteria on direct microscopic examination of Gram-stained bronchoalveolar lavage fluid
5) Histopathological evidence of pneumonia

Histological landmark of VAP is multifocal disease favouring dependant lung segments, often at different stages of development and severity with cultures growing heterogenous microbial flora.
Pathogenesis:

VAP that occurs within 48 hours after tracheal intubation is usually termed as early onset often resulting from aspiration, which complicates intubation process\(^2\). VAP occurring after this period is late onset. Early onset VAP is often due to antibiotic sensitive bacteria (eg. oxacillin-sensitive Staphylococcus aureus, Hemophilious influenza and Streptococcus pneumoniae), whereas late onset VAP is frequently caused by antibiotic resistant pathogens (eg. oxacillin-resistant Staphylococcus aureus, Pseudomonas aeruginosa, acinetobacter species and enterobacter species)\(^{23,24,25}\)

The pathogenesis of VAP usually requires that two important processes take place:

1. **Bacterial colonisation of the aero-digestive tract**
2. **Aspiration of contaminated secretions into the lower airway**\(^{26}\).

Therefore, the strategies to prevent VAP usually focus on reducing the burden of bacterial colonisation in the aero-digestive tract, decreasing the incidence of aspiration or both.

The presence of invasive medical devices is an important contributor to the pathogenesis and development of VAP\(^{27}\). Many patients have nasogastric tubes that predispose them to gastric reflux and increase the potential for aspiration. Endotracheal tubes facilitate bacterial colonisation of the tracheo-bronchial tree and lower airway aspiration of contaminated secretions through mucosal injury, pooling of contaminated secretions above the endotracheal tube cuff and elimination of the cough reflex\(^{28}\). The ventilator circuit and the respiratory-therapy equipment may also contribute to the pathogenesis of VAP if they become contaminated with bacteria, which usually originate in the patient’s secretions\(^{26,28}\).

**Prevention:**

The National Institute of Clinical Excellence (NICE) in collaboration with National Patient Safety Agency (NPSA) is examining four technical patient safety solutions for the prevention of VAP and in the process of publishing guidelines. The latest technical patient safety solutions for VAP was published in August 2008 which says

1. **Body position**-mechanically ventilated and intubated patients should be positioned with their upper body elevated for as long as possible. This may be inappropriate in some patients, eg. spinal injuries.

2. **Oral antiseptics** e.g. 2% chlorhexidine should be included as part of oral hygiene regimen for all patients who are intubated and ventilated. There is insufficient evidence to recommend any particular antibiotic regimen.

3. **Use of kinetic beds** - a lack of robust evidence meant the Committee was unable to make recommendations for action on the use of kinetic beds.

4. **Care bundles** - although the evidence supported the use of elements of care bundles; there was insufficient evidence to recommend a care bundle of any specific design.

The Department of Health published the following ‘High impact intervention’ for ventilated patients in June 2007

- Elevation of the head of bed to 35-40 degrees
- Sedation holding
- Deep Vein Thrombosis prophylaxis
- Gastric ulcer prophylaxis
- Appropriate humidification of inspired gas
- Appropriate tubing management
- Suctioning of respiratory secretions (including use of gloves and decontaminating hands before and after the procedure)
- Routine oral hygiene as per local policy

In addition the following also may contribute to the prevention of VAP

- Prolonged nasal intubation (more than 48hrs) should be avoided because of the association between nosocomial sinusitis and ventilator-associated pneumonia\(^{29}\).
- Several investigations have suggested that secretions that pool above the inflated endotracheal tube cuffs may be a source of aspirated material and thus VAP. Endotracheal tubes with separate dorsal lumen above the cuff to suction pooled secretions from the subglottic space are now available. The pressure of the endotracheal tube cuff should be adequate to prevent the leakage of colonised subglottic secretions into the lower airway\(^{26,30}\).
Antibiotic Administration:

Previous administration of antibiotics is an important risk factor for VAP because of the presence of antibiotic-resistant bacteria\(^4\). In an attempt to reverse the trend towards increasing rates of antimicrobial resistance among hospital acquired infections, more effective strategies for using antibiotics have been advocated that restrict antibiotic use or offer guidelines for their use \(^32,33\). Eliminating or reducing the unnecessary use of antibiotics should be the primary goal in reducing antibiotic-resistant nosocomial infections\(^32\).

The routine use of prolonged courses of empirical therapy i.e. therapy not supported by results of clinical cultures should be avoided to minimise the subsequent development of antibiotic-resistant infections.

The use of aerosolised antibiotics for the prevention of VAP has been abandoned because of its lack of efficacy and subsequent emergence of antibiotic-resistant infections\(^24\).

Similarly, the routine use of selective digestive tract decontamination has not gained acceptance in the UK and USA because of its lack of demonstrated effect on mortality, emergence of antibiotic resistant infections and additional toxicity. NICE is currently in consultation for selective decontamination of digestive tract guidelines. The technical patient safety solutions for VAP in adults were published in August 2008.

The Committee examined evidence, which suggested that selective decontamination of the digestive tract (SDD) using topical antibiotics may reduce the incidence of VAP and that SDD regimes that include systemic antibiotics may also reduce mortality. However, Specialist Advisers stated that UK intensive care specialists had particular concerns about the risk of infection with *Clostridium difficile* and the induction and/or selection of resistant, including multiresistant, microorganisms as a result of SDD. Therefore the Committee recommended further research into SDD in a UK setting.

Use of broad-spectrum antibiotics is also not recommended for the prevention of VAP because of increasing antibiotic resistance among subsequent hospital acquired infections. Targeted antibiotic therapy with appropriate dose of appropriate antibiotic is the sensible thing to do.

Vaccines:

Various vaccination programmes in adults and children have reduced the incidence of pneumonia caused by specific pathogens including H.influenzae type B, Streptococcus Pneumoniae and Influenza virus \(^34,37\). Vaccinations against these may prevent some hospital acquired infections. Pneumococcal and influenza vaccination must be considered before hospital discharge or included in the discharge planning for all patients at increased risk for subsequent respiratory infections including VAP.

Newer Developments:

There have been new advances in equipment and techniques to help prevention of VAP

3. Endotracheal and tracheostomy tubes with an extra subglottic port to avoid pooled secretions above the endotracheal and tracheostomy tube cuff.

4. Continuous suctioning of the subglottic secretions.

5. Endotracheal tubes with specially designed cuffs that do not allow pooled secretions above the cuff to trickle down causing micro-aspiration and ultimately leading to VAP. eg. Endotracheal tubes with microthin polyurethane cuff.

6. Specially designed closed Tracheal Suctioning Systems (TSS) as compared to open tracheal suctioning systems. However, a meta analysis of randomised controlled trial showed that closed suctioning system is not associated with a lower incidence of VAP or mortality as compared to open suctioning\(^30\).

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COMPETING INTERESTS

None Declared

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