

The prevention of ventilator associated pneumonia in clinical practice – What can be done to lower the risk?

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Introduction

Nosocomial infections are an important and frequent cause of morbidity and mortality in hospitals.¹ Besides these factors, there are tremendous cost implications (reducing already scarce resources in taking care of our patients). Microorganisms that are resistant to multiple drugs are becoming prevalent worldwide.² Common pathogens with resistance include extended spectrum beta lactamase *Klebsiella pneumoniae* and *Escherichia Coli*, vancomycin resistant *Enterococci* and vancomycin intermediate *Staphylococcus aureus* (VISA), penicillin resistant *Streptococcus pneumoniae* and multidrug resistant (MDR) *Pseudomonas aeruginosa*. It is therefore not surprising that antimicrobial resistance is becoming one of the greatest threats to patient health in the 21st century.³

Although urinary tract infection is the most common nosocomial infection – pneumonia remains the second most common nosocomial infection, the most prevalent serious nosocomial infection, and is the most common nosocomial infection in the Intensive Care Unit.^{4,5} The most serious form of this infection is Ventilator Associated Pneumonia (VAP). All patients are already at high risk for comorbid disease and are usually exposed to the most resistant pathogens within the hospital setting, i.e. microorganisms that are commonly present in the intensive care unit.

Table 1: Common pathogens in hospital settings and VAP⁶

- Coagulase negative *Staphylococcus aureus*
- *Staphylococcus aureus*
- *Pseudomonas aeruginosa*
- *Enterococcus* species
- *Enterobacter* species
- *Escherichia coli*
- *Candida albicans*
- *Klebsiella pneumoniae*

Pathophysiology

The two key factors for the development of a VAP are:^{7,8}

- Bacterial colonisation of the aerodigestive tract
- Aspiration of contaminated secretion into the lower respiratory tract

This leads to the development of focal areas of bronchiolitis that can expand to more progressive bronchopneumonia, and with time, a complete lobar pneumonia.

Other important factors for the development of VAP are:

- Sources of pathogens that include healthcare devices, the environment (air, water, equipment, and fomites – including gowns, caps and stethoscopes), and commonly the transfer of microorganisms between the patient and staff or other patients (cross-infection).
- Inhalation or direct inoculation of pathogens into the lower airway, haematogenous spread from infected intravenous catheters, and bacterial translocation from the gastrointestinal tract lumen.
- Infected biofilm that is present in the endotracheal tube, with subsequent embolisation to distal airways, may be important in the pathogenesis of VAP.⁹

Risk factors

There are various ways to classify risk for developing VAP. From the point of view of preventing or reducing risk of VAP it can be viewed in terms of non-modifiable risk factors and modifiable risk factors.

Being aware of non modifiable risk factors will increase awareness of patient risk and these include:¹⁰

- Extremes of age
- Chronic lung disease (especially bronchitis, chronic obstructive pulmonary disease [COPD] and asthma)
- Abdominal or thoracic surgery
- Endotracheal intubation
- Duration of mechanical ventilation
- Immunosuppression
- Nasogastric tubes
- Prior antimicrobial use

The following category would then be modifiable risk factors that are clearly areas where prevention strategies should be aimed at and implemented. (These will be discussed in more detail in the last section of this article.) These factors can be summarised as follows: intubation, duration of mechanical ventilation, aspiration, body position (supine vs semi-recumbent), route of feeding, modulation of colonisation, stress ulcer prophylaxis, transfusions, and hyperglycaemia.

Diagnosis

The diagnosis of VAP is based on clinical evaluation and guided by the following criteria:

- New radiographic infiltrates
- Fever
- Leucocytosis

- Purulent tracheal secretions
- Microorganisms isolated from endotracheal aspirates
Plus
- Clinical assessment

However, many non-infectious diseases can manifest in the same way e.g. atelectasis, pulmonary oedema, Acute Respiratory Distress Syndrome (ARDS), pulmonary embolism and chemical aspiration. Polk et al have indicated that the use of these clinical criteria can be misleading.¹¹ The additional use of quantitative cultures by means of bronchoalveolar lavage (BAL) has been recommended as a more accurate means of diagnosing VAP.¹²

Preventative strategies

The *American Thoracic Society* revised their guidelines for the management of VAP and Hospital Acquired Pneumonia (HAP) in 2004 and these were subsequently published in 2005 (first version originally published 1996).¹³ The most important preventative strategies can be outlined as follows:

Intubation and mechanical ventilation

- Intubation and re-intubation should be avoided (increasing the risk for VAP 6 to 21 fold), if possible, as it increases the risk of VAP. Non-invasive ventilation (positive pressure ventilation through a specialised mask connected to a ventilator) should be used whenever possible in selected patients with respiratory failure.
- Orotracheal intubation and orogastric tubes are preferred over nasotracheal intubation and nasogastric tubes to prevent nosocomial sinusitis and to reduce the risk of VAP.
- Continuous aspiration of subglottic secretions can reduce the risk of early-onset VAP, and should be used.
- The endotracheal tube cuff pressure should be maintained at greater than 20 cm H₂O to prevent leakage of bacterial pathogens around the cuff into the lower respiratory tract.
- VAP can be related to colonisation of ventilator circuits – numerous trials have shown that the frequency of ventilator circuit change does not affect the incidence of VAP, but the accumulation of condensate however does lead to an increase in VAP. Therefore contaminated condensate should be carefully emptied from ventilator circuits and condensate should be prevented from entering either the endotracheal tube or inline medication nebulisers.
- Passive humidifiers or heat-moisture exchangers decrease ventilator circuit colonisation, but have not consistently reduced the incidence of VAP, and thus they cannot be regarded as a pneumonia prevention tool.
- Reduced duration of intubation and mechanical ventilation may prevent VAP and can be achieved by protocols to improve the use of sedation and to accelerate weaning. Daily interruption or lightening of sedation to avoid constant heavy sedation should be used and paralytic agents should be avoided where possible since these both can depress cough and thereby increase the risk of HAP.
- Maintaining adequate staffing levels in the ICU can reduce length of stay, improve infection control practices, and reduce duration of mechanical ventilation.¹⁴

Aspiration, body position, and enteral feeding

- Patients should be kept in the semi-recumbent position (30 to 45°) rather than supine to prevent aspiration, especially when receiving enteral feeding.
- Enteral nutrition is preferred over parenteral nutrition to reduce the risk of complications related to central intravenous catheters and to prevent reflux villous atrophy of the intestinal mucosa, that may increase the risk of bacterial translocation.

Modulation of colonisation: Oral antiseptics and antibiotics

- Routine prophylaxis of HAP with oral antibiotics (selective decontamination of the digestive tract or SDD), with or without systemic antibiotics, reduces the incidence of ICU-acquired VAP, has helped contain outbreaks of MDR bacteria, but is *not recommended for routine use*, especially in patients who may be colonised with MDR pathogens.
- In one study, prophylactic administration of systemic antibiotics for 24 hours at the time of emergent intubation has been demonstrated to prevent ICU-acquired HAP in patients with closed head injury, but its *routine use is not recommended* until more data become available.
- Modulation of oropharyngeal colonisation by the use of oral has prevented ICU-acquired HAP in selected patient populations such as those undergoing coronary bypass grafting, but its *routine use is not recommended* until more data become available.

Stress bleeding prophylaxis, transfusion, and hyperglycaemia

- *Stress ulcer prophylaxis*: Comparative data from randomised trials suggest a trend towards reduced VAP with sucralfate but there is a slightly higher rate of clinically significant gastric bleeding, compared with H₂ antagonists. If needed, stress bleeding prophylaxis with either H₂ antagonists or sucralfate is acceptable. Intravenous proton pump inhibitors can also be used and have been proven to be at least as effective.¹⁵
- *Transfusion* of red blood cell and other allogeneic blood products should be *restricted* to a transfusion trigger policy only; furthermore when transfusion is required *leukocyte-depleted red blood cell* transfusions can help to reduce VAP in selected patient populations. Standard triggers used currently will be a Hb of < 10 g/dL if coronary artery disease or active bleeding is present otherwise a Hb of < 7 g/dL would be viewed as a trigger for transfusion.^{16,17,18}
- *Intensive insulin therapy* (including non-diabetic patients) is recommended to maintain serum glucose levels between 80 and 110 mg/dl (4.4–6.1 mmol/l) in ICU patients to reduce nosocomial blood stream infections, duration of mechanical ventilation, ICU stay, morbidity, and mortality.¹⁹ However a blood glucose of less than 145 mg/dl (8.1 mmol/l) seems to be reasonable and decreases the risk of hypoglycaemia and aiming too low can be harmful (as has recently been proven).^{20,21,22}

General infection control measures are very important as well. Effective general strategies include strict infection control, alcohol-based hand disinfection, use of microbiologic surveillance with timely availability of data on local MDR pathogens, monitoring and early removal of invasive devices, and programmes to reduce or alter antibiotic-prescribing practices.^{23,24}

Apart from general infection control measures, a practical ten point summary for the implementation of preventive strategies for VAP can be suggested:

1. Using non-invasive ventilation (NIV) where possible. If a patient does definitely require invasive ventilation, orotracheal intubation and orogastric tubes are preferred.
2. Regular clearance of subglottic secretions is recommended.
3. Cuff pressure should be monitored frequently and kept above 20 cm H₂O.
4. Clearance of contaminated condensate in ventilator circuits is essential.
5. Frequent (daily) sedation interruption is recommended to facilitate a shorter ventilation period.
6. Patient should be positioned in a semi-recumbent position.
7. Enteral feeding should be started as soon as possible and be protocol driven.
8. Stress ulcer prophylaxis should be routinely used.
9. Transfusions should only be done where essential and according to specific parameters or ICU protocol.
10. Reasonable glucose control in the critically ill is of significant benefit aiming for a blood glucose level of < 145 mg/dl (< 8.1 mmol).

Conclusion

There are clearly non-modifiable risk factors for the development of VAP and VAP cannot be eliminated from everyday clinical care in the intensive care unit (ICU) setting. However, as outlined in this article, there are numerous practical measures that can be implemented and have been proven by clinical evidence to significantly reduce the risk of VAP.^{4,24} For these measures to be successful their implementation should be checked at regular intervals (at least daily) by the ICU team when taking care of the individual patient. Reduction of VAP risk will go a long way to reducing morbidity and mortality that is associated with this condition.

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