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Microbes and Infection 7 (2005) 275–278
Microbes and Infection
www.elsevier.com/locate/micinf

Current focus

Pneumonia in patients who require prolonged mechanical ventilation

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Available online 08 January 2005

Abstract

Nosocomial pneumonia is the most important infectious disease in patients who require prolonged mechanical ventilation. Understanding of the etiology helps to prevent ventilator-associated pneumonia (VAP). VAP can develop in four ways: by aspiration, inhalation, hematogenous spread and by contiguous spread. The two most common are aspiration from the oropharyngeal region and inhalation, usually from manipulation of tubing or infected equipment. VAP is prevented by hand-washing, keeping the head of the bed at 45 and, in some cases, by treating the surface bacteria which usually cause VAP. Sputum can be used for the diagnosis of VAP in most of these patients instead of invasive bronchoscopy. However, if the patients are critically ill, then bronchoscopy is used. Treatment in these patients depends on the bacteria. *Pseudomonas* is treated by two drugs (beta-lactam plus a quinolone or aminoglycoside), *Acinetobacteria* by ampicillin/sulbactam or carbapenam, extended-spectrum beta-lactam-producing bacteria by carbapenums, and *Staphylococcus* by vancomycin or linezolid.

Keywords: Ventilator-associated pneumonia; Nosocomial pneumonia; Prevention; Etiology; Diagnosis; Treatment; Mechanical ventilation

1. Introduction

Prolonged mechanical ventilation is defined as ventilation for more than 4 weeks [1]. These patients often develop a variety of infections including the respiratory tract, central lines, urinary tract infections, infection with *Clostridium difficile* and vancomycin-resistant enterococccus [1]. Respiratory infections associated with prolonged mechanical ventilation include sinusitis, bronchitis, and pneumonia. Ventilator-associated pneumonia (VAP) is the most serious nosocominal disease that occurs in or out of the intensive care unit, and the prevalence ranges from 9% to 70% [1,2]. In long-term ventilated patients, the national average is usually about 16 per 1000 ventilated days. The attributable mortality of VAP ranges from 13% to 55% [1,2]. VAP is often preventable, but when it does occur, it should be aggressively diagnosed and treated [3]. In this article, the major issues of pathogenesis, prevention, diagnosis, and treatment of VAP will be addressed. The difference between VAP of long and short term is addressed in Table 1.

2. Pathogenesis of VAP

There are only four routes that organisms can take to infect the lower respiratory tract. The appreciation of this and the knowledge of the pathogenesis of VAP can often lead to the prevention of this significant disease. These routes are inhalation, aspiration, hematogenous and contiguous spread. In general, inhalation and aspiration are the two major ways that the organisms infect the lower respiratory tract. Hematogenous and contiguous routes are not as important [1,4].

2.1. Inhalation

If respiratory equipment is contaminated with waterborne organisms, they will be introduced into the lower respiratory tract, causing pneumonia. The ventilator tubing is usually cold and dry, and when the humidified and heated air is passed through it, water will precipitate into the tubing itself. The condensate is often contaminated with organisms usually arising from the patient. If this condensate is then aspirated into the lungs, pneumonia will develop. The best way to handle this contaminated water in the tubing is to allow it to drain into the in-line cups and then suction it out. Hands must then be washed before touching the patient to prevent transmitting pathogenic bacteria. Changing the ventilator cir-

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doi:10.1016/j.micinf.2004.12.002

Table 1
A comparison of VAP in patients who are ventilated acutely versus those who require prolonged mechanical ventilation

Characteristic	VAP in patients who are ventilated acutely	VAP in patients requiring prolonged mechanical ventilation
Time on ventilation	Less than 4 weeks	More than 4 weeks
Endotracheal tube	Most patients	Few patients
Tracheostomy tube	Few patients	Most patients
Prevalence	9–70% patients	16 per 1000 ventilator days
Pathogenesis	Mostly aspiration	Mostly aspiration
Sinusitis	Common	Rare
Chlorhexidine/mucopiricin wash	No published studies	Decreases MRSA VAP
Trimethoprim sulfa prophylaxis	In burn patients, decreases MRSA VAP	No published studies
Raised head of bed prophylaxis of VAP	Yes	Yes
Hand-washing	Yes	Yes
Isolation of MRSA	Necessary	Not necessary if chlorhexidine protocol
Diagnosis	More invasive test needed (tracheal aspirate if quantitative cultures only)	Tracheal aspirate useful
ICU patients	Most	Few
Treatment with de-escalation therapy	Yes	Yes

cuit does not decrease the prevalence of VAP and, therefore, it should only be changed every week. Closed suction systems decrease environmental contamination and may also decrease the risk of VAP [1,4].

2.2. Aspiration of oropharyngeal secretions

Aspiration is the main route used by bacteria to invade the lower respiratory tract and cause VAP. The vocal cords are separated forcibly by the endotracheal tube during translaryngeal intubation. The patients cannot cough efficiently and maintain the upper airways. This pre-disposes aspiration of infected oropharyngeal organisms through the open vocal cords. Once aspirated, the secretions pool above the inflated endotracheal cuff. Mechanical ventilation causes changes in the cuff pressures, deforming it and allowing the secretions to be transported around it by capillary action. Over time, the endotracheal tube develops a biofilm on its inner surface. The secretions are aspirated into the distal air spaces, coughed up and infect this biofilm. Repeated suctioning and ventilation causes showers of infected material to disseminate throughout the dependent parts of the lungs, causing pneumonia. Early tracheotomy has been shown to decrease pneumonia, mortality and the time on mechanical ventilation. In general, patients on long-term mechanical ventilation already have a tracheotomy in place. Re-intubation is another cause of VAP and is based on the same pathogenesis [1,4].

2.3. How do the bacteria get to the oropharynx?

2.3.1. Oropharyngeal colonization

Only about 6% of normal people will have their oral pharynx colonized. The prevalence increases to 35% in hospitalized patients and to 75% in critically ill patients. Those patients who are intubated for a prolonged period of time will have colonization almost 100% of the time. Colonization in these patients is generally by hospital-acquired bacteria. Those patients who have their oropharynx colonized are more likely to develop pneumonia than those who do not [1,3,4].

Pseudomonas may be one of the few organisms which can colonize in the lower respiratory tract without first colonizing the oropharynx. Transcolonization is the term used to describe bacterial colonization of the oropharynx from contiguous structures like dental plaque, periodontal tissues, and sinuses. The bacteria which ultimately colonize the oropharynx come from the hands of health-care workers who transport bacteria from one patient to another. They may even transport bacteria from the patient's own rectum to their upper airways [1,4].

2.3.2. Sinusitis

The development of sinusitis is more common in patients on mechanical ventilation who are intubated with an endotracheal tube. Those on prolonged mechanical ventilation usually have tracheotomies, and therefore the prevalence of VAP is significantly reduced [1,4].

2.3.3. Gastric aspiration

Many workers believe that the stomach is a source of bacteria responsible for VAP. This is the so-called "gastropulmonary" hypothesis. Prevention of VAP using this hypothesis has included keeping the head of the bed at 45°, digestive decontamination, and special stress ulcer prophylaxis. Many of these methods have been shown not to decrease VAP, and many do not believe that gastrointestinal bacteria are the major cause of VAP [1,3,4]. There have been many problems with studies looking at this question [5].

2.4. How do we reconcile the seemingly disparate evidence?

The bacteria aspirated from the oropharynx are pivotal in the development of VAP. All the factors that cause regurgitation of stomach contents may also cause aspiration of the oropharyngeal bacteria as the stomach contents are aspirated into the lungs. Therefore, it is not only the stomach contents themselves which are aspirated, but also what is aspirated from the oropharynx with the stomach contents [4].