



Prevention and Treatment of Respiratory Associated Pneumonia in a Patient with Prolonged Artificial Lung Ventilation

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Received 29th Nov 2021,
Accepted 31st Dec 2021,
Online 21st Jan 2022

Abstract: This article provides information about treatment of pneumonia. While critically ill patients experience a life-threatening illness, they commonly contract ventilator-associated pneumonia. This nosocomial infection increases morbidity and likely morbidity as well as the cost of health care. This article reviews the literature with regard to diagnosis, treatment, and prevention. It provides conclusions that can be implemented in practice as well as an algorithm for the bedside clinician and also focuses on the controversies with regard to diagnostic tools and approaches, also treatments.

Key Word: treatment in patients, diagnostic tools, mechanical ventilation, nosocomial infection, morbidity.

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Introduction

Patients in the intensive care unit (ICU) are at risk for dying not only from their critical illness but also from secondary processes such as nosocomial infection. Pneumonia is the second most common nosocomial infection in critically ill patients, affecting 27% of all critically ill patients. (170). Eighty – six percent of nosocomial pneumonias are associated with mechanical ventilation and are termed ventilator-associated pneumonia. (VAP). Between 250,000 and 300,000 cases per year occur in the United States alone, which is an incidence rate of 5 to 10 cases per 1,000 hospital admissions (134,170).

The mortality attributable to VAP has been reported to range between 0 and 50 (10,41,43,96,161). Studies have provided different results when determining attributable mortality, in part because of very

different populations (less-acute trauma patients, acute respiratory distress syndrome [ARDS] patients, and medical and surgical ICU patients) and in part as a result of variances in appropriate empirical medical therapy during the initial 2 days. Furthermore, the organisms recovered have an impact on outcome, with higher mortality rates seen in VAP caused by *Pseudomonas aeruginosa*, *Acinetobacter* sp, and *Stenotrophomonas maltophilia*. Beyond mortality, the economics of VAP include increased ICU lengths of stays (LOS) (from 4 to 13 days), and incremental costs associated with VAP have been estimated at between \$5,000 and \$20,000 per diagnosis.

Ventilator-associated pneumonia is defined as pneumonia occurring more than 48 h after patients have been intubated and received mechanical ventilation. Diagnosing VAP requires a high clinical suspicion combined with bedside examination, radiographic examination, and microbiologic analysis of respiratory secretions. Aggressive surveillance is vital in understanding local factors leading to VAP and the microbiologic milieu of a given unit.

Judicious antibiotic usage is essential, as resistant organisms continue to plague intensive care units and critically ill patients. Simple nursing and respiratory therapy interventions for prevention should be adopted. Over the past several decades our understanding of VAP has grown significantly with regard to pathogenesis, risk factors, diagnostic testing, therapies, and prevention by modifying risk factors. This paper is deemed for the practicing actually, clinician in addressing diagnosis, useful treatment.

There are other clinical diagnosis. Ventilator-associated pneumonia is usually suspected when the individual develops a new or progressive infiltrate on chest radiograph, leukocytosis, and purulent tracheobronchial secretions. Unfortunately, and unlike for community-acquired pneumonia, accepted clinical criteria for pneumonia are of limited diagnostic value in definitely establishing the presence of VAP. In a postmodern study by Fabregas et al, when findings on histologic analysis and cultures of lung samples obtained immediately after death were used as references, a new and persistent infiltrate on chest radiograph plus two of criteria

When all three clinical variables were required for the diagnosis, the sensitivity declined further (23%); the use of a single variable resulted in a decrease in specificity (33%). The poor accuracy of clinical criteria for diagnosing VAP should not be surprising considering that purulent tracheobronchial secretions are invariably present in patients receiving prolonged mechanical ventilation and are seldom caused by pneumonia.

In addition, the systemic signs of pneumonia such as fever, tachycardia, and leukocytosis are nonspecific; they can be caused by any state that releases the cytokines interleukin-1, interleukin n-6, tumor necrosis factor alpha, and gamma interferon. Examples of such conditions include trauma, surgery, the fibroproliferative phase of ARDA, deep vein thrombosis, pulmonary embolism, and pulmonary infarction. Reasonable clinical criteria for the suspicion of VAP include a new and persistent or progressive radiographic infiltrate plus two of the following: temperature of > 38 or $< 36^{\circ}\text{C}$, blood leukocyte count of $> 10,000$ cells/ml or $< 5,000$ cells/ml, purulent tracheal secretions, and gas degradation.

The sensitivity of the clinical criteria for VAP outlined above is even lower in patients with ARDS, where it may be difficult to detect new radiographic infiltrates. In the setting of ARDS, Bell et al. reported a false-negative rate of 46% for the clinical diagnosis of VAP. Consequently, suspicion for VAP in the setting of ARDS should be high. The presence of even one of the clinical criteria for VAP, unexplained hemodynamic instability, or an unexplained deterioration in arterial blood gases should prompt consideration of diagnostic testing.

purulent sputum, a positive sputum culture, fever, and leukocytosis are present without a new lung infiltrate, the diagnosis of nosocomial tracheobronchitis has been associated with a longer ICU stay

and time on the ventilator, without increased mortality. In one randomized trial of intubated patients with community-acquired tracheobronchitis, antibiotic therapy resulted in a decreased incidence of subsequent pneumonia and mortality.

However, prospective, randomized, controlled trials are required before antibiotic therapy can be recommended for the routine treatment of nosocomial tracheobronchitis. Furthermore, differentiation of tracheobronchitis from pneumonia is dependent upon the radiograph, which in the ICU is portable and often of poor quality. Hence, the clinician should utilize a clinical pulmonary infection score (CPIS) (see below) to show. .

There are also radiologic diagnosis. While the portable chest radiograph still remains a mandatory component in the diagnosis of ventilated patients with suspected pneumonia, as with clinical criteria for diagnosing VAP, it too has problems with both sensitivity and specificity. Poor-quality films further compromise the accuracy of chest X rays. Although a normal chest radiograph makes VAP unlikely, in one study of surgical patients, 26% of opacities were detected by computed tomography (CT) scan but not by portable chest X ray.

In addition, asymmetric pulmonary infiltrates consistent with VAP can be caused by numerous noninfectious disorders, including atelectasis, chemical pneumonitis, asymmetric cardiac pulmonary edema, pulmonary embolism, cryptogenic organizing pneumonia, pulmonary contusion, pulmonary hemorrhage, drug reaction, and asymmetric ARDS. The overall radiographic specificity of a pulmonary opacity consistent with pneumonia is only 27 %

In CONCLUSION, because of their high specificity, certain chest radiograph findings can be useful in establishing the diagnosis of pneumonia when present. Based on several studies, including an autopsy study by Wunderink et al., these useful findings include rapid cavitation of the pulmonary infiltrate, especially if progressive; an air space process abutting a fissure (specificity , 96%); and an air bronchogram, especially if single (specificity, 96%). Unfortunately, although there are the most useful such radiographic abnormalities are very uncommon, or less learnt

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