

Ventilator-Associated Pneumonia (VAP)

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Editorial

Ventilator-associated pneumonia (VAP) is a pulmonary infection that occurs more than 48 hours after patients have been intubated and received mechanical ventilation. The incidence of VAP ranges from 6 to 52% and can reach 76% in some specific settings [1]. The incidence depends on several factors, the duration of mechanical ventilation, reintubation, supine position, advanced age and altered consciousness. The VAP group was classified into two groups, early-onset type (within 48-96 h) and late-onset type (>96 h) [2].

Ventilator-associated bacterial pneumonia is an important intensive care unit (ICU) acquired infection in mechanically ventilated patients. Early and correct diagnosis of VAP is difficult but is an urgent challenge for an optimal antibiotic treatment [3]. Diagnosing VAP requires a high clinical suspicion combined with bedside examination, radiographic examination, and microbiologic analysis of respiratory secretions. Pugin et al., [4] developed a composite clinical score, called the clinical pulmonary infection score (CPIS), based on six criteria: temperature, blood leukocyte count, volume and purulence of tracheal secretions, oxygenation, pulmonary radiography, and semiquantitative culture of tracheal aspirate. The score varied from 0 to 12. A CPIS of >6 had a sensitivity of 93% and a specificity of 100%. Samples from the respiratory tract were collected by bronchoscopy from pneumonic cases for quantitative microbiological examination in a specific, constant sequence and with the following thresholds: tracheobronchial secretion, 10^5 and 10^6 colony-forming units (CFU)/ml; protected specimen brush, 10^3 CFU/ml and bronchoalveolar lavage, 10^4 CFU/ml [3].

For respiratory infection to occur, at least one of the three following factors must be present, immunodeficiency in the host, an inoculum of microorganisms into the lower respiratory tract that is large enough to overwhelm the host's immune system, or a highly virulent organism. Many ICU patients have prevalent underlying factors associated with immunodeficiency, such as diabetes mellitus or glucocorticoid therapy; these factors increase the risk of infection [5]. The organisms recovered have an impact on outcome, with higher mortality rates seen in VAP caused by *Pseudomonas aeruginosa*, *Acinetobacter baumannii* and *Stenotrophomonas maltophilia* [6].

Montero et al., [7] reported imipenem as a drug of choice unless there is a very high level of carbapenems resistance. Rifampins are used either as monotherapy or in combination with imipenem or tobramycin against highly carbapenem-resistant *Acinetobacter* strains; colistin had little effect either singly or in combination with rifampin *in vitro*. Ampicillin/sulbactam and cilastin/imipenem have equivalent efficacy for *Acinetobacter* spp. Intravenous colistin therapy has better results in patients with multidrug resistant *Acinetobacter* spp. and *Pseudomonas* spp.

The mortality attributable to VAP has been reported to range between 0 and 50% [8-10]. However, it is not certain that pneumonia is responsible for death or whether fatal outcome is caused by other risk factors for death that exist before the onset of pneumonia [11]. Simple and effective preventive measures for VAP might include noninvasive mechanical ventilation, diligent respiratory care, hand hygiene, elevation of head, oral instead of nasal cannulation, minimization of sedation, institution of weaning protocols, proper antibiotics administration according to semiquantitative culture and antibiogram for respiratory secretions [12].

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