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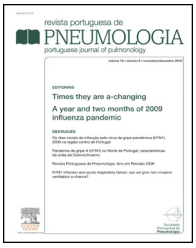

REVIEW

Prevention of ventilator-associated pneumonia

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Abstract Invasive mechanical ventilation (IMV) represents a risk factor for the development of ventilator-associated pneumonia (VAP), which develops at least 48 h after admission in patients ventilated through tracheostomy or endotracheal intubation. VAP is the most frequent intensive-care-unit (ICU)-acquired infection among patients receiving IMV. It contributes to an increase in hospital mortality, duration of MV and ICU and length of hospital stay. Therefore, it worsens the condition of the critical patient and increases the total cost of hospitalization. The introduction of preventive measures has become imperative, to ensure control and to reduce the incidence of VAP. Preventive measures focus on modifiable risk factors, mediated by non-pharmacological and pharmacological evidence based strategies recommended by guidelines. These measures are intended to reduce the risk associated with endotracheal intubation and to prevent microaspiration of pathogens to the lower airways.

PALAVRAS-CHAVE
Pneumonia associada ao ventilador;
Ventilação mecânica;
Prevenção

Prevenção de pneumonia associada ao uso do ventilador

Resumo A ventilação mecânica invasiva representa um fator de risco para o desenvolvimento da pneumonia associada ao ventilador (PAV), que se desenvolve 48 horas ou mais após a admissão hospitalar, em doentes ventilados através de traqueostomia ou intubação endotraqueal. A PAV é a infeção adquirida na unidade de cuidados intensivos (UCI) mais frequente entre os doentes submetidos a ventilação mecânica invasiva. Contribui para o aumento da mortalidade hospitalar, da duração da ventilação mecânica e do tempo de internamento na UCI e no hospital. Por conseguinte, agrava o estado de saúde do doente crítico e aumenta o custo total da hospitalização. A adoção de medidas preventivas é imprescindível, de modo a garantir o controlo e a diminuição da incidência da PAV. As medidas preventivas incidem sobre os fatores de risco modificáveis, sendo aplicadas estratégias não farmacológicas e farmacológicas baseadas na evidência e recomendadas por *guidelines*. As medidas preventivas têm como

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finalidade diminuir o risco associado à intubação endotraqueal e prevenir a microaspiração de microrganismos patogénicos para as vias aéreas inferiores.

Introduction

Invasive mechanical ventilation is a risk factor for the development of pneumonia,[1,2] being the ventilator-associated pneumonia (VAP) a public health problem. VAP is a hospital-acquired pneumonia which occurs in patients who were subjected to invasive mechanical ventilation, whether through tracheostomy or endotracheal intubation, at least 48 hours before the onset of infection and that were ventilated at the onset of the pneumonia.[3] This disease is also classified according to the time elapsed from the beginning of the mechanical ventilation (MV) to the onset of pneumonia; it is considered as early-onset if it occurs within 4 days of the start of MV, and late-onset if it occurs after 5 or more days of MV onset.[4,5] However, not all the studies consider early- and late-onset VAP within the same time range frame (Table 1).

VAP occurs primarily in intensive care units (ICU),[6] where the most debilitated patients are hospitalized, often requiring ventilatory support. It is estimated that from 8 to 28% of the patients receiving MV develop pneumonia, the risk is between 3 and 10 times higher compared to patients who do not receive MV.[7] Furthermore, almost 90% of episodes of nosocomial pneumonia registered in ICU occur during MV.[5] The predisposing risk factors for the development of the disease are innumerable and are divided into three groups as schematized in Table 2: related with the host, the hospitalization process and with drug therapy.

An episode of VAP may be due to a single pathogen or can have polymicrobial origin.[5,8,9] The etiology of VAP is quite diverse: bacterial, fungal and viral; fungi and viruses represent a greater role when the immune system of the patients is weakened.[8,10] The most common bacteria are listed in Table 3.

VAP is one of the major factors contributing to morbidity and mortality in the ICU.[11] A meta-analysis found that the average attributable mortality to VAP is 32.5% in the ICU,[12] supported by another study[13] that found 33%. VAP increases the ICU and hospital length of stay, as well as the time the patient requires ventilatory support.[13,14] This pathology is also responsible for more than half of the prescribed antibiotics in the ICU[5] and for the increased cost of hospital internment, registering a wide range of values for the average cost attributable to the disease (between 2089.13€ and >29431.70€).[14–18] Due to all this, the search for preventive measures in order to reduce these parameters, as well as to prevent the onset of the disease, has become imperative.[19,20]

Table 1 Different definitions of early and late-onset VAP.

Citation	Early-onset VAP	Late-onset VAP
Park, 2005[10]	≤4–7 days	>7 days
Olaechea et al., 2010[85]	≤7 days	>7 days
Erbay et al., 2004[16]	≤3–5 days	>3–5 days

Pathogenesis

There are several sources of VAP pathogens that can be classified as exogenous and endogenous in relation to the patient (Fig. 1). The exogenous sources are mostly from aerosols of the contaminated air, medical devices (humidifier, ventilatory circuit, catheter and bronchoscope), health professionals and other patients. The endogenous sources are represented by the oral, pharyngeal and gastric flora of the patient.[21,22]

Microorganisms reach the lower respiratory tract mainly by microaspiration of oropharyngeal secretions or secretions that are aspirated to the oropharynx through gastric reflux; and secondarily by direct extension of a contiguous infection, inhalation of contaminated aerosols or by hematogenous spread of microorganisms from other sites of infection.[21,23]

The defense mechanisms against lung infection in a healthy non smoker include: the anatomy of airways, cough reflex, mucus production, mucociliary clearance, lactoferrin, basement membrane and the immune system.[21,24] Not all defense mechanisms are operational in critically ill patients due to underlying diseases, sedative medication, poor nutrition and medical devices,[22] such as the endotracheal tube (ETT) which is used in the MV and compromises the cough reflex and the mucociliary clearance (ETT increases mucous secretion and stagnation of secretions) and causes lesions on the surface of the tracheal epithelium.[21,25] The ETT cuff prevents the aspiration of large volume secretions; however, it is not completely airtight, since there is the possibility of establishing microchannels between the tracheal mucous and the cuff when it is distended, which increases the probability of microaspiration of the accumulated secretions above the cuff (subglottic secretions) to the lower airways.[25] In addition, pathogens that reach the ETT cuff are able to colonize the interior of the tube, ensuring access to the distal airways with the aid of the inspiratory flow from the MV, establishing posteriorly the lung infection.[24] Previous surgeries and medication, particularly antibiotherapy, may also predispose the patient to the disease.[5]

Prevention of ventilator-associated pneumonia

VAP prevention is performed through pharmacological and non-pharmacological measures that mainly focus on modifiable risk factors. With this review our intention was to approach the most consensual preventive measures