

EDITORIAL:

VENTILATOR ASSOCIATED PNEUMONIA

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Ventilator Associated Pneumonia (VAP) is pneumonia occurring in a patient within 48 hours or more after intubation with an endotracheal tube or tracheostomy tube and which was not present before¹. It is also the most common and fatal infection of ICU². Early onset VAP occurs within 48 hours and late onset VAP beyond 48 hours of tracheal intubation. VAP increases length of ICU stay by 28%³ and each incidence of VAP results in manifold increase in financial burden.

Ventilator-associated pneumonia (VAP) continues to complicate the course of 8 to 28% of patients receiving mechanical ventilation (MV). In contrast to infections of more frequently involved organs (e.g., urinary tract and skin), for which mortality is low, (1 to 4%), the mortality rate for VAP ranges from 24 to 50% and can reach 76% in some specific settings or when lung infection is caused by high-risk pathogens.

Because of this large disease burden and the resultant attributable morbidity and mortality, there is great interest in accurately diagnosing, treating, and preventing this complication.

Despite the high incidence, diagnosis remains challenging because many conditions common to ICU patients like ARDS, sepsis, cardiac failure and lung atelectasis have similar clinical signs. More than 50% of patients clinically diagnosed with VAP do not have the disease whereas among 1/3rd of actual cases diagnosis is missed⁴. Unfortunately there is no clearly accepted gold standard for diagnosis of VAP. The efficacy of diagnostic and preventive strategies remains somewhat controversial. Diagnosis by invasive methods requires a considerable commitment of resources but can potentially reduce cost of care; however, mortality benefit from this approach has not been demonstrated.

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As such, in most institutions, VAP best diagnosed using traditional clinical criteria⁵.

Centres for disease control and prevention (CDC) national healthcare safety network has defined VAP based on following criteria. At least one of the following clinical findings, temperature > 38 deg C (with no other recognized cause) or Leucocytosis > 12000WCC/uL or leucopenia (<4000 WCC /uL) and for adults 70 years or older, altered mental status with no other recognisable cause can be suggestive Radiologically criteria includes 2 or more serial chest x-rays with at least one of the following; new or progressive and persistent infiltrate, consolidation or cavitation. Besides this new onset of purulent sputum, or change in character of sputum, or increased respiratory secretions, or increased suctioning requirements or new-onset or worsening cough, or dyspnoea or tachypnoea or rales or bronchial breath sounds or worsening gas exchange (eg. O₂ desaturations [PaO₂/FiO₂ ≤ 240], increased O₂ requirements, or increased ventilation demand) may be highly suggestive of VAP.

VAP that occurs within 48 hours after tracheal intubation is usually termed as early onset often resulting from aspiration, which complicates intubation process. VAP occurring after this period is late onset^{6, 7}. Early onset VAP is often due to antibiotic sensitive bacteria (eg oxacillin-sensitive *Staphylococcus aureus*, *Hemophilious influenza* and *Streptococcus pneumoniae*), whereas late onset VAP is frequently caused by antibiotic resistant pathogens (eg. oxacillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Acinetobacter* and *Enterobacter* species).

-In the current issue a study reported by Aneela Altaf et al was conducted in a medical ICU over a period of one year Microbiological diagnosis was established on specimens collected through deep tracheal aspirate. Forty four percent of ICU patients acquired VAP among them 77% had Gram negative infection, 20% having *Staphylococci* infection whereas 56% had polymicrobial infection. Multi drug resistance was found in 42% of

Acinobacter infection, while other gram negative organism showed variable susceptibility.

The pathogenesis of VAP usually requires that two important processes i.e. bacterial colonization of the aero-digestive tract and aspiration of contaminated secretions into the lower airway tract⁸. Therefore, the strategies to prevent VAP usually focus on reducing the burden of bacterial colonization in the aero-digestive tract, decreasing the incidence of aspiration or both.

The presence of invasive medical devices is an important contributor to the pathogenesis and development of VAP⁹. Many patients have nasogastric tubes that predispose them to gastric reflux and increase the potential for aspiration. Endotracheal tubes facilitate bacterial colonization of the tracheo-bronchial tree and lower airway aspiration of contaminated secretions through mucosal injury, pooling of contaminated secretions above the endotracheal tube cuff and elimination of the cough reflex. The ventilator circuit and the respiratory-therapy equipment may also contribute to the pathogenesis of VAP if they become contaminated with bacteria, which usually originate in the patient's secretions¹⁰. In ICU setting one of the microbiological criteria can also be used to diagnose VAP such as; positive growth in blood culture not related to another source of infection, or positive growth in culture of pleural fluid, or positive quantitative culture from bronchoalveolar lavage ($\geq 10^4$ colony forming units/ml) or protected specimen brushing ($\geq 10^3$ colony forming units/ml), or 5% or more of cells with intracellular bacteria on direct microscopic examination of Gram-stained bronchoalveolar lavage fluid, or histopathological evidence of pneumonia. Histological landmark of VAP is multifocal disease favoring dependant lung segments, often at different stages of development and severity with cultures growing heterogenous microbial flora.¹¹

The predominant organisms responsible for infection are *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Enterobacteriaceae*, but etiologic agents widely differ according to the population of patients in an intensive care unit, duration of hospital stay, prior antimicrobial therapy or reintubation^{12, 13, 14}. Because appropriate antimicrobial treatment of patients with VAP significantly improves

outcome, more rapid identification of infected patients and accurate selection of antimicrobial agents represent important clinical goals. When fiberoptic bronchoscopy is not available to physicians treating patients clinically suspected of having VAP, a simplified nonbronchoscopic diagnostic procedure is justified. Selection of the initial antimicrobial therapy should be based on predominant flora responsible for VAP at each institution, clinical setting, information provided by direct examination of pulmonary secretions, and intrinsic antibacterial activities of antimicrobial agents and their pharmacokinetic characteristics. Prompt administration of appropriate antibiotics seems to be the only intervention that alters outcome once the diagnosis is established. The routine use of prolonged courses of empirical therapy i.e. therapy not supported by results of clinical cultures should be avoided to minimize the subsequent development of antibiotic-resistant infections. Use of broad-spectrum antibiotics is also not recommended for the prevention of VAP because of increasing antibiotic resistance among subsequent hospital acquired infections. Targeted antibiotic therapy with appropriate dose of appropriate antibiotic is the sensible thing to do. Several strategies seem to reduce pneumonia incidence. Preventive measures include; elevation of the head of bed to 35-40 degrees, holding sedatives, gastric ulcer prophylaxis, appropriate humidification of inspired gas and routine oral hygiene as per local policy. In addition, the following also may contribute to the prevention of VAP; prolonged nasal intubation (more than 48hrs) should be avoided, endotracheal tubes with separate dorsal lumen above the cuff to suction pooled secretions from the subglottic space, and the pressure of the endotracheal tube cuff should be adequate to prevent the leakage of colonized subglottic secretions into the lower airway¹⁵.

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