Strategies to Prevent Ventilator-Associated Pneumonia in Neonates

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- Ventilator-associated pneumonia
- Health care-associated infection • Neonate

Ventilator-associated pneumonia (VAP) is defined by the Centers for Disease Control and Prevention (CDC) as an episode of pneumonia in a patient who requires a device to assist or control respiration through a tracheostomy or endotracheal tube within 48 hours before the onset of the infection.[1] Health care-associated infections have a large impact on neonatal morbidity, survival, hospital costs, and length of stay.[2,3] VAP is a common cause and accounts for 6.8% to 32.2% of health care-acquired infections among neonates.[4–8] This article summarizes epidemiology, suspected pathogenesis, diagnosis, and strategies to prevent VAP in neonates.

EPIDEMIOLOGY

The exact rate of neonatal VAP is difficult to establish, because radiographic identification of pneumonia is difficult, especially among neonates with significant underlying lung disease, and diagnostic procedures commonly used in adults are rarely used in the neonatal intensive care unit (NICU). Differences in study methodology and case mix also influence the reported incidence of neonatal VAP.[9] National Nosocomial Infections Surveillance system data from 2004 showed that VAP rates for neonates weighing less than 1000 g ranged from 2.4 to 8.5 episodes per 1000 ventilator days.[10] In a cross-sectional study of 12 NICUs in children's hospitals, the incidence of VAP among neonates weighing less than 1000 g was 0 to 21.2 (median 3.5) per 1000 ventilator days.[11] Other investigators have reported rates varying from 12.5 to 52 infections per 1000 ventilator days.[4,12–16] Differences in study design and the case mix are likely responsible for the wide range of reported rates.

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Developmental abnormalities in the neonate's immune system including greater permeability of the skin and mucous membranes, decreased complement activity, and lower levels of immunoglobulins increase the susceptibility to health care-acquired infections. In a cohort of 742 neonates,[4] low birth weight (odds ratio [OR] 1.37; 95% confidence interval [CI], 1.01, 1.85] and mechanical ventilation (OR 9.7; 95% CI, 4.6, 20.4) increased pneumonia risk. Intravenous antibiotics were protective (OR 0.37; 95% CI, 0.21, 0.64). In another cohort of 229 ventilated neonates weighing 2000 g or less, VAP was more likely to occur in neonates who had a previous bloodstream infection (OR 3.5; 95% CI, 1.2, 10.8).[17] Organisms responsible for bloodstream infections were different from those causing VAP, suggesting that bloodstream infections may serve as a surrogate for severity of illness in the population. Although prolonged intubation before the episode of pneumonia did not reach statistical significance in this cohort, it was associated with VAP in the cohort reported by Yuan and colleagues.[12] Opiate treatment for sedation (OR 3.8; 95% CI, 1.8, 8.5), frequent endotracheal suctioning (OR 3.5; 95% CI 1.6, 7.4), and reintubation (OR 5.3; 95% CI, 2.0, 14.0) increased VAP risk in this study of ventilated neonates. Pneumonia is less common in neonates treated with nasal continuous positive airway pressure (NCPAP) when compared with those intubated on mechanical ventilation (12.5/1000 ventilator days vs 1.9/1000 NCPAP days, $P = .04$).[13] Finally, NICU design and staffing may affect VAP rates. Neonatal VAP decreased significantly when a NICU was moved from a crowded space to a larger unit with 50% more staffing.[18]

PATHOGENESIS

VAP occurs when bacterial, fungal, or viral pathogens enter the normally sterile lower respiratory tract and lung parenchyma. Under normal circumstances, anatomic barriers, cough reflexes, tracheobronchial secretions, mucociliary lining, cell-mediated and humoral immunity, and the phagocytic system of the alveolar macrophages and neutrophils protect the lung parenchyma from infection. If these defenses are impaired, absent, or overcome by a high inoculum of organisms or those of unusual virulence, pneumonitis ensues.

Microorganisms responsible for VAP can originate from endogenous or exogenous sources (**Figs. 1** and **2**). Oropharyngeal or tracheobronchial colonization (endogenous source) with pathogenic bacteria begins with the adherence of microorganisms to the epithelial cells of the respiratory tract. Organisms causing VAP are often noted in the posterior pharynx.[19,20] Several investigators have highlighted[21–25] the role of pharyngeal and subglottic secretions in the development of VAP in adults. Contaminated oral and gastric secretions can pool above the cuff of the endotracheal tube in adult patients and gain access to the lower aspect of the respiratory tract by leaking around the cuff. Neonates are likely at greater risk for such aspiration of contaminated oral secretions, because endotracheal tubes used to ventilate neonates are not cuffed. Gram-positive organisms in the mouth colonize the trachea and endotracheal tubes within the first 48 hours of mechanical ventilation.[26,27] Gram-negative bacilli begin colonizing the endotracheal tube and trachea after 48 hours of respiratory support. VAP early after intubation tends to be more benign when compared with episodes that occur later in the hospital stay when gram-negative organisms begin to colonize the endotracheal tube.[27–29]

Support for the role of oropharyngeal colonization and subsequent tracheal colonization in the pathogenesis of VAP in neonates comes from studies showing a role of positioning in the acquisition of airway colonization with potential pathogens. Elevation of the head of the bed may reduce the risk of aspiration of contaminated