Introduction

Hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) remain important causes of morbidity and mortality despite advances in antimicrobial therapy, better supportive care modalities, and the use of a wide range of preventive measures (Craven et al. 1986, ATS 1996, Niederman 1996).

HAP is defined as pneumonia that occurs 48 hours or more after admission and that was not being incubated at the time of admission (Craven et al. 1986, Niederman 1996). HAP may be managed in a hospital ward or in the intensive care unit (ICU) when the illness is more severe. VAP is pneumonia that arises more than 48 hours after endotracheal intubation (Craven et al. 1986). Although not included in this definition, some patients may require intubation after developing severe HAP and should be managed in the same way as patients with VAP. Because most of the current data have been collected from patients with VAP, and microbiologic data from nonintubated patients may be less accurate, most of our information is from those with VAP, but by extrapolation can be applied to all patients with HAP.

Epidemiology

HAP accounts for up to 25% of all ICU infections and for more than 50% of all antibiotics prescribed (Richards et al. 1999). VAP occurs in between 9% and 27% of all intubated patients (Chastre and Fagon 2002, Rello et al. 2002). In ICU patients, nearly 90% of the episodes of HAP occur during mechanical ventilation.

Time of onset of pneumonia is an important epidemiologic variable and a major risk factor for specific pathogens and different outcomes in patients with HAP and VAP. Early onset HAP and VAP, defined as occurring within the first 4–5 days of hospitalization, usually have a better prognosis, and are more likely to be caused by antibiotic-sensitive bacteria. Late-onset HAP and VAP (5 days or more) are more likely to be caused by multidrug-resistant (MDR) pathogens, and are associated with increased patient mortality and morbidity. However, patients
with early onset HAP who have received antibiotics or been hospitalized within the previous 90 days are at greater risk of colonization and infection with MDR pathogens and should be treated in the same way as patients with late-onset HAP or VAP (Trouillet et al. 1998).

The crude mortality rate for HAP may be as high as 30% to 70%, but many of these critically ill patients with HAP die of their underlying disease rather than of pneumonia. Mortality related to HAP—attributable mortality—has been estimated to be between 33% and 50% in several case-matching studies of VAP.

Diagnosis

Diagnostic procedures are requested for two purposes: to define whether pneumonia is the explanation for a constellation of new signs and symptoms, and to determine the etiologic pathogen when pneumonia is present. Unfortunately, currently available tools cannot always reliably provide this information.

HAP is suspected if the patient has a radiographic infiltrate that is new or progressive and clinical findings suggesting infection (including new onset of fever, purulent sputum, leukocytosis, and decline in oxygenation). When fever, leukocytosis, purulent sputum, and a positive culture of a sputum or tracheal aspirate are present without a new lung infiltrate, a diagnosis of nosocomial tracheobronchitis should be considered. The accuracy of the clinical diagnosis of VAP has been investigated using autopsy findings or quantitative cultures of either protected specimen brush (PSB) or bronchoalveolar lavage (BAL) samples as the standard for comparison. The presence of chest infiltrates, plus two of three clinical criteria (fever, purulent sputum, leukocytosis) resulted in 69% sensitivity and 75% specificity (Fabregas et al. 1999).

Although these criteria should lead us to suspect HAP or VAP, confirmation of the presence of pneumonia is much more difficult, and clinical parameters cannot be used to define the microbiologic etiology of pneumonia. The etiologic diagnosis generally requires a lower respiratory tract culture, but only rarely can it be made from blood or pleural fluid cultures. Respiratory tract cultures include endotracheal aspirates, BAL or PSB specimens. Overall, the sensitivity of blood cultures is less than 25%, and when positive, the organisms may frequently originate from an extrapulmonary source. Although an etiologic diagnosis is made from a respiratory tract culture, colonization of the trachea precedes development of pneumonia in almost all cases of VAP, and thus a positive culture cannot always distinguish a pathogen from a colonizing organism. However, a sterile culture from the lower respiratory tract of an intubated patient, in the absence of a recent change in antibiotic therapy, is strong evidence that bacterial pneumonia is not present, and an extrapulmonary site of infection should be considered (Souweine et al. 1998). In addition, the absence of MDR microorganisms from any lower respiratory specimen in intubated patients, with no change in antibiotics during the previous 72 hours, is strong evidence that they are not the causative pathogen.