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The Diagnosis of Nosocomial Pneumonia

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Nosocomial pneumonia (NP) is a controversial and challenging topic. National University Hospital describes their experience in this issue of the Journal[1]. In their medical intensive care unit, they managed 24 patients with nosocomial or hospital acquired pneumonia (HAP) over a six-month period. Of these patients, 11 had ventilator-associated pneumonia (VAP). A microbiological diagnosis was established using expectorated sputum or endotracheal aspirates (EA). Quantitative cultures were not performed. There was no information on the length of stay or severity of illness, which would have better defined their study population.

They found a prevalence rate of 17% for HAP and 12% for VAP. Of their 11 patients with VAP, 9 died but only 1 death was directly attributed to VAP. The others died of their primary illness with and not because of pneumonia.

All patients with VAP and HAP had specimens sent for microbiology. MRSA, *Pseudomonas* or polymicrobial growth were the most frequent findings in patients with VAP. Patients with HAP had MRSA or *Klebsiella* isolated frequently and 27% of cases had no bacteria isolated. None of these patients had a positive blood culture. This could help in diagnosing a pneumonic process if both blood and sputum or EA were positive for the same organism.

In contrast, specimens were not obtained in 41% of their patients identified as having no pneumonia. In this group of patients, 44% had no bacteria isolated. In the 15% of patients with no pneumonia but positive cultures, *Klebsiella* and *Acinetobacter* were the most frequent organisms isolated.

This brings us to the controversial issues in the diagnosis of NP or VAP. The International Consensus Conference and American Thoracic Society have defined this group of patients so that studies can have a common enrolment criteria[2,3].

The first issue is the causes of fever and pulmonary infiltrates in patients with a clinical diagnosis of VAP. Meduri, in his classic study, reported that VAP was confirmed in only 42% of patients with a clinical diagnosis of VAP[4]. Their study protocol enrolled patients with clinical diagnosis of VAP and subjected them to an exhaustive protocol including bronchoscopy, sinus CT scans, evaluations for drug fever, atelectasis and thromboembolism and in selected cases, targeted studies like abdominal radiology and lumbar punctures.

The second issue is how to obtain specimens in patients with NP. The standardisation of bronchoscopic techniques to investigate VAP was recommended[2]. However in patients who are not intubated, it would be challenging to obtain uncontaminated specimens. Realising that the upper aerodigestive tract in these patients becomes colonised by Gram negative organisms, it then becomes difficult to interpret the expectorated sputum specimens obtained. The current consensus in diagnostic studies for retrieval of airway secretions for patients with NP can be summarised as follows[3].

1. For non-intubated patients, expectorated sputum is neither sensitive nor specific for pyogenic organisms.
2. In intubated patients, non-quantitative EA is sensitive but not specific. Specificity can be improved by performing quantitative cultures.
3. In intubated patients, bronchoscopic bronchoalveolar lavage (BAL) or protected specimen brush (PSB) gives better sensitivity and specificity provided it is done with rigorous adherence to technique, in the absence of prior antibiotic therapy and processed quantitatively.

The third issue is interpretation of the results of diagnostic testing and the final issue explores the impact on mortality. The results of 2 recent studies may lead us to reconsider our approach. Wermert et al were able to show that quantitative EA may be the best way to identify the causative organisms in nosocomial pneumonia[5]. They used a pig model of post-obstructive pneumonia comparing EA, PSB and BAL with lung biopsies. Half the animals were on antibiotic therapy and the second group on none. They were able to study VAP in a manner not confounded by prior antibiotic therapy or lack of histologic confirmation of pneumonia. The clinical implications of this study are important given the 24-hour availability of EA and the cost savings with the elimination of bronchoscopic techniques.

Sanchez-Nieto, in a landmark study in VAP, demonstrated no differences in mortality when comparing invasive and non-invasive culture sampling in the management of patients with VAP[6]. Briefly, 51 consecutive patients with VAP were randomised

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to bronchoscopic PSB/BAL and quantitative EA or to quantitative EA only. They noted that bronchoscopy led to more frequent antibiotic changes with no differences in mortality. The EA did not miss any organism picked up by PSB or BAL.

What is ahead in the future? We can expect to see more studies attempting to validate the findings of Wermert and Sanchez-Nieto. Perhaps the local institutions can move away from the present qualitative or semiquantitative analysis of EA and perform quantitative cultures instead. With an acceptable method of identifying the pathogens in NP or VAP, we can better describe our local epidemiology. Whether this will improve outcome in NP or VAP is something that is less certain.

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