STROKE-ASSOCIATED PNEUMONIA: THE NEED FOR STRICTER CASE DEFINITIONS

Dear Sir,

I read with interest the article "Stroke-associated pneumonia: microbiological data and outcome", wherein the authors describe the demographical, laboratory, radiological, microbiological data and outcome of patients with stroke-associated pneumonia[1]. However, there are a number of methodological limitations associated with the study which need further discussion.

Firstly, and the major point is regarding the diagnosis of pneumonia. The diagnosis of pneumonia is suspected if the patient has an infiltrate on chest radiograph that is new or progressive, along with clinical findings suggesting infection, which include the 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 present without a new pulmonary infiltrate, the diagnosis of nosocomial tracheobronchitis should be considered[2]. In fact, if we follow this criteria, the majority (75%) of the patients described in the current study do not fit into pneumonia, but will fit into the classification of nosocomial tracheobronchitis. Moreover as per CDC criteria which the authors have used to define pneumonia, the presence of radiographical infiltrates is mandatory for the diagnosis of pneumonia[3]. This point is further represented by the fact, that on a multivariate analysis in this study, the presence of infiltrates was an independent factor for mortality signifying that these were actually the patients who had pneumonia and were at an increased risk of dying.

Secondly, the authors do not state the number of patients who were mechanically ventilated, the method used to retrieve tracheal specimens, and the culture technique followed (qualitative versus quantitative). It is well known that microscopical evaluation and qualitative cultures of tracheal secretions and/or expectorated sputum are not representative of the lower respiratory tract in patients clinically suspected of having pneumonia, because the upper respiratory tract of most patients in the hospital is colonised with potential pulmonary pathogens, whether or not parenchymal pulmonary infection is present[4].

Thirdly, the authors have divided patients into two groups at 48 hours based on the assumption that pneumonia which develops early in the course is community-acquired, while that which develops late is usually hospital-acquired. Although logically correct, the authors also need to give us the duration of stay in other centres or healthcare facilities prior to admission at their hospital. In fact, the new American Thoracic Society/Infectious Disease Society of America guidelines for hospital-acquired pneumonia have recognised that stay in another healthcare facility is a risk factor for acquisition of multi-drug resistant pathogens and terms this category as healthcare- associated pneumonia[2]. This may explain the similarity of organisms in the two groups in the present study.

Finally, the authors state that on a multivariate analysis, presence of tracheal secretions were an independent factor for death but the authors failed to adjust it for other significant factors that affect hospital outcomes such as requirement of mechanical ventilation, renal failure, and baseline disease severity (as assessed by APACHE II scores)[5]. This makes the conclusions of the multivariate analysis clinically invalid as the factors included in the logistic regression model themselves are incomplete.

Yours sincerely,

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