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STROKE-ASSOCIATED PNEUMONIA: THE NEED FOR STRICTER CASE DEFINITIONS

Dear Sir,

We are thankful to Dr Agarwal who gave thoughtful comments, and raised some very important and valid concerns. According to the CDC definition of pneumonia, the patient must meet one of the two criteria, namely: (a) rales or dullness on chest percussion on examination and either new onset purulent sputum and/or change in its character, and/or positive blood culture or isolation of pathogen from specimen obtained by transtracheal space, brochial brushing or biopsy; (b) chest radiographs showing new or progressive infiltrate, consolidation, cavitation or pleural effusion and either new onset purulent sputum and/or change in its character, and/or positive blood culture or isolation of pathogen from specimen obtained by transtracheal space, bronchial brushing or biopsy and/or isolation of virus or viral antigen detection from respiratory secretions and/or diagnostic single antibody titre (IgM) or fourfold increase in sera (IgG) for pathogen and/or histopathological evidence of pneumonia[1]. All of our patients had abnormal chest examination, purulent sputum and were febrile and they met the first criterion of CDC definition.

We agree that traceobronchitis is a very pertinent and important diagnostic consideration. Although it is characterised by purulent secretions, fever, leukocytosis but does not cause rales or dullness on percussion on physical examination of chest. Furthermore, the low yield of chest radiographs in stroke-associated pneumonia is well reported. Hilker et al reported a yield of 69% in their cohort[2]. We assume that even lower yield in our cohort is due to short time lag between symptom onset and getting chest radiographs. However, we do not have sufficient data owing to the retrospective nature of our study to address the issue. We did not suggest an effect of chest radiogaphs infiltrates on outcome. Although length of stay was increased in patients with chest radiogaphical infiltrates, the mortality among radiograph-positive and radiograph-negative patients was not different.

The tracheal specimens were retrieved through a large suction catheter after saline nebulisation and chest percussion. The cultures were qualitative and we agree that positive cultures may be the result of colonisation. Colonisation of the upper airway with normal or nosocomial flora is always a matter of concern but such colonisation would not occur before 48-72 hours. The likelihood of colonisation in our cohort is low, as two-thirds of our patients had pneumonia within 48 hours and 22 out of 39 patients with positive cultures had pneumonia onset within 48 hours.

Transfer from another healthcare facility is a very valid point of concern. As only two patients in our cohort were transferred from another healthcare facility, healthcare-associated pneumonia is not a valid explanation of similarity of microbiological data between patients who manifested within 48 hours and those after 48 hours. Similarity of organisms between two groups in our study remains a dilemma. Possible explanations include contamination of probes, failure to immediately culture, and higher prevalence of *P. aureginosa* and *S. pneumonae* in our community.

Stroke-associated pneumonia significantly increases mortality of stroke patients and prolongs their hospital stay. Predictors of death in stroke-associated pneumonia are not well established.

We did not found any predictor of mortality in our cohort. However, chest radiographical infiltrates and positive cultures were associated with prolonged hospital stay. We excluded patients on mechanical ventilation but other variables like baseline functional status might have contributed to prolonged length of stay. Further prospective studies are warranted to resolve some of the unanswered queries.

Yours sincerely,

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