LETTER TO THE EDITOR

H1N1 influenza pneumonia and bacterial coinfection

ABSTRACT

The model described by Bewick et al seems to be able to distinguish between H1N1 influenza-related pneumonia and non-H1N1 community-acquired pneumonia (CAP) based on five criteria. However, bacterial infection in the influenza group has not been accurately excluded. Therefore, this model could misidentify these patients and lead to an inappropriate treatment. We conducted a prospective observational study to compare mixed pneumonia versus viral pneumonia. In the mixed pneumonia group, patients were older, had higher levels of procalcitonin and higher scores of severity. In our cohort, the model proposed by Bewick et al would not identify patients with coinfection.

Bewick et al recently published a model that identifies H1N1 influenza-related pneumonia based on five criteria.

The model seems to be able to distinguish between H1N1 influenza-related pneumonia and non-H1N1 community-acquired pneumonia (CAP). However, in the H1N1 influenza-related pneumonia cohort, there is no available information about the diagnostic testing procedures applied to identify bacterial infections associated with influenza.

Mixed infection due to the influenza virus and bacterial pathogens has been well described in the pandemics that occurred in the last century. In fact, in the last pandemic period, the incidence of bacterial infection in association with the 2009 H1N1 influenza was up to 20%. Remarkably, this percentage is probably an underestimate of the real figure. There are important methodological limitations in the pandemic reports, mainly, bacterial diagnostic tests were not performed in all patients and most patients received antibiotics close to the time of culture collection.

Bewick et al recognise that C reactive protein levels and leukocyte counts are affected by bacterial infections. It has been previously reported that clinical presentation, severity and outcome differs between pure viral pneumonia and coinfected patients. It is possible that the accuracy of the present model could be lower in coinfected patients and therefore it could misidentify patients with bacterial and influenza infections. It is common practice to treat with antiviral drugs and antibiotics those patients with CAP even when only influenza has been identified. However, in the group of patients with viral and bacterial infection, a lower sensitivity to detect influenza with the reported model could result in a delay in the initiation of antiviral treatment. This fact is crucial, as early antiviral treatment in severely ill patients with pneumonia has been associated with shorter length of stay, duration of ventilation and better survival rates.

We have recently conducted a prospective observational study of patients with CAP. The aim was to determine the aetiology of CAP among patients admitted to hospital and to compare the clinical and laboratory features of patients with mixed pneumonia (bacterial and viral pneumonia) versus those with viral pneumonia. Mixed pneumonia and viral pneumonia were diagnosed in 25 and 22 patients, respectively. Patients with mixed pneumonia were older (74 vs 56 years, p<0.001), had higher levels of procalcitonin (5.5 vs 0.8 ng/ml, p=0.03) and higher scores of severity indices. In this cohort, the diagnostic prediction model proposed by Bewick et al, probably, would not identify patients with coinfection.

REFERENCES

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Thorax published online October 12, 2011

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