LETTERS TO THE EDITOR

Authors' response

The H1N1 pneumonia cohort studied was a subset of a much larger cohort of adults hospitalised in the UK with confirmed pandemic influenza A/H1N1 2009 infection (FLU-CIN cohort, n=1046); part of that cohort has been described elsewhere.[1] The depth and breadth of bacteriological testing of patients recruited into FLU-CIN was at the discretion of attending physicians; only 3 of 1046 patients had evidence of bacterial co-infection recorded, probably an underestimate of the true burden of bacterial co-infection. Patients with identified bacterial co-infection were similar in age to patients without co-infection (mean age 27.0 (SD 13.1) years vs 39.5 (SD 16.4) years). The inclusion of patients with bacterial co-infection in the cohort of patients with H1N1 pneumonia would be expected to *reduce* any differences between the two study cohorts if patients with co-infection are indeed clinically distinguishable.

We are not aware of any publications arising from the 2009 pandemic demonstrating that patients with H1N1 pneumonia and bacterial co-infection can be reliably differentiated from patients without co-infection on clinical or demographic grounds alone. Some groups have examined the role of procalcitonin in this regard,[2] and ongoing investigations may provide additional information. Conclusions from studies of viruses other than H1N1/09 should be interpreted cautiously with regard to H1N1/09 disease patterns and vice versa.

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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 vs viral pneumonia. In the mixed pneumonia group patients were older, had higher levels of procalcitonine and higher scores of severity. In our cohort the model proposed by Bewick *et al* would not identify patients with coinfection.

Bewick *et al*[1] 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.[2] In fact, in the last pandemic period, the incidence of bacterial infection in association with the 2009 H1N1 influenza was up to 20%.[3] 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.[4] 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 sensibility 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.[5]

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.

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Contributors All the authors contributed equally to the conception and design of the study. ARP, ASGB, MEVB and SBG collected data. BBL analysed the data. All the authors were involved in interpretation of the results. VPG and ECS drafted the manuscript, which has been read and approved by all the authors.

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