

LETTER

Non-invasive ventilation in acute respiratory failure related to 2009 pandemic Influenza A/H1N1 virus infection

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See related research by Rello et al., http://ccforum.com/content/13/5/R148

Non-invasive ventilation (NIV) is considered first-line intervention for different causes of acute respiratory failure [1]. However, Rello and colleagues [2] show high rates of NIV failure in pandemic Influenza A/H1N1 virus infection (PH1N1).

We describe a patient with PH1N1 in whom NIV was effective. A 53-year-old male was admitted in November 2009 with cough, dyspnea, and hemoptysis. His temperature was 38.9°C, he was tachypneic, with diffuse rhonchi and bilateral crackles, and oxygen saturation was 96% (4 L/min oxygen). Arterial partial pressure of oxygen (PaO₂) and arterial partial pressure of carbon dioxide (PaCO₂) were 76 and 23 mm Hg, respectively. Creatine kinase (2,278 U/L) and brain natriuretic peptide (3,544 pg/ mL) were increased. Acute myocardial infarction was excluded. Chest x-ray showed bilateral interstitial infiltrates and cardiomegaly. Echocardiogram showed severe left ventricular systolic dysfunction. PH1N1 pneumonia was suspected, and oseltamivir was administered in

association with antibiotics and diuretics. On day 2, a nasopharyngeal swab was positive for PH1N1. The patient was subsequently transferred to a negativepressure ward. He was still tachypneic, with basal crackles and a PaO₂/fraction of inspired oxygen (FiO₂) ratio of 246. NIV (BiPAP Vision; Philips Respironics, Murrysville, PA, USA) through an oro-nasal mask in bilevel positive airway pressure mode (inspiratory positive airway pressure [IPAP] = 16 cm H₂O, expiratory positive airway pressure [EPAP] = 8 cm H₂O) was started. Due to patient preference, the mode was changed to continuous positive airway pressure (CPAP) at 10 cm H₂O and an FiO₂ of 25%. After 1 hour, PaO₂/FiO₂ increased to 364, and CPAP was stopped after 12 hours.

Recently, Djibré and colleagues [3] demonstrated the effectiveness of NIV in acute respiratory distress syndrome related to PH1N1 pneumonia. Our case further supports its role in a hypoxemic patient with cardiogenic pulmonary edema and PH1N1 pneumonitis.

Authors' response

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We appreciate the interest from Winck and Marinho in our article and their insightful observations regarding non-invasive ventilation (NIV) in severe influenza A (H1N1)v. The use of NIV in hypoxemic respiratory failure is controversial, and the etiology of hypoxemia appears to be an important determinant of its success. A metaanalysis [4] suggests that non-invasive positive-pressure ventilation does not decrease the need for intubation, so

there is not enough evidence to support its use in acute respiratory distress syndrome. Our experience [2] is consistent with other reports [5,6]; 25% to 30% of patients were non-invasively ventilated on admission, but 70% to 85% of these patients required subsequent intubation and invasive ventilation. There are only a few patients with H1N1-related respiratory failure who seem to benefit from NIV alone, so it should be reserved for patients with milder disease. Guidelines endorsed by the European Respiratory Society and European Society of Intensive Care Medicine [7] conclude that, as a general rule, NIV not be recommended as an alternative to invasive ventilation in patients affected by H1N1. In spite of this, selected patients with hypoxemia and additional cardiac compromise (severe left ventricular systolic dysfunction)

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or presenting with exacerbation of chronic obstructive pulmonary disease might benefit from this alternative therapy as it has been reported.

Abbreviations

CPAP, continuous positive airway pressure; FiO₂, fraction of inspired oxygen; NIV, non-invasive ventilation; PaO₂, arterial partial pressure of oxygen; PH1N1, pandemic Influenza A/H1N1 virus infection.

Competing interests

The authors declare that they have no competing interests.

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