CASE REPORT

Failure of NIV in acute asthma: case report and a word of caution

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Noninvasive ventilation (NIV) is the provision of ventilatory support without the need for an invasive airway, and has revolutionized the management of patients with diverse forms of respiratory failure. The advantages of NIV include improved patient comfort and reduced need for sedation, while avoiding the complications of endotracheal intubation, including upper airway trauma, sinusitis, otitis, and nosocomial pneumonia. In selected patients, NIV has also been shown to improve survival. The role of NIV in acute severe asthma is at best controversial. In this case report, we describe a patient with acute severe asthma who was initially managed and failed with NIV, and was successfully managed with invasive ventilation. We also review the pathophysiological mechanisms of benefit of NIV in acute severe asthma, and the current literature on the use of NIV in acute asthma. In conclusion, a trial of NIV in acute asthma may be justified in carefully selected and monitored patients who do not respond to initial medical therapy. However, as its role is not clear and as the condition of an asthmatic patient may deteriorate abruptly, extreme caution is advisable to recognize failure of NIV as in the case presented here. Facilities for immediate endotracheal intubation and next level of treatment should be readily available.

A 50 year old woman, a known asthmatic for the past 30 years, presented to the emergency department with a 2 day history of worsening breathlessness and cough. There was no fever, expectoration, chest pain, haemoptysis, or limb swelling. On examination, the patient was conscious and afebrile. She was diaphoretic and could not speak in complete sentences. Pulse rate was 126 beats/min, and blood pressure was 150/94 mm Hg with a pulsus paradoxus of 25 mm Hg. Respiratory rate was 40 breaths/min with accessory muscles of respiration in use. Chest auscultation revealed bilateral polyphonic inspiratory and expiratory wheeze. The remaining physical examination was unremarkable.

Oxygen saturation was 88% by pulse oximetry. Arterial blood gas showed type 1 respiratory failure (O_2 3 l/min, pH 7.45, PaO_2 8.4 kPa, $PaCO_2$ 4.9 kPa, HCO_3^- 26 mEq/l). Supplemental oxygen, nebulised salbutamol 5 mg and ipratropium 250 µg, intravenous methyl prednisolone 40 mg, deep venous thrombosis, and stress ulcer prophylaxis were administered. After 30 minutes of medical treatment, there was no clinical improvement, so the patient was moved to the respiratory intensive care unit, where non-invasive ventilation (NIV) was initiated using a full face mask and bi-level positive airway pressure (VPAP II; ResMed) at inspiratory and expiratory pressures of 80 and 40 mm H₂O respectively. An arterial line was placed through the right radial artery. Serum electrolytes, renal and liver function

tests, and complete blood counts were normal. In view of worsening dyspnoea and falling saturation, pressures were gradually increased to 150 and 80 mm H₂O respectively. Arterial blood gases repeated after two hours revealed type 2 respiratory failure (O_2 6 l/min, pH 7.27, PaO_2 10.3 kPa, $PaCO_2$ 6.3 kPa, HCO_3^- 27 mEq/l). As the patient was not improving on NIV and was becoming hypoxaemic and agitated, she was given 2 mg/kg of propofol, and oral endotracheal intubation was performed. She was mechanically ventilated with assist/control mode at tidal volumes of 400 ml, rate of 12, positive end expiratory pressure (PEEP) of 50 mm H₂O, and peak inspiratory flow of 70 l/min. There was no auto-PEEP and her peak and plateau pressures were 350 and 180 mm H₂O respectively. She was sedated intermittently with intravenous midazolam. Other medications and supportive medical care were continued. Arterial blood gases measured after 2 hours of mechanical ventilation were normal (pH 7.48, PaO_2 10.5 kPa, $PaCO_2$ 4 kPa, HCO_3^- 26 mEq/l, FiO_2 0.3). On day 2 following admission, she developed left lower collapse, for which a fiberoptic bronchoscopy was performed, and a mucus plug occluding the left lower bronchus was removed. She was weaned off the ventilator and extubated uneventfully on day 3, and discharged after 5 days of admission.

DISCUSSION

NIV has revolutionised the management of patients with diverse forms of respiratory failure.¹ The role of NIV in acute asthma (AA) is at best controversial. While no guidelines have been established, a reasonable approach would be to use NIV in patients who do not respond to initial medical therapy, and have no contraindications for the use of NIV. The advantages of NIV include improved patient comfort and reduced need for sedation, while avoiding the complications of endotracheal intubation, including upper airway trauma, sinusitis, otitis, and nosocomial pneumonia. Patients with AA have a significant increase in both inspiratory and expiratory indices of airway resistance, which leads to increased effort in breathing, resulting in inspiratory muscle fatigue and ventilatory failure.² Endotracheal intubation is associated with a high rate of complications and results in increased airway resistance. The success of NIV in treating patients with chronic obstructive pulmonary disease (COPD) and acute respiratory failure raises the possibility that it would also benefit patients with AA. There are several theoretical advantages of NIV in AA. These include a decreased need for intubation, reduced airway resistance and effort of breathing, bronchodilation, re-expansion of atelectatic lung segments, and mobilisation of secretions. In addition, expiratory positive airway pressure may offset intrinsic PEEP, thereby decreasing the adverse haemodynamic effects of large swings

Abbreviations: AA, acute asthma; COPD, chronic obstructive pulmonary disease; NIV, non-invasive ventilation; PEEP, positive end expiratory pressure

in pleural pressures.³ Some authors have suggested that aerosolised medication may be more effectively delivered via the NIV circuit than by a standard nebuliser.⁴ However, no randomised controlled trials have confirmed these hypotheses, and we cannot assume that AA would respond to NIV in the same way as acute exacerbation of COPD.

NIV has previously been used in patients with AA, and small series have been published describing this approach. In one study, only two of 17 asthmatic patients required intubation after starting therapy with NIV, and its use was associated with a reduction in PaCO₂ and improvement in dyspnoea.⁵ In a retrospective analysis of 33 asthmatic patients, the outcomes of patients managed with invasive mechanical ventilation were compared with those who had been managed with NIV.⁶ Although the NIV patients were less hypercapnic than the other group, gas exchange and vital signs improved rapidly in the NIV group, and only three patients eventually required endotracheal intubation. A prospective, randomised, placebo controlled study compared 15 patients with AA who received NIV plus conventional therapy versus conventional therapy alone, and found improved lung function and decreased hospitalisation rate in the NIV group.⁷ In contrast, another randomised controlled trial found no significant advantages of NIV in patients with AA.⁸

The case presented here was managed initially with conventional medical therapy, and once the patient did not respond to optimum medical treatment, NIV was administered within 60 minutes of admission to the emergency room. We kept a strict watch on the patient, and she was mechanically ventilated at the earliest sign of deterioration.

In conclusion, a trial of NIV in AA may be justified in carefully selected and monitored patients. However, as its role is not clear and the condition of an asthmatic patient

may deteriorate abruptly, extreme caution is advisable to recognise failure of NIV as in the case presented here. Facilities for immediate endotracheal intubation and next level of treatment should be readily available.

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