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Pneumothorax following non-invasive ventilation for acute asthma

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ABSTRACT We describe a case of pneumothorax occurring in an asthmatic following NIV in a high dependency setting. He had been persistently hypoxaemic related to bilateral lower lobe collapse from extensive mucus plugging with no other secondary cause for his hypoxaemia or mucus plugging found. There was no obvious evidence of initial pneumothorax and we presume that barotrauma related to NIV was responsible. We review the limited evidence for NIV in acute asthma and the recommendations from national guidelines.

We conclude that NIV should only be considered in acute asthma in the context of a randomised controlled trial and applied only in a high dependency or intensive therapy unit. Pneumothorax must be excluded in asthmatics that deteriorate suddenly on NIV.

KEYWORDS Asthma, non-invasive ventilation, pneumothorax.

LIST OF ABBREVIATIONS Arterio-venous malformation (AVM), bi-level positive airways pressure (BiPAP), chest X-ray (CXR), chronic obstructive pulmonary disease (COPD), computerised tomography (CT), continuous positive airways pressure (CPAP), expiratory positive airways pressure (EPAP), high dependency unit (HDU), inspiratory positive airways pressure (IPAP), intensive therapy unit (ITU), non-invasive ventilation (NIV), peak expiratory flow (PEF)

DECLARATION OF INTERESTS No conflict of interests declared.

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CASE HISTORY

A 22-year-old kitchen fitter with asthma was admitted with a four-day history of infective symptoms. He was diagnosed on the basis of steroid responsive symptoms, significantly reversible airways obstruction and diurnal peak flow variation. His asthma had been poorly controlled for some few weeks with nocturnal symptoms and a reduction in PEF to 250 (from his usual 350) despite Seretide 250 two puffs twice daily (metered-dose inhaler via aerochamber). He continued to smoke 15 cigarettes daily with a five pack year history.

On examination, he had pectus excavatum, and was only able to speak in short sentences. He was tachycardic (120/min) and tachypnoeic (30/min) with low saturations on air (87%). Chest examination was consistent with bilateral lower lobe collapse, confirmed on CXR, with no evidence of pneumothorax and hyperinflated lung fields (see Figure 1). Arterial blood gases on air confirmed type I respiratory failure (pO_2 6·3, pCO_2 4·0).

He was treated with high flow oxygen, steroids, bronchodilators, antibiotics and physiotherapy. Aspergillus was seen in his sputum but no specific bacteria were isolated. He failed to oxygenate well and was transferred to HDU. Continuous positive airways pressure at 10 cm water pressure (10 cm H_2O) via a BiPAP Vision ventilator (Respironics) and physiotherapy were used to improve oxygenation and promote re-expansion. Initially pO_2 rose 1·6 kPa but he still required 100% oxygen. Subsequent oxygenation remained poor despite increasing CPAP pressure (15 cm H_2O) and a repeat CXR showed minimal change.

After three days of CPAP, he was changed to BiPAP, at 20 cm IPAP and 15 cm EPAP but unfortunately after five minutes, his oxygen saturations fell to 80% on 100% oxygen with signs of respiratory distress. A repeat CXR confirmed a right sided pneumothorax in addition to his bilateral lower lobe collapse (see Figure 2). He required emergency intubation, ventilation and insertion of two chest drains. The first axillary drain failed to achieve good re-expansion, and a second apical large bore chest drain was required with suction.

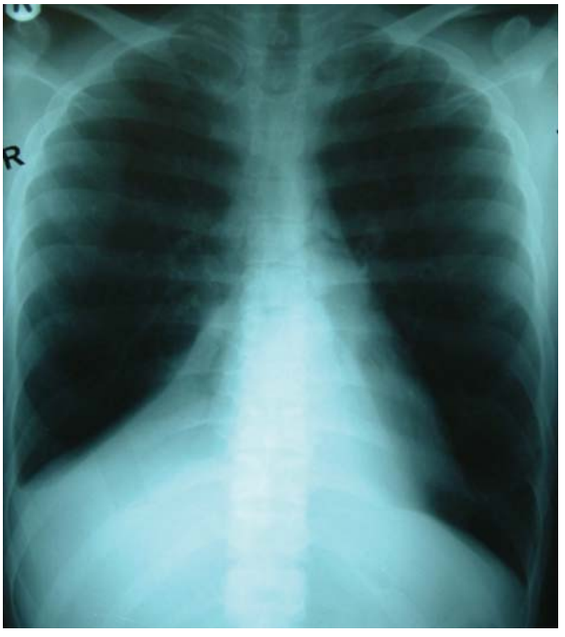

FIGURE 1 Initial CXR. Bilateral lower lobe collapse, hyperinflated lung fields.

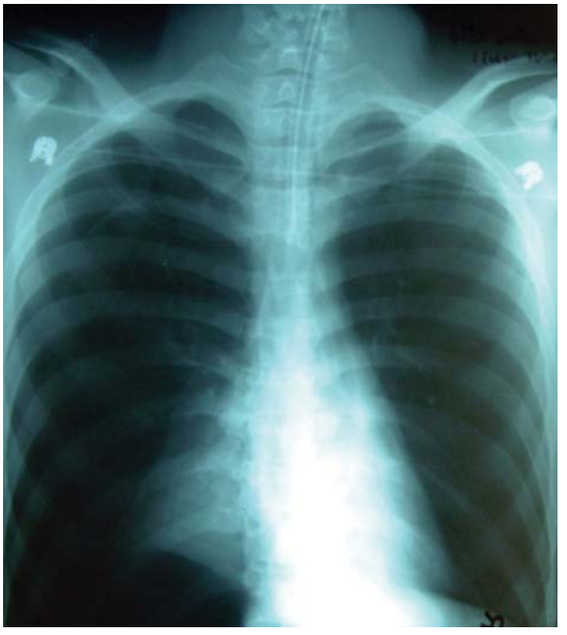

FIGURE 2 Chest X-ray on HDU at time of desaturation. New right sided pneumothorax.

His oxygen requirements remained high. Echocardiography and a CT scan of chest excluded any intracardiac shunt or other underlying lung disease, broncho-pleural fistula or AVM contributing to shunting. The CT confirmed the hydropneumothorax, mucus plugging and lobar collapse (see Figures 3A and 3B). He eventually improved and was discharged after a prolonged stay in ITU and HDU (complicated by nosocomial pleural and respiratory tract infection requiring a further chest drain possibly related to the initial chest drain). Subsequent immunological investigations off steroids excluded allergic bronchopulmonary aspergillosis. At follow-up in three months, the collapse had entirely resolved radiographically and his asthma control improved allowing him to return to his kickboxing.

DISCUSSION

Non-invasive ventilation is an established hospital treatment in acute and chronic respiratory disease, most commonly used in acute hypercapnic COPD exacerbations.[1, 2] In this case initially CPAP and later NIV were used both to promote re-expansion of the bilateral collapsed lower lobes and to improve oxygenation.

We presume the pneumothorax occurred as a direct result of barotrauma related to the high pressures used with BiPAP. Barotrauma is a well recognised risk of mechanical ventilation quoted at 0·5 – 40% in various studies. Chest X-ray prior to BiPAP had shown no evidence of pneumothorax, although this does not completely exclude this possibility (a lateral upright CXR may detect up to 14% of pneumothoraces not seen on a postero-anterior view[3]). In addition, because of the bilateral lower lobe collapse, only the middle and upper lobes were being ventilated perhaps exposing them to effective higher airway pressures.

In acute asthma there is limited evidence for the use of NIV. One small prospective trial of 17 patients with acute asthma in respiratory failure (mean pH 7·25) did report a physiological improvement with NIV.[4] Only two patients required intubation following 16+/-21 hours of NIV with a mean airways pressure of 18+/-5 cm (maximum 25 cm) and no pneumothoraces occurred. At present, national guidelines do not recommend routine use except in a randomised controlled trial.[1, 5]

In conclusion, NIV can result in pneumothorax and this should be excluded in patients who acutely deteriorate on NIV. Non-invasive ventilation may have a role in acute asthma but this requires further controlled studies on a larger scale.

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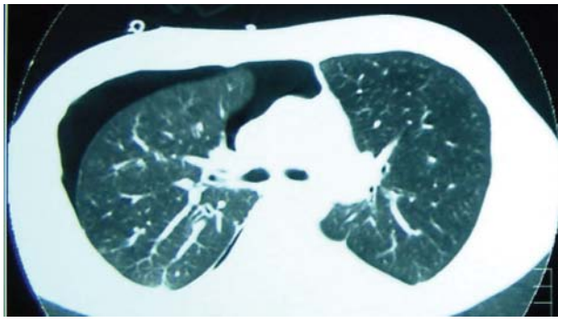

FIGURE 3A CT chest. Lung windows showing right sided pneumothorax, and mucus plugging with centrilobular nodular appearance.

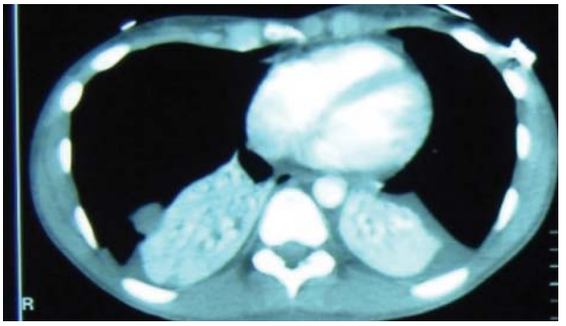

FIGURE 3B CT chest. Mediastinal windows showing bilateral lower lobe collapse and pleural fluid

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