Case Report

Fatal Re-expansion Pulmonary Oedema in a Mechanically Ventilated Patient

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ABSTRACT

Fatal course of re-expansion pulmonary oedema (REPO) is infrequent and very rarely documented in mechanically ventilated patients. We report a case of fatal REPO following tube thoracostomy for a right-sided pneumothorax in an elderly patient of chronic obstructive pulmonary disease (COPD) with respiratory failure on mechanical ventilation.
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Key words: Re-expansion pulmonary oedema, Pneumothorax, Mechanical ventilation, Respiratory failure.

INTRODUCTION

Re-expansion pulmonary oedema (REPO) is an infrequent iatrogenic complication of rapid pleural drainage of a large volume of fluid or air. Many patients with REPO are asymptomatic and detected only on computed tomography (CT) of chest. The usual course of symptomatic REPO is benign with a low mortality (1.2%).[1] Pre-existing pulmonary disease is a risk factor of severe REPO. However, the fatal outcome of REPO in chronic obstructive pulmonary disease (COPD) with respiratory failure is rarely reported.[2] We report a case of fatal REPO following tube thoracostomy for pneumothorax in a patient with COPD and respiratory failure on mechanical ventilation.

CASE REPORT

A 65-year-old male was admitted with severe breathlessness and cough of four days duration. There was no history of fever. He has been diagnosed to have COPD for the past four years. He smoked *bidi* (average 20 per day) for about 35 years and quit four years ago. On examination, the patient was drowsy, cyanosed and in respiratory distress. There was no jugular venous distension or peripheral oedema. The pulse rate was 124 beats per minute, blood pressure, 90/60 mmHg, respiratory rate, 32 breaths per minute and oxygen saturation on pulse oximetry was 78% at room air. Chest examination revealed signs of pneumothorax on the right side. Room air arterial blood gas (ABG) had a pH of 7.14, partial pressure of carbon dioxide ($PaCO_2$) of 71mmHg, partial pressure of oxygen (PaO_2) of 58mmHg and bicarbonate (HCO_3) of 24.2 mEq/L. The patient was intubated and mechanical ventilation (pressure support synchronised intermittent mandatory ventilator [pSIMV] mode with positive end expiratory pressure [PEEP] of 5cm of water) was started with supplemental fraction of inspired oxygen (FiO_2) at 0.8. Other standard and supportive treatment for COPD was initiated simultaneously. Blood investigations revealed a haemoglobin (Hb) of 10.6g/dL, a total leukocyte count (TLC) of 12,900/cumm with 90% polymorphonuclear leukocytes. The random blood sugar was 186mg/dL while other blood chemistry parameters were within normal limits. Chest radiograph (anteroposterior view) revealed pneumothorax on the right side and hyperinflated lung on the left side (Figure 1). Microbiological examination of endotracheal secretions and blood revealed no pathogens. A 12-lead echocardiogram (ECG) was normal and transthoracic echo-cardiogram showed mild pulmonary arterial hypertension. Subsequent ABG analysis on ventilatory support showed improvement with a pH of 7.345, $PaCO_2$ of 65.5mmHg, PaO_2 of 87mmHg, and HCO_3 of 30.7mEq/L. A tube thoracostomy was performed on the right side and connected to underwater seal. The ventilator parameters improved and FiO_2 was decreased to 0.6 with the oxygen saturation constantly above 90 percent. Two hours after chest tube insertion, suddenly copious frothy secretions were seen gushing through the

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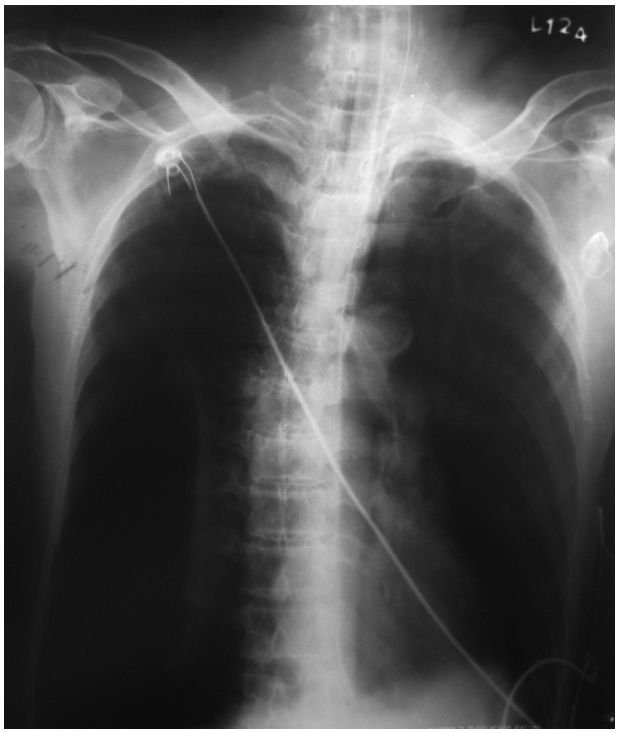

Figure 1. Chest radiograph (antero-posterior view) showing right pneumothorax and hyperinflated left lung.

endotracheal tube and the blood pressure fell to 80/50mmHg. On examination, chest tube was patent without air leak. Inspiratory crackles were heard over the right lower lobe on auscultation. At this time, the ABG analysis showed a pH of 7.164, $PaCO_2$ of 74mmHg, PaO_2 of 49mmHg and HCO_3 of 26mEq/L. Oxygen saturation remained below 80% despite ventilatory support with maximum supplemental oxygen (FiO_2 of 1.0). A supine chest radiograph was obtained that showed poorly-defined alveolar opacities in the right mid and lower zones with subcutaneous emphysema. Cardiac evaluation was negative. Repeat blood counts showed a Hb of 11.6g/dL and TLC of 10,000/cumm with 88% polymorphonuclear leukocytes. A diagnosis of ipsilateral REPO was made. For hypotension, intravenous dopamine infusion at 5mcg/kg/min and intravenous fluid therapy were started but there was no improvement in blood pressure despite increasing the dose of dopamine to 20mcg/kg/minute. The blood pressure was stabilised at 96/60mmHg only after noradrenaline infusion was added and maintained at 0.5mcg/kg/minute. Next day, the patient became comatose. There was again a fall in the blood pressure to 70/38mmHg. The ABG showed worsening of respiratory acidosis with metabolic acidosis (pH:6.892; $PaCO_2$:109.0mmHg; PaO_2: 58.0mmHg and HCO_3; 20.5mEq/L). Repeat chest radiograph showed an increase in the alveolar shadows in right hemithorax (Figure 2).

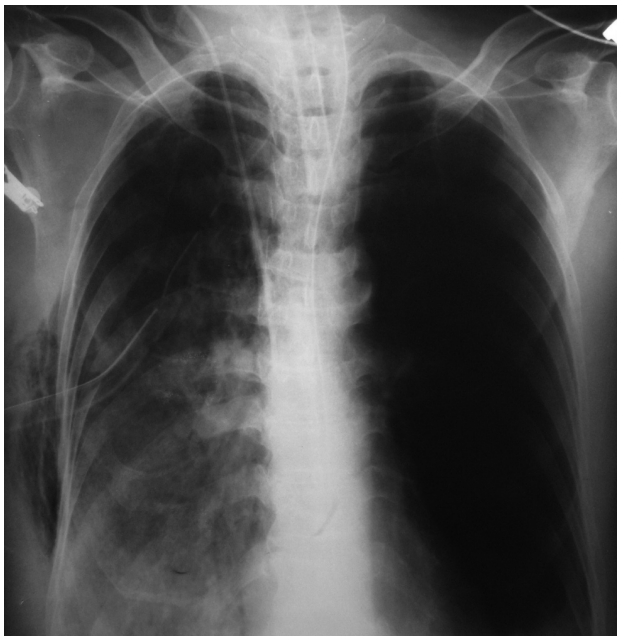

Figure 2. Post tube thoracostomy supine chest radiograph showing alveolar opacities in the right lung with minimal subcutaneous emphysema.

Mechanical ventilation and optimum supportive therapies were failed to stabilise the patient and he had sudden cardiac arrest and died approximately 36 hours after the tube thoracostomy.

DISCUSSION

Re-expansion pulmonary oedema should be suspected whenever there is a sudden respiratory or haemodynamic deterioration following pleural drainage of air or fluid with or without negative suction. There is no definitive diagnostic test for REPO. The diagnosis of REPO is made by history, suggestive clinical and chest radiographic features. REPO must be differentiated from pneumonia, haemorrhage and effusion. The usual clinical features of REPO include persistent cough, frothy sputum, agitation, tachycardia, tachypnoea, unresponsive hypoxaemia despite 100% oxygen lasting upto 48 hours and it usually resolves within three to seven days.[3] Our patient survived for 36 hours after developing REPO. The exact mechanism for the development and resolution of REPO is not known. The speed of pleural drainage of air or fluid as well as the duration of lung collapse are important predisposing factors for REPO.[4] Generation of excessive negative pleural pressure, an increase in the pulmonary capillary permeability and re-perfusion injury with the release of inflammatory mediators possibly resulting from hypoxic injury to the collapsed lung are possible mechanisms involved in the development of REPO.[5]

REPO is a permeability pulmonary oedema as a result of damage to the pulmonary micro-vessels caused by the mechanical stress when these are stretched during re-expansion of the collapsed lung. It is postulated that when mechanical stress during re-expansion of collapsed lung is small and does not induce biological injury, REPO does not develop.[6] In a study[7] of REPO between 1958 to 1987, 11 out of 53 patients died. Interestingly, no deaths occured in two subsequent studies which recorded an incidence of REPO at 14.4%[8] and 0.9%.[9] Pre-existing cardiovascular diasease is a risk factor for severe REPO characterised by hypotension and decreased organ perfusion that may be fatal.[10] Careful performance of pleural drainage procedure is essential when the left ventricular reserve is poor.[11] There is no specific treatment for REPO. In severe REPO, mechanical ventilatory support with positive end-expiratory positive pressure and haemodynamic support may be appropriate and successful.[12] Non-invasive continuous positive airway pressure ventilation has also been used effectively for severe REPO following chest tube insertion for a large left-sided pneumothorax.[13] Keeping the patient in lateral decubitus position with the side having REPO up may be a simple technique that can increase oxygenation by decreasing the intrapulmonary shunt.[14] REPO may run a fatal course without any pre-morbid illness despite mechanical ventilation and maximum haemodynamic support.[15] In our case, REPO following the tube thoracostomy drainage of pneumothorax, without negative suction, in pre-existing COPD with respiratory failure resulted in death despite maximum ionotropic support and mechanical ventilation. To conclude, following tube thoracostomy drainage of pneumothorax in COPD with respiratory failure, a careful watch for REPO is advisable.

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