FATAL RE-EXPANSION PULMONARY OEDEMA

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SYNOPSIS

A middle-aged Chinese male with a history of chronic bronchitis developed a massive right pneumothorax. Insertion of intercostal catheter resulted in acute ipsilateral pulmonary oedema. Resuscitative measures were unsuccessful.

INTRODUCTION

A case of fatal unilateral pulmonary oedema following evacuation of air from a spontaneous pneumothorax is presented. Ipsilateral pulmonary oedema resulting from aspiration of air or fluid is a well recognised phenomenon and has recently attracted much interest. Though usually it resolves spontaneously, it could occasionally be life threatening especially in those whom the cardiopulmonary function is already compromised. Inadequate knowledge about the pathophysiology of this condition has hindered a rational approach to the management in acute cases.

CASE REPORT

A 49 year old Chinese male was admitted on 25.1.85 with complaints of breathlessness and right chest pain aggravated by deep breathing for five days. On clinical examination he was noted to be dyspnoeic with a respiratory rate of 30/min and cyanosed. He was afebrile, had a pulse rate of 100/min and blood pressure of 110/70 mmHg. The air entry was markedly diminished over the right chest and crepitations were heard over the left lung base. He had been smoking 10 cigarettes per day over the last 20 years and complained of productive cough and dyspnoea on exertion for the past one year. He had two previous admissions for exacerbation of chronic bronchitis.

Investigations revealed a haemoglobin of 18.0g%, serum Na^+ 133mEq/L, serum K^+ 3.8mEq/L, serum Cl^- 81mEq/L. the arterial blood gases while breathing air were pH 7.39, PCO_2 52.8 mmHg, HCO_3 31.4mEq/L, PaO_2 58.4 mmHg, 0_2 saturation 88.3%. ECG showed a sinus tachycardia with 'P' pulmonale in lead II and right axis deviation. Chest x-ray (Fig 1) revealed a large right pneumothorax occupying approximately 80% of the right hemithorax and depressing the right hemidiaphragm. An intercostal catheter was introduced at the 4th right intercostal space at the anterior axillary line and connected to an underwater seal. No negative pressure suction was applied to evacuate the pneumothorax. The patient was also given 24% oxygen at 3 litres/min. via a ventimask. The patient felt much relieved after the introduction of the intercostal catheter. However, an hour later he complained of increasing breathlessness. He was noted to be tachypnoeic, centrally cyanosed, sweaty and clammy. His pulse rate was 120/min. and his systolic pressure was 80 mmHg. The diastolic pressure could not be determined. Auscultation revealed inspiratory rhonchi bilaterally and coarse crepitations over the right chest.

A repeat arterial blood gases while breathing 24% oxygen were pH 7.35, PCO_2 44.3 mmHg, HCO_3 24.3mEq/L, PaO_2 49.7 mmHg and oxygen saturation 82%. Chest radiograph (Fig 2) showed partially re-expanded lung which was more or less homogeneously opacified. He was administered intravenous Lasix, Aminophylline and Dopamine. At intubation the patient expectorated copious amounts of pink frothy fluid. He collapsed before he could be placed on a respirator and resuscitative measures were unsuccessful.

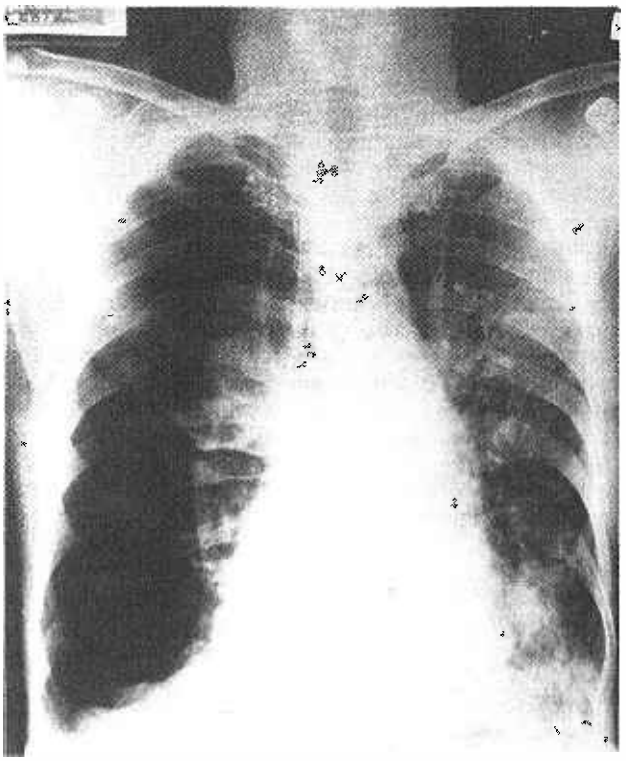

Fig 1 Massive right pneumothorax left lower zone pneumonitis

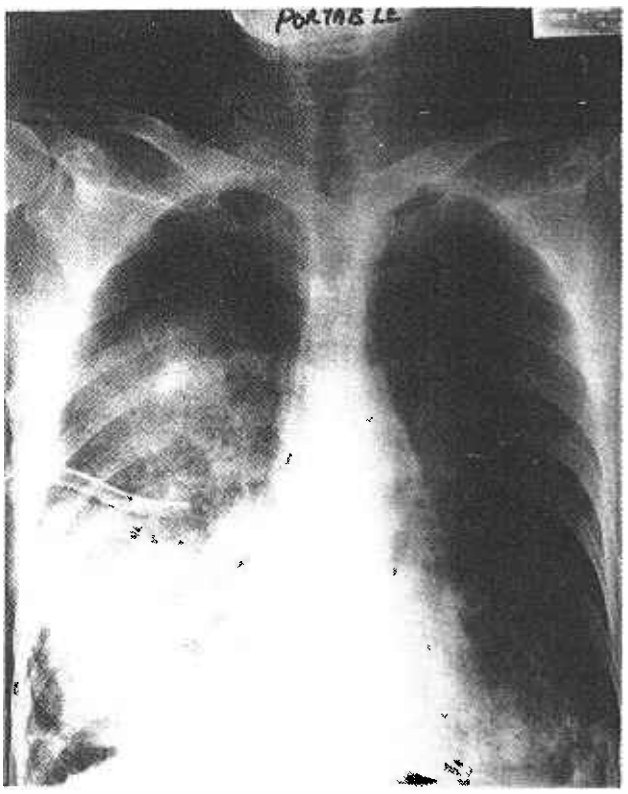

Fig 2 Partially re-expanded right showing homogeneous opacification

DISCUSSION

Re-expansion pulmonary oedema is one of the less common causes of non cardiac pulmonary oedema. Unilateral pulmonary oedema following evacuation of air or fluid has been known to occur for over a hundred years. The first well documented case was reported by Foucart (1) in 1875. In view of the relatively small number of case reports, pulmonary oedema following aspiration of pneumothorax is believed to be rare. However, in a series of 40 consecutive cases of pneumothorax Cheee t al (2) observed seven cases of asymptomatic pulmonary oedema intercostal catheter drainage.

Though the mechanisms of re-expansion pulmonary oedema are as yet controversial, prolonged lung collapse (3) and rapid evacuation (4, 5) are two causes often mentioned. These definitely are not the sole factors involved as it is known to occur even in pneumothorax of less than 72 hours' duration (6) and in the absence of application excessive negative intrapleural pressure as in our patient. Marland et al (7) analysed the pulmonary oedema fluid and found the protein content to be 4.6gm/dl and the pulmonary oedema serum protein ratio to be 0.85. From these observations they postulated pulmonary capillary damage as a result of prolonged lung collapse or rapid re-expansion as a factor in the development of oedema.

In the large majority of reported cases re-expansion pulmonary oedema is a radiological manifestation

which resolves spontaneously over 5-6 days without any sequela. To date only two fatalities have been described (3, 8). Though it is impossible to anticipate this complication, development of pulmonary oedema in patients with impairment of cardio pulmonary function is particularly liable to be life threatening (8). In our patient the underlying chronic bronchitis and pneumonitis in his left lung probably contributed to his fatal outcome. Treatment of symptomatic patients has been along conventional lines. Re-collapse of lung (9) and transient occlusion of the ipsilateral pulmonary artery (8) are some of the measures advocated in severely hypoxic patients. As pulmonary oedema usually occurs within one to two hours after re-expansion of the collapsed lung, close observation during this critical period in high risk patients is essential. Early detection and use of double lumen endotracheal tube will prevent aspiration of oedema fluid into the contralateral lung and permit positive pressure ventilation of the ipsilateral lung in severe cases.

Simple aspiration of spontaneous pneumothorax appears to be a safe and effective mode of treatment (10) and will ensure a relatively slower re-expansion. It is worth evaluating the effectiveness of this method in preventing re-expansion pulmonary oedema.

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