Recurrent re-expansion pulmonary oedema complicating rapid decompression of large pneumothoraces

Liam Chong Kin, MBBS, MRCP Lecturer

Shaikh Jaafar, MBBS Medical Officer

Department of Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur

Summary

A young male who developed ipsilateral pulmonary oedema on two occasions as a complication of treatment of pneumothoraces involving the left lung is reported. The importance of large pneumothoraces, the rapidity of decompression and the application of suction to the pleural space as factors predisposing to the development of re-expansion pulmonary oedema is well demonstrated by this case. The re-expansion pulmonary oedema was more severe on the second occasion when the lung had collapsed for a longer duration compared to the first pneumothorax which was of more recent onset.

Key words: Large pneumothorax, recurrent, re-expansion pulmonary oedema.

Introduction

A type of noncardiogenic pulmonary oedema that occurs following re-expansion of a lung after collapse due to pneumothorax has been described. It is important to recognize this rare phenomenom which can occur in the reduction of pneumothoraces. We report a case of recurrent pulmonary oedema which developed on two occasions following rapid evacuation of large pneumothoraces involving the same lung.

Case Report

A 19 year old boy presented with a 4 hour history of sudden left sided chest pain, non-productive cough and dyspnoea. He had no previous chronic respiratory symptoms. He smoked about 10 cigarettes daily since 13 years old. He was tall and thin with long digits and a high arched palate, but no other features of Marfan's syndrome. He was tachypnoeic but acyanotic. Clinical signs of a left pneumothorax were present. Chest radiograph (Fig. 1) showed a near complete left pneumothorax. An intercostal chest tube was inserted through the sixth left interspace in the mid-axillary line.

The chest tube was connected to an underwater seal and the pneumothorax was evacuated by applying a negative suction pressure of 10 cm H2O. A repeat chest radiograph obtained an hour later revealed complete re-expansion of the left lung with some alveolar infiltrates indicative of mild ipsilateral pulmonary oedema (Fig. 2). The patient felt less breathless.



Fig. 1: Chest x-ray showing a near complete left pneumothorax.

Fig. 2 : Chest x-ray showing ipsilateral re-expansion pulmonary oedema.

An attempt at removal of the chest tube after two days was not successful because the left lung recollapsed on clamping the chest tube. Suction was re-applied to re-expand the left lung but the chest tube was found to be blocked. A second chest tube was inserted through the second intercoastal space anteriorly, and removed 3 days later after radiographic confirmation that the left lung had remained fully expanded for 48 hours. The patient was sent on leave for a week. On his return chest radiography again demonstrated a near complete collapse of fhe left lung. The patient had experienced left-sided chest pain and a recurrence of breathlessness for about three days. The third intercostal drain was inserted through the second interspace anteriorly and a suction of 10 cm H2O was applied. Immediately he developed a non-productive cough and became distressed and centrally cyanosed. Auscultation disclosed widespread crepitations throughout the left lung. He was afebrile and his blood pressure maintained at 110/80 mm Hg but his heart rate rose from 80 to 100 beats per minute. Chest radiogaphy (Fig. 3) showed complete re-expansion of the left lung with changes consistent with unilateral left-sided pulmonary oedema. His arterial blood gas tensions while on supplemental oxygen at 3 1/min through nasal prongs were pH 7.37, PO2 6.1 kPa and PCO2 5.3 kPa. He was not given any diuretics. Over the next few hours his clinical condition improved and the lung crepitations became less and disappeared after three days. Nine days later, the patient underwent a left thoracotomy during which stapling of apical lung bullae and abrasion of the parietal pleura to induce pleurodesis were performed.



Fig. 3 : Chest x-ray showing recurrent ipsilateral re-expansion pulmonary oedema.

Discussion

The development of unilateral pulmonary oedema in the re-expanded lung is a rare but well recognised complication of evacuation of a pneumothorax.¹ The course of re-expansion pulmonary oedema is variable. In most instances, pulmonary oedema is simply noted on a post-aspiration chest radiograph and is not associated with major clinical consequences.² However, occasionally the oedema is severe enough to cause respiratory failure. The oedema is usually evident within two hours after expansion of the collapsed lung and may progress for 24 to 48 hours. It usually disappears in five to seven days without any sequelae.²

Various factors have been implicated in the development of re-expansion pulmonary oedema. Prolonged duration of lung collapse of three or more days, large pneumothoraces, and the rapidity with which air is removed from the pleural cavity appear to be important factors,¹ as demonstrated in the patient described. The pulmonary oedema which developed when the lung was re-expanded within a

few hours after the onset of the first episode of pneumothorax in the patient described was very mild. In contrast, he developed more severe re-expansion pulmonary oedema during the treatment of the second episode of an equally large pneumothorax when the lung had collapsed for 3 days. The application of excessive negative pressures to the pleural space is thought to be another factor. However, in some cases no negative suction was applied.³

The pathogenesis of re-expansion pulmonary oedema could be due to a combined effect of the increased permeability of the pulmonary capillaries as a result of anoxic damage during prolonged lung collapse and the sudden increase in pulmonary capillary blood flow during re-expansion of the lung.²The pulmonary oedema-serum protein ratio in re-expansion pulmonary oedema has been found to range from 0.74 to 0.85^{4,5}This is typical of non-cardiogenic pulmonary oedema whereas the ratio is less than 0.5 in cardiogenic pulmonary oedema.

The management of re-expansion pulmonary oedema is mainly supportive with supplemental oxygen and in severe cases mechanical ventilation with positive pressure ventilation. In some patients with life-threatening oedema, re-collapse of the lung by introducing air into the pleural space may be helpful.² The lung is later allowed to re-expand slowly.

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