Re-expansion Pulmonary Oedema in Chronic Pneumothorax

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ABSTRACT

A case of unilateral re-expansion pulmonary oedema in a chronic pneumothorax is presented. The patient had a long-standing left-sided pneumothorax. Intercostal drainage tube was inserted following which the patient developed severe hypotension and respiratory failure. Chest radiograph (postero-anterior view) showed partial lung expansion with unilateral pulmonary oedema. The patient responded to the standard management of pulmonary oedema and the lung was fully re-expanded. [Indian J Chest Dis Allied Sci 2010;52:165-167]

Key words: Pulmonary oedema, Pneumothorax.

INTRODUCTION

Re-expansion pulmonary oedema may be seen after rapid removal of air in pneumothorax or drainage of a pleural effusion, resulting in a rapid deterioration of the clinical condition of the patient. Sparse literature is available on this topic from India. It is a potentially serious complication requiring urgent management.

CASE REPORT

An 18-year-old male student presented with complaints of left-sided chest pain and mild breathlessness. On examination, pulse was 140/min, blood pressure was 120/80mmHg and respiratory rate was 24 per minute. On respiratory system examination, accessory muscles of respiration were active and breath sounds were decreased on the left side on auscultation. Oxygen saturation on room air was 99 percent. Chest radiograph (PA view) showed a left-sided hydropneumothorax with complete collapse of the left lung (Figure 1). An earlier chest radiograph done a month ago for similar complaints also showed left-sided pneumothorax for which no active intervention had been done. He gave history of having taken a complete course of antituberculosis treatment three months ago.

An intercostal drainage tube was inserted in the 5th intercostal space in the anterior axillary line on the left side. During the procedure, the patient collapsed. Immediately, the patient was treated with injection hydrocortisone 100mg intravenous, high flow oxygen and intravenous fluids. After two hours of intercostal

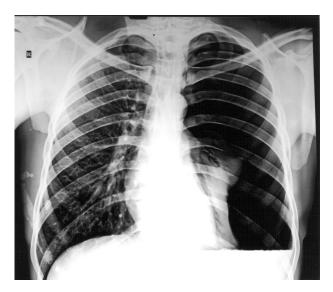


Figure 1. Chest radiograph (postero-anterior view) showing the left-sided pneumothorax and collapsed lung.

drainage tube insertion the patient started complaining of chest pain that was centralised, non-radiating and associated with dry cough. A chest radiograph was done, that revealed the left lung had re-expanded partially with a generalised ill-defined opacity suggestive of left-sided pulmonary oedema (Figure 2).

The patient continued to be hypotensive, became drowsy and was shifted to the intensive case unit (ICU). The blood pressure was 90/70 mmHg and oxygen saturation was 70% on room air. A central line was inserted on the left side and central venous pressure was measured that was 2cm of water.

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Figure 2. Chest radiograph (postero-anterior view) showing left-sided pulmonary oedema with partially re-expanded lung.

Ionotropic support was started along with intravenous fluids and high flow oxygen was given. The blood pressure started rising gradually. However, the size of pneumothorax also increased on the subsequent radiograph taken on the second day of hospitalisation. The intercostal drainage tube was repositioned followed which the lung showed complete expansion on the third day but with persistent left lower zone opacities (Figure 3). The left lung opacity gradually cleared up over the next one week. The vital parameters improved after five days and the intercostal drainage tube was subsequently removed.

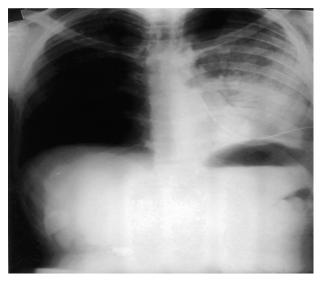


Figure 3. Chest radiograph (postero-anterior view) showing fully expanded lung with ill-defined opacity in the middle and the lower zones with intercostal drain *in situ*.

DISCUSSION

Re-expansion pulmonary oedema is a rare but serious complication of rapid evacuation of pleural effusion or pneumothorax. A mortality as high as 20% has been reported.1 Although it was first described in 1853, only 60 cases were reported between 1958 and 1999. Some authors suspect that re-expansion pulmonary oedema results from an increased endothelial permeability accompanying loss of integrity of the alveolarcapillary membrane. This capillary overflow is reported to be due to mechanical stresses and distention within the re-inflated lung. Additionally, the leakage is potentially worsened by increased nitric oxide and inflammatory mediators from neutrophils. Re-perfusion injury has also been offered as a potential explanation. With re-inflation of the previously non-ventilated, non-perfused collapsed lung, oxygen-free radicals may form, with resultant injury. This theory is supported by the findings that increased levels of oxygen and the administration of antioxidants prevents oedema formation. Low levels of surfactant in chronically collapsed lungs is another possible cause.

The exact cause of re-expansion pulmonary oedema, however, has yet to be evaluated. Severe hypotension during re-expansion pulmonary oedema that was fatal has been reported earlier.²

There are no definitive guidelines for prevention of re-expansion pulmonary oedema. It is believed that re-expansion pulmonary oedema is more likely to occur in a lung that has been collapsed for more than three days.3 In our patient, the pneumothorax had been present for over a month as suggested by his previous chest radiograph. In a patient with pleural effusion, if the pleural pressure is not monitored during the procedure, then no more than one liter of fluid should be removed in one sitting. The goal is to keep the pleural pressure above -20cm of water. According to Light⁴ if pleural pressure are being monitored, there is no limit on the amount of fluid that can be removed. The procedure should be halted if the patient has sudden chest tightness or the sudden onset of a cough. In our patient, intercostal drainage tube was clamped on suspicion of re-expansion pulmonary oedema to prevent its further worsening.

If re-expansion pulmonary oedema occurs, the treatment is mostly supportive, including supplemental oxygen, positive pressure ventilation, intravenous fluids, appropriate hemodynamic monitoring, vasopressors, and inotropic agents. Diuresis should be avoided.⁵ However, if there is radiographic evidence of re-expansion pulmonary oedema but the patient is asymptomatic and stable, no treatment is necessary. Certain precautions are

advised, such as avoidance of excessive ventilatory pressures, avoidance of application of negative pressure to the pleural space, and lateral decubitus positioning with the affected side facing up, to reduce intra-pulmonary shift and improve oxygenation. In severe cases, the patients may require endotracheal intubation and application of positive end-expiratory pressure. Differential two lung ventilatory strategies may be required in refractory cases.³

Re-expansion pulmonary oedema is a rare but serious complication of thoracocentesis. Physicians should always consider the possibility and be alert to the varied presentation of this complication.

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