CASE REPORT

Fatal re-expansion pulmonary edema in a young adult following tube thoracostomy for spontaneous pneumothorax

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SUMMARY

Re-expansion pulmonary edema (RPE) is a rare but potentially fatal complication that can occur following rapid lung expansion while managing patients with pleural effusion or pneumothorax. In this case, fatal outcome occurred due to RPE in a previously healthy young adult male patient subsequent to tube thoracostomy for spontaneous pneumothorax. While managing patients with pneumothorax or large pleural effusions, precautions should be taken to avoid rapid re-expansion of the previously collapsed lung in order to reduce the probability of development of this complication.

BACKGROUND

Re-expansion pulmonary edema (RPE) is a rare complication which is attributed to the rapid drainage of air or fluid from the pleural cavity. It has been reported to occur following either thoracentesis or tube thoracostomy. The attributed pathological mechanism is the abrupt reduction in the intrapleural pressure, especially as a result of extensive and rapid pneumothorax/pleural effusion drainage or rapid pulmonary expansion in cases where long-term pulmonary collapse has been present.^{1 2} These patients may develop varying degrees of hypoxia and hypotension and occasionally, pulmonary oedema which becomes bilateral, and the patient may require endotracheal intubation and mechanical ventilation. Rarely, the syndrome is fatal in otherwise healthy, young adult subjects without any underlying comorbid illnesses who develop this complication.

CASE PRESENTATION

A 20-year-old male patient presented to the emergency department at another healthcare centre with a history of sudden onset shortness of breath and left side chest pain, persistent for 5 days. The pain was sudden in onset, pleuritic in character and was not associated with radiation to any site. There was no history of fever, haemoptysis, wheezing, expectoration or any other comorbid illnesses. At the time subsequent to a chest radiograph (figure 1, left panel), a diagnosis of large left side spontaneous pneumothorax was made and an intercostal tube was inserted. Following tube thoracostomy, the patient developed worsening cough and shortness of breath associated with expectoration of pink frothy sputum. As there was progressive worsening of symptoms, the patient was referred to our centre.

At time of examination in the emergency room, the patient was cyanosed and pulse oximetric saturation (SpO₂) was 80% while breathing oxygen (FiO₂ 50%) with venturi mask. His pulse rate was 120/min, respiratory rate 35 bpm with visible use of accessory muscles of respiration and blood pressure was 90/68 mm Hg. Coarse crepitations were audible in the left infrascapular and axillary areas. Rest of the systemic examination was unremarkable. Chest radiograph demonstrated diffuse alveolar infiltrates on the left hemithorax suggestive of unilateral pulmonary oedema (figure 1, right panel). A diagnosis of RPE was made.

The patient was endotracheally intubated, mechanical ventilation was initiated and he was immediately shifted to respiratory intensive care unit. Mechanical ventilation with low tidal volume, high FiO₂ and PEEP (positive end expiratory pressure) was delivered. Simultaneously supportive treatment, intravenous fluids and inotropes to maintain haemodynamic parameters were started. Despite the best available supportive treatment and mechanical ventilation, the patient developed refractory hypoxaemia and worsening haemodynamic instability. Refractory ventricular fibrillation ensued from which the patient could not be revived and the patient expired.

DISCUSSION

RPE may be considered an iatrogenic complication because of rapid emptying of the pleural cavity which leads to rapid re-expansion of the collapsed lung. Pinault in 1853 first reported re-expansion oedema following pleural fluid aspiration and since then many reports of re-expansion oedema following pleural fluid aspiration have been reported. In 1959, Carlson *et al*³ reported RPE in the setting of pneumothorax. In the following years, there were several case reports on the occurrence of RPE after drainage of spontaneous pneumothorax, resection of mediastinal tumours, thoracoscopy or talc pleurodesis.^{4 5} The reported incidence of RPE following drainage of pleural effusion/pneumothorax is between 0% and 1%.^{1 2 6} This is likely an underestimate as the syndrome of RPE may be silent and only manifest radiologically in some cases. Young patients (20–40 years of age) with large pneumothorax and duration of pneumothorax greater than 7 days have been reported to be at the greatest risk of development of RPE.

The proposed pathological mechanism in RPE is increased capillary permeability, which is evident

To cite: Sharma S, Madan K, Singh N. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-010177

Figure 1 (left panel) Posteroanterior chest radiograph showing the presence of a large left side pneumothorax with near total collapse of the underlying left lung. (right panel) Chest radiograph following tube thoracostomy demonstrating unilateral alveolar opacities in the left hemithorax confirming the diagnosis of re-expansion pulmonary edema.

from the high protein content of the oedema fluid, in both animal and human studies.

Mechanical stresses to the lung during re-expansion cause damage to the capillary wall and leads to oedema formation. Another proposed possible mechanism is reperfusion injury due to reactive oxygen species (ROS). However, several animal studies^{7–9} had shown a decrease in neutrophilic infiltration but not the amount of oedema when ROS-scavenging compounds such as dimethylthiourea, catalase or superoxide dismutase were administered in experimental situations.

In humans, most cases of RPE develop when pneumothorax or pleural effusion have been present for at least 3 days and when negative pressure in the form of suction have been applied to the pleural space. Symptoms of RPE are pernicious coughing or chest tightness during or immediately after tube thoracostomy or thoracentesis which are progressive for 12–24 h. Serial chest radiographs reveal progressive unilateral pulmonary oedema, which may progress to involve the contralateral lung. Treatment is primarily supportive, with the administration of supplemental oxygen and diuretics, and endotracheal intubation and mechanical ventilation depending upon the individual case scenarios. Administration of CPAP (continuous positive airway pressure) within the first hour of the development of the syndrome was effective in one case report.¹⁰ Although most of the patients recover within 5–7 days, death rates may reach as high as 20% in patients who develop this complication.⁶ A literature review in 1988 revealed 53 cases of RPE with a fatality rate of 21% (n=11).¹¹

RPE can be fatal as highlighted by the index case; therefore, the major focus should be on the prevention of its occurrence. While performing tube thoracostomy for spontaneous

pneumothorax, the tubes should initially be connected to an underwater-seal drainage apparatus rather than to negative suction. If underwater-seal drainage does not cause re-expansion of the underlying lung within 24–48 h, then negative pressure (preferably <20 cm H₂O) can be applied to the pleural space.⁶ If a patient develops tightness of the chest or experiences coughing during thoracentesis, the procedure should be terminated.¹² The volume of fluid drained during thoracostomy should usually be limited to 1–1.5 Litres. RPE as a diagnostic possibility must be considered in the differential diagnosis of any patient with a large pneumothorax or pleural effusion who develops worsening respiratory symptoms following tube thoracostomy or thoracentesis.

Contributors SS and KM were involved in patient management, manuscript preparation and data collection. NS was involved in patient management and manuscript preparation.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- Re-expansion pulmonary edema is a rare but potentially fatal condition which can occur while managing patients with pneumothorax or pleural effusion.
- While performing thoracentesis, the amount of fluid removed should usually be limited to 1–1.5 litres and in case the patient develops chest tightness or worsening cough/shortness of breath during the procedure, it should be terminated.
- Management of re-expansion pulmonary edema is primarily supportive.

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