Re-expansion pulmonary oedema following spontaneous pneumothorax

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Re-expansion pulmonary oedema may occur after chest tube drainage of pneumothorax and can give rise to cardiopulmonary manifestations which range from the mild to the severe. In order to evaluate the prevalence and the clinical manifestations of this complication, all patients with spontaneous pneumothorax managed with chest tube drainage were evaluated over an 8-yr period (1986-1994). A chest radiograph was performed routinely in all patients within 4 h of tube insertion. Lung expansion and the appearance of infiltrates within the lungs were investigated specifically. Re-expansion oedema was noted in three of 320 episodes (0.9%). Two of the three patients needed rapid and extensive clinical treatment.

Introduction

Re-expansion unilateral pulmonary oedema (RPO) is an uncommon condition occurring after expansion of a collapsed lung. It has been reported after evacuation of pleural effusion (1,2), relief of bronchial obstruction (3) or following chest tube insertion for spontaneous pneumothorax (1,4-7).

The clinical manifestations of RPO can vary considerably. In some cases, symptoms may be absent and RPO may be revealed only by typical radiological findings, or it may be associated with severe cardiorespiratory insufficiency and circulatory shock. Death has been reported in up to 19% of cases (1).

There are few reports of RPO in the radiological literature and the prevalence is unknown. These case reports detail the authors’ experience over an 8-yr period of 320 episodes of pneumothorax managed by insertion of a chest drain, with an emphasis on the variable presentation and the significance of early recognition of this potentially lethal condition.

Patients and Methods

The chest X-rays of all patients with spontaneous pneumothorax who were managed with chest tube drainage from January 1986 to January 1994 were studied prospectively. Chest radiography was performed routinely 1-4 h following chest tube insertion. Emergency chest radiography was performed immediately when a patient complained of cough, chest pain or shortness of breath.

Diagnosis of RPO was based on the classical radiological finding of pulmonary oedema involving at least one lobe on the side of the pneumothorax, regardless of accompanying clinical manifestations. During this period, 180 patients with 320 episodes of spontaneous pneumothorax were managed. Re-expansion pulmonary oedema was diagnosed in three patients (0.9%). Management of RPO consisted of bed rest only, or treatment with oxygen, intravenous fluids and morphine sulphate depending on the clinical findings. Management was adjusted according to the patients’ condition (Table 1).

Case Reports

CASE 1

A 24-year-old man of previous good health presented with left-sided chest pain of 8 days duration and dry cough. Chest radiograph revealed a large left-sided pneumothorax (Plate 1). A chest tube was inserted and connected to an underwater seal without suction. A routine follow-up radiograph showed that the left lung had expanded completely. A fluffy infiltrate was seen radiating from the left
Table I Clinical data of three patients with re-expansion pulmonary oedema

<table>
<thead>
<tr>
<th>Sex/age</th>
<th>Underlying disease</th>
<th>Previous pneumothorax</th>
<th>Duration of ptx. before drainage</th>
<th>Active suction</th>
<th>Symptoms of RPO</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>M, 24</td>
<td>None</td>
<td>None</td>
<td>8 days</td>
<td>No</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>M, 22</td>
<td>Marfan</td>
<td>Multiple</td>
<td>6h</td>
<td>No</td>
<td>Cough</td>
<td>O₂, Mo</td>
</tr>
<tr>
<td>M, 85</td>
<td>Emphysema</td>
<td>None</td>
<td>&gt;3 days</td>
<td>Yes</td>
<td>SOB, BP₁</td>
<td>O₂, i.v.</td>
</tr>
</tbody>
</table>

Ptx., pneumothorax; SOB, shortness of breath; BP₁, drop in blood pressure; O₂, supplemental oxygen; Mo, Morphine sulphate; i.v., intravenous fluids. RPO, re-expansion pulmonary oedema.

A repeat chest radiograph the following day showed that the lung remained expanded and all evidence of pulmonary oedema had disappeared. The tube was removed and the patient was discharged 1 day later.

CASE 2

A 22-year-old man with Marfan's syndrome was admitted with a right-sided spontaneous pneumothorax. He had a history of recurrent bilateral spontaneous pneumothoracies. The patient was managed with a chest tube for 3 days, which led to full expansion of the lung.

Several hours after removal of the chest tube, a tension pneumothorax developed again on the right side (Plate 3). Another chest tube was introduced followed by severe cough. A chest radiography was immediately performed showing expansion of the right lung and the appearance of a right lung density with indistinct vessel margins radiating from the hilum compatible with RPO (Plate 4).

The patient was managed with supplemental oxygen and morphine sulphate. The chest radiograph on the following day showed disappearance of the oedema and good expansion of the lung. The air leak persisted for 10 days after which parietal pleurectomy and pleural abrasion were performed. This case was reported in detail elsewhere (8).

CASE 3

An 85-year-old man presented with increasing shortness of breath. He was known to suffer from emphysema and was being treated with

**Plate 1** Chest radiograph: Tension pneumothorax on the left side.

**Plate 2** Chest radiograph after chest tube insertion. Good expansion of the left lung. Fluffy infiltrate radiating from the left hilum indicating left-sided pulmonary oedema.
Re-expansion pulmonary oedema is an uncommon complication which may occur after expansion of a collapsed lung. It may be asymptomatic and detected only on a routine chest radiograph as in the first case. It can present with a dry cough and dyspnoea as in the second case, but it can manifest itself also with hypotension and severe cardiopulmonary collapse as in the third patient.

Cases of fatal RPO have been reported in the literature. Fanning et al. reported a woman with Stage IV ovarian adenocarcinoma who died from RPO following thoracocentesis (2). Sautter et al. had a similar experience with a 69-year-old woman after treatment of spontaneous pneumothorax (9). Three cases of hypotension following rapid evacuation of long-standing pneumothorax were reported by Pavlin (10). Mahfood et al. (1) in an extensive review article

Plate 3  Chest radiograph shows right-sided tension pneumothorax. Adhesion is seen between the apex and the collapsed lung.

Plate 4  After insertion of a chest tube. Good expansion of the right lung with minimal residual pneumothorax and a small effusion. The right lung shows increased densities radiating from the hilum with blurring and indistinct lung vessels as signs of pulmonary oedema.

Plate 5  Chest radiograph after insertion of a chest tube on the right side. Coalescent inhomogeneous infiltrate in the right lower lobe and ill-defined fluffy infiltrate of the right upper lobe compatible with right-sided pulmonary oedema.

managed with a chest tube connected to an underwater seal and low pressure suction. This procedure was followed immediately by severe shortness of breath.

The systolic blood pressure fell to 80 mmHg and increased to 110 mmHg after administration of i.v. fluids. A chest radiograph showed a right lower lobe inhomogeneous infiltrate and ill-defined fluffy infiltrate of the right upper lobe compatible with RPO (Plate 5). The patient was treated with i.v. fluids and supplemental oxygen which resulted in clinical improvement. The oedema resolved radiologically within 2 days.

Discussion

Re-expansion pulmonary oedema is an uncommon complication which may occur after expansion of a collapsed lung. It may be asymptomatic and detected only on a routine chest radiograph as in the first case. It can present with a dry cough and dyspnoea as in the second case, but it can manifest itself also with hypotension and severe cardiopulmonary collapse as in the third patient.

Bronchodilators and corticosteroids. A chest radiograph revealed a right-sided pneumothorax which gradually increased over a 3-day period and was
described one case and collected 53 other cases of RPO. In that review, the outcome of RPO following spontaneous pneumothorax was fatal in nine of 47 such cases (19%) in whom clinical data were available for analysis.

Matsuura et al. (7) reported 21 cases of RPO in 146 patients who developed spontaneous pneumothorax (14%). These numbers are very high and most other authors have considered RPO to be a rare complication which is usually published as a single case report, or in very small series. In fact in a large series of 376 patients with spontaneous pneumothorax, 225 of whom were treated by thoracostomy tube drainage, none developed RPO (11). In the authors’ institute, RPO was identified in only three out of 320 episodes (0.9%).

Almost all cases of RPO are on the side of the pneumothorax and this was the case in the present three patients. However, Mahfood reported three patients with contralateral RPO, two of whom died (1). A case of bilateral RPO was also described (12).

The pathogenesis of RPO is unknown. Various hypotheses regarding the mechanism of RPO have been suggested. It may be related to an interplay of multiple factors causing increased pulmonary vascular permeability, such as hypoxic injury both to the capillary and alveolar membrane, and decreased surfactant (1).

Protein leakage and polymorphonuclear leukocyte (PMN) accumulation has also been noted accompanied by various fluid mediators in the oedema fluid. These mediators may be involved in chemotaxis and activation of PMN in the development of RPO (13). Rapid re-expansion of the collapsed lung may cause fluid transfer across injured membranes. A major contributory factor in the development of RPO is considered to be the length of time of the collapse. Some cases, however, can develop even after collapse of only a few hours duration (4). Ravin described a case of RPO appearing after 4.5 h of pulmonary collapse following the positioning of an endotracheal tube which was inadvertently positioned in the right mainstem bronchus (3).

Re-expansion pulmonary oedema developed in one of the present patients 6 h after the lung collapse. One may assume that the rapidity of re-expansion can also play a role—where active suction may increase the parenchymal damage and thus the risk of RPO (1). This is not supported by the authors’ experience in two patients managed without suction, but who still developed RPO.

The differential diagnosis of RPO on the chest radiograph includes pneumonia, especially if only one lobe is mainly involved, and unilateral cardiogenic pulmonary oedema. The time sequence of oedema immediately after tube insertion, the absence of fever, the presence of a normal white blood cell count and normal cardiac function usually rule out the other options. Rapid radiological resolution of the infiltrates helps to establish the correct diagnosis.

As the mechanism of RPO is different from that of cardiogenic pulmonary oedema, the treatment of RPO is also different. It consists of a combination of i.v. fluids, oxygen and morphine depending on the clinical condition of the patient. Diuresis may be fatal and should be avoided.

In view of these facts, the possibility of RPO after treatment of pneumothorax, though uncommon, should be kept in mind. If it occurs, RPO should be regarded as a potentially lethal complication warranting the proper degree of attention.

References