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Re-expansion pulmonary oedema and pleural bleeding following suction drainage of pneumothorax

Abstract
We report a case of a 26-year-old male with a large left-sided spontaneous pneumothorax, whose manifestations developed 5 days prior to hospitalisation. Suction drainage was complicated by unilateral re-expansion pulmonary oedema (REPO) and bleeding into the left pleural cavity. During thoracotomy oozing of blood from multiple sites on the pleural surface was observed. Both the temporal relationship and the nature of this bleed could suggest a pathomechanism similar to that in REPO. As a result of symptomatic treatment the patient improved over the next several days and the radiological manifestations resolved. The risk factors for REPO include: young age, large volume of pneumothorax and collapse exceeding 3 days, as it was in case of our patient. In order to reduce this risk a slower re-expansion without suction drainage or with the use of reduced pressure in the initial period of treatment is recommended.

Key words: pulmonary oedema, haemothorax, re-expansion, pneumothorax


Introduction
Pneumothorax is one of the most common emergencies in pneumonology. It usually requires interventional treatment with drainage. Complications of this procedure develop in 9–30% of the cases. The case we report below illustrates the pitfalls in the diagnosis and treatment of one of the less common complication of pleural drainage.

Case presentation
A 26-year-old male, a smoker, with no previous history, presented to the admissions department of the Institute of Tuberculosis and Lung Diseases in Warsaw, Poland, with pain in the left side of his chest accompanied by shortness of breath on effort. The symptoms appeared five days earlier and were unrelated to any exercise and were not accompanied by fever or cough. The patient denied any other symptoms. Based on the history and chest X-ray (Figure 1) a diagnosis of left-sided spontaneous pneumothorax with total lung collapse. The patient was admitted to the Department of Chest Surgery at the Institute, where a chest tube was inserted into his left pleural cavity and about 100 ml of a bloody liquid was evacuated directly following the chest tube placement. During the first 24 hours of treatment with suction drainage the patient was gradually deteriorating and developed signs of bleeding into the left pleural cavity with a blood loss of about one litre. Blood tests revealed: a mildly elevated white blood cell count (11.2 thousand/ml) with an elevated neutrophil differential count (78%), anaemia with a haemoglobin concentration of 10 g%, elevated levels of D-dimers (1821 μg/l) and fibrinogen (6.3 g/l), decreased serum protein (6 g%) and mildly prolonged activated partial thromboplastin time (aPTT) (43 s). Blo-
aching 59 mm Hg. A chest X-ray revealed progression of the extensive opacities in the left lung (Figure 3), while a computed tomography (CT) scan of the chest demonstrated unilateral ground glass opacities (Figure 4). The patient received intravenous antibio-

od gas analysis using arterialised capillary blood revealed hypoxaemia (62 mm Hg) and normocapnia. A chest X-ray (Figure 2) followed by bronchoscopy were performed, which revealed no macroscopic changes in the bronchi and no blood or purulent discharge. On the second day of hospitalisation the patient underwent left thoracotomy with the only abnormal finding being “oozing of blood from the pleura, which easily detached from the wall”. Haemostasis was achieved with hot ribbon gauze and argon. During the next day the volume drained from the left pleural cavity decreased to about 150 ml and the level of haemoglobin was stable. The patient, however, developed fever; his white blood cell count increased to 15 thousand/ml, inflammation markers increased (C-reactive protein [CRP] 144 mg%), a further prolongation of aPTT to 48 s was observed and the hypoxaemia was worsening with PaO₂ re-
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Table 1. Changes in laboratory parameters during hospitalization

<table>
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<th>Parameter</th>
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<th>Doba 1</th>
<th>Doba 5</th>
<th>Doba 14</th>
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<tr>
<td>WBC [tys./ul]</td>
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<tr>
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<td>10,7</td>
<td>11</td>
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<tr>
<td>CRP [mg/dl]</td>
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<td>3</td>
<td></td>
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<td>pO2 [mm Hg]</td>
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<td>59</td>
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<td>84</td>
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<tr>
<td>aPTT [s]</td>
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<td>48</td>
<td>37</td>
<td></td>
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<tr>
<td>Białko [g/dl]</td>
<td>6</td>
<td>5,1</td>
<td>6,7</td>
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</tr>
</tbody>
</table>

WBC—white blood cell; Hb—haemoglobin; CRP—C-reactive protein; aPTT—activated partial thromboplastin time

Figure 5. Chest Xray. Fifth day. Evident partial remission of pleural and parenchymal lesions

tics, oxygen at the rate of 3 L/min, 2 units of packed red blood cells, 2 units of fresh frozen plasma and albumins. As a result of this treatment the patient gradually improved and the fever subsided. On the fourth day post-thoracotomy the patient was transferred to the Third Department of Lung Diseases at the Institute with suspected alveolar haemorrhage for further evaluation. The evaluation at the Department revealed no signs of coagulopathy, haemostatic abnormalities or the presence of autoantibodies (ANCA, ANA). Echocardiography revealed no abnormal findings and no pathologies in the other organs were demonstrated. The patient’s condition during hospitalisation at the Department was good and the abnormal values of haematologic parameters (elevated white blood cell counts), coagulation parameters, biochemical parameters and blood gas analysis parameters normalised (Table 1). Subsequent chest X-rays revealed resolution of the abnormalities in the left lung (Figure 5). Based on the clinical picture a final diagnosis of re-expansion pulmonary oedema and pleural bleeding following treatment of left-sided pneumothorax with suction drainage was established. The patient was discharged home on the 14th day of hospitalisation. The patient has now been followed up for 3 years and his condition has been good with no recurrence of pneumothorax.

Discussion

Re-expansion pulmonary oedema (REPO) is a rare complication of surgical and, much less commonly, endoscopic procedures during which a collapsed lung is quickly re-expanded. The most common of these procedures include suction drainage for pneumothorax and evacuation of pleural fluid. Some of the less common examples of these procedures include removal of a tumour or a bulla compressing the lung or a bulla compressing the lung or restoration of airway patency (by removing a foreign body, eliminating oedema or removing a tumour) [1–3].

The possible pathomechanisms of REPO include, first of all, hydrostatic factors associated with a rapid increase of blood pressure in pulmonary vessels and the possible increase in vascular permeability associated with an increased expression of proinflammatory cytokines [4, 5].

Oedema always develops in a re-expanded lung, although sometimes (in 5–9% of the cases) the oedema is bilateral, which is most commonly associated with compression of the contralateral lung in the setting of tension pneumothorax or presence of a considerable amount of fluid in the pleural cavity [6, 7].

The incidence of REPO in groups of patients undergoing the above procedures varies. REPO complicates about 2% of thoracocenteses with evacuation of fluid and develops in 2–7% of patients with pneumothorax managed by suction drainage. In the group described by Matsuura et al. [8], REPO developed in as many as 21 out of 146 patients with pneumothorax managed by suction drainage. Kim et al. reported changes consistent with pulmonary oedema on computed tomography in as many as 29% of patients undergoing interventions for pneumothorax with 1/3 of the patients accounting for cases with unilateral and asymptomatic changes [3, 8–10].
The volume of pneumothorax is the most commonly mentioned factor predisposing to REPO in patients with pneumothorax. Matsuura et al. [8] observed an increased incidence of pulmonary oedema in patients with tension (44%) and large pneumothorax (17% in cases of total lung collapse) versus patients with moderate (7%) and small pneumothorax (0% in cases of patients with pneumothorax occupying < 1/3 of the pulmonary field). The risk of REPO is also increased by a delay in interventional treatment of more than 3 or 5 days after the onset of pneumothorax and the type of treatment, with more than 80% of the cases occurring in patients managed by suction draining (≥ 12 cm H2O). REPO is also more common in young adults between 20 and 39 years of age (23%) compared to patients 40 years of age or older (2%) [6, 8]. Therefore, in the case of our 26-year-old patient with a large pneumothorax managed with suction draining on the fifth day after the onset of symptoms, all the suggested risk factors for this complication were present.

The diagnosis of REPO is based on the clinical picture and imaging studies of the lungs. Symptoms develop in the first 24 hours following the procedure leading to re-expansion of the collapsed lung, usually in the first few hours post-procedure, and include worsening of the dyspnoea accompanied by a decreasing PaO2 and increasing (usually, and include worsening of the dyspnoea accompanying the re-expansion of the lung in with the underlying mechanism being abnormalities of vascular permeability similar to those observed in REPO.

High resolution computed tomography (HRCT) is helpful in the recognition and differential diagnosis of changes in the course of REPO. The typical changes, which were also present in our patient, include ground glass areas in the lungs and areas of parenchymal consolidation present in 50% of the cases, which are characterised by a “geographical” distribution. In almost 90% of the cases these changes show a predilection for peripheral regions of the lungs. This picture is not, however, pathognomonic for REPO and requires differential diagnosis that would first of all include all infections [9]. The radiological changes usually resolve within 1–10 days (within 4 days on average).

Treatment in symptomatic cases includes oxygen and diuretics. In severe cases, requiring invasive or non-invasive ventilation, continuous positive airway pressure (CPAP) can be helpful. Preventive measures in patients at risk of REPO is also recommended, such as the use of lower pressures for suction drainage (<10 cm H2O) or periodic interruptions of suction to slow down pulmonary re-expansion [3, 12].

References