

## Re-Expansion Pulmonary Edema Post-Pneumothorax

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### Abstract

### Case Report

Vacuum pulmonary edema is a rare yet potentially serious complication, associated with a mortality rate of 15 to 20%. It typically arises as an uncommon consequence of draining a pneumothorax or pleural effusion, presenting with symptoms such as cough, chest pain, and hypoxemia. In severe cases, it can lead to shock and even death, with symptoms generally appearing within 24 hours following thoracentesis. Management is primarily supportive, involving treatments that range from oxygen supplementation to both non-invasive and invasive ventilation techniques. Preventive measures include using low negative pressure ( $< -20$  cm H<sub>2</sub>O) during suction in thoracentesis and avoiding excessive pleural fluid drainage if the patient experiences chest discomfort. We present a case of a patient who developed vacuum pulmonary edema subsequent to the drainage of a pneumothorax.

**Keywords:** Re-Expansion Pulmonary Edema, Pneumothorax, Chest CT scan.

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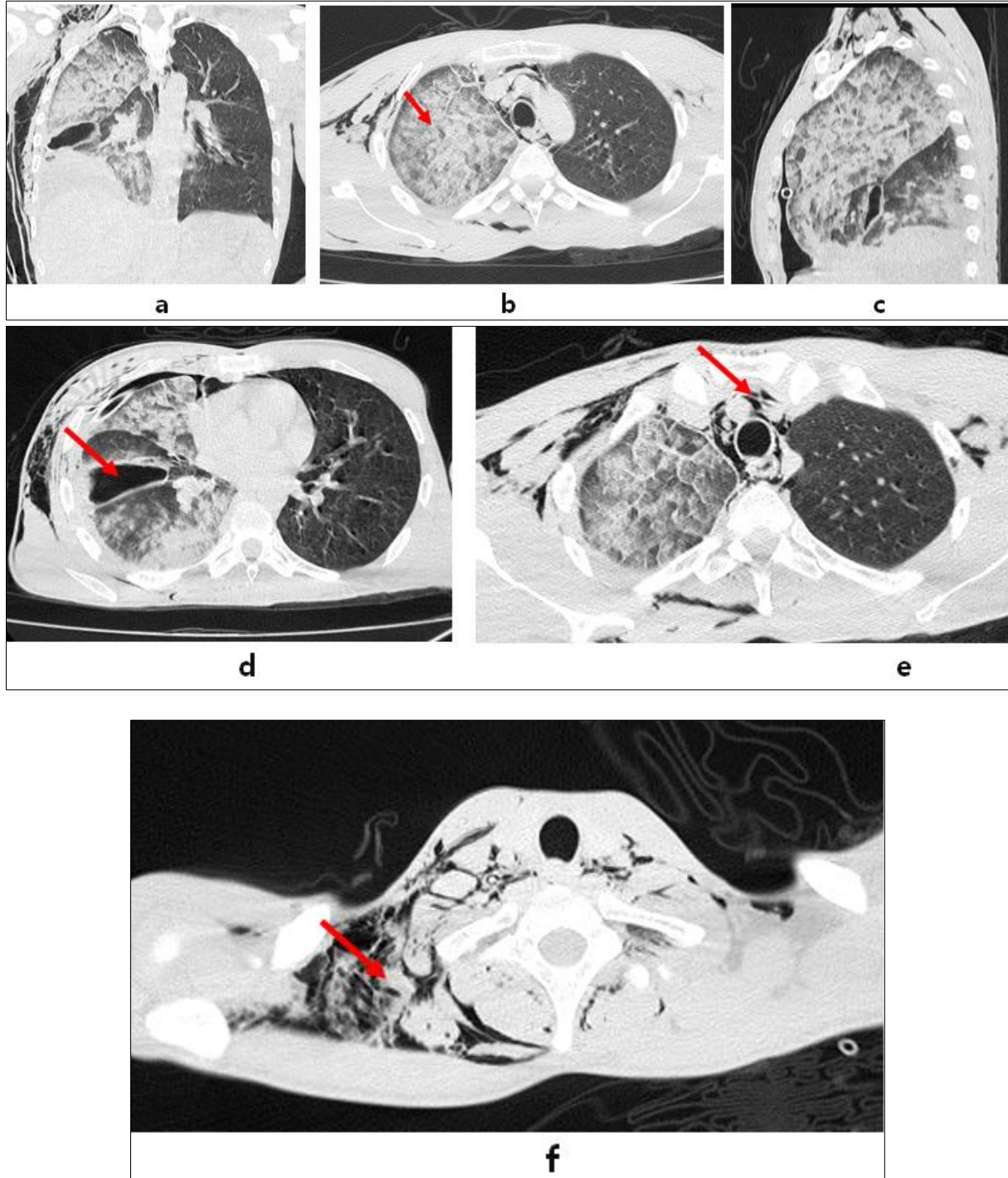
## INTRODUCTION

Re-expansion pulmonary edema (REPE) is a rare complication that can arise in patients with lung collapse, particularly in cases involving extensive and prolonged pneumothorax or pleural effusion. Following thoracentesis or thoracic tube drainage, the re-expansion of collapsed lungs can lead to acute lung edema, which may affect either one or both lungs within a short timeframe. While the reported incidence of REPE is less than 1% [1], it can have severe, potentially life-threatening repercussions, as indicated by several studies [2]. This article describes the case of a young male patient who experienced REPE following a traumatic pneumothorax.

## OBSERVATION

A 33-year-old man was admitted to the emergency department with sudden onset dyspnea. His medical history included chronic smoking (30 pack-years) and chronic cannabis use. Upon clinical examination, the patient was conscious but exhibited unstable respiratory function, with a respiratory rate of 35 breaths per minute. An initial chest X-ray revealed a large right pneumothorax. Biological tests indicated a white blood cell count of 10,000 and a C-reactive protein

level of 125. Emergency thoracic drainage was performed, and follow-up radiological monitoring showed a poorly defined alveolar opacity in the right upper lobe, along with moderate pneumothorax. A thoracic CT scan was conducted for further assessment, which demonstrated nodular and patchy intraparenchymal areas of ground-glass opacity and pulmonary consolidation in the right lung, sparing some segments of the middle lobe and left inferior lobe, with subpleural regions involved in some areas. There was also interlobular reticulation and thickening of the septal lines, creating a "crazy paving" appearance in places. The imaging findings included a small right hydropneumothorax with an encysted scissural pneumothorax on the same side, moderate right pleural effusion with passive atelectasis of the adjacent parenchyma, and moderate pneumomediastinum. No pulmonary nodules or micronodules were detected. Additionally, significant subcutaneous emphysema was observed, extending into the bilateral cervico-axillary and right thoracic soft tissues. These findings raise the suspicion of either pulmonary edema "a vacuo" or re-expansion edema occurring post-drainage. The patient was put on oxygen therapy with reduced pleural aspiration pressure. Evolution was favorable with respiratory stability.



**Figure 1: Thoracic angioscan, parenchymal window in axial, coronal and sagittal sections with MIP acquisition:**

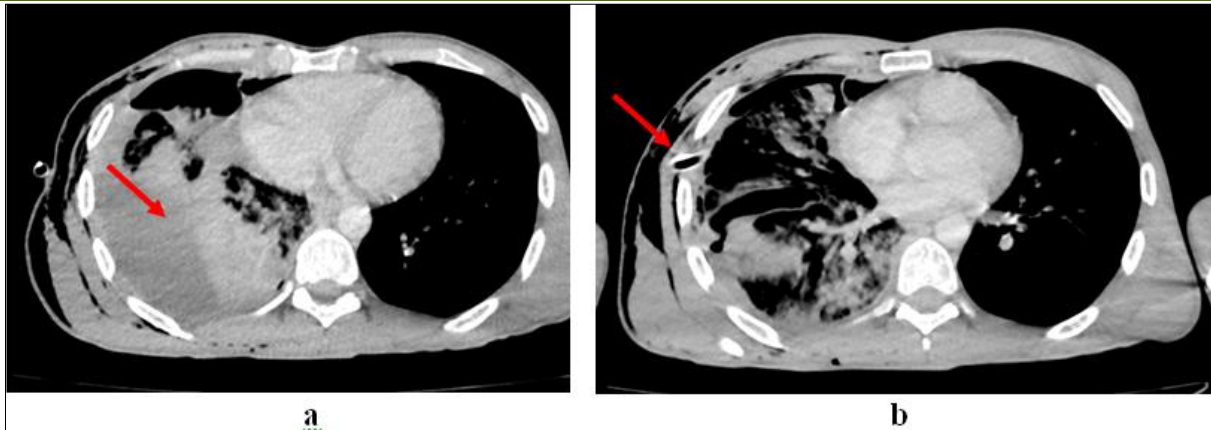
✓ **Figures a, b and c:**

Nodular and patchy intraparenchymal focus of ground-glass and pulmonary condensation in the right lung hemichamber, sparing some segments of the middle lobe and LID, and the subpleural regions in places, associated with inter-lobular reticulation and thickening of the septal lines giving the appearance of crazy paving in places.

✓ **Figure d:** Small right hydro-pneumothorax with homolateral encysted scissural pneumothorax.

✓ **Figure e:** Pneumomediastinum of moderate abundance.

✓ **Figure f:** Significant subcutaneous emphysema dissecting bilateral cervico-axillary and right thoracic soft tissue.



**Figures 2: Thoracic angioscan, mediastinal window in axial slices, showing:**

- ✓ **Figure a. Moderate right pleural effusion with passive atelectasis of adjacent parenchyma**
- ✓ **Figure b: Evidence of the distal end of the drainage probe at the ventral level of the LSD**

## DISCUSSION

Re-expansion pulmonary edema (REPE) is regarded as an iatrogenic complication that occurs when patients undergo rapid re-expansion of collapsed lungs, particularly following pleural effusion and pneumothorax. The first case of REPE was documented after thoracentesis in 1853, and subsequent reports emerged after the treatment of pneumothorax in 1958 [3]. Since then, numerous instances of REPE following pleural fluid aspiration and pneumothorax have been recorded. Although the exact pathophysiological mechanisms of REPE remain unclear, it is widely believed that increased vascular endothelial permeability and microvascular damage are significant contributing factors [4]. Collapse of the lung due to pleural effusion or pneumothorax can result in histological changes, such as thickening of the pulmonary microvasculature. When the lung is re-expanded, particularly after substantial or rapid drainage, mechanical stretching can injure the pulmonary microvasculature and elevate its permeability [5]. Therefore, it is advised that the volume of pleural effusion drained should not exceed 1–2 liters every 2 hours [6]. Inflammatory factors, such as interleukin-8 and reactive oxygen species, have also been implicated in the pathophysiology of REPE [7], while the role of pulmonary surfactant remains a topic of debate [8]. The diagnosis of REPE primarily relies on radiological findings [9]. Imaging studies may reveal ground-glass opacities or consolidations in the affected lung or in the contralateral lung, which can occur with or without respiratory symptoms. Common manifestations include cough, chest pain, and dyspnea. Despite the low reported incidence of REPE, it can have life-threatening consequences, emphasizing the need for heightened preventive measures. When draining pneumothorax, it is crucial to connect the drainage tube to an underwater-sealed device rather than directly to a negative-pressure suction system to facilitate monitoring of the drainage rate. During the early stages of drainage, close observation of the patient's signs and symptoms is

essential. Should the patient experience cough, chest pain, dyspnea, or a drop in oxygen saturation, drainage should be halted. Treatment for REPE is generally conservative and supportive, with approaches tailored to the patient's condition, including oxygen supplementation, diuretics, endotracheal intubation, and mechanical ventilation. It is important to reserve suction for pneumothorax cases requiring mechanical ventilation and to ensure that it is applied progressively. Clinical reports indicate that REPE following closed thoracic drainage after pneumothorax is rare and lacks a clear correlation with thoracic air volume, drainage velocity, or pleural pressure, yet it carries a high mortality rate [10]. The optimal rate and volume for pneumothorax drainage are not well-defined, but it is believed that pneumothorax is closely linked to REPE due to challenges in quantifying air volume. In this paper, we present a systematic account of a case of REPE following pneumothorax. While unexplained pneumothorax after trauma is relatively common, REPE resulting from closed thoracic drainage after pneumothorax is uncommon. Given the potentially severe and complex nature of REPE, careful prevention and consideration of treatment strategies for pneumothorax are essential. Pneumothorax management should be approached with greater caution than pleural effusion, as air drainage can be difficult to quantify and easily overlooked.

## CONCLUSION

Re-expansion pulmonary edema (REPE) is a rare complication that can arise in patients experiencing lung collapse. While the primary treatment involves oxygen therapy and minimizing pleural aspiration pressures, prevention remains the most effective strategy.

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