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# Re-expansion pulmonary edema after pleurocan catheterization: a case report

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## ABSTRACT

Re-expansion pulmonary edema (RPE) is a rare but potentially hazardous complication following evacuation of the pleural region. Symptomatic RPE occurs in less than 1% of patients after pleural drainage. Early diagnosis and treatment determines the progression of the disease and it is life-saving. The present case describes a 68-year-old man who developed RPE with the ipsilateral collapsed lung 6 hours after pleural drainage of a non-malignant effusion. He was intubated and 6 hours after aggressive treatment with mechanical ventilation support oxygenation was improved and the patient's blood gas analysis recovered. Over the course of his 12-day hospitalization, he was extubated and oxygen support was slowly weaned down. Mortality rate of RPE in severe cases is approximately 20%, therefore preventive interventions gain importance. In spite of the rare incidence of RPE, being aware of this potential condition can allow for early and proper management.

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***Keywords:*** Re-expansion pulmonary edema, pleural catheter, intensive care

## Introduction

Re-expansion pulmonary edema (RPE) is a rare but potentially hazardous complication which may occur in every kind of chronically collapsed lung, following evacuation of the air or fluid from the pleural region. Most cases are clinically mild and detected incidentally on radiography so the true incidence is still skeptical (0.9-20%), but symptomatic RPE occurs in less than 1% of patients after pleural drainage [1].

Mostly, RPE is limited to the ipsilateral lung after relief of collapse and the main signs are tachypnea, tachycardia, and crackles on the affected side of the lung. Generally, a chest radiograph is sufficient for diagnosis, but the suspect of the clinician is essential. The radiographic diagnosis includes the presence of opacities in the previously collapsed lung, following thoracentesis. Because of the clinical and radiological diversity of RPE, cardiogenic pulmonary edema, pulmonary infections and pneumonitis should be kept in mind in the differential diagnosis. Early diagnosis and treatment determines the progression of the disease and it is life-saving [2]. The present case describes a 68-year-old man who developed RPE with the ipsilateral collapsed lung 6 hours after pleural drainage of a non-malignant effusion.

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## Case Presentation

A 68-year-old man with cholangiocellular carcinoma diagnosis who had been operated in our hospital followed up for respiratory distress. He had progressive dyspnea over the course of several days. The initial radiographic assessment revealed massive pleural effusion in the right hemithorax (Figure 1). At first, an 8.3-French pig tail catheter drainage for the pleural effusion was performed and the cytology findings of the effusion were negative for malignancy. Then pleurocan catheter was implanted under local anesthesia and the initial amount of drainage was serosanguinous fluid of 1000 ml in 1 hour period. The patient tolerated the procedure well and his symptoms improved. However, 6 hours after the pleural drainage the clinical manifestation of tachypnea, dyspnea and tachycardia was observed, and his oxygen saturation dropped to 80% on room air. A chest radiograph taken at that time showed bilateral opacities resembling ARDS, suggesting RPE in the whole lung field (Figure 2).

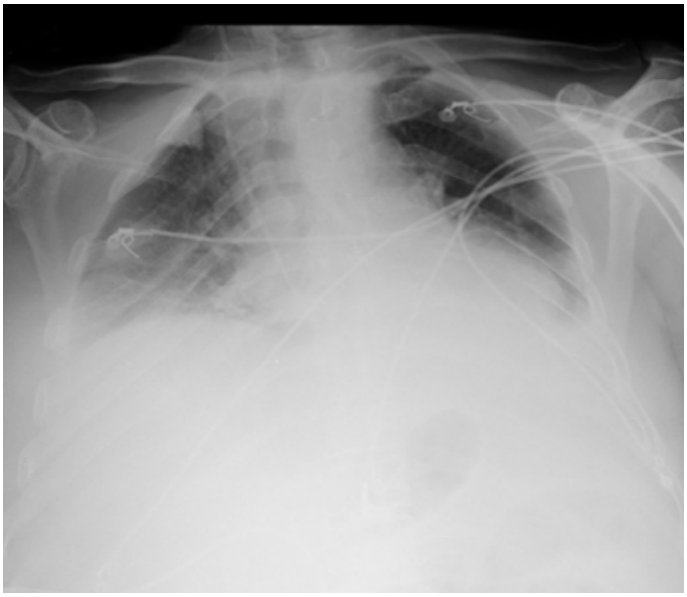

**Figure 1**. Massive pleural effusion in the right hemithorax.

As a conservative treatment fluid intake was restricted, diuretic and supportive oxygen therapy started. Cardiogenic edema was completely excluded by the mean of transthoracic echocardiography examination. More aggressive diuresis was avoided to protect from hypotension and cardiovascular collapsed in the setting of RPE.

The patient clinic situation worsened with extensive crackles in the bilateral lung auscultation. Blood gas analysis of the patient revealed pH: 7.13,

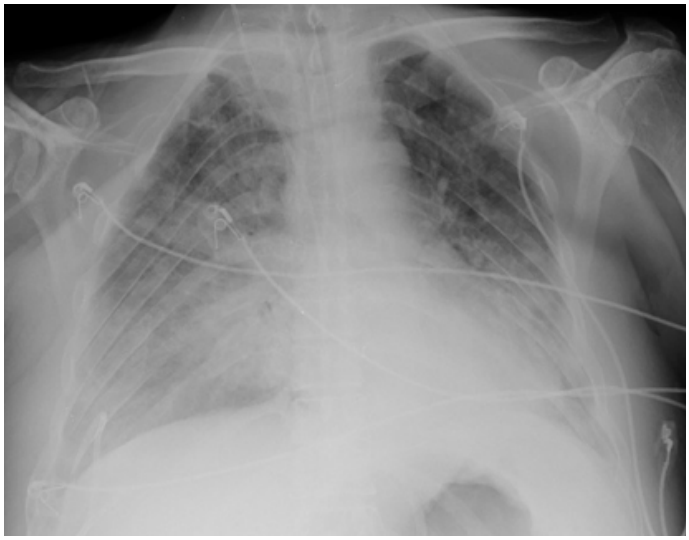

**Figure 2**. A chest radiograph taken at that time showed bilateral opacities resembling ARDS, suggesting re-expansion pulmonary edema in the whole lung field.

PaCO2: 56.4 mmHg, PaO2: 36.2 mmHg, HCO3: 18 mEq / L, base deficit: (-7) and orotracheal intubation became inevitable. He was placed on pressure-regulated pressure control with FiO2: 80%, PEEP: 8 cmH2O, frequency: 15/min and pressure support: 18 cmH2O. Six hours after this aggressive treatment with mechanical ventilation support oxygenation was improved and the patient's blood gas analysis recovered (pH: 7.42, pCO2: 28.4 mmHg, pO2: 104 mmHg). Ventilator settings were weaned to a PEEP of 5 and FiO2 of 40%. It was observed that the chest x-ray findings also recovered within 24 hours and the RPE resolved (Figure 3). Over the course of his 12-day hospitalization, he was extubated and oxygen was slowly weaned down.

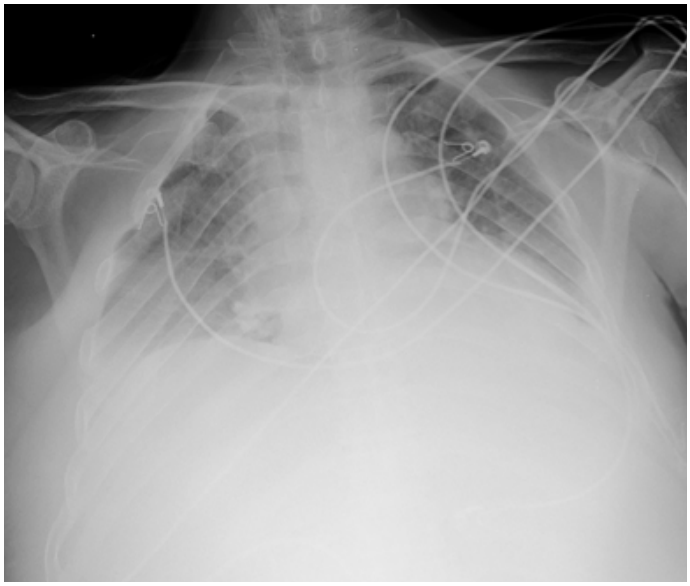

**Figure 3**. Chest x-ray findings also recovered within 24 hours and the re-expansion pulmonary edema resolved.

## Discussion

Mostly, RPE is limited to the ipsilateral collapsed lung after relief of collapse and occurs within 24 hours. The clinical appearance of RPE can range from asymptomatic radiographic findings to fatal hypoxia. Although there is no consensus for exact mechanism for RPE, major risk factors that have been proposed are rapid re-expansion, drainage with the use of negative intrapleural pressure, and chronicity of lung collapse [3]. Increased permeability of the pulmonary capillaries as a result of inflammation is mainly accused for pathophysiology [2]. In some studies, it was shown that ventilation and reperfusion of a previously collapsed lung may lead to an inflammatory response increasing capillary permeability and results in RPE [4].

Pressure-induced mechanical disruption of the alveolar capillaries, and altered lymphatic clearance are the other accused factors for RPE development. We speculate that in our case, those factors and surgical stress during abdominal operation may have induced a subclinical pulmonary inflammation, that may have ensured a second impact mechanism for the development of the RPE. Even though these factors might partake to the formation of RPE, maybe none of them is indispensable. This might explain the difficulty of prediction in the occurrence of RPE [5].

Early recognition is essential in ensuring successful treatment of RPE and the anchor of treatment remains generally conservative and supportive like sufficient oxygen supplementation, while in severe cases more intensive treatments such as mechanical ventilation is required [3]. In patients requiring orotracheal intubation and mechanical ventilation support, as in our case, positive end expiratory pressure (PEEP) usage improves symptoms after 24-48 h. There is often underestimation of the morbidity and mortality associated with pleural interventions . Mortality rate of RPE in severe cases is approximately 20%, therefore preventive interventions, including the use of low negative pressure for suction and limiting drainage to about 1 to 1.5 L of pleural fluid gain importance, though the data is limited. [6, 7].

## Conclusions

In spite of the rare incidence of re-expansion pulmonary edema, being aware of this potential condition can allow for early and proper management.

*Informed consent*

Written informed consent was obtained from the patient for the publication of this case report.

*Conflict of interest*

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

*Authors' contributions*

All authors participated in the design of the case report and coordination and helped to draft the manuscript. All authors read and approved the final manuscript.

## References

[1] Dias OM, Teixeira LR, Vargas FS. Reexpansion pulmonary edema after therapeutic thoracentesis. Clinics 2010;65:1387-9.
[2] Kim JJ, Kim YH, Choi SY, Jeong SC, Moon SW. Contralateral reexpansion pulmonary edema with ipsilateral collapsed lung after pleural effusion drainage: a case report. J Cardiothorac Surg 2015;10:68.
[3] Verhagen M, van Buijtenen JM, Geeraedts LMG. Reexpansion pulmonary edema after chest drainage for pneumothorax: A case report and literature overview. Respir Med Case Rep 2015;14:10-12.
[4] Kasmani R, Irani F, Okoli K, Mahajan V. Re-expansion pulmonary edema following thoracentesis. CMAJ 2010;182:2000-2.
[5] de Wolf SP, Deunk J, Cornet AD, Elbers PW. Case Report: Bilateral reexpansion pulmonary edema following treatment of a unilateral hemothorax. Research 2014:3:318.
[6] Corcoran JP, Psallidas I, Wrightson JM, Hallifax RJ, Rahman NM. Pleural procedural complications: prevention and management. J Thorac Dis 2015;7:1058-67.
[7] Özsin KK, Toktaş F, Sanrı US, Kahraman N, Yavuz Ş. Re-expansion pulmonary edema caused by pleural effusion drainage in late period after coronary artery bypass surgery: case report. Turkiye Klinikleri Arch Lung 2017;18:25-8.