



doi • 10.5578/tt.69524

Tuberk Toraks 2020;68(2):188-191

Geliş Tarihi/Received: 23.04.2020 • Kabul Ediliş Tarihi/Accepted: 13.05.2020

OLGU SUNUMU  
CASE REPORT

# A very rare fatal complication: bilateral reexpansion pulmonary edema after treatment of single side total pneumothorax

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\* Our case was presented as a e-poster at the 2018 TÜSAD congress.

## ABSTRACT

### A very rare fatal complication: bilateral reexpansion pulmonary edema after treatment of single side total pneumothorax

Reexpansion pulmonary edema is a rare but fatal clinical condition that develops during the treatment of pneumothorax, pleural effusion and collapsed lung after atelectasis. A 31-year-old male patient was referred to our clinic with the complaint of stinging back pain that started 3 days ago and dyspnea developed during the last 24 hours. Physical examination and radiologic examinations revealed total pneumothorax in the right hemithorax. After tube thoracostomy, his general condition deteriorated and bilateral reexpansion edema developed in the lungs. The patient was admitted to the intensive care unit and was discharged on the 5<sup>th</sup> day after medical treatment. Our case is the first case of bilateral reexpansion pulmonary edema seen after unilateral spontaneous pneumothorax when literature review is performed. In order to emphasize the importance of rapid diagnosis and treatment, it is presented in the light of the literature.

**Key words:** Pneumothorax; reexpansion pulmonary edema; bilaterally; fatal complication

**Cite this article as:** Dumanlı A, Günay E, Öz G, Aydın S, Çilekar Ş, Gencer A, et al. A very rare fatal complication: bilateral reexpansion pulmonary edema after treatment of single side total pneumothorax. Tuberk Toraks 2020;68(2):188-191.

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**ÖZ****Çok nadir görülen ölümcül bir komplikasyon: tek taraf total pnömotoraks tedavisinden sonra gelişen bilateral reekspansiyon akciğer ödemi**

Reekspansiyon akciğer ödemi; pnömotoraks, plevral efüzyon ve atelektaziye sekonder olarak, kollabe olmuş akciğerin tedavisi sırasında gelişen nadir görülen, ancak ölümcül olabilen klinik bir durumdur. Otuz bir yaşında erkek hasta üç gün önce başlayan batıcı tarzda sırt ağrısı ve son 24 saatte gelişen nefes darlığı şikayeti ile kliniğimize refere edildi. Yapılan incelemelerde sağ hemitoraksta total pnömotoraks saptandı. Tüp torakostomi uygulandıktan hemen sonra genel durumu kötüleşti ve bilateral akciğerlerde reekspansiyon ödemi gelişti. Yoğun bakıma alınan hasta medikal tedavi sonrası beşinci gün komplikasyonsuz olarak taburcu edildi. Olgumuz, literatür araştırması yapıldığında, tek taraflı spontan pnömotoraks sonrası olarak görülen ilk bilateral reekspansiyon akciğer ödemi olgusudur. Çok nadir görülmesi, hızlı tanı ve tedavinin hayati önemini vurgulamak amacıyla literatür eşliğinde sunuldu.

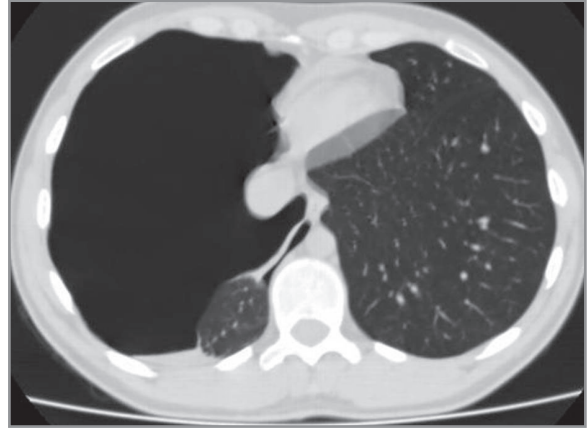
**Anahtar kelimeler:** Pnömotoraks; reekspansiyon akciğer ödemi; bilateral; ölümcül komplikasyon

**INTRODUCTION**

Reexpansion pulmonary edema (RPE) is a complication that occurs during the treatment of long-standing collapsed lung. It usually occurs during the rapid treatment of total pneumothorax or massive pleural effusion. It is seen rare but can cause to death. Mortality rate is reported to be approximately 20%, especially in bilateral and sudden onset RPE. Its pathophysiology is not fully known. It has been blamed for a series of inflammatory reactions that lead to decreased surfactant production and increased endothelial permeability in the lung. These reactions are associated with increased cytotoxic oxygen metabolites as a result of neutrophil flux (1). Early diagnosis and treatment determines disease progression and is life-saving (2-5). Bilateral lung re-expansion pulmonary edema is, as would be expected, more severe and fatal than unilateral re-expansion pulmonary edema. According to our researches, although very rare, bilateral RPE can be seen after treatment of massive pleural effusion, hemothorax and decortication operation. Our case is the first case of bilateral RPE after spontaneous pneumothorax and is presented in the light of literature.

**CASE REPORT**

A 31-year-old male patient was admitted to the emergency department with complaints of stinging back pain and shortness of breath that developed over the last 24 hours. The arterial blood pressure was 115/75 mmHg, pulse 115 /min, fever 36.9°C, respiratory rate was 24 /min and O<sub>2</sub> saturation was 89%. In the physical examination, respiratory sounds decreased in the right hemothorax. Total pneumothorax was detected in the right hemithorax on computerized thorax tomography and was referred to our clinic (Figure 1). Patient's general condition deteriorated immediately after tube thoracostomy. Blood pressure arterial 95/55 mmHg, pulse 143 /min, fever 37.1°C, respiratory rate

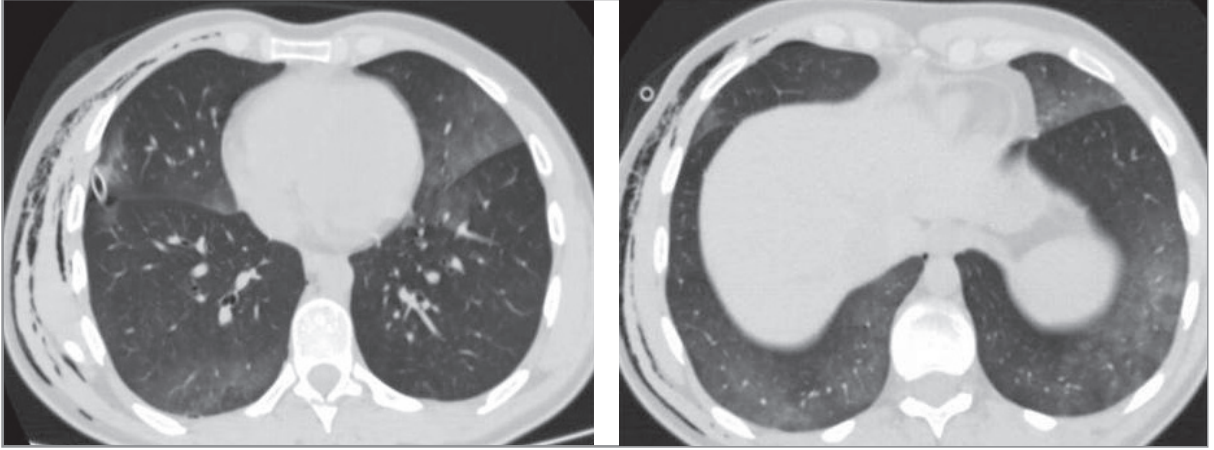


**Figure 1.** Right total pneumothorax.

33 min, O<sub>2</sub> saturation was 86%. Blood gas; pH 7.33, PaCO<sub>2</sub> 29.1 mmHg, PaO<sub>2</sub> was 63.9 mmHg. Radiological imaging was performed again and the patient was admitted to the intensive care unit considering bilateral RPE (Figure 2). Corticosteroid, diuretic, oxygen (4 L/min) treatment was given and intravenous fluid restriction was performed. Vital signs improved 48 hours after treatment without intubation. The control chest X-ray was repeated five days after admission and he was discharged with healing.

**DISCUSSION**

RPE was first described in 1958 by Carlson et al. (6,7). RPE is an important complication to keep in mind in patients requiring tube thoracostomy. The incidence of RPE is reported to be between 1-14% (5). The etiology is not fully known (2,7). However, it is thought that the cause is a destructive pathological process that develops as a result of increased endothelial permeability (2). In this process, the alveolo-capillary membrane is damaged and the ischemia-reperfusion injury cycle begins (6). Prolonged collapse (> 72 hours) leads to hypoxemia in the lungs, damage to the capillary wall, and consequent-



**Figure 2.** Bilateral reexpansion pulmonary edema.

ly reduced surfactant production. Rapid opening of the collapse causes sudden and rapid blood flow to this area and sudden distension of the alveoli (3). In this process, which results in increased capillary permeability and hydrostatic pressure, fluid flows into the alveoli and interstitium with high protein content and edema develops (5).

Although RPE usually occurs in the collapsing lung, it can rarely be seen in bilateral lungs (5,8-10). Therefore, vasoactive substances such as histamine, prostaglandin or neurogenic factors are thought to be effective in the development of RPE (11). Wolf et al. after unilateral hemothorax treatment and Paksu et al. also after empyema treatment has reported bilateral RPE (9,10). Our case is the first RPE developed after single-sided total pneumothorax treatment.

Collapses lasting more than three days and rapid drainage of excess pleural fluid (> 1500 mL) are risk factors for the development of RPE (3). Other risk factors for RPE include pulmonary hypertension that changes alveolar dynamics and impaired gas exchange hypoxemic conditions (12). Although RPE causes fatal respiratory failure, it may also spontaneously regress. Cobanoğlu et al. has reported in their study that 11 (21%) of 53 cases died (5). Our patient was discharged five days after hospitalization due to early diagnosis and treatment.

Monitoring of the patient, invasive or noninvasive hemodynamic support, oxygen supply, diuresis, discontinuation or reduction of fluid intake and the use of inotropic agents are vital in the treatment of RPE. Prevention of RPE depends on carefully pleural evacuation procedures.

### CONFLICT of INTEREST

The authors reported no conflict of interest related to this article.

### AUTHORSHIP CONTRIBUTIONS

Concept/Design: AD

Analysis/Interpretation: EG, AG

Data Acquisition: EG, GÖ

Writing: AD, SA

Critical Revision: ŞÇ, AB

Final Approval: İGÇ, AB

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