A Case of Pneumothorax in Which Progression of Reexpansion Pulmonary Edema Was Arrested

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Abstract
We report a case of pneumothorax secondary to emphysema in which we arrested reexpansion pulmonary edema. The patient was a 58-year-old male. A chest drainage tube was inserted to manage the secondary left pneumothorax, but further aggravation of dyspnea and frothy airway secretions were observed shortly after drainage. Because reexpansion pulmonary edema was suspected, we intentionally chose to perform oxygen administration and opening of the drain. As symptoms subsided, the patient was put in a right lateral decubitus, and gradual expansion of the lungs was attempted. As chest X-ray showed suspected edema in the left lung, a potent diuretic was administered. Since no exacerbation of symptoms or decrease in transcutaneous arterial oxygen pressure was noted, the patient was kept under observation without requiring further medication. In a clinical situation where a patient shows aggravation of dyspnea and appearance of frothy sputum during reexpansion of collapsed lungs, it is critical to prevent the development of fatal reexpansion pulmonary edema by arresting rapid expansion of the lungs and avoiding the increase in blood flow into the reexpanding lungs.

Key words: Pulmonary edema, Pneumothorax, Pulmonary blood flow, Blood vessel permeability, Dyspnea, Frothy sputum

Introduction
Reexpansion pulmonary edema (RPE) is a type of pulmonary edema that may develop when collapsed lungs are reexpanded. It is a rare but fatal condition. RPE is less likely to occur during reexpansion after a short period of pulmonary collapse, and the incidence increases after prolonged pulmonary collapse.1 In this article, we report a case in which we arrested the development of RPE following chest drainage in a patient with pneumothorax secondary to pulmonary emphysema.

The Case
The patient was a 58-year-old man, who had been treated by a local physician for pulmonary emphysema. Precordial pain and dyspnea developed about 9 a.m., and the patient visited our hospital at about 16:00 of the same day because of aggravating dyspnea. Chest X-ray revealed a left pneumothorax (Fig. 1), which was considered to have been present for about 7 hours. A chest drainage tube was inserted to manage the secondary pneumothorax, but further aggravation of dyspnea and the sudden appearance of frothy airway secretions were observed shortly after drainage. Because RPE was suspected, we intentionally chose to open the chest tube to release the negative pressure in the left thoracic cavity for the purpose of preventing mechanical damage to pulmonary alveoli and avoiding the increase in pulmonary blood flow that might result from the rapid reexpansion of the lungs.
Oxygen was administered and the patient was put in a sitting position. As the symptoms subsided in about 10 minutes, the patient was put in a right lateral decubitus, and gradual expansion of the lungs was attempted by water-seal drainage and repeated drain clamping. During drain clamping, the development of subcutaneous emphysema was prevented by strong compression on the drain insertion site. As chest X-ray showed suspected edema in the left lung (Fig. 2), a potent diuretic was administered. Since no exacerbation of symptoms or decrease in transcutaneous arterial oxygen pressure was noted, the patient was kept under observation without requiring further medication. CT examination on the following day did not indicate apparent aggravation of signs suggesting pulmonary edema (Fig. 3). Although the complete disappearance of ground-glass opacities in the lungs was achieved subsequently, the pulmonary air leak did not disappear. Consequently, thoracoscopic partial lung resection under general anesthesia was performed 11 days after drainage. Postoperative examinations demonstrated the disappearance of pulmonary air leak and the absence of RPE. The chest tube was removed 2 days after operation. As of the time of this writing, 7 months after surgery, the patient has been doing well without any relapse of the pneumothorax or the development of pulmonary edema.

Discussion

Treatment methods for pneumothorax include rest, thoracocentesis, chest drainage, and surgery. The guidelines and consensuses in such countries as the U.S. and the U.K. state that chest drainage is the method of choice for secondary pneumothorax resulting from pulmonary emphysema or pulmonary fibrosis. In the present case, we selected hospitalization and chest drainage following these examples.

RPE generally tends to develop when the
lungs are reexpanded rapidly after a long period of pulmonary collapse. This fact suggests the involvement of the mechanical and chemical damage to pulmonary microcirculation during prolonged pulmonary collapse, resulting in enhanced blood vessel permeability. In the present case, RPE developed as shortly as about 7 hours after the onset of symptoms. Although the cause of this early development of RPE is unknown, a reasonable explanation is that damage to pulmonary alveoli and microcirculation might have existed as a result of pulmonary emphysem. Rapid reexpansion of collapsed lungs is considered likely to induce RPE, as it promotes the influx of pulmonary arterial blood. In our case, early symptoms of RPE such as aggravation of dyspnea and appearance of frothy sputum were observed despite the fact that the lungs were reexpanded gradually using the water-seal method after chest tube insertion. This observation coincides with various authors reporting that RPE can develop even during gradual reexpansion of the lungs, as the development of RPE is dictated by blood vessel permeability and pulmonary blood flow. For this reason, we should keep the patient under close observation for a certain period after chest tube insertion, remembering that RPE can develop in any situation. RPE is most likely to occur during the 24 hours, particularly during the first hour, after reexpansion of the lungs, and care should be taken not to leave the patient unattended during this period.

Once RPE has become established, the condition should be treated with diuretics, cardiotonics, and bronchodilators, as well as artificial respiration management. The aggravation of RPE may be prevented by the timely detection of early symptoms and stopping pulmonary blood flow into the reexpanding lungs. After these measures are taken, medication to reverse the enhanced permeability of blood vessels in the lungs should be selected appropriately. In the present case, we attempted to address the aggravation of dyspnea shortly after chest drainage by opening the chest drain to release the negative pressure in the left thoracic cavity, induce the collapse of the left lung and blood vessels therein as much as possible, and limit the blood flow into the left lung. In this procedure, the open end of the drain tube was protected with gauze to reduce the risk of retrograde infection. Subsequently, the patient was put in a sitting position and oxygen was administered to improve dyspnea and lessen anxiety of the patient, and then he was put in a right lateral decubitus as promptly as possible. The purpose of this maneuver was to use gravitation for increasing blood flow in the right lung and decreasing that in the left lung. When complete collapse of the left lung is hindered by intrathoracic adhesion as in this case, it is impossible to stop the blood flow to the lower left lung field as long as the patient is in a sitting position.

Our judgment that the patient was about to develop RPE was based on clinical and imaging findings. We cannot say for sure whether or not the patient would have developed RPE without the treatment described above. However, fully developed RPE is often fatal, and treatment of the patients after successful life saving would require much time. We report this case to emphasize the fact that physicians performing thoracic drainage should always consider the possibility of RPE, and any aggravating signs or symptoms observed after drainage should be treated promptly.

References