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Reexpansion Pulmonary Edema after Drainage of a Spontaneous Pneumothorax

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Abstract: We report a case of life-threatening reexpansion pulmonary edema following chest tube drainage of spontaneous pneumothorax. Reviewing the literature, pathophysiology, symptoms and therapy of this complication were discussed. Physicians must be aware of the occurrence of reexpansion pulmonary edema following reinflation of the collapsed lung.

Key Words: reexpansion pulmonary edema, pneumothorax, chest tube drainage

Reexpansion pulmonary edema is a rare complication that occurs following drainage of spontaneous pneumothorax (1), thoracentesis of a large amount of pleural effusion (2) or removal of a huge intrathoracic tumor (3).

We report a case of life-threatening reexpansion pulmonary edema following insertion of a chest tube for spontaneous pneumothorax.

Case report

A 40-year-old male was admitted with complaint of 20 day history of exertional dyspnea, cough and sputum. He had no previous history of lung disease. Physical examination revealed a blood pressure of 114/70mmHg, pulse of 114 and respiratory rate of 42. The percussion note was hyperresonant and the breath sounds were decreased over the left side of the chest. No heart murmur was audible. An admission chest roentgenogram revealed left pneumothorax and complete collapse of the left lung with shift of the mediastinum to the right (Fig. 1). An intercostal chest tube was inserted in the 5th intercostal space in the midaxillary line without difficulty and suction was applied utilizing a negative 8cm H₂O pressure. Immediately after drainage, cough appeared but subsided during next 15 minutes. The patient was placed on supplemental oxygen with a face mask. However, 30 minutes after chest tube insertion he com-

plained of severe dyspnea and expelled frequently copious amount of frothy yellow sputa. He was cyanotic and inspiratory rales were audible diffusely on the left side. A repeat chest roentgenograph showed the left lung fully expanded and diffuse opacification in the left upper lung field (Fig. 2). A reexpansion pulmonary edema was suspected. Suction to the chest tube drainage was discontinued to underwater seal only. The patient was given 100% oxygen, and intravenous administration of crystalloids, diuretics, steroid and inotropic agents. His condition became deteriorated, cyanosis increased and inspiratory rales became audible over the right side of the chest. Endotracheal intubation was instituted. The patient's blood pressure has fallen to 60mmHg systolic. The chest roentgenogram revealed contralateral pulmonary opacification (Fig. 3). Forty minutes after tracheal intubation, apnea and profound hypotension appeared despite aggressive use of pressors and intravenous fluids. External cardiac massage and artificial respi-

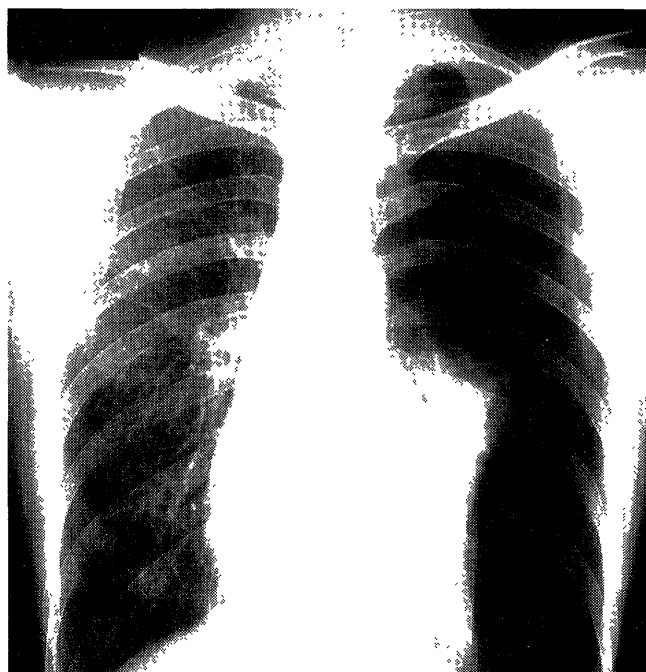


Fig. 1. Chest radiograph on admission showing left pneumothorax.

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Fig. 2. Chest radiograph one hour after insertion of chest tube showing unilateral left sided pulmonary edema.

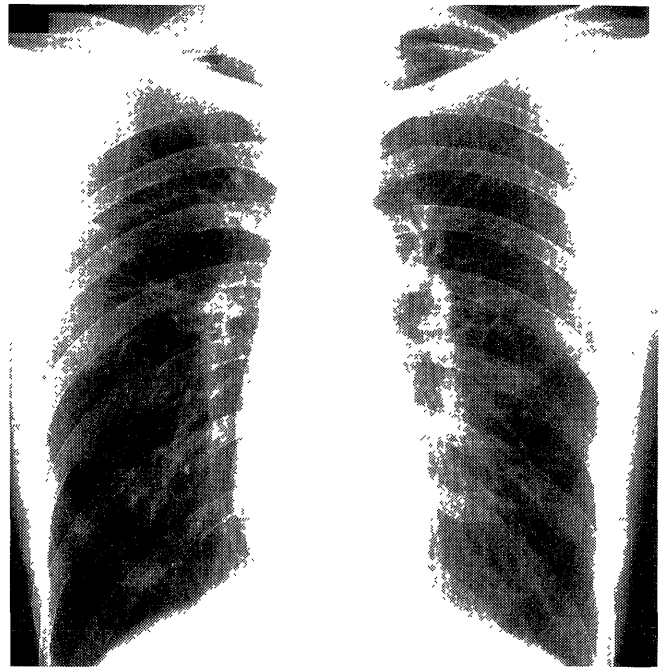


Fig. 3. Chest radiograph after intubation showing contralateral pulmonary opacification.

ration were started. He was resuscitated 10 minutes later. Mechanical ventilation was instituted and inotropic agents and crystalloids were continued. Blood pressure was 72/50mmHg and pulse rate 150. Foamy yellow sputa decreased gradually and cyanosis was improved. Twenty-five hours after chest tube insertion, he was weaned from respirator and the tracheal tube was removed. The blood pressure was 111/75mmHg, pulse rate 85 and respiration 20. The arterial blood gas values on seven liters of oxygen by face mask revealed PH 7.46, PaO₂ 67.6 and PaCO₂ 40.8. The chest roentgenogram showed decrease of opaque shadow in the left lung. Two days after chest tube insertion, chest x-ray revealed increase of left pneumothorax. Then, suction was re-established but bradycardia and ventricular premature capture occurred. Again water-seal drainage was established for the next four days. The chest roentgenogram 6 days later showed resolution of the pulmonary infiltrate. Suction drainage of 6cm H₂O was started and continuous airleakage from the chest tube was observed. Arterial blood gas values on room air were PH 7.47, PO₂ 84mmHg and PCO₂ 42mmHg. After pleurodesis with minocyclin, air leakage was ceased with complete reflation of the left lung. The chest tube was removed one month later after drainage (Fig. 4). His general condition is well and recurrence of pneumothorax was not observed 18 months later.

Comment

Reexpansion pulmonary edema following pneumothorax

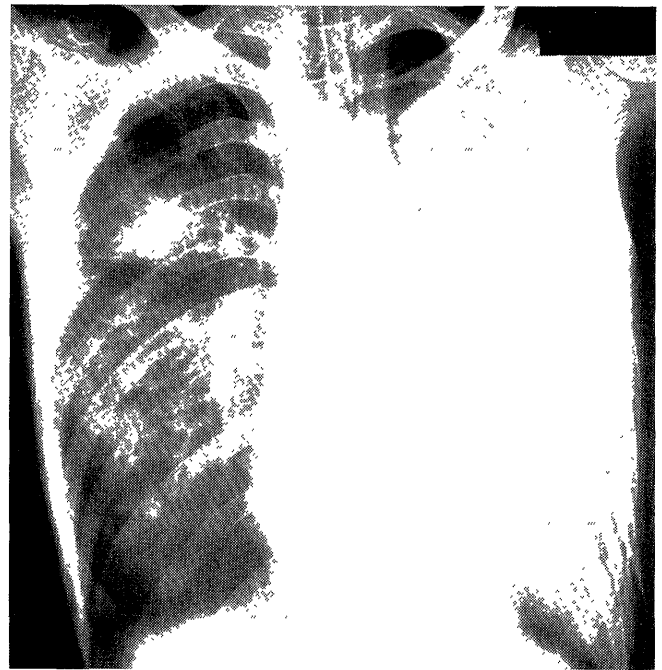


Fig. 4. Chest radiograph after removal of chest tube showing complete reflation of the left lung.

was first reported in 1958 by Carlson and his associates (4). The pathogenesis of pulmonary edema following reexpansion of a pneumothorax is unclear. Application of negative intrapleural pressure, decreased surfactant content, bronchial obstruction, pulmonary artery pressure changes and increased pulmonary vascular permeability were reported to contribute to the development of pulmo-

nary edema (5, 6). Recent evidence from experimental and clinical observations supports increased pulmonary permeability as a major etiologic factor. Marland and Glauser (7) described high concentration of total proteins in edema fluid and a normal pulmonary capillary pressure in a patient with pulmonary edema after removal of a large pleural effusion, which suggested that increased pulmonary capillary permeability rather than a hydrostatic mechanism was responsible for reexpansion pulmonary edema. In the previous reports, anoxic damage to the alveolar-capillary basement membrane has been suggested by many authors (1, 5, 6, 8, 9). However, the recent studies suggest that a reoxygenation injury with the lung tissue producing excess superoxide and other toxic metabolites such as protease cause the endothelial cell injury and the increased permeability of pulmonary capillaries (10, 11).

The etiology of the appearance of contralateral pulmonary opacification in our case was unknown. There have been 3 reported cases of contralateral pulmonary edema following reexpansion of pneumothorax (8). Only one case with bilateral reexpansion pulmonary edema was reported following pleurocentesis (12). Opacification of the contralateral lung could be resulted from spillage of edema fluid from the reexpanded ipsilateral lung or concomitant pulmonary edema. Contralateral pulmonary edema following unilateral reexpansion of pneumothorax has been reported experimentally, and neuro-humoral factors released from the ipsilateral lung were suggested to produce this complication (13).

Predisposing factors in the development of reexpansion pulmonary edema were pneumothorax more than 3 days, complete lung collapse and rapid re-expansion of the collapsed lung (6, 14). However, as documented in the literature, reexpansion pulmonary edema can develop in any collapsed lung, regardless of the duration of the lung collapse and without suction (15, 16). The patient who has a pneumothorax of over three days duration should have a chest tube attached to an underwater seal. The applications of negative pressure should be deferred for several hours and reexpansion encouraged by coughing and chest physiotherapy. The reexpansion pulmonary edema usually begins within minutes to hours after chest tube drainage and the outcome varies from roentgenographic findings alone to mild or severe cardiorespiratory insufficiency, shock, coma, and death (17). Mahfood and his associates (6) reported that re-expansion pulmonary edema was fatal in 20% of cases. Depending on the severity of clinical symptoms, treatment varies from monitoring of serial chest films to intubation and mechanical ventilation as observed in our case. Some patients received no specific therapy; others were treated with a variety of modalities and drugs, including oxygen, inotropic agents, digitalis, steroids, diuretics, bronchodilators and sedatives. Hypovolemia can occur due to sequestration of a large volume of fluid in the lung in the

patient with reexpansion pulmonary edema. Hypotension and low cardiac output must be managed by volume replacement and inotropic agents with careful hemodynamic monitoring. The independent artificial ventilation by using two respirators might be indicated in desperate situations (11).

It usually occurs unexpectedly and dramatically following reexpansion of the collapsed lung, the physician who manages the patient with a spontaneous pneumothorax, must be aware of reexpansion pulmonary edema (18).

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