Reexpansion Pulmonary Edema a Rare Complication after Intercostal Drainage Tube for Pneumothorax: A Case Report and Literature Overview

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ABSTRACT

In the emergency room, a middle-aged female patient with pneumothorax received treatment with an intercostal drainage tube. After a while, she experienced reexpansion pulmonary edema and was moved to the intensive care unit to receive more ventilatory assistance. After a while, the edema and dyspnea subsided, and the patient was released with good clinical results.

Rapidly progressing hypoxemia and respiratory discomfort following an intercostal drainage tube are the hallmarks of reexpansion pulmonary edema. Early detection of the symptoms and signs of reexpansion pulmonary edema is crucial Early intervention may result in the best outcome.

Keywords: Pneumothorax, Pulmonary edema, Intercostal drainage tube,Reexpansion

1. INTRODUCTION

Reexpansion pulmonary edema is an uncommon but potentially fatal illness that can arise when a collapsed lung expands quickly after a tube thoracostomy is performed to drain fluid or air from the pleural space (pleural effusion or pneumothorax). Increased permeability of pulmonary capillaries, which harmed by mechanical stress during lung reexpansion, is the cause of reexpansion pulmonary edema. Increased capillary permeability may also brought on by reperfusion injury brought on by free radicals. Symptomatic therapy should produce positive results.

Following the installation of an intercostal drainage tube to treat pneumothorax, the patient developed reexpansion pulmonary edema, which we summarise below. This condition is a

relatively uncommon, potentially fatal intercostal drainage tube problem. The best results would come from early detection of this problem.

2. CASE REPORT

2a. Patient information

A middle-aged woman who had been coughing continuously for the previous week arrived at the emergency room in respiratory distress. Uncomfortable chest pain on the left side followed these concerns. Nothing odd has been found in the family history or the patient's history of respiratory illness.

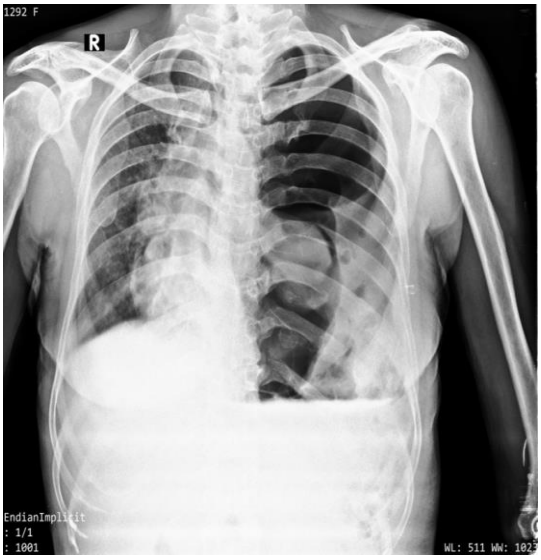
2b. Clinical findings and Timeline

Her entrance blood pressure was 130/80 mm Hg, her heart rate was 104 beats per minute, and her breathing rate was 30 beats per minute. With the addition of extra oxygen (3 lt/min via nasal prongs), the SpO2 increased from 88% on room air to 95%. In the left hemithorax, which was tympanitic on percussion, a systemic examination indicated absent breath sounds. Rightward displacement of the mediastinum. The remaining portion of the systemic evaluation was not helpful.

2c. Diagnostic Assessment

The clinical diagnosis of left-sided hydropneumothorax was supported by the chest X-ray (Figure. I-A).

Figure I-A. Left-Sided Hydropneumothora*x*



2d. Therapeutic intervention

After the left anterior axillary line, in the seventh intercostal space, she underwent intercostal tube drainage while under local anesthetic. Drainage was commenced with a 24Fr chest tube and no negative pressure suction. Serosanguineous fluid drained with air in an amount of about 300 ml.

2e. Follow-up and outcomes

The patient experienced strong coughing and the formation of pinkish sputum an hour after the treatment. They also experienced significant dyspnea. Blood pressure readings were 70/50 mm Hg and pulse 150/min, respectively. To 78%, the SpO2 decreased. Unlike the right, which was completely clean, the left hemithorax was rife with crepitations. On the left lung field of the chest x-ray, there were several fluffy opacities (Figure I-B). Using a nonrebreathing face mask, the patient obtained additional oxygen to make up for hypoxemia. She was given antibiotics, inotropes (Dopamine 5–15 mcg/kg/min), and IV fluids. On the third post-admission day, the inotrope support was discontinued because the patient had gradually improved clinically. The vital signs didn't change. As long as there was drainage and no air leak, the intercostal tube was left in place. The patient fully recovered with no lingering hypoxemia. The left mid and bottom zones of the repeat x-ray at that time had only very slight opacities (Figure II). In addition to respiratory physiotherapy, conservative treatment was maintained.

Figure I-B. Fluffy Opacities Scattered Along the Whole Left Lung Field

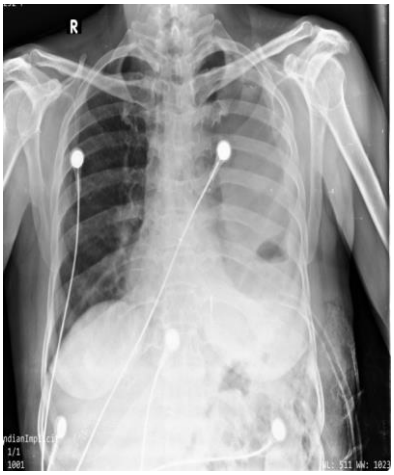
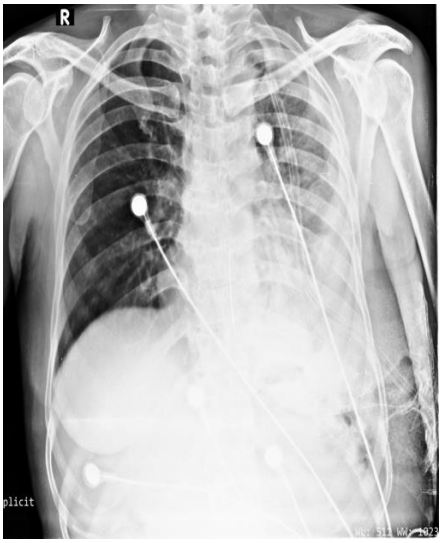


Figure II. Minimal Opacities Over the Left Mid and Lower Zones



3. DISCUSSION

Reexpansion pulmonary edema is defined by the rapid reinflation of a collapsed lung following a variable period of collapse caused by a pleural effusion or pneumothorax (1). This rapid reinflation causes unilateral pulmonary edema. Increased pulmonary vascular permeability appears to cause the reexpansion of pulmonary edema. The high protein content of edema fluid in both humans (2) and rabbits (3) suggests that the cause of the edema is leaky capillaries rather than a higher hydrostatic pressure gradient. According to Pavlin et al.'s(4), pulmonary edema is caused by capillary injury brought on by the mechanical pressures placed on the lung during reexpansion. No proof exists that the collapsed lung's permeability had increased before reinflation (4)Reexpansion pulmonary edema may be caused by reperfusion damage, according to a different but increasingly accepted theory(5) Atelectasis can cause severe hypoxia of the atelectatic lung due to reduced oxygen delivery to the lung caused by missing ventilation and hypoperfusion (6). After then, oxygen-free radical production is encouraged when the hypoxic zones are reperfused, which may lead to lung injury. Sometimes the reexpansion edema only affects the atelectatic portion of the lung when the lung is only partially deflated (7).

The discovery that re-expanding the lungs without first administering a higher Fio2 (40%) for the duration of the pneumothorax decreases edema supports the reperfusion injury hypothesis (8). Even if the lung is deflated, more oxygen eliminates systemic hypoxemia. The finding that giving antioxidants to rabbits before reexpansion reduces their permeability edema and level of inflammation adds more evidence in favor of this theory (9).

4. CLINICAL INDICATORS

Patients with reexpansion pulmonary edema frequently have severe coughing or chest tightness during or right away after thoracentesis or chest tube insertion. Large volumes of frothy pink sputum might occasionally produce by the cough. Other symptoms include cyanosis, dyspnea, tachypnea, tachycardia, fever, hypotension, and nausea. A chest radiograph shows pulmonary edema across the ipsilateral lung and a progression of the symptoms over 24 to 48 hours. Additionally, the opposing lung may have pulmonary edema (10). Recovery is typically finished if the patient does not pass away within the first 48 hours (11).

Reexpansion pulmonary edema occurred in 2% of 500 patients who underwent thoracoscopy and talc insufflation for the treatment of recurrent pleural effusion, according to Milanez de Campos et al. (12).

5. PREVENTION

If a patient has extensive pleural effusions or pneumothoraxes that last more than a few days, he or she should evaluate for the possibility of reexpansion pulmonary edema. In light of the animal studies by Miller et al (13) and Pavlin and Cheney, the tubes should attach to an underwater-seal drainage system rather than a negative pressure apparatus when performing tube thoracostomy for spontaneous pneumothorax (14). Negative pressure can administer to the pleural space if underwater-seal drainage fails to cause the underlying lung to expand again within 24 to 48 hours. If patient experiences chest tightness, chest pain, shortness of breath, or more than mild coughing during a therapeutic thoracentesis, the procedure is withheld. The amount of fluid that is withdrawn, however, does not require a strict cap to be set, as stated in the text before.

6. TREATMENT

Intravenous fluids, oxygen, and morphine are the main supportive therapies used to treat reexpansion pulmonary edema. Diuresis should be avoided because it could be harmful (15). Intubation, mechanical ventilation, and PEEP are suggested as escalating levels of treatment for severe hypoxemia; for moderate to mild hypoxemia continuous oxygen should be given in the form of face mask and nasal prongs, volume replacement, and inotropic agents are suggested as escalating levels of treatment for hypotension with low cardiac output; and no treatment is suggested for an abnormality on radiography alone (16).

7. CONCLUSION

REPE can be relatively harmless or life-threatening. When something is serious, it appears suddenly and dramatically. patients who present with symptoms do so within 1 hour of reexpansion, and all are symptomatic within 24 hours. Symptoms include a severe persistent cough and chest pain. In more severe cases, patients experience hypoxemia, tachypnea, tachycardia, and hypotension. Symptoms may worsen for 24 to 48 hours. Chest radiographs show patchy or diffuse alveolar infiltrates in the expanded lung. If the patient survives the first 48 hours, recovery is usually complete. Treatment is primarily supportive, with mechanical ventilation used in severe cases. The best strategy intervention, despite the lack of randomized trials to guide practice. When performing tube thoracostomy for a spontaneous pneumothorax of unknown duration, it is recommended that the tube be initially connected to a water seal rather than to negative pressure. If the lung does not fully expand after 12 to 24 hours, negative pressure can applied to the pleural space.

8. LIMITATIONS

The results of our study are not without limitations. The Current Study was administered within a single hospital setting in an urban area and thus may not be generalizable to other facilities.

9. FINANCIAL SUPPORT AND SPONSORSHIP- The authors have indicated they have no financial relationships relevant to this article to disclose.

10. CONFLICT OF INTEREST- The authors declare that they have no conflict of interest.

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