

Reexpansion pulmonary edema after treatment of secondary spontaneous pneumothorax

Babaji D Ghewade, Saood Ali, Swapnil Chaudhari, Smaran Cladius

Department of Respiratory Medicine, Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha, Maharashtra, India.
Correspondence to: Babaji D Ghewade, E-mail: bghewade@rediffmail.com

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Abstract

Reexpansion pulmonary edema is an unusual, but commonly fatal, clinical state. It is denoted by the occurrence of unilateral pulmonary edema in a lung that has been speedily reinflated following a variable duration of collapse secondary to a pleural effusion or pneumothorax. Unilateral pulmonary edema is connected with a variable degree of hypoxia and hypotension, occasionally needing intubation and mechanical ventilation, and at times causing lethality. The exact pathophysiologic anomalies linked with this disorder are still not known, although reduced pulmonary surfactant levels and a proinflammatory status are supposed mechanisms. Early diagnosis is important because prognosis is based on early recognition and right treatment. Preventive means are still the best applicable approach for patient handling. Here, we report a case of a 25-year-old male patient who developed reexpansion pulmonary edema after intercostal drainage for secondary spontaneous pneumothorax.

KEY WORDS: Pneumothorax, reexpansion pulmonary edema, BIPAP

Introduction

Reexpansion pulmonary edema is a life-threatening condition characterized by development of unilateral pulmonary edema in a lung that has been rapidly reinflated following a variable period of collapse secondary to a pleural effusion or pneumothorax. The method is unknown and has been linked to surfactant depletion or owing to hypoxic capillary damage causing heightened capillary permeability. The incidence of reexpansion pulmonary edema varies from 0.9% to 29.8%, the mortality rate associated with RPE can be as high as 20%. It is related to a variable degree of hypoxia and hypotension, occasionally needing intubation and mechanical ventilation, and at times causing fatality. Early diagnosis is important because prognosis is based on early recognition and correct treatment.

Case Report

A 25-year-old male patient came in casualty with complaints of sudden onset of breathlessness since 2 days, which had increased in intensity over a period of 2 days to Modified Medical Research Council Grade III without any diurnal or postural variation. Patient also complained of right-sided chest pain since 2 days, which was squeezing, diffuse, and nonradiating in nature. Patient showed no history of allergic rhinitis, bronchial asthma, orthopnea, paroxysmal nocturnal dyspnea, hemoptysis, hypertension, or diabetes mellitus. Patient showed a history of tuberculosis 2 years ago and had taken antitubercular treatment Category (CAT)1 Directly Observed Treatment Strategy (DOTS) for 6 months.

On examination, patient was febrile with temperature 100° F, and there was no pallor, icterus, lymphadenopathy, or pedal edema. His pulse was 114/min, regular, normovolumic, and peripheral pulses were felt bilaterally. Respiratory rate was 46/min, thoracoabdominal, blood pressure was 120/74 mm Hg in right arm in supine position, and oxygen saturation was 68% on room air. Immediately, supplemental oxygen was started with face mask.

On respiratory system examination, upper respiratory tract was normal, no congestion or deviated nasal septum was seen. Lower respiratory tract examination showed movements

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decreased on right side, decreased tactile vocal fremitus, and hyperresonant notes were heard on percussion in all lung fields on right side. On auscultation, breath sounds were absent on right side. Hence, a diagnosis of secondary spontaneous pneumothorax was made clinically.

Chest X-ray PA view showed right-sided pneumothorax with mediastinal shift toward left side along with fibrocavitary lesions in both upper lobes [Figure 1]. Urgent intercostal drainage tube was inserted in right fifth intercostal space in midaxillary line. Procedure was uneventful, and patient tolerated the procedure well. Gush of air was relieved after tube insertion. Water column movement and a large bronchopleural fistula were seen. Patient felt relieved immediately after tube insertion, and his saturation improved to 88%. But after few minutes, patient started having rigorous dry cough associated with right-sided chest pain and shortness of breath. His pulse was 122/min, blood pressure was 106/70 mmHg, and SaO₂ came down to 72%. PostICD chest X-ray PA view was done, which showed expansion of right lung and homogeneous opacity in right lower zone suggestive of unilateral right-sided pulmonary edema [Figure 2].

Patient was shifted to respiratory intensive care unit. Oxygen supplementation was continued with face mask, and arterial blood gas was done, which showed Type I respiratory failure with pH: 7.46, pO₂: 67.8, pCO₂: 41.1, and HCO₃⁻: 29.0. Patient continued to be tachypneic, and oxygen saturation did not improve above 85%. Patient was put on noninvasive ventilation using bilevel positive airway pressure (BIPAP) for almost 12 h. Repeat arterial blood gas analysis was done which was normal. BIPAP was removed, and patient was shifted to ward. Chest X-ray PA view was also repeated, which showed marked improvement both in resolution of pneumothorax and pulmonary edema [Figure 3].

His laboratory investigations showed hemoglobin: 15.1 g %, WBC: 10,500/cmm with 66% neutrophils, normal platelet counts, and ESR: 53 mm at the end of 1 h. Random blood sugar, liver function and kidney function tests were normal. ELISA test for HIV was nonreactive.

His sputum examination was positive for acid-fast bacilli (1+), while cytology was negative for malignant cells.

Considering old antitubercular history, patient was started on CAT II DOTS. Patient improved over a span of 5 days, and chest X-ray was done after the fifth day, which showed complete resolution of pneumothorax and pulmonary edema [Figure 4]. Intercostal drain was removed on the sixth day, and, postICD removal, chest X-ray was carried out [Figure 5].

Discussion

Pinault in 1853, first described reexpansion pulmonary edema as a complication after thoracentesis.^[1] In 1959, Carlson *et al.*^[2] described reexpansion pulmonary edema that occurred after treatment of pneumothorax. In 1988, Mahfood *et al.* published an extensive review of cases of reexpansion edema. Several criteria for this condition were defined, characterizing



Figure 1: Chest X-ray PA view on admission.

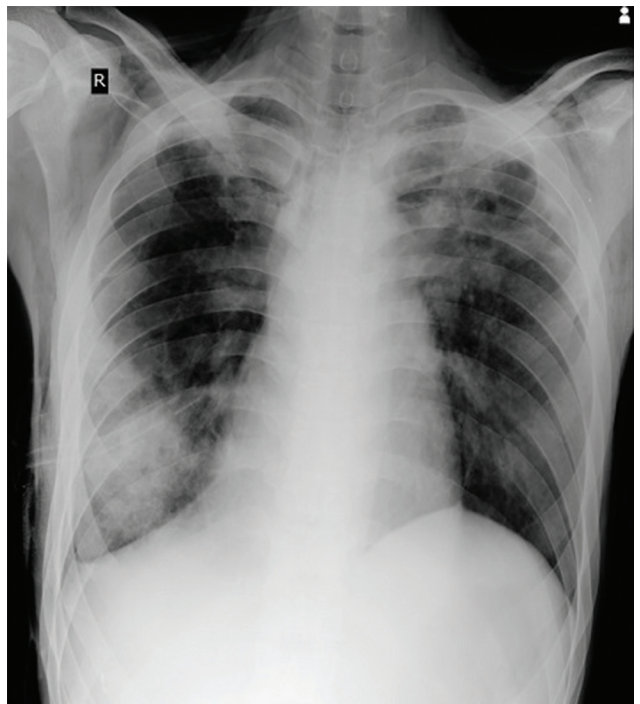


Figure 2: PostICD chest X-ray PA view.

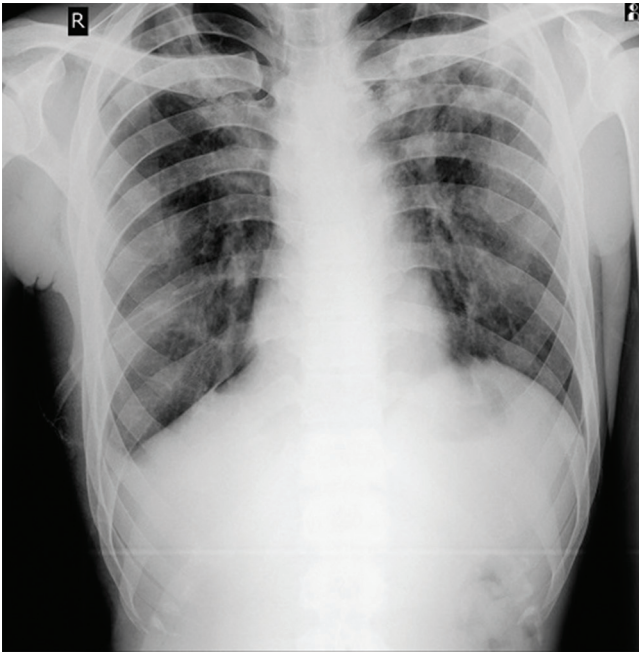


Figure 3: Chest X-ray PA after 24 h of ICD insertion.

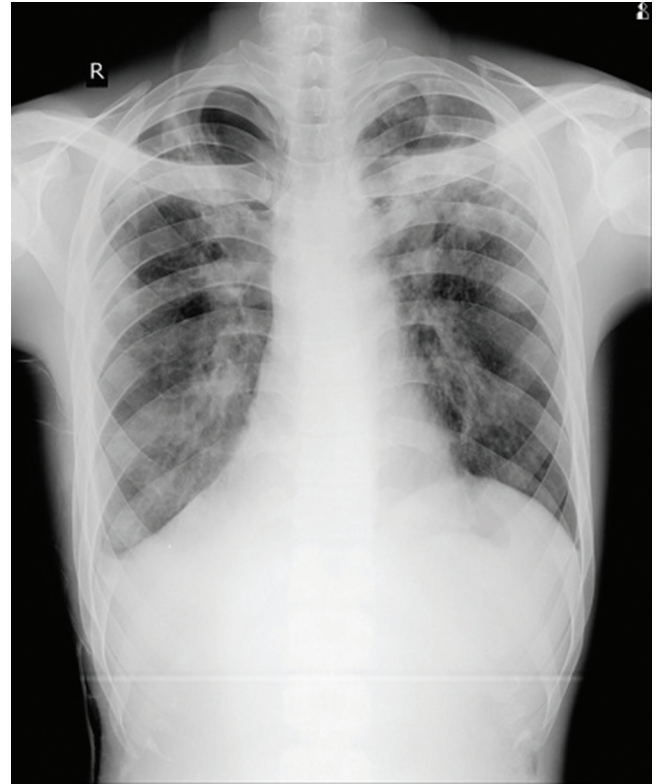


Figure 5: Chest X-ray after ICD removal.



Figure 4: Chest X-ray PA after fifth day.

it as being the consequence of hypoxemia and alveolus-capillary mechanical lesion owing to a prolonged lung collapse, occurring independently from the technique used for pleural emptying on either pleural effusion or pneumothorax (pleural suction, Heimlich valve, water stamp, or positive-pressure ventilation). Since then, many cases have been reported.

The exact mechanisms causing reexpansion pulmonary edema are not clearly understood. The pathogenesis is certainly multifactorial. Many factors are supposed to be associated with the development of edema such as chronicity of collapse, volume of effusion, reexpansion technique, pulmonary vascular permeability, bronchial obstruction, loss of surfactant, and alteration of pulmonary artery pressure. Pulmonary collapse with more than 72 h of evolution is the most important factor involved in development of reexpansion pulmonary edema.^[3]

Pavlin and Cheney,^[4] in a study in rabbits, found that more extensive reexpansion pulmonary edema occurred in lungs that had been collapsed for 7 days than in those that had been collapsed for 3 days. Contralateral pulmonary edema also developed in some cases but was less extensive. However, in humans, some cases of reexpansion pulmonary edema have occurred when no negative pressure was applied to the pleural space. When the pneumothorax or pleural effusion is present for more than 3 days, chances of development of reexpansion edema are very high. There seems to be association of increased permeability of the pulmonary vasculature to the development of reexpansion pulmonary edema.^[3]

Pavlin *et al.*^[5] have suggested that capillary damage occurs after the mechanical stresses to the lung during reexpansion, which leads to the development of pulmonary edema. But, there is no evidence that the collapsed lung has increased permeability before reinflation.

An alternate hypothesis has been proposed where reperfusion injury results in reexpansion pulmonary edema.^[6] Absent ventilation and hypoperfusion owing to atelectasis leads to severe hypoxia of the atelectatic lung. Reperfusion of the hypoxic areas promotes oxygen-free radical formation and lung injury. In partially collapsed lungs, the reexpansion edema sometimes occurs only in the collapsed parts of the lungs.^[7]

Patients typically develop pernicious coughing or chest tightness during or immediately following thoracentesis or chest tube placement. They also experience new or worsening of the existent cough, which may be sometimes productive of copious amounts of frothy pink sputum. Dyspnea, tachypnea, tachycardia, fever, hypotension, nausea, vomiting, and cyanosis may be the other symptoms. The symptoms may worsen for the first 24–48 h, and the chest radiograph shows pulmonary edema throughout the ipsilateral lung. Pulmonary edema may also develop in the contralateral lung.^[8] If the patient does not die within the first 48 h, prognosis is very good.

The diagnosis of reexpansion pulmonary edema is based on the history, clinical presentation, and radiological features. The radiological evidence is the pulmonary edema with interstitial opacity, consolidations, air bronchogram, and Kerley's "B" lines.^[8] The prognosis may vary from spontaneous resolution to life-threatening respiratory failure.

The treatment of reexpansion pulmonary edema is primarily supportive with intravenous fluids, oxygen, and morphine. Diuresis may be detrimental and should be avoided. Suggested escalating levels of treatment include no treatment for an abnormality on radiography alone; nasal supplemental oxygen for mild hypoxemia; continuous positive airway pressure through face mask for moderate hypoxemia^[9] and intubation, mechanical ventilation, and positive end-expiratory pressure for severe hypoxemia; and volume replacement and inotropic agents for hypotension with low cardiac output.

In our case, patient showed symptoms for 2 days, which is comparatively a short time for development of reexpansion edema when compared with the earlier reported cases in literature. Pneumothorax for less than 72 h may have led to a favorable outcome in his condition. Within 12 h of noninvasive ventilatory support, patient showed signs of improvement, and the reexpansion edema disappeared completely within 5 days.

Many similar cases have been recently reported by various authors. In 2013, Kwon *et al.*^[10] published a case report of a 46-year-old male patient with simultaneous bilateral spontaneous tension pneumothorax. Severe reexpansion pulmonary edema developed after bilateral tube thoracostomy, but he recovered after 2 days of ventilator care. After bilateral wedge resection and talc pleurodesis, he was discharged without complications and had remained well and without recurrence during the 8-year follow-up.

Another similar case was reported by Harner and Crawley^[11] in 2014, where they reported a case of a 48-year-old female patient who had a radiographically severe but clinically mild reexpansion edema following decompression of a spontaneous pneumothorax.

Conclusion

Reexpansion pulmonary edema is a rare complication after tube thoracostomy because of rapid reinflation of collapsed lung. Considering the high mortality rates related to it, preventive measures are still the best available strategy. Even though there have been no extensive studies on the risk factors and on the strategy for extensive pneumothorax or large effusion, there are empirical data which reveal that removing over 2 L of air or effusion from the pleura considerably rises the risk of reexpansion edema. The possibility of this complication is often neglected. We should be aware of this complication, the corresponding risk factors such as young patient, large and protracted pneumothorax (more than 24 h), rapid reexpansion of the lung, and the possible prophylactic procedures. Even though these procedures cannot reliably prevent reexpansion edema, awareness of this life-threatening complication allows early diagnosis and rapid initiation of the necessary treatment.

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