J Med Sci 2016;36(1):22-24
DOI: 10.4103/1011-4564.177172

CASE REPORT

Reexpansion Pulmonary Edema Following Tube Thoracostomy for Spontaneous Pneumothorax in an Elderly Male

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Reexpansion pulmonary edema (RPE) is an important cause of unilateral pulmonary edema that rarely occurs following drainage of pleural effusion or pneumothorax. Most patients develop symptoms within an hour of lung expansion. The presentation is usually rapid and dramatic and may be fatal at times. The duration of lung collapse more than 3 days, large-size pneumothorax, rapidity of lung expansion, and application of negative pleural suction are well-known risk factors for the development of RPE. We present here an elderly male with diabetes mellitus who presented with a large pneumothorax and developed RPE shortly after insertion of a chest tube.

Key words: Pneumothorax, tube thoracostomy, reexpansion pulmonary edema, diabetes mellitus

INTRODUCTION

The edema occurring in a lung that re-expands following a reasonable period of collapse is known as reexpasion pulmonary edema (RPE). Usually, this has been described as a rare complication after drainage of pleural effusion or pneumothorax;[1,2] however, occasional cases of RPE after excision of the large hepatic cyst, giant mediastinal tumor, or decortication have been reported.[3] The presentation of RPE is usually rapid and dramatic and can be fatal at times.[3] We report here a patient with diabetes mellitus who developed RPE after placement of chest tube for removal of spontaneous pneumothorax.

CASE REPORT

A 62-year-old male presented with mild breathlessness and left-sided boring chest pain for 3 weeks. He recalled the onset of chest pain as acute in nature 3 weeks ago; however, he ignored then as the severity of symptoms subsided in few minutes. He was a farmer and current smoker with a smoking index of 350. He had not suffered from previous tuberculosis or interstitial lung disease. Past medical history was notable for diabetes mellitus of 7 years duration on oral glucose lowering agents. On admission, his vitals were as follow: Heart rate 84 beats/min, blood pressure 140/90 mmHg, respiratory rate 24 breaths/min, oral temperature 38°C, and oxygen saturation of 97% in room air. Physical examination of the chest revealed intercostal fullness, decreased vocal fremitus, hyperresonanat percussion note, and amphoric bronchial breathing on the left side. Chest radiograph showed a large pneumothorax on the left side with minimal fluid [Figure 1A]. An intercostal tube with underwater seal drainage was placed. About 5 min after the procedure patient complained of increasing breathlessness and a sense of chest tightness and sweating was noticed over the face. His heart rate increased to 124 beats/min, respiratory rate was 35 breaths/min, blood pressure 140/100 mmHg, and room air oxygen saturation dropped to 86%. Lung auscultation revealed fine inspiratory crackles on the left. Suspecting reexpansion edema chest tube was clamped immediately, and high-flow oxygen, nebulized bronchodilator, and intravenous crystalloid were administered. He improved symptomatically, and a repeat chest radiograph 4 h later with a chest tube *in situ* in clamped position showed complete expansion of the left lung, albeit with the appearance of peripheral airspace opacification that was denser in mid and lower zones and had a coarse granular pattern in the upper zone [Figure 1B]. Expectedly, the radiographic abnormality resolved completely in next 72 h [Figure 1C] reaffirming our suspicion of RPE. He was well at follow-up 4 weeks later. Spirometry confirmed a diagnosis of chronic obstructive pulmonary disease as the underlying cause for the spontaneous pneumothorax.

DISCUSSION

RPE was first reported by Pinault in 1853 following removal of pleural effusion. Subsequently, in 1959, Carlson *et al*.

Received: August 26, 2015; Revised: September 12, 2015; Accepted: November 09, 2015
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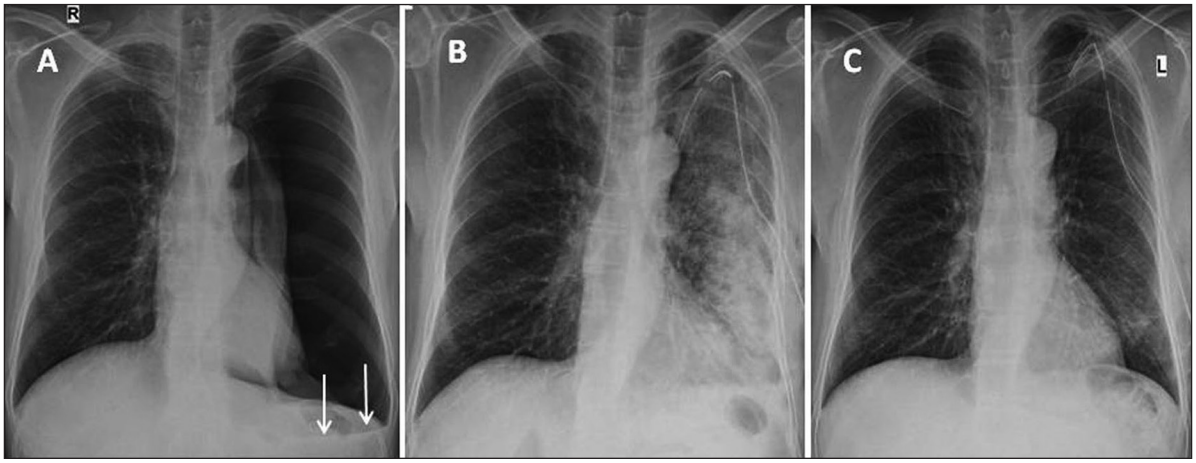

Figure 1: Serial chest radiographs. (A) On presentation showing a large pneumothorax with minimal fluid (arrows) on left side. (B) After 4 h of tube thoracostomy showing complete expansion of left lung with peripheral ground-glass haziness in upper and dense air-space opacity over mid and lower zones with chest tube *in situ.* (C) After 72 h showing complete resolution of the radiographic abnormality

reported the first case of pneumothorax related RPE after manual aspiration of air. Since then only a few 100 cases of RPE have been reported so far suggesting its rarity.[3] In a recent series of 185 patients with pleural effusion undergoing large-volume (>1L) thoracentesis, the incidence of clinical and radiographic RPE was 0.5% (one patient) and 2.2% (four patients), respectively.[4]

The risk factors for developing RPE include duration of lung collapse more than 3 days, evacuation volume more than 2000 ml, large pneumothorax, application of significant negative pleural suction, rapid lung expansion, and diabetes mellitus.[1-3,5] A recent study concluded that RPE is rare after large-volume thoracentesis and is independent of the volume of fluid removed, pleural pressures and pleural elastance and recommends drainage of pleural fluid safely so long the patient tolerates.[4] RPE usually develops within an hour of lung expansion and almost always within 24 h. Reportedly, the incidence of RPE is more if negative pleural suction is applied than simple tube thoracostomy with under-water-seal drainage. Mostly, it occurs in the ipsilateral expanding lung. However, occasional instances of RPE in the contralateral lung has been reported. Increasing dyspnea, chest tightness, tachycardia, and auscultatory crackles with or without desaturation following the pleural procedure suggests the onset of RPE. Severe forms of RPE may present with respiratory failure, cardiovascular collapse, shock, coma, or even death.[3] The previous study has reported mortality as high as 20% and seems to be associated with bilaterality and sudden onset of RPE.[1] Some cases of RPE may have an only radiographic presentation without any clinical manifestations, thereby likely to remain undiagnosed. However, in most instances the onset of RPE is dramatic that need immediate attention. Typically, postprocedure chest radiograph shows ground-glass and air-space opacity in the reexpanded lung.[3] The most common computed tomography findings of RPE include ipsilateral ground-glass opacities, septal thickening, foci of consolidation, and areas of atelectasis.[6]

Pathophysiologically, RPE represents a form of increased capillary permeability edema. Other factors implicated in the pathogenesis are hypoxic injury to capillary and alveolar membrane, mechanical stress, and decreased surfactants.[3] Recently, diabetes mellitus has been found to be an independent risk factor for the development of RPE in patients with spontaneous pneumothorax.[5] Diabetic microangiopathy is the fundamental pathophysiological abnormality that results in several end-organ damage in diabetes. Microangiopathy leading to pulmonary alveolar basement membrane damage may be the reason for predisposition to RPE in diabetic patients. Autopsy studies have shown alveolar basement membrane thickening in diabetic patients.[5,7] Furthermore, the relationship between basement membrane thickening and increased capillary permeability has been demonstrated in high-glucose condition.[8] Therefore, this ultrastructural alteration of basement membrane and composition of extracellular matrix in diabetic patients increases the capillary permeability and possibly contribute for RPE.[5] Our patient had 7 years history of diabetes mellitus and a large pneumothorax of long duration, both favorable for the development of RPE.

Most patients with pneumothorax present with acute chest pain and breathlessness that usually resolve after the evacuation of pleural air. Surprisingly, our patient had increased symptoms after insertion of a chest tube, and postprocedure chest radiograph showed consolidation in the expanded lung. In such scenario, one may be tempted to consider this radiographic abnormality as underlying tuberculosis or bacterial pneumonia and the pneumothorax its complication. However, lack of fever and signs of clinical toxicity preceding the procedure, negative sputum staining for acid fast bacilli, and spontaneous resolution of radiopacity

within 3 days practically excluded an infectious etiology. Besides RPE, other possibility of postprocedure deterioration of patient's clinical status may be due to misplaced chest tube causing lung contusion, diaphragmatic injury, or perforation of abdominal viscera that needs evaluation by appropriate imaging.[9] The pneumothorax in our patient was attributed to underlying chronic obstructive pulmonary disease.

Treatment is largely supportive and guided by the severity of presentation. Mild cases may be managed with administration of oxygen, intravenous fluids, and nebulized bronchodilator whereas those with hemodynamic instability or unresponsive hypoxemia will require vasopressor and noninvasive or invasive ventilation.[1-3] Noninvasive ventilation as continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) has been highly beneficial in improving cardiogenic pulmonary edema. A recent meta-analysis concluded that both CPAP and BiPAP are equally effective with similar adverse effects profile in the management of cardiogenic pulmonary edema.[10] There are reports of successful use of CPAP in the management of RPE.[11,12] Some authors have reported using prostaglandin analog misoprostil and ibuprofen or indocin suppositories in RPE for their cytoprotective or anti-inflammatory actions.[13] Contralateral decubitus positioning of the patient seems to be a reasonable step that may help in reducing the progression of edema and improving the intrapulmonary shunt. Most RPE usually resolves in 24-72 h.[13]

The index case was managed with a nebulized bronchodilator, supplemental oxygen, and intravenous fluid therapy and inhaled bronchodilator was prescribed for underlying chronic obstructive pulmonary disease. The physician should anticipate RPE while performing tube thoracostomy even if negative pleural suction is not applied and the patient should be explicitly informed beforehand regarding such complication. Slow and sequential drainage of fluid or air is the best safeguard in preventing RPE.

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