Literature Review

Re-expansion Pulmonary Edema

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Abstract

Re-expansion pulmonary edema (RPE) is a rare complication of pleural puncture (thoracentesis) and chest tube insertion. The incidence of RPE is low (1%), but mortality can be up to 20%. The main pathophysiological mechanism is pulmonary edema due to increased permeability and increased hydrostatic pressure in the pulmonary capillaries. Risk factors include duration of lung collapse (>3 to 7 days), size of pneumothorax (>30%), volume of aspirated air or fluid (>1.5 to 3 L), excessive negative intrapleural pressure, diabetes mellitus, and chronic hypoxemia. Prevention includes limiting the volume of aspirated air or fluid (<1.5 L), air or fluid evacuation in a controlled manner, and preventing excessive negative intrapleural pressure. Treatment is supportive care through cardiovascular and respiratory monitoring, oxygen and decubitus positioning.

Keywords: re-expansion pulmonary edema, pathogenesis, clinical management

INTRODUCTION

Pleural effusion and pneumothorax are common conditions. In the United States, each year there are 1.5 million patients with pleural effusion and 40.000 patients with pneumothorax, which causes pleural puncture (thoracentesis) and chest tube insertion to be common medical procedures.[1,2] The collapsed lung due to pneumothorax or pleural effusion after a chest tube insertion or pleural puncture in some circumstances leads to re-expansion pulmonary edema (RPE), which is a condition that rarely occurs but can be fatal.[2]

A case of RPE was first reported in 1853 when Pinault removed 3 L of pleural fluid from a patient with pleural effusion. A case report of RPE in a pneumothorax patient was initially reported by Carlson in 1958. The prevalence of RPE is relatively low (less than 1%), however, there are differences from various studies with a prevalence range between 0.3% and 32.5%. This is due to different definitions (clinical versus radiological), small sample sizes, and different patient populations.[2,3]

Mortality from RPE has been reported to be as high as 20%, nonetheless, recent studies in patients with pneumothorax have shown lower rates, making it difficult

to determine the prognosis in patients with RPE. The pathophysiology and contributing factors of RPE are not fully understood, and prevention is the best medical approach. Prevention is one of the reasons for expert agreement on the amount of fluid that can be removed in a pleural puncture for one attempt (1 to 1.5 L).[3] This literature review will discuss RPE, especially in patients with pleural effusion and pneumothorax.

PATHOGENESIS AND PATHOPHYSIOLOGY OF RPE

The pleural cavity consists of the parietal pleura lining the chest wall, diaphragm and mediastinum, and also the visceral pleura lining the lung. In normal conditions, there is fluid between the two pleural layers. The volume of pleural fluid in a normal individual is 0.26 mL per kilogram of body weight. Pathological conditions can increase the volume of the pleural cavity both in the form of increased fluid volume (effusion) and the entry of air into the pleural cavity (pneumothorax). This leads to impaired lung development and requires invasive management such as pleural puncture and chest tube insertion.[1]

Pneumothorax causes an increase in intrapleural pressure. The increase in intrapleural pressure that occurs is not very large, and usually the upper lobe of the lung is more affected because the pressure gradient at the apex is greater than the basal lung. Pleural effusion will cause a greater pressure gradient at the lowest part of the lung, so that in an upright state, the lower lobe of the lung, which is the dependent part, will be more affected than the upper lobe of the lung.[4]

The mechanism of RPE is not well known and study on this subject is limited. Data from existing studies indicate that two important mechanisms cause RPE, namely impaired permeability at the alveolar-capillary barrier and increased hydrostatic pressure in the lung microvasculature.[5,6]

The pathological processes that occur are changes in the lung microvasculature, tissue damage by oxygen free radicals, increased local cytokine production, decreased surfactant function in the collapsed lung, decreased pulmonary lymphatic vessel flow, and excessive negative pressure in the pleural cavity during lung expansion.[5,7]

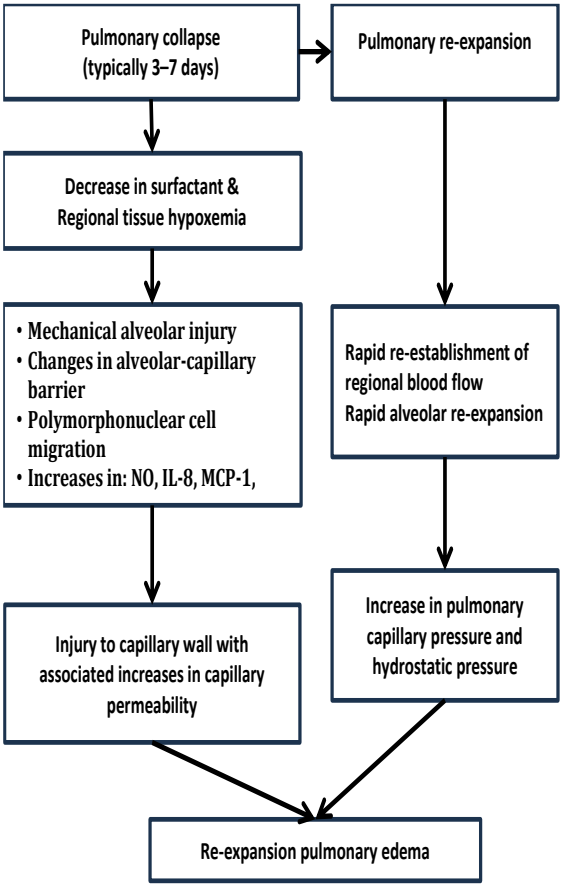

Figure 1. Pathophysiology of RPE[5–8]

Table 1. Components of diagnosis RPE[2,6,7]

Category	Description
Anamnesis	1. Abnormalities or space-occupying lesions of the thoracic cavity that cause lung collapse for more than 3-7 days
	2. Air or liquid aspiration within the previous 24-48 hours
Examination finding	1. Coughing for more than 20 minutes, pink or pollen sputum, chest discomfort, shortness of breath, tachycardia
	2. Rales on the ipsilateral, sometimes bilateral, rarely contralateral only
	3. Signs of hemodynamic instability, hypoxia, and hypovolemia
Radiologic examination	1. Unilateral radiologic abnormalities in the collapsed lung, sometimes bilateral, rarely contralateral only
	2. Alveolar filling pattern (infiltrates and consolidation) on thoracic radiographs
	3. Ground glass opacity, consolidation and thickened septa on CT scan
	4. Resolution of radiological features after a few days (in non-fatal cases)

CLINICAL MANIFESTATION

The diagnosis criteria for RPE have not yet been established. The clinical diagnosis of RPE requires a critical review of the history of the disease, clinical symptoms and signs, and radiological findings (Table 1).

Risk Factors

The mechanism of RPE is not well known and is multifactorial, so several risk factors may underlie the process. The duration of lung collapse is a major risk factor for RPE. Lung collapse of more than 3–7 days increases the risk of RPE compared to lung collapse of less than 72 hours. A possible mechanism is that pathological changes in the lung microvasculature take several days to have a significant effect on permeability when lung re-development occurs. Risk factors for RPE include:[7,8]

1. Duration of lung collapse (>3–7 days)
2. The large size of pneumothorax (>30%), tension pneumothorax
3. Concurrent effusion with pneumothorax
4. Age less than 40 years old
5. Aspiration fluid volume (>1.5–3 L)
6. Excessive negative pressure on the pleural cavity
7. Chronic hypoxemia
8. Diabetes mellitus

MANAGEMENT OF RPE

Prevention

The RPE is difficult to prevent due to its unknown etiology and pathophysiology, as well as the multifactorial risk factors. The risk of RPE remains, although all interventions have been implemented. Identification of risk factors and the possibility of RPE occurring in patients must be carried out. Identified risk factors must be adequately managed so that the risk of RPE can be reduced as much as possible. Patients with hypoxemia or high risk for RPE should be monitored and given oxygen during and after the procedure.[6]

Clinical Management

The primary management in the case of RPE is close respiratory and cardiovascular monitoring plus oxygen administration. Such conservative

management is sufficient in the majority of RPE cases.[9] The lateral decubitus strategy of placing the affected side in a dependent position can reduce intrapulmonary shunts and improve oxygenation.[6,7,10] Patients who do not improve with conservative management require respiratory support using non-invasive ventilation with continuous positive airway pressure (CPAP) or bi-level positive airway pressure (BiPAP).[7,9]

Mechanical ventilation with tracheal intubation and positive end-expiratory pressure (PEEP) may be considered in patients who are worsening or unstable. Hemodynamic support with vasopressor or inotropic drugs may be required in such patients.[7,10] Patients who continue to have respiratory failure after maximal management may be considered for differential ventilation.[6,7,10] The benefits of steroid administration have not been supported by strong evidence while diuretic administration can be detrimental because it aggravates the hypovolemia that occurs, so if it is needed, it is given under close supervision.[6,9]

Experimental treatments such as the administration of prostaglandin analog misoprostol and non-steroidal anti-inflammatory drugs (NSAIDs) ibuprofen and indomethacin at the onset of RPE diagnosis to obtain cytoprotective and anti-inflammatory effects have been reported but to date have not been supported by strong evidence.[3,6,10]

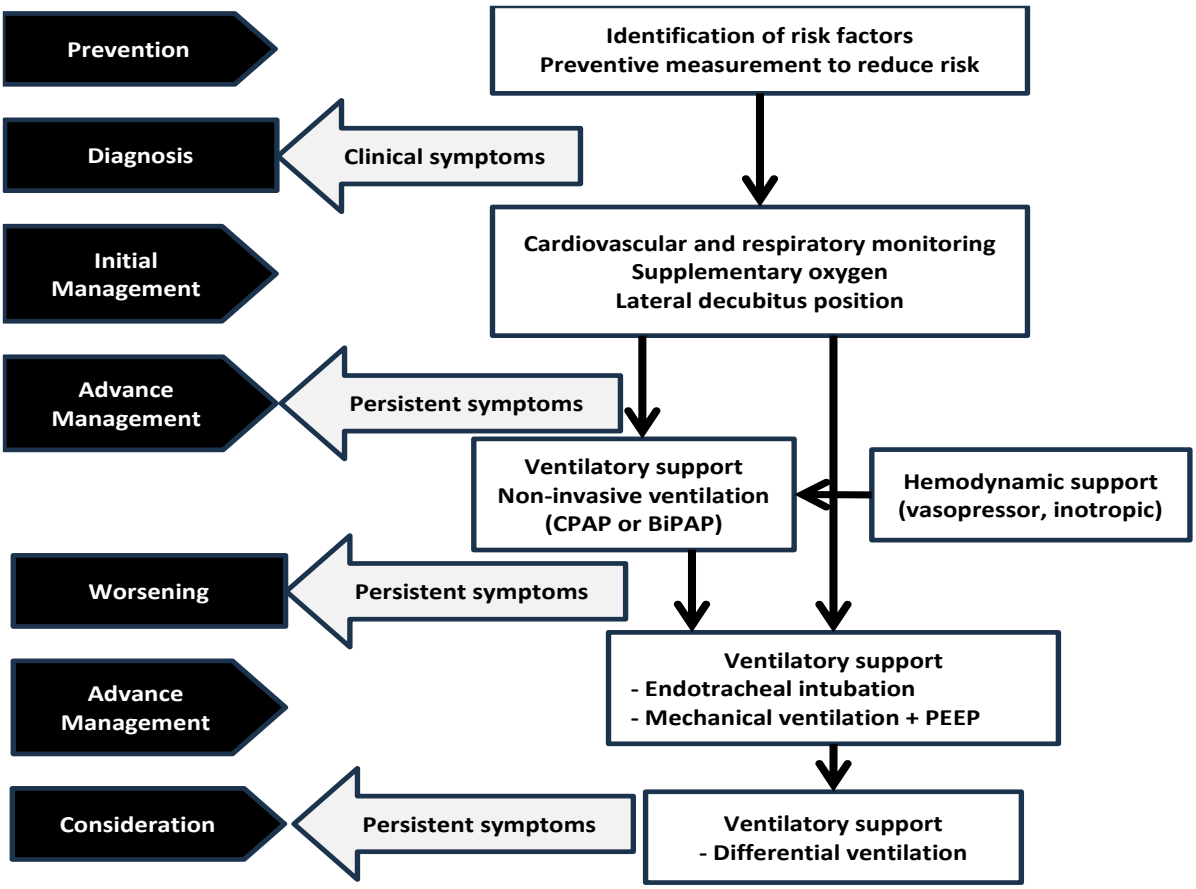

Figure 2. Comprehensive Management of RPE[3,6,7,9,10]

Administration of anti-IL-8 antibodies to reduce the inflammatory process that occurs during the development of collapsed lung and administration of alpha lipoic acid (ALA) as an antioxidant gave positive results in experimental animals, however, there is no evidence of benefit in humans.[3,6,10]

CONCLUSION

The RPE is a rare complication but is related to many cases encountered in the clinic, namely pleural effusion and pneumothorax. Identification and management of risk factors are important to minimize the risk of RPE. Most cases of RPE can be managed with cardiovascular and respiratory monitoring, as well as oxygen administration alone. Respiratory and circulatory support via non-invasive ventilation or mechanical ventilation and vasopressor/inotropic administration can be given in severe RPE.

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