



Reexpansion Pulmonary Edema (REPE) an Uncommon but Important Complication of Thoracocentesis: Case Report

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Abstract

Re-expansion Pulmonary Edema (REPE) is rare complication that occurs mainly after tube thoracostomy in pneumothorax and thoracocentesis in large pleural effusion is attributed to rapid pressure changes in the pleural cavity and free radical injury to the pulmonary vasculature. Usually REPE seen in cases of extensive pneumothorax and large pleural effusion with lung atelectasis of long duration. Authors report a case of re-expansion pulmonary edema following chest tube insertion in a case of iatrogenic pneumothorax.

Keywords: *Pneumothorax, thoracocetasis, reexpansion pulmonary edema (REPE)*

Introduction

Re-expansion pulmonary edema (REPE) though uncommon but important complication associated with re-expansion of lung that occurs after chest tube insertion while managing pneumothorax, hemothorax, thoracocentesis of massive pleural effusion, after lobectomy and even in single lung ventilation.^{1,2} Though exact mechanism of REPE is not known and has been attributed to rapid pressure changes in pleural cavity, free radical and hypoxic injury to pulmonary vasculature resulting in increased permeability especially in cases of pneumothorax and massive pleural effusion. The onset can be sudden, characterized by hypotension, tachycardia fall in

O₂ saturation and despite being infrequent mortality is reported up to 20%.^{1,3}

Case Report

Twenty four year female patient was referred to our ICU setting as case of purporeal sepsis. Despite on supportive measures patient had persistent dyspnea with fall in O₂ saturation detailed clinical history revealed that patient had undergone diagnostic pleural tap a day before. On examination patient had tachycardia with pulse rate of 120/min, tachypnea with respiratory rate or 28/min, chest examination revealed absent breath sounds on left side, chest x-ray done revealed pneumothorax in left hemithorax with shifting of

mediastinum to right side fig1. Tube thoracostomy was done, however clinically there was no improvement as fall in o₂ saturation continued along with dyspnea and tachycardia. Post chest tube thoacostomy chest x-ray revealed diffuse opacity in left hemithorax with no airfluid level and mediastinal structures in normal position, the right lung was normal as before with small pleural effusion fig 2. Echocardiography was done to rule out any myocardial involvement and to rule out any thrombus in main pulmonary trunk which did not reveal any diagnostic abnormality and ECG showed only sinus tachycardia, these findings suggestive of non-cardiogenic pulmonary edema and diagnosis of REPE made. Patient was managed symptomatically with oxygenation supplementation, nebulization with bronchodilators and started improving clinically after 24hrs and chest x-ray after 48 hrs revealed clear lung field in left hemi thorax, on third day she was able to maintain normal oxygen levels on room air, and subsequently shifted to the parent unit.



Fig. 1: Chest roentgenogram showing pneumothorax on left side and mediastinum shifted to right side with pleural effusion on right side.

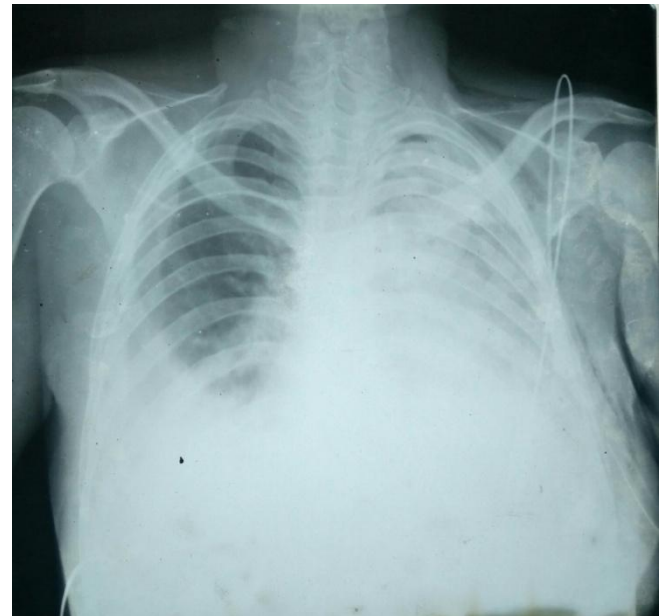


Fig. 2: Diffuse opacity in the left hemothorax and mediastinum in normal position with ICD in situ on left side.



Fig. 3: Bilateral clear lung fields with mediastinum in normal position and ICD in situ on left side.

Discussion

Re-expansion pulmonary edema is considered as iatrogenic complication associated with thoracocentesis of pleural effusion and chest tube thoracostomy of pneumothorax. Incidence has been reported less than 1% with mortality up to 20% in literature.^{1,3} The common risk factors include young patients with pneumothorax, massive pleural effusion and longer duration of

pulmonary collapse however it can occur even in pulmonary collapse of shorter duration. Exact pathophysiological mechanism of REPE remains yet to be defined and is considered multifactorial, various mechanisms have been proposed. One explanation being after re-expansion of collapsed lung the ventilation and reperfusion sets in leading to inflammatory response resulting in increased vascular permeability and altered lymphatic drainage. This damage is due to production of superoxide radicals and reactive oxygen species and inflammatory mediators including TNF, leukotriene B₄ and interleukin 8. Another mechanism proposed is the hydrostatic pressure changes in the pulmonary vasculature after re-expansion leading to mechanical dysfunction of pulmonary capillaries and decreased surfactant levels.^{1,4,5}

Clinical presentation of REPE varies depending upon the extent of edema majority of the patients are symptomatic within few hours and the duration of presentation may vary up to 24hrs. Characteristic features include tachypnea, tachycardia and crepitations on the effected side others falling oxygen saturation, hemodynamic instability and persistent coughing with pink frothy sputum and immediate chest tightness also suggest REPE. REPE usually involves whole lung occasionally single lobe or both lungs can be involved.^{3,6} Radiological signs include presence of opacities on the side of re-expanded lung which follow thoracocentesis/tube thoracostomy and reverting rapidly. CECT shows ground glass appearance with thickening of interlobular septa, peribronchovascular thickening.^{2,5}

Treatment

High degree of clinical suspicion and immediate recognition ensure the successful management of REPE. Treatment is usually supportive but varies depending upon the severity, oxygen supplementation in patients with mild symptoms may prove adequate but those with severe symptoms may require intubation and mechanical

ventilation. Noninvasive ventilation is also recommended in severe cases with good results, other measures include lateral decubitus position on the effected side which helps in reducing pulmonary shunt and improving oxygenation.^{2,4,7} The use of steroids, diuretics, prostaglandins analogues as misoprostol and ibuprofen has been reported but remains to be clarified. Preventive strategies involve use of low negative pressure <-20cm H₂O during tube thoracostomy with limited drainage of less than 1L, care should be taken during the drainage if patient complains of chest discomfort and uncontrolled coughing procedure should be stopped. Pleural pressure manometry is being recommended recently for drainage of large pleural effusions.^{4,8}

References

1. Murat A, Arslan A, Balci AE. Re-expansion pulmonary edema. *Acta Radiol* 2004;45:431-3.
2. Dias OM, Teixeira LR, Vargas FS. Reexpansion pulmonary edema after therapeutic thoracentesis. *Clinics (Sao Paulo)* 2010;65:1387-9.
3. Miller WC, Toon R, Palat H. Experimental pulmonary edema following re-expansion of pneumothorax. *Am Rev Respir Dis* 1973;8:664-6.
4. Kasmani R, Irani F, Okoli K, Mahajan V. Reexpansion pulmonary edema following thoracentesis. *CMAJ* 2010;182.
5. Sherman SC. Reexpansion pulmonary edema: a case report and a review of the current literature. *J Emerg Med* 2003;24:23-7.
6. Chowdhary M, Peng EWR, Sarkar PK. The risk of fatal reexpansion pulmonary oedema in poor left ventricular reserve. *Interact Cardiovasc Thorac Surg* 2009;9:350-51.
7. Volpicelli G, Fogliati C, Radeschi G, Frascisco M. A case of unilateral re-expansion pulmonary oedema successfully

treated with non invasive continuous positive, airway pressure. Eur J Emerg Med 2004;11:291-4.

8. Beng ST, Mahadevan M. An uncommon life threatenign complication after chest tube drainage of penumothorax in the ED. Am J Emerg Med 2004;22:615-9.