Case report:

Re-expansion pulmonary edema- case reports and review of literature

Basanta Hazarika[1], Suresh Sharma[2], Jugesh Sarma[3], Kripesh Ranjan Sarmah[4]

[1]Associate Professor, [2] Resident Physician, [3] Professor and HOD, [4] Assistant Professor, Department of Pulmonary Medicine, Gauhati Medical College, Guwahati

Abstract

Re-expansion pulmonary edema, described rarely in literature, can occasionally complicate decompression procedures in different lung diseases. Proper and prior assessment of the possibility, adoption of preventive strategy, and appropriate treatment may even save lives. The issue is elaborated with presentation of two predominantly ipsilateral re-expansion pulmonary edema following chest tube insertion.

Ke Words: Reexpansion pulmonary edema (REPE), Spontaneous pneumothorax, Refractory pleural effusion, pleurodesis. (The Pulmo - Face; 14:2, 46-48)

Abbreviations:

REPE: reexpansion pulmonary edema,

CKD: chronic kidney disease

INTRODUCTION:

Re-expansion pulmonary edema (REPE) is a rare entity and was first described by Carlson in 1958 [(1)]. It usually develops on rapid re-expansion of collapsed lungs [(2)] possibly through increased capillary permeability secondary to decompression [(3)]. The onset of REPE can be sudden and dramatic [(4)]. Hypoxemia, hypotension, and even death have been observed with the mortality reported as high as 20% [(5)]. Here, we present two cases of pulmonary re-expansion related oedema with two different settings as refractory pleural effusion and pneumothorax.

THE CASES:

The case - 1:

A sixty two years old patient of CKD (with diabetes been controlled on insulin) had right sided pleural effusion persisting for one year despite optimal medical management, maintenance haemodialysis and repeated therapeautic thoracocentesis. An intercostal tube drainage followed by chemical pleurodesis was planned. At presentation with shortness of breath, his general physical examination was essentially normal while the chest examination suggested massive right sided pleural effusion that was obvious on the chest radiograph (figure 1a). The pleural fluid on analysis was transudative according to the Light's criteria. He was slightly anaemic (hemoglobin as 10.3 %) with his blood urea and creatinine levels being 68 and 6.2 (mg % respectively) and his liver functions parameters and serum electrolytes were within normal limits.

Corresponding author:
Dr. Basanta Hazarika
Associate Professor, Department of Pulmonary Medicine, Guwahati Medical College, Guwahati-781032, Assam, India.
E-mail: *drbasantahazarika@yahoo.com*

We performed an intercostals tube placement and a volume of 1.6 litres of fluid was drained within 30 minutes or so. Two hours later the patient developed breathlessness and chest discomfort with restlessness. Soon he showed cyanosis and hypoxemia (SaO_2 falling to 78%) with feeble pulse and low blood pressure of (86/48 mmHg). Auscultation revealed coarse crepitations much more extensive over the right lower hemithorax than the left. His blood gas reveled no acidosis but a PaO2/ FiO2 ratio of 100 with a SaO_2 been 89 % on 50% FiO_2. The ECG was normal and Troponin-T test came negative. An urgent portable chest x-ray showed heterogeneous opacity in right lower zone (Figure 1b). Based on the clinic-radiological findings, the diagnosis of re-expansion pulmonary edema was made. The patient was treated with intravenous fluids and oxygen supplementation through face mask. He recovered slowly and was comfortable and hemo-dynamically stable (blood pressure 142/68 mmHg) with a SpO2 of 98% on oxygen (FiO_2: 28 %) after 24 hours. Chest radiograph (taken after three days) showed marked resolution of pulmonary edema (Figure 1c). The patient underwent pleurodesis (with 10% povidone iodine) after another three days and was discharged after removal of the intercostals tube in course of time.

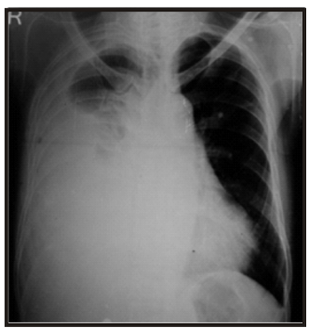

Figure 1a

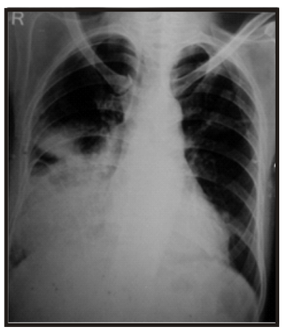

Figure 1b

Figure 1: 1a showing massive right sided pleural effusion, Figure **1b** displays complete resolution of the condition

The case - 2:

A 23 year old male patient presented 6 days after the onset of a sudden right-sided pleuritic chest pain accompanied by exertional shortness of breath. His medical history was unremarkable and the patient was in good general health with normal vital signs. However, he had diminished breath sounds on the left hemithorax. The hemogram, liver functions and serum electrolytes were within normal limits. The chest x-ray carried by the patient (done 6 days prior to the presentation) confirmed the clinical suspicion of a left sided pneumothorax (figure 2a). An intercostal tube drainage (in the right 4^{th} intercostals space in the anterior axillary line) relieved the symptoms but within an hour of the procedure, the patient developed severe cough, dyspnoea with hypoxemia (SaO_2 as 85% despite oxygen supplementation with nasal prongs at 2L/minute). The arterial blood gas analysis under higher oxygen supplementation (FiO_2 roughly 40 %) showed hypoxemia (PaO_2 59.9 mmHg, SaO_2 88%). An urgent portable chest X-ray showed expanded right lung with heterogeneous alveolar opacities in right lower zone (Figure 2b). Again, based on the setting and the findings, a diagnosis of re-expansion pulmonary edema was done. The patient stabilized and steadily improved with augmented oxygen supplementation alone. HRCT thorax (done on the 2nd day) showed dense and ground glass opacities in right lower lobe (Figure-2c). Finally, the chest tube could be removed on 3^{rd} day with complete re-expansion of the lung and no evidence of pulmonary edema (figure-2d). He was soon discharged in a satisfactory state.

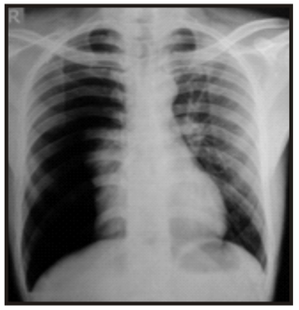

Figure 2a

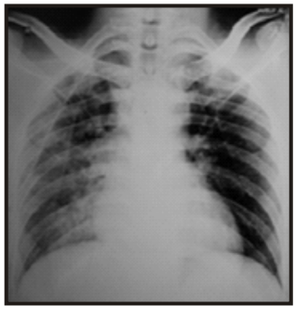

Figure 2b

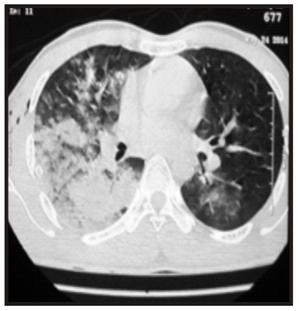

Figure 2c

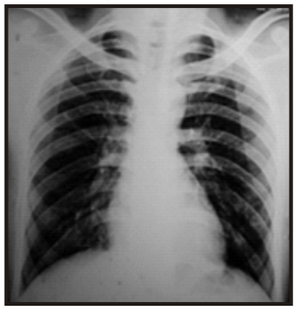

Figure 2d

Figure 2: 2a shows right sided pneumothorax,**2b** and **2c** elaborates the heterogeneous alveolar opacities in right middle and lower one suggesting REPE and **2d** displays complete recovery.

DISCUSSION:

The two cases of REPE reported here had two different clinical settings (pleural effusion in one and pneumothorax in the other) both occurring after intercostal tube drainage. Both the patients suffered from similar symptoms, featured suggestive of pulmonary edema on radiological evaluations, and recovered uneventfully with adequate oxygenation. Although rare, REPE is usually a self limiting entity and should be suspected particularly when a patient's condition worsens following the initial amelioration of symptoms after decompressing a lung through rapid evacuation of air or fluid. A rapid onset of dyspnea and tachypnea ensues most often within one hour of re-expansion of the collapsed lung [6]. Coughmay precede the development of REPE and hypotension may also occur due to third spacing of intravascular fluid into lung parenchyma [6, 7]. A mortality rate of as high as 20% has been described [5].

The degree and duration of lung collapse, the pace of re-expansion after decompression (closed aspiration or placement of chest tube) with or without the use of negative pressure treatment are the risk factors for development of REPE [8]. The longer the duration of collapse and the faster the decompression, the higher is the chance of REPE. REPE is noted following drainage of hemopneumothorax, large pleural effusion, pneumothorax, after lobectomy, or even during single-lung ventilation [2]. Spontaneous pneumothorax, however, remains the commonest underlying condition for REPE [2]. Rarely in pleurodesis from talc insufflations REPE may occur secondary to talc related inflammatory reaction [9].

Animal models have been studied to help elucidate the unclear pathogenesis of REPE. It is suggested that following collapse, the blood flow in the affected lung is significantly reduced from hypoxic pulmonary vasoconstriction. With rapid re-expansion, the pulmonary vasoconstriction resolves acutely. This leads to rapid oxygenation and exposure to reactive oxygen species. Concomitantly, there is endothelial damage from an increased presence of lipid and polypeptide mediators and immune complexes along with altered and increased trafficking of the inflammatory cells that release mediators in the affected lung to result in pulmonary edema [10].

REPE may be prevented by controlled decompression allowing slow and small re-expansion at several sittings. The treatment is essentially supportive with oxygen supplementation [11]. Occasionally, non-invasive continuous positive pressure ventilation or even invasive mechanical ventilation deems necessary [12, 13]. Differential lung ventilation may be attempted in extreme situations [14]. The role of pharmaceutics in treating REPE remains uncertain.

CONCLUSION:

The patients been presented are classical cases of REPE with risk factor of prolonged collapse of the affected

lungs and rapid decompression by intercostals drainage. They also elaborate the resuscitation and recovery with proper oxygen supplementation with or without intravenous fluid administration.

REFERENCES:

1. Carlson R I, Classen K L, Gollan F, Gobbel W G Jr, Sherman D E, Christensen R O: Pulmonary edema following the rapid reexpansion of a totally collapsed lung due to a pneumothorax: a clinical and experimental study. Surg Forum 1958; 9:367-371.
2. Murat A, Arslan A, Balci A E. Re-expansion pulmonary edema. Acta Radiol 2004; 45:431-433.
3. Miller W C, Toon R, Palat H, et al. Experimental pulmonary edema following re-expansion of pneumothorax. Am Rev Respir Dis 1973; 8:664-666.
4. Trachiotis G D, Vricella L A, Aaron B L, Hix W R. Reexpansion pulmonary edema. Updated in 1997. Ann Thorac Surg 1997; 63:1207.
5. Mahfood S, Hix W R, Aaron B I, et al. Re-expansion pulmonary edema. Ann Thorac Surg 1988; 45:340-345.
6. Sherman S C. Reexpansion pulmonary edema: A case report and review of the current literature. J Emerg Med 2000; 24:23-27.
7. Cinnella G, Dambrosio M, Birenza N, et al. Reexpansion pulmonary edema with acute hypovolemia. Intensive Care Med 1998; 24:1117.
8. Matsura Y, Nomimura T, Murakami H, Matsushima T, Kakehashi M, Kajihara H. Clinical analysis of reexpansion pulmonary edema. Chest 1991; 100:1562-1566.
9. De Campos J R M, Vargas F S, Werebe E D C, et al. Thoracoscopy talc poudrage. Chest 2001; 119:801-806.
10. Sivrikoz M C, Tuncozgur B, Cekmen M, et al. The role of tissue reperfusion in the reexpansion injury of the lungs. Euro J Cardiothorac Surg 2002; 22:721-727.
11. Rozenman J, Yellin A, Simansky D A, Shiner R J. Re-expansion pulmonary oedema following spontaneous pneumothorax. Respir Med 1996; 90(4):235-238.
12. Tariq S M, Sadaf T: Images in clinical medicine. Reexpansion pulmonary edema after treatment of pneumothorax. N Engl J Med 2006; 354(19):2046.
13. Papakonstantinou D K, Gatzioufas Z I, Tzegas G I, et al. Unilateral pulmonary oedema due to lung re-expansion following pleurocentesis for spontaneous pneumothorax. The role of noninvasive continuous positive airway pressure ventilation. Int J Cardiol 2006.
14. Cho S R, Lee J S, Kim M S: New treatment method for reexpansion pulmonary edema: differential lung ventilation. Ann Thorac Surg 2005; 80(5):1933-1934.