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RESEARCH ARTICLE

ASYMPTOMATIC RE-EXPANSION PULMONARY EDEMA - A RARE OCCURENCE IN CLINICAL PRACTICE

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ABSTRACT

Re-expansion pulmonary oedema (RPE) is a rare but often fatal complication which occurs due to rapid re-expansion of collapsed lung following treatment of conditions like a spontaneous pneumothorax or massive pleural effusion. The patho-physiology is still largely unknown, some of the reasons postulated include decreased surfactant levels, regional hypoxia, and hardened pulmonary microvasculature in collapsed lung and pro-inflammatory environment. Clinical manifestations vary from mild or no symptoms to life threatening hypoxia and cardio-respiratory failure. Early diagnosis and prompt treatment can save lives. Because of rarity of this fatal condition, we report a case of chronic obstructive pulmonary disease (COPD) with spontaneous pneumothorax who developed RPE following insertion of intercostal drainage (ICD) tube.

INTRODUCTION

Cases of Re Expansion Pulmonary Odema have been reported as early as 1853 (by Pinault). Patients who are commonly predisposed to RPE include male population, rapid re expansion of collapsed lung (> 3 days) and pleural tap volume of > 2000ml. Symptoms of RPE most commonly start within the first two hours after pulmonary expansion and last for 1-2 days. Symptoms usually disappear in a week. Clinical features range from being asymptomatic to dyspnoea of varying intensity, chest pain, cough with blood tinged sputum, cyanosis and on clinical examinations crackles are heard in the expanded lung. Some patients may develop respiratory distress and hemodynamic instability needing ionotropic & invasive/non-invasive ventilatory support. Chest skiagram may show pulmonary edema, interstitial shadows, consolidation, & air bronchograms. Clinicians should promptly differentiate this from cardiogenic causes of pulmonary odema (ECG/2D-Echocardiography).

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CASE REPORT

A 60 years old male reported to emergency department with complaints of on and off breathlessness since 7-10 days which was insidious in onset, initially it was present on slight exertion but now present even at rest (grade II NYHA to grade IV NYHA). History of recurrent cough with scanty whitish non foul smelling sputum since last 7 days. Chest pain since 4-5days which was insidious in onset, vaguely present all over chest, but since 6hrs it became very severe, present on right half of chest and patient said his breathing difficulty increased during that period. This was not associated with palpitation, sweating, syncope, swelling of limbs and facial puffiness. Patient was a known case of COPD for the last 7 years and has been on irregular treatment with inhalers and oral bronchodilators (tab deriphylline retard 300mg od). Patient was a known beedi smoker (40 beedi's/day for last 30yrs) and occasional alcoholic (1quarter local whisky every week). Patient is illiterate and manual labourer by occupation and belongs to low socio- economic strata. No significant past or family history present. On examination, patient had a pulse of 118 beats/min, regular, thready in nature, blood pressure was

98/58 mmHg in right upper limb in supine position, respiratory rate was 32/min, accessory muscles of respiration were being used. Pallor was present. No clubbing, cyanosis, odema or significant lymphadenopathy present. BMI was 18.5 kg/m2. Chest examination revealed decreased expansion on right side; trachea shifted towards left; hyper-resonant percussion note with absent breath sound on the right side. SpO2 was 80%, which improved to 93% on oxygen inhalation at the rate of 6l/min. The patient was shifted to ICU for hemodynamic monitoring and respiratory support. Urgent ABG done revealed pH – 7.23, pO2 – 51, pCO2 – 55 HCO3 – 19, sodium – 141, potassium – 4.0. ECG, RBS done was within normal limits. Urgent CXR- PA view (Fig.1) showed pneumothorax on the right side with collapse of lung (Fig.1).

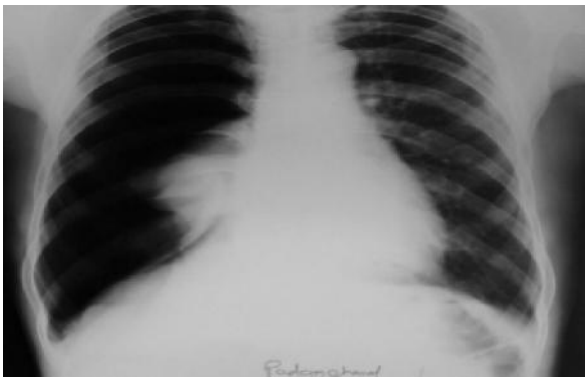

Fig 1. Pneumothorax on right side with collapse of ipsilateral lung

Resuscitation started with 500ml of ringer lactate given fast over 15min, followed by NS/DNS @ 75ml/hr. After informed consent, emergency ICD was performed under local anaesthesia using 28G PVC ICD tube and connected to under water seal drainage. Air leak was present. Patient was apparently relieved of dyspnoea and his SpO2 improved to 99% and oxygen was slowly tapered off in next 4hrs. Pulse rate improved to 75/min, BP increased to 110/65mm Hg and respiratory rate was 20/min. He was started on injection-ceftriaxone 1gm i.v. twice daily, inj pantocid 40mg once daily, inj ondansetron 4mg iv sos, iv inj tramadol 100mg iv sos along with nebulized salbutamol + ipratropium bromide three times a day. A CXR-PA view done later showed complete expansion of lung (Fig.2).

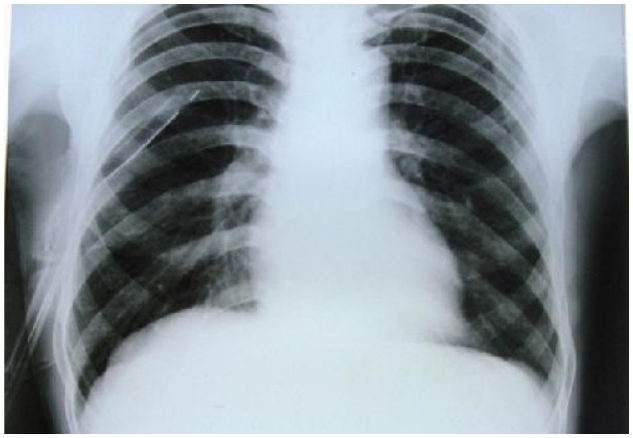

Fig 2. Post ICD insertion, expanded right lung with ICD in situ

Blood investigation (hemoglobin, TLC/DLC) was within normal limits. 24 hrs after ICD insertion (day-2), chest examination revealed crepitations over entire right lung. Rest of the clinical examination was within normal limits. He was having cough but without much expectoration, was afebrile and haemodynamically stable and his respiratory rate was 23/min, regular SpO2 was 91% without oxygen which improved to 99% with O2 face mask @ 5l/min. A CXR-PA view was taken, which showed non- homogenous opacity involving predominantly middle and lower zone of the right lung (Fig.3).

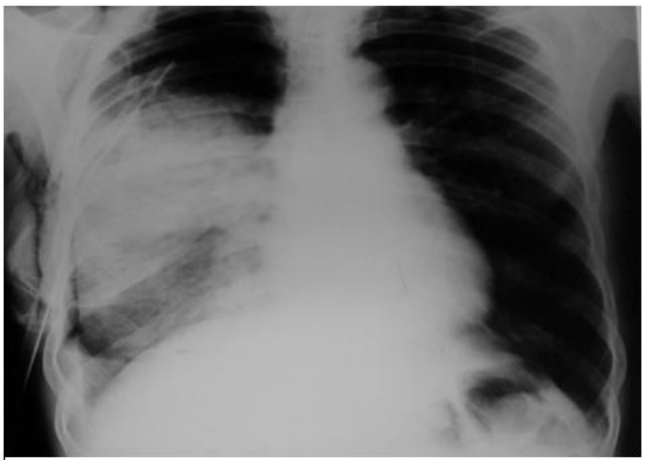

Fig 3. Expanded lung following ICD insertion along with an opacity in right mid and lower zones

ABG done depicted pH– 7.28, pO2 – 63, pCO2 – 52, HCO3 – 19, sodium – 144, potassium – 3.6. Cardiology opinion was sought and 2D Echo done, showed no regional wall motion abnormality, no valvular abnormality, no vegetations, ejection fraction of 60%, no signs of cor-pulmonale. Sputum was sent for microbiological analysis (gram stain, culture sensitivity and AFB). Blood investigation (hemoglobin, TLC/DLC, total protein/ serum albumin) was within normal limits. CECT/HRCT (thorax) was advised to patient which was refused because of financial constrains. As the patient improved on oxygen support, no further changes in management was done. Patient continued to improve clinically and was shifted out of ICU on day-3. Sputum analysis report on day-5 was sterile. Air leak from ICD stopped completely on day-7 and CXR done showed clearing of lung opacity (Fig.4).

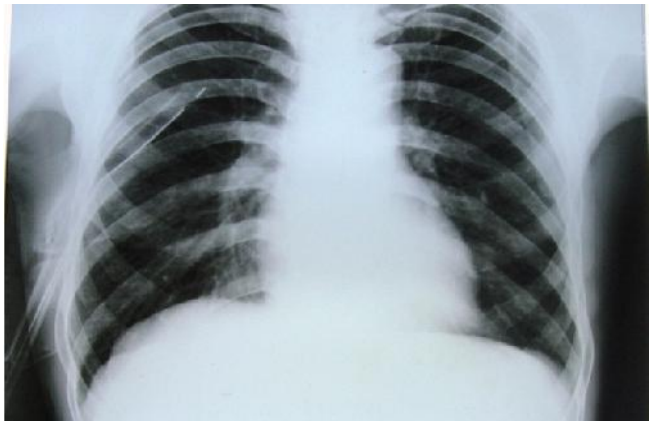

Fig 4. Follow up CXR showing clearing of opacity on right side

ICD was subsequently removed on day-8. Patient was discharged on day-9. He was advised to come after 15 days for further follow up and management.

DISCUSSION

RPE is defined as pulmonary odema occurring in a rapidly re-expanded collapsed lung (of usually >3day duration). This case being reported by us shows asymptomatic re-expansion pulmonary edema after insertion of emergency ICD for right pneumothorax which is a rare occurrence in clinical practice. Patient was a known case of COPD on irregular treatment and acute breathlessness with decreased air entry and suggestive CXR showed probably rupture of subpleural bulla. He was relieved of acute breathlessness after insertion of ICD. His spontaneous pneumothorax was probably > 3day old and respiratory distress made him seek medical advice. Fortunately for us and the patient, the reexpansion pulmonary edema remained symptom free, which seldom occurs. Other causes for pulmonary odema like cardiac causes/hypoprotinemia was sequentially ruled out by us. Pneumonia as the cause of infiltrates was also ruled out as there was no fever, purulent sputum or leucocytosis. In 1853, Pinault probably reported for the first time, a case of pulmonary edema and respiratory failure after pleurocentesis of a massive pleural effusion (Kernodle *et al.,* 1984). Carlson (1958) described the first case of RPE following pneumothorax drainage (Carlson *et al.,* 1959). There are reports of RPE in patients after removal of large quantities of cystic fluid from giant hepatic cyst (Fakuda *et al.,* 1989), after excision of mediastinal tumour (Matsumiya *et al.,*1991) and after decortications (Yamanaka *et al.,* 1998). In context of pathogenesis of RPE, Mahajan *et al.* (1979) suggested that edema resulted from rapid blood flow during reexpansion which increased pulmonary capillary pressure leading to overflow of protein and fluid into the alveoli and interstitium. In 1882, Mariend and Glauser (Matsuura *et al.,* 1991) confirmed this hypothesis by comparing protein contents of tracheal secretion in cardiogenic (0.5) and reexpansion (0.85) pulmonary edema. Mahfood *et al.* (1988) suggested hypoxia and alveolar- capillary lesions due to prolonged lung collapse as major contributors of RPE. Nakamura in 1994 demonstrated the role of polymorphnuclear cell, interleukin-8, and monocyte chemotactic protein-1 in the genesis of RPE. In 1997, Trachiotis (1997) published another extensive review and concluded, majority (83%) of the RPE cases occurred in patient with prolonged pulmonary collapse (more than 72 hrs) leading to alteration in the permeability and capillary pressure and surfactant loss. In their review of 47 cases of RPE in pneumothorax cases, Mahfood *et al.* (1988) found that RPE is more common among males in a ratio of 38:9 with an average age of 42 years (range- 18- 84 years). Thirty nine (83%) out of these 47 cases had collapsed lung for more than 3 days. Thirty (64%) had onset within 2 hrs after reexpansion, and in all patients the onset occurred within 24 hrs. Three patients also had edema of contralateral lungs, 2 of them died. The overall mortality was 19% (9 out of 47 died), 7 (80%) were above 50 years. It is very important to differentiate RPE from pulmonary infection/ pneumonia, cardiogenic pulmonary edema, especially in elderly patients as the treatment and outcome varies with the diagnosis. Timely arterial blood gas analysis, CXR, blood investiagations, sputum microscopy, electrocardiogram and echocardiogram help in differentiating various causes of pulmonary odema/infection. Once a diagnosis of RPE is confirmed, treatment is based on oxygen supplementation and respiratory (invasive/non-invasive ventilatory) support depending upon the seriousness of clinical condition of the patient. Prevention of RPE is based on careful gradual graded pleural tapping or Intercostal drainage procedures. Avoiding rapid re-expansion of chronically collapsed lung is the most reliable way of preventing extensive pulmonary microvascular injury and RPE. Some suggest use of steroid may stabilise pulmonary microvasculature and provide some benefit in management of RPE. Use of lodoxamide has been shown to limit leukocyte sequestration and plasma leakage in some studies on RPE. Risk factors like time of evolution of the pleural effusion longer than 72 hours and estimated volume more than 1500 ml, pulmonary hypertension, and cardiovascular- pulmonary diseases should be observed and corrected. Several studies suggest that the ideal volume should not be more than 1000 ml (Fakuda *et al.,* 1989; Matsumiya *et al.,* 1991; Mahajan *et al.,* 1979). Mahajan *et al.* (1989) suggested a safe limit of 1500 ml. Pleural pressure monitoring (Light *et al.,* 1980) done to drain large amount of fluid has in some studies shown benefit in preventing RPE.

Conclusion

RPE is a permeability pulmonary odema associated with injury of pulmonary micro-vessels. RPE is a rare clinical entity with a high mortality, particularly in elderly age group. It is very important for all practising physicians to be aware of this complication while treating commonly occurring conditions like pneumothorax or pleural effusion. All precautionary measures should be taken to avoid this complication. The clinicians should always be watchful of this complications and should never under-estimate the importance of timely ECG and 2D-Echo in ruling out cardiac cause of pulmonary odema. We in this case have come across re-expansion pulmonary odema which remained asymptomatic and didn't require any active intervention. So, a watchful observation of clinical condition (pulse, spO2, BP) in some patients may avoid any further active intervention (invasive/non-invasive ventilation) as chest skiagram may reveal dubious findings.

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