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CASE REPORT

IPSILATERAL RE-EXPANSION PULMONARY OEDEMA AFTER DRAINAGE OF A SPONTANEOUS PNEUMOTHORAX

K. N. Mohan Rao[1], Tarun Sharma[2], Venkata Siva Prasad Reddy[3]

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ABSTRACT: We report a case of an ipsilateral re-expansion pulmonary edema occurring after the insertion of an intercostal drainage tube in a young male patient with spontaneous pneumothorax. The patient was managed with oxygen via a non-rebreathing face mask to compensate for his hypoxemia. In about 24 hours after the event, the patient gradually improved and recovered completely without any residual hypoxemia. Re expansion pulmonary edema occurring after the insertion of an intercostal drainage tube for pneumothorax or pleural effusion is a rare complication with a high mortality rate up to 20%. This condition should be considered if the patient develops cough, dyspnea and hypoxemia following the insertion of a chest tube. The exact pathophysiology leading to this complication is still unknown. The risk factors for re-expansion pulmonary edema should be evaluated and considered prior to the insertion of chest tubes. Treatment remains supportive only.

INTRODUCTION: In 1853, Pinault[1] described REPE following pleural effusion in a patient who had a large volume of pleural fluid rapidly removed by thoracentesis. This is probably the first report of REPE. In 1958, re expansion pulmonary edema (REPE) was first reported following pneumothorax by Carlson and colleagues[2] and was comprehensively reviewed by Ziskind and colleagues[3] in 1965. In 1991 Matsuura in his clinical analysis of 146 patients summarized that 21 of 146 cases of spontaneous pneumothorax which were treated by Thoracentesis and continuous low negative pressure suction drainage (- 12cm H20) of the pleural space developed REPE, The rate of REPE was higher in patients 20 to 39 years of age than in those over the age of 40.[4] REPE can occur on the ipsi- or contralateral side, can be bilateral and can even be asymptomatic.[5-9] The exact pathophysiology for this complication is unknown. Factors that have been implicated in the pathogenesis of this complication include chronicity of collapse, technique of re-expansion, increased pulmonary vascular permeability, airway obstruction, loss of surfactant and pulmonary artery pressure changes.[10] Oxygen radicals are produced during the hypoxemia in the collapsed lung. Moreover, the activity of different cytokines such as interleukin 8 and monocyte chemo attractant protein 1 (MCP-1),[11] or the activity of xanthine oxidase[12] have been implicated in the pathogenesis of REPE. Additionally, the GJP-binding protein Rho and its target Rho-Kinase (ROCK) identified by Sawafuji et al. has been implicated in permeability changes causing RPE as well.[13]

CASE PRESENTATION: A 27 years old male patient presented to our emergency department 12 hours after the onset of a sudden left-sided chest pain with associated breathlessness. His medical history was unremarkable. He was an electrician and denied any history of strenuous work or any trauma. The patient was otherwise a healthy well built male with normal vital signs, including respiratory rate (16/min) and oxygen saturation (96% on room air). His height was 184 cm and

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weight was 58 kilograms; rest of the general physical examination was normal. On auscultation he had diminished breath sounds on the left side. His labs showed RBS-90 mg/dL, Urea 18 mg/dL, creatinine 0.72 mg/dL, sodium 139 meq/L, potassium 4.2 meq/L, Haemoglobin 14.5 g/dL, total leukocyte count 6000 cells/cumm. DLC showed -N 67%, L 30%, M 3%, Platelets were 2.54 L/cumm

An emergency Chest X-ray was done, which confirmed the clinical suspicion of a left sided large pneumothorax with left side total collapse of the lung with slight mediastinal shift probable starting of a tension pneumothorax (figure 1). A chest tube was inserted under local anesthesia in the left mid axillary line without complications and connected to underwater seal drainage system in the emergency department.

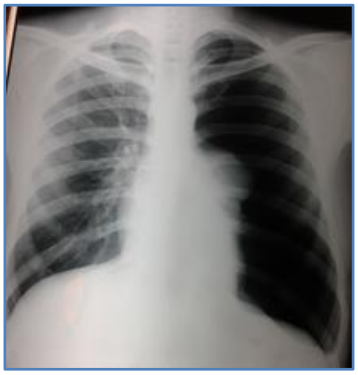

Fig. 1: CXR suggestive of large left sided pneumothorax

After about 3 hours of intercostal drain insertion and relief of symptoms, the patient complained of cough with whitish expectoration, dyspnea and increasing pain in the left side of chest. Chest auscultation revealed left-sided inspiratory crackles. Patient had increased breathlessness with RR of 35/min and BP 100/70 mmHg. Oxygen saturation was decreased to 85% despite the addition of oxygen (initially 2 l/min via nasal cannula). Later oxygen saturation stabilized at 90% with 12lt/min oxygen via a non-rebreather face mask.

The arterial blood gas analysis under 12lt/min oxygen showed hypoxemia: pO2 60 mm Hg (80 to 100 mm Hg), pCO2 35 mm Hg (35-45 mm Hg), pH 7.36 (7.35 - 7.45), bicarbonate 24 mEq/L/l (21–28 mEq/L) and oxygen saturation of 90%. A second chest X-ray was done which, demonstrated an expanded left lung, but also an ipsilateral pulmonary edema (figure 2).

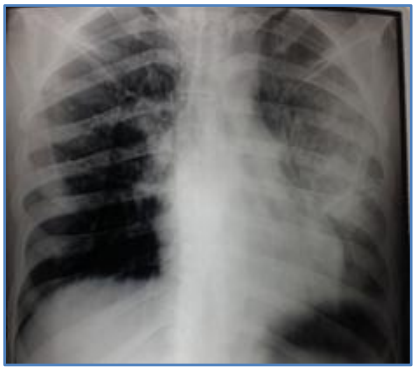

Fig. 2: CXR suggestive of Pulmonary edema in the left lung

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The patient stabilized under continuous oxygen (12 l/min via a non-rebreather face mask) and his O_2 saturation gradually normalized. He did not require any further medical management. The patient was transferred to the respiratory ward from emergency ward. He was continuously observed in the ward and hourly clinical observations were done, which were unremarkable over the course of next 12 hours. His oxygen was gradually tapered off and stopped after 24 hrs and a chest x-ray was repeated which showed decrease in the pulmonary edema. (figure 3)

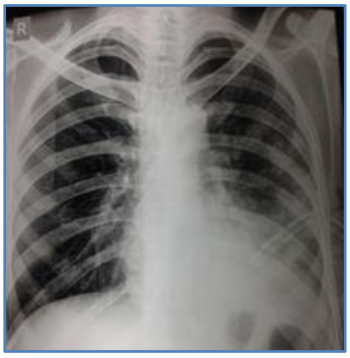

Fig. 3: Chest X-Ray showing reduction in the pulmonary edema with ICD in situ

The chest drainage was removed when no more air leakage was detected on day 2 of hospitalization. A repeat CXR showed complete resolution of the pulmonary edema. The patient was discharged home without any complications. He was called to the outpatient clinic after 15 days and then after 2 months and was found to be in excellent health with normal oxygen saturations. His CT was deferred in view of normal follow-up chest x-rays.

DISCUSSION: REPE is a known entity in pneumothorax, which is more commonly seen in elderly people with underlying lung co-morbidity and having long standing pneumothorax.

Usually young people with pneumothorax withstand the procedures well, which was not seen in our case, who is a young man of 27 years who can withstand the procedure related complications but developed REPE and the history was very short for any structural changes in the lung parenchyma.

Our patient was diagnosed with this rare complication but had a benign clinical course. He was successfully managed with only supplemental oxygen via a non-rebreather face mask. His oxygen saturation gradually improved and stabilized and he was weaned off oxygen within 24 hours.

Usually REPE is self limited and can even be asymptomatic.[5] However, a mortality rate as high as 20-22% has been described.[10,14] It is thought to be caused by increased pulmonary capillary permeability. Factors that have been postulated to contribute to altered permeability include lung hypoxia, chronicity of pulmonary collapse, rapid re-expansion and subsequent occurrence of strong negative pressure in the thorax, application of excessive intrathoracic suction pressure, rapid increase in blood flow to the involved lung during re-expansion, an increase in the pressure gradient between the alveolar space and the pulmonary capillary, disturbance of lymphatic flow in the 1ung, and destruction of lung surfactant.[4]

The clinical features of REPE are observed in patients with a lung collapse period of 3 days or more, an evacuation volume of 2000 ml or more, a period of less then one hour from re-expansion to the onset of REPE; and the type of pulmonary edema is permeability pulmonary edema.[15]

As the patient's height is 184, which is also a predisposing condition for pneumothorax, rapid re-expansion in tall statured young individuals might have predisposed to an alveolar capillary membrane injury leading to pulmonary edema. Hence we thought it is an interesting case and for the readers to be cautious and be aware of this complication which can occur in any setting

Summarizing, REPE is a rare but severe complication after the insertion of a chest tube for spontaneous pneumothorax and can be lethal. Slow re-expansion of the pneumothorax and minimal or no negative pressure should be applied if risk factors are present in order to avoid this life threatening complication.

CONCLUSION: Re-expansion pulmonary edema is a rare complication with high mortality rate in situations without ICU facilities occurring after thoracic drainages for pneumothorax or pleural effusions. Clinicians should be familiar with this complication and the associated risk factors. Treatment is symptomatic.

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AUTHORS:

1. K. N. Mohan Rao
2. Tarun Sharma
3. Venkata Siva Prasad Reddy

PARTICULARS OF CONTRIBUTORS:

1. Professor & HOD, Department of Respiratory Medicine, Rajarajeswari Medical College & Hospital, Mysore Road, Bangalore.
2. Post Graduate, Department of Respiratory Medicine, Rajarajeswari Medical College & Hospital, Mysore Road, Bangalore.
3. Post Graduate, Department of Respiratory Medicine, Rajarajeswari Medical College & Hospital, Mysore Road, Bangalore.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. K. N. Mohan Rao,
Department of Respiratory Medicine,
Rajarajeswari Medical College & Hospital,
Mysore Road, Bangalore – 560074.
E-mail: kotnur.rao@gmail.com

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