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ORIGINAL RESEARCH

Case report- re expansion pulmonary edema [REPE] with underlying alcoholic cardiomyopathy: A deadly duo

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Introduction
Reperfusion Edema [REPE] is a rare but near fatal complication which can occur after chest tube insertion for treatment of pneumothorax or pulmonary effusion. Clinical presentation includes cough, chest discomfort and hypoxemia; if the edema is severe, shock and death may ensue. Symptoms are usually noted within 24 hours after thoracentesis. Treatment is generally supportive, ranging from oxygen supplementation to noninvasive and invasive ventilation. Since the complication can be life threatening, it needs to be recognised and treated promptly. Preventive strategies include the use of low negative pressure (< −20 cm H_2O) for suction during thoracentesis and limiting drainage of pleural fluid if the patient reports chest discomfort. REPE occurring in a patient with underlying undiagnosed alcoholic cardiomyopathy can have deadly consequences if not managed promptly. Here we report the successful management of a young male with previously undiagnosed alcoholic cardiomyopathy who developed REPE following chest tube insertion for drainage of massive pleural effusion.
Keywords: REPE, Chest Tube, Alcoholic Cardiomyopathy

Case report
A 40 year old male reported to emergency with chief complaints of pain in right upper abdomen and bleeding per rectum which was frank red in colour since 2 days. Patient gave history of intermittent fever since 10 days which was not associated with any chills or rigors and history of constipation since 5 days. He also complained of reduced appetite since 10 days. Personal history revealed a history of smoking since 20 years along with history of alcohol consumption since last 10 years which approximated to 240 ml per day. Past history revealed history of jaundice one year back which was managed conservatively. On general physical examination, patient was haemodynamically stable. Respiratory examination revealed reduced breath sounds in right basal area on auscultation. Upon abdominal examination, it was observed that the abdomen was soft, non distendedbut tenderness was noted in the right upper quadrant. Rest of examination was within normal limits. Baseline haematological investigation showed Hb: 6.9 g/dl , TLC: 12,100, potassium (K): 2.9 mmol/L, total bilirubin: 2 mg/dl, total protein: 5.6 g/dl, albumin 2.1, AST: 30 U/L, ALT: 21 U/L, ALP-304 U/L and non reactive viral markers. Rest of the investigations were within normal limit .Chest Xray showed mild pleural effusion on(R)side .ECG showed sinus tachycardia. USG abdomen revealed multiple, heterogeneous(17cm) hypoechoic space occupying lesions

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(SOL) in liver, largest being 50.71cc in segment VI of liver likely abscess, mild splenomegaly , thickened and oedematous GB wall with minimal pericholecystic fluid and minimal inter bowel free fluid. Patient was diagnosed as a case of USG documented liver abscess and managed conservatively for 48 hrs post admission. Subsequently, patient developed tachycardia and multiple episodes of fever. In view of underlying clinical suspicion of ruptured liver abscess, patient was planned for emergency exploratory laparotomy. Preoperatively the patient's vital signs were pulse rate (PR) of 104 beats per minute (bpm), blood pressure (BP) measuring 126/74 mmHg, respiratory rate (RR) of 25 breaths per minute, Spo2 94% on room air and a temperature of 98.6. Due to the patient's inadequate fasting status a modified rapid sequence intubation (RSI) was performed. Intraop analgesia was provided by Inj fentanyl (100ug) and inj. paracetamol(1gm). Intraoperative findings revealed an adherent omentum in the subhepatic area, and a ruptured liver abscess cavity in the liver's VI segment. About 1.5 liters of seropurulent fluid was drained from the peritoneal cavity. Intraoperatively patient was haemodynamically stable and extubation was uneventful at the conclusion of surgery. Intraoperatively patient received one unit of PRBC and was kept under monitoring in the surgical intensive care unit (SICU). The patient was shifted to ward on the Ist POD in a clinically stable condition on oxygen supplementation via nasal prongs @6 lit/min. Over the course of the next 24 hours, he was managed in the ward, where he had been complaining of increasing shortness of breath. His oxygen requirements had increased and he was put on high flow oxygen face mask. On 2nd POD, Chest x ray was done which showed right sided moderate pleural effusion. In view of clinical deterioration and chest Xray findings, an intercostal drainage tube (ICD) was inserted on the right side. About 2 hours following the insertion of ICD, the patient started complaining of of severe difficulty in breathing, accompanied by excessive perspiration, restlessness and irritability. Consequently patient was emergently transferred back to the SICU for further intervention and management. On arrival in SICU, he was alert but restless, agitated, tachycardic and tachypneic with profuse perspiration . The patient's vital signs were a HR of 141 bpm, BP of 151/100 mmHg, RR of 45 breaths per minute, and oxygen saturation (SpO2) of 55% on room air and 62% on non-rebreathing mask (NRBM) at a flow rate of 12 liters per minute. The auscultatory examination revealed the presence of bilateral coarse crepitations with ECG showing sinus tachycardia. Arterial blood gas (ABG) showed a pH: 7.07, pCO2: 60 mmHg, pO2: 51 mmHg, lactate: 8.4 and HCO^{3-}: 17.4.The chest tube drainage bag was blood filled with 1.5 liter of serosanguineous fluid. The patient received supplemental oxygen therapy via non-rebreathing mask (NRBM) at a flow rate of 14 liters per minute along with IV frusemide and morphine. Patient"s saturation failed to improve with high flow oxygen therapy and hence was immediately intubated in view of impending respiratory failure. On intubation pink frothy secretions were seen in the ET tube following which thorough endotracheal suctioning was done and mechanical ventilation started. The chest radiograph revealed bilateral blunting of costophrenic angle and diffuse pulmonary vascular congestion in both lung fields suggestive of pulmonary edema. 2D ECHO was done which revealed left atrial enlargement, left ventricular enlargement, left ventricular ejection fraction (LVEF) of 30-35%, global hypokinesia, and dilation of the inferior vena cava (IVC).Subsequently, the patient received all-encompassing care, which included restricted fluid intake, diuretics, sedation, analgesia, and high positive end expiratory pressure (PEEP) mechanical ventilation. The patient's condition improved overnight, and his blood gas values normalized. Following improvement in clinical, ventilator and ABG parameters, patient was gradually weaned off the ventilator over next 24 hours and extubated successfully. Patient was monitored and managed conservatively in ICU over next 48 hrs and uneventfully shifted to ward.

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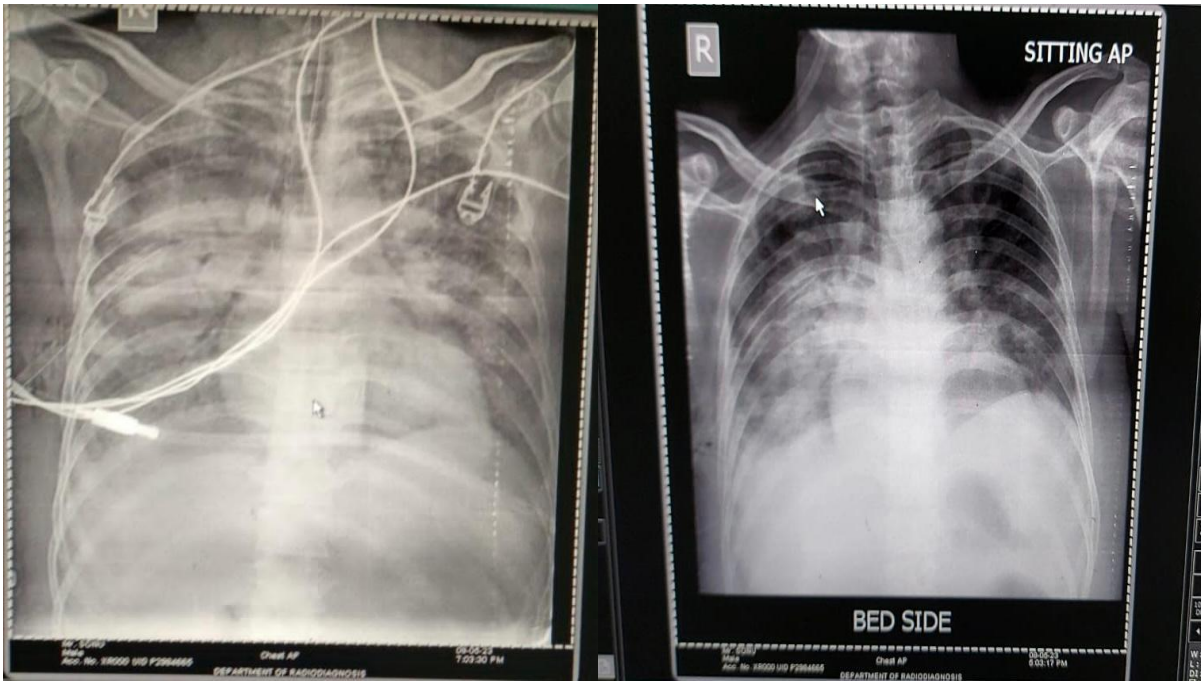

Discussion

The possibility of re-expansion pulmonary edema following drainage of pleural effusion or pneumothorax has been recognized for decades.[**1**] The reported incidence following drainage of a pleural effusion and pneumothorax has been between 0% and 1% in most studies.**[2]** These estimates likely reflect widespread under-reporting, since re-expansion pulmonary edema in many instances is clinically mild and detected only using radiography.[**3]**The incidence of REPE, including all etiologies, is 0.9% to 20%, but the majority occurs when the pleural effusion is rapidly drained, and upon pneumothorax, the incidence is <1%[**5-7]** REPE is a rare complication with an incidence of < 1 % in pneumothorax but a mortality rate of up to 20 % and so is very fatal [**5,6,8]** REPE typically manifests unilaterally and arises when the lung undergoes swift re-expansion due to the active removal of substantial quantities of air or fluid from the pleural cavity. [**6]** .Although the pathophysiology of re-expansion pulmonary edema is multifactorial and poorly understood, new investigations are uncovering possible mechanisms. One of the more promising theories suggests that the root of the condition is increased permeability of the pulmonary capillaries as a result of inflammation. Ventilation and reperfusion of a previously collapsed lung may lead to an inflammatory response, with production of reactive oxygen species and superoxide radicals, a sequence of events that ultimately results in increased capillary permeability. Inflammatory mediators, including interleukin 8, leukotriene B4 and monocyte chemotactic activating factor, are pivotal in this inflammatory response[**4]** Another recent study identified a signaling pathway of the small guanosine triphosphate-binding protein Rho and its target protein ROCK (Rho-associated coiled–coil-forming protein kinase) as a possible mechanism. The activation of Rho via the action of its target protein causes phosphorylation of myosin light chains, actomyosin contraction and dysfunction of the endothelial barrier cells.[**11]**Alternatively, research suggests that mechanisms such as increased pulmonary hydrostatic pressure caused by enhanced venous return, pressure-induced mechanical disruption of the alveolar capillaries, decreased levels of functional surfactant, increased pressure across the capillary–alveolar membrane from bronchial obstruction and altered lymphatic clearance may also lead to re-expansion pulmonary edema in some patients**.[3]** Increased permeability of damaged pulmonary blood vessels due to rapid re-expansion of lung tissue, histological changes in the lung parenchyma, and reperfusion of

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ischemic lungs after re-expansion, increased oxygen free radicals and anoxic stress, thereby resulting in damage to the vascular endothelium. These damages have been suggested as the onset mechanism of REPE[**8,9,10]**

Risk factors of REPE include young age (<40 years old),large pneumothorax (>30%) or large amount pleural effusion, long duration of symptoms and lung collapse (>3 days), rapid reexpansion of the lungs(<10 min)and pleural drainage of 1.5 litres or more at once, and negative pressure suction drainage.[**8,9,1213]**. Several authors recommend that no more than 1 L of air or fluid be drained at a time, and that a water valve device should be used instead of suction**.[8,10,14–16**] The maximum amount of drainage at once should not exceed 1200 to 1800 ml.[**8]** However, pneumothorax, unlike pleural effusion, is difficult to drain them progressively, and the intermittent clamping of a chest tube may cause subcutaneous emphysema.[**9,17]** Thus, some authors suggest that, if there are such risk factors, inserting a chest tube after repeatedly aspirating less than 1000 cc of air with a syringe may be a safe and practical solution.[**14]** Recent evidence suggests that large-volumes can be safely drained as long as pleural pressures are monitored.[**1,18**] If the patient reports vague chest pressure during thoracentesis, this may indicate a precipitous drop in intrapleural pressure, and the thoracentesis s*hould be stopped. Pleural manometry is being increasingly advocated for the drainage of large pleural effusions.*[**18]**

Typically, symptoms manifest within a 24-hour timeframe, with around 64% of patients experiencing symptom onset within 1 to 2 hours subsequent to lung re-expansion [**3]** Our patient too had risk factors such as young age and rapid and massive drainage of pleural fluid(1.2 litres stat within 5 minutes). The diagnosis mostly relies on radiological findings which reveals the presence of ground-glass opacity or consolidation in either the collapsed lung or the lung on the opposite side. This manifestation may or may not be accompanied by respiratory symptoms [**5]**. Clinical features include cough, chest pain, intractable cough with frothy sputum, cyanosis, hypoxemia, hypotension, opacification of lung field. Expectoration of frothy sputum is hallmark of severe REPE and patient needs immediate treatment [**4]**. Although most patients completely recover within five to seven days, severe re-expansion pulmonary edema can lead to sequestration of large quantities of fluid in the lung, which may result in shock and possibly death.[**3,4]**

In cases of (REPE), the standard approach to treatment typically involves conservative and supportive measures. Various interventions can be implemented based on the specific condition of the individual, including the administration of supplemental oxygen, the application of diuretics, endotracheal intubation, and the use of mechanical ventilation [**5]** Management is generally supportive but varies by severity of the condition. Whereas oxygen supplementation may prove adequate in patients with mild symptoms, those with severe symptoms require endotracheal intubation and mechanical ventilation.[**3]** In patients with worsening symptoms, the use of noninvasive ventilation with bi-level positive airway pressure may help to circumvent the need for endotracheal intubation.[**19**] Having the patient lie on his or her unaffected side is therapeutic in unilateral pulmonary edema. The lateral decubitus position allows a patient to bring the affected area upwards, and so to reduce shunting and increase oxygenation. Unilateral ventilation is rarely required.[**8,20]** Since intrapulmonary shunting can cause hypoxia or hypovolemia, fluid supply or use of inotropes is required. Also, the use of diuretics should be avoided as they may worsen hypovolemia.[**8,9,13**] Evidence supporting the use of diuretics, bronchodilators, prostaglandin analogues (e.g., misoprostil), ibuprofen and steroids remains anecdotal.[**19**]]

In our case the patient had undergone chest tube insertion in view of progressively increasing pleural effusion but there had been a rapid stat drainage of 1 lire of pleural fluid which was subsequently followed by acute respiratory distress and fall in oxygen saturation rapidly progressing to respiratory failure which was managed promptly by initiation of mechanical

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ventilation. Post REPE, a bed side ECHO was done which revealed a dilated LA,LV., left ventricular ejection fraction (LVEF) of 15-20%, global hypokinesia, and dilation of the inferior vena cava (IVC), thus suggesting a probable diagnosis of underlying Alcoholic cardiomyopathy. Our patient had undergone an uneventful emergency exploratory laparotomy 48 hrs before and there was no history whatsoever suggestive of cardiac disease in the patient .The EcHO findings suggestive of Left atrial and ventricular dilation with low ejection fraction in presence of long standing history of heavy alcohol consumption points towards a background alcoholic cardiomyopathy. The patient presented in flash pulmonary edema post chest tube insertion although ECHO findings demonstrated that his cardiac functions were already compromised due to underlying undiagnosed alcoholic cardiomyopathy. The patient had been in a delicate cardiac compensated state which got disturbed by the episode of REPE and pushed the patient into a state of acute decompensation. The interaction of REPE with underlying alcoholic cardiomyopathy can have fatal consequencences if not managed promptly. Our patient experienced an acute on chronic insult which could have had fatal consequences.Alcohol use is an important cause for non-ischemic cardiomyopathy and accounts for 10% of all cases of dilated cardiomyopathies Alcohol-induced toxicity leads to non-ischemic dilated cardiomyopathy characterized by loss of contractile function and dilatation of myocardial ventricles. These findings are coupled with a clinical history of heavy alcohol use in the absence of coronary artery disease as a supportive etiology.The major risk factor for developing ACM is chronic alcohol abuse; however, there is no specific cutoff value for the amount of alcohol consumption that would lead to the development of ACM.[21]Changes in ventricular function may depend on the stage, in that asymptomatic ACM is associated with diastolic dysfunction, whereas systolic dysfunction is a common finding in symptomatic ACM patients. The pathophysiology of ACM is complex and may involve cell death (possibly due to apoptosis) and changes in many aspects of myocyte function. [22]The postulated mechanism includes mitochondria damage, oxidative stress injury, apoptosis, modification of actin and myosin structure, and alteration of calcium homeostasis.[21]

Our patient too had a long standing history of alcoholic consumption Investigative parameters such as gamma-glutamyl-transpeptidase (GGT), high transaminases (AST, ALT), and raised INR, are commonly observed in cases of liver injury and can indicate consumption of alcohol[21] Patients are to maintain a continuous and permanent state of refraining from consuming any form of alcohol. Treatment also includes use of an ACE inhibitor and digoxin for those presenting with symptomatic left ventricular (LV) dysfunction, alongside the utilization of diuretics to relieve symptoms. In addition to conventional treatments, administration of beta blockers, have been used in stable patients who show no signs of decompensated heart failure [22]

As such, REPE can be caused suddenly, worsen rapidly, and result in sometimes life-threatening conditions. In this regard, it is necessary to prepare in advance in mind that REPE may occur before chest tube insertion in patients with risk factors. Notably, following chest tube insertion, it is necessary to observe carefully whether REPE occurs, and to recognize it early and take appropriate measures as soon as possible when symptoms suspected of REPE appear.[2,5,15] Prompt recognition is paramount in ensuring successful treatment of re-expansion pulmonary edema. Preventive measures involve the utilization of low negative pressure (< -20 cm H2O) for suction during the procedure of tube thoracostomy, as well as the restriction of drainage to around 1 to 1.5 L of pleural fluid

In conclusion, it is important to consider the possibility of pulmonary edema when observing a deterioration in the patient's respiratory status following lung re- expansion. Physicians and surgeons managing cases of chronic lung collapse should be mindful of this complication and make efforts to prevent its occurrence, as it is far from being harmless. The optimal

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management of alcoholic cardiomyopathy necessitates an inter-disciplinary approach, wherein physicians and cardiologists collaborate closely.

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