RE-EXPANSION PULMONARY EDEMA: A COMPLEX HEART-LUNG INTERACTION IN ACUTE HEART FAILURE

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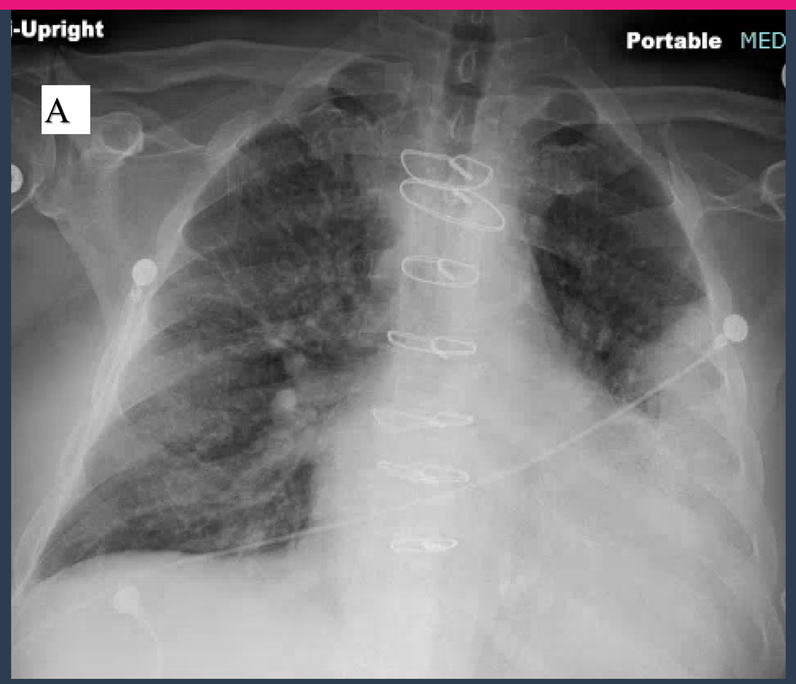
INTRODUCTION

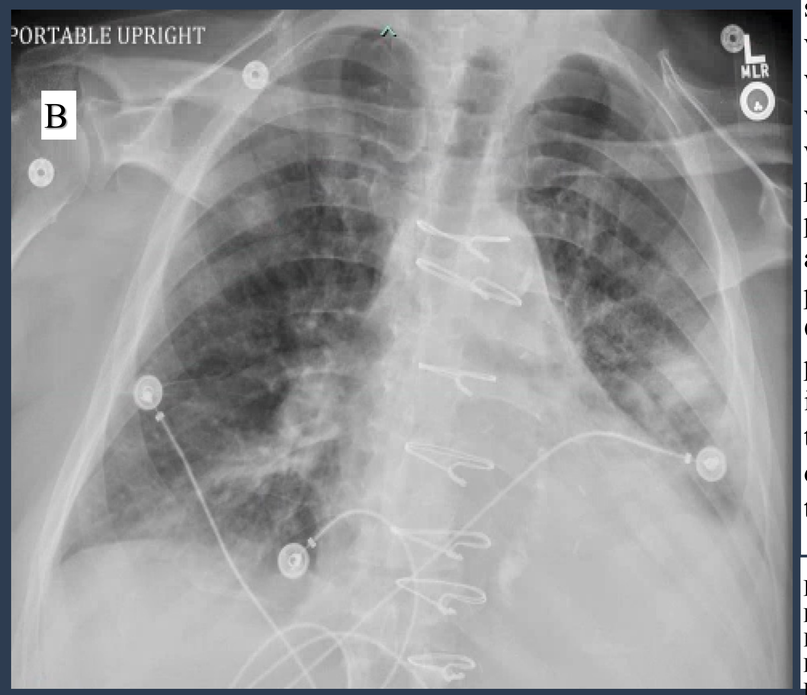
Re-expansion pulmonary edema (RPE) is a rare but serious complication manifested as hypoxic respiratory failure after lung expansion with signs of pulmonary edema on chest imaging. Possible contributory factors are high volume thoracentesis, applied negative suction pressure, duration of lung collapse, depletion of surfactant, and transcapillary hydrostatic pressure gradient in rapidly expanding lung; however, clear mechanism and pathogenesis remains unclear. We report two cases of RPE in patients with heart failure and chronic kidney disease which are suggestive of complex heart and lung interaction as the etiology of RPE.

CASE 1

68-year-old-male with heart failure with reduced ejection fraction (HFrEF), chronic kidney disease stage 3, and coronary artery disease (CAD) status post cardiopulmonary bypass surgery in the recent past, presented with worsening dyspnea, orthopnea, and edema of bilateral upper and lower extremities. Patient was hypoxic and chest radiograph (CXR) showed large left pleural effusion. Echocardiogram showed left ventricle ejection fraction (LVEF) of 45-50%, mild mitral regurgitation (MR) and moderate left ventricle (LV) concentric hypertrophy. In addition to diuretic therapy, thoracentesis was performed and 2,650 ml of transudative fluid was removed. Within 6 hours of thoracentesis, patient developed worsening dyspnea with increased supplemental oxygen requirement and repeat CXR showed worsening of bilateral pulmonary vascular congestion suggestive of RPE. Patient was treated with bilevel positive airway pressure support and an additional dose of furosemide with clinical improvement noticed within 24 hours. Patient was discharged to rehabilitation facility for physical therapy after 10-days of hospitalization.

CASE 1 IMAGES





CASE 2

64-year-old male with end-stage renal disease on hemodialysis, HFrEF, and CAD presented with dyspnea and worsening bilateral lower extremities edema. On examination, patient had diminished breath sounds in the right lung and was requiring 4-liter per minute supplemental oxygen. CXR showed large right sided pleural effusion and echocardiogram showed LVEF of 25% and moderate MR, moderate tricuspid regurgitation (TR) and moderate pulmonary hypertension. To relieve dyspnea, thoracentesis was performed, and 2,000 ml of fluid was removed. Patient remained asymptomatic at the time of procedure, however, post-thoracentesis CXR showed decreased pleural effusion but significant pulmonary alveolar and interstitial edema. Unfortunately, the patient developed severe hypoxemia due to RPE complicated by cardiac arrest within the next 4 hours of thoracentesis and did not survive the event despite resuscitation and mechanical ventilator support.

DISCUSSION

RPE is a serious condition and can lead to severe hypoxic respiratory failure and may require non-invasive or invasive mechanical ventilator support. In patients with systolic heart failure, removal of even small volume of pleural fluid has been documented to cause worsening of left ventricular end diastolic pressure (LVEDP) with a reduction in stroke volume (SV) and resulted in RPE. Our observation of more frequent RPE with increased severity in patients with chronic heart failure and a baseline poor kidney function suggest changes in heart function as a possible pathophysiologic mechanism of RPE. We need prospective studies to assess the impact of thoracentesis on cardiac hemodynamics in patients with chronic heart disease.

Conclusion: Therapeutic thoracentesis should be prudently performed in patients with compromised LV function as pleural drainage causes a drop in intrathoracic and pleural pressures which in turn can lead to a higher LV transmural pressure and an increased LV afterload which could be detrimental for a compromised left ventricle. Further studies are needed to elucidate the heart lung interaction during and after thoracentesis.

References:
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