Reexpansion Pulmonary Edema Following Thoracentesis

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INTRODUCTION
Thoracentesis is a common procedure performed for both diagnostic and therapeutic purposes. As with any procedure there are potential complications associated with removing pleural fluid from the intrathoracic space. Among these is the phenomenon known as reexpansion pulmonary edema (RPE).

Case Report
An 89-year-old gentleman with a past medical history significant for severe aortic stenosis and atrial fibrillation on warfarin therapy presented to the Providence VA Medical Center for placement of a right-sided chest tube. Several weeks prior, the patient had fallen at home and developed a hemothorax after sustaining several rib fractures. At the time of his fall, he had undergone a thoracentesis with 1 liter of bloody fluid removed. He returned for a repeat thoracentesis as he had persistent symptoms of shortness of breath and a repeat chest x-ray demonstrated re-accumulation of the pleural fluid (Figure 1).

The thoracentesis procedure itself went well without any significant complications and 2 liters of bloody fluid were removed. During the procedure, the chest tube was placed and attached to wall suction. The patient was subsequently admitted to the hospital for post-thoracentesis observation. A short time later, a rapid response was called due to hypoxia and respiratory distress when the patient’s oxygen saturation noted to be in the low 50’s despite being on a 100% nonrebreather. He was placed on BiPAP, given Furosemide 60mg and Morphine 2mg, both intravenously. The patient was subsequently transferred to the ICU and had a repeat chest x-ray (Figure 2) that demonstrated improvement in the size of the pleural effusion but the interval development of a new right lower lobe interstitial infiltrate in the area of the re-expanded lung. The diagnosis of reexpansion pulmonary edema was made on this basis and BiPAP was continued for a total of 24 hours.

The patient’s respiratory status improved over subsequent days and serial chest x-rays demonstrated improvement of the right lower lobe opacities. During this period the patient became leukopenic with a white blood cell count dropping from a baseline of 10.8 k/mm³ to a nadir of 3.9 k/mm³. On the day of discharge his white blood cell count had recovered and was noted to be 5.8 k/mm³. His chest x-ray on the day of discharge demonstrated almost complete resolution of the opacities (Figure 3).
animal models have been studied to help elucidate the cause. It is thought that RPE occurs most frequently when a chronically collapsed lung is rapidly reinfated using high suction. In a collapsed lung, blood flow is significantly reduced because of hypoxic pulmonary vasoconstriction. With reexpansion of the lung pulmonary vasoconstriction resolves and the alveoli become oxygenated. The lung reperfuses bringing in oxygen supply and reactive oxygen species may form. During reperfusion, there are increases in lipid and polypeptide mediators and immune complexes leading to endothelial damage. This alters the flow of monocytes, macrophages, and polymorphonuclear leukocytes (PMNs) to the alveolar-capillary membrane. Interestingly, due to this inflammatory response, there could be a paradoxical systemic leukopenia on routine blood work. The endpoint of the reexpansion injury is an increase in permeability of the endothelial cells, which then can lead to pulmonary edema.

Molecules that have been linked to the pro-inflammatory process in RPE include IL-8 and monocyte chemoattractant proteins. Xanthine oxidase (XOD), a main endogenous source of reactive oxygen species was shown in a rat model to induce apoptosis of the endothelium, increasing vascular permeability and pulmonary edema. Additionally, the GTP-binding protein Rho and its target Rho-Kinase (ROCK) identified by Sawafuji et al. has been implicated in permeability changes causing RPE as well.

In terms of prevention of development of RPE the consensus statement of the American College of Chest Physicians states that a chest catheter or tube be used to reexpand the lung, the catheter or tube should be attached to a Heimlich valve or water seal without suction; however, suction should be used if the lung does not reexpand adequately with water seal drainage and it may be applied immediately after chest tube insertion in a clinically unstable patient or even in a stable patient. The guidelines of the British Thoracic Society (BTS) state that suction should not be applied initially to a chest tube that has been placed to treat a pneumothorax, but it can be applied after 48 hours if there is still an air leak or pneumothorax present. If suction is needed, 10 to 20 CmH2O has been recommended.

Treatment for RPE remains supportive. The cornerstone is positive-pressure mechanical ventilation and utilization of positive end-expiratory pressure (PEEP) to help reexpand collapsed alveoli, increase functional residual capacity, and reduce shunting. Treatment also may include diuresis and vasopressor support. The use of the prostaglandin analog misoprostil, ibuprofen, and indocin has been reported but its benefit is not clear. Other possible therapeutic options include placing the patient in lateral decubitus position with the involved side nondependent to help reduce perfusion and edema and intrapulmonary shunting. In the lateral decubitus position, there would be greater perfusion to the dependent lung because of the effects of gravity. The role of pharmacuetics in treating RPE remains uncertain; only anecdotal evidence exists supporting the use of any drug or combination of drugs. Four of 11 published
cases reviewed did not indicate the use of any drugs in the acute management of RPE.\textsuperscript{19,20,21,22} In RPE case reports where pharmaceutical interventions were reported, diuretics such as furosemide were the most commonly used agents.\textsuperscript{23} Colloids, crystalloids, or albumin were used in several cases to influence osmotic pressure.\textsuperscript{24,25,26,27} Epinephrine and dopamine have been used for vasopressor support.\textsuperscript{28} Two review articles cited the potential for misoprostol, a prostaglandin analogue, and indomethacin and ibuprofen, non-steroidal anti-inflammatory drugs (NSAIDS), to be used for their “cytoprotective and anti-inflammatory effects.”\textsuperscript{29,30} Methyprednisolone was used in two cases, including once to no therapeutic effect in combination with furosemide, glycopyrrolate, diphenhydramine, epinephrine, and inhaled albuterol.\textsuperscript{31,32} Nevertheless, a proper drug regimen in the management of REPE is not readily apparent. The decision to use or not use pharmaceuticals in treating RPE should be based on the prevailing needs of the individual patient.

References


