Medical Image of the Month: Reexpansion Pulmonary Edema

Figure 1. Large right hydrothorax with mild mediastinal shift to the left.

Figure 2. Status post right pleural pigtail drain placement with interval improvement of the now small right pleural effusion with re-expansion of the right lung and early edema.

Figure 3. Moderate right pleural effusion and worsening reexpansion pulmonary edema.
A 54-year-old woman with decompensated alcoholic liver cirrhosis presented to the emergency department with exertional dyspnea. She was afebrile, tachycardic (110), with oxygen saturation of 74% on 5 liters/minute (L/min), in moderate respiratory distress and was subsequently placed on a non-rebreather. On examination, she had absent breath sounds throughout her right lung with chest radiograph revealing large right-sided pleural effusion (Figure 1). A pigtail catheter was placed, draining approximately 4 liters of fluid (Figure 2), resulting in improved oxygenation to 93% on 3 L/min. On admission to internal medicine, the chest tube was clamped immediately. In the next 24 hours, patient developed increased oxygen requirements, with worsening tachypnea and tachycardia, requiring bilevel positive airway pressure and admission to the medical intensive care unit for reexpansion pulmonary edema (Figure 3).

Hepatic hydrothorax is a complication of cirrhosis and portal hypertension, defined as pleural effusion without any underlying pulmonary or cardiac etiologies. Though the pathophysiology is not completely understood, it is widely believed that the pleural effusion is caused by negative intrathoracic pressures allowing peritoneal fluid to enter the pleural cavity through diaphragmatic defects. Management of hepatic hydrothorax includes sodium restriction, diuresis, therapeutic thoracentesis, and transjugular intrahepatic portosystemic shunt. Repeated thoracentesis is the routine procedure to remove pleural fluid in refractory hepatic hydrothorax (1).

Though relatively safe, thoracentesis is associated with reexpansion pulmonary edema (RPE). RPE is believed to occur due to increased permeability of the pulmonary capillaries as a result of inflammation caused by ventilation and reperfusion of previously collapsed lung. Symptoms of RPE include chest discomfort and cough with onset typically within 24 hours of lung reexpansion. Signs of RPE include tachypnea, tachycardia, lung crackles, and hypoxemia refractory to oxygen therapy. Risk factors are young age (20-40 years), long duration of lung collapse, use of negative pressure during thoracentesis, large volume drainage, and rapid lung reexpansion. Management is largely supportive and ranges from diuresis to endotracheal intubation with mechanical ventilation (2).

Unfortunately, the amount of fluid that can be safely removed from the pleural effusion in order to prevent RPE has not been clearly defined. Feller-Kopman (3) reported that only one patient (0.5%) of 185 participants experienced clinical RPE, while four patients (2.2%) had radiographic RPE without symptoms. Our case demonstrates that removal of large volume from the pleural effusion via the chest tube resulted in clinical and radiographic RPE, thus, necessitating the need for clearly defined guidelines.

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References