A 16-Year-Old With Left-sided Pneumothorax*

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A 16-year-old boy with features of Marfan’s syndrome, previously in good health, had left sided chest pain and dyspnea of 6 days’ duration, which had worsened considerably on the day of admission. A posteroanterior chest radiograph showed complete pneumothorax on the left side and a partial pneumothorax on the right side. A chest tube was inserted on the left side and connected to an underwater seal drain. The patient became more dyspneic immediately. A repeat chest radiograph showed air space shadowing over the entire left lung field and an increase in the size of the pneumothorax on the right side (Fig 1). Subsequently, another chest drain was inserted on the right side and connected to an underwater sealed drainage. No negative pressure was applied to either of the drains. Over the next 30 min, the patient’s BP dropped to 70/50 mm Hg, and his oxygen saturation was recorded at 70% by pulse oximetry. Arterial blood gas analysis showed a PaO₂ of 7.7 kPa and saturation of 90% while breathing 35% oxygen via face mask. Further chest radiographs revealed increasing air space shadowing over the rest of the left lung field with complete expansion of both lungs. Arterial BP initially responded to transfusion of IV fluids, but the patient remained hypotensive despite the administration of 1.5 L of IV gelatin solution.

Video-assisted thoracoscopy was performed to exclude intrathoracic bleeding. On intubation of the trachea, copious amounts of frothy secretions were noted through the endotracheal tube; the patient became hypotensive and tachycardic. He required inotropic support to maintain hemodynamic stability. During operation, no bleeding was found in the chest cavity.

Postoperatively, the patient was admitted to the ICU. A pulmonary artery flotation catheter was inserted; the pulmonary artery wedge pressure was 10 mm Hg, the cardiac index was 3.26 L/min/m², and the systemic vascular resistance index was 1,427 dynes·cm⁻⁵/m². The patient was treated with positive end-expiratory pressure (PEEP), IV fluids, and inotropic support (epinephrine 0.2 µg/kg/min). The patient was weaned off inotropic support within 8 hs, and gas exchange improved over the following 12 h. The patient required ventilatory assistance for 36 hs. He was discharged home on the seventh day with complete resolution of his pulmonary edema.

What is the diagnosis?

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Diagnosis: Reexpansion pulmonary edema

Reexpansion pulmonary edema (RPE) is a recognized complication of rapid evacuation of a large pneumothorax or a large pleural effusion.1–3 Pulmonary edema occurs in the collapsed lung after reexpansion. It is most likely to occur when the lung has been collapsed for more than 3 days,4–6 but it can develop in any collapsed lung regardless of the duration of collapse. It usually appears unexpectedly and dramatically—immediately or within 1 h in 64% of patients and within 24 h in the remainder.7 The edema may progress for 24 to 48 h and persist for 4 to 5 days. The clinical features of RPE vary from asymptomatic radiographic findings alone to severe cardiopulmonary manifestations.8 In one of the reviews, the outcome was fatal in 20% of the cases.7 Various mechanisms have been proposed for the development of RPE. These include the following: increased microvascular permeability,9–11 increased hydrostatic pressure from vascular flooding of the reexpanded lung as result of negative intrapleural pressure; and altered capillary permeability from hypoxic injury of the collapsed lung.12,13 On reexpansion and reintroduction of oxygen to the relatively hypoxic lung, oxygen-derived free radicals are generated and are thought to damage the alveolar epithelial and endothelial cells and may cause increased vascular permeability. Activated neutrophils may also provide a major source of free radicals after reexpansion.12 Woodring13 noted that RPE developed in the portion of the lung that has been collapsed and did not affect the portion of lung that is aerated, which again suggests that hypoxic injury to the atelectatic lung is a possible cause for RPE. Hence, both the risk of developing RPE and the severity of RPE increase as the duration of collapse and hypoxia increases.

Our patient had a complete left-sided pneumothorax possibly of 6 days’ duration. The insertion of a chest tube resulted in sudden reexpansion of the left lung which precipitated pulmonary edema on that side. There were no radiologic or clinical signs of pulmonary edema on the right side. Hence, the mechanism of pulmonary edema could cause hypoxic injury to the left lung.

References