Case report - Thoracic general

Re-expansion pulmonary edema following video-assisted thoracic surgery for recurrent malignant pleural effusion

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Abstract

A rare case of a unilateral re-expansion pulmonary edema following video-assisted thoracic surgery for malignant pleural effusion is described.

Keywords: Pulmonary edema; Unilateral; VATS

1. Introduction

Re-expansion pulmonary edema (RPE) is a rare and potentially lethal complication of thoracostomy tube placement for pneumothorax, pleural effusion and severe atelectasis. Its onset is sudden and dramatic. RPE is very rarely described in association with anesthesia and video-assisted thoracic surgery (VATS) and only four cases are described in the medical literature. A case of a unilateral RPE following VATS for recurrent malignant pleural effusion is reported.

2. Case presentation

A 68-year-old man with a history of gastric adenocarcinoma was admitted with severe dyspnea and a right chest pain for three weeks. Clinical examination and chest roentgenogram confirmed a large right pleural effusion with nearly complete collapse of the right lung. The patient, under general anesthesia with double lumen tube, underwent a VATS drainage. Approximately 2.6 l of serosanguinous fluid was evacuated gradually. Pleural and lung biopsies (frozen section) confirmed the presence of metastatic disease. One chest tube was placed in the right pleural cavity and the right lung was fully expanded. The chest tube was connected to −20 cm H₂O suction. The patient extubated rapidly in the operating room. Pulse oximetry was measured at 100% on a 2 l nasal cannula approximately 1 h after the procedure.

Three hours later the patient’s condition deteriorated. He developed severe respiratory distress, cyanosis, coughing with a large amount of frothy secretions, tachypnea and tachycardia. With oxygen requirements increasing, a respiratory rate of 40 and an SpO₂ of 80–85% on an FiO₂ of 0.7–0.8 via a non-rebreathing face mask, the patient was transferred to the intensive care unit. Chest auscultation revealed diffused rhonchi over the whole right hemithorax while the left lung fields were clear. Chest X-ray indicated unilateral pulmonary edema (Fig. 1). Negative pressure applied to the pleural space was stopped immediately. Tracheal intubation followed by mechanical ventilation with an FiO₂ of 1.0 and PEEP was required. Fiberoptic bronchoscopy revealed frothy secretions originating from the right lung. Furosemide and dopamine were administered intravenously. The patient diuresed more than 2 l in the first two hours with continued decrease in oxygen requirements. A follow-up chest roentgenogram revealed clearing of the edema (Fig. 2). Despite this initial improvement the patient’s course was further complicated by sepsis, presumably from a pulmonary source. A tracheostomy was performed on the 7th postoperative day to provide access for frequent pulmonary toilet and bronchoscopy. The patient died two weeks later due to septic shock and multiple organ failure.

3. Discussion

Re-expansion pulmonary edema (RPE) is a complication which develops in a lung that has been rapidly reinflated after varied periods of collapse secondary to a pneumothorax or a pleural effusion of large volume. Its incidence is relatively low but mortality is up to 21%. RPE is usually seen within 2 h, occasionally delayed by 24–48 h, of re-expansion of a lung collapsed for at least 72 h. The RPE can be lobar or multilobar, probably related to prior degree of atelectasis. RPE is usually short-lived but rarely – as in our case – can become lethal.

The clinical signs of RPE include the presence of rales in the affected lung field, frothy sputum production from the...
RPE is unknown, although several potential mechanisms have been suggested. Enhancement of venous return and increase in pulmonary hydrostatic pressure, hypoxia-induced cardiac dysfunction and pressure-induced mechanical alveolar-capillary disruption promoting capillary permeability and favoring transudation of fluid in the lung have been proposed [7]. There is no specific literature to correlate open or endoscopic thoracic surgery with RPE. However, one lung ventilation may change the partitioning of blood flow between the non-dependent and dependent lungs as well as thoracic epidural anesthesia could be another factor that may affect the sympathetic control of pulmonary circulation [5].

Different strategies for prevention of RPE have been proposed. Although RPE has been reported without negative pressure suction, reduced levels (< −10 cm H2O) and slow re-expansion have been recommended [8]. Some authors suggest that it is not as much the negative pressure as the rate of re-expansion that is important. No human study has been performed prospectively to determine whether the incidence of RPE would be less if the chest tube is put to water seal only. There is only one experimental animal study. The authors studied RPE in monkeys and found that after a large pneumothorax of three days duration, six out of six monkeys developed RPE if their lungs were re-expanded with −10 cm H2O vs. none of the six animals who had their lungs re-expanded on water seal only [9].

Video-assisted thoracoscopic surgery is being used more often for both diagnostic and therapeutic interventions with a low rate of complications. There are four post-VATS REP cases in the medical literature [5, 10]. Our case was lethal. Early recognition and supportive measures are important for bringing these cases to a successful conclusion.

In summary, our case demonstrates a rare association of unilateral RPE with VATS for malignant pleural effusion. Negative pressure pleural suction may be one of the predisposing factors. Slow re-expansion by low or without negative pressure is recommended.

Other factors that have to be investigated are the degree of preoperative infection, inspissated secretions, lymphangitis carcinomatosa and systemic inflammatory response syndrome.

References