Ipsilateral Pulmonary Reexpansion Edema During Video-Assisted Thoracoscopic Surgery

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INTRODUCTION

Reinflation of a collapsed lung may lead to REPE. REPE is potentially fatal iatrogenic complication with mortality rate of %20 that is far from benign. It is very rare complication of VATS. Preventive measures and knowing risk factors is important to cope this stressful condition.

CASE REPORT

A 54 year old man after traffic accident a month ago was admitted thoracic surgery department with progressive dyspnea. He had no chest pain, cough or fever. He had history of diabetus mellitus.

Vital signs were stable. He looked confortable and wasn’t in respiratory distress. On physical examination breath sounds couldn’t be heard on right hemithorax. The left lung fields were clear. Other findings were normal on examination.

Blood analysis, biochemical values were normal. A chest radiograph revealed complete right sided opacity, left mediastinal and trakeal deviation. Broncoscopy was done, compressed right main bronchus with moderate amount of bronchial secretion was seen and aspired.

Right thoracentesis drained defibrininated hemorrhagic fluid. He was scheduled for VATS.

VATS was performed under one lung ventilation with left lateral position. 4000 cc defibrininated hemorrhagic fluid was drained. Under thoracoscopic view right lung expanded easily, it’s pinky and occupied entire right thorax. After operation the patient woke easily, was extubated and taken into the intensive care unit.

Just after the operation the patient was confortable, breath sounds were normal on both hemithoraces. Blood gas analysis revealed mild hypoxia with PH: 7.34, PCO2: 42.6 mm Hg, PO2: 60.4 mm Hg and HCO3: 22.7 mmol/L. The patient received supplemental oxygen via a nasal canule to compensate for hypoxemia.

On the second postoperatif hour the patient gradually developed respiratory distress and his condition deteriorated. He became tachycardic and tachypneic. On auscultation breath sounds were decreased on right hemithorax. A chest radiograph (fig: 1) showed diffuse opacity over entire right lung. Blood gas analysis at that time revealed respiratuar acidosis with PH: 7.16, PCO2: 72.8 mm Hg, PO2: 144 mm Hg and HCO3 25.3mmol/L. Except moderate leucocytosis (15200) blood count and biochemical parameters were in normal range.

The patient was intubated secondary to respiratory insufficiency and mechanically ventilated. Steroids and diuretics are prescribed. By the next ten hours blood gas values improved markedly to PH 7.44, PCO2 32.7 mm Hg, PO2 90 mm Hg and HCO3 21.8mmol/L.
On the first postoperatif day breath sounds were heard on right hemithorax. Blood gas analysis revealed mild hiperoxi with PH 7.43, PCO2 34.5 mm Hg, PO2 151 mm Hg, HCO3 22.5mmol/L. Chest radiograph obtained that time demonstrates rapid resolution of the infiltrates (fig:2).

Leucocytosis gradually decreased. The patient was extubated on the following day and recovered uneventfully

**DISCUSSION**

VATS is safe and effective procedure. VATS with low morbidity and mortality rates, patient comfort and with decreased hospital stay is superior to thorocotomy. Prolonged air leak, hemorrhage and infection are some of known complications of VATS but REPE is very rare complication in VATS experience, until now 5 cases present in English literature (1-2).

REPE can be fatal iatrogenic complication that occurs after rapid reexpansion of a collapsed lung. (3). The precise incidence isn’t known but ranged from 0.9 % to 14 % after tube thorocostomy(4). VATS needs general anesthesia with one lung ventilation (OLV). OLV can have adverse effects on pulmonary circulation because of hypoxic vasoconstriction of nonventilated lung and partitioning of blood flow between dependent and nondependent lungs (5) Several cases of pulmonary edema associated with OLV have been reported (2).

We know something about the aetiology, pathophysiology and preventive measures of REPE after thoracocentesis or tube thorocostomy but there is limited information on short term lung collaps and reinflation as seen in OLV (2).

Although the exact pathophysiology of REPE is unknown. Increased permeability of pulmonary capillaries, prolonged hypoxia, restoration of pulmonary circulation and increased negative intrapleural pressure may be important in developing REPE (4). Elevated levels of pro-inflammatory cytokines are detected in edema fluid and blood suggests inflammatory and immunologic nature of REPE (4-6). Presence of proteins, red blood cells, neutrophils in bronchial aspires supports certain degree of diffuse alveolar damage explains inefficency of oxigenation despite high FiO2 (7).

REPE is usually seen in ipsilateral entire lung but contrlateral or bilateral cases also present (6). Single lobe or segment rarely may be involved. Correlation between radiological and clinical findings isn't rule. Radiographic findings without clinical correlation don’t require therapy. Radiologic apperance of REPE is usually indistinguishable from other forms of pulmonary edema (7)

The clinical course is often rapid and variable(4). Situation is usually self limited (6). Cough, dyspnea, tachypnea, and tachycardia are frequent symptoms, rarely large amounts of frothy pink sputum is seen (7). Persistant cough heralds the development of pulmonary edema. The progression of edema results in hypoxia. Oxygen therapy alone is usually insufficient to improve blood oxgenation because of fluid filled alveolar space. Symptoms usually increase in severity.
Early recognition of REPE is important because the disease proves fatal in up to 20% of cases (5). The situation is unpredictable but younger age, large pneumothorax (> % 30), prolonged collabs, excess negative intrapulmoner pressure application and drainage of more than 1500 cc fluid once are risk factors (6-8).

Therapy is supportive. Mechanical ventilation with positive end expiratory pressure and hemodynamic support may be appropriate (3-4-8).

Here, prolonged collabs (> a month), excess drainage (4000 cc) and OLV can be risk factors in developing REPE. Large volume of drainage isn't recommended for thoracocentesis. Similar recommendation may be relevant for VATS. OLV may have adverse effect on pulmonary circulation but, right hemithorax was full of fluid compressed entire lung with no ventilation. The ongoing hypoxic vasoconstriction wasn't result of OLV and could exclude the explanation of some of OLV’s adverse effect in this situation. Timing is typical here, REPE developed two hours after operation. And, also rapid clearing of the chest graphy wasn’t consistent with pneumonia, lung contusion or acute respiratory distress syndrom supports the diagnosis of REPE. Unfortunately, we didn’t have bronchial aspirates after REPE developed to measure the concentration of protein content, cellular elements etc, to eliminate other factors contribute in pulmoner edema formation.

References
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