

Ipsilateral Pulmonary Reexpansion Edema During Video-Assisted Thoracoscopic Surgery

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Citation

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Abstract

Reinflation of collapsed lung sometimes may lead to pulmonary edema (REPE). REPE is potentially fatal iatrogenic complication with mortality rate of %20 is far from benign. The exact pathophysiology of reexpansion pulmonary edema is unknown; mechanical and inflammatory processes are incriminated. Treatment is supportive. Young age, longer duration, large pneumothorax are encountered risk factors in developing REPE. We report a case of (REPE) after video thoracoscopic surgery (VATS) in 54 year old man suffered progressive dyspnea. REPE is very rare complication in VATS experience, until now five cases present in English literature

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 diabetes mellitus.

Vital signs were stabile. He looked comfortable 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 tracheal deviation. Bronchoscopy was done, compressed right main bronchus with moderate amount of bronchial secretion was seen and aspirated.

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

VATS was performed under one lung ventilation with left lateral position. 4000 cc defibrinated hemorrhagic fluid was drained. Under thoroscopic view right lung expanded

easily, it's pink 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 comfortable, 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 postoperative 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 respiratory acidosis with PH: 7.16, PCO₂: 72.8 mm Hg, PO₂: 144 mm Hg and HCO₃: 25.3 mmol/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 21.8 mmol/L.

On the first postoperative 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

Figure 1

Figure1: Development of pulmonary reexpansion edema two hours after operation



Figure 2

Figure 2: clearing of reexpansion pulmonary edema in the right lung



DISCUSSION

VATS is safe and effective procedure. VATS with low morbidity and mortality rates, patient comfort and with decreased hospital stay is superior to thoracotomy. 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 thoracostomy (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 thoracostomy but there is limited information on short term lung collapse 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 aspirates supports certain degree of diffuse alveolar damage explains inefficiency of oxygenation despite high FiO₂ (7).

REPE is usually seen in ipsilateral entire lung but contralateral 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 appearance 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). Persistent cough heralds the development of pulmonary edema. The progression of edema results in hypoxia. Oxygen therapy alone is usually insufficient to improve blood oxygenation because of fluid filled alveolar space. Symptoms usually increase in severity for 24- 48 hours then slowly resolves next 5-7 days (7).

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 collapse, excess negative intrapulmonary 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 collapse (> a month), excess drainage (4000 cc) and OLV can be risk factors in developing REPE. Large volume of drainage isn't recommended for thoracentesis. 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 syndrome 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 pulmonary edema formation.

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