

Re-expansion Pulmonary Oedema Following One-lung Ventilation

-A Case Report

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Abstract

Acute ipsilateral or bilateral pulmonary oedema following lung re-expansion after pleurocentesis or treatment of pneumothorax is an unusual clinical phenomenon that may have serious consequences. It usually occurs when the chronically collapsed lung is rapidly re-expanded. There are, however, no reported cases of this complication following one-lung ventilation used to facilitate surgery. A report of the occurrence of unilateral re-expansion oedema in a young female undergoing scoliosis correction is described.

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Case Report

A 15-year-old female patient weighing 40 kg with asymptomatic but severe scoliosis was admitted for correction of a skeletal deformity via a thoracoscopic anterior release approach and posterior instrumentation. Preoperative assessment revealed good effort tolerance. Chest radiographs showed a marked scoliotic spine with convexity to the right. Lung function test was characteristic of the restrictive pattern (FVC 65%, FEV₁ 60%). Baseline blood gases were normal (PaO₂ 95 mmHg, PaCO₂ 34 mmHg at FiO₂ 0.21).

Intraoperative monitoring consisted of electrocardiography (ECG), pulse oximetry, nasopharyngeal temperature, radial artery and central venous catheterization. She was anaesthetised with propofol 2 mg.kg⁻¹, fentanyl 2 µg.kg⁻¹ and vecuronium 0.1 mg.kg⁻¹. A left-sided #28 French gauge Brochocath® double-lumen tube (DLT) was inserted and position confirmed via a bronchoscope. Anaesthesia was maintained with isoflurane 1% to 1.5% in an oxygen-air mixture and muscle paralysis continued via a vecuronium infusion. One-lung ventilation (OLV) with collapse of the right lung was achieved without significant hypoxaemia (SaO₂ 99% and PaO₂ 153 mmHg at FiO₂ of 0.5). The first stage of the surgery consisted of an anterior release procedure done thoracoscopically. After 75 minutes of OLV, she remained haemodynamically stable with no episodes of desaturation. There was an increased in the end-tidal CO₂ from 38 to 48 mmHg. The lungs were re-expanded with positive ventilation for 5 minutes with peak airway

pressures at 20 to 30 mmHg and FiO₂ of 1.0. OLV was then reinstituted at a FiO₂ of 0.5 and the procedure continued. However, desaturation was noted 10 minutes later with the pulse oximetry reading at 92%. The lungs were again re-expanded with manual bagging until SaO₂ was 100% and the position of the DLT checked, after which the procedure was continued. Fifteen minutes later, however, desaturation to 87% occurred despite the use of a FiO₂ of 1.0 to the dependent lung. Fortunately, the operative procedure had finished and manual ventilation of both lungs corrected the hypoxaemia. It was then noted that pink frothy secretions (about 10 ml) were present in the bronchial (non-dependent) lumen of the DLT while the dependent side was clear. She was normotensive and the central venous pressure was 7 mmHg at this time. Chest X-ray revealed alveolar infiltrates in the right lung 20 minutes after the onset of the oedema (Fig. 1). The drainage of oedema fluid subsided 15 minutes later. The posterior instrumentation stage of the operation was postponed and the patient was extubated 8 hours later in the intensive care unit.

Discussion

In this patient, pulmonary oedema fluid was present only in the lung that had been collapsed and re-expanded. The use of a DLT clearly differentiated the involved side. While surgical trauma to the lungs may caused bleeding and manifest as such, there was minimal use of lung retractors in this case as exposure was excellent.

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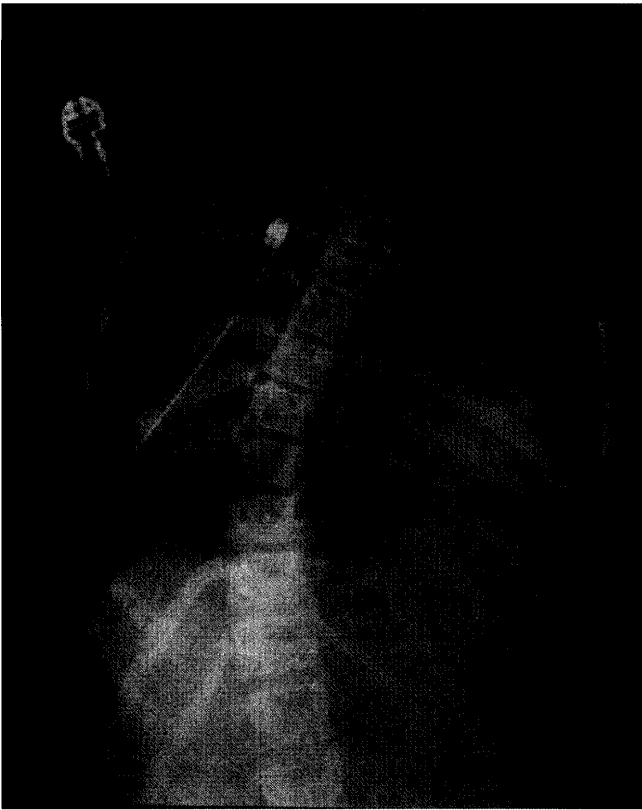


Fig. 1. Chest X-ray showing unilateral pulmonary oedema.

Chronicity of the collapsed lung is an important risk factor for the development of re-expansion pulmonary oedema (REPE).¹ It typically occurs after a large quantity of air or fluid is rapidly removed from the pleural space that has been present for more than three days. REPE occurring during general anaesthesia for elective surgery in lungs that were not chronically collapsed as in this patient is rare.

The precise pathogenesis of REPE remains unclear. The common end-point involved enhanced endothelial permeability secondary to alveolar-capillary membrane disruption and ischaemia-reperfusion mediated injury.* That unilateral involvement of the collapsed side is present while both lungs are subjected to the same positive pressure ventilation during re-expansion indicated that the reduction of blood flow in the atelectatic lung caused by hypoxic pulmonary vasoconstriction may have resulted in abnormal microvascular permeability.^{3,4} The development of REPE in this patient is probably related to the stretching and distension of the collapsed alveoli membranes and the increased pulmo-

nary blood during the period of re-expansion. Once this process is initiated and resulted in oxygen desaturation, more vigorous manual bagging must have exacerbated the destructive process.

Most REPE would resolve in 24 to 72 hours with supportive treatment. Mechanical ventilation with positive end-expiratory pressure at low peak airway pressures, diuresis, and positioning the patient with the affected side up may help reduce oedema. Anti-inflammatory drugs such as non-steroidal anti-inflammatory drugs have been advocated

Pulmonary oedema secondary to negative pressure developed as a result of occlusion of the endotracheal tube or laryngospasm during anaesthesia is a well-documented phenomenon.^{5,6} Re-expansion pulmonary oedema occurring in a patient undergoing general anaesthesia with one-lung ventilation is rare and there is no similar previous case report in the English literature. Early recognition, maintenance of a clear airway and adequate oxygenation are essential therapeutic modalities. The natural course of this patient seemed to follow other patients who developed negative pressure pulmonary oedema during anaesthesia and had resolution of the symptoms fairly rapidly. As such, further invasive haemodynamic monitoring or aggressive drug therapy with inotropic agents was felt not to be necessary. A heightened awareness among anaesthesiologists of the varied causes of intraoperative pulmonary oedema may help reduce the occurrence and facilitate early management of the potential complications.

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