

**RE-EXPANSION PULMONARY EDEMA:
A SEVERE COMPLICATION OF THORACIC SURGERY.**

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ABSTRACT

Re-expansion pulmonary edema is a relatively rare condition that develops during rapid expansion of a collapsed lung due to pleural effusion, pneumothorax or atelectasis. The exact pathophysiology of this complication is still unknown but involves histological abnormalities of the pulmonary microvessels as well as mechanical stress during re-expansion. The evolution under artificial ventilation with positive expiratory pressure has improved the prognosis but it remains potentially fatal with a mortality that can reach 21%. We herein describe the case of a 61 year-old man, a smoker, who presented a suspicious apical mass of the right upper lobe with tumoral invasion of the superior vena cava associated with a right hydropneumothorax who was scheduled for a thoracoscopic biopsy. The patient benefited from a surgical drainage of the hydropneumothorax and was complicated by a postoperative respiratory distress, which revealed a re-expansion pulmonary edema.

Patient management using noninvasive ventilation and diuretics was unsuccessful. The patient's condition worsened afterwards complicated thereafter with a state of shock and death by multiorgan failure.

Keywords: Re-expansion pulmonary edema; Thoracic surgery

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*****Published in Marsh 10, 2022.

doi: 10.46327/msrjg.1.000000000000212

doi url: <https://doi.org/10.46327/msrjg.1.000000000000212>

INTRODUCTION

Re-expansion pulmonary edema (RPE) is an under reported condition of non-cardiac pulmonary edema that can be lethal in 21 % of cases. [1] The lack of an effective treatment makes the prevention as well as the early recognition of this complication a matter of great importance. In this paper, we will report the case of a fatal re-expansion pulmonary edema complicating the drainage of a large hydropneumothorax.

CASE REPORT

The patient was 61 years old with a history of chronic smoking (30 pack-years) weaned 2 months before and hypertension treated for 7 years using calcium channel blockers. He presented first a superior vena cava syndrome. Radiological exploration revealed a highly suspicious mass in the apical segment of the right upper lobe associated

with an adenopathy in Baret's space, infiltrating the superior vena cava (thrombosis of the superior vena cava (SVC)) and a moderate bilateral pleural effusion. The patient was scheduled for biopsy of the mass and the pleura.

The pre-operative anesthesia visit found a polypneic patient with a respiratory rate of 30 breaths per minute, a saturation at 94% in room air, blood pressure at 140/70 mmhg and heart rate of 93 beats per minute. The lung examination found a reduced breath sounds on the right hemithorax. The cardiovascular exam revealed superior vena cava syndrome with facial edema and thoracic collateral venous circulation as well as right heart failure symptoms: hepatomegaly, jugular veins distension, lower limbs edema and a hydrocele. The chest X-ray showed a right hydropneumothorax (Fig. 1 & 2); the electrocardiogram showed a sinusal rhythm, a microvoltage and repolarization disorders.

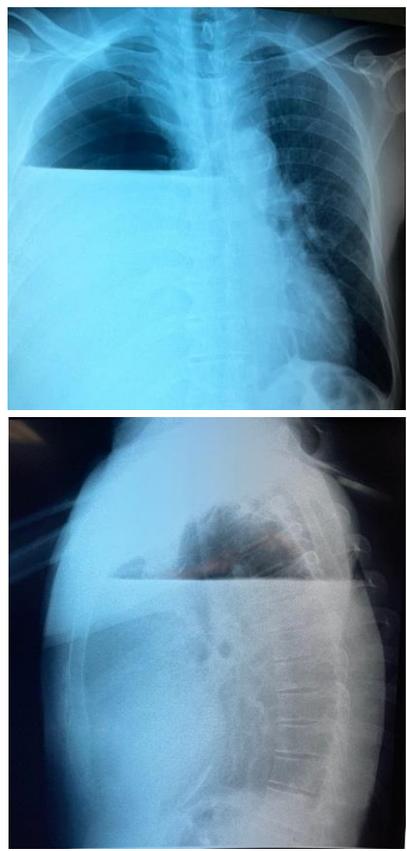


Fig. 1 & 2: Frontal and side chest X-ray showing a large right hydropneumothorax

The echocardiography found no hypertrophy nor dilatation of the left ventricle with a preserved systolic function, a moderate circumferential pericardial effusion in front of the right cavities as well as a slight collapse of the right atrium. Blood investigations were normal except for a CRP of 12.6 mg/L. It was decided to perform a preoperative thoracic drainage in the operating room, which allowed a rapid aspiration of 2500 ml of fluid before clamping. Anesthetic induction was performed using fentanyl, propofol and rocuronium after pre oxygenation at normal tidal volume for 3 min with a FIO₂ of 1. A left selective bronchial intubation was performed using a Carlens tube n° 37. Intra-operatively, a left single lung ventilation was performed; thoracoscopy allowed the aspiration of an additional 1500 ml of pleural fluid; Exploration found a pleura free of macroscopic nodules and an apical mass of the right upper lobe; the biopsy of the pleura and the tumor mass was performed. A chest tube was placed and attached to 20 cm H₂O underwater-seal suction followed by a switch to a normal ventilation allowing pulmonary re-expansion. During the surgery, respiratory and hemodynamic status were stable with oxygen saturation of ranging between 96% and 100%, HR between 80 and 90 bpm and BP between 120-

130/60-70 mmhg. After the emergence from anesthesia, the patient was extubated and transferred to a postoperative intensive care unit. Upon admission to the ward, the respiratory assessment found: RR at 22 bpm, SpO₂ at 98% on 6l/min of oxygen, and well perceived vesicular murmurs bilaterally. One hour later, the patient developed respiratory distress: polypnea at 45 bpm, SpO₂ at 70 % under high concentration oxygen mask; Lung auscultation found diffuse crackles in the right hemithorax. A bedside chest X-ray showed an alveolar-interstitial syndrome in the right lung evoking a re-expansion pulmonary edema (**Fig. 3**).



Fig. 3: Frontal chest X-ray showing a unilateral pulmonary edema

The suction was immediately stopped; the patient was put on diuretics and non-invasive ventilation with the following parameters (FiO₂ at 100%, PEEP at 8 and Inspiratory pressure support at 15 cmH₂O). The worsening of the respiratory condition under NIV indicated his intubation; the patient developed circulatory failure and was put under vasoactive drugs. A chest X-ray showed a slight improvement of the edema (**figure 4**). The evolution was towards the patient's death 24 hours later, following a multi-organ failure.



Fig. 4: Frontal chest X-ray showing a unilateral pulmonary edema (4 hours later)

DISCUSSION

Re-expansion pulmonary edema is well known as a major complication of thoracentesis for over 150 years. It was first described by Pinault in 1853 as a complication of thoracentesis [2], but it was not until 1958 that Carlson et al. reported it after the treatment of a pneumothorax [3]. The pathogenesis of RPE is still unclear but it is certainly multifactorial involving the alteration of capillary permeability, the release of inflammatory mediators, surfactant reduction and increased hydrostatic pressure [4]. The reported incidence of RPE is between 0% and 1 % but it is surely underestimated. Some series that opted for the use of systematic chest CT scan after pulmonary expansion found much higher incidence (ranging from 32.5% to 74 %) [5-6]. This sizeable difference could be explained by the higher rate of asymptomatic RPE. The risk factors for RPE remain unclear. Several risk factors have been proposed, including the duration of symptoms, a larger size of the pneumothorax, younger age and a rapid rate of re-expansion. The presence of pleural effusion coincident with pneumothorax was also associated with the development of RPE [5-6]. RPE symptoms usually appear during the first two hours after pulmonary re-expansion, and may last for 24 to 48 hours [7]. The severity of the symptoms depends on the extension of the edema. Radiographical changes such as ipsilateral infiltrates should aid the diagnosis. Although most of the patients recover within 5–7 days, death rates may reach up to 21% in patients who develop this complication. [1]. Treatment consists generally of support measures based on oxygen supplementation as well as ventilatory support, either by invasive or non-invasive ways, associated to hemodynamic support by volume expansion, inotropes and even diuretics. New therapeutic options are described: Rapid pleural space re-expansion, which consists of the reintroduction of the drained pleural fluid into the patient's pleural space, is an interesting option for mild cases. For severe cases, differential lung ventilation can be a compelling alternative to conventional ventilation. [8-9]

Lacking a therapeutic consensus, management is essentially based on prevention, which includes careful pleural drainage procedures and avoiding negative pleural aspiration for large volume pleural effusion.

CONCLUSION:

Re-expansion pulmonary edema is a largely unknown complication that develops in a lung rapidly reinflated after varied periods of collapse, which can make it an iatrogenic complication.

While the treatment of RPE is mainly supportive (and is in most instances effective), considering its high mortality, as highlighted by the index case, the primary goal should be the prevention of this complication in clinical practice.

DECLARATION OF INTEREST:

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

FUNDING SOURCES: None

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