A Case of Re-Expansion Pulmonary Oedema – is Thoracotomy a Risk Factor?

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Abstract

Re-expansion pulmonary oedema (RPO) is an uncommon clinical phenomenon encountered during the correction of chronic lung collapse. It has a mortality rate of 20%. Identification of the risk factors for RPO development may help in preventing the process. We presented an acute case of RPO complication which occurred within 3 hours following immediate water seal drainage of a right sided pneumothorax developed after the thoracotomy, and discussed the possible risk factors, mechanisms and preventive treatment options.

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Abbreviation: *RPO: Re-expansion pulmonary oedema*

Introduction

Re-expansion pulmonary oedema (RPO) is an uncommon complication of the treatment of chronic lung collapse (1). It has usually been reported after the pleurocenthesis and chest tube drainage of pneumothorax. The clinical picture varies from very mild to fatal, and may necessitate immediate intervention. Despite every precaution, occurrence of RPO could not be prevented in the majority of the cases. If the risk factors for RPO could be identified, its prevention would be possible. But the previous investigations could not specify any risk factor.

One fact, that has not been noticed until now, is that around 20% of RPO cases developed either during the management of the lung collapse which had occurred following thoracic surgery or following the surgical correction of the chronic lung collapse (2-4). The rate of RPO complication encountered after thoracic surgeries is too high, and suggestive to be regarded as a risk factor in the etiopathogenesis.

Here, we presented an acute RPO case after water seal drainage of a right sided pneumothorax, developed in a patient who had undergone left thoracotomy for carcinoma of the oesophagus, and discussed the possible risk factors,

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mechanisms and prophylactic treatment modalities with the aid of the prior literature.

Case Report

A 35-year-old man diagnosed as having a carcinoma of the oesophagus was operated on by thoracotomy. Any complication during or after the operation did not occur and the patient was weaned off from the mechanical ventilation on the second postoperative day. The daily chest roentgenograms were normal except the surgical sequel. His spontaneous breathing was not problematic and the vital signs were completely normal. Total blood count and arterial blood gas analysis revealed mild anemia and leucocytosis (Hgb: 10.8 g/dl, WBC: 14800 cells/mm^3), and mild hypoxemia ($PaCO_2$: 38 mm Hg, PaO_2: 77 mm Hg and pH: 7.41).

On the 3rd postoperative day, the patient complained of sudden chest pain on the right side and difficulty in breathing. He became tachypneic (31/min) and tachycardic (136 beats/min). His chest roentgenogram revealed total collapse of the right lung (Figure 1). Arterial blood gas analysis resulted as $PaCO_2$: 33.3 mm Hg, PaO_2: 63.7 mm Hg and pH: 7.47. He was immediately placed on 100% O_2 with a face mask and connected to water seal drainage avoiding the application of negative intra-pleural pressure. The general condition of the patient got better with the relief of dyspnea in 2 hours and the subsequent chest roentgenogram showed the total re-expansion of the right lung. Approximately 3 hours following the first complaint, the patient suddenly developed cyanosis, severe dyspnea, cough, hypotension (80/50 mm Hg) and tachycardia (152 beats/min). Blood gas analysis demonstrated hypercapnia, severe hypoxemia and acidosis ($PaCO_2$: 62.8 mm Hg, PaO_2: 49.5 mm Hg and pH: 7.20). Simultaneous chest roentgenogram revealed a complete heterogenous opacification on the right lung (Figure 2). The patient was expectorating copious amounts of clear to pink frothy sputum. The patient was diagnosed as having an re-expansion pulmonary oedema and connected to the mechanical ventilation with PEEP (5 cm H_2O). Despite the support given with volume expanders, dobutamine, tracheal aspiration and high dose corticosteroid infusion (250 mg), the negative clinical condition with cardiopulmonary collapse continued deteriorating and the patient died 6 hours after the onset of RPO.

Discussion

Since it is relatively rare, the knowledge about the etiopathogenesis of RPO is limited. The implicated mechanisms are fast re-inflation, application of excess negative intra-pleural pressure, chronicity of the lung collapse, loss of surfactant and progressive inflammatory process triggered by the exudate extravasated from pulmonary vasculature (5). The prevalence of RPO is thought to be less than 1% but it is generally believed that many cases set sub-clinically and resolve spontaneously (5).

Whatever the reason is, prolonged lung collapse leads the loss of surfactant, increased alveolo-capillary membrane

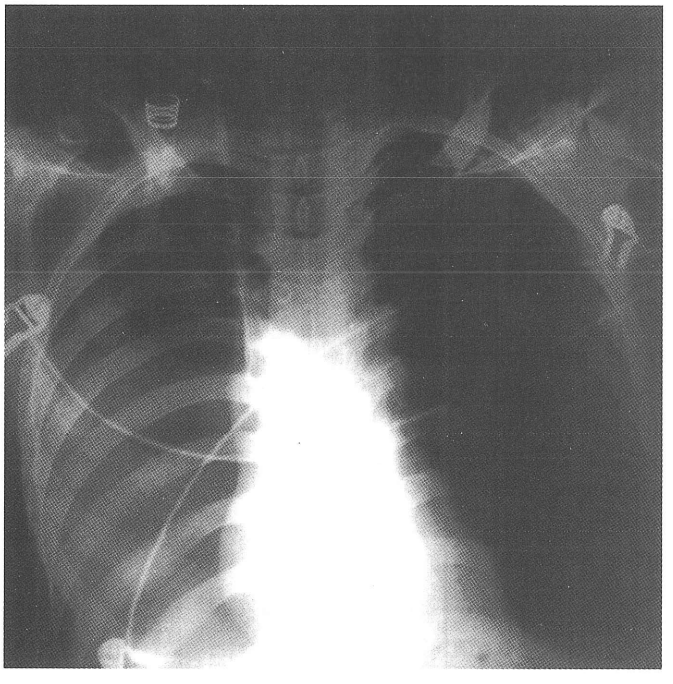

Fig. 1. Chest roentgenogram of the patient demonstrating the total collapse of the right lung.

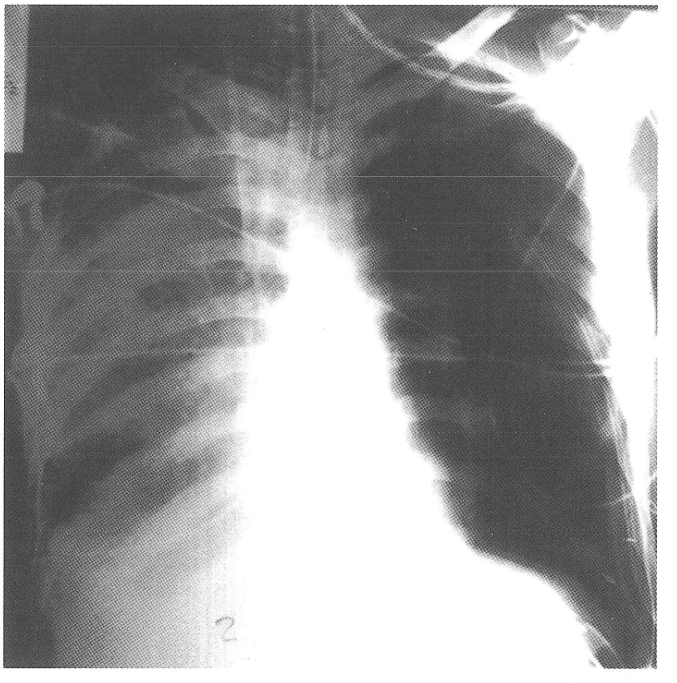

Fig. 2. Chest roentgenogram of the patient demonstrating the complete heterogenous opasification of the right lung.

pressure and anoxic damage to the capillaries. All of these factors accomplish the necessary environment for RPO, and any inappropriate attempt of re-inflation triggers the start of the cascade (exudation from already damaged or stressed capillaries, then inflammatory process and progressive pulmonary oedema). In the inflammatory stage, various mediators within the exudate attracts and activates PMNL and leads the generation of further inflammatory substances, like O_2 radicals (6,7).

Since the mortality rate is around 20%, prevention of RPO complication gains the highest priority. But by now, identification of the patients at risk could not be possible. In the pathogenesis of RPO, the stress on lungs created by the thoracic surgery performed for re-inflation may have an additive effect on the already present stress by the chronicity of collapse. Also other conditions like recent thoracic surgery as in our case, and clinical or sub-clinical pulmonary inflammation may increase the stress on lungs, thus increasing the risk for RPO, if lung collapse has occurred. Twenty percent of previously reported RPO cases were carrying at least one of these risk factors. Theoratically all these stress factors may cause some interstitial oedema by exudation from capillaries which is the initial phase of the RPO cascade. Recent laboratory studies tested the ability of some anti-inflammatory agents, like superoxide dismutase and allopurinol, to reverse RPO (7,8). However, only corticosteroid administration was mentioned anectodally in a limited number of clinical cases (9). All of these trials included the already established RPO cases and found little benefit of anti-inflammatory substances, if any. Indeed it is not surprising because once the inflammatory process has been started and inflammatory substances are generated, anti-inflammatory treatment is not supposed to have any remarkable effect.

Our patient's preoperative assessment of cardiopulmonary status by physical examination, detailed history, P.A. chest roentgenogram, spirometry and 12 lead ECG was completely normal. Despite this fact, recent thoracotomy for carcinoma of the oesophagus might have established such a suitable background that the time interval elapsed from the first symptom of pneumothorax to the development of RPO was only 3 hours. To our knowledge, this is comparably the shortest duration reported by now. Table 1 describes thoracotomy-related RPO cases in the literature and the duration of their lung collapse. In all of the previous cases (2-4), except one (10), lung collapse had already been established before the thoracotomy. Since lung collapse developed in our patient after the thoracotomy, they differ from our case in this aspect. The situation in the other case (10) is also different in that the right lung was intentionally collapsed during the thoracotomy phase of the surgery for oesophageal cancer. The common point in the previous cases and our's is exposition of the lungs to extra stress and inflammation created by thoracotomy. Immediate application of water seal drainage without active evacuation and intensive care could not restrain the development of RPO in our patient. If such clinical predisposing factors are present, water seal drainage without the application of negative intra-pleural pressure may not be sufficient, and controlled re-expansion by intermittent clamping of the chest tube should be considered. Thus permitting even slower than usual re-expansion.

Table 1. Presentation of the thoracotomy-related re-expansion pulmonary oedema cases.

References	Underlying pathology leading to thoracotomy	Duration of lung collapse
Morikawa et al.[2]	Intrathoracic haematoma	2 weeks
Waller et al.[3]	Pleural tumour	Chronic event
Angel et al.[4]	Lung tumour	Chronic event
Khoo et al.[10]	Oesophageal cancer	4 hours
Günen et al. (present report)	Oesophageal cancer	3 hours

We think that the surgical correction of chronic lung collapse and lung collapse which happens after thoracic surgery or on a clinical or sub-clinical pulmonary inflammatory basis should be regarded as hazardous for RPO development. In patients carrying one or more of these risk factors, intermittent re-inflation, avoidance of active evacuation and initiation of prophylactic anti-inflammatory medication before the surgery or tube thoracostomy may be considered to prevent RPO complication.

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