

Re-expansion pulmonary edema in a boy with spontaneous pneumothorax during an influenza B virus infection*

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ABSTRACT

Re-expansion Pulmonary Edema (RPE) is an uncommon complication following rapid re-expansion of the lungs, and the most common disorder associated with RPE is spontaneous pneumothorax. The majority of patients with RPE associated with pneumothorax have been adults; only 4 cases have been reported in children. We present a patient who developed RPE after treatment of spontaneous pneumothorax that occurred during an influenza B virus infection. His condition improved only with oxygen supplementation and intravenous corticosteroids. Infection with influenza B virus leads to increased production of lung epithelial oxidants, which might have contributed to the development of RPE in our patient.

Keywords: Reexpansion Pulmonary Edema; Influenza Virus; Pulmonary Complication

1. INTRODUCTION

Re-expansion Pulmonary Edema (RPE) is an uncommon complication that occurs when a collapsed lung is rapidly re-expanded after evacuation of air or fluid from the pleural space [1]. Although it is possible for RPE to occur in every type of chronically collapsed lung that can be re-expanded, the most common disorder associated with RPE is spontaneous pneumothorax [2]. The majority of patients with RPE associated with pneumothorax have been adults [1]; only 4 cases have been reported in children [3-5]. We report a patient who developed RPE after treatment of spontaneous pneumothorax that occurred during an influenza B virus infection.

2. PATIENT REPORT

A previously healthy, 6-year-old boy was admitted to our hospital after 2 days of cough, dyspnea and high-grade

fever. On admission, the patient was febrile with a body temperature of 39.6°C, and his oxygen saturation was 94% while breathing ambient air.

Physical examination showed tachypnea and diminished breath sounds over the left hemi-thorax. A nasopharyngeal swab tested positive for influenza B on a rapid influenza antigen detection test. Routine laboratory tests were normal except for a slightly elevated C-reactive protein concentration. Chest radiography revealed a left-sided pneumothorax (**Figure 1(a)**). His symptoms improved immediately after placement of a chest tube with application of a negative pressure of 10 cm H₂O. However, after 30 minutes, the patient again exhibited tachypnea and his oxygen saturation decreased to 88%. Chest auscultation revealed left-sided inspiratory crackles. Repeat chest radiography showed a fully expanded left lung, but also left-sided pulmonary edema (**Figure 1(b)**).

The patient was diagnosed with RPE occurring after treatment of pneumothorax. He was administered oxygen supplementation (5 L/min via face mask) and intravenous corticosteroids. His condition gradually improved over the next 2 days, and after an additional 3 days, his pneumothorax and RPE completely resolved. The patient was discharged after the chest tube was removed, without further sequelae. The patient remained well over a 6 months follow-up period.

3. DISCUSSION

RPE has been reported after rapid evacuation of large pneumothorax or large pleural effusion [4]. The clinical features of RPE include the following: a collapsed lung over period of 3 days or more; an evacuation volume of 2000 ml or more; a period of less than 1 hour from re-expansion to the onset of RPE; and the pulmonary edema is classified as permeability pulmonary edema [2]. Risk factors for RPE include the degree of lung collapse and a rapid re-expansion [1]. The symptoms of RPE range from asymptomatic presentation to cardiopulmonary insufficiency [4] and the reported mortality rate has ranged from 0% to 20% [1].

*Conflict of interest: There is no conflict of interest with regard to this manuscript.

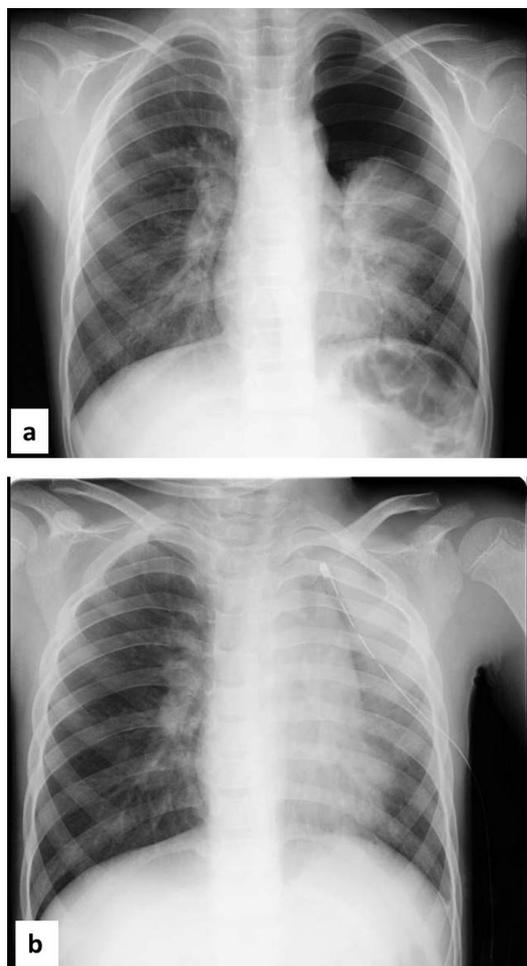


Figure 1. (a) Chest radiograph on admission showing spontaneous pneumothorax of the left lung; (b) Chest radiograph after insertion of the chest tube showing re-expansion pulmonary edema of the left lung.

Despite the fact that the rate of occurrence of RPE after drainage of a pneumothorax varies from 14.4% to 27%, there have only been 4 reports from the pediatric population [3-5]. Pediatric cases of RPE with diseases other than pneumothorax are also rare; RPE has been reported in children with pleural effusion due to nephrotic syndrome [6] or non-Hodgkin's lymphoma [7,8], with reinflation of a retracted lung following patent ductus arteriosus ligation [9] or following general anesthesia [10], and with pleural empyema [11].

Treatment for RPE remains supportive, using oxygenation, positive-pressure mechanical ventilation and utilization of positive end-respiratory pressure, diuresis and hemodynamic support. In addition, the use of prostaglandin analogs or corticosteroids has been also reported [1,2,12]. However, the best treatment is to avoid rapid lung re-expansion [2].

Although the precise pathogenic mechanism for the development of RPE remains unclear, the common end-

point in the pathological process is probably enhanced endothelial permeability caused by a combination of alveolar-capillary membrane disruption and ischemia-reperfusion-mediated injury [1,2]. Oxygen radicals and interleukin 8 (IL-8) have also been reported to be contributing factors in the development of RPE [1,2].

In our patient, RPE occurred following treatment of pneumothorax associated with influenza B virus infection. Infection with influenza virus leads to increased production of lung epithelial oxidants, which directly and/or via IL-8 expression results in injury to the lung epithelial cells [13]. Influenza virus induction of these mediators might have contributed to the development of RPE in our patient.

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