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Reexpansion pulmonary edema following reconstruction of a traumatic hernia of the diaphragm

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Introduction

Acute ipsilateral pulmonary edema following lung reexpansion after pleurocentesis or the treatment of pneumothorax is a well-described clinical phenomenon that may have serious consequences [1–6]. This complication is believed to occur only when a collapsed lung is reexpanded by means of the evacuation of large amounts of air or fluid. We report here the occurrence of ipsilateral acute pulmonary edema after inflation with positive pressure ventilation during the reconstruction of a traumatic hernia of the diaphragm.

Case report

A 57-year-old man with a history of ischemic heart disease, who had already undergone percutaneous transluminal coronary angioplasty (PCTA) three times, was admitted to the hospital as the result of a traffic accident. He was suffering from multiple fractures (left tibia, left femoral bone, right portion of the pelvis) and a traumatic hernia of the left part of the diaphragm. Reconstruction of the left femoral bone fracture and of the diaphragmatic hernia were scheduled 3 days after the injuries, but the operations were postponed for 2 weeks due to liver dysfunction induced by the administration of antibiotics. Laboratory tests before the operations disclosed the following findings: white blood cell count, 7000 mm^3, red blood cell count, $309 \times 10^4/mm^3$; hemoglobin, 9.9 $g \cdot dl^{-1}$; hematocrit, 30.6%; glutamic pyruvic transaminase, 31 $IU \cdot L^{-1}$; glutamic oxaloacetic transaminase, 53 $IU \cdot L^{-1}$; lactate dehydrogenase, 285 $IU \cdot L^{-1}$; cholinesterase 0.59 ΔpH; blood urea nitrogen, 20 mg·dl; creatinine, 0.7 mg·dl; total bilirubin, 4.8 mg·dl; and total protein, 6.4 g·dl. A chest roentgenogram revealed the herniation of the stomach which caused collapse of the left lung and left pleural effusion (Fig. 1).

Atropine sulfate (0.5 mg) and hydroxyzine chloride (50 mg) were administered intramuscularly 60 min before the induction of anesthesia. Anesthesia was induced with thiopental (200 mg, i.v.) and vecuronium (10 mg, i.v.) together with isoflurane and N_2O inhalation. The patient was monitored with blood pressure, ECG, and pulse oximeter during the operation. After the induction of anesthesia, arterial oxygen saturation (Spo_2) was maintained at around 98% under 50% of FIo_2. During the first 2 h after the induction of anesthesia, the reconstruction of the fracture of the left femoral bone was performed smoothly. After that, the abdomen was opened and the stomach was withdrawn to the abdominal cavity. Immediately after this process, Spo_2 fell to 85% and a large amount of yellowish fluid issued from the endotracheal tube. One hundred percent oxygen was administered and anesthesia was maintained by administration of pentazocine and diazepam during the rest of the operation. After completion of the operation, the patient was transferred to the intensive care unit of Mie University Hospital. On admission, an arterial catheter and a Swan-Ganz catheter were inserted. Arterial blood gas value on 100% oxygen was as follows: Po_2, 66 mmHg; Pco_2, 69.8 mmHg; pH, 7.192. The data obtained from the Swan-Ganz catheter were as follows: cardiac index, $2.15^{-1} \cdot m^{-2}$, pulmonary arterial pressure, 28/16 (22) mmHg; pulmonary capillary wedge pressure, 1 mmHg; pulmonary vascular resistance, 349 dyn·sec·cm^{-5}; systemic vascular resistance, 2332 dyn·sec·cm^{-5}. A

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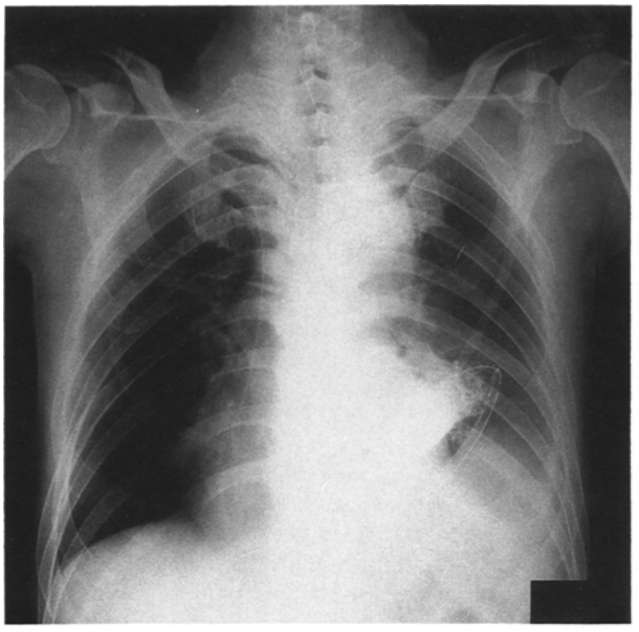

Fig. 1. Chest roentogenogram before operation showing traumatic hernia of the left porition of the diaphragm

chest roentogenogram revealed a diffuse parenchymal shadow in the reexpanded lung (Fig. 2).

The patient was treated with a variety of modalities and drugs, including oxygen, steroids, albumin, and the application of positive end-expiratory pressure under mechanical ventilation. Twenty-four hours after treatment, there was radiographic improvement (Fig. 3) without a fatal outcome.

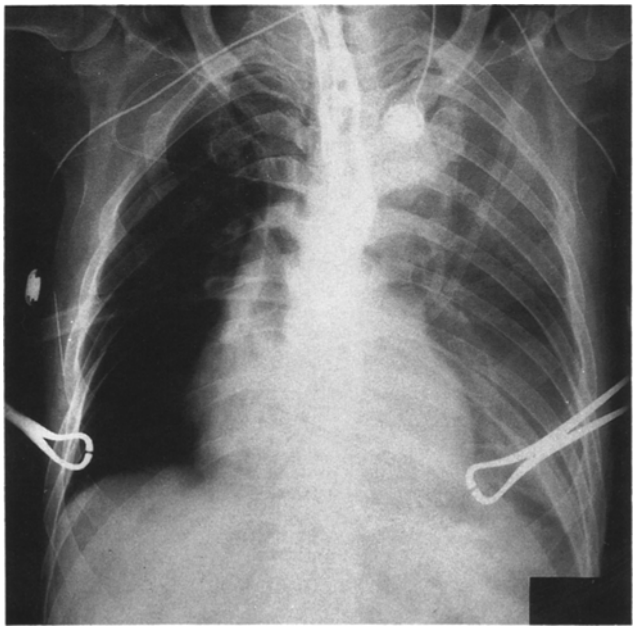

Fig. 2. Chest roentogenogram 2 h after completion of reconstruction of diaphragmatic hernia showing development of left reexpansion pulmonary edema

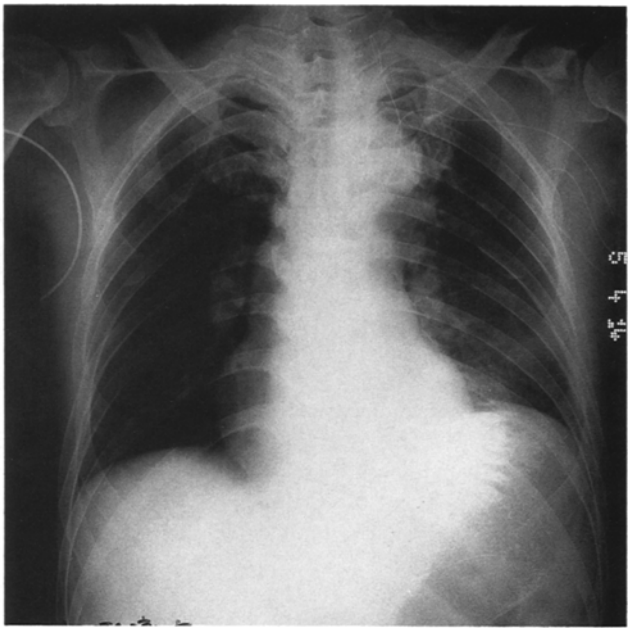

Fig. 3. Rapid clearing of lung edema

Discussion

In this case, a yellowish fluid issued from the endotracheal tube after the reconstructin of the diaphragmatic hernia and a chest roentogenogram revealed a diffuse parenchymal shadow in the reexpanded lung. These findings indicate the occurrence of reexpansion pulmonary edema (RPE) arising from the inflation of the collapsed lung. RPE occurs when a chronically collapsed lung is rapidly reexpanded by the evacuation of large amounts of air or fluid, usually with the application of high negative intrapleural pressure [1,2]. In the present case, however, the RPE occurred after inflation of the collapsed lung with the application of positive pressure ventilation.

Although the exact cause of RPE is unkown, increased pulmonary vascular permeability is considered to be a major etiological factor in its development [3,4]. The mechanisms that have been implicated in the pathogenesis of increased permeability are as follows [2]: (1) ischemic injury during lung collapse, (2) pulmonary epithelial cell and vascular endothelial cell injury due to a loss of surfactant, (3) mechanical damage due to reexpansion, (4) chemical mediators, and (5) oxygen free radicals. Probably at least two of the factors cited above contribute to the development of reexpansion pulmonary edema.

It is generally believed that the major contributing factors in the development of RPE are the chronicity of collapse (usually more than 3 days) and the rapidity of reexpansion by negative intrapleural pressure [5,6]. Experimental studies on animal specimens have

ocnfirmed these findings [7,8]. Therefore, it has been suggested that the slow evacuation of air or fluid from the pleural space [8] or repeated aspirations of less than 1000 ml of fluid or air [9] may aid in preventing the onset of RPE. However, RPE may occur after acute pulmonary collapse even if reexpansion is slow and evacuation is not employed [2].

In the present case, the collapsed lung was reexpanded by inflation with positive pressure. Under the general anesthesia associated with the reconstruction of the diaphragmatic hernia, it was difficult to reexpand the collapsed lung slowly because the patient was ventilated with positive pressure. In such a case, the anesthesiologist should use a double-lumen endotracheal tube and control the intrapulmonary pressure of the collapsed lung.

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