IPSILATERAL PULMONARY OEDEMA AFTER RAPID RE-EXPANSION OF PNEUMOTHORAX

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SYNOPSIS

A case report on the rare complication of ipsilateral pulmonary oedema following rapid re-expansion of the pneumothorax is described. The contributory factors and pathogenesis are discussed.

INTRODUCTION

A reduction in the period of hospitalisation and an earlier return to normal activity are arguments for the insertion of an intercostal catheter attached to a suction apparatus as a means of treating spontaneous pneumothorax. This is, however, not without danger under certain circumstances. In this paper we report on a case of pulmonary oedema following rapid re-expansion of a pneumothorax.

CASE REPORT

A 68-years-old Chinese male was admitted on Dec. 5 1975, with a history of exertional breathlessness and an occasional unproductive cough for 8 days. There was no other history of note. For the past 40 years, he had been smoking 20 cigarettes daily but stopped the habit a year ago.

On examination, he was slightly dyspnoeic. The trachea was shifted to the left with signs of a right pneumothorax (Fig. 1) with a slight shift of the mediastinum to the left.

A Jacques rubber catheter (size 11) was then inserted in the second right intercostal space lateral to the mid-clavicular line. Air was felt gushing out. The tube was connected to a Gomco pump with continuous suction at minus 10cm water via an underwater seal. The patient was then noted to be slightly more dyspnoeic and the chest X-ray done an hour later showed expansion of the right lung with diffuse pulmonary opacities. The ipsilateral pulmonary opacities increased on the next day (Fig. 2) and subsequently disappeared spontaneously with bed rest on the 6th day (Fig. 3). His course in hospital was otherwise uncomplicated. Subsequent investigations showed active minimal pulmonary tuberculosis in the right upper lobe and anti-tuberculous treatment was commenced. The patient was discharged on the 15th day.

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DISCUSSION

Pulmonary oedema is a rare complication following the rapid re-expansion of a pneumothorax. It appears to occur with a large pneumothorax, where the pulmonary collapse has been prolonged for many days and with the introduction of negative intrapleural pressure suction (Ziskind *et al*, 1965). The earliest reported cases were probably by Carlson *et al* (1959). Since then 12 cases have been reported (Waqaruddin *et al*, 1975). The duration of pneumothorax averaged 18 days with a minimum of three days with the exception of one patient (Humphreys *et al*, 1970) in whom the pneumothorax was a complication from the insertion of a central venous pressure catheter and was re-expanded within one hour. Five of the 12 pneumothoraces were re-expanded under negative pressure suction.

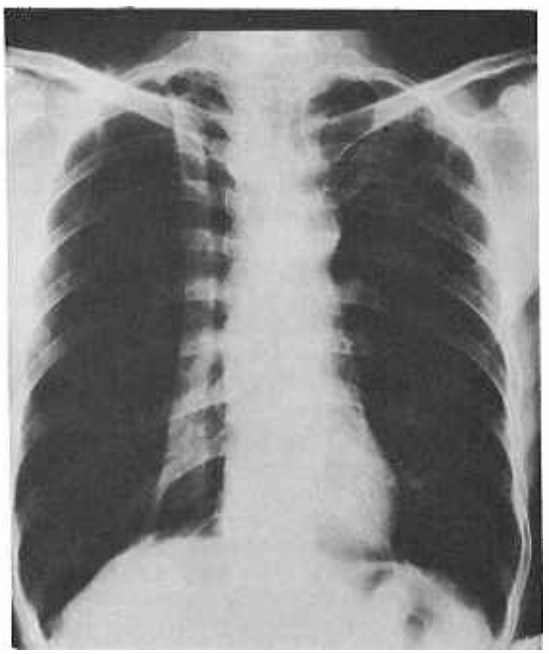

Fig. 1. Large right pneumothorax with slight mediastinal shift to the left.

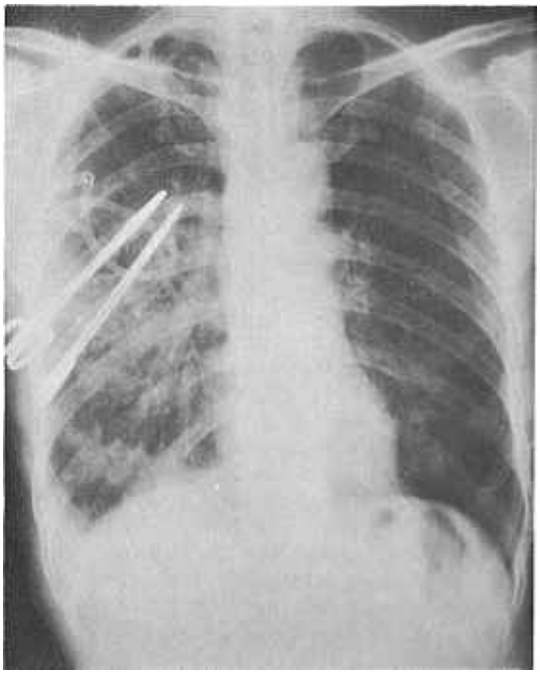

Fig. 2. Ipsilateral pulmonary oedema after pleural aspiration under negative pressure.

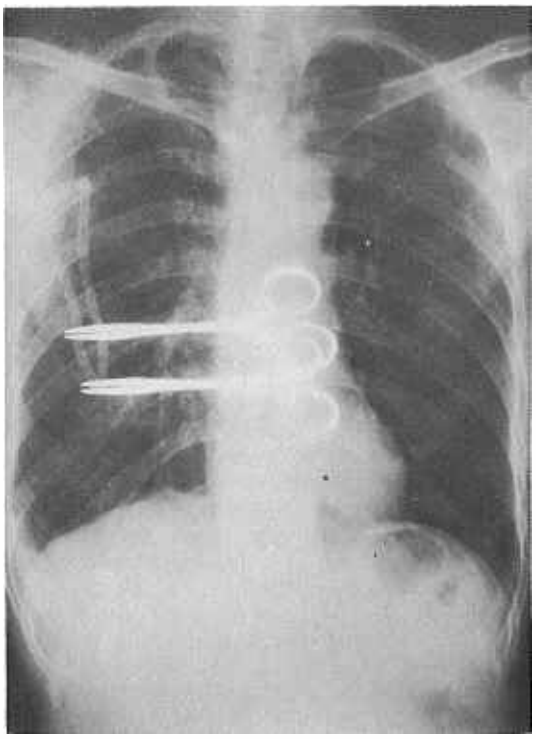

Fig. 3. Complete disappearance of pulmonary oedema on 6th day.

Various theories have been postulated for the cause of the pulmonary oedema. Prolonged compression and atelectasis with hypoperfusion of the ipsilateral lung have been postulated to have an hypoxic effect on the pulmonary capillaries with a predisposition to leakage of fluid on re-expansion (Carlson *et al*, 1959 and Humphreys *et al*, 1970). A sudden and excessive negative intrapleural suction pressure has also been noted to cause the leakage of fluid from the circulation into the tissue but an occlusion of the bronchus as by a thick mucus plug is necessary to produce the effective pressure gradient (Childress *et al*, 1971). However, such an occlusion has not been shown in any of the cases or experimentally by Miller *et al* (1973). Trapnell *et al* (1970) and Sautters *et al* (1971) further postulate that the increased alveolar surface tension with the decrease of surfactant in the chronically collapsed lung also contributes to the development of pulmonary oedema but Robin *et al* (1973) have shown that oedema can develop in a degassed lobe without an air-liquid interface.

An experiment to identify the contributory factors mentioned above, was performed by Miller *et al* (1973). They studied the experimental pulmonary oedema in the lungs of Rhesus Monkeys following re-expansion of the iatrogenic unilateral pneumothorax with 80 to 100% collapse. The observation was that a large pneumothorax of three or more days' duration and the application of large negative pressure to re-expand the lungs were factors predisposing to pulmonary oedema. Our patient had a large pneumothorax of eight days re-expanded under negative pressure resulting in unilateral pulmonary oedema.

The clinical course of the pulmonary oedema varied among the cases reported but two deaths have been recorded (Trapnell *et al*, 1970 and Sautter *et al*, 1971). Our patient had a spontaneous recovery by the sixth day. No active treatment for the pulmonary oedema was given as the patient was comfortable but he was kept under close observation. Whatever the clinical course might be, the proper management would be to recognise the predisposing factors and take special care to prevent and treat the complication.

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