IPSILATERAL PULMONARY OEDEMA AFTER DRAINAGE OF SPONTANEOUS PNEUMOTHORAX

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SYNOPSIS

Seven cases of ipsilateral pulmonary oedema following insertion of a chest tube for drainage of the pneumothorax are described. This complication is not uncommon. Contributory factors and pathogenesis are discussed and the literature reviewed.

INTRODUCTION

Spontaneous pneumothorax is not rare. Drainage via an intercostal catheter with or without attachment to a suction apparatus is carried out when indicated although the use of suction is arbitrary. Of 40 episodes of unilateral spontaneous pneumothoraces in patients requiring catheter drainage seen in the medical unit over the period December 1975 to March 1978, eight cases of ipsilateral pulmonary oedema after drainage of the pneumothoraces were encountered. The first case has been reported (Ooi and Leong 1976). The other seven cases are now presented.

CASE REPORTS

Case 1 In March 1976, a 58 year old man who presented with a spontaneous left pneumothorax of eight days' duration had it drained under suction with no sequelae. One week later he developed a right pneumothorax with dyspnoea for one day. This was similarly treated but he developed radiological evidence of ipsilateral pulmonary oedema though the dyspnoea improved. Fig. 1 (a) and 1 (b). He was treated for pulmonary tuberculosis from 1961 — 64.

Case 2 A 20 year old female developed sudden left chest pain in April 1976 but sought hospital admission one month later when she was found to have a left pneumothorax. After two chest aspirations, the lung expanded and she went home. One week later in May she was re-admitted for the same condition and this time an intercostal chest tube was con-

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nected to an underwater seal for drainage but no suction was applied. A chest radiograph the next day showed diffuse pulmonary opacities in the left lung field. She was sent for pleurodesis. Fig. 2 (a) and 2 (b).

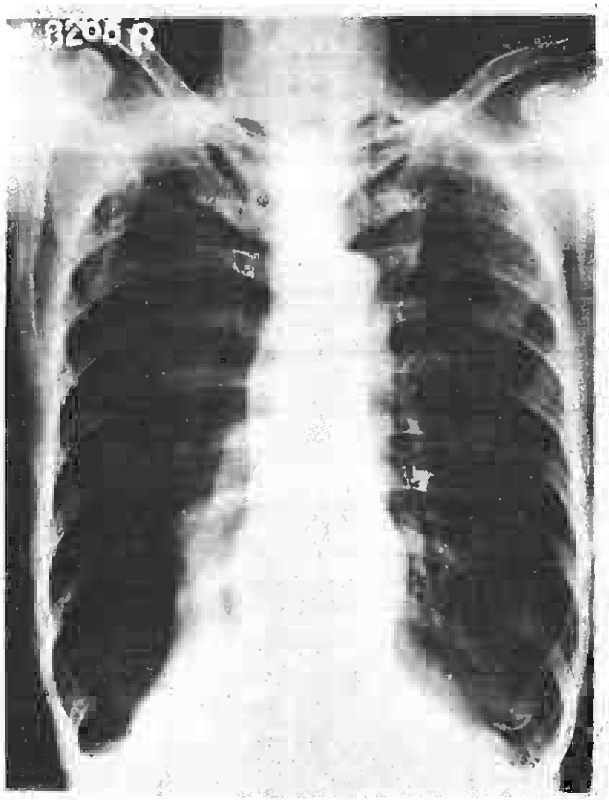

Fig. 1 (a) Large right pneumothorax. Old tuberculosis scars at apices.

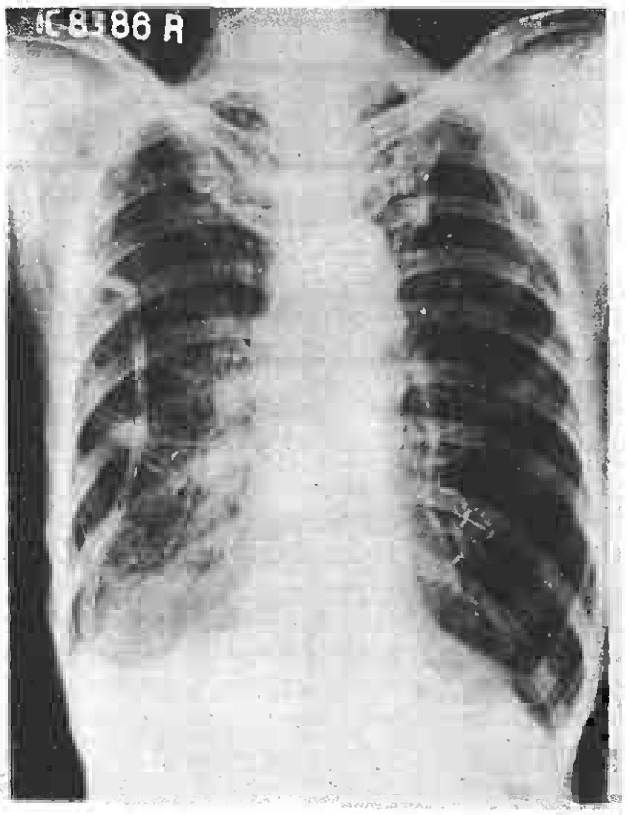

Fig. 1 (b) Radiological evidence of pulmonary oedema in the right middle and lower lobes.

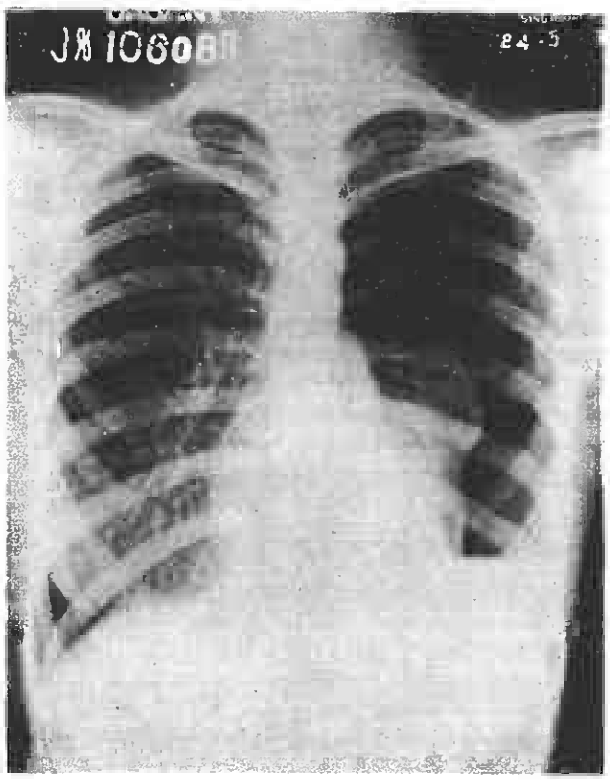

Fig. 2 (a) Left pneumothorax

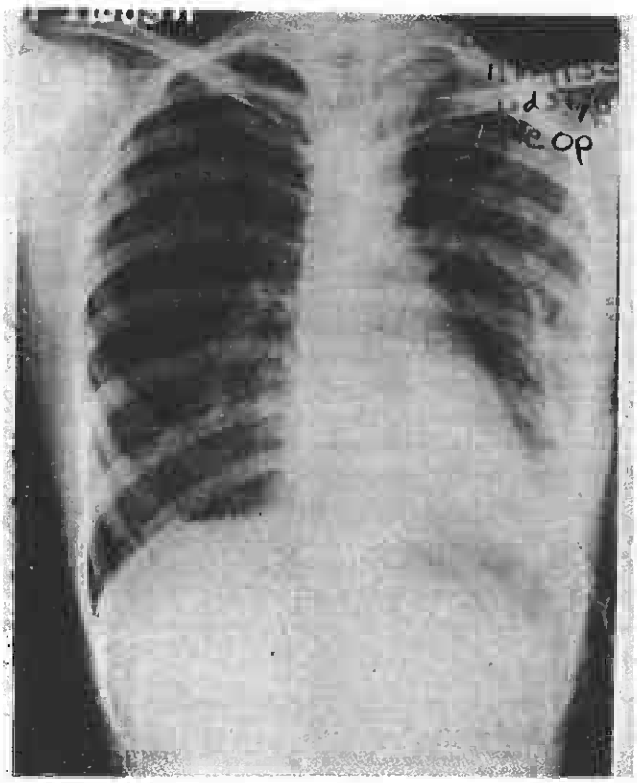

Fig. 2 (b) Diffuse pulmonary opacities in left lung field after drainage.

Case 3 In June 1976 an apparently healthy 30 year old male had dysponea for one week and chest pain for two days due to a right pneumothorax. Fig. 3 (a). This was drained by a rubber chest tube and connected to a suction apparatus at a pressure of minus 10 cm. of water. Though he improved clinically, a chest radiograph showed ipsilateral pulmonary oedema. Fig. 3 (b).

Case 4 A 72 year old woman developed a right pneumothorax with cough and dyspnoea for two weeks before she was hospitalised in August 1977. She was treated for pulmonary tuberculosis seven years before. The pneumothorax was drained without suction. The chest radiograph the next day showed marked right pulmonary opacities. Fig. 4 (a) and 4 (b). She improved clinically and was no more dyspnoeic.

Case 5 In August 1977 a 63 year old male, treated for pulmonary tuberculosis 30 years previously, developed dyspnoea and cough progressively worsening over two weeks. He was also an opium addict. His

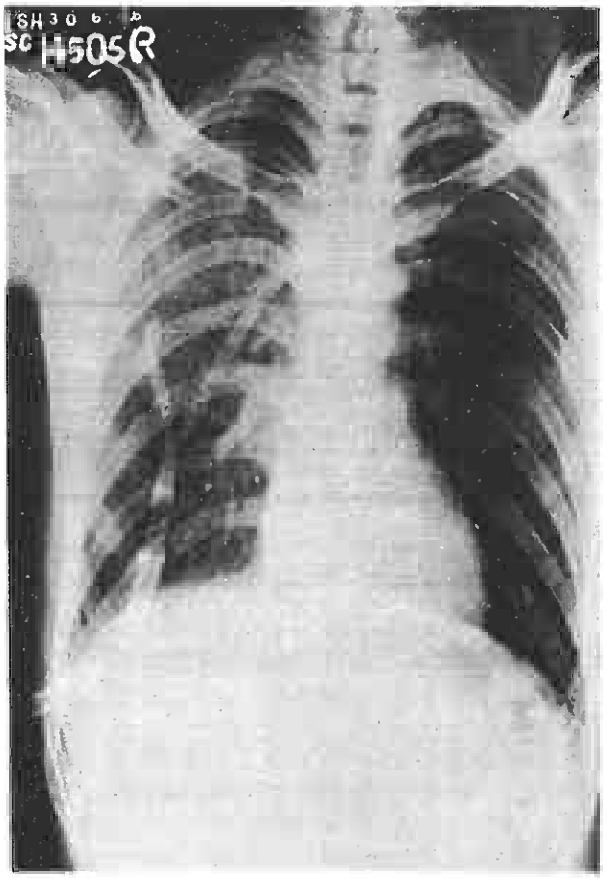

Fig. 3 (b) Ipsilateral pulmonary oedema

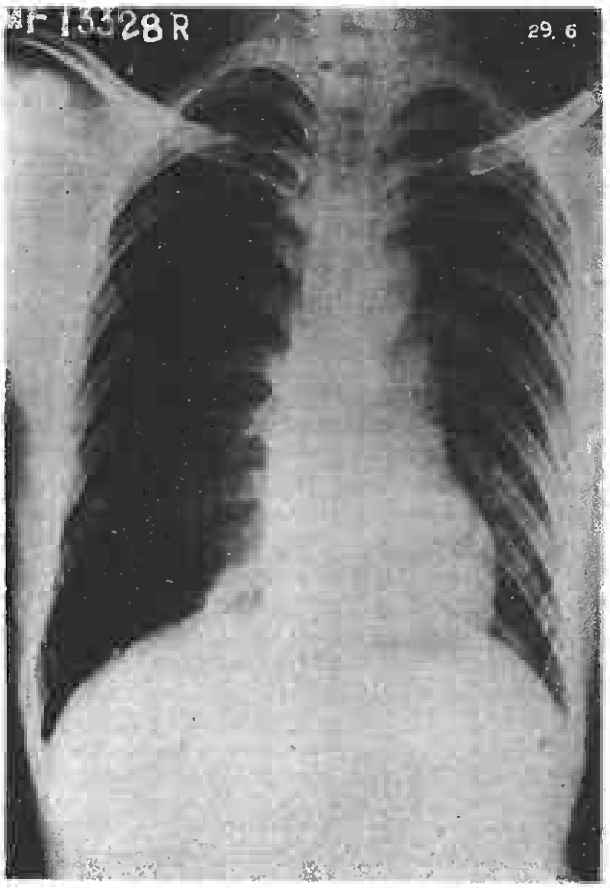

Fig. 3 (a) Large right pneumothorax

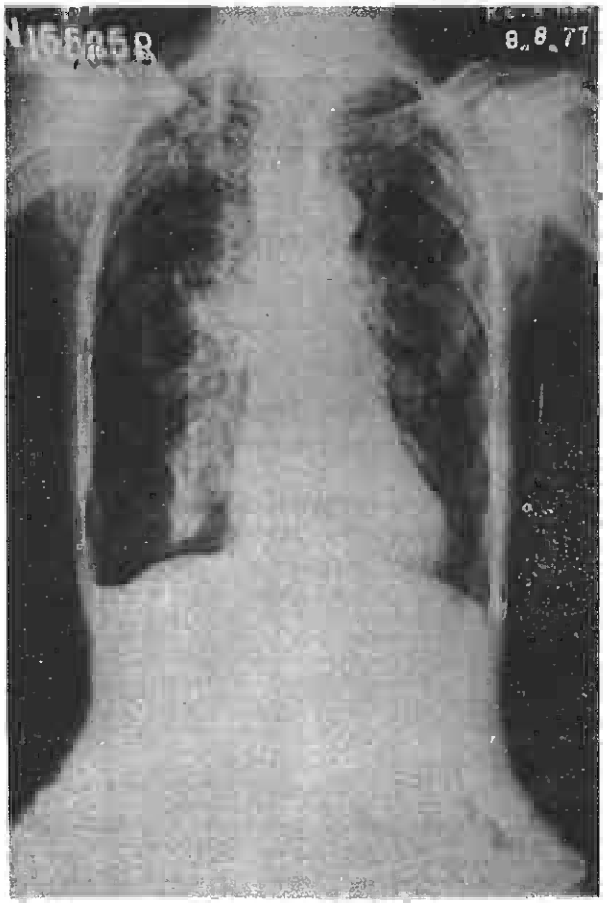

Fig. 4 (a) Right pneumothorax

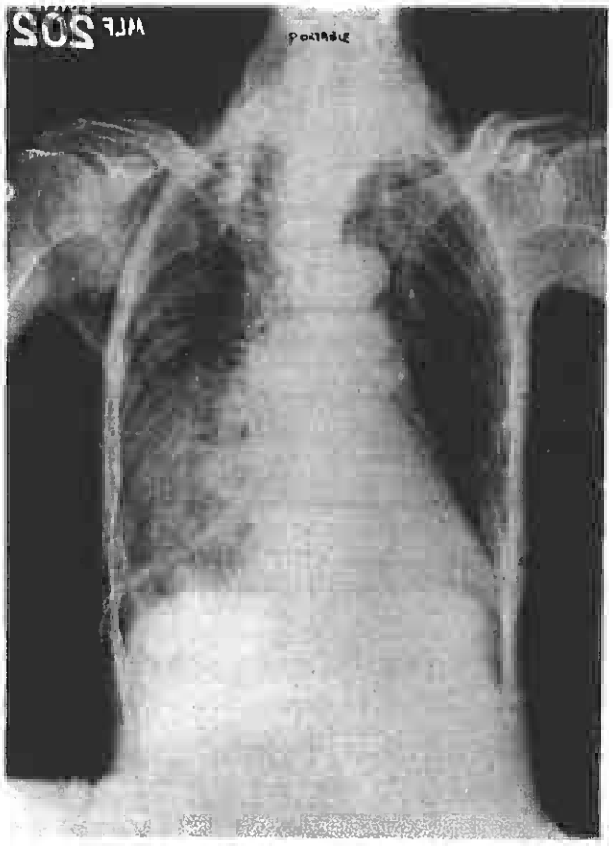

Fig. 4 (b) Ipsilateral pulmonary oedema

left pneumothorax was drained and suction at minus 10 cm. water applied. He developed radiological evidence of pulmonary oedema. Fig. 5 (a) and 5 (b).

Case 6 A healthy 20 year old female was hospitalised in November 1977 for a left pneumothorax. She had complained of sudden left chest pain with dyspnoea two months previously but only the pain had subsided since. Chest tube drainage without suction was used and the chest radiograph the following day showed evidence of ipsilateral pulmonary oedema. Fig. 6 (a) and 6 (b).

Case 7 In February 1978 a 57 year old male, treated for pulmonary tuberculosis in 1970, was admitted for a right pneumothorax of one week's duration. In 1972 he had a right pneumothorax which re-expanded after intercostal chest tube drainage without suction, and there was no pulmonary oedema. This time the same treatment was given but hazy opacities were seen in the chest radiograph taken the following day. Fig. 7 (a) and 7 (b).

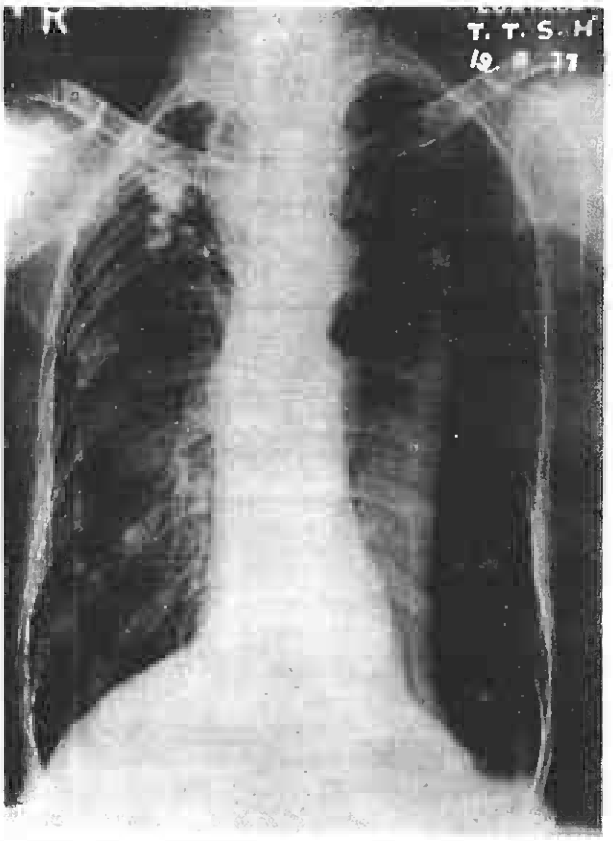

Fig. 5 (a) Large left pneumothorax. Old tuberculosis scars at apices.

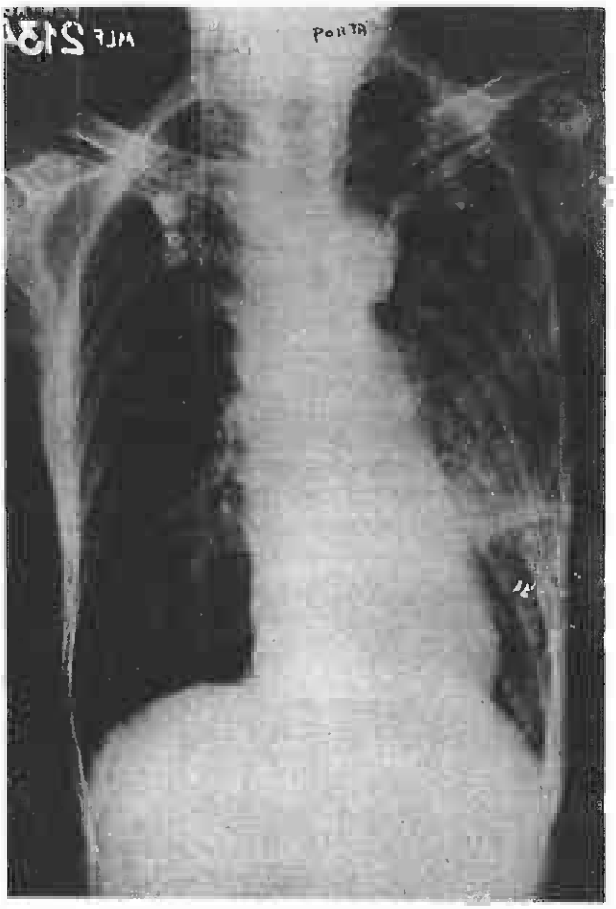

Fig. 5 (b) Left lower pulmonary opacities after drainage of pneumothorax

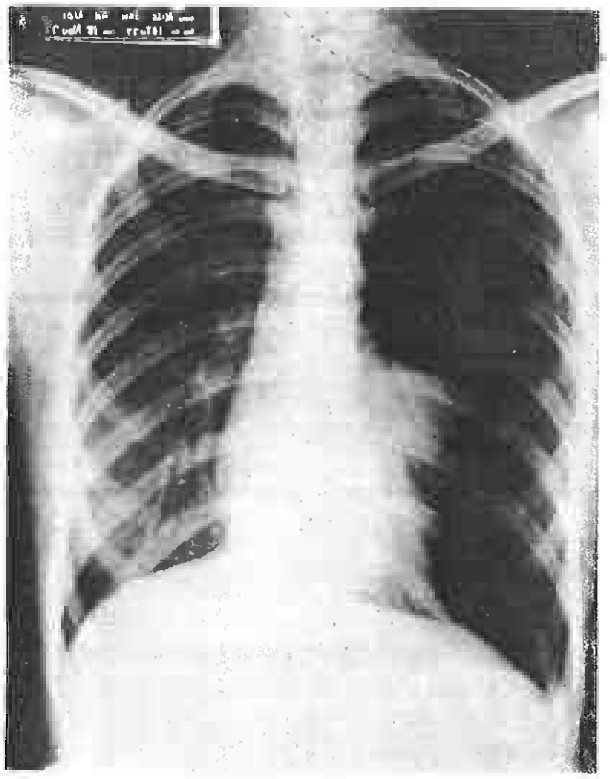

Fig. 6 (a) Left pneumothorax

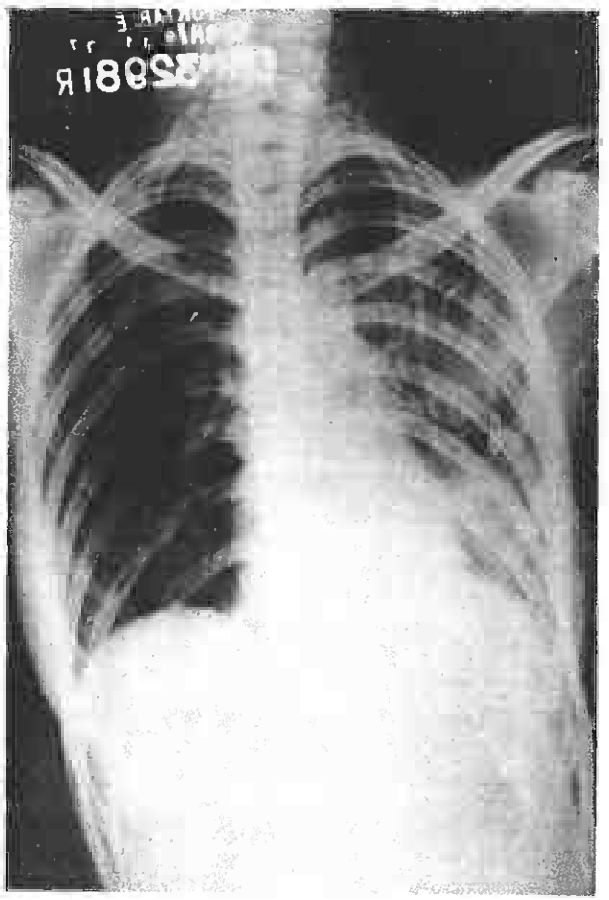

Fig. 6 (b) Ipsilateral left pulmonary oedema

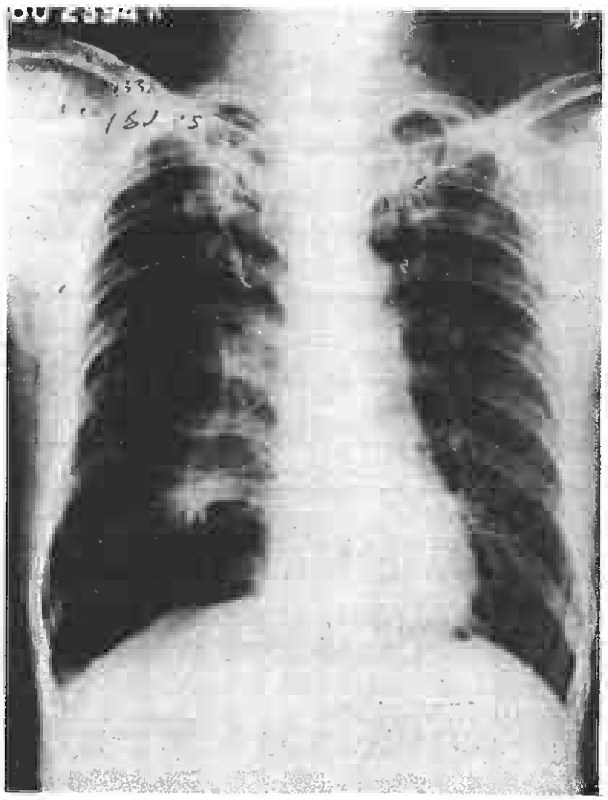

Fig. 7 (a) Right pneumothorax. Old tuberculosis scars at apices.

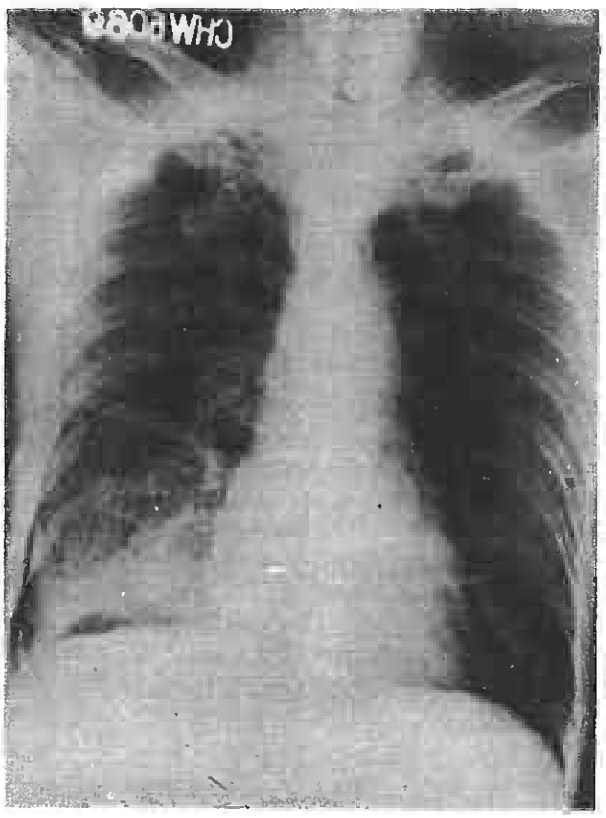

Fig. 7 (b) Ipsilateral pulmonary oedema after drainage of pneumothorax

As these seven patients improved after insertion of the chest tubes to drain their pneumothoraces, the chest radiographs done the next day were a routine following the chest tube insertion. No daily radiographs were done but it was noted that by the fifth day after insertion of the chest tube, the pulmonary opacities in the chest x-ray had cleared. None of the patients deteriorated clinically despite the radiological picture of pulmonary oedema. Clinical examination failed to reveal lung crepitations at that stage. All were closely observed but none required specific therapy for the oedema.

DISCUSSION

Carlson et al in 1959 reported the first case of pulmonary oedema following the rapid re-expansion of a totally collapsed lung due to a pneumothorax. To-date twelve other cases have been published but the reports were of one or two cases. This report details seven cases. A summary of the salient features of all these twenty cases is shown in Table I.

Childress et al (1971) reported a case of severe unilateral pulmonary oedema that followed evacuation of a spontaneous pneumothorax. The oedema occurred one minute after suction was applied. The patient was in shock and received oxygen, intravenous fluids, aminophylline and diuretics, recovering after six days. Analysing this case, the authors concluded that two factors were required for the development of acute pulmonary oedema viz. bronchial occlusion and a very negative intrapleural pressure combining to cause fluid transudation from the pulmonary capillaries into the alveoli and interstitial tissue.

Miller et al (1973) experimented with eighteen Rhesus monkeys. Six had a unilateral 80 — 100% pneumothorax induced and left for one hour before being re-expanded under minus 10 mmHg. suction pressure. No pulmonary oedema occurred. The other twelve had the pneumothorax untreated for three days. Then six were re-expanded with underwater seal and no suction: none developed pulmonary oedema. The other six had pulmonary oedema in two hours after rapid re-expansion with suction present at minus 10 mmHg. Thus the duration of the pneumothorax was also a factor.

Table I: Twenty cases of ipsilateral pulmonary oedema after drainage of spontaneous pneumothorax

Author/Year	No. of patients	Duration of Pneumothorax	Use of Suction
Carlson et al 1959	1	5 weeks	Yes
Ziskind et al 1970	1	3 days	Yes
Humphreys & Berne 1970	2	14 days	No
		1 hour	No
Trapnell & Thurston 1970	2	8 days	No
		3 days	Yes
Childress et al 1971	1	6 days	Yes
Sautter et al 1971	1	81 days	No
Ratcliff et al 1973	1	3 days	Yes
Saini 1974	1	7 days	No
Waqaruddin 1975	2	5 weeks	No
		3 days	No
Ooi & Leong 1976	1	8 days	Yes
Present series 1978	7	1 day	Yes
		1 day	No
		7 days	Yes
		14 days	No
		14 days	Yes
		8 weeks	No
		7 days	No

It would seem therefore that a large pneumothorax of prolonged duration with rapid evacuation of the air under high negative intrapleural pressure predisposes to the development of unilateral ipsilateral pulmonary oedema. Clinically the picture is variable ranging from asymptomatic patients (present series) to those with severe dyspnoea, shock and even death.

Treatment of the pulmonary oedema is along conventional lines though severe hypovolemia with profound hypotension requires massive fluid administration. (Hoffmann 1977).

The two reported deaths are by Trapnell & Thurston (1970) and Sautter et al (1971). An eighteen year old male with spontaneous left pneumothorax had suction applied and in four hours developed pulmonary oedema. Venous tourniquets, intravenous aminophylline, morphine, frusemide and digoxin were administered to no avail. Surfactant given with inspired oxygen nebulised with alcohol was also tried. He had in addition other congenital anomalies and also old pulmonary tuberculosis. The case reported by Sautter et al was a 69 year old male with a right pneumothorax of three weeks' duration. He also had hypertension, aortic incompetence with several past episodes of congestive cardiac failure. He died one hour after drainage of the pneumothorax.

The present series has seven cases all with large pneumothoraces five of which were of greater than a week's duration. The first two patients had recent pulmonary problems and were discharged only one week prior to their being admitted again for the pneumothorax. Only three had negative intrapleural pressure applied. It would appear that the duration and size of the pneumothorax are most important in the pathogenesis of the ipsilateral pulmonary oedema and this could be related to the lack of surfactant in the collapsed lung.

Our experience indicated that ipsilateral pulmonary oedema following drainage of pneumothorax is not as rare as it would appear from a review of the literature. Eight cases of this complication occurred in forty episodes of unilateral spontaneous pneumothorax in patients seen over a period of twenty eight months.

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