

LIFE THREATENING RE-EXPANSION HYPOTENSION AND PULMONARY OEDEMA FOLLOWING TREATMENT OF A PNEUMOTHORAX

K N Sin Fai Lam, L S Chew

ABSTRACT

A 27-year old man was admitted with a right-sided pneumothorax of 2-3 weeks duration. A chest tube was inserted and connected to an underwater seal drainage system without the application of external suction. Three hours later, the patient developed unilateral re-expansion pulmonary oedema and severe hypotension. Active management consisted of ventilating the patient with the addition of PEEP, and the administration of liberal amounts of fluids, including plasma and gelatin solution. The mechanism of re-expansion pulmonary oedema is different from that of cardiogenic pulmonary oedema, and the treatment consequently different. The cause of the hypotension may be due to hypovolaemia, from rapid pooling of fluid within the thorax, pre-existing volume depletion and myocardial depression. One must specially be aware of this possible complication when the pneumothorax is large and of more than 3 days, and it is to be stressed that suction should never initially be used in the treatment of a pneumothorax.

Keywords: Pneumothorax, Re-expansion, Pulmonary oedema, hypotension

SING MED J. 1989; NO 30: 502-505

INTRODUCTION

The treatment of a pneumothorax is usually a simple procedure with little complication. However, in rare instances, it can lead to a life-threatening situation.

The case reported is of interest because of the occurrence of severe unilateral pulmonary oedema associated with haemodynamic instability following treatment of a pneumothorax.

CASE REPORT

A 27-year old Chinese man was admitted on 6 October 1988 with a history of chest pain, cough and dyspnoea, the onset of which was two to three weeks previously. He had seen a private practitioner who did not initially make the appropriate diagnosis but at a subsequent visit, a chest x-ray was carried out and it showed a near complete right pneumothorax. He was previously healthy, a non-smoker and had participated in various sports. On admission, the vital signs were as follows: pulse 96/min. Blood pressure 140/90. Temperature 37°C. The patient was not short of breath at rest. Investigations done on admission: Haemoglobin 16.2 G/dl, White cell count $6.5 \times 10^9/L$, serum sodium 136 mmol/L, serum potassium 3.5 mmol/L, chloride 109 mmol/L, urea 27 mg/dl. A right lateral chest tube was inserted at 3.45 a.m. and connected to an underwater seal drainage system without the application of external suction.

Department of Medicine
Alexandra Hospital
Alexandra Road
Singapore 0314

K N Sin Fai Lam, MB, MRCPI
Senior Registrar

L S Chew, MBBS, FRACP
Consultant Physician and Head

At 7.00 a.m. the patient complained of cough, sweating and slight breathlessness. The blood pressure was 110/70. Crepitations were heard in the right lung. Oxygen was prescribed at 40 % concentration. A chest x-ray was carried out and showed florid unilateral pulmonary oedema. (Fig. 1). At 8.30 a.m. the patient was noted to be very breathless, sweaty and cyanosed. He coughed up copious amounts of pink frothy sputum. Arterial blood gases with an FiO_2 40% were as follows: pH 7.248, pCO_2 33.9 mm Hg, pO_2 46.4 mm Hg, Std Bicarbonate 14.4 mmol/L. The patient was hypotensive and the blood pressure dropped to a low of 60 mm Hg systolic.

The patient was intubated and ventilated using the Bird Percussionaire VDR 3 Autocycle (R) respirator. FiO_2 was set at 40%. Further laboratory values were obtained as follows: Arterial blood gases: pH 7.420, pCO_2 22 mm Hg, pO_2 80.2 mm Hg, Std Bicarbonate 18.2 mmol/L. Haemoglobin 23.3 G/dl, Haematocrit 69%, White cell count $27.8 \times 10^9/L$.

The patient remained hypotensive; the systolic blood pressure ranged from 60-80 mm Hg. From 7.00 a.m. to 12.00 pm, only 20 mls of urine was produced. The initial CVP reading was -4 cm H_2O . Over the next 12 hours, 4,500 mls of fluid were infused. This included one litre of pooled plasma and one litre of a 3% gelatin solution. The amount of fluid infused was titrated against the CPV reading (maintained at +12 cm H_2O), the systemic blood pressure and the urine output. PEEP of 8 cm was added when the systolic blood pressure rose to 100 mm Hg.

With correction of the hypovolaemia, the urine output improved and the total output over the 24 hours was 1500 mls.

His subsequent progress over the next few days was satisfactory. The pulmonary oedema cleared gradually and completely (Fig. 2). Artificial ventilation was discontinued on 9 October, and the patient was extubated on 10 October. The chest tube was removed on 14 October.

The Haematology profile on 10 October, five days after admission was as follows: Haemoglobin 14.9G/dl, White cell count $11.7 \times 10^9/L$, and Haematocrit 45.7%.

The patient made a complete recovery and was discharged.

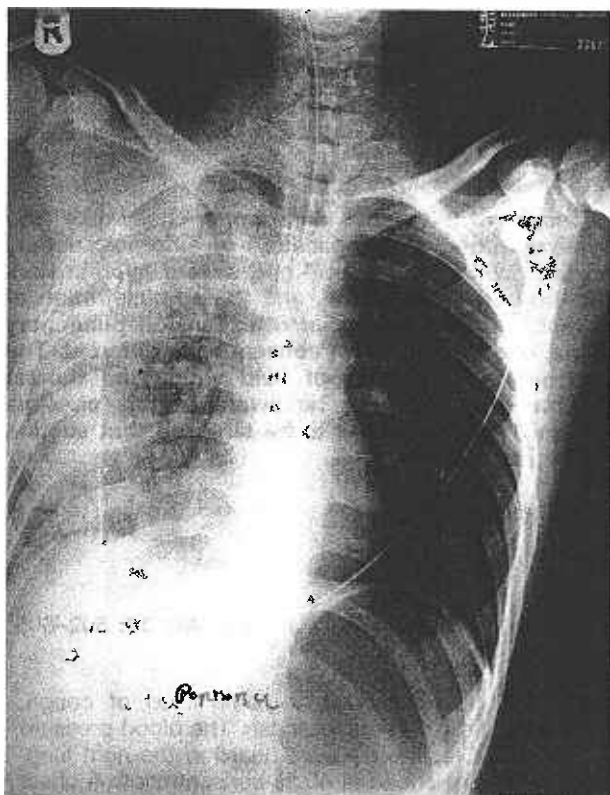


Fig 1: 7 October 1988
Unilateral re-expansion pulmonary oedema

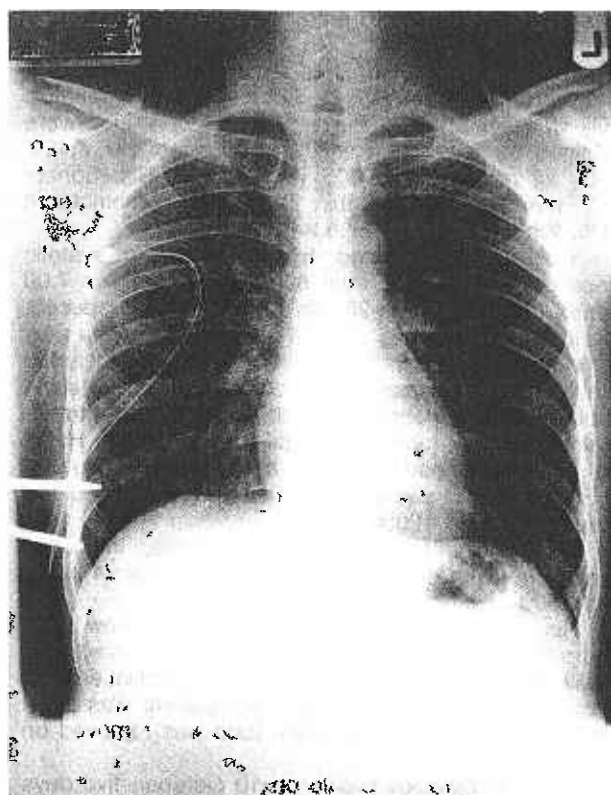


Fig 2: 13 October 1988
Complete clearing of pulmonary oedema

DISCUSSION

Reports of pulmonary distress following thoracentesis for pleural effusion have appeared as far back as 1853. Pinault reported that rapid evacuation of a large pleural effusion was followed by a form of respiratory distress entitled "acute albuminous expectorations". Subsequently, Foucart in 1875, Ortner in 1899, and Hartley (1) in 1906 have also described this complication.

However, pulmonary oedema after re-examination of pneumothorax has been described less commonly. The first description was by Carlson, Classen and Gollan (2) in 1959, followed by others (3-26). It has also been described in the local literature (24-26). Chee (25) in 1979 described 7 cases of ipsilateral pulmonary oedema following the insertion of a chest tube for drainage of a pneumothorax. External suction was utilised in only 3 out of 7 cases. However, in all of the 7 cases, the patient was asymptomatic and required no other specific treatment. Narendran K (26), however, in 1986 described a case of fatal re-expansion pulmonary oedema which was similar to our present patient in that it was associated with hypotension and haemodynamic disturbance. Re-expansion pulmonary oedema occurs rapidly in the underlying lung as it re-expands and is associated with variable degree of respiratory distress. Some of these patients develop a shock-like state with hypotension and impaired organ perfusion.

It has been quoted that the development of pulmonary oedema is a relatively benign condition (Bernstein, 12). In most instances, it has been described as a radiological diagnosis without major clinical consequence (Mahajan, 19). However, several reports have emphasised the potentially life threatening nature of this complication, and several deaths have been reported. This is well-illustrated by our patient who would surely have perished without intensive management.

Many factors have been put forward as being the underlying cause of re-expansion pulmonary oedema. It is unlikely that a single one is responsible. Chronic collapse of the lung of more than 3 days is said to be a pre-requisite although a report by Sherman (21) illustrates re-expansion pulmonary oedema developing after treatment of a large pneumothorax of less than one day's duration. However, a negative pressure of 20 cm H₂O was applied to the pleural space. A negative intrapleural pressure is thought to be another important factor. Nevertheless, in some cases (23), as well as in our case, no negative suction was applied. One must include therefore that it is not the level of negative pressure, but the rate of re-expansion which is critical. The pressure of airways obstruction and high negative intrapleural pressure enables a pressure gradient to develop between the pulmonary capillaries and alveolar space. However, in most instances, there is no evidence of bronchial obstruction and in experimental studies by Carlson (2) and Miller (29), no obstruction was present. Other factors involved include increased alveolar-capillary membrane permeability and loss of tissue surfactant.

The mechanism of the pulmonary oedema therefore is different from that of cardiogenic pulmonary oedema. Several researchers have carried out haemodynamic monitoring and evaluation of pulmonary oedema in patients with re-expansion pulmonary oedema. They have found a pulmonary oedema-serum protein ratio range of 0.81(8), 0.74(27), 0.85(28), which is typical of non-cardiogenic pulmonary oedema. The ratio is < 0.50 in cardiogenic pulmonary oedema. In addition, the mean pulmonary artery wedge pressures were normal.

Hypotension has been noted to be a significant finding in the recently described cases of re-expansion of pulmonary oedema (23). Again the mechanism of

TIME	Haemoglobin (G/dl)	White cell count (x10 ⁶ /L)	Haematocrit %
0300 hrs	16.2	6.5	—
1000 hrs	23.3	27.8	69
DAY 5	14.9	11.7	45.7

Table I
HAEMATOLOGICAL INDICES AT VARIOUS STAGES OF PATIENT'S ILLNESS

TIME AFTER ADMISSION	CENTRAL VENOUS PRESSURE	BLOOD PRESSURE	TOTAL URINE OUTPUT	TOTAL FLUID INFUSED
0	—	140/90	0	0
8 hrs	-4 cm	systolic 60 mm	20 mls	500 mls
24 hrs	+12 cm	120/80	1500 mls	5000 mls

Table II
RELATIONSHIPS BETWEEN TOTAL FLUID INFUSED AND CVP, BLOOD PRESSURE AND URINE OUTPUT

hypotension is multifactorial. Pavlin (23) suggested: 1) Acute vascular volume depletion caused by rapid pooling of fluid within the thorax following re-expansion of the lung. 2) Pre-existing volume depletion as a result of persistent pneumothorax and hypoxaemia. 3) Myocardial depression following the evacuation of the pneumothorax.

Our patient was in a state of shock when he developed re-expansion pulmonary oedema. He had decreased organ perfusion with minimal urine output. That he had hypovolaemia is well demonstrated by the elevated Haemoglobin of 23.3 G/dl (on admission Hb was 16.2 G/dl, and a Haematocrit of 69% (Table 1). In cardiogenic pulmonary oedema, we would be treating with diuretics and restricting fluid intake, both these measures being harmful in re-expansion oedema. In our patient, a total of 4,500 mls of fluid was given over a period of 12 hours. Other cases (23) have been described when up to 11 litres of fluid were required over a period of 24 hours to correct the hypovolaemia, the

hypotension and maintain organ perfusion. (Table II)

In summary, the development of re-expansion pulmonary oedema and re-expansion hypotension cannot be predicted in any patient treated for pneumothorax. However, the presence of a large pneumothorax, prolonged duration of pulmonary collapse for more than 3 days, hypoxaemia, an elevated or rising haemoglobin or haematocrit are signs which should alert us as to its possible development. The radiological diagnosis of a pneumothorax does not usually constitute an emergency situation unless there is evidence of tension or bleeding into the pleural cavity. Therefore re-expansion of the lung can be allowed to take place gradually. There is never any indication to apply suction to the pleural cavity initially, unless a persistent air leak prevents full expansion of the lung, and even then, the negative suction should not be higher than 10 cm of water. Vigorous fluid therapy is necessary to preserve haemodynamic stability and organ perfusion in the presence of pulmonary oedema.

REFERENCES

- Hartley P: Albuminous expectoration following paracentesis of the chest. St Bartholomew's Hospital Rep 1906; 41:77.
- Carlson RI, Classen KL, Gollan F: Pulmonary oedema following the rapid expansion of a totally collapsed lung due to a pneumothorax; A clinical and experimental study. Surg Forum 1959; 9:367.
- Shanahan MX, Monk I, Richards HJ: Unilateral pulmonary oedema following re-expansion of pneumothorax. Anaesth Intensive Care 1975; 3:19-30.
- Grant MJA: Acute unilateral pulmonary oedema following re-expansion of a spontaneous pneumothorax. NZ Med J 1971; 74:250-1.
- Ratliff JL, Chavez CM, Jamchuk A, Forestner JE, Conn JH: Re-expansion pulmonary oedema. Chest 1973; 64:654-6.
- Brenna NJ, Fitzgerald MX: Anatomically localised re-expansion pulmonary oedema following pneumothorax drainage. Respiration 1979; 38:233-7.
- Humphreys RL, Berne AS: Rapid re-expansion of pneumothorax: a cause of unilateral pulmonary edema. Radiology 1970; 509-12.

8. Sprung CL, Loewenherz JW, Baier H, Hauser MJ. Evidence for increased permeability in re-expansion pulmonary edema. *Am J Med* 1981; 71:497-500.
9. Poulias GE, Prombonas E. Massive unilateral pulmonary edema as a rapid re-expansion sequel. *Scand J Thorac Cardiovasc Surg* 1974; 8:67-9.
10. Childress ME, Moy G, Mottram M. Unilateral pulmonary edema resulting from treatment of spontaneous pneumothorax. *Am Rev Respir Dis* 1971; 104:119-21.
11. Rogaly E, Mervitz MD. Unilateral pulmonary edema after drainage of a spontaneous pneumothorax. *S Afr Med J* 1975; 49:1611-3.
12. Wagaruddin M, Bernstein A. Re-expansion pulmonary edema. *Thorax* 1975; 30:54-60.
13. Sautter RD, Dreher WH, MacIndoe JH. Fatal pulmonary edema and pneumonitis after re-expansion of chronic pneumothorax. *Chest* 1971; 60:399-401.
14. Ziskind MM, Weil H, George RA. Acute pulmonary edema following the treatment of spontaneous pneumothorax with excessive negative intrapleural pressure. *Am Rev Respir Dis* 1965; 92:632-6.
15. Shaw TJ, Caterine JM. Recurrent re-expansion pulmonary edema. *Chest* 1984; 86:784-6.
16. Milne B, Spence D, Lynn RB, Sleemen D. Unilateral re-expansion pulmonary edema during emergence from general anaesthesia. *Anaesthesiology* 1983; 59:244-5.
17. Saini GS. Unilateral pulmonary edema after drainage of a spontaneous pneumothorax. *Br Med J* 1974; 1:615.
18. Gurman C, Collins GJ, Fradis M, Podishin L. Unilateral pulmonary edema after bilateral pneumothorax. *Anesthesia* 1978; 33:613-6.
19. Mahajan VK, Simon M, Huber GL. Re-expansion pulmonary edema. *Chest* 1979; 75:192-4.
20. Peatfield RC, Edwards PR, Johnson NM. Two unexpected deaths from pneumothorax. *Lancet* 1979; i:356-8.
21. Sherman S, Ravikrishnav KP. Unilateral pulmonary edema re-expansion of pneumothorax of brief duration. *Chest* 1980; 77:714.
22. Kassis E, Philipsen E, Clausen KH. Unilateral pulmonary edema following spontaneous pneumothorax. *Eur J Respir D* 1981; 62:102-6.
23. Pavlin DJ, Raghu G, Rogers T, Cheney FW. Re-expansion hypotension: A complication of rapid evacuation of prolonged pneumothorax. *Chest* 1986; 89:70-4.
24. Ooi KH, Leong D. Ipsilateral pulmonary oedema after rapid re-expansion of pneumothorax. *Sing Med J* 1976; 17:252.
25. Chee YC, Gill DS, Poh SC. Ipsilateral pulmonary oedema after drainage of spontaneous pneumothorax. *Sing Med J* 1979; 20:283-9.
26. Narendran K. Fatal re-expansion pulmonary oedema. *Sing Med J* 1986; 27:350-2.
27. Buezko GB, Grossman RF, Goldberg M. Re-expansion pulmonary edema: evidence for increased capillary permeability. *Can Med Assoc J* 1981; 125:460-1.
28. Marlan AM, Glauser FL. Haemodynamic and pulmonary edema protein measurements in a case of re-expansion pulmonary edema. *Chest* 1982; 81:250-1.
29. Miller WC, Toon R, Palat H and Lacroix J. Experimental pulmonary edema following re-expansion of a pneumothorax. *Am Rev Respir Dis* 1973; 108:664-6.