DEXTROSE PNEUMONITIS - A COMPLICATION OF INTRAPLEURAL INSTILLATION OF 50% DEXTROSE FOR PNEUMOTHORAX WITH PERSISTENT AIR LEAK - A CASE REPORT

C B E Chee, Y T Wang, S C Poh

ABSTRACT

A 51-year-old man with active pulmonary-pleural air leak suffered acute cardiovascular and respiratory collapse with pneumonitis as a complication of intrapleural instillation of 50% dextrose.

Keywords: *Dextrose, active pulmonary-pleural air leak, pneumonitis.*

SINGAPORE MED J 1992; Vol 33: 641-642

INTRODUCTION

Chemical pleurodesis with a number of sclerosing agents (tetracycline in particular) has been carried out safely but with variable success in patients with active pulmonary pleural air leaks who were unwilling or at high risk for surgery[1,2]. Despite the theoretical risk of reflux of the sclerosing agent through the fistula into the pulmonary parenchyma thereby causing pneumonitis and lung damage, there has been, to our knowledge, no report of this complication of the procedure.

We report a case of pneumonitis as a complication of intrapleural instillation of 50% dextrose in a patient with active pulmonary-pleural air leak. We believe that reflux of the dextrose through the fistula also induced closure of the air-leak by the process of local inflammation and fibrosis as opposed to induction of chemical pleuritis as originally intended.

CASE REPORT

A 51-year-old Chinese man, a heavy cigarette smoker with hypertension and chronic obstructive airways disease complicated by cor pulmonale and hypercapnic respiratory failure, was admitted with his first episode of a spontaneous left-sided pneumothorax. A chest tube was inserted with complete re-expansion of the left lung. However, there was continuous air leakage which persisted for the next eighteen days.

He was discharged home with the chest tube to allow him to celebrate the local festive season, but was re-admitted two days later with increasing breathlessness. A chest X-ray did not reveal any recurrence of the pneumothorax but he continued to have persistent air leakage.

Ten days later, there was a recurrence of the pneumothorax. A new chest tube was inserted and the pre-existing tube removed. There was near-complete re-expansion of the left lung but the air leakage continued for a total of forty-one days.

Department of Medicine III
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

C B E Chee, MBBS, MRCP(UK), MMed(Int Med)
Registrar

Y T Wang, MBBS, MMed(Int Med), MRCP(UK), FAMS
Physician

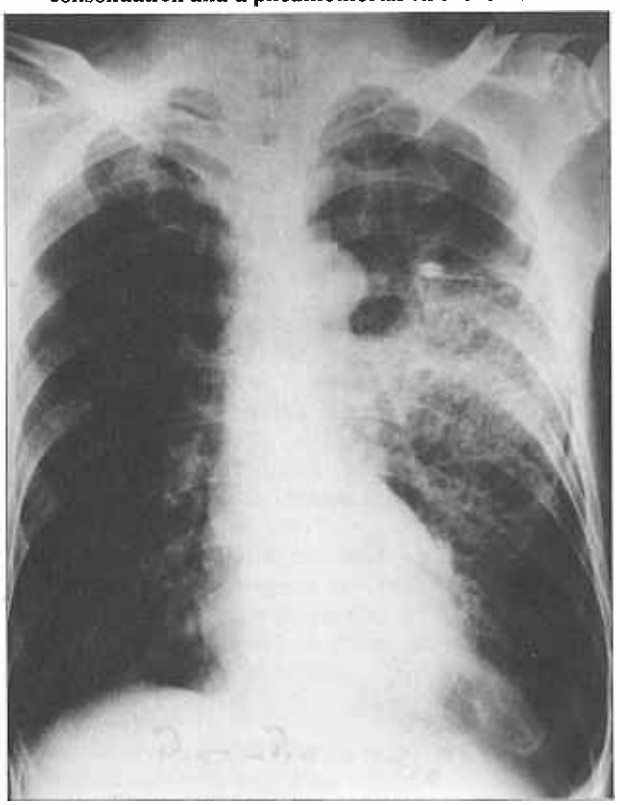
S C Poh, MBBS, MRCP(Edin), FRCP (Edin), FAMS
Senior Physician & Head

Correspondence to: Dr C B E Chee

A decision was made for an attempt at pleurodesis using 50% dextrose solution. Immediately after the intrapleural instillation of 50 ml of 50% dextrose, the patient experienced severe breathlessness followed by cardiovascular and respiratory collapse. Cardiopulmonary resuscitation with endotracheal intubation was carried out with return of spontaneous pulse, respiration and blood pressure.

After the resuscitation, it was noted that there were copious amounts of secretions from the endotracheal tube. The aspirate from the endotracheal tube was glucose, as shown by a dipstick. The post-resuscitation chest X-ray showed a small left pneumothorax and non-homogeneous patchy consolidation in the left midzone (Fig 1). The air leak persisted for another five days and then ceased spontaneously. Serial chest X-rays over the next six days showed re-expansion of the left lung and clearing of the patchy consolidation. The chest tube was removed eight days after the pleurodesis and the patient was discharged home well.

Fig 1 – Chest X-Ray showing non-homogeneous patchy consolidation and a pneumothorax on the left side.



The patient was reviewed regularly in the outpatient clinic over the ensuing ten months during which his clinical condition as well as lung function remained stable.

DISCUSSION

Studies involving animal models have demonstrated the destruction of lung parenchyma in rabbits by the intrapulmonary injection of trisodium citrate and acid-citrate-dextrose[3].This results in degeneration and necrosis of alveolar pneumocytes, and bronchial and bronchiolar epithelial cells and leads to the proliferation of fibroblasts which produce fibrous connective tissue followed by pulmonary fibrosis in one week. This is followed by the proliferation of the type II pneumocytes which produce and release large amounts of surfactant which permit the reopening of collapsed and adhesive air spaces causing the fibrous areas in the pathological lungs to become smaller and/or appear normal by four weeks.

The successful use of a 30% dextrose solution for chemical pleurodesis was first reported by Splengler in 1923. Early reports by Hennell et al in 1939 on chemical pleurodesis have advocated the use of 67% aqueous solution of dextrose in amounts varying from 50 ml to 60 ml[4]. The use of dextrose has been superseded in recent years by apparently more effective agents such as talc, quinacrine and tetracycline. The choice of dextrose over tetracycline as a sclerosing agent in our patient was influenced by the distressing pain experienced in our own series of patients[2] as well as in another report[1] when tetracycline was used.

A literature search failed to produce any report of dextrose-induced parenchymal lung damage in clinical practice. Our patient developed pneumonitis acutely with cardiovascular and respiratory collapse as a result of reflux of dextrose through the fistula into the lung tissue, as evidenced by the glucose-rich aspirate obtained from the endotracheal tube post-resuscitation. The resulting pulmonary oedema could have been caused by the high tonicity of the dextrose or by increased permeability since the 50% dextrose is highly irritating. The rapid onset of the symptoms within minutes of dextrose instillation would suggest a major role of osmotic pulmonary oedema in the initial period. It is likely that inflammatory permeability oedema also contributed subsequently, as the time course of radiological clearing (5 days) would suggest.

Fortunately, efforts at cardiopulmonary resuscitation were successful, and the patient did not suffer any long-term consequences of the collapse. The reflux of the dextrose through the pulmonary-pleural fistula, we believe, also induced local inflammation and fibrosis causing closure of the air leak within the next five days. There was complete radiological clearing of the pneumonitis within one week, and to date there has been no evidence of any progressive pulmonary fibrosis clinically, radiologically or on pulmonary function testing.

This case report illustrates a potentially acutely fatal complication of chemical pleurodesis for active pulmonary-pleural air leak.

REFERENCES

1 Stephenson LW. Treatment of pneumothorax with intrapleural tetracycline. Chest 1985; 5: 803-4.

2. Wang YT, Ng KY, Poh SC. Intrapleural tetracycline for spontaneous pneumothorax with persistent air leak. Singapore Med J 1988; 29: 72-3.

3 Mitsushashi T, Shimazaki M, Chanoki Y, Kuwahara H, Sakai T, Masuda H. Experimental pulmonary fibrosis induced by trisodium citrate and acid-citrate-dextrose. Exp Mol Pathol 1985; 42(2): 261-70.

4. Hennell H, Steinberg MF. Tense pneumothorax - treatment of chronic and recurrent forms by induction of chemical pleurodesis. Arch Intern Med 1939; 63: 648-63.