Re-expansion pulmonary oedema after evacuation of iatrogenic tension pneumothorax: a case report

S Goel, B Singh
Department of Anaesthesiology and Intensive Care, G.B. Pant Hospital, New Delhi, India

Abstract
A 20- year male, ASA physical status grade I, was operated on for a duodenal fistula. The intra- and postoperative periods were uneventful. 10 days later, the right internal jugular catheter was substituted with a triple lumen catheter in the right subclavian vein. Immediate chest X- ray revealed a right haemothorax. A chest drain was inserted immediately which was removed after the complete expansion of the lung. 24 hours later, the patient's general condition deteriorated, and he was clinically diagnosed to have a right-sided tension pneumothorax. A 'gush of air' after insertion of the chest drain confirmed the presence of a pneumothorax. After a few hours, the patient developed features of pulmonary oedema, and a diagnosis of re-expansion pulmonary oedema was made. The patient's general condition deteriorated rapidly, and he did not survive. The possible causes for his demise, and a review of the literature are both discussed.

Key Words: Re-expansion pulmonary oedema, Pneumothorax, Haemothorax

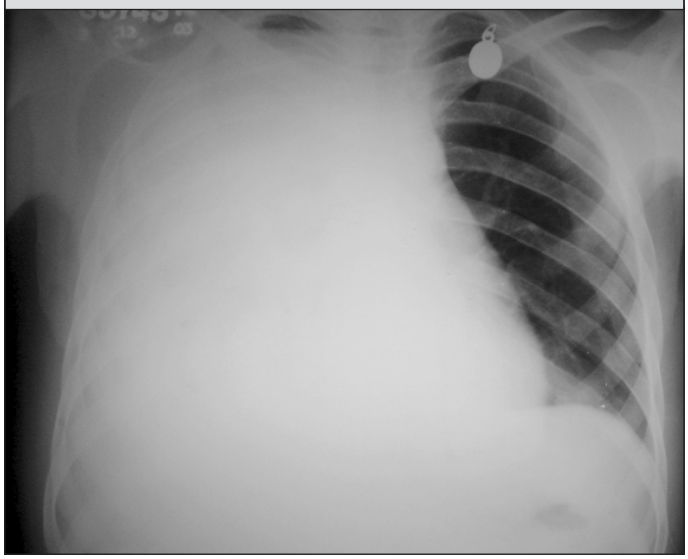
Introduction

Re-expansion pulmonary oedema (REPE) is a rare and potentially lethal complication following the placement of a chest drain to evacuate a large pneumothorax and pleural effusion. The majority of patients recover after a few days, but the mortality may be as high as 20%. REPE is generally unilateral but may also be bilateral. We report a case of bilateral REPE with rapid haemodynamic deterioration following evacuation of a tension pneumothorax.

Case history

A 20-yr-old 50 kg male with no known medical problem underwent repair of a duodenal fistula. The intraoperative period was uneventful. The right internal jugular vein (IJV) was cannulated intraoperatively for central venous pressure (CVP) monitoring and administration of total parenteral nutrition in the postoperative period. 10 days later, the IJV catheter was substituted with a triple lumen catheter in the right subclavian vein. One hour later, the patient complained of difficulty in breathing. On examination, the blood pressure (BP) was 90/60 mm Hg, heart rate (HR) 120 beats/ min and he had grossly diminished air entry on the right side of his chest. Immediate chest x-ray revealed a homogenous opacity in the right hemithorax (figure 1). A diagnosis of right haemothorax was made and a chest drain (28 FG) was inserted in the 4th intercostal space at the anterior axillary line under local anaesthesia. 750 mL of blood was drained through the chest drain over the following 4 hours, and 1500 mL of crystalloid and 350 mL of blood was transfused during this period. The patient's general condition improved and an x-ray of his chest two days later revealed a fully

Figure 1: Chest X-ray- showing homogenous opacity in the right hemithorax.



Correspondence:
Dr Sanjay Goel
Email- drsan_goel@yahoo.co.in

expanded lung (figure 2). The chest drain was removed on the 3rd day after insertion, when there was no further drainage. The wound was sealed and patient was moved to the ward. A day later, the patient again complained of respiratory difficulty with chest pain and was re-admitted to the intensive care unit (ICU). On examination, his BP was 80/46 mm Hg, HR 150 beats/min, respiratory rate (RR) 40/min, and grossly reduced air entry was found on the right side of his chest. An arterial blood gas (ABG) revealed a haemoglobin (Hb) of 8.5 gm%, with the pH 7.20, PaO_2 54, $PaCO_2$ 47 mmHg and SpO_2 79.5%. A chest x-ray was requested, but could not be done due to some technical reasons. A chest drain was therefore inserted immediately in the 4th intercostal space in the anterior axillary line. A ''gush of air'' was heard on entering the pleural cavity, and 50 mL of blood-tinged fluid drained via the chest drain into the water seal drainage system, without application of negative pressure. Over the next two hours, the patient's general condition and haemodynamic status continued to deteriorate: BP 74/48 mmHg, HR 170 beats/min, RR 40/min. The ABG revealed a Hb of 13.5 gm%, pH 7.20, PaO_2 47, $PaCO_2$ 45 mmHg and SpO_2 70%. On auscultation of the chest, coarse crepitations were present bilaterally, with distinctly more on the right side. The patient also produced copious pink frothy sputum. A diagnosis of re-expansion pulmonary oedema (REPE) was made. As the peripheral oxygen saturation did not improve and the patient also developed an altered sensorium, the trachea was intubated and controlled mechanical ventilation instituted, at a rate of 14/min, tidal volume of 500 mL with a positive end expiratory pressure (PEEP) of 5 cm of H_20 using 100% oxygen. A dopamine infusion of 5 μg/kg/min was also started, to improve the haemodynamics. Adrenaline was then added to maintain the systolic pressure above 100 mmHg. The patient was also given conventional medications for pulmonary oedema, such as morphine (10mg), frusemide (40mg) and of course 100% oxygen and PEEP. We were reluctant to increase the PEEP to more than 5 cm of H_2O due to the persistent hypotension. The patient's condition did not improve and the ABG revealed a Hb of 14.5 gm%, pH 7.30, PaO_2 54 and $PaCO_2$ 40 mmHg. Chest x-ray showed bilateral haziness (figure 3). Despite the maximal inotropic and full ventilatory support, the systolic BP did not rise above 80 mmHg. The patient suffered a cardiac arrest 4 hours later and could not be resuscitated.

Discussion

Amongst the various causes of pulmonary oedema[1], the diagnosis of REPE in our patient was made after the exclusion of other common causes such as hypoalbuminaemia, drug related causes, overtransfusion, septicaemia and cardiogenic pulmonary oedema. This patient was young with a normal cardiovascular system, was on jejunal feeding and had a good urine output. In addition, the overall clinical picture was suggestive of REPE.

REPE is a recognized complication of rapid evacuation of a large pneumothorax or a large pleural effusion.[2] It may also occur uncommonly following resection of a thoracic tumour[3] and after single lung ventilation.[4] Pavlin is of the opinion that it usually occurs after re-expansion of a collapsed lung that is three or more days old[5], but may develop in any patient with a collapsed lung, regardless of the duration of collapse (as in our case, where the pneumothorax was less than 24 hours old). Recently, however, it has been reported to occur unexpectedly, and either immediately or within 1 hour of re-expansion.[6] The exact incidence of REPE is not known but ranges between 0.9 % and 14 %.[7,8] Various mechanisms have been proposed, such as increased hydrostatic pressure from vascular flooding of the re-expanded lung as a result of negative intrapleural pressure, altered capillary permeability from hypoxic injury of the collapsed lung[9], and re-perfusion injury as reflected by increased inflammatory mediators.[10] On expansion and reintroduction of oxygen to the relatively hypoxic lung, oxygen derived free radicals are generated and are thought to damage the alveolar epithelial and endothelial cells, leading to increased vascular permeability. In addition, activated neutrophils may also provide a major source of free radicals after the re-expansion.[11] The development of oedema bilaterally in our patient supports the theory that inflammatory mediators are released during re-expansion and re-perfusion, since the contralateral lung could only have been affected by the humoral mechanisms.

The usual clinical presentation of the REPE is persistent spasmodic cough, chest tightness, and unresponsive hypoxaemia despite 100% oxygen administration. Recovery usually occurs if the patient survives the next 48 to 72 hours.[12] However, the outcome may be fatal in up to 20% of the cases, despite aggressive treatment as in our patient.[6] Patients with REPE are reported to be hypovolaemic due to rapid pooling of fluid within

Figure 2: Chest X- ray- showing right-sided fully expanded lung with chest drain in situ.

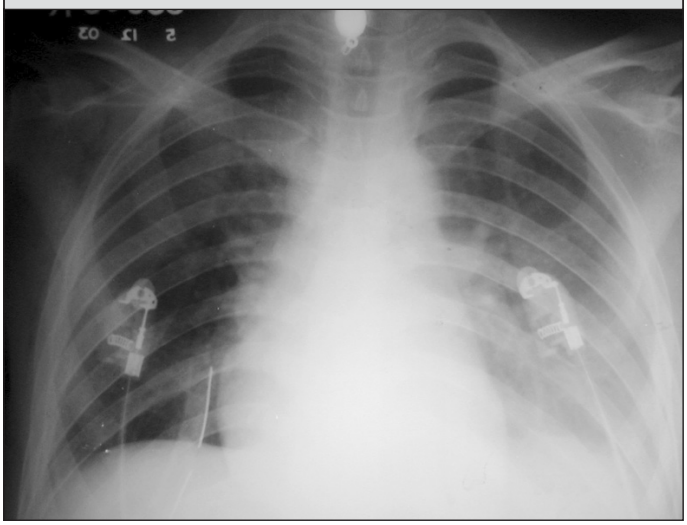
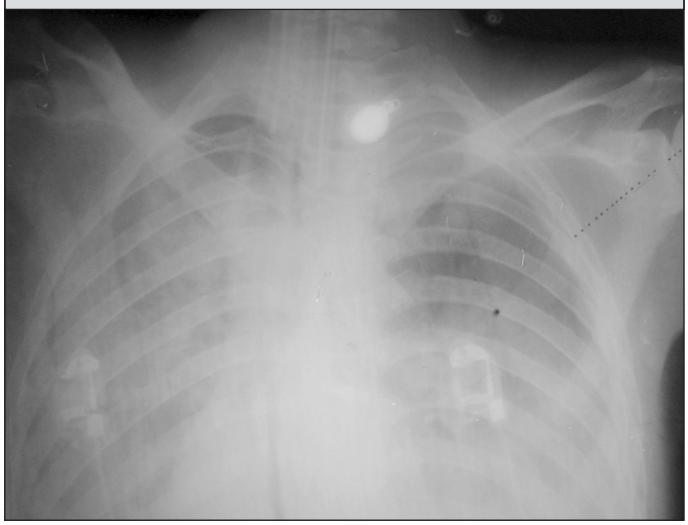


Figure 3: Chest X- ray- showing bilateral haziness (right side> left side)



the thorax following pulmonary re-expansion, and pre-existing volume depletion leads to hypotension and shock.[13] Hypovolaemia may be further compounded by myocardial depression secondary to hypoxaemia and hypotension. Thus vigorous fluid therapy in such patients may be advantageous in preserving optimal circulatory dynamics despite the presence of pulmonary oedema. The authors feel that the persistent deterioration in the haemodynamic status in this patient could have been due to the inadequate administration of fluids compounded by an already reduced circulatory volume. In addition, myocardial function could have been depressed by various mechanisms, such as the use of vasopressors that increase left ventricular afterload, an increase in the blood viscosity due to hypovolaemia (Hb increased from 8.5 gm % to 14.5 % in our case), severe tachycardia, and positive pressure ventilation with PEEP. Retrospectively, we feel that the CVP monitoring in this patient could have guided us to more precise administration of fluids. However, stabilizing the patient was the first priority at the time, and because of the complication of the central venous cannulation earlier, we were conservative about securing another central venous line. Establishing a central line by cannulating the basilic vein on both sides was attempted, but was unsuccessful.

It has been observed that the development of REPE is related to three factors; longer duration and greater size of the pneumothorax, and a rapid rate of re-expansion. However, controlling one of these factors may not prevent its development if the other two are present. Furthermore, young patients (<40 years) have been observed to be at greater risk for developing REPE.[7] Pneumothorax was not of long duration (< 24 hours) in our patient, although the presence of the other two factors (besides the young age) seem to have contributed to the development of REPE. It can also develop suddenly after chest drain insertion, particularly, if negative pressure is applied.[6]

It is difficult to explain as to how a tension pneumothorax developed after the removal of the chest drain in this case. Improper sealing of the wound after removal of the chest drain leading to sucking in of air is unlikely to cause tension pneumothorax. X-ray after removal of the chest drain was not done. This may have alerted us earlier to the presence of the pneumothorax. Another theory is that it could have occurred as a result of pleural or lung parenchymal damage, which might have been masked by the presence of the massive haemothorax immediately after subclavian vein cannulation. However, the leak from the collapsed lung stopped but when the lung re-expanded following evacuation of the haemothorax, it is possible that the air leak opened up again. Since the site of the chest drain insertion was properly sealed, the continued leak in valvular fashion from the parenchymal damage may have led to the development of the tension pneumothorax.

Haemothorax, as a result of right subclavian vein cannulation is rare, but has been reported.[14,15] Generally, it occurs with inexperienced operators (i.e. staff who have performed less than 12 supervised cannulations) and using wrong technique.[16] In the present case, the resident was experienced, but encountered difficulty while pulling out the J wire from the catheter. Although it is difficult to explain the exact cause of the pneumo- and haemothorax the following possibilities can be considered. Firstly, the pleura might have been damaged while inserting the cannula. Secondly the cannula could have transfixed the subclavian vein while passing the 'J' wire, thus once the catheter was inserted in the vein, there was difficulty in taking out the 'J' wire. This could have created a tear in a vessel that led to the development of a haemothorax. In addition, there has been growing concern regarding major vessel injury due to overinsertion of the dilator.[17] As the haemothorax developed very rapidly, the possibility of subclavian artery damage cannot be ruled out. Autopsy in this case was necessary to determine the exact cause but was refused by the relatives.

In conclusion, bilateral REPE can occur after the management of a large pneumothorax or pleural effusion. It may be further complicated with a severe degree of cardiovascular instability and impaired organ perfusion. Furthermore, a large volume infusion guided by central venous pressure monitoring, minimizes the complications arising from a hypovolaemic state.

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