Clinical Case Reports

Severe localized re-expansion pulmonary oedema: An unusual instance

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ABSTRACT

A lobar re-expansion pulmonary oedema (REPO) after pleural drainage procedure is rare and usually asymptomatic. We report a 56-year-old man with severe left lower lobar REPO after tube thoracostomy drainage of a loculated hydropneumothorax of 2 days' duration with underlying chronic obstructive pulmonary disease. The clinical manifestations were immediate and disproportionate to the radiological extent of REPO. The severity of lobar REPO was probably related to the pre-existing emphysematous changes and airway obstruction. Supplemental oxygen and intermittent pleural drainage led to clinical recovery within 24 hours, followed by radiological clearance of alveolar opacities within 3 days.

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INTRODUCTIONS

Re-expansion pulmonary oedema (REPO) is a known complication of rapid re-expansion of a collapsed lung, most often after drainage of a pleural disease such as pneumothorax, pleural effusion or haemothorax. REPO following drainage of pneumothorax occurs within one hour in a majority of patients and usually involves the whole re-expanded lung.[1] A clinically important lobar REPO is rare and is often detected as an incidental finding through imaging such as a chest computed tomography (CT).[2] We report a severe lobar REPO following tube thoracostomy drainage of a loculated hydropneumothorax of 2 days' duration in a patient with chronic obstructive pulmonary disease (COPD).

THE CASE

A 56-year-old man, ex-smoker, with COPD was admitted for sudden onset left-sided pleuritic chest pain and shortness of breath for 2 days. The patient was on dried powder inhaled therapy with ipratropium bromide and levosalbutamol. Physical examination including vital parameters were unremarkable. Respiratory system examination showed central trachea and decreased breath sounds in the left hemithorax. Laboratory reports of complete blood count, room air arterial blood gas analysis, blood biochemistry and viral serology were non-contributory. A posteroanterior chest X-ray (Fig. 1a) showed left-sided loculated air and fluid and CT scan of the chest (Fig. 1b) in addition showed apical bullae. A 2D transthoracic echocardiogram showed mild pulmonary arterial hypertension and normal left ventricular function. A trocar-guided chest tube (28F) was inserted through the fifth intercostal space in the left midaxillary line and connected to a disposable plastic underwater seal drainage system. Immediately after insertion of the chest tube, the patient had severe cough, tachypnoea and breathlessness. The blood pressure was 96/60 mmHg and the room air oxygen saturation decreased to 86%. The oxygen saturation and the symptoms improved quickly with supplemental oxygen and after clamping the drainage tube for 30 minutes. After the chest tube was clamped, the patient was instructed to apply pressure around the tube thoracostomy wound with fingers if there was cough to prevent the escape of air into the subcutaneous tissue due to high intrathoracic pressure generated during coughing.

Intermittent pleural drainage and supplemental oxygen were continued for about 24 hours till the patient became asymptomatic. The patient also received nebulized levosalbutamol, ipratropium bromide and intravenous hydrocortisone. The pleural fluid reports did not contribute. Post-tube thoracostomy chest X-ray (Fig. 1c) showed re-expanded left lung with alveolar infiltrates in the lower lobe. The chest X-ray (Fig. 1d) on day 3 showed clearance of alveolar opacities in the re-expanded left lung establishing the diagnosis of unilateral lobar REPO. Two days later, the chest tube was removed, and the patient was discharged. The patient remained stable on inhaled therapy for COPD without recurrence of pneumothorax during the following 6 months.

DISCUSSION

The first report of REPO localized to the right middle and lower lobes following drainage of a pneumothorax was published in 1979 and was believed to be due to occlusion of bronchus intermedius.[3] Later, right upper lobar REPO following drainage of a pneumothorax was reported as an incidental radiological finding and was attributed to total collapse of the upper lobe compared to other lobes of the lung.[4] The exact cause of REPO whether lobar or multilobar is unclear but is related to the degree of collapse of the lung, speed of re-expansion of the collapsed lung on the affected side and the duration of collapse, which may be a major contributing factor.[5] The risk of REPO is more when a portion of the lung remains collapsed and structures within the lung parenchyma get thickened and become stiff. Due to the decreased flexibility, the integrity of the alveolar–capillary membrane may be lost and pulmonary blood vessels may get damaged when stretched suddenly during re-expansion of the lung resulting in increased vascular endothelial permeability.[6] When blood flow returns to the injured pulmonary blood vessels, oxygen-derived free radicals are produced and induce migration of leucocytes into the lung and both these events may cause further damage to the pulmonary vasculature

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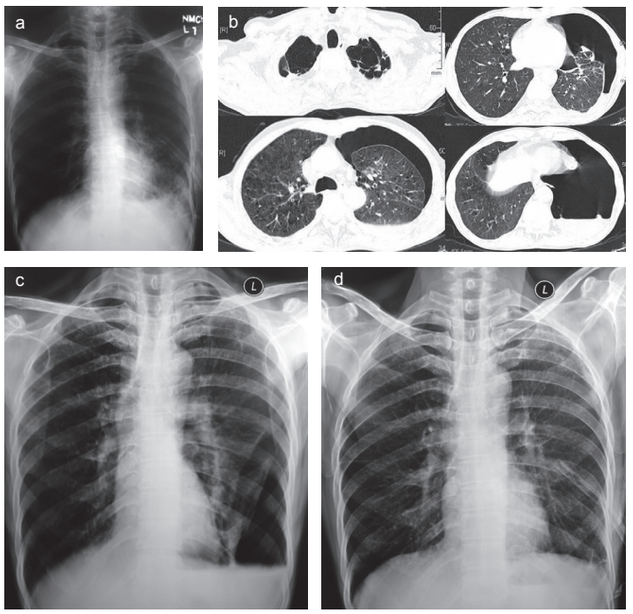

FIG 1. (a) Posteroanterior chest X-ray showing left-sided loculated air and fluid; (b) sections of computed tomography scan of the chest showing apical bullae in addition to loculated air and fluid; (c) post-tube thoracostomy chest X-ray showing alveolar infiltrates in the lower lobe of the re-expanded left lung and (d) post-tube thoracostomy chest X-ray on day 3 showing resolution of alveolar infiltrates in the re-expanded left lung

leading to permeability oedema. Other factors such as airway obstruction, excessive negative intrapleural pressure, decreased surfactant activity and pulmonary arterial pressure change may play a role in the formation of REPO.[7] In our patient, the lobar REPO was probably due to complete collapse of the left lower lobe within the loculated hydropneumothorax and pre-existing emphysematous changes and airway obstruction might have caused severe clinical manifestations. Breathlessness, tachypnoea and oxygen desaturation are the most frequent clinical manifestations of REPO followed by cough with or without sputum.[8] Our patient had all these features except that there was no sputum. There are no specific chest X-ray or CT features of REPO, but the development of unilateral diffuse or localized alveolar opacities in the re-expanded lung within 24 hours of a pleural drainage procedure is considered as diagnostic once other causes such as cardiogenic pulmonary oedema, pneumonia or pulmonary haemorrhage are excluded.

REPO whether lobar or multilobar is usually self-limiting and resolves in 24–72 hours. Supplemental oxygen, haemodynamic support and ventilatory support are the treatment options depending on the severity of REPO. Keeping the patient in the lateral decubitus position with the affected side up may reduce the intrapulmonary shunt.[9] Once the patient becomes breathless, relief may be obtained after stopping the pleural drainage for 15–20 minutes; otherwise, full-blown REPO may develop.[10] In our patient, early clamping of the chest tube followed by intermittent drainage might have helped limit the progress of REPO.

To conclude, a lobar REPO following drainage of a loculated pleural collection of air and fluid with severe clinical manifestations is rare. The predisposing factors may be a total collapse of a lung lobe and concomitant respiratory diseases such as COPD.

Conflicts of interest. None declared

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