**A spontaneous massive pleural effusion**

K Ongeti and J Ogeng’o

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**Abstract**

A 62-year-old male presented with a progressing 3-week history of respiratory distress, tachypnoea, right-sided chest stony dullness, and mediastinal shift to the left. He had no clinical, laboratory, or radiological evidence of pulmonary tuberculosis or malignancy and could not remember any history of chest trauma. Chest X-ray revealed massive right-side pleural effusion. A computerised tomography (CT) scan showed six consecutive rib (ribs 5–10) fractures with no callus formation. Chest tube insertion drained 4.7 L of straw-coloured effusion that did not recur subsequently. We suspect that multiple rib fractures irritated the pleura, resulting in a massive pleural effusion. A review of the literature indicates this to be a rare finding.

**Introduction**

Exudative pleural effusions often present a diagnostic challenge. The most frequent causes are tuberculosis (TB), malignancy, paramalignant effusion, pneumonia, empyema, and general infection, with only a few cases being related to trauma. Using a systematic approach based on the patient’s history, physical examination, routine laboratory studies, and chest roentgenograms, the clinician will accurately establish the presence and location of pleural effusion in the majority of cases. We present a case of a massive pleural effusion resulting from unnoticed rib fractures that constituted a diagnostic challenge.

**Case presentation**

Patient T M, a 62-year-old male, came to our hospital complaining of chest pain, cough, and difficulty in breathing for 3 weeks. He was a known hypertensive, drug compliant and controlled. Prior to admission he had sought help from two other hospitals with the same problem. He had been consistently treated using co-amoxiclav antibiotics. He was not a smoker and had never been treated for pulmonary TB. There was no history of chest trauma. He was dehydrated and had reduced breath sounds on the right lung fields with stony dullness. Other systems were normal. The chest radiograph showed a right-sided pleural effusion (see Figure 1). He had an erythrocyte sedimentation rate (ESR) of 4 mm/hour, the full haemogram showing a mild elevation of white blood cells at $11 \times 10^9/L$ with a normal peripheral blood film. The electrocardiogram (ECG), electrolytes, creatinine, and urea were normal. The patient was started on antituberculosis drugs and continued on co-amoxiclav. TB was ruled out and the antituberculosis drugs were stopped 2 days later when all sputum samples were negative for AAFB (alcohol and acid fast bacilli). The patient was discharged 3 days later only to be readmitted 2 weeks later in severe respiratory distress, with tachypnoea and right-sided stony dullness with the mediastinum displaced to the left (see Figure 2). The repeat chest radiograph showed a massive right-sided pleural effusion obliterating the right lung field. An intercostal chest drainage tube was inserted. It drained 4.7 L of straw-coloured pleural fluid in a single...
instance. The fluid had 6.3 mmol/L glucose, microprotein of > 400 mg/dL, 2-3 pus cells, 5–10 red blood cell (RBCs), there were no bacteria on Gram stain, the Zeihl Nielsen (ZN) for AAFB was negative. The repeat ESR was 46 mm/hour. The white blood cells were elevated at 14 × 10⁹ cells/L.

Chest and abdominal CT scans were done with i.v. and oral contrast after the pleural fluid was drained (see Figures 3 and 4). The abdominal region was normal. There was residual right pleural effusion with the chest tube in situ. The right lung had partial atelectatic changes. The pleura did not have any significant thickening, neither did it show any nodules or septations. The mediastinum tracheobronchial tree, hilar regions and left lung were normal. There were no mass lesions or adenopathy in the chest. Six sites of mildly displaced linear fractures were noted on the ribs on the right side. There were no fractures on the left. The fractures had mild sclerosis without callus formation (see Figure 5). The thoracic and lumbar spines were normal. There was no evidence of a neoplastic cause of the pleural effusion.

The patient was followed up a month after the chest drain had been removed. He still had a small pleural effusion on the same side.

Discussion
Observations from this patient suggest that the effusion was related to irritation by the rib fractures. Pertinent observations of the present case in support of the suggestion, however, are that the CT scan confirmed the fractured ribs; the effusion was exudative and lymphocytic in nature. What is surprising is that this patient does not recall having any chest trauma. Our patient did not have a haemothorax, as confirmed by the straw-coloured fluid that was drained as well as the low number of RBCs in the pleural aspirate. The fluid equally took about 3 months to accumulate. A possible explanation is that the trauma had occurred much earlier and he could not just remember. Indeed in literature, pleural effusion has been diagnosed 3 years after rib fracture. In cases of delayed pleural effusion, patients could have recurrent lymphocytic exudative effusion. This rare entity is probably related to sub pleural trauma, and may recur. As in the present study, it can be treated with pleural drainage. The cause of the rib fracture is classified as benign because the bones did not have any malignant changes.

In conclusion, multiple rib fractures should be considered as rare causes of exudative pleural effusions, especially in the elderly.

References