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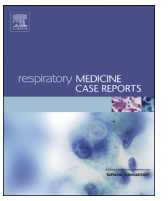

Case Report

Tension hydrothorax in a patient with a history of pulmonary tuberculosis

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ABSTRACT

A tension hydrothorax is a massive pleural effusion that leads to hemodynamic instability. Here we present a case of tension hydrothorax secondary to poorly differentiated carcinoma. A 74-year-old male smoker presented after a one-week history of dyspnea and unintentional weight loss. Physical exam demonstrated tachycardia, tachypnea, and decreased breath sounds diffusely over the right lung. Imaging revealed a massive pleural effusion causing mass effect on the mediastinum with tension physiology. Chest tube placement revealed an exudative effusion with negative cultures and cytology. Pleural biopsy revealed atypical epithelioid cells consistent with poorly differentiated carcinoma.

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Author participation

Authors JS, LR, JJ, and AA all participated equally in the creation of this manuscript and have made substantial contributions to this work. All authors participated directly in patient care.

1. Background

A pleural effusion is the accumulation of fluid between the parietal and visceral pleural, known as the pleural cavity. It is estimated that about 1.5 million patients in the US suffer a pleural effusion, most commonly from congestive heart failure, pneumonia, and cancer [1]. They are commonly associated with dyspnea, chest discomfort, and potential respiratory compromise. However, in large effusions, the increased intrapleural pressure can be transmitted to the pericardial space, which can lead to hemodynamic instability and impaired cardiac filling [2,3]. This is known as a tension hydrothorax, a massive pleural effusion resulting in hemodynamic compromise secondary to mediastinal compression [4]. This phenomenon is rare in the setting of malignancy and tuberculosis infection [4–6]. We present an interesting teaching case of a patient with a history of pulmonary tuberculosis that presented to our institution with a pleural effusion of unclear etiology with tension physiology.

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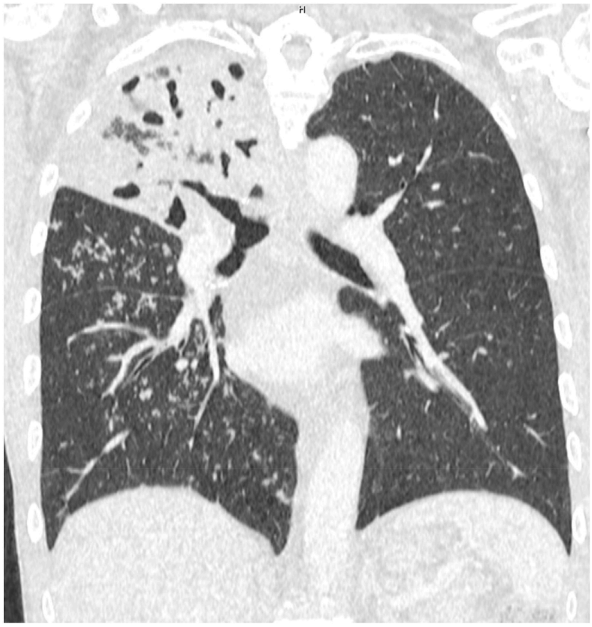

Fig. 1. Computed tomography of the chest (coronal view) showing a right upper lobe cavitation and endobronchial spread of pulmonary tuberculosis diagnosis, 7 years prior to presentation.

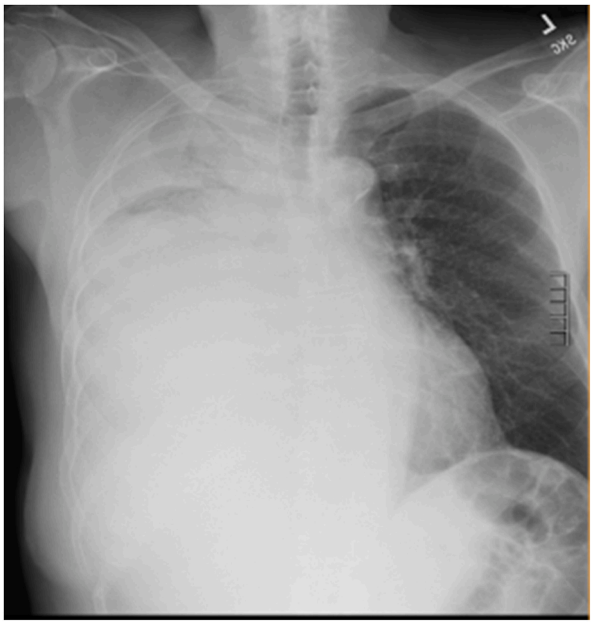

Fig. 2. Chest x-ray demonstrating a large, right lung 'white out' consistent with tension hydrothorax with tracheal deviation and mediastinal shift.

2. Case description

A 74-year-old male with a history of tuberculosis with known cavitary lesion (Fig. 1) and 75 pack-year smoking history presented to a local emergency department with a one-week history of dyspnea on exertion and unintentional weight loss. He was afebrile with sinus tachycardia at 120 beats per minute, respiratory rate of 22 respirations per minute, blood pressure of 166/89 mmHg, and oxygen saturation of 94% on room air. Physical exam revealed scant expiratory wheezing on the left lung field and markedly diminished breath sounds and dullness to percussion on the right posterior thorax. A chest x-ray (CXR) was notable for complete opacification of the right hemithorax (Fig. 2). Computed tomography (CT) of the chest revealed mass effect on right atrium and right paratracheal lymphadenopathy. Point of care ultrasound (POCUS) prior to 14-Fr tube thoracostomy revealed right atrial and ventricular collapse during cardiac cycle and a large volume complex-homogenous pleural effusion with inverted right diaphragm (Figs. 3 and 4A, Suppl 1,2). During the procedure, 3500 mL of bloody-appearing fluid was drained and repeat POCUS revealed normal lung volumes and di-

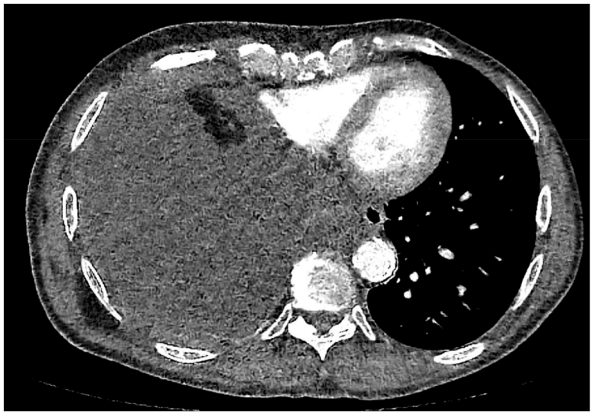

Fig. 3. Computed tomography of the chest (axial view) showing mediastinal shift with right atrial compression.

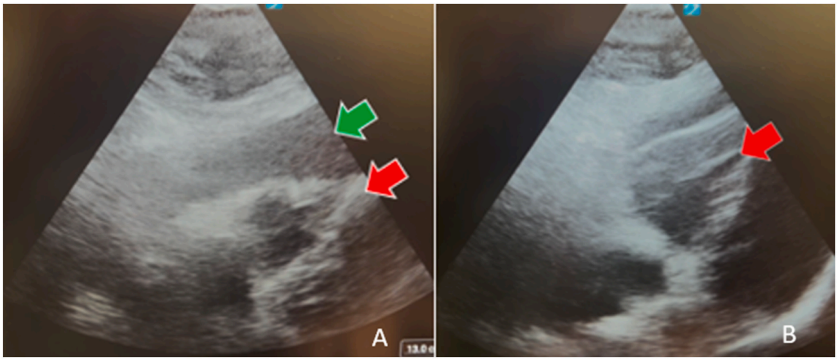

Fig. 4. A; bedside ultrasonography showing homogenously complex pleural effusion (green) compressing the right ventricle (red). B; bedside ultrasonography demonstrating the right ventricle (red) post thoracostomy tube placement. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

aphragm function post-procedure (Suppl 3). Additionally, point of care cardiac ultrasonography immediately post-drainage demonstrated normalization of the pleural fluid compromising right ventricular function (Fig. 4B, Suppl 4). Pleural fluid analysis demonstrated pH 7.34, Glucose 49, lactate dehydrogenase (LDH) 1050 U/L (serum 203), total protein 3.6 g/dL (serum 5.9), white blood count 570 (lymphocytes 82, monocytes 18), adenosine deaminase (ADA) 14 U/L, and cultures with no growth. Sputum acid-fast bacilli (AFB) cultures were negative. Repeat chest CT revealed right sided pleural thickening and nodularity with interstitial septal thickening, as well as paratracheal and periaortic lymphadenopathy.

Pleural cytology was unremarkable, and with an unclear source of the effusion and concern for malignancy, thoracoscopy with right pleural and chest wall mass biopsy was completed. Surgical pathology revealed atypical epithelioid cells consistent with poorly differentiated carcinoma. It was negative for mesothelioma markers calretinin, CK5/6, WT1, D2-40, Germ cell tumor marker SALL4, and other organ or origin specific markers including GATA4, PAX8, TTF-1, Napsin A, NKX3.1, synaptophysin, chromogranin, CK20, and mucicarmine. The patient is currently undergoing further work-up with NextGen sequencing and plans for palliative chemotherapy/immunotherapy.

Supplementary video related to this article can be found at https://doi.org/10.1016/j.rmcr.2023.101868

3. Discussion

Our case highlights the uncommon occurrence of tension hydrothorax in the setting of malignancy, particularly in a patient with known history of tuberculosis. Pleural effusions occur as a result of increased fluid production and/or decreased reabsorption in the pleural space. Normal rate of pleural fluid production is approximately 0.3 mL/kg and lymphatic resorption is approximately 0.28 mL/kg/hr [7]. Tension hydrothorax is a massive pleural effusion resulting in mediastinal compression, which can impair diastolic filling and cardiac output. The pathophysiology of a tension hydrothorax mimics a cardiac tamponade; therefore, early detection and intervention is imperative to prevent hemodynamic collapse. Treatment involves immediate thoracentesis or tube thoracostomy, with a maximum of 1500 mL drained within the first hour to prevent re-expansion pulmonary edema [4].

Exudative pleural effusions are defined by Light's Criteria as a pleural protein to serum protein ratio greater than 0.5, pleural LDH to serum LDH ratio greater than 0.6, and pleural LDH greater than ⅔ the upper limit of normal. Tuberculosis and malignancy are common causes of exudative pleural effusions, and they have similar biochemical markers on pleural fluid analysis, including elevated lymphocytes and low glucose levels. Adenosine deaminase, a marker of activated T lymphocyte activity, can be used to differentiate

tuberculosis (ADA >30–35 U/L) and malignant pleural effusions (ADA <40 U/L). In adults, tuberculosis effusions most often occur during primary disease. These tend to be smaller, occupying 2/3 or less of the hemithorax and slightly favor the right side [8]. Pleural fluid analysis typically demonstrates a lymphocyte-predominance, protein concentration >3.0 g/dL, elevated LDH, pH < 7.40, glcose between 60 and 100 mg/dL, and lymphocyte-to-neutrophil ratio >.75, which can be used to make a presumptive diagnosis with negative AFB smear and culture [1]. In terms of thoracic ultrasound findings of a tuberculous effusion, it may demonstrate a thickened pleural. In our patient, his complex homogenous effusion indicated an exudative effusion pre-pleural drainage [9].

Malignant pleural effusions develop when malignant cells infiltrate the pleural space disrupting lymphatic drainage. While not all effusions with lung cancer are malignant, up to 15% of patients will have a malignant effusion [10]. Cytology should also be performed if etiology is unknown.

Cytology has a sensitivity of 60% in malignant pleural effusions thus, pleural biopsy may be warranted to establish diagnosis. A malignant effusion will preclude curative resection, surgical thoracoscopy should follow negative cytology to further evaluate the pleural space [11]. Overall, this case highlights the diagnostic findings of a tension hydrothorax and the clinical approach to a massive effusion of unclear etiology.

Declaration of competing interest

Dear Editors for Respiratory Medicine Case Reports, On behalf of all authors, the corresponding author states that there are no conflicts of interest. This manuscript has not been submitted elsewhere, is not under review by another journal, and has not been published previously. The authors have read the manuscript and approve its submission. None of the authors have any conflict of interests or potential for financial gain associated with the publication of this manuscript. All authors had access to the data and had a role in writing the manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2023.101868.

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