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Internal Medicine Section

Splenic Haematoma as a Rare Cause of Pleural Effusion: A Case Report

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ABSTRACT

Pleural effusion has various aetiologies, mostly located either in the lung parenchyma or in the pleura. Subphrenic causes, leading to exudative pleural effusion are uncommon. Authors hereby, presented a case of a 50-year-old female with recurrent left sided pleural effusion. The effusion was haemorrhagic, exudative with low Adenosine Deaminase (ADA), and no malignant cells on cytology. Contrast Enhanced Computed Tomography (CECT) thorax and upper abdomen showed an incidental splenic haematoma. With conservative treatment, splenic haematoma and adjoining pleural effusion resolved completely. Thus, this case highlights splenic haematoma as a rare but important cause of pleural effusion.

Keywords: Exudative, Pleural effusion, Splenic haematoma, Splenic injury

CASE REPORT

A 50-year-old non smoker female came to Pulmonary Medicine Outpatient Department (OPD) with left-sided chest pain, fever, and progressive dyspnoea since 15 days. The pain was sudden in onset and dull in nature which was present over the lower chest radiating to back, and was not associated with palpitations or sweating. The fever was continuous in nature and accompanied by rigor and chills. About 14 years ago, the patient had been treated for right sided tubercular pleural effusion.

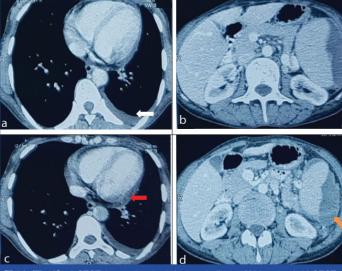
On chest examination, breath sounds were diminished in intensity over the left lower lung field. There was mild tenderness seen in the left hypochondrium that worsened on inspiration. Chest roentgenogram (Chest X-ray) was done that was suggestive of left sided pleural effusion [Table/Fig-1]. Routine haematological tests showed anaemia (haemoglobin was 6.9 g/dL, haematocrit was 21%) and leucocytosis (total leucocyte count were 17.7×10⁹/L). Ultrasound guided diagnostic thoracocentesis demonstrated a haemorrhagic

pleural fluid (haematocrit was 8.3%), that was exudative (proteins

[Table/Fig-1]: Chest X-ray showing the initial left pleural effusion (white arrow).

was 4.7 g/dL) with low Adenosine Deaminase (ADA) (30.18 IU/L) and mixed cellularity (neutrophils was 38%, mesothelial cells was 22%, mononuclear cells was 40%, no malignant cells). Routine bacterial and fungal cultures were sterile. Serum amylase (20 IU/L) and lipase levels (13 IU/L) were within normal limits that excluded pancreatitis as a possible aetiology. D-dimer and bilateral compression ultrasound were done to screen pulmonary thromboembolism that were both normal.

The patient was started on an empirical course of antibiotics followed by therapeutic thoracocentesis however, there was no improvement in pain or dyspnoea. Then, repeat pleural fluid investigations were done, that were again inconclusive. Thereafter, the patient underwent CECT thorax and upper abdomen that showed small left pleural effusion, underlying passive atelectasis, small to moderate pericardial effusion along with multiple splenic lacerations (grade III as per American Association for the Surgery of Trauma (AAST)}, subcapsular haematoma, and mild haemoperitoneum [Table/Fig-2a-d] [1].



[Table/Fig-2]: a) CECT thorax showing left pleural effusion (White arrow); b) CECT Abdomen showing subcapsular splenic haematoma and splenic laceration; c) CECT thorax showing left pleural effusion, mild pericardial effusion (Red arrow); d) CECT Abdomen showing subcapsular splenic haematoma and splenic laceration (Orange arrow).

On retrospective questioning, patient admitted to have a history of fall over the bricks, three to four days before the initiation of symptoms which caused splenic haematoma. With a diagnosis of splenic haematoma, mild haemoperitoneum with left pleural effusion, consultation for surgery was taken, where the patient

was advised conservative management with observation, tablet Clopidogrel 75 mg OD and symptomatic treatment. Patient had an uneventful course and was discharged after three days. On OPD follow-up after seven days, patient showed gradual improvement in her symptoms. Chest X-ray (CXR) was repeated after 14 days that showed marked improvement [Table/Fig-3].



[Table/Fig-3]: Follow-up Chest X-ray after 14 days, showing marked improvement (Black arrow).

DISCUSSION

Pleural effusion is a common manifestation of many pulmonary and extrapulmonary diseases. Most of the aetiologies have pathogenetic mechanisms operating in the lung or pleura itself. Unilateral pleural effusions are usually exudative in nature and commonly seen in infections like tuberculosis, various types of pneumonia and carcinomas. Other, less common causes includes pulmonary infarction, thromboembolism, collagen vascular diseases, and subphrenic abscess [2]. Physicians rarely try to look for abdominal causes in exudative effusions, except when are ruling out with metastasis or dissemination of tuberculosis. Splenic haematoma is an uncommon cause of pleural effusion and is rarely documented in the literature [3,4].

In 1980, Koehler PR and Jones R reported three cases of subcapsular splenic haematoma secondary to trauma that was associated with left sided pleural effusions. The diagnosis in these cases was delayed because the search centered around common intrathoracic causes of pleural effusion. The effusions responded poorly to thoracentesis and cleared only after splenectomy [5]. Similarly, in present case significant time was lost as patient denied the history of trauma and our differentials were centered around malignancy and tuberculosis in the first place. Also, in our case patient did not respond to therapeutic thoracocentesis. However, it was later when CECT was done that authors encountered splenic haematoma as the cause of effusion. Koehler PR and Jones R in their case series, could not document the mechanism behind the effusions. The reason why a pleural effusion was associated with the splenic trauma was thought to be caused by a sympathetic reaction, or associated trauma to the diaphragm, pleura, chest wall or the lung [5]. There has been a case report by Suliman I et al., relating splenic injury and pleural effusion [6]. Also, a study by Kulaylat AN et.al, found an incidence of pleural effusion in 4.4% of paediatric population with blunt splenic injury thus proving it to be an important cause [7].

In lymphatic anatomy, the costal pleura drains into the internal mammary and intercostal lymphatic system and the visceral pleura drains into the mediastinal lymph nodes and there are unidirectional transdiaphragmatic vessels which descends and drain the posterior thorax, receiving tributaries from the diaphragm and splenic mesentery [8,9]. Thus, as per this basic lymphatic anatomy, the exudative effusion found in our patient could be explained by a combination of: (1) direct compression of the posterior lymphatics by the enlarged infarcted spleen; and (2) filtration of haemorrhagic splenic fluid into the pleural space due to increased permeability caused by perisplenic inflammation. The CECT in the present patient demonstrated haemoperitoneum that reinforces the above theory.

The important differential diagnosis to suspect in unilateral haemorrhagic pleural effusions include malignancy, trauma, tuberculosis, pulmonary embolism, pancreatitis, postcardiac injury syndrome, aortic dissection and aneurysm. Usually, pleural fluid investigations supplemented by imaging studies are helpful in narrowing down the diagnosis. However, splenic haematoma as a cause of pleural effusion can only be confirmed, if we suspect and do a targeted imaging study. Previously, angiography was used for the diagnosis of subcapsular splenic haematoma, but, currently non invasive techniques such as CT scanning can easily identify such injury [10].

The treatment of pleural effusion secondary to splenic injury involves the management of the underlying injury. The management is done as per the AAST [1]. Immediate surgery for abdominal exploration may be the only and immediate treatment option, especially in a haemodynamically unstable patient in order to control the active bleeding. In haemodynamically stable patients, any injury in the presence of a shattered spleen, splenic vascular injury, active bleeding confined within the splenic capsule or parenchymal laceration involving segmental or hilar vessels (producing >25% devascularisation) are managed by angiography and embolisation, whereas the rest of the splenic injuries are managed conservatively [1].

Timely abdominal imaging is useful in suspected cases to confirm the diagnosis. With the increasing incidence of spleen sparing treatment options for splenic injury, symptomatic pleural effusion may become more common in the future. Being that reaccumulation has been described in the literature, these patients need close follow-up after standard management and investigation for pleural effusion [6].

CONCLUSION(S)

The present case highlights splenic haematoma as an important yet unsuspected cause of left sided haemorrhagic pleural effusion. Careful history for possible injury should be inquired from all the patients with repeated or persistent left sided pleural effusions, followed by targeted imaging studies.

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