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Fatal diffuse pleural calcification due to Tuberculosis- An unexplained entity

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ABSTRACT

Diffuse pleural thickening has many causes and often need to be diagnosed early as delay in treatment can be lethal. Diffuse pleural thickening can be due to calcifications and may occur as a result of chronic infections including Tuberculous effusion. Primary pleural calcification due to Tuberculosis is extremely rare. A 28 year old patient was presented with exertional dyspnea, chronic cough and pleuritic type chest pain for a period of 4 months. CXR showed left sided diffuse pleural calcifications and ultrasonography showed calcified pleura with thick echogenic material suggestive of an empyema. Further evaluation with a CECT showed left sided diffusely calcified, septated pleura with empyema and contralateral early pleural and peritoneal calcification. Pleural aspiration showed a hemorrhagic exudative lymphocytic effusion with high ADA titer. Cytology did not reveal malignant cells. His serum calcium level was normal. He was diagnosed to have extra-pulmonary tuberculosis and was treated with standard anti TB treatment with an intercostal tube drainage. Despite TB treatment he passed away due to respiratory distress caused by pleural thickening. This case highlights the importance of timely initiation of anti TB treatment and the capacity of Tuberculosis to cause diffuse pleural calcification which can be fatal in an untreated setting.

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Introduction

Tuberculosis accounts for 30-80% of the pleural effusions in developing countries and is associated with lethal outcome in an untreated setting[1]. Generally it causes a unilateral straw colored effusion which can be hemorrhagic in certain situations. Long term untreated plural effusion can get complicated with empyema causing pleural calcifications which is extremely rare. Diffuse pleural calcification caused by Tuberculosis is always a diagnostic challenge as certain diagnostic tools like pleural biopsy is not possible.

Case report

A 28 year old previously well patient presented with progressively worsening shortness of breath associated with a dry cough and left sided pleuritic type chest pain for 4 months. He was experiencing loss of appetite with weight loss for the same duration. There was no history of hemoptysis, body swelling or night sweats. He had a contact history of Tuberculosis and denied to have asbestos exposure.

On examination he was in respiratory distress at rest with a respiratory rate of 35/min with reduced chest expansion on the left. He did not have clubbing or lymphadenopathy. His trachea was central and respiratory system examination showed absent breath sounds with stony dullness suggestive of a left sided effusion. There was no organomegaly.

His complete blood count showed a white blood cell count 11600/ul (N – 78%), Hb – 10g/dl, platelet – 453000/ul. He had elevated inflammatory markers with ESR of 89mm/hr and CRP of 43mg/dl. A CXR which was done 2 weeks before at a primary care hospital showed a left sided effusion with an air bronchogram. Repeat CXR done on admission showed a left sided diffuse pleural calcifications involving the entire pleura. Further evaluation with bed side chest ultrasonography showed pleural calcification with septae and thick echogenic material suggestive of empyema. Pleural aspiration was hemorrhagic. Pleural fluid report showed total cell count 16700/ul (L-80%), Potein 3.7g/dl, LDH -1200 U/L, ADA – 88U/L with a sugar level of 41mg/dl. Pleural fluid Ph was 7.0 and cytology did not show abnormal cells. Pleural fluid for pyogenic culture, gram stain, fungal and TB culture were negative.

He was further investigated with serum ionized calcium and phosphate which were in normal range. Sputum studies for sputum AFB, Gene X –pert, AFB culture were negative. Retro viral screening and fasting blood sugar values were within the normal range.

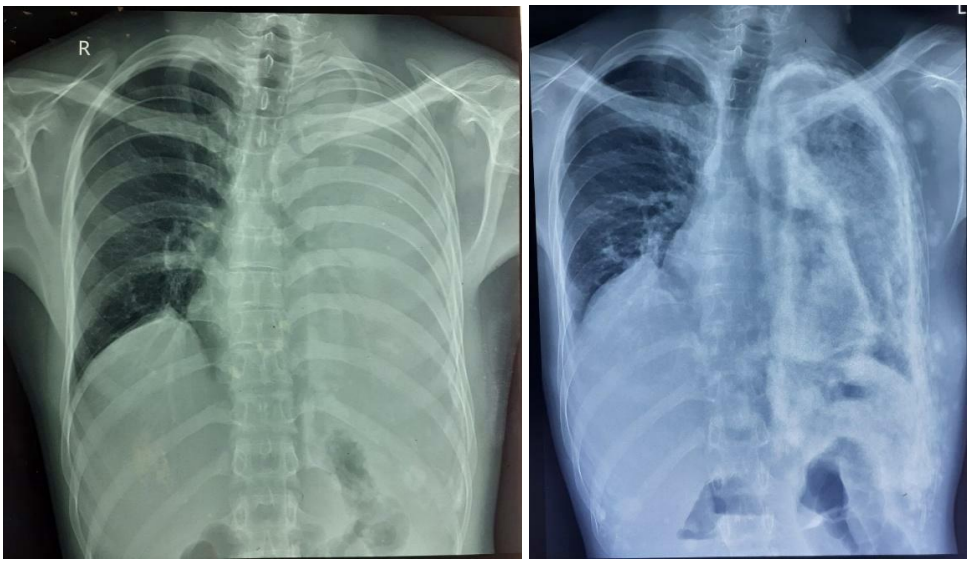

Figure 1 – CXR taken two weeks apart showing an initial left sided pleural effusion with an air bronchogram followed by left sided pleural calcification with central localized fluid collection.

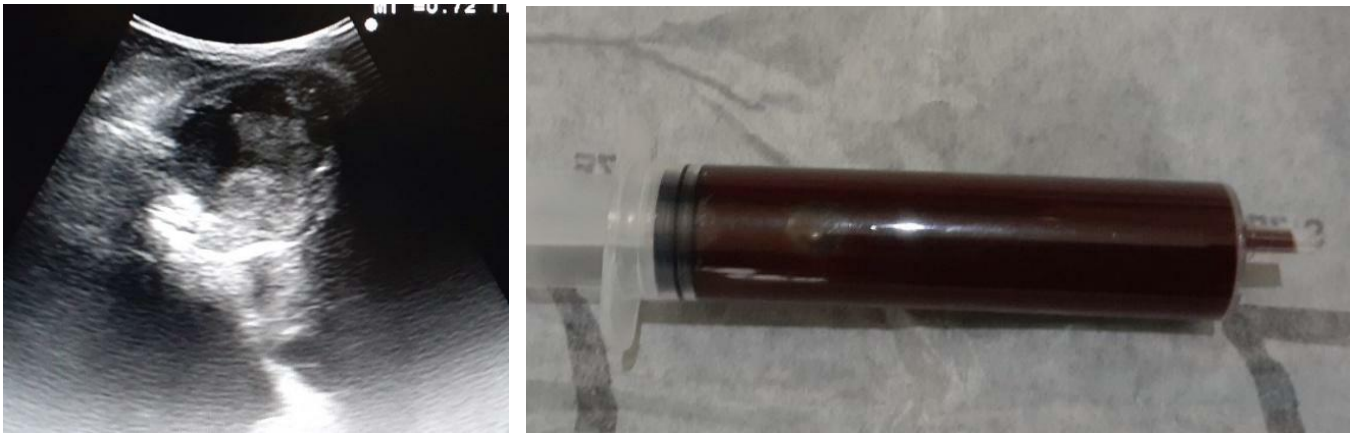

Figure 2 - Ultrasound scan showing pleural calcifications and pleural aspirate showing hemorrhagic fluid

CECT chest showed diffuse pleural calcifications with multiple septae formation. There was evidence of contralateral pleural involvement with calcified lymph nodes in the mediastinum, hilar region and in the axilla. There were no lung parenchymal changes, other mediastinal abnormalities and abdominal abnormalities except for peritoneal calcification. His mantoux test became strongly positive (17mm).

Considering the overall picture a diagnosis of extrapulmonary TB was made and he was treated with standard anti TB treatment and intercostal tube was inserted as there was evidence of empyema with other supportive measures. Pleural biopsy was not possible and thoracoscopy was not attempted as he was not stable for the procedure.

Despite vigorous treatment his respiratory distress did not resolve and passed away on the sixth day following admission.

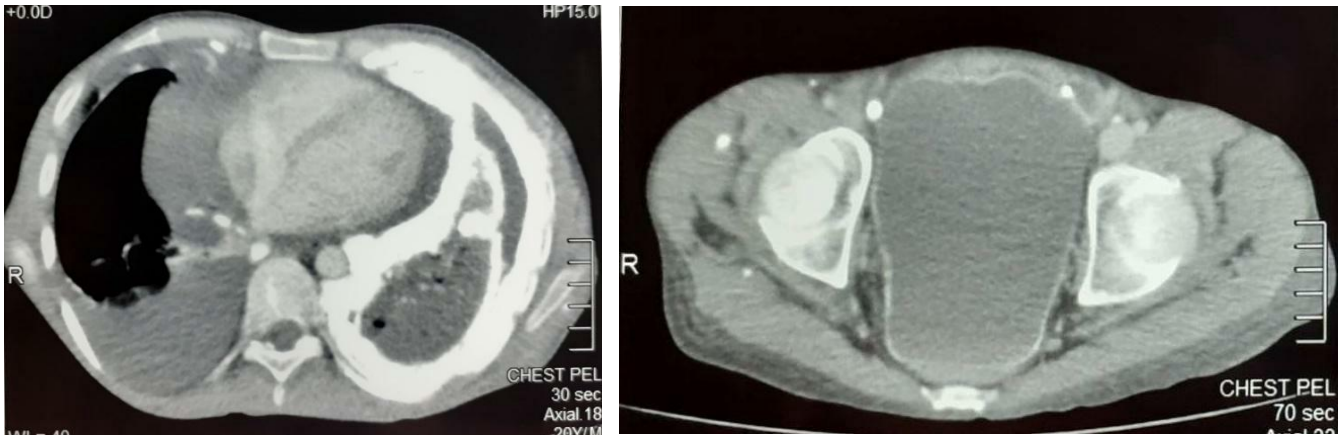

Figure 3 – CECT showing diffuse pleural calcifications, septae formation of left lung with early right sided pleural and peritoneal involvement

Discussion

Pleural tuberculosis is the most common presentation of extrapulmonary tuberculosis and the most common cause of pleural effusion worldwide [4, 5]. Manifestation of pleural Tuberculosis as pleural plaques, calicifications was previously described in case reports [2, 3]. However our case report become extraordinary due to the diffuse involvement of the pleura which was not described earlier. Primary diffuse pleural calcification due to tuberculosis is a rarely described entity which is seldom seen. This may be due to delayed diagnosis or drug resistant forms. On the other hand, there is a rapid progression of

the disease which was evident when comparing X rays taken two weeks apart.

It has to be understood that pleural effusion usually occurs in the acute stage of the illness while pleural thickening and calcifications occur at a later stage [3] due to persistent chronic inflammation. Co-existing disease with concurrent effusion and calcifications is extremely rare making this case extraordinary. On the other hand concurrent disease involving bilateral pleurae and peritoneum was never described before highlighting the unique nature of the case report.

The diagnosis of pleural TB was made based on strong contact history of Tuberculosis, strongly positive mantoux test, hemorrhagic lymphocyte predominant exudative pleural effusion, elevated ADA level, negative cytology and calcification of lymph nodes.

There were some limitations of evaluation. Firstly, pleural biopsy was not possible due to diffuse and calcifying nature of the disease. Secondly, we were unable to subject him to a thoracoscopy, which is a good diagnostic tool in these types of challenging cases but he was not fit enough for the procedure. Thirdly there was no microbiological evidence of Tuberculosis in the pleural fluid. However detecting Tuberculous bacilli or particles from pleural fluid is extremely difficult.

Extensive pleural calcification is associated with several differential diagnosis. This includes malignant mesothelioma mostly due to chronic asbestos exposure, pleural lymphoma, pleural metastases, pleural tuberculosis and untreated long term hemothorax. Out of these mesothelioma is the most common etiology. Our patient did not have asbestos exposure and usually this is seen after fifth decade of life following long term exposure to asbestos which is not applicable in our case. There was no evidence of traumatic events and tuberculosis is a well-known cause of hemothorax. Pleural metastases can cause pleural calcifications but it is unlikely to cause homogenous diffuse disease as it usually causes patchy nodular infiltrate of the pleura. Isolated pleural lymphoma is an extremely rare disease and calcified lymph nodes, with high ADA titer was in more favor for Tuberculosis.

There are number of reasons for the poor outcome of the patient. His pleura was diffusely involved directly affecting his respiratory mechanism causing persistent respiratory distress. Secondly, there was a concurrent empyema in an encased area with limited antibiotic penetration. Thirdly, diagnosis was delayed by several weeks causing an undue delay in initiation of anti TB treatment. Finally he was not fit enough to undergo surgical intervention such as decortication.

Conclusion

Pleural tuberculosis is a rare cause of diffuse pleural calcification. Early diagnosis is mandatory in order to prevent lethal outcomes.

Declarations

Ethics approval and consent to participate

Not Applicable

Consent for publication

Available on request

Availability of data and materials

Not Applicable

Competing interests "The authors declare that they have no competing interests"

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Authors' contributions

Sugeesha Wickramasinghe – Literature review gathering data and writing the article

Bandu Gunasena – Reviewing the article before submission and guiding its intellectual content

Sumudu Palihawadene - Literature review and guiding its intellectual content

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