Pleural Manifestations of Tuberculosis

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**Learning objectives**

To illustrate pleural manifestations of tuberculosis.

To describe in detail each of these manifestations and discuss the differential diagnosis.

**Background**

Pleura is a serous membrane investing the lung (visceral layer) and lining the thoracic wall (parietal layer). These two layers enclose a space called pleural space.

Tuberculosis is a bacterial infectious disease caused by mycobacterium tuberculosis. Parenchymal disease is the most common presentation of pulmonary tuberculosis.

Pleural tuberculosis most commonly spreads by rupture of subpleural parenchymal focus into the pleural space or less commonly via hematogenous spread / contamination of adjacent infected lymph nodes.

Our exhibit focuses on the pleural manifestations which mimic other pleural disease patterns and throws light on the less common pleural features which one should be aware of.

**Findings and procedure details**

Pleura is imaged using ultrasound and multidetector computed tomography.

All cases are proven cases of tuberculosis (microbiological testing/immunological testing/pleural biopsy)

The following are the varied 14 pleural manifestations of tuberculosis

**Pleural effusion**
Tuberculous pleural effusion is the second most frequent extrapulmonary form of presentation. In case of primary TB, it manifests as unilateral free large effusion, without loculations. It occurs 3-6 months after infection and is induced when the mycobacterium releases antigenic protein into the pleural cavity, thus triggering an imperfectly understood delayed hypersensitivity reaction which increases the pleural fluid formation and and decreased pleural fluid removal by obstruction of lymphatic pores in the parietal pleura.[fig.1]

In analysis of TB pleural effusion, pleural fluid is invariably as protein-rich exudate and occasionally demonstrates an elevated level of adenosine deaminase (ADA), a sensitive marker of TB pleural effusion.

**Complex TB effusion**

Usually seen in post primary tuberculosis and is associated with parenchymal lesions. Delayed diagnosis and/or treatment of TB pleurisy may cause disordered fibrin turnover in the pleural cavity with subsequent fibrin deposition and loculation of pleural fluid. This pleural fluid is enriched in proteins, inflammatory cells, and various pro-inflammatory and profibrotic cytokines.

They can be complex septated, complex non septated or have a homogeneously echogenic pattern.[fig2]

**Chyliform/pseudochylos effusion**

The most common cause of high-lipid nonchylous effusion is tuberculous empyema. The diseased pleura may result in an abnormally slow transfer of cholesterol and other lipids, originating from degenerated red and white blood cells, out of the pleural space and lead to accumulation of cholesterol in the pleural fluid. CT shows a fat-fluid or fat-calcium level. [fig.3]

**Pleural thickening**

Pleural thickening in tuberculosis is sequelae to tuberculous pleurisy and can be focal or diffuse.[fig.4] Diffuse pleural thickening is defined as thickening of pleura (more than 5 mm) with combined area of involvement more than 25% of chest wall if bilateral and 50% involvement if unilateral.
Trapped lung is a dreaded complication of dysfunctional pleural healing after pleurisy that results in a restrictive visceral pleural peel. Surgical decortication is the only available therapy.

Apical pleural thickening is a normal aging process, but if the thickening is more than 2 cm, it requires further work-up. Differentials of diffuse pleural thickening are asbestosis, hemothorax, pulmonary fibrosis, irradiation, previous surgery, trauma and drugs.

**Nodular pleural thickening**

Nodular pleural thickening is usually a manifestation of malignancy and is seen as secondary deposits from various primaries, mesothelioma, lymphoma and invasive thymoma. On Computed Tomography (CT) scan, malignant pleural thickening is nodular (>1 cm), shows circumferential involvement, and involves the mediastinal pleura. At our institution we came across few cases of already proven tuberculosis (parenchymal) which also had nodular pleural thickening as one the many myriad manifestations of tuberculosis.[fig.5] A case of nodular pleural masses was initially diagnosed as malignant on imaging but histopathological diagnosis was positive for epitheloid granulomas suggestive of tuberculosis.[fig.17]

**Pleural calcification**

Results due to end stage tuberculous pleurisy. In the setting of concomitant parenchymal lesions and old history, the cause of pleural calcification can be attributed to tuberculosis. [fig.6] Other differentials to be kept in mind are asbestosis, previous hemothorax, surgery and extraskeletal osteosarcoma.

**Chronic Tuberculous Empyema**

Tuberculous pleurisy progresses to become chronic tuberculous empyema, which may be defined as persistent, grossly purulent pleural fluid containing tubercle bacilli. However, it may be difficult to culture the bacilli in chronic empyema.

Empyema usually develops in three distinct phases - exudative, fibrinopurulent and organizing. In the fibrinopurulent phase, CT shows thickened visceral and parietal pleurae separated by fluid, the "split pleura" sign.[fig.7] In the organizing phase it reveals a loculated pleural fluid collection with thickened pleural peel and variable degree of calcification with or without proliferation of extrapleural fat.[fig.8]
Fibrothorax

End result of tuberculous pleurisy is fibro thorax. It is characterized by volume loss, diffuse thickening, calcification and impaired lung function. Absence of pleural effusion suggests inactivity and is characteristic of fibro thorax[fig.8],[fig.9]

Bronchopleural Fistula

Fistulous communication can occur following spread of disease, tension pneumothorax, trauma or surgical procedure.

CT is the investigation of choice and may demonstrate the exact site of communication between the pleural space and the bronchial tree or lung parenchyma.

Appearance of air-fluid level in empyema suggests communication with the bronchial tree (bronchopleural fistula).[fig.10] It presents as increasing expectoration, air in the pleural space, and changing air-fluid level.

Bronchocutaneous fistula

Pneumatocele or subcutaneous emphysema is a very rare manifestation of rupture of a pulmonary cavity into subcutaneous tissue resulting from bronchocutaneous fistula.[fig.11] Drainage of a cold abscess from TB may be responsible for a potential weakness in the pleura and chest wall leading to the development of BCF

Other causes of fistulous communication are pulmonary operations, perforating chest trauma, empyema, lung abscess, pneumonia or massive pulmonary infarction.

Pneumothorax

Pneumothorax secondary to tuberculosis often heralds severe and extensive pulmonary involvement by the infectious process and the onset of bronchopleural fistula and empyema. It occurs in approximately 5% of patients with postprimary tuberculosis, usually in severe cavitary disease but rarely in miliary tuberculosis. The pathogenesis involves pleural caseous infiltrates that undergo liquefaction, resulting in pleural necrosis and rupture.[fig12] If any apical abnormality is seen after reexpansion of a spontaneous pneumothorax, active tuberculosis should be considered.
Empyema necessitans/Chest Wall Tuberculosis

Spontaneous discharge of empyema through the parietal pleura into the chest wall forms a subcutaneous abscess which is termed *empyema necessitans*. [fig 13] It can also spread to involve other sites such as the esophageal, breast, retroperitoneal, peritoneal, pericardial and paravertebral regions. Bone and cartilage may be intact or destroyed in chest wall tuberculosis. Rarely, it is associated with development of retromammary or intramammary tuberculous abscesses.

Bone/cartilage destruction

Tuberculosis occasionally involves the sternum, the sternoclavicular joint, or a rib, leading to destruction and localized abscess formation.[fig 14] Such involvement may occur by direct extension from a pleuropulmonary tuberculous lesion or by hematogenous spread from a distant focus.

Pleural mass

Pleural tuberculosis can rarely present as benign multiple pleural nodules and masses with or without parenchymal involvement or lymphadenopathy. These lesions may demonstrate calcification/rim enhancement.[fig.15][fig 17] Differentials to be kept in mind are pleural metastases, mesothelioma, lymphoma and malignant thymoma.

The occurrence of malignant neoplasm is a relatively rare but critical complication of chronic tuberculous empyema. Pathologic cell types of malignancy associated with longstanding empyema are variable and include malignant lymphoma, squamous cell carcinoma, mesothelioma, malignant fibrous histiocytoma, sarcoma, and hemangioendothelioma. CT can reveal an abnormal mass with soft-tissue attenuation around the empyema and usually contrast enhancement in the mass.[fig.16]

Images for this section:
**Fig. 1:** pleural effusion

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**Fig. 2:** complex effusion with sepatations, loculation (arrow) and echogenic material (star)

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Fig. 3: pseudochylous effusion with fat fluid level(arrow) in a known case of tuberculosis(pleural calcification)

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**Fig. 4:** pleural thickening

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Fig. 5: nodular thickening of pleura

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Fig. 6: pleural calcification

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Fig. 7: empyema with split pleura sign (fibropurulent stage)

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Fig. 8: empyema in organising stage (star) fibrothorax with calcifications (arrows)

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Fig. 9: fibrothorax with volume loss and mediastinal shift

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Fig. 10: bronchopleural fistula (red arrow) hydropneumothorax (yellow arrow)
Fig. 11: bronchocutaneous fistula

Fig. 12: pneumothorax (black arrow) parenchymal tuberculosis (red arrow)

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**Fig. 13:** empyema necessitans

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**Fig. 14:** rib erosion seen in a case of tuberculous lesion

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**Fig. 15:** pleural mass with rim calcification

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**Fig. 16:** old case of tuberculous fibrothorax with newly diagnosed mass causing rib erosion-adenocarcinoma.
Fig. 17: pleural nodular masses
Conclusion

Pleural manifestations of tuberculosis are many and knowledge of various radiological features is valuable for appropriate diagnosis and management.

Personal information

References

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