# **Tuberculosis of the Chest Wall with Massive Tuberculous Pleural Effusion**

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## **Abstract**

Primary tuberculosis of components of the chest wall is a rare entity. Involvement of skeletal muscle by tuberculosis without any primary focus is also rare. Here, we report a case of tuberculosis of chest wall without pulmonary or bone involvement, that invaded into the pleural space leading to a massive pleural effusion. [Indian J Chest Dis Allied Sci 2016;58:63-65]

Key words: Chest wall, Tuberculosis, Pleural effusion.

#### Introduction

Primary tuberculosis of the components of chest wall as well as involvement of any skeletal muscle without any primary focus is rare. Chest wall tuberculosis constitutes 1% to 5% of all cases of musculo-skeletal tuberculosis and 1% to 2% of all tuberculosis cases.¹ Chest wall tuberculosis may occur as an extension from an affected lymph node group or underlying bone involvement.² It may also result from a direct invasion from an underlying pleural or pulmonary parenchymal disease or as a result of haematogeneous spread from an occult primary focus.³ We report a case of tuberculous chest wall abscess without pulmonary and bone involvement, that invaded into the pleural space, resulting in a massive pleural effusion.

# **Case Report**

A 30-year-old male was admitted with a history of dry cough and intermittent low-grade fever for one month, associated with breathlessness at rest for two days and loss of weight and appetite. There was no past history of tuberculosis or contact with a case. He was a chronic alcoholic but had given up since last six months.

The patient was admitted to general surgery ward two weeks prior with a non-tender swelling on the anterior aspect of the chest on the left side that had gradually increased in size over a period of three weeks. Aspiration of the swelling had revealed thick pus and the swelling was incised and drained. Reports of investigations were awaited.

When he presented to us, he was febrile with a pulse rate of 120 per minute, a blood pressure of 140/90mmHg and a respiratory rate of 30 per minute. There was no pallor, icterus, clubbing, palpable lymph nodes or pedal oedema. There was a healthy incision wound on

the anterior chest wall on the left side measuring 7cm×4cm in size. Examination of the respiratory system revealed a shift of the trachea to the left, with decrease movements on the left side associated with a decrease in the vocal fremitus in the left mammary area, axillary area, infra-axillary area and infra-scapular area. On percussion, there was a stony dull note in these areas and on auscultation, breath sounds were absent with decrease in vocal resonance in the same areas. Other systems examination were within normal limits. The findings suggested a left-sided pleural effusion.

Laboratory investigation revealed: haemoglobin 11.6g%, total leucocyte count 10000 cells/cmm, with neutrophils 66%, lymphocytes 21%, monocytes 12%, basophils 1%; blood urea 10mg/dL and serum creatinine 0.7mg/dL; serum bilirubin (Total 2.2mg/dL, direct 0.9mg/dL, serum glutamic-oxaloacetic transaminase [SGOT] 110IU/L and serum glutamic-pyruvic transaminase [SGPT] 69IU/L, alkaline phosphatase 130IU/L and total proteins 5.8gm/dL. Repeat liver function tests after treatment with two weeks of hepato-protective drugs were as follows: serum bilirubin-total 1.5mg/dL, direct 0.9mg/dL, SGOT 81IU/L and SGPT 50IU/L. Serum amylase was 53IU/L. Enzyme-linked immunosorbent assay test for human immunodeficiency virus was non-reactive.

The chest radiograph showed a massive left-sided pleural effusion (Figure 1). Abdominal ultrasonography revealed a mildly enlarged liver (18cm) with grade 1 fatty changes, while the rest of the ultrasonography was normal. Contrast enhanced computed tomography (CECT) of the thorax done two weeks earlier during evaluation of the chest-wall swelling had shown a bulky left pectoral muscle with a well-defined round to oval heterogeneous area within and multiple enlarged lymph nodes in the axillary area. On post-contrast, the lesion was 8.5cmx3.5cm in size with

[Received: October 20, 2014; accepted: February 17, 2015]

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extension to the extra-pleural space (Figure 2). The underlying lung parenchyma, airways and mediastinum and trachea and central bronchi had no abnormality. Underlying bones were normal.

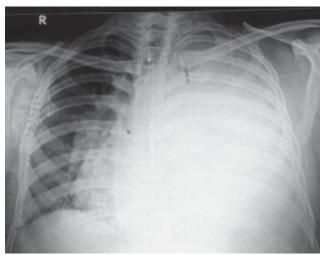


Figure 1. Chest radiograph (postero-anterior view) showing left-sided massive pleural effusion.



Figure 2. Contrast enhanced computed tomography of thorax showing left-sided chest wall abscess with extension into extra-pleural space.

A repeat CECT of the thorax done during the current admission revealed an encysted pleural effusion along the anterior and lateral chest wall on the left side with the basal segment of the left lower lobe showing collapse and consolidation. There was a slight shift of the mediastinum to the right. Enlarged lymph nodes were noted in the axilla. Few subcentimetric pretracheal, para-tracheal, pre-vascular lymph nodes were also noted without any remarkable features. There was no evidence of bone involvement (Figures 3 and 4).

The patient underwent thoracocentesis and a total of three litres of clear, straw-coloured fluid was aspirated. Pleural fluid examination showed 100% lymphocytes, proteins 4.1g/dL, sugar 154mg/dL, chloride 96mEq/L and an amylase level of 52IU/L.

Adenosine deaminase (ADA) levels were 81.2U/L. Culture for pyogenic organisms was sterile and smear for acid-fast bacilli was negative. Thus, the pleural fluid investigations were suggestive of tuberculous aetiology.



Figure 3. Coronal CT image showing drained chest wall abscess with encysted pleural effusion along anterior and lateral chest wall on left-side, no evidence of bone involvement.

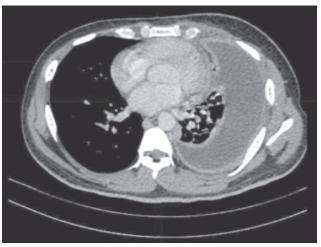


Figure 4. The CECT of thorax showing encysted pleural effusion along anterior and lateral chest wall on the left side, no evidence of hone involvement.

By this time, the histopathology report of the wall of abscess that had been drained earlier was available. It showed granulation tissue enclosing casseating epitheloid cell granulomas surrounded by Langhans giant cells and fibroblasts suggestive of tubercular granulation tissue. This was suggestive of tuberculosis of the chest wall. A final diagnosis of tuberculosis of the chest wall skeletal muscle with tuberculous pleural effusion was made.

The patient was prescribed anti-tuberculosis treatment with non-hepatitinic regimen because of altered liver function tests. He received injection streptomycin 0.75g, with oral ciprofloxacin 750mg, and ethambutol 800mg daily to which he showed good improvement. The chest wall incision healed rapidly and there was no recurrence of pleural effusion. He became afebrile at the end of two weeks.

## Discussion

Explaining the pathogenesis of chest wall tuberculosis, Burke<sup>5</sup> proposed that tuberculous bacilli invade the pleural space, leading to local or widespread pleuritis. Some of the bacilli are transported to chest wall lymph nodes where caseous necrosis and liquefaction occurs. This necrotic material tracks either anteriorly or posteriorly into the chest wall causing a chest wall abscess.<sup>5</sup> Our patient initially presented with tuberculous chest wall abscess with extra-pleural extension and subsequently developed pleural effusion. The pleural effusion was likely due to an extra-pleural extension of the chest wall abscess.

Similar cases of tuberculous chest wall abscess without pulmonary involvement have been reported in the past by Gaude and Reyas<sup>6</sup> and Unsal *et al*<sup>7</sup>. Kim *et al*<sup>8</sup> have reported chest wall abscess with pleural effusion, where 23.1% of patients with chest wall abscess had a loculated pleural effusion.

Chest wall tuberculosis may involve bones, like sternum, costochondral junctions, rib shafts, costovertebral joints and vertebrae. Cases of tuberculous chest wall abscess due to rib involvement has been reported by Keny<sup>10</sup> in the past. Our patient had no evidence of pulmonary or bone involvement.

Medical management is similar to extra-pulmonary tuberculosis. Some series in the past have reported good outcomes with anti-tuberculosis drugs alone.<sup>11</sup> If the medical treatment is not adequate then debridement may be required along with anti-tuberculosis drugs. Paik *et al*<sup>12</sup> reviewed 89 patients who underwent surgical resection of cold abscess of the chest wall. They recommended post-operative tuberculosis medication for a minimum of 12 months.<sup>12</sup> In our patient, incision

and drainage of the chest wall abscess, and thoracocentesis of the pleural effusion was carried out and anti-tuberculous therapy was initiated with injection streptomycin, and oral ciprofloxacin and ethambutol as his liver function tests were deranged. He responded well to the treatment and became afebrile after two weeks of therapy with rapid healing of the incision wound on the chest wall.

To conclude, a tuberculous aetiology must be considered in a case of chest wall abscess, especially when a pyogenic process is not established.

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