Chest Wall Tuberculosis without Pulmonary Involvement

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INTRODUCTION

Tuberculosis causes significant public health problem especially in the developing country like India. Chest wall tuberculosis is rare and can cause significant diagnostic and therapeutic challenge. The characteristic presentation of chest wall tuberculosis is cold abscess. Rarely can it present with no lesion in the lung parenchyma or in the ribs [1].

CASE REPORT

A 48-year-old male patient came with complaints of painless swelling over the chest wall. This swelling has been present for more than 5 months in duration. Initially this was a non-progressive swelling but later it increased in size and burst open two days before he came to the institution. After it burst open, thick pus started discharging from the site. He had no complaints of cough, expectoration, hemothysis, chest pain, wheeze and prior history of any TB infection. Patient complained of fever when he started developing the swelling two months back for seven days. He had no history loss of weight or loss of appetite. He had no significant past medical history with no history of any substance abuse. He was an auto driver by occupation. Physical examination was unremarkable with no clubbing or lymphadenopathy. Local examination of the chest wall revealed a 2 x 2 cms ulcer with serous discharge. The ulcer was seen over the sternum at his level of fifth rib with undermined edges [Fig.1]. Respiratory examination was unremarkable.

His complete blood count, liver function test, renal function, serum electrolytes, urine routine examination was normal. His HIV was non reactive and HbA1c was normal. Mantoux was 17 mm after 48 hours. Chest X ray was normal [Fig.2]. CT thorax scan done showed a soft tissue dense lesion of size 2.6 x 1.2 cms with surrounding inflammatory changes in the subcutaneous plane overlying the sternum at fifth rib level and extending to the fifth costochondral junction. No evidence of pleural, parenchymal or mediastinal involvement [Fig 3]. Biopsy from the edge of the ulcer showed granulomas consisting of epithelioid cells, lymphocytes and Langhan’s type of giant cells, which was conclusive for TB [Fig.4].

Thus the diagnosis of chest wall tuberculosis without lung involvement was made and he started on anti tuberculous drugs for the duration of 6 months. After completing 4 months of ATT the ulcer completely healed and there was good clinical response.
Fig-1: Lesion seen in the chest wall

Fig-2: Normal CXR

Fig-3: CT showing Soft tissue dense lesion with surrounding inflammatory changes in the subcutaneous plane overlying the sternum at fifth rib level and extending to the fifth costochondral junction

Fig-4: Histopathology (Hematoxylin and Eosin staining, 200x) shows Langhan’s giant cell with granuloma
**DISCUSSION**

Despite the improvement in the treatment TB still remains a major cause of mortality and morbidity especially in India [1]. Chest wall tuberculosis infections constitute 1 – 5% of all musculoskeletal TB, which constitutes 1 – 2% of all Tb cases [2]. The incidence is not expected to stay so low in the future because of the rapid emergence of the drug resistant bacilli and increase in the number of immunocompromised patients [3]. Chest wall TB infections can involve sternum, rib shafts, costochondral junctions, costovertebral joints and vertebrae, frequently involving the margins of the sternum and rib shafts in almost 60% of the cases [4] as was evident in our case. There are three main mechanisms by which chest wall TB infection can occur (i) lymphatic spread, (ii) hematogenous dissemination, (iii) direct extension from pleural or parenchymal lesions [1]. In our case we believe that the most probable route of spread is lymphatic. Faure et al. [5] suggested lymph nodes of the chest wall become caseous and necrotic then burrow externally to form an abscess. These findings support the possibility of lymphatic spread in our case.

Laboratory investigations like CBC, ESR is usually unremarkable, as seen in our case [6]. Morris BS et al. [4] suggested CT thorax typically manifests cartilaginous destruction and soft tissue masses with rim enhancement and calcifications along with osseous and cartilaginous destruction. These findings were seen in our case. Histopathological examination usually establishes the diagnosis [7], as seen in our case showing caseating granuloma and Langerhans giant cells, which are characteristic of tuberculosis. Other possibilities of granuloma include sarcoïdosis, atypical mycobacterial infection, crohn’s disease, Histoplasmosis, Actinomycosis, Wegener’s granulomatosis and Langerhans cell histiocytosis [8]. There is increase in number of atypical mycobacterial infection cases reported world wide our case had giant cells and necrosis in the histopathology, which is more in favour of mycobacterial tuberculosis [9]. Even though not performed in our case, real time PCR on GeneXpert platform can be used to establish the diagnosis [1]. Diagnosis of chest wall tuberculosis is usually challenging and delayed as seen in our case [1].

Treatment of chest wall tuberculosis is controversial. There are some series reporting good results with only anti tuberculous drugs while others reporting the requirement of surgery, although WHO recommends only standard tubercular drugs [2].

**CONCLUSION**

Our case had a 2.6 x 1.2 cms indolent ulcer over the chest wall with serous discharge. There was no evidence of any parenchymal, mediastinal or pleural involvement in the CT thorax and with histology showing caseating granulomas consisting of epithelioid cells, lymphocytes and Langhan’s type of giant cells, the diagnosis of primary chest wall tuberculosis was made.

**REFERENCES**