

Primary tuberculous abscess of chest wall in a young immunocompetent female

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ABSTRACT

Chest wall tuberculosis (TB) is rarely reported in immunocompetent individuals and is usually a part of disseminated disease. A tuberculous abscess may mimic pyogenic abscess and unless strongly suspected, tuberculous etiology may be missed due to paucibacillary nature of extrapulmonary lesions. Molecular diagnostic tests can prove useful by providing a rapid and accurate diagnosis in such cases. We present here a case of chest wall tuberculous abscess without any evidence of immunosuppression or previous history of TB.

Key words: Chest wall abscess, Extrapulmonary, Tuberculosis

India accounts for one-fourth of the global tuberculosis (TB) burden. It features among the high-burden country lists for TB, TB/HIV, and multidrug-resistant TB defined by the World Health Organization (WHO). As per the WHO Global Report on TB 2016, 16% of all freshly diagnosed cases in India are extrapulmonary [1]. Extrapulmonary TB (EPTB) is rarely smear-positive; it is generally accepted that the contagious potential of this form is negligible, and therefore, it has never been a priority in the campaigns undertaken by national TB control programs. The new Revised National TB Control Program (RNTCP) strategy to offer cartridge-based nucleic acid amplification technique (CBNAAT) upfront for the diagnosis of EPTB will improve the program's capacity to diagnose EPTB patients [2]. Tuberculous abscess of the chest wall is usually a part of disseminated TB. Most cases present with soft tissue swelling (cold abscess) or discharging sinus over anterior chest wall [3]. Here, we report a case of primary tubercular chest wall abscess in an immunocompetent individual with no history of contact or previous antitubercular therapy, confirmed by CBNAAT.

CASE REPORT

A 25-year-old female presented with a lump on the anterior chest wall in the left paramedian region. It was preceded by pain and had gradually increased in size over a period of about 4 weeks. There was no history of trauma or similar lesion in the past. There were no respiratory complaints, fever, and loss of appetite or weight. There was no history of TB or contact with a case of TB in the family. She had no chronic illness other than hypothyroidism for which she was being treated with thyroxine (50 mcg).

On examination, the patient was average built, afebrile. No abnormality was detected on systemic examination. A painless swelling was present in the left paramedian region, medial to the left breast. It was about 2–3 cm in size, soft, mobile, and tender. There was no redness, localized rise of temperature, or venous prominence over the swelling. There was no regional lymph node involvement. The differential diagnosis included cold abscess, pyogenic abscess, and costochondritis.

Laboratory investigations revealed: Hemoglobin 10.3 g/dl; total leukocyte count 8200/cu.mm with a differential count of 67% neutrophils, 29% lymphocytes, 3% monocytes, and 1% eosinophils. Other biochemical investigations, including liver and kidney function tests, were within normal limits. Serology for HIV was non-reactive. Ultrasound evaluation revealed a hypoechoic area in the soft tissues superficial to the left costochondral junction (Fig. 1). Fine-needle aspiration from the swelling was done and yielded pus. Cytological examination showed dense neutrophilic infiltrate with few lymphocytes and macrophages in a background of extensive caseous necrosis. Aerobic bacterial culture of the pus was sterile, but acid-fast bacilli were detected on Ziehl–Neelsen staining (Fig. 2). CBNAAT confirmed the presence of *Mycobacterium* TB sensitive to rifampicin, while culture on Lowenstein–Jensen media was lost to contamination. Chest radiograph was normal with no evidence of hilar lymphadenopathy (Fig. 3). No clinical, radiological, or microbiological evidence of pulmonary TB could be established.

The patient was started on Category I antitubercular treatment with four drugs, namely, isoniazid, rifampicin, ethambutol, and pyrazinamide. On follow-up after 2 months, the abscess showed evidence of resolution and had decreased in size.

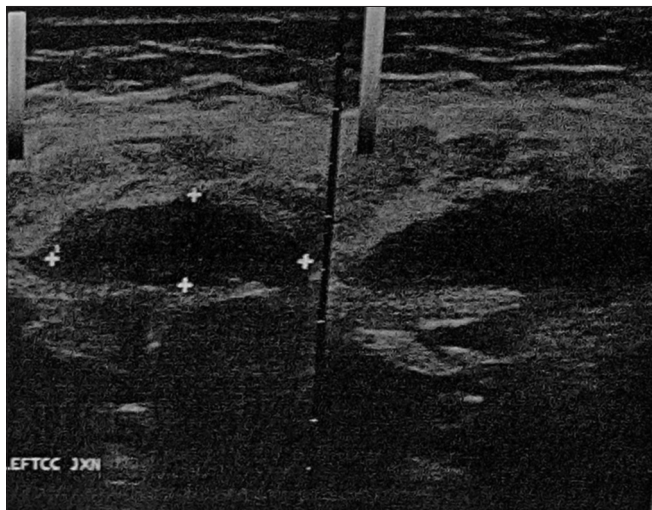


Figure 1: Ultrasound showing hypoechoic area in the soft tissue superficial to left costochondral junction

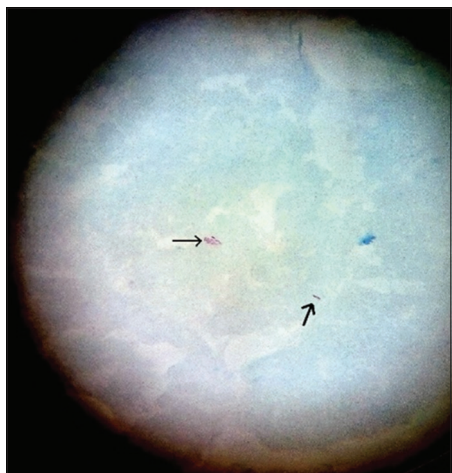


Figure 2: Ziehl-Neelsen staining of aspirated pus showing acid-fast bacilli

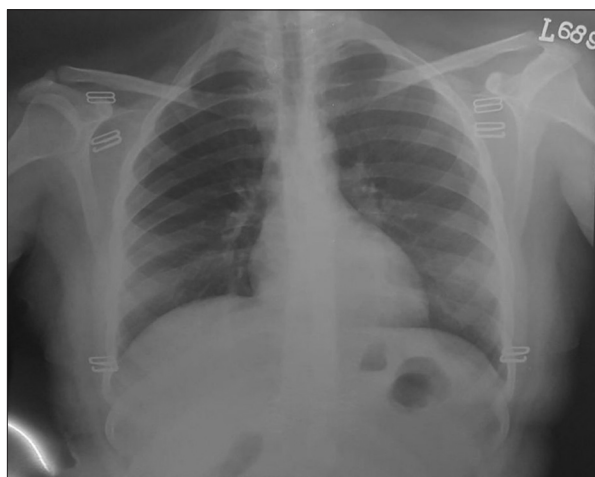


Figure 3: Chest X-ray showing normal lung parenchyma and absence of hilar lymphadenopathy

DISCUSSION

EPTB accounts for 10–20% of TB in immunocompetent patients and more than 40% of HIV-positive individuals. [4]

Chest wall TB is rare form of EPTB and accounts for 1–5% of all musculoskeletal TB, which itself represents 1–2% of the total cases of TB. Sternum is the most common site to be involved though rib shaft and costochondral junction, and vertebral bodies can also be involved [5]. There are several reports of tuberculous subcutaneous abscesses of the thoracic wall [5–8]. Three mechanisms are described in the pathogenesis of chest wall abscess: Direct extension from pleural or pulmonary parenchymal disease, hematogenous dissemination of a dormant tuberculous focus, or direct extension from lymphadenitis of the chest wall [3].

In a study of tubercular chest wall abscesses by Keum *et al.*, 45.6% of cases had a past or current history of TB [7]. There are only few reports of tubercular abscesses without a history of TB [5,8]. Similarly, no primary focus of TB could be found in our patient.

Cold abscesses of the chest wall are generally solitary, but multiple lesions are possible. In a case series of 68 patients with chest wall cold abscesses, 64 were solitary [7]. Our patient also had solitary abscess. Cases have been reported of chest wall abscess with progression to a discharging sinus and ulcer formation [7,9]. Sometimes, abscesses make a fistulous tract to the pleural cavity and destroy underlying bone or cartilage [7,8]. In most cases, tubercular cold abscess has an insidious course with non-specific symptoms, and therefore, patients may remain undiagnosed for a long time and may present with complications. Swelling was the only presenting complaint in our case.

Biopsy and culture of mycobacteria are considered as the gold standard, though fine-needle aspiration cytology (FNAC) is the most commonly used technique for establishing a diagnosis [9]. In our case also, the pus sample after FNAC was used to achieve the microbiological diagnosis by Ziehl-Neelsen staining and CBNAAT. Culture, though highly sensitive and specific for TB diagnosis, requires 6–8 weeks to yield results, and hence, not useful for early diagnosis. In tropical countries like India, the rate of culture contamination is high, making novel diagnostic techniques such as CBNAAT all the more useful. Nucleic acid amplification technique provides rapid (2 h) and accurate diagnosis of TB by detecting *M. tuberculosis* and rifampicin resistance (*rif* gene). Under RNTCP, it was earlier used in cases of pulmonary TB only, but now it is used upfront in EPTB as well. In our patient, *M. tuberculosis* was confirmed by CBNAAT leading to early institution of therapy and prevention of possible complications such as ulceration, involvement of underlying structures as well as systemic dissemination.

Antitubercular therapy is the mainstay of management. Surgical intervention is secondary in the form of either ultrasonography or computerized tomography-guided aspiration or open drainage which is usually reserved for patients in whom medical treatment has failed [10].

CONCLUSION

Primary tubercular abscess of the chest wall in an immunocompetent patient without involvement of other structures is rare, and diagnosis requires a high index of suspicion

and microbiological examination to confirm *M. tuberculosis*. In a country like India, with a high burden of TB and limited availability of culture and molecular diagnostics, there is a possibility that EPTB may be missed or misdiagnosed.

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