

Tuberculous abscess of the thoracic wall secondary to nodular tuberculosis: Atypical presentation in an older adult

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Abstract

Tuberculosis (tb) of the chest wall is uncommon and it represents less than 5% of all cases of musculoskeletal tb and only 1-2% of tb. We present the case of an elderly woman with tb of the chest wall secondary to a nodal tb with an unusual presentation. The diagnosis of this entity is difficult because the disease often mimics other diseases such as pyogenic abscess, chest wall.

KEY WORDS: Tuberculosis. Tuberculous abscess. Nodal tuberculosis.

Introduction

Tuberculosis (TB) remains an important global health problem. In 2013, 9 million people were estimated to have developed this disease and there were 1.5 million deaths caused by it, including 360,000 deaths of patients with HIV¹.

Over the past two decades, TB cases have decreased, especially pulmonary TB; in 2012, 0.8 million cases of extrapulmonary TB were reported worldwide², with tuberculous lymphadenitis being the most common form, which is responsible for 43% of peripheral lymphadenopathy in the world^{3,4}.

TB presentation on the chest wall is uncommon, and it is often secondary to a history of pulmonary, pleural or lymph node TB, as in the case we next present⁵.

Case presentation

This is the case of an 81-year-old woman with no relevant personal history or chronic-degenerative conditions such as diabetes mellitus. On the directed interrogatory she denied a history of cohabitation with

people with TB. Her symptoms had started 2 weeks prior to her admission with volume increase at the left infraclavicular region, with no change of temperature or color at said region; 1 week later, pain in the area was added, as well as malaise, diaphoresis and non-quantified hyperthermia of nocturnal predominance that required physical measures and acetaminophen to be controlled. These symptoms persisted on subsequent days and she therefore attended our hospital. At her admission, during physical examination, a left infraclavicular tumor was found of 8 x 6 cm in size at fixed tension, with burning pain on superficial palpation, and no changes of color or temperature; in addition, ipsilateral axillary chain lymphadenitis was found, the largest size of which was approximately 1.5 cm (Fig. 1). The rest of examination was normal. Laboratory data indicated the presence of leukocytosis of 16,900/mL at the expense of 15,717/ μ L neutrophilia; a chest X-ray was also performed (Fig. 2 A).

Continuing with the protocol, a chest computed tomography (CT) was performed, where a lesion was observed occupying the left hemithorax, posterior and inferior to the pectoralis minor muscle, with

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Figure 1. Clinical photograph showing the volume increase located at the left pectoral infraclavicular region, without hyperemia or skin alterations.

4.3 x 5.3 mm in diameter, heterogeneous density, multiple adenopathies of the ipsilateral axillary chain and a left supraclavicular lymph node mass associated with cutaneous and fatty involvement, with diffuse interstitial thickening and presence of isolated pulmonary bullae (Fig. 2 B-D).

A pyogenic abscess was suspected and empiric management was started with moxifloxacin for 6 days with a decrease of the mass volume, and the patient was therefore discharged owing to clinical improvement. One month after the discharge, she started with 38 °C fever of nocturnal predominance; she self-medicated with acetaminophen and the temperature decreased. This clinical picture was repeated for the 3 subsequent days and she attended the hospital again. On physical examination, an 11 x 6 cm lesion was found on the left sternocleidomastoid muscle with the same characteristics of the previous one and with ipsilateral cervical lymph nodes present. The rest of the examination was normal.

Plain and contrasted chest CT revealed a 100 x 50 mm hypodense image located below the left sternocleidomastoid muscle extending to the midline, pushing the trachea to the right and compressing the vascular structures of the left cervical region. The contrast revealed peripheral enhancement, which indicated the diagnosis of an abscess at this level. No abdominal or renal lymph nodes were found.

The abscess was drained under fluoroscopy guidance, with 90 cc of purulent material being obtained; Ziehl-Neelsen staining revealed the presence of acid-alcohol-resistant bacilli (AARB) and the sample was sent for culture (Fig. 3 A).

Two days after the puncture, an increase of the abscess volume was found and surgical drainage was carried out, as well as left jugular lymph node resection, with histological report of tuberculous granulomatous lymphadenitis (Fig. 3 C, D). Purified Protein Derived test (PPD) was positive; three bacilloscopies were negative, and the Quanti-FERON-TB assay for TB had an indeterminate result. At 8 weeks of the drainage, *Mycobacterium tuberculosis* growth was reported in Löwenstein-Jensen culture medium (Fig. 3 B), and treatment was therefore started with rifampicin (RIF), isoniazid (INH), pyrazinamide (PZA) and ethambutol (EMB). The patient is currently on follow-up as an outpatient with report of clinical improvement and no relapse of the abscess so far.

Discussion

Tuberculous involvement of the chest wall, as the presented case, is infrequent and accounts for less than 5% of musculoskeletal TB cases and only 1-2% of TB overall; usually, it is 8-fold more common in males than in females and more than half the patients has a previous history of pulmonary TB, unlike the presented case, where there was no history of pulmonary involvement^{5,6}.

In a series of 181 cases of tuberculous lymphadenitis, Smaoui et al. demonstrated that 55.2% (n = 100) had extended lymphadenopathy and that most common localizations were cervical (83.4%), axillary (6.6%) and unilateral (78.4%). In addition, they found concomitant extrapulmonary TB in 13 patients: miliary in 5, pleural in 2 and abdominal, skin and others only in 1⁷.

TB involvement in soft tissues is generally associated with an underlying condition, such as collagenopathies, immunosuppressive therapy or local lesions; however, our patient had none, and reaching a diagnosis was therefore difficult right from the start⁸.

Some tissues, such as bone marrow, liver and spleen tissues, are almost always invaded, but rarely allow replication, while others promote growth (pulmonary apices, kidney, brain and bone)⁹. Chest wall TB abscesses are more commonly found at the sternum margins, along the rib axes and also can be located at the costochondral and costovertebral joints. The low incidence of chest TB may be due to the fact that

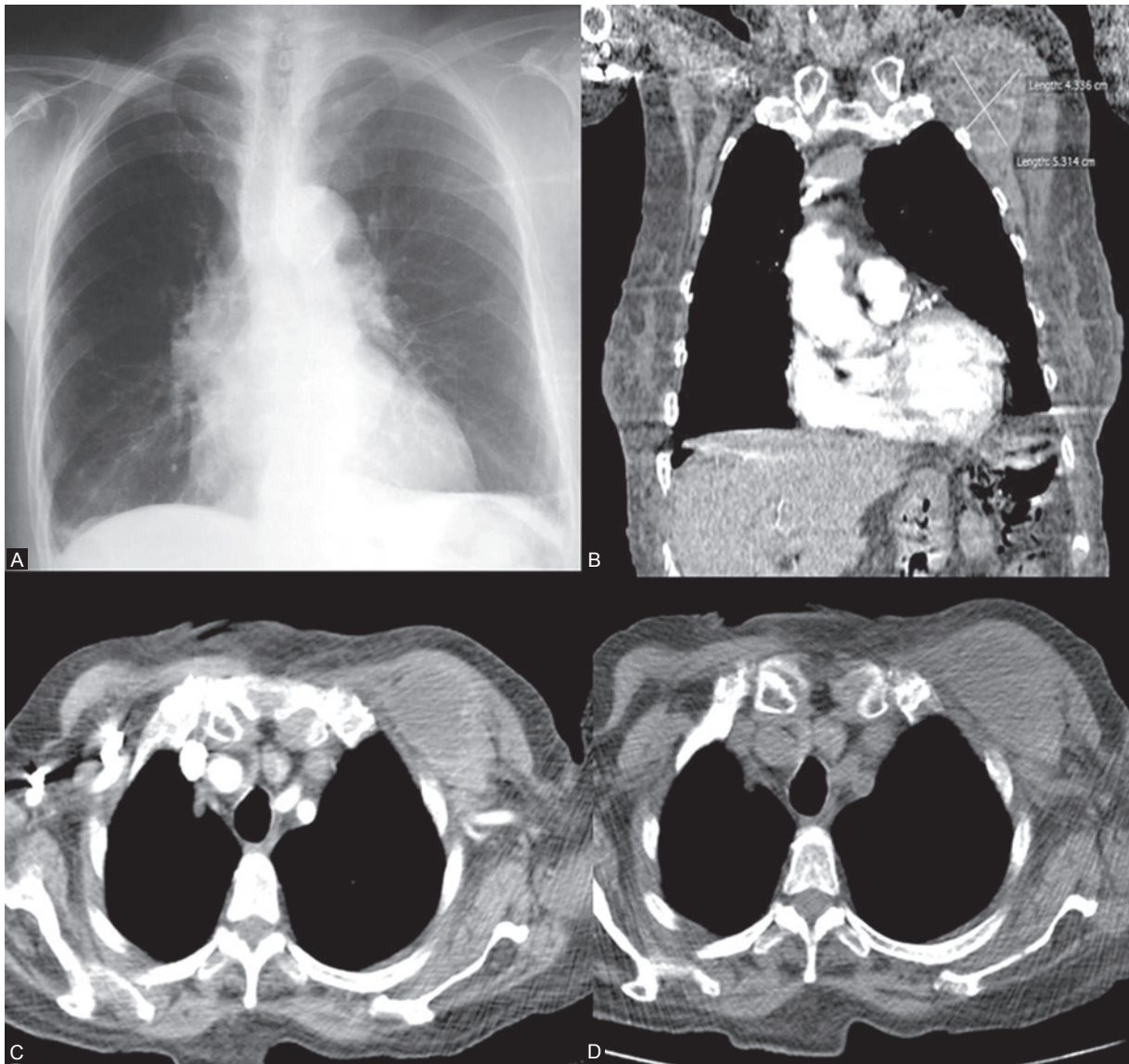


Figure 2. A: Chest X-ray with evident asymmetry between both infraclavicular regions and left clavicle upward shift. Note the increased density of the ipsilateral hemithorax upper portion. B: computerized axial tomography (CAT) coronal reconstruction showing the left infraclavicular region tuberculous abscess. CAT sections at simple (C) and contrasted (D) phases showing a left subpectoral collection with thick capsule that enhances in an annular shape after intravenous administration of non-ionic iodized contrast, of semi-liquid content.

breast and musculoskeletal tissues appear to be relatively resistant to TB⁶.

Clinically, it occurs as a painful, thick, soft or firm tumor, as in the case of our patient⁶. Other of the described systemic clinical manifestations are weight loss, fever, asthenia and nocturnal diaphoresis⁷.

Sometimes, abscesses can form fistulae towards the pleural cavity with underlying destruction of bone and cartilage. There are three mechanisms in the pathogenesis of a chest wall tuberculous abscess: 1) direct extension of underlying pleural or pulmonary TB, 2) direct extension of chest wall lymphadenitis and 3) hematogenous dissemination owing to the presence of latent TB¹⁰.

There is one classification by location and level of involvement on CAT scan: 1) lesion confined to the chest wall, 2) lesion confined to the interior chest wall below the ribs and also protruding at the pleural space and 3) lesion that compromises most part of the chest wall and that also protrudes at the pleural space¹⁰.

Diagnosis of the disease is complicated, since TB is known as “the great pretender” and its clinical manifestations occur according to the affected organ. Owing to this, the possibilities of abscess versus neoplasm were the two main probable diagnoses¹¹. Nonaka et al. reported AARB positivity only in 35% of cases and positive cultures only in 60% of patients with chest wall TB¹².

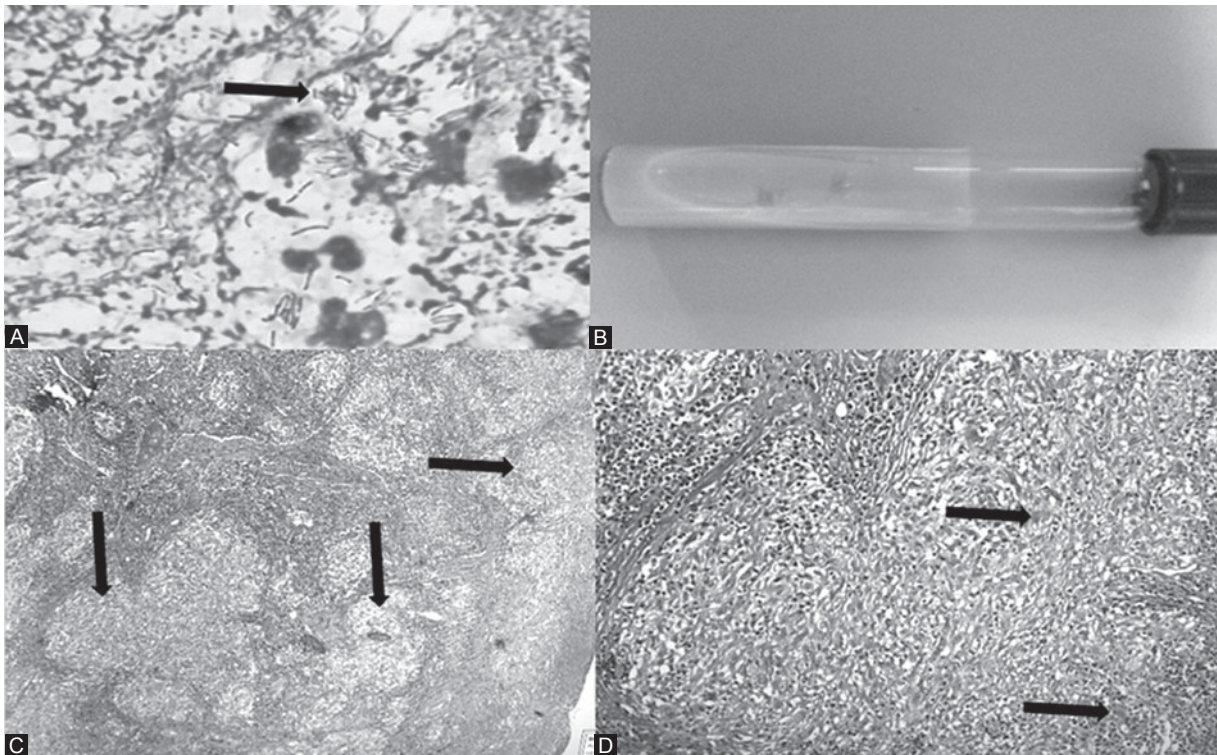


Figure 3. A: Ziehl-Neelsen staining with abundant AARB (100X lens with immersion oil). **B:** positive Löwenstein-Jensen culture. **C:** lymph node with architectural distortion caused by the multifocal granulomatous lesion (lens 5X). **D:** granulomatous lesion with giant multinucleated and epithelioid cells (lens 25X).

The treatment of chest wall tuberculous abscess is controversial, but the combination of medical and surgical treatment is the best approach with a recommended duration of 6 to 12 months of medical treatment with 2 months of daily quadruple antitubercular medications (INH, RIF, EMB and PZA) and 4 months of daily dual therapy with INH and RIF or thrice-weekly with at least two of the main antitubercular drugs prescribed during the course of treatment. There is currently no clear global consensus for treatment, including its duration^{5,7}.

Surgical treatment methods are controversial. The literature refers that this disease is best treated with antibiotics, drainage and debridement, and excision is reserved for most extended cases. Optimal surgical approach consists in extirpating the abscess with wounds primary closure, but the resection extent is not defined. However, extirpation and medical treatment with antitubercular drugs during the postoperative period are mandatory to achieve an acceptable cure rate and the prognosis is generally good if detection is opportunely made¹⁰.

Conclusion

Chest TB diagnosis is always difficult, since its clinical presentation often mimics pyogenic abscess or chest

wall tumors, but its likelihood should always be suspected even in the absence of a history of TB disease.

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