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CASE REPORT
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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2026, 43(Συμπλ 1):109–113

A tuberculous cold abscess masquerading as a chest wall swelling

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Tuberculosis (TB) is a chronic infectious disease caused by *Mycobacterium tuberculosis,* primarily affecting the lungs and can also present as extrapulmonary TB. Among extrapulmonary forms, musculoskeletal TB is relatively rare. Herein, we report a case of a 59-year-old female who presented with complaints of anterior chest wall swelling and shortness of breath on exertion. The patient underwent incision and drainage of the chest wall abscess. Pus culture showed no growth. Acid-fast bacilli and gene-Xpert are negative. A biopsy taken from the abscess wall showed necrotising granulomatous inflammation with Langerhans giant cells and multinucleated giant cells. The patient was started on empirical anti-tubercular therapy. Upon follow-up, the patient exhibited a favourable clinical response, with resolution of the swelling. This case highlights an unusual site of tuberculous cold abscess.

Ένα φυματιώδες ψυχρό απόστημα εμφανιζόμενο ως οίδημα του θωρακικού τοιχώματος

Περίληψη στο τέλος του άρθρου

Key words

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Tuberculosis (TB) is a leading cause of infectious disease killer in low-income and developing countries. It is a chronic infection caused by *Mycobacterium tuberculosis*. TB is a ubiquitous disease that can affect multiple organs. The lungs are usually the primary site of infection and can manifest as primary TB, reactivation TB, or endobronchial TB. Extrapulmonary tuberculosis (EPTB) can virtually affect any organ outside the lungs, such as the pleura, lymph nodes, abdomen, genitourinary tract, bones, meninges, etc. Cervical lymphadenopathy is the most common extrapulmonary manifestation of TB. Extrapulmonary TB has a myriad of presentations, accounting for late diagnosis and inevitable delay in treatment.[2] TB can present as a collection of pus called a cold abscess, lacking the signs of inflammation. It presents as a soft to firm mass that progressively increases in size, with or without destruction of the surrounding rib or cartilage, which is commonly seen in immunocompromised patients.[3] It is seen in lymph nodes, especially the cervical group of lymph nodes, spine, iliopsoas region, breast and chest wall. Skeletal tuberculosis accounts for about 1–5% of EPTB. A chest wall abscess accompanies about 0.1% of musculoskeletal TB. It presents in a non-specific, insidious manner, thus causing both a delay and difficulty in the diagnosis of the condition.

CASE PRESENTATION

A woman in her late fifties, who has recently been diagnosed with type 2 diabetes mellitus (T2DM), presented with complaints of breathlessness on exertion for six months and swelling over the anterior chest wall for two months. The swelling over the chest wall progressively increased in size over the past 2 months, associated with intermittent dull aching type of pain. The patient also reported weight loss of 4–5 kg over 3 months, myalgia and loss of appetite. She had fever for 2 weeks, which was low-grade, intermittent and relieved with tablet paracetamol, and shortness of breath which progressed from Modified Medical Research Council (mMRC) grade 2 to mMRC grade 3. The patient denied experiencing orthopnoea, paroxysmal nocturnal dyspnoea, cough, hemoptysis, palpitations, hoarseness of voice, dysphagia, or odynophagia.

On clinical examination, the patient's anthropometric measurements were as follows: height 152 cm and weight 101 kg. Notable findings included pallor and a right cervical lymphadenopathy. There was no evidence of icterus, cyanosis, clubbing, pedal oedema, or thyromegaly. Local examination revealed a 6×4 cm swelling over

the right anterior chest wall, soft to firm in consistency, without associated redness, tenderness, or cough impulse (fig. 1). Bilateral breast examination was unremarkable. Additionally, a 3×2 cm hyperpigmented, hyperkeratotic plaque was observed in the left gluteal region. Baseline investigations revealed normocytic anemia, secondary to chronic disease, and an elevated acute-phase reactants (tab. 1). Viral markers were non-reactive, and blood and urine cultures yielded no growth. Chest radiography was within normal limits. Electrocardiography showed normal sinus rhythm along with non-specific T wave changes. Computed tomography (CT) of the thorax revealed a well-defined lesion located in the deep subcutaneous plane of the right upper medial chest wall, with extension into the pectoralis musculature (fig. 2). No evidence of lung parenchymal mass, consolidation, mediastinal lymphadenopathy, pleural effusion, or pericardial effusion was observed. Contrast-enhanced computed tomography (CECT) of the neck showed a well-defined peripherally enhancing collection in the anterior chest wall, measuring 6×4.5×5.9 cm, noted to cause erosion of the medial end of the right clavicle (fig. 3). Other findings noted were hyper-dense foci-bony spicules in the abscess, right level-IV enlarged cervical lymph nodes and another peripherally enhanc-

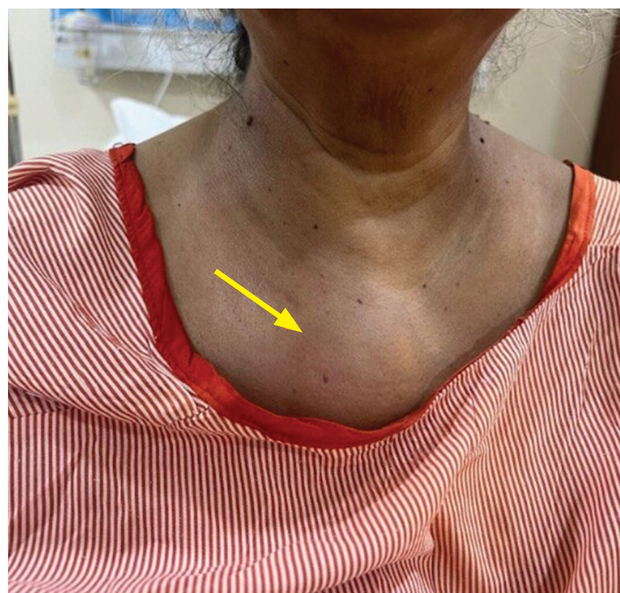

Figure 1. Right anterior chest wall globular swelling measuring approximately 6×4 cm, without redness or induration (arrow).

Table 1. Baseline blood investigations, including viral markers and acute-phase reactants.

Parameter	Patient value	Reference range
Hemoglobin	7.3	12–15 g/dL
Mean corpuscular volume	89.4	83–101 fL
Total white cell count	7,430 (polymorph: 74%, lymphocytes: 14%)	4,000–11,000/µL (polymorphs: 45–70%, lymphocytes: 25–40%)
Platelet count	3,810,000	150,000 to 450,000/µL
Blood urea nitrogen	6	7–18 mg/dL
Creatinine	0.7	0.6–1.3 mg/dL
Sodium	136	134–144 mmoL/L
Potassium	4.3	3.5–5 mmoL/L
Chloride	101	96–108 mmoL/L
Bicarbonate	25	21–29 mmoL/L
Uric acid	6.6	2.6–7.2 mg/dL
AST	18	0–35 IU/L
ALT	14	0–41 IU/L
Alkaline phosphatase	88	45–129 IU/L
Albumin	3.9	3.2–4.8 g/dL
Total bilirubin	0.35	0.1–1.2 mg/dL
LDH	176	100–190 IU/L
HbA1c	6.3	4–6%
HBsAg	Negative	Negative
Anti-HCV	Negative	Negative
HIV-1/2 with p24Ag capture assay	Negative	Negative
ESR	54	4–19 mm/hour
CRP	5.1	<0.6 mg/dL

ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein; LDH: Lactate dehydrogenase; HBsA: Hepatitis B surface antigen

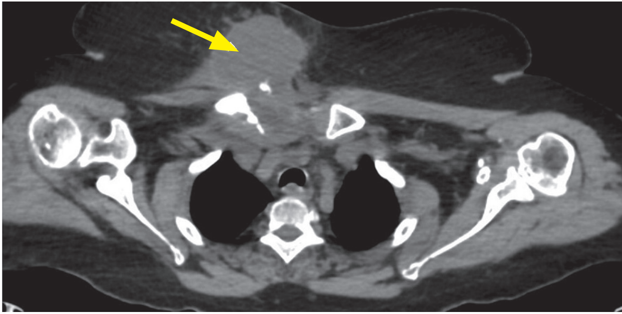

Figure 2. Axial non-contrast computed tomography (CT) thorax shows a well-defined lesion involving the deep subcutaneous plane of the right upper medial chest wall with extension into the pectoralis muscles.

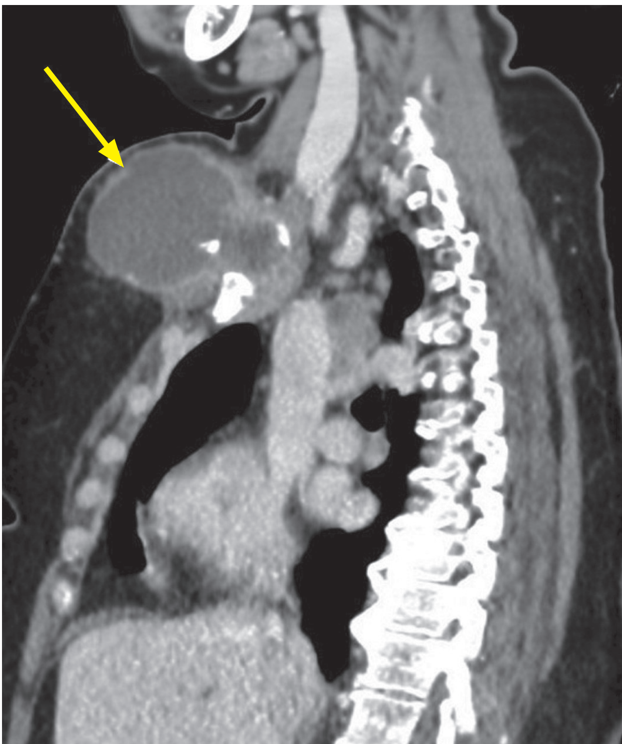

Figure 3. Computed tomography (CT) thorax **(**sagittal view) post-contrast images show the lesion with central areas of necrosis and a thick peripherally enhancing capsule.

ing soft tissue density collection measuring 2.7×2.8×3 cm. General surgery opinion was taken, and incision and drainage of the chest wall abscess under general anaesthesia was done; 40 mL of pus was drained. Pus culture showed no growth. Acid fast bacilli and gene-Xpert for *M. tuberculosis* are negative. Biopsy taken from the abscess wall showed necrotising granulomatous inflammation with multinucleate giant cells, and stains for acid fast bacilli and fungal smear were negative. Positron emission computed tomography (PET-CT) whole body showed a small collection extending from the right sternoclavicular joint, forming a sinus tract extending to the skin surface with destruction of the right sternoclavicular joint, permeative lytic lesion in the superior and lateral walls of the right maxillary sinus and mediastinal lymphadenopathy.

In view of the histopathological findings –negative for malignancy and consistent with necrotising granulomatous inflammation– and a negative acid-fast bacilli smear, empirical antituberculous therapy (ATT) was initiated with informed verbal patient consent. The patient was started on isoniazid (300 mg), rifampicin (600 mg), pyrazinamide (1,500 mg), ethambutol (1,000 mg) along with pyridoxine 40 mg once daily. Her liver function parameters and uric acid were monitored regularly on follow-up. Serial monitoring of liver function tests, serum uric acid, blood sugars was done. Oral hypoglycaemic agents, along with premix insulin, were initiated for T2DM. Post-initiation of ATT, defervescence was noted, and resolution of swelling size was observed at 2 months after therapy. This case report highlights the presentation of a cold abscess in an unusual site of the chest wall.

COMMENTS

Skeletal tuberculosis is a rare manifestation of TB, accounting for about 1–5% of presentations. A chest wall abscess accompanies about 0.1% of musculoskeletal TB.[4] It is often mistaken to be a pyogenic abscess or soft tissue tumour. TB would not be considered the first differential in the background of a lack of pulmonary involvement. It would lead to delays in diagnosis and treatment, paving the way for the formation of deformities. The important risk factors for TB include lower socioeconomic status, malnourishment, diabetes mellitus, smoking, and immunocompromised states like human immunodeficiency virus infection (HIV).[1] Compared to females, males are affected twice, with the predominant age being 15 to 30 years. Chest wall TB can affect all anatomical structures, but typically affects the sternal margins and body of the ribs.[5,6] It presents as predominantly painless swelling, gradually progressively increasing in size with no signs of inflammation and bone pain, with minimal constitutional symptoms. Complications may include secondary infections, rib or sternum fractures, and compression or erosion of major blood vessels. Commonly considered differentials include other granulomatous diseases, chronic infections including fungal and parasitic infections and tumours of soft tissue, bone and cartilage. Musculoskeletal TB develops directly from the pleura or lung parenchyma, bloodstream spread or spread through lymphatics. Infection reaches the bone through the blood or lymphatics, where the metaphyseal part of the bone is infected first, owing to its rich blood supply. TB bacilli invade the end artery, causing end arteritis, resulting in bony destruction at the epiphysis. Once through the epiphysis, bacilli can either drain into a joint space, causing arthritis or form a sinus tract. *M. tuberculosis*

doesn't produce cartilage-destroying enzymes. Untreated infections may lead to "cold" abscesses around joints or bones. These abscesses can rupture, creating sinus tracts; a hallmark of musculoskeletal TB. Without timely initiation of treatment, a fistula can form rarely.

Radiological investigations, including X-ray, CT and magnetic resonance imaging (MRI) aid in evaluation and do not help to clinch the diagnosis. The diagnosis can be made with the identification of acid-fast bacilli in acid-fast stain or biopsy specimens, or cultures. Needle aspiration and biopsy can confirm the diagnosis by revealing caseating granuloma and acid-fast bacilli (AFB). The formation of granuloma is based on a delayed type of hypersensitivity reaction. The activated macrophages around the centre of the lesion neutralize tubercle bacilli without causing further tissue destruction. The necrotic material in the central part results in caseous necrosis. A weak macrophage response allows for tissue destruction to set in. The lesion increases in size, and the central necrosis liquefies, containing a large number of tubercular bacilli. Thus, a predominantly granulomatous lesion will yield little or no AFB, whereas a predominantly caseous necrotic lesion with no co-existent granuloma will show more AFB. Thus, there exists an inverse relation between granuloma and AFB.[7]

Samples obtained from these sites tend to be paucibacillary and thus have low sensitivity. Conventional smear microscopy has a sensitivity in the range of 0–40%, whereas the yields of mycobacterium culture vary from 30% to 80%, but it takes a few weeks for results, leading to a delay in diagnosis.[8] Nucleic acid amplification tests like Xpert MTB/RIF, commonly used in the diagnosis of pulmonary TB, may lack sensitivity in the setting of extrapulmonary TB, thus, a negative result doesn't rule out TB. The above factors lead to difficulty in establishing a diagnosis, and the patient is treated under high clinical suspicion. The management of skeletal TB includes a combination of both medical and surgical management. A needle aspiration should be performed, followed by anti-tuberculous therapy. Surgery should be considered if the lesion doesn't resolve or worsens after 1 to 3 months of medication. A radical excision of the swelling is commonly performed.[9] A 6-month treatment regimen may be suitable for patients who have undergone successful wide excision. For patients who have not had surgery, a 9- to 12-month course of medication is typically prescribed, though the optimal duration of chemotherapy has not been established.

In conclusion, chest wall cold abscess, which is a rare manifestation of TB, poses a significant diagnostic challenge due to its nonspecific presentation and often absent pulmonary involvement. Early recognition through imaging and confirmatory biopsy, followed by timely anti-tuberculous therapy, is essential to prevent severe complications such as bone destruction and deformity. Awareness of this entity is crucial for prompt diagnosis and effective management.

ΠΕΡΙΛΗΨΗ

Ένα φυματιώδες ψυχρό απόστημα εμφανιζόμενο ως οίδημα του θωρακικού τοιχώματος

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Η φυματίωση είναι μια χρόνια μολυσματική νόσος που προκαλείται από το *Mycobacterium tuberculosis,* το οποίο προσβάλλει κυρίως τους πνεύμονες, αλλά ενδέχεται επίσης να εμφανιστεί και ως εξωπνευμονική μορφή. Μεταξύ των εξωπνευμονικών μορφών, η μυοσκελετική φυματίωση είναι σχετικά σπάνια. Παρουσιάζεται η περίπτωση μιας 59χρονης γυναίκας που παρουσίασε οίδημα του πρόσθιου θωρακικού τοιχώματος και δύσπνοια κατά την άσκηση. Η ασθενής υποβλήθηκε σε παροχέτευση του αποστήματος του θωρακικού τοιχώματος. Η καλλιέργεια πύου ήταν αρνητική, καθώς και η αναζήτηση οξεάντοχων βακίλλων και η δοκιμασία gene-Xpert. Η ιστολογική εξέταση του τοιχώματος του αποστήματος έδειξε νεκρωτική κοκκιωματώδη φλεγμονή με παρουσία γιγάντιων κυττάρων Langerhans και πολυπύρηνα γιγαντιαία κύτταρα. Η ασθενής άρχισε εμπειρική αντιφυματική θεραπεία και εμφάνισε καλή κλινική ανταπόκριση, με υποχώρηση του οιδήματος. Επρόκειτο για μια ασυνήθιστη εντόπιση φυματιώδους ψυχρού αποστήματος.

Λέξεις ευρετηρίου: Απόστημα, Εξωπνευμονική, Οίδημα, Φυματίωση

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