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Case Report

Postauricular Tuberculous Lymphadenitis in A High-Risk Patient: A Diagnostic Challenge

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ABSTRACT

Background: Extrapulmonary tuberculosis (EPTB) accounts for 15–20% of tuberculosis cases, with lymph node involvement being the most common form. Postauricular lymphadenitis is a rare presentation and often mimics bacterial abscesses or neoplastic conditions, delaying diagnosis.
Case Report: We describe a 65-year-old female with type 2 diabetes mellitus, chronic kidney disease, coronary artery disease, hypertension, dyslipidemia, and a history of left MCA infarct, who presented with a painful postauricular swelling and fever. Investigations revealed anemia, leukocytosis, elevated ESR and CRP, renal dysfunction, and poor glycemic control. Ultrasound of the local site showed necrotic, matted lymph nodes. FNAC showed reactive lymphadenitis, but incision and drainage of pus revealed acid-fast bacilli, confirming tuberculous lymphadenitis. The patient was managed with anti-tubercular therapy (ATT) as per NTEP guidelines, along with antibiotics, incision and drainage, and optimization of comorbidities.
Conclusion: Postauricular tuberculous lymphadenitis is an uncommon form of EPTB. High clinical suspicion, microbiological confirmation, and early ATT initiation are essential for Favourable outcomes, particularly in patients with multiple comorbidities.

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INTRODUCTION

Tuberculosis (TB) remains a major global health challenge, with extrapulmonary TB (EPTB) contributing to 15–20% of cases. Among EPTB, lymph node tuberculosis is the most frequent manifestation, commonly affecting cervical and supraclavicular regions. Postauricular involvement is rare and may mimic bacterial abscess, fungal infection, or malignancy. Timely diagnosis is difficult, particularly in patients with comorbidities such as diabetes mellitus (DM) and chronic kidney disease (CKD), which impair immune response. Here, we report a rare case of postauricular tuberculous lymphadenitis in a patient with multiple comorbidities.

CASE REPORT

A 65-year-old female presented with a one-week history of swelling behind the left ear, which was insidious in onset, progressively increasing in size, and associated with a painful, pus-filled lesion. She also reported intermittent low-grade fever for two days. History was significant for an acute left MCA infarct (15/03/25) without haemorrhagic transformation, and she was a known case of coronary artery disease, type 2 diabetes mellitus, hypertension, chronic kidney disease, and dyslipidemia. There was no prior history of tuberculosis. On general examination, she was conscious, oriented, with mild pallor but no icterus, cyanosis, clubbing, generalized lymphadenopathy, or pedal edema. Systemic examination revealed stable cardiovascular, respiratory, abdominal, and neurological systems. Local examination showed a diffuse, tender, indurated 8x5 cm swelling in the left postauricular region, with warmth and active discharge.

Investigations revealed anemia (Hb 9.1 g/dL), neutrophilic leucocytosis (total count 28,500 → 11,800 post-treatment), elevated inflammatory markers (CRP 120 mg/L, ESR 85 mm/hr), deranged renal function (urea 308 mg/dL, creatinine 3.85 mg/dL), proteinuria and glycosuria, mildly abnormal liver function tests, dyslipidemia, and poor glycemic control (HbA1c 11.1%). Echocardiography showed CAD with mild LV dysfunction (EF 45%). Ultrasound of the abdomen revealed bilateral grade 1 renal changes with a left renal microlith, while ultrasound of the local site demonstrated multiple necrotic, matted lymph nodes with surrounding edema. FNAC shows reactive lymphadenitis, whereas pus obtained after incision and drainage was positive for acid-fast bacilli. CBNAAT was done from pus aspirated from the swelling, which was negative, and pus culture showed no growth of non-tuberculous mycobacteria or Nocardia, confirming tuberculous lymphadenitis. She was initiated on anti-tubercular therapy. After a week of initiation of anti-tubercular therapy, the patient improved symptomatically, and the lesion started to regress. She was continued on anti-tubercular therapy and is in a follow-up period.

Fig 1: LYMPHNODE

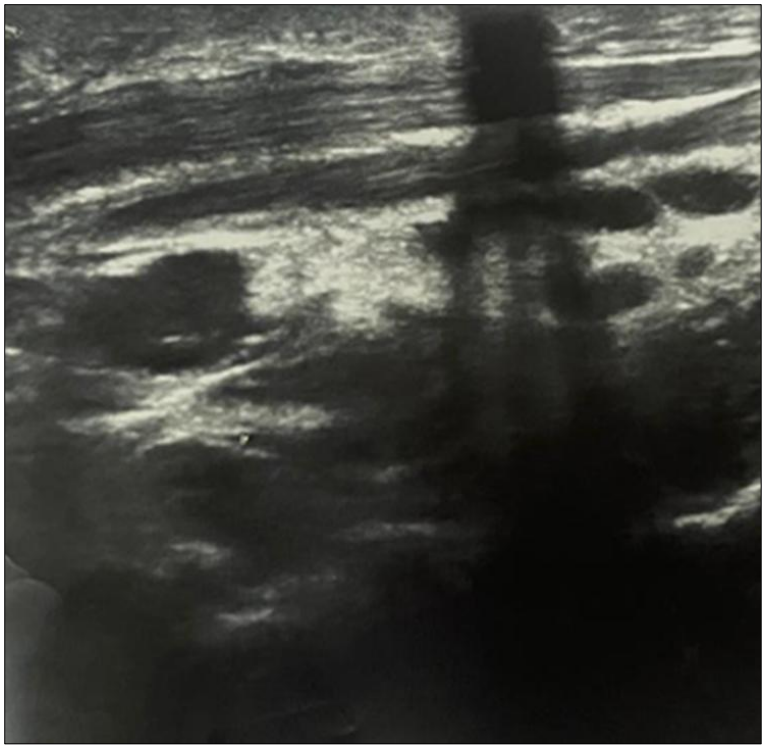

Fig 2: USG Neck

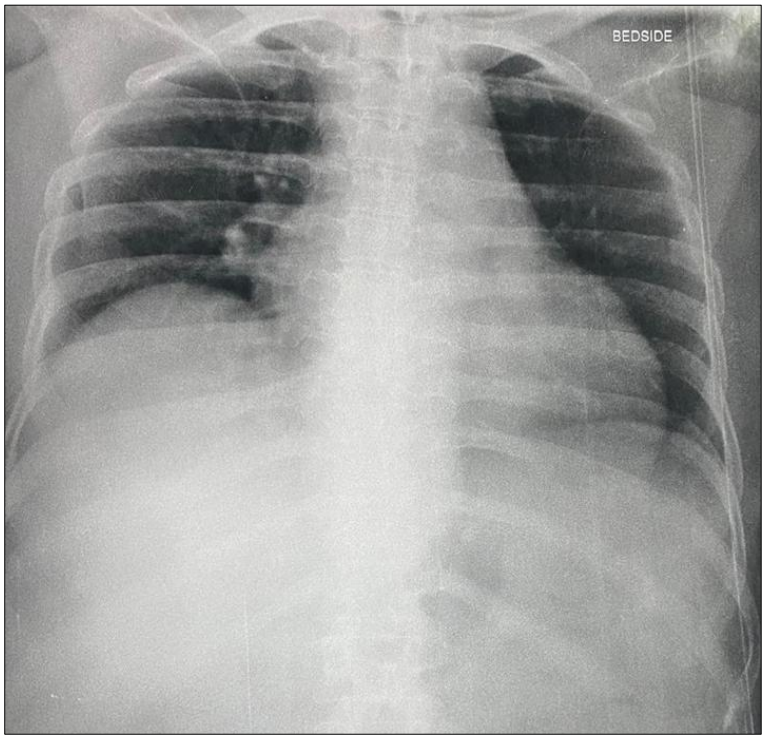

Fig 3: Chest X-ray

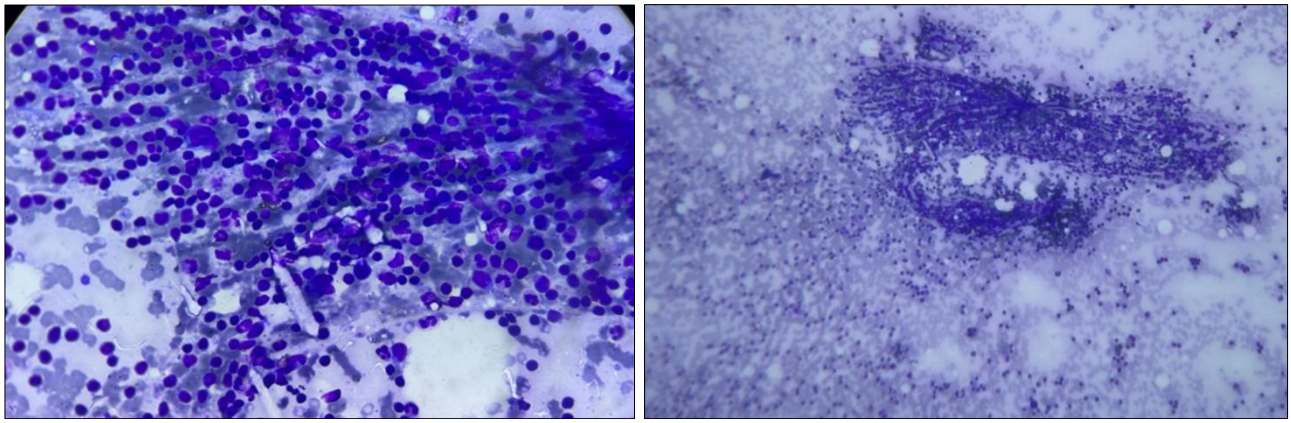

Fig 4: Histopathological examination

Light Microscopy (MGG, H&E Stain)
USG guided FNAC from left cervical lymph node swelling is sparsely cellular, showing a polymorphous population of lymphoid cells, such as small lymphocytes, centrocytes, mitotic activity present, immunoblasts, and plasma cells in a haemorrhagic background.

DISCUSSION

Role of Lymph Nodes in Tuberculosis: Infection, Immunity, and Reactivation:

Early Events in MTB Infection: Inhaled droplet nuclei containing *Mycobacterium tuberculosis* (Mtb) reach the alveoli, where infection begins. Insights from Poulsen's Faroe Islands study (1950s) revealed that the incubation period is around 40 days between exposure and the first clinical sign, which is usually fever. Radiologically, hilar adenopathy and dense shadows were commonly observed and could persist for 1–2 years. Among tuberculin skin test (TST) converters, 64% showed hilar lymphadenitis (78% in children vs. 56% in adults). However, only about 10% of these individuals progressed to active TB, suggesting that most contain the infection effectively [1, 2].

Dynamics of Lymph Node Infection
Lymph nodes (LNs) act as long-term reservoirs for Mtb, with complete elimination being rare. Granulomas disrupt the LN architecture, leading to impaired antigen presentation. Unlike lung granulomas, LN granulomas lack B-cell–rich tertiary lymphoid structures, which reduces their ability to mediate bacterial killing. This difference in granuloma structure contributes to the persistence of infection within the nodes [3].

Immune Response in Lymph Nodes
Within LNs, both Th1 (IFN-γ, TNF, IL-2) and Th17 (IL-17) responses are activated. Interestingly, IL-10 production by CD11b+ cells is associated with bacterial clearance, whereas an excess of TNF-producing CD4+ T cells correlates with higher bacterial loads. Human studies of tuberculous lymphadenitis (TBLN) reveal an upregulation of inflammatory, TLR, and Th1-related transcripts, although some variability exists between adult and paediatric cases. Overall, a fine balance between pro-inflammatory and regulatory responses, particularly IL-10, is critical in determining disease outcome.

Reactivation
Evidence from both non-human primate (NHP) and human studies suggests that reactivation of TB often originates within lymph nodes. PET-CT imaging shows FDG-avid nodes as predictors of reactivation. During conditions such as SIV/HIV co-infection or TNF neutralization, new lung granulomas frequently arise from LN seeding, highlighting their role in reactivation dynamics [4, 5].

Diagnosis of extrapulmonary tuberculosis (EPTB)
remains a clinical challenge due to its protean manifestations and the relatively low bacillary load in affected tissues, which makes direct smear demonstration of acid-fast bacilli difficult. Clinical features largely depend on the organ system involved, and hence, a high index of suspicion is required. A range of investigations may assist in diagnosis, including fine-needle aspiration cytology (FNAC) with direct smear examination, excision biopsy with histopathology, cytology and biochemical analysis of body fluids, and imaging modalities such as X-ray and ultrasonography. In lymph node tuberculosis, FNAC has a diagnostic yield of up to 80%, while biopsy may reveal granulomas with or without caseous necrosis [6]. Although AFB smear positivity confirms TB, mycobacterial culture remains the gold standard but is limited by delayed results.
In recent years, molecular techniques such as Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) and line probe assays have gained importance, offering rapid detection of *M. tuberculosis* and rifampicin resistance. According to national guidelines, CBNAAT on extrapulmonary specimens can establish a microbiologically confirmed diagnosis if positive, while indeterminate or invalid results require repeat testing or referral for liquid culture and drug susceptibility testing. In cases where microbiological confirmation is not possible, diagnosis relies on clinical, radiological, histopathological, and

immunological evidence, and such patients are classified as clinically diagnosed EPTB [7].

Among the various sites of EPTB, lymph nodes are the most commonly affected, followed by pleura, genitourinary tract, bones/joints, meninges, peritoneum, and pericardium. Tuberculous lymphadenitis typically evolves from discrete nodes to matted necrotic nodes with or without sinus formation. Classically painless, they may present with tenderness when secondary infection occurs. Differential diagnoses include infectious conditions such as bacterial abscesses and fungal infections, neoplastic causes like lymphoma or metastatic carcinoma, and rare entities such as Kikuchi's disease, Kimura's disease, and Castleman's disease. Delay in diagnosis can lead to life-threatening outcomes. Therefore, early suspicion, appropriate use of molecular diagnostics, and multidisciplinary evaluation are crucial in the management of EPTB.

CONCLUSION

Postauricular tuberculous lymphadenitis is a rare manifestation of EPTB. Clinicians must maintain a high index of suspicion in patients with chronic necrotic lymphadenopathy, especially in endemic regions. Early microbiological confirmation and prompt ATT initiation are key to preventing complications, particularly in individuals with multiple comorbidities.

CONFLICTS OF INTEREST: Nil

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