

Tuberculous Mastoiditis with Parotid Swelling in a Child: A Case Report

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Abstract

Case Report

Background: Tuberculous mastoiditis is a rare manifestation of extrapulmonary tuberculosis, particularly in the pediatric population. Its clinical presentation may be insidious and atypical, mimicking chronic otitis media or parotid gland pathology, which can significantly delay diagnosis. **Case Presentation:** We report the case of a 6-year-old child who presented to our otorhinolaryngology department with a fistulized right mastoiditis, a pre-fistulized parotid swelling, and fistulized right laterocervical lymphadenopathy evolving over several months. Biological workup revealed elevated inflammatory markers and a positive tuberculin skin test. Cervicofacial ultrasound demonstrated a heterogeneous collection in the parotid region with adjacent necrotic lymphadenopathy, and magnetic resonance imaging (MRI) of the temporal bone and neck revealed opacification of the right middle ear and external auditory canal with a retroauricular fistulous tract, and multiple necrotic intraparotid and jugulocarotid lymph nodes with skin fistulization. Histopathological examination of a biopsy specimen obtained through the mastoid fistula confirmed the diagnosis of tuberculosis, showing epithelioid granulomas with central caseous necrosis. The patient was referred for standard antitubercular therapy and demonstrated favorable clinical outcome at follow-up. **Conclusion:** This case highlights the importance of considering tuberculosis in the differential diagnosis of pediatric mastoiditis presenting with parotid and laterocervical lymphadenopathy, even in the absence of classic pulmonary features. Early histopathological analysis through accessible fistulous tracts can expedite diagnosis and avoid unnecessary surgical procedures.

Keywords: tuberculous mastoiditis; extrapulmonary tuberculosis; laterocervical lymphadenopathy; parotid lymphadenopathy; pediatric; fistula; case report.

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1. INTRODUCTION

Tuberculosis (TB) remains a major global health concern, with the World Health Organization estimating approximately 10 million new cases annually. While pulmonary TB is the most prevalent form, extrapulmonary manifestations account for 15–20% of all cases, with the proportion being even higher in children and immunocompromised individuals. Among extrapulmonary sites, head and neck involvement — including cervical lymphadenitis, laryngeal TB, and sinonasal TB — is well documented, yet mastoid tuberculosis remains exceedingly rare.

Tuberculous otomastoiditis was first described in the 19th century but has since become an uncommon entity in high-income countries following the widespread implementation of vaccination and antitubercular therapy programs. However, its incidence persists in endemic regions, and cases continue to be reported

worldwide, often presenting diagnostic challenges due to atypical clinical features.

Tuberculous otomastoiditis typically presents with otorrhea, tympanic membrane perforation, conductive hearing loss, and perimastoid swelling, often with a protracted course unresponsive to conventional antibiotic therapy.

We report a case of tuberculous mastoiditis in a 6-year-old child who presented with a fistulized right mastoiditis, a pre-fistulized parotid swelling with disrupted overlying skin, and fistulized right laterocervical lymphadenopathy, in whom diagnosis was established by histopathological analysis of a biopsy specimen through the fistulous tract, and who responded favorably to antitubercular therapy.

2. CASE PRESENTATION

A 6-year-old child, with no significant past medical history and no known contact with active tuberculosis, was referred to our otorhinolaryngology department for evaluation of a fistulized right mastoiditis associated with a pre-fistulized parotid swelling and fistulized right laterocervical lymphadenopathy, evolving over approximately four months. The lesions were painless and the patient had no history of trauma, prior ear surgery, or systemic symptoms such as fever, night sweats, or weight loss at the time of presentation. There was no reported odynophagia or trismus.

On clinical examination, a tender, indurated swelling was noted in the right parotid region extending anteriorly and toward the mastoid process, with a fistulous opening identified over the mastoid cortex with seropurulent discharge and granulation tissue. Multiple firm, non-tender fistulized lymph nodes were palpated along the right laterocervical chain. Otoloscopic examination of the right ear revealed a central tympanic membrane perforation with mucopurulent otorrhea. Facial nerve function was normal.



Figure 1: Clinical appearance at presentation: fistulized mastoid and upper cervical swelling with crusted seropurulent discharge, granulation tissue, and a pre-fistulized parotid region swelling with disrupted overlying skin

2.1 Investigations

Laboratory workup revealed a mildly elevated erythrocyte sedimentation rate (ESR) of 58 mm/h and C-reactive protein (CRP) of 18 mg/L. White blood cell count was 9,200/mm³ with lymphocytic predominance. Hemoglobin was 11.2 g/dL, consistent with anemia of chronic disease. Mantoux tuberculin skin test (TST) was strongly positive, with an induration of 22 mm at 72 hours. HIV serology was negative.

Cervicofacial ultrasonography revealed a heterogeneous hypoechoic collection measuring 38 × 25 mm in the right parotid region, with internal echogenicities suggestive of a necrotic or suppurative process. Posterior acoustic enhancement was noted, and the lesion appeared to communicate with the mastoid cortex via a fistulous tract.

Magnetic resonance imaging (MRI) of the temporal bone and neck was performed with gadolinium contrast, using axial T2, coronal T2, sagittal T1, FLAIR,

DWI, and T1 post-contrast sequences. At the level of the right temporal bone, MRI demonstrated opacification of the middle ear and external auditory canal, with intermediate T2 signal and restricted gadolinium enhancement, associated with thickening and enhancement of the external auditory canal walls. A fistulous tract was individualized, originating from the external auditory canal and directed toward the retroauricular skin. The inner ear structures, semicircular canals, vestibule, and cochlea showed normal T2 signal. No labyrinthine enhancement or acoustic nerve abnormality was identified. At the cervicofacial level, multiple right intraparotid and jugulocarotid lymph nodes were identified, containing central T2 hyperintense areas consistent with necrosis, the most prominent measuring 15 × 13 mm in axial diameter, with one node fistulized to the skin. Intense enhancement of the right parotid gland parenchyma was noted. Bilateral high jugular and spinal lymphadenopathy was also observed. No intracranial extension was identified.

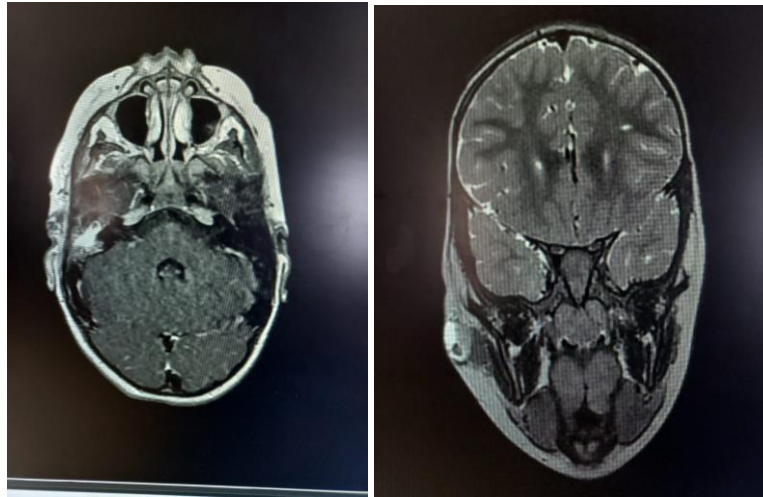


Figure 2: MRI of the temporal bone and neck (T1 post-gadolinium). Left: axial view showing opacification of the right middle ear and external auditory canal with restricted enhancement and individualization of a retroauricular fistulous tract. Right: coronal view demonstrating right intraparotid necrotic lymphadenopathy (15 × 13 mm) with parotid gland enhancement and skin fistulization

2.2 DIAGNOSIS

Given the clinical, radiological, and biological findings, a biopsy was performed through the mastoid fistulous tract under local anesthesia. The specimen was sent for histopathological examination and microbiological culture (including Löwenstein–Jensen medium for mycobacterial culture and GeneXpert MTB/RIF assay). Histopathological analysis revealed necrotizing epithelioid granulomas with Langhans-type multinucleated giant cells and areas of central caseous necrosis, consistent with tuberculosis. Ziehl–Neelsen staining demonstrated acid-fast bacilli within the granulomas. GeneXpert MTB/RIF assay confirmed the presence of *Mycobacterium tuberculosis* complex, with no detected rifampicin resistance. Mycobacterial culture subsequently yielded growth of *M. tuberculosis* at week 4, sensitive to all first-line agents (isoniazid, rifampicin, pyrazinamide, ethambutol).

2.3 Treatment and Outcome.

The patient was referred to the department of pneumology and phthysiology and initiated on standard four-drug antitubercular therapy (isoniazid, rifampicin, pyrazinamide, ethambutol) for an initial two-month intensive phase, followed by a four-month continuation phase with isoniazid and rifampicin, in accordance with WHO guidelines for extrapulmonary tuberculosis in children.

Clinical follow-up at two months demonstrated progressive reduction in parotid swelling and closure of the mastoid fistula, with resolution of ear discharge. At six-month follow-up, the swelling had completely resolved with no clinical evidence of disease relapse. No adverse effects related to antitubercular therapy were noted.



Figure 3: Post-treatment appearance at six-month follow-up: complete closure of the mastoid and cervical fistulae with residual scarring, and full resolution of parotid and laterocervical lymphadenopathy.

3. DISCUSSION

Tuberculous mastoiditis is an exceptionally rare form of extrapulmonary tuberculosis, with fewer than 200 cases reported in the modern literature. It typically occurs as a result of hematogenous dissemination of *Mycobacterium tuberculosis*, or, less commonly, via direct extension from an adjacent site of infection or from the middle ear through the Eustachian tube. The pathophysiology involves the formation of necrotizing granulomas within the mastoid air cell system, which may erode the bony cortex, creating fistulous tracts and periauricular collections.

The pediatric population appears particularly susceptible, possibly due to the relative immaturity of cellular immunity and the greater vascularization of the developing mastoid bone. In endemic regions, TB should be included in the differential diagnosis of any child presenting with a chronic mastoid or periauricular swelling, especially when the clinical course is protracted and unresponsive to conventional antibiotic therapy.

The diagnostic challenge in this case lies in the atypical presentation: the prominence of parotid and laterocervical lymphadenopathy as the chief complaint could have misled clinicians toward a primary lymph node pathology such as bacterial lymphadenitis, lymphoma, or non-tuberculous mycobacterial infection. The close anatomical proximity of the mastoid process and the parotid gland — separated only by the parotid-masseteric fascia and partially by the cartilaginous portion of the external auditory canal — facilitates contiguous spread of infection from the mastoid to the parotid and laterocervical lymph node chains.

MRI proved essential in this case by delineating the full anatomical extent of the disease: opacification of the right middle ear and external auditory canal with individualization of a retroauricular fistulous tract, multiple necrotic intraparotid and jugulocarotid lymph nodes with skin fistulization, and intense parotid gland enhancement, while excluding intracranial complications. Several authors have emphasized the superiority of MRI over CT in characterizing the soft tissue components and necrotic content of tuberculous cervicofacial lesions, as well as in identifying fistulous tracts and distinguishing tuberculous lymphadenopathy from other etiologies.

The diagnostic confirmation in this case was achieved through histopathological analysis of a biopsy specimen obtained via the pre-existing mastoid fistula. This minimally invasive approach, avoiding formal surgical exploration, proved both safe and diagnostically sufficient. The demonstration of necrotizing granulomas with Langhans giant cells, acid-fast bacilli on Ziehl–Neelsen staining, and the positive GeneXpert MTB/RIF assay provided unambiguous confirmation of tuberculosis. The GeneXpert assay, as a molecular

diagnostic tool, is increasingly recommended in pediatric TB given its high specificity and rapid turnaround time.

The favorable response to antitubercular therapy, with complete resolution of swelling and fistula closure within six months, further underscores the importance of early and accurate diagnosis. Unnecessary surgical procedures — including total parotidectomy, mastoidectomy, or drainage operations — can be avoided when tuberculous etiology is identified promptly, thus sparing the patient from the risks of facial nerve injury and anesthesia-related morbidity.

To our knowledge, the association of tuberculous mastoiditis presenting as fistulized mastoiditis with parotid and laterocervical lymphadenopathy in a young child, diagnosed through transdermal fistula biopsy, has been rarely described in the literature. Our case adds to the growing body of evidence supporting a low diagnostic threshold for tuberculosis in children presenting with atypical head and neck inflammatory lesions in endemic regions.

4. CONCLUSION

This case illustrates that tuberculous mastoiditis, though rare, should be included in the differential diagnosis of any young child presenting with fistulized mastoiditis, pre-fistulized parotid swelling, and fistulized laterocervical lymphadenopathy, even in the absence of overt pulmonary involvement. A systematic diagnostic approach combining clinical evaluation, cervicofacial ultrasound, MRI, tuberculin skin testing, and histopathological analysis of accessible specimens is essential to establish an early and accurate diagnosis. Antitubercular therapy remains the cornerstone of treatment, yielding excellent outcomes when initiated promptly.

Patient Consent: Written informed consent was obtained from the patient's legal guardian for the publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Conflicts of Interest: The authors declare no conflicts of interest.

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