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Refractory Otitis Media as an Early Manifestation of Systemic Tuberculosis: A Case-Based Diagnostic Approach

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Abstract

Background: Refractory otitis media represents a frequent yet challenging scenario in clinical practice, particularly when standard antimicrobial therapy fails. Although most cases are attributed to chronic inflammatory or infectious conditions, a subset may reflect underlying systemic disease. Tuberculous otitis media (TOM) is an uncommon but clinically significant entity, often underrecognized due to its atypical presentation and low diagnostic yield of local testing. This study aims to highlight a structured diagnostic approach to refractory otitis media, emphasizing the role of systemic evaluation. **Case Presentation:** We report the case of a 35-year-old man with a 6-month history of persistent otorrhea, aural fullness, and conductive hearing loss unresponsive to prolonged antibiotic therapy. Otoscopic examination revealed a thickened and retracted tympanic membrane without perforation, and temporal bone computed tomography showed soft tissue density in the middle ear and mastoid without bony erosion. Initial microbiological and histopathological analyses of middle ear specimens were inconclusive. Given the discordance between clinical evolution and local findings, a stepwise diagnostic reassessment was undertaken, including systemic evaluation. Chest imaging revealed findings suggestive of active pulmonary tuberculosis, and diagnosis was confirmed by polymerase chain reaction for *Mycobacterium tuberculosis* from bronchoalveolar lavage fluid. Anti-tuberculosis therapy was initiated, resulting in progressive clinical, endoscopic, and audiometric improvement, with complete resolution and no recurrence at 18 months. **Discussion:** This case underscores key diagnostic pitfalls in refractory otitis media, including over-

reliance on local testing and delayed consideration of systemic etiologies. Based on this experience and available literature, we propose a practical diagnostic framework incorporating clinical red flags, limitations of local microbiological studies, and indications for systemic screening. Early identification of TOM can prevent unnecessary surgical interventions and allow effective medical management. **Conclusion:** Refractory otitis media with atypical features should prompt a structured diagnostic approach that extends beyond the ear. Incorporating systemic evaluation into clinical decision-making is essential for timely diagnosis of tuberculous otitis media and for avoiding misdiagnosis and overtreatment.

Keywords

Tuberculosis, Pulmonary, Otitis Media, Hearing Loss, Conductive, *Mycobacterium tuberculosis*

1. Introduction

Otitis media encompasses a heterogeneous spectrum of inflammatory and infectious disorders of the middle ear and remains among the most frequently encountered conditions in otolaryngological practice. Although most episodes follow a predictable clinical course and respond to conventional medical or surgical management, persistent or refractory middle-ear disease constitutes a substantially different diagnostic scenario. Failure to improve despite appropriate antimicrobial therapy, recurrent or persistent otorrhea, progressive hearing impairment, granulation tissue, unexplained tympanic membrane abnormalities, or radiological findings disproportionate to the presumed diagnosis should prompt reconsideration of the initial etiological hypothesis. In such circumstances, the differential diagnosis must extend beyond conventional bacterial infection to include cholesteatoma, chronic inflammatory disorders, fungal and atypical infections, neoplastic processes, granulomatous diseases, autoimmune conditions, and systemic infections with secondary otologic involvement.

Tuberculosis remains one of the most consequential infectious diseases worldwide and represents a persistent diagnostic challenge because of its remarkable clinical heterogeneity and ability to involve virtually any organ system. Although pulmonary disease is its predominant manifestation, extrapulmonary tuberculosis comprises a broad spectrum of presentations that may occur independently or in association with active pulmonary infection. Tuberculous otitis media (TOM) is an uncommon extrapulmonary manifestation of *Mycobacterium tuberculosis* infection and represents only a small proportion of chronic middle-ear disease. Its clinical relevance, however, is disproportionate to its rarity because delayed recognition may result in progressive conductive, sensorineural, or mixed hearing loss; facial nerve paralysis; labyrinthine involvement; temporal bone destruction; and, in advanced disease, intracranial complications [1]-[3].

Historically, TOM has been associated with a classical clinical constellation of painless otorrhea, multiple tympanic membrane perforations, and facial nerve paralysis. However, contemporary series and systematic analyses demonstrate that this classical phenotype is inconsistently present and may be entirely absent, particularly during the early stages of disease. Instead, patients may initially present with nonspecific symptoms such as persistent otorrhea, aural fullness, conductive hearing loss, tympanic membrane thickening, middle-ear effusion, granulation tissue, or findings indistinguishable from conventional chronic otitis media or cholesteatoma [1] [3] [4]. This phenotypic variability represents one of the major reasons for diagnostic delay and frequently leads to repeated courses of empirical antibiotics, prolonged observation, and occasionally unnecessary surgical intervention before the underlying mycobacterial etiology is recognized.

The pathogenesis of middle-ear tuberculosis is likely heterogeneous and may vary according to age, host immune status, pulmonary disease burden, and anatomical factors. Several routes of infection have been proposed, including hematogenous dissemination from a distant tuberculous focus, retrograde spread through the Eustachian tube following exposure to infected respiratory secretions, direct extension from adjacent anatomical structures, and, less commonly, direct inoculation through a pre-existing tympanic membrane perforation [2] [5]. The presence of concomitant pulmonary tuberculosis may therefore provide a crucial diagnostic clue but is neither universally present nor required to establish the diagnosis of TOM. Conversely, otologic manifestations may occasionally precede recognition of systemic tuberculosis, creating a particularly challenging diagnostic sequence in which the ear represents the first clinically apparent site of disease.

The otoscopic appearance of TOM is similarly heterogeneous and evolves throughout the course of the disease. Multiple tympanic membrane perforations, traditionally regarded as a hallmark of the condition, may coalesce into a single larger perforation or may never develop. Some patients present with an intact but thickened or retracted tympanic membrane, persistent middle-ear effusion, pale granulation tissue, or polypoid changes. In early-stage disease, the clinical picture may be particularly subtle, and molecular testing may identify *M. tuberculosis* before the development of more characteristic destructive or granulomatous changes. Clinical studies examining the spectrum of TOM have demonstrated that polymerase chain reaction may be particularly valuable in early disease, including patients presenting with middle-ear effusion behind an intact tympanic membrane [4] [6].

Radiological assessment is essential for characterizing the extent of middle-ear and temporal bone involvement but generally lacks pathognomonic findings. High-resolution computed tomography may demonstrate soft-tissue density within the tympanic cavity and mastoid, variable preservation or destruction of the ossicular chain, mastoid opacification, cortical erosion, or more extensive temporal bone involvement. Importantly, the absence of bony erosion does not exclude TOM, particularly in earlier stages of the disease. Clinical-radiological studies have emphasized the substantial overlap between TOM and other forms

of chronic otitis media, reinforcing that imaging findings must be interpreted in conjunction with otoscopy, audiological evolution, microbiological results, histopathology, and systemic evaluation [7] [8].

The diagnosis is further complicated by the limitations of local microbiological and histopathological testing. Otologic specimens are frequently paucibacillary, resulting in low sensitivity of direct acid-fast bacilli staining. Mycobacterial culture remains microbiologically important but requires prolonged processing and may yield negative results because of inadequate sampling, previous antimicrobial exposure, secondary bacterial contamination, or a low local mycobacterial burden. Histopathological examination may reveal epithelioid granulomas, Langhans-type giant cells, and caseous necrosis, but these findings are not invariably present and may vary according to disease stage and specimen quality. Molecular methods, particularly polymerase chain reaction-based detection of *M. tuberculosis*, have substantially improved diagnostic capabilities and may provide greater sensitivity in selected clinical settings. Nevertheless, no single diagnostic modality is sufficiently sensitive to exclude TOM in every patient, and negative local investigations should not terminate the diagnostic process when the clinical trajectory remains atypical [1] [4] [6] [9].

This limitation becomes particularly important when otologic disease precedes recognition of tuberculosis elsewhere in the body. An exclusively ear-centered diagnostic approach may create false reassurance after negative local cultures, stains, biopsies, or molecular tests. In contrast, persistent discordance between the expected natural history of conventional otitis media and the observed clinical evolution should trigger broader diagnostic reassessment. Failure of adequate antimicrobial therapy, persistent or recurrent otorrhea, unexplained conductive or mixed hearing loss, granulation tissue, cranial neuropathy, atypical radiological findings, epidemiological risk factors, constitutional symptoms, or evidence of disease outside the temporal bone should be regarded as diagnostic red flags warranting consideration of atypical infections and systemic evaluation [1] [3] [5] [9].

From an otological perspective, early recognition has direct therapeutic consequences. Although surgery remains appropriate for selected complications, extensive destructive disease, diagnostic tissue acquisition when less invasive methods are nondiagnostic, or management of structural sequelae, uncomplicated TOM is fundamentally a medically treatable infectious disease. Delayed identification may expose patients to repeated antimicrobial regimens or surgical procedures that fail to address the underlying pathological process. Several reports and clinical series have documented patients undergoing mastoidectomy or tympanoplasty under alternative preoperative diagnoses, with tuberculosis only subsequently recognized through histopathological examination or additional systemic investigation [2] [3] [5] [10].

Appropriate antituberculous therapy generally results in progressive resolution of otorrhea and middle-ear inflammation, although auditory recovery depends on the type, severity, and duration of hearing impairment and on the presence of irreversible ossicular, cochlear, or neural damage. Early diagnosis is therefore es-

sential not merely for microbiological cure but also for preservation of hearing and prevention of facial nerve dysfunction, temporal bone destruction, and intracranial extension [1] [3] [7].

The contemporary diagnostic challenge is therefore not simply to recognize a predefined constellation of classical manifestations, but rather to identify tuberculosis within the broader and more heterogeneous population of patients presenting with refractory middle-ear disease. This requires an integrated diagnostic strategy incorporating serial otoscopic findings, audiological assessment, temporal bone imaging, microbiological and histopathological studies, epidemiological context, response to previous therapy, and, critically, evidence of systemic disease. Such an approach is especially important in tuberculosis-endemic regions but remains relevant worldwide because of international migration, immunosuppression, socioeconomic disparities, and the persistent global burden of *M. tuberculosis* infection.

In this report, we describe a 35-year-old man with a six-month history of persistent otorrhea, aural fullness, and conductive hearing loss refractory to prolonged antimicrobial therapy. Otoscopic examination revealed an atypical middle-ear process, while temporal bone computed tomography demonstrated soft-tissue density involving the middle ear and mastoid without bony erosion. Initial microbiological and histopathological investigations of middle-ear specimens were inconclusive. The persistent discordance between the clinical evolution and nondiagnostic local findings prompted a broader systemic reassessment, ultimately revealing active pulmonary tuberculosis confirmed by polymerase chain reaction for *M. tuberculosis* in bronchoalveolar lavage fluid. Initiation of antituberculous therapy resulted in progressive clinical, otoscopic, audiometric, and radiological improvement, with complete resolution and no recurrence at 18 months.

Beyond documenting an uncommon manifestation of tuberculosis, this case illustrates a broader diagnostic principle of direct relevance to otological practice: **when refractory otitis media fails to behave as expected, the diagnostic field must extend beyond the temporal bone**. We therefore present a case-based diagnostic approach emphasizing clinical red flags, the limitations of exclusively local testing, and the circumstances in which systemic evaluation should become an integral component of the diagnostic pathway.

2. Case Presentation

A 35-year-old man presented to the otorhinolaryngology service with a six-month history of persistent unilateral otologic symptoms characterized by otorrhea, aural fullness, and progressive hearing impairment. He had no known history of immunosuppressive disease or previous tuberculosis and was not receiving corticosteroids, immunosuppressive agents, or other immunomodulatory therapies [**HIV serology performed during the diagnostic evaluation was nonreactive.**] He reported **[no known household or occupational contact with a patient with active tuberculosis]** and had no relevant chronic comorbidities. At initial presentation,

[he denied fever, night sweats, clinically significant weight loss, and other constitutional symptoms; respiratory symptoms were absent/minimal]. The patient lived in Colombia, a country in which tuberculosis remains endemic, which became epidemiologically relevant as the diagnostic evaluation progressed.

During the months preceding specialized evaluation, the patient had been treated for presumed bacterial otitis media with several courses of antimicrobial therapy. These included **[amoxicillin/clavulanate for 7 - 10 days, followed by an oral fluoroquinolone/alternative systemic antibiotic, together with topical fluoroquinolone ear drops]** [**A transient reduction in otorrhea occurred during treatment, but symptoms recurred shortly after antimicrobial discontinuation.]** Despite these sequential treatments, no sustained clinical response was achieved, and persistent otorrhea, aural fullness, and progressive subjective hearing impairment led to referral to the otology team.

At initial otologic assessment, examination of the affected ear demonstrated **hyperemia, thickening, and retraction of the tympanic membrane with purulent otorrhea**, without the multiple tympanic membrane perforations classically associated with tuberculous otitis media. No facial nerve dysfunction was identified. Serial photographic documentation was subsequently obtained throughout follow-up, providing a longitudinal record of the otoscopic findings from the initial presentation through 18 months after treatment (Figure 1(a)-(e)).

Pure-tone audiometry performed during the initial evaluation demonstrated conductive hearing loss in the affected ear, characterized by an air-bone gap consistent with middle-ear dysfunction. Serial audiometric examinations were subsequently obtained during follow-up to objectively assess changes in auditory function (Figure 1(f)-(h)).

As part of the diagnostic evaluation, computed tomography of the temporal bones was performed. Initial imaging demonstrated soft-tissue density occupying the middle-ear cavity and extending into the mastoid air-cell system, without significant ossicular or other bony erosion (Figure 1(i)). The absence of an erosive mass, together with the otoscopic findings, provided no radiological evidence strongly suggestive of cholesteatoma or an invasive neoplastic process.

Because of the persistence of otologic manifestations despite conventional antimicrobial therapy, additional local diagnostic studies were performed [**Middle-ear secretion/granulation tissue obtained from the affected ear]** was submitted for microbiological and histopathological evaluation [**Direct acid-fast bacilli staining was negative, mycobacterial culture showed no growth, and molecular testing for *Mycobacterium tuberculosis* in the local specimen was negative/not performed.]** Histopathological examination **[showed nonspecific chronic inflammatory changes without caseating granulomas, malignant cells, or fungal elements]** [**Routine bacterial and fungal cultures did not identify a pathogen capable of explaining the persistent clinical course.]** Thus, the local studies were considered nondiagnostic and did not provide direct microbiological confirmation of *M. tuberculosis* infection of the middle ear.

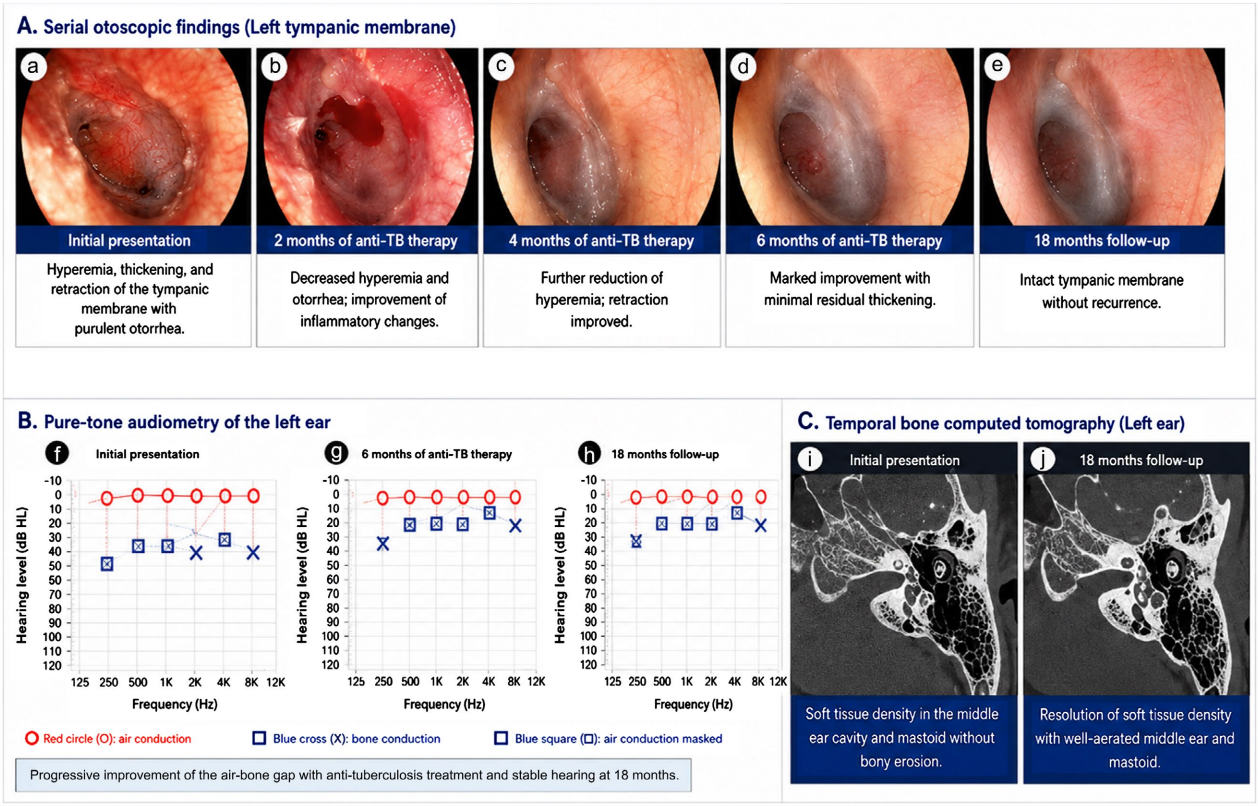

Figure 1. Clinical, audiometric, and radiological evolution of the left ear following anti-tuberculosis therapy. (A) Serial otoscopic findings of the left tympanic membrane: initial presentation and follow-up at 2, 4, 6, and 18 months, demonstrating progressive resolution of hyperemia, tympanic membrane thickening and retraction, and otorrhea, with restoration of an intact tympanic membrane and no recurrence at 18 months. (B) Pure-tone audiometry of the left ear: initial presentation, 6 months after initiation of anti-tuberculosis therapy, and 18-month follow-up, demonstrating progressive improvement of the air-bone gap and sustained hearing improvement. (C) Temporal bone computed tomography of the left ear: initial presentation showing soft-tissue density in the middle-ear cavity and mastoid without bony erosion, and 18-month follow-up demonstrating resolution of the soft-tissue density with restoration of middle-ear and mastoid aeration.

The combination of persistent symptoms, failure of appropriate conventional antimicrobial therapy, nondiagnostic local studies, and an atypical clinical course prompted expansion of the diagnostic evaluation beyond the temporal bone. Systemic assessment included chest imaging, which demonstrated **[upper-lobe/upper-lung parenchymal opacities with nodular/tree-in-bud changes ± cavitation, or insert the actual CT findings]** considered suspicious for active pulmonary tuberculosis. These findings prompted bronchoscopic evaluation with bronchoalveolar lavage.

Bronchoalveolar lavage fluid was subsequently analyzed using **[Xpert MTB/RIF or the actual molecular platform]**, which detected *Mycobacterium tuberculosis* complex DNA, thereby microbiologically confirming active pulmonary tuberculosis [**No rifampin resistance was detected by the molecular assay.] [Mycobacterial culture of the BAL specimen subsequently grew *M. tuberculosis*, with drug-susceptibility testing demonstrating susceptibility to first-line antituberculous agents.]** These systemic findings, considered together with the refractory

middle-ear disease and absence of a convincing alternative local diagnosis, supported a working diagnosis of **presumed tuberculous otitis media associated with microbiologically confirmed pulmonary tuberculosis**, rather than microbiologically confirmed tuberculous involvement of the middle ear.

Alternative causes of refractory unilateral middle-ear disease were reassessed during this process. Cholesteatoma was considered unlikely because otoscopy and temporal bone CT did not demonstrate the characteristic keratinizing lesion or associated bony erosion [**Fungal studies were negative and histopathological examination demonstrated no fungal organisms.] [Mycobacterial culture/molecular evaluation of the local specimen did not identify nontuberculous mycobacteria.]** Neoplastic disease was considered unlikely because imaging showed no destructive mass and **[histopathological examination demonstrated no dysplasia or malignant cells]**. Together, these findings substantially reduced the likelihood of the principal alternative diagnoses.

Following microbiological confirmation of pulmonary tuberculosis, standard antituberculous therapy was initiated with isoniazid, rifampin, pyrazinamide, and ethambutol **[for the initial 2-month intensive phase]**, followed by isoniazid and rifampin **[for a 4-month continuation phase, for a total treatment duration of 6 months]** [**Treatment was administered under/according to the Colombian national tuberculosis program, and adherence was documented throughout therapy without clinically significant interruptions.] [No additional systemic antibacterial treatment was administered after initiation of antituberculous therapy; concomitant local otologic management was limited to routine ear care/no additional otologic antimicrobial therapy.]** Thereafter, the patient underwent serial clinical, otoscopic, audiometric, and radiological follow-up.

At approximately two months after initiation of antituberculous treatment, the patient reported improvement in his otologic symptoms, with a reduction in otorrhea and aural fullness. Otoscopic examination demonstrated improvement of the previously documented inflammatory abnormalities (**Figure 1(b)**).

At the four-month follow-up, further clinical improvement was observed. The patient reported continued reduction of otologic symptoms, while otoscopic examination showed progressive resolution of the initial abnormalities (**Figure 1(c)**). Repeat pure-tone audiometry demonstrated improvement in conductive hearing function, with reduction of the previously documented air-bone gap (**Figure 1(g)**).

At approximately six months of follow-up, the patient remained clinically improved, without recurrence of persistent otorrhea. Otoscopic examination demonstrated continued resolution of the middle-ear abnormalities (**Figure 1(d)**). Importantly, improvement occurred in parallel with antituberculous therapy and was sustained after completion of treatment.

Follow-up computed tomography of the temporal bones was subsequently performed and demonstrated marked resolution of the previously identified soft-tissue density involving the middle-ear cavity and mastoid air cells, without development of new osseous abnormalities (**Figure 1(j)**).

Long-term otologic surveillance was maintained. At 18 months of follow-up, the patient remained clinically stable, with no recurrent otorrhea and no evidence of active middle-ear inflammation on otoscopic examination (Figure 1(e)). Final audiometric evaluation demonstrated sustained improvement in conductive hearing function, with progressive closure of the air-bone gap compared with the initial examination (Figure 1(h)). The concordant clinical, otoscopic, audiometric, and radiological response, in the setting of microbiologically confirmed pulmonary tuberculosis and without direct demonstration of *M. tuberculosis* in the middle ear, provided additional support for the diagnosis of **presumed tuberculous otitis media**.

The complete clinical course, from the onset of refractory otologic symptoms through local diagnostic investigations, identification of pulmonary tuberculosis, initiation of antituberculous therapy, and subsequent clinical follow-up, is summarized in Figure 2.

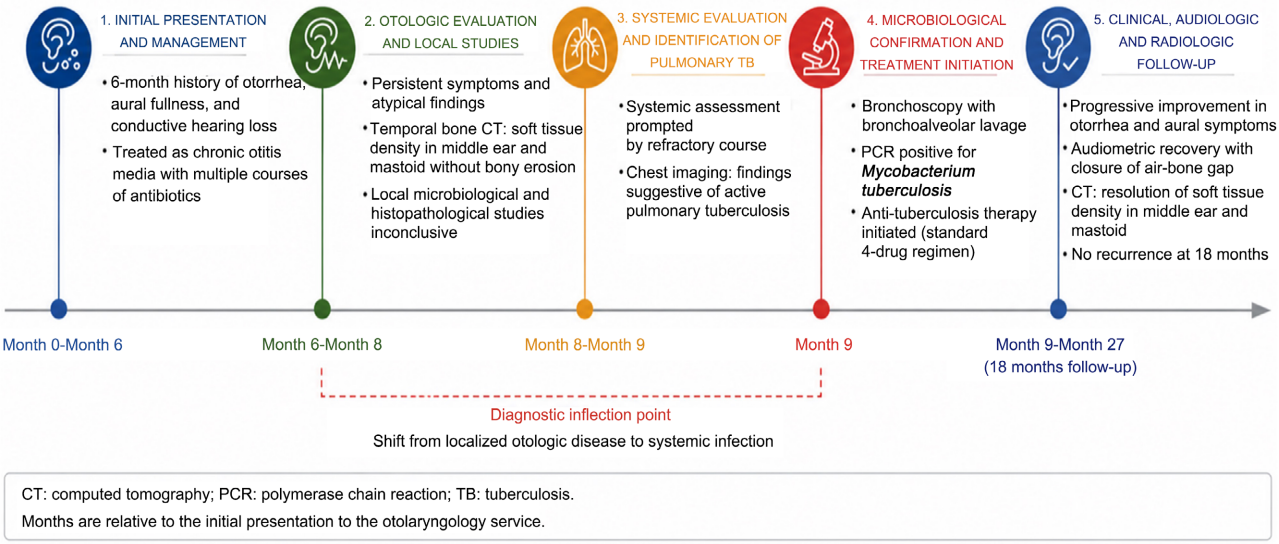

Figure 2. Timeline of the clinical course, diagnostic evaluation, treatment, and follow-up.

The timeline depicts the six-month history of persistent otorrhea, aural fullness, and hearing impairment; previous antimicrobial treatment; otologic evaluation; temporal bone imaging; local microbiological and histopathological investigations; subsequent systemic evaluation; identification of pulmonary tuberculosis confirmed by polymerase chain reaction for *Mycobacterium tuberculosis* in bronchoalveolar lavage fluid; initiation of antituberculous therapy; and serial otoscopic, audiometric, and radiological follow-up through 18 months.

3. Discussion

Tuberculous otitis media (TOM) is a rare extrapulmonary manifestation of tuberculosis and remains a considerable diagnostic challenge because its clinical presentation is often nonspecific and may closely resemble chronic bacterial otitis

media. The historically described triad of painless otorrhea, multiple tympanic membrane perforations, and facial nerve paralysis is currently observed in only a minority of patients, whereas persistent otorrhea, conductive or mixed hearing loss, tympanic membrane thickening, granulation tissue, and refractory middle-ear inflammation are considerably more frequent presentations. Consequently, diagnostic delays remain common, particularly when patients initially receive repeated courses of conventional antimicrobial therapy without sustained improvement [11]-[13].

The present case illustrates this diagnostic difficulty. The patient experienced six months of unilateral otorrhea, aural fullness, and hearing loss despite conventional treatment for chronic otitis media. Otoscopy demonstrated hyperemia, thickening, and retraction of the tympanic membrane with purulent otorrhea, without the classical multiple perforations or facial nerve involvement traditionally associated with TOM. This atypical phenotype emphasizes that the absence of classical otoscopic findings should not exclude tuberculosis from the differential diagnosis of persistent or treatment-refractory middle-ear disease [14] [15].

Hearing loss is one of the most frequently reported manifestations of TOM and may be conductive, sensorineural, or mixed depending on the anatomical extent of the disease. Conductive hearing loss may result from middle-ear effusion, inflammatory tissue, tympanic membrane abnormalities, or ossicular involvement, whereas sensorineural impairment may occur in more advanced disease involving the inner ear [16]. In our patient, pure-tone audiometry initially demonstrated conductive hearing loss with a significant air-bone gap. Serial audiometric evaluations at 6 and 18 months showed progressive narrowing of this gap and sustained hearing improvement, paralleling the resolution of otorrhea and normalization of the tympanic membrane.

Temporal bone computed tomography is useful for defining the extent of middle-ear and mastoid involvement, although no radiological feature is pathognomonic for TOM. Reported abnormalities include soft-tissue opacification of the tympanic cavity and mastoid, ossicular erosion, cortical destruction, labyrinthine involvement, and facial canal abnormalities. Importantly, the absence of bone destruction does not exclude tuberculous disease, particularly in earlier or less aggressive forms [17] [18]. In the present case, initial CT demonstrated soft-tissue density within the middle-ear cavity and mastoid without significant bony erosion, followed by complete radiological resolution and restoration of aeration at 18 months.

A major diagnostic consideration in this case is the absence of direct microbiological confirmation of *Mycobacterium tuberculosis* from the middle ear. Local microbiological and histopathological investigations may have limited sensitivity because TOM is frequently paucibacillary, and diagnostic yield may be further reduced by previous antimicrobial exposure or inadequate sampling [19] [20]. In our patient, pulmonary tuberculosis was subsequently confirmed by PCR detection of *M. tuberculosis* in bronchoalveolar lavage fluid. Although microbiological

confirmation at a distant anatomical site does not independently prove tuberculous involvement of the middle ear, the persistent refractory otologic disease, absence of a definitive alternative diagnosis, and subsequent concordant response to anti-tuberculosis treatment strongly supported the diagnosis of **presumed tuberculous otitis media associated with microbiologically confirmed pulmonary tuberculosis**.

The therapeutic response constitutes one of the most relevant features of this case. After initiation of standard anti-tuberculosis therapy with isoniazid, rifampin, pyrazinamide, and ethambutol, followed by continuation therapy with isoniazid and rifampin, progressive improvement was observed across multiple independent domains. Otorrhea resolved, tympanic membrane inflammation and thickening progressively decreased, the conductive hearing deficit improved, and follow-up CT demonstrated complete resolution of middle-ear and mastoid soft-tissue abnormalities. Anti-tuberculosis pharmacotherapy remains the cornerstone of TOM treatment, whereas surgery is generally reserved for obtaining diagnostic tissue, treating complications, removing sequestra, or managing persistent disease despite adequate medical therapy [21] [22].

The principal strength of this report is its prolonged multimodal follow-up. Serial otoscopic examinations documented progressive anatomical recovery from active inflammatory disease to an intact tympanic membrane without recurrence at 18 months. This evolution was accompanied by objective audiometric improvement and complete radiological resolution. The concordance among clinical, otoscopic, functional, and radiological outcomes provides compelling evidence supporting the tuberculous nature of the middle-ear disease despite the absence of direct local microbiological confirmation.

Several differential diagnoses must be considered in chronic refractory otorrhea, including conventional chronic suppurative otitis media, cholesteatoma, fungal infection, nontuberculous mycobacterial disease, granulomatosis with polyangiitis, sarcoidosis, syphilis, and neoplastic processes [23]. The present case emphasizes that tuberculosis should be considered when middle-ear disease persists despite appropriate conventional treatment, particularly when systemic tuberculosis is demonstrated or epidemiological and clinical factors increase the pretest probability.

This report has an important limitation: *M. tuberculosis* was not directly identified in middle-ear specimens. Therefore, the case should not be classified as microbiologically confirmed TOM. Nevertheless, the temporal association between refractory otologic disease and microbiologically confirmed pulmonary tuberculosis, together with the progressive and sustained resolution of symptoms, otoscopic abnormalities, conductive hearing loss, and radiological findings following anti-tuberculosis therapy, provides strong clinicoradiological support for the diagnosis.

In conclusion, TOM may present without multiple tympanic membrane perforations, facial paralysis, or destructive temporal bone changes and may closely

mimic conventional chronic otitis media. Persistent middle-ear disease that fails to respond to standard therapy should prompt consideration of atypical infections, including tuberculosis. The present case demonstrates the diagnostic value of integrating systemic microbiological findings with serial otoscopy, audiometry, imaging, and therapeutic response, particularly when direct microbiological confirmation from the middle ear cannot be obtained.

4. Conclusions

Tuberculous otitis media remains a rare but clinically relevant manifestation of extrapulmonary tuberculosis whose diagnosis is frequently delayed because of its nonspecific presentation and resemblance to conventional chronic otitis media. The absence of classical findings—including multiple tympanic membrane perforations, facial nerve paralysis, or destructive temporal bone changes—should not exclude the diagnosis, particularly in patients with persistent unilateral otorrhea, hearing loss, and middle-ear abnormalities refractory to standard antimicrobial therapy [24] [25].

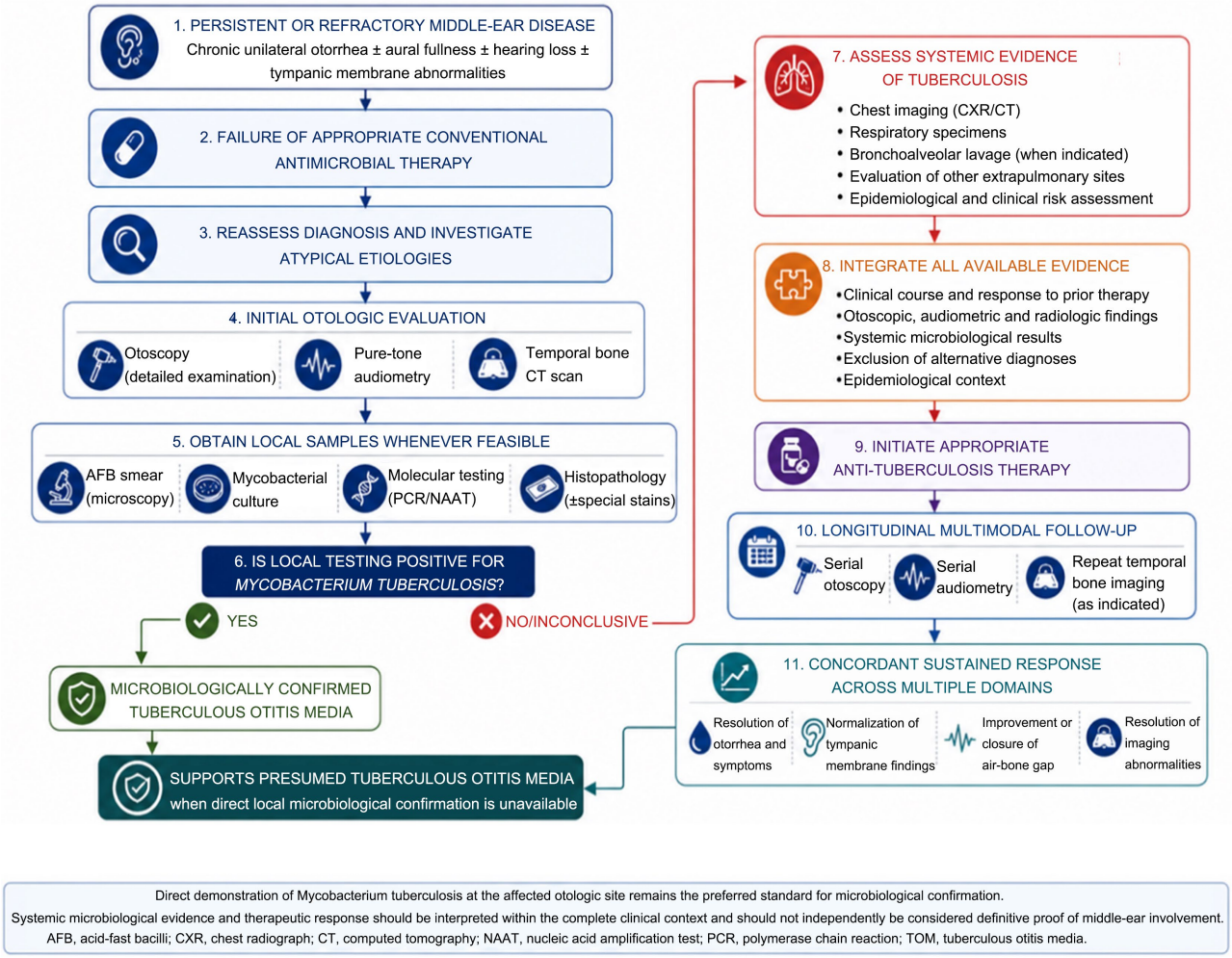

Figure 3. Clinical algorithm for the diagnosis and management of suspected tuberculous otitis media (TOM).

This case highlights the importance of integrating clinical evolution, serial otoscopy, audiometry, temporal bone imaging, systemic investigation, and therapeutic response when direct microbiological confirmation from the middle ear cannot be obtained. Although pulmonary identification of *Mycobacterium tuberculosis* does not independently establish otologic involvement, the concordant and sustained resolution of otorrhea, tympanic membrane abnormalities, conductive hearing loss, and radiological changes following anti-tuberculosis treatment provides strong support for the diagnosis of presumed tuberculous otitis media associated with microbiologically confirmed pulmonary tuberculosis [26] (**Figure 3**).

For otologists and otorhinolaryngologists, refractory middle-ear disease should prompt reconsideration of the initial diagnosis and investigation for atypical infectious causes. Earlier recognition of TOM may prevent unnecessary antimicrobial exposure, repeated procedures, irreversible hearing impairment, facial nerve involvement, and other potentially severe complications [27].

Ethical Statement

Written informed consent was obtained from the patient for the publication of this case report and all accompanying clinical images. The patient's identity and confidentiality were protected throughout the preparation of the manuscript.

Data Availability

All relevant data are included within the article. Additional information is available from the corresponding author upon reasonable request.

Declaration of Generative AI and AI-Assisted Technologies in Manuscript Preparation

During the preparation of this manuscript, the authors used Claude (Anthropic) and ChatGPT (OpenAI) solely as AI-assisted editorial tools. Their use was limited to language and copy editing, including correction of grammar, spelling, syntax, and punctuation; refinement of selected sentences to improve semantic clarity, readability, and academic style; and assistance in improving the wording and description of the figures and their corresponding legends. These tools were not used to generate the scientific content of the manuscript, formulate the clinical interpretation or conclusions, analyze data, make diagnostic or therapeutic decisions, or generate or select references. All AI-assisted suggestions were critically reviewed, verified, and revised as necessary by the authors. The authors take full responsibility for the accuracy, integrity, and final content of the manuscript.

Author Contributions

All authors contributed to the conception, preparation, critical revision, and final approval of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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