

## Original Research Article

# Tubercular otitis media: demystifying its nuances and an update on the tenets of emerging perspectives

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## ABSTRACT

**Background:** Tuberculous otitis media (TOM), a rare cause of chronic otitis media, is easily confused with nonspecific chronic otitis media owing to its unexpectedly changing and variable presentation, low suspicion and low prevalence which causes difficulty in its early diagnosis. The purpose of this study was to analyze its changing clinical pattern and to formulate an acceptable management protocol.

**Methods:** This retrospective observational, descriptive study included 457 cases of Chronic otitis media who were operated between January 2017 to July 2020 in a tertiary care center. Histo-pathological examination of tissue from the middle ear and mastoid were sent and positive results were corroborated retrospectively to the clinical findings.

**Results:** Diagnosis of TOM was established post-operatively in five cases (1.09%) with a positive histopathology report in four out of five cases and by polymerase chain reaction (PCR) in one case. Three cases (60%) presented with facial palsy and one with profound hearing loss (20%). All cases received 6 months course of anti-tubercular therapy postoperatively and were in regular follow up.

**Conclusions:** Tuberculosis of the middle ear can present unexpectedly either as a complication or completely as a non-specific chronic otitis media. The extent of pathological findings might not be in commensuration with the clinical features. In operated chronic otitis media cases, specimen for histopathological examination must be sent in all cases and further specific microbiological investigations may as well be advised accordingly.

**Keywords:** Tuberculous otitis media, Otomastoiditis, anti-tubercular therapy, facial nerve palsy, tympanic membrane, acid fast bacilli

## INTRODUCTION

Tuberculosis has been a global health care concern for long, and in developing countries like India it is a major cause for increased morbidity and mortality. It has been reported that more than nine million new tubercular cases and 1.7 million fatalities occur annually throughout the world.<sup>1</sup> The reported incidence in India is 1.5 per 1000 population, whereas 4 per 1000 population are bacteriologically positive.<sup>2</sup> Extra-pulmonary tuberculosis manifesting as tubercular otitis media (TOM) is a rare atypical presentation which accounts for 0.05-0.9% of

infections of middle ear and TOM accounts for 4% of head and neck TB.<sup>3,4</sup>

The incidence of this entity has remained high in India but it has shown its resurgence in recent years due to an increase in HIV infection and its association with tuberculosis, emergence of multidrug-resistant TB, increase in population living in poor socio-economic conditions, increasing incidence of immunocompromised cases, better diagnostic facilities, growing familiarity with disease among clinicians, migration from endemic areas and access to better medical care. These

cases are frequently misdiagnosed due to the limitations of conventional diagnostic methods and non-availability of newer diagnostic modalities as well. This serious and contagious pathology can engage any part of the temporal bone. The clinical suspicion of the disease should be high in any atypical case of chronic suppurative otitis media (CSOM) not responding to conventional treatment, particularly in high-risk groups.

This study was carried out with an aim of understanding the enigmatic presentation of this recalcitrant condition, getting an insight into the recent advances in its diagnostic realm and proposing a working algorithm helping clinicians to appropriately manage this condition.

## METHODS

This retrospective observational, descriptive study was carried out in the department of otorhinolaryngology of a tertiary care center from January 2017 to June 2020. This study comprised 457 cases of CSOM who underwent middle ear surgery and histopathological reports of those cases were examined and corroborated to physical findings in positive cases.

We reviewed the medical records of all CSOM cases. Detailed history and routine orhinolaryngologic evaluation were done in all patients. Otologic findings with respect to perforation-size, site and number; presence or absence of ear discharge; granulation tissue/polyps; condition of middle ear mucosa, post aural swellings or fistulas, facial nerve paralysis and other complications were noted. Pure tone audiogram, appropriate radiological and routine investigations were done in all cases. All the biopsies from middle ear cleft surgeries are routinely sent for histopathological examination as a standard protocol of our department. Retrospective analysis of all available histopathological reports and their case sheets was done. Based on histopathological report and other microbiological tests, the diagnosis of TOM was established.

## RESULTS

The clinical records of all 457 operated cases of chronic otitis media were scrutinized retrospectively. Female to male ratio was found to be 1.37:1 (193 males and 264 females). Age of the patients ranged from six to sixty-five years. Among the 457 cases, 203 cases had right ear disease, 248 cases had left ear disease and 6 cases had bilateral ear involvement and 6 cases had bilateral ear disease. Right ear surgery was done in 206 cases, whereas the left ear was operated in 251 cases.

Of 457 cases of chronic otitis media, the diagnosis of TOM was made in five cases (1.09%). It were the per-operative findings in three cases that led the author to suspect TOM while in two cases the diagnosis was made after the routine HPE report. Out of five cases in which a diagnosis of TOM was made, canal wall down

mastoidectomy was performed in two cases and decompression of the facial nerve was done in three cases. The extensive granulation /polypoidal mass found in the middle ear cleft was sent for histopathology in all these cases and a confirmed diagnosis of TOM was made in four out of five cases. In one case, for which the initial histopathology report was inconclusive, final diagnosis was established after re-exploration after second surgery done for persistent pain and mass on HRCT. TB-PCR and histopathology were both positive for TB. Post-operatively Anti-tubercular therapy (ATT) was given to all patients for at least 6 months. Postoperative follow up of all cases were kept for 6-9 months and adequate healing was obtained in all cases. Here we have described the clinical, pathological, radiological and surgical attributes of TOM cases in a tabulated form (Table 1).

### Case 1

A 52-Year-old male patient presented with the complaints of bilateral ear discharge for 3 years, bilateral decreased hearing for one year and inability to close right eye for five days. The discharge bilaterally was scanty, odorless and painless. Otoscope examination of left ear revealed medium sized central perforation. In the right ear a medium sized central perforation and pale middle ear mucosa was seen after removing thick discharge. The patient was a known case of diabetes mellitus for five years under irregular treatment. On the left side there was a grade V (House Brackman) facial palsy.



**Figure 1: (A) Pre-operative facial palsy and immediate postoperative facial status, (B) axial and coronal HRCT temporal bone opacity in epitympanum, aditus, antrum and mesotympanum, (C) facial appearance after 6 months of ATT, (D) decompressed facial nerve and granulations in attic.**

**Table 1: Clinical, audiological, radiological and surgical profiles of all TOM cases.**

Age (year)	Sex	Clinical spectrum	Radiology	Hearing status	Operation done	Intra-op findings	Postoperative status	co-morbidities	Final diagnosis based on
52	M	b/l otorrhea b/l decreased hearing right sudden onset facial nerve palsy grade V	X-ray- pneumatized mastoid air cells with clouding of air cells. HRCT temporal bone- soft tissue in right mastoid antrum, middle ear	Right ear- 95 db SNHL left ear- severe SNHL	Intact canal wall mastoidectomy with type III tympanoplasty with facial nerve decompression in right ear	Pale middle ear mucosa, pale cheesy granulations in mastoid antrum	No hearing improvement, intact TM, grade III facial palsy	Diabetes mellitus	HPE +ve
23	M	Left otorrhea left decreased hearing	X-ray- sclerosed left mastoid	Left ear- 45 db (AC) CHL	Cortical mastoidectomy with type I tympanoplasty in left ear	Pale & friable middle ear mucosa, no granulations in mastoid antrum	Hearing improvement 38 db, intact TM,	-	HPE +ve
54	F	Right otorrhea right decreased hearing	X-ray- diploic right mastoid	Right ear- 50 db (AC) CHL	Cortical mastoidectomy with type III cartilage tympanoplasty in right ear	Pale fleshy granulations in mastoid antrum, epitympanum & mesotympanum	Hearing improvement 40 db (AC), intact TM,	-	HPE +ve
65	F	Right decreased hearing right otalgia right sudden onset facial nerve palsy grade IV	HRCT temporal bone s/o right sided erosion of tegmen tympani and facial canal with otomastoiditis, repeat HRCT temporal bone s/o right sided soft tissue in antrum	Right ear- severe mixed hearing loss	Cortical mastoidectomy with type III cartilage tympanoplasty with facial nerve decompression in right ear revision surgery: right ear modified radical mastoidectomy	Granulations over facial canal revision surgery- mastoid antrum filled with polypoidal mass	No hearing improvement, improvement in facial palsy grade II	-	Initial HPE -ve Reoperated HPE +ve TB- PCR +ve
48	M	right ear otorrhea right ear decreased hearing right-otalgia vertigo right sudden onset facial nerve palsy II	HRCT temporal bone-soft tissue density in right mastoid and middle ear	Right ear- severe mixed hearing loss	Right modified radical mastoidectomy with facial nerve decompression	Granulations in mastoid antrum, epitympanum and retro tympanum	No hearing improvement, no improvement in facial palsy	Diabetes mellitus	HPE +ve

On Laboratory investigations ESR was elevated (78 mm/1<sup>st</sup> hour) and chest X-ray revealed no abnormality. Pure tone audiometry showed profound sensory-neural hearing loss of 95 dB in right ear and severe sensorineural hearing loss in left ear. X-ray mastoid showed pneumatic mastoid with clouding of air cells. Computed tomography of temporal bone revealed soft tissue in right mastoid air cells and antrum, aditus and middle ear cavity. Ossicular chain was intact. The patient was planned for surgery and intact canal wall mastoidectomy (with type III tympanoplasty) and facial nerve decompression was done. Facial nerve was delineated along the mastoid and tympanic segment. Intra-operatively we found pale middle ear mucosa and attic and antrum were filled with cheesy material like granulation tissue. Intra-operatively, the middle ear mucosa was pale and granulation tissue from mastoid antrum and middle ear was sent for a biopsy which revealed tubercular otitis media. The patient was treated with ATT for a 6 months. After 6-month follow-up, patient's TM was to be found intact with facial palsy grade III (Figure 1), but there was no hearing improvement.

#### Case 2

A 23-year-old man presented with complaints of chronic left ear discharge and decreased hearing for last three years. He was treated with antibiotic therapy for several months. On Otoscopy there was a large tympanic membrane perforation with purulent discharge. Audiometry revealed 45 dB conductive hearing loss in left ear and X ray mastoid showed sclerotic pattern and surgery was advised. Tympanoplasty along with mastoidectomy was done and no granulations, sequestra or hyperplastic mucosa was found. Per-operatively the middle ear mucosa was found strikingly pale and friable. A part of pale mucosa occupying the middle ear was removed and sent for HPE. Biopsy revealed tuberculous infection and subsequently patient was started with ATT. It is worth mentioning that the patient did not have any typical features of tubercular infection in pre-operative period. The patient was followed-up regularly and after 6 months of follow up his TM was found to be intact and hearing improved to 38 dB.

#### Case 3

A 54-year-old female complained of chronic right otorrhea and decreased hearing for one year. The discharge was mucopurulent and non-foul smelling. On examination, there was a small central perforation in Antero-inferior quadrant with pulsatile discharge. Audiometry showed a 50 dB conductive hearing loss and X ray mastoid exhibited diploic pattern. The patient underwent type III cartilage tympanoplasty with mastoidectomy and intraoperatively there were exorbitant pale fleshy granulations in mastoid antrum, aditus, epi- and mesotympanum. These granulations surrounded the ossicles and incudo-stapedial joint was found to be eroded. Facial canal was dehiscence at second genu. Histopathological examination (HPE) revealed features

of chronic granulomatous disease suggestive of tuberculosis with the presence of tubercular bacilli. ATT was commenced (2 HRZE/4 HR) based on the histopathological report and continued for 6 months. The patient's hearing improved to 40 dB and with intact neo-membrane.

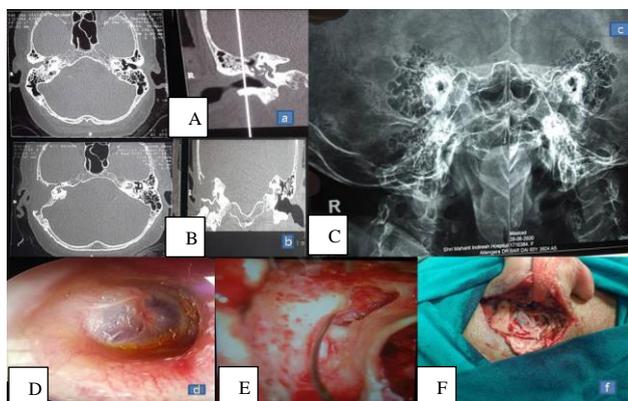
#### Case 4

A 65-year-old female presented with complaints of decreased and progressive hearing loss of gradual onset in right ear for 6 months and sharp and intermittent pain in right ear for 5 days and facial palsy for 12 days. There was no previous history of tuberculosis oto-endoscopic. TM appeared apparently intact on otoscopic examination with polypoidal tissue abutting it and sagging of (postero-superior quadrant) meatal wall. The patient also had tragal as well as mastoid tip tenderness with grade IV facial nerve palsy. Pure tone audiogram revealed right severe mixed hearing loss. HRCT temporal bone indicated right sided otomastoiditis with the possible cholesteatoma formation with erosion of tegmen tympani, tympanic part of the facial nerve. Routine blood investigations were within normal limits except high ESR (51 mm/1<sup>st</sup> hour). The patient was diagnosed as a case of right active squamosal CSOM with mastoiditis and facial nerve palsy with mixed hearing loss.

The patient underwent intact canal wall mastoidectomy (type III augmentation tympanoplasty) and facial nerve decompression. Intra-operatively, exuberant granulation tissue was found over dehiscence facial nerve area (second genu) only and cholesteatoma was conspicuously absent. A suspicion of patient having tubercular otitis media cropped up but the histopathological examination of granulation sent from mastoid revealed an infiltration of mixed inflammatory cells comprising lymphocytes, plasma cells, histiocytes, occasional neutrophils and eosinophils along with lymphoid aggregates suggesting features of chronic otitis media. Also, no well-formed granuloma or caseation necrosis was seen. However, the patient was empirically put-on anti-tubercular treatment according to DOTS- category-I (directly observed treatment, short course).

Postoperatively, after one month, the patient developed severe pain in right ear, which was continuous and disturbing to the patient. She did not respond to conservative management. Examination revealed tenderness of mastoid tip and cyma concha. MRI brain was normal and HRCT temporal bone revealed soft tissue in the mastoid air cells with erosion of tegmen tympani. The patient was taken up for re-exploration and intraoperatively soft tissue polypoidal mass was seen filling mastoid antrum which bled on removal (Figure 2). There was exuberant pale granulation tissue surrounding the ossicles and facial nerve. All diseased tissue was removed and canal wall was lowered. Tissue was sent for TB-PCR, Gene-Xpert and histo-pathological examination. HPE was suggestive of chronic granulomatous condition with TB-PCR positive for mycobacterium tuberculosis, postoperatively patient was

relieved of pain but had persistent grade IV facial nerve palsy with no hearing improvement. After 9 months patient had a grade II facial palsy with dry mastoid cavity and no clinical complaints.



**Figure 2: (A) Preoperative HRCT temporal bone axial and coronal cuts of soft tissue density in mesotympanuma and epitympanum, (B) Repeat HRCT temporal bone before re-exploration (axial and coronal cuts) of soft tissue opacity in antrum, (C) pre-operative X-ray mastoid revealed pneumatic pattern, (D) apparently normal looking TM (otoscopically) pre-operatively, (E) decompressed facial nerve, (F) On re-exploration, a soft polypoidal mass was seen in mastoid antrum completely filling it.**

### Case 5

A 48-year-old male, having a previous history of type II diabetes mellitus presented with right ear discharge for two years, pain in right ear for seven days, giddiness for seven days and decreased hearing in right ear for five days. On clinical examination, Patient had grade II facial nerve palsy, there was no nystagmus and fistula test was negative. Otoscopic Examination revealed grade III retraction of the pars tensa with a small central perforation (in posterior quadrant). Pure tone audiogram showed a severe mixed hearing loss. HRCT temporal bone revealed soft tissue density filling right mastoid air cells and middle ear cavity. Routine blood investigations were normal except ESR which was raised (27 mm/1<sup>st</sup> hour). The patient was diagnosed with a right chronic otitis media and facial nerve palsy with severe mixed hearing loss. Right Modified radical mastoidectomy with facial nerve decompression was planned in this patient. Intra-operatively pale granulation tissue was seen in antrum, epi-tympanum and retro-tympanum which was removed and sent for histopathological examination. The histopathology report showed ill formed epithelioid granuloma, which was suggestive of tuberculosis, following which a 6-month course of ATT was given. However, the patient had no improvement in hearing and facial palsy on follow up.

### DISCUSSION

The tuberculous affliction of the temporal bone has been familiar since 18<sup>th</sup> century, when it was first described as tuberculous mastoiditis by Jean Louis Petit. The classical presentation of tuberculosis of the ear was first described by Wilde in 1853 whereas in 1878, Schwartze noticed the cheesy infiltration and tubercles of the mucosa. In 1882 Koch discovered the bacillus responsible for tuberculosis and later Esche found the bacillus in ear discharge.<sup>5</sup> The typical clinical features of TOM were then reported again in 1953 by Wallner.<sup>6</sup> The clinical presentation of TOM is characterized by indolent course and an non-specific and heterogeneous clinical characteristics that usually occur in individuals having concurrent chronic middle ear infection.

The tubercle bacilli may gain access to middle ear cleft either through pre-existing tympanic membrane perforation, or via the Eustachian tube and rarely from maternal systemic or genitourinary infection to the infant.<sup>4</sup> Invasion to middle ear may also occur by hematogenous spread, either from pulmonary or extrapulmonary tuberculosis, and contiguous spread from adjacent intracranial tubercular by foci through temporal bone. Tuberculous otitis media in adults, most commonly occurs in association with advanced pulmonary or extrapulmonary tuberculosis but in children it may occur in isolation. TOM is more prevalent in children and younger age group with 84% of all cases, aging below 15 years.<sup>7</sup> Additionally, clinical features of TOM seen in infants and children are different from those seen in adults because initial symptoms are more extensive and often go unnoticed in infants.<sup>5</sup> The study age range of positive cases in our study was 23 years to 65 years.

Characteristic clinical presentation of the disease described in the literature is now rarely noticed in clinical practice. The surprising presentation of middle ear tuberculosis lies in the fact that changes in its clinical behavior have been evolving over time in an unpredictable and perplexing way, characteristically the most common features of TOM observed are moderate/severe hearing loss, painless ear discharge, single perforation of the tympanic membrane, hyperemic middle ear mucosa with some pale granulations.<sup>2</sup> The granulations are usually described as pale, flabby and exuberant but surprisingly, they recur vociferously after removal and we observed the same in our study also.<sup>5</sup>

Triad of signs, including regional lymphadenitis in the absence of systemic TB, refractory otitis media resistant to conventional treatment and complications such as sensorineural hearing loss or facial nerve palsy must alert the otologist to exclude the presence of TOM.<sup>8</sup> Plester et al in 1980 described that a dull, deep seated aching pain due to tension or mild pressure effected by granulation tissue was diagnostically significant.<sup>9</sup> Persistent pain was a significant complaint in onset case four in this study, which recurred postoperatively for which surgical re-

exploration was done. Hearing impairment occurs in the majority of cases, usually severe and early. Conductive hearing loss is usually the commonest presentation in the majority of these cases, whereas indolent character of the pathology may result in mixed or sensorineural hearing loss which may persist even after complete treatment of the disease. In this study one case presented with sensorineural hearing loss, two cases with conductive and two cases presented with mixed hearing loss and no post-treatment hearing improvement was noticed in 3 cases. Complaints like tinnitus and dizziness may also be present.

Facial nerve paresis, being the most common complication, occurs early in the course of disease and has been observed in 16% of adult patients and 35% of children with tubercular otitis media.<sup>7</sup> In this series, we noticed facial palsy in three cases and post-operative recovery of facial nerve function was seen only in two cases.

The clinical diagnosis of TOM is very often missed in the early stages and usually made only after getting the histopathological report. In this study, the probable diagnosis of TOM was made in three out of five cases due to their clinical presentation or per-operative findings, whereas in two cases the diagnosis of TOM could be made only after getting HPE report. Despite of low clinical suspicion and high prevalence of TOM in regions like India it is recommended to send the ear discharge and granulation tissue for culture, sensitivity and histopathological examination with AFB stain to rule out Tuberculosis.

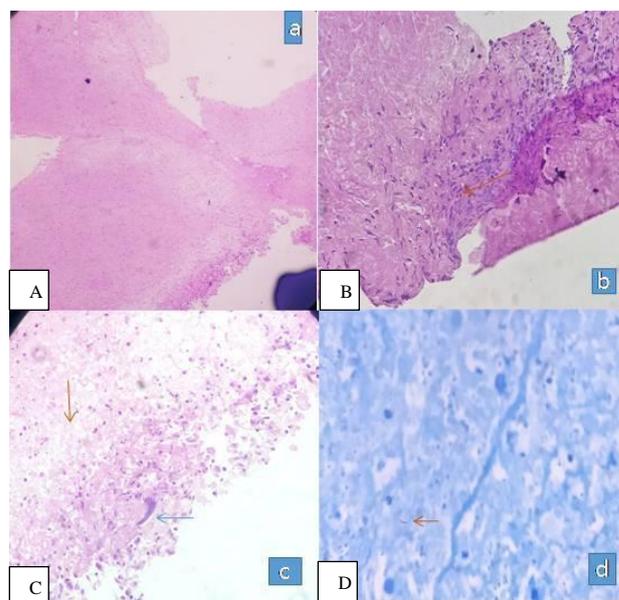
A complete diagnostic workup includes chest and mastoid X-rays, audiogram, ear, culture/sensitivity of ear discharge, skin testing and blood investigations. Skin testing with purified protein derivative (Mantoux test) is not a very reliable test in an anergic patient or if he is on steroid medication, especially in endemic areas.<sup>10,11</sup> The chest radiograph is more conclusive of pathology and it is positive in about 50 % of cases and some authors believe that it may reach up to 94% of cases.<sup>12</sup> In this study none of the five TOM cases had any evidence of pulmonary tuberculosis on chest x ray during pre-operative work up. Chirch et al described that CT of the thorax, along with sputum culture and smear examination, might prove an important diagnostic adjunct in precluding the possibility of concurrent pulmonary involvement.<sup>13</sup>

A CT scan of the temporal bone is the standard radiological investigation for evaluation of chronic otitis media. It delineates the extent of pathology, but specific findings are not attributable to tubercular infection only and findings might be dependent on the duration of pathologic processes. CT scans usually shows soft tissue densities in the middle ear with clouding of mastoid air cells, multifocal erosion of mastoid or petrous parts, destruction of ossicles and involvement of auditory nerve.<sup>13</sup> Rho et al cited that soft tissue in middle ear cavity, preservation of mastoid air cells without sclerotic

change, mucosal thickening of the bony external ear canal, or a soft tissue extension to the external canal without erosion of scutum appeared to be specific CT findings that might be helpful in early diagnosis of TOM. There may not be any bone erosion.<sup>14</sup> In this study CT temporal bone was done in three cases who presented with complications (facial nerve palsy) where pneumatic pattern was observed and soft tissue opacities were seen in middle ear.

Demonstration of tubercular bacilli in the ear discharge is quite difficult due to superadded infection and this high rate of secondary bacterial infection, interfering with the growth of Koch's bacillus, limits the chances of identification of acid-fast bacilli on either staining or culture. Smear positivity for AFB in ear discharge extends up to 20% of cases, whereas positive cultures are seen in 5 to 35% of cases.<sup>15</sup> Repeated examinations might improve the yield, hence repeated cultures of the ear discharge must be done in cases with atypical clinical features.

The gold standard for diagnosing TOM includes the culture and HPE of material obtained from the middle ear and mastoid. A definitive diagnosis needs a demonstration of acid-fast bacilli by either culture or tissue staining (Figure 3). However, false negative rate of up to 10 percent are reported in the first histopathological report.<sup>7</sup> Nevertheless, in case of strong clinical suspicion, despite the negative results of first biopsy, one should assess the progression and clinical response to a routine antibiotic line of management. In this study initial HPE report was inconclusive in one case and the biopsy sent for HPE after re-exploratory surgery confirmed the diagnosis of TOM.



**Figure 3: (A) Large areas of caseous necrosis (H and E, 10X), (B) arrow showing scattered epithelioid cells forming ill-defined granuloma (H and E, 20X), (C) arrow showing Langhans giant cell (H and E, 20X), (D) arrow showing acid fast bacilli (ZN stain).**

TB PCR analysis is another important investigation, which could be done to confirm the diagnosis. However, the availability of microbial culture, serological tests, polymerase chain reaction and histological analysis can be a limiting factor particularly in developing countries like India. DNA amplification by polymerase chain reaction method is quick, easy to automate, economic, and quite specific. But, preferably CB-NAAT (Cartridge based nucleic acid amplification test) for the quick, simultaneous detection of tuberculosis and rifampicin resistance (GeneXpert MTB/RIF assay) is advisable in all adults and children with suspected TOM.<sup>2</sup> The importance of cartridge based nucleic acid amplification test (CBNAAT) with an efficacy to diagnose tuberculosis and rifampicin resistance within a few hours is encouraging. We suggest, based on our experience, that histopathology should always be sent along with amplification tests, if facilities are available.

The differential diagnosis of TOM includes, histoplasmosis, blastomycosis, midline granuloma, Wegener's granulomatosis, histiocytosis X, atypical mycobacterial infections, malignant otitis externa, cholesteatoma, syphilis, sarcoidosis, nocardiosis and lymphoma.<sup>16</sup>

The treatment of the disease comprises both medical and surgical modalities which results in faster healing with better results. Antitubercular (ATT) treatment is advised for a period of 6 months with a longer treatment needed in cases of disseminated tuberculosis and cases with intracranial complications. The recovery of hearing loss, particularly conductive component can be accomplished, after resolution of ear discharge, by tympanoplasty. In contrast, the recovery of sensori-neural hearing loss does not usually occur with the healing process.

Facial palsy will improve partially or completely. The rapidity and extent of recovery depends on the time duration between the appearance of facial paralysis and the institution of proper treatment. Its prognosis and recovery does not depend on decompression, but on prompt diagnosis and early institution of treatment. If the duration between facial palsy and the commencement of treatment is less than five days, a full recovery should be anticipated. If treatment is initiated after two months of symptoms, recovery of the facial palsy is unlikely.<sup>17,18</sup>

The role of surgery in the treatment of TOM is very important. Surgery is required to remove bone sequestrations and granulation tissue, functional restoration of hearing by tympanoplasty, establishing ear drainage and ventilation, removing a nidus of infected debris, to obtain histological or biological samples and in cases not responding to medical management. Intraoperative findings such as pale and extensive granulations in the middle ear cleft, sequestra and pale, avascular mucosa might call attention to the probability of tubercular infection. We have summarized a few points where TOM must be suspected during intra-operative/postoperative period (Table 2). The complications of the TOM are extradural abscess, tubercular meningitis, mastoiditis, osteomyelitis, petrositis, labyrinthitis profound hearing loss and facial nerve palsy. The labyrinth seems to be at greatest risk in adults, and the facial nerve and meninges are the most vulnerable risk areas in pediatric cases.<sup>19</sup> Surgery in the absence of any complications has been criticized by some authors.<sup>20</sup> Some studies have demonstrated higher rates of dry ears when surgery precedes ATT.<sup>21</sup> We therefore, present a roadmap for proper management of a suspected case of TOM in a clinical setting (Figure 4).

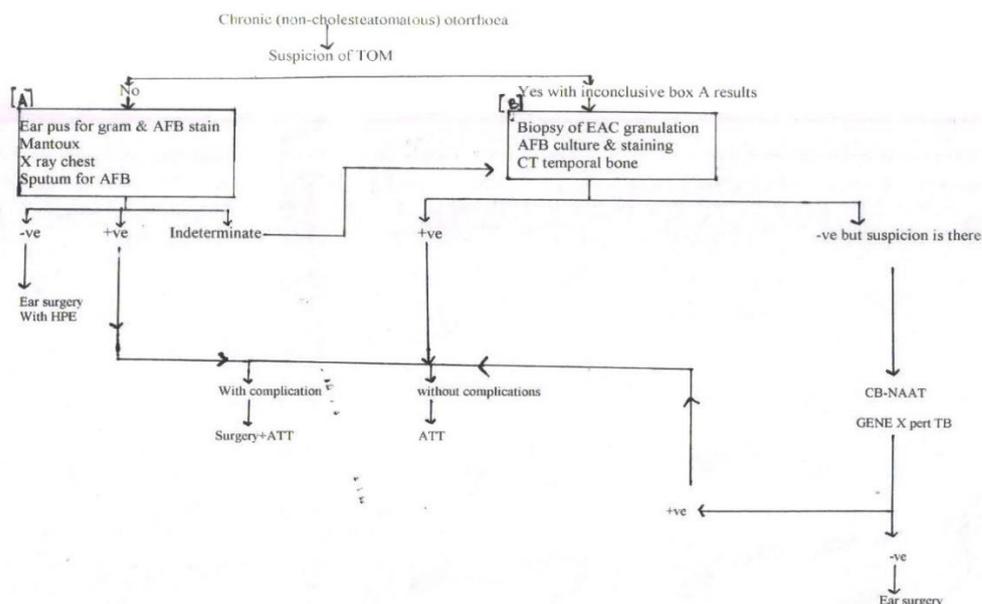


Figure 4: Flowchart of the proposed work up in a suspected TOM case.

**Table 2: Intra/post-operative features and suggestions which might point towards possible tubercular infection.**

S. no.	Intra/post-operative features
1.	If the pain/facial paresis does not improve/recurs/worsens even though HPE is negative for TB.
2.	If there is preoperative facial palsy and intra-operatively, we find disease disproportionate to the extent of facial palsy.
3.	If middle ear cleft refills rapidly again with soft tissue opacity (on CT), even if the previous HPE report is negative.
4.	Wherever suspected, always send the TB-PCR (If facilities available) along with HPE.
5.	Try to send HPE in every ear operated, if possible.
6.	Two samples should be sent for HPE to two different labs.

**Table 3: Clinical presentations helpful for suspecting a case of TOM.**

S. no.	Clinical presentations
1.	Persistent and chronic middle ear discharge refractory to medical treatment even with no clinical evidence of TB elsewhere.
2.	Short duration with rapid development of complications.
3.	Clinical findings or their duration inconsistent with complaints.
4.	Persistent otalgia in CSOM cases.
5.	Sudden or progressive sensorineural hearing loss in case of chronic otorrhea.
6.	Preauricular swelling/neck swelling with CSOM features.
7.	Pale granulations in external ear coming out of perforation.
8.	History of multiple failed surgery.
9.	A soft tissue mass in middle ear cleft on CT, and /or microbiological/radiological signs of active pulmonary tuberculosis
10.	Pneumatic pattern on HRCT but soft tissue mass in meso-, epitympanum, aditus ad antrum and antrum.
11.	Patient belonging to endemic zone or h/o migration from endemic zone.
12.	Past history of tuberculosis/contact/active pulmonary/nasopharyngeal tuberculosis at present.
13.	Thickened drum/+HL/pre/post-auricular swelling/mastoid tenderness.
14.	Unilateral hearing loss with/without painless otorrhea, and perforated or moth-eaten TM or intact TM

**CONCLUSION**

Every case of chronic otitis media must be assessed keeping the possibility of tuberculosis in mind. Most often the characteristic presentation of TOM is evident of either complications or features indistinguishable from nonspecific chronic otitis media. Diagnosis of TOM must be suspected in a chronic non-cholesteatomatous case presenting with following features (Table 3).

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