International Journal of Otorhinolaryngology and Head and Neck Surgery
Tiwari V et al. Int J Otorhinolaryngol Head Neck Surg. 2020 Aug;6(8):1572-1575
http://www.ijorl.com
pISSN 2454-5929 | eISSN 2454-5937

DOI: http://dx.doi.org/10.18203/issn.2454-5929.ijohns20203220

Case Report

Tuberculous otitis media: rare cause of recurrent facial nerve palsy

Vibhu Tiwari, Rohit Singh, V. Anand*

Department of ENT, Armed Forces Medical College, Pune, Maharashtra, India

Received: 10 June 2020
Accepted: 09 July 2020

***Correspondence:**
Dr. V. Anand,
E-mail: anandjayas@gmail.com

ABSTRACT

Tuberculous otitis media (TOM) is a rare form of tuberculosis, due to tubercular otitis media, thick caseous material and granulation tissue gets deposited in middle ear cleft causing bone erosion, sequestration, ossicular destruction, heading loss which can be conductive or mixed and rarely facial paralysis. In this case report we discuss tubercular otitis media as a cause of recurrent facial nerve paralysis. 54 year old male patient with no known previous comorbidities presented to ENT OPD on 12 October 19 with complaints of scanty, mucopurulent foul smelling occasionally blood stained continuous otorrhoea (R)×02 years, painless progressive and persistent hearing loss ×1 year and recurrent (R) sided facial nerve palsy, 03 episodes in last 2 years which were spontaneous and sudden in onset, showed gradual recovery with oral medication. Tubercular otitis media is not a very common diagnosis with which an otolaryngologist comes across. It generally presents with otorrhea and hearing loss which mimics more commoner ear disease. Typical findings of multiple tympanic membrane perforations are not necessarily seen. There can be involvement of facial nerve and presence of refractory otorrhoea with atypical features may be considered indicative of tubercular pathology. A clinician should have high index of suspicion whenever there are signs which outlies symptoms in form of severity. This case highlights the diagnostic challenge and high index of clinical suspicion required for diagnosis. Combination of appropriate diagnostic modality along with good clinical vigilance will aid in early diagnosis and start of early medication which reduces chances of any subsequent complications.

Keywords: Tubercular otitis media, Bony sequestrum, Facial nerve palsy, Chronic otitis media with complication

INTRODUCTION

Tuberculous otitis media (TOM) is a rare form of tuberculosis with its incidence being 0.04% of total cases of chronic otitis media, TOM can occur using two different pathways of spread.[1-3] It is classified as secondary where pathway of spread is the Eustachian tube which allows ascending infection. Hematogenous seeding called as primary where otitis media develops due to tubercular infection is another pathway of spread of tuberculosis, more commonly seen in geriatric age group, the characteristic features of primary tuberculosis. Whereas, secondary tubercular otitis media is more common in paediatric age group, most common source being primary pulmonary tuberculosis which leads to spread of infection through Eustachian tube.[4]

Due to tubercular otitis media, thick caseous material and granulation tissue gets deposited in middle ear cleft causing bone erosion, sequestration, ossicular destruction, facial nerve paralysis and heading loss which can be conductive or mixed.[3,5,6]

Clinical features of tubercular otitis media are insidious otorrhoea which is the initial symptom in 70% cases.[2] Discharge of TOM is not characteristic, it may be serous initially in 70% of cases and may turn progressively purulent and foul smelling. This is followed by painless and progressive hearing loss which occurs early in the course of disease. Periauricular lymphadenitis may also be present on the affected side which is a reliable indicator of tubercular nature of disease.[2] Facial nerve paralysis may also occur in around 8% of the cases

however the incidence has been quoted differently by different authors in various case series otalgia, fever and giddiness are progressively rare and may indicate an impending/overt complication.[7]

On otoscopic examination, there may be thick discharge, denuded mucosa with or without bony sequestrum and abundant granulation tissue, the characteristic multiple perforations of tympanic membrane are seldom seen as small perforations coalesce to form a single subtotal perforation.[2,6,8] Granulation tissue is also seen and may alter the characteristic of otorrhoea into a serosanguinous one. Ossicles are often seen embedded in granulation tissue and bony erosion with sequestration is seen in the course of the disease.[6]

Conventional gram staining doesn't show AFB as the thick waxy cell wall of M Tb doesn't stain well with gram stain, special alcohol staining and subsequent washing with dilute HCl shows the acid fast bacilli (AFB) from ear swab/tissue. Recent advances in molecular biology and genetics testing may also differentiate between the different atypical mycobacteria from M Tb and also predict the probable drug resistance (if any) by genetic testing, obviating the need for culture and biochemical typing of mycobacteria which is a tedious and time consuming process taking over 06 weeks for confirmed microbiological diagnosis and typing of species /sub species.[9-11] Routine haematology may be unremarkable if the primary focus is in the ear, however, presence of pulmonary tuberculosis (PTB) may show lymphocytic leucocytosis with anaemia with raised ESR in cases of advanced PTB.

Imaging studies range from plain X-ray mastoid which may show bony sclerosis to diffusion weighted fat suppressed contrast enhanced MRI which is more specific in detecting intracranial extension, high resolution CT temporal bone is more useful in detecting the bony erosion which occurs invariably with TOM.

Tubercular otitis media is managed conservatively using regime for extrapulmonary tuberculosis as per RNTCP guidelines 2017. The conventional 2x (HRZE) + 4x (HRZ) for a total of 06 months has been found to be adequate treatment for Tubercular otitis media. Surgical management is indicated in cases where complications are impending or have already taken place. Surgical intervention may encompass mastoidectomy to remove the mastoid reservoir and tympanoplasty to eradicate the disease from middle ear, the specimen may also be subjected to various molecular tests for confirmation of diagnosis, the bacterial yield of caseous material from middle ear and mastoid may be having lower bacterial count to be positively confirmed using staining however, with advent of cartridge based nucleic acid amplification technique (CBNAAT), even very small amount of MTb DNA can be amplified and detected which is much faster than conventional methods and also indicates the presence of genes responsible for ATT resistance.[9-11]

Management of complications like facial nerve palsy is largely conservative as the prognosis of facial nerve paralysis due to TOM is usually guarded.

CASE REPORT

54 year old male patient with no known previous comorbidities presented to Civil ENT OPD on 12 Oct 19 with complaints of scanty, mucopurulent foul smelling occasionally blood stained continuous otorrhoea (R) ×02 years, painless progressive and persistent hearing loss ×1 year and recurrent (R) sided facial nerve palsy, 03 episodes in last 2 years which were spontaneous and sudden in onset, showed gradual recovery with oral medications. Last episode of facial nerve palsy occurred 1 month back with complete recovery in 20 days. Past, family and personal history were unremarkable, the patient took treatment from a private hospital for facial nerve palsy for which he was treated with oral medications, details of which aren't available.

Examination findings

Preauricular, pinna and post auricular regions in both ears were normal, no cervical lymphadenopathy was noted, External auditory canal was filled with mucopurulent discharge with granulation tissue at the bony cartilaginous junction. Extensive areas of bare bone with no overlying mucosa were seen in posterior, inferior and anterior aspect of deep bony EAC. Tympanic membrane showed loss of normal landmarks, pars tensa was completely adherent to promontory and medial wall of middle ear with granulation tissue in superior part of tympanic membrane. Handle of malleus visualised partially; no keratin debris seen (Figure 1). Grade V lower motor neuron type facial nerve palsy (L) was present, no features of exposure keratitis seen.

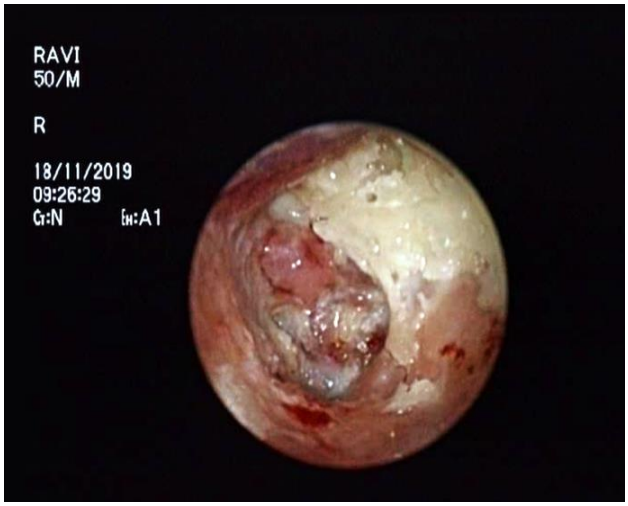

Figure 1: Otoendoscopic image of right ear showing exposed bone in external auditory canal and presence of extensive granulation with loss of normal landmarks.

Investigations: Haematological investigations were normal, HIV serology was non-reactive, chest X ray was done to rule out pulmonary tuberculosis was normal. Pure tone audiometry showed profound hearing loss (L) ear, Pus swab from EAC was processed to reveal E. coli sensitive to piperacillin and tazobactam.

Imaging: HRCT temporal bone revealed homogenous opacities filling mastoid air cells, attic, epitympanum, sinus tympani and facial recess, ossicles not visualised, scutum, tegmen and sinus plate thinned and eroded, tympanic part of facial canal eroded, hypodense opacities are also seen near internal carotid artery anteriorly.

CEMRI was done to evaluate the intracranial extension and status of internal carotid artery which showed the presence of (L) sigmoid sinus thrombosis and sequestration of (L) temporal bone suggestive of Infective or Inflammatory pathology, no intracranial extension or ICA involvement visualised.

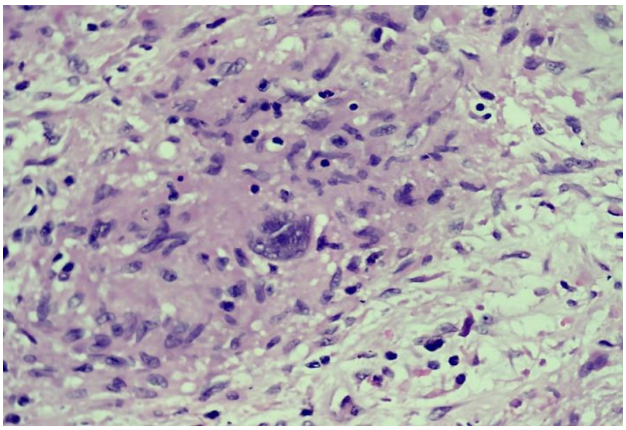

Figure 2: Photomicrograph of granulation tissue showing granuloma at 40X magnification.

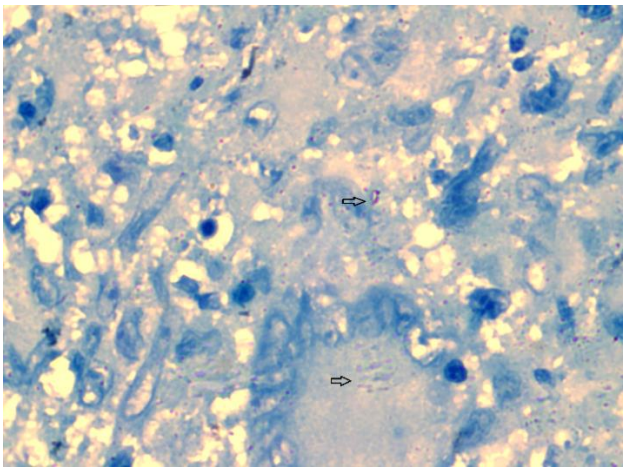

Figure 3: Photomicrograph of AFB on ZN staining (see arrow).

Histopathological examination of granulation tissue was done which revealed partially necrotic tissue with giant cell granuloma formation (Figure 2) and AFB was detected on ZN staining (Figure 3).

Gene Xpert showed very low MTb DNA with no Rif resistance indicating a non-drug resistant MTb

Management: The patient was managed medically as per RNTPC revised guidelines 2017 as category I, Anti Tubercular therapy with 2× (HRZE) intensive phase was started after DOTS registration, there was no noticeable improvement of facial nerve function, and patient was advised conservative management in form of eye protection and facial physiotherapy to limit disability and allow facial nerve to regain function. Patient is currently completed intensive phase ATT and is on continuation phase ATT and shows symptomatic improvement in decreased otorrhoea.

DISCUSSION

Tubercular otitis media was first diagnosed around 100 years ago and since then its incidence has gradually declined. It is not a very common diagnosis with which an otolaryngologist comes across very often. It generally presents with otorrhea and hearing loss which mimics more commoner ear disease. Typical findings of multiple tympanic membrane perforations are not necessarily seen. A clinician should have high index of suspicion whenever there are signs which outlies symptoms in form of severity.[13]

It is mentioned in literature that its presenting symptoms has changed over years. Initially, it was thought to present with painless otorrhea, multiple perforations of the tympanic membrane, early onset ipsilateral LMN type facial palsy, abundant granulations however these classical features are seldom seen in today's practice.

Otorrhea which is described as 'painless' can be painful at times and as described by Plester et al in 1980 and Yaniv in 1987, it could be the predominant complaint with which the patient presents to ENT OPD. The cause of otalgia was described as pressure exerted by granulation tissue over nociceptive receptors present in middle ear and mastoid. Multiple perforations of tympanic membrane which were once considered the hallmark of tubercular otitis media are rarely seen now. Presence of ipsilateral UMN type facial palsy in a case of otitis media should raise suspicion of tubercular etiology although its absence cannot rule out this diagnosis. The prevalence of facial palsy is reported as much as 21% by Mjoen et al in 1992 and our case also had history of recurrent facial palsy when he presented to us. Another significant finding reported is presence of abundant granulation which was also present in our case.[13,14]

Due to such varied presenting symptoms which also mimics more commoner ENT ailments the diagnosis of tubercular otitis media is often delayed. A clinician should have an open mind and should suspect tubercular

etiology in a case of otitis media which doesn't responds to standard therapy. A past history of pulmonary tuberculosis or a history of close contact with a tuberculosis patient should be brought out. As reported by Yaniv in 1987, as much as 50 percent patients of tubercular otitis media also have presence of pulmonary tuberculosis when investigated. Histopathological and microbiological examination of granulation tissue needs to be done as was done in our case and it is supported as a diagnostic modality by Takahashi et al, 1985. Anti-tubercular therapy (ATT) remains the mainstay of treatment and ATT is administered as per category 1 of RNTCP 2017. The disease has a very good prognosis and hearing rehabilitation is done later with hearing aids.[14]

This case highlights the fact that how closely tubercular otitis media mimics other common ear diseases as chronic otitis media squamous and also malignancy of ear. A clinical suspicion should always be there to make a diagnosis. The second point which this case highlights is necessity of obtaining microbiological confirmation of disease which can be sometimes difficult to get because of presence of thick cell wall of mycobacteria. Histological diagnosis also rules out other causes like cholesteatoma, malignancy or other systemic granulomatous diseases like Wegener's granulomatosis or granulomatosis with polyangiitis.[15]

CONCLUSION

This case highlights the diagnostic challenge and high index of clinical suspicion required for diagnosis. This case also reinforces the fact that apart from microbiological testing of acid-fast bacilli, newer testing modality based on polymerase chain should also be incorporated when deemed necessary. Combination of appropriate diagnostic modality along with good clinical vigilance will aid in early diagnosis and start of early medication which reduces chances of any subsequent complications.

ACKNOWLEDGEMENTS

The authors of the article want to acknowledge the contribution of the patient Mr Ravi who has allowed the use of medical data for this study. We thank the department of microbiology who gave us the photomicrographs.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Weiner GM, O'Connell JE, Pahor AL. The role of surgery in tuberculous mastoiditis: Appropriate chemotherapy is not always enough. J Laryngol Otol. 1977;111:752-3.
2. Myerson MC, Gilbert JG. Tuberculosis of the middle ear and mastoid. Arch Otolaryngol Head Neck Surg. 1941;33(2):23150.
3. V. Vital, A. Printza, T. Zaraboukas, Tuberculous otitis media: a difficult diagnosis and report of four cases, Pathol Res Pract. 2002;198:31-5.
4. McKibben BG, Sheehy JL. Tuberculosis of the middle ear and nasal passages. AMA Arch Otolaryngol. 1956;63(3):286-9.
5. Davidson S, Creter D, Leventon G, Katznelson D. Tuberculosis of the middle ear in an infant. Arch Otolaryngol Head Neck Surg. 1989;115:876-7.
6. Kirsch CM, Wehner JH, Jensen WA, Kagawa FT, Ampagna A, Tuberculous otitis media Southern Med J. 1995;88(3):363-6.
7. Saunders NC, Albert DM. Tuberculous mastoiditis: when is surgery indicated? Int J Pediatr Otorhinolaryngol. 2002;65:59-63.
8. Rajesh Gandham N, Sardar M, Jadhav SV, Vyawahare C, Misra R. Tuberculous Otitis with Proteus mirabilis Co-Infection: An Unsuspected Presentation Encountered in Clinical Practice. J Clin Diagn Res. 2014;8(5):1-3.
9. Kasat S, Biradar M, Deshmukh A, Jadhav S, Deshmukh H. Effectiveness of CBNAAT in the diagnosis of extrapulmonary tuberculosis. Int J Res Med Sci. 2018;6:3925-8.
10. Sharma SK, Kohli M, Chaubey J, Yadav RN, Sharma R, Singh BK, et al. Evaluation of Xpert MTB/RIF assay performance in diagnosing extrapulmonary tuberculosis among adults in a tertiary care centre in India. Eur Respir J. 2014;44(4):1090-3.
11. Chaudhary A, Barve K, Desai U, Joshi J. Utility of GeneXpert in diagnosis of multidrug-resistant extrapulmonary tuberculosis. Inter J Recent Surg Med Sci. 2017;3(2):85-7.
12. World Health Organization. Global tuberculosis report 2019. Geneva, WHO, 2019. Available at: http://apps.who.int/iris/bitstream/handle/10665/329368/978924156 5714-eng.pdf?ua=1. Accessed on 3 March 2020.
13. Vaamonde P, Castro C, García-Soto N, Labella T, Lozano A. Tuberculous otitis media: a significant diagnostic challenge. Otolaryngol Head Neck Surg. 2004;130(6):759-66.
14. Farrugia EJ, Raza SA, Phillipps JJ. Tuberculous otitis media-a case report. J Laryngol Otol. 1997;111(1):58-9.
15. Dale OT, Clarke AR, Drysdale AJ. Challenges encountered in the diagnosis of tuberculous otitis media: case report and literature review. J Laryngol Otol. 2011;125(7):738-40.

Cite this article as: Tiwari V, Singh R, Anand V. Tuberculous otitis media: rare cause of recurrent facial nerve palsy. Int J Otorhinolaryngol Head Neck Surg 2020;6:1572-5.