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Tubercular otitis media: an under diagnosed entity

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ABSTRACT

Background: Tubercular otitis media (TOM) is a well described rare entity in literature. Its characteristic diagnostic delay leads to severe irreversible complications. Diagnosis is much more difficult when it co-exists with aural cholesteatoma. Its Actual incidence is thought to be much more, than what is reported in literature. This study was carried out to establish TOM as an under diagnosed entity and to study its various presentations.
Methods: A prospective study was performed in 617 cases (both recurrence and new) of chronic otitis media who gave a fully informed consent, for being investigated for a probable diagnosis of TOM, based on history and clinical findings. Histopathological examination of the granulation tissue retrieved during biopsy/mastoid exploration surgery and mycobacterial culture of the aural discharge was performed in all cases. A final diagnosis was made with either of tests being reported as positive for mycobacterium tuberculosis infection. All confirmed cases were treated with a 6 month course of anti-tubercular therapy.
Results: Diagnosis of TOM was confirmed in 12 cases. M:F ratio was 1:1.4. Cholesteatoma was found to co-exist in 4 cases (33.33%). A positive histopathology report was obtained in 11 cases (91.67%) while the mycobacterial culture yielded growth in 3 cases (25%) only. Canal wall down mastoidectomy was performed in 9 cases. In all cases, a follow-up with a 6 months course of anti-tubercular therapy gave satisfactory healing with no complications.
Conclusions: The incidence of TOM, a potentially dangerous entity is on a rise. Role of surgery is crucial in procuring tissue for histopathological and bacteriological examination. It is a curable disease if diagnosed in time with early institution of anti-tubercular therapy.

Keywords: Tubercular otitis media, Granulation, Facial paralysis, Anti-tubercular therapy

INTRODUCTION

Tuberculosis is a major health concern for developing countries like India. It mainly affects lung, lymph nodes and visceral organs. Involvement of temporal bone in the form of tubercular mastoiditis and tubercular otitis media are uncommon occurrences. While primary tubercular otitis media is a rarer entity, majority of cases of secondary involvement are from lesion in lungs, larynx, pharynx and nose.[1]

Tubercular bacilli can gain entry into the temporal bone via various routes – insufflated through eustachian tube, haematogenous spread, contiguous involvement from adjacent intracranial or extracranial infected focus, directly through external auditory canal via perforated tympanic membrane.[2,3] Transplacental transmission and acquirement via infected birth canal have also been reported.[4]

The diagnosis of tubercular otitis is quite challenging because of nonspecific symptoms and varied clinical presentation. Co-existence of aural cholesteatoma with tubercular otitis media further complicates the situation.[5] Significant morbidity frequently results owing to the prolonged clinical course, diagnostic dilemma and

management delay. The aggressiveness of chronic otitis media and severity of complications is much enhanced with tuberculosis as its cause.[6] Assessment of its true incidence is frequently biased, as most of the available literature and documented case series come from hospitalized patients with confirmed diagnosis.[1,6]

In lieu of nonspecific clinical features, diagnostic delay and significant morbidity, a prospective study was carried out with the following objectives.

- To study the various presentations of tubercular otitis media.
- To analyze statistical data obtained from the present study in light of available literature and establishing TOM as an under diagnosed entity.

METHODS

With prior Institutional Ethical Committee approval, a prospective study was undertaken at Hi-Tech Medical College and Hospital, Bhubaneswar. During a period of 3 year, ranging from July 2011 to June 2014, a total of 3643 patients of chronic otitis media (both new and recurrence cases) presented in ENT department outdoor facility. All cases underwent routine ENT and general medical evaluation. Ear findings with respect to perforation – size, site, number, margin, state of drum remnants; presence or absence of ear discharge/granulation tissue/polyps; condition of middle ear mucosa, post aural swellings or fistulas, facial nerve paralysis and tuning fork tests, pure tone audiometry findings were noted. X-ray mastoid-schullers view, both sides to look for pneumatization and to rule out any associated mastoid pathology was done.

In them a diagnosis of tubercular otitis media was suspected on the following grounds:

Pre-operatively

- Chronic middle ear infection with painless ear discharge unresponsive to routine antibiotic therapy and no growth in routine microbiological culture of discharge
- Hearing loss – disproportionate to the disease extent clinically
- Tympanic membrane perforations – multiple/total
- Pale granulations and caseous discharge
- Non-progressive facial nerve paralysis

Post-operatively

- Poor wound healing, wound gaping/dehiscence.
- Persistent otorrhoea
- Post-mastoidectomy recurrence of granulation tissue.

From the total outdoor case load, 617 patients gave their consent for being considered as a part of the study, which was aimed for their betterment. These patients were specifically counselled to undergo investigations pertaining to tubercular otitis media. From these patients additional information was obtained in the form of demographic data, history of tuberculosis in past or a history of contact with a tubercular patient, ESR, HIV status, chest X-ray, Mantoux test, CT-Scan temporal bone. Granulation tissue and/or pus collected either on OPD basis or during biopsy/mastoid exploration surgery were sent for histopathological analysis and mycobacterial culture. A specially prepared proforma was used to document the details of history, investigations, pre-operative examination, intra-operative and follow-up findings. The diagnosis was confirmed on the basis of positive mycobacterial culture and/or histopathology report.

RESULTS

In the present study covering 617 patients of chronic otitis media, the diagnosis of tubercular otitis media was confirmed in 12 cases. There were no dropouts and patient complications in the study. Patient details and symptoms have been tabulated in Table 1. Amongst these 12 cases, there were 5 male and 7 female patients. M:F ratio was 1:1.4. There were 4 pediatric cases (age <15 years). There were 8 new cases while 4 were recurrence cases. Findings of clinical examination, pure tone audiometry, CT scan temporal bone, chest x-ray, Mantoux test test and HIV status have been summarized in Table 2. Left ear (8 cases, 66.67%) was more commonly affected than the right ear. Facial nerve paralysis was seen in 5 cases (41.67%). Of these, there were 2 pediatric cases (50%) and 3 adult cases (37.5%). Positive chest x-ray suggestive of active tubercular infection was found in 8 cases (66.67%). Mantoux test was positive in 83.33% (10) cases. Treatment details, Intra-operative findings, results of histopathology examination and mycobacterial culture have been depicted in Table 3. In 9 cases canal wall down mastoidectomy was performed, while in 3 cases (all 3 were recurrence cases) diagnosis was confirmed by biopsy of granulation tissue. Cholesteatoma was found to co-exist in 4 cases (33.33%). A positive histopathology report was obtained in 11 cases (91.67%) while the mycobacterial culture yielded growth in 3 cases (25%) only. Post-operatively Anti-tubercular therapy was given to all cases for 6 months. Post-operatively all cases were followed up for 6 months and satisfactory healing was achieved in all 12 cases.

DISCUSSION

Although tuberculous mastoiditis has come a long way following the earliest descriptions by Jean Louis Petit (18th centuary) and detailed clinical insights provided by Wilde (1853), its diagnosis still remains an enigma.

Involvement of middle ear cleft can be primary or secondary in origin. Primary involvement is exceedingly

rare. Although secondary involvement of temporal bone is the commoner type, a high incidence of primary type was reported by Plester et al.[7] The various routes by which the tubercular bacilli gains access into the middle ear cleft are – aspiration of infected mucus through Eustachian tube, haematogenous spread, contiguous involvement from adjacent intracranial or extracranial infected focus, and rarely as a direct seedling through external auditory canal via perforated tympanic membrane.[2,3] Other rare routes mentioned are transplacental transmission and via infected birth canal.[4]

Table 1: Patient symptoms.

S.no.	Age (years)	Sex	Case type	Pain	Ear discharge duration (years)		Deafness		Tinnitus		Vertigo
					R	L	R	L	R	L	
1	8	M	N	Mild	-	5	-	+	-	-	-
2	55	F	REC	-	15	2	+	-	+	-	Occasional
3	10	M	N	-	-	6	-	+	-	-	-
4	33	F	REC	-	4	-	+	-	+	-	Occasional
5	23	F	N	-	-	3	-	+	-	-	-
6	49	M	N	Mild	1	4	-	+	-	-	-
7	17	F	REC	-	-	3	-	+	-	-	-
8	9	M	N	-	5	-	+	-	+	-	-
9	27	F	REC	-	-	5	-	+	-	+	Occasional
10	22	F	N	-	-	4	-	+	-	+	-
11	45	M	N	Mild	2	3	+	+	-	-	-
12	10	F	N	-	-	3	-	+	-	-	-

R-Right ear, L-Left ear, M-Male, F-Female, + Present, - Absent, N-New, REC- Recurrence

Table 2: Signs observed.

S.no	TM perforation		Middle ear	Facial paralysis		Deafness		CT scan findings	CXR	HIV	Mantoux
	R	L		R	L	R	L				
1	-	TP	G	-	-	-	SEV	M- hazy & bone erosion, ME- soft tissue opacity & Ossicular erosion	-	-	+
2	TP	CP	G	+	-	SEV	MIL	M-Cortical erosion, ME- Ossicular erosion	+	-	+
3	-	G	G	-	+	-	SEV	ME- Ossicular erosion	-	-	+
4	STP	-	G	-	-	MOD	-	M-hazy, ME-soft tissue opacity	+	+	-
5	-	STP	-	-	-	-	MOD	M-hazy, ME-Ossicular erosion	+	-	+
6	CP	TP	G	-	+	MIL	SEV	M- hazy & bone erosion, ME- soft tissue opacity	+	-	+
7	-	STP	G	-	-	-	MOD	ME- soft tissue opacity	+	-	+
8	TP	-	G	+	-	SEV	-	M- hazy & bone erosion, ME- soft tissue opacity & Ossicular erosion	-	-	+
9	-	TP	G	-	+	-	MOD	ME- soft tissue opacity	+	+	-
10	-	G	G	-	-	-	SEV	M- hazy & bone erosion, ME- soft tissue opacity	-	-	+
11	STP	CP	G	-	-	SEV	MIL	M- hazy & bone erosion, ME- soft tissue opacity & Ossicular erosion	+	+	+
12	-	STP	G	-	-	-	SEV	M- hazy & bone erosion, ME- soft tissue opacity	+	-	+

R-Right ear, L-Left ear, CXR- Chest x-ray, CP-Central perforation, AR-Attic retraction, STP-Subtotal perforation, TP-Total perforation, G-Granulation tissue, + Present, - Absent, MIL- Mild, SEV-Severe, Mod-Moderate, M-mastoid, ME-Middle ear.

Table 3: Treatment, intra-operative findings, H.P. Study and culture.

S.no	Treatment			Intra-operative findings		H.P. Study	Culture
	ATT	R	L	R	L		
1	+	-	CWD-M	-	M- G ME- G, OE, C	+	-
2	+	CWD-M	MP	M- G ME- OE, C	-	+	+
3	+	-	CWD-M	-	ME- OE, G	+	-
4	+	BIOPSY	-	-	-	+	-
5	+	-	CWD-M	-	M- G ME- G, OE	+	+
6	+	MP	CWD-M	-	M- G ME- G, OE, C	+	-
7	+	-	BIOPSY	-	-	+	-
8	+	CWD-M	-	M- G ME- G, OE	-	+	-
9	+	-	BIOPSY	-	-	+	-
10	+	-	CWD-M	-	M- G ME- G, OE, C	-	+
11	+	CWD-M	MP	M- G ME- G, OE	-	+	-
12	+	-	CWD-M	-	M- G ME- G, OE	+	-

R-Right ear, L-Left ear, CWD-M Canal wall down mastoidectomy, + Positive, - Negative, MP-Myringoplasty,M-Mastoid,ME-Middle ear,OE-Ossicle erosion,G-Granulation, C-Cholesteatoma.

Disease onset is generally insidious. Initially the aural discharge is generally serous, which changes to thick, mucoid or mucopurulent with superadded bacterial infection.[3] The otorrhoea is usually painless. However, in certain cases there is neither a tympanic membrane perforation nor the associated otorrhoea.[7] Multiple perforations of tympanic membrane, though once considered a hallmark of TOM, is not a valid diagnostic criteria now. It is believed that these multiple perforation coalesce to result in a single large perforation. Pale granulomatous middle ear mucosa and relative avascularity, is a significant feature.[8]

With further progression of the disease process, granulation tissue dissolutes the ossicles and fills the mastoid air cells system and blocks aditus ad-antrum. Unchecked bone destruction continues which ultimately results in Osteomyelitis of the temporal bone.[3] In comparison with non-cholesteatomatous or simple type of chronic otitis media, the complications (facial paralysis, mastoiditis, labyrinthitis, sensorineural hearing loss) in TOM are significantly higher and severe.[9]

Hearing loss is inconsistent with the disease extent.[10] Although a conductive hearing loss is appreciable in 90% cases, with prolonged duration a mixed or sensorineural hearing loss is also seen.[3,11] Associated preauricular, postauricular or cervical lymphadenopathy is a regular finding.[12]

Towards the end of 19th century, approximately 3-5% of all chronic suppurative otitis media cases were of tubercular origin. However with the advent of anti-tubercular therapy, the incidence dropped significantly.[13] Incidence of TOM as reported by various researchers over the last century includes 0.37% (Singh B, 1991) and 2.8 % (Turner and Eraser, 1915), 0.5% (Ormoldd FC, 1931), 0.9% (Palva et al, 1973), 0.04% (Weiner et al, 1977).[8,14-17] In the present study, an overall three year incidence of 1.95% (12 cases amongst 617 chronic otitis media cases) was recorded. This apparent rise in incidence can be attributed to increased prevalence of immunocompromised states (HIV-AIDS, immune-suppression therapy, diabetes mellitus) and rising antibiotic resistance in the tubercular bacilli. For paediatric population (age <15years) the incidence recorded was 0.65%, while it was 0.44% as reported by Singh B, 1991.[8]

Amongst confirmed TOM cases, a M:F ratio of 1:1.4 (5 male and 7 female) was observed. Male to female ratio reported by other authors include 1:3 (Vital et al, 2002) and 1.5:1 (Meher et al, 2006).[12,18] It was noticed that left ear (8 cases, 66.67%) was more commonly affected than the right ear, and tubercular lesion was noted only on one side, even though a few patients had bilateral chronic otitis media. Involvement of middle ear cleft by tuberculosis is generally unilateral, cases with bilateral involvement have also been reported.[19,20]

The subjective complaint of deafness was confirmed in all cases by pure tone audiometry, which showed presence of conductive hearing loss (moderate degree) in 4 cases and mixed hearing loss in 8 cases (severe degree). The degree of hearing loss was graded according to the American Speech Language hearing Association classification (Clark, 1981). Facial nerve paralysis is present in 16% adult cases of TOM, while incidence in pediatric cases is around 35%.[8] In our study the incidence of facial paralysis was 41.67% (5 cases), of which there were 2 pediatric (50%) cases and remaining 3 were adult (37.5%). The overall incidence of facial paralysis reported by other workers is 39.54% (Singh B, 1991) and 39.13% (Samuel J and Fernandes CM) respectively.[8,21] In 3 patients, who complained of occasional vertigo as part of their symptoms, a positive Rhomberg sign was appreciated during clinical examination.

We found TOM to co-exist with aural cholesteatoma in 33.33% (4) cases. Records from available literature report an incidence of coexistence in 14.29% (Chaturvedi et al, 1986), 15.15% (Yaniv E, 1987) and 20% (Brian L, 1972) cases.[22-24] Cases of coexistent cholesteatoma and tubercular otitis constitute a real diagnostic challenge since both are non-responders to conservative treatment and cause extensive bone destruction.[5]

The diagnosis of TOM is challenging, requires a high level of suspicion and is based on pre and post-operative histological and bacteriological findings.[12] During histopathological study, well-formed epithelioid granulomas and few Langhan's giant cells in background of lymphocytes and histiocytes, and necrosis are suggestive of tuberculous etiology. In this study granulomas suggestive of tuberculosis were found in 91.67% (11) cases. Other researchers have found them in 100% (Chaturvedi et al, 1986), 85.71% (Plava et al, 1973) and 80% (Varty et al, 2000) cases respectively.[16,22,26]

Smear positivity for acid fast bacilli in aural discharge reaches upto 20% of cases.[3] Superadded infection makes it difficult to demonstrate tubercular bacilli in ear discharge, and culture also provides a low yield.[26] Culture for *Mycobacterium tuberculosis* (performed using BACTEC system) was positive in 25% (3) cases in this study, while others have found culture positivity to the extent of 16.13% (Yaniv E, 1987), 28.57% (Plester et al, 1980) and 90.91% (Windle Taylor PC and Bailey CM, 1980).[6,7,23]

Mantoux test was positive in 83.33% (10) cases, while it was 100% in the study of Yaniv.[23] A history of having received BCG vaccine was available in all cases. 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, and especially in endemic areas.[27,28]

Chest X-ray showed evidence of active pulmonary tuberculosis in 66.67% (8 cases). Results from other reports include a positivity to the extent of 42.86% (Plester et al, 1980), 51.61% (Yaniv E, 1987) and 93.02% (Singh B, 1991).[7,8,23] The imaging modality of choice in cases of TOM is CT-scan. It not only helps in visualizing disease extension, but also identifies bony sequestrum and thus helps to plan surgery. MRI can be of help in diagnosing intracranial extension.[29]

In the present study, 3 patients (25%) were found to be HIV positive. In order to identify resistant bacilli, drug sensitivity testing must be performed following isolation of bacilli in culture.[30] Polymerase chain reaction (PCR), of the ear discharge can aid in analysis of treatment results and identifying bacilli in aural discharge.[28]

The diagnosis of TOM was established by biopsy and histopathological study of the granulation tissues obtained on outdoor basis in 3 cases (all 3 being recurrence cases). With the institution of ATT the granulations resolved and ear became dry. Amongst remaining 9 cases, cholesteatoma was found to co-exist with tubercular granulations in 4 cases. In all 9 cases the disease extent necessitated a canal wall down mastoidectomy (CWD-M). The granulations collected intraoperatively, turned out positive for mycobacterium tuberculosis (by culture and/or histopathological study). Hence all these cases were put on ATT which resulted in attainment of a dry and safe ear.

Amongst these 12 cases of TOM, there were 3 cases of bilateral chronic otitis media. In all 3 cases, the routine antibiotic therapy (prescribed on outdoor basis) rendered an ear dry only unilaterally, while the other continued to discharge. Myringoplasty was performed in these 3 ears which resulted in successful closure of tympanic membrane perforation. In all the 3 cases the other ear was operated after 4-6 months, wherein a diagnosis of TOM was established and managed accordingly.

Anti-tubercular therapy (ATT) for a period of 6 to 12 months remains the mainstay of treatment for tuberculous otitis media.[3] Within 2 months of institution of ATT the otorrhoea usually subsides,[11] and its character in follow serves as a marker to monitor treatment efficacy.[7,11] Role of surgery is limited to biopsy in doubtful cases and in management of complications like facial palsy, postauricular fistula, subperiosteal abscess, removal of bony sequestrum and intracranial extension.[29] Failure to start appropriate anti-tubercular therapy following a tympano-mastoid surgery in TOM cases can be catastrophic and cause non healing suture lines, fistulae and surgical failure.[3]

CONCLUSION

Tubercular otitis media is a potentially dangerous rare entity. There is a rising trend in its incidence. The characteristic diagnostic delay is responsible for associated severe morbidity. Its probability should be kept in mind while dealing with cases of intractable

otorrhoea associated with pale granulation tissue in the middle ear cleft. Surgery plays a vital role in procuring tissue for histopathological and bacteriological examination. It is a curable disease if diagnosed in time with early institution of anti-tubercular therapy.

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