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Clinicopathological Evaluation of Tympanosclerosis

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Abstract

Introduction: Tympanosclerosis is a disorder associated with calcareous deposits in various parts of the middle ear. It is an irreversible end result of any unresolved specific or nonspecific inflammatory disease of the middle ear characterized by anatomical distortion resulting in conductive type of hearing loss.[1]

Objective: This study was conducted in a tertiary care teaching hospital to know the incidence, to study clinical features, histopathology and the quantum of hearing loss secondary to tympanosclerosis (TS) in cases with chronic otitis media (COM) undergoing surgical treatment.

Methodology: All patients with chronic otitis media (COM), either mucosal or squamosal type undergoing surgery were included in the study.

Results: Out of 160 cases operated for COM, 52 cases (32.5 percent) had tympanosclerotic plaques. In 52 cases with TS plaques, 5 cases (all are males) were squamosal type with histopathological evidence of cholesteatoma and rest 47 cases were of mucosal type (24 males & 23 females). On histopathological evaluation of the TS plaques, only 7 cases showed extensive hyalinization and the reminder showed fibrotic changes, granulation tissue, dense collagen, scattered fibroblasts and inflammatory cells.

Conclusion: TS is a common association, clinically and pathologically with COM more with mucosal type. TS causes significant hearing loss compared to similar cases without TS. TS patches over critical areas like stapes foot plate, facial nerve canal and semicircular canals may be difficult to manage because of their strategic location.

Keywords: Tympanosclerosis; Chronic otitis media; Cholesteatoma.

Introduction

Tympanosclerosis is a condition associated with hardening of the middle ear cleft. (Tympanum-middle ear; sclero-hardening; osis-condition). It is a non-functional and non-changeable inert repair phenomenon.[1] The condition is characterised by calcareous deposits in the tympanic membrane, middle ear cavity, ossicular chain and occasionally in the mastoid.[2] Von Troltsch (1873) was the first person to coin the term Taukensklerose to describe sclerotic changes in the middle ear mucosa. Zollner (1955) later called it tympanosclerosis.[3] The fibrosis occurs in the submucosal connective tissue layer covering the auditory ossicles, lining the bony walls of the tympanic cavity and forms the middle fibrous layer of the tympanic membrane.[4] Sclerosis is the final stage of the dynamic inflammatory process.[1] TM(Tympanic membrane) is the most common site for tympanosclerotic patches where it is called myringosclerosis. It is considered as a long term sequelae of Chronic Otitis Media (COM).[5]

It is proposed that TS is not usually associated with active mucosal disease and with cholesteatoma.[6] As tympanosclerosis has a mysterious nature, this study was planned to know the incidence at our place, the clinical features, histopathology, level of hearing impairment and to plan the management of tympanosclerosis in both mucosal and squamosal type of chronic otitis media.

Materials and Methods

Study design

Patients coming to the ENT outpatient department of our tertiary care referral hospital, diagnosed with chronic otitis media either mucosal or squamosal type and posted for surgery, were included in the study. Patients with otosclerosis and non-chronic otitis media cases undergoing surgery, those with intracranial complications were excluded from the study. The study was conducted for a period of 18 months.

Sample size

A minimum of 50 patients was the sample size proposed for the study. However, the final sample size was 160 in 18 months.

Methodology

Ethical clearance was obtained from the institutional ethics committee. Relevant clinical and demographic data were obtained from the patients. A detailed clinical examination was performed. Informed and written consent was taken from all the patients. Audiological assessment (pure tone and impedance audiometry), radiological assessment (X ray bilateral mastoids lateral oblique view and in selected cases, HRCT temporal bone), laboratory investigations (complete blood count, bleeding time/clotting time, random blood sugar, blood urea, serum creatinine and urine analysis) and histopathology of excised plaques were done.

Results

Out of 160 cases operated for chronic otitis media, 52 (32.5%) had histopathological evidence of tympanosclerotic plaques with slightly higher preponderance in males. (56%) 5 cases were associated with cholesteatoma. (10%) All these 5 cases were males. Rest of 47 cases with TS patches were having male to female ratio of 24: 23 (Table 1).

Table 1: Gender distribution of squamosal disease (SPSS version 22.0).

	Male	Female	Total	p-value
TS patches associated with cholesteatoma	5	0	5	0.04* Fisher's Exact
TS patches not associated with cholesteatoma	24	23	47	test *P<0.05 statistically significant
Total	29	23	52	

Out of 52 cases, 29 underwent tympanoplasty. 16 underwent cortical mastoidectomy and tympanoplasty. 4 cases underwent modified radical

Table 2: Types of Surgeries (SPSS version 22.0).

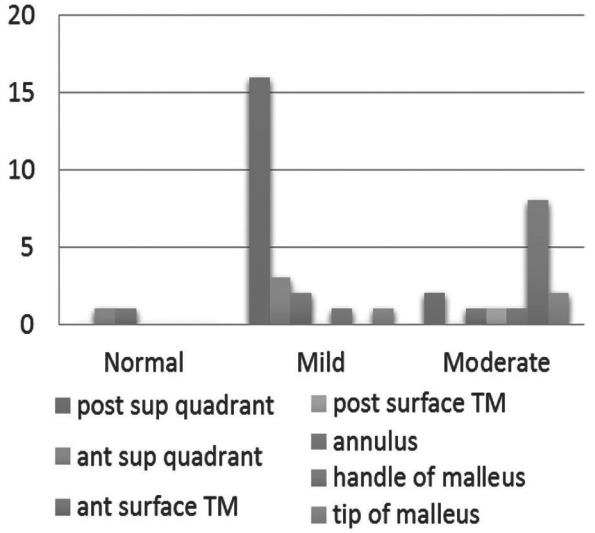
	Tympanoplasty	Cortical + Tympanoplasty	Intact Canal wall Mastoidectomy + Atticotomy + Tympanoplasty	MRM + Tympanoplasty	
Type 1`	25	12	2	1	0.048*
Type 2	4	4	1	1	Fisher's Exact test.
Type 3 & above	0	0	0	2	*P<0.05 statistically significant

mastoidectomy and tympanoplasty. Other 3 cases underwent intact canal wall mastoidectomy with atticotomy and tympanoplasty (Table 2).

Out of 52 intraoperative cases with TS patches, 40 cases (77%) had intact and mobile ossicular chain. Seven cases (13%) had partially eroded malleus, three cases (6%) were having absent malleus, one case (2%) both malleus and incus were absent and other one case (2%) where all the ossicles were absent except foot plate of stapes.

Hearing loss and distribution of TS patches where ossicular chain was intact and mobile in 40 cases were studied. (figure no.1) TS patches were more in posterosuperior quadrant of TM with mild CHL in 16 cases and moderate CHL seen in 8 cases with distribution of TS patches in handle of malleus. (Graph 1 -Y axis denotes number of cases).

Graph 1: Hearing loss & distribution of TS patches.



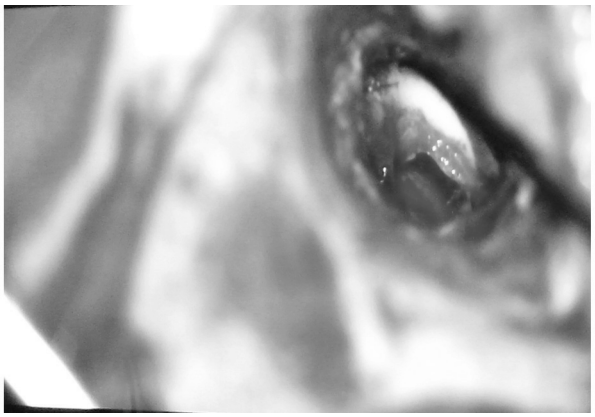


Fig. 1: Intraoperative TS patch in the anterosuperior quadrant of TM with intact handle of malleus.

Considering degree of hearing loss in 52 patients, out of the 5 cases associated with squamosal disease, 3 cases showed moderately severe mixed hearing loss and one each with moderate and mild CHL. Rest of the 47 cases had hearing pattern as per table no.3. The middle ear mucosa was healthy in 16 cases who underwent only tympanoplasty. Remaining cases of mucosal disease had thickened and oedematous mucosa. The mucosa was unhealthy in all the squamosal cases.

Table 3: Degree of hearing loss (SPSS version 22.0).

Hearing Status	Without Cholesteatoma	Cholesteatoma + TS patches	Total
Normal hearing	2	0	2
mild CHL	24	1	25
moderate CHL	14	1	15
moderately severe mixed HL	7	3	10
Total	47	5	52
	Fisher's exact value = 4.95, p= 0.128(NS) Fisher's exact test P<0.05 statistically significant p>0.05 non-significant, NS		

Regarding histopathology of TS patches, fibrotic changes were more evident with fragments of keratin material. Cases associated with Cholesteatoma showed granulation tissue, dense collagen, scattered fibroblasts and inflammatory cells. Only 13 cases showed extensive hyalinization and calcification, on which 2 cases were cholesteatoma associated. Two cases had associated refractory foreign material with nonspecific granulation tissue surrounded with chronic inflammatory infiltrate.

Discussion

Cassebohm described this entity as `chalky patches' in the tympanic membrane as early as in 1734. Gibb AG (1976) called this as the 'cinderella of the middle ear disease.[1] The presentation of the disease is variable and aetiologies may be different. There are too many hypothesis on the aetiology of TS. It is proposed to be secondary to infection associated with necrosis and granulation, ischemic alterations, and/or stasis of infected materials. It could be caused by immunologic reaction. Connective tissue components are stimulated because of infection, inflammation or trauma and this initiates the local immunological reaction in the submucosa.[7]

Tympanosclerosis though a relatively benign condition, causes significant conductive type of hearing loss. The severity of hearing loss depends on the extent of the tympanosclerosis plaques and their location. which can be judged only after formal exploration of the middle ear.[5] The sclerotic changes

may damage the middle ear both anatomically and functionally.[8] It is usually because of inflammation, but rarely may appear after trauma or surgery or any non-treated inflammatory process may be the cause.[9]

The incidence of tympanosclerosis in chronic otitis media has been reported to range from 9% to 38%. It appears histologically as an acellular hyalinization of the subepithelial connective tissue of the tympanic membrane and middle ear associated with calcification and neo osteogenesis. Ossicular fixation occurs most frequently in the attic and is associated with the heads of the malleus and body of incus. When plaques occur within the tympanic membrane, they are limited to the lamina propria.[10] Microscopically, there is hyalinized collagen in the submucosa with a lamellar arrangement. Mature fibrocytes may be seen between collagen fibers.[11]

Under light microscopy, four different phases in the development of tympanosclerosis can be observed histologically. During the early phase, vesicles of around 50-300 nm in diameter are formed in the fibroblasts, inflammatory cells, and epithelium cells of the extracellular collagenous matrix followed by the appearance of crystalline like inclusions called calcospherules in these vesicles. Calcium and phosphate precipitate in these spherules and mineralization begins. During the last phase, masses that are completely mineralized appear and they are then called plaques.[12]

Under otoendoscope or microscope, plaques are classified as type I (soft), type II (moderately hard) and type III (very hard).[13] In type I, fibroblasts and collagen fibers are equally abundant in typical loose connective tissue. A few small calcium crystals are also seen. In type II TS, large bundles of collagen fibres, proliferation of fibroblasts and focal calcification points are seen. In type III, round shaped chondroblast like cells located in lacunae and intense calcification points are evident.[13]

Another way to classify TS is based on their morphological and histological aspects. Histological classification is based on the maturation of the tissue, and thereby helps to grade the disease. In type I tympanosclerosis, even after surgical excision, the underlying process may go on and new sclerotic tissue formation can be expected. Type III sclerotic tissue is associated with limited and inactive disease. Progress of the disease and the patient's benefit from surgery can be interpreted according to this classification.[13]

Radiologically tympanosclerosis appears as unifocal or multifocal punctate or web like calcifications in the middle ear cavity or on the tympanic membrane. New bone formation (fibro-osseous sclerosis) is usually seen in the attic and is the least common manifestation. Thick bony webs or generalized bony encasement may be seen in computer tomography (CT).[14]

Tympanoplasty and ossicular reconstruction in ears with tympanosclerosis carry higher risk of cochlear damage than in other middle ear diseases because of the extensive dissection that is required in these ears and the coexistence of labyrinthine fistula.[10] The dense plaque on the tympanic membrane reaching the perforation edge should be removed from the medial aspect to facilitate vascularization of the graft.[15]

In tympanosclerosis with intact tympanic membrane, if there is a significant conductive hearing loss, it indicates ossicular chain involvement and surgery is indicated. After removing tympanosclerosis plaques and involved ossicles, ossiculoplasty with or without tympanic membrane reconstruction is performed.[16] On the other hand, if the stapes is fixed, a staged operation is necessary in which a first stage myringoplasty and management of the malleus and the incus is followed by a second stage surgery after a few months' interval.[16]

Several studies were conducted regarding tympanosclerosis. Most of the studies were focussing on the incidence, histopathology, distribution of plaques and degree of hearing loss.

Pal I and Sengupta A did a study on the distribution of tympanosclerosis, their clinical presentation, and the possible surgical treatment, the results of those surgeries and the histopathological nature of tympanosclerosis plaques. They found that TS occurs equally in both sexes and the commonest site being tympanic membrane affecting any quadrant of pars tensa sparing pars flaccida, associated commonly with conductive hearing loss and very rarely sensorineural hearing loss (SNHL). Histopathology revealed dense bundles of collagen with hyaline degeneration and scattered areas of calcification.[1]

Kaur K, Sonkhya N, Bapna as did another study to identify the incidence of tympanosclerosis amongst patients with chronic suppurative otitis media and also to study the correlation between the degree of hearing loss and the site of tympanosclerosis. Audiometric and operative findings of 200 patients of chronic suppurative otitis media were analysed. The incidence of tympanosclerosis was found to be 19% (out of 200 patients). The hearing loss associated

with tympanosclerosis was of the conductive type in the majority of cases. Ossicular mobility was found to be normal in 71.1% of the cases.[3]

In our study, males have slightly more incidence of TS patches (M: F - 24:23). Also, TM was the most common site for tympanosclerotic patches with majority seen in posterior superior quadrant of the TM (mild CHL), followed by handle of malleus (moderate CHL). Regarding histopathology of TS patches, fibrotic changes were more evident with fragments of keratin material. Two cases with cholesteatoma had refractory foreign material with nonspecific granulation tissue surrounded with chronic inflammatory infiltrate. In our study, 40 (77%) had intact and mobile ossicular chain. Seven cases (13%) had partially eroded malleus, three cases (6%) were having absent malleus, one case (2%)–both malleus and incus were absent and other one case (2%) where all the ossicles were absent except foot plate of stapes.

Asiri S, Hasham A, Anazy F A, Zakzouk S, Banjar A conducted a study to estimate the incidence of tympanosclerosis among patients with chronic suppurative otitis media (COM), its association with cholesteatoma and also the type of hearing loss as well as its relation to the degree and site of tympanosclerosis. They concluded that incidence of tympanosclerosis was found to be 11.6 per cent (90 patients out of 775 COM cases) and also found that association of cholesteatoma and tympanosclerosis may be regarded as uncommon (2.2 percent).[17]

In our study, out of total 160 cases operated for chronic otitis media, 52 (32.5%) has histopathological evidence of tympanosclerotic plaques. Ten percent (5 cases) also showed association with squamosal disease, which was significantly higher compared to previous studies. All of the 5 cases who were associated with squamosal disease were males.

Yetiser S, Hidir Y, Karatas E, Karapinar U conducted a study to review the previous reports and to analyze the long-term surgical outcome of 30 patients who have been operated for tympanosclerosis. They concluded that the success of the surgery was dictated by the location and the extent of tympanosclerotic involvement.[18]

In our study, out of 52 cases, 29 underwent tympanoplasty. 16 patients underwent cortical mastoidectomy and tympanoplasty. Modified radical mastoidectomy with tympanoplasty done in 4 cases and other 3 underwent intact canal wall mastoidectomy with atticotomy and tympanoplasty.

Summary and Conclusion

The purpose of this study was to evaluate the patients with TS patches in chronic otitis media of both mucosal and squamosal type. Total of 160 cases were operated for chronic otitis media, 52 (32.5%) had histopathological evidence of tympanosclerotic plaques. Males were showing slightly more incidence. Association of squamosal disease (cholesteatoma) with TS patches was seen significantly higher compared to previous studies. Previously not more than 5 percent were seen; however, our study showed a 10 percent association. Interestingly all the subjects with cholesteatoma were males. The TS patches were seen more in the posterosuperior quadrant of TM where the hearing loss is only mild, followed by handle of malleus with moderate CHL.

On histopathology, Cholesteatoma associated cases showed granulation tissue, dense collagen, scattered fibroblasts and inflammatory cells. Fibrotic changes were more evident with fragments of keratin material. Only 13 cases showed extensive hyalinization and calcification, of which 2 cases were associated with cholesteatoma.

TS is commonly associated clinically and pathologically with COM more with mucosal type. It causes significant hearing loss compared to similar cases without TS. TS patches over critical areas like stapes foot plate, facial nerve canal and semi-circular canals may be difficult to manage because of their strategic location. The present study starred a significant association of TS with COM. The associated clinical manifestations, hearing loss and management of these cases as done in this study, add to the existing information in the literature.

More studies are needed to unfold the mystery of TS patches in future. The surgeon has the key role depending on expertise to remove the plaques with less morbidity.

Abbreviations

TS – Tympanosclerosis

COM – Chronic Otitis media

TM – Tympanic membrane

CHL – Conductive hearing loss

HRCT – High Resolution Computed Tomography

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