CLINICOPATHOLOGICAL AND AUDIOLOGICAL STUDY OF TYMPANOSCLEROSIS

Indranil Pal, A Sengupta

**ABSTRACT:** Tympanosclerosis is the terminal irreversible stage of a pathological process based on inflammation of the middle ear mucosa which has not regressed to restitutio ad integrum and which does not continue as an inflammatory process. It is a non functional and inert repair phenomenon. However in spite of its inertness, it is an important clinicopathological entity as it can cause stiffness of the sound conducting mechanism of the ear. This study aims to understand the distribution of tympanosclerosis, their clinical presentation, the possible surgeries, the results of those surgeries and the histopathological nature of the tympanosclerotic plaques.

**Key words:** tympanosclerosis, plaques, histopathology.

**INTRODUCTION**

The term tympanosclerosis literally means hardening of the middle ear. *(Tympanum - middle ear; sclero - hardening; osis - condition).*

Tympanosclerosis is described as an irreversible, though not immutable end result of any unresolved specific or nonspecific inflammatory disease of the middle ear, characterized by anatomical distortion resulting in functional impairment that is hearing loss of the conductive type. It is a nonfunctional and inert repair phenomenon which is however not changeable. There is no overall evidence of resolution of the sclerotic changes with time (Maw A R, 1991). The inflammation is dynamic and evolutionary in nature, whereas the sclerosis is the final stage of a process (Ferlito 1979).

**AIMS AND OBJECTIVES**

Our study was aimed at the following objectives:-

a. Age and sex prediction for the occurrence of tympanosclerosis
b. Common sites of involvement in the middle ear cleft and tympanic membrane.
c. To determine the hearing status in patients afflicted with tympanosclerosis. Hearing status in these patients after surgery for removal tympanotic plaque as far as practicable.
d. Histological pattern of the excised plaques
e. Whether mastoid exploration is needed in the cases of mastoid involvement with tympanosclerosis

**MATERIALS AND METHODS**

**Patients**

For this study, 50 patients were selected from the general pool of patients attending the ENT OPD of Medical College Hospital, Kolkata, between the dates of 01 September 2000 to 31 August 2002, with a clinical diagnosis of tympanosclerosis. There were no other specific selection criteria other than the presence of tympanosclerotic plaques.

**METHOD**

a. Recording of history.
b. Clinical examination.
c. Radiological assessment of the mastoids and tympanic cavity
d. Audiological assessment
e. Middle ear compliance- Tympanometry and tests for acoustic reflexes.
f. Examination under microscope.
g. Findings during surgery.
h. Postoperative hearing assessment.

**HISTORY**

The first description of tympanosclerosis was made by Cassebohm in 1734.

Von Troltsch(1873) first described the term ‘Paukensklerose’ or ‘Typanosclerosis’. Thereafter Walb(1893) also carried on research on this subject. It seems that since 19th century, there was a gap of almost 50 years, when tympanosclerosis was forgotten totally in the world literature. The term was then

Tympanosclerosis is thus the ‘Cinderella of the middle ear disease’ (Gibb, 1976), because it is a forgotten and neglected entity in the practice of otology.

Further pathological and clinical studies on tympanosclerosis have been carried out by other authors (Goodhill, 1960; House and Sheehy, 1960; Harris, 1961; Harris and Weiss, 1962; Sheehy and House, 1962; Joseph and Gordon, 1963; Chang, 1969; Friedmann, 1971; Smyth, 1972; Tos and Bak-Pedersen, 1974 etc.)

The aetiology is basically unknown. However, various hypotheses exist:-

a. A sequelae of severe acute otitis media, either bacterial or viral (Gibb 1976).

b. Stagnation of secretion into the mucosal fold(Smyth,1972)

c. Disordered fibrogenesis during healing following longstanding inflammation:

d. Immunological basis (Schiff and Yoo, 1985).

e. The role of osteopontin (Makiishi - Shimobayashi C. et al, 2001)

f. Role of HLA antigens HLA-B35 and HLA- DR 3 may play an important role (Dursun et al,1997)

g. Degenerative processes in otitis media (Friedmann, 1993)

HISTOPATHOLOGY

Tympanosclerotic plaques clinically vary from small patches to a diffuse process involving and enveloping the entire tympanic cavity.

Light microscopy of tympanosclerotic plaques shows dense layers and bundles of collagenous fibrous tissue arising from the granulation tissue covering and replacing the mucosa of the middle ear cavity. The lesion shows hyaline degeneration and calcification. The lesion appears to be avascular. These changes may also occur in the underlying bone.

PATHOGENESIS

The first stage of tympanosclerosis is effusion in the mastoid air cells. With progression if resorption of the exudates doesn’t take place, the characteristics of the exudates change with increased cellular infiltrate and increased protein content. It is then called successively serous, mucous and suppurative otomastoiditis.

The three types of otomastoiditis may:

a. Regress to ‘restitutio ad integrum’.

b. Change in severity of inflammation, thus progressively becoming acute suppurative otomastoiditis, as also it may become chronic causing chronic suppurative otomastoiditis.

c. Evolve towards tympanosclerosis

Clinicopathological Considerations

The plaques typically occur in certain sites of election, most commonly in the ear drum, stapes and oval window area, subfallopian groove and upper promontory. Adjacent structures such as the long process of the incus, stapedius tendon and horizontal part of the fallopian canal are also involved. Deposits are common in the epitympanum and in relation to the malleus, but tympanosclerosis in the Eustachian tube area, the hypotympanum and the round window niche is rare while deposits in the mastoid are uncommon (Gibb 1971).

Tympanosclerosis destroys the ossicular chain by the process of strangulation effect on the blood supply and not by direct destructive effect. Erosion of the long process of incus and stapes crura is the commonest causes of discontinuity (Gibb, 1976).

The anatomical & physiological impairment is greater if the area involved is the epitympanum. If it involves the head of malleus & body of incus assembly, it can give rise to the fixed malleus head syndrome (Powers et al, 1967; Goodhill, 1966; Sleeckx, 1967 and Davies, 1968 - as quoted by Ferlito A, 1979). Also the stapes footplate may be fixed at the oval window by a tympanosclerotic plaque causing pseudo otosclerosis.

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>0-10</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>11-20</td>
<td>1</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>21-30</td>
<td>10</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>31-40</td>
<td>4</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>41-50</td>
<td>5</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>51-60</td>
<td>-</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>24</td>
<td>26</td>
<td>50</td>
</tr>
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Table 2: Associated otological findings

<table>
<thead>
<tr>
<th>Central Perforation</th>
<th>- 27</th>
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<tr>
<td>Retracted/ Atelectatic TM</td>
<td>- 10</td>
</tr>
<tr>
<td>Cholesteatoma</td>
<td>- 1</td>
</tr>
<tr>
<td>Intact TM</td>
<td>- 20</td>
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</tbody>
</table>
The diagnosis is mainly by otoscopy or otomicroscopy when characteristic chalk white patches are seen on the tympanic membrane or the middle ear through a perforation. The mobility of the tympanic membrane may be reduced or may even disappear completely if the plaque is adherent to the sulcus tympanicus or to the handle of malleus or makes contact with the promontory.

Tuning fork tests, full audiometric investigations and tympanometry are important aids for hearing assessment in these patients. Tympanometry is of value only if the TM is intact.

There is no direct relationship between extent of tympanosclerosis and severity of hearing loss according to Bhaya et al (1993). He also concluded that the only audiometric finding of any consequence was a mixed hearing loss in the presence of middle ear tympanosclerosis.

TREATMENT
The primary objective of treatment is relief of deafness. Since it is seldom possible to assess the extent and severity of the disease process preoperatively, the surgeon must be experienced and skilled enough to cope with any situation which may arise at operation.

<table>
<thead>
<tr>
<th>Table 3: Involvement of the tympanic membrane</th>
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<table>
<thead>
<tr>
<th>Tympanic membrane</th>
<th>48</th>
</tr>
</thead>
</table>

Ossicles

<table>
<thead>
<tr>
<th>Malleus</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incus</td>
<td>5</td>
</tr>
<tr>
<td>Stapes</td>
<td>2</td>
</tr>
</tbody>
</table>

Middle ear mucosa 7

Mastoid air cells 1

Tympanosclerosis is an insidious disease and its incidence is higher than that reported in the literature. In fact at times, the diagnosis may only be made at operation (Sheehy and House, 1962). Sorensen and True (1972) state that tympanosclerosis has been histologically proven in some cases in which it had not been suspected on gross examination. The same observation has been made by Tos and Bak-Pedersen (1974). In our series the commonest presenting complaint was deafness of varying degrees (Seen in 60% of the cases). Amongst these almost 83.3% were of the conductive type. The impairment of hearing was mainly due to the involvement of the middle ear sound conducting system by the inflammatory process that is
the precursor for tympanosclerosis (Sorensen & True, 1976). Significantly 14% of the patients didn’t have any otological complaints. These patients had come to the OPD for other non otological complaints and in them tympanosclerosis was just an incidental finding.

In the current series 5.6% of the cases had sensorineural hearing loss. Gibb (1971) had suggested that sensorineural hearing loss may occur due to the involvement of the inner ear by tympanosclerosis. Uripin et al (2000) in their study of 412 cases of chronic otitis media, of which 26 cases had tympanosclerosis, found all of them to have conductive or mixed hypoacusis. In our series we had encountered 2 cases with purely sensorineural hearing loss and 4 with mixed type. At least 2 of these 6 patients were of the older age group and their sensorineural hearing loss could be attributed to presbyacusis. Two of the cases with mixed hearing loss were operated upon and in them there was no evidence of tympanosclerosis in or around the round or oval window niches. It can thus be surmised that in these two cases tympanosclerosis did not take any part in causing the sensorineural deafness. The explanation in the remaining two cases remains obscure. It may be due to microscopic involvement of the cochlea by hyaline degeneration of the membranous labyrinth.

In our study, we did not correlate the degree of hearing loss with the extent of tympanosclerosis as the degree of hearing loss is also related to other associated pathologies like central perforation of tympanic membrane, cholesteatoma, otosclerosis etc and such a correlation is liable to be fallacious. According to the otological findings in our series, the posterosuperior quadrant of the pars tensa of the tympanic membrane was most commonly involved area by tympanosclerotic plaques, closely followed by the anteroinferior and posteroinferior quadrants. The pars flaccida was not involved in any of the cases of our series. Different studies have stated different findings with respect to site of involvement by tympanosclerotic plaque, like Bhaya et al (1993) reported the anterior and posteroinferior quadrants to be the most frequently involved, according to Yabe et al (1995), it were the anterosuperior and posterosuperior quadrants of the pars tensa and according to Jaisinghani (2000), it was the anteroinferior quadrant (According to him however the maximum area of involvement was in the posteroinferior quadrant). However the pars flaccida was always spared according to all studies. The lack of unanimity of opinion regarding the commonest quadrant of involvement probably suggests that tympanosclerosis actually does not have any predilection for any particular site on tympanic membrane. According to Sade (1966) and Tos and Bak Pedersen (1974) the tympanotic plaques occur in areas where the mucous gland population is lowest and in areas where cilia are scanty i.e. factors which favour stagnation of inflammatory exudates. The attic region of the middle ear fits this description. In spite of that, though the head of malleus body of incus, both housed in the attic, are often involved by tympanosclerosis, the pars flaccida is never involved. We have not been able to provide any satisfactory explanation for this.

By far the commonest otological finding in ears with tympanosclerosis was a central perforation (54%). The next most common finding was an intact tympanic membrane. Gibb (1976) reported in his series that roughly two thirds of the cases with tympanosclerosis had a tympanic membrane, which is roughly similar to our data of 54%.

We had encountered a solitary case of cholesteatoma with tympanosclerosis. The association of cholesteatoma with tympanosclerosis is mired with controversy. Plester (1972) considered it to be purely coincidental. Gibb (1979) also had a similar opinion. However others like Harris and Weiss (1962), Bonnau (1971) and Jerlito (1972) had reported a definite association between the two. Considering the fact that we had encountered only one case of cholesteatoma out of 50 cases of tympanosclerosis, we are endued to support the former view.

Impedance audiometry was done in 11 cases with non perforated tympanic membrane, where tuning fork tests indicated a conductive deafness and Sieglisation indicated impaired mobility. 7 out of 11 cases showed a type B curve. Gibb (1977) reported that commonest type of tympanometric curve in cases with tympanosclerosis was the As type indicating stiffness of the sound conducting mechanism. In our series we had encountered adhesive changes in the middle ear of 20% of the cases, which accounts for the type B curve being the commonest type.

Surgical exploration revealed the extent of involvement of the middle ear by tympanosclerosis. A total of 31 patients in our series underwent surgery of some type or the other. Tympanoplasties of various types were done in 29 cases with either a central perforation or atelectasis. Atticotomy was done in 4 cases. Three of them had plaques extending to the attic and one of them had attic granulation. According to Gibb (1970), the tympanosclerotic plaques are common in the epitympanum and in relation to the malleus. Kamal (1997) reported that in the middle ear cavity tympanosclerotic plaques commonly affect the eardrum, promontory, the oval window niche, ossicular chain and the facial nerve. After surgery the grafts were well taken up in the majority of cases (80%). Upto 80% patients reported some subjective
improvement of hearing after surgery. Thus the overall results of surgery in ears with tympanosclerosis is good, both in terms of graft uptake and in terms of hearing improvement. This included those cases where some amount of plaque was left behind as the surgeon had assessed that the plaques by virtue of their location were not hampering the sound conducting apparatus of the middle ear.

Gibb (1976) had concluded that the success rates of myringooplasty in cases with tympanosclerosis was roughly similar to that achieved in tympanic membrane repair where no evidence of tympanosclerosis exists. He opined that the removal of plaques prior to grafting was not always advisable since grafts applied over the plaques survived surprisingly well. Moreover plaque removal risks tearing of the tympanic membrane and enlargement of the perforation (Gibb, 1979). This viewpoint was also supported by Wielinga et al (1995). Santos et al (1979) however had reported poor functional results in such cases.

Radiological appearance of the mastoids showed 73% of the patients to have a sclerotic mastoid. This suggests that most of the ears with tympanosclerosis had a chronic inflammatory or infective disease of the middle ear cleft.

Histopathology of the plaques after haematoxylin and eosin staining revealed dense bundles of collagen with hyaline degeneration and scattered areas of calcification. In certain areas they were seen to overlie granulation tissue where their avascular appearance contrasted sharply with the vascular appearance of the granulation tissue.

CONCLUSION

After collecting the data and its extensive study and analysis, the following conclusions can be drawn:

1. Tympanosclerosis is a clinicopathological entity of immense importance and is a sequela of long standing inflammation of the middle ear cleft, most commonly seen in CSOM with central perforation.
2. It can affect almost any part of the middle ear cavity and is often symptomatic but may often be an incidental finding in otologically asymptomatic patients.
3. It doesn’t have any gender predilection and occurs equally in both sexes.
4. Its incidence is commonest in the 3rd and 4th decades of life though it is not infrequent in other age groups.
5. The commonest site of tympanosclerosis is the tympanic membrane where it can affect any quadrant of the pars tensa but always spares the pars flaccida.
6. Tympanosclerosis is associated with a predominantly conductive type of hearing loss, which is the chief complaint. However in some cases sensorineural deafness may be encountered.
7. Hearing loss can be alleviated to a great extent in a majority of patients by an appropriate surgery. The results of surgery are good and are not affected by the presence of the plaque unless it is present at a site which causes some fixity of the sound conducting mechanism.
8. Histopathological examination of the plaques by light microscopy after haematoxylin and eosin staining reveals dense bundles of collagen with hyaline degeneration and scattered areas of calcification.

REFERENCE


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