The Journal of Laryngology and Otology
December 1999, Vol. **113,** pp. 1076–1080

Tympanosclerosis: review of literature and incidence among patients with middle-ear infection

Saad Asiri, M.D.*, Alaa Hasham, M.D.*, Fatma Al Anazy, M.D.†, Siraj Zakzouk, M.D., F.R.C.S.*, Adel Banjar, M.D., F.R.C.S.‡

Abstract

The aim of the study was to review the literature of tympanosclerosis especially its pathogenesis, to study the general incidence of tympanosclerosis among patients with chronic suppurative otitis media (CSOM), its association with cholesteatoma and also the type of hearing loss as well as its relation to the degree and site of tympanosclerosis.

Seven hundred and seventy-five patients with CSOM were studied retrospectively. A full history was taken and thorough ENT examinations were carried out. Pure tone audiograms (PTA) of all patients were done and analysed. The operative finding of tympanosclerosis as well as middle-ear status were inspected.

The incidence of tympanosclerosis was found to be 11.6 per cent (90 patients out of 775 CSOM cases). Most tympanosclerosis cases had dry ear, (85.6 per cent). Of the 57.8 per cent who had myringosclerosis, their PTA showed an AB gap 20-40 dB. When sclerosis affect both the tympanic membrane and middle ear, 61 per cent of patients had an AB gap >40 dB. The association of cholesteatoma and tympanosclerosis may be regarded as uncommon, 2.2 per cent.

The exact aetiology and pathogenesis of tympanosclerosis is as yet not well known. Our study concentrated on the clinical picture of tympanosclerosis among patients with CSOM. The majority of hearing loss associated with tympanosclerosis was of the conductive type.

Key words: Otitis media, suppurative; Sclerosis; Hearing loss, conductive

Introduction

Tympanosclerosis a term introduced into the English literature by Zollner (1956) is a common sequela of middle ear infection. The aim of this study was to review the literature regarding this sequela and to study the general incidence of tympanosclerosis among 775 patients with CSOM, admitted for surgery during the period 1992 to 1995 in three main hospitals. It was also to report on the incidence of dry ears, association with cholesteatoma, myringosclerosis, intratympanic sclerosis and the type of hearing loss found in these cases as well as its relation to the degree and site of tympanosclerosis.

Definition

Tympanosclerosis is an abnormal condition of the middle-ear cleft in which there are calcareous deposits in the tympanic membrane, tympanic cavity, ossicular chain and occasionally in the mastoid (Wielinga and Kerr, 1993). It is considered as a long-term sequela of chronic middle-ear infection, characterized by the laying down of large amounts of collagen fibres in the submucosal connective tissue of those sites. Thickening and fusion of the collagen may follow with the formation of a homogenous hyaline mass into which deposition of scattered intra- and extra-cellular calcium and phosphage crystals may take place (Slack *et al.*, 1984). Tympanosclerosis affects the tympanic membrane either alone, in which case it is given the name myringosclerosis (Doyle, 1975) or in association with other parts of the middle-ear cleft.

Tympanosclerosis was first described in 1734 by Cassebohm as 'chalky patches' in the tympanic membrane, cited by Gibb (1976). The term sclerosis was first applied in 1873 by Von Trolsch, who gave a detailed account of 'Paukensklerose' (tympanic cavity sclerosis) relating the condition to chronic ear infections (Wielinga and Kerr, 1993). He described how tympanosclerosis could render the mucosa stiff and inflexible with fixation of the ossicular chain. The histopathological characteristics were recognized by Toynbee in 1860 and Politzer in 1883 which helped in the distinction between the features of tympanosclerosis and those of otosclerosis. Differentiating these two conditions was difficult clinically until the middle of this century when

From the Departments of ENT, Security Forces Hospital*, King Abdul Aziz University Hospital†, Riyadh, and Ohud Hospital‡, Madina, Saudi Arabia.
Accepted for publication: 24 August 1999.

Zollner (1956), aided by the operating microscope, was able to give a comprehensive picture of this condition. He introduced the term 'tympanosclerosis' into the English literature as a translation for 'Paukenskerlose'. Thereafter, the characteristics of this disease entity were extensively studied, but still the exact aetiology and pathogenesis are obscure and in addition, its surgical management is also debatable.

Incidence

The incidence of tympanosclerosis varies widely between seven to 33 per cent in different series among patients with chronic middle-ear infections (Emmett and Shea, 1978). Among 1000 operations performed for all types of middle-ear infections, Austin (1988) reported the incidence of tympanosclerosis to be 32 per cent, only 6.4 per cent of which were clinically significant. Myringosclerosis alone is much commoner than tympanosclerosis ranging from 24 per cent to 51 per cent in chronic otitis media. Bilateral cases occur in around 40–60 per cent of cases (Gibb and Pang, 1994).

Myringosclerosis can occur at any age and has been reported in a two-year-old child (Miller, 1984). The incidence is highest in children and is related to the increased incidence of secretory otitis media in this age and the associated use of ventilation tubes. The incidence of myringosclerosis in children was reported to affect from 28 to 61 per cent of all cases who had insertion of ventilation tubes (Wielinga and Kerr, 1993). Some authors claim an incidence of five to seven per cent of all children around five to six years of age (Tos *et al.*, 1983). In contrast, the incidence of intratympanic tympanosclerosis is higher in adults. Kinney (1978), quoted that 90 per cent of patients were over 30 years of age and had had a history of ear disease for over 20 years.

Aetiology and pathogenesis

The exact aetiology and pathogenesis of tympanosclerosis is as yet not well known. It is usually considered as a healed inflammation or a particular form of scar tissue following recurrent otitis media (Friedman, 1971). Several factors are postulated in the pathogenesis of tympanosclerosis.

(1) Post-inflammatory. Tympanosclerosis may occur as a result of long-standing middle ear inflammation either purulent or serous in nature. Many authors suggested three stages, an initial (reversible) stage in which the inflammatory exudate will lead to breakdown of collagen fibres with either organization of stagnant exudate in the recesses and folds of the middle ear (Ferlito, 1979) or formation of granulation tissue due to recurrent or long-standing inflammation (Igarashi *et al.*, 1970). The reparative (irreversible) stage then ensues with invasion and increased activity of fibroblasts leading to increased collagen production and formation of hyaline masses. The final stage is also irreversible in which calcification or even ossification takes place.

(2) Otitis media with effusion (OME) Post-inflammatory factors due to the associated oedema and inflammation in the submucosa in cases of long-standing effusion lead to degeneration of the fibrous layer (Miller, 1984). In this damage to the fibrous layer in the lamina propria occur due to some factors which all lead to the breakdown of collagen fibres. The second cause is the direct action of hydrolytic enzymes in serous fluid on the lamina propria (Wielinga *et al.*, 1988). A third factor is the traumatic effect either due to eardrum retraction exerting long-standing pressure on the tympanic membrane (Lesser *et al.*, 1988) or due to myringotomy and insertion of ventilation tubes. There may be intraepithelial haemorrhage, after tube insertion, that heals by fibrosis (Parker and Maco, 1990).

Pathological changes are situated in the lamina propria of the drum and middle-ear mucosa. Microscopically, tympanosclerosis appears as a dense network of interlacing of collagen fibres with irregular fibrillar structure found in the lamina propria. Degeneration can occur with loss of fibrils and calcium deposition resulting in an amorphous hyaline appearance. Electron microscopy show that calcification starts in the mitochondria and lyosomes of fibroblasts; later on they become extracellular and fuse together to form calcospheres in the surrounding connective tissue. These calcospheres enlarge and form amorphous hyaline masses which appear clinically as chalky patches.

(3) Autoimmune factors were also postulated to have a role in the pathogenesis of tympanosclerosis. Schiff *et al.* (1980) and Schiff and Yoo (1985), concluded that tympanosclerosis is an immune – complex reaction resulting from otitis media in an ear already sensitized by a previous inflammatory episode. They claimed that infection will lead to autolytic enzymes production and oedema which in turn will stretch and damage the ground substance in the lamina propria of the tympanic membrane. The breakdown products act as antigenic stimulants this presensitizing the tissues. On subsequent infection, a pronounced immune response involving complement will take place. However, Miller and Nilsen (1986), failed to find responsible antibodies in electron microscopic studies.

The association between cholesteatoma and tympanosclerosis has long been a matter of great debate in the literature. The conflicting views range from enhancement of one disease by the other (Ferlito, 1979) to a negative association between the two conditions (Schiff *et al.*, 1989). At the other end of the scale, many authors believe that the coexistence of both disorders is rare and more or less accidental (Plester, 1972).

Site and clinical type of tympanosclerosis

Clinically, tympanosclerosis can either be myringosclerosis or intratympanic. Myringosclerosis occurs when the disease affects only the tympanic membrane and appears as white 'chalky patches'. In most cases, it is asymptomatic or have little effect on hearing (Tos *et al.*, 1983), but if the plaques involve large areas of tympanic membrane or if they are adherent to the bony annulus, handle of malleus or

promontory, the mobility of the drum will be greatly reduced resulting in marked hearing loss (Wielinga and Kerr, 1993). It might be either open (80 per cent) with tympanic membrane perforation or closed (20 per cent) with intact drumhead. The intratympanic type is frequently associated with marked conductive or mixed hearing loss. The degree of hearing loss depends on the extent of tympanosclerotic involvement of the ossicular chain (Kamal, 1997).

Diagnosis

Diagnosis is usually easy on otoscopic examination except in the closed intratympanic type when the tympanic membrane is quite normal. In the open type, chalky patches of variable size can be seen on the tympanic membrane, promontory or oval window. The middle ear mucosa is healthy and dry in 80 to 92 per cent of cases (Austin, 1988). In closed tympanosclerosis, otoscopic examination is usually of limited value unless the tympanic membrane is grossly affected. Patients usually present with conductive hearing loss and sometimes a false carhart notch may lead to confusion with otosclerosis. Differentiation is usually by a negative family history of otosclerosis, positive history of recurrent otitis media, presence of sclerotic patches on the drumhead and acellular mastoid on X-ray (Ferlito, 1979). CT of the middle ear can identify calcification or ossification, in case of tympanosclerosis, as uni or multifocal deposits (Swartz *et al.*, 1985).

Surgery of tympanosclerosis

Treatment of tympanosclerosis is aimed at improving the patient's hearing either by hearing aid or surgery. Controversy still remains concerning surgical management. While some surgeons consider tympanosclerosis as 'the least amenable' middle ear disease for surgical treatment and which can even constitute a probable contraindication to surgery (Smyth, 1972), others claim that in experienced hands, surgical treatment of tympanosclerosis is a safe procedure and that most patients have hearing results comparable to surgery for other chronic ear disease (Giddings and House, 1992). In myringosclerosis, removal of sclerotic plaques is done if they are likely to affect the mobility of the drum or impair healing of the graft. In fixation of malleus – incus complex in the attic, exposure of the epitympanum is performed with either mobilization of the ossicles, which carry a great risk of refixation within a short period of time, or removal of malleus head and incus with subsequent reconstruction of the sound transforming mechanisms. In case of stapedal fixation, many surgeons prefer to perform surgery in two stages, the first stage is to graft the tympanic membrane followed after a period of at least six to 18 months by stapes surgery. Mobilization of the stapes always carries the risk of refixation, hydraulic trauma to the cochlea and perilymph fistula with resulting irreversible sensorineural hearing loss in up to 53 per cent of cases (Smyth, 1972).

Because of these effects, many surgeons believe that stapedectomy followed by closure of the oval window by vein, fat or tragal perichondrium is the treatment of choice for oval window tympanosclerosis. If there is a coexisting malleus – incus fixation in the attic, removal of the incus and malleus head can be done with reconstruction of the sound transformation mechanism by incus, cartilage strut, TORP or PORP (Wielinga and Kerr, 1993). The overall results in the majority of studies of total stapedectomy procedures in tympanosclerosis are slightly better than the results obtained by mobilization. On the other hand, many other authors consider that, all procedures for tympanosclerosis showed good results at short-term follow up, but later on these results showed deterioration with time due either to refixation of the remaining ossicles or due to the normal progress of the chronic otitis process.

Considering all the above mentioned, the indication of surgery must be very guarded; even with initial improvement a hearing aid will often still be required in the long term follow up of many patients.

Materials and methods

Seven hundred and seventy-five cases of chronic suppurative otitis media were studied retrospectively in the period from February 1992 to December 1995 in three main hospitals, two in Riyadh and one in Madina to detect the number of cases with tympanosclerosis. Ninety patients were found to have a varied degree of tympanosclerosis and were selected for our study.

In all the 90 patients, a full history was recorded regarding age, sex, duration of illness and symptoms such as hearing loss earache, otorrhoea, tinnitus, vertigo and any previous trauma surgery of middle ear. A thorough ENT clinical examination was carried out of the ear, nose and throat, including otoscopy, tuning fork test, pure tone audiometry using interacaustic AC 30 audiometer where both air condition (AC) and bone conduction (BC) were tested at the frequency of 250, 500, 1000, 2000, 4000 and 8000 Hz. Both AC and BC were taken as the average of 500, 1000 and 2000 Hz values. All 90 patients with tympanosclerosis underwent surgery to treat their chronic ear problem. During surgery, the ossicular mobility, middle ear mucosa and degree of tympanosclerosis in middle ear and ossicles were inspected. Nine patients had a tympanosmastoidectomy because of cholesteatoma or severe granulation and polypi while the remaining 81 patients had tympanoplasty ± ossiculoplasty.

Results

The records of 775 patients with history of CSOM were reviewed, 90 patients who had tympanosclerosis were found with an incidence of 11.6 per cent. These were 49 females and 41 males. The age ranged from four years to 52 years with average age of 31 years (Table I).

https://doi.org/10.1017/S0022215100157937 Published online by Cambridge University Press

TABLE I
AGE AND INCIDENCE OF TYMPANOSCLEROSIS

Age group	No.	%
<5 yrs	1	1.1
5–15 yrs	9	10
15–30 yrs	36	40
>30	44	48.9
Total	90	100%

TABLE II
SYMPTOMS PREVALENT AMONG 90 PATIENTS WITH TYMPANOSCLEROSIS

Symptoms	No.	%
Otorrhoea	13	14.4
Hearing impairment	86	95.6
Tinnitus	22	24.4
Otalgia	6	6.7
Vertigo	4	4.4

TABLE III
STATUS OF TYMPANIC MEMBRANE, OSSICLES AND MIDDLE EAR MUCOSA

Findings	No.	%
T.M.		
— Perforated	76	85
— Parstensa	69	77
— Pars flaccida	7	13
— Intact	14	16
Middle ear mucosa		
— Dry	77	86
— Polyps	4	4
— Granulations	7	8
— Cholesteatoma	2	2
Ossicles		
— Mobile	55	61
— Fixed	27	30
— Eroded	8	9

The main clinical presentation of the patients with tympanosclerosis is shown in Table II. The status of tympanic membrane, ossicles and middle ear mucosa is shown in Table III while the relation between the extent of the disease and hearing loss is shown in Table IV.

The pathological finding in 12 cases with mixed hearing loss is shown in Table V.

Discussion

Tympanosclerosis is a long term sequelae of chronic suppurative otitis media. The incidence varies widely in the literature between seven and 33 per cent of all patients with chronic middle ear infection. Our results showed an incidence of (11.6 per cent) which falls within this range. There was a slight predominance towards females (54.4 per cent).

In the literature, the incidence of intratympanic tympanosclerosis is much higher in adults. Few cases have been reported in the first and second decades of life. The youngest patient reported in the literature was eight years old (Giddings and House, 1992). We found in our study that only 44 patients (49 per cent) were over 30 years of age, while the number of cases in the younger age group (from five to 30 years) was 45 patients or 50 per cent, five cases had intratympanic tympanosclerosis while the youngest patient in our series was five years old. Due to these factors namely, the young age incidence and the occurrence of intratympanic pathology in children, we suggest that this possibly denotes a more aggressive nature and rapid course of the disease which might be attributable to the very dry weather prevalent in this part of the world especially the (central area of Saudi Arabia) where humidity may reach as low as 10 per cent.

Clinically, a characteristic feature of tympanosclerosis is the absence of suppurations in the ears affected, usually for a long time. The incidence of dry ears with healthy mucosa quoted by different authors ranged from 80 to 90 per cent. In our series, Table VI, only 13 patients had otorrhoea, while the incidence of dry ears was 86 per cent which corresponds well to the literature. Our study showed that 76 of cases (84.4 per cent) had tympanic membrane perforation, 69 of which were in the pars tensa. These results compare favourably to those as mentioned in the literature where the open type is four to five times more common than the closed variety (Austin, 1988; Tos *et al.*, 1990) and that perforation are invariably sited in the pars tensa (Plester, 1972; Kamal, 1997). In our series we found only two cases (two per cent) of cholesteatoma, and based on the great clinical differences between the two diseases, it is our contention that the association of cholesteatoma and tympanosclerosis may be regarded as uncommon and coincidental.

TABLE V
TYPE OF PATHOLOGY IN THE EAR IN 12 CASES WITH MIXING HEARING LOSS

Myringo-sclerosis	M.E. sclerosis	Granulation and/or polypi	Cholesteatoma
3	6	2	1

TABLE IV
RELATION BETWEEN EXTENT OF TYMPANOSCLEROSIS AND DEGREE OF HEARING LOSS

Site of tympanosclerosis	No. of patients	No. of patients with AB gap <20 AB	AB gap 20–40 dB	AB gap >40 dB
TM	45 (50%)	9	26	10
Middle ear	27 (30%)	3	13	11
Both	18 (20%)	–	7	11
Total	90 (100%)	12 (13%)	46 (51%)	32 (36%)

https://doi.org/10.1017/S0022215100157937 Published online by Cambridge University Press

In our study, we found that 45 cases (50 per cent) of cases had myringosclerosis, while in 18 cases (20 per cent of patients), the disease has affected both tympanic membrane and middle ear. These results conform with those found in the literature.

The degree and extent of tympanosclerosis plaques will eventually determine the severity of hearing loss. Only 10 out of 45 of our patients with myringosclerosis (22 per cent) exhibited an air-bone gap more than 40 dB. In these patients, tympanosclerotic plaques either involved large areas of the drum or were found to be adherent to the bony anulus and the promontory causing severe impairment of the tympanic membrane mobility.

Among 27 patients with intratympanic involvement, 41 per cent of cases had A-B gap more than 40 dB, while 11 of 18 cases (61 per cent) where the disease affected both tympanic membrane and middle ear showed an A–B gap more than 40 dB. These results will explain the direct relation between the degree of hearing loss and the increased extent of the disease.

References

Austin, D. F. (1988) Reconstructive techniques of tympanosclerosis. *Annals of Otorhinolaryngology* **97:** 670–674.

Doyle, D. E. (1975) Myringosclerosis: An offering of a new term. *Eye Ear Nose and Throat Monographs* **54:** 399–400.

Emmett, J. R., Shea, J. J. (1978) Surgical treatment of tympanosclerosis. *Laryngoscope* **88:** 1642–1648.

Ferlito, A. (1979) Histopathogensis of tympanosclerosis. *Journal of Laryngology and Otology* **93:** 25–37.

Friedmann, I. (1971) Tympanosclerosis. *Annals of Otology, Rhinology and Laryngology* **80:** 4110–4113.

Gibb, A. G. (1976) Tympanosclerosis. *Proceedings of Royal Society of Medicine* **69:** 155–162.

Gibb, A. G., Pang, Y. T. (1994) Current considerations in the etiology and diagnosis of tympanosclerosis. *Archives in Otorhinolaryngology* **251:** 439–451.

Giddings, N. A., House, J. W. (1992) Tympanosclerosis of the stapes: Hearing results for various surgical treatments. *Otolaryngology - Head and Neck Surgery* **107:** 644–650.

Igarashi, M., Konishis, S., Alford, B. R., Guilford, F. R. (1970) The pathology of tympanosclerosis. *Laryngoscope* **80:** 233–243.

Kamal, S. A. (1997) Surgery of tympanosclerosis. *Journal of Laryngology and Otology* **111:** 917–923.

Kinney, S. E. (1978) Postinflammatory ossicular fixation in tympanoplasty. *Laryngoscope* **88:** 821–838.

Lesser, T. H. J., Williams, K. R., Skinner, D. W. (1988) Tympanosclerosis, grommets and shear stresses. *Clinical Otolaryngology* **13:** 375–380.

Miller, P. (1984) Tympanosclerosis of the eardrum in secretory otitis media. *Acta Otolaryngologica (Suppl.) (Stockholm)* **414:** 171–177.

Miller, P., Nilsen, R. (1986) Tympanosclerosis in children. An electron microscopic and immunohistochemical study. Proceedings in the International Conference on Acute Secretory Otitis Media, Jerusalem. Kugler Publications, Amsterdam. As cited in Wielinga, E. W. J., Kerr, A. G. (1993). Tympanosclerosis review. *Clinical Otolaryngology* **18:** 341–349.

Parker, A. J., Maco, A. R. (1990) Intratympanic membrane bleeding after grommet insertion and tympanosclerosis. *Clinical Otolaryngology* **15:** 203–207.

Plester, D. (1972) Tympanosclerosis. *Australian Journal of Otolaryngology* **3:** 325–326.

Schiff, M. Poliquin, J., Catanzaro, A., Ryan, A. (1980) Tympanosclerosis: a theory of pathogenesis. *Annals of Otology, Rhinology and Laryngology* **89: (Suppl. 70)** 1–16.

Schiff, M., Yoo, I. J. (1985) Immunologic aspects of otologic disease. An overview. *Laryngoscope* **95:** 256–269.

Slack, R. W., Maw, A. R., Capper, J. W. R., Kelly, S. (1984) Prospective study of tympanosclerosis developing after grommet insertion. *Journal of Laryngology and Otology* **98:** 771–774.

Smyth, G. D. (1972) Tympanosclerosis. *Journal of Laryngology and Otology* **86:** 9–14.

Swartz, J. D., Wolfson, R. J., Marlowe, F. I., Popky, G. L. (1985) Post inflammatory ossicular fixation: CT analysis with surgical correlation. *Radiology* **154:** 697–700.

Tos, M., Bonding, P., Poulsen, G. (1983) Tympanosclerosis grommets. A prospective, comparative study. *Journal of Laryngology and Otology* **97:** 489–496.

Tos, M., Lau, T., Arndal, H., Plate, S. (1990) Tympanosclerosis of the middle ear: late results of surgical treatment. *Journal of Laryngology and Otology* **104:** 685–689.

Wielinga, E. W. J., Kerr, A. G. (1993) Tympanosclerosis, Review. *Clinical Otolaryngology* **18:** 341–349.

Wielinga, E. W. J., Kuijpers, W., Tonnaer, E. L. G. M., Jap, P. H. K. (1988) An experimental model for tympanosclerosis: a preliminary report. *Acta – Otolaryngologica (Stockholm)* **105:** 537–543.

Zollner, F. (1956) Tympanosclerosis. *Journal of Laryngology and Otology* **70:** 77–85.

Address for correspondence:
Dr Fatma H. Al-Anazy,
ENT Department,
King Abdul Aziz University Hospital,
P.O. Box 245,
Riyadh 11411,
Saudi Arabia.

Fax: (966) 1-477 5748