

NEW TRENDS IN THE MANAGEMENT OF CHOLESTEATOMA

Essay

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Master Degree

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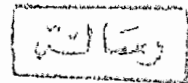
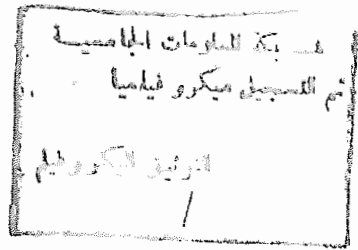
Otorhinolaryngology

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

«أَقْرَأْ بِاسْمِ رَبِّكَ الَّذِي خَلَقَ،

خَلَقَ الْإِنْسَانَ مِنْ عَلَقٍ، اقْرَأْ وَرَبُّكَ الْأَكْرَمُ،

الَّذِي عَلَّمَ بِالْقَلَمِ، عَلَّمَ الْإِنْسَانَ مَا لَمْ يَعْلَمْ»

صدق الله العظيم



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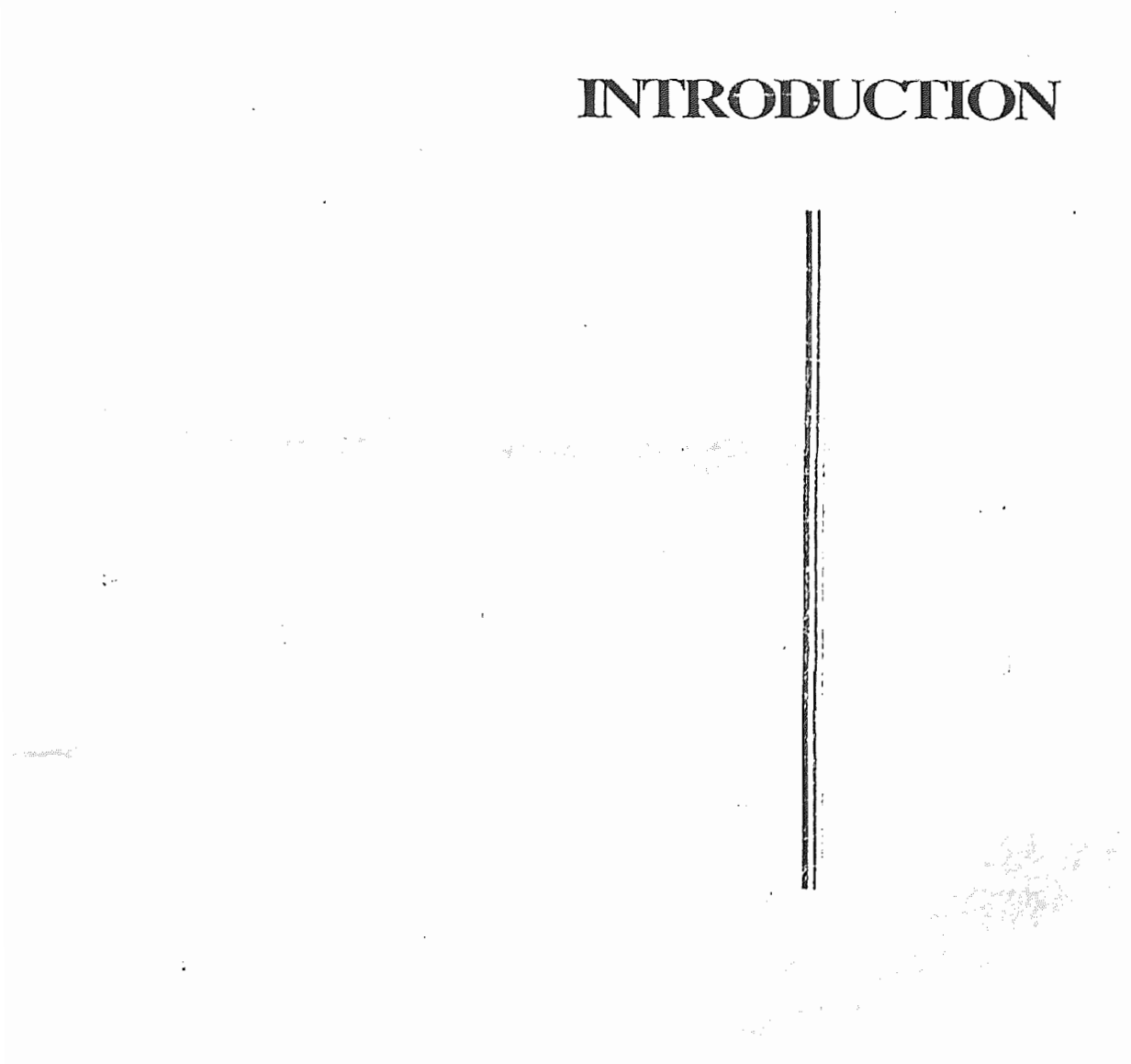
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INTRODUCTION





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Aural cholesteatoma or more Precisely (Keratoma) (Schuknecht, 1974) is defined as, the presence of keratinizing stratified squamous epithelium with accumulation of desquamated epithelium or keratin within the middle ear or other pneumatized portion of the temporal bone.

Cholesteatoma is a misnomer, it is neither a tumour nor does it necessarily contain cholestrin. The simplest definition of cholesteatoma is (skin in a wrong place) (Gray, 1964).

As early as the begining of the century, otologists made a distinction from the clinical point of view and for purposes of prognosis and treatment between attico-antral suppuration (cholesteatoma) and tubotympanic suppuration. (Wayoff & Lacher, 1987).

Today the treatment of cholesteatoma must include an element of prevention which implies a good understanding of the pathogenesis of cholesteatoma and the pathophysiology of the middle ear. (Mawson, 1974).

Opinoins vary about the best method of surgical treatment of cholesteatoma. The choice is between the wide-access or (open) approaches or canal wall down techniques on one hand and the (closed) or canal wall up techniques that retain the bony canal wall on the other hand. (Stell & Maran, 1979).

A few surgeon, however who were already concerned to avoid needless mutilation suggested the use of the modified or partial radical operation. (Heath & Bondy, 1908).

One of these surgeon was (Sourdille, 1906) who asked four essential questions that are still important today about the surgical treatment:

- 1- What is the necessary anatomical modification?
- 2- How in view of the lesion can it heal?
- 3- When the lesion has healed can there be a further improvement?
- 4- Is cholesteatoma to be considered as a complication that necessarily involves radical surgery?

Heath, as early as, (1906) mentioned that, the main aim was to extenterate the antromastoid cavity leaving intact, those elements in the tympanic cavity which were functional.

The state of the tympanic cavity was thus already the determining factor in deciding whether a conservative approach was to be used or not.

The primary goal of the surgical treatment of cholesteatoma is its complete eradication in order to provide the patient with a safe and dry ear. This is done by removing all the cholesteatoma, diseased bone and granulation tissue.

An important but secondary goal is to improve or preserve hearing acuity by reconstructing the tympanic membrane and ossicular mechanism and restoring an aeriated middle ear cleft. (Sheehy, 1982)

The ambiguous term radical mastoidectomy or modified radical mastoidectomy are of historical interest but do not convey a specific operative procedure and their use should be abandoned (Smyth, 1976)

The procedure in which the posterior canal wall is removed as a part of mastoidectomy should be classified as (canal wall down).

The alternative approaches are those in which the mastoid and middle ear surgery is performed while preserving the posterior canal intact, these approaches should be classified as (canal wall up). (Smyth, 1985).

Jansen, (1977) stated that the selection of a specific operative technique should be determined by:

- 1- The extent of the cholesteatoma invasion.
- 2- Clinical assessment of the Eustachian tube function.
- 3- Degree of the mastoid pneumatization.
- 4- Anatomical configuration of the patient's mastoid.

DEFINITION OF CHOLESTEATOMA

A cholesteatoma is a non - malignant accumulation of keratin medial to the outer layer of the tympanic membrane, enclosed at least originally, by a membranous stroma of connective tissue.

Mc Guckin, (1961) prefers the term (keratosis) and maintains that the majority of cases of so called (chronic otitis media) arise by cholesteatomatous or keratotic invasion from outside the middle ear, that is, in the skin of the deep part of the bony external meatus and epithelial layer of the tympanic membrane. The same entity may be described also as epidermosis by (Tumarkin, 1961) or cholesteatosis by (Young, 1980).

In the meanwhile, Shambough, (1959), defined it as epidermoid cholesteatoma consisting of a matrix or a germinal layer attached to the bony wall of the attic or antrum, from which masses of squamous epithelium are cast off, these are concentrically arranged, with here and there crystals of cholestrin. To the naked eye, it generally appears as a smooth, glistening pearly body. The layers of epithelium accumulate and as cholesteatoma expands, osteitis and bone absorption occur.(Friedman, 1977).

PATHOLOGY OF CHOLESTEATOMA

It is a tumour like growth, characterized by a bone-eroding skin lined cavity filled with concentric layers of desquamated epithelium. (Fernandez, 1959).

The normal desquamation of the outer cornified layers of skin proceeds to collect in concentric onion-like layers of whitish debris containing crystals of cholesterol. (Marquet,J, 1980).

This cholesteatoma debris offers a favourable culture medium for various pathogenic and putrifactive bacteria from the external meatus.

Microscopically, the cholesteatoma consists of keratinizing squamous epithelium (matrix), covering a stroma of fibrous granulation tissue of variable thickness (perimatrix), which lines the usually cystic structure the keratinized layers being innermost.(Nager,G.,T, 1975).

Desquamated epithelium, either in thick lamellae or smaller keratin scales, often with a generous admixture of pus, form the contents. The microscopical diagnosis rests entirely on the presence and identification of squamous epithelium and on lamellated keratinized matter.

The subepithelial fibrous granulation tissue may also contain some elements of the middle ear mucosa-gland like structures or columnar epithelium.

It was frequently observed that in the same, specimen, both columnar epithelium of the middle ear and the squamous lining are present, indicating that

squamous epithelium has migrated into the mucosa of the middle ear.

The ossicles, malleus and incus, in particular are covered by the lining of the epidermoid cholesteatoma and display osteoporotic changes or purulent osteitis. (Friedman, 1977)

Escher, (1959) noted that squamous epithelium can spread from the tympanic membrane directly to the incudo-stapedial joint, and from a central perforation, it may even, spread to the head of the stapes and into the area of the oval window niche-cholesterol deposits may be present, and the combination of the two lesions might lead to some confusion.

In 1951, the morphological analysis of the cholesteatoma was recognized by Mendel according to serial sectioning of many specimens at the ultrastructural level. It is shown that the thickness of the epithelial layer can vary considerably, not only between individual cholesteatomas but also within the same tissue. The basal cells of the epithelium have an extensive network of edges towards the stroma but in general do not penterate the basal membrane. There are microvilli towards the adjacent cells.

MECHANISM OF BONE EROSION IN CHOLESTEATOMA

The mechanism of bone erosion in cholesteatoma is unknown, two theories have been put forward to explain it.

The earlier anoxia-necrosis theory (Ruedi, 1968) and (Tumarkin, 1988), based on influence of direct pressure by the cholesteatomatous masses upon the bone.

Thomsen, et,al, (1977) stated that the influence of increased pressure is not interpretable as ischaemia, but as an irritation and foreign body reaction, leading to hyperaemia which promotes resorption.

Although the anoxia-necrosis theory has been abandoned by Sade', et,al,(1977), in favour of the inflammation enzymatic theory, the influence of pressure upon bone resorption was confirmed again in (1979) by Tos.

He relied upon the fact that the erosion was corresponding to the most compact parts of the cholesteatoma in many cases in which there were several spherical, firm cholesteatomas in the epitympanum, incarcerated between the superior wall of the epitympanum and the malleus-incus complex.

Congenital cholesteatoma, and especially post-traumatic cholesteatoma, which are not associated with infection, may cause appreciable bone resorption.

Histological studies have demonstrated inflammatory reaction close to the resorbed bone (Grippaudo, 1968), (Harris, 1962), (Thomsen, et,al,1974), (Sade' and Berco, 1974), all these authors always found inflammatory

tissue between the bone and the squamous epithelium. Histochemical studies have suggested that the important factor in bone destruction might be hydrolytic, lysosomal or protein-splitting enzymes in the subepithelial granulation tissue of a cholesteatoma. (Abramson, 1969).

Sade' et,al, (1977) felt squamous epithelium played no role in bone resorption. However, these enzymes by themselves may not be sufficient to cause bone resorption.

Bernsterin, et,al,(1977), have showed a factor which stimulated the release of minerals from bone and they suggested that the product of cholesteatoma was capable of producing calcium resorption, osteoclastic activation and inhibition of bone synthesis. It was assumed that possible factors included endotoxin (Howsmann, et,al,1972), prostaglandin (Klein & Raisz, 1970) and also osteoclastic activating factors. (Kaneko, et,al, 1980)

Tos, (1979) also showed that squamous epithelium have a marked-presumably enzymatic influence upon bone resorption due to the following observations:-

Bone resorption was on the whole for more cammon in cholesteatoma than in granulation osteitis.

Resorption of the malleus head and incus body was found only in cases with cholesteatoma, significantly most often in attic cholesteatoma.

The presence of squamous epithelium close to the resorption bone or at its site was an invariable finding and its absence, during operation does not mean that it