How I See the Development of Acquired Cholesteatomas based on 3 Fundamental Factors: Tympanic Membrane Retraction, Mesotympanum's Medial Wall Mucosal Cells and Its Recess, The Burial of these Mucosal Cells by the Tympanic Membrane Retraction

Opinion

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Opinion

There are several theories explaining the acquired cholesteatoma's etiopathogenesis, but there still are obscure points. Only the flaccid or tense tympanic membrane retraction - Bezold's theory (1888) - is accepted by most. Based on the knowledge acquired through my years of experience, I present the hypothesis that three elements are fundamental in cholesteatoma development.

The retraction of the entire pars tensa, or, more commonly, only in the posterosuperior and posteroinferior portions, destroys the long process of the incus and stapes, creating a space where the retracted membrane (never the meatal skin) penetrates to the attic and sometimes to the antrum, keratinizing, building up and forming a dry cholesteatoma, which destroys, from medial to lateral, the rest of the incus and the head of the malleus, resting on a smooth bed of regular contour - characteristics that are very clearly seen upon CT scans and surgery, evidently. This dry cholesteatoma can be diagnosed by seeing a dark crust adhered to the lateral wall of the attic, behind the neck of the malleus. It is a pathognomonic lesion and its removal should not be attempted without surgery, because it hurts substantially and bleeds, but then one knows that the cholesteatoma is there.

The posteroinferior tympanic membrane retraction, including the niche of the round window, buries the mucosa cells of this region, leaving it without ventilation from the Eustachian tube and without oxygen, thus causing it to swell and hypertrophy, producing granuloma and secretion, which increasing in volume breaks the thin membrane, exposing the granular hypertrophied mucosa, which subsequently infects, stimulates the keratinization of the rest of the retracted membrane and its recesses, increasing the dry cholesteatoma keratinization in the attic and antrum, - thus forming the SECONDARY CHOLESTEATOMA that everyone knows.

What would prove this thesis? Patients taken from a series of more than 2,400 cholesteatoma surgeries, more than 90% having been submitted to conservative canal-wall down technique, all performed by the author during 47 years, and followed by him (sometimes with the help of residents) for 3, 4 or 5 months, until complete cavity epithelization (a sine qua non condition, it either heals, or it heals), whom remained with their dry cavities for 10, 15, 20, 30 years, and then developed granulation in the posteroinferior portion of the mesotympanum, including the round window niche.

Those who came for a return visit only many months or even years after they felt e fetid odor or secretion in the ear presented, in addition to granulation, the entire mastoid cavity completely keratinized, thus meaning a cholesteatoma. Showing the importance of granulation in its formation. The infection is secondary to the granulation. They were treated in the outpatient ward, with warm water rinsing, removal and granulation cauterization. Without granulation, the dry cholesteatoma would remain dry, as it happens with the external acoustic meatus cholesteatoma, and with a small cholesteatoma that develops with a partial destruction of the lateral wall of the attic, resting on the side face of the body of the incus and the head of the malleus, without burying the mucosa, it remains dry forever.

The posteroinferior region of the mesotympanum, including the round-window niche, which I, for more than thirty years, have called a true Achilles heel of open mastoidectomies, presents granulations more frequently because the cells there can be in contact with all the mastoid cells through the cells located medially to the descending facial nerve and, with cells all the way to the tip of the petrous bone, through cells of the hypotympanum cells and infra-labyrinthine cells. They are cells that also run out of oxygen when the attic and addictus are blocked by the dry cholesteatoma.

In primary cholesteatomas, when the membrane retracts to the attic and into the antrum of a well-pneumatized temporal bone, it buries many mucosal cells, quickly forming granulation tissue; unlike the secondary cholesteatoma, which sits on the medial attic wall or in the antrum of a sclerotic temporal bone. The secondary cholesteatoma matrix is always regular, smooth and without granulation, that can and should be spared in open mastoidectomies; while the primary colesteatoma matrix always causes much granulation, and it needs to be removed.

To avoid short and long-term postoperative granulation, one must remove all possible cells from the temporal bone, such as: cells of the anterior tympanic sinus or Kerstner recess; cells of the medial attic wall; supra-labyrinthine cells; solid angle cells; Trautman's triangle cells; sinodural angle cells; middle fossa plate and posterior fossa plate cells; zygomatic cells; mastoid tip cells; cells between the sigmoid sinus and the facial nerve, retro and medial to the facial nerve; lateral cells to the second bend of the facial nerve - always found when one completely lowers the canal wall; facial recess cells; cells of the round window niche – being careful with the cochlear membrane; cells of the posterior tympanic sinus and hypotympanum (caution with the jugular bulb). For more than 40 years, I have sometimes placed a cartilage to strengthen the membrane in the posteroinferior region, in addition to removing the cells with diamond burrs and sealing them with bone powder. Even then, granulation tissue develops in some cases, because of connection between cells of that region with cells of the mastoid and cells of the tip of the petrous bone (it is a true marsh).

The cartilage removal from the inferior and anterior portions of the external meatus to strengthen the membrane gave me a Eureka cry 30 years ago, noticing that the cartilaginous canal became completely loose, being possible to move it up and back, leaving the entrance to the meatus thoroughly normal, without blind spots in the open mastoidectomy. It facilitates the fitting of hearing aids, provides for better esthetics and freedom for swimming or diving.

Controversies

In primary cholesteatomas there is a retraction that goes to the attic and antrum, forming the cholesteatoma, which should also be classified as a secondary cholesteatoma, since it is always preceded by a retraction of the attic wall of variable dimensions, it is always found in the ear contralateral to the cholesteatoma. It can also be seen in the ears of children, in which we put a tube to avoid cholesteatomas.

I rarely saw perforations, sometimes in the tuba, and only then, where the retracted membrane is very thin, treated infections may leave a perforation. The granulations of the secondary colesteatoma are not perforations, but rather wounds in the retracted membrane or matrix. If there are not many, or if it is only one, it is possible, with its removal and cauterization, to obtain a scarred and normal retracted membrane.

In all secondary cholesteatomas, I could identify the entire tympanic anulus and the pars tensa of the retracted membrane as the cholesteatoma matrix. There is no need for skin to migrate from the external meatus, since the membrane is already there, retracted, the migrated skin would have to pass over the normal tympanic anulus. I do not think this happens.

Total perforations prevent cholesteatomas. In atelectasis cases, if we were to remove the entire pars tension 40%, we could avoid the formation of a secondary cholesteatoma. A dry cholesteatoma could develop in the attic. There would be no mucosa burial in the tympanic cavity, nor the development of granulation tissue, indispensable for cholesteatoma formation. Some time, complete perforations are found with the retracted membrane remains, over the malleus and the incus, forming a dry cholesteatoma in the attic.

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