DIFFERENTIAL DIAGNOSIS OF OTOSCLEROSIS

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Abstract: The article is devoted to the study of the state of otosclerosis is a condition in which the bone tissue inside the ear grows, causing sound transmission problems and hearing loss, and may also cause tinnitus. The causes of otosclerosis are not entirely clear, but there is a genetic predisposition and the condition most commonly affects middle-aged white women. Treatment may include conservative treatments such as bisphosphonates and surgery such as stapedoplasty, which restores hearing in most patients.

Introduction

Mixed hearing loss occurs when someone experiences both conductive hearing loss (a problem in the outer or middle ear) and sensorineural hearing loss (a problem in the inner ear or auditory nerve) simultaneously. This combined type of hearing impairment can result from causes such as ear infections, genetic factors, aging, noise exposure, head injuries, or certain medications. An audiogram can identify mixed hearing loss by showing both a reduced bone conduction response and an air-bone gap. Treatment often requires a combination of medical intervention and hearing aids. Mixed hearing loss has elements of both conductive hearing loss and sensorineural hearing loss. This means there is damage to both the outer and inner ear. The outer ear cannot conduct sound properly to the inner ear, and the inner ear can’t process the sound to be sent to the brain. The sensorineural component (inner ear) is usually permanent, but the conductive hearing loss (outer ear) may not be. Many people with mixed hearing loss experience sounds as very soft in volume and difficult to understand.

Pathophysiology

Otosclerosis is one of the most common causes of progressive hearing loss, in particular in people of working age. The effectiveness of stapedial surgery largely determines the interest in studying the problems and prospects for the development of technologies, namely, the improvement of prostheses, surgical techniques, and assistance during interventions. It is worth noting that the main attention in the

scientific literature is paid to the description of the clinic, the diagnosis of otosclerosis, as well as the methods of stapedoplasty.

However, the question of the etiology and pathogenesis of this disease remains relevant and open for discussion, despite the many domestic and foreign works in this field. The emergence of new research methods, including molecular-genetic ones, contributed a to the transition of research to a new level and the development of several new theories..

Otosclerosis should be differentiated from a number of middle ear diseases accompanied by conductive hearing loss. In these cases, in addition to the absence of a family history, a number of support diagnostic signs characteristic of a specific pathology are revealed.

Exudative otitis is characterized by the presence of progressive hearing loss, conductive in the early stages, and mixed in later stages. The disease often begins after an acute respiratory viral infection. An important differential diagnostic sign is the tympanogram: type B, preservation of the ipsi- and contralateral acoustic reflex. On CT of the temporal bones, pathological contents are observed in the cells of the mastoid process and tympanic cavity without bone changes.

Consequences of purulent otitis (adhesive otitis media, tympanosclerosis), more often unilateral. Otoscopy reveals foci of myringosclerosis or areas of eardrum atrophy, retraction pockets. Tympanogram: type E or D, ipsi- and contralateral acoustic reflex in the ear being examined may be absent.

Ossicular chain rupture. History of trauma, unilateral hearing loss. Changes in the eardrum are possible during otoscopy. The audiogram reveals a uniform increase in air conduction thresholds across the entire tone scale, with a large air-bone interval; during tympanometry, type Ad is determined, absence of ipsi- and contralateral acoustic reflex.

Fixation of the malleus (incus). The process is usually unilateral. On the audiogram: conductive hearing loss, a possible Carhart-like tooth at a frequency of 0.5 kHz. Tympanogram with the presence of the on/off effect (there is a narrow peak at the moment the reflex begins to "turn on"). When performing CT of the temporal bones, dislocation of the malleus and visualization of the ossified external ligament are possible (which is the most common cause of fixation). Congenital malformations of the middle ear. Unilateral process, develops in early childhood. During examination, pathology of the auricle or changes in the auditory canal are often

found. On the audiogram: conductive hearing loss without a sensorineural component. Tympanogram: type A or Ad, ipsi- and contralateral acoustic reflex is absent. When performing CT, lesions of the bone structures of the tympanic cavity are revealed in the form of dysplasia or aplasia of the auditory ossicles, dystopia of the facial nerve canal, bone changes in the area of the windows of the labyrinth.

Discussion.

A large number of scientific papers are devoted to the role of metabolic disorders in the course of changes in mineral metabolism and the progression of otosclerosis. AurbaL et al. (1992) drew attention to the role of parathyroid hormones (parathyroid hormone, PTH), namely the PTH-mediated effect of osteoblasts in the process of bone tissue metabolism. Hearing loss develops gradually, gradually progressing, although the rate of this process is quite variable.

According to the rate of progression of hearing loss, the following are distinguished:

• slowly progressing forms (socially unsuitable hearing develops on average within 9-10 years from the onset of the disease);

• fulminant forms (almost complete deafness develops within a few months due to the involvement of the nervous elements of the inner ear in the process);

• protracted forms (onset in old age).

Features of the manifestation of hearing loss

1. Hearing loss is almost always bilateral, but often the patient complains of unilateral hearing loss, since hearing loss is always asymmetrical and the patient thinks the better hearing ear is healthy. The difference in hearing acuity of both ears is usually insignificant. 2. With otosclerosis, there is no complete deafness, the patient hears himself even with a high degree of hearing loss, due to which speech deformation does not develop, as happens with sensorineural hearing loss.

3. The onset of the disease in women is often associated with pregnancy and childbirth.

4. Characteristically, hearing improves in a noisy environment (paracusis Willisii), decreased speech intelligibility when swallowing and chewing (deprecusis Scheer), when several people talk at the same time (Toynbee symptom) and with intense attention (Urbanich-Walbe symptom).

Tinnitus usually tends to increase as hearing loss progresses, and is much more difficult for patients to bear than with other forms of hearing loss. In some cases, tinnitus deprives patients of sleep and appetite and sometimes leads to despair, close to suicide. Subjective tinnitus is most often low-pitched and resembles the sound of falling water, wind, surf, rustling leaves, the hum of wires, and may resemble buzzing, hissing, or clicking. The noise is mostly constant, but may

increase under the influence of alcohol, mental stress, fatigue, during a runny nose, after physical work, or sports activities.

The severity of subjective noise has 3 degrees:

I degree - the patient is almost not bothered by tinnitus, and the presence of this symptom is revealed during an active survey;

II degree - complaints of tinnitus are presented along with other complaints;

III degree - the sensation of tinnitus is the leading complaint of the patient.

Dizziness in patients with otosclerosis is quite rare. Patient complaints vary from vague instability to attacks of systemic dizziness. Positional vertigo is typical, occurring in a certain position, with a certain turn of the head, with rapid bending and throwing back of the head, and quickly getting out of bed. The cause of vertigo is considered to be intoxication of the nerve endings of the stat kinetic receptor when otosclerosis spreads to the semicircular canals and internal auditory canal, as well as physical or physiological obstruction of the vestibular aqueduct, biochemical changes in the composition of the perilymph when the bone capsule of the inner ear is involved, and hydrops of the labyrinth in the initial stage of the disease.

Oxidative stress. Oxidative stress and reactive oxygen species (ROS) are actively associated with the development of age-related hearing loss and drug-induced ototoxicity. It has been proven that under the influence of ROS, the process of oxidative phosphorylation is disrupted and signaling molecules are produced to enhance the production of angiotensin II. Also, ROS, namely 4-hydroxynonenal (4-NH), acts as a regulator of the production of TOB-β.

In otosclerosis, ROS can arise in otosclerotic foci and spread to the inner ear, which leads to damage to the cochlea and sensorineural hearing loss.

Conclusion

Studying the etiopathogenetic aspects of otosclerosis remains relevant to this day. The introduction of molecular genetic research methods has contributed to the transition of research to a new level and the development of a number of new theories. However, none of the theories can fully explain the process of otosclerosis development, which provides grounds for future large-scale research in this area.

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