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ETIOLOGY, PATHOGENESIS OF ACUTE SENSORINEURAL HEARING LOSS OF INFECTIONAL GENESIS

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ABOUT ARTICLE

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Abstract: According to a recent epidemiological survey, the incidence of acute sensorineural hearing loss (ASNHL) is increasing yearly. The cause of ASNHL is of great interest in research. To date, viral infection, vascular occlusion, abnormal cellular stress responses within the cochlea, and immune-mediated mechanisms are considered the most likely etiologies of this disease. Among these etiologies, the relationship between viral infection and sudden deafness has been unclear. According to the course of the disease, sudden, acute and chronic forms are distinguished. When the disease starts suddenly, it develops unilaterally in a few hours without any symptoms. The acute form develops slowly over several days. The chronic form is characterized by a long period (more than 1 month) and slow hearing loss.

INTRODUCTION

Sensorineural hearing loss is a hearing loss caused by damage to the inner ear structures, the vestibulocochlear nerve, and the central parts of the auditory analyzer (the auditory cortex of the brain) [1-6]. According to the course of the disease, sudden, acute and chronic forms are distinguished. When the disease starts suddenly, it develops unilaterally in a few hours without any symptoms. The acute form develops slowly over several days. The chronic form is characterized by a long period (more than 1 month) and slow hearing loss[7-12].

1. Infectious diseases. Most often, hearing loss is observed after the defeat of the influenza virus, measles, as well as after scarlet fever, diphtheria and syphilis.

2. Toxic effects, including iatrogenic nature: ototoxic drugs (aminoglycosides, loop diuretics, chemotherapy drugs).
3. Genetic factors or hereditary predisposition - 75% of cases of hereditary hearing loss are related to recessive non-syndromic hearing impairment, i.e. there is no damage to other organs and systems associated with hearing loss and is inherited
4. Diseases of the cervical spine, for example, spondylosis, spondylolisthesis of the cervical spine.
5. Pathology of the circulatory organs: hypertension, ischemic disease, cerebrovascular accidents, etc.
6. Age-related hearing loss, or presbycusis [3,13].

It is widely believed that viral infection is the key etiology of acute sensorineural hearing loss (ASNHL). Sensorineural hearing loss occurs with viral infections, including herpes simplex virus (HSV), mumps virus, measles virus, rubella virus, hepatitis virus, human immunodeficiency virus, Lassa virus, and enterovirus [14,15]. The relationship of hearing loss with diseases caused by these viruses is confirmed, along with clinical manifestations, also by serological indicators - an increase in the titer of the corresponding antibodies. It is generally accepted, for example, that the danger of sudden hearing loss with parotitis occurs 4 days before the swelling of the salivary glands and submandibular lymph nodes characteristic of this disease and persists for another 18 days after it disappears. In the absence of obvious clinical signs of mumps, a high titer of specific antibodies can be detected within 2-3 weeks. from the moment of unilateral hearing loss, and the titer of class M immunoglobulin remains elevated for another 3 months [16].

Pathogenesis of acute sensorineural hearing loss of infectional genesis:

ASNHL can develop in viral infection based on three different mechanisms: the first mechanism is based on the development of neuritis as a result of direct invasion of the cochlear nerve by the virus or the development of cochleitis under the influence of perilymph and endolymph; The second mechanism is via the reactivation of latent virus within tissues of the inner ear, under certain conditions. The third mechanism is through a virus indirectly triggering SSNHL, which involves a systemic or distant viral infection triggering an antibody response that cross-reacts with an inner ear antigen (an example of the immune-mediated hypothesis) or that triggers a circulating ligand, causing pathologic activation of cellular stress pathways within the cochlea (an example of the stress response hypothesis) [17].

Damage to the inner ear in parotitis is a direct effect of infection. The virus can damage the inner ear by spreading directly through the blood or through the cerebrospinal fluid to the perilymph space [18-24].

When examining an animal model infected with the mumps virus, it can be observed that the outer hair cells are atrophied, the vestibule and drum scales are affected by fibrosis. Viral capsids can be observed in the fibers of the auditory nerve, and viral antigens are located throughout the shell.

Ramsey Hunt syndrome is a typical example of virus reactivation. The varicella virus can remain latent for a long time in the nodular ganglion, vestibular ganglion, and spiral ganglion, and when the reactivity of the body decreases, it can activate and cause neuritis of the auditory nerve and facial nerve. Systemic infection activates the shell's innate immune system and causes a strong immune response. The immune response against viral peptides is explained by the production of antibodies directed at phospholipids [19,25]. Psillas was the first to describe the development of paresis of the facial nerve and bilateral ASNHL as a result of autoimmune diseases. The immune response against viral peptides is explained by the production of antibodies against phospholipids. Psillas et al. stated that a 33-year-old patient had vesicular herpetic eruptions on the external auditory canal and paresis of the facial nerve. After two weeks, the patient suddenly lost his hearing. The serum of the patient was examined and it was determined that the amount of IgG and IgM against herpes was increased.

Adhesion molecules and cytokines play a key role in the immune response of all mammalian tissues. Systemic inflammatory diseases, viral infection, physical and mental stress provoke an innate immune response by increasing the production of cytokines and the active form of oxygen in the inner ear. Because a cellular immune response is needed to overcome a viral infection [20,21,22].

With regard to the herpes virus, data were also obtained on the intactness of the inner ear when animals were infected through the large cisterna of the brain, that is, directly in the subarachnoid space [26]. It can probably be assumed that viral labyrinthitis occurs under certain conditions, but they do not determine the pathogenesis, clinic and frequency of sudden hearing loss. In other words, sudden hearing loss, being a nosologically independent form of sensorineural auditory pathology, is characterized by pathogenetic independence as a complication of acute viral infections. Another thing is what this independence consists of.

The incidence of acute respiratory viral infections is still high. It is known that the clinical manifestations of acute respiratory viral infections, including influenza, are not always clearly expressed, the range varies widely from "almost a healthy state" to very violent severe manifestations. Sometimes self-healing of ASNHL is explained by activation of cochlear immune response. But the activation of the permanent immune response leads to irreversible sensorineural changes [27]. Experiments conducted in animals infected with the Lass virus and developing ASNHL show that an

uncontrolled immune response against the infection is observed with damage to the auditory nerve [28].

The results of investigations have shown that tumor necrosis factor is also related to the pathogenesis of ASNHL development. Tumor necrosis factor (TNFα) activates sphingosine-1-phosphate and brings the shell into the constructive state of the microcirculatory ring [16]. Tumor necrosis factor is an evaluative factor in the treatment of ASNHL. Investigations by Zinovia et al. show that the recovery of hearing in the treatment of ASNHL with corticosteroids is associated with a decrease in the amount of TNFα. It has been found that there is a positive correlation between the increase in the amount of interleukin (IL)-6 during the treatment of ASNHL with corticosteroids and hearing rehabilitation. IL-6 plays an antioxidant and antiapoptotic role in the immune response of the inner ear. Increased IL-6 increases antiapoptotic gene expression [26,27].

Gukovich V.A. (1983) conducted a systematic clinical and serological examination (observations over a single team of 383 people aged 15-22 years). During this period, 163 people fell ill (56 had A2 viral influenza, 27 had viral influenza B, 19 had parainfluenza, and 2 had adenovirus infection). Out of 104 patients with an established type of virus, a clinically clear expression of the manifestations of the disease was noted in 28 (27%). Serological examination of all other persons revealed an increase in the titer of antibodies to respiratory viruses in 161 (42.3%) clinically healthy patients who did not seek medical help. These data characterize a high degree of infection with viruses in the surveyed contingent during the period (autumn-winter) of an increase in the incidence [29-33].

The mechanism of development of acute cochleitis and labyrinthitis can be explained by characteristic viral lesions of small vessels and subsequent pathoanatomical changes in the tissues of the inner ear: increased permeability of the vascular wall, extravasation and edema, which cause an increase in intralabyrinthine pressure, a violation of intracochlear conduction, and a sharp hypoxia of receptors [34-37]. This manifests itself in a fluctuating (rarely permanent) hearing loss of a perceptual or mixed (if there is a violation of endolymph mobility) type, usually of a moderate degree (up to 20-30 dB with an ascending type of curves with an air-bone gap of 10-20 dB), a feeling of fullness in the ear, low-pitched tinnitus, slight dizziness. Depending on the location of the spertensia (in the cochlea or the vestibular region), the symptoms may vary mainly in the direction of predominance or hearing loss or dizziness [38].

CONCLUSIONS

Thus, based on experimental and clinical studies of domestic and foreign authors, it can be concluded that ASNHL in influenza is a manifestation of primary sensorineural hearing loss and is due to the specific vasotropic and peirotropic effects of a viral infection on the inner ear. We have reviewed and summarized the relevant literature aiming to prove the etiology of ASNHL. Most studies have shown that viral infection is one etiology of SSNHL. However, there is little research proving or clearly indicating the pathogenesis of this disease. Thus, further research is needed to elucidate the precise etiopathogenesis of ASNHL to enable better understanding of the disease and establish more suitable treatment to restore patients' hearing.

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