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ETIOLOGY, PATHOGENESIS AND DIAGNOSIS OF ACUTE SENSORINEURAL HEARING LOSS WITH VASCULAR GENESIS

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ABOUT ARTICLE

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Abstract: Acute sensorineural hearing loss is an urgent condition that can develop irreversible and serious complications if is not treated on time. Hearing loss can develop suddenly or over several hours. Patients complain of congestion in the affected ear, noise in the ears, and often dizziness. Hearing loss is usually unilateral, and in 2% of cases it is bilateral. According to epidemiological data, the disease rate is 5-27 cases per 100,000 population. Unlike conductive tinnitus, sensorineural tinnitus is characterized by damage to the sound-receiving part of the unilateral auditory analyzer. The cranial artery is the only terminal artery that supplies the cranium with blood. A.auditivae is separated from the internal basilar artery in different situations. It separates from the lower anterior cerebral artery in 65% of cases, from the main one in 29%, and from the lower posterior cerebral artery in 6% of cases.

INTRODUCTION

Acute sensorineural hearing loss is an urgent condition that can develop irreversible and serious complications if is not treated on time. Hearing loss can develop suddenly or over several hours. Patients complain of congestion in the affected ear, noise in the ears, and often dizziness. Hearing loss is usually unilateral, and in 2% of cases it is bilateral [1]. According to epidemiological data, the disease is 5-27 cases per 100,000 population [2]. Unlike conductive hearing loss, sensorineural hearing loss is characterized by damage to the sound receiving part of the unilateral auditory analyzer [3].

According to etiological factors, the following forms of sensorineural hearing loss are differrented:

- Vascular insufficiency
- inflammation
- viral infection
- Toxic effects
- Trauma (mechanical ear trauma, acute trauma, barotrauma)

Despite of, questions about the causes of acute sensorineural hearing loss remain open. In acute sensorineural hearing loss, outer and inner hair cells of the cochlea, which convert sound vibrations into electrical impulses, are damaged [4].

Diagnosis of acute sensorineural hearing loss. Collecting the patient's complaints and anamnesis is of great importance in the diagnosis of acute sensorineural hearing loss. Most of the patients complain of a sudden change in hearing, ear blockage [5]. Patients may complain of tinnitus and dizziness along with a sudden change in hearing. When listening to patients' complaints, it is necessary to pay attention to how long the patient has noticed a decrease in hearing acuity, how the nature of hearing loss has changed over time, and to determine the probable etiological factor. It is necessary to ask whether the patient had any colds or infectious diseases before the onset of hearing loss, what medications he regularly takes, and whether he has started taking any medications.

It is necessary to carry out laboratory tests such as general blood analysis, coagulogram, biochemical analysis of blood, determination of the amount of lipids in blood [5]. Certain blood tests, such as inflammatory markers and immunological tests, can help determine the cause of the disease, but are not necessary to start treatment. Because hyperglycemia is observed in the majority of patients with sensorineural deficits of unknown cause. Cerebrospinal fluid should be checked in patients with neurological problems.

All patients are required to undergo an endoscopic examination of the ear, otomicroscopy, examination of the patency of the auditory canal, camerton tests and an audiogram. Differentiating acute sensorineural hearing loss from other hearing problems is an important first step for the physician. Otoscopy is almost normal in SSNHL patients. Weber and Rinne camerton tests are performed to distinguish SSNHL from conductive hearing losss [6]. Tonal threshold audiometry is more reliable in differentiating SSNHL from conductive hearing loss than tonal tests [7]. Absence of the air-bone interval and descending audiogram are characteristic for SSNHL. MSCT of the temporal bone and impedancemetry are of great importance in the diagnosis of SSNHL patients. Examination of tympanometry and acoustic reflexes shows that middle ear structures are involved in the process. In

order to rule out organic changes in one-sided hearing loss, MRI of the brain is performed together with visualization of the spinal cord. Some studies show that 95% of patients with vestibular schwannoma have hearing loss, and 7-20% have sudden hearing loss[9,10].

Vascular etiology of SSNHL. Cardiovascular diseases are one of the etiological factors of SSNHL development. SSNHL can develop mainly under the influence of the following vascular factors: violation of vascular permeability and rheological properties of blood (circulation disorders in the carotid artery, formation of blood clots, hemorrhages in the endo- and perilymphatic areas, and vascular immunoallergic processes) as a result. Labyrinth artery is the only terminal artery that supplies blood to the skull. A.auditivae is separated from the internal basilar artery in different situations. It separates from the lower anterior cerebral artery in 65% of cases, from the main one in 29%, and from the lower posterior cerebral artery in 6% of cases[11]. When the labyrinth artery enters the internal auditory canal and reaches the modiolus, it is divided into cochlea, cochlea-vestibular and vestibular branches. The cochlea artery enters the central canal of the cochlea and divides into 3 groups of capillaries in the modiolus: one group supplies blood to the bundle of vessels, lig spiralis; the second group gives a network to the spiral plate, the bazal membrane and the lumbus; the third group supplies the spiral ganglion with blood. The basal part of the cochlea receives a branch from the cochlea-vestibular artery. The wall of the inner ear artery consists of smooth muscles, and therefore the blood flow in the pinna depends on the general blood pressure and blood circulation in the brain. Therefore, the oxygen demand of the snail is extremely high. Therefore, the hairy cells of the shell are resistant to a decrease in oxygen supply. The fact that the high frequencies when the internal carotid artery is obstructed, and the low frequency sounds when the carotid artery is blocked is explained by its blood supply [12]. The cochlea is very sensitive to the inflammatory process, the effects of ototoxic drugs, noise and acoustic traumas, and the effects of active forms of nitrogen responsible for intracellular oxidation processes[13]. In 2011, the rheological properties of blood, lipids of patients with acute sensorineural hearing loss of various genesis, examined for many years, and the results of examination of brachiocephalic vessels were published. The results of the investigation show that in the development of acute sensorineural hearing loss, the microcirculation in the inner ear is disturbed, which leads to the disruption of the metabolism of nerve fibers and neuroepithelium and the death of hair cells. The majority of patients with acute sensorineural hearing loss have lipid metabolism disorders, which are observed with an increase in the amount of triglycerides and low-density lipoproteins [14].

Violation of blood supply to the inner ear can lead to changes in its structure. This shows how important the role of microcirculation is in the development of hearing loss [16]. The development of SSNHL in

the form of acute blood circulation in the brain is of great importance. In 2016, otorhinolaryngological examinations were conducted in 66 patients with mild ischemic stroke in inpatient conditions. The age of the patients ranged from 37 to 67 years. The main complaints of the patients were dizziness, ringing in the ears and speech disorders. When performing a tonal threshold audiogram for all patients, unilateral hearing impairment of 1 degree was detected in 47% of patients[15]. The amount of oxygen in the cochlea is very limited, and therefore, blood circulation disorders in it first lead to functional changes, and then to morphological changes. Investigations by Tsunao (2001) show that cochlea oxygen concentrations are as low as 0.47 microliters per 1 mm3, but functional changes are observed when oxygen concentrations decrease to 0.1 microliters per 1 mm3.

Chau and others believe that cardiovascular diseases, angioneurological diseases, hemorrhages in the brain and brain bridge areas, and transient ischemia are important in the development of SSNHL [17]. According to the vascular theory of SSNHL, a decrease in blood flow to the inner ear leads to local ischemia, and in this case, it is necessary to increase the amount of oxygen in the perilymph. The results of the tests show that the partial pressure of oxygen in the blood is higher in hyperbaric conditions [18]. In addition, a violation of blood supply in the inner ear can indicate an acute violation of blood circulation in the spinal cord [18].

It is well known that how long it takes to start treatment affects the recovery of hearing and the recovery of SSNHL symptoms. Therefore, the manifestation of etiological factors plays a key role in choosing an adequate treatment.

CONCLUSIONS

The fact that the cochlea is located in the temporal bone, its blood supply is limited to only one labyrinth artery, and the hair cells of the cochlea are extremely sensitive to oxygen show the importance of the vascular theory of SSNHL. Labyrinth artery is the only terminal artery that supplies blood to the skull. A.auditivae is separated from the internal basilar artery in different situations. It separates from the lower anterior cerebral artery in 65% of cases, from the main one in 29%, and from the lower posterior cerebral artery in 6% of cases. The basal part of the cochlea receives a branch from the cochlea-vestibular artery. The wall of the inner ear artery consists of smooth muscles, and therefore the blood flow in the pinna depends on the general blood pressure and blood circulation in the brain. Therefore, the oxygen demand of the snail is extremely high. Therefore, the hairy cells of the shell are resistant to a decrease in oxygen supply. The fact that the high-frequency sounds when the internal carotid artery is blocked, and the low-frequency sounds when the carious artery is closed, is explained by its blood

supply. Manifestation of vascular factors in the development of SSNHL is important for timely initiation of complex treatment.

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