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Glutathione - Involvement in the antioxidant defense of the auditory system

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Introduction Glutathione (GSH) is the most abundant low molecular weight thiol at the cellular level and is mostly located in the plasma membrane. GSH is one of the most important antioxidants in the human body and plays a important role against free radicals, specifically Reactive Oxygen Species (ROS) in systems that are subject to oxidative stress. However, this molecule does not act exclusively as an antioxidant agente, because it also has other functions at the cellular level such as intervention in metabolism and regulation. The association with the increase of oxidative stress and the metabolism of GSH, in the hair cells of the cochlea, has been related as one of the factors of hearing loss due to exposure to high sound levels. However, it is important to see if the mechanisms to increase the concentration of this substance contribute to prevent the death of the hair cells and, consequently, prevent hearing loss. There are two types of cell death: necrosis and apoptosis. Necrosis is usually induced by a noxious agent and apoptosis - or programmed cell death - is an active mechanism that plays a role in regulating the amount of cells in the body. Oxidative stress is one of the intracellular stimuli that can lead to its activation of the mechanism of apoptosis.

Objectives Determine if GSH plays a major role in hearing protection and identify what strategies exist to increase the concentration of this antioxidant in the human body, in particular in the cochlea.

Methodology Systematic review of existing literature on Glutathione as an agent in the antioxidant defense of the auditory system, with a detailed approach of the literature to answer the question: *what is the GHS involvement in the antioxidant defense of the auditory system?*

Conclusions Regarding GSH, it seems clear that it is the most important substance in the process of free radical neutralization (ROS) in the tissues of the cochlea. This increase seems to be related to several factors: exposure to high sound levels, aging, genetic factors, health status (diabetics, smoker…) and ototoxic agents. The use of N-acetylcysteine amide (NAC) - as a precursor of GSH - appears to be the most effective in hearing protection since it leads to an increase in GSH in cochlear tissues.

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Sudden sensorineural hearing loss related to vertebrobasilar arterial insufficiency – a challenge in clinical practise

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Keywords: Sudden sensorineural hearing loss, stroke

Background: Sudden sensorineural hearing loss (SSNHL) is defined as greater than 30 dB hearing reduction, over at least three contiguous frequencies, occurring over a period of 72 hours or less. It is a medical emergency with multifactorial causes, of which viral, autoimmune and vascular insufficiency are the most common. In clinical studies isolated SSNHL has been rarely related to vertebrobasilar arterial insufficiency (VAI).

Material and methods: In our observational study we analysed 22 cases affected by SSNHL eventually associated to vestibular symptoms but in absence of other neurological involvement to describe clinical course and report the incidence of SSNHL related to VAI. In the period from 1st January to 31st October 2018 we treated 70348 patients in our Emergency Center. Those with sudden hearing loss had been referred for diagnostic treatment to otorhinolaryngologist. We diagnosed 22 patients (15 males/7 females; aged 21 to 85 years) with idiopathic SSNHL by careful history, physical examination, looking for infections, systemic diseases, ototoxic medications and by documenting recent decline in hearing. For the diagnostic audiologic evaluation we performed pure tone audiometry, tympanometry, measuring otoacoustic emissions and brainstem auditory evoked potentials. In all patients we performed balance tests and Computer Tomography (CT) or Magnetic Resonance Imaging (MRI) of the brain.

Results: In all cases only one ear was affected. 20 patients (90,90%) had tinnitus and fullness in the ear and 5 (22,73%) had vertigo. 13 patients (59,09%) had cardiovascular risk factors. In 2 cases (9,09%) we diagnosed infratentorial stroke. Both patients were men with cardiovascular risk factors.

Conclusion: SSNHL is the presenting symptom of an emergency for which we have to find a convincing course and reliable treatment. The clinical presentation of our cases emphasizes that a careful follow-up of any patient with SSNHL is warranted. Our results confirm 9,09% incidence of VAI as the cause of SSNHL. This should be suspected in patients with cardiovascular risk factors. Acute audiovestibular disturbance can be the harbinger of more widespread posterior circulatory ischemic stroke. We underline the necessity of its early recognition and proper management.