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Sudden sensorineural hearing loss: an otorhinolaryngologic emergency

Abstract

Sudden Sensorineural Hearing Loss (ISSNHL) is sudden hearing loss, greater than 30 dB in 3 consecutive frequencies, can be accompanied by tinnitus and sometimes vertigo. The highest incidence occurs from the age of 45 between 55 and 65 years. Its etiology is unknown, but there are several hypotheses such as viral, immunoallergic and vascular causes. Its diagnosis should be made in the shortest possible time in order to guide the etiopathogenic possibilities and start treatment as soon as possible. The injection of intratympanic corticosteroids is the treatment of choice in order to achieve the recovery of hearing thresholds.

Keywords: deafness, suddenness, hearing loss, idiopathic

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Sudden sensorineural hearing loss (ISSNHL)

Sudden Sensorineural Hearing Loss (ISSNHL) is defined as hearing loss which has changed more than 30 db, across 3 consecutive frequencies, within a 72 hour period. It is accompanied by tinnitus, a sensation of aural fullness and transient signs of sound distortion, such as hyperacusis, algiacusia, autophony, diploacusis, as well as peripheral vertigo associated or not with neurovegetative courtship.[1,2]

Etiopathogenesis

Although the triggering causes are unknown, multiple hypothesis have been explored. Among the most interesting hypothesis are viral, vascular, allergic, pressure, trauma, autoimmune and related issues, which will be addressed below.

Viral infectious hypothesis

The existence of infectious antecedents such as upper respiratory tract flu-like illnesses are well-known. These infectious viral outbreaks may be subclinical and the patient may not be aware of them and may not report them. Greater incidence has been observed in the 3rd and 4th decade of life, which may present with very fast onset and variable predisposition to recovery. Factors which impact recovery are not known but may include viral dose, duration of active exposure, susceptibility of the individual and more.

Neuronal deterioration due to viral infection may include, atrophy of the organ of Corti, folding of the tectorial membrane and endolymphatic hydrops. Multiple viral agents show capacity to trigger am imappropriate, exaggerated or misinterpreted immune response, with generation of antigen-antibody immunocomplexes which may be present in the organ of Corti.

Vascular hypothesis

Vascular Hypothesis is perhaps considered when ISSNHL occurs between the ages of 55 and 65. Hypoxia and ischemia evolved in the context of a deteriorated vascular bed, especially in the microcirculation. The situation would be more acute if one thinks of a terminal vascular system such as the cochlear, lacking collateral circulation and anastomotic vicariants of sufficient entity to supply metabolic needs.

Cochlear vascularization derives from the labyrinthine artery, a branch of the anteroinferior cerebellar artery.

There are risk factors related to (ISSNHL)[3–6]

Hemorrhagic: alterations in central venous drainage, hypertension, leukemias, bone marrow aplasias and hemostatic disorders such as thrombocytopenia, liver disease or anticoagulant or antiplatelet medication.

Obstructive: atheromatosis or cardiogenic embolism (arrhythmia, valvular disease, metabolic microangiopathy), prothrombotic coagulopathies (antithrombin III deficiency, protein C and/or S, haemopathies and dysfibrinogenemias), hyperviscosity syndrome with slowed blood flow (macroglobulinemias, polycythemia, sickle cell disease, platelet hyper aggregability, dyslipidemias, diabetes).

Vascular spasm: explains the reversibility and recurrence of some episodes of SBI and the therapeutic effect of vasodilatory agents. Tension instability and vegetative imbalance would favor these disorders.

Allergic hypothesis

It is assumed that type I hypersensitivity mechanisms (anaphylactic reactions) would determine HLSS due to disorders in capillary permeability and edema in the vascular stria.

Hypothesis of increased pressure

Endolymphatic hydrops occurs with specific or preferential involvement of the anterior vestibule, which may be accompanied by vestibular symptoms, as well as the possibility of recovery and relapse, depending on the dynamic disorders that the endolymph may experience, either due to hypersecretion from the vascular stria or hyporesorption in the endolymphatic sac.

Traumatic hypothesis

It can occur in some surgical acts performed in the middle ear with injury to the oval or round window that communicates it with the labyrinth. It may present with peripheral vertiginous syndrome and a positive fistula sign.

Autoimmune hypothesis

There are some data that can justify this hypothesis, such as: bilaterality (although not synchronous), the relapsing nature of some episodes, the optimal response of some cases to corticosteroid therapy, the increasing detection of specific anticochlear antibodies in tension vascular pathology and the concomitant association of

other autoimmune conditions (SLE, antiphospholipid syndrome, Sjögren's syndrome, Hashimoto's thyroiditis, vasculitis, recurrent polychondritis, panarteritis nodosa, Wegener's disease, Cogan's syndrome.

Epidemiology

There is a greater predisposition in women to immune disorders and hormonal variations, and in men to vascular conditions. The highest incidence occurs from the age of 45 between 55 and 65 years.

Viral, allergic and autoimmune processes speak in favor of the first peak of involvement and vascular processes for the second.

Symptomatology

The way in which HLSS is established makes the patient consult quickly for unilateral hearing loss that is easily accurate at the time of onset. The synchronous involvement of cases does not exceed 1%, although approximately 5% of cases develop a similar episode in the other ear over time. There are no prodromes or auras, but they can be associated with tinnitus or other symptoms of sound distortion, as well as symptoms of peripheral vertigo with Romberg maneuver and vestibulospinal tests positive for the diseased ear and horizontal rotating nystagmus, if detected, of rapid component to the healthy ear.

Prognostic factors

Age of the patient: better prognosis in those under 40 years of age, probably due to a less damaged microcirculatory system and more receptive to systemic medication.

Intensity of the initial hearing loss: the lower the hearing loss with which the HLSS begins, the greater the possibility of regression of it, since the mild functional disorder means that the cytolytic mechanisms have not yet been fully activated and their arrest and reversal is feasible. Hearing loss with a loss of less than 40-45 db seems to reflect contractile alterations in the external hair cells (SCCs) that are reversible spontaneously or after treatment. Loss greater than 65-70db would imply damage to the inner hair cells (ICCs), the sensory receptors themselves and therefore less susceptible to recovery.

Type of audiometric curve: from worst to best prognosis, cophosis, descending curves, flat curves, concave curves, convex curves, scotomas and ascending curves would tend to be pigeonholed. In cases where treatment is started beyond the sixth day after clinical onset, the response to any therapy applied seems to be a function of the audiometric pattern.

Hearing function of the healthy ear: the significant association between the indemnity of the ear contralateral to injury and a greater probability of full recovery from the HLSS is recognized.

Associated symptomatology: the existence of tinnitus or vertiginous syndrome of synchronous onset with hearing loss raises the possibility of a greater affectation of labyrinthine damage, in the same way persistence will be related to a lower rate of spontaneous or post-therapeutical recovery.

Early initiation of treatment: time between the appearance of hearing loss and the performance of audiometry since the potential vascular damage takes a few hours to establish itself and the metabolic involvement is still reversible. A rapid therapeutic option would stop the cascade of mechanisms that would lead to the annulment of CCIs and would even determine full cochlear replacement in the first days of treatment.

Early onset of clinical improvement: the chance of recovery is considered slim when the patient does not experience symptomatic relief in the first 7 days. About 30% of those affected by sudden deafness recover completely; another 30% do so incompletely, and another 30% do not recover. It is also suggested that some patients recover completely without medical intervention during the first three days of experiencing the loss, which is called spontaneous recovery. Others improve slowly within a period of one to two weeks, and 15% of Sudden SensoriNeural Hearing Loss (ISSNHL) sufferers experience hearing loss that will worsen over time. Recovery is always better in the bass (lower) frequencies. There are delayed recoveries when a long time has passed since the end of treatment. Some may recur (recur) and in such a case, they are called fluctuating deafness.[5,6,7]

Diagnosis

Sudden SensoriNeural Hearing Loss is a sensorineural emergency and its early diagnosis should be considered in the shortest possible time in order to guide the etiopathogenic possibilities of these diseases, modify any risk factors, control any concomitant pathology and initiate a specific therapeutic regimen.

The anamnesis should be taken as soon as possible, in detail and all the data regarding the circumstances of the appearance of the condition should be included, both on the period of time that has elapsed since its appearance and on the clinical signs that occur. Personal history of previous infectious episodes, especially mumps or colds of the upper respiratory tract, cardiovascular, hematological, and hepatic or dysmetabolic history, as well as a history of tumor, allergic, autoimmune diseases, will be collected. It is helpful to get an idea of cochlear function prior to the onset of the condition by asking about your hearing.[8–10]

A family history of deafness, as well as hereditary diseases, vascular disorders, or chronic infections, should also be investigated.

The medication that the patient uses must be known, emphasizing the intake of ototoxic, cardiotonic, anticoagulants, antiplatelet agents, oral contraceptives.

Physical exam

Otoscopy: normal

Examination of diapazones: Weber lateralized to the healthy ear, Rinne: shortened positive, Schwabach: shortened.

Liminal tonal audiometry: Sensorineural hearing loss with an intensity of more than 30 dB at three successive frequencies.

Supraliminal tonal audiometry: guides the topodiagnosis of the lesion.

Speech audiometry: does not include 100% of phonemes.

Complementary tests: They will be indicated, according to the pathological history of each patient and the possible cause, if detected in the anamnesis: (blood count, hemochemistry, coagulogram, serological, virological and immunological studies).

MRI: to rule out acoustic neuroma, tumors, infarctions, thrombi, demyelination.

Treatment

Different treatments have been proposed without being able to carry out a universally accepted protocol, due to the fact that there is no specific cause, only the empirical application and the presumption of certainty of some of the etiopathogenic hypotheses that support

the treatment. Over time, antivirals, vasodilators, anticoagulants, mineral and antioxidant supplements, and hyperbaric oxygen have been used.[10,11]

Currently, steroids are considered the most effective and commonly treated approved. Steroids can be administered orally, intravenously, or locally by intratympanic injection. The intratympanic administration of corticosteroids has two advantages: the high perilymphatic concentration it achieves and the reduction of adverse effects achieved with systemic absorption. In addition, it has been used as a therapy for sudden refractory hearing loss refractory to treatment in the middle ear. The injection of intratympanic corticosteroids should be performed under microscopic vision, after placing the patient in the supine position, with topical anesthesia with 10% lidocaine for 20 minutes. Dexamethasone 1ml is injected into the posteroinferior quadrant of the tympanic membrane. This procedure is performed once a week, for 4 weeks waiting for recovery results from hearing loss up to 2 months after treatment.[12,13]

Conclusion

ISSNHL is considered an otorhinolaryngological emergency, so this diagnosis should be thought about in order to start treatment as soon as possible and to be able to obtain better recovery from hearing thresholds.

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Conflicts of interest

The author declares that there is no conflicts of interest.

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