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Treatments for Idiopathic Sudden Hearing Loss: Literature Review

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ABSTRACT

Idiopathic sudden hearing loss is characterized by an acute onset of hypoacusis, usually unilateral, occurring in less than 72 hours and with a loss equal to or greater than 30 decibels (dB) in at least three consecutive audiometric frequencies. In 71% of cases, the etiology is idiopathic. The symptoms are unpleasant and limiting, so a proper therapeutic plan is needed to minimize the impact on patients' lives. The purpose of this study is to describe treatment modalities, characterize the action mechanism, describe the efficacy and identify the risks of the therapeutic modalities. This is a study of narrative literature review; the articles were taken from Pubmed, Scielo, National Library of Medicine and Google Scholar databases. 35 articles from 1995 to 2023 were analyzed. In the studies, it was reported that treatment of idiopathic sudden hearing loss is controversial due to the different etiological and pathophysiological theories, being based on: systemic corticosteroid therapy, intratympanic corticosteroid therapy, hyperbaric oxygen therapy, antivirals and vasoactive or vasodilating substances. It is concluded that, although there is little high-quality evidence to prove the superiority of any treatment, those with the best risk-benefit ratio should be used. Systemic corticosteroid therapy may be used, associated or not with intratympanic corticosteroid therapy and hyperbaric oxygen therapy according to availability. Antivirals and vasodilators may be disregarded.

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Introduction

The human auditory system is composed of three parts: the outer ear, the middle ear and the inner ear. The external ear, formed by the ear auricle and the external auditory meatus, is responsible for collecting and forwarding sound waves; in addition, it protects the tympanic membrane. The middle ear consists of the tympanic membrane, the tympanic cavity, the auditory ossicles and the auditory tube. In addition, it is responsible for equalizing the impedances of the middle and inner ear. The inner ear is composed of the bony labyrinth, membranous labyrinth and internal acoustic meatus, being responsible for the transduction of mechanical energy into electrical energy [1].

Hearing loss is characterized as the reduction or absence of the ability to hear certain sounds and can be classified as: conductive, sensorineural and mixed hearing loss [2]. Transmission or conductive hearing loss is characterized by the change in the amount of sound, that is, in the outer ear or the middle ear, thus preventing the transmission of sound waves. In sensorineural hearing loss, the abnormalities are located in the inner ear or in transmission to the brain, with changes in sound quality. In the mixed type, both conduction and sensorineural occur in the same individual [3]. The degree of hearing loss is classified as mild (21 to 40 dB), moderate (41 to 70 dB), severe (71 to 90 dB) and profound (above 95 dB), based on the average value of 500, 1000 and 2000Hz frequency thresholds [4].

Idiopathic sudden hearing loss is characterized by the acute onset, usually unilateral, of hearing loss, occurring in less than 72 hours and with a loss equal to or greater than 30 decibels (dB) in at least three consecutive audiometric frequencies. It usually manifests itself between 50 and 60 years of age, being similar in both genders [5]. Previously, the incidence of sudden hearing loss was estimated to be between five and 20 cases per 100,000 individuals per year. However, more recent studies show a considerably higher incidence, from 160 to 400 cases per 100,000 individuals per year [6]. There is a wide etiological variety in sudden hearing loss, including neoplastic (2.3%), vascular or hematological causes (2.8%), trauma (4.2%), ear diseases (4.7%), infectious diseases (12.8%), idiopathic (71%) and others (2.2%). Therefore, it appears that, in the vast majority of cases, a cause is not identified, which is called idiopathic etiology [7].

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In idiopathic cases, pathophysiological mechanism is uncertain, and some of the most accepted hypotheses are: vascular impairment, rupture of the cochlear membrane, autoimmune mechanisms and viral infection [8]. Unilateral hypoacusis occurs in 98% of cases and generates difficulties in speech perception in spaces and in sound localization, in addition to tinnitus discomfort, vertigo and depression, which can lead the patient to social isolation. The signs and symptoms of idiopathic sudden hearing loss (ISHL) are unpleasant and limiting, so an adequate therapeutic plan is important to minimize the impact on the patient's life [6]. The prognosis of ISHL depends on some factors, such as age, presence of vertigo at the onset of symptoms, degree of hearing loss, audiometric curve pattern and, as the main prognostic factor, the early initiation of therapy [9].

In cases of sudden hearing loss with a predefined cause, treatment is based on the cause of the disease itself. In ISHL, due to different etiological and pathophysiological theories, treatment is controversial, being based on one or more of the following modalities: systemic corticosteroid therapy (SCT), intratympanic corticosteroid therapy (ITCT), hyperbaric oxygen therapy (HBOT), antivirals and vasoactive or vasodilating substances. Their ultimate goal is to recover the hearing of the affected individual [9].

In view of the increase in cases and etiological and pathophysiological controversies, therapeutic modalities available for ISHL are based on theories, so there is no consensus on the treatment for this pathology, therefore being a therapeutic challenge. In this sense, the purpose of this article was to review the medical literature seeking to present the effectiveness of treatments for sudden idiopathic hearing loss, aiming to describe the therapeutic modalities. In addition to it, to characterize the action mechanism, evaluate the efficacy and risks of each modality to thus obtain better clarification of treatments and verify if there is any therapeutic superiority.

Material and Methods

This was a study of narrative literature review. The literature search was carried out through the databases of Pubmed, Scielo, National Library of Medicine and Google Scholar. 35 articles were analyzed from 1995 to 2023, using a yearly temporal cut. Only articles on idiopathic sudden hearing loss, their treatments and mechanisms were used. Letters to the editor and manuscripts that were outside the scope of this study were excluded. Ethical care and information accuracy guarantee were respected.

The descriptors used for the literature searches were: "sudden hearing loss", "idiopathic sudden hearing loss", "sudden hearing loss treatment", "sudden hearing loss corticosteroid", "hyperbaric oxygen therapy", "hearing loss antivirals" and "hearing loss vasodilators". Firstly, data and information from articles on sudden hearing loss were collected. Secondly, articles on treatments were used, in which the action mechanism of each modality was reported. Finally, efficacy and risks were analyzed.

Results and Discussion

In cases of sudden hearing loss, hypoacusis is noticed upon waking up or trying to use the affected ear, such as by wearing headphones, for example. In addition, other symptoms such as tinnitus (90%), aural fullness/capped ear sensation (80%), vestibular symptoms (57%), can be reported by the patient. The clinical presentation is, in most cases, unilateral [8]. Initially, clinical evaluation should distinguish sudden sensorineural hearing loss (SSHL) from conductive hearing loss (CHL), as they have a similar clinical picture, but prognosis and treatment are different. Diagnosis is based on a combination of clinical history, objective examination and audiometry [10].

The objective physical examination includes otoscopy and acumetry (Rinne and Weber's test). In patients with SSHL, otoscopy will almost always have no changes, unlike cases of CHL. The Rinne test in the SSHL is expected to be positive, that is, air conduction is superior to bone conduction in the tested ear. The presence of sound lateralization in Weber's test occurs for the ear without pathology [11].

It is not recommended to routinely perform laboratory tests, such as blood count, coagulogram, electrolytes or erythrocyte sedimentation rate, as it was found that there is no improvement in patient management and has associated costs. Regarding serological investigation for infectious causes, this should be guided by clinic and epidemiology, but it should also not be requested as a routine [12]. On the other hand, the neurological physical examination is encouraged to evaluate the cranial and cerebellar pairs, seeking differential diagnoses [8].

The gold standard exam is tonal and vocal audiometry, which will evaluate the degree and pattern of sensorineural hearing loss [13]. Tonal and vocal audiogram should be performed as soon as possible, being mandatory for the diagnosis. Hearing loss must be equal to or greater than 30 dB in three consecutive frequencies, in 72 hours [12]. Brain magnetic ressonance imaging (MRI) with attention to the ears is the imaging test of choice to evaluate the cochlea and auditory pathways; it is useful to exclude cerebellopontine angle tumors and retrocochlear abnormalities [14].

The cerebellopontine angle is an irregularly shaped latent space located in the posterior cerebral fossa; 80 to 90% of the tumors that occur in this angle are Vestibular Schwannomas. These are benign, slow-growing, which over time compress the VII and VIII cranial pairs, leading to hearing loss, tinnitus and other vestibular symptoms. In addition, sudden hearing loss occurs in most cases, and investigation for correct diagnosis is relevant [15]. Regarding treatment, systemically administered corticosteroids (orally or intravenously) are used as initial therapy in ISHL, offering better recovery in the first two weeks after the onset of symptoms and little benefit after four to six weeks. Prednisone 1mg/kg/day (maximum dose of 60 mg/day), methylprednisolone 48 mg/day and dexamethasone 10 mg/day are used [9]. The action of these drugs is uncertain. It is believed to improve prognosis due to its ability to decrease inflammation and edema in the auditory organs [16].

As corticosteroid therapy is nonspecific, the different etiologies of sudden hearing loss may have benefits in this treatment. The use is based on the occurrence of an inflammatory process in the inner ear, which may be due to an autoimmune mechanism,

a viral infection or sequelae due to ischemia and infarctions [17]. SCT is the most widely used initial therapy, having anti-inflammatory action; a better patient prognosis is noted [9]. Generally, the side effects of systemic corticosteroids are controllable and known, such as weight gain, gastritis, increased thirst, as well as changes in appetite, mood and sleep. Moreover, hypertension, hyperglycemia, cataracts and avascular necrosis of the hip, these are more worrisome [18].

In cases of tuberculosis, diabetes mellitus, rheumatic disease, active atherosclerotic vascular disease, severe psychiatric disease, use of immunosuppressants, chemotherapy agents, radiotherapy, pancreatitis, hepatitis C or B infection, chronic renal failure, alcohol abuse, active herpes zoster, osteoporosis and history of head and neck cancer, the use of SCT alone is contraindicated [18]. In 1996, Silverstein reported the use of ITCT for the first time in sudden hearing loss. In this modality, the drug in question is administered directly over the round window membrane, thereby generating high concentrations of the drug, in addition to being an organ-specific therapy. Dexamethasone and methylprednisolone are the drugs of choice in this type of application [19].

The anti-inflammatory effects of dexamethasone and methylprednisolone are important for the best prognosis. The concentration of dexamethasone solution is 2-4 mg/mL to 25 mg/mL and methylprednisolone from 32mg/dL to 62.5 mg/dL, with the amount injected being 0.3 to 0.5 mL, approximately the volume of the middle ear [20]. ITCT can be used as a primary therapy, that is, as an initial treatment, without the use of systemic corticosteroid therapy. In addition, it can also be used as adjuvant therapy, along with the systemic one, and as rescue therapy when SCT treatment fails. The technique of ITCT has differences as in the methods of administration. There is transtympanic needle injection, myringotomy, myringotomy with ventilation tube, myringotomy with special capillary for perfusion and implantable infusion pump in the middle ear. Treatment time in this modality is variable, ranging from a single dose to weekly injections to an implantable infusion pump [21].

Regarding the advantages of intratympanic therapy modality, we can mention the following: it is relatively painless, can be used soon after diagnosis, easy to administer, there is high concentration of the agent when administered directly into the affected ear, can be done under local anesthesia - thus avoiding the use of general anesthesia - and it is possible to use it in patients in whom SCT are contraindicated [22]. The risks associated with the use of ITCT include vertigo, pain, tympanic membrane perforation, serous otitis media and the lack of evidence of having a superior efficacy over systemic therapy [19]. The combined use of systemic prednisone and intratympanic dexamethasone showed statistically significant improvements compared to the isolated use of both. Combined therapy modalities increased more than 15 dB in recovery. It is believed that, due to the high doses and because it is administered by two routes, this therapy prevents hair cell apoptosis, in addition to helping restore blood flow to the organ of Corti [23]. Oxygen consumption is high in stria vascularis and organ of Corti. In vascular etiological theory, hypoperfusion, due to phenomena of partial or temporal obstruction or blood hyperviscosity, would lead to a decrease in oxygen, and cellular apoptosis may occur. With HBOT, it is possible to increase the oxygen tension in the cochlea by up to 450% of the baseline value, compensating for any blood perfusion deficit. The sessions take place in a hyperbaric chamber with oxygen at 100% [24]. After diagnosis, HBOT treatment should be started as soon as possible. The application of oxygen therapy is strongly recommended during the two weeks following diagnosis. Treatment in conjunction with systemic corticosteroid therapy is recommended. The pressure should be 1.5 to 2.5 atmosphere (atm) for 90 minutes on a daily basis, in a total of 15 to 20 sessions [25]. Due to the high cost, access is limited [26]. Since ISHL may have an ischemic origin, the use of oxygen is among one of the therapeutic options; thus, HBOT promotes an increase in the partial oxygen pressure in the blood, acting as an anti-inflammatory, in addition to helping the decrease in the production of cyclooxygenase-2 and prostaglandin E2. It is usually combined with SCT [27].

In HBOT therapy, the risks are reduced; however, we can mention barotrauma, sinus and lung lesions due to the change in pressure, worsening of myopia, claustrophobia, oxygen poisoning and, the most common, auditory tube dysfunction [28]. Based on the viral infection theory, antivirals such as acyclovir and ganciclovir are used in the treatment, in most cases combined with corticosteroids [29]. The viral theory is based on the histological findings of the temporal bones, specific virus seroconversion and previous history of upper airway infection or other viral diseases. The main latent viruses are from the Herpesvirus group (Herpes simplex, Herpes simplex type 2, Varicella-Zoster, Cytomegalovirus and Epstein-Barr). Depending on the viral agent, hearing loss has a certain pattern, but almost all are unilateral. Inner ear damage generated by acute or latent attack can lead to hearing loss. It is known that the virus has an affinity for nerve tissue and is widely distributed in the body. Thus, in these cases, degeneration is more diffuse and the ear is subjected to fibrous tissue invasion, leading to cochlear ossification [30]. Other infectious etiologies include Syphilis, Toxoplasmosis and Rubella [31].

The main etiological theory is based on a viral cause; however, there is no evidence for the use of antiviral drugs associated with steroids in the treatment of ISHL [29]. Some authors reported that no antiviral was effective in the treatment of ISHL [32].

In theory, vasodilators improve blood supply to the cochlea, reversing hypoxia [31]. Considering that sudden hearing loss may have a vascular origin, it is believed that pathophysiological capillary and microvascular changes can lead to SHL, that is, due to embolism, hypercoagulability, vasospasm, intracochlear hemorrhage, arteriosclerosis, systemic vascular disease and connective tissue disorders. Vasodilators are used because they can improve blood flow. Vasoactive substances, on the other hand, change blood viscosity. Papaverine or nicotinic acid are vasodilators, other drugs are intravenous contrast (Hypaque), calcium channel blockers, prostaglandin E1 infusion and ethacrynic acid. Treatment time varies from days to months, depending on the agent used [33].

Vasodilators increase blood flow; based on the vascular change theory, their use in the therapy of ISHL has been proposed. However, it did not obtain scientific evidence of its use [9]. Prognosis of ISHL depends on early treatment initiation. Therapy before seven days has better rates of absolute recovery when compared to use above it. There is no significant difference between the onset before 48 hours and up to seven days. In addition to the delay in starting treatment, the worst prognosis is also related to vertigo, profound hearing loss and altered hearing in the contralateral ear [34].

Conclusion

In cases of idiopathic sudden hearing loss, there is no consensus on therapy. Thus, the most used rescue treatments are based on cause theories and the modalities used are: systemic corticosteroid therapy, intratympic corticosteroid therapy, hyperbaric oxygen therapy, antivirals and vasoactive or vasodilating substances.

In the analysis of therapeutic modalities, it was observed that the action mechanisms are different. Systemic and intratympanic corticotherapies act by decreasing inflammation and edema through anti-inflammatory action. Hyperbaric oxygen therapy, on the other hand, acts by increasing the oxygen tension in the cochlea, improving blood perfusion. Antivirals act by interrupting the infectious process and vasodilators act by increasing blood flow, reversing hypoxia.

Regarding the efficacy of treatments, it was found that systemic corticosteroid therapy was the best therapeutic option when used in the first two weeks. In addition, its contraindications and side effects are well known. In the use of intratympanic corticosteroid therapy, as it is a specific organ, high concentrations of drugs can be administered with good results, in addition to being a good option in cases of oral corticosteroid contraindication. The combined use of systemic and intratympanic corticosteroid therapy presents superior results when compared to their isolated use. Therapy with hyperbaric oxygen therapy is also an option, with good results if used in the first two weeks and combined with systemic corticosteroids. Antivirals and vasodilators do not demonstrate effectiveness in treatment.

While carrying out the therapeutic plan, risks also need to be considered. When using systemic corticosteroid therapy, the following may occur: drowsiness, weight gain, gastritis, cataracts, hypertension and avascular necrosis of the hip. The risks associated with intratympanic corticosteroid therapy are: vertigo, pain, tympanic membrane perforation and serous otitis media. When using hyperbaric oxygen therapy, auditory tube dysfunction, barotrauma, sinus lesions, lung lesions, worsening of myopia, claustrophobia and oxygen poisoning may occur. Based on the available studies, it is concluded that, currently, although there is little high-quality evidence to prove the superiority of any treatment, preference should be given to those with the best risk-benefit ratio, depending on the patient's comorbidities. Systemic corticosteroid therapy may be used, associated or not with intratympanic corticosteroid therapy and hyperbaric oxygen therapy, also according to availability. Antivirals and vasodilators may be disregarded. It is noteworthy, however, the need for controlled and randomized studies for better discussion of the subject.

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