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Meniere's Disease Treatment

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<http://dx.doi.org/10.5772/67981>

Abstract

The Meniere's disease is a chronic condition that requires treatment for long time and whose control is not always easy to achieve, requiring some multidrug treatments, and sometimes even procedures. We have many drugs and procedures to the treatment of Meniere's disease which may be taken according to the stage of disease and individual features.

Keywords: Meniere's disease, treatment, endolymphatic hypertension, betahistine, diuretics, endolymphatic surgery

1. Introduction

Meniere's disease (MD) symptoms are caused by the accumulation of endolymph in the membranous labyrinth, with consequent endolymphatic hypertension, that has been demonstrated in anatomical-pathological studies [1]. Endolymphatic hypertension leads to malfunction or irreversible damage to the sensory cells of the anterior and posterior labyrinths with their consequent symptoms. It is a multifactorial disorder and involves the participation of genetic and external factors.

The treatment of Meniere's symptoms is based on the control and reversal of endolymphatic hypertension which can be done through medications, change in life habits, and others. Conservative treatments are aimed at normalizing the membranous labyrinth system homeostasis and controlling the evolution of the disease as well as its symptoms. When symptom control cannot be achieved by conservative treatments, one can consider using invasive treatments that may assist in the control of endolymphatic hypertension, or destroy the labyrinth sensory cells.

It is worth noting that the clinical spectrum of Meniere's disease is broad, with the possibility of remission and recurrence of symptoms, and even very unfavorable evolution, with irreversible hearing loss and permanent damage to the vestibular function. A good treatment must be individualized for each patient, taking into consideration such possibilities, the stage of the disease, and the possible consequences that have been promoted.

The more common treatments include salt restriction, diuretics and betahistine, intratympanic gentamicin and steroids, ablative surgical therapies and endolymphatic sac surgery, and Meniett device. Many ways of hearing and vestibular rehabilitation are available for chronic damage treatment, according to each situation (**Table 1**).

Treatment	Level of evidence
Salt restriction	5
Diuretics	2b
Betahistine	2b
Steroids	1b
Gentamicin	1b
Endolymphatic sac surgery	2b
Vestibular nerve section	2b
Labyrinthectomy	2b
Meniett device	No evidence level

Table 1. Level of evidence of treatments according to the Oxford Centre for evidence-based medicine.

2. Treatment modalities

2.1. Salt restriction and other dietary modifications

The observation that water retention can exacerbate the symptoms of Meniere's disease (MD) was first documented in 1929 [2]. Studies on the increase of sodium levels inducing hydrops attacks have been performed since then, with numerous related publications [3]. More recent studies also suggest that the restriction of salt, associated with the use of diuretics, gets better symptom control in patients with Meniere's disease [4]. The endolymph disturbances of volume and electrolytes are the main cause of the symptoms experienced by patients with MD. High salt intake can affect the concentration of electrolytes in the blood, which affects the composition of endolymph. This fluctuation in the composition and volume of endolymph contributes to the floating nature of symptoms experienced by people suffering from MD [5].

A low-salt diet is an important treatment for patients with MD. The effectiveness of treatment is shown when sodium intake is reduced to less than 3 g per day. A low-salt diet can induce an

increase in plasma concentration of aldosterone, which can enable the transport of ions to the absorption of endolymph in the endolymphatic sac. Other dietary changes include limiting alcohol and caffeine intake as evidenced by [4]. Both alcohol and caffeine can lead to vasoconstriction and a decrease in blood supply to the inner ear which can make the symptoms of patients more intense.

2.2. Diuretics in Meniere's symptoms

Diuretics affect the balance of electrolytes in the endolymph, leading to the reduction of the volume and pressure, which can occur by increased drainage of endolymph or by reducing its production. They are usually used to control vertigo, hearing loss, tinnitus, and aural fullness in patients with Meniere's disease.

The article published by Ref. [6] makes an analysis of the retrospective medical records of patients with Meniere's disease and was designed to evaluate the effect of acetazolamide and chlorthalidone at the rate of hearing loss. Three groups were compared as follows: (1) 79 patients treated with clortalidona from 5 to 13.4 years; (2) 42 patients treated with acetazolamide between 5 and 7.8 years; and (3) a control group of 71 patients who received only the symptomatic treatment for intermittent dizziness, followed by 5–24.1 years. In the short-term, after 2–6 weeks of treatment, a statistically significant reduction of average hearing loss was observed with both clortalidona and acetazolamide. In the long run, more than 5 years, no preventive effect on the deterioration of hearing loss can be detected. Both acetazolamide and chlorthalidone may be useful for diagnostic purposes, causing a fluctuation of hearing, as well as to control the attacks of vertigo, but is not useful for the long-term prevention of deterioration in hearing on Meniere's disease [6].

Another double-blind randomized controlled study published in 1982 [7] comparing the betahistine use with hydrochlorothiazide. Patients were initially kept under observation for 3 months without medication beyond symptomatics. Then patients were divided into two groups, each with 16 patients receiving betahistine or hydrochlorothiazide during 6 months. Before and during treatment, subjective symptoms, such as dizziness, bouts of dizziness, tinnitus, aural fullness, and general well-being, were evaluated every 4 weeks. At the moment, betahistine seems to be the drug of choice for Meniere's disease with floating hearing thresholds. In all patients of this study, an improvement and reduction in the severity, frequency, and duration of vertigo attacks in 6 months of treatment was observed. In contrast, hydrochlorothiazide showed a distinct therapeutic effect on general well-being and vertigo, notably during the first months of treatment in patients with stable hearing thresholds [7].

Klockhoff et al. in 1976 [8] observed 34 patients with Meniere's disease who were treated with chlorthalidone. Twenty-six patients had drug-related improvements, especially a reduction in the prevalence and severity of vertigo. In four patients, the effect was minor, other four patients seemed to have resistance to chlorthalidone despite positive glycerin tests, and two of them needed surgery. Chlorthalidone was also given to 220 severely disabled patients who were hospitalized for further examination and possible surgical procedure. The improvements were obtained in such a way that the operation was avoided in 133 patients (60%).

Regular or long-term treatment with chlorthalidone that produces a symptomatic relief is considerable in many patients during the active phase of the disease. It reduces the need for surgical intervention and helps patients to maintain an active life, but not arrest the course of degenerative disease [8].

However, Brookes and Booth [9] published an observational study that was conducted in 14 patients who received acetazolamide with duration ranging from 1 week to 9 months and the therapeutic efficacy of this medication. There was improvement in four patients and improvement was not maintained in two of these, while the other had to cease the medication due to the development of kidney stone. The worsening of symptoms was observed in 3 cases and adverse side effects were observed in 6 of the 13 patients (46.2%) that fulfilled the dosage of drugs. It is suggested that this high incidence of side effects can be consistent with the general metabolic difference between Meniere's disease patients and normal individuals. This work concluded that acetazolamide has no place in the medical treatment of Meniere's disease [9].

More recently, a new review conducted by Duke University in 2016, based on boots with diuretics in MD for the last 10 years sought to assess, including any oral diuretic study in adult patients, hearing results reported, results of vestibular symptoms, effects, and complications of diuretic treatment. In this revision, 19 studies were included with considerable heterogeneity in the population of patients evaluated, design of studies, as well as type of diuretics, dosage, follow-up, and results. Most of these studies reported improvement in vestibular symptoms, but little improvement of hearing in these patients. As with other conditions faced by otorhinolaryngologists, diuretic therapy for Meniere's disease is often initiated as first-line therapy, although low level of evidence is present to justify its use (2b) [10].

Last survey of Cochrane [11] found no evidence of high quality to evaluate the efficacy of diuretics on Meniere's disease to not to introduce controlled double-blind randomized trials using placebo for diagnosis and outcome evaluation. Despite the lack of evidence of high quality, some studies have reported an improvement in patients' vertigo during use of diuretics in the short-term.

2.3. Betahistine on Meniere's symptoms

Betahistine is a drug that has pharmacological and structural properties similar to histamine. Betahistine is a heteroreceptor H3 antagonist and agonist H1 receiver that improves the microcirculation in the inner ear, promoting and facilitating central vestibular compensation [12]. The circulatory effects of betahistine have been demonstrated in laboratory animals and in humans. Betahistine increases the regional blood flow in patients with degenerative cerebrovascular disease and significantly improves cognitive function in the elderly [13].

Mira et al., in 2003 [14], compared the efficacy and safety of betahistine dihydrochloride to placebo in recurring dizziness resulting from Meniere's disease (MD) or benign paroxysmal positional vertigo (BPPV) of vascular origin. In this double-blind, parallel-group, multicenter, and randomized study, a group was treated with betahistine (MD: 34/BPPV: 41) and another with placebo (MD: 40/BPPV: 29). Betahistine had a significant effect on the intensity, frequency, and duration of vertigo's attacks compared with placebo, also with a better quality of life [14].

Lezius et al. [15] evaluated the clinical benefits and side effects of high doses of betahistine dihydrochloride (288–480 mg/day) in patients with severe Meniere's disease. In this series, 11 MD patients who have not responded well enough to 144 mg/day dosage of betahistina were treated individually with daily doses between 288 and 480 mg. As a result, the frequency and severity of dizziness were significantly reduced in all patients. The side effects were mild, self-limited, and do not require any changes in treatment strategy. Despite the considerable limitations of an observational study, high doses of betahistine between 288 and 480 mg/day appear to be effective in patients who are not sufficient to respond to lower doses. In addition, these doses are well tolerated.

In contrast, Adrion et al. [16] indicate that the incidence of attacks related to Meniere's disease did not differ among the three treatment groups ($P = 0.759$). Compared with placebo, crisis rates were 1.036 (95% confidence interval -1.140 to 0.942) and 1.012 (0.919–1.114) to a betahistine low-dose and high-dose betahistine, respectively. The global monthly attack rate dropped significantly by 0.758 factor (0.705–0.816; $P < 0.001$). Based on the monthly average incidence population on average over the period of evaluation was of 2.722 (1.304–6.309), 3.204 (1.345–7.929), and 3.258 (1.685–7.266) for the placebo groups, betahistine low-dose and high-dose betahistine, respectively. The results were consistent with no important side effects. The placebo effect could not be evaluated.

The Cochrane survey [17] reveals no conclusion evidence of betahistine use on Meniere's treatment despite the good results.

2.4. H1 receptor blockers and calcium antagonist

H1 receptor blockers and calcium antagonist, flunarizine and cinnarizine, inhibit vasoconstriction and act as sedatives vestibular, being used in the treatment of central and peripheral vertigo. Both are contraindicated in patients with extrapyramidal disorders [13], being useful for symptomatic treatment and during crises.

2.5. Benzodiazepines

Benzodiazepines act in a way that increases the inhibitory effect of gamma-aminobutyric acid in the vestibular nuclei and is useful in the therapy of vertigo, in the control of anxiety, and panic attacks in patients dizzying. One may experience drowsiness, fatigue, and drug dependence.

2.6. The Ginkgo Biloba (EGb 761)

The EGb 761's hemodynamic, hemorheological, metabolic, and neural effects are studied in Ref. [18]. It is used in the treatment of vertigo of peripheral origin [19].

Headache, hypotension, and gastrointestinal disturbance are the main adverse effects.

2.7. Intratympanic corticosteroid and Meniere's symptoms

The aim of these treatments is to use the medication that will affect the inner ear by entering the ear through the round window. Corticosteroids reduce the inflammation in the ear and

can increase labyrinth circulation and also there have been some suggestions that the steroids affect the metabolism of salt in the inner ear [20].

In a retrospective analysis by She et al. [21] patients with intractable Meniere's disease were treated with intratympanic methylprednisolone perfusion. These 16 patients were followed for more than 2 years. The vertigo control rates in the short- and long-term were 94 and 81%, respectively; the improvements of functional activities in the short-term were 94 and 88% in the long-term. The tonal average did not change significantly. In patients with intractable disease with good hearing preservation, intratympanic methylprednisolone can control vertigo and functional improvement, being a viable alternative for an intractable Meniere's disease.

A retrospective analysis performed by Boleas-Aguirre et al. states that control of dizziness was retrieved from 117 (91%) of 129 individuals and needed only 1 injection of dexamethasone 37%, 2 shots in 20%, 3 shots in 14%, and 4 injections in 8%; 21% needed more than 4 injections; 96 patients had follow-up data after 2 years. Of these, 91% had control of dizziness with intratympanic dexamethasone, and some needed more injections of dexamethasone or associated intratympanic gentamicin [22].

A review is published by the Cochrane in 2011 through randomized clinical trial of intratympanic dexamethasone versus placebo in patients with Meniere's disease. Only 22 patients were included. After 24 months of study, a statistically significant improvement in vertigo was confirmed compared to placebo. Change in score dizziness handicap inventory (60.4 against 41.3) and average improves subjective vertigo (90 versus 57%). The scheme of treatment described by the authors involves daily injections of dexamethasone 4 mg/ml solution for consecutive 5 days. These results were clinically significant. No complications were reported [23].

2.8. Intratympanic gentamicin injection

The first publication about the use of intratympanic aminoglycosides (streptomycin) on Meniere's disease in 1950 was of Schuknecht [24]. The aim of this therapy is to cause chemical damage or perform the ablation maze sick, in order to stop the floating labyrinth malfunction, causing the symptoms of Meniere's disease, and create a lasting situation of hypofunction where the brain can compensate for. This treatment can decrease the episodes of dizziness in Meniere's disease.

This chemical ablation of the labyrinth has some advantages over the classic surgical ablation (labyrinthectomy or vestibular nerve section), such as it can be performed on an out-patient basis under local anesthesia. Gentamicin is more vestibulotoxic than ototoxic, so it may be possible to preserve hearing. There is no consensus on the best dosing schedule to minimize hearing damage, but many authors argue that intermittent dosing with long intervals between two injections to check if hearing loss occurred is a safer approach in the maintenance of hearing [25].

The procedure is started after anesthesia of tympanic membrane topically with any phenol or EMLA® cream (2.5% lidocaine and 2.5% prilocaine). A small ventilation hole is made with a 25 gauge needle preferably earlier, and then the drug is injected postinferiorly until the middle ear space bottom is filled. The patient is then instructed to keep the ear where it was

injected the drug up to 20 min, to allow for absorption by the round window. In most studies, the application of gentamicin dose varies from 30 to 40 mg/mL (1 vial), applied between 6 weeks and 6 months, or 12 shots with a maximum of 360 mg, the following suggested ranges around 6–28 months, the American Academy of Otolaryngology Head and Neck Surgery (AAO-HNS) recommends a 2-year follow-up [26, 27].

Contraindications include active, middle ear infection only with hearing or ear balance function. The most commonly seen complications are hearing loss and unilateral vestibular hypofunction. Most of the patients need only an injection and most of these patients are able to avoid the ablative surgery due to significant improvement of treatment with intratympanic gentamicin.

The side effects of either intratympanic gentamicin or corticosteroids are minimal and a good result is achieved in about 90% with steroids at an early stage or gentamicin at a later stage, a very significant number when compared to the 30% success rate from placebo [28].

2.9. Endolymphatic sac surgery

Endolymphatic mastoid sac surgery (EMSS) has been known since the age of the Renaissance and remains a conservative popular procedure for the treatment of MD up to date [29]. Historically, vestibular neurectomy and transmastoid labyrinthectomy were provided to vestibular control, 90–95% of control rate. Vestibular neurectomy has a high potential for morbidity and neurosurgical complications. Hearing is eliminated in all cases of labyrinthectomy.

It is due to these possibilities of complications that today we opt for less destructive procedures to relieve vertigo.

In particular, EMSS appears to have regained popularity recently due to its low-risk safety profile, effectiveness in controlling episodes of vertigo, and in some cases improvement in hearing [30].

The procedure is initiated with the patient on general anesthesia and monitoring of the facial nerve. Antibiotics are administered and a standard mastoidectomy is performed followed by decompression of sigmoid sinus. Then the endolymphatic sac is identified behind the posterior semicircular canal, along the plate posterior fossa, below the Donaldson line. In a decompression surgery, bone about the sac is largely removed. A “stent” (Silastic® or Teflon®) is placed inside the sac that directs endolymph to mastoid cavity or to the cerebrospinal fluid compartment. The more common contraindications are active mastoid or middle ear disease. Among complications related to this surgical procedure mainly are hearing loss, dizziness, cerebrospinal fluid loss, damage to the sigmoid sinus, facial palsy, addition of anesthetics, and general surgical risks. Within the surgical technique terms, similar results are achieved with decompression of the endolymphatic sac against shunting maneuvers.

Samy et al. concluded in a retrospective chart review of 456 consecutive patients between 1997 and 2006, that EMSS did not significantly affect hearing outcomes at 2-year follow-up. Of all patients in the study, 60% had no clinically significant change in hearing, whereas 24% improved and 16% worsened. The distribution of posttreatment hearing changes between the medical and surgical groups was statistically insignificant ($P = 17$) [31].

The safety of EMSS surgery has also been established in elderly patients with Meniere's disease. In a Paparella et al. study with 62 patients (age ≥ 65 years) submitted to 78 EMSS surgeries without significant complications, 1.6% of major complications, mainly cardiac arrhythmia and 77% of patients achieved complete resolution of the symptoms for up to 2 years after the procedure.

2.10. Vestibular nerve section (VNS)

The great brain surgeon Walther Edward Dandy (1886–1946) of Baltimore described the vestibular nerve section (VNS) in 1928. Until 1941, he had already operated 401 cases with only one fatality [31]. There was renewed interest in vestibular nerve section after House introduced the middle-fossa vestibular neurectomy in 1961. Fisch and Glasscock and colleagues modified House's middle-fossa approach to include inferior vestibular neurectomy for improved control of vertigo. Silverstein and Norrell described the first retrolabyrinthine vestibular nerve section for Meniere's disease [32].

Patients with bilateral vestibular disease are not considered for a VNS because of the oscillopsia and permanent imbalance that can result from bilateral vestibular loss.

Vestibular nerve section can be performed through a retrosigmoid approach or retrolabyrinthine, associated with the monitoring of the facial nerve and auditory evoked potential. In the retrosigmoid approach, lower and upper vestibular nerves are identified and divided in internal acoustic pores not taking care to injure the facial nerve or cochlear. The identification of the vestibular nerves can be facilitated by the decompression internal auditory canal laterally, in order to locate definitely references such as the horizontal and vertical ridges (Bill) and the unique nerve after semicircular canal ampulla.

Already in the retrolabyrinthine approach, a mastoidectomy is performed, with decompression of sigmoid sinus and posterior semicircular canal and the vertical segment of the facial nerve. The dura mater of the posterior fossa is then identified and the internal auditory meatus is decompressed to view individual nerves. The vestibular nerve and the facial cochlear are identified and the vestibular nerve is carefully cut with subsequent placement of fat graft inside the mastoidea cavity to prevent loss of CSF (cerebrospinal fluid). Central compensation after vestibular neurectomy is key for postoperative recovery of balance. This makes any indication of central nervous system diseases such as cerebellar dysfunction, multiple sclerosis, physiologic old age, and poor medical condition a relative contraindication for vestibular nerve section.

Vestibular nerve sectioning is one of the most effective procedures for treating intractable vertigo in patients with no hearing in a unilateral Meniere's disease ear. In the literature [33–37], vertigo control rates between 78 and over 90% have been reported.

The contraindications are rare for this type of surgery, complications can exemplify: loss of CSF, meningitis, cranial Neuropathies, seizures, stroke, and death, in addition to general surgical and anesthetic risks. One of the major setbacks of the postoperative period is the need for hospitalization of the patient around 2–5 days. Effective medical treatment and dietary control, combined with intermittent use of oral steroids and middle ear perfusion of steroids

or gentamicin has substantially reduced the number of patients with intractable vertigo needing vestibular neurectomy.

2.11. Labyrinthectomy

The goal of the surgery is the removal of vestibular end organs of five neuroepithelium: the three semicircular canals, the utricle, and saccule. In patients with severe hearing loss who do not respond to other surgical and medical treatments, labyrinthectomy is typically the last choice for unilateral MD. Bilateral MD is a contraindication for this procedure, because of the oscillopsia and permanent imbalance that can result from bilateral vestibular loss, as for VNS.

Several studies have shown excellent control of vertigo in up to 97% of patients. There is a 3% risk of CSF leak and a 2% risk of facial nerve injury [38].

Classically indicated when the audiometry shows loss greater than 60 dB and discrimination less than 50%.

The patient is placed under general anesthesia and monitoring of facial nerve after antibiotic administration of a standard mastoidectomy is performed. The tegmen tympani, sigmoid sinus, horizontal channel, and facial nerves are all identified. Gain access to the horizontal semicircular canal, followed thereafter by posterior semicircular canal, which in turn is followed to the raw columna to identify the upper channel of the semicircular canals to the lobby, and finally removes the saccule and utricle, after removal of all neuroepithelium, the surgical wound is closed and a bandage on the mastoid is applied.

It is normal to find the post or horizontal nystagmus and lateral superior oblique (LSO), a slope deviation can be occasionally observed due to acute interruption of the utricular unilateral entry. Complications include dizziness, loss of CSF, sigmoid sinus damage, facial paralysis, in addition to the anesthetic and surgical risks.

Often the vestibular therapy is useful in the postoperative period to assist the central compensation and the return to functionality.

Nevertheless, literature research concerning cases of labyrinthectomy and cochlear implantation in patients suffering Meniere's disease is limited and is being performed in cases of bilateral Meniere's disease or even 20 years after labyrinthectomy. Zwolan et al. performed to our knowledge the first simultaneous labyrinthectomy and cochlear implantation in a single patient [39]. As the results show, the combination of labyrinthectomy and cochlear implantation is an efficient concept for the treatment of patients with Meniere's disease and single-sided deafness in case where the above preconditions have been implemented. An excellent control of vertigo symptoms could be achieved using this therapeutic concept.

For patients with single-sided Meniere's disease and profound sensorineural hearing loss, a simultaneous labyrinthectomy and cochlear implantation are efficient method for the treatment of vertigo and rehabilitation of the auditory system [40].

3. Meniett device

During the decade of 1970, the demand for a more effective and a nondestructive method for the treatment of MD, Inglestad et al. [41] observed that some patients reported improvement with changes of pressure in a pressure chamber. Densert et al. [42] showed that the manipulation of the middle ear pressure influences the pressure in the inner ear; later, improved hearing and dizziness in patients with MD were described after the application of positive pressure in the middle ear.

Additionally, there was improvement of the cochlear electrical potentials after administration of positive pressure in the middle ear, which finally led to the development of the device known as the Meniett (Medtronic Xomed Surgical Products, Jacksonville, FL).

The Meniett device emits a pulse of repeated pressure of 0.6 second in the range of 0–20 cm H₂O at 6 Hz. Treatment consists of three to four cycles of a sequence of treatment of 5 min, this device requires only a short-term ventilation tube (Sheppard) to allow the transmission of impulses in an auditory external pressure to the middle ear.

The FDA approved the use of the device in 2002, demand is still low, despite recent work showing the effectiveness of the device. Meniett reduces the frequency of dizziness in patients with Meniere's disease activity, an improvement of AAO-HNS Meniere's disease functional level scale, but the device does not significantly improve hearing, showing a safe option for patients with refractory diseases to the conventional treatments [43].

4. Vestibular rehabilitation

Vestibular rehabilitation is a form of body and eye movement stimulation therapy designed to improve vestibular function and mechanisms of central adaptation and compensation. It is mainly useful to treat the MD squeal; vestibular adaptation exercises to prevent falls have proven to be particularly effective. This type of treatment is quite effective for patients with stabilized vestibular function [44].

5. Conclusion

Treatments in Meniere's disease are generally aimed at reducing the acute symptomology vertiginous episodes. The cure currently does not exist. To date, the treatment has convincingly been shown to be effective in altering the natural course of the disease, thereby preventing end organ damage, which results in hearing loss and vestibular impairment. The clinical spectrum of the disease of Meniere's symptoms is wide, with the possibility of remission and recurrence of symptoms, and even fairly unfavorable developments, with irreversible hearing loss and damage the vestibular function. A good treatment must be individualized for each patient, taking into consideration these possibilities, the stage of disease, and the possible consequences that have been promoted.

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