

Optimal management of Ménière's disease

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Abstract: Confusion in the nomenclature of Ménière's disease and lack of a standard definition of the disorder until 1995 has hampered accurate assessment of treatment efficacy since the presently defined disorder was first described in 1938. The lack of a widely accepted mechanism of the disease has also delayed the development of rational treatments. Past treatments have focused on relieving elevated pressures in the hydropic ear and more recently on treatment of underlying migraine. Present dietary methods of control include sodium restriction and migraine trigger elimination. Pharmacologic treatments include diuretics, migraine prophylactic medications, histamine analogs, and oral steroids. Surgical procedures include intratympanic steroid perfusion, shunts, and ablative procedures when conservative treatments fail. External pressure devices are also used. Evidence of efficacy is lacking for most interventions other than ablation.

At our institution, Ménière's disease is treated as a cerebrovascular disorder. Control of risk factors for cerebrovascular ischemia is combined with treatment of pressure dysfunction in the hydropic ear. Screening for risk factors is performed at presentation. Migraine, dyslipidemia, obesity, diabetes, sleep apnea, hypertension, and atherosclerosis are among the major factors that often require medical management. Migraine prophylactic medications, magnesium supplementation, sodium restriction, migraine trigger elimination, diuretics, anticoagulants, and antihypertensives are among the treatments used initially. Steroids administered orally or intratympanically are used if control is not achieved medically, and ablation remains the definitive treatment in unilateral cases experiencing treatment failure.

Keywords: endolymphatic hydrops, migraine, cerebrovascular disorders, ischemia

Introduction

Progress in controlling the symptoms of Ménière's disease has been frustratingly slow. For the first few decades after Meniere identified the ear as the source of vertigo spells in 1861, his name was applied indiscriminately to a host of chronic dizziness disorders that lacked a common pathology. It was not until the 1938 description of the disorder by Hallpike and Cairns¹ that the definition of the disease began to approach its present form, and only since the 1995 American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) criteria² has a clear, unambiguous, and sufficiently narrow definition of the disorder been available to researchers. This confusion in nomenclature means that many articles purporting to treat Ménière's disease over the past 75 years have included patients with unrelated disorders, interfering with proper assessment of treatment efficacy.

A second factor that has impaired treatment progress is the lack of consensus on a mechanism for the disorder. There have been two major opposing factions since the Hallpike and Cairns paper was published.¹ One broad group believes that endolymphatic hydrops and Ménière's disease are synonymous and that treatments directed at hydrops should be sufficient to control symptoms.³ Within this "hydrops group", it is currently believed that the onset of hydrops is probably multifactorial,

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including viral infection, autoimmunity, and other disorders as causes. Since the 1950s, the spells have been ascribed to sudden silencing of the inner ear due to contamination of the perilymph with endolymph after rupture of the membrane,³ although this mechanism has been seriously questioned over the past few decades.⁴ There is no longer a clear consensus on the mechanism by which hydrops results in attacks in this group, but treatments continue to focus on decompressing the hydropic ear either through medications or by surgical intervention.

The other major group, the “migraine group” believed early on that migraine explains many cases of the disorder, perhaps through the mechanism of ischemia due to vasospasm.^{5,6} Some researchers have rejected hydrops as causal in the disorder.⁷ Genetic factors or the presence of an ion channelopathy have been invoked to explain the association.⁸ Treatment strategies involve using medications to prevent migraine-associated vasospasm or neurosensory disturbances.⁹

Both sides have developed a considerable arsenal of papers supporting their positions. In the absence of consensus, treatments for Ménière’s disease have been based on empirical success with patients rather than on a formal pathologic rationale. At the University of Colorado, we combine treatments from both groups along with additional treatments because we believe that Ménière’s disease results from a combination of hydrops with a variety of risk factors for cerebrovascular disease, including maladies as disparate as migraine, sleep apnea, atherosclerosis, and autoimmune disorders.^{10,11} We have hypothesized that hydrops acts as an intermittent Starling resistor to lower perfusion pressure in the inner ear, which is additive with other causes of lowered perfusion such as cerebrovascular diseases. In our theory, progressive damage is attributed to calyceal and dendritic ischemia–reperfusion injury (excitotoxicity). The treatments we advocate are directed at mitigating the vascular risk factors that trigger spells and also at lowering pressure fluctuations in the affected ear.

The current AAOHNS definition of the disorder describes a very specific type of hours-long attack, during which the sufferer experiences acute hearing loss, roaring tinnitus, a sensation of fullness in the ear, and intense vertigo with the illusion of environmental rotation. Over time, a progressive hearing loss develops in the affected ear. No treatment has thus far been shown to stop the inevitable progression to deafness in the afflicted ear. Vertigo spells can be controlled with high efficacy, but the favored means are destructive: gentamicin ablation, vestibular neurectomy,

and labyrinthectomy. The rationale for these treatments is to functionally disconnect the labyrinth, relieving the most intrusive symptom of vertigo, after which the ear continues to follow its progressive downhill audiologic course. Destructive procedures are not usually used in bilateral cases because this results in the disabling symptom of oscillopsia.

Given the destructive nature of the most effective procedures, treating physicians usually prefer to temporize for months or years after a case presents itself. Delaying destructive treatments allows time to ascertain that only one ear is involved; it provides time for interim treatments that, if successful, may render destruction unnecessary, and it allows repeated testing to ensure that the ear is inevitably declining. This review will focus on these interim treatments.

Dietary methods

Sodium restriction

This has been widely used to control symptoms of Ménière’s disease since 1931.¹² Reduction of dietary sodium combined with diuretics has been reported to slow progression of hearing loss in Ménière’s disease.¹³ Some advocate moderate regulation of sodium intake rather than harsh restriction.⁹ The literature lacks controlled studies of this therapy based on modern Ménière’s disease definitions.

Therapeutic rationale

Members of the hydrops group were early advocates of this treatment because it was thought to lower pressure in the hydropic ear, reducing the risk of membrane rupture. Sodium restriction has not been advocated as a migraine treatment, but reduction of monosodium glutamate intake as a part of sodium restriction maybe helpful in migraine because glutamate is a known trigger for migraine.¹⁴ A low-sodium diet is also useful in controlling hypertension, a vascular risk factor.¹⁵

Migraine trigger elimination

Common migraine food triggers include monosodium glutamate, chocolate, red wine, fermented dairy products including yogurt, and aged or pickled foods.¹⁶ Trigger elimination is advocated as treatment for migraine-associated vestibular disorders,¹⁷ but controlled studies demonstrating the efficacy of this treatment in Ménière’s disease are lacking.

Therapeutic rationale

The migraine group recommends avoidance of these foods with the belief that Ménière’s disease will fluctuate and

worsen if migraines are uncontrolled.⁹ There is no expectation that these triggers modify hydrops, but they are often included in dietary regimens for hydrops control based on their empiric efficacy.¹⁸ Our protocol is to recommend avoidance of these food triggers in patients with migraine headaches, aura, or in Ménière's patients under the age of 50 years because migraine is the most common vascular risk factor in that group.

Pharmacologic methods

Diuretics

A combination of hydrochlorothiazide and triamterene is the most commonly used medical treatment for Ménière's disease. Acetazolamide, furosemide, and spironolactone are also used by some. Combining these with sodium restriction has slowed progression of hearing loss.¹³ A recent Cochrane review regarding diuretics in Ménière's disease noted that no articles supporting diuretic use that meet accepted review standards are presently available in the literature.¹⁹

Therapeutic rationale

Like the sodium-restricted diet described earlier, diuretics have been used in the belief that pressures in the hydropic ear may be reduced by the drug. Acetazolamide is also used in the treatment of migraine with aura, so it has been used by both the hydrops and migraine groups. In our center, we use hydrochlorothiazide with triamterene in Ménière's disease patients without migraine, particularly for those with hypertension and atherosclerosis. Acetazolamide is used for those who have migraine with aura or as a second-line agent in patients with migraine who do not respond to other migraine prophylactic medications. There is a risk for renal calculi with this medication, so patients are advised to increase fluid and citrus intake.

Migraine prophylactic medications

Amitriptyline, beta blockers, calcium channel blockers, acetazolamide, and topiramate are commonly used in the prevention of migraine headaches¹⁴ and are also used for vestibular symptoms in migraineurs.¹⁷ Migraine prophylactic medications are used by some centers for Ménière's disease but controlled studies are lacking.

Therapeutic rationale

Migraine and hydrops have not been shown to be causally related, so migraine prophylactic medications are generally used only by the migraine group with the belief that vasospasm and other migraine phenomena can be controlled by

these drugs. Calcium channel blockers such as verapamil, and in Europe, flunarizine, are effective in migraineurs.²⁰ The carbonic anhydrase inhibitors have some diuretic effects and so may impact hydrops directly in addition to treating migraine. The blockers are also used in the treatment of hypertension and so may be of use in Ménière's patients who also have migraine and hypertension.

Histamine and related medications

Histamine was first advocated over 50 years ago by the migraine group because the intense facial flushing it causes was hoped to reflect increased perfusion to the ear. This has not stood the scrutiny of time. Niacin was also used for a similar reason, and like histamine it has faded from use.

Betahistine is a modern analog, although the mechanism of action has been attributed to central vestibular effects as well as to vascular effects. Recent work suggests that it has positive effects on cochlear blood flow.²¹ A 2001 Cochrane review has found no definitive evidence of efficacy in the treatment of the vertigo spells, and no effect on hearing or tinnitus.²² It continues to be a popular drug in Europe and Canada, but is not approved in the United States based on lack of efficacy.

Oral steroids

Oral steroids have been used for treatment with the belief that inflammation or immunologic factors or a direct effect on the vestibular nucleus may reduce the severity of spells.²³ This study using 18 months of steroid treatment reported a 50% improvement in vertigo spell frequency.

Intratympanic perfusion Steroids

Intratympanic steroids have been noted to have a calming effect on Ménière's ears.²⁴ The number and severity of spells seem to decrease for up to 2 years after use.²⁵

Therapeutic rationale

Steroids are not useful in migraine and there is no plausible mechanism for alteration of hydropic distention by steroids. Improvement in Ménière's disease has been hypothesized to occur when hydrops is due to inflammation or autoimmune factors.²⁴ Steroids are used to reduce edema after ischemic stroke,²⁶ so steroid-responsive Ménière's disease also supports ischemia as a mechanism of attacks. Steroids are also used successfully to treat sudden sensorineural hearing loss,²⁷ with improvement attributed to a reduction in inflammation and improvement in blood flow to the cochlea.²⁸

Surgery directed at hydrops Surgeries to relieve distention of the endolymphatic system

Endolymphatic shunts of various kinds, cochleosacculotomy, and endolymphatic sac decompressions have been used for many decades. Improvement in vertigo control has been achieved in about two-thirds of treated patients.²⁹ This is similar to the improvement seen in nonsurgically treated cases over time.³⁰ None have been shown to be as effective as destructive procedures.³¹ The possibility that any improvement found in prior studies is due to the placebo effect has been repeatedly raised.³² On autopsy studies, shunts have been found to be malpositioned or occluded, suggesting that any impact might be temporary.³³

Shunt procedures are still popular and are the major non-destructive surgical treatment for the disorder. Over 80% of otologists use shunts for medically refractory Ménière's disease. They are appealing for patients with bilateral Ménière's because ablative procedures cannot be used in that group. There is no evidence that shunts alter the natural history of the disease.³⁰

Therapeutic rationale

The migraine group does not advocate these surgeries because they believe hydrops is not a critical factor in causation of attacks. The hydrops group, however, has long felt that elevated endolymph pressures cause the distention of hydrops, and so have devised these treatments to shunt endolymph and relieve this pressure. At the University of Colorado, shunts are performed infrequently, more often in those with bilateral disease, and when control cannot be achieved with treatment of vascular risk factors. Complete, lifelong control of vertigo is rarely achieved by shunting.

Devices

External pressure device (Menniott)

While a trend toward improvement in vertigo has been identified with this device,^{34,35} improvement also occurs in the natural history of the disease.³⁰ A long-term follow-up showed 75% improvement in frequency and severity of spells,³⁶ which is similar to the improvement in untreated patients. This is a relatively expensive device, and a cost-benefit analysis is lacking.

Therapeutic rationale

This device is advocated by the hydrops group and the purported mechanism of action is either transmission of pulsed pressure to the endolymph space, presumably relieving hydrops by massaging fluid out of the endolymph space, or another as yet unknown mechanism.³⁵

Suppressant medications

Benzodiazepines, meclizine, other antihistamines such as promethazine or diphenhydramine, and transdermal scopolamine patches have all been used to control the sensation of vertigo during acute attacks. These medications have no effect on the progression of the disorder. However, they are useful in controlling vegetative symptoms during attacks and can help patients during the interval before ablative surgery is contemplated.³⁷ Transdermal scopolamine patches require 4 hours to reach a therapeutic blood level of scopolamine and continue to release drug for 72 hours,³⁸ which is a poor match for typical Ménière's attacks of sudden onset and duration of a few hours. Meclizine is often useful to reduce nausea for milder spells, oral benzodiazepines are well tolerated for more severe spells, and promethazine suppositories can be used for the most severe spells with intractable vomiting.

The University of Colorado protocol

The cornerstone of our treatment protocol is the identification and control of disorders that impair cerebral and inner ear vascular perfusion. Patients with a history of classic Ménière's attacks are screened for a number of common vascular disorders (Table 1). Initial screening is by history and examination. All patients with this diagnosis undergo magnetic resonance imaging of the internal auditory canals as part of the screening process.

Migraine is closely associated with Ménière's attacks in adults under the age of 50 years. Serum testing is only done in these younger patients when there is no personal or family history of migraine or when other vascular risk factors are evident in the history or on examination. All obese patients and those over the age of 50 years receive serum screening consisting of a standard metabolic panel (glucose, blood urea nitrogen, creatinine, electrolytes), complete blood count, erythrocyte sedimentation rate, C-reactive protein, lipid screening (cholesterol, lipoprotein profile, triglycerides), and coagulation screening (platelets, prothrombin time, activated partial thromboplastin time, fibrinogen). Additional selected tests may be performed on patients with more complex disorders, such as autoimmune disease.

Treatments are tailored to the underlying vascular risk factor (Table 2). Migraine patients and all patients under the age of 50 years without other known vascular disorders are managed with migraine trigger elimination and daily magnesium supplements. Those who continue to have attacks are given migraine prophylactic medications. All patients over the age of 50 years are given diuretics, daily low-dose aspirin (80 mg), and magnesium supplementation. Modest

Table 1 Cerebrovascular risk factor screening in Ménière's patients

Risk factor by history	Serum testing	Other tests
Migraine		Magnetic resonance imaging
Dyslipidemia	Lipoproteins Triglycerides Total cholesterol	
Obesity	Lipoproteins Triglycerides Total cholesterol	
Hypertension	Electrolytes, BUN, creatinine	
Smoking	Lipoproteins Triglycerides Total cholesterol	
Diabetes	Fasting glucose Hemoglobin A1C Lipoproteins Triglycerides Total cholesterol	
Sleep apnea	C-reactive protein Fasting glucose Lipoproteins Triglycerides Total cholesterol	Polysomnography
Myocardial infarction, stroke	C-reactive protein	Magnetic resonance imaging
Transient ischemic attacks	Lipoproteins	Electrocardiography
Known atherosclerotic disease	Triglycerides Total cholesterol	
Autoimmune disease	ESR C-reactive protein	
Coagulopathies	CBC, PT, APTT Lupus anticoagulants (selected patients)	

Abbreviations: BUN, blood urea nitrogen; ESR, erythrocyte sedimentation rate; CBC, complete blood count; PT, prothrombin time; APTT, partial thromboplastin time.

Table 2 Treatment matrix for vascular risk factors

Risk factor	Medication	Other treatments
Migraine	Tricyclics Calcium channel blockers Carbonic anhydrase inhibitors Topiramate Acetazolamide	Migraine trigger elimination Magnesium supplements Refer treatment failures to neurology
Dyslipidemia	Statins Low-dose aspirin Diuretic	Nutritional counseling
Obesity	Low-dose aspirin	Nutritional counseling
Hypertension	Low-dose aspirin antihypertensives Diuretics Beta blockers Calcium channel blockers	Coordinate management with internist
Smoking	Low-dose aspirin Diuretic Statins	Smoking cessation program
Diabetes	Low-dose aspirin Diuretic Statins	Referral to endocrinology
Sleep apnea	Low-dose aspirin Diuretic	CPAP or surgical treatments
Myocardial infarction, stroke	Low-dose aspirin	Coordinate management with internist
Transient ischemic attacks	Diuretic	
Known atherosclerotic disease	Statins	
Autoimmune disease	Transtympanic or oral steroids Diuretic	Referral to rheumatology
Coagulopathies	Low-dose aspirin Diuretic	Referral to hematology

Abbreviation: CPAP, continuous positive airway pressure.

sodium restriction (2,000 mg/day) is also recommended. Each identified vascular risk factor is treated either in our clinic or by coordination with the patient's other care providers. Transtympanic or oral steroids are used after initial treatment failure in all groups. Four or more classic unilateral attacks during a year or more of treatment is considered inadequate control, and these patients are usually referred for ablative procedures. Because bilateral ablation is disabling, bilateral patients who fail conservative treatment may be offered other options such as shunting and pressure devices.

Disclosure

The author has no conflict of interest to report regarding this work.

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