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Meniere's Disease is a Manifestation of Migraine

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Abstract

Purpose of review—To discuss the theory that Meniere's disease (MD) is a variation of otologic migraine rather than an isolated inner ear condition.

Recent findings—In contrast to the approximately 12% of the general population suffering from migraine headaches, 51–60% of patients with MD experience migraine headaches. While pathognomonic for MD, endolymphatic hydrops has also been identified in patients with vestibular migraine. Treatment with the integrative neurosensory rehabilitation approach (diet and lifestyle changes, magnesium and riboflavin supplementation, and when needed, prophylactic medication) to treat the underlying migraine process has been highly effective in patients with MD.

Summary—MD can be understood as a manifestation of migraine such that patients with MD can be effectively treated with migraine therapies.

Keywords

Meniere's disease; migraine; otologic migraine; endolymphatic hydrops; integrative neurosensory rehabilitation

INTRODUCTION

Meniere's disease (MD) was first described by Prosper Meniere in 1861 through his publication of a series of patients with episodic hearing loss, vertigo, and tinnitus that identified an inner ear mechanism. At that time, these symptoms were understood to be of cerebral origin and Meniere's new theory of an internal ear origin was the center of heavy debate. Meniere himself recorded the frequent presentation of headaches in his cohort of patients.(1) Meniere did not disregard the consistent association of headache and his identified clinical triad as coincidence but rather considered a common etiology. Among his original papers, he stated "… it is not less certain that cerebral states, called migraine, give place in the end to similar attacks, and the deafness which arises in these circumstances would seem to us inevitably to be related to a disease of the same nature."(2)

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In more recent decades, MD is diagnosed clinically using the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) criteria as follows: two or more spontaneous vertigo attacks for 20 minutes to 12 hours, fluctuating low to mid frequency sensorineural hearing loss of affected ear that is audiometrically documented, fluctuating aural symptoms including tinnitus, or fullness.(3) For patients that do not meet criteria, patients may be diagnosed with probable MD. This entity is defined as at least two vertigo or dizziness attacks lasting 20 minutes to 24 hours and fluctuating aural symptoms. MD is typically unilateral with both vertigo and hearing loss isolated to one side. Symptoms may go into remission for years therefore it can take multiple years to establish a diagnosis. MD remains a clinical diagnosis with no highly accurate tests for the disorder.

Vestibular migraine (VM) is among the differential for a patient presenting with episodic vertigo. The combination of vertigo in association with migraine has been recognized since the 19th century with the term Vestibular Migraine coined in 1999.(4) The diagnostic criteria are reported independently from MD by the Barany Society and International Headache Society. To meet these criteria, a patient will have at least 5 episodes of moderate to severe vestibular symptoms lasting 5 mins to 72 hours, current or previous history of migraine, and one or more migraine features co-occurring with at least half of the vestibular episodes. (5) Migraine features include visual aura, photophobia, phonophobia, headache with one sided location, pulsating, moderate to severe pain, and aggravation by routine physical activity. In patients with fluctuating hearing loss, differential diagnoses can include cochlear migraine (CM). This condition was first proposed in 2018 following recognition of a cohort of fluctuating hearing loss and migraine.(6, 7) Proposed major diagnostic criteria included recurrent or fluctuating unilateral sensorineural hearing loss, aural fullness of affected ear, and without vertigo therefore not meeting criteria for VM or MD.

In this review of recent literature, we shall discuss our hypothesis that the above conditions of MD, VM, and CM are not distinct but rather variations of the singular entity that is otologic migraine and part of a spectrum of disorders related to central sensitivity.

EPIDEMIOLOGY

The prevalence of MD in the general population is 0.2%(8) while VM has been reported at 2.7%.(9) Studies investigating prevalence of MD have noted difficulty in distinguishing between MD and VM.(8) In contrast to the approximately 12% of the general population suffering from migraine headaches, 51–60% of patients with MD experience migraine headaches.(10, 11). Auditory symptoms- that often differentiate MD from VM- have been reported in 61% of patients with VM.(12) Additionally, the incidence of cochlear disorders was found to be significantly higher among patients with migraine compared to those without (12.2% *vs.* 5.5%) with an adjusted hazard ratio of 2.71 (95% CI: 1.86–3.93).(13) Independent associations have been found between migraine and subjective hearing loss (25% *vs.* 16.6%, $p<0.001$; OR=1.2, 95% CI 1.3–1.7) or tinnitus (34.6% *vs.* 16.9%, $p<0.001$; OR= 2.1, 95% CI 1.9–2.3).(14)

There are many reports of comorbid MD and VM. After identifying 10 patients who met a combination of MD and VM criteria for definite diagnosis, Murofushi *et al.* proposed the

VM/MD overlapping syndrome.(15) In a study of 190 subjects with MD, 20.9% experienced migraine and 42.9% experienced headaches.(16) A propensity score match study found migraine and non-migraine headache to be associated with MD with odds ratios of 2.5 and 1.7 respectively.(17) A retrospective study of 600 patients with chief complaint of dizziness, found 21 patients met criteria for both MD and VM. MD symptoms were present in VM patients with 38% reporting episodic hearing loss, aural pressure, and tinnitus.(18) Additionally, indicators of migraine were found in nearly half of the patients meeting criteria for MD alone that did not meet full migraine criteria. When detailed questionnaires or interviews are used, migraine co-occurrence with MD can be found at much higher rates than utilizing diagnostic codes.

In a study consisting of two independent longitudinal population database studies with a total population of over 142,000 participants, Kim *et al.* described a reciprocal association between MD and VM.(19) Presence of both migraine and MD were higher in patients with either diagnosis than in the general population. Of the 8.6% of total subjects with a history of migraine, 5% had a history of MD. There was a history of MD in 1.8% of included subjects, of which 24.7% had a history of migraine. MD was found to have a 2.22-fold greater hazard ratio of for migraine with an E value as high as 3.31. Additionally, migraine was found to have a 1.95-fold greater hazard ratio for MD.

When CM and VM occur together as otologic migraine, the presentation is that of MD.(20, 21) (Figure 1) Overlap or comorbidity of VM, CM, and MD is therefore accounted for under our theory that MD is a manifestation of migraine. Patients may not display all symptoms at initial presentation. Vestibular or auditory symptoms may occur first with additional symptoms developing later. A longitudinal study of VM patients found 18% of patients developed hearing loss and presence cochlear symptoms increased from 15% to 49%.(22) Diagnosis of VM has been found to significantly decrease with age (p=0.035).(23) While patients are more likely to be diagnosed with VM under the age of 40(9), MD onset is reported typically between ages 40–70(8). In a study aimed at characterizing vestibular symptoms in patients with VM and MD, it was found that patients with MD have a longer history of symptoms than patients with VM.(24) Patients who seek care for symptoms of vertigo before the age of 40 may not have developed symptoms of hearing loss that would direct the diagnosis to MD over VM.

MECHANISM OF MIGRAINE

The physiology of migraine is understood to be due to activation and sensitization of the trigeminal vascular system as a result of spreading cortical depression.(25–27) Neurotransmitters including substance P, neurokinin A, and calcitonin gene related peptide are released by perivascular nerve endings leading to vasoconstriction and inflammation. Subsequent to the vasoconstriction which causes aura symptoms (e.g., scintillating scotomas, roaring tinnitus, etc.), prolonged vasodilation may occur which leads to many of the symptoms of migraine (e.g., headache, vertigo, hearing loss). The headache of migraine is due to the activation of nociceptors by the trigeminal system. Disturbances of the cochleovestibular neurovasculature as a result of spreading cortical depression induces the triad of symptoms of MD.(28) The ophthalmic branch of the trigeminal nerve (V1) has been

found to be intimately involved with the posterior cerebral circulation. The cochleovestibular vasculature including the stria vascularis have also been found to be heavily innervated by V1.(29) (Figure 2) When V1 has been stimulated electrically in an animal model, fluid extravasation within the cochlea has been observed within 60 minutes of the V1 stimulation.(30) Vestibulocochlear symptoms are secondary to a combination of changes of blood flow and actions of diffused neuromodulators. Chronic deficits in perfusion of the inner ear secondary to vasospasm and/or vasodilation of the spiral modiolar artery can ultimately lead to endolymphatic hydrops (ELH).(10) We believe that based on the animal studies, primary cochleovestibular vasodilation leads to ELH from fluid extravasation. This initial ELH and repeated vasospasm/vasodilation damaging the endolymphatic fluid homeostasis causes permanent ELH.

ENDOLYMPHATIC HYDROPS

While not included in the diagnostic criteria, the presence of histologic ELH is highly associated with MD.(31) ELH is an enlargement of endolymphatic compartment leading to distention of the structures surrounding the scala media as well as the utricle and saccule. Variation in mechanical compliance of the endolymphatic component membranes has been proposed to explain differences in the extent of distension.(32) Symptoms of MD have been thought to occur secondary to ELH as the distension of the endolymphatic duct predisposes the ear to acute changes in pressures. This has been thought to lead to tears in Reissner's membrane which can cause the potassium rich endolymph to periodically enter the perilymphatic space. ELH can be congenital or acquired while most are deemed idiopathic. While nearly all ears affected with MD have ELH, not all ears with ELH will produce symptoms of MD. Postmortem histopathology revealed ELH in all 13 patients with a history of MD. Notably, 6 of 106 patients with no history of vestibular symptoms were found to have hydrops. Similarly, temporal bone analysis found ELH in all included 28 MD cases and 9 without vertigo were identified to have ELH.(33) Why some patients with ELH develop the symptoms of MD is unknown. Vasoconstriction, vasodilation, or neurogenic inflammation as seen in migraine headaches have been proposed as theories to explain the development of ELH and as the cause of symptoms in MD.(10) ELH in MD is likely a result rather than the cause of cochlear homeostasis dysfunction. The dysfunctional homeostasis in the cochlea is likely caused by damage to the cochlea from repeated vasoconstriction/vasodilation attacks from migraine (trigeminal nerve (V1) stimulation).

While ELH has been thought to be pathognomonic for MD, it is known that not all ears with ELH produce MD symptoms.(31) It is unsurprising that ELH has been found in patients diagnosed with VM without meeting MD criteria. ELH may be temporary in some patients. One patient with VM was reported to have fluctuating ELH on delayed gadolinium-enhanced magnetic resonance imaging of the inner ear (iMRI).(34) The specific sequence used was a cisternographic three-dimensional T2 and delayed intravenous-enhanced three-dimensional fluid-attenuation inversion recovery (DIVE-3D-FLAIR) sequence. Another study utilizing iMRI findings reported ELH in 2 of 25 patients with VM and 2 of 8 patients meeting criteria for both VM and MD.(35) Multiple patients with VM have been found to have ELH as determined by ELH presumption testing.(36) These tests include Futaki's method and furosemide loading vestibular evoked myogenic potential (VEMP). Futaki *et*

al.'s method is positive when the maximum slow-phase velocity of eye movement increases by more than 9.4% 40 minutes after an intravenous injection of 20 mg furosemide.(37) The test has been shown to be positive in 80% of MD patients. In patients with VM, 35.7% of patients had a positive response. Furosemide loading VEMP compares cervical-VEMP 60 minutes after injection of 20 mg furosemide. Positivity is determined by an amplitude increase greater than 14.2% or the development of a biphasic wave. ELH was present per this test in 70.6% of patients with MD and 50% of VM patients.(36) The presence of ELH has been thought to be indicated by electrocochleography (EcochG) and calculation of cochlear summating potential and auditory nerve action potential (SP/AP) ratio greater than 0.5.(38, 39) Recent studies have shown 12 of 30 VM cases with episodic audiologic dysfunction positive for ELH, 3 of 21 cases of VM with no audiologic component positive for ELH, and 8 of 21 VM patients with unspecified cochlear symptoms.(38–40)

Anatomical variations in ears of patients diagnosed with MD or VM may represent possible predisposing factors in the development of ELH. The distance between the posterior semicircular canal and posterior fossa was found to be shorter and visibility of the vestibular aqueduct was poorer in patients with MD than VM.(41) The short distance between the posterior semicircular canal and posterior fossa has been thought to be the cause of a small or poorly functioning endolymphatic sac. Of note, these findings have a low diagnostic accuracy to differentiate between VM or MD. It is possible that those with VM and these anatomic differences are more likely to develop ELH in association with MD symptoms of hearing loss and tinnitus.

CLINICAL FEATURES AND TESTING

Efforts have been made to differentiate MD and VM via clinical testing as symptom profile is so similar in these two conditions.(42) The role of many of these tests is to isolate a peripheral or central source of vestibular dysfunction. For example, vertical nystagmus suggests a central origin compared to horizontal. Both patients with VM and MD can have horizontal or vertical nystagmus(42). The head shaking test and subsequent head shaking nystagmus was positive in 68–78% of MD patients and 50% of VM.(43, 44) Caloric testing abnormalities have been detected in both MD and VM.(45–47) Both ocular and cervical VEMPs can have abnormalities including increased amplitudes in patients with MD and VM.(48, 49) So far, no clinical testing has been able to distinguish VM from MD, likely because the two conditions are along a spectrum of the same origin and not two distinct disorders.

TREATMENT

Patients diagnosed with MD respond well to migraine treatment (50). A cohort of 25 patients with definite MD who did not qualify for the VM diagnostic criteria and failed diuretic therapy were treated with migraine prophylactic therapy incorporating lifestyle modification and pharmaceuticals (integrative neurosensory rehabilitation regimen).With a minimum of 18 months of follow up, quality of life was improved in 23 (92%) of the respondents in every metric measured, unchanged in 1 (4%), and poorer in 1 (4%) of 25 patients after migraine prophylaxis treatment. On average, the patients experienced

8.3 attacks per month prior to treatment compared to 0.8 vertigo attacks per month post treatment.(50) An additional case report of a pediatric patient with definitive MD was treated with migraine lifestyle modifications, riboflavin, and magnesium had significant improvement in hearing and had resolution of vertigo.(51)

The integrative neurosensory rehabilitation approach to migraine treatment incorporates broad lifestyle modifications with diet changes and supplements. (Table 1) Lifestyle modifications target reduction in migraine triggers such as stress, dehydration, hunger, and inadequate sleep (including treatment of insomnia and sleep apnea). Patients are counseled in stress management strategies, drinking a minimum of 2 liters of water, eating at regular intervals, and sleep hygiene. Dietary triggers of migraine -including tyramine, caffeine, histamine, and glutamate- can be avoided through elimination or reduction of chocolate, most forms of alcohol, nuts, fermented/aged products, and packaged foods. Elimination of salt, which is often advised for MD, may be actually effective from the inadvertent reduction of glutamate, tyramine, and histamine that are found in salty foods (pre-packaged, frozen, canned foods, nuts, etc.). Rather than restricting sodium, patients should be encouraged to maintain adequate hydration as dehydration and a relative increase in serum sodium concentration is the likely trigger rather than an increased salt load. We routinely tell our MD patients to not restrict sodium but drink more than 2 liters per day if they do consume more salt. Alcohol elimination, advised commonly in the treatment of MD, is actually eliminating tyramine (found at high levels in wine and beer) from the diet. We have found that highly distilled alcohol (e.g., vodka) does not cause attacks as long as patients maintain the extra hydration required after consuming alcohol due to its diuretic effect.

For medical prophylaxis, pharmacologic options include calcium channel blockers (verapamil), tricyclic antidepressants (nortriptyline) with anti-epileptics (topiramate) with escalation.(52, 53) Other classes to consider include beta blockers, gepants, and monoclonal antibodies to CGRP. Patients with symptoms of MD may benefit from this migraine prophylaxis in addition to (or instead of) the standard thiazide diuretic therapy. For severe refractory MD symptoms, we rarely perform surgery as migraine treatment is highly effective. For patients with pressure sensitive MD or vertigo in general, we perform a tympanostomy tube on the affected side or bilaterally when the ear of origin is unknown.(54) Over the last 15 years of our practice using migraine treatment for MD, we have not had to perform a destructive procedure in our patients given the highly effective migraine therapies.

CONCLUSION

The symptoms of MD are likely a manifestation of migraine rather than an independent inner ear condition. Neurogenic changes during migraine can impact cochleovestibular blood flow, which can induce episodic vertigo, tinnitus, and hearing loss. While ELH is typically pathognomonic for MD, ELH has been found in patients with VM and clinical testing has yet to differentiate MD from VM. MD can be effectively treated with migraine regimens such as the integrative neurosensory rehabilitation approach using lifestyle/diet changes, magnesium and riboflavin supplementation, and migraine prophylactic medications when necessary.

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KEY POINTS

- Meniere's disease (MD) is a manifestation of migraine. Migraine can cause vertigo (vestibular migraine) and migraine can cause hearing loss (cochlear migraine). MD is migraine that causes both cochlear and vestibular symptoms (otologic migraine).
- The prevalence of migraine in MD is more than four times higher than the general population.
- Symptoms of MD can be effectively treated with migraine prophylaxis including lifestyle and diet modifications, magnesium and riboflavin supplementation, and migraine prophylactic medications.

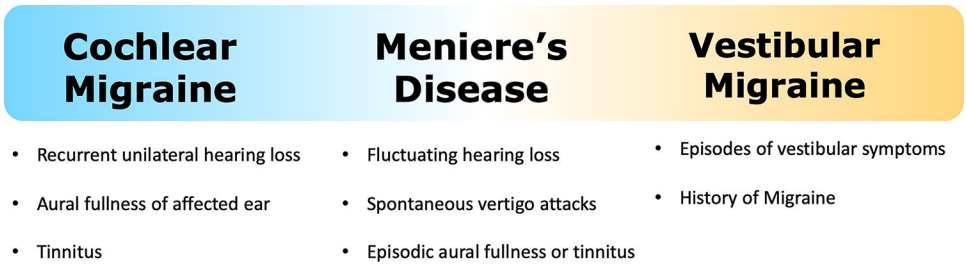

Figure 1.
Meniere's disease as otologic migraine

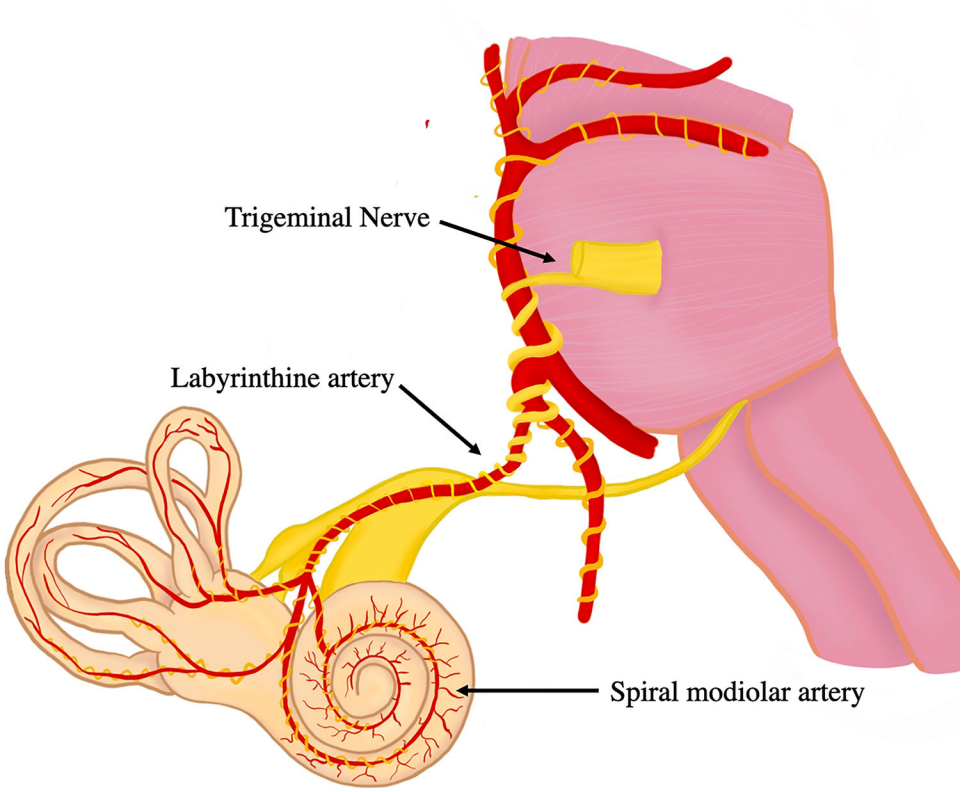

Figure 2.
Trigeminal stimulation of cochleovestibular vasculature

Table 1.

Integrative neurosensory rehabilitation approach

RECOMMENDATIONS	DETAILS
Lifestyle Modifications	
Stress and stimulation management	Regular exercise Practice meditation or yoga Efforts to reduce stressors
Sleep hygiene	Evaluate for sleep apnea or insomnia Consistent sleep schedule of 6–8 hours Go to bed and wake up around the same time daily (weekends and weekdays)
Avoid dehydration and hunger	2 liters of water daily Eat frequent meals on a consistent schedule
Diet Elimination	
Histamine	Nuts, citrus, chocolate, pole or broad beans, lima beans, lentils, fava beans, onions, avocado, fig, banana, papaya
Tyramine	Fermented products including alcohol (beer, wine), aged, aged or processed meats (bologna, pepperoni, salami, sausage, jerky), processed soy (tofu, soy sauce, teriyaki sauce, etc.), dried fruit, overly ripened fruit Egg whites, yogurt, kimchi
Glutamate (including monosodium glutamate)	Pre-packaged foods including chips, canned, and frozen meals Soy sauce, canned soup, bouillon cubes
Caffeine	Coffee, tea, soda, Excedrin™
Supplements	Magnesium 400mg bid, riboflavin 200mg bid
Medications	
Calcium channel blockers	Verapamil
Antidepressants	Nortriptyline, amitriptyline, paroxetine, venlafaxine
Anti-epileptics	Topiramate, gabapentin, valproate
Beta blockers	Propranolol
Gepants	Ubrogepant, atogepant and rimegepant
Monoclonal antibodies	Erenumab, Eptinezumab, Fremanezumab, Galcanezumab