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Migraine Features in Patients With Meniere's Disease

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Objectives/Hypothesis: To better understand the features of migraine in Meniere's disease (MD).

Study Design: Retrospective review of prospectively obtained surveys in an outpatient clinic of a tertiary medical center.

Methods: Detailed questionnaires on headaches and dizziness were given to consecutive patients presenting with dizziness. The responses were verified by the clinician with the patient. The data, in addition to the clinical history and audiogram, were used to diagnose patients with migraine headaches and MD using criteria set by the International Headache Society (IHS) and the American Academy of Otolaryngology–Head and Neck Surgery, respectively. The prevalence of migraine-like symptoms in those patients with MD, who did not fit the diagnostic criteria for migraine, was evaluated.

Results: Thirty-seven patients with definite MD were included. There was a predominance of females (female/male:26/11). Mean age of patients was 52 ± 14 years. Nineteen patients (51%) had migraine headaches. Fifteen patients fulfilled the criteria for definite vestibular migraine. Of those who did not fulfill the IHS migraine criteria, a majority had characteristics such as a family history of migraine, visual motion sensitivity, or lifelong motion sickness that were highly suggestive of a migraine disorder.

Conclusions: A majority of patients with MD have migraine headaches as defined by the IHS. Sensitivity to visual motion, light and sound, head motion, smells, weather changes, or medication was present in 95% of all patients with definite MD and 82% of non-IHS migraine MD patients. This may suggest that MD may be an atypical variant of migraine.

Key Words: Meniere's disease, migraine-related vestibulopathy, vestibular migraine.

Level of Evidence: 4

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INTRODUCTION

Several studies have shown that there is significant overlap between the symptoms of vestibular migraine (VM) and those of Meniere's disease (MD).[1–3] Patients with VM may experience symptoms of MD including episodic vertigo, tinnitus, constant or fluctuating sensorineural hearing loss (SNHL), and/or aural fullness.[1] Dizziness attacks are seen in a majority of VM patients, and these attacks are independent of headaches.[2] Therefore, sometimes patients with VM are misdiagnosed as MD patients.[3] In these circumstances, differentiation of these two diseases is often difficult. Currently, there are no known definitive diagnostic tests that can reliably distinguish the two conditions. Diagnosis is primarily based on clinical criteria. MD is diagnosed by the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) criteria,[4] and VM is diagnosed based on the criteria defined by the International Headache Society (IHS).[5]

The overlap of MD and VM has been described by a multitude of authors. Rassekh and Harker found a migraine prevalence of 81% in patients with vestibular MD compared with 22% in patients with classic MD.[6] These groupings of vestibular and classical MD, as well as cochlear and vestibular MD, have fallen out of favor, and the Committee on Hearing and Equilibrium of the AAO-HNS had recommended that these cochlear or vestibular variants of MD be excluded from classical MD.[7] The AAO-HNS suggested that this phenomenon of recurrent vestibulopathy without hearing loss was not related to the same pathological disorder of classical MD of idiopathic endolymphatic hydrops. Parker also recognized that migraine occurs more often in patients with MD than in the general population, and that the classical symptoms of MD and migraine may be related.[8] A common genetic and vascular origin of migraine and MD was proposed, where abnormal blood flow to the brain, cochlea, and vestibule can cause paroxysmal cochlear and vestibular symptoms. Minor et al. noted that there are similarities in the presentations of patients with migraine and MD.[9] There is clearly a significant overlap between VM and MD, which may suggest that the two conditions may be variants of each other.

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It has been observed that patients with VM generally had normal pure-tone average hearing, whereas patients with definite MD had significantly worse pure-tone average hearing, especially in the lower frequencies. Based on this observation, it has been recommended that SNHL be the main characteristic in distinguishing the two disorders.[10] However, it was noted that there were several patients with definite MD according to the AAO-HNS criteria who presented with normal hearing, though a review of later audiograms identified a low-frequency sensorineural hearing loss.[10] Additionally, there were VM patients with hearing loss. These patients with VM improved with migraine prophylactic medications. The cochlear symptoms of VM include tinnitus, aural fullness, and hearing loss that may fluctuate, as with MD. The vestibular manifestations of VM are variable, ranging from true vertigo, with episodic vertigo attacks, motion sensitivity, and chronic nonspecific vestibulopathy.[9,10]

In our practice, we have found that the vast majority of the patients with MD also have histories that are consistent with the presence of migraine. These patients, however, may not completely fulfill the IHS criteria for migraine headaches with or without aura. In this study, we set out to determine the prevalence of migraine headaches as defined by the IHS criteria in our Meniere's disease cohort. In addition, we specifically examined the characteristics of the MD patients who did not fulfill the criteria for migraine to determine the frequency of other migraine-related characteristics without the IHS definition of migraine.

MATERIALS AND METHODS

Patients

All adult patients who presented to the neurotology clinic at our institution with a complaint of dizziness between April 2013 and January 2014 were asked to fill out a questionnaire detailing their dizziness, headaches (if any), and other migraine-related symptoms. Patients with a diagnosis of MD based on the AAO-HNS criteria were included in our study.[4] Criteria were met using clinical evaluation, questionnaire, the audiogram, and vestibular testing (when available) (Table I). If a patient's diagnosis remained uncertain, his/her record was excluded from the analysis. Institutional review board approval was obtained from our institution.

Data Collection

A written questionnaire was developed based on the criteria set by the IHS for migraine with and without aura, VM, and MD (Table II).[5] In addition, a list of symptoms and clinical features potentially related to migraine was determined through a review of the literature. Data regarding these migraine-related factors were collected through the questionnaire and on clinical evaluation (Table III). This questionnaire was provided to patients as part of their clinical evaluation. Audiograms and vestibular testing (when available) were evaluated to assist in the diagnosis of MD. For each patient, the examiner confirmed the validity of their responses by rechecking the written responses with the patient. All diagnoses were confirmed by the senior author during the patient encounter. Patients were then followed up for at least 3 months, and their response to migraine prophylactic treatment was recorded. A positive response was considered as at least a 50% reduction in the frequency and subjective severity of the attack.

TABLE I.
The 1995 American Academy of Otolaryngology–Head and Neck Surgery Diagnostic Criteria for Meniere's Disease.

Certain Meniere's disease	Definite Meniere's disease plus histopathologic confirmation
Definite Meniere's disease	Two or more definitive spontaneous episodes of vertigo 20 minutes or longer
	Audiometrically documented hearing loss on at least one occasion
	Tinnitus or aural fullness in the treated ear
	Other causes excluded
Probable Meniere's disease	One definitive episode of vertigo
	Audiometrically documented hearing loss on at least one occasion
	Tinnitus or aural fullness in the treated ear
	Other causes excluded
Possible Meniere's disease	Episodic vertigo of the Meniere's type without documented hearing loss, or sensorineural hearing loss, fluctuating or fixed, with disequilibrium but without definitive episodes
	Other causes excluded

Statistical Analysis

The frequencies of patients, who fulfilled the criteria for definite, probable, and possible MD, as well as patients who fulfilled the criteria for migraine with and without aura and VM, were calculated. The prevalence of migraine-related symptoms and clinical features were also determined in all patients and among those who did and did not fulfill the IHS criteria for migraine (non-IHS migraine patients). χ^2 test was used to make comparisons between the subgroups based on the IHS criteria. Fisher exact test was used wherever the χ^2 test's assumptions were not met. As a secondary analysis, three sets of migraine-related symptoms and clinical features that were similar to each other were defined (Table IV). Category A included the sensitivities and motion sickness; category B included family histories of migraine, MD, or motion sickness; and category C included head and neck pain associated with various stimuli. Prevalence of these groups of symptoms was calculated. All statistical analyses were performed using PASW 18.0 (SPSS Inc., Chicago, IL). Because multiple comparisons were made on the same data, the Bonferroni correction was applied to reduce the occurrence of type I errors. Therefore, a P value of $<.002$ was considered as significant.

RESULTS

Sixty-nine patients with MD comprised our study sample. Twenty-seven had possible, five had probable, and 37 had definite MD. The mean age of the patients was 52 years $\pm$ 14 (range, 18–87 years). The female to male ratio was 26 (70%) to 11 (30%). According to the IHS criteria, 19 patients (51%) had migraine headaches, nine (24%) without aura, and 10 (27%) with aura. Fifteen patients fulfilled the criteria for VM according to

TABLE II.
The International Classification of Headache Disorders, Third Edition, Criteria for Migraine With and Without Aura, and Vestibular Migraine.

Migraine without aura	A. At least five attacks fulfilling criteria B–D
	B. Headache attacks lasting 4–72 hours (untreated orunsuccessfully treated)
	C. Headache has at least two of the following fourcharacteristics:
	1. Unilateral location
	2. Pulsating quality
	3. Moderate or severe pain intensity
	4. Aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
	D. During headache at least one of the following:
	1. Nausea and/or vomiting
	2. Photophobia and phonophobia
	E. Not better accounted for by another ICHD-3 diagnosis
Migraine with aura	A. At least two attacks fulfilling criteria B and C
	B. One or more of the following fully reversible aura symptoms:
	1. Visual
	2. Sensory
	3. Speech and/or language
	4. Motor
	5. Brainstem
	6. Retinal
	C. At least two of the following four characteristics:
	1. At least one aura symptom spread gradually over $\geq$5 minutes, and/or two or more symptoms occur in succession
	2. Each individual aura symptom lasts 5–60 minutes
	3. At least one aura symptom is unilateral
	4. The aura is accompanied or followed within 60 minutes by headache
	D. Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded
Vestibular migraine	A. At least five episodes fulfilling criteria C and D
	B. A current or past history of migraine without aura or migraine with aura
	C. Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours
	D. At least 50% of episodes are associated with at least one of the following three migrainous features:
	1. Headache with at least two of the following four characteristics:
	i. Unilateral location
	ii. Pulsating quality
	iii. Moderate or severe intensity
	iv. Aggravation by routine physical activity
	2. Photophobia and phonophobia
	3. Visual aura
	E. Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder.

ICHD-3 = International Classification of Headache Disorders, Third Edition.

the IHS criteria (41% of all patients and 79% of those with migraine according to the IHS criteria).

The symptoms and clinical features related to migraine among patients with MD include scalp allodynia; family history of migraine; facial, head, or neck pain when exposed to cold stimuli; and aura-like or dizziness symptoms when having a migraine (Table III). Sensitivity to visual motion, light and sound, head motion, smells, weather changes, or medication was present in 95% of all patients with definite Meniere's and 82% of non-IHS migraine MD patients. Among these, head motion sensitivity was the most common (84% of all patients with definite MD). Fifty-nine percent of all and 50% of non-IHS migraine patients had motion sickness. Headache was also present in 65% and 39% of all and non-IHS migraine MD patients, respectively. Comparing those who fulfilled the IHS criteria for migraine and those who did not, only neck stiffness was significantly different in incidence (Table III). There were no other significant differences after applying the Bonferroni correction.

Thirty-two (87%) patients of the definite MD cohort had at least one of the symptoms in category A, 19 (51%) patients had at least one of the family histories in category B, and 33 patients (89%) had at least one of the symptoms in category C. All 37 definite Meniere's patients in our study sample (100%) met at least one of the above features, had at least one of the symptoms in category A, one of the family histories in category B, or one of the symptoms in category C.

DISCUSSION

We found that 51% of the 37 patients with definite MD had migraine headaches according to the IHS criteria. Additionally, 48% of the overall MD patients had VM based on the IHS criteria. Of the 28 patients who fulfilled the AAO-HNS criteria for MD but did not fulfill the IHS criteria for migraine, all had symptomatology that was highly suggestive of a migraine background.

The pathogenesis of the vestibular dysfunction in both disorders is still unclear. Endolymphatic hydrops is considered as the main histopathological correlate of MD, which may have been caused by another pathophysiologic mechanism. VM is thought to be related to

TABLE III.
Prevalence of Symptoms and Clinical Features Related to Migraine Among Patients With Definite Meniere's Disease and Comparison of Differences Between Those Who Fulfilled IHS Criteria for Migraine and Those Who Did Not.

Clinical Feature	All Patients With Definite MD		Patients Not Fulfilling IHS Criteria for Migraine		Patients Fulfilling IHS Criteria for Migraine		*P* Value*
	Frequency, N = 37	%	Frequency, N = 18	%	Frequency, N = 19	%	
Sensitivity							
Visual-motion sensitivity	19	51%	9	50%	10	53%	.99
Light sensitivity	20	54%	8	44.4%	12	63%	.25
Sound sensitivity	19	51%	9	50%	10	53%	.87
Head-motion sensitivity	31	84	7	39%	11	58%	.89
Smells sensitivity	11	30%	4	22%	7	37%	.33
Weather-change sensitivity	12	32%	6	33%	6	32%	.9
Medication sensitivity	8	22%	4	22%	4	21%	.93
Any of the above	33	89%	15	83%	18	95%	.63
Motion sickness	22	59%	9	50%	13	68%	.25
Mental confusion (head fog)	25	68%	10	55.5%	15	79%	.12
Family history							
Family history of migraine	12	32%	5	26%	7	39%	.56
Family history of Meniere's disease	3	8%	2	11%	1	5%	.52
Family history of motion sickness	3	8%	1	5%	2	11%	.58
Any of the above	19	51%	8	44%	11	58%	.32
History of using medication for migraine	12	32%	4	22%	8	42%	
Sinus pain, facial pressure, or headache when exposed to wind or air conditioner	11	30%	2	11%	9	47%	.01
Pain in scalp or face from unusual stimuli	5	13%	0	0%	5	26%	.02
History of getting headache when eating ice cream	25	68%	12	67%	13	68%	.91
History of sinus headaches	22	59%	8	44%	14	74%	.07
Neck stiffness	20	54%	7	39%	13	68%	.07
Hearing loss							
Sensorineural in at least one ear	35	95%	12	67%	13	68%	.91
Mixed hearing loss		%	6	33%	9	47%	.39
All types of hearing loss	35	95%	16	89%	19	100%	.14

*Based on the Bonferroni correction. *P* values <.002 were considered statistically significant.
IHS = International Headache Society.

the neural input of the trigeminal nerve on the vasculature to the inner ear.[11] In addition, central vestibular abnormalities, neurogenic inflammation, and possibly changes in blood flow may contribute to the effect of migraine on the inner ear or the symptoms seen in patients with VM. Specific effects on the peripheral end organ other than increase in vascular permeability have not been elucidated.[11] Therefore, MD is thought to be a vestibular end-organ disease, and VM is a central or peripheral vestibular dysfunction caused by a central phenomenon. Some MD patients have characteristics and features that correspond to the VM criteria, and more interestingly, these patients respond to the same medications that are used for VM patients.[12] The evidence for this treatment effect is currently observational. Even though this appears to support a similar underlying cause for VM and MD, it could also be partially due to medication effects that potentially control dizziness where the two disorders could have different pathophysiologic mechanisms. In our experience, medications with no apparent effect on dizziness (e.g., calcium channel blockers and tricyclic antidepressants), have provided relief in a significant group of patients with MD.

Based on similarities in cervical and ocular vestibular-evoked myogenic potential (VEMP) responses,[13] Zuniga et al. found that there was no one VEMP test that distinguished the two diseases. They theorized that patients with VM might have end-organ injury in the inner ear, causing endolymphatic hydrops. They recognized that certain VM patients might actually be MD patients who do not have large enough of an audiometric loss to meet the AAO-HNS criteria for MD. Similarly, it has been found that neither a motion sensitivity questionnaire nor caloric testing could distinguish MD and VM patients.[14] We believe that clinical testing cannot distinguish MD from VM, possibly due to the common origin of the two conditions.

The pathophysiology of migraine headaches is partially understood. Migraine aura is believed to be due to the release of neuropeptides, such as substance P, neurokinin A, and calcitonin gene-related peptide into the dural circulation,[15] with the activation and sensitization

TABLE IV.
Migraine-Related Symptoms and Clinical Features Categorization.

Category A	Motion sickness
	Sensitivity to head motion
	Sensitivity to light and sound
	Sensitivity to visual motion
	Sensitivity to smells
	Sensitivity to weather changes
	Sensitivity to medication*
Category B	Family history of migraine
	Family history of Meniere's disease
	Family history of motion sickness
Category C	Sinus pain, facial pressure, or headache when exposed to wind or air conditioner
	Pain in scalp or face from unusual stimuli
	History of getting headache when eating ice cream
	History of sinus headaches
	Neck stiffness

*Defined as significant side effects to low doses of many medications.

of the trigeminal vascular system,[16] and subsequent spreading cortical depression.[16,17] Vasospasm in the posterior circulation has been proposed as a mechanism for migraine-associated damage to the inner ear[18–20] and may underlie some forms of migrainous vertigo. Additionally, it has been proposed that imbalance and motion intolerance may result from abnormal central processing, possibly from vasospasm or spreading depression to the vestibular cortex.[21] Stimulation of the trigeminal nerve with noxious stimuli in guinea pigs caused changes in vascular permeability in the cochlea and basilar artery with orthodromic and antidromic activation of trigeminal sensory fibers.[11] From this animal model, the authors proposed that vertigo, tinnitus, and hearing deficits associated with migraine could arise by excitation of the trigeminal nerve fibers in the cochlea.

Endolymphatic hydrops is a histopathological diagnosis that is not confined to MD, and MD is a clinical diagnosis that is not always associated with hydrops.[22] Other factors, including autoimmune reactions,[23] food sensitivities, and vascular disturbances may play a role in MD.[22] As of yet, there is no clear explanation of why the hearing loss in MD is in the low frequencies. The labyrinthine artery, which is usually a branch of the anterior inferior cerebellar artery, then branches into the end-organ arteries of the cochlear and vestibular arteries.[24] The cochlear artery supplies the basal turn primarily and the spiral modiolar artery supplies the apex and the middle turn. A drop in blood flow to the cochlea via the spiral modiolar artery has been proposed to cause a low-frequency hearing loss.[25] We conjecture that endolymphatic hydrops may be the end result of recurrent assaults to the cochlea from recurrent vasospasms of the spiral modiolar artery, which primarily supplies the apical turn of the cochlea[26] and not an intrinsic cochlear disorder.

We theorize that MD is most likely a variant of VM and that the symptoms are a consequence of a migraine etiology and not an intrinsic inner ear disorder. We hypothesize that involvement of a combination of the anterior vestibular artery, vestibulocochlear artery, its individual branches (cochlear and vestibular arteries), and the spiral modiolar arteries are affected. The variance in the hearing loss may be due to the difference in anastomotic patterns among individual patients.[26] Some patients may develop low-frequency hearing loss, some high, some low and high with preserved mid frequencies (peak shaped audiogram),[27] and some flat.[28] A reversible vasospasm of the spiral modiolar artery alone would cause only a low frequency sensorineural hearing loss, and no vestibular symptoms as has been described in what was previously termed cochlear hydrops. Additional involvement of the cochlear artery can lead to high-frequency hearing loss seen in the high- and low-frequency hearing loss.[28] The involvement of the anterior vestibular artery, which supplies the superior canal, horizontal canal, and the utricle can lead to the more significant vertigo attacks. Finally, the vestibular artery involvement can cause dysfunction of the posterior canal and saccule, which may potentially be the cause of positional vertigo seen in MD.[29] Although this hypothesis for the pathophysiologic mechanism of this disorder is unproven and untested, further studies are warranted to elucidate this mechanism. Potential studies in animals modeled after the study by the Vass et al. group[11] to selectively stimulate or partially occlude the various arteries of the inner ear with subsequent histopathologic examination will help in understanding the pathophysiology. For example, if changes in vascular flow or recurrent stimulation of the trigeminal nerve were found to affect hearing in the animals and cause endolymphatic hydrops, it could help support the above hypothesis.

All the patients with Meniere's disease in our cohort responded to the migraine lifestyle/dietary changes and migraine prophylactic therapy with control of their vertigo and stabilization of their hearing. Our recommendation is that patients with MD and migraine receive treatment for their migraine disorder. Other patients, who do not fulfill the criteria for migraine headaches or VM, should be treated with migraine prophylactic agents prior to surgical or destructive intratympanic therapy. We believe that there may be several separate types of VM: one that causes hearing loss and has clinical manifestations of MD via a centrally modulated dysfunction of the cochlear and vestibular arteries and possibly end organs, and one that does not cause hearing loss (selective dysfunction of the vestibular artery or anterior vestibular artery). This second group of patients is also prevalent in our practice, but is not described in this study.

CONCLUSION

A majority of patients with MD have migraine headaches as defined by the IHS. Of those who do not fulfill the IHS migraine criteria, a majority has characteristics such as a family history of migraine, visual

motion sensitivity, or lifelong motion sickness that are highly suggestive of a migraine etiology. All Meniere's patients who did not fulfill the IHS criteria for migraine had either a family history of migraine-related problems, at least two sensitivities to external stimuli, or atypical head and neck pain associated with migraine. All MD patients in our cohort responded well to migraine lifestyle/dietary changes and migraine prophylactic therapy, with control of their vertigo and stabilization of their hearing. We recommend that patients with MD and migraine receive treatment for their migraine disorder.

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