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Migraine-associated vestibulopathy

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Abstract

Purpose of review: In the past few years, otologists have been seeing an increasing number of patients with vestibular disorders due to migraine-associated vestibulopathy. This article reviews some of the latest developments in the understanding of this disease process, specifically its incidence, symptoms, diagnosis, and treatment.

Recent findings: Migraine-associated vestibular symptoms may include episodic true vertigo, movement-provoked dysequilibrium, imbalance/unsteadiness, and complaints of lightheadedness. The pathophysiology of migraine-associated vestibulopathy is not completely understood; however, both peripheral and central deficits have been observed. Although the International Headache Society classification does not include migraine-associated vestibulopathy as a subclassification of migraine, there is emerging evidence to support this development, which should then lead toward improved diagnosis and treatment. Currently, migraine-associated vestibulopathy is still considered a diagnosis of exclusion.

Summary: Treatment of migraine-associated vestibulopathy is effective and includes lifestyle changes, such as reducing triggers that increase susceptibility to migraines (e.g. stress, poor diet, nicotine, or irregular sleep patterns), prophylactic and abortive medications, vestibular therapy, or a combination of these. Further research is needed to better understand migraine-associated vestibulopathy and improve treatment.

Keywords: dizziness, migraine, migraine-associated vestibulopathy, vertigo

Introduction

Dizziness and headaches are two of the most common symptoms for which patients seek medical attention. One subtype of headache, migraines, affects at least 16% of the adult population. Women are more commonly affected by migraines than men, especially after menarche [1-6]. A large percentage of individuals with migraine are affected by dizziness, also known as migraine-associated vestibulopathy (MAV) [7]. Migraine headaches and MAV may occur at any age [5, 8, 9]. However, patients typically present with a history of headaches years before vestibular symptoms develop; these patients also often have a family history of migraine sufferers [5, 6]. In a 2007 study of 24 vertiginous children and 12 age-matched and sex-matched controls, Niemensivu et al. [10] found that 10 of the vertiginous children had a history of migraine and four had migraine-associated dizziness. The authors concluded that headaches and migraines were more common among vertiginous children than among healthy controls. However, clinical findings on neurological and audiological examinations revealed no significant difference between groups.

Clinical presentation

A migraine is described as a recurring headache with varying characteristics. Migraines can present with reversible, focal neurological changes that may precede or coincide with the headache event (also known as an aura). Some patients suffering from migraines will not describe a headache at all, but they will instead report the associated aura-related neurological symptom, such as dizziness. The International Headache Society (IHS) has developed a classification system for migraines: migraine with or without aura, migraine with prolonged aura, basilar migraine, migraine aura without headache, childhood periodic syndromes, benign paroxysmal vertigo of childhood, and migrainous infarction [11] .

Migraine-associated vestibular symptoms may include episodic true vertigo, movement-provoked dysequilibrium, imbalance/unsteadiness, and complaints of lightheadedness [5, 8]. Symptoms vary for each individual, and the duration and frequency of vestibular symptoms may change over time [12]. In a 2007 retrospective review, Vuković et al. [13] compared the prevalence of dizziness among those

with and without migraines. The authors reported that 51.7% of 327 migraine patients experienced vertigo in their lifetime compared with 31.5% of 324 people in the control group. The authors noted that vertigo symptoms coincided with the migraine attacks in only 23.2% of the patients. Patients with MAV also have a propensity to motion sickness [14]. Although imbalance is commonly reported, few patients display difficulty walking without assistance [12]. Additionally, patients with MAV have also reported photophobia, phonophobia, and other auras, which represent a well established connection with migraine. Auditory symptoms reported in individuals with migraine-related vertigo include fluctuating hearing loss, tinnitus, and aura fullness, potentially complicating the differential diagnosis and differentiation of the disease from Ménière's syndrome [9,15].

Pathophysiology of migraine-associated vestibulopathy

The pathophysiology of MAV is not well understood. Both peripheral and central deficits have been observed. A spreading, global central nervous system depression that also affects the brainstem may account for central findings [5, 8]. Peripheral cochleovestibular dysfunction may be attributed to vasospasm of the internal auditory artery causing ischemia to the labyrinth [16-18]. However, others have attributed central and peripheral deficits to the release of neurotransmitters such as calcitonin-gene-related peptide during the migraine attack [8]. Evidence of ion-channel dysfunction and calcium-channel disturbances of the inner ear and its central connections also offer a promising hypothesis for the diagnosis and treatment of MAV [19].

Diagnosis

Dizziness may result from a variety of disease entities, including migraines [15]. At present, although the IHS classification does not include MAV as a subclassification of migraine [11], Neuhauser and Lempert [20] have proposed diagnostic criteria for MAV (Table 1). This may pave the way toward improved diagnosis and treatment. At this time, MAV is still considered a diagnosis of exclusion; that is, when no evidence exists of other peripheral or central nervous system disorders to account for the dizziness, this diagnosis is considered appropriate. The diagnostic challenge occurs because the vestibular symptoms often occur during headache-free periods [5, 6, 8, 21]. Specifically, one study [21] found that 50% of patients with vestibular symptoms never reported an accompanying headache.

The otoneurologic examination is typically unremarkable for patients with MAV. Unfortunately, there is not a single diagnostic test specific for MAV. However, peripheral and central vestibular system abnormalities noted during electronystagmography (ENG) testing (particularly caloric and ocular motor subtests) have been reported [22]. In a 2007 study of the clinical features and pathophysiology of MAV, Celebisoy et al. [23] detected peripheral and central findings on balance function tests in 35 patients. In particular, 20% exhibited caloric unilateral weakness. Cutrer and Baloh [8] identified spontaneous and positional nystagmus (often central-type) on ENG testing. Researchers have also noted directional preponderance on rotational chair testing [9].

Patients with MAV often present symptoms in common with other medical diagnoses, such as Ménière's disease (particularly vestibular Ménière's). Aural fullness and fluctuating hearing loss are common in patients with Ménière's disease and in those patients with migraine; these similar auditory symptoms have led some researchers to propose a link between Ménière's disease and migraines. Radtke et al. [24] found migraine-type symptoms in over 50% of patients with Ménière's disease. In a 2008 study, Cha et al. [25*] investigated the association and pathological link between migraine, episodic vertigo, and Ménière's disease in six families. In confirming this frequent association in closely related individuals (including identical twins), they then argued for the concept of heritability of a migraine-Ménière's syndrome. The researchers speculated that mi-

Table 1. Diagnostic criteria for migraine-associated vestibulopathy as proposed by Neuhauser and Lempert

Migrainous vertigo type	Symptoms
Definite migrainous vertigo	Recurrent episodic vestibular symptoms (moderate severity) History of migraine (current or previous and meets criteria of the IHS) Associated migrainous symptoms such as migrainous headache, photophobia, phonophobia, and other auras occurring during at least two vertiginous episodes Other causes ruled out by appropriate investigations
Probable migrainous vertigo	Recurrent episodic vestibular symptoms (moderate severity) One of the following: current or previous history of migraine according to the criteria of the IHS or migrainous symptoms during ≥ 2 attacks of vertigo or migrainous precipitants (food triggers, sleep irregularities, hormonal changes) before vertigo in more than 50% of attacks or responses to migraine medications in more than 50% of attacks Other causes ruled out by appropriate investigations

Data from Neuhauser and Lempert [20]. IHS, International Headache Society.

graine may contribute to permanent damage of the inner ear, subsequently predisposing the ear to Ménière's disease. In a related retrospective case-control study, Cha et al. [26] compared the clinical findings of patients with Ménière's disease and migraine and patients with Ménière's disease only. They discovered that the age of onset of vertigo symptoms was significantly lower in patients with Ménière's disease and migraine, as suggested in the 2008 study.

Management of migraine-associated vestibulopathy

Research on MAV has traditionally focused on the diagnostic results of various balance disorders with little emphasis on appropriate treatment options. The appropriate treatment plan is similar to that for classic migraine. This includes lifestyle changes, such as stress reduction, nutritious diet, avoiding nicotine, or avoiding irregular sleep patterns. In addition, the physician can also prescribe abortive or prophylactic medications. Patients may also benefit from vestibular therapy. In a retrospective review of the various management plans and their efficacy, Johnson [15] found that a comprehensive approach to control migraine headaches resulted in a substantial improvement in a majority of the patients.

Medications given for prophylaxis are often the drugs of choice when migraine-related vertigo attacks are severe and unresponsive to the nonmedical treatment options elucidated above [12]. Calcium-channel blockers, β-blockers, tri-cyclic amines, and antidepressants have all been advocated [5, 27, 28]. A prospective study by Maione [29] reported either complete resolution or considerable control of migraine-related vertigo symptoms when combinations of commonly used prophylaxis medications (β-blockers, calcium antagonists, and antidepressants) were administered in 36 patients with recurrent vertiginous spells.

Çeliker et al. [30*] investigated valproic acid to determine its effects on vestibular symptoms and ENG results in 37 women who were divided into three groups: 13 patients with vertigo, 13 patients with nonvertigionous dizziness, and 11 women without any vestibular symptoms. In ENG evaluations performed before and after valproic acid treatment, the investigators found a statistically significant decrease in the number of vestibular episodes in both groups with vertiginous symptoms. The drug was equally effective at reducing migraine headache in all groups. However, there were not any statistically significant differences noted on ENG. The results suggest that while the effect of migraine on the vestibular system may be permanent (as noted on ENG), valproic acid could be used to effectively reduce both the headache and MAV symptoms. In a 2007 retrospective review, Iwasaki et al. [31] reported on the effectiveness of lomerizine as a prophylactic treatment of MAV. The calcium-channel blocker, which was administered to 22 Japanese patients, effectively reduced headache and dizziness symptoms in 87% of the patients.

Conclusion

Although the IHS currently has not recognized MAV in the classification of migraine, there is emerging clinical evidence to support this diagnosis. The relationship between vestibular symptoms and migraine is commonly observed. However, further research, involving multidisciplinary and multicenter approaches, is needed to better understand the pathophysiologic mechanisms behind MAV. Ultimately, this will result in the earlier diagnosis and improved treatment of patients with MAV.

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