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Migraine-Associated Vertigo

Updated: Mar 09, 2017
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OVERVIEW

Overview

The manifestations of migraine-associated vertigo are quite varied and may include episodic true vertigo, positional vertigo, constant imbalance, movement-associated dysequilibrium, and/or lightheadedness. [1, 2]

Symptoms can occur before the onset of headache, during a headache, or, as is most common, during a headache-free interval. Consequently, many patients who experience migraines have vertigo or dizziness as the main symptom rather than headache. For this reason, this article is devoted to the description of migraine-associated vertigo.

Since the 19th century, repeated references have been made to the clinical association of migraine and dizziness. Over the years, several syndromes of episodic vertigo associated with migraine have been reported, including benign paroxysmal vertigo of childhood and benign recurrent vertigo in adults. [3, 4] Some authors have even suggested an association between migraine and Ménière disease.

Vertigo is also a symptom of basilar migraine, a migraine variant that is also known as Bickerstaff syndrome [5] (and that was previously known as basilar artery migraine).

See Vertigo: 5 Case-Based Diagnostic Puzzles, a Critical Images slideshow, to help recognize diagnostic clues in vertigo cases.

Although the definition of migraine-related vertigo and the continuum of the symptom complex remains poorly defined, the relationship is clearly more than a chance association.

In a well-controlled study that evaluated 200 patients from a migraine clinic, a dizziness clinic, and a control group from an orthopedic clinic, the group presenting with vertigo showed a higher lifetime prevalence of migraine (38%) than did a similar group of patients in the control group (24%). Similar findings have been seen in studies evaluating migraine patients. Vertigo, as well as chronic, nonspecific symptoms of vestibular system dysfunction, can be related to all forms of migraine.

A study by Wang et al indicated that persons with migraine-associated vertigo experience greater cognitive impairment than do persons who suffer from simple migraine. Subjects with migraine-associated vertigo scored lower on cognitive tests than did those with simple migraine, while magnetic resonance imaging (MRI) demonstrated a greater incidence of deep brain, peripheral lateral ventricle, and total white matter lesions in the migraine-associated vertigo group than in the other. [6]

Definitions of migraine and vertigo

Migraine headaches are recurrent headaches that are often accompanied by nausea and light sensitivity

and that are separated by symptom-free intervals. The headaches typically have a throbbing quality, are relieved after sleep, and may be accompanied by visual symptoms, dizziness, or vertigo. Patients often have a family history of migraine.

Migraine can be divided into 2 categories: migraine without aura (common migraine, 90% of migraine headache cases) and migraine with aura (classic migraine, 10% of cases).

Vertigo is an illusion of movement of the environment or of the patient in relation to the environment.

Epidemiology

Migraine is an extremely common disorder worldwide; in the United States alone, this condition occurs in 18% of women and in 6% of men aged 12-80 years, a total of 25-28 million people. [7] Women of childbearing age are most affected, with an approximate prevalence of 25% in 35-year-old women. [8]

In 1984, Kayan and Hood reported a significant increase in the frequency of vertigo in people with migraines versus people with tension headaches. [9]

Overall, episodic vertigo occurs in about 25-35% of all migraine patients. Using these figures, roughly 3.0-3.5% of people in the US have episodic vertigo and migraine. [7] Comparatively, the prevalence of Ménière disease (a peripheral vestibular disorder with symptoms overlapping that of migraine-associated vertigo) is estimated to be 0.2% of the US population. [10]

A Turkish study, by Batu et al, of 100 children with vertigo who presented to a pediatric neurology referral center found that migraine-associated vertigo was the fourth most common form of the condition (11%) among these patients, behind benign paroxysmal vertigo of childhood (39%), psychogenic vertigo (21%), and epileptic vertigo (15%). [11]

For patient education information, see the Headache and Migraine Center, as well as Migraine Headache, Vertigo, Dizziness, and Understanding Migraine and Cluster Headache Medications.

IHS Migraine Classification

Migraine categories from the International Headache Society (IHS) include childhood period syndromes, migrainous infarction, and migraines with or without aura. These are detailed below. [12]

Migraine without aura

Migraine without aura, formally called common migraine, has at least 2 of the following characteristics:

- Unilateral location
- Pulsating quality
- Moderate or severe intensity that inhibits or prohibits daily activities
- Aggravation by walking up stairs or similar routine physical activity

Left untreated, the headache attacks last 4-72 hours; in children younger than 15 years, the headache may last 2-48 hours. During the headache, at least 1 of the following occurs: (1) nausea and/or vomiting or (2) photophobia and phonophobia.

During the clinical evaluation, at least 1 of the following occurs:

- History and physical examination findings do not suggest another disorder

- History and physical examination findings do suggest another disorder, but the other disorder is ruled out by appropriate investigations (eg, magnetic resonance imaging [MRI] or computed tomography [CT] scanning of the head).

Migraine with aura

Migraine with aura, formally known as classic migraine, is categorized by headache and aura features. The headache characteristics are the same as those for migraine without aura, above. The aura is characterized by at least 2 attacks of the following:

- One reversible aura symptom indicating focal central nervous system (CNS) dysfunction - Ie, vertigo, tinnitus, decreased hearing, ataxia, visual symptoms in 1 hemifield of both eyes, dysarthria, double vision, paresthesias, paresis, decreased level of consciousness
- Aura symptom that develops gradually over more than 4 minutes or 2 or more symptoms that occur in succession
- No aura symptom that lasts more than 60 minutes unless more than 1 aura symptom is present
- Headache occurring before, during, or up to 60 minutes after the aura is completed

Migraine with prolonged aura fulfills criteria for migraine with aura; however, the aura lasts more than 60 minutes (but less than 7 days).

Basilar migraines fulfill the criteria for migraine with aura, but 2 or more of the following aura symptoms occur:

- Vertigo
- Tinnitus
- Decreased hearing
- Ataxia
- Visual symptoms in both hemifields of both eyes
- Dysarthria
- Double vision
- Bilateral paresthesias
- Bilateral paresis
- Decreased level of consciousness

Migraine aura without headache (replaces migraine equivalent or acephalic migraine) fulfills criteria for migraine with aura, but no headache occurs.

Childhood period syndromes

Childhood periodic syndromes are those that may be precursors to or associated with migraines.

Benign paroxysmal vertigo of childhood is characterized by brief sporadic episodes of dysequilibrium, anxiety, and, often, nystagmus or vomiting. The neurologic examination findings are normal, as are findings on electroencephalography.

Migrainous infarction

Patients with migrainous infarction (replaces complicated migraine) have previously fulfilled the criteria for migraine with aura. Their present attack is typical of previous attacks, but neurologic deficits are not completely reversible within 7 days, and/or neuroimaging demonstrates ischemic infarction in the relevant area. Other causes of infarction are ruled out by appropriate investigations.

Etiology and Neuropathophysiology

Migraine headache and migraine-associated vertigo are often triggered by certain factors, including stress, anxiety, hypoglycemia, fluctuating estrogen, certain foods, and smoking. However, although central and peripheral defects have been observed, the etiology of migraine-associated vertigo is not completely understood. No single hypothesis explains the headache or dizziness process in migraine at this time. Thus, the causes of the symptoms of migraine remain controversial.

Genetics

The genetic cause of a rare type of migraine has been discovered. Familial hemiplegic migraine, a form of migraine with aura, is associated with mutations in the *CACNA1A* gene located on chromosome arm 19p13. [13] This gene codes for a neuronal calcium channel. Defects involving this gene are also involved in other autosomal dominant disorders that have neurologic symptoms (see Table 1, below). One example is that of episodic ataxia type 2 (EA2), which is also known as periodic vestibulocerebellar ataxia and acetazolamide-responsive hereditary paroxysmal cerebellar ataxia. In cases of EA2, a pH abnormality has been discovered, and it often resolves with medication (eg, acetazolamide, valproic acid, calcium channel blocker).

Table 1. *CACNA1A* Gene Defects Associated With Autosomal Dominant Disorders With Neurologic Symptoms (Open Table in a new window)

GENE Defect	Syndrome	Symptoms and Signs
Point mutation	Familial hemiplegic migraine	Episodic hemiparesis for 60min or less, followed by headache; gaze-evoked and downbeat nystagmus may persist after spells
Point mutation	Episodic ataxia type 2 (EA2)	Episodic ataxia and vertigo, gaze-evoked and downbeat nystagmus, abnormal pursuit on electronystagmography (ENG)
CAG repeats	Spinocerebellar ataxia type 6 (SCA6)	Progressive ataxia, gaze-evoked and downbeat nystagmus, abnormal pursuit on ENG
Adapted from Tusa, 1999. [14]		

Spreading depression theory

In 1992, Cutrer and Baloh developed the most commonly accepted theory regarding the pathophysiology of migraine-associated vertigo. [15] They proposed that episodes of dizziness of a duration similar to that of a migraine aura (< 60min) that are time-locked with the headache most likely have the same pathophysiologic mechanism (eg, spreading wave of depression) as other aura phenomena.

According to the spreading depression theory, some type of stimulus (eg, chemical, mechanical) results in a transient wave front that suppresses central neuronal activity. This depression spreads in all directions from its site of origin. Neuronal depression is accompanied by large ion fluxes, including

increases in extracellular potassium (K^+) and decreases in extracellular calcium (Ca^{++}). These changes result in a reduction in cerebral blood flow in the areas of spreading depression. However, most patients with migraine-associated vertigo have dizziness independently of the headache.

Cutrer and Baloh suggested that when the dizziness is unrelated to the headache, the dizziness results from the release of neuropeptides (ie, neuropeptide substance P, neurokinin A, calcitonin gene–related peptide [CGRP]). [15] Neuropeptide release has an excitatory effect on the baseline firing rate of the sensory epithelium of the inner ear, as well as on the vestibular nuclei in the pons.

Asymmetrical neuropeptide release results in the sensation of vertigo. When neuropeptide release is symmetrical, the patient feels an increased sensitivity to motion due to an increased vestibular firing rate during head movements.

Cutrer and Baloh also proposed that CGRP and other neuropeptides may produce a prolonged, hormonelike effect as these peptides diffuse into the extracellular fluid. [15] This may explain the prolonged symptoms in some patients with migraine-associated vertigo, as well as the typical progression of persistent spontaneous vertigo, followed by benign positional vertigo and then motion sensitivity.

Alternative proposed mechanisms

Some authors have suggested that peripheral cochleovestibular dysfunction in migraine patients may be attributed to vasospasm of the internal auditory artery causing ischemia to the labyrinth. [16] Furthermore, Lee et al have reported a positive association of the progesterone receptor (PGR) with migraine-associated vertigo. [17]

Serotonin (5-hydroxytryptamine [5-HT]) has also been found to be an important substrate in the development of migraine. Interestingly, 5-HT has direct effects on the firing rate of vestibular nucleus neurons. The serotonergic and the peptidergic pathways possibly play a role in the development of the short and prolonged periods of dizziness in migraine-associated vertigo.

Patient History and Physical Examination

As with any type of dizziness evaluation, the history is the most important means to diagnose migraine-associated vertigo. [1, 18, 19] Patients with migraine-related vestibulopathy typically experience a varied range of dizzy symptoms throughout their life and even within individual attacks. [2, 18] These symptoms may be solitary or may be a combination of vertigo, lightheadedness, and imbalance.

A thorough headache history is also important when evaluating patients for possible migraine-associated vertigo. Many patients with recurrent headaches are unaware that their headaches may be from migraine. Therefore, the examining physician should have a thorough knowledge of the strict diagnostic criteria for migraine diagnosis.

Dizziness and vertigo

At the time of presentation, dizziness symptoms may have been present for a few weeks or for several years. Vertigo may occur spontaneously, provoked by head motion or by visual stimuli. Symptoms may last for a few minutes or may be continuous for several weeks or months. In women, dizziness may often occur during the menstrual cycle.

Patients with migraine-associated vertigo often provide a long history of motion intolerance during car, boat, or air travel—or all 3. Some patients are very sensitive to motion of the environment and to busy

environments. Vertigo, which is an illusion of movement of the environment or of the patient in relation to the environment, is the most common type of dizziness reported, and it is present at some time in approximately 70% of patients. The attacks of vertigo may awaken patients and are usually spontaneous, but they may be provoked by motion.

The duration of the vertigo can also be quite variable. The following list delineates the frequencies of different durations of vertigo spells in migraine-associated vertigo:

- Duration of seconds - 7%
- Duration of minutes to up to 2 hours - 31%
- Duration of 2-6 hours - 5%
- Duration of 6-24 hours - 8%
- Duration longer than 24 hours - 49%

When vertigo is present, it may be indistinguishable from the spontaneous vertigo of Ménière disease. One clue that the vertigo is not of the Ménière type is that the vertigo of migraine-associated vertigo may last longer than 24 hours. In fact, a rocking sensation may be a continuous feeling for many weeks to months. In contrast, the vertigo of Ménière disease typically does not last longer than 24 hours. (See Table 2).

Headache

Patients may or may not have a history of concurrent migraine headaches. In fact, most patients have dizziness symptoms during headache-free intervals or even numerous years following their last migraine headache. [8] Some patients with migraine-associated vertigo have never experienced a migraine headache but have a family history of migraine.

Physical findings

Findings on a complete neurotologic examination are often normal. Horizontal rotary spontaneous nystagmus may be present during an acute attack of vertigo. Dix-Hallpike examination may elicit symptoms of vertigo or nonvertigo dizziness, each without nystagmus.

Diagnostic Criteria

No diagnostic tests exist for migraine-associated vertigo. As with any type of dizziness evaluation, the history is the most important means to diagnose this condition. [1, 18, 19] When the history is unclear, the diagnosis is made by a therapeutic response to treatment.

A definite diagnosis of migraine-associated vertigo can be made when patients have migraine with aura that is accompanied by concurrent episodes of vertigo or when they have migraine without aura that is repeatedly associated with vertigo immediately before or during the headache.

A probable diagnosis of migraine-associated vestibulopathy is suggested when patients experience recurrent or continuous vertigo or dizziness sensations without neurologic symptoms, when the dizziness is not time-locked to headache, when a past or family history of migraine headaches exists, and when the dizziness cannot be fully explained by other vestibular disorders. In these patients, a trial of migraine therapy can be started for diagnostic and therapeutic purposes.

Proposed diagnostic criteria

Neuhauser and Lempert proposed the following criteria for the diagnosis of definite migraine-associated

vertigo [20] :

- Episodic vestibular symptoms of at least moderate severity - Rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance (ie, sensation of imbalance or illusory self or object motion that is provoked by head motion)
- Migraine according to the International Headache Society (IHS) criteria
- At least 1 of the following migrainous symptoms during at least 2 vertiginous attacks - Migrainous headache, photophobia, phonophobia, visual or other auras
- Other causes ruled out by appropriate investigations

Proposed criteria for the diagnosis of probable migraine-associated vertigo include the following:

- Episodic vestibular symptoms of at least moderate severity - Rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance
- At least 1 of the following - Migraine according to the criteria of the IHS, migrainous symptoms during vertigo, migraine-specific precipitants of vertigo (eg, specific foods, sleep irregularities, hormonal changes), response to antimigraine drugs
- Other causes ruled out by appropriate investigations

Differential Diagnosis

Ménière disease versus migraine-associated vertigo

The principal differential is with Ménière disease. The overlapping symptoms of Ménière disease and migraine-associated vertigo include episodic vertigo, sensorineural hearing loss, and tinnitus. Differentiating migraine-associated vertigo from Ménière disease may be difficult, because of the overlapping nature of the symptoms of these diseases. However, often the patient's history offers clues that may help to make the diagnosis. (See Table 2, below.)

Table 2. A Comparison of the Symptoms of Migraine-Associated Vertigo and Ménière Disease (Open Table in a new window)

Symptom	Migraine-Associated Vertigo	Ménière Disease
Vertigo	May last >24h	Lasts up to 24h
Sensorineural hearing loss	Very uncommon; when present, often low frequency; very rarely progressive; may fluctuate in cases of basilar migraine	Nearly always progressive; most often unilateral; may be bilateral; fluctuation is common
Tinnitus	May be unilateral or bilateral; rarely obtrusive	May be unilateral or bilateral; often of significant intensity
Photophobia	Often present; may or may not be associated with dizziness	Never present unless a concurrent history of migraine exists

When vertigo is present, it may be indistinguishable from the spontaneous vertigo of Ménière disease. One clue that the vertigo is not of the Ménière type is that the vertigo of migraine-associated vertigo may last longer than 24 hours. In fact, a rocking sensation may be a continuous feeling for many weeks to

months. In contrast, the vertigo of Ménière disease typically does not last longer than 24 hours.

Symptoms that would support the diagnosis of migraine-associated vertigo as opposed to Ménière disease include photophobia, nonprogressive sensorineural hearing loss, vertigo of longer than 24 hours in duration, a long-standing history of motion intolerance, and dizziness occurring only during the menstrual cycle. Childhood benign positional vertigo is strongly related to migraine-related vertigo.

Migraine and vestibular disease can coexist. [21] Patients who meet the clinical criteria for Ménière disease should be treated appropriately for Ménière disease, even if a history of migraine headache exists.

Sensorineural hearing loss in Ménière disease and basilar migraine

Although unexplained sensorineural hearing loss has been reported in 0-31% of unselected patients with migraine, [22] such changes are rarely a significant feature of migraine-related vertigo and thus help to differentiate it from other causes of vertigo, especially Ménière disease.

Up to 80% of patients with basilar migraine have been reported to have sensorineural hearing loss, which often affects the lower frequencies and may be bilateral. [23] Fluctuation is also possible, similar to the sensorineural hearing loss of Ménière disease. However, unlike in Ménière disease, the sensorineural hearing loss of basilar migraines rarely progresses.

Differentials

The differential diagnosis of migraine-associated vertigo includes peripheral and central vestibular disorders. Peripheral disorders include the following:

- Ménière disease
- Perilymphatic fistula
- Benign paroxysmal positional vertigo
- Recurrent vestibular neuritis
- Recurrent vestibulopathy

Central disorders include the following:

- Multiple sclerosis
- Central paroxysmal positional vertigo
- Vertebrobasilar artery insufficiency
- Cervicomedullary compression from abnormalities of the craniovertebral junction

The following conditions should also be considered in suspected cases of migraine-associated vertigo:

- Acute laryngitis
- Central nervous system causes of vertigo
- Labyrinthitis

A study by Goto et al indicated that photic-driven electroencephalographic responses can be used to diagnose migraine-associated vertigo. Such responses differed significantly on average between persons in the study with migraine-associated vertigo (21 patients) and those with other vestibulopathies (15 patients) at frequencies of 10, 12, and 15 Hz. [24]

Diagnostic Testing

No pathognomonic abnormalities on imaging studies or vestibular testing confirm migraine-associated vertigo. When the clinical history is wholly consistent, no other evaluation should be necessary to confirm the diagnosis.

Audiometric evaluation

Full audiometric evaluation, including pure-tone audiometry, word recognition scores, and reflex testing, is appropriate for any patient being evaluated for dizziness.

Electronystagmography

Electronystagmography (ENG) is typically not helpful in differentiating migraine-associated vertigo from Ménière disease. However, for patients with a several-year history of dizziness, normal findings on ENG are suggestive of migraine-associated vertigo.

Electrocochleography

Patients with a several-year history of Ménière disease often have a reduced vestibular response on at least 1 side. Electrocochleography (ECoG) may help to differentiate Ménière disease and perilymphatic fistula from migraine-associated vertigo.

Vestibular evoked myogenic potential

Both ocular and cervical vestibular evoked myogenic potentials (VEMPs) are receiving attention for their potential clinical utility. The more studied cervical VEMP is based on a reflex relation of the sternocleidomastoid muscle in response an acoustic signal. VEMP testing in migraine patients is very similar to that seen in Ménière disease patients, which again emphasizes the similarities between these 2 conditions. [25]

Caloric testing

Celebisoy et al detected peripheral and central findings on balance function tests in 35 patients with migraine-associated vertigo. [26] Of note, 20% of these patients exhibited caloric unilateral weakness, whereas all of the migraine patients in the control group without vertigo had normal caloric testing.

MRI

MRI of the brain with gadolinium is necessary when patients present with unilateral symptoms or signs or if the patient's symptoms do not respond to appropriate treatment. If the patient’s symptoms are those of unilateral sensorineural hearing loss or tinnitus, the MRI should be directed to the internal auditory canals.

Gadolinium warning

Gadolinium-based contrast agents (gadopentetate dimeglumine [Magnevist], gadobenate dimeglumine [MultiHance], gadodiamide [Omniscan], gadoversetamide [OptiMARK], gadoteridol [ProHance]) have been linked to the development of nephrogenic systemic fibrosis (NSF) or nephrogenic fibrosing dermopathy (NFD). For more information, see the Medscape Reference topic Nephrogenic Systemic Fibrosis. The disease has occurred in patients with moderate to end-stage renal disease after being given a gadolinium-based contrast agent to enhance MRI or magnetic resonance angiography (MRA) scans.

NSF/NFD is a debilitating and sometimes fatal disease. Characteristics include red or dark patches on the skin; burning, itching, swelling, hardening, and tightening of the skin; yellow spots on the whites of the eyes; joint stiffness with trouble moving or straightening the arms, hands, legs, or feet; pain deep in the hip bones or ribs; and muscle weakness.

Pharmacologic and Other Therapies

Because most patients equate migraine with headache exclusively, convincing them that symptoms other than headache are due to migraine may be difficult. Dizziness secondary to migraine usually responds to the same treatment used for migraine headaches.

The 3 broad classes of migraine headache treatment include reduction of risk factors, abortive medications, and prophylactic medical therapy. [27, 28, 29, 30] Vestibular rehabilitation therapy may be of benefit in patients with movement-associated disequilibrium.

Migraine and vestibular disease can coexist. Patients who meet the clinical criteria for Ménière disease should be treated appropriately for Ménière disease, even if a history of migraine headache exists.

Abortive medication and risk-factor reduction

In general, drugs used to abort migraine headaches have not been found effective in treating dizziness secondary to migraine. Reduction of risk factors includes an attempt to avoid certain conditions (eg, stress, anxiety, hypoglycemia, fluctuating estrogen, certain foods, smoking) that can trigger migraine. Elimination of birth control pills or estrogen replacement products may be helpful.

Prophylactic pharmacotherapy

Prophylactic medical therapy should be used when migraine-associated vertigo occurs several times a month, is continuous over several weeks or months, or has severely affected the patient's lifestyle. First-line prophylactic medications include calcium channel blockers (verapamil), tricyclic antidepressants (nortriptyline), and beta blockers (propranolol). Second-line treatments include topiramate, valproic acid, venlafaxine, and methysergide. Acetazolamide and lamotrigine have also been reported as an effective treatment by several authors. However, these 2 medications seem to be primarily effective for only the vestibular symptoms and not headaches. [31]

The actual mechanism of action for migraine control with these medications is unknown. However, the calcium channel blockers, tricyclic antidepressants, beta blockers, and methysergide are believed to block the release of neuropeptides into dural blood vessel walls, because of their antagonist effect on serotonin (5-HT)-2 receptors.

One class of prophylactic medication does not seem to be more effective than the others. Therefore, unless contraindicated, verapamil is often used initially, because this medication has the lowest side-effect profile among the prophylactic medications.

If dizziness is not controlled with one class of medication, another class should be used. If dizziness is controlled with one of these medications, the drug should be administered continuously for at least 1 year (except for methysergide, which requires a 3- to 4-week drug-free interval at 6mo). The medication can be restarted for another year if the dizziness returns after discontinuing therapy.

Dietary restrictions

Avoiding certain foods helps less than 25-30% of all people who experience migraines. In general, the

following foods should be avoided:

- Monosodium glutamate (MSG)
- Certain alcoholic beverages - Eg, red wine, port, sherry, scotch, bourbon
- Aged cheese - Eg, Colby, Roquefort, Brie, Gruyere, cheddar, bleu, mozzarella, Parmesan, Boursault, Romano
- Chocolate (including carob)
- Aspartame

MSG is often found in certain soups, Chinese food and fast food, soy sauce, yeast, yeast extract, meat tenderizers, seasoned salt, and several salad dressings.

An elimination diet for 1 month may be prescribed. If, after 1 month, symptoms are not better, diet modification is not helpful. If foods are a trigger for symptoms, the offending food(s) can be identified by adding back 1 food at a time until the symptoms return.

A food diary is an alternative option to an elimination diet, because certain foods cause migraine symptoms almost immediately (eg, red wine, MSG), whereas other foods (eg, chocolate, cheese) may cause symptoms the next day. The diary should include all foods consumed for 24 hours before the onset of a dizzy spell.

Vestibular rehabilitation therapy

Vestibular rehabilitation therapy is recommended when movement-associated dysequilibrium is present, either as the predominant symptom, or it may be a continuing symptom despite adequate vertigo control with prophylactic medication. In either case, vestibular rehabilitation is quite beneficial. However, this therapy is not indicated for the treatment of spontaneously occurring vertigo.

Consultations

Consultation with a neurologist is warranted if the patient has or develops focal neurologic deficits, if the patient develops migrainous infarction, or if the examining physician is uncomfortable using prophylactic medications that may be appropriate in the treatment of migraine-associated vertigo.

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Table 1. *CACNA1A* Gene Defects Associated With Autosomal Dominant Disorders With Neurologic Symptoms

GENE Defect	Syndrome	Symptoms and Signs
Point mutation	Familial hemiplegic migraine	Episodic hemiparesis for 60min or less, followed by headache; gaze-evoked and downbeat nystagmus may persist after spells
Point mutation	Episodic ataxia type 2 (EA2)	Episodic ataxia and vertigo, gaze-evoked and downbeat nystagmus, abnormal pursuit on electronystagmography (ENG)

CAG repeats	Spinocerebellar ataxia type 6 (SCA6)	Progressive ataxia, gaze-evoked and downbeat nystagmus, abnormal pursuit on ENG
Adapted from Tusa, 1999. [14]		

Table 2. A Comparison of the Symptoms of Migraine-Associated Vertigo and Ménière Disease

Symptom	Migraine-Associated Vertigo	Ménière Disease
Vertigo	May last >24h	Lasts up to 24h
Sensorineural hearing loss	Very uncommon; when present, often low frequency; very rarely progressive; may fluctuate in cases of basilar migraine	Nearly always progressive; most often unilateral; may be bilateral; fluctuation is common
Tinnitus	May be unilateral or bilateral; rarely obtrusive	May be unilateral or bilateral; often of significant intensity
Photophobia	Often present; may or may not be associated with dizziness	Never present unless a concurrent history of migraine exists

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Received income in an amount equal to or greater than $250 from: The Physicians Edge, Cliexa
 Received stock from RxRevu; Received ownership interest from Cerescan for consulting; .

Acknowledgements

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