Published in final edited form as:

Semin Neurol. 2010 April ; 30(2): 167–174. doi:10.1055/s-0030-1249225.

Migraine-Associated Vertigo: Diagnosis and Treatment

Yoon-Hee Cha, M.D.[1]

[1]Department of Neurology, University of California Los Angeles, Los Angeles, California

Abstract

Migraine-associated vertigo has become a well-recognized disease entity diagnosed based on a clinical history of recurrent vertigo attacks unexplained by other central or peripheral otologic abnormalities, which occurs in the patient with a history of migraine headaches. There is no international agreement on what spectrum of symptoms should be covered under this diagnosis, or what terminology should be used. The headaches and vestibular symptoms of migraine-associated vertigo may not be temporally associated, which often obscures the association. Diagnostic tests usually show nonspecific abnormalities that are also seen in patients with migraine who do not experience vestibular symptoms. Management generally follows the recommended treatment of migraine headaches, and includes dietary and lifestyle modifications and medical treatment with β blockers, calcium channel blockers, and tricyclic amines. Small case series show that acetazolamide and lamotrigine appear to be more effective for the vertigo attacks than headaches. Vestibular rehabilitation has also been shown to be helpful in several studies. In this review, the epidemiologic and clinical features of the disorder, as well as the current state of knowledge on pathophysiology, diagnostic testing, and treatment are described.

Keywords

Migraine; vertigo; vestibular; dizziness

The association of migraine and episodic vertigo has been well-established as several large studies have shown that the prevalence of migraine in patients with otherwise unexplained episodes of vertigo is significantly higher than the population prevalence of migraine.[1–3] Unfortunately, a wider recognition of migraine-associated vertigo as a distinct disease entity has also led to numerous variations on terminology and on criteria for diagnosis. Terms such as benign recurrent vertigo,[2,4–6] vestibular migraine,[7] migraine-associated dizziness,[8,9] migraine-associated vertigo,[10,11] migrainous vertigo,[12] and migraine-related vestibulopathy[13] are just a few. Some investigators restrict the definition to true spontaneous rotational vertigo[2,4]; others accept other illusions of motion and imbalance[12,14]; others are unclear as to what is meant by the term "vertigo."[11] Criteria for definite and probable migrainous vertigo proposed by Neuhauser et al[12] (Table 1) have been adopted in more recent investigations.[15–19] For the purposes of this review, the terminology used by each investigator will be used in the description of referenced studies.

Address for correspondence and reprint requests: Yoon-Hee Cha, M.D., Visiting Assistant Professor, Department of Neurology, University of California Los Angeles, 710 Westwood Plaza, Box 951769, Los Angeles, CA 90095 (yhcha@mednet.ucla.edu).

EPIDEMIOLOGY

Studies of the general population and those performed in clinics specializing in dizziness both report a higher than chance association of migraine and vertigo.[20] In a series of 200 patients seen consecutively in a dizziness clinic, 38% met International Headache Society (IHS) criteria for migraine with a lifetime prevalence of 7% of definite migrainous vertigo. In a group of 200 patients with migraine, 9% met criteria for definite migrainous vertigo compared with 0.5% of 200 control orthopedic patients.[12]

A German Telephone Health Survey screened 4869 participants for a history of vestibular vertigo defined as (1) rotational vertigo, (2) positional vertigo, or (3) recurrent dizziness with nausea and either oscillopsia or episodic imbalance. One-thousand three of these participants reported moderate to severe dizziness or vertigo and were able to complete both a screening interview and a detailed neurotologic interview. Of the 1003 participants, 24% reported a lifetime history of vestibular vertigo. The overall lifetime prevalence of vestibular vertigo was reported at 7.4%, a 1-year prevalence at 4.9%, and a 1-year incidence at 1.4%.[14] A subsequent study with this dataset showed a 3.2% prevalence of both migraine and vestibular vertigo in the same participant and a 0.9% prevalence of migrainous vertigo specifically.[21]

CLINICAL FEATURES

In general, the age of onset of migraine headaches precedes the onset of vertigo attacks by many years, which often obscures the association.[2,7,16] However, in individual patients, the age of onset of headaches and vertigo can occur simultaneously or vertigo attacks can precede the onset of headaches. The age of onset of migraine headaches is reported to be in the teens to early 20s, whereas the onset of vertigo averages in the 30 to 40s. Onset after age 50 appears to be less common.[1,2,4,10,12]

The difficulty in making the diagnosis often rests on the inconsistent temporal association between the headache and vestibular symptoms. The percentage of patients who report a consistent relationship between headache and vertigo is reported to be as low as 5%, but most reports indicate that ~30 to 70% of patients report at least some episodes of concurrent headache and vertigo.[2,4,7,9,22]

The majority of clinical reports of migrainous vertigo, regardless of the criteria used for vertigo, has shown that at least 30% of patients who meet the criteria for migraine headaches and vertigo have at least some spells of vertigo without any headache.[2,9,10,12,22] A large fraction of patients with migraine and vertigo spells experience some vertigo spells with migraine symptoms and others without, raising the question of whether the temporal association of the two conditions is really that critical or represents a difference in pathophysiology.[2,4,9]

The range of the duration of attacks may be seconds, minutes, hours, or days, but the most common durations reported are on the order of hours to days.[2,7,10,12] Though audiologic features like tinnitus, phonophobia, and aural fullness may be experienced during migrainous vertigo attacks, baseline audiometry is reported to be normal.[23] Most studies are

careful, however, to exclude patients with hearing loss when diagnosing migraine-associated vertigo, however. It must be borne in mind that if a significant hearing loss were found in a patient with recurrent vertigo and aural symptoms, they would meet the criteria for Ménière disease.[24]

RELATED DISORDERS

Benign Recurrent Vertigo of Childhood

Basser first reported on the benign nature of recurrent vertigo attacks in children, which usually occurred before the age of 8 and had a benign prognosis.[25] Fenichel later showed the connection between these early vertigo attacks and the later development of migraine headaches.[26] Long-term follow-up studies have shown that recurrent vertigo is one of the childhood periodic syndromes, such as cyclical vomiting and recurrent abdominal pain, which are precursors to migraine headaches.[27–30] Mild degrees of vestibular paresis and other less-specific vestibular function abnormalities have been noted in these patients.[25,31]

Basilar-type Migraine

In the second edition of The International Classification of Headache Disorders (ICHD-2) criteria, vertigo is recognized as an aura symptom of basilar-type migraine, but not as a separate migraine-equivalent syndrome.[32] Because of the often lack of temporal association with headache and the typically longer duration of vertigo than the 60-minute limit for aura, patients with migrainous vertigo rarely meet the criteria for basilar-type migraine.[2,7,12]

There are some differences in baseline vestibular function testing in patients who meet criteria for basilar-type migraine versus migrainous vertigo. One study showed that patients with basilar-type migraines showed more baseline saccadic dysmetria, are more prone to baseline motion sickness, and have more frequent attacks than patients with migrainous vertigo.[33] The prevalence of caloric paresis is also reported to be higher in patients with basilar-type migraine.[34,35]

Ménière Disease

The main competing diagnostic entity to migrainous vertigo is Ménière disease: both conditions present with spontaneous episodes of vertigo with a similar time course.[36] A prior separate designation of “cochlear Ménière” and “vestibular Ménière” was called into question when a much higher prevalence of migraine in vestibular Ménière was shown, and that these patients infrequently went on to develop cochlear symptoms.[37] Thus, the 1995 American Academy of Otolaryngology-Head and Neck Surgery criteria no longer recognizes this distinction.[24]

Previous reports that either used older criteria or no accepted criteria reported migraine prevalence between 22 to 76% in patients with Ménière disease.[37–39] A more recent study using strict criteria for definite Ménière disease and migraine showed that 56% of patients with Ménière disease also had a lifetime history of migraine. Forty-five percent of these patients always experienced migraine symptoms like headache, aura, and photophobia during their Ménière attacks.[40]

A history of migraine in a patient with Ménière disease may be relevant as an earlier age of onset, increased tendency for onset of bilateral symptoms, and a greater prevalence of a family history of vertigo was seen in such patients as compared with those without migraine.[41] That Ménière disease and migrainous vertigo may be related genetically was shown in a study of six families in which an index patient with Ménière disease had a first-degree relative who suffered from migrainous vertigo without auditory symptoms. In two of these cases, the relative was an identical twin.[42]

DIAGNOSTIC TESTING

Unilateral vestibular pareses in patients with vestibular migraine have been reported with a prevalence of between 8.1 to 23.8%.[7,9,13,16,22,43,44] Two studies have shown that patients with basilar-type migraine specifically have a higher prevalence of a reduced vestibular response (60% in each report),[34,35] including 12% who were reported as having bilateral reduced vestibular responses.[35] The specificity of this is unclear as vestibular paresis and nonspecific ocular motor abnormalities have also been reported in patients with migraine without a history of vertigo.[45,46] Vestibular hyperresponsiveness was reported in five of 30 patients with migrainous vertigo in a recent study, using a combined slow-phase velocity of 140 deg/s as criteria for hyperresponsiveness.[17]

An interesting aspect of caloric testing, incidentally noted during one investigation of patients with migrainous vertigo, was that patients with *definite* migrainous vertigo were four times more likely to experience emesis and abort testing than patients with *probable* migrainous vertigo or other vestibular disorders.[19] A relatively newer addition to the vestibular function testing armamentarium are vestibular evoked myogenic potentials (VEMPs). Vestibular evoked myogenic potentials permit the evaluation of a vestibulocollic reflex by using sound stimulation of the saccule to elicit an inhibitory postsynaptive potential in the ipsilateral sternocleidomastoid muscle.[47] They are an important complement to traditional caloric testing as they provide information on otolith function (saccule), which sends information through the inferior branch of the vestibular nerve and acts through a neural pathway that descends from the vestibular nuclei to the cervical cord. Caloric irrigation stimulates the horizontal canal, which transmits information through the superior vestibular nerve and synapses on brainstem neurons that ascend from the vestibular nuclei to ocular motor nuclei.[48]

Vestibular evoked myogenic potentials have been shown to be delayed or absent in 10 of 20 patients with basilar artery-type migraine, a finding also seen in brainstem stroke. These abnormalities were reported to reverse with successful treatment of the headaches.[49,50] These findings were not replicated in patients with vestibular migraine.

Migraine sufferers do show a potentiation in VEMP responses by the end of the click stimulus train, whereas normal controls show a habituating response.[51] Overall VEMP amplitudes are lower in individuals with migraine than controls without differences between patients with a history of vertigo.[15,52] Because the amplitude of the response is lower, but the response does not habituate, these studies suggest that there is abnormal brainstem processing of the vestibular stimuli.

A clear central or peripheral localization cannot be ascribed for the ocular motor abnormalities in migrainous vertigo. Nonlocalizing vestibular abnormalities such as static positional nystagmus, directional preponderance, and spontaneous nystagmus have been observed in patients with migraine-associated dizziness.[9] von Brevern performed eye movement recordings in 20 patients during an acute migrainous vertigo attack and found central features in 10 patients, peripheral features in three, and indeterminant findings in seven.[53]

PATHOGENESIS

Genetic Risk

Case series report a genetic risk factor prominent in patients with migraine-associated vertigo, but the risk cannot be attributed to any specific genetic mutation. A study of 24 patients with benign recurrent vertigo, as well as 220 family members who underwent a structured interview, showed that 40% of first-degree relatives also experienced recurrent vertigo attacks. This was in contrast to only 2% of unrelated spouses.[4] A genome wide association study of 20 families with an apparent autosomal dominant transmission of benign recurrent vertigo showed linkage to 22q12 with a logarithm of odds (LOD) score of 4.02, but there was marked genetic heterogeneity.[54] Given the higher prevalence of migraine in women, one study examined the association of migraine-associated vertigo with the progesterone receptor (PGR) and the estrogen receptor (ESR1) in 150 women with migraine-associated vertigo with 145 controls. A single nucleotide polymorphism (SNP) at position rs1042838 was found to be in high linkage disequilibrium with the PROGINS variant of the progesterone receptor.[55]

Though migrainous vertigo behaves much as other episodic disorders caused by ion channel mutations, it does not appear to be associated with the genes known to cause familial hemiplegic migraine types I (CACNA1A), II (ATP1A2), or III (SCN1A).[56]

Vasospasm

A role for reversible vasospasm in the posterior circulation has been proposed based on the association of vertigo attacks with other symptoms often attributed to vasospasm such as amaurosis fugax, hemiplegia, and angina.[57] It has also been proposed as a mechanism for ischemic damage to the inner ear.[58] Reversible vasospasm has been observed in retinal migraine, which can be treated with calcium channel blockers, a class of drugs commonly used in migraine-associated vertigo.[59,60]

The CAMERA (Cerebral Abnormalities in Migraine, an Epidemiological Risk Analysis) Study recently reported an odds ratio of 13.7 for the development of subclinical infarcts in the posterior circulation, given a history of migraine with aura.[61] These studies provide circumstantial evidence that a vasospastic process in migraine may contribute to small vessel ischemic disease in the posterior circulation, which could theoretically cause inner ear symptoms.

Neuropeptides

Cutrer and Baloh first proposed a role for calcitonin gene-related peptide (CGRP) in migraine-associated dizziness in a case series of 91 patients with migraine-associated dizziness.[9] They proposed that the release of neuroactive peptides, including CGRP, could increase firing of primary afferent vestibular nerves and lead to symptoms such as vertigo or motion sensitivity. This hypothesis is supported by early work on rat brain slices with retrograde tracing techniques, which revealed the presence of neurons in the caudal pons that provide efferent vestibular projections to vestibular hair cells. These efferent fibers coexpress CGRP and choline acetyltransferase (ChAT), a marker for acetylcholine expression.[62] Application of CGRP to the lateral line of frogs increases afferent activity, showing that efferent vestibular activity may play a role in modulating afferent activity.[63]

A recent trial of the CGRP antagonist MK-0974 (telcagepant®; Merck & Co., Inc., Whitehouse Station, NJ) compared with rizatriptan showed a similar 2-hour treatment response for migraine headache and was well-tolerated.[64] Testing the efficacy of this class of drugs in migrainous vertigo would not only be logical and potentially increase treatment choices, it would also add information regarding the pathophysiology of the disorder.

TREATMENT

Symptomatic

In the absence of trials of acute therapy for vertigo attacks, the mainstay of treatment remains the use of vestibular suppressants such as anticholinergics (scopolamine, homatropine), antihistamines (meclizine, hydroxyzine, promethazine, diphenhydramine, dimenhydrinate), anti-dopaminergics (prochlorperazine, metoclopramide), and benzodiazepines.[65] The mechanism of action of these drugs are not pure, however, and are often complicated by sedation, cognitive slowing, and extrapyramidal side effects.

The only randomized placebo controlled trial of a triptan for the treatment of acute migrainous vertigo was inconclusive due to small sample size. In this study, three of eight attacks (38%) of vertigo treated with 2.5 mg of zolmitriptan versus two of nine (22%) attacks of vertigo treated with placebo improved from moderate/severe to mild/none.[66] In a related study, five patients with a history of migrainous vertigo were compared with five patients with a history of migraine headaches without vertigo in a motion sickness provocation study. Patients were randomized in a crossover trial to receive 10 mg of rizatriptan or placebo prior to motion sickness provocation. Only patients with a history of migrainous vertigo received benefit from pretreatment with rizatriptan suggesting that they are more sensitive to changes in serotonin tone.[67]

Preventative

MIGRAINE HEADACHE PREVENTION STRATEGY—The efficacy of typical migraine prophylactic medications on the treatment of migrainous vertigo was examined prospectively in 53 patients who experienced recurrent vertigo or dizziness attacks with either a personal or family history of migraine.[68] Forty-nine patients experienced recurrent vertigo attacks. Patients were treated with propranolol, metoprolol, flunarizine,

amitriptyline, or clonazepam and could switch between medications based on efficacy. Six-month follow-up was available on 36 patients (33 of whom experienced rotational vertigo). Twenty-seven (81.8%) had a greater than 50% reduction in their vertigo attacks.

Substantial reductions in migraine-related vestibular symptoms with combined medical and lifestyle/diet changes have been reported in a study of 89 patients. Treatment was tailored to each patient's symptoms and consisted of medical therapy, physical therapy, lifestyle and diet modifications and at times, acupuncture.[43] Using a combination of therapies, 92% of the patients reported substantial reduction in symptoms. However, it should be noted that a large percentage of these patients were treated with benzodiazepines, which are associated with problems of tolerance and dependency.

A later study of 81 patients with migraine-associated dizziness used a stepwise approach and reported significant improvement (>75% reduction) of both headache and dizziness symptoms in 72% of subjects. Only 5% reported no improvement at all. The algorithm started with dietary modification followed by low-dose nortriptyline, then a β blocker. If no response was seen after at least 4 weeks of therapy, the patient was referred to a neurologist. Other therapies added by the neurologist included calcium channel blockers, selective serotonin reuptake inhibitors (SSRIs), valproic acid, carbamazepine, and gabapentin.[44]

An indication that medical therapy is more effective than conservative therapy with lifestyle modification, physical therapy, and muscle relaxation was supported by a recent study of 100 patients with migrainous vertigo (29 definite, 71 probable). The majority of the 74 medically treated patients received β blockers and showed statistically significant reductions in the frequency, duration, and intensity of vertigo attacks, whereas the 26 conservatively treated patients only noted decreased intensity.[18]

TOPIRAMATE—A small open label prospective trial of topiramate was performed on 10 patients with an average total dose of 100 mg a day. All of the patients had reported auditory symptoms, eight of which were unilateral, two bilateral. The authors reported that the patients became symptom free during a follow-up period of 6 to 16 months, but there were no details provided for the clinical features of migrainous vertigo, the frequency of attacks, or the nature of the auditory symptoms.[69] A follow-up study would certainly be warranted, as topiramate has become a commonly used drug for migraine headache prevention.

LAMOTRIGINE—A retrospective chart review of 19 patients with migraine and migraine-related vertigo treated with lamotrigine titrated to 100 mg daily, showed reduction in the frequency but not the duration of vertigo attacks.[70] The vertigo frequency was reduced by 50% in 18 patients and was unchanged in one patient. Five patients reported complete relief. There was no significant reduction in headache frequency, similar to prior reports that lamotrigine is more effective for auras than for headache.[71,72]

ACETAZOLAMIDE—Episodic vertigo may be a feature of episodic ataxia type II (EAII), a channelopathy caused by mutations in the P/Q type voltage gated calcium channel.[73] Acetazolamide markedly reduces attacks in EA II.[74–76] Using this rationale, a family with

episodic vertigo, migraine, and essential tremor were treated with acetazolamide with marked improvement of the vertigo attacks and tremor.[77]

FLUNARIZINE, LOMERIZINE, BETAHISTINE—Flunarizine has antihistaminergic and calcium channel blocking properties, and is a frequently used medication for migraine prophylaxis in Europe.[7,78,79] A recent trial of lomerizine, also a calcium channel blocker in 22 patients with migraine-associated vertigo, showed significant symptom reduction in 87% of patients.[80] Betahistine (L-histidine) is the immediate precursor to histamine and has been used in Europe (not available in the United States) for the treatment of Ménière disease.[81] There is also good support for it's use in migrainous vertigo.[82,83]

Vestibular Rehabilitation

Focused vestibular rehabilitation has been shown to benefit patients with migraine-related vestibulopathy as well as patients with migraine who have additional vestibular disorders. One early study compared 14 patients with a diagnosis of migraine-related vestibulopathy to 25 patients with migraine and apparently unrelated vestibular dysfunction, and showed that both groups improved significantly on both subjective and objective outcome measures.[84] This finding has been supported by another study of 34 patients with migraine-associated dizziness using IHS criteria and vestibular symptoms defined as imbalance, true vertigo, dizziness, or unsteadiness.[44]

A study comparing 30 patients with a vestibular disorder and migraine to 30 patients with a vestibular disorder without migraine showed that the migraine sufferers had a greater perception of disability, even though their physical performance measures were the same.[85] Development and persistence of concurrent anxiety and affective disorders are more common in patients with vestibular migraine than in patients with other vestibular disorders.[86,87] Thus, additional emotional or psychosocial factors may need to be addressed in migraine patients with vestibular symptoms to maximize treatment efficacy.

CONCLUSION

Growing recognition of migraine-associated vertigo as a separate recognizable disorder has led to a recent increase in publications on epidemiology, diagnostic testing, and treatment. Though many studies are using the Neuhauser et al[12] criteria for definite and probable migrainous vertigo, there is still a lack of consensus on diagnostic criteria.

The diagnosis still rests on the association of episodic vertigo with a personal or strong family history of migraine headaches and ruling out otologic causes of symptoms, namely Ménière disease. Decreases in vestibular responses on caloric and VEMP testing are common in patients with migraine regardless of whether they have a history of vertigo, so such tests cannot be used to distinguish competing diagnoses. As yet, the mechanism for vertigo symptoms has not been clarified, but support for competing possibilities warrants clinical trials with each mechanism in mind.

Acute therapies need to be explored, as currently available vestibular suppressants are often sedating and occasionally associated with cognitive and extrapyramidal side effects. The

limited data on the use of triptans in the treatment of acute vertigo suggests that they are not associated with undue side effects, but there is little evidence for their efficacy over placebo. Further trials of triptans and possibly the CGRP antagonists would not only expand the repertoire, but also reveal important aspects of the pathophysiology of the disorder.

Preventative treatment largely remains based on those that are effective in migraine headaches, though there is some indication that lamotrigine and acetazolamide may be more effective for the vertigo attacks than for headaches. Topiramate may be effective but requires more clinical data. Most treatment rests on the use of β blockers, calcium channel blockers, tricyclic amines, and conservative measures like lifestyle and dietary modification. If available, a focused rehabilitation program directed toward migrainous vertigo, with attention paid to the additional mood and anxiety disorders, is likely to be helpful.

Acknowledgments

This work has been supported by NIH grant U54 RR019482 and a Research Seed Grant from the UCLA Department of Neurology.

References

1. Lee H, Sohn SI, Jung DK, et al. Migraine and isolated recurrent vertigo of unknown cause. Neurol Res. 2002; 24(7):663–665. [PubMed: 12392202]
2. Cha YH, Lee H, Santell LS, Baloh RW. Association of benign recurrent vertigo and migraine in 208 patients. Cephalalgia. 2009; 29(5):550–555. [PubMed: 19170697]
3. Aragones JM, Fortes-Rego J, Fuste J, Cardozo A. Migraine: an alternative in the diagnosis of unclassified vertigo. Headache. 1993; 33(3):125–128. [PubMed: 8486507]
4. Oh AK, Lee H, Jen JC, Corona S, Jacobson KM, Baloh RW. Familial benign recurrent vertigo. Am J Med Genet. 2001; 100(4):287–291. [PubMed: 11343320]
5. Slater R. Benign recurrent vertigo. J Neurol Neurosurg Psychiatry. 1979; 42(4):363–367. [PubMed: 458483]
6. Lee H, Jen JC, Wang H, et al. A genome-wide linkage scan of familial benign recurrent vertigo: linkage to 22q12 with evidence of heterogeneity. Hum Mol Genet. 2006; 15(2):251–258. [PubMed: 16330481]
7. Dieterich M, Brandt T. Episodic vertigo related to migraine (90 cases): vestibular migraine? J Neurology. 1999; 246:883–892.
8. Bikhazi P, Jackson C, Ruckenstein MJ. Efficacy of antimigrainous therapy in the treatment of migraine-associated dizziness. Am J Otol. 1997; 18(3):350–354. [PubMed: 9149830]
9. Cutrer FM, Baloh RW. Migraine-associated dizziness. Headache. 1992; 32(6):300–304. [PubMed: 1399552]
10. Brantberg K, Trees N, Baloh RW. Migraine-associated vertigo. Acta Otolaryngol. 2005; 125(3): 276–279. [PubMed: 15966697]
11. Savundra PA, Carroll JD, Davies RA, Luxon LM. Migraine-associated vertigo. Cephalalgia. 1997; 17(4):505–510. discussion 487. [PubMed: 9209771]
12. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology. 2001; 56(4):436–441. [PubMed: 11222783]
13. Cass SP, Furman JM, Ankerstjerne K, Balaban C, Yetiser S, Aydogan B. Migraine-related vestibulopathy. Ann Otol Rhinol Laryngol. 1997; 106(3):182–189. [PubMed: 9078929]
14. Neuhauser HK, von Brevern M, Radtke A, et al. Epidemiology of vestibular vertigo: a neurotologic survey of the general population. Neurology. 2005; 65(6):898–904. [PubMed: 16186531]
15. Baier B, Stieber N, Dieterich M. Vestibular-evoked myogenic potentials in vestibular migraine. J Neurol. 2009; 256(9):1447–1454. [PubMed: 19377861]

16. Celebisoy N, Gökçay F, Sirin H, Biçak N. Migrainous vertigo: clinical, oculographic and posturographic findings. Cephalalgia. 2008; 28(1):72–77. [PubMed: 17999681]
17. Teggi R, Colombo B, Bernasconi L, Bellini C, Comi G, Bussi M. Migrainous vertigo: results of caloric testing and stabilometric findings. Headache. 2009; 49(3):435–444. [PubMed: 19220504]
18. Baier B, Winkenwerder E, Dieterich M. "Vestibular migraine": effects of prophylactic therapy with various drugs. A retrospective study. J Neurol. 2009; 256(3):436–442. [PubMed: 19266150]
19. Vitkovic J, Paine M, Rance G. Neuro-otological findings in patients with migraine- and nonmigraine-related dizziness. Audiol Neurootol. 2008; 13(2):113–122. [PubMed: 18057875]
20. Neuhauser HK. Epidemiology of vertigo. Curr Opin Neurol. 2007; 20(1):40–46. [PubMed: 17215687]
21. Neuhauser HK, Radtke A, von Brevern M, et al. Migrainous vertigo: prevalence and impact on quality of life. Neurology. 2006; 67(6):1028–1033. [PubMed: 17000973]
22. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain. 1984; 107(Pt 4):1123–1142. [PubMed: 6334543]
23. Battista RA. Audiometric findings of patients with migraine-associated dizziness. Otol Neurotol. 2004; 25(6):987–992. [PubMed: 15547431]
24. Committee on Hearing and Equilibrium Guidelines for the Diagnosis and Evaluation of Therapy in Menière's disease. American Academy of Otolaryngology-Head and Neck Foundation, Inc. Otolaryngol Head Neck Surg. 1995; 113(3):181–185. [PubMed: 7675476]
25. Basser LS. Benign paroxysmal vertigo of childhood (a variety of vestibular neuronitis). Brain. 1964; 87:141–152. [PubMed: 14152207]
26. Fenichel GM. Migraine as a cause of benign paroxysmal vertigo of childhood. J Pediatr. 1967; 71(1):114–115. [PubMed: 5293847]
27. Lanzi G, Balottin U, Fazzi E, Rosano FB. The periodic syndrome in pediatric migraine sufferers. Cephalalgia. 1983; 3(Suppl 1):91–93. [PubMed: 6616613]
28. Lanzi G, Balottin U, Fazzi E, Tagliasacchi M, Manfrin M, Mira E. Benign paroxysmal vertigo of childhood: a long-term follow-up. Cephalalgia. 1994; 14(6):458–460. [PubMed: 7697708]
29. Drigo P, Carli G, Laverda AM. Benign paroxysmal vertigo of childhood. Brain Dev. 2001; 23(1): 38–41. [PubMed: 11226728]
30. Abu-Arafeh I, Russell G. Paroxysmal vertigo as a migraine equivalent in children: a population-based study. Cephalalgia. 1995; 15(1):22–25. discussion 4. [PubMed: 7758093]
31. Marcelli V, Piazza F, Pisani F, Marciano E. Neuro-otological features of benign paroxysmal vertigo and benign paroxysmal positioning vertigo in children: a follow-up study. Brain Dev. 2006; 28(2): 80–84. [PubMed: 16168599]
32. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders, 2nd Ed. Cephalalgia. 2004; 24(Suppl 1):1–160.
33. Wang CT, Lai MS, Young YH. Relationship between basilar-type migraine and migrainous vertigo. Headache. 2009; 49(3):426–434. [PubMed: 19178580]
34. Eviatar L. Vestibular testing in basilar artery migraine. Ann Neurol. 1981; 9(2):126–130. [PubMed: 7235628]
35. Olsson JE. Neurotologic findings in basilar migraine. Laryngoscope. 1991; 101(1 Pt 2, Suppl 52): 1–41.
36. Perez-Garrigues H, Lopez-Escamez JA, Perez P, et al. Time course of episodes of definitive vertigo in Meniere's disease. Arch Otolaryngol Head Neck Surg. 2008; 134(11):1149–1154. [PubMed: 19015442]
37. Rassekh CH, Harker LA. The prevalence of migraine in Menière's disease. Laryngoscope. 1992; 102(2):135–138. [PubMed: 1738284]
38. Parker W. Menière's disease. Etiologic considerations. Arch Otolaryngol Head Neck Surg. 1995; 121(4):377–382. [PubMed: 7702810]
39. Hinchcliffe R. Headache and Menière's disease. Acta Otolaryngol. 1967; 63(4):384–390. [PubMed: 6033661]
40. Radtke A, Lempert T, Gresty MA, Brookes GB, Bronstein AM, Neuhauser H. Migraine and Ménière's disease: is there a link? Neurology. 2002; 59(11):1700–1704. [PubMed: 12473755]

41. Cha YH, Brodsky J, Ishiyama G, Sabatti C, Baloh RW. The relevance of migraine in patients with Meńière's disease. Acta Otolaryngol. 2007; 127(12):1241–1245. [PubMed: 17851970]
42. Cha YH, Kane MJ, Baloh RW. Familial clustering of migraine, episodic vertigo, and Ménière's disease. Otol Neurotol. 2008; 29(1):93–96. [PubMed: 18046258]
43. Johnson GD. Medical management of migraine-related dizziness and vertigo. Laryngoscope. 1998; 108(1 Pt 2):1–28.
44. Reploeg MD, Goebel JA. Migraine-associated dizziness: patient characteristics and management options. Otol Neurotol. 2002; 23(3):364–371. [PubMed: 11981397]
45. Bir LS, Ardiç FN, Kara CO, Akalin O, Pinar HS, Celiker A. Migraine patients with or without vertigo: comparison of clinical and electronystagmographic findings. J Otolaryngol. 2003; 32(4): 234–238. [PubMed: 14587563]
46. Kuritzky A, Toglia UJ, Thomas D. Vestibular function in migraine. Headache. 1981; 21(3):110–112. [PubMed: 6973556]
47. Zhou G, Cox LC. Vestibular evoked myogenic potentials: history and overview. Am J Audiol. 2004; 13(2):135–143. [PubMed: 15903139]
48. Welgampola MS, Colebatch JG. Characteristics and clinical applications of vestibular-evoked myogenic potentials. Neurology. 2005; 64(10):1682–1688. [PubMed: 15911791]
49. Liao L-J, Young Y-H. Vestibular evoked myogenic potentials in basilar artery migraine. Laryngoscope. 2004; 114(7):1305–1309. [PubMed: 15235366]
50. Chen C-H, Young Y-H. Vestibular evoked myogenic potentials in brainstem stroke. Laryngoscope. 2003; 113(6):990–993. [PubMed: 12782810]
51. Allena M, Magis D, De Pasqua V, Schoenen J, Bisdorff AR. The vestibulo-collic reflex is abnormal in migraine. Cephalalgia. 2007; 27(10):1150–1155. [PubMed: 17784856]
52. Roceanu A, Allena M, De Pasqua V, Bisdorff A, Schoenen J. Abnormalities of the vestibulo-collic reflex are similar in migraineurs with and without vertigo. Cephalalgia. 2008; 28(9):988–990. [PubMed: 18624802]
53. von Brevern M, Zeise D, Neuhauser H, Clarke AH, Lempert T. Acute migrainous vertigo: clinical and oculographic findings. Brain. 2005; 128(Pt 2):365–374. [PubMed: 15601663]
54. Lee H, Jen JC, Cha YH, Nelson SF, Baloh RW. Phenotypic and genetic analysis of a large family with migraine-associated vertigo. Headache. 2008; 48(10):1460–1467. [PubMed: 18081823]
55. Lee H, Sininger L, Jen JC, Cha YH, Baloh RW, Nelson SF. Association of progesterone receptor with migraine-associated vertigo. Neurogenetics. 2007; 8(3):195–200. [PubMed: 17609999]
56. von Brevern M, Ta N, Shankar A, et al. Migrainous vertigo: mutation analysis of the candidate genes CACNA1A, ATP1A2, SCN1A, and CACNB4. Headache. 2006; 46(7):1136–1141. [PubMed: 16866717]
57. Viirre ES, Baloh RW. Migraine as a cause of sudden hearing loss. Headache. 1996; 36(1):24–28. [PubMed: 8666532]
58. Lee H, Whitman GT, Lim JG, et al. Hearing symptoms in migrainous infarction. Arch Neurol. 2003; 60(1):113–116. [PubMed: 12533097]
59. Tippin J, Corbett JJ, Kerber RE, Schroeder E, Thompson HS. Amaurosis fugax and ocular infarction in adolescents and young adults. Ann Neurol. 1989; 26(1):69–77. [PubMed: 2774503]
60. Winterkorn JM, Kupersmith MJ, Wirtschafter JD, Forman S. Brief report: treatment of vasospastic amaurosis fugax with calcium-channel blockers. N Engl J Med. 1993; 329(6):396–398. [PubMed: 8326973]
61. Kruit M, van Buchem M, Launer L, Terwindt G, Ferrari M. Migraine is associated with an increased risk of deep white matter lesions, subclinical posterior circulation infarcts and brain iron accumulation: the population-based MRI CAMERA study. Cephalalgia. 2009 Jun 8. (Epub ahead of print).
62. Ohno K, Takeda N, Yamano M, Matsunaga T, Tohyama M. Coexistence of acetylcholine and calcitonin gene-related peptide in the vestibular efferent neurons in the rat. Brain Res. 1991; 566(1–2):103–107. [PubMed: 1814528]

63. Sewell WF, Starr PA. Effects of calcitonin gene-related peptide and efferent nerve stimulation on afferent transmission in the lateral line organ. J Neurophysiol. 1991; 65(5):1158–1169. [PubMed: 1651373]
64. Ho TW, Mannix LK, Fan X, et al. MK-0974 Protocol 004 study group. Randomized controlled trial of an oral CGRP receptor antagonist, MK-0974, in acute treatment of migraine. Neurology. 2008; 70(16):1304–1312. [PubMed: 17914062]
65. Zajonc TP, Roland PS. Vertigo and motion sickness. Part II: Pharmacologic treatment. Ear Nose Throat J. 2006; 85(1):25–35. [PubMed: 16509240]
66. Neuhauser H, Radtke A, von Brevern M, Lempert T. Zolmitriptan for treatment of migrainous vertigo: a pilot randomized placebo-controlled trial. Neurology. 2003; 60(5):882–883. [PubMed: 12629256]
67. Marcus DA, Furman JM. Prevention of motion sickness with rizatriptan: a double-blind, placebo-controlled pilot study. Med Sci Monit. 2006; 12(1):PI1–PI7. [PubMed: 16369474]
68. Maione A. Migraine-related vertigo: diagnostic criteria and prophylactic treatment. Laryngoscope. 2006; 116:1782–1786. [PubMed: 17003732]
69. Carmona S, Settecase N. Use of topiramate (topamax) in a subgroup of migraine-vertigo patients with auditory symptoms. Ann N Y Acad Sci. 2005; 1039:517–520. [PubMed: 15827012]
70. Bisdorff AR. Treatment of migraine related vertigo with lamotrigine an observational study. Bull Soc Sci Med Grand Duche Luxemb. 2004; (2):103–108. [PubMed: 15641316]
71. Lampl C, Buzath A, Klinger D, Neumann K. Lamotrigine in the prophylactic treatment of migraine aura—a pilot study. Cephalalgia. 1999; 19(1):58–63. [PubMed: 10099861]
72. D'Andrea G, Granella F, Cadaldini M, Manzoni GC. Effectiveness of lamotrigine in the prophylaxis of migraine with aura: an open pilot study. Cephalalgia. 1999; 19(1):64–66. [PubMed: 10099862]
73. Ophoff RA, Terwindt GM, Vergouwe MN, et al. Familial hemiplegic migraine and episodic ataxia type-2 are caused by mutations in the Ca2+ channel gene CACNL1A4. Cell. 1996; 87(3):543–552. [PubMed: 8898206]
74. Jen J, Kim GW, Baloh RW. Clinical spectrum of episodic ataxia type 2. Neurology. 2004; 62(1): 17–22. [PubMed: 14718690]
75. Zasorin NL, Baloh RW, Myers LB. Acetazolamide-responsive episodic ataxia syndrome. Neurology. 1983; 33(9):1212–1214. [PubMed: 6684259]
76. Calandriello L, Veneziano L, Francia A, et al. Acetazolamide-responsive episodic ataxia in an Italian family refines gene mapping on chromosome 19p13. Brain. 1997; 120(Pt 5):805–812. [PubMed: 9183251]
77. Baloh RW, Foster CA, Yue Q, Nelson SF. Familial migraine with vertigo and essential tremor. Neurology. 1996; 46(2):458–460. [PubMed: 8614512]
78. Rascol O, Hain TC, Brefel C, Benazet M, Clanet M, Montastruc JL. Antivertigo medications and drug-induced vertigo. A pharmacological review. Drugs. 1995; 50(5):777–791. [PubMed: 8586026]
79. Rascol O, Clanet M, Montastruc JL. Calcium antagonists and the vestibular system: a critical review of flunarizine as an antivertigo drug. Fundam Clin Pharmacol. 1989; 3(Suppl):79s–87s. [PubMed: 2693294]
80. Iwasaki S, Ushio M, Chihara Y, Ito K, Sugasawa K, Murofushi T. Migraine-associated vertigo: clinical characteristics of Japanese patients and effect of lomerizine, a calcium channel antagonist. Acta Otolaryngol Suppl. 2007; (559):45–49. [PubMed: 18340570]
81. Schmidt JT, Huizing EH. The clinical drug trial in Menière's disease with emphasis on the effect of betahistine SR. Acta Otolaryngol Suppl. 1992; 497:1–189. [PubMed: 1496890]
82. Brandt T, Zwergal A, Strupp M. Medical treatment of vestibular disorders. Expert Opin Pharmacother. 2009; 10(10):1537–1548. [PubMed: 19527184]
83. de Bock GH, Eelhart J, van Marwijk HW, Tromp TP, Springer MP. A postmarketing study of flunarizine in migraine and vertigo. Pharm World Sci. 1997; 19(6):269–274. [PubMed: 9443168]
84. Whitney SL, Wrisley DM, Brown KE, Furman JM. Physical therapy for migraine-related vestibulopathy and vestibular dysfunction with history of migraine. Laryngoscope. 2000; 110(9): 1528–1534. [PubMed: 10983955]

85. Wrisley DM, Whitney SL, Furman JM. Vestibular rehabilitation outcomes in patients with a history of migraine. Otol Neurotol. 2002; 23(4):483–487. [PubMed: 12170150]

86. Best C, Eckhardt-Henn A, Tschan R, Dieterich M. Psychiatric morbidity and comorbidity in different vestibular vertigo syndromes. Results of a prospective longitudinal study over one year. J Neurol. 2009; 256(1):58–65. [PubMed: 19221849]

87. Eckhardt-Henn A, Best C, Bense S, et al. Psychiatric comorbidity in different organic vertigo syndromes. J Neurol. 2008; 255(3):420–428. [PubMed: 18338198]

Table 1

Proposed Criteria for Probable and Definite Migrainous Vertigo

Definite migrainous vertigo
Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance, i.e., sensation of imbalance or illusory self or object motion that is provoked by head motion)
Migraine according to the IHS criteria *
At least one of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual, or other auras
Other causes ruled out by appropriate investigations
Probable migrainous vertigo
Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance)
At least one of the following: migraine according to the criteria of the IHS *; migrainous symptoms during vertigo, migraine-specific precipitants of vertigo, e.g., specific foods, sleep irregularities, hormonal changes; response to antimigraine drugs
Other causes ruled out by appropriate investigations

* Mild, did not interfere with daily activities; moderate, interfered with but did not impede daily activities; severe, could not continue daily activity.

IHS, International Headache Society.

From Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology 2001;56(4):436–441.