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Vestibular Migraine: How to Sort it Out and What to Do About it

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Background: Vestibular migraine (VM) is the most common neurologic cause of vertigo in adults and results in significant utilization of health care resources, but remains under-recognized and underdiagnosed.
Evidence Acquisition: Review of literature in PubMed using the following terms: vestibular migraine, migraine-associated vertigo, vertiginous migraine, benign recurrent vertigo, migraine-associated dizziness, migraine, migraine treatment, Meniere disease (MD), vertebrobasilar ischemia (VBI), posterior circulation stroke, benign paroxysmal positional vertigo, and episodic-ataxia Type 2 (EA2).
Results: VM can manifest with a variety of vestibular symptoms, including spontaneous vertigo, triggered vertigo, positional vertigo, and head-motion dizziness. Patients may report more than 1 vestibular symptom. Episodes of vertigo are often, but not always, accompanied by headache. Auditory symptoms are frequently associated with VM attacks and may mimic the manifestations of MD. Other migrainous features that accompany VM attacks include photophobia, phonophobia, osmophobia, and visual aura. Interictally, patients may suffer from persistent dizziness or isolated paroxysmal vestibular symptoms. Mood disorders (particularly anxiety) are often found in VM. Abnormal neuro-otologic findings are not uncommon in patients with VM. Differential diagnoses for VM include MD, VBI, EA2, and migraine with brainstem aura. For rescue treatment, triptans, vestibular suppressants, and/or antiemetic agents may be considered. Pharmacologic migraine preventives (antiepileptics, beta-blockers, and antidepressants) are often useful.
Conclusions: The keys to correctly diagnosing VM is identifying a relationship between vestibular symptoms and migrainous features and being aware of the heterogeneity of manifestations of this enigmatic, but treatable, condition. The principles of treatment of VM include rescue therapy, lifestyle modification, nonpharmacologic migraine preventives, pharmacologic migraine prophylaxis, and treatment of comorbidities.

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A relationship between vertigo and migraine has been recognized since the time of ancient Greece. In the second century AD, Aretaeus of Cappadocia observed attacks of vertigo that were associated with migraine headache ("heterocrania"), photophobia ("avoid light and feel relief when in the dark"), possible visual aura ("flying threads float before their eyes"), nausea, vomiting, and even nystagmus ("the eyes… move to and fro forcedly") (1). The term "vestibular migraine" (VM) was coined in 1999 (2), and in 2012, the International Headache Society and Bárány Society published consensus criteria for the diagnosis of VM (3), which have been adopted by the International Classification of Headache Disorders (ICHD-3) (4).

METHODS

We reviewed PubMed for the following terms: vestibular migraine, migraine-associated vertigo, vertiginous migraine, benign recurrent vertigo, migraine-associated dizziness, migraine, migraine treatment, Meniere disease (MD), vertebrobasilar ischemia (VBI), posterior circulation stroke, benign paroxysmal positional vertigo (BPPV), and episodic-ataxia Type 2 (EA2).

DEMOGRAPHICS

VM has a prevalence of between 1% and 2.7% of the adult population and is the most common neurologic cause of vertigo in adults (5,6), but remains a very underdiagnosed condition (5–7). VM predominantly affects women (1.5–5.6:1 female preponderance) in their late 30s to mid 40s (2,5,8–21). Preceding the onset of vestibular symptoms, most patients have a history of migraine headache

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(2,13,16,19,21,22) and motion sickness (8,12,19,21,23,24). In addition, a family history of migraine is common (2,9,10,12,19,21).

CLINICAL FINDINGS

A wide variety of vestibular symptoms may be described by patients with VM, which leads to diagnostic confusion, and explain why VM is under-recognized. The different vestibular symptoms that occur during VM attacks include spontaneous vertigo (2,5,9,10,18–21,25–28), positional vertigo (2,5,8,12,15,18,19,21,23,28–30), head motion-induced dizziness/vertigo (5,9,10,15,18,21,23,28), postural unsteadiness (5,14,17,19,21,27), oscillopsia (5,15,19,21), visually induced dizziness/vertigo (19,21,23), and directional pulsion (19,21). Further complicating matters, most patients with VM may complain of more than 1 vestibular symptom during VM episodes (21). The duration of vestibular symptoms varies widely between and within patients (from seconds to weeks), but the 2012 diagnostic criteria specify that the episodes used to diagnose VM must be between 5 minutes and 72 hours (3).

Another point of confusion among clinicians and patients is the variable relationship of headaches to VM attacks. Headaches may or may not accompany VM episodes and are often less severe compared with their typical migraine-related headaches (2,5,9,12,13,15,16,20,21,28). Instead of a painful headache, some patients may only perceive a sensation of head pressure or fullness (2,21).

Because headaches may not be associated with vestibular symptoms, to make the diagnosis of VM, it is important to inquire about other migrainous features, including photophobia, phonophobia (2,13,15,16,19,21,26), and visual aura (5,13,21,26). Other migrainous symptoms, which are not part of the diagnostic criteria, include osmophobia (15,21), neuropsychiatric symptoms (e.g., fatigue, emotional lability, cognitive, and word-finding trouble) (2,21), autonomic symptoms (e.g., pallor, dry mouth, diaphoresis, and diarrhea) (21), nonspecific sensory changes (21), visual symptoms (e.g., blurry vision, visual snow, and palinopsia) (21), and Alice in Wonderland syndrome (AIWS) dysperceptions (31).

Aural symptoms accompany VM attacks in more than two-thirds of patients, including tinnitus (the most common), aural pressure, and muffled hearing (2,5,8,10,12,16,17,19,21,28,32). The presence of aural symptoms, particularly unilateral, may lead to confusion with MD (see later). Some unusual aural symptoms may be described as well, including bubbling, pulsating, or vibrations in the ear (21).

Persistent interictal dizziness, independent of VM attacks, may occur as well (12,17,21,28,33,34). Patients with VM commonly complain of head motion- and visually-induced dizziness (10,21) in the interictal period. This population of patients will report constant dizziness or vertigo and may lead to premature exclusion of VM unless more detailed questioning is undertaken to reveal a pattern of constant, baseline dizziness with superimposed episodes of vestibular symptoms accompanied by migrainous features. VM with persistent interictal dizziness can be likened to chronic migraine, where patients suffer from headache for at least 15 days out of a month with superimposed migraine attacks (4). In addition, some patients may describe brief, isolated paroxysms of vestibular symptoms in the attack-free period (21).

Psychiatric comorbidities are very common in VM (21,35), consistent with the relationship between vestibular symptoms and mood disorders, especially anxiety (36–40). A history of anxiety and depression also portends a higher risk of developing VM (6). Functional neurological disorders may also be present, particularly in those with long-standing symptoms (21). Patients complaining of vestibular symptoms and exhibiting anxiety are at risk of being dismissed as having a "psychogenic" or "psychiatric" disorder only. Furthermore, some patients with VM may experience AIWS dysperceptions between VM episodes (31), which are often bizarre, and may be mistaken for psychosis. A careful evaluation is needed to detect a vestibular disorder accompanied by mood disturbances.

The "classic" migraine triggers are also triggers for VM and include stress, bright lights, sleep deprivation, missing meals, weather changes, red wine, caffeine, chocolate, and menses (21). Prolonged or excessive exposure stimuli that provoke visually induced dizziness/vertigo (e.g., attending a concert or sports event and riding in a glass elevator), as well as tasks that require excessive head movements, may also trigger VM episodes. (21).

VESTIBULAR TESTING AND OTHER INVESTIGATIONS

A history of episodic vestibular symptoms of an appropriate duration accompanied by migrainous features in a patient with a current or previous history of migraine is often enough to help make the diagnosis of VM. In addition, the description of a combination of various vestibular symptoms can help distinguish VM from other vestibular disorders that usually manifest with monosymptomatic vertigo (41).

Brain MRI should be obtained in all patients with new-onset episodic vestibular symptoms that are not typical of BPPV; the lack of any structural lesions supports the diagnosis of VM and can assuage patients' anxiety. Audiological testing should always be ordered in patients in whom MD is suspected or in those whose vestibular episodes are accompanied by aural symptoms. Vestibular testing is often ordered on patients with vertigo, particularly if they had consulted with otolaryngologists. Abnormal vestibular tests are common in, but not specific to, VM;

however, vestibular testing can help exclude some disorders and reassure patients.

Neuro-otologic abnormalities are commonly encountered in patients with VM, both during attacks and the interictal period. Central and peripheral patterns of spontaneous, positional, and mixed patterns of nystagmus have been reported during acute VM episodes (15,42). Less than 10% of patients have spontaneous interictal nystagmus (8–10,20,43), and slightly more have nystagmus with fixation removed (2,21).

Interictal gaze-evoked nystagmus occurs in less than 10% of patients (8,21,28,43–47). Published studies report interictal positional nystagmus in VM ranging from 0% to 28% (2,8–10,21,28). Other findings include head-shaking (21,28,46–48), vibration-induced (21,48), and hyperventilation-induced nystagmus (21,49).

The presence of saccadic pursuit in VM varies widely (3%–57%) (2,10,28,43–45,47,50–52); this may be due to differences in testing procedure (bedside assessment vs eye movement recording) and dependence on patient cooperation (41). Although it may be tempting to attribute the interictal ocular motor abnormalities to vestibular dysfunction underlying the specific etiopathogenesis of VM, it is important to note that central positional nystagmus, head-shaking nystagmus, and saccadic pursuit have been observed in individuals with migraine without a history of vestibular symptoms (45–47,53).

Caloric testing reveals unilaterally reduced responses in about 10%–20% of patients with VM (2,9,10,28,43–45,47,50–52). Diminished bilateral caloric responses have been described in up to 11% of patients (8,54,55). Of note, this is not specific to VM because similar abnormal caloric responses have been reported in patients with migraine without previous vestibular symptoms (45,47,56–58). Of note, video head-impulse testing (which assesses the high-frequency vestibulo-ocular reflex) in VM reveals fewer abnormalities compared with caloric testing (59); the reason for the discrepancy between caloric and video head-impulse testing is unclear.

Vestibular myogenic-evoked potentials (VEMPs) assess otolithic organ function; cervical VEMP (cVEMP) and ocular VEMP (oVEMP) assess saccular and utricular function. In VM, cVEMPs and oVEMPs have yielded mixed results. Some studies describe diminished (60,61) or even absent cVEMPs (62). Others report no cVEMP difference between patients with VM and migraineurs without vertigo (63), and healthy controls (64,65). Although some report diminished oVEMPs in VM (64,66), others report no difference compared with healthy controls (61). Latencies in VEMPs are rarely (67) or never prolonged (60–62) in VM. VEMPs do not seem to be useful in distinguishing VM from MD (60,61).

Auditory brainstem response (ABR) consists of a sequence of volume-conducted waves recorded at the scalp after brief acoustic clicks. The response consists of 7 waveforms (I–VII), with each waveform representing a different location in the auditory pathway; waves I (auditory nerve), III (cochlear nuclei), and V (inferior colliculi) are the most consistent components and are those on which the ABR interpretation is based (68). Wave I delay or absence of all waveforms indicates peripheral auditory dysfunction. Prolongation of the I–III interpeak interval or absence of waves III and V indicates a lesion between the auditory nerve and lower pons. Prolongation of the III–V interpeak interval or absence of wave V (in the presence of a clear wave III) reflects dysfunction between the lower pons and midbrain (68). ABR abnormalities have been observed during attacks of VM (69) and migraine with brainstem aura (MBA) (70). However, ABR abnormalities also occur in migraine (during and/or between attacks), even in the absence of vestibular symptoms (71–76). Although ABR can help identify peripheral auditory or brainstem dysfunction, VM has no specific ABR abnormalities.

In summary, abnormal ocular motor findings and vestibular testing are common in VM, but there are no pathognomonic or specific findings for VM. These abnormalities must be interpreted in the context of the patient's history and examination.

DIAGNOSTIC CRITERIA: WHICH TO FOLLOW?

In 2001, Neuhauser et al (13) proposed a set of diagnostic criteria for definite and probable VM that have been used in many studies (Fig. 1). The Bárány Society and the International Headache Society collaborated to refine these criteria in 2012 (3) (Fig. 2). The 2001 criteria are less stringent that the 2012 criteria. Which criteria should be used to make the diagnosis of VM? In my opinion, VM can be diagnosed clinically with either criteria because the goal is to permit a reasonably confident diagnosis and institute treatment to alleviate the patient's suffering. However, for research purposes, it is better to use the current 2012 criteria to ensure uniformity and accuracy of diagnosis and data.

DIFFERENTIAL DIAGNOSES FOR EPISODIC VERTIGO

The differential diagnoses of single attack of vertigo lasting more than 24 hours (i.e., acute vestibular syndrome) are myriad but are beyond the scope of this review. Conditions such as mal de debarquement syndrome, chronic subjective dizziness, somatoform dizziness, visual vertigo, and persistent perceptual postural dizziness usually cause a chronic, constant pattern of dizziness instead of episodic vestibular symptoms and will be discussed in a future review. Here, the focus will be on disorders that cause recurrent episodes of vestibular symptoms similar to VM.

Definite:

- Episodic vestibular symptoms of at least moderate severity.
- Migraine according to IHS criteria.
- At least one of the following migrainous symptoms during at least 2 vertiginous attacks: photophobia, phonophobia, visual or other aura, and migrainous headache.
- Not better accounted for by another diagnosis.

Probable:

- Episodic vestibular symptoms of at least moderate severity.
- At least one of the following: migraine according to IHS criteria; migrainous symptoms during vertigo; migraine-specific triggers of vertigo for example, specific foods, sleep deprivation, hormonal changes; and response to antimigraine medications.
- Not better accounted for by another diagnosis.

FIG. 1. The Neuhauser 2001 Criteria for VM (13).

Meniere Disease

MD classically manifests as episodic vertigo accompanied by unilateral low-pitched roaring tinnitus, aural pressure, and diminished hearing (77). The diagnosis is based on the 2015 clinical criteria proposed by the International Classification Committee for Vestibular Disorders of the Bárány Society (78). MD is a rare disease in the general population (affecting 8.2–157 per 100,000 individuals per year) (77), and therefore, the likelihood of encountering a patient with VM is much higher. However, when confronted with a patient experiencing episodic vertigo and aural symptoms, distinguishing MD from VM is often challenging.

The classic triad of MD (vertigo, tinnitus, and hearing loss) only occurs in 40% of patients (79); it is not uncommon for episodic vertigo without aural symptoms in the first year (80). Vertigo in MD typically lasts between 20 minutes and 12 hours (77), which falls within the attack duration of VM (3). Complicating matters, there is a higher prevalence of migraine among patients with MD (about one-third) (48,81). Furthermore, headache and migrainous symptoms frequently accompany MD attacks (48,82–85) because vestibular activation can provoke migraine attacks (86), and hence, MD attacks that trigger migraine episodes can often masquerade as VM. Confounding matters even further, there is significant overlap between these disorders, with some patients suffering from both (83,87).

As discussed earlier, vestibular testing cannot reliably differentiate VM from MD. The best distinguishing characteristic of MD is unilateral, low-frequency, progressive, or fluctuating sensorineural hearing loss (SNHL). Audiometrically documented fluctuating low-frequency unilateral SNHL is the key to diagnosing MD (77). By contrast, VM-related SNHL has been described in up to 20% of patients (55) but is usually mild and symmetric (28,88,89). As such, a patient with a long history of episodic vertigo with aural symptoms who only has mild, symmetrical hearing loss most likely has VM. On the other hand, a patient with profound unilateral low-frequency SNHL most likely has MD.

When evaluating a patient with both vestibular and aural symptoms, it is important to refer the patient to an

Definite VM:

A. At least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 minutes to 72 hours.
B. Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD).
C. One of more migraine features with at least 50% of the vestibular episodes:

- Headache with at least 2 of the following characteristics: unilateral, pulsating, moderate or severe intensity, aggravated by routine physical activity;
- Photophobia and phonophobia;
- Visual aura.

D. Not better accounted for by another vestibular or ICHD diagnosis.

Probable VM:

A. At least 5 episodes of vestibular symptoms of moderate or severe intensity, lasting 5 minutes to 72 hours.
B. Only one of the criteria B and C for definite VM is fulfilled.
C. Not better accounted for by another vestibular or ICHD diagnosis.

FIG. 2. International Headache Society-Bárány Society 2012 Criteria for VM (3).

otolaryngologist (especially one with vestibular expertise) and for audiometry. The lack of unilateral, low-frequency SNHL (particularly in patients with episodic vertigo over a year) favors the diagnosis of VM. Patients with episodic vertigo accompanied by migrainous features (even those with clinically established MD) should be offered a trial of migraine therapy if there are no contraindications. There is evidence that patients with definite MD who fail conventional medical therapy (but who do not fulfil the criteria for VM) benefit from treatment with nortriptyline and verapamil (90).

Benign Paroxysmal Positional Vertigo

BPPV is the most common cause of vertigo in adults (91,92). It typically presents with episodic positional vertigo lasting less than a minute which is triggered by changes in head position relative to gravity (92), spontaneously remits (93,94), and recurs at a rate of about 15%–50% per year (91,92). Positional vertigo is one of the recognized manifestations of VM (3) and has been known to mimic BPPV (95,96). Complicating matters, migraine is 3 times more common in patients with BPPV compared with the general population (97), and migraine attacks can be provoked by vestibular activation (86).

Several clues can help distinguish VM-related positional vertigo from BPPV. First, an association between migrainous features and attacks of positional vertigo is more characteristic of VM (96). Second, direct observation of the nystagmus pattern during acute episodes with positional testing can help distinguish between VM and BPPV. The typical nystagmus in BPPV relates to the affected canal (e.g., left posterior canal BPPV causes an upbeating and left torsional nystagmus in the left Dix–Hallpike position), lasts less than 60 seconds, and is fatigable (92). On the other hand, VM often causes central positional nystagmus, which is characterized by a nystagmus that does not correspond to a particular semicircular canal function, lacks latency, is not fatigable, and persists as long as the patient's head is maintained in the provoking position (15,21,42,95,96). Third, positional testing in the interictal period should be normal in those with BPPV, but may reveal positional nystagmus in VM (discussed earlier). Fourth, because BPPV is often cured by canalith repositioning maneuvers (98), positional vertigo that fails to improve with such maneuvers (especially when performed by experienced clinicians or vestibular therapists) should raise the possibility of VM (96).

Vertebrobasilar Ischemia

A fifth of all ischemic strokes involve the posterior circulation (99). More than 60% of those diagnosed with VBI have at least 1 episode of dizziness/vertigo (100). Abrupt episodic vertigo lasting minutes is the most common initial sign of VBI and is more frequent in the 90-day period leading up to a vertebrobasilar stroke (101,102). Visual symptoms (diplopia, field defects, and illusions), cerebellar ataxia, unilateral limb weakness, and headaches often accompany vestibular symptoms in VBI (99,100,103–105).

It is important to remember that isolated vestibular symptoms only account for 0.7% of strokes (106), and very few patients (0.18%–0.7%) diagnosed with peripheral vertigo return to the emergency department within 30 days with a stroke (107–109). However, dizziness or vertigo is the symptom that is most associated the symptom that is most associated with a missed diagnosis of stroke (110), and VBI can often mimic peripheral causes of dizziness (111–113). Posterior circulation strokes often carry significant morbidity and mortality (114), underscoring the need to reliably identify patients with VBI, and differentiate them from those with VM or peripheral vestibulopathy.

A 3-step bedside examination can reliably and rapidly discriminate between vestibular neuritis and central vestibulopathy—the head-impulse test, gaze-holding for nystagmus, and alternate cover test for skew deviation (HINTS; Head Impulse, Nystagmus, Test of Skew) (115–117). Bilaterally normal head-impulse tests, nystagmus in more than 1 direction, and a skew deviation are ominous signs pointing to a posterior fossa stroke (115–117). By contrast, vestibular neuritis causes a unilaterally abnormal head-impulse test, unidirectional nystagmus, and no skew deviation (115–117).

Although computed tomography (CT) is sensitive for acute hemorrhagic strokes, it is rare for such strokes to present with isolated vertigo (118). MRI is far superior to CT for detecting posterior fossa infarcts (114,119), but even MRI with diffusion-weighted imaging may miss 10%–20% of posterior fossa strokes within the first 48 hours (115–117) and should be repeated 3–7 days after symptom onset to evaluate for new infarcts (110,115,116). Notably, the HINTS examination is more sensitive than early MRI in detecting posterior fossa stroke (115–117).

MRA or CT angiography (CTA) should be performed in patients with vascular risk factors who experience new-onset episodic vestibular symptoms. Contrast-enhanced MRA is better for visualizing extracranial vessels compared to MRA without contrast; CTA is as good as contrast-enhanced MRA (120). In those with VBI/stroke, the presence of vertebrobasilar stenosis of at least 50% portends a 3-fold higher risk of recurrent VBI or stroke (compared to those without such stenoses) in the first 90 days (121,122); pooled analysis of these 2 studies show that the risk of recurrent VBI/stroke was 7% in those without stenosis, 16% with extracranial stenosis, and 33% with intracranial stenosis (123).

Recurrent episodes of vertigo in a patient with a history of migraine favors VM rather than VBI, particularly if most episodes of vertigo are accompanied by migrainous features

and are triggered by the usual migraine triggers. However, the first episode of VM may be indistinguishable from a posterior fossa stroke; these patients should undergo a thorough evaluation for stroke (especially if their HINTS examination indicates a central vestibular disorder and if they have vascular risk factors).

Migraine with Brainstem Aura

For the diagnosis of MBA (formerly, basilar-type migraine), there must be at least 2 posterior circulation symptoms (vertigo, diplopia, tinnitus, impaired hearing, ataxia, or encephalopathy) lasting between 5 and 60 minutes and be followed by a migraine headache (3,4). Most patients with VM do not meet the criteria for MBA because the vestibular symptoms do not fit in the aura time window, and even fewer experience it immediately before headache onset (3).

Episodic Ataxia Type 2 (EA2)

EA2 is an autosomal dominant disorder that begins before 20 years of age and manifests with episodic vertigo and ataxia (lasting hours to days), in patients with a history of migraine; rarely, it manifests after 50 years of age (124). These attacks are usually induced by physical exertion, emotional stress, or alcohol; the interictal duration varies widely, from daily to years (124,125). Such attacks may be difficult to distinguish from VM. Interictal central ocular motor abnormalities can be appreciated in most patients (124,126); similar interictal findings are common in VM as well. Unlike VM, cerebellar atrophy, particularly in the anterior vermis, may be seen on MRI in patients with EA2 (124). Acetazolamide effectively attenuates or prevents attacks in half to three-quarters of patients with EA2 (124). Although it may be difficult to distinguish EA2 from VM on the basis of symptoms alone, clues from the age of onset, triggers, brain MRI, family history, and response to acetazolamide can be helpful.

Cervicogenic Dizziness

Cervicogenic dizziness (CD) is a controversial entity, but there is evidence to support dizziness and vestibular symptoms caused by neck disturbances. CD occurs in patients with degenerative cervical spinal disease and is attributed to perturbations of proprioceptive input from cervical receptors (127). A history of neck pain preceding the onset of dizziness is often present (128). Patients have tender points on the neck, as well as pain radiation to the shoulders and head (127,128). The dizziness is episodic and can be triggered by neck turning (127). Examination and vestibular testing in patients with CD is normal (127,128). Finally, CD often improves or resolves with neck physical therapy and/or surgical treatment of degenerative cervical disease (127,128).

Bow Hunter syndrome (rotational vertebral artery syndrome) is a very rare vascular disorder caused by dynamic compression of the dominant vertebral artery at the atlantoaxial joint during neck turning (127,129). It typically affects older men and rarely presents with isolated vertigo, but is usually accompanied by other posterior circulation symptoms (127). The diagnosis is made when reduced posterior circulation is observed with transcranial Doppler or dynamic digital subtraction angiography with neck turning (127).

TREATMENT

Acute Treatment

Only 1 small randomized, double-blind, placebo-controlled clinical trial evaluated rescue treatment in VM; although it showed improvement in vertigo at 2 hours with zolmitriptan, the study was underpowered, and the results were inconclusive (130). Sumatriptan was beneficial in a retrospective study (131). Rizatriptan reduced visually induced motion sickness in patients with VM (132).

It is reasonable to offer the patient a triptan if they have attacks that last more than an hour because triptan therapy would not be useful for short attacks. In cases of triptan failure or contraindication, benzodiazepines, meclizine, or dimenhydrinate may be offered for acute treatment of vertigo; patients should be warned that these agents cause sedation. Transdermal scopolamine may be used in patients with infrequent attacks of VM that last for more than 24 hours. Transdermal scopolamine should be used very judiciously because of the potential for sedation, mydriasis, confusion, and dry mouth; it must not be used for more than 72 hours because of the risk of withdrawal (causing headache, dizziness, and nausea) on removal of the patch (133). VM attacks that last much longer than a patient's typical episodes ("vestibular status migrainosus") may be ameliorated by intravenous methylprednisolone (134).

Antiemetics can be used in conjunction with the aforementioned rescue medications if nausea and vomiting are problematic; promethazine, prochlorperazine, or ondansetron may be used, but promethazine and prochlorperazine may be better for VM because their antidopaminergic action also targets migrainous activity within the trigeminocervical complex (135).

Prevention

All patients should be counseled about lifestyle modifications that improve migraine control: trigger avoidance (if possible), proper sleep hygiene, regular meals, exercise, and stress management. Diet modification (avoiding trigger foods and eliminating caffeine) alone may be effective in controlling VM in some patients (12,14,16,136). Low-impact physical exercise (20 minutes daily, 3 times a week for 6 weeks) reduced the number of vertiginous attacks and proinflammatory markers in a small study of patients with VM (137).

TABLE 1. The big five of vestibular migraine treatment

Category	Treatment Options
Acute treatment	Triptans Dimenhydrinate Meclizine Benzodiazepines Antiemetics Analgesics (if headache is present) Corticosteroid burst or taper
Lifestyle modification	Trigger avoidance Stress management Physical exercise Diet modification Sleep hygiene Regular meals
Vitamin/herbal migraine preventives	Riboflavin Magnesium Coenzyme-Q10 Vitamin D Butterbur extract
Pharmacologic migraine prophylaxis	Tricyclic antidepressants Amitriptyline and nortriptyline Beta-blockers Propranolol and metoprolol Calcium-channel blockers Verapamil and flunarizine Antiepileptic drugs Topiramate, lamotrigine, and valproic acid Selective norepinephrine reuptake inhibitors Venlafaxine Cyproheptadine (in pediatric patients)
Treatment of comorbidities	Mood disorders Psychiatric referral Psychology referral Counseling Antidepressant therapy Persistent dizziness Vestibular therapy Motion sickness prevention (for long travel) Transdermal scopolamine Premedication with meclizine, dimenhydrinate, or benzodiazepines Persistent light sensitivity FL-41 optical tint Persistent sound sensitivity Vibes earplugs Tinnitus retraining therapy Desensitization (“pink noise therapy”)

To date, there are only 2 randomized controlled trials in VM prophylaxis. One showed that flunarizine improved vertigo frequency and severity, but not headache (138). The other showed that venlafaxine and propranolol reduced vertigo attacks and dizziness, but venlafaxine was superior at addressing comorbid mood disorders in VM (65). Two prospective studies showed that topiramate successfully controlled the frequency and severity of vertigo and headache (139), and auditory symptoms (140). Treatment with either propranolol or venlafaxine improved vertigo attack severity and frequency in an open-label, prospective parallel group study (141). Another open-label, prospective study showed that venlafaxine, flunarizine, and valproic acid decreased vertigo attacks and overall dizziness in VM (142).

Lamotrigine reduced the frequency of vertigo but not headache in a retrospective, open-label study (143). Other retrospective, open-label studies observed a reduction in vertigo and headache with cinnarizine (144) and

acetazolamide (145). Retrospective cohort studies reported a reduction in duration, intensity, and frequency of vertiginous attacks with valproic acid, beta-blockers, calcium-channel blockers, topiramate, lamotrigine, benzodiazepines, tricyclic antidepressants, selective serotonin-reuptake inhibitors, venlafaxine, methysergide, and cyproheptadine (12,14,16,20,31,55,60,131,136,146,147). If nonpharmacologic therapy is preferred, riboflavin (148), magnesium (149), butterbur extract (60), and coenzyme-Q10 (150) are effective for migraine prevention and may be tried for VM (Table 1). Table 1 summarizes the treatment strategies for VM.

It is reasonable to select a migraine preventive that suits a patient's needs and medical history. For example, topiramate can help obese patients lose weight but is potentially teratogenic, while amitriptyline helps insomnia, but should be judiciously used in patients with cardiac arrhythmias. Cyproheptadine is usually used in the pediatric population (147), but the potential weight gain and appetite increase limit its use among adults. Although monotherapy is preferred, combination therapy is sometimes required for optimal VM control (personal experience).

Treatment of Comorbidities

As discussed before, mood disorders (particularly anxiety) often accompany VM. Addressing these mood disorders (including psychological and psychiatric care if needed), in addition to the appropriate migraine prophylactic and rescue medication, is essential for successful treatment of VM. Venlafaxine may be a good choice in VM patients with depression or anxiety because it may help both disorders.

Persistent interictal dizziness (including head motion- and visually-induced dizziness) often affects patients' ability to perform optimally in their jobs or engage in family or social activities (21). From my personal observation, this persistent dizziness often improves with VM control; although vestibular therapy is not very useful with only episodic vertigo from VM, it should be considered in patients suffering from persistent interictal dizziness (20,151–154). Short-term disability may be needed in patients who find that their dizziness interferes with their job performance.

Other interictal migrainous features include motion sickness, and persistent light and sound sensitivity (21). Motion sickness often improves with migraine control (155). To prevent motion sickness on long car rides, air travel, or cruises, transdermal scopolamine patches may be used (with strict instructions on removing the patch on completion of the journey), or premedication with meclizine, dimenhydrinate, or benzodiazepines can be considered. Patients with persistent light sensitivity should be strongly discouraged from wearing sunglasses indoors to avoid retinal dark adaptation which in turn aggravates photophobia; FL-41 optical tints are often useful for such patients (156). Similar to how indoor usage of sunglasses aggravates photophobia, patients with persistent sound sensitivity should avoid using constant use of earplugs since this will exacerbate sound intolerance (157). Some of my patients report benefits from Vibes earplugs which are designed to reduce decibel levels without affecting clarity. If sound sensitivity is severe, patients may need to be referred for specialist care for consideration of tinnitus retraining therapy and desensitization ("pink noise therapy") (157).

CONCLUSIONS

In summary, VM is the most common neurologic cause of vertigo in adults, but is under-recognized, and significantly affects quality of life and economic productivity (6,158). It is important to be aware of the manifestations of this disorder, diagnose it correctly, exclude potential mimics, help patients understand this disorder, and institute the appropriate rescue and preventive therapies.

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