

The Treatment of Vestibular Migraine: A Narrative Review

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Abstract

Vestibular migraine (VM) is one of the most debilitating chronic diseases that is currently underdiagnosed and undertreated. The treatment of VM is a dynamic and rapidly advancing area of research. New developments in this field have the potential to improve the diagnosis and provide more individualized treatments for this condition. In this review, we discussed the progress of evidence-based treatment of VM, including pharmacotherapy and nonmedical methods. A search of the literature was conducted up to September 2019. In order to control or cure VM, patients should follow three steps. First, patients should comply with diet and behavioral medication; Second, during the attack of VM, patients should take medicine to control the symptoms. These acute attack treatment of VM consists of antiemetic medications (e.g., dimenhydrinate and benzodiazepines), anti-vertigo medicine, and analgesics (e.g. triptans). Third, prophylactic medicine (e.g., propranolol, topiramate, valproic acid, lamotrigine, and flunarizine) can be used to reduce the frequency and severity of VM attack. Also, vestibular rehabilitation (VR) treatment should be considered for all VM. Meanwhile, we also propose to establish a culture of prevention which is essential for reducing the personal, social and economic burden of VM.

Keywords: Migraine, prophylaxis, treatment, vertigo, vestibular migraine

INTRODUCTION

Vestibular migraine (VM) is considered to be the second most common cause of vertigo after benign paroxysmal positional vertigo and the most common cause of spontaneous episodic vertigo.^[1] It has been increasingly recognized as a frequent cause of vertigo, which affected up to 1% of the general population, with female preponderance.^[2] Although previously used names including migraine-associated vertigo/dizziness, migraine-related vertigo, migraine-related vestibulopathy, benign recurrent vertigo, VM, vertiginous migraine, and migrainous vertigo were given to the association of migraine and vertigo, the term “VM” has been convincingly advocated as a condition that stresses the particular vestibular manifestation of migraine, and thus best avoids confounding with nonvestibular dizziness associated with migraine.^[3] The International Headache Society (IHS), the Barany Society (International Society for Neuro-Otology), neurologists, otorhinolaryngologists, and other scientists have reached an agreement document with consistent diagnostic criteria for VM.^[4] This classification and the corresponding diagnostic criteria were initially released as a beta document in 2014; since then, these internationally diagnostic criteria have been widely accepted and should now form the basis of all diagnosis and further promote the management of VM in clinical practice as well as in systemic research.^[5] In May 2018, the International Headache Society published the diagnostic criteria for VM in the appendix of the new ICHD-3 (the third edition of the International Classification of Headache Disorders) version of the international headache classification.^[6] As of now, it remains an ongoing discussion about whether VM is an autonomous disorder with distinct

pathophysiological features or whether it simply represents a subform of migraine that also includes vestibular symptoms.^[7] Since the mechanisms underlying VM remain insufficiently known, treatment trials that specifically address VM are few and most treatment recommendations for VM are extrapolated from knowledge gathered from clinical trials on migraine with and without aura.^[4] Like migraine, there are specific treatment and prophylactic treatment for VM. This review summarizes the knowledge of current treatment options that are specific for VM.

SEARCH METHODS

As this is a narrative review, we did not conduct a systematic literature search. However, a search of the PubMed database was conducted to collect all available published data on the treatment of VM in September 2019, with no date limits, using the search terms “vestibular migraine,” “migrainous vertigo,” “migraine-associated vertigo,” “migraine-associated dizziness,” “migraine and vertigo,” and “treatment”, and the

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Received: 07.11.2019 **Revision:** 22.11.2019

Accepted: 25.12.2019 **Published:** 11.02.2020

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DOI: 10.4103/aian.AIAN_591_19

results were screened for relevance to the review topic. Articles were also added based on the authors' knowledge of the area.

Epidemiology of vestibular migraine (VM)

VM is more prevalent than other vestibular disorders^[8] and its prevalence is different in the various study population and clinical context.^[9] Stolte *et al.* in a population-based study found that the lifetime prevalence of migraine and vertigo was approximately 16% and 7%, respectively in Western industrial nations taking Germany as a representative.^[7] Neuhauser *et al.* performed similar research and found that a 1-year prevalence was 0.9% and the lifetime prevalence was 1%.^[10] Meanwhile, many people, young or old, might suffer from VM in any period of life. However, it has been proved that women are to a greater extent subjected to VM compared with men, and the female-to-male predominance is about 5:1, with a mean age of onset of 37.7 years for women and 42.4 years for men.^[11] Familial occurrence has been reported in some patients with an autosomal dominant pattern of inheritance and decreased penetrance in men.^[12]

Clinical presentation and diagnostic criteria for VM

Either a headache or vertigo of VM might present as the initial symptom, and there is no fixed pattern.^[13] The headache might be from moderate to severe.^[12] However, nearly 30% of VM attacks found no associated headache. Only a quarter or less of the patients tend to have throbbing migraine attacks.^[12] In addition, migraine and vertigo can never occur together. The International Headache Society (IHS) published a consensus document of diagnostic criteria for VM, which was added in the appendix of the new ICHD-3 version of the International Headache Classification^[6] [Table 1].

Acute attack treatment of VM

The treatment of VM aims to reduce the frequency of migraine attacks and vertigo. Various treatment options are available for patients with VM, including pharmacotherapy, VR exercises, and reduction of triggers.^[8,14] Due to the unclear mechanism of VM and lack of diagnostic criteria, the specific evidence-based treatment is scarce and insufficient, and no enough randomized controlled trials for the treatment of VM have ever been carried out. An abortive therapy is currently used to treat an acute attack of headache or vertigo.^[15]

Although some researchers strongly opposed to using triptans in treating basilar migraine, it has been proposed that triptans may be used in VM.^[3] Two randomized controlled clinical studies have been performed with triptans (the serotonin agonist) on the specific treatment of VM.^[16,17] Another study examined the benefit of zolmitriptan in treating VM attacks.^[18] A randomized, double-blind, placebo-controlled, crossover-after-one-attack trial of 2.5 mg oral zolmitriptan for the treatment of acute migrainous vertigo was also performed.^[16] Zolmitriptan was thought to improve central nervous system penetration due to its less hydrophilic nature compared to sumatriptan.^[19] Obermann and Strupp found out 38% (three of eight episodes) of patients with VM attacks could benefit from zolmitriptan, whereas in the placebo group, a positive effect was observed

Table 1: Diagnostic criteria of vestibular migraine by the International Headache Society in 2018^[6]

A.	At least five episodes fulfilling criteria C and D
B.	A current or past history of migraine without aura or migraine with aura according to the International Classification of Headache Disorders (ICHD)
C.	Vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 h
D.	At least half of episodes are associated with at least one of the following three migrainous features: <ol style="list-style-type: none"> 1. Headache with at least two of the following four characteristics: <ol style="list-style-type: none"> a. Unilateral location b. Pulsating quality c. Moderate or severe intensity d. Aggravation by routine physical activity 2. Photophobia and phonophobia 3. Visual aura
E.	Not better accounted for by another ICHD-3 diagnosis or by another vestibular disorder.

in only 22% (two of nine episodes).^[4] Nevertheless, the study had some limitations such as the large confidence intervals and the small number of patients recruited ($n = 10$) with only 17 reported attacks.^[16] Marcus and Furman found that rizatriptan could prevent the development of motion sickness and severe motion sickness symptoms in patients with migrainous vertigo.^[20] A double-blind, randomized, placebo-controlled study examined the benefit of rizatriptan in treating motion sickness in response to a complicated vestibular stimulus. Compared to placebo, rizatriptan reduced the motion sickness in 13 of 15 subjects suffering from vestibular-induced motion sickness ($P < 0.02$).^[21] However, this positive effect was not seen after exposure to more provocative vestibular stimulation.^[4,17]

A retrospective multicenter open-label trial with almotriptan was studied by Cassano *et al.* for the acute treatment of VM. The study suggests that almotriptan is effective and safe in reducing both vertigo and headache among patients who suffer from VM.^[22]

In a retrospective cohort study treated with a variety of prophylactic and abortive medications for migraine-associated dizziness and vertigo, sumatriptan (orally or intramuscularly) demonstrated efficacy for headache and vertigo.^[23]

The same drugs used for vertigo can also provide relief from vestibular episodes. Antiemetic medications (e.g., dimenhydrinate and benzodiazepines) may be also useful during the acute phase of vestibular.^[24] Other drugs encompass promethazine, betahistine, and metoclopramide (an antiemetic agent). These drugs help treat dizziness, motion sickness, nausea and vomiting, and some other symptoms as well.

The episodes of severe migrainous vertigo of more than 1-day duration in four patients reported by Prakash were successfully terminated by intravenous methylprednisolone (1,000 mg/d, 1–3 days) in two patients. Two other patients who had attacks of migrainous vertigo almost daily also showed complete response to intravenous methylprednisolone.^[25]

There was a retrospective chart review of patients with VM treated with noninvasive vagus nerve stimulation in a single tertiary referral center between November 2017 and January 2019. The study provides preliminary evidence that noninvasive vagus nerve stimulation may provide rapid relief of vertigo and headache in acute VM.^[26]

Prophylactic treatment of VM

Prophylactic medications [Table 2] are mainstays for the management of VM.^[24] In general, the scientific literature suggests that medications used for migraine prophylaxis can be used to treat VM.^[18,27] Prophylactic treatment was analyzed recently in The Cochrane Collaboration for randomized controlled trials in adults with the diagnosis of VM or probable VM according to the Barany Society/International Headache Society criteria.^[28] In a recent article, Strupp *et al.* reviewed pharmacotherapy options for VM and noted that preventive agents may include β -blockers, valproic acid, topiramate, tricyclic antidepressants, and lamotrigine.^[29]

Flunarizine is safe and effective for the prophylactic treatment of VM. A randomized controlled study was performed in patients with definite VM. In this study, the frequency, duration, and intensity of vertiginous episodes showed a significant improvement in both group A receiving 10 mg flunarizine daily for 3 months along with betahistine 12 mg tid for 48 h during episodes and group B receiving only betahistine for 48 h during episodes. No severe adverse events were found.^[30] Another randomized control trial was taken by Lepcha *et al.* in a tertiary academic referral center to evaluate the efficacy of flunarizine in patients with migrainous vertigo compared to

betahistine and vestibular exercises. The study demonstrated that flunarizine (10 mg orally) is effective in patients with migrainous vertigo who suffer from considerable vestibular symptoms.^[31] In a retrospective cohort study, patients with VM receiving flunarizine 10 mg daily had significant overall clinical improvement of vestibular symptoms and headaches.^[32] In another separate retrospective review, 30 patients were administered with flunarizine (average dose 10 mg) and 68% responded with an improvement in vestibular symptoms ($P < 0.001$).^[33]

As a selective calcium channel blocker, cinnarizine has been used for the treatment of vertigo. A study group included a case series of 13 subjects with VM (10 females and 3 males). They were complaining of positional vertigo and were treated with cinnarizine or topiramate. The findings showed that prophylactic therapy of VM (cinnarizine or topiramate) cured positional vertigo with a success rate of up to 92% in the study group.^[34] A retrospective, single-center, open-label investigation of the effects of cinnarizine on VM with associated vertigo were put into effect by Taghdiri *et al.* They showed that the mean frequency of vertigo and also the mean frequency, duration, and intensity of migraine headaches per month were reduced significantly after 3 months of cinnarizine therapy.^[35] In a non-randomized prospective open-label non-placebo controlled trial, the researchers studied the association of cinnarizine 20 mg and dimenhydrinate 40 mg in a group of 22 patients affected by definite VM. Subjects treated with dimenhydrinate and cinnarizine found a decrease in vertigo attacks from 5.3 to 2.1 and headaches from 4.3 to 1.7 during the 6 months of therapy. Among these subjects, 68% reported a decrease of at least 50% of vertigo attacks, and 63% of headaches as well. They reported a consistent reduction of vertigo spells in 60–80% patients.^[36]

Baloh *et al.* studied the effectiveness of acetazolamide in a family with migraine, vertigo, and essential tremor. All five patients who were treated showed a marked decrease in the frequency of headaches, vertigo spells, and the severity of the essential tremor.^[37] In a study by Baloh *et al.*, the researchers found that acetazolamide stopped or markedly decreased the frequency of vertigo attacks in three patients treated but had little effect on the chronic vestibular loss.^[38] In a retrospective cohort study, 39 patients with VM were prescribed acetazolamide 500 mg/d. Vertigo and headache frequency were determined by a number of attacks per month, and the severity was determined by visual analog scales measured in centimeters from 0 to 10 which were collected from the records. The results demonstrated that acetazolamide was effective in reducing both the frequency and severity of vertigo and headache attacks and this effect was more prominent for vertigo frequency and severity.^[39]

In a randomized comparison trial study, the efficacy and safety of venlafaxine, flunarizine, and valproic acid in VM prophylaxis were investigated. The data confirmed the efficacy and safety of venlafaxine, flunarizine, and valproic acid in

Table 2: Medication used in vestibular migraine prophylaxis^[30]

Drug	Side effects
Propranolol 40-240 mg ^[31]	Fatigue, impotence, depression, nightmares, bronchial constriction, hypotension, falls, bradycardia
Metoprolol 50-200 mg ^[32]	Fatigue, hypotension, impotence, depression, nightmares, bronchial constriction, falls, bradycardia
Topiramate 50-100 mg ^[33,34]	Paresthesia, fatigue, memory and concentration difficulty, sedation, appetite disturbances, depression, weight loss
Amitriptyline 50-100 mg ^[35]	Sedation, orthostatic hypotension, dry mouth, weight gain, constipation, urinary retention, conduction block
Acetazolamide 250-750 mg ^[36]	Paresthesia, nausea, sedation, hypokalemia, hyperglycemia
Flunarizine 5-10 mg ^[32]	Weight gain, depression, sedation, reversible parkinsonism

the prophylaxis of VM. Venlafaxine had an advantage in terms of emotional domains. Venlafaxine and valproic acid were also shown to be preferable to flunarizine in decreasing the number of vertiginous attacks, but valproic acid was shown to be less effective than venlafaxine and flunarizine in decreasing vertigo severity.^[40] On the contrary, in a retrospective chart review, the researchers thought specific prophylactic antimigraine medications including verapamil, topiramate, gabapentin, amitriptyline, valproic acid, and quetiapine were not associated with improved outcomes in post-traumatic migraine-associated dizziness patients.^[41] Celiker *et al.* investigated the effects of valproic acid on vestibular symptoms and electronystagmography findings in patients with migraine-related vestibulopathy and determined that prophylactic low-dose (500 mg/d) valproic acid could decrease the frequency of headache and vestibular symptoms, although it does not cause any statistically meaningful change in electronystagmography findings.^[42]

Topiramate is used to treat seizures as an antiepileptic medication that reduces neuronal hyperexcitability and proved to be effective for the prevention of migraine.^[19] A study was conducted by Salmito *et al.* to evaluate the efficacy of prophylactic treatment used in patients from a VM outpatient clinic. It was found that 80.9% of the patients showed improvement with prophylaxis ($P < 0.001$) and amitriptyline, flunarizine, propranolol, and topiramate improved vestibular symptoms and headache. The four drugs were proved to be effective with a statistical significance. There was also a positive statistical correlation between the time of vestibular symptoms and clinical improvement.^[32] The aim of another study at a pediatric vestibular clinic was to evaluate the diagnostic and response to therapy of VM in children. The study elucidated that medications effectively reduced reported vestibular symptoms in 88% of those treated with tricyclics, 86% of those treated with cyproheptadine, 80% of those treated with topiramate, 80% of those treated with triptans, and 25% of those treated with gabapentin. The authors concluded that VM is a common cause of vertigo in the pediatric population that is commonly responsive to medical therapy.^[43] In a prospective study, topiramate was shown to be beneficial in reducing the severity and frequency of vertigo and headache attacks. Both doses of 50 mg/d and 100 mg/d were equivalently effective.^[44] A retrospective chart review study was to evaluate a therapeutic pathway for VM and complex dizziness of undetermined etiology with caffeine cessation and pharmacotherapy. Patients were recommended to stop the intake of caffeine and other putative migraine-triggering agents. Pharmacotherapy was initiated with nortriptyline or topiramate if symptoms persisted despite diet modification. The authors demonstrated that VM and complex dizziness of undetermined etiology can be treated effectively with a therapeutic pathway consisting of caffeine cessation followed by pharmacotherapy.^[45] In a retrospective study of 100 patients, 26 received the non-pharmacological intervention and 74 received drugs, mainly beta-blockers (propranolol or

metoprolol), anticonvulsants (valproic acid, topiramate, or lamotrigine), or butterbur root extract. The study reported a reduction of frequency, duration, and severity of vestibular attacks as well as headaches. The effect was more marked for the pharmacological treatments.^[1,46] In brief, there is an extensive evidence base provided by double-blind, placebo-controlled trials, showing that topiramate is a safe, effective, and well-tolerated drug in the management of migraine and its variants, being especially promising in the management of the migraine-vertigo syndrome.^[47]

A retrospective study of 19 patients treated with lamotrigine 25 mg every morning for 2 weeks, then 50 mg for 2 weeks, to reach a target dose of 100 mg after weeks showed a significant reduction in vertigo but not in headache frequency.^[1,48]

A study by Celik *et al.* was to determine the efficacy of propranolol treatment in patients with VM by the visual analog scale, dizziness handicap inventory, vertigo symptom scale, and vestibular disorders activities of daily living scale and its effect on the quality of life. The results showed that the severity, frequency, and the number of attacks and disability scores were reduced, and the quality of life was improved in patients with VM with propranolol treatment.^[49] A prospective, randomized, controlled clinical trial was implemented by Salviz *et al.* They compared the effectiveness of venlafaxine and propranolol for the prophylaxis of VM. The study provided evidence that venlafaxine and propranolol show equal effectiveness as prophylactic drugs for ameliorating vertiginous symptoms in VM. However, venlafaxine may be superior to propranolol in ameliorating depressive symptoms.^[50] In a retrospective study about epigone migraine vertigo, the researchers reported three illustrative clinical cases among 28 patients collected during an observation period of 13 years. They suggested that the patients use prophylactic treatment with flunarizine (5 mg per day) and/or acetylsalicylic acid (100 mg per day), or propranolol (40 mg twice a day). The data showed that all patients considerably improved symptoms with therapy.^[51]

A study was designed to test whether rizatriptan is also effective in protecting against visually-induced motion sickness and to test whether rizatriptan blocks the augmentation of motion sickness by head pain. Using the randomized double-blind, placebo-controlled methodology, 10 females, 6 with migrainous vertigo and 4 without vertigo received 10 mg rizatriptan or placebo 2 h prior to being stimulated by optokinetic stripes. These pilot data suggest that rizatriptan does not consistently reduce visually induced motion sickness in migraineurs. Rizatriptan may diminish motion sickness potentiation by cranial pain.^[52]

Nonmedical treatment options for VM

1. VR exercises

VR is a therapeutic approach to treat dizziness and balance dysfunction and is based on central mechanisms of neuroplasticity, which includes adaptation, habituation, and substitution that facilitate vestibular compensation.^[15] Because

of the availability, VR treatment should be considered for all VM patients.

2. Behavioral modification

Researchers aren't certain what causes VMs, but some believe that the abnormal release of chemicals in the brain plays an important role. Some of the same factors that trigger other kinds of migraines can also trigger a VM, including stress, lack of sleep, dehydration, weather changes, or changes in barometric pressure, menstruation. Meanwhile, certain foods and drinks can trigger a VM attack as well. These foods and drinks include chocolate, red wine, aged cheeses, monosodium glutamate, processed meats, coffee, and sodas with caffeine. Therefore, some researchers propose that it is of great significance to establish regular sleep patterns, stress reduction, a migraine diet (avoiding chocolate; aged cheeses; red wine and port; monosodium glutamate found in fast food, soy sauce, yeast, and meat tenderizers), and eliminate caffeine or habitual analgesic use.^[10,53]

CONCLUSION

In the current research, VM is considered to be the second most common cause of vertigo and the commonest cause of spontaneous episodic vertigo in the general population. At present, VM remains underdiagnosed and undertreated despite its relatively high prevalence. Until better medications become available, the principles of migraine treatment are appropriate for the management of patients with VM. The treatment strategies consist of avoidance of triggers, pharmacotherapy, and VR. First, the treatment for VM patients includes diet style and behavior modification. Then, we can use migraine-suppressing drugs such as calcium channel blockers, beta-blockers, antidepressants, or antiepileptic drugs. For patients with frequent episodes, prophylactic medication is useful. VR is often helpful for all patients with VM. We expect that more results of randomized controlled trials may provide more robust evidence on the treatment of VM in the future.

Financial support and sponsorship

Nil.

Conflicts of interests

On behalf of all authors, the corresponding author states that there is no conflict of interest regarding the publication of this paper.

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