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Review Article

Vestibular migraine and its management in Indian clinical setting: a narrative review

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ABSTRACT

Vestibular migraine (VM) is a disorder connecting two clinical conditions, migraine and vertigo. A high prevalence of VM (~30%) is reported among Indians suffering from dizziness. Yet, there is no gold standard for diagnosis and treatment. Trials specifically for patients with VM are scarce. This review provides an overview on VM and its management in the Indian clinical setting. An evidence-based discussion and review of literature on VM was performed by expert panelists, and opinions were gathered. Experts opined that in vertigo clinics, around 50% of patients are diagnosed with VM, with a higher predominance in females aged 30-50 years, while in children, the age of manifestation is 12-14 years. The most common presenting symptoms of VM among adults are spontaneous vertigo, followed by positional vertigo with headache, nausea, vomiting, photophobia and phonophobia, while intolerance to loud sounds and sudden falls are common symptoms in children with VM. Common comorbidities associated with VM are benign paroxysmal positional vertigo (BPPV) and Meniere's disease (MD). Experts opined that there are currently no approved standardized treatment protocols for VM. The steps in the management of VM include diet and lifestyle modification, along with medicines for symptomatic relief (abortive) and prophylaxis and vestibular rehabilitation. Globally, prochlorperazine, a vestibular suppressant, is widely used for the management of vertigo and its associated symptoms. According to experts, prochlorperazine 5 mg twice or thrice daily for 5-7 days can play a significant role as an abortive treatment in the management of various patient profiles of VM.

Keywords: Vestibular migraine, Acute vertigo, Prochlorperazine, Migraine-associated vertigo

INTRODUCTION

In the general population, migraine and vertigo are the two most prevalent conditions.[1] The term 'vestibular migraine was first used by Dieterich and Brandt to describe the concomitant occurrence of vestibular symptoms and migraine.[2] VM is characterized by episodes of spontaneous or positional vertigo lasting seconds to days along with migraine symptoms.[3] Diagnostic criteria by the International Headache Society and the committee for the International classification of vestibular disorders (ICVD) of the Bárány Society help in differentiating definite and probable VM (Table 1).[4] There are multiple care gaps in the diagnosis and management of VM, including an unclear spectrum of VM disorder, under diagnosis, the association of VM with multiple unrecognized comorbidities, absence of gold standard tests or markers for diagnosis of VM, and a lack of appropriate treatment strategy.[1,7,8] To address these gaps, an advisory board meeting with 12 otorhinolaryngologists from different regions of India was conducted to elicit insights on the management of VM in India.

Table 1: Definite and probable VM.[4-6]

Definite vestibular migraine	Probable vestibular migraine
(A) At least five episodes fulfilling criteria (C) and (D)	(A) At least five episodes with vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours.
(B) A current or past history of 1.1 migraines without aura or 1.2 migraines with aura	(B) Only one of criteria (B) and (D) for VM fulfilled (migraine history or migraine features during the episode)
(C) Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours[4]	
(D) At least 50% of episodes associated with at least one of the following three migrainous features; Headache with at least two of the following four characteristics: unilateral headache, pulsating quality, moderate to severe intensity, or aggravation by routine physical activity. Photophobia and phonophobia. Visual aura.	(C) Not better accounted for by another ICHD-3β diagnosis or by another vestibular disorder
(E) Not better accounted for by another ICHD-3β diagnosis or by another vestibular disorder	

A search of the PubMed database was conducted to collect all available published data on the treatment of VM, with no date limits, using the search terms "vestibular migraine," "migrainous vertigo," "migraine-associated vertigo," "migraine-associated dizziness," "migraine and vertigo," and "treatment," "diagnosis" and the results were screened for relevance to the review topic. Based on their clinical practice, the panelists provided their opinions on the epidemiology and diagnosis of VM in Indian clinical settings along with a place in therapy for prochlorperazine in different patient profiles. Based on the discussion, expert consensus statements were derived and summarized in this review.

Burden of disease

The prevalence of VM ranges from 4.3%-29.3% and that of probable VM varies from 4.0%-5.7%, with a higher predominance of VM in females versus males (5.1:1.5).[1-8] Among Indian patients with dizziness, researchers reported that 30.35% fulfilled the criteria of VM.[9] The onset of VM is commonly seen at the mean age of 38-50 years.[1] VM is also found to be prevalent in children, affecting about 24%-56% of those suffering from vertigo, with 30% of children fulfilling the criteria of definite VM.[10,11] According to experts, in Indian clinical practice, among patients with vertigo, 20% have VM. However, clinical evidence demonstrates a higher proportion of patients with vertigo (~30%) have VM. Among adults, VM is reported in 5%-10% of patients in multispecialty hospitals, 30% of patients in ENT specialist clinics and 50% of patients in vertigo clinics. Individuals aged 30-50 years are more likely to have VM, with a higher predominance in females. Among children, VM is found to be common in those aged 12-14 years with higher prevalence in young girls.

Clinical presentation and comorbidities associated with VM

Spontaneous vertigo is the most frequent vestibular symptom associated with migraine; it is observed in 67% of patients, followed by positional vertigo in 24%.[12] Along with this, patients may also experience head motion intolerance vertigo, head motion-induced dizziness, visual vertigo and non-vertiginous dizziness such as light-headedness or a "boat-like" rocking feeling.[3,12] Acute VM is also frequently accompanied by nausea and imbalance. Vertigo duration ranges from seconds to several days. About 10%-30% of patients have vertigo with the typical duration of a migraine aura, i.e., 5 to 60 minutes.[3] Vertigo may precede headache or begin together or may occur in the late phase of the headache. Other symptoms include photophobia, phonophobia, osmophobia and visual or other auras.[2] Auditory symptoms, including hearing loss (mild and transient),[3,12,13] tinnitus, and aural pressure are also observed in patients with VM (38%).[1,13]

An early manifestation of VM in children is the benign paroxysmal vertigo of childhood. The common characteristics include brief attacks of vertigo or disequilibrium, anxiety, and nystagmus or vomiting, recurring for months or years in otherwise healthy young children. Many of these children, after years of cessation of vertigo attacks develop migraine.[13] VM is also associated with comorbidities, including benign paroxysmal positional vertigo (BPPV) and Meniere's disease (MD). Among patients with VM, more than half (57%) have at least one comorbid neurotologic condition causing vestibular symptoms.[14] Shin et al reported the occurrence of VM in about 35% of patients with MD.[15] VM also co-exists with psychiatric comorbidities, including anxiety and somatoform dizziness/chronic subjective dizziness.[7] Persistent postural perceptual dizziness (PPPD), a chronic vestibular disorder also has VM as a precipitating factor (in about 25% of patients).

PPPD is characterized by presence of one or more symptoms of dizziness, unsteadiness or non-spinning vertigo for most of the day during a duration of ≤3 months.[16]

DIFFERENT PATIENT PROFILES OF VM

Vestibular symptoms along with migraine are experienced in various patient profiles as shown in (Table 2).

Table 2: Profiles of patients presenting with vertigo and migraine.[17-20]

Condition	Patient profile
Migraine with aura	Experience of sensory symptoms (neurologic, gastrointestinal, and autonomic) including flashes of light, blind spots, or tingling in the hands or face before or during a migraine episode
Migraine without aura	Recurrent headache attacks that last for 4-72 hours. Unilateral location, pulsating quality, moderate or severe pain intensity, and aggravation by routine physical activity are the typical features of an attack
VM with MD	Probable or definite VM with bilateral, low-frequency sensorineural hearing loss
VM with BPPV	Probable or definite VM with atypical positional nystagmus and/or vertigo during acute attack
Recurrent VM	Migraine triggers including altered sleep patterns, monosodium glutamate, menstrual cycle and food, such as chocolate, ripened or aged cheese and red wine can result in episodes in VM patients

According to the experts, common presenting symptoms of VM in adults include spontaneous vertigo followed by positional vertigo, and sometimes head motion-induced vertigo, headache, nausea, and vomiting, photophobia and/or phonophobia. In children, the common presenting symptoms are intolerance to loud sounds and sudden falls. BPPV and MD are common overlapping comorbidities in majority of patients with VM.

DIAGNOSIS OF VM

History

A detailed history of the patient is imperative. Past history of headaches is common among patients with VM (70%).[19]

Diagnostic tests

There are no pathognomonic clinical signs or laboratory tests that can help in confirming the diagnosis of VM.[21] A few functional tests that can help in the differential diagnosis of VM are mentioned in (Table 3).

Owing to absence of any conclusive diagnostic tests for vestibular migraine, videonystagmography can be an effective and reliable tool to diagnose vestibular migraine. A case report by Bijlani et al of a 31-year-old woman with complaints of persistent postural imbalance, non-rotatory dizziness and previous history of positional vertigo and migrainous headaches showed a saccadic vertical smooth pursuit (MRI done to rule out central etiology), low amplitude up-beating nystagmus on removal of fixation and left beating horizontal nystagmus on gaze testing without fixation on videonystagmography.

Usually, videonystagmography findings show non-specific abnormalities, but in this woman the presence of up beating nystagmus was described as a differentiating feature from other vestibular syndromes.[24]

Clinical and laboratory findings

In patients with VM, normal neurological finding is observed between the attacks. However, a few patients may have some abnormalities between and during the attacks.[4]

Between attacks, gaze-induced nystagmus has been observed in 27% patients, spontaneous nystagmus in 11% patients, persistent positional nystagmus and positional nystagmus in 12%-28% patients, vertical saccadic pursuit in 48% patients, horizontal saccadic pursuit in 22% patients, subtle saccadic pursuit in 20%-63% patients, unilateral canal paresis in 8%-22% patients, bilateral vestibular failure in 11% patients, low-frequency, mild cochlear loss in 3%-12% patients, and mild bilateral sensorineural hearing loss in 18% patients.

During attacks, spontaneous nystagmus has been observed in 19% patients; nystagmus provoked by horizontal headshaking in 35% patients; low-velocity, sustained, central positional nystagmus in all patients; pathologic nystagmus with spontaneous or positional nystagmus in 70% patients, central vestibular dysfunction in 50% patients, peripheral vestibular dysfunction in 15% patients, and unclear mixture in 35% patients.

Biomarkers for VM

Biochemical analysis can help rule out other coexisting comorbidities like MD (Table 4).[22]

Table 3: Functional tests for the diagnosis of VM.[8,22,23]

Test	Observation in patients with VM
Otoacoustic emission (OAE) suppression test (Audiological tests)	A reduced OAE suppression
Auditory middle latency response (AMLR) (Audiological tests)	Lack of habituation (or potentiation) of Na-Pa amplitude in AMLR
Posturography tests	Compromised body balance and higher visual dependency
Vestibular-evoked myogenic potential (VEMP)	Low electromyography itudes, loss of cervical VEMP responses, maximum VEMP response shifting from 500 to 1000 Hz, and increased latencies
Caloric electronystagmography	Differentiates peripheral vestibular diseases from VM. Reduced caloric response in peripheral vestibular hypofunction, labyrinthitis and vestibular neuritis
Video head impulse test (vHIT)	Differentiates vestibular disorders from VM, vHIT gain along with PR score helps in differentiating different vertigo diseases
Radiology and neuroimaging	A raised gray matter mass of the frontal lobe, temporal lobe of the left side, left thalamus and occipital lobe along with reduced gray matter in the left cerebellum is reported
Videonystagmography (VNG)	Saccades, smooth pursuit, optokinetic test, spontaneous nystagmus, high frequency headshake, hyperventilation gaze test, Dix hallpike test, McClure, Pagnini test, Head position tests

Table 4: Biomarkers for VM.

Biomarker	Observation
IL-1β, IL-1 receptor antagonist, IL-6 and TNF-α	Higher levels in MD patients vs. healthy controls
IL-1β, CCL3, CCL22 and CXCL1	Help in differentiating VM from MD
IL-1β, TNF- α, Interferons (IFN)-γ, IL-2, IL- 6 and IL-8.	Elevated levels in VM vs. healthy controls

CCL, chemokine (C-C motif) ligand; CXCL: chemokine (C-X-C motif) ligand; IFN, interferon; IL, interleukin; MD, Meniere's disease; TNF, tumor necrosis factor; VM, vestibular migraine.

Differential diagnosis

MD is the main differential diagnosis of VM. Prominent features of MD include low-frequency hearing loss, ear fullness or pain preceding the attack or at attack onset along with tinnitus and/or hearing loss during the attack.[12] Another common differential diagnosis for VM is BPPV, which is a common cause of recurrent vertigo. However, recurrent vertigo associated with migraine occurs many times a year or a month, with a duration ranging from hours to a few days. However, the episodes in BPPV are short, with vertigo typically lasting weeks to months without therapy. The analysis of positional nystagmus helps to distinguish positional VM from BPPV during acute vertigo attacks.[12] Vestibular paroxysmia (VP), another cause of *recurrent spontaneous attacks* of vertigo, is also a differential diagnosis for VM.[25,26] However, episodes of VP are of short duration, lasting from seconds up to one minute versus minutes to hours in case of VM.[26] Besides, along with vertigo, patients with VP experience tinnitus, pressure/numbness around ears, minor headache, or pressure on the head versus vertigo accompanied by headache or other migrainous symptoms.[27] According to the experts, clinical history is important for the diagnosis of VM. Points to be considered while diagnosing VM include history or current presentation of headache, quality of headache as per diagnostic (Table 1), at least five recurrent episodes of vestibular symptoms, photophobia and/or phonophobia, history of visual aura, mild and bilateral hearing loss, and history of motion sickness and fear of height. Systemic illness should be assessed to confirm the right diagnosis. Conditions like episodic vertigo, BPPV, MD and VP should be excluded to diagnose VM. The experts suggested that VM is a diagnosis of exclusion because there are no diagnostic tests/biomarkers for diagnosing VM.

MANAGEMENT OF VM

Various treatment strategies for VM include lifestyle and diet modification, medication and vestibular rehabilitation.[28]

Lifestyle modification

This treatment strategy includes recognizing and avoiding the triggers that cause headaches and vertigo by modifying a person's lifestyle and diet. Common triggers that can cause migraines and lead to vertigo attacks include skipping meals, sleep pattern changes, menstruation, air pressure changes, alcohol, sweeteners, processed meats,

aged cheeses, excess caffeine consumption, monosodium glutamate, and chocolate.[20] However, patients can require additional medical therapy despite lifestyle and diet modifications.[29]

Abortive treatment

This treatment strategy aims to suppress the vestibular system and is used during acute vertigo attacks in VM.[20,21,29] The commonly used drugs for the abortive treatment of VM are given in Table 5. However, abortive drugs should be used for less than 10 days per month.

Prophylactic treatment

Patients with over three attacks per month, with very long-lasting or disabling attacks, or those not responding to acute treatment are provided prophylactic treatment.[32] These are the mainstay for the management of VM.[4] Evidence suggests that prophylactic medicines demonstrate a reduction in the duration, intensity, and frequency of episodic vertigo.[32] Drugs used for prophylactic treatment of VM are listed in (Table 6).[32] For management of migraine and associated symptoms prophylaxis therapy can be given for about six months.[33]

Table 5: Treatment options for acute attacks of vestibular migraine.[28,30,31]

Acute treatment	Symptoms managed	Medication	Dose
Triptans	Headache	Zolmitriptan	2.5 mg oral
		Rizatriptan	10 mg oral
Simple analgesics/NSAID		Paracetamol	1 g qid
		Aspirin	600-900 mg 4 times a day
		Ibuprofen	400-800 mg every eight hours
Vestibular sedatives	Vertigo, nausea, vomiting	Prochlorperazine	10-15 mg/day
		Cinnarizine	75 mg/day

Table 6: Prophylactic treatment for VM.

Class	Drug	Dosage
Beta-blockers	Metoprolol	100-200 mg oral
	Propranolol	40-160 mg oral
Anti-epileptic drugs	Valproic acid	600 mg oral
	Topiramate	50-100 mg oral
	Lamotrigine	75-100 mg oral
Anti-depressants	Amitriptyline	10-100 mg oral
	Nortriptyline	25-75 mg oral
Calcium channel blockers	Flunarizine	5-10 mg oral
	Cinnarizine	37.5-75 mg oral
Others	Magnesium	400 mg
	Clonazepam	0.25-1 mg oral

Vestibular rehabilitation

Patients who become overly dependent on environmental and visual stimuli and those who have impaired physiologic adaptation are recommended to undergo VR.[28] This type of management strategy is useful if attacks are very frequent, have concurrent persistent postural-perceptual dizziness (PPPD) or have disabling avoidance behaviours.[29] This therapy is beneficial in VM especially with complications such as anxiety, visual dependence or loss of confidence in the balance system.[34,35] It is based on central mechanisms of neuroplasticity, which include adaptation, habituation, and substitution that help in vestibular compensation.[35] Adaptation exercises, through head and eye movements, focus on the adaptation of the vestibulo-ocular reflex. Habituation exercises work by constantly repeating the trigger movements that cause the symptoms and in turn controlling the symptoms. Besides, to help patients improve the use of proprioceptive afferents, static and dynamic exercises are included in the substitution exercises.[28]

MANAGEMENT OF DIFFERENT PATIENT PROFILES

Migraine with and without aura

Treatment of migraine with and without aura is largely similar.[36] For acute treatment of migraine, the first-line medications are over-the-counter analgesics, including nonsteroidal anti-inflammatory drugs (NSAIDs), acetylsalicylic acid, ibuprofen and diclofenac potassium. Triptans are considered a second-line treatment in patients who have inadequate pain relief with over-the-counter analgesics. However, triptans are not used in patients with aura. If patients on triptans show no or insufficient therapeutic response in at least three consecutive attacks, then gepants (ubrogepant or rimegepant) or ditans (lasmiditan) could be used. For the management of nausea

and/or vomiting, antiemetics can be used as an adjunct to oral medications.[18] Patients who are adversely affected on at least 2 days per month are considered for preventive treatment. Besides, overuse of acute medication is also an indication of preventive therapy.[18]

VM with comorbidities like MD and BPPV

No literature suggests the management strategy for patients with VM and MD/BPPV. Patients with overlapping symptoms need to be treated for the causes MD, BPPV and VM separately.

Recurrent episodes of VM

Acute or prophylactic treatment is warranted when attacks of VM are severe and frequent.[1] The mainstay of treatment for acute attacks of VM is vestibular suppressants.[37] Migraine Trust suggests that in patients with VM, a short course of prochlorperazine may be beneficial as a potential vestibular sedative.[38]

VM in children

Drugs found to be efficacious in the management of pediatric VM include tricyclic antidepressants, cyproheptadine, topiramate, triptans and gabapentin.[10] Besides, anti-dopaminergic agents like metoclopramide and prochlorperazine are widely used for the treatment of nausea and vomiting associated with migraine attack. Prochlorperazine is efficacious in aborting migraine attacks and has demonstrated a higher response rate than metoclopramide. It has also shown improvement in the symptoms of VM in children.[10,39] Therefore, it can be used in children above 2 years of age or more than 10 kg weight at a dose of 2.5 mg/kg.[40]

Role of prochlorperazine in the management of VM

Antivertigo agents are efficacious in symptomatic relief of vertigo.[41] Prochlorperazine is a suitable drug for managing acute attacks of vertigo due to its faster onset of action (within 30 mins) and low chances of reoccurrence.[42] It has multimodal action. It acts as a vestibular suppressant by blocking on H1 receptors in the vestibule and brain. It also has antidopaminergic (D2) and anti-serotonergic (5HT3) action. It also blocks the chemoreceptor trigger zone, cholinergic and alpha-adrenergic receptors and thus provides relief from associated symptoms of vertigo.[42]

Efficacy and safety of prochlorperazine in management of acute vertigo: Indian evidence

In dizziness patients, prochlorperazine is reported to be one of the most commonly used drugs (34.1%). Prochlorperazine 5mg TID for seven days is found to significantly reduce number of episodes of dizziness, nausea and vomiting and is reported to be safe.[41,43]

Prochlorperazine for the management of migraine headache

In a systematic review and meta-analysis of 11 studies (n=771), among patients with acute migraine headaches, prochlorperazine was found to be more effective than placebo, metoclopramide, and other active comparators for relieving headaches.[44]

Place of prochlorperazine in evidence-based journals and clinical practice guidelines

American Academy of Family Physicians, Indian Academy of Neurology guidelines in Vertigo, National Health Service, UK; Italian Journal of Medicine, Australian Family Physician and ENT UK recommend using prochlorperazine for the management of acute vertigo. There are currently no approved standardized treatment protocols for VM.[40,45-49] Lifestyle modifications and abortive and prophylactic medications are used to manage VM patients. The treatment strategy for different patient profiles is detailed below.

Migraine with/without aura

Acute treatment remains the same for both migraine with/without aura. Prochlorperazine can play a role as an abortive treatment in migraine with/without aura. If prochlorperazine is contraindicated, then the second abortive option includes a combination of cinnarizine and dimenhydrinate. NSAIDS should be given for managing migraine headaches. Patients with migraine with aura are potential candidates for prophylactic treatment. Prophylaxis treatment should be considered in patients experiencing attacks significantly affecting activities of daily living despite acute treatment, ≥3 debilitating episodes per month, and contraindication to or failure of acute treatment modalities.

Vertigo preceding migraine

For acute episodes, prochlorperazine is the preferred drug for control of vertigo and its associated symptoms at a dose of 5 mg BID/TID for five to seven days.

VM with MD

Prochlorperazine can be used as an abortive treatment. For treating headache, naproxen and triptans can be prescribed. Diuretics like acetazolamide 125-250 mg OD and betahistine 48 mg OD can be used as a prophylactic treatment.

VM with BPPV

In VM patients with BPPV, maneuvers are the mainstay of treatment for BPPV. Post-maneuver patients can be prescribed prochlorperazine for 2-3 days till symptoms subside. Prophylactic therapy can be given in these patients if symptoms persist.

Recurrent VM

Prochlorperazine can be used as for management of recurrent VM along with vestibular rehabilitation.

VM in children

Prochlorperazine 2.5 mg/kg can be given in children above 10 kg or more than 2 years of age as an abortive treatment in VM.

CONCLUSION

This review summarizes the epidemiology and diagnosis of VM in Indian clinical settings and highlights the current gaps in the diagnosis and treatment of VM, along with treatment strategies for different patient profiles. Experts opined that about half of the patients in vertigo clinics are diagnosed with VM, with a higher prevalence in people aged 30-50 years with BPPV and MD as the common comorbidities associated with VM. While in children, VM is common in age groups 12-14 years. Detailed clinical history is important for diagnosis of VM, which is mostly by exclusion. The management of VM includes dietary and lifestyle modification along with abortive and prophylactic treatment. As an abortive treatment in the management of various patient profiles of VM, oral prochlorperazine 5 mg BID/TID for 5-7 days, can play a significant role in providing relief. For prophylaxis, calcium channel blockers, propranolol and triptans can be used.

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