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Vestibular migraine- a dilemma no more

Renu Rajguru[1]*, Inder Deep Singh[2], Abhishek Gupta[1]

[1]Department of ENT, Armed Forces Medical College, Pune, Maharashtra, India
[2]Department of ENT, Command Hospital (Southern Command), Pune, Maharashtra, India

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***Correspondence:**
Dr. Renu Rajguru,
E-mail: renurajguru@yahoo.com

ABSTRACT

Background: Vestibular migraine (VM) is an increasingly recognized cause of episodic vertigo. However, the pathophysiology of VM is still a matter of speculation and it is not known to what extent the dysfunction is located in the central or peripheral vestibular system. Though in its earlier version International Headache Society recognized only adult onset VM in the setting of basilar migraine, but in its latest 3rd edition beta version in consensus with Barany Society (2013), VM is included in the International Headache Society classification of migraine in appendix 1. It does not figure in the main list because it is yet to be validated by scientific research.
Methods: The purpose of this study is to record and describe the spectrum of clinical findings of VM patients, to study the vestibular system and find out the site of vestibular dysfunction. In this study we studied 20 patients with acute VM in the symptomatic and asymptomatic phase.
Results: Abnormal findings in the vestibular work up were present in all 20 VM patients (100%). Central pathology in the vestibular system was seen in 10 patients (50%), 6 patients had peripheral vestibular pathology (30%), and in 4 patients (20%) the site of vestibular dysfunction was indeterminate as they had features of both central and peripheral dysfunction and the exact site of dysfunction could not be determined with certainty.
Conclusions: Acute VM should be considered in the differential diagnosis of vertigo. It can present both as a central and a peripheral vestibular disorder. However there are no definitive signs to pin-point the diagnosis. A careful clinical history combined with clinical findings and exclusion of other causes of vertigo, is fundamental for assessing the profile of patients with vestibular migraine.

Keywords: Vestibular migraine, Migraine, Clinical findings, Diagnosis

INTRODUCTION

Vestibular migraine (VM) refers to a disorder in which vestibular symptoms are an integral part of migraine symptomatology. Though in its earlier version International Headache Society recognized only adult onset Vestibular migraine in the setting of basilar migraine, but in its latest 3rd edition beta version in consensus with Barany Society (2013), VM is included in the International Headache Society classification of migraine in Appendix 1.[1-3] Presently VM is only in appendix and not in the main body of classification because it is novel entity that is not sufficiently validated by research conducted so far. Though it is believed to be real but requires better scientific evidence before it can be formally accepted, and with valid research to back it up, it may move into the main body of classification at the next revision. The current article is a step in this direction.

The diagnostic criteria of VM and probable VM given by "international classification of headache disorders (ICHD) in consensus with Barany Society in 2013" is shown in Table 1.

Table 1: Diagnostic criteria for vestibular migraine.[3]

	1. Vestibular migraine
A.	At least 5 episodes with vestibular symptoms of moderate or severe intensity lasting 5 min to 72 h
B.	Current or previous history of migraine with or without aura according to ICHD (1.1, 1.2)
C.	One or more migraine features with at least 50% of the vestibular episodes • Headache with at least two of the following characteristics: one-sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity • Photophobia and phonophobia • Visual aura
D.	Not better accounted for by another vestibular or ICHD diagnosis
	2. Probable vestibular migraine
A.	At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 h
B.	Only one of the Criteria B and C for vestibular migraine is fulfilled (migraine history or migraine features during the episode)
C.	Not better accounted for by another vestibular or ICHD diagnosis

Vestibular symptoms, as defined by the Barany Society's classification of vestibular symptoms and qualifying for a diagnosis of VM, include the following:

(a) Spontaneous vertigo including internal vertigo (a false sensation of self-motion) and external vertigo (a false sensation that the visual surround is spinning or flowing), (b) positional vertigo, occurring after a change of head position, (c) visually induced vertigo, triggered by a complex or large moving visual stimulus, (d) head motion-induced vertigo, occurring during head motion, (e) head motion-induced dizziness with nausea. Dizziness is characterized by a sensation of disturbed spatial orientation. Other forms of dizziness are currently not included in the classification of VM. One symptom is sufficient during a single episode, though different symptoms may occur during different episodes. Also associated symptoms may occur before, during, or after the vestibular symptoms. The purpose of this study is to record and describe the spectrum of clinical findings of VM patients in symptomatic and asymptomatic period and find out the site of vestibular dysfunction.

METHODS

The present study was an analytical cross sectional and observational study done over a period of two years (March 2011 to March 2013) at a tertiary care centre at Bangalore, Karnataka. A total of 20 patients were studied during the symptomatic phase and asymptomatic phase. Ethical Committee clearance to conduct the study was taken. The patients who were included in the study were 18 years to 60 years old of both sexes who reported with complaints of vertigo, headache or other migrainous symptoms. Patients with history of other neurological, vestibular, opthalmologic and ENT disorders, those with hearing abnormality as measured by audiometry or taking medications that could possibly influence test results were excluded from the study. Pregnant patients were also excluded from the study.

Patients were examined during acute attack as well as during symptom free interval. A thorough clinical examination was performed after taking adequate history and informed written consent, followed by neuro-otologic examination, audiometry, tympanometry, Video-nystagmography (VNG) test battery with the bithermal water caloric test. In some patients MRI was also performed to rule out other causes of vertigo. An opinion of neurologist was sought in selected cases.

Diagnosis of VM was made according to the ICHD-3 Beta criteria by means of a semi-structured face-to-face interview and clinical examination.[3]

RESULTS

Fifteen female and five male patients presented with VM. The presenting complaint in these patients was vertigo of variable intensity and duration. Five patients (25%) reported external vertigo, ten patients (50%) reported head motion intolerance and five patients (25%) reported internal vertigo. Migraine without aura was reported by 14 (70%) and Migraine with aura was reported by 6 patients (30%). Headache was the commonest associated migrainous symptom during the recorded attack in 14 patients (70%). Nausea and vomiting were also common associated complaints found in 12 patients (60%). Phonophobia was reported by 8 patients (40%), 5 patients complained of photophobia (25%) and 6 patients complained of both (30%). History of motion sickness was present in all the patients. Family history was present in 8 patients (40%). Age of onset ranged from 13 years to 37 years and 12 (60%) patients complained that migraine appeared first whereas vestibular symptoms appeared later in the course of the disease. The duration of each episode ranged from 5–60 minutes in 12 patients (60%), 1–24 hours in 6 patients (30%), and 24–72 hours in two patients (10%).

Clinical Examination showed abnormal gait in five patients (25%), spontaneous nystagmus in 6 patients (30%), and positioning nystagmus in 7 patients (35%).

Table 2: Clinical characteristics of 20 VM patients by history.

S. No	Sex	Age (years)	Age at onset	Vestibular complaint	Migraine Tympto m	Associated Symptoms during MV	Duration of each episode	H/O Motion Sickness	Family History
1	F	36	13	EV*	MO§	H ¶, Phono**, N‡‡, V††	20 mints	+	+
2	F	41	20	IV †	MO§	-	5 mints	-	+
3	M	25	24	HMI‡	MO§	H ¶, Photo††, N‡‡	24 Hours	+	-
4	F	42	28	HMI‡	MO§	Phono**, N‡‡	15 mints	+	+
5	F	56	34	HMI‡	MO§	H ¶, Phono**	45 mints	+	-
6	F	43	28	EV	MA ‖	H ¶, Phono**, N‡‡	12 Hours	-	-
7	M	49	34	IV	MA ‖	H ¶, Photo††, Phono**, N‡‡, V ††	8 Hours	+	+
8	F	47	27	HMI‡	MA ‖	Photo††, Phono**, N‡‡	01 Hour	-	-
9	F	29	17	HMI‡	MA ‖	H ¶, Photo††, Phono**, N‡‡, V ††	48 Hours	+	+
10	F	45	30	HMI‡	MA ‖	Photo††	3 Hours	+	+
11	F	39	23	IV	MO§	H ¶, Phono**	30 mints	-	-
12	F	43	25	EV	MO§	Photo††	45 mints	-	-
13	M	35	24	EV	MO§	H ¶, Photo††	1 Hour	+	+
14	F	42	28	HMI‡	MO§	Phono**	15 mints	-	-
15	F	52	35	HMI‡	MO§	H ¶, Phono**	24 Hours	+	+
16	F	41	18	EV	MA ‖	H ¶, Phono**, N‡‡, V††	10 mints	-	-
17	M	49	37	IV	MA ‖	H ¶ , Photo, Phono**	48 Hours	+	+
18	F	44	22	HMI‡	MA ‖	H ¶, Photo††, Phono**, N‡‡	45 mints	+	+
19	M	31	27	HMI‡	MA ‖	H ¶ , Photo, Phono**, N‡‡, V††	3 Hours	+	+
20	F	45	30	HMI ‡	MA ‖	Photo††	24 Hours	+	+

EV* = External vertigo; IV† = External vertigo; HMI‡ = Head motion intolerance, MO§ = Migraine without aura; MA ‖ = Migraine with aura; H ¶ = Headache; Phono** = Phonophobia; Photo†† = Photophobia; N‡‡= Nausea; V††= Vomiting.

Table 3: Clinical characteristics of 20 VM patients by examination during an acute episode.

S.No	Sex	Gait	Nystagmus	TM	PTA & Tympanogram	VNG	Caloric Test	MRI	Site of vestibular dysfunction
1	F	AB*	Spont‡	N†	N	AB*	Bil dec response	N	Central
2	F	N	Posi‡	N	N	AB*	Uni CP§	-	Peripheral
3	M	N	-	N	N	N	N	-	Indeterminate
4	F	AB*	Spont‡	N	N	AB*	N	-	Central
5	F	N	Posi‡	N	N	N	Uni CP§	-	Peripheral
6	F	N	-	N	N	N	N	-	Central
7	M	AB*	Spont‡	N	N	AB*	N	N	Central
8	F	N	Posi‡	N	N	N	Uni CP§	-	Peripheral
9	F	N	-	N	N	AB*	N	-	Central
10	F	AB*	Spont‡	N	N	AB*	N	N	Indeterminate
11	F	N	-	N	N	AB*	Bil inc response	-	Central
12	F	N	Posi‡	N	N	N	Uni CP§	-	Peripheral
13	M	N	Spont‡	N	N	AB*	Uni CP§	N	Indeterminate
14	F	N	-	NI	N	AB*	N	-	Central
15	F	N	Posi‡	N	N	N	N	-	Peripheral
16	F	N	-	N	N	N	N	-	Central
17	M	AB*	Spont‡	N	N	AB*	N	N	Central
18	F	N	Posi ‡	N	N	N	Uni CP§		Peripheral
19	M	N	-	N	N	N	N	-	Central
20	F	N	Posi ‡	N	N	AB*	Bil dec response	-	Indeterminate

AB*=Abnormal; N†= Normal; Spont‡=Spontaneous; Posi‡=Positional; Uni CP§ = Unilateral Canal Paresis.

All patients had normal tympanic membrane, normal audiometry and tympanometry. The VNG test battery showed abnormal findings in 11 patients (55%) with VM, the abnormalities being gaze-evoked nystagmus, spontaneous nystagmus, saccadic pursuit, delayed slow phase velocity. All types of nystagmus were observed (vertical, horizontal, and torsional) in VM patients. 4 (20%) patients showed impaired vestibulo-oculomotor reflexes. Five patients (25%) showed unilateral canal paresis, two patients (10%) had bilateral increased responses whereas one patient had bilateral decreased response in bithermal caloric test (5%).

Central vestibular dysfunction was indicated by purely vertical or torsional spontaneous nystagmus, which worsened or showed <50% decrease of SPV of spontaneous nystagmus with gaze fixation, was multi-directional or direction changing. Peripheral vestibular dysfunction was shown by Direction-fixed, predominantly horizontal spontaneous nystagmus which showed >50% decrease of SPV of spontaneous nystagmus with fixation and absence of signs of CNS involvement. Central pathology in the vestibular system was seen in 10 patients (50%), 6 patients had peripheral vestibular pathology (30%), and in 4 patients (20%) the site of vestibular dysfunction was indeterminate as they had features of both central and peripheral dysfunction. Our data are consistent with the wide variability in the duration of vestibular episodes. Although, the findings in the remaining 4 patients (20%) reflected asymmetry in the vestibular system, yet the exact site of dysfunction could not be determined with certainty.

In the symptom free interval, no vestibular findings were detected in all patients. None of the patients had auditory complaints or neurologic symptoms apart from vertigo and abnormal gait in some patients during their episodes. No neurological deficits were seen in patients with VM in symptomatic and symptom free interval.

DISCUSSION

Clinical examination in all 20 VM patients revealed some vestibular dysfunction during the acute phase, thus confirming the vestibular origin of such disease entity.[4, 5] Acute VM manifested with variable findings suggesting that vestibular system may be involved at any level from the brainstem, cerebellar connections down to the periphery with variable percentage. This heterogeneity of symptoms in VM was reflected in several case series.[5-8] The neural mechanisms of VM appear to be multifactorial, and the variability in the symptoms and clinical findings during and between attacks highlights the interaction of migraine with the vestibular system at various levels.[9]

Various pathophysiologic mechanisms have been proposed for VM, all of which are derived from the presumed pathophysiology of migraine.[10,11] Cortical spreading depression, role of neurotransmitters like serotonin and dopamine, vasospasm of labyrinthine artery, genetic channelopathy with mutations in neuronal Ca2+ channels (CACNA1A gene coding for the α1A subunit), and lower stimulation threshold for crosstalk between trigeminal and vestibular nuclei are some of these mechanisms. The brain and the inner ear may share critical ion channels that are affected in migraine attacks. This is the basis of trigeminovascular hypothesis which explains the peripheral component of VM wherein stimulation of the V nerve ganglion can lead to permeability changes in the vertebrobasilar as well as cochlear vasculature, thereby leading to vertigo, which can be peripheral, central, or mixed.

VM should be differentiated from other conditions like benign paroxysmal positional vertigo, Meniere's disease, and vascular compression of eighth nerve, vertebrobasilar transient ischemic attacks, perilymph fistula, vestibular schwannoma, autoimmune inner ear disease and insufficient compensation of unilateral vestibular loss.[9]

Once the diagnosis is made, the management aims at the treatment of the acute attack and prevention of further attacks by nonpharmacological and pharmacological means.[11-16] The management of acute attack includes a combination of the following:

(a) Vestibular suppressants like prochlorperazine, cinnarizine, flunarizine, promethazine, (b) antiemetics like metoclopromide, domperidone, (c) NSAIDS like ibuprofen, diclofenac, aspirin, paracetamol, (d) benzodiapines like clonazepam, alprazolam, lorazepam, or prazepam, (e) triptans like sumatriptan and zolmitriptan.

In those patients who experience severe, long and frequent attacks of VM, prophylaxis is considered. Also the patient should be explained the exact nature of the disease and this will help alleviate the anxiety which might be associated with the attack.

Prophylaxis usually employs a multifaceted approach which comprises of a combination of the following: (a) identification and avoidance of triggers and environmental stimuli, (b) dietary changes, stress management, sleep improvement, exercise, (c) beta-blockers eg. metoprolol (50-100 mg/day), propranolol 40 mg once or twice a day, (d) calcium antagonists eg. flunarizine (10-20 mg/day), (e) anticonvulsants eg valproic acid (600-1200 mg/day), lamotrigine (50-100 mg/day), topiramate (100 mg/day), which act by raising the threshold for cortical spreading depression, (f) antidepressants eg amitriptyline 50–100 mg.

CONCLUSIONS

With inclusion of VM in the new classification of migraine by ICHD in the Appendix, there is an emerging clinical evidence to support this diagnosis. The relationship between vestibular symptoms and migraine

is commonly observed but unfortunately, there is not a single diagnostic test specific for VM. So more often than not, due to lack of awareness of this condition, the diagnosis of VM is often missed and the patient continues to be erroneously managed as BPPV, Meniere's Disease etc. But the typical history, presence of peripheral and central vestibular system abnormalities during an acute attack and their absence during the asymptomatic interval are suggestive towards this diagnosis. However, further research, involving multidisciplinary and multicenteric approaches is needed to better understand the pathophysiologic mechanisms behind VM as it is common and a clinically relevant disorder. This will result in the earlier diagnosis and improved treatment of patients with VM.

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REFERENCES

1. Neuhauser HK. Epidemiology of vertigo. Curr Opin Neurol. 2007;20(1):40-6.
2. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders, 2nd edition. Cephalalgia. 2004;24(1):9-160.
3. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33(9):629–808.
4. Büttner U, Helmchen C, Brandt T. Diagnostic criteria for central versus peripheral positioning nystagmus and vertigo: a review. Acta Otolaryngol. 1999;119: 1-5.
5. Dieterich M, Brandt T. Episodic vertigo related to migraine (90 cases): vestibular migraine? J Neurol. 1999;246:883-92.
6. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain. 1984;107:1123-42.
7. Neuhauser H, Leopold M, von Brevern M, Lempert T. The interrelations of migraine, vertigo and Vestibular migraine. Neurology. 2001;56:684-6.
8. Cutrer F, Baloh RW. Migraine-associated dizziness. Headache. 1992;32:300-4.
9. Neuhauser H, Lempert T. Vertigo and dizziness related to migraine: a diagnostic challenge. Cephalalgia. 2004;24:83-91.
10. Espinosa-Sanchez JM, Lopez-Escamez JA. New Insights into Pathophysiology of Vestibular Migraine. Front Neurol. 2015;6:12.
11. Bikhazi P, Jackson C, Ruckenstein MJ. Efficacy of antimigrainous therapy in the treatment of migraine-associated dizziness. Am J Otol. 1997;18:350-4.
12. Bisdorff AR. Treatment of migraine-related vertigo with lamotrigine: an observational study. Bull Soc Sci Med Grand Duche Luxemb. 2004;2:103-8
13. Carmona S, Settecase S. Use of topiramate (Topamax) in a subgroup of mirgraine-vertigo patients with auditory symptoms.Ann NY Acad Sci. 2005;1039:517–20.
14. Johnson GD (1998) Medical management of migraine-related dizziness and vertigo. Laryngoscope. 1998;108:1–28.
15. Maione A. Migraine-related vertigo: diagnostic criteria and prophylactic treatment. Laryngoscope. 2006; 116(10):1782-86.
16. Neuhauser H, Radtke A, Von Brevern M, Lempert T. Zolmitriptan for treatment of migrainous vertigo: a pilot randomized placebo-controlled trial. Neurology. 2003;60:882-3.

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